WBUHS

(2006-2010)

MS- PAPER – I -IV

Guide for Answering theory questions in MS Surgery

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MS (General Surgery) Examination, 2010

PAPER I

Time Allowed: 3 Hours **Full Marks: 100**

Attempt all questions

1. Discuss the micro- and macroanatomy of lymph nodes. Mention the imaging criteria for metastatic lymph nodes. Discuss the role of sentinel node biopsy in common malignancies. 6+4+10

2. Discuss Calcium metabolism in health and surgical disease. 8+12.

3. Write short notes on the following : 6 x 5

a) **Systemic Inflammatory Response Syndrome.**
b) **Allograft rejection.**
c) **Postoperative Ventillatory Support.**
d) **Enteral Nutrition.**
e) **Tumour Markers and their role in Surgery.**

4. Write brief answers on the following: $7^1/_2$

a) **Pathology of Vascular Malformations in childhood.**
b) **Role of blood component in Surgery.**
c) **Cervical rib and its effects.**
d) **Tumours of Endocrine Pancreas.**

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1. **Discuss the micro- and macroanatomy of lymph nodes. Mention the imaging criteria for metastatic lymph nodes. Discuss the role of sentinel node biopsy in common malignancies. 6+4+10.**

Answer. Lymph nodes:

- The lymph nodes are small, round or ovoid bodies placed along the course of lymphatic vessels.
- They are surrounded by a fibrous capsule from which trabeculae penetrate into the nodes.
- The node can be differentiated into an **outer cortex** and **an inner medulla**.
- In the cortex are accumulations of lymphocyte primary lymphoid follicles) within which germinal centres (secondary follicles) develop during antigenic stimulation.

The follicles contain - proliferating lymphocytes
-dendritic macrophages which capture and process the antigen.

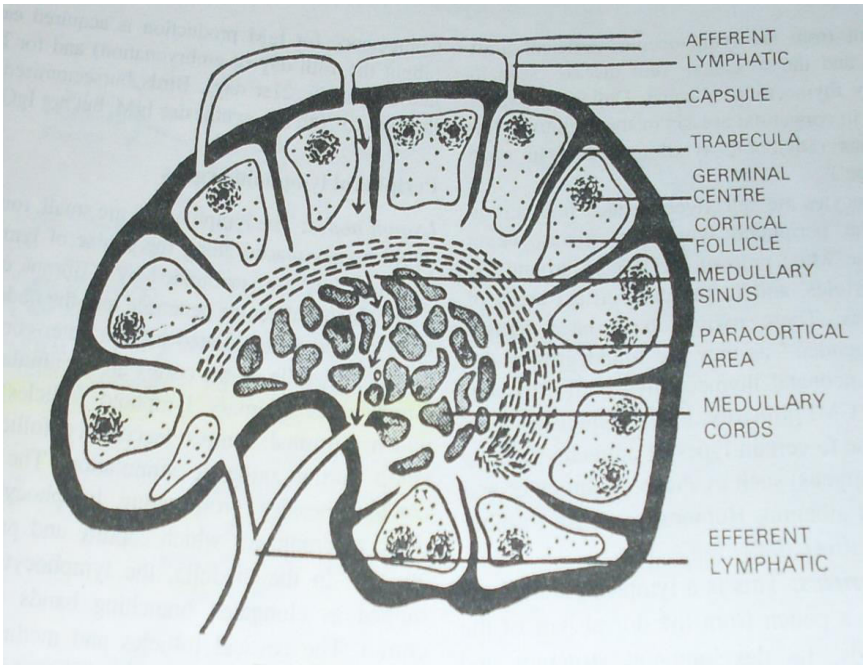
- In the medulla, the lymphocytes are arranged as elongated branching bands (medullary cords).

The cortical follicles and medullary cords contain B lymphocytes and constitute the bursa dependent areas.

- Between the cortical follicles and medullary cords, theres is a broad, ill defined intermediate zone (paracortical area) which contains T lymphocytes and constitutes the thymus dependent area.

Functions of Lymph Node:

- Lymph nodes act as a filter for the lymph, each group of nodes draining a specific part of the body.
- They phagocytose foreign materials including microorganisms.
- They help the proliferation and circulation of T and B cells.
- They enlarge following local antigen stimulation**.**



Diagramatic section of a lymph node.

Imaging criteria for metastatic lymph nodes:

<u>USG criteria:</u>

- Size of the lymph node greater than 1.0 cm,
- Necrosis,
- Shape of a lymph node is determined by measurements taken vertically (maximum longitudinal or minimal axial diameter) L/T ratio greater than1,
- Eccentric enlargement of nodal cortex

<u>CT and MRI criteria:</u>

• A discrete mass greater than 1.0 cm; • An ill-defined mass in a lymph node area; • Multiple nodes of 6–15 mm; • Obliteration of tissue planes around vessels in a nonirradiated neck. • A nodal mass with central low density is specifically indicative of tumor necrosis. • A discrete mass greater than 1.0 cm; • An ill-defined mass in a lymph node area; • Multiple nodes of 6–15 mm; • Obliteration of tissue planes around vessels in a nonirradiated neck.	• A nodal mass with central low density is specifically indicative of tumor necrosis. • If neoplastic process progresses, lymph node gains a spheric appearance due to an increase in node size. • Original node structure shows defeats due to tumor infiltration, desmoplastic reaction and necrosis. • In this stage either a decrease or an increase can be seen in vascularisation under influence of angiogenic factors. neovascularisation.

- The assessment of lymph nodes using these modalities relies on lymph node anatomy rather than function and physiology.
- On cross-sectional imaging, a normal lymph node usually measures <1 cm in size, has a smooth and well-defined border, and shows uniform, homogeneous density or signal intensity.
- Most benign nodes have a central fatty hilum, which has a distinctive feature on CT and MRI.

Role of sentinel lymph node biopsy in different malignancies:

- **Carcinoma breast:**

- Sentinel lymph node biopsy is primarily used in women with early breast cancers (T1 and T2, N0).
- It also is accurate for T3 N0 cancers, but nearly 75% of these women will have nonpalpable axillary lymph node metastases.
- In women undergoing neoadjuvant chemotherapy to permit conservation surgery, sentinel lymph node biopsy may be used.

Contraindications to the procedure include

- Palpable lymphadenopathy,
- Prior axillary surgery, chemotherapy or radiation therapy, and
- Multifocal breast cancers.

- The combination of intraoperative gamma probe detection of radioactive colloid and intraoperative visualization of isosulfan blue dye (Lymphazurin) is more accurate than the use of either agent alone.
- On the day prior to surgery, or on the morning of surgery, the radioactive colloid is injected. Using a tuberculin syringe and a 25-gauge needle, 0.5 mCi of 0.2-micron technetium-99 sulfur colloid in a volume of 0.2 to 0.5 mL is injected (three to four separate injections) at the cancer site or subdermally.
- Subdermal injections are given in proximity to the cancer site or subareolar.
- Subsequently, in the operating room, 4 mL of isosulfan blue dye (Lymphazurin) is injected in a similar fashion, but with an additional 1 mL injected between the cancer site and the overlying skin.
- For nonpalpable cancers, the injection is guided by either intraoperative ultrasound or by a localization wire that is placed preoperatively under ultrasound or stereotactic guidance.
- It is helpful for the radiologist to mark the skin overlying the breast cancer at the time of needle localization using an indelible marker.
- In women who have undergone previous excisional biopsy, the injections are made around the biopsy cavity but not into it.
- Women are told preoperatively that the isosulfan blue dye injection will impart a change to the color of their urine and that there is a very small risk of allergic reaction to the dye (1 in 10,000).
- Anaphylactic reactions have been documented. The use of radioactive colloid is safe and radiation exposure is very low.

- By using a combination of isotope lymphatic mapping, an intraoperative hand-held gamma probe, and intraoperative injection of blue dye, the SLN could be identified in more than 95% of cases in the groin and axilla, with identification in the head/neck region being slightly lower (85%).

- A hand-held gamma counter is then employed transcutaneously to identify the location of the sentinel lymph node.
- The gamma counter is employed to pinpoint the location of the sentinel lymph node.
- As the dissection continues, the signal from the probe increases in intensity as the sentinel lymph node is approached.
- The sentinel lymph node also is identified by visualization of isosulfan blue dye in the afferent lymph vessel and in the lymph node itself.
- The lowest false-negative rates for sentinel lymph node biopsy have been obtained when all blue lymph nodes and all lymph nodes with radiation counts greater than 10% of the 10-second ex vivo count of the sentinel lymph node are harvested (10% rule).
- . This procedure is repeated until residual radioactivity in the surgical bed is less that 10% of the 10-second ex vivo count of the most radioactive sentinel lymph node. ii.

- **Malignant melanoma: Management of Regional Lymph Nodes**:

- After WLE of the primary tumor, the most common sites of first recurrence are regional (lymph nodes, in-transit metastases, and local recurrences).
- Nodal metastases generally appear in the basin or basins draining from the primary site. This is a predictable pattern for extremity melanomas; however, truncal and head and neck melanomas may drain to more than one site.
- Lines of drainage for truncal melanomas are divided by the midline and the line of Sappey, which extends from the umbilicus across the iliac crest and around to the spine at the level of L2.
- This sequence of recurrences led surgeons to conclude that resection of nodal basins containing occult metastases could provide an increase in survival. This procedure, termed elective lymph node dissection (ELND), was commonly practiced.

- As prognostic factors became better understood, it was postulated that patients with thin tumors (<1 mm in thickness) would have a low risk of metastases at any site and patients with thick tumors (>4 mm in thickness) would have a high risk of distant as well as regional metastases. In contrast, patients with intermediate-thickness melanoma (1-4 mm) would have an elevated risk for nodal metastases without a high risk for distant disease.

Development of the SLN concept ended one debate over ELND, changed clinical management, and opened a new series of questions about the tumor biology of melanoma.

- There was great anatomic variation resulting in drainage to multiple or uncommon sites.
- Detailed pathologic analysis of the sentinel nodes via step sections enabled detection of micrometastases that could be missed by standard techniques.
- The probability of finding a positive sentinel node can be predicted by using a nomogram derived from multifactorial analysis.
- In most cases a positive sentinel node was the only positive node.
- No prognostic factors were found that accurately identified a subpopulation of SLN-positive patients at zero risk of harboring other positive nodes.

▪ **Carcinoma penis:** Not used nowadays. Biopsy of the lesion is taken to confirm the diagnosis.

2. Discuss Calcium metabolism in health and surgical disease. 8+12.
Answer.

Distribution (mmol/L) of Calcium in Normal Human Plasma.		
Total diffusible		***1.34***
Ionized (Ca^{2+})	1.18	
Complexed to HCO_3^-, citrate, etc	0.16	
Total nondiffusible (protein-bound)		***1.16***
Bound to albumin	0.92	
Bound to globulin	0.24	
Total plasma calcium		***2.50***

- The normal plasma level of intact PTH is 10–55 pg/mL. The half-life of PTH is approximately 10 minutes, and the secreted polypeptide is rapidly cleaved by the Kupffer cells in the liver into midregion and carboxyl terminal fragments that are probably biologically inactive. PTH and these fragments are then cleared by the kidneys. It is interesting in this regard that a synthetic polypeptide containing the amino terminal 34 amino acid residues of PTH has all the known biologic effects of the full molecule.
- PTH acts directly on bone to increase bone resorption and mobilize Ca^{2+}. In addition to increasing the plasma Ca^{2+} and depressing the plasma phosphate, PTH increases phosphate excretion in the urine. This **phosphaturic action** is due to a decrease in reabsorption of phosphate in the proximal tubules. PTH also increases reabsorption of Ca^{2+} in the distal tubules, although Ca^{2+} excretion is often increased in hyperparathyroidism because the increase in the amount filtered overwhelms the effect

on reabsorption. PTH also increases the formation of 1,25-dihydroxycholecalciferol, and this increases Ca^{2+} absorption from the intestine.

On a longer timescale, PTH stimulates both osteoblasts and osteoclasts. The net effect varies, but with mildly elevated plasma PTH levels it is usually anabolic.

Actions of Major Calcium-Regulating Hormones

	BONE	KIDNEY	INTESTINE
Parathyroid hormone	Stimulates resorption of calcium and phosphate	Stimulates resorption of calcium and conversion of $25(OH)D_3$; inhibits resorption of phosphate and bicarbonate	No direct effects
Vitamin D	Stimulates transport of calcium	Inhibits resorption of calcium	Stimulates calcium and phosphate absorption
Calcitonin	Inhibits resorption of calcium and phosphate	Inhibits resorption of calcium and phosphate	No direct effects

Hypoparathyroidism: Hypoparathyroidism is an endocrine disorder in which hypocalcemia and hyperphosphatemia are the result of a deficiency in PTH secretion or action.

Causes of hypoparathyroidism are:

- Iatrogenic: damage to the parathyroid glands during thyroidectomy (most common), but it also can occur after parathyroid exploration.
- It can occur as part of a multiglandular endocrine deficiency syndrome (type 1) characterized most commonly by hypoparathyroidism, adrenal insufficiency, and mucocutaneous candidiasis.
- Disorders in which there is abnormal or absent formation of the parathyroid glands are associated with hypocalcemia. For example, DiGeorge's syndrome occurs when the third and fourth branchial pouches develop abnormally.
- Parathyroid gland function can be impaired by infiltrative involvement of the glands in diseases such as hemochromatosis, Wilson's disease, sarcoidosis, tuberculosis, or amyloidosis.
- Exposure to external radiation or very large doses of ^{131}I for Graves' disease or well-differentiated thyroid cancer has rarely been associated with hypocalcemia.
- Abnormalities in magnesium levels are associated with a reversible abnormality of PTH secretion.
- Parathyroid gland function can be impaired by infiltrative involvement of the glands in diseases such as hemochromatosis, Wilson's disease, sarcoidosis, tuberculosis, or amyloidosis. Exposure to external radiation or very large doses of ^{131}I for Graves' disease or well-differentiated thyroid cancer has rarely been associated with hypocalcemia. Finally, abnormalities in magnesium levels are associated with a reversible abnormality of PTH secretion.

The signs and symptoms of hypocalcemia are caused by neuromuscular excitability from reduced plasma ionized calcium. Early manifestations include perioral numbness and tingling in the fingers. Anxiety or confusion can follow, and it is important for the surgical team to reassure patients early to reduce psychiatric and neurocognitive symptoms. Anxiety often results in hyperventilation, which can then lead to respiratory alkalosis and a

further reduction in the serum calcium level. Tetany, marked by carpopedal spasm, convulsions, or laryngospasm (or any combination of the three), may follow and can be fatal. Physical examination includes testing for a Chvostek sign, which is contraction of the facial muscles after tapping on the facial nerve anterior to the ear. Approximately 15% of normal individuals have a positive Chvostek sign.

Causes of Hypercalcemia

Disease	**Confirmatory Test**
Primary Hyperparathyroidism	
Sporadic	Intact parathyroid hormone
Familial	
Malignancy	
Humoral/multiple myeloma	Parathyroid hormone-related protein
Osteolytic	
Endocrinopathy	
Hyperthyroidism	Thyroid stimulating hormone
Addison's disease	Adrenocorticotropic hormone stimulation test
Drug-related	
Vitamin D intoxication	25-hydroxyvitamin D_3
Thiazide diuretics	
Granulomatous Disease	
Sarcoidosis	Angiotensin-converting enzyme 1,25-dihydroxyvitamin D_3
Familial hypocalciuric hypercalcemia	Urine calcium: creatinine ratio

Hyperparathyroidism:

- Primary hyperparathyroidism (PHPT) - is a syndrome of inappropriate secretion of parathyroid hormone (PTH) by one or more abnormal parathyroid glands. (PHPT) is a syndrome of inappropriate secretion of parathyroid hormone (PTH) by one or more abnormal parathyroid glands. Most cases of PHPT occur sporadically, with a female preponderance of nearly 4 to 1. Familial syndromes of PHPT account for relatively few cases. These syndromes include multiple endocrine neoplasia, types 1 and 2; familial isolated hyperparathyroidism; and hereditary hyperparathyroidism jaw tumor syndrome.

A single parathyroid adenoma accounts for between 85% and 90% of cases of PHPT. Multiple gland disease, in the form of either multiple adenomas or hyperplasia of all parathyroid glands, occurs in the remaining 10% to 15%. Parathyroid carcinoma is a rare (less than 1%) cause of PHPT. The molecular mechanisms underlying most sporadic cases of PHPT remain poorly understood.

Clinical presentation:

- The parathyroid glands function to preserve serum calcium in a tightly controlled range (8.5 to 10.2 mg/dl).
- Most patients present with hypercalcemia and few, if any, symptoms.
- Most patients come to clinical attention by identification of hypercalcemia on routine chemistry panels.
- Increasingly, otherwise asymptomatic patients are being identified during evaluation of osteoporosis.
- Classic manifestations of moderate PHPT include nephrolithiasis, osteoporosis, arthralgia, and myopathy.
- Subtle manifestations of PHPT may include hypertension, neuropsychiatric disorders, dyspepsia, and constipation.
- Severe manifestations of PHPT occur uncommonly and include osteitis fibrosa cystica, osteoclastomas (Brown tumors), and nephrocalcinosis.
- Severe PHPT may still be seen today in populations not routinely screened with the multichannel serum chemistries.
- Familial cases of PHPT are uncommon but must be discerned through careful determination of family history, in order to either exclude the possibility of familial hypocalciuric hypercalcemia (FHH) or alert the surgeon to the possibility of multiple gland disease.

- Secondary hyperparathyroidism (2HPT) is the result of chronic overstimulation leading to hypersecretion and hyperplasia of intrinsically normal parathyroid glands in response to derangements in mineral metabolism. This phenomenon occurs to some degree in nearly all patients with chronic renal failure (CRF) but can also be seen in patients with celiac disease, malabsorption, and vitamin D resistance. For practical purposes virtually all operative cases of 2HPT are associated with end-stage renal failure (ESRF). Prior to current medical therapeutic regimens, approximately 5% of dialysis patients required parathyroidectomy, but recent studies show that only about 1% of ESRF patients will need parathyroidectomy.
- Tertiary hyperparathyroidism (3HPT) occurs in the setting of longstanding 2HPT in which long-standing parathyroid hyperplasia leads to autonomous function despite correction of the underlying cause, usually by means of a kidney transplant. It is manifest as either failure of resolution of hyperparathyroidism after renal transplantation or refractory hypercalcemia despite aggressive medical management in a patient with previously medically controlled 2HPT.
- From the perspective of the surgeon managing the care of these patients, the distinction between 2HPT and 3HPT is not critical because an understanding of the underlying mineral disorders causing the disease applies to both conditions. Unlike the multiple gland abnormalities seen in 2HPT, some have reported that the autonomous function seen in 3HPT can be related to a single abnormal gland.

3. Write short notes on the following : 6 x 5

a) Systemic Inflammatory Response Syndrome.
b) Allograft rejection.
c) Postoperative Ventillatory Support.
d) Enteral Nutrition.
e) Tumour Markers and their role in Surgery.

Answer.
(a)Systemic Inflammatory Response Syndrome.
Answer.

Criteria for Four Categories of the Systemic Inflammatory Response Syndrome
Systemic Inflammatory Response Syndrome (SIRS) Two or more of the following: ▪ Temperature (core) >38°C or <36°C ▪ Heart rate >90 beats/min ▪ Respiratory rate of >20 breaths/min for patients spontaneously ventilating or a $Paco_2$ <32 mm Hg ▪ White blood cell count >12,000 cells/mm^3 or <4000 cells/mm^3 or >10% immature (band) cells in the peripheral blood smear
Sepsis Same criteria as for SIRS but with a clearly established focus of infection
Severe Sepsis Sepsis associated with organ dysfunction and hypoperfusion Indicators of hypoperfusion: ▪ Systolic blood pressure <90 mm Hg ▪ >40 mm Hg fall from normal systolic blood pressure ▪ Lacticacidemia ▪ Oliguria ▪ Acute mental status changes
Septic Shock Patients with severe sepsis who ▪ Are not responsive to intravenous fluid infusion for resuscitation ▪ Require inotropic or vasopressor agents to maintain systolic blood pressure

Protocol for Resuscitation of Adult Hypotensive Patients With Suspected Sepsis

- Culture relevant body fluids, including blood.
- Infuse a balanced electrolyte solution of 500 mL/15 min. Monitor the systolic blood pressure response.
- Insert a central venous or pulmonary artery catheter.

- If after a 500-mL bolus of saline the patient remains hypotensive and CVP is <8-12 mm Hg or PAWP is <8-12 mm Hg, infuse another 500-mL bolus of fluid; repeat as needed.

- If CVP is >15 or PAWP is 15-20 and the patient remains hypotensive (<65 mm Hg), start an infusion of the inotropedobutamine or dopamine. The goal is a mean systemic pressure >65 mm Hg and a pulse rate <120 beats/min.

- Determine the cardiac index and systemic vascular resistance.

- If after infusion of fluid and inotropes SVR is <600, infuse avasopressor—either norepinephrine or vasopressin—to increase SVR.

- Monitor mixed venous oxygen saturation and urine output as an indication that therapeutic interventions have improved perfusion.
- CVP, central venous pressure; PAWP, pulmonary artery wedge pressure; SVR, systemic vascular resistance

(b) Allograft rejection.

Answer. Allograft: an organ or tissue transplanted from one individual to another. Allografts trigger a graft rejection response because of allelic differences at polymorphic genes that give rise to histocompatibility antigens(transplant antigens), of which ABO blood group antigens and human leucocyte antigens (HLA) are the most important.

	Class I	Class II
HLA loci	HLA – A,-B& -C	HLA-DR,-DP & -DQ.
Structure	Heavy chain & B2microglobulin	alpha & beta - chain
Distribution	All nucleated cells	B cells, dendritic cells, macrophages.

There are several types allograft of rejection:

- Hyperacute rejection (HAR) is immediate graft rejection is mediated by preformed antibodies that bind to antigens of ABO blood groups, non-self HLA, and xenografts that are similar to antigens found on bacteria and viruses. It results in immediate tissue oedema, haemorrhage, and thrombosis.
- Acute rejection is a function of both the innate and the adaptive immune system, triggered by the recognition of foreign MHC and foreign peptides presented by self-

MHC, by T cells, and results in tissue destruction over days to many months(during the first 6 months) after transplant. It is reversible.

Diagnosis: Clinical features include low grade fever, malaise, reduced exercise tolerance, pericardial rub, supraventricular arrhythmias, low cardiac output, and signs of congestive cardiac failure. Blood tests reveal a lymphocytosis. As all patients are on immunosuppression, symptoms may be minimal until rejection is quite advanced, so routine surveillance is undertaken. Haemodynamic measurements with a PA catheter may be helpful, but the gold standard diagnostic tool is RV endomyocardial biopsy: usually via a right internal jugular percutaneous approach. This is carried out every 7-10 days for a month, tapering to 2-6 month intervals. Lymphocyte infiltration and myocyte necrosis is used to grade the severity of cellular rejection. Biopsy negative cardiac dysfunction raises the possibility of accelerated coronary artery disease or vascular rejection. **Management:**

- Asymptomatic mild rejection is monitored but not routinely treated.
- Myocyte necrosis is a definite indication for increased immunosuppression, but intermediate grades are treated according to clinical context.
- Up to 3 days IV methyprednisolone 1000mg/day for acute rejection within 3 months of transplant, or for severe episodes.
- 100mg/day PO methylprednisolone on a reducing dose over several weeks for all other episodes.
- Repeat endomyocardial biopsy after 7-10 days: repeat steroid course if no improvement, or give rescue therapy if haemodynamically unstable.
- Rescue protocols include methylprednisolone + OKT3, or methotrexate.
- Total lymphoid irradiation and photophoresis have been used in refractory rejection.
- Retransplantation is no longer performed in many centres for refractory rejection as results are extremely poor.

1. Chronic rejection occurs after first 6 months. Non-immune factors may contribute to pathogenesis. It is the most common cause of graft rejection and is characterized by myo-intimal proliferation in great arteries leading to ischaemia and fibrosis. Chronic rejection manifests as allograft coronary artery disease, detectable in over half of cardiac transplant patients within 5y of transplantation, and within months in a few patients. Other processes that have been associated with allograft coronary disease include increased donor age, hyperlipidaemia, and CMV infection. Ischaemia is frequently silent as the heart is dennervated. The clinical picture includes ventricular arrhythmias, congestive cardiac failure, and sudden death. Coronary angiography is the gold standard investigation but may underestimate the extent of the disease as the morphology is diffuse, smooth, intimal proliferation. There is no effective treatment apart from retransplantation. Prophylaxis centres on the same risk reduction strategies used in native ischaemic heart disease.

Immunosuppressive agents:

- Corticosteroids:Corticosteroids inhibit the immune response at many levels. They decrease production of interferon and interleukins that would normally cause upregulation of the lymphocyte response; and reduce macrophage function
- Cyclosporin (CYA):Cyclosporin is a calcineurine inhibitor: it inhibits the production of IL-2 by T helper cells, selectively reducing the cytotoxic T-cell response.

- Tacrolimus (FK506):Tacrolimus is also a calcineurin inhibitor with a similar profile
- Mycophenolate mofetil (MMF): MMF inhibits purine synthesis in lymphocytes, reducing clonal expansion and lymphocyte count.
- Azathioprine (AZA):Azathrioprine causes dose-related bone marrow suppression by suppression of purine synthesis.
- Sirolimus (rapamycin):Sirolimus stops IL-2 triggering clonal expansion of T lymphocytes
- Daclizumub, basiliximab:IL-2 receptor blockers that prevent clonal expansion of T cells
- OKT3: OKT3 is a monoclonal antibody produced in mice that binds the CD3 receptor site on cytotoxic T cells, preventing antigen recognition and clonal expansion
- Polyclonal antibodies, e.g. antithymocyte globulin:These are produced by animals after immunization with HLA: they attach to most circulating lymphocytes effecting a reduction in cell counts to less than 10% of normal.
- Simvastatin:Simvastatin has a moderate protective effect against chronic rejection: it suppresses T-cell function.

(c) Postoperative Ventillatory Support.
Answer. Mechanical ventilation is often utilized and required in patients with postoperative respiratory failure.

The indications for tracheal intubation include

- Inadequate oxygenation or ventilation,
- Inadequate airway patency or protection,
- Excessive secretions,
- Increased work of breathing, and
- Impaired physiologic reserve.

Early intubation should be the goal to avoid airway and hemodynamic compromise, preferably in the presence of someone specifically trained in routine and difficult airway management. Equipment for managing difficult airways must be readily available, and placement should always be confirmed with an end-tidal carbon dioxide detector.

- <u>Modes of mechanical ventilation</u>
- Volume-limited modes
  - Assist-control (A/C) ventilation delivers a preset tidal volume at a set rate. As the machine senses each inspiratory effort by the patient, it delivers the set tidal volume. If the patient's respiratory rate is below the machine's set rate, ventilator-initiated breaths are delivered to make up the difference between the set rate and the patient's. A/C ventilation minimizes the work of breathing because the ventilator assists all breaths (hence, the term full support); however, for this reason, this mode is uncomfortable if the patient's breaths are dyssynchronous with those delivered by the ventilator. Respiratory alkalosis from hyperventilation may develop in agitated patients.
  - Intermittent mandatory ventilation (IMV), like A/C ventilation, delivers a preset tidal volume at a set rate. IMV does not assist spontaneous respiratory efforts; it is commonly used with pressure support.
- Pressure-limited modes

    - Pressure-support ventilation delivers a preset inspiratory pressure but at no set rate. Constant inspiratory pressure continues until the inspiratory flow of gas falls below a predetermined level and the exhalation valve opens. Thus, tidal volumes are generated only when the patient is breathing spontaneously. This allows the patient to maintain control of inspiratory and expiratory time and thus tidal volume; as a result, this mode is the most comfortable for spontaneously breathing patients. The disadvantages of pressure-support ventilation are that (1) all ventilation depends on patient effort and (2) sudden increases in airway resistance decrease tidal volumes. Small amounts (5 to 8 cm H_2O) of pressure-support ventilation are used routinely to overcome the resistance to air flow caused by the ET tube and the inspiratory demand valves of the ventilator.
    - Pressure-control ventilation delivers a preset inspiratory pressure (as opposed to tidal volume) at a set rate. This mode is used in patients with poor (low) lung compliance who develop high inspiratory pressures when they are ventilated with the more traditional modes described previously. Thus, the advantage of this mode is that it allows the physician to set the airway pressure and thereby minimize barotrauma. The disadvantage is that the tidal volume varies depending on compliance. The sudden development of an increase in airway resistance (coughing, thick secretions, a kink in the ET tube, a Valsalva maneuver), for example, increases airway pressures and decreases tidal volumes to dangerously low levels.
- BiLevel ventilation is a style of ventilator support that allows a patient to breathe spontaneously at two levels of positive end-expiratory pressure (PEEP). The time at the lower PEEP level may be limited so that all breaths are taken at the upper PEEP level and the pressure is then released just long enough to allow the lung volume to decrease (airway pressure release ventilation). Alternatively, spontaneous breathing may occur at both levels. There is evidence to suggest that this improves patient comfort and synchrony with the ventilator.
- High-frequency oscillatory ventilation (HFOV) uses substantially faster rates (180 to 300/minute) and smaller tidal volumes than conventional modes. The result is a relative decrease in diaphragmatic excursion, lung movement, and airway pressures. The physical mechanisms responsible for gas movement are complex and incompletely understood.
        - Ventilator management
- Choice of ventilator mode. Patient needs should be matched with the appropriate ventilator mode by considering each mode's advantages and disadvantages.
- FIO_2 should be adjusted to ensure adequate arterial oxygenation, which is a blood hemoglobin saturation of 92% in lighter-skinned individuals and 95% in darker-skinned patients. The lowest possible FIO_2 (ideally ≤0.40) should be used to achieve these levels of arterial saturation to prevent pulmonary oxygen toxicity.
- Tidal volume. There is no consensus on the optimal tidal volume for the postoperative patient who requires short-term mechanical ventilatory support. However, in ARDS, low tidal volumes are associated with improved survival.
- Ventilatory rate. Once the tidal volume has been determined, the rate is chosen (typically 8 to 16 breaths per minute) to provide adequate minute ventilation (the product of rate and tidal volume). The rate is adjusted to optimize arterial pH and $PaCO_2$; an end-tidal CO_2 monitor is useful in this regard.
- Inspiratory-expiratory (I:E) ratio. The normal I:E ratio is 1:2 to 1:3. Longer expiratory times allow patients with obstructive lung disease (high compliance) to exhale fully and prevent stacking of breaths. In contrast, longer inspiratory times, which decrease peak

airway pressures, are useful in patients with low pulmonary compliance. Inverse-ratio ventilation takes advantage of breath stacking, using I:E ratios from 1:1 to 4:1. Used only in patients with severe consolidating lung disease, inverse ratio ventilation is believed to improve gas exchange by progressive alveolar recruitment (mean airway pressures are higher, keeping a larger number of alveoli open for a greater percentage of the respiratory cycle). Inverse-ratio ventilation is used most commonly with pressure-control ventilation.

- Positive end-expiratory pressure increases functional residual capacity, increases lung compliance, and improves ventilation–perfusion matching by opening terminal airways and recruiting partially collapsed alveoli. PEEP of 5 cm H_2O is considered physiologic; higher levels are used when hypoxemia is moderate to severe. PEEP significantly increases intrathoracic pressure and therefore decreases CO, reduces venous return to the heart, increases airway pressure, and alters pulmonary vascular resistance. PEEP levels of greater than 15 cm H_2O significantly increase the risk of barotrauma and spontaneous pneumothorax. PEEP applied to the spontaneously ventilating patient without inspiratory ventilatory support is called continuous positive airway pressure (CPAP).
- Sedation and neuromuscular paralysis. Sedation is often necessary in mechanically ventilated patients to control anxiety, allow the patient to rest, and synchronize breathing. The need for paralysis is rare, except in patients with severe respiratory failure that benefit from increased pulmonary compliance due to decreased chest wall elastic recoil. If paralytics are necessary, they should be discontinued as soon as possible because long-term use is associated with paresis, which may last for weeks to months.
- Prone positioning is one of several techniques that may have benefit as “rescue” strategies in patients with severe acute lung injury or acute respiratory distress syndrome.

Mechanical ventilation is associated with significant morbidity, mortality, and costs of care. Therapies available to reduce this burden are listed below.

Concurrent Evidence-Based Therapies

Semirecumbency
Peptic ulcer disease prophylaxis
Deep venous thrombosis prophylaxis
Holding sedation such that patients can follow commands once a day
Daily assessment of respiratory function
Tight glucose control <110 mg/dl

Criteria for Extubation

Parameter	**Value**
Pa_{O_2} on Fi_{O_2}	>80 mm Hg on <0.6
Pa_{CO_2}	<45 mm Hg
Respiratory rate	<35
Tidal volume	>5 ml/kg
Vital capacity	>10 ml/kg
Minute ventilation	<10 l/min

Parameter	Value
Negative inspiratory force	-20 cm H_2O or more negative
Rapid shallow breathing index	<80
FiO_2, Fractional inspired oxygen.	

(d) Enteral Nutrition.

Answer. The need for nutritional support should be assessed continually in patients preoperatively and postoperatively.

- In general, the enteral route is preferred over the parenteral route. Enteral feeding is simple, physiologic, relatively inexpensive, and well tolerated by most patients. Enteral feeding maintains the GI tract cytoarchitecture and mucosal integrity (via trophic effects), absorptive function, and normal microbial flora. This results in less bacterial translocation and endotoxin release from the intestinal lumen into the bloodstream.
- Enteral feedings are indicated for patients who have a functional GI tract but are unable to sustain an adequate oral diet.
- Enteral feedings may be contraindicated in patients with an intestinal obstruction, ileus, GI bleeding, severe diarrhea, vomiting, enterocolitis, or a high-output enterocutaneous fistula.
- Choice of appropriate feeding site, administration technique, formula, and equipment may circumvent these problems.

- Feeding tubes. Nasogastric, nasojejunal (e.g., Dobhoff), gastrostomy, and jejunal tubes are available for the administration of enteral feeds. Percutaneous gastrostomy tubes can be placed endoscopically or under fluoroscopy.
- Enteral feeding products. A variety of commercially available enteral formulas are available. Standard solutions provide 1 kcal/mL; calorically concentrated solutions (>1 kcal/mL) are available for patients who require volume restriction. The available dietary formulations for enteral feedings can be divided into polymeric (blenderized and nutritionally complete commercial formulas), chemically defined formulas (elemental diets), and modular formulas.
  - Blenderized tube feedings can contain any food that can be blenderized. Caloric distribution of these formulas should parallel that of a normal diet.
  - Nutritionally complete formulas (standard enteral diets) vary in protein, carbohydrate, and fat composition. Several formulas use sucrose or glucose as carbohydrates and are suitable for lactose-deficient patients. Commercial formulas are convenient, sterile, and inexpensive. They are recommended for patients with minimal metabolic stress and normal gut function.
  - Chemically defined formulas (elemental diets). The nutrients are provided in predigested and readily absorbed form. They contain protein in the form of free amino acids or polypeptides. They are hyperosmolar, which may cause cramping and diarrhea. Elemental diets are efficiently absorbed in the presence of compromised gut function. However, they are costlier.
  - Modular formulations are designed for use in specific clinical situations (e.g., pulmonary, renal, or hepatic failure or immune dysfunction).

Enteral feeding protocols:

- Bolus feedings are reserved for patients with nasogastric or gastrostomy feeding tubes. Feedings are administered by gravity, begin at 50 to 100 mL every 4 hours, and are increased in 50-mL increments until goal intake is reached (usually 240 to 360 mL every 4 hours). Tracheobronchial aspiration is a potentially serious complication because feedings are prepyloric. To reduce the risk of aspiration, the patient's head and body should be elevated to 30 to 45 degrees during feeding and for 1 to 2 hours after each feeding. The gastric residual volume should be measured before administration of the feeding bolus. If this volume is greater than 50% of the previous bolus, the next feeding should be held. The feeding tube should be flushed with approximately 30 mL of water after each use. Free water volume can be adjusted as needed to treat hypo- or hypernatremia.
- Continuous infusion administered by a pump is generally required for nasojejunal, gastrojejunal, or jejunal tubes. Feedings are initiated at 20 mL/hour and increased in 10- to 20-mL/hour increments every 4 to 6 hours until the desired goal is reached. The feeding tube should be flushed with approximately 30 mL of water every 4 hours. Feedings should be held or advancement should be slowed if abdominal distension or pain develops. For some patients, the entire day's feeding can be infused over 8 to 12 hours at night to allow the patient mobility free from the infusion pump during the day.
- Conversion to oral feeding. When indicated, an oral diet is resumed gradually. In an effort to stimulate appetite, enteral feeding can be modified by the following measures:
    - Providing fewer feedings.
    - Holding daytime feedings.
    - Decreasing the volume of feedings. When oral intake provides approximately 75% of the required calories, tube feedings can be stopped.
- Administration of medications. Many oral medications can be administered through feeding tubes. The elixir form is preferred but is not always available. Medications that are not suitable for administration through a feeding tube include the following:
    - Enteric-coated medications.
    - Drugs in gelatinous capsules.
    - Medications that are designed for sublingual use.
    - Most sustained-release medications.

Complications:

- Metabolic complications. Abnormalities in serum electrolytes, calcium, magnesium, and phosphorus can be minimized through vigilant monitoring. Hyperosmolarity (hypernatremia) may lead to the development of mental lethargy or obtundation. Hyperglycemia may occur in any patient but is particularly common in individuals with preexisting diabetes or sepsis. Insulin should be used to control serum glucose level.
- Clogging can usually be prevented by careful routine flushing of the feeding tube. Wire stylets should not be used to unclog a feeding tube because of the risk of tube perforation and injury to the GI tract.
- Tracheobronchial aspiration of tube feeds may occur with patients who are fed into the stomach or proximal small intestine and may lead to the development of pneumonia. Patients at particular risk are those with central nervous system abnormalities and those who are sedated. Testing tracheal aspirateswith glucose strips or adding

methylene blue (1 mL/L) to the tube feeds aids in assessing for aspiration. Historically, jejunal feeding has been the preferred route for patients who are at risk for aspiration. High gastric residuals as a result of outlet obstruction, dysmotility, intestinal ileus, or bowel obstruction may limit the usefulness of nasogastric or gastrostomy feeding tubes.

- Diarrhea occurs in 10% to 20% of patients; however, other causes of diarrhea (e.g., Clostridium difficile colitis) should be considered. Diarrhea may result from an overly rapid increase in the volume of hyperosmolar tube feedings, medications (e.g., metoclopramide), a high-fat diet, or the presence of components not tolerated by the patient (e.g., lactose). If other causes of diarrhea can be excluded, the volume or concentration of tube feedings should be decreased. In surgical patients, C. difficile is a frequent cause of diarrhea due to the common use of perioperative antibiotics. Diagnosis can be confirmed with a C. difficile toxin assay or colonoscopy. Treatment includes stopping unnecessary antibiotics, followed by either oral or intravenous metronidazole or vancomycin orally or as a retention enema**.**

(e) Tumour Markers and their role in Surgery.

Answer. Tumor markers are indicators of cellular, biochemical, molecular, or genetic alterations by which neoplasia can be recognized. These surrogate measures of the biology of the cancer provide insight into the clinical behavior of the tumor. This is particularly useful when the cancer is not clinically detectable. The information provided may

- Be diagnostic and distinguish benign from malignant disease
- Correlate with the amount of tumor present (so-called tumor burden)
- Allow subtype classification to more accurately stage patients
- Be prognostic, either by the presence or absence of the marker or by its concentration
- Guide choice of therapy and predict response to therapy

The ideal tumor marker has three defining characteristics:

1. The marker is expressed exclusively by the particular tumor
2. Collection of the specimen for the tumor marker assay is easy.
3. The assay itself is reproducible, rapid, and inexpensive.

Tumor markers fall into three broad categories—proteins, genetic mutations, and epigenetic changes. All three may be found in the tumor tissue itself. Tumor markers found in body fluids, particularly blood and urine, have the greatest potential for clinical application because of the ease of access to these fluids for analysis and because repeated sampling allows in vivo monitoring of the malignancy for such features as disease progression or recurrence, metastasis, and response to therapy.

Potential Nonprotein Tumor Markers
RNA-Based Markers
Overexpressed/underexpressed transcripts
Regulatory RNA (e.g., micro-RNA)
DNA-Based Markers
Single-nucleotide polymorphisms (SNPs)
Chromosomal translocations—bcr-abl (Philadelphia)
Changes in DNA copy number
Microsatellite instability
Epigenetic changes (e.g., differential promoter region methylation)

Protein Tumor Markers

Carcinoembryonic Antigen

Carcinoembryonic antigen (CEA) is probably the most studied cancer tumor marker and is predominantly used clinically in patients with cancer of the colon and rectum. It is an oncofetal protein that is normally present during fetal life but can be seen in low concentration in healthy adults.

Testing Normal serum levels are less than 2.5 ng/mL, borderline if 2.5 to 5.0 ng/mL, and elevated if greater than 5.0 ng/mL. Borderline levels occur with benign disorders such as inflammatory bowel disease, pancreatitis, cirrhosis, and chronic obstructive pulmonary disease, and smoking can also increase CEA—the upper limit of normal in smokers is considered 5 ng/mL.

Screening CEA is not useful as a screening test because of its low sensitivity in early-stage disease—elevated CEA levels occur in only 5% to 40% of patients with localized disease.

Prognosis Elevated CEA levels reflect the burden of tumor present. The degree of CEA elevation correlates with increasing stage of disease, and therefore CEA levels have prognostic value. Preoperative serum CEA is an independent predictor of survival—the higher the preoperative serum level, the poorer the prognosis. This effect persists even after patients are stratified for resectability and extent of local tumor invasion. Five-year survival is significantly worse in patients with elevated preoperative CEA levels than in those with a normal preoperative CEA level.

Monitoring The most common application of CEA is to monitor patients for recurrent disease. CEA is most sensitive for hepatic or retroperitoneal metastasis and relatively insensitive for local, pulmonary, or peritoneal involvement. About 75% of patients with recurrent colorectal cancer have an elevated serum CEA level before the development of symptoms. However, the pattern or magnitude of the rise in CEA levels is of no value in distinguishing localized recurrence from distant disease. Because elevations of CEA may be transient, repeat measurement is performed as confirmation of the trend. A confirmed rising trend in CEA prompts evaluation for recurrent disease.

α-Fetoprotein: α-Fetoprotein (AFP) is used for the detection and management of HCC. It is an oncofetal antigen that consists of a single-chain polypeptide with a molecular weight of 700 kd. Levels are elevated in the fetus, decrease sharply after birth, and are increased during pregnancy.

Testing AFP is measured with immunoassay kits, either enzyme-linked immunoassays or radioimmunoassays. The upper limit of normal for a healthy, nonpregnant adult is less than 25 ng/mL. Ten percent to 20% of HCCs do not have detectable levels of AFP. Levels are also raised in nonseminomatous testicular cancer, for which it is a valuable tumor marker (see discussion later). Twenty percent of patients with gastric or pancreatic cancer and 5% of patients with colorectal or lung cancer have significant elevations (>5 ng/mL) in serum AFP levels. Elevated levels are also seen in hepatitis, inflammatory bowel disease, and cirrhosis.

Screening AFP has an estimated sensitivity of 25% to 75%, a specificity of 76% to 94%, and a positive predictive value of 9% to 50%. However, note that the sensitivity and specificity vary with the cutoff value chosen. If the cutoff is set at 20 ng/mL, the sensitivity and specificity are 30% and 87%, respectively, but if raised to 100 and 400 ng/mL, the sensitivity and specificity vary from 72% to 56% and 70% to 94%, respectively.

Prognosis The AFP concentration reflects tumor size, with levels higher than 400 ng/mL being associated with larger tumors. As a result, it has been shown that AFP correlates with stage and prognosis. The rate of increase, expressed as AFP doubling time, has also been associated with poorer prognosis.

Monitoring AFP has been shown to decline after resection or ablation. After complete resection, AFP levels should drop and remain at less than 10 ng/mL. Tumor regrowth after chemoembolization does not correlate with rate of increase in AFP or tumor burden. AFP levels usually decline in response to effective chemotherapy. Monitoring of AFP therefore avoids prolonged use of ineffective and potentially toxic chemotherapy.

Carbohydrate Antigen 19-9

Carbohydrate antigen 19-9 (CA 19-9) is widely used as a serum marker for pancreas cancer, but its use is limited to monitoring response to therapy, not as a diagnostic marker.The CA 19-9 epitope is normally present within the biliary tree. Biliary tract disease, both acute and chronic, can elevate serum CA 19-9 levels.

Testing CA 19-9 is detected with an immunoassay, and the upper limit of normal for a healthy adult is 37 U/mL. Patients with negative Lewis^{a} blood group antigen cannot synthesize CA 19-9, and therefore it is not used as a serologic marker in these individuals, who make up about 10% of the population. Patients with benign biliary tract disease can have levels up to 400 U/mL, with 87% having concentrations higher than 70 U/mL. Significant numbers of patients with pancreatitis, either acute or chronic, also have elevated levels. Third, besides pancreatic cancer, CA 19-9 levels are also elevated in patients with other cancers, including those of the biliary tree (95%), stomach (5%), colon (15%), liver (HCC, 7%) and lung (13%). For colorectal cancer, CA 19-9 levels add little clinically useful information to determination of CEA levels.

Screening CA 19-9 is not useful as a screening modality because of its low sensitivity in early-stage disease. With increasing levels of CA 19-9, the diagnosis of pancreatic cancer becomes more accurate. Because of its frequent elevation in benign biliary tract disease, CA

19-9 is not useful in distinguishing benign from malignant distal common bile duct strictures.

Prognosis In patients with pancreatic cancer who have CA 19-9 detectable in their serum, the level has been shown to correlate with tumor burden. For example, higher CA 19-9 levels typically correlate with higher tumor stage, and more than 95% of patients with unresectable disease have levels higher than 1000 U/mL. Of patients who undergo curative resection, those whose CA 19-9 levels returned to normal survived longer than those whose levels fell but never normalized.

Monitoring Serial measurement of CA 19-9 is used to monitor response to therapy. A rise in CA 19-9 after curative resection has been shown to precede clinical or computed tomographic evidence of recurrence by 2 to 9 months. In patients with unresectable/metastatic disease, failure of CA 19-9 levels to fall with chemotherapy reflects poor tumor response. However, in both settings, the lack of alternative effective therapies limits the utility of serial monitoring of CA 19-9.

Prostate-Specific Antigen

Prostate-specific antigen (PSA) is a serine protease that is formed in the prostatic epithelium and secreted into the prostatic ducts. PSA is considered a tissue-specific rather than a prostate cancer–specific marker—patients who have undergone curative radical prostatectomy, as well as females, have no detectable PSA.

Testing PSA is detected with an immunoassay. Besides BPH, other instances in which serum PSA levels may be elevated include prostatitis, prostatic massage, prostatic biopsy, and digital rectal examination. Initial studies set the upper limit of normal for PSA at 4 ng/mL, with levels greater than 10 ng/mL being suspicious for malignancy and levels of 4 to 10 ng/mL being indeterminate.

Screening PSA is widely used as a screening tool for prostate cancer because it enables early detection and diagnosis of this disease. Screening detects prostate cancer earlier. However, much concern has been raised about the risk of overdiagnosis. Autopsy studies have found that prostate cancer can be found in 55% of men in their 5th decade of life and 64% in their 7th decade, thus indicating that a significant proportion of these cancers are not lethal. Only one in eight screening-detected cancers is likely to kill its host if left untreated.

Monitoring Response to Therapy After operative resection, the PSA level is expected to normalize after 2 to 3 weeks. In patients whose PSA level remained elevated 6 months after radical prostatectomy, recurrent disease eventually developed. In contrast, it takes 3 to 5 months for PSA to normalize after radiotherapy. However, failure of the PSA level to normalize after radiotherapy also predicts relapse. A rise in serum PSA is usually the first sign of either local recurrence or metastatic progression. In patients with advanced disease, PSA levels are also used to monitor response to systemic therapy.

Carbohydrate Antigen 125 : Carbohydrate antigen 125 (CA 125) is a carbohydrate epitope on a glycoprotein carcinoma antigen. It is present in the fetus and in derivatives of the coelomic epithelium, including the peritoneum, pleura, pericardium, and amnion. In healthy adults, CA 125 has been detected by immunohistochemistry in the epithelium of the

fallopian tubes, endometrium, and endocervix. However, neither adult nor fetal ovarian epithelium expresses CA 125.
Testing CA 125 levels are measured with an immunoassay, with the upper limit of normal set at 35 U/mL. Elevated levels are detected in 80% of patients with ovarian cancer. In patients with ovarian masses, an elevated CA 125 level has a sensitivity of 75% and a specificity of approximately 90% for malignancy. It is also detectable in a high percentage of patients with cancer of the fallopian tube, endometrium, and cervix, as well as in nongynecologic malignancies of the pancreas, colon, lung, and liver. Benign conditions in which CA 125 is elevated include endometriosis, adenomyosis, uterine fibroids, pelvic inflammatory disease, cirrhosis, and ascites. As for CA 19-9 in patients with pancreatic cancer, CA 125 is an adjunct to diagnosis rather than being diagnostic by itself.

Screening Alone, CA 125 is not useful as a screening tool for ovarian cancer because of its poor specificity.

Prognosis Patients with elevated CA 125 levels at the time of diagnosis have a worse prognosis than patients with normal levels do. Absolute levels of CA 125 do not clearly correlate with tumor stage, although with increasing stage, greater percentages of patients have elevated CA 125 levels—50% of stage I patients, 70% of stage II patients, 90% of stage III patients, and 98% of stage IV patients.

Monitoring Response to Therapy CA 125 is of value in monitoring the disease course. Partial or complete response to therapy is associated with a decrease in CA 125 levels in more than 95% of patients. Increasing levels of CA 125 correlate with disease recurrence and precede clinical or imaging evidence of recurrence by a median of 3 months. When rising CA 125 levels are used as an indication for second-look laparotomy, recurrent disease is found approximately 90% of the time.

CA 125 levels in peritoneal fluid may be more sensitive than serum levels. Thus, in patients whose serum CA 125 level normalizes during therapy, peritoneal fluid CA 125 levels may better be able to distinguish patients with residual disease from those without. The upper limit of normal for peritoneal fluid CA 125 is 200 U/mL.

<u>α-Fetoprotein and Human Chorionic Gonadotropin in Testicular Germ Cell Tumors</u>

Nonseminomatous testicular cancers comprise several different histologic types, including embryonal carcinoma, syncytiotrophoblasts (choriocarcinoma), yolk sac tumors, and teratomas. Marker expression can be predicted on the basis of the predominant histologic type—human chorionic gonadotropin (HCG) is detected in more than 90% of choriocarcinomas, whereas AFP is expressed by 90% to 95% of yolk sac tumors, 20% of teratomas, and 10% of embryonal carcinomas.

Diagnosis Of patients with proven nonseminomatous testicular germ cell tumors, about 50% will have elevated serum levels of HCG and 60% will have elevated AFP, with either marker being elevated in 90% of cases. Determination of both marker levels is very important because nearly half these tumors secrete only one of these substances. In addition to the high rate of marker positivity, there have been very few cases of spuriously elevated serum levels of HCG or AFP in patients without testicular cancer. The presence of a testicular tumor in combination with an elevated level of AFP or HCG is suggestive of

testicular cancer, without being diagnostic. Elevated levels of these markers in a man younger than 40 years without signs of a testicular tumor may indicate extratesticular germ cell cancer.

Prognosis An absolute AFP concentration greater than 500 ng/mL or an HCG level higher than 1000 ng/mL is predictive of a poor prognosis. These tumor markers are useful in identifying biologically distinct categories of morphologically similar tumors.

Monitoring In the majority of patients with nonseminomatous germ cell tumors, tumor marker levels correlate with response to chemotherapy. The rate of marker decline (half-life), calculated from weekly determinations after initiation of chemotherapy, can be used for early identification of patients who will respond poorly to chemotherapy. Half-lives longer than 3.5 days for HCG or longer than 7 days for AFP suggest that very aggressive therapy is required, such as high-dose chemotherapy in combination with stem cell transplantation. However, there is a significant percentage of patients whose levels of tumor markers fall despite failure of their tumors to regress with therapy.

After completion of primary therapy, increasing marker concentrations, even in the absence of other features of recurrence, may lead to salvage chemotherapy. Therefore, it is important to exclude false-positive results. The HCG level needs to be measured in urine, where the concentration is generally similar to that in serum; however, interfering substances are not excreted into urine. Intensive chemotherapy may induce hypogonadism with associated HCG levels of up to 5 to 10 IU/L. It can be differentiated from relapse by measurement of luteinizing hormone and follicle-stimulating hormone—similar to the postmenopausal state in women, levels higher than 30 to 50 IU/L indicate that HCG is derived from the pituitary.

DNA-Based Markers

Specific mutations in oncogenes, tumor-suppressor genes, and mismatch repair genes can serve as biomarkers. These mutations may be germline, such as the ret proto-oncogene of MEN 2 and the APC gene of FAP, or somatic mutations, such as the occurrence of p53 mutations in a wide variety of tumors. Chromosomal abnormalities such as the 9:22 translocation that creates the bcr-abl oncogene are also useful biomarkers. Specific single-nucleotide polymorphisms have been identified that are associated with increased risk for specific cancers, and haplotype assessment has been shown to predict susceptibility to several cancers, including prostate, breast, lung, and colon cancer.

Epigenetic Changes

Testing for epigenetic changes is still at an early discovery stage and has not yet reached the clinic. First, DNA assays for aberrant methylation are easier and more sensitive than those for point mutations. Second, cancer-specific DNA methylation patterns can be detected in tumor-derived free DNA in the bloodstream and in epithelial tumor cells shed into the lumen. This ease of access to sample medium may facilitate efforts at detection and monitoring of cancer. Third, DNA-methylation profiles are more chemically and biologically stable than RNA or most proteins. As a result, they may be more reliably detected in diverse biologic fluids.

Methylation biomarker studies have been performed in a variety of cancers, including breast, esophageal, gastric, colorectal, and prostate cancer. Sources of the DNA have included plasma/serum, urine, sputum, and saliva.

Combining DNA methylation assays may complement existing screening methods with high sensitivity but low specificity, such as PSA in prostate cancer. The use of panels of methylation targets in these studies improved the clinical sensitivity of the assay.

Potential Applications

1. Early detection. Although abnormal epigenetic silencing of genes can occur at any time during carcinogenesis, it appears to occur most frequently early in the transformation process. Aberrant crypt foci that contain preneoplastic hyperplastic colonic epithelial cells have been found to demonstrate abnormal methylation in promoter regions of genes involved in abnormal activation of the Wnt signaling pathway. Detection of abnormal methylation patterns in histologically normal cells may emerge as a useful marker for assessment of cancer risk.
2. Predict response to therapy. Methylation of specific genes can be linked to the biologic behavior of the tumor.
3. Prognostication. Abnormal methylation of combinations of genes has been associated with a poor outcome.

On an opposite note, loss of methylation is increasingly being recognized as an important event in carcinogenesis.Hypomethylated CpG islands have been associated with the activation of nearby genes. For example, hypomethylation of the promoter for the cancer/testis antigen CAGE correlates with increased expression of the gene and is found in premalignant lesions of the stomach. Similar instances of demethylated promoters activating their downstream genes have been found in numerous other cancers, including those of the colon, pancreas, liver, uterus, lung, and cervix. In a recent study of ovarian carcinogenesis, hypomethylation of centromeric and juxtacentromeric satellite DNA was found to be increased in tumors of advanced stage or high grade, and this strong hypomethylation was an independent marker of poor prognosis. Furthermore, genome-wide hypomethylation has also been detected in cancer cells and may contribute to genomic instability.

DNA-methylation profiles in which both hypermethylation and hypomethylation are examined may provide greater insight into tumor behavior than possible with either profile alone.

RNA-Based Markers

RNA-based markers have been identified in the context of global mRNA expression by high-throughput technologies. These microarrays (“gene chips”) allow us to measure the expression of 30,000 to 40,000 human genes in a single experiment. Statistical modeling then allows selection of groups of genes, or so-called fingerprints, that best distinguish disease states.

Proteomic Profiling : Proteomics is the study of all the proteins expressed by the genome. Proteomic profiling using mass spectrometry technologies generates complex fingerprints

of ion peaks corresponding to protein concentrations, which can be correlated with disease states. Numerous studies using samples of blood (plasma or serum), urine, and pancreatic juice have demonstrated the feasibility of this technology for discovery of biomarkers and early detection of ovarian, breast, prostate, and pancreatic cancer. Identification of reproducible protein signatures of specific diseases has the potential to achieve much higher diagnostic sensitivity and specificity than possible with currently available biomarkers. Proteomic profiling lacks a standardized methodology and remains time and labor intensive.

Biomarkers and Biologically Targeted Therapies

CANCER	BIOMARKER	THERAPY
Breast	Estrogen receptor, progesterone receptor	Tamoxifen/aromatase inhibitors
Lymphoma	CD20	Rituximab
Chronic myelogenous leukemia (CML)	bcr-abl	Imatinib
Gastrointestinal stromal tumor (GIST)	c-kit	Imatinib
Non–small cell lung cancer	EGFR mutation	Gefitinib
Breast	HER2/neu	Trastuzumab

4. Write brief answers on the following: $7\frac{1}{2}$

a) Pathology of Vascular Malformations in childhood.
b) Role of blood component in Surgery.
c) Cervical rib and its effects.
d) Tumours of Endocrine Pancreas.

Answer.

(a) Pathology of Vascular Malformations in childhood.

Answer. Vascular malformations

Key facts:

Classified broadly into two principal groups.

Primary haemangiomas (with endothelial hyperplasia)

- All congenital or idiopathic.
- Mostly sporadic but may rarely be part of a familial syndrome, e.g. von Hippel-Lindau.
- Pulmonary haemangiomas commonly seen in hereditary haemorrhagic telangiectasia are linked to a deficiency in endoglin (endothelial growth factor).

True arteriovenous malformations (AVM)

AVMs have three main causes.

- Congenital: origin/cause unknown. Mostly sporadic but may rarely be part of a congenital syndrome (e.g. Klippel-Trenaunay).
- Traumatic: may follow relatively minor trauma.

- latrogenic: following a variety of surgical /interventional procedures.

Pathological features: Vascular malformations are histologically categorized as capillary, venous, lymphatic, or mixed in type, depending on the predominant vessel type affected, and subdivided into low or high flow varieties.

Clinical features:

- Congenital AVMs are usually evident at birth and the superficial lesion may only represent a part of the overall abnormality.
- Symptoms are dependent on the size, site, and type of vessel affected, and whether the AVMs are high or low flow.

Low flow

- May result in considerable cosmetic deformity if large (e.g. Klippel-Trenaunay).
- Pain may be a feature due to spontaneous thrombosis of some/all of the venous elements.
- Classically the symptoms are worse after exercise when blood flow is maximized.

High flow

- These are largely asymptomatic but there may be a detectable venous hum or bruit.
- They may result in local hyperhidrosis, heat, ulceration or present with profuse bleeding.
- May lead to high output cardiac failure if large and untreated.

Go through: Diagnosis and investigation:

- **Colour duplex: diagnoses lesion, can estimate flow rate, and is useful for follow-up monitoring.**
- **MRI has replaced CT as the best imaging modality and gives both the extent and related anatomy for complex lesions.**
- **Angiography is reserved for high flow lesions when suitability for embolization**

Treatment:

Largely conservative:

- **Congenital avms frequently reduce in size with growth of the child and treatment is rarely easy;**
- **Adult AVMs only require treatment for complications or occasionally cosmesis.**

Interventional radiology

- **Percutaneous embolization using wire coils or foam sclerosant under radiological guidance.**

- **Risks include:**
    - **Risks of percutaneous puncture (infection, false aneurysm formation, embolization);**
    - **Inadvertent embolization of adjacent vessels;**
    - **Tissue necrosis after successful lesion embolization;**
    - **Postembolization syndrome may occur with pain at the site of embolization accompanied by malaise, fever and leucocytosis, hyperkalaemia. This usually settles with symptomatic treatment in 24-48h. due to tissue necrosis and cytokine release.**

Surgery:

- **Small lesions may be excised completely.**
- **Obliteration of small superficial venous malformations can be undertaken by direct puncture and injecting a sclerosant such as STD (sodium tetradecyl sulphate).**
- **Open surgery is mostly confined to high flow lesions after preoperative embolization.**

(b) Role of blood component in Surgery.

Answer. Role of blood component in Surgery:

- Platelet transfusions:

Indications. Platelet transfusions are used to control bleeding that is caused by thrombocytopenia or platelet dysfunction and to prevent spontaneous bleeding in situations of severe thrombocytopenia. Platelet transfusions should be considered for a platelet count of less than 10,000/μL to prevent spontaneous bleeding. In cases of bleeding or for minor surgical procedures, the transfusion threshold is often increased to a platelet count of less than 50,000/μL. For severe ongoing hemorrhage and before major operations, platelet counts greater than 100,000/μL should be the goal. Anticoagulation medications

- Principles and indications. Anticoagulation is used to prevent and treat thrombosis and thromboembolic events. Before therapy is instituted, careful consideration must be given to the risk of thromboembolism and to anticoagulation-induced bleeding complications. Specific indications for anticoagulation therapy are discussed in detail in other chapters; they include atrial fibrillation, mechanical prosthetic heart valves, venous thromboembolism, stroke prevention, and acute arterial or graft occlusion.
- Heparin
    - Unfractionated heparin (MW 10,000 to 20,000) acts by potentiating the action of AT, leading to accelerated inhibition of thrombin, factor Xa, and other coagulation proteases.
        - Administration. Heparin is administered parenterally, either subcutaneously or intravenously. PTT should be measured before initiation of heparin, 6 hours after the initial bolus, and 6 hours after each change in dosing. A therapeutic PTT of 1.5 to 2.5 times the control (approximately 50 to 80 seconds) should be maintained. Platelet counts should be measured daily until a maintenance dose of heparin is achieved and periodically thereafter.
-

    - LMWH preparations (enoxaparin, dalteparin, and tinzaparin) are depolymerized, yielding fragments of 3,000 to 7,000 daltons. The anticoagulant effect of LMWH is predominantly due to factor Xa inhibition, and LMWH results in less thrombin inhibition than unfractionated heparin. The advantages of LMWH include a more predictable anticoagulant effect, less platelet interaction, and a longer half-life. Dosing is based on weight, and laboratory monitoring is not typically needed. LMWH may be used for longer-term therapy in patients with a contraindication to oral anticoagulant treatment (e.g., pregnant patients who cannot take warfarin). Because LMWH has a longer half-life and no effective antidote, it must be used with caution in surgical patients and in those in whom a bleeding risk has been substantiated.
- Direct thrombin inhibitors are a class of compounds that bind to free and fibrin-bound thrombin. These agents inhibit thrombin activation of clotting factors, fibrin formation, and platelet aggregation.
    - Hirudin, an anticoagulant originally derived from leeches, has been formulated as lepirudin and bivalirudin. Lepirudin, recombinant hirudin, binds irreversibly to thrombin, providing effective anticoagulation. The drug is approved in patients with HIT but may be considered in other severe clotting disorders. Lepirudin is cleared unmodified by the kidney, so dosing is adjusted in patients with renal insufficiency. Monitoring of anticoagulation is by the PTT, similar to heparin. Bivalirudin is a truncated form of recombinant hirudin that targets only the active site of thrombin. Bivalirudin is Food and Drug Administration approved for use during percutaneous coronary angioplasty and stenting.
    - Argatroban is a synthetic thrombin inhibitor that is also approved for treatment of HIT. Therapy is monitored by the PTT. Elimination is primarily through the liver and should be used with caution in patients with hepatic insufficiency.

Fibrinolytic therapy

- Indications and contraindications. Thrombolytic therapy is most often used for iliofemoral deep venous thrombosis (DVT), superior vena caval thrombosis, pulmonary embolism (PE) resulting in a hemodynamically unstable patient, acute thrombosis of peripheral, mesenteric, and coronary arteries, acute vascular graft occlusion, thrombosis of hemodialysis access grafts, and occlusion of venous catheters. Dosage depends on the agent and the indication. Tissue plasminogen activator (alteplase) or a recombinant analog (reteplase), as well as urokinase (Abbokinase), are used for lysis of catheter, venous, and peripheral arterial thrombi.

Transfusion products for coagulopathy

- FFP contains all the coagulation factors. However, factors V and VIII may not be stable through the thawing process and are not reliably recovered from FFP. Therefore, it can be used to correct coagulopathies that are due to deficiencies of any other coagulation factor and is particularly useful when multiple factor deficiencies exist (e.g., liver disease or massive transfusion). FFP effects are immediate and typically last about 6 hours. Factor VIII and IX deficiencies are best treated using specific factor concentrates.

Contraindications to Fibrinolytic Therapy

Absolute contraindications
Intolerable ischemia (for arterial thrombosis)
Active bleeding (not including menses)
Recent (<2 mo) stroke or neurosurgical procedure
Intracranial pathology such as neoplasm
Relative contraindications
Recent (<10 d) major surgery, major trauma, parturition, or organ biopsy
Active peptic ulcer or recent gastrointestinal bleeding (within 2 wk)
Uncontrolled hypertension (blood pressure >180/110 mm Hg)
Recent cardiopulmonary resuscitation
Presence or high likelihood of left heart thrombus
Bacterial endocarditis
Coagulopathy or current use of warfarin
Pregnancy
Hemorrhagic diabetic retinopathy

- Cryoprecipitate is the cold-insoluble precipitate of fresh plasma and is rich in factor VIII and vWF as well as fibrinogen, fibronectin, and factor XIII. Cryoprecipitate may be used as second-line therapy in vWD or hemophilia but is most often used to correct fibrinogen deficiency in DIC or during massive transfusion.
- Recombinant human factor VIIa (rhFVIIa) is used primarily in the treatment of patients with hemophilia or factor VIII inhibitors. However, it may be considered for patients with difficult bleeding problems for which therapy is inadequate or not available. The recommended dose is 100 μg/kg, which can be repeated at 1- to 2-hour intervals if needed. The PT is used to monitor drug effect. Use of rhFVIIa in blunt trauma patients requiring massive transfusions has been shown to reduce the overall blood transfusion requirements. There was also a trend in reduction of the incidence of multiple system organ failure and acute respiratory distress syndrome with its use.

(c) Cervical rib and its effects.

Answer. Arterial compression is almost universally associated with a bony abnormality of the thoracic outlet. Cervical ribs, which occur in 0.5% to 1.5% of the normal population, are present in more than 95% of patients with arterial TOS. Repetitive compression and trauma to the subclavian artery lead to intimal injury/ulceration at the site of compression or poststenotic dilation/aneurysmal degeneration of the artery just distal to the point of compression. Mural thrombus forming at the site of the damaged intima or within an aneurysm can then embolize down the arm, leading to ischemic symptomatology.

Effects of cervical rib:

- Patients with symptomatic arterial TOS most commonly present with symptoms of digital ischemia related to microembolization of platelet aggregates formed on intimal lesions or mural thrombus from poststenotic aneurysms.
- Early symptoms may include unilateral Raynaud's syndrome, or complaints of digital cyanosis, pallor, coldness, pain, and paresthesias. Findings are most common in the thumb and index finger because of the direct pathway of the radial artery.

- If the arterial lesion is not diagnosed and corrected, repeated embolization will lead to obliteration of the distal arterial bed, with worsening ischemia leading to digital ulceration and gangrene.
- Macroembolization to the larger arteries of the arm presents as acute limb ischemia, the severity of symptoms depending on the patency of the distal arterial tree. Even a relatively small embolus can be limb threatening if preceded by multiple episodes of microembolization.
- Thrombotic occlusion of the subclavian artery can also occur. Physical examination may be normal or reveal signs of ischemia of varying degrees and extents.
- A supraclavicular bruit or pulsatile mass may occasionally be detected.
- Concomitant symptoms of neurogenic TOS may be present but are often overshadowed by ischemic symptomatology.
- Patients with Raynaud's symptoms or subtle signs of digital ischemia should always undergo segmental pressure testing including the digital waveforms to rule out an occlusive process.
- In the absence of a documented collagen vascular disorder or severe arteriosclerosis, the presence of small artery (palmar/digital) occlusive disease should always lead to arteriography.
- Patients with macroemboli and no obvious cardiac source should also undergo arteriography to exclude a proximal arterial source.Arteriography should include complete views from the aortic arch to the fingertips.
- If the subclavian artery appears normal in the neutral position, repeat view should be obtained with the arm abducted. Magnified views of the hand following vasodilator injection are helpful in distinguishing embolic occlusions from intrinsic disease.
- Arteriographic findings can be subtle, ranging from minor wall irregularities and minimal poststenotic dilation to frank aneurysms.
- Duplex scanning may be useful in patients who present with pulsatile supraclavicular masses or questionable findings on arteriography.

(d) Tumours of Endocrine Pancreas.

Answer. Tumours of Endocrine Pancreas are:

- Gastrinoma (Zollinger-Ellison syndrome) – origin gastrin producing G cell.
- Insulinoma - origin B cell.
- VIP oma – origin D2 cell.
- Glucagonoma - origin A cell. .
- Somatostatinoma - origin D cell.
- PPoma – origin PP(F) cell.

Methods of Localization of Endocrine Tumors of the Pancreas:

MODALITY
Noninvasive
Ultrasonography
Octreotide radioimaging (SRS)
CT
MRI
Invasive

Endoscopic ultrasonography
Selective angiography
Portal venous sampling
Provocative angiography: Calcium for insulinoma; secretin for gastrinoma. SRS, somatostatin receptor scintigraphy.

Gastrinoma (Zollinger-Ellison syndrome):
Clinical Presentation: Many patients with ZES present with abdominal pain, peptic ulcer disease, and severe esophagitis.

Aspects of Peptic Ulcer Disease That Raise Suspicion for Zollinger-Ellison Syndrome

1. Ulcers in atypical locations.
2. Multiple ulcers
3. Ulcers that fail to respond to conventional treatment
4. Ulcers that recur after conventional treatment
5. Peptic ulcer disease in association with diarrhea
6. Ulcers in association with hyperparathyroidism

- Serologic Testing: The diagnosis of ZES is made by measuring the serum gastrin level. It is important that patients stop taking proton pump inhibitors for this test. In most patients with gastrinomas, the level is greater than 1000 pg/ml, but it can be in the 150 to 1000 pg/ml range or even normal.
- Tumor Localization: The next step is to localize the gastrinoma(s) and to determine if metastases are present. This is best accomplished preoperatively if possible. In 70% to 90% of patients, the primary gastrinoma is found in Passaro's triangle; an area defined by a triangle with points located at the junction of the cystic duct and common bile duct, the second and third portion of the duodenum, and the neck and body of the pancreas.
- The test of choice is somatostatin receptor scintigraphy in combination with computed tomography (CT).
- Endoscopic ultrasound (EUS) is another new modality that assists in the preoperative localization of gastrinomas. It is particularly helpful in localizing tumors in the pancreatic head or duodenal wall where gastrinomas are usually less than 1 cm. A combination of octreoscan and EUS detects more than 90% of gastrinomas.

Surgery: Pharmacologic control of acid secretion has rendered total gastrectomy unnecessary. Omeprazole therapy is so effective that every patient with ZES is a candidate for a tumor removal operation until proved otherwise because of systemic illness or widespread metastases. Although gastrinomas have a high rate of malignancy, efforts at surgical cure are clearly justified.

- Every attempt is made to localize the tumor before surgery, and CT and MRI are effective with larger tumors and especially with hepatic metastases. Gastric secretion is controlled during the perioperative period with either oral or parenteral proton pump inhibitors.

- Intraoperative ultrasonography plus palpation is effective in localizing 90% to 98% of pancreatic gastrinomas.

Tumors within the pancreas are enucleated if at all possible. If they are adjacent to a duct, care must be taken to not injure the duct, but if the duct is injured, it is fine-sutured and a drain placed. Large tumors located distally can be excised by distal pancreatectomy. Tumors within the head are enucleated if at all possible.

Treatment of metastatic disease has undergone serial changes but is still unsatisfactory. Radiation therapy and chemotherapy are largely ineffective. The combination of doxorubicin, streptozotocin, and 5-fluorouracil has a low, temporary response rate, but it is highly toxic and has no impact on survival.Surgical treatment of distant metastases by cytoreduction procedures (debulking) appears to be useful, and some patients with solitary localized metastatic disease have prolonged postoperative disease-free survival.

Insulinoma: Insulinoma is the most common functioning tumor of the pancreas,

Clinical features and diagnosis: The diagnostic hallmark of the syndrome is the so-called Whipple triad, namely, symptoms of hypoglycemia (catecholamine release) and low blood glucose (40-50 mg/dL) and relief of symptoms after the IV administration of glucose.

- The symptom complex may be due to autonomic nervous overactivity, as expressed by fatigue, weakness, fearfulness, hunger, tremor, sweating, and tachycardia, or alternatively, a central nervous system disturbance with apathy (or irritability or anxiety), confusion, excitement, loss of orientation, blurring of vision, delirium, stupor, coma, or convulsions.
- The pathognomonic finding is an inappropriately high (>5 mU/mL) level of serum insulin during symptomatic hypoglycemia. A possible mechanism for this high level of insulin in the face of hypoglycemia may be overexpression of the insulin splice variant.
- A diagnostic ratio of blood insulin (in microunits per milliliter) to glucose (in milligrams per deciliter) of greater than 0.4 or C peptide levels higher than 2 nmol/L have proved valuable in diagnosis. The best way to induce hypoglycemia is with fasting: two thirds of patients will experience hypoglycemic symptoms in 24 hours, and nearly all other patients experience symptoms by 72 hours of fasting.

Localization:
- Insulinomas are small (usually <1.5 cm), usually single (only 10% are multiple and those are usually associated with MEN 1 syndrome), usually benign (only 5%-10% are malignant), and generally hard to find.
- Plain abdominal radiographic and ultrasound studies are rarely helpful, but contrast-augmented computed tomography (CT) and magnetic resonance imaging (MRI) locate 50% to 60% of tumors.
- Because few insulinomas have many somatostatin receptors, somatostatin receptor scintigraphy (SRS) is not highly successful. Success in localization by selective arteriography varies with the size of the tumor; 90% accuracy rates have been reported with insulinomas.
- Selective portovenous sampling is also helpful for measurement of insulin levels in pancreatic venous tributaries, a method that does not absolutely localize the site of the

tumor but, in about 75% of cases, does provide accurate information on the region of the pancreas from which high levels of insulin are released.

- Highly promising test has been developed for localizing insulinomas by means of selective intra-arterial injection of calcium (into the gastroduodenal, superior mesentery, right hepatic, or splenic arteries) and obtaining samples for radioimmunoassay of insulin from the right hepatic vein.
- Localization study is useful in identifying the tumor by intraoperative ultrasound.

Surgery: Treatment of insulinoma is surgical and performed by either open or laparoscopic approaches.

- Most insulinomas are benign and can be enucleated. Nutrient vessels in the bed of the adenoma need to be cauterized. Care needs to be taken during enucleation to avoid injury to ductal structures, and if a duct is injured, it is sutured and drained. If malignant, the tumor is resected in a cancer-type operation, and if metastatic, it is worthwhile to try to remove all primary and metastatic tumor tissue in an effort to minimize persistent hyperinsulinism.
- Persistent hyperinsulinemia after surgery for metastatic islet cell tumors may be managed by hepatic artery tumor embolization, by diazoxide, or by streptozotocin plus fluorouracil.

Verner-Morrison Syndrome (VIPoma):

- VIPomas are endocrine tumors usually arising from pancreatic islets that secrete VIP and cause a syndrome of profound watery diarrhea, hypokalemia, and achlorhydria.
- The diarrhea persists despite fasting (which qualifies it as a secretory diarrhea) and despite nasogastric aspiration (which differentiates it from the diarrhea of ZES).
- Verner-Morrison syndrome is highly variable. Constant features are diarrhea, hypovolemia, hypokalemia, and acidosis; variable features are achlorhydria or hypochlorhydria, hypercalcemia, hyperglycemia, and flushing with rash.

- The diagnostic triad in Verner Morrison syndrome is a secretory diarrhea, high levels of circulating VIP, and a pancreatic tumor. Diarrhea volumes are often massive, 3 to 5 L/day, and the diagnosis of VIPoma is unlikely if stool volume is less than 700 mL/day. VIPomas alone show elevated levels of VIP; normal levels are lower than 200 pg/mL, and VIPoma patients have levels ranging from 225 to 2000 pg/mL.
- The best localization is achieved either by SRS or by endoscopic ultrasound. Most tumors are large, and localization is often achieved with enhanced CT or MRI or with arteriography. About 50% of patients have metastatic spread by the time of diagnosis. If abdominal studies fail to locate the tumor, thoracic CT is performed because as many as 10% of the tumors are intrathoracic.
- As soon as the diagnosis is established, treatment with the long-acting somatostatin analogue octreotide is used to control fluid loss.
- Surgical removal of VIPoma is attempted in all patients.
- Most VIPomas can be excised by distal pancreatectomy.
- The adrenals and retroperitoneal tissue are carefully examined if no pancreatic tumor is found.
- In the 50% of patients with metastatic disease, local excision of as much tumor as can be safely removed (debulking) is indicated.
- Partial pancreatectomy plus resection of liver metastases has been reported to bring about resolution of recurrent Verner-Morrison syndrome. In patients with

nonresectable tumors, chemotherapy is rarely effective, but octreotide is helpful in the control of diarrhea.

Glucagonoma: A tumor of islet alpha cells, glucagonoma causes a syndrome of a characteristic rash, diabetes mellitus, anemia, weight loss, and elevated circulating levels of glucagon. The characteristic skin lesion, a necrolytic migrating erythema. Glucagonoma was found to be associated with anemia, glossitis, and most importantly, a low level of amino acids; parenteral administration of amino acids was found to bring about disappearance of the skin lesions. Diabetes is usually mild.

- The diagnosis of glucagonoma is made from the characteristic skin lesion, elevated levels of glucagon (whose release can be provoked by secretin, if necessary), and a pancreatic tumor. The upper limit of normal for glucagon is 150 to 190 pg/mL; glucagonoma patients have levels of 200 to 2000 pg/mL. The islet tumor may be demonstrated by enhanced CT or MRI or by selective angiography.
- Once the diagnosis is made, the patient is prepared by the administration of total parenteral nutrition containing amino acids, along with simultaneous octreotide for symptomatic relief. Tumors are best localized by CT, MRI, and intraoperative ultrasonography. Because a third of these patients have been reported to have thrombotic complications after surgery, perioperative heparin is indicated.

Treatment is surgical excision of the tumor, which usually lies in the body or tail of the pancreas. Nearly all glucagonomas are malignant, but an aggressive approach to removal of the primary and metastatic tumor is warranted. Even so, the cure rate appears to be only 30%, and long-term chemotherapy has proved disappointing for metastatic disease. Symptomatic relief can be achieved with octreotide.

Somatostatinoma: All endocrine tumors of the pancreas are rare; somatostatinomas are exceedingly rare.

- Full syndrome - (steatorrhea, diabetes mellitus, hypochlorhydria, and gallstones).
- The features of the syndrome are variable and do not always coincide with the predictable effects of high circulating levels of somatostatin.
- Most patients have mild diabetes, but 10% have symptoms of hypoglycemia.
- This illustrates the unpredictability of hor-mone-hormone interactions: in the case of diabetes, the suppressive effect of somatostatin on insulin release predominates; in hypoglycemic patients, the predominant inhibition affects glucagon.
- The clinical findings are unpredictable; obstructive jaundice caused by pressure on the common bile duct by tumor has developed in some patients, whereas initial findings in other patients have been diarrhea and gallstones. Some duodenal somatostatinomas have been associated with von Recklinghausen's neurofibromatosis.
- Tumors can be localized by CT, MRI, arteriography, and even SRS because somatostatinomas do possess functioning somatostatin receptors.
- Treatment is surgical. Seventy percent to 90% of tumors have been reported to be malignant. Most tumors are located in the tail of the pancreas, and caudal pancreatectomy is indicated.
- Localization is rarely a problem because the tumors are generally large. Hepatic metastases are common, and debulking of metastatic tumor tissue is indicated. The large size and frequency of malignancy dictate resection rather than enucleation of the pancreatic tumor.

- Metastases are, of course, excluded before consideration is given to a Whipple resection of the head of the pancreas. Small duodenal tumors can be treated by local excision. At surgery, cholecystectomy is performed regardless of whether the patient has gallstones because if they are not present, they will probably occur.

Still-Rarer Tumors:

- Various functional tumors of the endocrine pancreas have been reported, with some secreting GRF, neurotensin, PTHrP, PP, or ACTH, with the appropriate resultant endocrine syndrome.
- GRFomas are invariably associated with MEN 1 syndrome; 30% of GRF tumors originate in the pancreas, 50% in the lung, and 10% in the small bowel.
- Forty percent of patients with GRFoma have ZES, and 40% have Cushing's syndrome.
- Patients with ACTH-secreting tumors usually have other endocrine syndromes, most frequently ZES. They have classic symptoms of Cushing's syndrome (which occurs in 5% of all ZES patients and in 20% of patients with both ZES and MEN 1).
- Neurotensinomas cause hypokalemia, weight loss, hypotension, cyanosis, flushing, and diabetes. They are usually malignant.
- PPomas are associated with high circulating PP levels and no characteristic symptoms, although patients with PPomas have been reported with watery diarrhea and rash. PPomas are almost always large and, except when associated with MEN 1 syndrome, are usually solitary and in the head of the pancreas. Elevated levels of PP are often seen with other islet cell tumor syndromes.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES

MS (General Surgery) Examination, 2009

PAPER I

Time Allowed: 3 Hours **Full Marks: 100**

Attempt all questions

1. **Describe the surgical anatomy of the thyroid gland. How do you evaluate its function. (10+10)**
2. **2. Classify shock and discuss the pathophysiology of septic shock. How do you manage it? 5+7+8**

3. Write short notes of the following: 5 x 6

a) Deep Venous Thrombosis

b) Metabolic changes in pyloric obstruction.

c) Diagnostic studies for obstructive jaundice.

d) Universal precaution.

e) Malrotation of gut.

4. Answer briefly on the following: $7^1/_2$

a) Diagnosis and management of gastro-oesophageal reflux disease.

b) Role of nutrition, its methods in surgical patients.

c) Pain relief in surgery.

d) Post splenectomy problems.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES
MS (General Surgery) Examination, 2009

May 2009

PAPER I

Time Allowed: 3 Hours **Full Marks: 100**

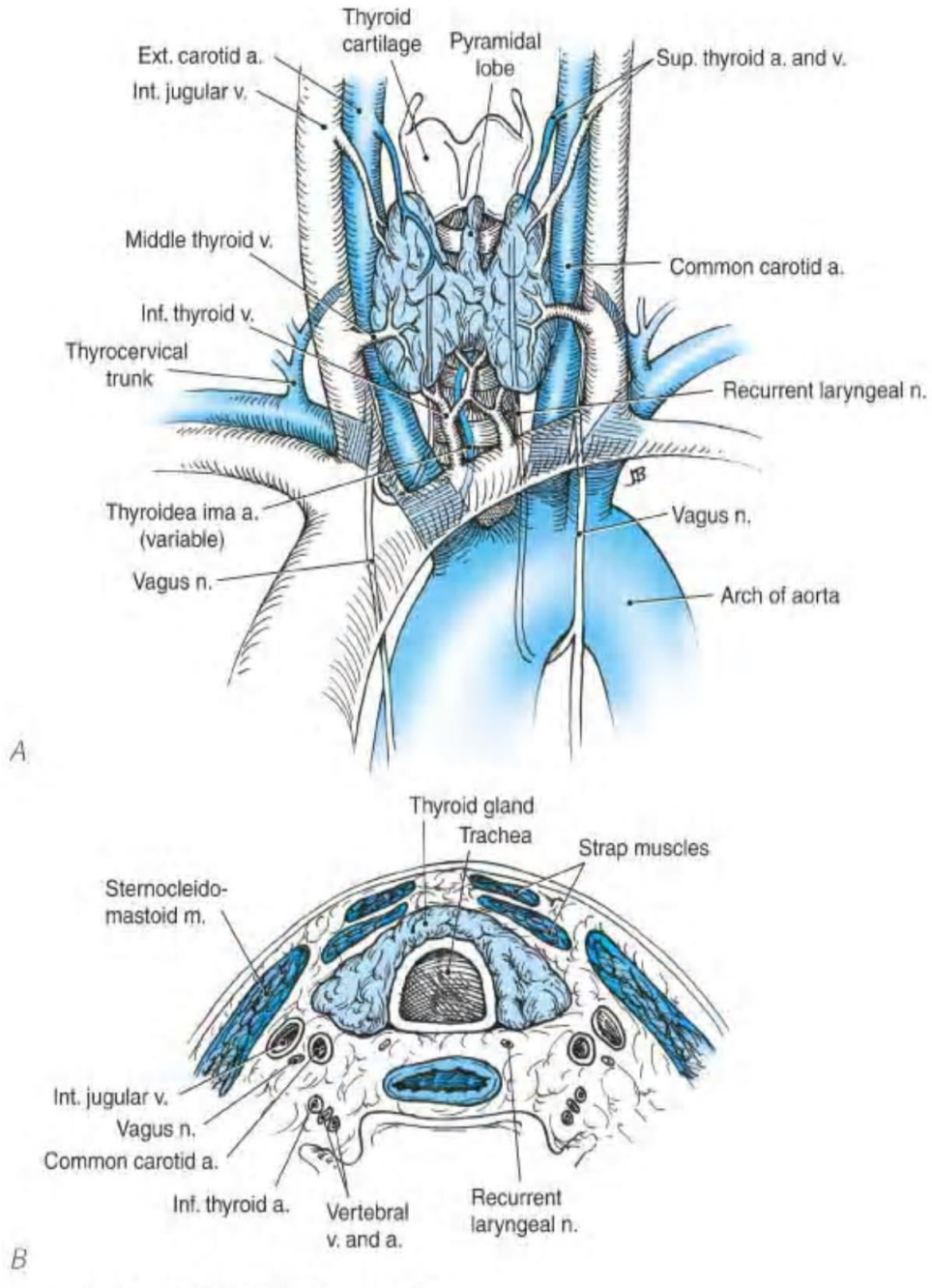
Attempt all questions

1. Describe the surgical anatomy of the thyroid gland. How do you evaluate its function. (10+10)

Answer.

Thyroid Anatomy:

- Figure given below depicts the anatomic relations of the thyroid gland and surrounding structures.
- The adult thyroid gland is located posterior to the strap (sternothyroid and sternohyoid) muscles.
- The normal thyroid gland weighs approximately 20 g, but gland weight varies with body weight and iodine intake.
- The thyroid lobes are located adjacent to the thyroid cartilage and connected in the midline by an isthmus which is typically located just inferior to the cricoid cartilage.
- A pyramidal lobe, which represents the most caudal end of the thyroglossal duct, is found in approximately 50% of individuals having thyroid operations.
- The thyroid lobes extend to mid-thyroid cartilage superiorly and lie adjacent to the carotid sheaths and sternocleidomastoid muscles laterally.
- The strap muscles (sternohyoid, sternothyroid and superior belly of the omohyoid) are located anteriorly and are innervated by the ansa cervicalis (ansa hypoglossi).
- The thyroid gland is enveloped by a loosely connecting fascia that is formed from the partition of the deep cervical fascia into anterior and posterior divisions.
- The true capsule of the thyroid is a thin, densely adherent fibrous layer that sends out septa that invaginate into the gland, forming pseudolobules. The thyroid capsule is condensed into the posterior suspensory or Berry's ligament near the cricoid cartilage and upper tracheal rings.



Anatomy of the thyroid gland and surrounding structures, viewed anteriorly (A) and in cross section (B).

Blood Supply:

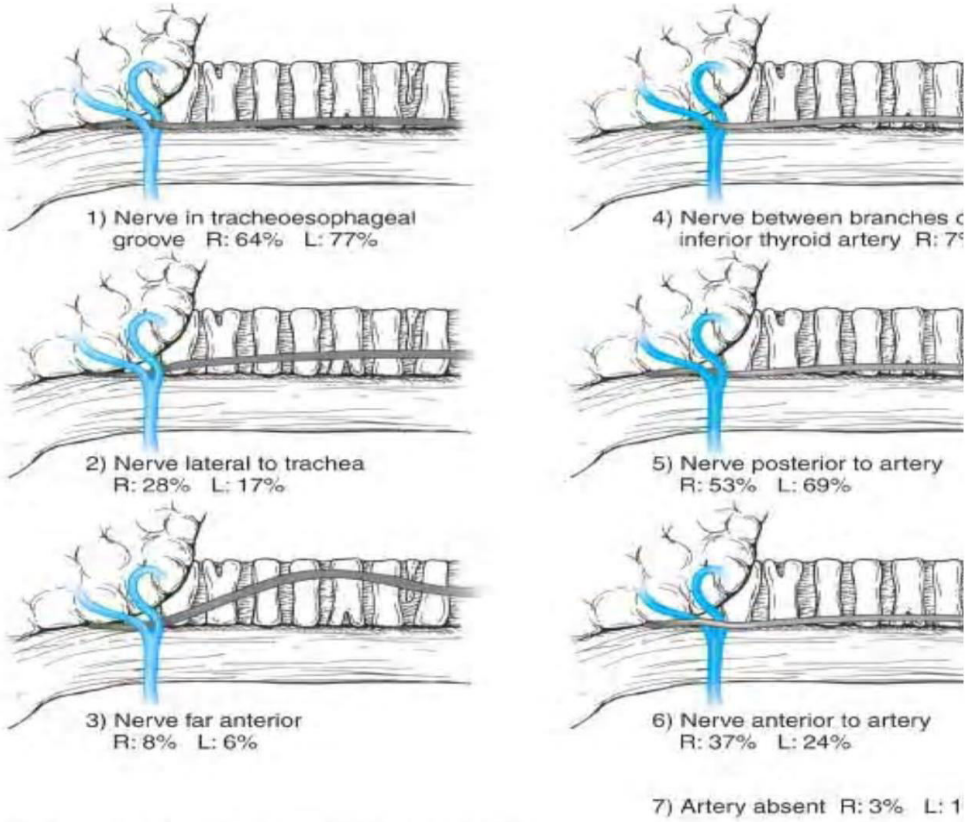
- The superior thyroid arteries arise from the ipsilateral external carotid arteries and divide into anterior and posterior branches at the apices of the thyroid lobes.
- The inferior thyroid arteries are derived from the thyrocervical trunk shortly after their origin from the subclavian arteries. The inferior thyroid arteries travel upward in the neck posterior to the carotid sheath to enter the thyroid lobes at their midpoint.

- A thyroidea ima artery arises directly from the aorta or innominate in 1 to 4% of individuals, to enter the isthmus or replace a missing inferior thyroid artery.
- The inferior thyroid artery is intimately associated with the recurrent laryngeal nerve (RLN), necessitating identification of the RLN before the arterial branches can be ligated.
- Venous drainage of the thyroid gland occurs via multiple small surface veins, which coalesce to form three sets of veins—the superior, middle, and inferior thyroid veins. The superior thyroid veins run with the superior thyroid arteries bilaterally. The middle vein or veins are the least consistent.
- The superior and middle veins drain directly into the internal jugular veins, the inferior veins often form a plexus, which drains into the brachiocephalic veins.

Nerves:

- The left RLN arises from the vagus nerve where it crosses the aortic arch, loops around the ligamentum arteriosum and ascends medially in the neck within the tracheoesophageal groove.
- The right RLN arises from the vagus at its crossing with the right subclavian artery. The nerve passes posterior to the artery before ascending in the neck, its course being more oblique than the left RLN.
- Along their course in the neck, the RLNs may branch, and pass anterior, posterior or interdigitate with branches of the inferior thyroid artery.
- The right RLN may be nonrecurrent in 0.5 to 1% of individuals and is often associated with a vascular anomaly in this situation.
- 2 Nonrecurrent left RLNs are rare, but have been reported in patients with situs inversus and a right-sided aortic arch.
- The RLN may branch in its course in the neck, and identification of a small nerve should alert the surgeon to this possibility. Identification of the nerves or their branches often necessitates mobilization of the most lateral and posterior extent of the thyroid gland, the tubercle of Zuckerkandl, at the level of the cricoid cartilage.
- The last segments of the nerves often course below the tubercle and are closely approximated to the ligament of Berry.
- Branches of the nerve may traverse the ligament in 25% of individuals, and are particularly vulnerable to injury at this junction. The recurrent laryngeal nerves

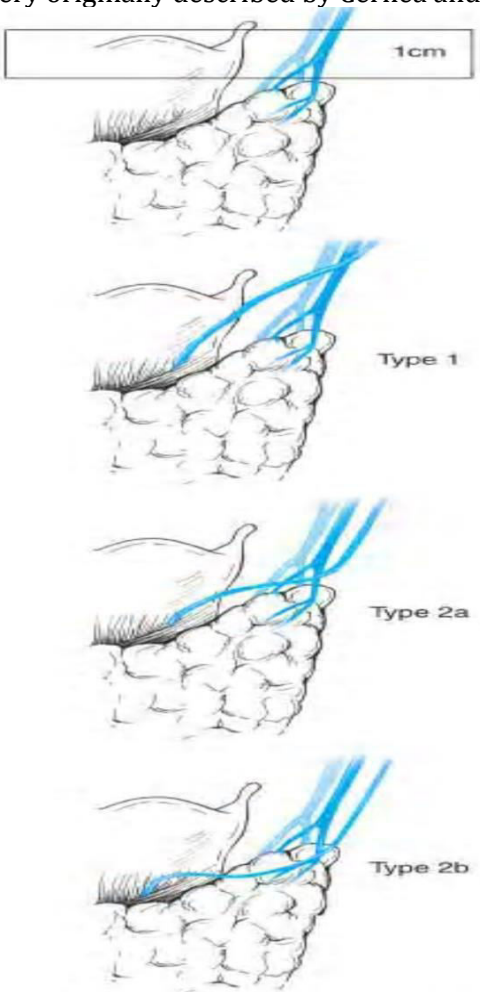
terminate by entering the larynx posterior to the cricothyroid muscle.



- Relationship of RLN to the inferior thyroid artery. The superior parathyroid is characteristically dorsal to the plane of the nerve, whereas the inferior gland is ventral to the nerve.
- The RLNs innervate all the intrinsic muscles of the larynx, except the cricothyroid muscles, which are innervated by the external laryngeal nerves.
- Injury to one RLN leads to paralysis of the ipsilateral vocal cord, which comes to lie in the paramedian or the abducted position. The paramedian position results in a normal, but weak voice, whereas the abducted position leads to a hoarse voice and an ineffective cough.
- Bilateral RLN injury may lead to airway obstruction, necessitating emergency tracheostomy, or loss of voice. If both cords come to lie in an abducted position, air movement can occur, but the patient has an ineffective cough and is at increased risk of repeated respiratory tract infections from aspiration.

- The superior laryngeal nerves also arise from the vagus nerves. After their origin at the base of the skull, these nerves travel along the internal carotid artery and divide into two branches at the level of the hyoid bone.
- The internal branch of the superior laryngeal nerve is sensory to the supraglottic larynx. Injury to this nerve is rare in thyroid surgery, but its occurrence may result in aspiration.
- The external branch of the superior laryngeal nerve lies on the inferior pharyngeal constrictor muscle and descends alongside the superior thyroid vessels before innervating the cricothyroid muscle.

- Cernea and associates proposed a classification system to describe the relationship of this nerve to the superior thyroid vessels. The type 2a variant, in which the nerve crosses below the tip of the thyroid superior pole, occurs in up to 20% of individuals and places the nerve at a greater risk of injury.
- Therefore, the superior pole vessels should not be ligated en masse, but should be individually divided, low on the thyroid gland, and dissected lateral to the cricothyroid muscle.
- This nerve also has been called the Amelita Galla Curci or "high note" nerve after the opera singer. Injury to this nerve leads to inability to tense the ipsilateral vocal cord and hence difficulty "hitting high notes," projecting the voice, and voice fatigue during prolonged speech.
- Relationship of the external branch of the superior laryngeal nerve and superior thyroid artery originally described by Cernea and associates.



In type 1 anatomy, the nerve crosses the artery 1 cm above the superior aspect of the thyroid lobe. In type 2 anatomy, the nerve crosses the artery <1 cm above the thyroid pole (2a) or below (2b) it.

- Sympathetic innervation of the thyroid gland is provided by fibers from the superior and middle cervical sympathetic ganglia. The fibers enter the gland with the blood

vessels and are vasomotor in action. Parasympathetic fibers are derived from the vagus nerve and reach the gland via branches of the laryngeal nerves.

Tests of Thyroid Function

Evaluation of the Pituitary-Thyroid Feedback Loop:

- Evaluation of serum TSH is an important screening test for the diagnosis of thyroid dysfunction. TSH is measured by an ultrasensitive radioimmunometric assay, which has greatly improved clinical diagnosis.
- This assay is especially important in the delineation of hypothyroid from euthyroid states. Additionally, clinically euthyroid patients may have suppressed TSH levels (subclinical hyperthyroidism), and the assay can therefore demonstrate hyperthyroidism before it becomes clinically manifested. The sensitivity of the TSH assay is less affected by nonthyroidal disease processes and remains unaffected by changes in thyroid hormone–binding proteins.

Serum Triiodothyronine and Thyroxine Levels:

- Thyroid production is initially screened by measuring serum free T_4. Total T_4, which measures both free and protein-bound hormone, can be affected by changes in hormone production or hormone binding to serum proteins; therefore, accurate evaluation of thyroid function requires measurement of free T_4 levelsCalcitonin

- In patients with thyroid masses and in whom multiple endocrine neoplasia type 2 (MEN 2) syndrome or isolated medullary carcinoma is suspected, a baseline calcitonin level can be calculated. If there is doubt about the diagnosis, pentagastrin- or calcium-stimulated calcitonin evaluation, a 4- to 5-hour test, can be performed. Additionally, calcitonin can be used as a screening test in families with MEN 2 syndrome to document clinically inapparent disease. Use of calcitonin screening in patients with a thyroid mass, however, is not cost-efficient or necessary.

Radioactive Iodine Uptake:

The radioactive iodine uptake test is becoming less widely used .This test has in the past involved oral administration of iodine 123 (^{123}I) and calculated its uptake with radioscintigraphy. A normal result is 15% to 30% uptake of the radionuclide after about 24 hours. Use of ^{123}I is preferable because of a shorter half-life and lesser radiation exposure than with ^{131}I, which is used to radioablate thyroid neoplasms.

Thyroid Autoantibody Levels:

Thyroid antigens are produced in autoimmune thyroid disorders (thyroid-stimulating immunoglobulin, antimicrosomal antibodies), including Graves' disease and Hashimoto's thyroiditis. Detection of autoantibodies can be extremely important if either of these autoimmune conditions is suspected. About 95% of patients with Hashimoto's thyroiditis and 80% with Graves' disease have detectable antimicrosomal antibodies. In Graves' disease, circulating antibodies have high affinity for TSH receptor (TSH-R) on thyroid

follicular cells. Newer assays have greater sensitivity and may allow earlier detection of Graves' disease and more accurate monitoring of the effects of thyroid medication.

2. Classify shock and discuss the pathophysiology of septic shock. How do you manage it? 5+7+8

Answer.

Classification Of Shock - According to Bailey and Love:
Classification of shock:

- Hypovolaemic shock
- Cardiogenic shock
- Obstructive shock
- Distributive shock
- Endocrine shock

Other classification:

a. **Cardiogenic** : Shock caused as a result of cardiac pump failure
   Causes:
   - Myocardial Infarction
   - Arrythmias (Atrial fibrillation, ventricular tachycardias, bradycardias, etc)
   - Mechanical abnormalities (valvular defects)
   - Extracardiac abnormalities (PE, pulm HTN, tension pneumothorax)

b. **Hypovolemic**: Shock caused by decreased preload due to intravascular volume loss.
   Causes:
   - Hemorrhagic – trauma, GI bleed, hemorrhagic pancreatitis, fractures
   - Fluid loss induced – Diarrhea, vomiting, burns

c. **Distributive**: Shock as a result of severely diminished systemic vascular resistance.

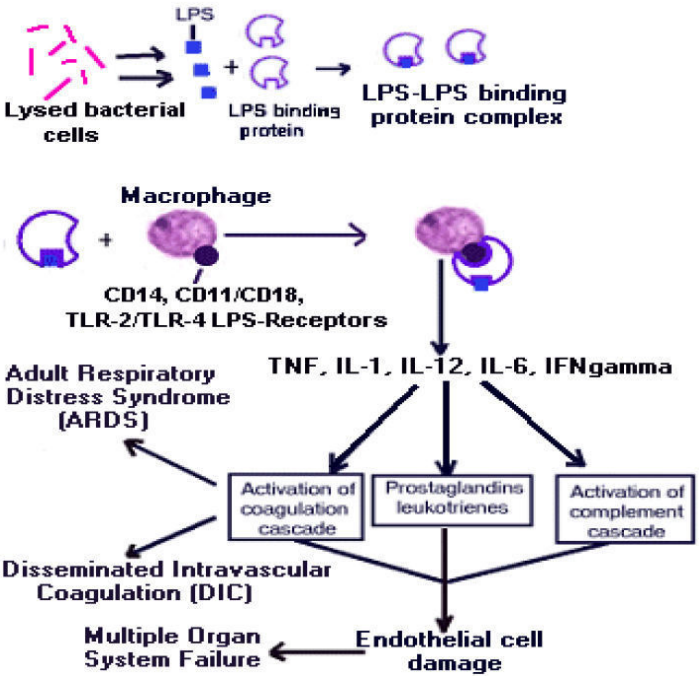
   i. Septic: secondary to an overwhelming infection

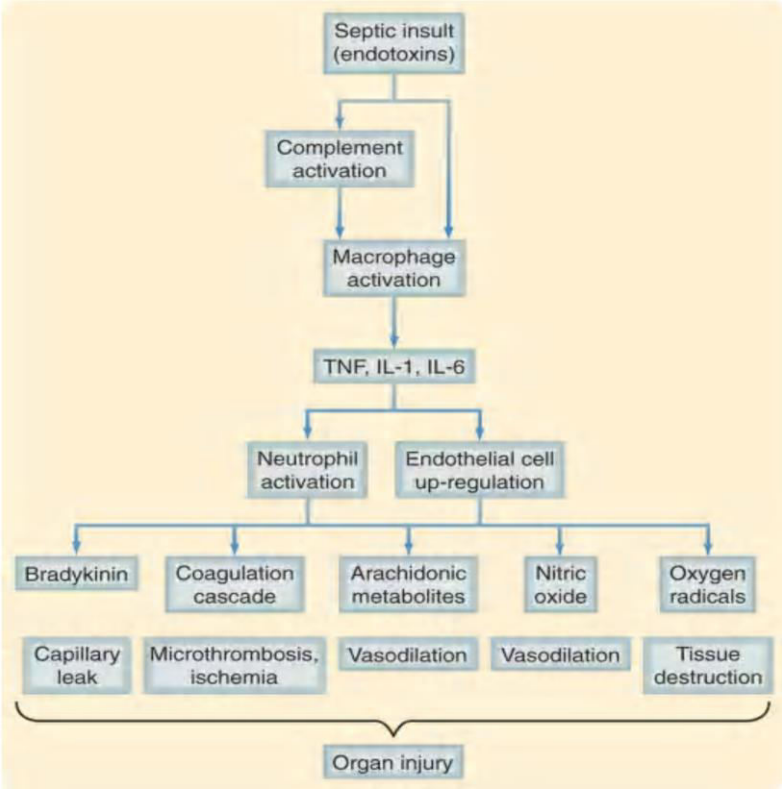
   ii. Anaphylactic: secondary to an overwhelming infection

   iii. Neurogenic: secondary to a sudden loss of the autonomic nervous system function.

d. **Combined**.

Pathogenesis of sepsis and septic shock:





Septic shock-mediated infl ammatory cascade. IL-1, interleukin-1; TNF, tumor necrosis factor.

Protocol for Resuscitation of Adult Hypotensive Patients With Suspected Sepsis

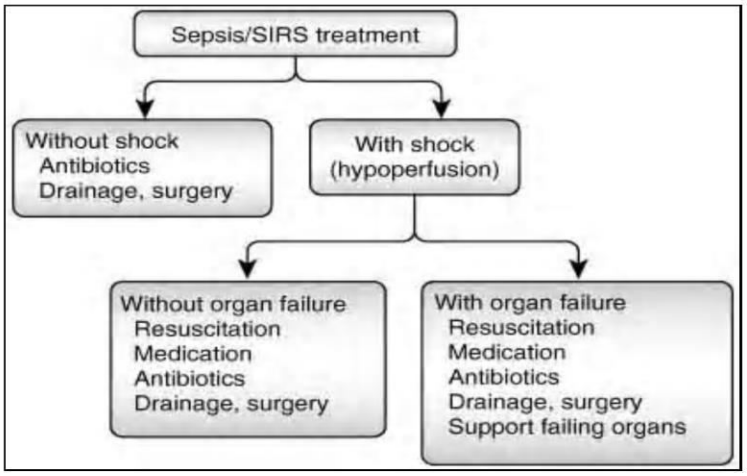
- Culture relevant body fluids, including blood.
- Infuse a balanced electrolyte solution of 500 mL/15 min. Monitor the systolic blood pressure response.
- Insert a central venous or pulmonary artery catheter.
- If after a 500-mL bolus of saline the patient remains hypotensive and CVP is <8-12 mm Hg or PAWP is <8-12 mm Hg, infuse another 500-mL bolus of fluid; repeat as needed.
- If CVP is >15 or PAWP is 15-20 and the patient remains hypotensive (<65 mm Hg), start an infusion of the inotrope dobutamine or dopamine. The goal is a mean systemic pressure >65 mm Hg and a pulse rate <120 beats/min.
- Determine the cardiac index and systemic vascular resistance.

    - If after infusion of fluid and inotropes SVR is <600, infuse a vasopressor—either norepinephrine or vasopressin—to increase SVR.

- Monitor mixed venous oxygen saturation and urine output as an indication that therapeutic interventions have improved perfusion. (**CVP, central venous pressure; PAWP, pulmonary artery wedge pressure; SVR, systemic vascular resistance.)**

Diagnostic Studies:

Blood	Complete blood count, amylase, lipase, renal function, liver function, serum lactate, arterial blood gases
Roentgenograms	Chest, abdomen, plain films of areas with suspected soft-tissue infection
Advanced imaging	Ultrasound, computerized tomography, magnetic resonance injury, nuclear imaging



A simple algorithm to match the therapeutic response to the patient's clinical condition. *SIRS,* Systemic inflammatory response syndrome.

3. Write short notes of the following: 5 x 6

a) Deep Venous Thrombosis
b) Metabolic changes in pyloric obstruction.

c) Diagnostic studies for obstructive jaundice.
d) Universal precaution.
e) Malrotation of gut.

Answer.

(a) Deep Venous Thrombosis

Answer. Causes and features:

- May develop in association with abnormalities of the vein wall, blood flow, or constituents of blood (Virchow's triad).
- May be due to vein compression or stasis (immobility, trauma, mass, bed rest, surgery, paralysis, long distance travel including airline travel).
- May be due to inherited hypercoaguability (factor V Leiden, protein C, protein S, or antithrombin insufficiency).
- May be due to acquired hypercoaguability (surgery, malignancy, polycythaemia, smoking, hormone replacement therapy, OCP, dehydration).

Severity may vary from isolated asymptomatic tibial/calf thrombosis to severe iliofemoral segment thrombosis with phlegmasia caerulea dolens (venous gangrene).

Documented risk factors for DVT:		
• Increasing Age • Obesity • Prolonged Immobility • Varicose Veins • Stroke	• Cardiac Dysfunction • Paralysis • Indwelling Central Venous Catheters • Previous Venous Thromboembolism • Inflammatory Bowel Disease	• Cancer And Its Treatment • Nephrotic Syndrome • Major Surgery • Pregnancy Or Estrogen Use. • Trauma

Clinical features

- Clinical manifestations may be absent.
- Local features of venous engorgement and stasis:
  - Limb swelling;
  - Pain;
  - Erythema and warmth to the touch;
  - Mild fever and tachycardia resulting from release of inflammatory mediators;
  - Homan's sign calf pain on dorsiflexion of the foot is very unreliable and should not be performed.
- Complications:
  - Pulmonary embolism;
  - Venous gangrene (phlegmasia dolens).

Diagnosis and investigations: Aim to confirm presence and extent of thrombosis (to decide on necessity and type of treatment, risk of embolization).

- **Fibrin, Fibrinogen Assays** The basis of fibrin or fibrinogen can be assayed by measuring the degradation of intravascular fibrin. The D-dimer test measures cross-linked degradation products, which is a surrogate of plasmin's activity on fibrin. It is shown that in combination with clinical evaluation and assessment, the sensitivity exceeds 90% to 95%. The negative predictive value is 99.3% for proximal evaluation and 98.6% for distal evaluation. In the postoperative patient, D-dimer is causally elevated due to surgery, and, as such, a positive D-dimer assay for evaluating for DVT is of no use. However, a negative D-dimer test in patients with suspected DVT has a high negative predictive value, ranging from 97% to 99%.
- Ascending venography: rarely used now.
- Duplex scan: investigation of choice. Visualizes anatomy and gives extent of thrombosis. Relies on flow of blood and compressibility of vein. Is operator-dependent and has lower sensitivity for calf DVT.
- VQ scan: If suspicion or evidence of pulmonary embolism.
  - CT pulmonary angiography (CTPA): safest, most sensitive, and most specific investigation for suspected pulmonary embolism.

Risk stratification for thromboembolism after surgery

Abbreviations*:* DVT: deep vein thrombosis; PE: pulmonary embolism.

Level of risk	Age(years)	Type of surgery	Additional risk factors	Incidence of proximal DVT (%)	Incidence of PE (%)
Low	<40	Minor	None	0.4	<0.5
Moderate					
A	Any	Minor	Present		
B	<40	Major	None		
C	40–60	Nonmajor	None		
High				4–8	2–4
A	>60	Nonmajor	±Other		
B	>40	Major	None		
C	<40	Major	Present		
Highest risk				10–20	4–10
B	>40	Major	Prior VTE		
			Cancer		
			Hypercoagulable state		

Recommendations:
Low Risk Patients – No prophylaxis is needed other than early ambulation

Moderate Risk – Low dose unfractionated heparin (LDUH) (5000 U) BID or low molecular weight heparin (LMWH) (< 3,400 U) QD or intermittent pneumatic compression stocking (IPCS). There is some data suggesting multiple modalities may be synergistic.

High Risk – LDUH (5000U) BID or TID or LMWH (< 3,400 U) QD or IPCS

Highest Risk – LDUH (5000U) BID or TID or LMWH (< 3,400 U) QD and IPCS

Arguments against of heparin prophylaxis:

1) Bleeding: 2 – 3% increase risk compared with placebo
2) Heparin induced thrombocytopenia: Occurs in 1-5% of people and usually resolves with discontinuation of heparin. Low molecular weight heparins have even a lower incidence.
3) Cost: Cost based analysis has shown heparin prophylaxis is far more cost effective when considering the decreased incidence of DVT and PE.

Treatment:

- Prophylaxis.
- Conservative measures: bed rest, elevation, and good hydration.
- Uncomplicated DVT:
  - Low molecular weight heparin (LMWH), initially in hospital; may be given on an outpatient basis via a dedicated DVT clinic. Subsequent treatment is with oral anticoagulation with warfarin for 3-6 months.
- Complicated DVT:
  - Initially with IV unfractionated heparin (UFH) whilst converting to oral anticoagulation with warfarin.
- Thrombolysis or surgical thrombectomy are reserved for severe thrombosis with venous gangrene.
- Vena caval filter percutaneously inserted via jugular vein into infrarenal IVC to catch thromboemboli and prevent PE.
  - Used for patients: with recurrent PEs despite treatment; at risk of major central PE; requiring urgent surgery despite high risk that DVT is present.
  - Risks include IVC obstruction, renal vein thrombosis, complications of insertion.

(b) Metabolic changes in pyloric obstruction.

Answr. The gastric outlet obstruction due to the pyloric obstruction impairs emptying of gastric contents into the duodenum. As a consequence, all ingested food and gastric secretions can only exit via vomiting, which can be of a projectile nature. The vomited material does not contain bile because the pyloric obstruction prevents entry of duodenal contents (containing bile) into the stomach.

This results in loss of water and gastric acid (hydrochloric acid). The loss of water leads to dehydration.The chloride loss results in hypochloremia which impairs the kidney's ability to excrete bicarbonate. This is the significant factor that prevents correction of the alkalosis.

A secondary hyperaldosteronism develops due to the hypovolemia. The high aldosterone levels causes the kidneys to:

- Avidly retain Na^+ (to correct the intravascular volume depletion)

- Excrete increased amounts of K^+ into the urine (resulting in hypokalaemia) and also excretion of H^+ion leading to aciduria. Hypokalemia later on leads to hypomagnesemia and hypocalcemia.

The body's compensatory response to the metabolic alkalosis is hypoventilation resulting in an elevated arterial pCO_2.

So there will be hypochloremic hypokalemic metabolic alkalosis with paradoxical aciduria.

(c) Diagnostic studies for obstructive jaundice.

Answer. Laboratory Tests:

Obstructive jaundice is commonly associated with both liver dysfunction and acute cellular injury with resultant elevations in liver function tests. Hepatocellular injury results in increased levels of unconjugated or indirect reacting bilirubin due to an increase in bilirubin production or a decrease in hepatocyte uptake with conjugation. Conjugated or direct hyperbilirubinemia is due to defects in bilirubin excretion (intrahepatic cholestasis) or extrahepatic biliary obstruction. In addition to hyperbilirubinemia, an increased alkaline phosphatase level is virtually pathognomonic of bile duct obstruction. Serum transaminase (aspartate and alanine) levels can also be mildly elevated in biliary system disease, either because of direct injury of the liver adjacent to an inflamed gallbladder or from the effect of biliary sepsis on hepatocellular membrane integrity. Leukocytosis, composed primarily of neutrophils, is often present with acute cholecystitis or cholangitis, but is a nonspecific finding that does not distinguish them from other infectious or inflammatory causes. Incresed GGT is alo indicative of obstructive jaundice.

Studies

Plain Radiographs

Although frequently obtained during the initial evaluation of abdominal pain, plain radiographs of the abdomen in patients with complaints localized to the right upper quadrant are rarely helpful. Only about 15% of gallstones contain enough calcium to render them radiopaque and therefore visible on plain abdominal films.

Ultrasonography

Ultrasound of the abdomen is an extremely useful and accurate method for identifying the cause of obstructive jaundice. It is often the initial screening test for patients with suspected extrahepatic biliary obstruction . Dilation of the extrahepatic (>10 mm) or intrahepatic (>4 mm) bile ducts suggests biliary obstruction. Intraoperative ultrasound is now used frequently to further evaluate intrahepatic lesions, assess resectability, and determine involvement of vascular structures.

Computed Tomography

Although abdominal CT scanning is probably the most informative single radiographic tool for examining intra-abdominal pathology, its overall value for the diagnosis of biliary tract disease pales in comparison to ultrasonography. The disadvantage is largely because gallstones and bile appear nearly isodense on CT; that is, it is difficult to distinguish

gallstones from bile, unless the stones are heavily calcified. Conversely, CT is more accurate at identifying the site and cause of extrahepatic biliary obstruction. Abdominal CT is a powerful tool for evaluating biliary tract disease when the differential diagnosis includes a question of hepatobiliary or pancreatic neoplasm. Use of CT cholangiogram provides improved definition of the biliary tract comparable to magnetic resonance cholangiography. Angiograms have now essentially been replaced by triple-phase liver CT angiogram.

Cholangiography

Cholangiography functionally involves the installation of contrast directly into the biliary tree and is the most accurate and sensitive method available to anatomically delineate the intrahepatic and extrahepatic biliary tree. It is most useful when the precise location or cause of biliary pathology needs to be ascertained. MRC is noninvasive and provides excellent anatomic detail. Both endoscopic retrograde cholangiopancreatography (ERCP) and percutaneous transhepatic cholangiography (PTC) are invasive procedures with a 2% to 5% risk of complications but offer the opportunity for a therapeutic intervention. ERCP is most useful in imaging patients with hepatobiliary malignancies and choledocholithiasis. It illustrates distal common bile duct or ampullary obstruction, can provide tissue samples for pathologic diagnosis, and can palliate patients with complete biliary obstruction using prosthetic stents. However, it gives no information regarding tumor size, local invasion, or distant spread, and is of limited use in staging.

Transhepatic cholangiography is the preferred technique in patients with proximal biliary obstruction or in patients in whom ERCP is not technically possible. Percutaneous transhepatic cholangiography can be followed by placement of transhepatic catheters, which can decompress the biliary system, function as anatomical landmarks during surgical reconstruction, or provide access for nonoperative dilation of strictures.

Scintigraphy

Biliary scintigraphy is useful to visualize the biliary tree, assess liver and gallbladder function, and diagnose several common disorders including cholecystitis. Although it is an excellent test to decide whether the common bile and cystic ducts are patent, biliary scintigraphy does not identify gallstones or give any detailed anatomic information. Nonvisualization of the gallbladder at 2 hours after injection is reliable evidence of cystic duct obstruction. Biliary scintigraphy followed by CCK administration is helpful for documenting biliary dyskinesia when gallbladder contraction accompanies biliary tract pain in patients without evidence of stones (CCK hepatobiliary 2,6-dimethyl-iminodiacetic acid [HIDA]). These agents are iminodiacetic acid (IDA)-based compounds and are processed in the liver and excreted (H originally stood for hydroxy, but today stands for hepatobiliary because other IDA derivatives, such as proisopropyl-IDA [PIPIDA], are more commonly used, but are still referred to as HIDA scans).

Laparoscopy

Advancement in laparoscopic skill has coincided with the increased use of laparoscopy for diagnosis and treatment of biliary tract disorders. It is most effective when used in conjunction with laparoscopic ultrasound in the staging and operative management of

biliary malignancies. Intraoperative ultrasound is now used frequently to further evaluate intrahepatic lesions, assess resectability, and determine involvement of vascular structures. Although the need for laparoscopy may have diminished as a result of advancements in radiologic techniques like CT, laparoscopy still best identifies micrometastases much beyond the discrimination of the CT scan; in addition, biopsy of micrometastases can be undertaken with the laparoscope.

FDG-PET Scanning

Fluorodeoxyglucose positron emission tomography (FDG-PET) is a whole-body technique that allows detection of unsuspected metastases that may lead to major changes in the surgical management of these patients. PET imaging with the fluorinated glucose analogue, ^{18}FDG, can be used to exploit the metabolic differences between benign and malignant cells for imaging purposes. Therefore, ^{18}FDG-PET imaging has become well established for differentiation of benign from malignant lesions, staging malignant lesions, detection of malignancy recurrence, and monitoring therapy for various malignancies. Recent studies have shown that ^{18}FDG-PET is accurate in predicting the presence of nodular cholangiocarcinoma (mass >1 cm) and gallbladder carcinoma (sensitivity, 78%). ^{18}FDG-PET is not useful for detection of carcinomatosis, and inflammatory changes related to biliary stents may cause interpretation difficulties.

(d) Universal precaution.

Answer. Universal precautions refers to the practice, in medicine, of avoiding contact with patients' bodily fluids, by means of the wearing of nonporous articles such as medical gloves, goggles, and face shields.

Under universal precautions all patients are considered to be possible carriers of blood-borne pathogens. The guideline recommends wearing gloves when collecting or handling blood and body fluids contaminated with blood, wearing face shields when there is danger of blood splashing on mucous membranes and disposing of all needles and sharp objects in puncture-resistant containers.

Universal precautions are designed for doctors, nurses, patients, and health care support workers who are required to come into contact with patients or bodily fluids. This includes staff and others who may not come into direct contact with patients.

Universal precautions are typically practiced in any environment where workers are exposed to bodily fluids, such as:

▪ Blood ▪ Semen ▪ Vaginal secretions	▪ Synovial fluid ▪ Amniotic fluid ▪ Cerebrospinal fluid	▪ Pleural fluid ▪ Peritoneal fluid ▪ Pericardial fluid

Bodily fluids that do not require such precautions include:

▪ Feces ▪ Nasal secretions ▪ Urine	▪ Vomitus ▪ Perspiration	▪ Sputum ▪ Saliva

Universal precautions are the infection control techniques that were recommended following the AIDS outbreak in the 1980s. Every patient is treated as if infected and therefore precautions are taken to minimize risk.

Protective clothing includes but is not limited to:

- Barrier gowns
- Gloves
- Eyewear (goggles or glasses)
- Face shields

1. Alt patients must be regarded as potentially infected with blood-borne pathogens.
2. Strict hand washing must be practiced before and after each patient contact. **Hands must be washed immediately if they are contaminated with blood or body fluids**
3. Gloves should be worn:
A. If soiling with blood or body fluids is anticipated,
b. For placement of intravenous lines.
C. Gloves should not be reused.
4. Gowns or aprons should be worn during procedures that are likely to generate splashes of blood or other body fluids and if soiling with blood or body fluids is likely.
5. Masks and protective eyewear (goggles) should be worn during procedures that are likely to generate droplets or splashes of blood or other body fluids to prevent exposure of mucous membranes of the mouth, nose arid eyes.
6. Articles contaminated with blood or body fluids should be discarded:
A. If disposable, in red bags labeled "infectious waste."
b. Non-disposable items should be cleaned with a hospital approved disinfectant and sent to Central Services for sterilization.
7. Care should be taken to avoid needle-stick injuries. Used needles should not be recapped or bent; they should be placed in a prominently labeled puncture resistant container designated specially for such disposal. You may prefer to administer all drugs, via stopcock, to avoid recapping needles.
8. Blood spills should be cleaned up promptly with a solution of 5.25% sodiumhypochforite diluted with Water (1:10 household bleach).
9. Health care workers who have exudative lesions or weeping dermatitis should refrain from all patient care and handling equipment until the condition is resolved.
10. To minimize the need for emergency mouth-to-mouth resuscitation, disposable mouth pieces, resuscitation bags or other ventilation devices should be available for use,
11. Pregnant health care workers should strictly adhere to precautions to minimize the risk of HIV transmission.
12. All specimens must be contained in a leak-proof plastic bag labeled 'bio-hazard."

(e) Malrotation of gut.

Answer. Malrotation and volvulus
Key facts and clinical features (neonates)
Malrotation

- This can present at birth or soon after and symptoms are due to rotation of the small bowel leading to duodenal obstruction. Intestine fails to undergo its normal rotation and fixation during embryologic development.
- Ladd's bands are occasionally the cause of the obstruction.
- Proximal duodenal distension leads to bile-stained vomiting.
- The caecum may be in an abnormally high or midline position.
- Incidence 1/500 to 1/6,000 live births.
- Associated anomalies are present in 30% to 59% of patients.

Volvulus

- Twisting, in clockwise direction, of malrotated, non-fixed midgut loop on its narrow-based mesentery through 360° or more.
- Results in obstruction of superior mesenteric blood vessels.
- Signs: sudden onset of abdominal pain; bile-vomiting; progression to shock; passage of blood per rectum.
- May be less dramatic. Most dangerous in newborn period because of delay in diagnosis and rapid development of gut ischaemia.
- Older children may present insidiously or as rapid onset of shock with less prominent other symptoms.

Diagnosis and investigations:

History and physical examination:

- Bilious emesis
- Hematemesis
- Abdominal distention
- Abdominal tenderness
- Hematochezia

If in doubt operate. Viability of twisted bowel is very time-dependent delays in diagnosis can be very serious.

- Plain abdominal X-ray: double bubble sign with some distal gas.
- Barium meal: obstruction of second part duodenum, non-rotation of duodenum/jejunum, and corkscrew appearance of proximal small bowel loops; absent C loop of duodenum.
- Ultrasound scan: reversed relation of superior mesenteric artery and vein.
- Doppler ultrasound: absent or abnormal small bowel blood.

Treatment:

- Resuscitation including decompression with nasogastric tube.
- Prompt surgery to avoid irreversible bowel damage.
- Laparotomy may reveal:
    - obstructed but viable bowel;
    - patchy ischaemic changes;
    - established necrosis.
- Resection of ischaemic gut may risk short gut syndrome.Second look laparotomy (24-48h) allows reassessment prior to resection.

4. Answer briefly on the following: $7^1/_2$

a) Diagnosis and management of gastro-oesophageal reflux disease.
b) Role of nutrition, its methods in surgical patients.
c) Pain relief in surgery.
d) Post splenectomy problems.

Answer.
(a) Diagnosis and management of gastro-oesophageal reflux disease.

Answer. Symptoms:

• Heartburn • Regurgitation • Abdominal pain • Cough	• Dysphagia for solids • Hoarseness • Belching • Bloating	• Aspiration • Wheezing • Globus

- The physical examination of patients with GERD rarely contributes to confirmation of the diagnosis.
- In patients with advanced disease, several observations may help identify the source of the patient's discomfort.
- A patient who constantly drinks water during the interview is facilitating esophageal clearance, which may be indicative of continual reflux or distal obstruction.
- Other patients with advanced disease will sit leaning forward and carry out the interview with their lungs inflated to near vital capacity. This is an attempt to keep the diaphragm flattened, the anteroposterior diameter of the hiatus narrowed, and, thus, the LES pressure elevated.
- Patients who have severe proximal reflux with regurgitation of gastric contents into their mouth may have erosion of their dentition (revealing yellow teeth due to the loss of dentin), injected oropharyngeal mucosa, or signs of chronic sinusitis.

Preoperative Evaluation

The preoperative workup in a patient being considered for operative treatment will help confirm the diagnosis, exclude other pathologic entities, and direct the operative intervention.

Endoscopy: Endoscopy is an essential step in the evaluation of patients with GERD who are being considered for operative intervention. The value of the study is in its ability to exclude other diseases, especially a tumor, and to document the presence of peptic esophageal injury. The degree of injury can be measured using a scoring system such as the Savary-Miller interpretation (1 indicates erythema; 2, linear ulceration; 3, confluent ulceration; and 4, stricture). The extreme of mucosal injury is Barrett's esophagus. Biopsy samples are taken to confirm the metaplastic transformation and to exclude dysplasia.

Manometry: A significant amount of information about the function of the esophageal body and the LES may be obtained from stationary esophageal manometry. The manometry catheter is a flexible tube with pressure-sensing devices (water perfused or solid state) arranged at 5-cm intervals . The upper esophageal sphincter is notoriously difficult to analyze because it migrates during the cervical phase of swallowing.

- The LES is analyzed for mean resting pressure. This may be determined in two ways: a station pull-through and a rapid pull-through. The majority of laboratories report the values recorded from the station pull-through.

- Normal pressures for a station pull-through at the LES range between 12 and 30 mm Hg. The sphincter generally relaxes to the pressure of the gastric baseline for several seconds when a swallow is initiated.
- Other information to be gained from the LES is the total length, the intra-abdominal length, and the location of the sphincter relative to the nares. The longer the length of the high-pressure zone and the longer the intra-abdominal component, the greater the barrier to reflux of gastric contents.
- The esophageal body is assessed to determine the effectiveness of peristalsis. With the four channels located at 3, 8, 13, and 18 cm above the LES, the patient is given a series (at least 10) of 5-mL aliquots of water to swallow. The peristaltic activity is reported as the percentage of initiated swallows that are transmitted to each channel successfully. Normally, a patient has greater than 80% peristalsis.
- The second characteristic of clinical importance is the amplitude of the peristaltic wave. The amplitude is simply the average of the pressures generated in the distal esophagus during effectively transmitted peristaltic waves.
- Ineffective esophageal motility (IEM) is defined as less than 60% peristalsis or distal esophageal amplitudes of less than 30 mm Hg and is often associated with significant GERD. It was traditionally thought that a 360-degree fundoplication is likely to cause an insurmountable obstruction to swallowing and to result in dysphagia, but this idea has been challenged recently.

pH Monitoring:

- The gold standard for diagnosing and quantifying acid reflux is the 24-hour pH test.
- The study is performed by placing a thin catheter containing one or more solid-state electrodes in the esophagus.

- A large amount of information may be gleaned from the study: total number of reflux episodes (pH<4), longest episode of reflux, number of episodes lasting longer than 5 minutes, extent of reflux in the upright position, and extent of reflux in the supine position. An overall score is obtained with the use of a formula that assigns a weight to each item according to its capacity to cause esophageal injury. This value, known as the DeMeester score, needs to be less than 14.7. A simpler way to determine whether abnormal reflux is occurring is to estimate the total percent time the pH is below 4 in the proximal and distal channels. The total percent time is calculated by dividing the time the pH was less than 4 by the total time of the study and multiplying by 100. In the proximal esophagus (15 cm above the LES), acid exposure normally occurs less than 1% of the time; in the distal esophagus (5 cm above the LES), it normally occurs less than 4%.

The patient's symptom diary needs to be correlated with episodes of reflux. The correlation of heartburn or chest pain with a drop in the pH has significant clinical value because it helps to confirm a cause-and-effect relationship. If there is symptom correlation with low pH measurements, the suspicion of reflux-induced disease may be confirmed, even if the total acid exposure is normal.

Esophagogram:

- The esophagogram provides valuable information in the evaluation of patients with symptoms of GERD when an operation is contemplated or when the symptoms do not respond as expected.
- Often, spontaneous reflux during the examination will be demonstrated. Although reflux may be induced in patients who do not have the disease, the occurrence of spontaneous reflux lends support to the diagnosis of abnormal gastroesophageal reflux.
- The true value of the study is to determine the external anatomy of the esophagus and the proximal stomach. The presence and size of a hiatal hernia may be characterized. Although this neither confirms nor refutes the presence of disease, it is extremely beneficial in planning the operation.
- A mediastinal gastroesophageal junction that does not reduce into the peritoneal cavity during the study is a predictor of a more difficult operation that may require an esophageal lengthening procedure. Peptic esophageal strictures may also be found on an esophageal contrast study. The presence of a stricture will taint the interpretation of the 24-hour pH study, especially if it is tight enough to prevent reflux.
- Other anatomic abnormalities, such as diverticula, tumors, and unexpected paraesophageal hernias, will be discovered during an esophagogram.

Other Tests:

In unique circumstances, other diagnostic tests may be valuable. Occasionally, a patient will not be able to tolerate nasoesophageal intubation. A scintigraphic study to evaluate esophageal clearance and reflux may provide evidence of motility disorder and gastroesophageal reflux. Gastric distention resulting from delayed emptying may also be diagnosed with a scintigraphic study. Although this condition may contribute to reflux, it is not clear whether a gastric emptying procedure (pyloroplasty) needs to be added to an antireflux procedure in a patient with delayed gastric emptying.

Some patients will have laryngeal symptoms of gastroesophageal reflux. Laryngoscopy and stroboscopic examinations will help provide objective evidence of extraesophageal reflux; findings include inflammation of laryngeal mucosa, muscle tension abnormalities, and, in severe cases, subglottic stenosis.

Treatment:

- Most people will experience symptoms of reflux during their life. A smaller percentage will proceed with self-treatment
- Lifestyle modifications are certainly helpful in avoiding gastroesophageal reflux. Cessation of smoking, decreased caffeine intake, and avoidance of large meals before lying down will help decrease transient episodes of LES relaxation.
- Elevation of the head of the bed and avoidance of constricting clothing will help prevent unfavorable pressure gradients across the gastroesophageal junction.

Medical Management:

- When a patient is first seen, a lengthy workup is not necessary if the history and examination are consistent with GERD. It would be prudent to check for chronic anemia in such a patient and to prescribe a 6-week course of acid suppression therapy. Double dose is preferred.
- If the symptoms persist after a trial of medical therapy, a more extensive evaluation, as described earlier, would be indicated. The medications available to treat acid reflux include antacids, motility agents, histamine-2 (H_2) blockers, and proton pump inhibitors.
- Pharmacologic treatment of GERD has been revolutionized by the advent of proton pump inhibitors. These drugs act by irreversibly binding the proton pump in the parietal cells of the stomach, thus effectively stopping gastric acid production. The maximal effect occurs after about 4 days of therapy, and the effects will linger for the life of the parietal cell. Thus, the acid suppression will persist for 4 to 5 days after therapy has ended. For this reason, the patient needs to be off therapy for 1 week before being evaluated with pH monitoring.
- Compared with H_2 blockers, proton pump inhibitors are more effective at healing esophageal ulceration secondary to acid exposure. The medications are relatively expensive but are well tolerated. The side effects may include headache, abdominal pain, and diarrhea.
- Long-term therapy appears to be safe even though it has been linked to gastric polyp formation. The polyps are usually hyperplastic and do not appear to be premalignant.

Surgical Therapy:

- The indications for surgical therapy have changed somewhat with the advent of proton pump inhibitors.
- Certainly, patients with evidence of severe esophageal injury (ulcer, stricture, or Barrett's mucosa) and incomplete resolution of symptoms or relapses while on medical therapy are appropriate to consider for operative intervention.
- Other patients with a long duration of symptoms or those in whom symptoms persist at a young age are considered for operative treatment initially. In these patients, operative

therapy is considered an alternative to medical therapy rather than a treatment of last resort.

- Some patients have absolutely no response of their symptoms to the use of proton pump inhibitors. They need to be scrutinized further before offering surgical treatment, as opposed to being considered medical failures who need operative treatment. Because the proton pump inhibitors are so effective at decreasing the acid production of the stomach, the diagnosis of GERD in such patients is questioned and must be demonstrated with objective testing.

In patients who have more than 10 years of life expectancy and are in need of lifelong therapy due to a mechanically defective sphincter, surgical therapy may be considered the treatment of choice.

360-Degree Wrap (Left Crus Approach):

The technique described here is the left crus approach to a 360-degree wrap (Nissen fundoplication), which is the procedure of choice for most patients. The left crus approach provides the advantage of a direct and early view of the short gastric vessels and the spleen. After this obstacle is negotiated, there is little chance of injuring the spleen during the remainder of the procedure.

Partial Fundoplication

- When esophageal motility is poor, a partial fundoplication may be considered to prevent obstruction to bolus propagation in the esophagus. Although this was thought mandatory in all patients with IEM (peristalsis <60% or distal esophageal amplitudes <30 mm Hg), this practice has been questioned in recent years.
- A total fundoplication can be performed in most patients with IEM (except perhaps those with absent peristalsis), without an increase in development of dysphagia. In fact, effective control of reflux with a total fundoplication usually improves premorbid dysphagia and often improves the esophageal motility. When needed, there are many types of partial fundoplications. Regardless of the type used, the initial dissection of the esophagus is the same.

If an anterior wrap (e.g., Thal, Dor) is to be performed, there is no need to disrupt the posterior attachments of the esophagus. The Dor and Thal fundoplications are created with the fundus folded over the anterior aspect of the esophagus. They are anchored to the hiatus and esophagus as in the 360-degree wrap. The experience with these repairs is limited in patients being treated for gastroesophageal reflux. They are more commonly used in patients with achalasia after an anterior myotomy has been performed.

If a posterior wrap (Toupet) is to be performed, the entire esophageal dissection is the same as for a 360-degree wrap, and the crura are reapproximated as well. The reconstruction of the posterior fundoplication is initiated by passing the posterior fundus behind the esophagus from left to right. The fundoplication is created by anchoring the posterior fundus to the crura and the esophagus. The most cephalad sutures of the wrap incorporate all three structures (fundus, crus, esophagus). The wrap is anchored posteriorly to the crura with two or three sutures. The fundus is then sutured to the esophagus along the anterolateral aspects, creating a 220- to 250-degree wrap.

Endoscopic Therapy

- Recently, several endoscopic techniques have been developed for the treatment of GERD. These procedures have sparked significant interest because they each promise a mechanical treatment for reflux with less invasion than a fundoplication. These techniques attempt to augment the LES by suturing, or biocompatible polymer injection.
- There is some interest in using these endoscopic techniques in patients with recurrent GERD after antireflux surgery. Although it is tempting to avoid complicated reoperation, there is not enough experience to recommend its use at this time. Overall, it is not clear what role endoscopic treatments have in the treatment of GERD; therefore, laparoscopic antireflux surgery remains the best alternative to medical therapy.

Outcome: The results of operative intervention may be measured by relief of symptoms, improvement in acid exposure, complications, and failures.

b) Role of nutrition , its methods in surgical patients.

Answer. Role of nutrition in surgical patients: Nutrition plays a vital and often overlooked role in the care of patients on a surgical service. Between 30% and 50% of hospitalized patients are malnourished, and malnutrition is clearly associated with increased morbidity and mortality in select general surgical patients. Although most healthy patients can tolerate 7 days of starvation (with adequate glucose and fluid replacement), those who have been subject to major trauma, a prolonged operative course, sepsis, cancer-related cachexia, or other physiologic stressors require earlier nutritional intervention.
Indications for nutritional support should consider the following:

1. The patient's premorbid state (healthy or otherwise)
2. Poor nutritional status (current oral intake meeting <50% of total energy needs)
3. Significant weight loss (initial body weight less than usual body weight by 10% or more or a decrease in inpatient weight by more than 10% of the admission weight
4. The duration of starvation (>7 days' inanition)
5. An anticipated duration of artificial nutrition (particularly total parenteral nutrition [TPN]) of longer than 7 days
6. The degree of the anticipated insult, surgical or otherwise
7. A serum albumin value less than 3.0 g/dL measured in the absence of an inflammatory state
8. A transferrin level of less than 200 mg/dL
9. Anergy to injected antigens

Clinical Sequelae of Impaired Nutrition: Numerous studies have clearly shown an increased incidence of nosocomial infection, longer hospital stay, and increased mortality in patients with significant unintentional weight loss (>10%) before their acute illness. Even in an individual with initially normal nutritional status, after 7 to 10 days of inanition, the body's ability to heal wounds and to support normal immune function begins to be impaired. Such deficits include diminished complement and immunoglobulin production,

poor cellular immunity, and impairment of various aspects of leukocyte action, including chemotaxis, phagocytosis, and oxidative burst. Other consequences of inadequate nutrition in the postoperative period include poor tissue repair and wound healing and loss of muscle function and strength as a result of progressive muscle wasting, which may contribute to reduced ventilatory performance and prolonged ventilator dependence. Overall, malnutrition will be limiting to all aggressive surgical and medical therapies.

Methods:

Enteral nutrition:
In general, the enteral route is preferred over the parenteral route. Enteral feeding is simple, physiologic, relatively inexpensive, and well tolerated by most patients. Enteral feeding maintains the GI tract cytoarchitecture and mucosal integrity (via trophic effects), absorptive function, and normal microbial flora. This results in less bacterial translocation and endotoxin release from the intestinal lumen into the bloodstream. Enteral feedings are indicated for patients who have a functional GI tract but are unable to sustain an adequate oral diet. Enteral feedings may be contraindicated in patients with an intestinal obstruction, ileus, GI bleeding, severe diarrhea, vomiting, enterocolitis, or a high-output enterocutaneous fistula. Choice of appropriate feeding site, administration technique, formula, and equipment may circumvent these problems.

- Feeding tubes. Nasogastric, nasojejunal (e.g., Dobhoff), gastrostomy, and jejunal tubes are available for the administration of enteral feeds. Percutaneous gastrostomy tubes can be placed endoscopically or under fluoroscopy.
- Enteral feeding products. A variety of commercially available enteral formulas are available. Standard solutions provide 1 kcal/mL; calorically concentrated solutions (>1 kcal/mL) are available for patients who require volume restriction. The available dietary formulations for enteral feedings can be divided into polymeric (blenderized and nutritionally complete commercial formulas), chemically defined formulas (elemental diets), and modular formulas.
    - Blenderized tube feedings can contain any food that can be blenderized. Caloric distribution of these formulas should parallel that of a normal diet.
    - Nutritionally complete formulas (standard enteral diets) vary in protein, carbohydrate, and fat composition. Several formulas use sucrose or glucose as carbohydrates and are suitable for lactose-deficient patients. Commercial formulas are convenient, sterile, and inexpensive. They are recommended for patients with minimal metabolic stress and normal gut function.
    - Chemically defined formulas (elemental diets). The nutrients are provided in predigested and readily absorbed form. They contain protein in the form of free amino acids or polypeptides. They are hyperosmolar, which may cause cramping and diarrhea. Elemental diets are efficiently absorbed in the presence of compromised gut function. However, they are costlier.
    - Modular formulations are designed for use in specific clinical situations (e.g., pulmonary, renal, or hepatic failure or immune dysfunction).
    - Conversion to oral feeding. When indicated, an oral diet is resumed gradually. In an effort to stimulate appetite, enteral feeding can be modified by the following measures:
        - Providing fewer feedings.
        - Holding daytime feedings.

        - Decreasing the volume of feedings. When oral intake provides approximately 75% of the required calories, tube feedings can be stopped.
    - Administration of medications. Many oral medications can be administered through feeding tubes. The elixir form is preferred but is not always available. Medications that are not suitable for administration through a feeding tube include the following:
        - Enteric-coated medications.
        - Drugs in gelatinous capsules.
        - Medications that are designed for sublingual use.
        - Most sustained-release medications.

Parenteral nutrition:
Parenteral nutrition is indicated for patients who require nutritional support but cannot meet their needs through oral intake and for whom enteral feeding is contraindicated or not tolerated.

- Peripheral parenteral nutrition (PPN) is administered through a peripheral intravenous catheter. The osmolarity of PPN solutions generally is limited to 1,000 mOsm (approximately 12% dextrose solution) to avoid phlebitis. Consequently, unacceptably large volumes (>2,500 mL) are necessary to meet the typical patient's nutritional requirements. Temporary nutritional supplementation with PPN may be useful in selected patients but is not typically indicated.
- Total parenteral nutrition (TPN) provides complete nutritional support. The solution, volume of administration, and additives are individualized based on an assessment of the nutritional requirements.
    - Access. TPN solutions must be administered through a central venous catheter. A dedicated single-lumen catheter or a multilumen catheter can be used. Catheters should be replaced for unexplained fever or bacteremia.
    - TPN solutions. TPN solutions generally are administered as a 3-in-1 admixture of protein, as amino acids (10%; 4 kcal/g); carbohydrate, as dextrose (70%; 3.4 kcal/g); and fat, as a lipid emulsion of soybean or safflower oil (20%; 9 kcal/g). Alternatively, the lipid emulsion can be administered as a separate intravenous “piggyback” infusion. Standard preparations are used for most patients. Special solutions that contain low, intermediate, or high nitrogen concentrations as well as varying amounts of fat and carbohydrate are available for patients with diabetes, renal or pulmonary failure, or hepatic dysfunction.
    - Additives. Other elements can be added to the basic TPN solutions.
        - Electrolytes (i.e., sodium, potassium, chloride, acetate, calcium, magnesium, phosphate) should be adjusted daily. A suggested formulation is often listed on a prewritten order sheet, with concentrations designed for patients with normal serum electrolytes and renal function. The number of cations and anions must balance; this is achieved by altering the concentrations of chloride and acetate. If the serum bicarbonate is low, the solution should contain more acetate. The calcium:phosphate ratio must be monitored to prevent salt precipitation. Medications such as albumin, H_2-receptor antagonists, heparin, iron, dextran, insulin, and metoclopramide can be administered in TPN solutions. However, not all medications are compatible with 3-in-1 admixtures. Regular insulin should initially be administered subcutaneously according to a sliding scale, based on a determination of the blood glucose level. After a stable insulin requirement has been

established, insulin can be administered in the TPN solution, generally at two thirds of the daily subcutaneous insulin dose.

- Other additives. Trace elements are added daily using a commercially prepared mixture (e.g., 1 mL trace element-5: 1 mg copper, 12 μg chromium, 0.3 μg manganese, 60 μg selenium, 5 mg zinc). Multivitamins generally are added daily using a commercially prepared mixture (e.g., 10 mL MVI-12). Vitamin K is not included in most multivitamin mixtures and must be added separately (10 mg once a week). Vitamins A and C and zinc are essential for proper wound healing.

- Routine physiologic and laboratory monitoring should occur on a scheduled basis. This can be performed less frequently for patients whose postoperative course has stabilized and who are receiving a consistent TPN regimen.
  The initial frequency of monitoring includes vital signs and serum glucose every 6 hours; weight, serum electrolytes, and blood urea nitrogen daily; and triglycerides, complete blood cell count, prothrombin time, liver enzymes, and bilirubin weekly.
- Administration of TPN. Orders, written daily, should reflect the patient's dynamic nutritional status and biochemical profile.
  - Introduction of TPN should be gradual. For example, approximately 1,000 kcal is provided the first day. If there is metabolic stability (i.e., normoglycemia), this is increased to the caloric goal over 1 to 2 days.
  - TPN solutions are delivered most commonly as a continuous infusion. A new 3-in-1 admixture bag of TPN is administered daily at a constant infusion rate over 24 hours. Additional maintenance intravenous fluids are unnecessary, and total infused volume should be kept constant while nutritional content is increased.
  - Cyclic administration of TPN solutions may be useful for selected patients, including (1) those who will be discharged from the hospital and subsequently receive home TPN, (2) those with limited intravenous access who require administration of other medications, and (3) those who are metabolically stable and desire a period during the day when they can be free of an infusion pump. Cyclic TPN is administered for 8 to 16 hours, most commonly at night. This should not be done until metabolic stability has been demonstrated for patients on standard, continuous TPN infusions.
  - Discontinuation of TPN should take place when the patient can satisfy 75% of his or her caloric and protein needs with oral intake or enteral feeding. The calories provided by TPN can be decreased in proportion to calories from the patient's increasing enteral intake. To discontinue TPN, the infusion rate should be halved for 1 hour, halved again the next hour, and then discontinued. Tapering in this manner prevents rebound hypoglycemia from hyperinsulinemia. It is not necessary to taper the rate if the patient demonstrates glycemic stability when TPN is abruptly discontinued (i.e., cycled TPN) or receives less than 1,000 kcal/day.

(c) Pain relief in surgery.

Answer. Most people have some pain after surgery. Pain can be harmful if it is not relieved after surgery. Pain can make it hard for the patient to take deep breaths, move and walk. This can cause problems like blood clots and pneumonia.

Pain control after surgery

- Keep patient's pain under control.
- Patient should be comfortable so that he or she can sleep, deep breathe, walk and visit with others.
- Manage pain to help the patient heal after surgery.

Different medicines used to control pain
After surgery, different medicines can be used to control pain. They work together so that less medicine is needed while getting better pain control.
Pain medicines: Patient may be given one or more different pain medicines, like opiate and NSAIDs.
Anti-anxiety medicines
Anti-nausea medicines
Patient Controlled Analgesia (PCA): Patient controlled analgesia (PCA) is a special pump that has pain medicine in it that connects to Patient's IV (intravenous) tube. When patient feels pain, he or she pushes a button and the pump gives a measured dose of medicine into your bloodstream. Only patient should push the button, not the family members or friends.
Epidural Analgesia
Epidural analgesia is another way pain medicine is given. A small plastictube called an epidural catheter is placed in patient's back just before surgery. A small pump is attached to the epidural catheter. The pump puts a small amount of analgesic medicine through the catheter in patient's back at a set rate. One can use epidural medicine until the pain has eased enough to take pain pills by mouth.
Local anaesthetic block can also be used.

Acute postoperative pain relief:

- Requires an expert team approach.
- Pain levels must be measured regularly.
- Analgesia is best given before pain breaks thtough.
- A combination of analgesics gives best result.
- Opioids should not be withheld.
- The dose of analgesia used must be adequate to control the pain.

Techniques for postoperative pain relief:

- Regular intramuscular injections – may get pain breakthrough.
- Local anaesthetic block – ideal if it works.
- Indwelling epidural – good pain control; opioids may depress respiration.
- Continuous infusions – reduce oscillations in pain relief but risk overdose.
- Patient-control-analgesia – pain relief titrared to patient's need.

(d) Post splenectomy problems.

Answer. Postoperative Complications Following Splenectomy

Bleeding Complications	**Pulmonary Complications**	**Septic Complications**	**Others**	
Intraoperative bleeding (requiring transfusion)	Atelectasis	Wound infection	Deep venous thrombosis	Postoperative ileus
Postoperative bleeding	Pneumonia and upper respiratory tract infection	Deep line sepsis	Gout flare	Hypophyseal insufficiency

Abdominal wall hematoma	Pulmonary embolus	Urinary tract infection	Myocardial infarction	Gastric stasis
Hemothorax	Pneumothorax		Vocal cord damage	Pancreatic tail injury

- Overwhelming systemic sepsis(opportunistic post splenectomy infection) usually associated with gram-positive encapsulated bacteria such as ***Streptococcus*.** The incidence of overwhelming postsplenectomy sepsis is highest in children and has been reported as high as 4%. The mortality rate from overwhelming postsplenectomy sepsis is approximately 50%. Increasing the incidence in adults are those who have undergone splenectomy for an associated malignancy or as an incidental procedure.

In children, those most at risk are those who have undergone splenectomy for either congenital or acquired anemias. The most common etiologic organisms are ***Streptococcus pneumoniae, Haemophilus influenzae*,** and ***Neisseria meningitis*.** As a result, vaccination against these organisms has been advocated. The timing of these vaccinations has been debated; however, most data would suggest that preoperative (typically 2 weeks) vaccination is best. When this is not possible, recent data have suggested that vaccination should be delayed until 2 weeks postoperative.
Given that the highest incidence of overwhelming postsplenectomy sepsis has been shown to be in the first asplenic year, some also advocate that low-dose prophylactic penicillin be provided to these patients the first year after splenectomy. Fever and rigor in an asplenic patient must prompt immediate aggressive empirical treatment with antibiotic coverage, even in the absence of culture data.

- Currently, the standard of care for postsplenectomy patients includes immunization with polyvalent pneumococcal vaccine (PPV23), ***H. influenzae*** type b conjugate, and meningococcal polysaccharide vaccine within 2 weeks of splenectomy. Even with vaccination, the development of a protective level of antibody against pneumococci is only about 50%. Currently available vaccines elicit a T-cell–independent response and do not produce a sustained increase in antibody titers. Subsequently, there is a lack of evidence that indicates a substantial increase in protection in most revaccinated persons.
- Therefore, routine revaccination of immunocompetent persons who were previously vaccinated with PPV23 is not recommended by the Centers for Disease Control and Prevention (CDC). Revaccination is, however, recommended in high-risk individuals. Candidates for revaccination with PPV23 include the following .

  - Persons who received the 14-valent vaccine who are at highest risk for fatal pneumococcal infection (e.g., asplenic patients)
  - Adults at highest risk who received the 23-valent vaccine 6 years prior
  - Adults at highest risk who have shown a rapid decline in pneumococcal antibody levels (e.g., patients with nephrotic syndrome, those with renal failure, and transplant recipients)
  - Children at highest risk (asplenia, nephritic syndrome, sickle cell anemia) who would be 10 years old at revaccination

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES
MS (General Surgery) Examination, 2008
PAPER I

Time Allowed: 3 Hours **Full Marks: 100**

Attempt all questions

1. Discuss the pathophysiological basis of fluid and electrolyte abnormalities in gastric outlet obstruction. How do you prepare a patient of such a condition before Surgery? (10+10)
2. Describe the anatomy of anal sphincter. How do you evaluate its function? (10+10)

3. Write short notes on: 5 x 6

a) AIDS and the Surgeon.
b) Pulmonary embolism.
c) Principles of diathermy.
d) Prophylactic antibiotics.
e) Systemic inflammatory response.

4. Answer briefly of the followings: 4 x $7^1/_2$
a) Pharmacotherapy of benign prostatic hypertrophy.
b) Repair of hypospadius.
c) Vaious syndromes associated with medullary carcinoma thyroid.
d) Tetanus prophylaxis.

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May 2008

PAPER I

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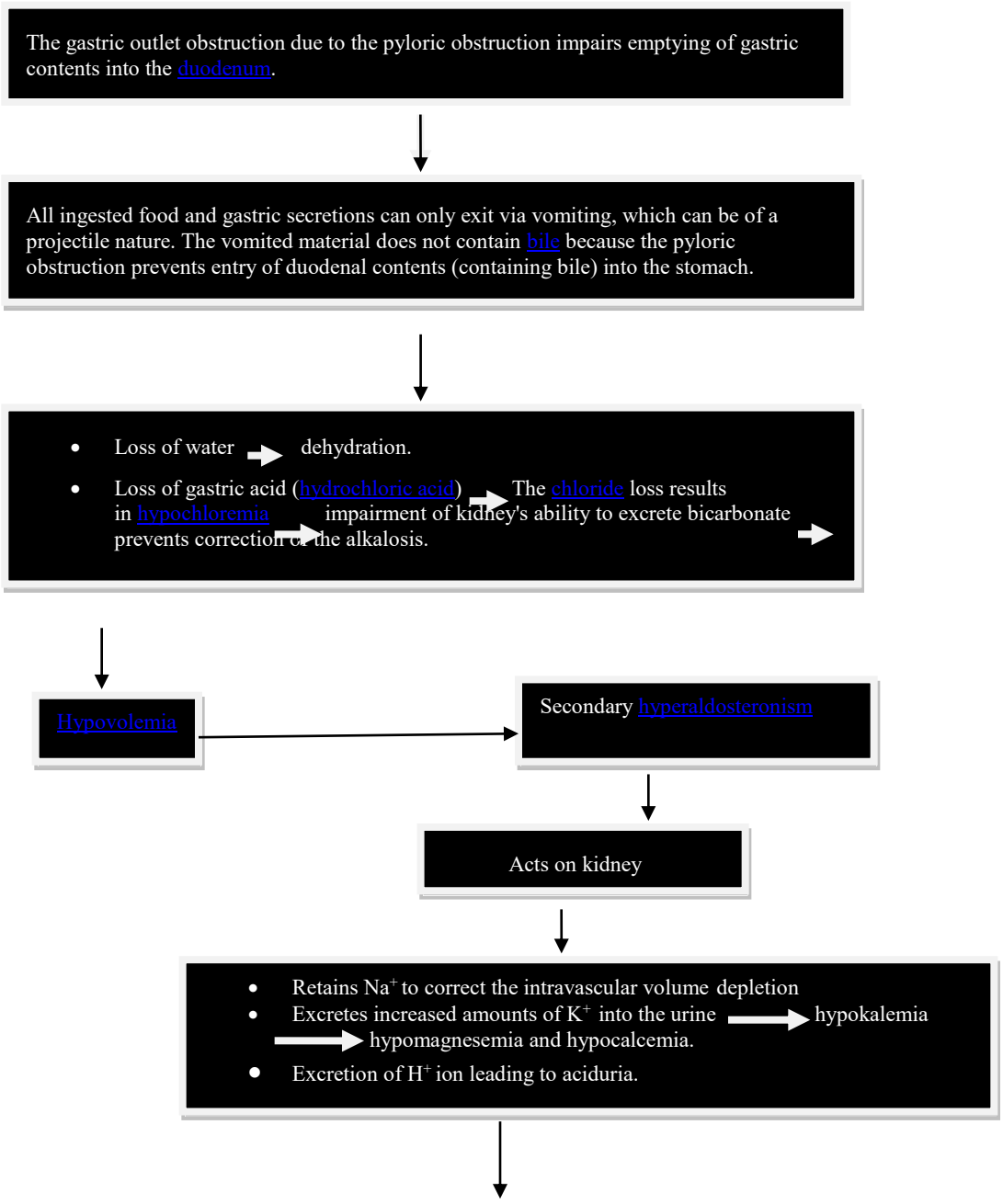
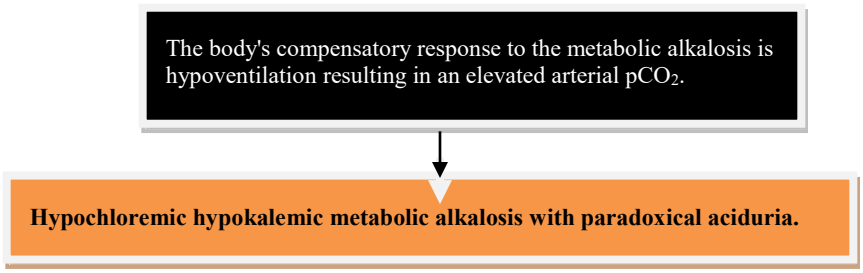
1. Discuss the pathophysiological basis of fluid and electrolyte abnormalities in gastric outlet obstruction. How do you prepare a patient of such a condition before Surgery. (10+10)

Answer.





Causes of GOO

Benign causes	Malignant causes
■ PUD ■ Gastric polyps ■ Ingestion of caustics ■ Pyloric stenosis ■ Congenital duodenal webs ■ Gallstone obstruction (bouveret syndrome) ■ Pancreatic pseudocysts ■ Bezoars ■ Gastric TB ■ Gastric crohn's	■ Pancreatic cancer ■ Carcinoma gall bladder ■ Ampullary cancer ■ Duodenal cancer ■ Cholangiocarcinoma ■ Gastric cancer ■ Lymphoma ■ Metastases to the gastric outlet

Preoperative management:

- First and foremost is hydration and correction of electrolyte imbalances
- Sodium chloride solution: initial IV fluid of choice.
- Potassium deficits must be corrected.
- NG tube: to decompress stomach.
- Nutrition should be maintained – if needed TPN.
- For malignant lesions anemia should be corrected – Hb% should be at least – 8gm%.
- To investigate for finding out the actual cause.

2. Describe the anatomy of anal sphincter. How do you evaluate its function? (10+10)

Answer. Surgical anatomy: The anal canal commences at the level where the rectum passes through the pelvic diaphragm and ends at the anal verge. The muscular junction between the rectum and anal canal can be felt with the finger as a thickened ridge – the anorectal 'bundle' or 'ring'.

Anal canal anatomy:

The anorectal ring

The anorectal ring marks the junction between the rectum and the anal canal. It is formed by the joining of the puborectalis muscle ,the deep external sphincter, conjoined longitudinal muscle and the highest part of the internal sphincter. The anorectal ring can be clearly felt digitally, especially on its posterior and lateral aspects.

The puborectalis muscle

Puborectalis, part of the funnel-shaped muscular pelvic diaphragm, maintains the angle between the anal canal and rectum and hence is an important component in the continence mechanism . The muscle derives its nerve supply from the sacral somatic nerves, and is functionally indistinct from the external anal sphincter. The position and length of the anal canal, as well as the angle of the anorectal junction, depend to a major extent on the integrity and strength of the puborectalis muscle sling. It gives off fibres that contribute to the longitudinal muscle layer.

The external sphincter

The external sphincter forms the bulk of the anal sphincter complex and, although traditionally it has been subdivided into deep, superficial and subcutaneous portions, it is a single muscle(Goligher), which is variably divided by lateral extensions from the longitudinal muscle layer. Some of its fibres are attached posteriorly to the coccyx, whereas anteriorly they fuse with the perineal muscles. Being a somatic voluntary muscle, the external sphincter is red in colour and is innervated by the pudendal nerve.

The intersphincteric plane

Between the external sphincter muscle laterally and the longitudinal muscle medially exists a potential space, the intersphincteric plane. This plane is important as it contains intersphincteric anal glands and is also a route for the spread of pus, which occurs along the extensions from the longitudinal muscle layer. The plane can be opened up surgically to provide access for operations on the sphincter muscles.

The longitudinal muscle

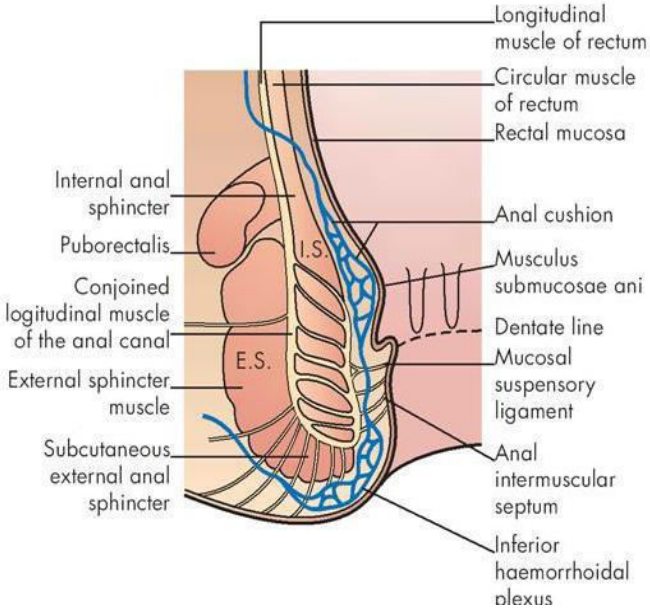
The longitudinal muscle is a direct continuation of the smooth muscle of the outer muscle coat of the rectum, augmented in its upper part by striated muscle fibres originating from the medial components of the pelvic floor. Most of the muscle continues caudally before splitting into multiple terminal septa that surround the muscle bundles of the subcutaneous portion of the external sphincter to insert into the skin of the lowermost part of the anal canal and adjacent perianal skin. Milligan and Morgan named the most medial of these septa, passing around the inferior border of the internal sphincter, the 'anal intermuscular septum'. As it descends, however, it gives off fibres that pass medially across the internal sphincter to reach the submucosal space, and laterally across the external sphincter and ischiorectal space to reach the fascia of the pelvic side walls. As well as providing a supportive mesh for the anal canal and other muscular components, its ramifications provide potential pathways for the spread of infection. During defaecation, its contraction widens the anal lumen, flattens the anal cushions, shortens the anal canal and everts the anal margin; subsequent relaxation allows the anal cushions to distend and thus contribute to an airtight seal.

The internal sphincter

The internal sphincter is the thickened (2–5 mm) distal continuation of the circular muscle coat of the rectum, which has developed special properties and which is in a tonic state of contraction. This involuntary muscle commences where the rectum passes through the pelvic diaphragm and ends above the anal orifice, its lower border palpable at the intersphincteric groove, below which lie the most medial fibres of the subcutaneous external sphincter, and separated from it by the anal intermuscular septum. When exposed during life, it is pearly-white in colour and its circumferentially placed fibres can be seen clearly. Although innervated by the autonomic nervous system, it receives intrinsic non-adrenergic and non-cholinergic (NANC) fibres, stimulation of which causes release of the neurotransmitter nitric oxide, which induces internal sphincter relaxation.

The epithelium and sub-epithelial structures: The pink columnar epithelium lining the rectum extends through the anorectal ring into the surgical anal canal. Passing downwards the mucous membrane becomes cuboidal and redder in colour whereas above the anal valves it is plum coloured. Just below the level of the anal valves there is an abrupt, albeit wavy, transition to stratified squamous epithelium, which is parchment coloured. This wavy junction constitutes the dentate line. The dentate line is a most important landmark both morphologically and surgically, representing the site of fusion of the proctodaeum and post-allantoic gut, and being the site of the crypts of Morgagni (synonym: anal crypts, sinuses). The latter are small pockets between the inferior extremities of the columns of Morgagni through which anal ducts that communicate with deeper placed anal glands open into the anal lumen. The squamous epithelium lining the lower anal canal is thin and shiny and is known as the anoderm; it differs from the true skin in that it has no epidermal appendages, i.e. hair and sweat glands. At the dentate line, the anoderm is attached more firmly to deeper structures. The mucosa and submucosa above the dentate line is uneven and thrown into folds, the so called anal cushions. There are variations in the numbers and positions of these cushions but there are usually three, corresponding to those seen in later life. These are described classically as occupying the left lateral, right posterior and right anterior positions, and they continue proximally as the primary rectal foldings. Secondary foldings (the rectal columns of Morgagni) lie both over and between the primary folds. This area is the caudal limit of the so called epithelialtransitional zone, below which the stratified squamous epithelium is richly innervated by sensory nerve endings serving several modalities including touch, pain and temperature. The bulk of the anal cushions themselves, situated in the upper part of the anal canal, receive only visceral afferent innervation and, although there is perception of stretching, sensitivity to noxious stimuli is much more blunted than distally. Between the epithelial layer and the internal sphincter lies the submucosa, consisting of vascular, muscular and connective tissue supportive elements. From the longitudinal muscle, medial extensions cross the internal anal sphincter and form part of the supporting meshwork of the submucosa, blending with the true submucosal smooth muscle layer and thereby supporting the mucosa itself. Parks described the increased density of fibres that insert into the mucosa of the anal crypts at the level of the dentate line, termed the ‘mucosal suspensory ligament’. One feature of this structure is that it separates the superior (portal) and inferior (systemic) haemorrhoidal plexuses, another is that the

mucosa is more firmly tethered to underlying tissues at this level than above. It is important to appreciate that the meshwork of supporting tissues (muscle fibres and connective tissue) within the subepithelial space is intimately linked to deeper structures within the anal sphincter complex, including the internal sphincter, longitudinal muscle layer and external anal sphincter, and indeed structures beyond the sphincter complex. With age, the smooth muscle component of this mesh is reduced and muscle fibres are gradually replaced with fibroelastic connective tissue, which in turn becomes fragmented.



Relevant anatomy of the anus

Blood supply

In addition to the meshwork support of the lining of the anal canal, the subepithelial space contains venous dilatations supported by the same fibroelastic connective tissue and smooth muscle scaffolding. Debate has centred on the nature of the vascular component of haemorrhoids, but the seminal anatomical studies of Thomson have clarified this issue. Venous dilatations are seen in the submucosa both above and below the level of the dentate line; they are much more numerous above although tend to be larger below. The historical description of the blood supply to the upper anal canal as constant, with bifurcation of the main trunk of the superior rectal artery into right and left branches and with subsequent division of the former into anterior and posterior divisions thereby determining the sites of haemorrhoids around the anal circumference. Later it was found that the divisions of the superior rectal artery were not constant and that, furthermore, the anal submucosa may sometimes can receive blood supply from the middle and inferior rectal arteries. There is also presence of free communications between tributaries of the superior, middle and inferior rectal veins, as well as tiny direct arteriovenous communications with the submucosal venous dilatations. These communications have been shown both histologically and radiologically, and the oxygen tension of the blood contained within the venous dilatations (as well as the colour) is more arterial than venous.

Venous drainage

The anal veins are distributed in a similar fashion to the arterial supply. The upper half of the anal canal is drained by the superior rectal veins, tributaries of the inferior mesenteric vein and thus the portomesenteric venous system, and the middle rectal veins, which drain into the internal iliac veins. The inferior rectal veins drain the lower half of the anal canal and the subcutaneous perianal plexus of veins: they eventually join the internal iliac vein on each side.

Lymphatic drainage

Lymph from the upper half of the anal canal flows upwards to drain into the postrectal lymph nodes and from there goes to the para-aortic nodes via the inferior mesenteric chain. Lymph from the lower half of the anal canal drains on each side first into the superficial and then into the deep inguinal group of lymph glands. However, if the normal flow is blocked, e.g. by tumour, the lymph can be diverted into the alternative route.

Examination of the anus:

- Careful clinical examination will be diagnostic in the vast majority of patients complaining of anal symptoms but it requires arelaxed patient who is informed of what the examination will entail, a private environment, a chaperone (for the security of both parties) and good light.
- Most commonly, the patient is examined in the left lateral (Sims) position with the buttocks overlying the edge of the examination couch and with the axis of the torso crossing, rather than parallel with, the edge of the couch.
- Alternatively, in younger patients, the prone jack-knife or knee–elbow positions may be used. The examining couch should be of sufficient height to allow easy inspection and access for any necessary manoeuvres. A protective glove should be worn.

Inspection

- The buttocks are gently parted to allow inspection of the anus and perineum: the presence of any skin lesions and whether they are confined to the perineum or evident elsewhere on general examination, e.g. psoriasis, lichen planus, or on genital examination, e.g. warts, candidiasis, lichen sclerosus et atrophicus, the vesicles of herpes simplex virus (HSV); evidence of anal leakage; whether the anus is closed or patulous; and the position of the anus and perineum at rest and on bearing down (the latter may reveal prolapse of haemorrhoids or even the rectum).
- Pain on parting the buttocks, perhaps together with the presence of a sentinel tag, may indicate the presence of an underlying fissure, but may also prompt the need for examination under anaesthesia to exclude more suspicious pathology, for example squamous cell carcinoma of the anal canal.

Digital examination with the index finger

With an adequately lubricated index finger, the soft tissues around the anus are palpated for induration, tenderness and subcutaneous lesions. The index finger is then introduced gently into the anal canal along its posterior aspect. At the apex of the canal, the sling of puborectalis is felt posteriorly; supralevator induration feels bony hard and is more easily appreciated if unilateral. The posterior surface of the prostate gland with its median sulcus can be palpated anteriorly in male patients; in female patients, the
uterine cervix can be palpated. The presence of any distal intrarectal, intra-anal or extraluminal mass is recorded. Sphincter length, resting tone and voluntary squeeze are assessed. On withdrawalmthe examining finger is inspected for the presence of mucus, blood or pus and to identify stool colour.

Proctoscopy

Proctoscopy, performed with the patient in the same position, allows a detailed inspection of the distal rectum and anal canal. Minor procedures can also be carried out through this instrument, e.g. treatment of haemorrhoids by injection or banding (see below) and biopsy. Asking the patient to bear down on slow withdrawal of the proctoscope may reveal a descending intussusceptions.

Sigmoidoscopy

Although this is strictly an examination of the rectum, it should always be carried out even when an anal lesion has been confirmed. Rectal pathology, e.g. colitis or carcinoma, is

frequently associated with an anal lesion, e.g. fissure or haemorrhoids. Not infrequently, rectal pathology is found that is independent of the anal lesion and which requires treatment.

Physiological Aspects of The Anal Sphincters And Pelvic Floor, And Special Investigations:

- Anal continence and defaecation are highly complex processes that necessitate the structural and functional integrity of the cerebral, autonomic and enteric nervous systems, the gastrointestinal tract (especially the rectum) and the pelvic floor and anal sphincter complex, any of which may be compromised and lead to disturbances of function of varying severity.
- The sphincter mechanism provides the ultimate barrier to leakage and its integrity can be assessed fairly simply and objectively in the physiology laboratory (Swash and Henry). Perineal position and degree of descent on straining (markers of pelvic floor and pudendal nerve function) can be quantified, and functional anal canal length, resting tone (reflective predominantly of internal sphincter activity) and squeeze increment (reflective of external sphincter function) can be measured by a variety of simple manometric techniques.
- The structural integrity of the sphincters can be visualised with endoluminal ultrasound, and neuromuscular function can be measured by assessment of conduction velocity along the pudendal nerve on each side, or, more painfully, by needle electromyogram (EMG) studies.
- In the elderly especially, but also in younger patients, disorders relating to rectal sensorimotor dysfunction can lead to 'overflow' of rectal contents through what may be an otherwise normal sphincter.
- The dynamics of defaecation can also be assessed radiologically by evacuation proctography, in which radio-opaque pseudo-stool is inserted into the rectum and the patient asked to rest, squeeze and then bear down to evacuate the rectal contents under real-time imaging.
- Proctography can be combined with synchronous EMG and pressure studies (Williams) to yield more information about possible reasons [mechanical (rectocoele, intussusception) or functional (anismus, lack of effort)] for disordered defaecation in an individual. Results of all physiological tests have to be compared with a normal range and within the context of the patient's symptoms, and are used to guide rational rather than empirical treatment strategies.

Electromyography:

Single fiber density determination is of historical interest and is also extremely painful for the patient. It is not routinely performed.Pudendal nerve terminal motor latency determination measures the conduction velocity of the nerve action potential through the terminal 4 cm of the pudendal nerve between Alcock's canal and the external sphincter. A delay in conduction reflects injury to the fast-conducting fibers of the nerve. This injury usually is the result of stretch, direct trauma, or systemic disease. The normal terminal motor latency is 2.2 ± 0.2 ms. Any delay in conduction velocity greater than this indicates nerve injury.

Transrectal Ultrasound

The most sensitive method for documenting sphincter injury may be the anal ultrasound using a 360° rotating 10-MHz transducer covered with an anal cap and inserted into the anal canal. The focal length of the anal probe is approximately 1–2 cm and allows evaluation of the anal

sphincter muscles in three dimensions as the probe is withdrawn from the rectum Scarring at the site of an injury is usually easily seen by endoanal ultrasound. A rectovaginal fistula can also be detected. An algorithm for the evaluation and management of anal incontinence can be produced using these diagnostic techniques.

3. Write short notes on: 5 x 6

a) AIDS and the Surgeon.
b) Pulmonary embolism.
c) Principles of diathermy.
d) Prophylactic antibiotics.
e) Systemic inflammatory response.

Answer.

(a) AIDS and the Surgeon.

Answer. Introduction: The incidence of HIV is steadily increasing in India. Screening of these patients by routine testing of all patients is not possible; so Universal Work Precautions take an important place to prevent transmission of HIV in the health care settings.Fundamentally this prevention can be achieved by the following means:

Barrier precautions:

- Use of gloves – A surgeon needs to wear gloves while doing any of the following procedures:
- Dressing a wound.
- Starting an IV drip.
- Taking blood samples.
- Doing any operative procedures.
- Doing a PR, PV or examination of the oral cavity.

Literature shows that two pairs of gloves afford better protection than one pair of gloves.

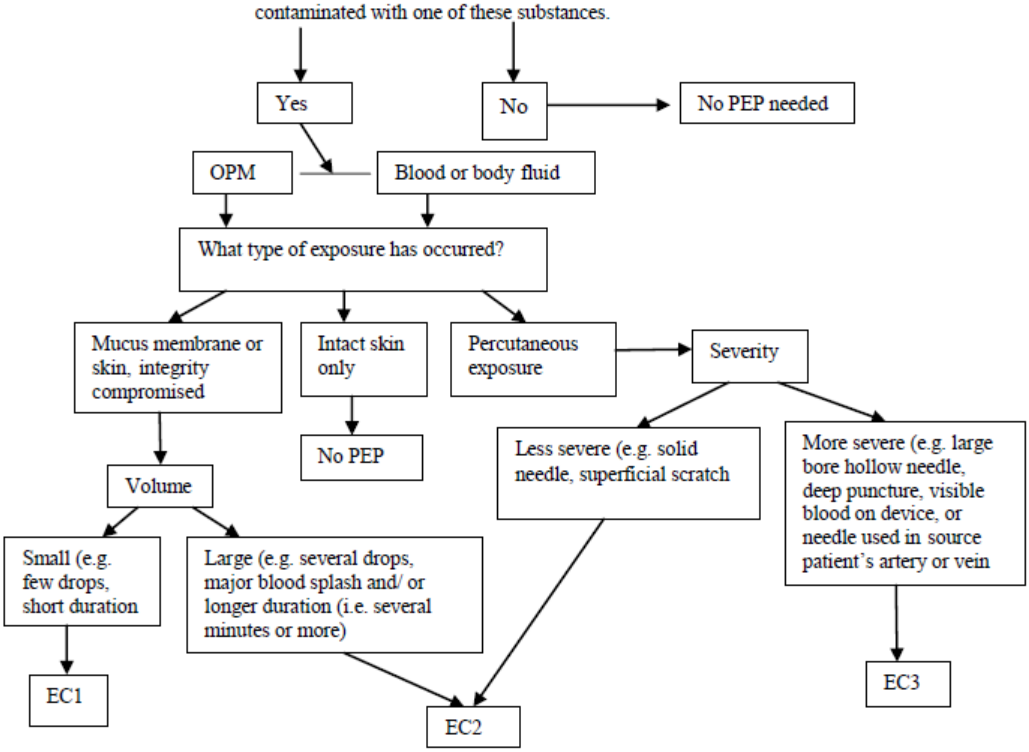
- Use of mask and cap.
- Use of glasses /special masks for protection of eyes.
- Use of plastic apron.
- Use of correct footwear.

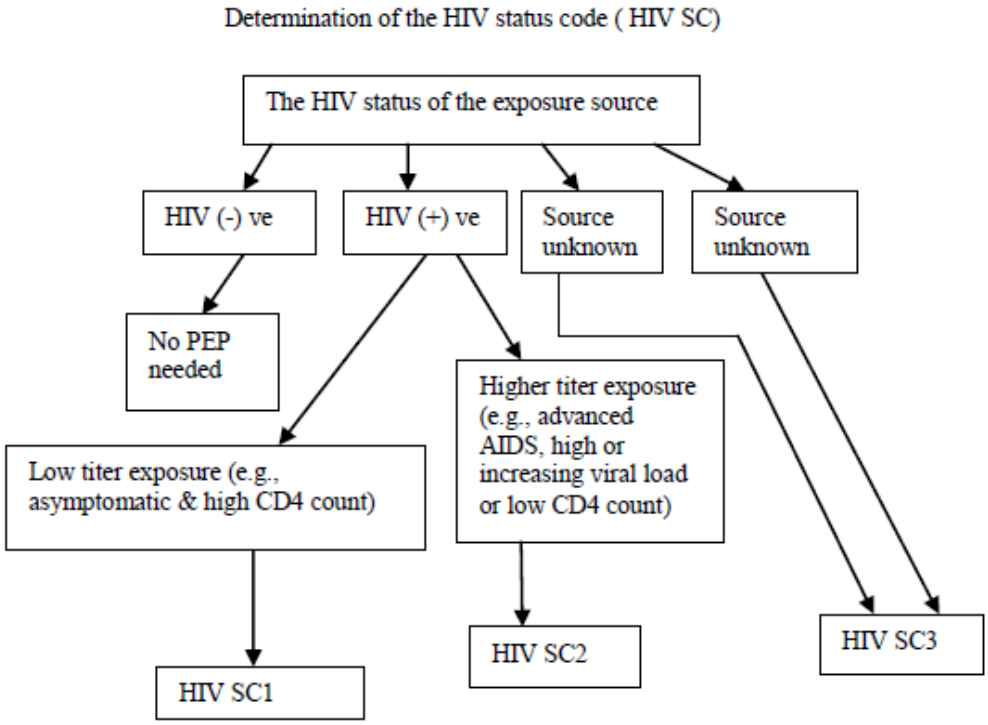
1. A methodical approach to all procedures.
2. Proper care and disposal of the sharps: like use of puncture proof containers in OT.
3. Doing all the procedures in the operation theatre in a controlled and deliberate manner.

Post Exposure Prophylaxis

Determination of the exposure code:

Is the source material blood, bloody fluid, other potentially infectious material(OPIM) , or an instrument contaminated with one of these substances.





Making a recommendation for PEP:

1. EC1+HIV SC1 : PEP is probably not required.
2. EC1+HIV SC2 : Basic regimen should suffice.
3. EC2+HIV SC1 : Basic regimen is recommended.
4. EC2+HIV SC2 : Expanded regimen is recommended.
5. EC3+HIV SC1 or SC2 : Expanded regimen is recommended.
6. EC2 or EC3+ HIV SCunknown : Basic regimen is recommended.

PEP should be started as early as possible after the exposure. The ideal time to start PEP is less than 6 to 12 hours. It is also known that PEP started after 72 hours of exposure is not beneficial and therefore is not recommended.

Basic regimen:

- Zidovudine (AZT) – 600 mg in divided doses (300 mg twice a day or 200 mg thrice a day) for 4 weeks.
- Lamivudine (3TC) – 150 mg twice a day for 4 weeks.

Expanded regimen:

- Zidovudine (AZT) – 600 mg in divided doses (300 mg twice a day or 200 mg thrice a day) for 4 weeks.
- Lamivudine (3TC) – 150 mg twice a day for 4 weeks.

- Indinavir – 800 mg thrice daily for 4 weeks. Alternatively , in place of Indinavir we can use Nelfinavir in the dosage of 750 mg thrice daily.

(b) Pulmonary embolism.

Answer. Introduction: Pulmonary thromboembolism (PE) constitutes a problem of immense clinical importance. Diagnosis is often difficult because PE may mimic a variety of cardiopulmonary conditions.

Aetiology:

- Respiratory difficulty in the setting of elevated risk for thrombosis indicates a likelihood of PE.
- The highest rates of PE are documented in patients older than 60 years and those in the highest quintile of weight.
- Heavy tobacco use and hypertension were also risk factors for PE.
- Women taking hormone replacements have a two to three times increased risk of PE depending on the formulation.
- Patients with cancer and those undergoing surgery are also at increased risk for PE.

Clinical features:

- Dyspnea is the most common presenting symptom, and tachypnea is the most common sign.
- Pleuritic chest pain, cough, and/or hemoptysis can also occur.
- Helpful findings on physical examination include those that indicate right ventricular dysfunction, namely, bulging neck veins with v waves, a left parasternal lift, an accentuated pulmonic component of S_2, and a systolic murmur at the lower left sternal border that increases in inspiration.
- The differential diagnosis includes pneumonia, bronchitis, myocardial infarction, pulmonary edema, aortic dissection, pneumothorax, worsening chronic obstructive pulmonary disease, carcinoma of the lung, and primary pulmonary hypertension.

Investigations:

- The diagnostic workup should include at the outset an electrocardiogram (ECG) and a chest radiograph. The most common ECG abnormality is T-wave inversion in the anterior precordial leads. Prominence of the S wave in lead I and a Q-wave and T-wave inversion in lead III may occur when right ventricular strain is present ($S_1Q_3T_3$ pattern).
- Abnormalities seen on chest x-ray film include focal oligemia (Westermark's sign), a peripheral wedged-shaped density above the diaphragm (Hampton's hump), and an enlarged right descending pulmonary artery (Palla's sign).
- Arterial blood gases may be helpful in the differential diagnosis and should be analyzed when PE is suspected. Hypoxemia and/or hypocapnia are suggestive of but not specific for the diagnosis, and PE can occur in the setting of normal blood gas analyses.

Three more specific tests are helpful in the diagnosis of PE.

- Perfusion lung scanning is the most useful screening test to rule out clinically significant PE. Normal results or results indicating a high probability of PE are useful. When the degree of clinical suspicion is high, low probability results should be considered nondiagnostic and further studies carried out.
- Spiral CT with contrast is most accurate for PE involving the proximal pulmonary arteries.
- Pulmonary arteriography should be used in the setting of high clinical suspicion with inconclusive scan results.

Ancillary tests include

- **D**-dimer enzyme-linked immunosorbent assay (ELISA) - The **D**-dimer lacks specificity and may be elevated in patients with myocardial infarction, pneumonia, heart failure, or cancer.
- Lower extremity venous ultrasound - Venous ultrasonography is accurate for the diagnosis of DVT, but a normal examination does not rule out a PE.
- Transthoracic echocardiography -Transthoracic echocardiography is useful in critically ill patients suspected of having a PE. .
- Venous ultrasonography is accurate for the diagnosis of DVT, but a normal examination does not rule out a PE.
- The combination of right ventricular pressure overload, right ventricular hypokinesis, and positive findings on venous ultrasound of the leg veins is pathognomonic of PE.

Treatment:

- Anticoagulation with heparin is the treatment of choice in all patients with PE unless contraindications such as active bleeding exist. Heparin should be given to all patients with a moderate or high clinical suspicion of PE during the definitive diagnostic workup. Unfractionated heparin should be given as an initial bolus of 5000 to 10,000 units followed by a continuous infusion of 18 U/kg of body weight per hour (not to exceed 1600 units per hour) with a target of a partial thromboplastin time of 60 to 80 seconds.
- Low-molecular-weight heparin (LMWH) may be given to hemodynamically stable patients with PE with comparable safety and efficacy. Heparin therapy is directed to the source of the embolus rather than the offending thrombus in the pulmonary artery.
- Thrombolysis is an established therapy for patients with PE and hemodynamic instability. These agents have been shown to dissolve thrombi and improve both pulmonary perfusion and right ventricular function.
- The patients with PE undergo echocardiography to assess the degree of right ventricular dysfunction and if moderate or severe receive intravenous alteplase in addition to heparin. Catheter-directed therapy allows the infusion of thrombolytic agents directly into the pulmonary artery thrombus. Wedging the catheter into the thrombus also allows for some thrombofragmentation with the potential for more rapid thrombolysis.
- Transvenous catheter embolectomy, percutaneous thrombofragmentation, or surgical embolectomy should be considered in patients with hemodynamic instability and contraindications to thrombolysis. Pulmonary embolectomy has been advocated as the procedure of last resort in patients with circulatory collapse and PE.
- In situations of hemodynamic collapse, percutaneous cardiopulmonary support systems can be used at the bedside as a bridge to emergent surgical embolectomy. These systems are compact, battery-operated portable units that employ femoral arterial and venous cannulas, a centrifugal pump, and a membrane oxygenator.

- **Indications for Aggressive Intervention in a Patient with a Pulmonary Embolism (at Least One of the Following Should Be Present)**

1. Arterial hypotension
2. Cardiogenic shock
3. Echocardiographic evidence of right ventricular failure
4. Precapillary pulmonary hypertension
5. Arterial-alveolar O_2 gradient >50 mm Hg
6. Contraindication to anticoagulation

Role of caval interruption:

- ❖ Interruption of the inferior vena cava by either ligation, plication, or insertion of an intravascular device has been used for the prevention of PE. In some centers, filters have been used as an alternative to heparin therapy for primary prevention of PE in patients with DVT of the lower extremity thought to have contraindications to anticoagulation.
- ❖ Filters should be reserved for secondary prevention or in those patients with contraindications for anticoagulation.
- ❖ Indications for inferior vena cava filter (IVCF) placement:

1. Contraindication to anticoagulation.

Indications for Vena Cava Filter Placement

Absolute indications
Failure of anticoagulation—PE or recurrent DVT while anticoagulated
Complications of anticoagulation
DVT/PE with contraindication to anticoagulation
Failure of previously placed filter
After pulmonary embolectomy
Relative indications
Large free-floating thrombus
DVT in patients with poor cardiopulmonary reserve
Protection during thrombolytic therapy
High-risk trauma patients
High-risk surgical patients
Cancer patients with DVT/PE
High-risk patients with prolonged bed rest
Pregnant patients
DVT, Deep venous thrombosis; PE, pulmonary embolism.

2. Complication with anticoagulation.
3. Recurrent PE despite anticoagulation or presence of another device.
4. Prophylaxis in the presence of DVT/PE.
5. Following pulmonary embolectomy.

Prevention: Recommendations for Venous Prophylaxis

Surgical Risk	Recommendation
Low: <40 yr, minor procedure	Early ambulation
Moderate: minor procedures with thrombosis risk factors, age 40–60 yr, major surgery in patients <40 yr	Elastic stockings or IPC or low-dose UFH (5000 IU SC q12h)
High: >60 yr, >40 yr with major procedures and risk factors for thrombosis	LMWH or IPC
Very high: >60 yr with associated risk factors for thrombosis	LMWH and IPC
Total joint replacement	LMWH or warfarin started preoperatively or immediately postoperatively and continued
Very high with additional high risk for bleeding	IPC
IPC, Intermittent pneumatic compression; LMWH, low-molecular-weight heparin; SC, subcutaneously; UFH, unfractionated heparin.	

(c) Principles of diathermy.

Answer. Surgical Diathermy

Introduction: Electrosurgical units (diathermy machines) were first introduced during the early twentieth century to facilitate haemostasis and/or the cutting of tissue during surgical procedures (Meeker & Rothrick 1999). This is achieved by passing normal electrical current via the diathermy machine and converting it into a high frequency alternating current (HFAC). This HFAC produces heat within body tissues to coagulate bleeding vessels and cut through tissue.

- There are 2 different types of electrosurgery: Monopolar and Bipolar.
- Monopolar electrosurgery is the emitance of the HFAC from the diathermy via an active electrode through the patients body tissues and then returned back to the diathermy machine via a dispersive electrode (patient return pad) .
- Bipolar electrosurgery is the emitance of the HFAC from the diathermy machine down one prong of a bipolar forcep through the tissue that has been placed between the forcep tips and returned to the diathermy machine via the second prong. Bipolar diathermy does not require a dispersive electrode (patient return pad).

Background

Electrosurgery has three effects on body tissue:

- Cut - generation of heat destroys tissue cell
- Coagulation - tissue cells contract to increase normal clotting
- Fulguration - cell walls destroyed through dehydration

Mechanism

Molecular rotation occurs in materials containing polar molecules having an electrical dipole moment, which will align themselves in an electromagnetic field. If the field is oscillating, as in an electomagnetic wave, these molecules rotate to continuously align with it. This is called dipole rotation. As the field alternates, the molecules reverse direction. Rotating molecules push, pull, and collide with other molecules (through electrical forces), distributing the energy to adjacent molecules and atoms in the material. Temperature is the average kinetic energy (energy of motion) of the atoms or molecules in a material, so agitating the molecules in this way by definition increases the temperature of the material. Thus, dipole rotation is a mechanism by which energy in the form of electromagnetic radiation is converted to heat energy in matter. (There are also many other mechanisms by which this conversion occurs.)

Dipole rotation is the mechanism normally referred to as dielectric heating, and is most widely observable in the microwave oven where it operates most efficiently on liquid water, and much less so on fats, sugars, and frozen water. This is caused by fats and sugars being far less polar than water molecules, and thus less affected by the forces generated by the alternating electromagnetic fields. Frozen water molecules fail to respond for a different reason: they are fixed in place and cannot freely rotate, so they cannot accelerate as much in response to the electromagnetic forces they experience from the electromagnetic waves.

Electrosurgery is the application of a high-frequency electric current to biological tissue as a means to cut, coagulate,desiccate, or fulgurate tissue. Its benefits include the ability to make precise cuts with limited blood loss. Electrosurgical devices are frequently used during surgical operations helping to prevent blood loss in hospital operating rooms or in outpatient procedures.

In electrosurgical procedures, the tissue is heated by an electric current. Although electrical devices may be used for the cauterization of tissue in some applications, electrosurgery is usually used to refer to a quite different method thanelectrocautery. The latter uses heat conduction from a probe heated to a glowing temperature by a direct current (much in the manner of a soldering iron). This may be accomplished by direct current from dry-cells in a penlight-type device. Electrosurgery, by contrast, uses alternating current to directly heat the tissue itself. When this results in destruction of small blood vessels and halting of bleeding, it is technically a process of electrocoagulation, although "electrocautery" is sometimes loosely and nontechnically used to describe it.

Often electrosurgery is mistakenly referred to as diathermy. Unlike Ohmic heating by electric current passing through the conductive tissue in conventional electrosurgery, diathermy means dielectric heating, produced by rotation of molecular dipoles in high frequency alternating electric field. This effect is most widely used in microwave ovens which operate at gigahertz frequencies.

Electrosurgery is commonly used in dermatological, gynecological, cardiac, plastic, ocular, spine, ENT, maxillofacial, orthopedic, urological, neuro- and general surgical procedures as well as certain dental procedures.

Electrosurgery is performed using an electrosurgical generator (also referred to as power supply or waveform generator) and a handpiece including one or several electrodes, sometimes referred to as an RF Knife. The apparatus when used for cutting or coagulation in surgery is still often referred to informally by surgeons as a "Bovie," after the inventor.

(d) Prophylactic antibiotics.

Answer. Which ever antibiotics are administered, the goal of therapy is to achieve antibiotic levels at the site of infection that exceed the minimum inhibitory concentration for the pathogens present. Each patient with a serious infection is evaluated daily or more frequently to assess response to treatment. If obvious improvement is not seen within 2 to 3 days one should think of the following:

The initial operative procedure was not adequate.

The initial procedure was adequate but a complication has occurred.

A superinfection has developed at a new site.

The drug choice is correct, but not enough is being given.

Another or a different drug is needed.

- The choice of antibiotics is not the most common cause of failure unless the original choice was clearly inappropriate, such as failing to provide coverage for anaerobes with an intra-abdominal infection.
- When choosing an antibiotic for empirical treatment, following guidelines should be practiced:

Choose an antibiotic that is able to reach the site of the infection. Specifically, for UTI and cholangitis, choose antibiotics with high renal and biliary concentrations, respectively. Consider skin, lung, and central nervous system tissue concentrations for infections at these sites.

Consider toxicity, particularly in critically ill patients, in whom bioavailability and the range of therapeutic and toxic levels are harder to predict. Once an antimicrobial with significant toxic side effects is started, closely monitor blood levels and organ function.

Whenever an infection that will need antibiotics is identified, aggressively dose the antibiotics. The volume of redistribution of these patients is unpredictable because they have usually undergone aggressive fluid replacements as part of their support or resuscitation.

Whenever starting an antibiotic regimen, set a time limit for the period the antibiotic will be given.

e) Systemic inflammatory response.

Answer. See the answer of question 3(a) of Paper – I of 2010.

4. Answer briefly of the followings: 4 x $7\frac{1}{2}$

a) Pharmacotherapy of benign prostatic hypertrophy.

b) Repair of hypospadius.
c) Vaious syndromes associated with medullary carcinoma thyroid.
d) Tetanus prophylaxis.

Answer.

(a) Pharmacotherapy of benign prostatic hypertrophy.

Answer. BPH is most commonly treated medically with α-blockers, such as doxazosin, terazosin, tamsulosin, and alfuzosin. Another class of medications, 5-α-reductase inhibitors, such as finasteride or dutasteride, are also used to treat BPH. The Medical Therapy of Prostatic Symptoms Trial (MTOPS) showed that combination therapy with an α-blocker and a 5-α-reductase inhibitor reduced the risk of overall clinical progression of BPH significantly more than did treatment with either drug alone.

Drugs used are:
- Alpha blockers are categorized by their selectivity for the AndrogenReceptor, and by their elimination half-life.
  - Non-selective: phenoxybenzamine effective symptom control, but high side-effect profile
  - Short acting: prazosin, alfuzosin, indoramin
  - Long-acting: terazosin, doxazosin, alfuzosin SR
  - Subtype selective: tamsulosin relatively selective for (1a-AR subtype compared to the 1±1b subtype).

No study has directly compared one alpha blocker with another in terms of efficacy or side-effects.
Indications for treatment: Bothersome lower urinary tract symptoms where watchful waiting has failed or the patient wishes to have treatment.
- 5alpha-reductase inhibitors inhibit the conversion of testosterone to dihydrotestosterone, the more potent androgen in the prostate. This causes shrinkage of the prostatic epithelium and therefore a reduction in prostate volume, thereby reducing the component of benign prostatic enlargement. This takes some months to occur, so urinary symptoms will not improve initially.
  - Finasteride is a competitive inhibitor of the enzyme 5alpha-reductase (type II isoenzyme) which converts testosterone to DHT. Finasteride therefore lowers serum and intraprostatic DHT levels.
  - Dutasride can also be used.
  - Epristeride is a dual inhibitor of 5alpha-reductase. Whether it has any clinically significant advantages over finasteride remains to be established.
- Combination therapy

A combination of an alpha blocker and a 5alpha-reductase inhibitor. Most studies, suggest that combination therapy is NO more useful than an alpha blocker alone.
iv. Alternative drug therapy
Anticholinergics: For a man with frequency, urgency, and urge incontinence symptoms suggestive of an overactive bladder consider prescribing an anticholinergic (e.g. oxybutynin, tolterodine, trospium chloride, or flavoxate). There is the concern that these drugs could precipitate urinary retention in men with BOO (because they block parasympathetic/cholinergic mediated contraction of the detrusor), but the risk of this occurring is probably very low, even in men with urodynamically proven BOO.
Phytotherapy: An alternative drug treatment for BPH symptoms, and one which is widely used in Europe and increasingly in North America, is phytotherapy. 50% of all medications

consumed for BPH symptoms are phytotherapeutic ones.These agents are derived from plants and include the African plum (Pygeum africanum), purple cone flower (Echinacea purpurea), South African star grass (Hipoxis rooperi), and saw palmetto berry (Seronoa Repens, Permixon).

(b) Repair of hypospadius.

Answer. Definition: Hypospadias is a congenital deformity where the opening of the urethra (the meatus) occurs on the underside (ventral) part of the penis, anywhere from the glans to the perineum.
Classification: Hypospadias can be classified according to the anatomical location of the urethral meatus.

- Anterior (or distal) - glandular, coronal, and subcoronal (~50%)
- Middle - distal penile, midshaft, and proximal penile (~30%)
- Posterior (or proximal) - penoscrotal, scrotal, and perineal (~20%)

Treatment:

- Surgery is indicated where deformity is severe, interferes with voiding, or is predicted to interfere with sexual function. Surgery is now performed between **6-12 months** of age. Local application of testosterone for 1 month pre-operatively can help increase tissue size.
- Surgery aims to correct penile curvature (orthoplasty), reconstruct a new urethra, and bring the new meatus to the tip of the glans using urethroplasy, glanuloplasty, and meatoplasty techniques.
- Severe cases may require staged procedures.
- Common operations for anterior hypospadias include meatal advancement and glanuloplasty (MAGPI), meatal-based flaps (Mathieu procedure), and tubularization of the urethral plate. Posterior defects require free grafts (buccal mucosa), onlay grafts, and preputial transfer flaps.

(c) Vaious syndromes associated with medullary carcinoma thyroid.

Answer. Multiple Endocrine Neoplasia II

MEN IIA
Medullary thyroid carcinoma
Pheochromocytoma
Hyperparathyroidism
Lichen planus amyloidosis
Hirschsprung's disease
MEN IIB
Medullary thyroid carcinoma
Pheochromocytoma
Marfanoid body habitus
Mucosal neuromas

Ganglioneuromatosis of the gastrointestinal tract

Multiple Endocrine Neoplasia Type 1 (MEN1)

The MEN1 gene

The MEN1 gene consists of ten exons, spanning about 10 kb, and encodes a 610 amino acid protein named menin. The first exon and the last part of exon 10 are not translated.

The Menin Protein:

Menin is a 610 amino acid (67Kda) nuclear protein, highly conserved from mouse (98%), rat (97%) and, more distantly, zebrafish (75%) and Drosophila (47%) (47-51). Human and mouse MEN1 amino acid sequences share 95.8% identity and 98.4% similarity. Analysis of menin amino acid sequence did not reveal homologies to any other known human or mammalian protein, sequence motif, or signal peptide..

Pathophysiology:

MEN1 follows Knudson's "two-hit" model for tumor suppressor gene carcinogenesis (30). The first hit is a heterozygous MEN1 germline mutation, inherited from one parent (familial cases) or developed in an early embryonic stage (sporadic cases) and present in all cells at birth. The second hit is a MEN1 somatic mutation, usually a large deletion, that occurs in the predisposed endocrine cell as loss of the remaining wild-type allele and gives cells the survival advantage needed for tumor development.

MEN1 mutations in multiple endocrine neoplasia patients and clinical genetics

MEN1 gene mutations can be identified in 70-95% of MEN1 patients and in about 20% of familial isolated hyperparathyroidism cases. Almost all patients are heterozygous for mutations. One affected family has been identified with individuals both homozygous and heterozygous for MEN1 mutations. In this family, there was no difference in disease history between the homozygous and heterozygous mutation carriers.

Fifty percent of patients develop signs and symptoms by 20 years of age and more than 95% have symptoms by 40 years of age. There is significant intra- and inter-familial variability in the age of onset, severity of disease, and tumor types. Despite numerous studies, no genotype-phenotype correlations have been established, suggesting that unknown genetic and environmental modifiers are involved in the expression of the MEN1 phenotype.\

Manifestations

Multiple Endocrine Neoplasia type 1 (MEN1) is a rare hereditary endocrine cancer syndrome characterized primarily by tumors of the parathyroid glands (95% of cases), endocrine gastroenteropancreatic (GEP) tract (30-80% of cases), and anterior pituitary (15-90% of cases). Other endocrine and non-endocrine neoplasms including adrenocortical and thyroid tumors, visceral and cutaneous lipomas, meningiomas, facial angiofibromas and collagenomas, and thymic, gastric, and bronchial carcinoids also occur. MEN1 should be suspected in patients with an endocrinopathy of two of the three characteristic affected organs, or with an endocrinopathy of one of these organs plus a first-degree relative affected by MEN1 syndrome.

MEN1 patients usually have a family history of MEN1. Inheritance is autosomal dominant; any affected parent has a 50% chance to transmit the disease to his or her progeny.

Many endocrine tumors in MEN1 are benign and cause symptoms by overproduction of hormones or local mass effects, while other MEN1 tumors are associated with an elevated risk

for malignancy. About one third of patients affected with MEN1 will die early from an MEN1-related cancer or associated malignancy. Entero-pancreatic gastrinomas and thymic and bronchial carcinoids are the leading cause of morbidity and mortality.

Recommended cancer surveillance:

A recommend surveillance program for Multiple Endocrine Neoplasia Type 1 has been suggested by the International Guidelines for Diagnosis and Therapy of MEN syndromes group.

Multiple endocrine neoplasia type 1

Multiple endocrine neoplasia type 1 (MEN-1 syndrome) or Wermer's syndrome is part of a group of disorders that affect the endocrine system.

Explanation

These disorders greatly increase the risk of developing multiple cancerous and noncancerous tumors in glands such as the parathyroid, pituitary, and pancreas. Multiple endocrine neoplasia occurs when tumors are found in at least two of the three main endocrine glands (parathyroid, pituitary, and pancreatico-duodenum). Tumors can also develop in organs and tissues other than endocrine glands

Although many different types of hormone-producing tumors are associated with multiple endocrine neoplasia, tumors of the parathyroid gland, pituitary gland, and pancreas are most frequent in multiple endocrine neoplasia type 1. Tumors cause an overactivation of these hormone-producing glands, leading to serious health problems such as severe ulcers (Due to gastrin hypersecretion, which stimulates secretion of hydrochloric acd). Overactivity of the parathyroid gland (hyperparathyroidism) is the most common sign of this disorder. Hyperparathyroidism disrupts the normal balance of calcium in the blood, which can lead to kidney stones, thinning of bones, weakness, and fatigue.

Neoplasia in the pituitary gland can manifest as prolactinomas whereby too much prolactin is secreted, suppressing the release of gonadotropins, causing a decrease in sex hormones such as testosterone. Pituitary tumor in MEN1 can be large and cause signs by compressing adjacent tissues.

Pancreatic tumors usually form in the islet cells, which over-secrete insulin, causing a decrease in blood glucose levels.

Multiple endocrine neoplasia is part of a group of disorders that affect the body's network of hormone-producing glands (the endocrine system). Hormones are chemical messengers that travel through the bloodstream and regulate the function of cells and tissues throughout the body. Multiple endocrine neoplasia involves tumors in at least two endocrine glands; tumors can also develop in other organs and tissues. These growths can be noncancerous (benign) or cancerous (malignant). If the tumors become cancerous, some cases can be life-threatening.

The two major forms of multiple endocrine neoplasia are called type 1 and type 2; they are often confused because of their similar names. Type 1 and type 2 are distinguished by the genes involved, the types of hormones made, and the characteristic signs and symptoms.

- Many different types of tumors are associated with multiple endocrine neoplasia. Type 1 frequently involves tumors of the parathyroid gland, pituitary gland, and pancreas. Tumors in these glands lead to the overproduction of hormones. The most common sign of multiple endocrine neoplasia type 1 is overactivity of the parathyroid gland (hyperparathyroidism). Hyperparathyroidism disrupts the normal balance of calcium in the blood, which can lead

to kidney stones, thinning of bones, nausea and vomiting, high blood pressure (hypertension), weakness, and fatigue.

- The most common sign of multiple endocrine neoplasia type 2 is a form of thyroid cancer called medullary thyroid carcinoma. Some people with this disorder also develop a pheochromocytoma, which is a tumor of the adrenal glands that can cause dangerously high blood pressure. Multiple endocrine neoplasia type 2 is divided into three subtypes: type 2A, type 2B, and familial medullary thyroid carcinoma (FMTC). These subtypes differ in their characteristic signs and symptoms and risk of specific tumors; for example, hyperparathyroidism occurs only in type 2A. The features of this disorder are relatively consistent within any one family.

Genetic effects

People with multiple endocrine neoplasia type 1 are born with one mutated copy of the MEN1 gene in each cell. Then, during their lifetime, the other copy of the gene is mutated in a small number of cells. These genetic changes result in no functional copies of the MEN1 gene in selected cells, allowing the cells to divide with little control and form tumors.

Signs and symptoms

Parathyroid

Hyperparathyroidism is present in ≥ 90% of patients. Asymptomatic hypercalcemia is the most common manifestation: about 25% of patients have evidence of nephrolithiasis or nephrocalcinosis. In contrast to sporadic cases of hyperparathyroidism, diffuse hyperplasia or multiple adenomas are more common than solitary adenomas.

Pancreas

Pancreatic islet cell tumors occur in 60 to 70% of patients. Tumors are usually multicentric. Multiple adenomas or diffuse islet cell hyperplasia commonly occurs; such tumors may arise from the small bowel rather than the pancreas. About 30% of tumors are malignant and have local or distant metastases. Malignant islet cell tumors due to MEN 1 syndrome often have a more benign course than do sporadically occurring malignant islet cell tumors.

About 40% of islet cell tumors originate from a β cell, secrete insulin (insulinoma), and can cause fasting hypoglycemia. β-Cell tumors are more common in patients < 40 years of age. About 60% of islet cell tumors originate from non-β-cell elements and tend to occur in patients > 40 years of age. Non-β-cell tumors are somewhat more likely to be malignant.

Most islet cell tumors secrete pancreatic polypeptide, the clinical significance of which is unknown.Gastrin is secreted by many non–β-cell tumors (increased gastrin secretion in MEN 1 also often originates from the duodenum). Increased gastrin secretion increases gastric acid, which may inactivate pancreatic lipase, leading to diarrhea and steatorrhea. Increased gastrin secretion also leads to peptic ulcers in > 50% of MEN 1 patients. Usually the ulcers are multiple or atypical in location, and often bleed, perforate, or become obstructed. Peptic ulcer disease may be intractable and complicated (Zollinger-Ellison syndrome—see Tumors of the GI Tract: Zollinger-Ellison Syndrome). Among patients presenting with Zollinger-Ellison syndrome, 20 to 60% have MEN 1.

A severe secretory diarrhea can develop and cause fluid and electrolyte depletion with non–β-cell tumors. This complex, referred to as the watery diarrhea, hypokalemia, and achlorhydria syndrome (WDHA; pancreatic cholera—see Tumors of the GI Tract: Vipoma), has been ascribed to vasoactive intestinal polypeptide, although other intestinal hormones or

secretagogues (including prostaglandins) may contribute. Hypersecretion of glucagon, somatostatin, chromogranin, or calcitonin, ectopic secretion of ACTH (causing Cushing's syndrome), and hypersecretion of growth hormone–releasing hormone (causing acromegaly) sometimes occur in non–β-cell tumors. All of these are rare in MEN 1.

Nonfunctioning pancreatic tumors also occur in patients with MEN 1 and may be the most common type of pancreatoduodenal tumor in MEN 1. The size of the nonfunctioning tumor correlates with risk of metastasis and death.

Pituitary

Pituitary tumors occur in 15 to 42% of MEN 1 patients. From 25 to 90% are prolactinomas. About 25% of pituitary tumors secrete growth hormone or growth hormone and prolactin. Excess prolactin may cause galactorrhea (see Pituitary Disorders: Galactorrhea), and excess growth hormone causes acromegaly clinically indistinguishable from sporadically occurring acromegaly. About 3% of tumors secrete ACTH, producing Cushing's disease. Most of the remainder are nonfunctional. Local tumor expansion may cause visual disturbance, headache, and hypopituitarism. Pituitary tumors in MEN 1 patients appear to be larger and behave more aggressively than sporadic pituitary tumors.

Other manifestations

Adenomas and adenomatous hyperplasia of the thyroid and adrenal glands occurs occasionally in MEN 1 patients. Hormone secretion is rarely altered as a result, and the significance of these abnormalities is uncertain. Carcinoid tumors, particularly those derived from the embryologic foregut, occur in isolated cases. Multiple subcutaneous and visceral lipomas, angiofibromas, and collagenomas may also occur.

Multiple endocrine neoplasia type 2

Multiple endocrine neoplasia type 2 (also known as "Pheochromocytoma and amyloid producing medullary thyroid carcinoma", "PTC syndrome," and "Sipple syndrome")is a group of medical disorders associated with tumors of the endocrine system. The tumors may bebenign or malignant (cancer). They generally occur in endocrine organs (e.g. thyroid, parathyroid, and adrenals), but may also occur in endocrine tissues of organs not classically thought of as endocrine.

MEN2 is a sub-type of MEN (multiple endocrine neoplasia) and itself has sub-types, as discussed below.

Classification

Before gene testing was available, the type and location of tumors determined which type of MEN2 a person had. Gene testing now allows a diagnosis before tumors or symptoms develop.

A table in the multiple endocrine neoplasia article compares the various MEN syndromes. MEN2 and MEN1 are distinct conditions, despite their similar names. MEN2 includes MEN2A, MEN2B and familial medullary thyroid cancer.

The common feature among the three sub-types of MEN2 is a high propensity to develop medullary thyroid carcinoma.

Presentation

MEN2 can present with a sign or symptom related to a tumor or, in the case of multiple endocrine neoplasia type 2b, with characteristic musculoskeletal and/or lip and/or gastrointestinal findings.

Medullary thyroid carcinoma (MTC) represent the most frequent initial diagnosis. Occasionally pheochromocytoma and primary hyperparathyroidism may be the initial diagnosis.

In MEN2A primary hyperparathyroidism occurs in only 10%-30% and is usually diagnosed after the third decade of life. It occur in children but this is rare. It may be the sole clinical manifestation of this syndrome but this =is unusual.

MEN2A associates medullary thyroid carcinoma with pheochromocytoma in about 20-50% of cases and with primary hyperparathyroidism in 5-20% of cases.

MEN2B associates medullary thyroid carcinoma with pheochromocytoma in 50% of cases, with marfanoid habitus and with mucosal and digestive ganglioneuromatosis.

In familial isolated medullary thyroid carcinoma the other components of the disease are absent.

Management

Management of MEN2 patients include thyroidectomy including cervical central and bilateral lymph nodes dissection for MTC, unilateral adrenalectomy for unilateral pheochromocytoma or bilateral adrenalectomy when both glands are involved and selective resection of pathologic parathyroid glands for primary hyperparathyroidism.

Familial genetic screening is recommended to identify at risk subjects who will develop the disease, permitting early management by performing prophylactic thyroidectomy, giving them the best chance of cure.

Prognosis of MEN2 is mainly related to the stage-dependant prognosis of MTC indicating the necessity of a complete thyroid surgery for index cases with MTC and the early thyroidectomy for screened at risk subjects.

Causes

The table in the multiple endocrine neoplasia article lists the genes involved in the various MEN syndromes. Most cases of MEN2 derive from a variation in the RET proto-oncogene, and are specific for cells of neural crest origin.

The protein produced by the RET gene plays an important role in the TGF-beta (transforming growth factor beta) signaling system. Because the TGF-beta system operates in numerous tissues throughout the body, variations in the RET gene can have effects in numerous tissues throughout the body.

MEN2 generally results from a gain-of-function variant of a RET gene. Other diseases, such as Hirschsprung disease, result from loss-of-function variants.

- Most cases of multiple endocrine neoplasia type 2 are inherited in anautosomal dominant pattern.

When inherited, multiple endocrine neoplasia type 2 is transmitted in an autosomal dominant pattern, which means affected people have one affected parent, and possibly-affected siblings and children. Some cases, however, result from spontaneous new mutations

in the RET gene. These cases occur in people with no family history of the disorder. In MEN2B, for example, about half of all cases arise as spontaneous new mutations.

Differences in presentation

As noted, all types of MEN2 include pheochromocytoma, and medullary thyroid carcinoma.

MEN2A is additionally characterized by the presence of parathyroid hyperplasia or tumor.

MEN2B is additionally characterized by the presence of mucocutaneous neuroma, gastrointestinal symptoms (e.g. constipation andflatulence), and muscular hypotonia.

MEN2B can present with a Marfanoid habitus

Multiple endocrine neoplasia type 2b

Multiple endocrine neoplasia type 3 (also known as "Mucosal neuromata with endocrine tumors", "Multiple endocrine neoplasia type 2B", "MEN2B", "Multiple mucosal neuroma syndrome", and "Wagenmann–Froboese syndrome") is a genetic disease that causes multiple tumors on the mouth, eyes, and endocrine glands. It is the most severe type of multiple endocrine neoplasia, differentiated by the presence of oral and submucosal tumors in addition to endocrine tumors. It was first described by Wagenmann in 1922.

MEN 2B typically manifests before a child is 10 years old. Affected individuals tend to be tall and lanky, with an elongated face and protruding, blubbery lips. Benign tumors (neoplasms) develop in the mouth, eyes, and submucosa of almost all organs in the first decade of life, followed by adrenal and thyroid tumors after puberty. Medullary thyroid cancer almost always occurs, and cancer of the adrenal glands (pheochromocytoma) occurs in 50% of cases.

A variety of eponyms have been proposed for MEN 2B, such as Williams-Pollock syndrome, Gorlin-Vickers syndrome, and Wagenmann-Froboese syndrome. However, none ever gained sufficient traction to merit continued use, and are no longer used in the medical literature.

It has an estimated prevalence of 1 in 40.000.

Causes

Like MEN 1 and MEN 2A, MEN 2B is inherited as an autosomal dominant trait, which means affected people have one affected parent, and possibly-affected siblings and children. However, about half of the cases appear to be spontaneous mutations. 95% of patients with MEN 2B have a single-point mutation of the RET proto-oncogene allele, which suppresses cancer formation. The mutation - a single methionine to threonine substitution in the intracellular tyrosine kinase domain - alters the substrate specificity of intracellular signal transduction, conferring dominant cancer-causing activity.

Fifty percent of MEN 2B cases appear to be from a de novo mutation, a copying error that occurs in either the sperm or ovum prior to fertilization. Like many de novo mutations, children born to older parents have an increased risk of spontaneous MEN 2B. The mutated gene is almost exclusively paternal, particularly from older fathers. The sex ratio is also uneven: sons are twice as likely to develop MEN 2B as daughters.

Symptoms

Patients are tall and lanky, with a "marfanoid" body type and occasional muscle wasting. Mucosal neuromas are the most consistent and distinctive feature, appearing in 100% of patients. Usually there are numerous yellowish-white, sessile, painless nodules on the lips or tongue, with deeper lesions having normal coloration. There may be enough neuromas in the

body of the lips to produce enlargement and a "blubbery lip" appearance. Similar nodules may be seen on the sclera and eyelids, sometimes causing the lid to turn inside-out.

Multiple mucosal neuromata is associated with diffuse ganglioneuromatosis, which can cause gastrointestinal problems such as diverticulosis or persistent diarrhea.

Pathology

Findings at postmortem evaluation indicate that symptoms can be attributed to neuroma formation: a characteristic adventitious plaque of tissue composed of hyperplastic, interlacing bands of Schwann cells and myelinated fibers overlay the posterior columns of the spinal cord. Mucosal neuromas are made up of nerve cells, often with thickened perineurium, intertwined with one another in a plexiform pattern. This tortuous pattern of nerves is seen within a background of loose endoneurium-like fibrous stroma.

Treatment and Prognosis

The mucosal neuromas of this syndrome are asymptomatic and self limiting, and present no problem requiring treatment. They may, however, be surgically removed for aesthetic purposes or if they are being constantly traumatized.

Almost all patients develop medullary thyroid cancer, in a more aggressive form than MEN 2A. Complete thyroidectomy is often recommended at a young age, before malignant tumors develop. The ideal age for surgery is 4 years old or younger, since cancer may metastasize before age 10. Pheochromocytoma - cancer of the adrenal glands - is also present in 50% of cases. Affected individuals are encouraged to get yearly screenings for thyroid and adrenal cancer, and it is strongly suggested that other family members also be evaluated for MEN 2B.

(d) Tetanus prophylaxis.

Answer.

- Tetanus is a serious disease characterized by muscle spasm and rigidity. The mortality rate is approximately 20% and is due to spasm of the muscles of respiration. Tetanus is an illness preventable through primary immunization and regular booster shots.
- The Emergency Department patient encounter provides an ideal opportunity to screen for adequate tetanus immunization and to provide it, when necessary. In North America, the vast majority of people seen in the Emergency Department will have received primary immunization. Groups that may have missed primary immunization include elderly patients and immigrants.
- Primary immunization involves a series of four toxoid injections for preschool children or three toxoid injections if started at age 7 or older. Following primary immunization, children receive a booster shot at age 5 and additional boosters every 10 years subsequent to that.
- Patients seen in the Emergency Department with clean, minor wounds are considered adequately immunized if they have received primary immunization and have had a booster within the past 10 years. If a wound is "dirty" (which includes wounds contaminated with saliva, feces or dirt, and burn injuries) then a booster within the past 5 years is necessary to ensure immunization.
- If the patient has not received primary immunization, (or if the patient is unsure) then passive immunity with tetanus immune globulin (T.I.G.) is provided. At the same time,

but with a different injection site, tetanus and diphtheria toxoid should be given. This initiates primary immunization but adequate follow-up should be arranged to ensure completion of the series. Note that the diphtheria toxoid is added to ensure adequate immunity to diphtheria in the population.

- Patients will occasionally present stating they have an allergy to the toxoid. Adverse reactions such as local pain, erythemia, fever, malaise or rash are common but should not preclude further immunization. A true anaphylactic or serious neurologic reaction to the toxoid are the only contraindications to further immunization with the tetanus and diphtheria toxoid.
- If a patient has had a true serious reaction in the past, they should receive a T.I.G. in the Emergency Department and then follow-up with an allergist to assess immunization status. The table below summarizes the CDC guidelines for tetanus prophylaxis.

Tetanus Immunization: **For clean, minor wounds:**

	Give patient Tetanus Diphtheria Toxoid?	Give patient Tetanus Immune Globulin?
Unknown or less than 3 doses of absorbed Tetanus Toxoid	Yes	No
Greater than 3 doses of of absorbed Tetanus Toxoid	No (unless >10 years since last booster)	No

For all other wounds:

	Give patient Tetanus Diphtheria Toxoid?	Give patient Tetanus Immune Globulin?
Unknown or less than 3 doses of absorbed Tetanus Toxoid	Yes	Yes
Greater than 3 doses of of absorbed Tetanus Toxoid	No (unless >5 years since last booster)	No

TETANUS PROPHYLAXIS GUIDELINES

TETANUS PROPHYLAXIS FOR CHILDREN 6 WEEKS THROUGH 6 YEARS				
History of Adsorbed Tetanus Toxoid	Clean, Minor Wounds		All Other Wounds[1]	
	DTaP[2]	TIG	DTaP[2]	TIG
Unknown or less than 3 doses	**Yes** (the primary series should be completed)	No	**Yes** (the primary series should be completed)	Yes
3 or more doses (If only 3 doses of fluid tetanus toxoid have been received, a fourth dose of toxoid, preferably an adsorbed toxoid, should be given)	**No** (Yes if the routine immunization schedule has lapsed)	No	**No** (Yes if the routine immunization schedule has lapsed or if it has been more than 5 years since the previous dose)	No

TETANUS PROPHYLAXIS FOR 7- 9 YEARS				
History of Adsorbed Tetanus Toxoid	Clean, Minor Wounds		All Other Wounds[1]	
	Td	TIG	Td	TIG
Unknown or less than 3 doses	**Yes**	No	**Yes**	Yes
3 or more doses (If only 3 doses of fluid tetanus toxoid have been received, a fourth dose of toxoid, preferably an adsorbed toxoid, should be given)	**No** (Yes if the routine immunization schedule has lapsed)	No	**No** (Yes if it has been more than 5 years since the previous dose)	No

TETANUS PROPHYLAXIS FOR 10 YEARS AND OLDER				
History of Adsorbed Tetanus Toxoid	Clean, Minor Wounds		All Other Wounds[1]	
	Tdap/Td[3]	TIG	Tdap/Td[3]	TIG
Unknown or less than 3 doses	**Yes**	No	**Yes**	Yes
3 or more doses (If only 3 doses of fluid tetanus toxoid have been received, a fourth dose of toxoid, preferably an adsorbed toxoid, should be given)	**No** (Yes if it has been more than 10 years since the previous dose)	No	**No** (Yes if it has been more than 5 years since the previous dose)	No

[1] Including, but not limited to wounds contaminated with dirt, feces, soil or saliva; puncture wounds, avulsions; wounds resulting from missiles (gunshots), crushing, burns or frostbite.

[2] DTaP is licensed for use in children 6 weeks up through 6 yrs of age. Use DT only if patient has a contraindication to acellular pertussis.

[3] Tdap vaccine is the preferred vaccine for adolescents and adults who have not previously received a dose of Tdap. There are 2 Tdap vaccines: Boostrix is licensed for persons 10-18 yrs of age. ADACEL is licensed for persons 11-64 yrs of age. Administer Td if Tdap is not available.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES

MS (General Surgery) Examination, 2007

PAPER I

Time Allowed: 3 Hours **Full Marks: 100**

Attempt all questions

1. Define & discuss subphrenic spaces & their surgical importance. (20)

2. Describe calcium metabolism and its various disorders seen in surgical practice. (20)

3. Write short notes on: 5 x 6

a) Physiology of blood coagulation & its importance in surgery.

b) Anatomy & physiology of spleen.

c) Development & descent of testis.

d) Complications of pneumoperitoneum induced during laparoscopy.

e) Metabolic changes in high output fistula.

4. Answer in brief: 4 x $7^1/_2$

a) Surgical anatomy of liver & its importance in liver resection.

b) Pathogenesis of septic shock.

c) Postoperative pain relief.

d) Surgical pathology of salivary gland tumours.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES
MS (General Surgery) Examination, 2007

April, 2007

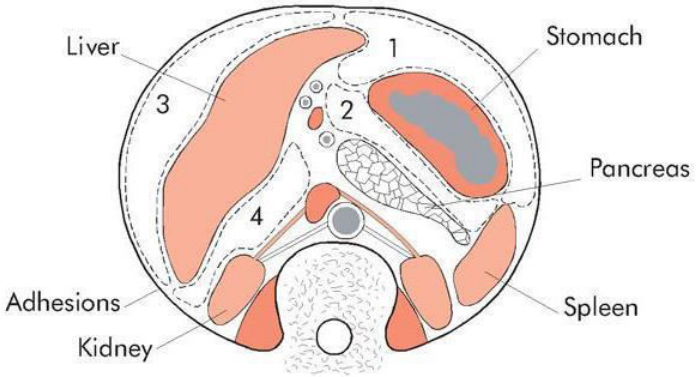
PAPER I

Time Allowed: 3 Hours **Full Marks: 100**

Attempt all questions

1. Define & discuss subphrenic spaces & their surgical importance. (20)

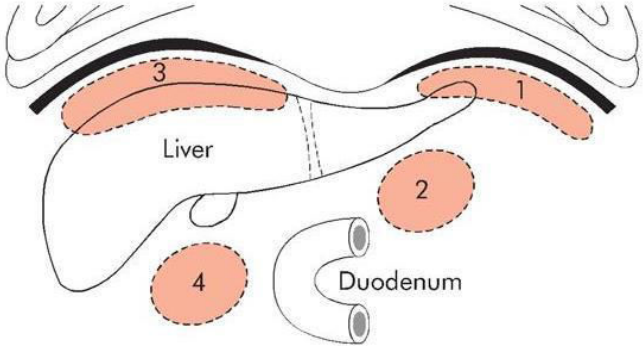
Answer. Introduction: The complicated arrangement of the peritoneum results in the formation of four intra peritoneal spaces in which pus may collect.



Intraperitoneal spaces on transverse section.

(1) The left subphrenic space; (2) left subhepatic space/lesser sac;

(3) right subphrenic space; (4) right subhepatic space.



Intraperitoneal spacses on sagittal section.

(1) Left subphrenic; (2) left subhepatic/lesser sac;

(3) right subphrenic; (4) right subhepatic space.

Left subphrenic space: This is bounded above by the diaphragm and behind by the left triangular ligament and the left lobe of the liver, the gastrohepatic omentum and the anterior surface of the stomach. To the right is the falciform ligament and to the left the spleen, gastrosplenic omentum and diaphragm.
The common cause of an abscess here is an operation on the stomach, the tail of thepancreas, the spleen or the splenic flexure of the colon.

Left subhepatic space/lesser sac:The commonest cause of infection here is complicated acute pancreatitis. In practice, a perforated gastric ulcer rarely causes a collection here because the potential space is obliterated by adhesions.

Right subphrenic space: This space lies between the right lobe of the liver and the diaphragm. It is limited posteriorly by the anterior layer of the coronary and the right triangular ligaments and to the left by the falciform ligament. Common causes of abscess here are perforating cholecystitis, a perforated duodenal ulcer, a duodenal cap 'blow-out' following gastrectomy and appendicitis.

Right subhepatic space:This lies transversely beneath the right lobe of the liver in Rutherford Morison's pouch. It is bounded on the right by the right lobe of the liver and the diaphragm. To the left is situated the foramen of Winslow and below this lies the duodenum. In front are the liver and the gall bladder and behind are the upper part of the right kidney and the diaphragm. The space is bounded above by the liver and below by the transverse colon and hepatic flexure. It is the deepest space of the four and the commonest site of a subphrenic abscess, which usually arises from appendicitis, cholecystitis, a perforated duodenal ulcer or following upper abdominal surgery.

Clinical features:
The symptoms and signs of subphrenic infection are frequently non-specific and it is well to remember the aphorism, 'pus somewhere, pus nowhere else, pus under the diaphragm'.

Symptoms: A common history is that, when some infective focus in the abdominal cavity has been dealt with, the condition of the patient improves temporarily but, after an interval of a few days or weeks, symptoms of toxaemia reappear. The condition of the patient steadily, and often rapidly, deteriorates. Sweating, wasting and anorexia are present. There is sometimes epigastric fullness and pain, or pain in the shoulder on the affected side, because of irritation of sensory fibres in the phrenic nerve, referred along the descending branches of the cervical plexus. Persistent hiccoughs may be a presenting symptom.

Signs: A swinging pyrexia is usually present. If the abscess is anterior, abdominal examination will reveal some tenderness, rigidity or even a palpable swelling. Sometimes the liver is displaced downwards but more often it is fixed by adhesions. Examination of the chest is important and, in the majority of cases, collapse of the lung or evidence of basal effusion or even an empyema is found.

Investigations:
A number of the following investigations may be helpful:
• Blood tests usually show a leucocytosis and raised C-reactiveprotein.
• A plain radiograph sometimes demonstrates the presence of gas or a pleural effusion. On screening, the diaphragm is often seen to be elevated (so called 'tented' diaphragm) and its movements impaired.
• Ultrasound or CT scanning is the investigation of choice and permits early detection of subphrenic collections.
• Radiolabelled white cell scanning may occasionally prove helpful when other imaging techniques have failed.

Differential diagnosis:
Pyelonephritis, amoebic abscess, pulmonary collapse and pleural empyema may give rise to diagnostic difficulty.

Treatment: The clinical course of suspected cases is monitored, and blood tests and imaging investigations are carried out at suitable intervals. If suppuration seems probable, intervention is indicated. If skilled help is available it is usually possible to insert a percutaneous drainage tube under ultrasound or CT control. The same tube can be used to instill antibiotic solutions or irrigate the abscess cavity. To pass an aspirating needle at the bedside through the pleura and diaphragm invites potentially catastrophic spread of the infection into the pleural cavity. If an operative approach is necessary and a swelling can be detected in the subcostal region or in the loin, an incision is made over the site of maximum tenderness or over any area where oedema or redness is discovered.
If no swelling is apparent, the subphrenic spaces should be explored by either an anterior subcostal approach or from behind after removal of the outer part of the 12th rib according to the position of the abscess on imaging. With the posterior approach, the pleura must not be opened and, after the fibres of the diaphragm have been separated, a finger is inserted beneath the diaphragm so as to explore the adjacent area. The aim with all techniques of drainage is to avoid dissemination of pus into the peritoneal or pleural cavities. When the cavity is reached, all of the fibrinous loculi must be broken down with the finger and one or two drainage tubes must be fully inserted. These drains are withdrawn gradually during the next 10 days and the closure of the cavity is checked by sonograms or scanning. Appropriate antibiotics are also given.

2. Describe calcium metabolism and its various disorders seen in surgical practice. (20)

Answer. See the answer of question 2 of Paper – I of 2010.

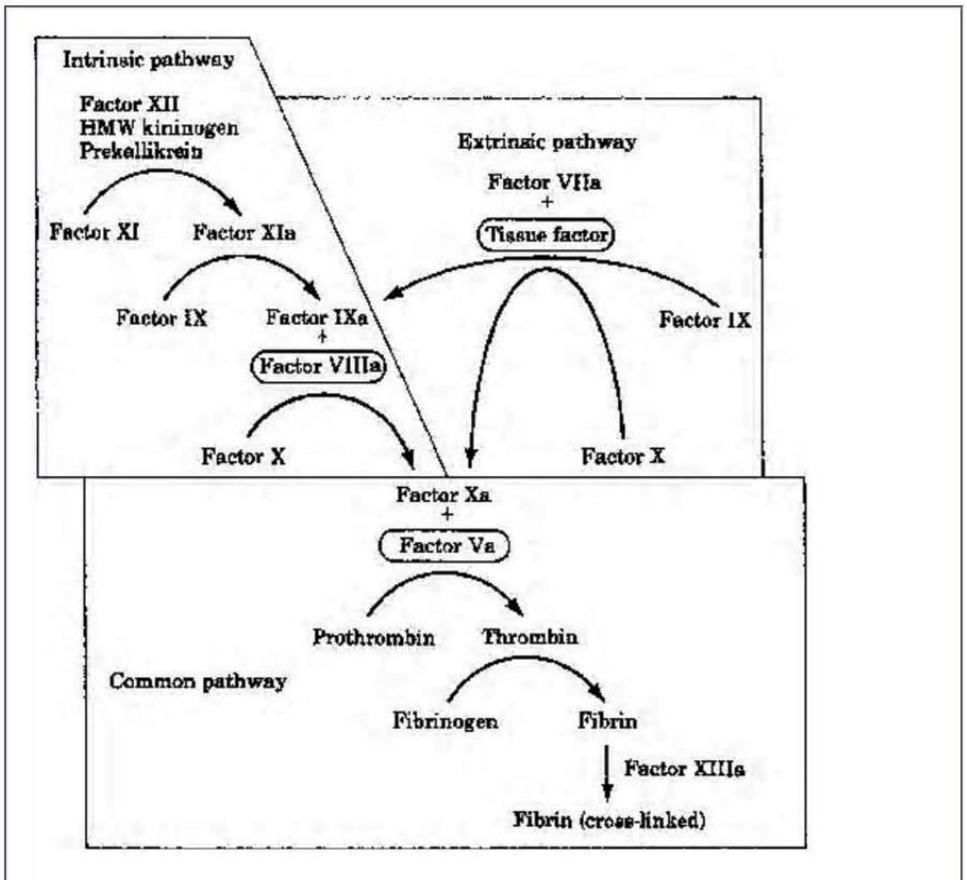
3. Write short notes on: 5 x 6

a) Physiology of blood coagulation & its importance in surgery.

b) Anatomy & physiology of spleen.
c) Development & descent of testis.
d) Complications of pneumoperitoneum induced during laparoscopy.
e) Metabolic changes in high output fistula.

(a) Physiology of blood coagulation & its importance in surgery.

Answer.



Blood coagulation cascade. Plasma zymogens are sequentially converted to active proteases (arrows). Nonenzymatic protein cofactors (ovals) are required at several stages of the cascade. Factors IX and X and prothrombin are activated on phospholipid surfaces. Thrombin cleaves fibrinogen, yielding fibrin monomers that polymerize to form a clot. HMW, high molecular weight.
Coagulation is a complex equilibrium among thrombotic, anticoagulant, and fibrinolytic processes. Disruption of these processes can lead to severe hemorrhagic or thrombotic complications.

Disorders of coagulation:

- Acquired factor deficiencies in surgical patients commonly result from a nutritional deficiency (e.g., vitamin K) or from medical conditions such as liver failure and DIC.

    - Vitamin K deficiency leads to the production of inactive, noncarboxylated forms of prothrombin, factors VII, IX, and X, and proteins C and S. The diagnosis should be considered in a patient with a prolonged PTT that corrects with a 50:50 mixture of normal plasma. Vitamin K deficiency can occur in patients without oral intake within 1 week, with biliary obstruction, with malabsorption, and in those receiving antibiotics or warfarin.
    - Sepsis stimulates the coagulation cascade, whereas levels of anticoagulant factors such as protein C, protein S, and antithrombin III are decreased. This represents an imbalance in hemostasis, causing microvascular thrombi to form. These thrombi further amplify injury and cause distal tissue ischemia and hypoxia. Therapy with activated protein C has been reported to decrease mortality in patients with severe sepsis as defined by an Acute Physiology and Chronic Health Evaluation (APACHE) II score greater than or equal to 25.
    - Liver dysfunction leads to complex alterations in coagulation through decreased synthesis of all clotting factors and inhibitors except vWF. The coagulopathy can be worsened by thrombocytopenia from associated hypersplenism. Spontaneous bleeding is infrequent, but coagulation defects should be corrected prior to invasive procedures. Fresh-frozen plasma (FFP) administration often improves the coagulopathy transiently.
    - Disseminated intravascular coagulation has many inciting causes, including sepsis, extensive trauma or burns, necrotic tissue, intravascular antibody–antigen immune reactions, malignancies, liver failure, obstetric complications, and intravascular prosthetic devices. The pathogenesis of DIC is due to the inappropriate generation of thrombin within the vasculature, leading to the formation of fibrin thrombi, platelet activation, and fibrinolytic activity. DIC often presents with complications from microvascular thrombi that involve the vascular beds of the kidney, brain, lung, and skin.
      Laboratory findings in DIC include thrombocytopenia, hypofibrinogenemia, increased FDPs, and prolonged TT and PTT. Therapy begins with treatment of the underlying cause. Management of hemodynamics and oxygenation is critical. Correction of coagulopathy with platelet transfusions, FFP, and cryoprecipitate should be undertaken for bleeding complications but should not be given empirically.
- Hemophilia is an inherited factor deficiency of either factor VIII (hemophilia A) or factor IX (hemophilia B, Christmas disease). The severity of the disease depends on the factor activity level. The diagnosis is suggested by patient history and an elevated PTT, normal PT, and normal bleeding time. Factor activity assays confirm the diagnosis. Minor bleeding can often be controlled locally without the need for factor replacement therapy. DDAVP stimulates the release of vWF into the circulation, which increases factor VIII levels two- to sixfold. This

may control minor bleeding in patients with mild disease. Major bleeding (e.g., during a surgical procedure) requires factor VIII replacement. Cryoprecipitate contains factor VIII, vWF, and fibrinogen and can be used to treat patients with hemophilia A for control of bleeding. Purified factor IX is the treatment of choice for hemophilia B.

- Other inherited factor deficiencies account for fewer than 10% of severe factor deficiencies. Deficiencies of factor XII, HMWK, or prekallikrein do not cause bleeding and require no treatment.
- Inherited hypercoagulable disorders place patients at risk for thrombosis and include deficiencies in body anticoagulants (antithrombin, protein C, and protein S deficiencies), hyperhomocystinemia, and prothrombin gene mutations. Indications for anticoagulation depend on type, severity, and clinical situation of the disorder.
    - Antithrombin deficiency is an autosomal dominant disorder that presents with recurrent venous thromboembolism, usually in the second decade of life. Assays for AT levels are typically decreased in the setting of acute thrombosis and also if the patient is receiving heparin. Patients with acute thromboembolism or previous history of thrombosis are typically anticoagulated. AT-deficient patients should have the AT level restored to more than 80% of normal activity with AT concentrate prior to operation or childbirth.
    - Protein C deficiency and protein S deficiency are risk factors for venous thrombosis. In a state of protein C or S deficiency, factors Va and VIIIa are not adequately inactivated, thereby allowing unchecked coagulation. Besides the inherited type, protein C deficiency is encountered in patients with liver failure and in those who are receiving warfarin therapy. Symptomatic patients are treated with heparin [or low-molecular-weight heparin (LMWH)] anticoagulation followed by warfarin therapy. In individuals with diminished protein C activity, effective heparin anticoagulation must be confirmed before warfarin initiation because warfarin transiently lowers protein C levels further and potentially worsens the hypercoagulable state. Patients with protein C or S deficiency but with no history of thrombosis typically do not require prophylactic anticoagulation.
    - Activated protein C resistance (factor V Leiden) is a genetic mutation in factor V that renders it resistant to breakdown by activated protein C, placing patients at increased risk for thromboembolism. Routine preoperative screening in asymptomatic patients is unnecessary. Therapy for venous thrombosis consists of anticoagulation with heparin followed by warfarin therapy. The role of long-term warfarin anticoagulation for patients with a single thrombotic event is undefined.
- Acquired hypercoagulable disorders
    - Antiphospholipid antibodies are immunoglobulins that are targeted against antigens composed in part of platelet and endothelial cell phospholipids. Antiphospholipid antibody disorders may be detected by lupus anticoagulant, anticardiolipin, or other antiphospholipid antibodies. Patients

with these antibodies are at risk for arterial and venous thrombosis, recurrent miscarriages, and thrombocytopenia.

- Other acquired hypercoagulable states include malignancies, pregnancy or the use of estrogen therapy, intravascular hemolysis (e.g., hemolytic anemia or after cardiopulmonary bypass), and the localized propensity for thrombosis in arteries that have recently undergone endarterectomy, angioplasty, or placement of prosthetic vascular grafts. Along with effective anticoagulation therapy, treatment should be directed at any identified underlying risk factor.

(b) Anatomy & physiology of spleen.

Answer. Anatomy of spleen:

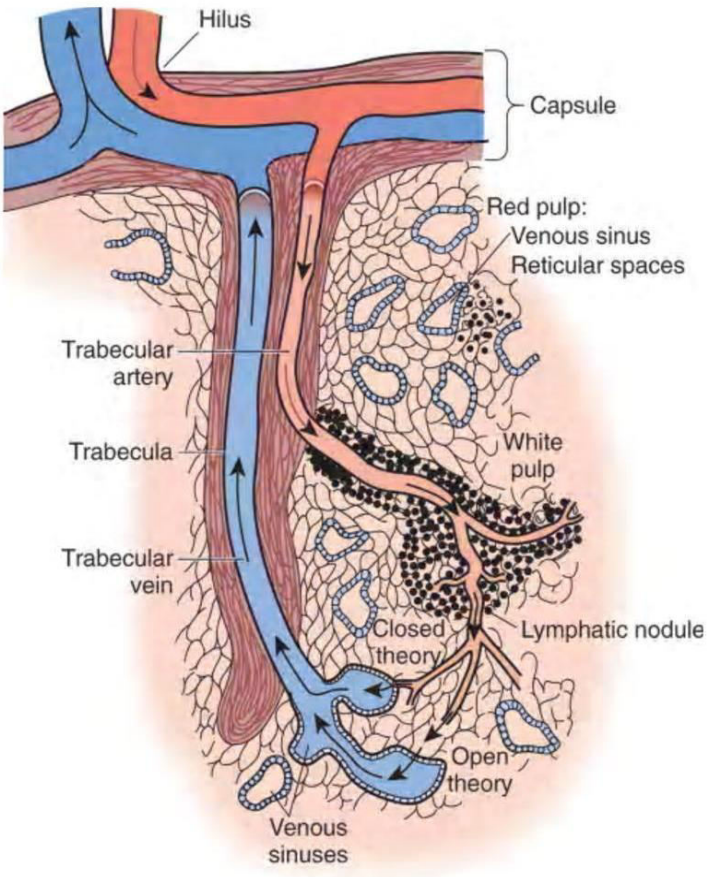
The spleen develops from mesenchymal cells in the dorsal mesogastrium during the fifth week of gestation. The spleen is located in the posterior left upper quadrant of the abdomen. The convex smooth surface of the spleen faces superiorly, posteriorly, and to the left in relation to the abdominal surface of the diaphragm. The diaphragm separates the spleen from the pleura, the left lower lobe of the lung, and the adjacent 9th, 10th, and 11th ribs. The costodiaphragmatic recess of the pleura extends down as far as the inferior border of the normal-sized spleen. The normal size and weight vary somewhat; in adults, the approximate size of the spleen is 12 cm in length, 7 cm in width, and 3 to 4 cm in thickness. The average spleen weight in an adult is 150 g, with a range of 80 to 300 g.

The visceral relationships of the spleen are with the proximal greater curvature of the stomach, the tail of the pancreas, the left kidney, and the splenic flexure of the colon. The parietal peritoneum adheres firmly to the splenic capsule, except at the splenic hilum. The peritoneum extends superiorly, laterally, and inferiorly, creating folds, which form the suspensory ligaments of the spleen. The splenophrenic and splenocolic ligaments are usually relatively avascular. The splenorenal ligament extends from the anterior left kidney to the hilum of the spleen as a two-layered fold in which the splenic vessels and the tail of the pancreas are invested. These two layers continue anteriorly and superiorly to the greater curvature of the stomach to form the two leaves of the gastrosplenic ligament through which the short gastric arteries and veins course. A fibroelastic capsule commonly known as the splenic capsule invests the organ, and from it trabeculae pass into the parenchyma, branching to form a trabec-ular network that subdivides the organ into small compartments.

The splenic artery is a tortuous vessel that arises from the celiac trunk; it courses along the superior border of the pancreas. The branches of the splenic artery include the numerous pancreatic branches, the short gastric arteries, the left gastroepiploic artery, and the terminal splenic branches. The splenic artery divides into several branches within the splenorenal ligament before entering the splenic

hilum, where they branch again into these trabeculae as they enter the splenic pulp. Small arteriolar branches leave the trabeculae, and their adventitial coat becomes replaced by a sheath of lymphatic tissue that accompanies the vessels and their branches until they divide into capillaries. It is these lymphatic sheaths that make up the white pulp of the spleen and that are interspersed along the arteriolar vessels as lymphatic follicles. The interface between the white pulp and the red pulp is known as the marginal zone. As the arterioles lose their sheaths of lymphatic tissue, they traverse the marginal zone and enter the red pulp, which is composed of large branching, thin-walled blood vessels called splenic sinuses and sinusoids, and thin plates of cellular tissue composing the splenic cords.

The venous sinusoids empty into the veins of the red pulp, and these veins drain back along the trabecular veins that empty into at least five major tributaries, ultimately joining to form the splenic vein in the splenorenal ligament. The splenic vein runs inferior to the artery and posterior to the pancreatic tail and body. It receives several short tributaries from the pancreas. The splenic vein joins the superior mesenteric vein at a right angle behind the neck of the pancreas to form the portal vein. The inferior mesenteric vein often empties into the splenic vein; it may also empty into the superior mesenteric vein at or near the confluence of the splenic vein and superior mesenteric vein.



Splenic function:

- The spleen has important hematopoietic functions during early fetal development, with both red and white blood cell production. By the fifth month of gestation, the bone marrow assumes the predominant role in hematopoiesis, and normally there is no significant hematopoietic function left in the spleen.
- Under certain pathologic conditions, however, such as myelodysplasia, the spleen can reacquire its hematopoietic function. Removal of the spleen does not usually result in anemia or leukopenia in an otherwise healthy person.
- Although the hematopoietic function is usually lost during fetal development, the spleen continues to function as a sophisticated filter because of the unique circulatory system and lymphoid organization, and it has blood cell monitoring and management functions as well as important immune functions throughout life.
- The functions of the spleen are closely linked to splenic structure and its unique circulatory system. The arteries flow through the white pulp (lymphoid tissues), after which part of the blood flow goes directly through endothelial cell–lined capillaries into the venous system ("closed" theory).
- Most of the blood flow, however, enters the macrophage-lined reticular meshwork, and the blood flows slowly back to the venous circulation through the venous sinuses ("open" theory). The formed blood elements must pass through slits in the lining of the venous sinuses; if they cannot pass, they are trapped in the spleen and ingested by splenic phagocytes. Experimental animal studies have demonstrated that an intact splenic arterial system is necessary for optimal control of infection. Removal of the spleen results in loss of both the immunologic and filtering functions.
- The most important function of the spleen is probably its mechanical filtration, which removes senescent erythrocytes and likely contributes to control of infection. The spleen is important in clearing circulating pathogens that reside within erythrocytes, for example, malarial parasites, or bacteria such as *Bartonella* species. Mechanical filtration by the spleen may also be important for removal of unopsonized, noningested bacteria from the circulation. It may be particularly important for clearing microorganisms for which the host has no specific antibody.
- Splenic filtering function is important for maintaining normal erythrocyte morphology and function. Normal red blood cells are biconcave and deform relatively easily to facilitate both passage through the microvasculature and optimal oxygen and carbon dioxide exchange. The spleen is an important site for the processing of immature erythrocytes and for repair or destruction of deformed or aged erythrocytes.
- As immature red blood cells pass through the spleen, they may undergo several types of repair, including removal of nuclei and excessive cell membrane from immature cells to convert them from a spherical nucleated to a biconcave anucleated mature morphology.
- Erythrocytes may also undergo repair by having surface abnormalities such as pits or spurs removed. In the asplenic condition, there are several characteristic

alterations in the morphologic appearance of the peripheral red blood cells, with the presence of target cells (immature cells), Howell-Jolly bodies (nuclear remnant), Heinz bodies (denatured hemoglobin), Pappenheimer bodies (iron granules), stippling, and spur cells. Aged red blood cells (120 days) that have lost enzymatic activity and membrane plasticity are trapped and destroyed in the spleen.

Biologic Substances Removed by the Spleen

Normal Subjects
Red blood cell membrane
Red blood cell surface pits and craters
Howell-Jolly bodies
Heinz bodies
Pappenheimer bodies
Acanthocytes
Senescent red blood cells
Patients With Disease
Spherocytes (hereditary-spherocytosis)
Sickle cells, hemoglobin C cells
Antibody-coated red blood cells
Antibody-coated platelets
Antibody-coated white blood cells
Particulate antigen

The filtering function of the spleen is also an important factor in anemic conditions associated with abnormal red blood cell morphology. Abnormal erythrocytes that result from hereditary spherocytosis, sickle cell anemia, thalassemia, or pyruvate kinase deficiency are trapped by the splenic filtering mechanism, resulting in worsening anemia, symptomatic splenomegaly, and occasionally splenic infarction. In autoimmune hemolytic anemia, immunoglobulin G (IgG) bound to the cell membrane targets the red blood cells for splenic destruction by splenic

macrophages. A similar IgG-dependent mechanism is involved in splenic platelet destruction in immune thrombocytopenic purpura (ITP).

Another major function of the spleen is the maintenance of normal immune function and host defenses against certain types of infectious agents. It is well established that people lacking a spleen are at a significantly higher risk for overwhelming postsplenectomy infection (OPSI) with fulminant bacteremia, pneumonia, or meningitis, as compared with those with normal splenic function. Major pathogens in OPSI are organisms such as *Streptococcus pneumoniae,* in which polysaccharide capsules requiring both antibody and complement are important in host defense against these organisms. Asplenic subjects have defective activation of complement by the alternative pathway, leaving them more susceptible to infection.

Asplenic patients have a normal response to reimmunization to an antigen first encountered before splenectomy but do not have an optimal response to new antigen exposure, especially if the antigen is administered intravenously (IV). For organisms such as the encapsulated bacteria, much higher quantities of antibody are necessary for effective clearance. The spleen, with its specialized circulatory system and large supply of macrophages that are capable of ingestion of organisms not optimally opsonized with antibody, greatly enhances their clearance. Asplenic subjects have been found to have subnormal IgM levels, and their peripheral blood mononuclear cells exhibit a suppressed immunoglobulin response.

The spleen is a major site of production for the opsonins properdin and tuftsin, and removal of the spleen results in decreased serum levels of these factors. Properdin can initiate the alternative pathway of complement activation to produce destruction of bacteria as well as foreign and abnormal cells. Tuftsin is a tetrapeptide that enhances the phagocytic activity of both polymorphonuclear leukocytes and mononuclear phagocytes. The spleen is the major site of cleavage of tuftsin from the heavy chain of IgG, and circulating levels of tuftsin are suppressed in asplenic subjects. Neutrophil function is decreased in asplenic patients, and the defect appears to result from the absence of a circulating mediator.

(c) Development & descent of testis.

Answer.

Descent of the testes

Between the 3rd month of pregnancy and its end the testes become transferred from the lumbar area (ventro-medial to the mesonephros) into the future scrotum. This transfer is due to a combination of **growth processes and hormonal influences.** The **gubernaculum testis** also plays a decisive role in this phenomenon.

The **gubernaculum testis** arises in the course of the 7th week from

the lower**gubernaculum**, after the mesonephros has atrophied. Cranially it has its origin at the testis and inserts in the region of the genital swelling (future scrotum).
At the same time, at the inguinal canal along the lower gubernaculum, an**evagination of the peritoneum** arises, the **vaginal process**, on which the testes will slide through the inguinal canal.

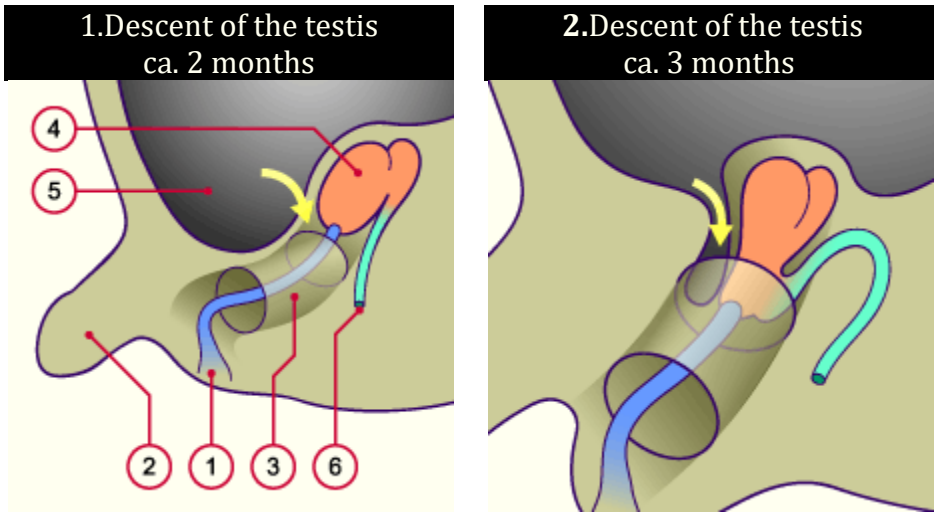
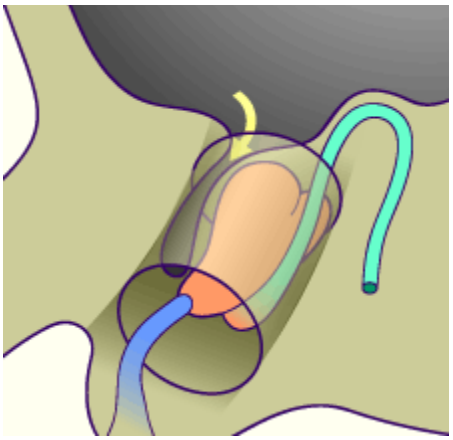


Fig.1

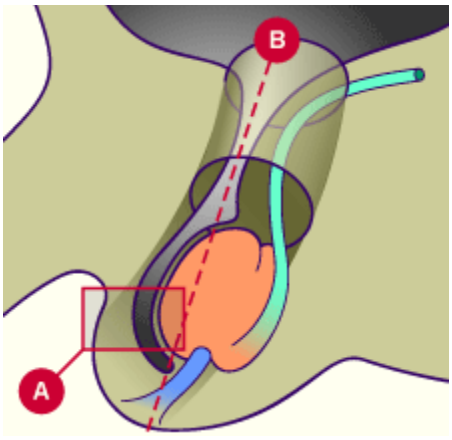
The yellow arrow shows the location of the protrusion of the peritoneum and the beginning of the testicular descent into the inguinal canal.

Fig. 2

In this diagram, the beginning of the formation of the vaginal process is visible. It enters with the testis into the inguinal canal. Shown in blue is the gubernaculum that becomes increasingly shorter.



1 Gubernaculum testis
2 Penis
3 Inguinal canal



4 Testis
5 Peritoneal cavity
6 Deferent duct

Fig. 3
Between the 3rd and 7th month of pregnancy the testes remain near the inguinal canal in order to pass through it. The vaginal process lengthens while the gubernaculum shortens, thereby drawing the testis, the deferent duct and its vessels on both sides downwards.

Fig. 4
In the 9th month of pregnancy (but also sometimes only after birth) the testes reach the scrotum. The vaginal process forms now a serous bilaminar structure on the front side of the testis.

In that the vaginal process lengthens downwardly, it takes the muscle fibers of the oblique

5 - Section through the scrotum at the time of birth according to A

internal muscle and the transverse muscle with it.

The muscle fascia of the transverse muscle is the innermost layer and in the scrotal region, it forms the **internal spermatic fascia** of the spermatic cord and the scrotum.

The muscle layer of the musculus cremaster is formed from **fibers of the oblique internal and transverse muscles**.

Externally, the external spermatic fascia is formed from the superficial**aponeurosis of the oblique external abdominal muscle**.

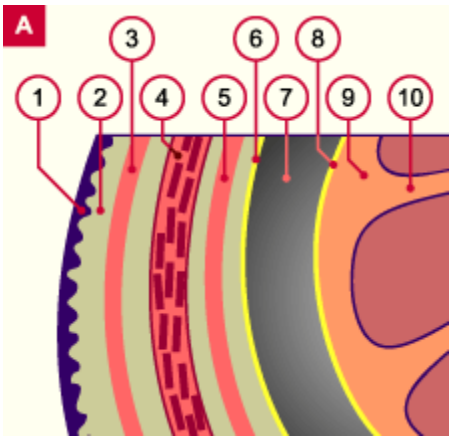


Fig. 5
Detail of the various layers that have formed in the scrotum by the end of the pregnancy.

1 Epidermis
2 Dermis (tunica dartos)
3 External spermatic fascia
4 Musculus cremaster
5 Internal spermatic fascia
6 Parietal lamina of the tunica vaginalis
7 Virtual cavity between the
8 two layers of the tunica vaginalis
9 Visceral lamina of the tunica
10 vaginalis
Tunica albuginea
Interlobular septum of the testis

The region, where the testes pass through the abdominal wall, is called the **inguinal canal**.
Between the 7th and the 12th week the **gubernaculum shortens** and pulls the testes, the deferent duct and its vessels downwards.
Between the 3rd and 7th month the **testes** stay in the area of the inguinal canal so they can enter into it. They reach the scrotum at roughly the time of birth under the influence of the androgen hormone.

(d) Complications of pneumoperitoneum induced during laparoscopy.

Answer. Introduction: Laparoscopic surgery is nowadays a common daily-performed procedure worldwide, replacing many types of open surgeries. It has the benefits of small incision, improved cosmetic aspects, less postoperative pain, and quick recovery time to normal activities.The most commonly used gas for insufflation is carbon dioxide. Carbon dioxide CO_2 pneumoperitoneum and

While in the first year of life the upper part of the vaginal process becomes obliterated, there remains only the **peritoneo-vaginal ligament**. The lower portion persists as the **tunica vaginalis testis**, which consists of a parietal and a visceral layer.

increased intraabdominal pressure can induce many pathophysiologic disturbances, requiring the anesthesiologist to be well alert during the operation for necessary management. Moreover, advanced laparoscopic surgeries are being used also on older patients and in critically ill patients, requiring technically demanding anesthesia.

Pathophysiologic changes

- **Respiratory changes:** The physiology of respiratory system is affected by pneumoperitoneum. With insufflation, causing an increase in intraabdominal pressure (IAP), the diaphragm is pushed upwards causing stiffness of the chest wall, causing the total volume of the lungs to be reduced. Hence the pulmonary compliance is decreased to 35–40% and also a non-negligible increase in the maximum respiratory system resistance Hypoxemia may occur from a ventilationperfusion mismatch and intrapulmonary shunting but is rare in healthy patients.

Carbon dioxide is usually administered at a rate of 1–2 ml/min. Being a highly soluble gas, it is readily absorbed into the circulation through the peritoneum, causing hypercapnia and acidosis.

Several studies have shown the effect of CO2 pneumoperitoneum on the arterial partial pressure of CO2 (PaCO2) and end-tidal CO2 (ETCO2)Carbon dioxide is mainly excreted by the lungs, depending on alveolar and mixed venous CO2 exchange rates, which are themselves controlled by the cardiac output, alveolar ventilation and respiratory quotient . Normal excretion of CO2 is 100–200 mL/min and is increased by 14–48 mL/min when CO2 is administered intraperitoneally. After a long laparoscopic operation, achieving a normal CO2 value can take several hours after desufflation, since high use of peripheral storage capacity will lengthen the duration of increased PaCO2.

- **Cardiovascular changes:** Cardiovascular system effects during CO2 pneumoperitoneum are caused mainly by hypercarbia followed by acidosis and increased intra-abdominal pressure. A euvolemic status is of great importance prior to surgery to reduce any cardiac depression via reduced preload caused by the pneumoperitoneum.

  Hypercarbia has direct and indirect sympathoadrenal stimulating effects on cardiovascular functions. These effects are not pronounced with mild hypercarbia (PaCO2 45–50 mmHg), whereas moderate to severe hypercarbia affects cardiac function since it is then a myocardial depressant and has direct vasodilatary effect.
- **Renal changes:** Oliguria is the most common renal effect of pneumoperitoneum. Different mechanisms are involved in the reduction of the urine amount during IAP. IAP also activates of the renin-angoitensin-aldosterone system following decreased renal perfusion, which results in renal cortical vasoconstriction.

Level of ADH, renin, and aldosterone significantly increased during laparoscopic GBP. Renal blood flow has been measured during increasing IAP, and a gradual decrease in RBF up to 75% was observed upon reaching a pressure of 15 mmHg.those with maintenance fluid, whereas this change was not noticed in well-

hydrated animals with adequate volume loading. Controversially, there are also studies that reported no RBF changes during pneumoperitoneum.

- **Splanchnic changes:** The splanchnic circulation is also affected during raised IAP. Depending on intra-abdominal pressures, studies in animals have show decrease in splanchnic macro and micro-circulation. Signs of hepatocytic damage [38] were noticed, with increase of glutamic oxaloacetic transaminase and glutamic pyruvic transaminase. Impaired Kupffer cell function and gastric intramucosal pH drop were also noticed.

Complications:

- Anesthetists should always bear in mind the possible pulmonary complications of pneumoperitoneum like gas embolism, barotraumas, hypoxemia, pulmonary edema, atelectasis, subcutaneous emphysema, pneumothorax, pneumomediastinum and pneumopericardium.
- Carbon dioxide embolism is rare, occurring in about 0.0014–0.6% of laparoscopic surgeries , but with a mortality rate of about 28%. Carbon dioxide enters the circulation through an opening in a damaged vessel under raised IAP. It can also occur if the Veress needle is misplaced into a vessel or parenchymal organ. Transesophagal echocardiography studies have shown bubbling of CO2 in the right heart chamber in 68% asymptomatic patients during pneumoperitoneum.
  Clinical manifestations of gas embolism are severe drop in blood pressure, cyanosis, cardiac arrhythmias or asystole. A mill-wheel murmur may be heard on auscultation of the heart, and ETCO2 will increase.
- Subcutaneous emphysema occurs in 0.3–3.0% laparoscopic surgeries. Mild to severe subcutaneous emphysema has generally not been shown to have clinical effects, but upper airway obstruction must be considered if there is neck involvement.
- Pneumothorax can occur following peritoneum visceral tear, parietal pleura tear during resection around the esophagus or congenital defect in the diaphragm through which CO2 gas travels . Extension of emphysema can also occur, causing pneumothorax and pneumomediastinum. It has been reported that even subcutaneous emphysema arising from extraperitoneal inguinal hernia repair has extended to cause pneumothorax and pneumomediastinum.

Pneumothorax should be differentiated with capnothorax following CO2 diffusion into the intrapleural space.

- With both pneumothorax and capnothorax, the ETCO2 increases, so capnothorax can be suspected if the mean airway pressure increases with a drop in SpO2, and confirmation should be made with a chest x-ray. Pneumopericardium can develop when CO2 is forced into the mediastinum and pericardium. It can also occur if CO2 enters the defect in the membranous portion of the diaphragm, resulting in a communication between the pericardial and peritoneal cavities .
- Cardiovascular complications such as hypertension, arrhythmias, hypotension and cardiac arrest have been reported with pneumoperitoneum. Hypertension seems to have a higher incidence at the beginning of insufflation when the blood volume in the splanchnic vasculature is reduced due to increased IAP, thereby increasing preload and arterial pressure. Arrhythmias occur in up to 14–27% of

laparoscopies. These must be differentiated from arrhythmias caused by release of catecholamine. Sinus tachycardia and ventricular extrasystoles are usually more benign, and dangerous ones like bradyarrhythmias (bradycardia, nodal rhythm, atrioventricular dissociation and asystole) are also seen. Bradyarrhythmias arise due to the vagal nerve mediated cardiovascular response following acute stretching of the peritoneum. Hypotension, which occurs in up to 13% of laparoscopies, a potentially serious complication . IAP of 20 mmHg or more results in compression of the inferior vena cava, reducing the venous return. Cardiac output is reduced, leading to hypotension. This complication is aggravated by high intrathoracic pressure.

- Vasovagal responses to quick intraperitoneal CO2 insufflation and gas embolism have both been related to cardiac arrest. The possible development of acute tubular necrosis in response to long lasting hypoperfusion from pneumoperitoneum is controversial.
- Free radicals are released by inflation and deflation of the peritoneum. Oxygen and organic free radicals may contribute to ischemia reperfusion phenomena or chemical carcinogenesis. Still, the impact of production of free radicals is smaller than with open surgery injury.

(e) Metabolic changes in high output fistula.

Answer. Fluid losses: The effect of fistula output is best appreciated by an analysis of daily gastrointestinal secretions.

Source	Amout of secretion per day
The salivary and gastric glands	1.5 L
Pancreas	1 L
Bile	1 L
Duodenal Brunner's glands	200 ml
Small intestine	1.8 L

Therefore, 8 to 10 L of fluid flows through the jejunum each day, depending on the oral intake. In the intact functioning intestine 98% of this fluid is (re)absorbed; leaving only 100 to 200 mL of fluid to be excreted in the stool. Most (7–8 L) of this absorption occurs proximal to the ileocecal valve.
In the case of proximal fistulae there is a potential for serious compromise of fluid balance even when there is a reasonable extent of more distal intact small intestine, in that large fluid volumes may exit through the fistula rather than proceeding distally to allow absorption to occur. Enzymes and electrolytes (especially sodium and magnesium) inevitably accompany fluid, as do essential nutrients. The nutritional and fluid balance issues are less with distal fistulae, as small intestinal absorption leaves less fluid and fewer nutrients to be lost as long as the small bowel proximal to the fistula is in good functional condition. Reduction of gastrointestinal tract fluid secretion is an integral part of fistula treatment, as discussed in the following.

Metabolic derangements: It is not only the volume that is critical with high-output fistulae but also the composition of the secretions at various levels of the gastrointestinal tract.

Gastric juice contains hydrogen, chloride, sodium, and potassium ions, the concentrations differing significantly between the fasting and fed states. Feeding increases the hydrogen ion concentration from about 50 mM to up to 100 mM, increases chloride from 90 to 120 mM, but decreases sodium from 40 to about 25 mM.

Duodenal fistulae consequently lead to loss of fluid rich in hydrogen and chloride ions, especially after eating, culminating in hypochloremic, hypokalemic metabolic alkalosis together with hypovolemia.

The combination of **Brunner's gland secretions, bile, and pancreatic secretions** serve to neutralize gastric acid secretion. The main electrolytes in bile are sodium and chloride, whereas bicarbonate and sodium make up most of the pancreatic juice. The sodium concentration of about 140 mM is independent of the activity state of the pancreas, but the bicarbonate concentration increases from 40 to up to 145 mM following stimulation. Fistulous discharge at the level of the **upper jejunum** is therefore associated with significant losses of sodium, chloride, and bicarbonate ions.

Enzymes and skin: By the time the gastric contents have reached the jejunum, an array of enzymes has been released and activated within the gut lumen. Salivary amylase and gastric pepsin, together with potent pancreatic proteolytic enzymes including trypsin, chymotrypsin, and carboxypeptidases, commence carbohydrate and protein digestion. Other pancreatic enzymes including lipase, amylase, phospholipase, and cholesterol esterase are also activated and are found in the effluent from high-output fistulae. These enzymes contribute to delay in wound closure and can lead to major excoriation of previously normal skin.

The role of gut hormones: Gastric acid secretion is accompanied by the release of gastrin, histamine, somatostatin, and cholecystokinin (CCK). Gastrin and histamine exert a paracrine and endocrine effect to activate the acid-secreting parietal cell and the somatostatin-secreting D cell. CCK is released from duodenal and upper jejunal mucosa when food enters the small bowel. In addition to stimulating pancreatic enzyme secretion, CCK is a potent gastrin stimulus and serves to perpetuate gastric acid secretion. It also stimulates the release of gastric somatostatin. Both of these actions are mediated in an endocrine manner. Somatostatin, on the other hand, is a potent inhibitory hormone and switches off gastric acid secretion by direct action on the parietal and enterochromaffin-like cells and indirectly by inhibiting gastrin secretion. Somatostatin thus mediates its actions in endocrine, paracrine, and neurocrine manners to regulate gastrointestinal motility and secretion.

Glucagon-like peptide 2 (GLP2) has attracted considerable attention as a therapeutic agent for intestinal injury since its identification as a potent stimulator of mucosal epithelial proliferation.[11] Preliminary trials in patients with short bowel syndrome have yielded improvements in intestinal absorption of both fluids and nutrients. A

GLP2-induced reduction of gastric acid secretion and gastric motility is perhaps particularly relevant in terms of its potential as a therapeutic agent for patients with ECF. As yet there are no studies specifically examining the effects of GLP2 on high-output fistulae.

Associated short bowel features: Fistulae related to intestinal surgery, inflammatory bowel disease, and radiation enteritis are often accompanied by short bowel syndrome, which may occur because the residual bowel is actually short, because it is functionally impaired, or because the dominance of effluent from a proximal fistula precludes reabsorption from distal (otherwise intact) intestine. Problems with fluid balance and reduced absorptive capacity for nutrients are then to be expected. Early recognition of these complicating factors is crucial to a good outcome.

4. Answer in brief: **$4 \times 7^1/_2$**

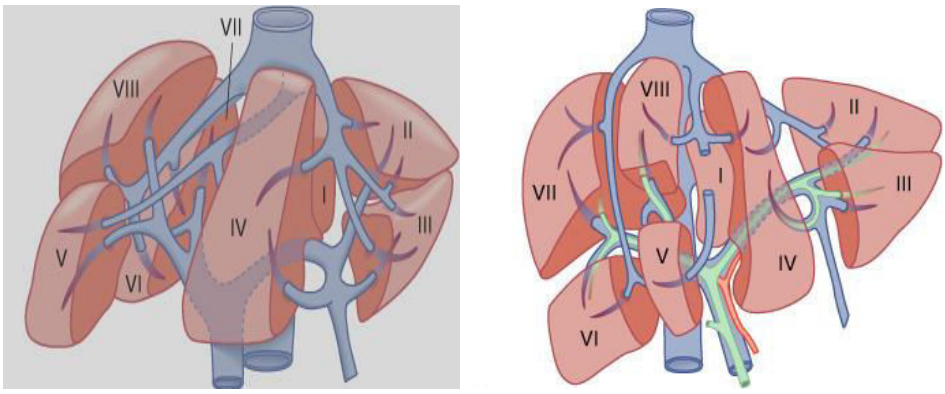
a) Surgical anatomy of liver & its importance in liver resection.
b) Pathogenesis of septic shock.
c) Postoperative pain relief.
d) Surgical pathology of salivary gland tumours.

(a) Surgical anatomy of liver & its importance in liver resection.

Answer. Historically, the liver was divided into left and right lobes by the obvious external landmark of the falciform ligament. Not only was this description oversimplified, but it was also anatomically incorrect in relationship to the blood supply to the liver. Later, more accurate descriptions of the lobar anatomy of the liver were developed. The liver was divided into right and left lobes determined by portal and hepatic vein branches. Briefly, a plane without any surface markings running from the gall-bladder to the left side of the IVC (known as the portal fissure, or Cantlie's line) divided the liver into right and left lobes. The right lobe was further divided into anterior and posterior segments. The left lobe was divided into a medial segment (also known as the quadrate lobe) that lies to the right of the falciform ligament and umbilical fissure and a lateral segment lying to the left. This system, although anatomically more correct, is only sufficient for mobilization of the liver and simple hepatic procedures. It does not describe the more intricate and functional segmental anatomy that is essential to understand before pursuing complex hepatobiliary surgery.

The functional anatomy of the liver is composed of eight segments, each of which is supplied by a single portal triad (also called a pedicle) composed of a portal vein, hepatic artery, and bile duct. These segments are further organized into four sectors that are separated by scissurae containing the three main hepatic veins. The four sectors are even further organized into the right and left liver (the phrase right and left liver is preferable to right and left lobe because there is no external mark that allows the identification of the right and left liver). This system was originally

described in 1957 by Woodsmith and Goldburne as well as Couinaud and defines hepatic anatomy as it is most relevant to surgery of the liver.



A B

- Segmental anatomy of the liver as seen at laparotomy in the anatomic position **(A)** and in the ex vivo position (B).
- The functional anatomy is more often seen as cross-sectional imaging.The main scissura contains the middle hepatic vein, which runs in an anteroposterior direction from the gallbladder fossa to the left side of the vena cava and divides the liver into right and left hemi-livers. The line of the main scissura is also known as Cantlie's line. The right liver is divided into an anterior (segments V and VIII) and posterior (segments VI and VII) sector by the right scissura, which contains the right hepatic vein. The right portal pedicle, composed of the right hepatic artery, portal vein, and bile duct, splits into right anterior and posterior pedicles that supply the segments of the anterior and posterior sectors.
- The left liver has a visible fissure along its inferior surface called the umbilical fissure. The ligamentum teres (containing the remnant of the umbilical vein) runs into this fissure. The falciform ligament is contiguous with the umbilical fissure and ligamentum teres. The umbilical fissure is not a scissura, does not contain a hepatic vein, and in fact, contains the left portal pedicle (triad containing the left portal vein, hepatic artery, and bile duct), which runs in this fissure, branching to feed the left liver. The left scissura runs posterior to the ligamentum teres and contains the left hepatic vein. The left liver is split into an anterior (segments III and IV) and posterior (segment II—the only sector composed of a single segment) sector by the left scissura.
- At the hilum of the liver, the right portal triad has a short extrahepatic course of about 1 to 1.5 cm before entering the substance of the liver and branching into anterior and posterior sectoral branches. The left portal triad, however, has a long extrahepatic course of up to 3 or 4 cm and runs transversely along the base of segment IV in a peritoneal sheath that is the upper end of the lesser omentum. The left portal triad, as it runs along the base of segment IV, is separated from the liver substance by connective tissue known as the hilar plate.The continuation of the left portal triad runs anteriorly and caudally in the umbilical

fissure and gives branches to segments II and III and recurrent branches to segment IV.

- The caudate lobe (segment I) is the dorsal portion of the liver and embraces the IVC on its posterior surface and lies posterior to the left portal triad inferiorly and the left and middle hepatic veins superiorly. The main bulk of the caudate lobe is to the left of the IVC, but inferiorly, it traverses between the IVC and left portal triad, where it fuses to the right liver (segments VI and VII). This part of the caudate lobe is known as the right portion or the caudate process. The left portion of the caudate lobe lies in the lesser omental bursa and is covered anteriorly by the gastrohepatic ligament (lesser omentum) that separates it from segments II and III anteriorly. The gastrohepatic ligament attaches to the ligamentum venosum (sinus venosus remnant) along the left side of the left portal triad.
- The vascular inflow and biliary drainage to the caudate lobe comes from both the right and left systems. The right side of the caudate (the caudate process) largely derives its portal venous supply from the right portal vein or the bifurcation of the main portal vein, whereas the left portion of the caudate derives its portal venous inflow from the left main portal vein. The arterial supply and the biliary drainage of the right portion are generally through the right posterior sectoral system and the left portion through the left main vessels. The hepatic venous drainage of the caudate is unique in that multiple small veins drain posteriorly directly into the IVC.
- The posterior edge of the left side of the caudate terminates into a fibrous component that attaches to the crura of the diaphragm and also runs posteriorly, wrapping behind the IVC and attaching to segment VII of the right liver. Up to 50% of the time, this fibrous component is composed either partially or completely of liver parenchyma, and thus liver tissue may completely encircle the IVC. This important structure is known as the IVC ligament and is important when mobilizing the right liver or the caudate lobe off of the vena cava.
- Anomalous development of the liver is uncommonly encountered. Complete absence of the left liver has been reported. A tongue of tissue extending inferiorly off of the right liver has been described (Riedel's lobe). Rare cases of supradiaphragmatic liver in the absence of a hernia sac have been noted.

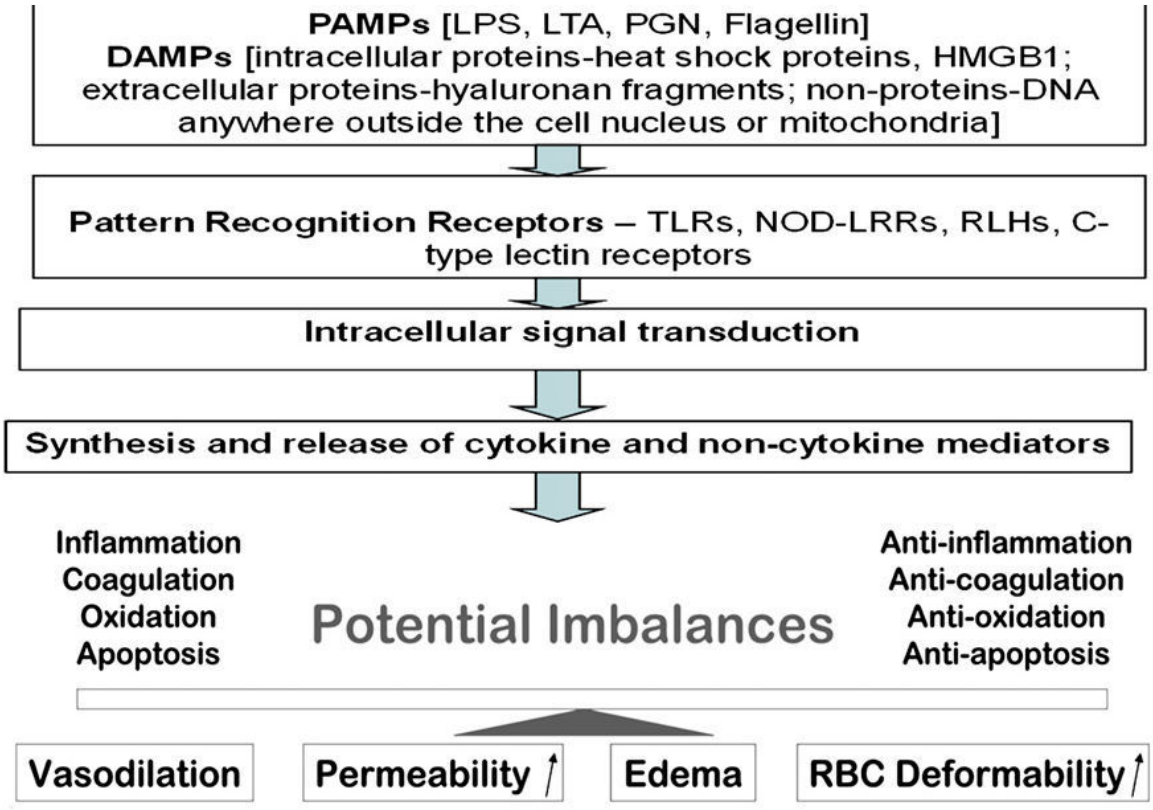
Nomenclature for Most Common Major Anatomic Hepatic Resections

SEGMENTS	**COUINAUD, 1957**	**GOLDSMITH AND WOODBURNE, 1957 BRISBANE, 2000**	
V-VIII	Right hepatectomy	Right hepatic lobectomy	Right hemi-hepatectomy
IV-VIII	Right lobectomy	Extended right hepatic lobectomy	Right trisectionectomy
II-IV	Left hepatectomy	Left hepatic lobectomy	Left hemi-hepatectomy

SEGMENTS	COUINAUD, 1957	GOLDSMITH AND WOODBURNE, 1957 BRISBANE, 2000	
II, III	Left lobectomy	Left lateral segmentectomy	Left lateral sectionectomy
II, III, IV, V, VIII	Extended left hepatectomy	Extended left lobectomy	Left trisectionectomy

(b) Pathogenesis of septic shock.

Answer. Most cases of septic shock (approximately 70%) are caused by endotoxin-producing Gram-negative bacilli. Endotoxins are bacterial wall lipopolysaccharides (LPS) consisting of a toxic fatty acid (lipid A) core common to all Gram-negative bacteria, and a complex polysaccharide coat (including O antigen) unique for each species. Analogous molecules in the walls of Gram-positive bacteria and fungi can also elicit septic shock. Free LPS attaches to a circulating LPS-binding protein, and the complex then binds to a specific receptor (CD14) on monocytes, macrophages, and neutrophils. Engagement of CD14 (even at doses as minute as 10 pg/mL) results in intracellular signaling via an associated "Toll-like receptor" protein 4 (TLR-4), resulting in profound activation of mononuclear cells and production of potent effector cytokines such as IL-1 and TNF-α. These cytokines act on endothelial cells and have a variety of effects including reduced synthesis of anticoagulation factors such as tissue factor pathway inhibitor and thrombomodulin. The effects of the cytokines may be amplified by TLR-4 engagement on endothelial cells. TLR-mediated activation helps to trigger the innate immune system to efficiently eradicate invading microbes. At high levels of LPS, the syndrome of septic shock supervenes; the same cytokine and secondary mediators, now at high levels, result in systemic vasodilation (hypotension), diminished myocardial contractility, widespread endothelial injury and activation, causing systemic leukocyte adhesion and diffuse alveolar capillary damage in the lung activation of the coagulation system, culminating in disseminated intravascular coagulation (DIC). The hypoperfusion resulting from the combined effects of widespread vasodilation, myocardial pump failure, and DIC causes multiorgan system failure that affects the liver, kidneys, and central nervous system, among others. Unless the underlying infection (and LPS overload) is rapidly brought under control, the patient usually dies.



(c) Postoperative pain relief.

Answer. See the answer of question 4(c) of Paper-I of 2009.

(d) Surgical pathology of salivary gland tumours.

Answer.

Tumors of the Major and Minor Salivary Glands
Benign
Pleomorphic adenoma
Warthin's tumor
Capillary hemangioma
Oncocytoma
Basal cell adenoma
Canalicular adenoma
Myoepithelioma
Sialadenoma papilliferum

Intraductal papilloma

Inverted ductal papilloma

Malignant

Acinic cell carcinoma

Mucoepidermoid carcinoma

Adenoid cystic carcinoma

Polymorphous low-grade adenocarcinoma

Epithelial-myoepithelial carcinoma

Basal cell adenocarcinoma

Sebaceous carcinoma

Papillary cystadenocarcinoma

Mucinous adenocarcinoma

Oncocytic carcinoma

Salivary duct carcinoma

Adenocarcinoma

Myoepithelial carcinoma

Malignant mixed tumor

Squamous cell carcinoma

Small cell carcinoma

Lymphoma

Metastatic carcinoma

Carcinoma ex pleomorphic adenoma

Pleomorphic adenoma:

- Because of their remarkable histologic diversity, these neoplasms have also been called mixed tumors.
- They represent about 60% of tumors in the parotid, are less common in the submandibular glands, and are relatively rare in the minor salivary glands.
- They are benign tumors that are derived from a mixture of ductal (epithelial) and myoepithelial cells, and therefore they show both epithelial and mesenchymal differentiation.
- They also reveal epithelial elements dispersed throughout a matrix along with varying degrees of myxoid, hyaline, chondroid (cartilaginous), and even osseous tissue. In some tumors, the epithelial elements predominate; in others, they are

present only in widely dispersed foci. Little is known about the origins of these neoplasms except that radiation exposure increases the risk.

- Equally uncertain is the histogenesis of the various components. A currently popular view is that all neoplastic elements, including those that appear mesenchymal, are of either myoepithelial or ductal reserve cell origin (hence the designation pleomorphic adenoma).
  - Morphology: Most pleomorphic adenomas present as rounded, well-demarcated masses rarely exceeding 6 cm in greatest dimension.Although they are encapsulated, in some locations (particularly the palate) the capsule is not fully developed, and expansile growth produces tongue like protrusions.

Gross pathology	• Histology
– Smooth – Well-demarcated – Solid – Cystic changes	– Mixture of epithelial, myopeithelial and stromal components – Epithelial cells: nests, sheets, ducts, trabeculae – Stroma: myxoid, chrondroid, fibroid, osteoid – No true capsule – Tumor pseudopods

Warthin's Tumor:

• Gross pathology	• Histology
▪ Encapsulated ▪ Smooth/lobulated surface ▪ Cystic spaces of variable size, with viscous fluid, shaggy epithelium ▪ Solid areas with white nodules representing lymphoid follicles.	○ Papillary projections into cystic spaces surrounded by lymphoid stroma ○ Epithelium: double cell layer ○ Luminal cells ○ Basal cells ○ Stroma: mature lymphoid follicles with germinal centers

Oncocytoma:

• Gross pathology	• Histology

– Encapsulated – Homogeneous, smooth – Orange/rust color	– Cords of uniform cells and thin fibrous stroma – Large polyhedral cells – Distinct cell membrane – Granular, eosinophilic cytoplasm – Central, round, vesicular nucleus

Mucoepidermoid Carcinoma:

• Gross pathology	• Histology
- Well-circumscribed to partially encapsulated to unencapsulated - Solid tumor with cystic spaces	▪ Low-grade ▪ Mucus cell > epidermoid cells ▪ Prominent cysts ▪ Mature cellular elements.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES

MS (General Surgery) Examination, 2006

PAPER I

Time Allowed: 3 Hours **Full Marks: 100**

1. Describe the surgical anatomy of chronic pancreatitis. Give a brief account of cysts in relation to the pancreas. (25)

2. Describe the structure of a 'nephron'. Discuss the role of the kidney in the maintainance of acid – base balance of the body. (25)

3. Describe the surgical anatomy of axilla. Discuss about the diseases that implicate it. (25)

4. Give a brief account of rational use of antibiotics in surgical practice. Comment about prevention of abuse of antibiotics. (25)

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES
MS (General Surgery) Examination, 2006

April, 2006

PAPER I

Time Allowed: 3 Ho **Full Marks: 100**

1. Describe the surgical anatomy of chronic pancreatitis. Give a brief account of cysts in relation to the pancreas. (25)

Answer. I don't know what the examiner is meant by surgical anatomy of chronic pancratitis. A detail discussion on chronic pancratitis has been done in the answer of question 1 of Paper II of 2008.

Here the surgical anatomy of pancreas is described.

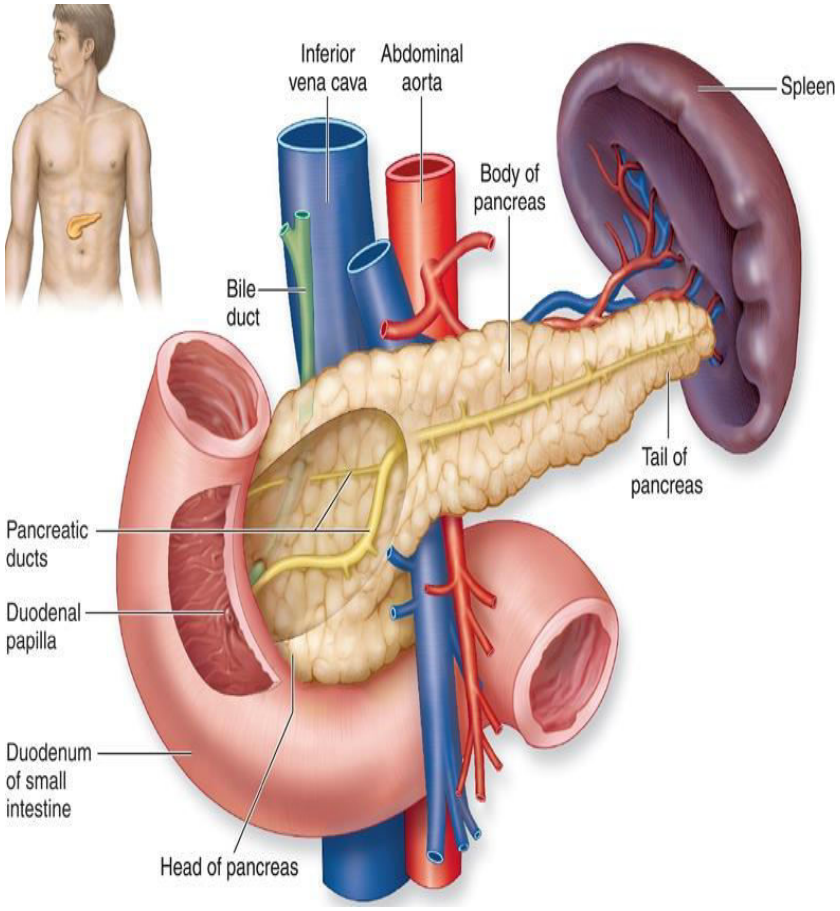


Fig: 1

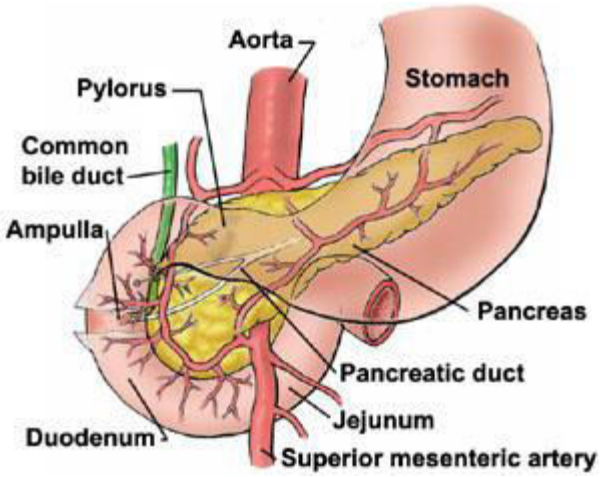
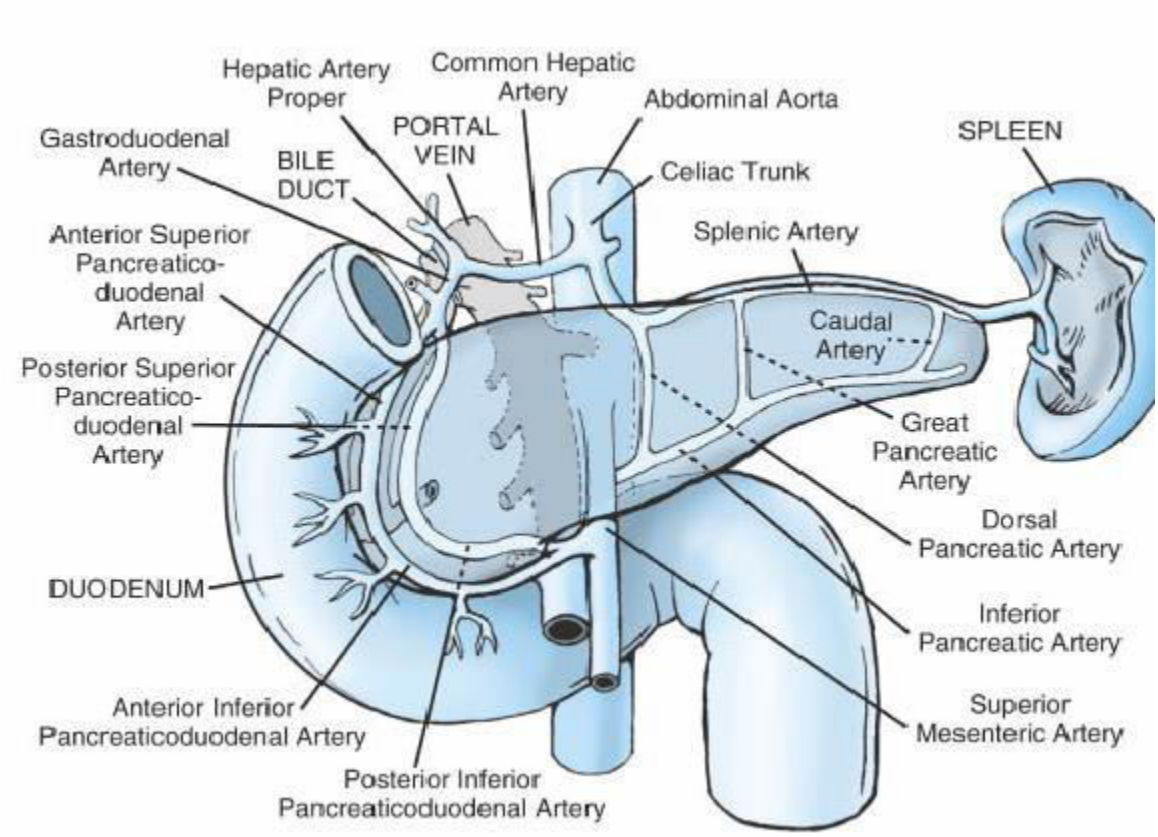


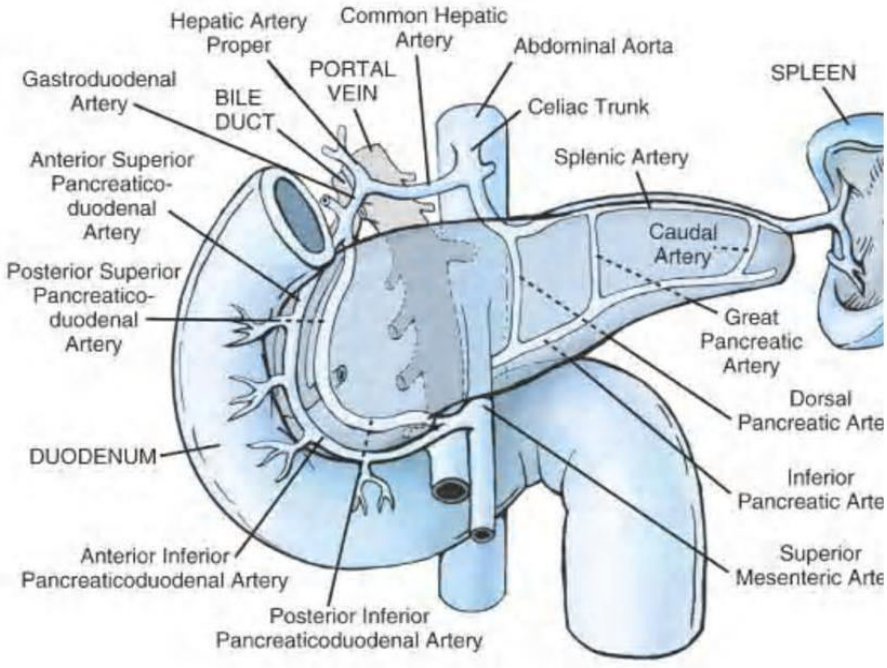
Fig: 2

- The pancreas lies across the upper abdomen. It extends from the inside of the C loop of the duodenum to the hilum of the spleen. It lies behind the stomach and the peritoneum (the thin lining of the abdominal cavity). The head of the pancreas lies in the C- shaped duodenal curve while the tail of the pancreas lies toward the spleen. The body of the pancreas lies between the head and the tail. (Figure 1)

- The blood supply comes from branches of the celiac, superior mesenteric and splenic artery.

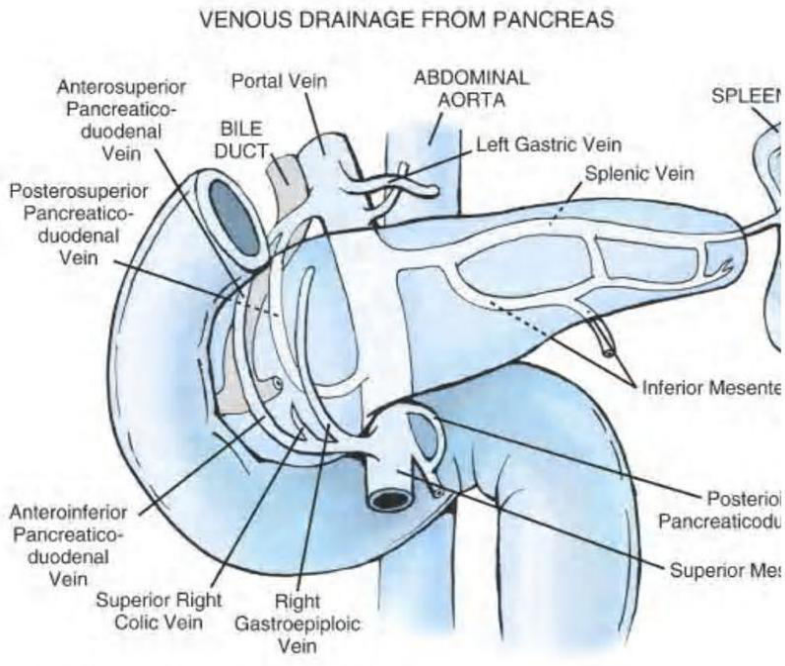


ARTERIAL SUPPLY TO THE PANCREAS



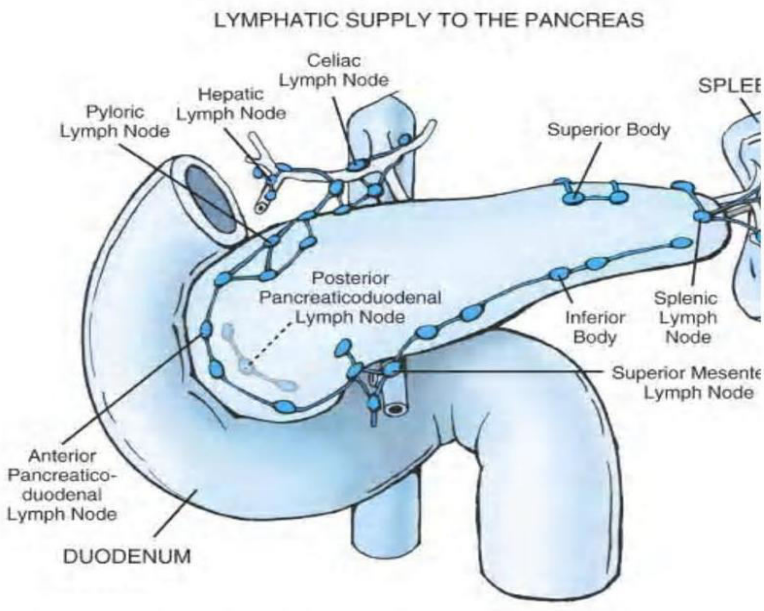
Arterial supply to the pancreas. Multiple arcades in the head and body of the pancreas provide a rich blood supply. The head of the pancreas cannot be resected without devascularizing the duodenum unless a rim of pancreas containing the pancreaticoduodenal arcade is preserved.

- Blood drains by veins to the large portal vein that drains into the liver.



- Venous drainage from the pancreas: The venous drainage of the pancreas follows a pattern similar to the arterial supply, with the veins usually superficial to the arteries. Anterior traction on the transverse colon can tear fragile branches along the inferior border of the pancreas, which then retract into the parenchyma of the pancreas. Venous branches draining the pancreatic head and uncinate process enter along the right lateral and posterior sides of the portal vein. There are usually no anterior venous tributaries, and a plane can usually be developed between the neck of the pancreas and the portal and superior mesenteric veins.
- The lymph nodes that come from the pancreas are plentiful and follow the major arteries and veins

  Lymphatic supply to the pancreas. The lymphatic drainage from the pancreas is diffuse and widespread, which explains the high incidence of lymph node metastases and local recurrence of pancreatic cancer. The pancreatic lymphatics also communicate with lymph nodes in the transverse mesocolon and mesentery of the proximal jejunum. Tumors in the body and tail of the pancreas are often unresectable because they metastasize to these lymph nodes and to the hilum of the spleen.



- The pancreas has both exocrine (having ducts) and endocrine (without ducts) histology (tissue as seen under the microscope)

1. The exocrine portion is analogous to a cluster of grapes with the grapes representing the secreting unit that produces the pancreatic digestive enzymes. The grape stems would be the pancreatic ducts coming together to form a single large duct (duct of Wirsung) that empties into the duodenum at a small raised area, the ampulla of Vater
2. The endocrine portion, called the islets of Langerhans, are scattered throughout the pancreas. The various cells that make up the islets of Langerhans produce numerous hormones

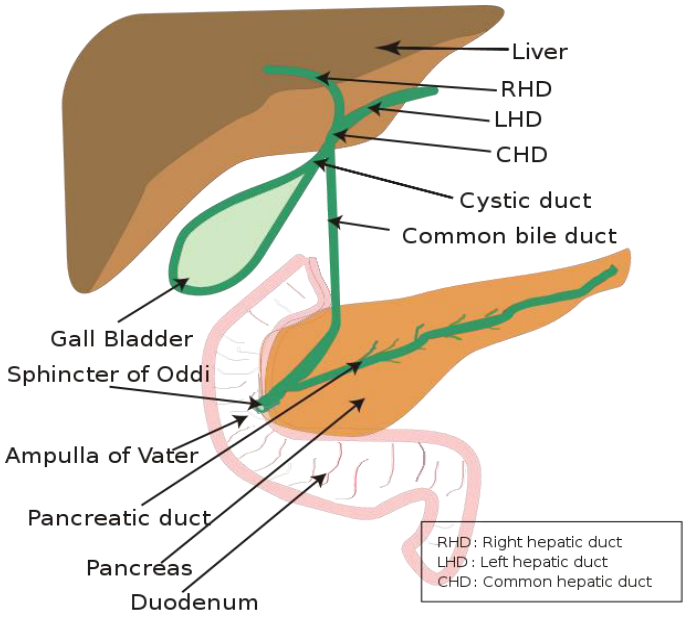
The ampulla of Vater, also known as the hepatopancreatic ampulla, is formed by the union of the pancreatic duct and the common bile duct. The ampulla is specifically located at the major duodenal papilla.

The ampulla of Vater is an important landmark, halfway along the second part of the duodenum, that marks the anatomical transition fromforegut to midgut (and hence the point where the celiac trunk stops supplying the gut and the superior mesenteric artery takes over).

Function

Various smooth muscle sphincters regulate the flow of bile and pancreatic juice through the ampulla: the sphincter of the pancreatic duct, thesphincter of the bile duct, and the hepatopancreatic sphincter (Sphincter of Oddi).

The sphincter of Oddi controls the introduction of bile and pancreatic secretions into the duodenum, as well as preventing the entry of duodenal contents into the ampulla.

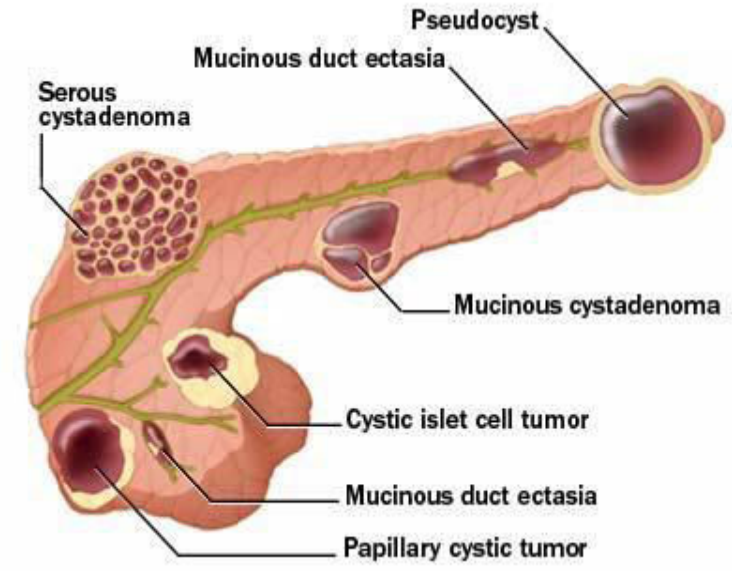


Pancreatic cysts are abnormal, sac-like pockets of fluid on or within your pancreas. Though it may be alarming to learn you have a pancreatic cyst, the good news is that most pancreatic cysts aren't cancerous — and many don't even cause symptoms.

In fact, many pancreatic cysts aren't technically cysts at all. Called pseudocysts, these noncancerous (benign) pockets of fluids aren't lined with the type of cells found in true cysts but, instead, with inflammatory or scar tissue.

But because some pancreatic cysts can be cancerous, your doctor may want to take a sample of the pancreatic cyst fluid to determine whether cancer cells are present. If your cyst is or can become cancerous, treatment involves surgical removal.

Cysts of pancreas:



Pathologic Classification of Cystic Neoplasms of the Pancreas: The WHO International Classification of Tumors, 2000
Serous cystic neoplasm (SCN)
Microcystic adenoma
Oligocystic adenoma
Mucinous cystic neoplasm (MCN)
Mucinous cystadenoma
Mucinous cystic tumor–borderline
Mucinous cystadenocarcinoma
Noninvasive (carcinoma-in-situ)
Invasive
Intraductal papillary mucinous neoplasm (IPMN)
Adenoma
Borderline
Carcinoma-in-situ
Invasive carcinoma

You may have pancreatic cysts, including pseudocysts, but experience no symptoms. Pancreatic cysts are often discovered when imaging tests of the abdomen are done for another reason. When signs or symptoms do occur, they typically include:

- Persistent abdominal pain, which may radiate to your back
- A mass you can feel in your upper abdomen, where your pancreas is located
- Nausea and vomiting

Cysts can become infected patient will be feverish and have persistent abdominal pain.

A ruptured pseudocyst is a medical emergency. When a cyst ruptures, the released fluid can damage nearby blood vessels and cause massive bleeding. It may also cause infection of the abdominal cavity (peritonitis). Seek emergency medical treatment if one have signs or symptoms of internal bleeding and shock, including:

- Fainting
- Severe abdominal pain
- Decreased consciousness
- Weak and rapid heartbeat
- Vomiting blood

Causes

In many cases, the cause of a particular pancreatic cyst is unknown. Genetic mutations can lead to cancerous cysts. Some cysts are associated with rare illnesses — such as von Hippel-

Lindau disease, which is a genetic disorder that can affect the brain, retina, adrenal glands, kidneys and pancreas.

Pseudocysts often follow a bout of pancreatitis, a painful condition in which the pancreas's digestive enzymes become prematurely active and digest some of the pancreas itself. Pseudocysts can also result from blunt trauma to the abdomen, such as during a car accident.

Risk factors

Heavy alcohol use and gallstones are risk factors for pancreatitis, and pancreatitis is a risk factor for pseudocysts — the most common type of pancreatic cyst.

Tests and diagnosis

Pancreatic cysts are diagnosed more often now than in the past, but not because more people have them. Instead, improved imaging technology has made pancreatic cysts easier to find. In fact, many pancreatic cysts are found during abdominal scans for other problems.

The biggest challenge of the diagnostic process is determining whether the pancreatic cyst is cancerous or not. The following tests are often used to help with diagnosis and to help plan treatment:

- Medical history. A previous history of pancreatitis or abdominal injury typically is an indication of a pseudocyst.
- CT scan. A CT scan of your pancreas combines a series of X-rays from different angles to give your doctor detailed information about the structure of a pancreatic cyst. It can sometimes help differentiate cancerous cysts from noncancerous ones.
- MRI scan. An MRI scan, which uses a magnetic field and radio waves to create images, can highlight subtle details of a pancreatic cyst structure, including whether there are any solid components to the cyst.
- Endoscopic ultrasound. Your doctor may also order an endoscopic ultrasound to visualize the cyst and to obtain fluid from the cyst for analysis. In this study, a thin flexible tube (endoscope) is passed through your mouth and into your stomach and upper small intestine. The endoscope is equipped with a small ultrasound probe used to obtain images of the cyst, and with a needle to obtain fluid from the cyst.
- Cyst fluid evaluation. Fluid taken from a cyst is examined in a laboratory for possible signs of cancer. One such sign, or marker, is a substance called carcinoembryonic antigen (CEA). Elevated levels of CEA in the fluid sample are suggestive of cancer.

  In some cases, the characteristics and location of the cyst in your pancreas — along with your age and sex — can help doctors pinpoint what type of cyst you have.

- Serous cystadenoma. This growth can become large enough to displace nearby organs, causing such symptoms as abdominal pain and a feeling of fullness. Serous cystadenomas occur most frequently in middle-aged women and become cancerous only rarely.
- Mucinous cystadenoma. This type of cyst is usually located in the body or tail of the pancreas and occurs most often in middle-aged women. It's precancerous and may already be cancerous when discovered. Larger cysts are more likely to be cancerous.

- Mucinous duct ectasia. More common in men, this type of cyst consists of dilated ductal segments, usually within the head of the pancreas. Also known as an intraductal papillary mucinous tumor, this type of growth is usually precancerous or cancerous.
- Papillary cystic tumor. A papillary cystic tumor — also known as a papillary cystic neoplasm, or solid and pseudopapillary neoplasm — occurs most often in young women and is usually located in the body or tail of the pancreas. It's usually cancerous.
- Cystic islet cell tumor. This type of pancreatic tumor is mostly solid but can have cyst-like components. It may be confused with other pancreatic cysts.

Treatments and drugs

Treatment differs depending on the type of cyst and whether it causes symptoms. Sometimes, no treatment is necessary.

Watchful waiting

A benign pseudocyst — even a large one — can be left alone.Because a serous cystadenoma so rarely evolves into cancer, it also can be left alone unless it causes symptoms or enlarges. Your doctor may want to follow its size over time by checking repeat scans, especially if a precancerous cyst can't be ruled out.

Drainage

A pseudocyst that is causing bothersome symptoms or growing larger may be drained with a needle, often with the use of an endoscope, a small flexible tube that's passed through your mouth to your stomach and small intestine. The endoscope can be equipped with a needle to drain the cyst.

Surgery

An enlarged pseudocyst or serous cystadenoma that's causing pain or other symptoms can be surgically removed. However, a pseudocyst that is removed may recur if you have ongoing pancreatitis.

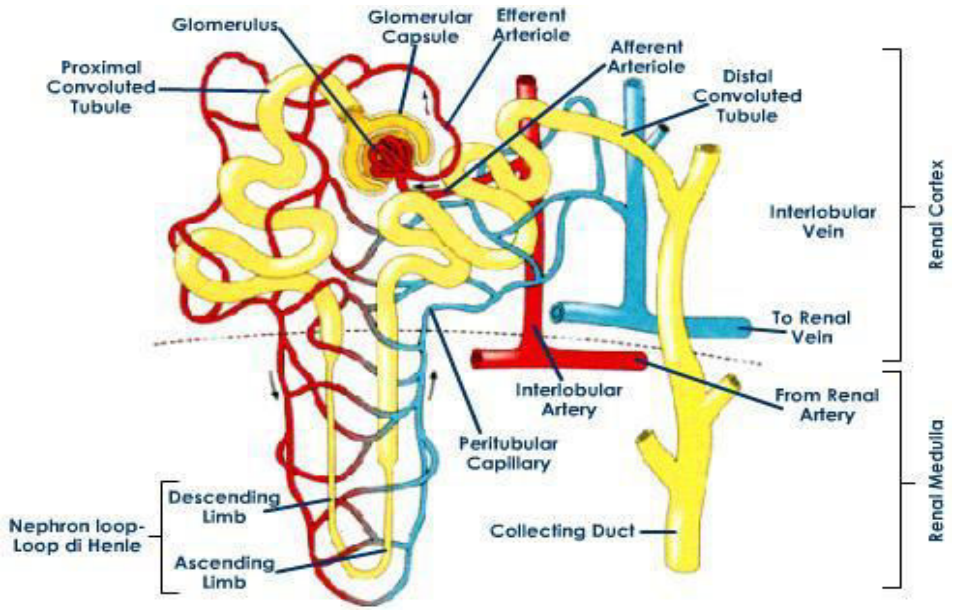
Treatment for most other types of lesions in the pancreas is generally surgical removal, because of the risk of cancer. Surgery is very effective, with little chance of recurrence.

Prevention

The best way to avoid pseudocysts is to avoid pancreatitis, which is usually caused by gallstones or heavy use of alcohol. If pancreatitis is due to alcohol abuse, abstaining from alcohol can reduce the risk. If gallstones are triggering pancreatitis, one may need to have his/her gallbladder removed.

2. Describe the structure of a 'nephron'. Discuss the role of the kidney in the maintainance of acid – base balance of the body. (25)

Answer.



Each nephron is composed of an initial filtering component (the "renal corpuscle") and a tubule specialized for reabsorption and secretion (the "renal tubule"). The renal corpuscle filters out large solutes from the blood, delivering water and small solutes to the renal tubule for modification.

Renal corpuscle

Composed of a glomerulus and the Bowman's capsule, the renal corpuscle (or Malpighian corpuscle) is the beginning of the nephron. It is the nephron's initial filtering component.

The glomerulus is a capillary tuft that receives its blood supply from an afferent arteriole of the renal circulation. The glomerular blood pressure provides the driving force for water and solutes to be filtered out of the blood and into the space made by Bowman's capsule. The remainder of the blood (only approximately 1/5 of all plasma passing through the kidney is filtered through the glomerular wall into the Bowman's capsule) passes into the narrower efferent arteriole. It then moves into the vasa recta, which are only found in juxtamedullary nephrons and not cortical nephrons. The vasa recta are collecting capillaries intertwined with the convoluted tubules through the interstitial space, in which the reabsorbed substances will also enter. This then combines with efferent venules from other nephrons into the renal vein, and rejoins the main bloodstream.

The Bowman's capsule, also called the glomerular capsule, surrounds the glomerulus. It is composed of a visceral inner layer formed by specialized cells called podocytes, and a parietal outer layer composed of a single layer of flat cells called simple squamous epithelium. Fluids from blood in the glomerulus are filtered through the visceral layer of podocytes, and the resulting glomerular filtrate is further processed along the nephron to form urine.

Renal tubule

The renal tubule is the portion of the nephron containing the tubular fluid filtered through the glomerulus. After passing through the renal tubule, the filtrate continues to the collecting duct system, which is not part of the nephron.

The components of the renal tubule are:

i.Proximal convoluted tubule

ii.Loop of Henle

- Descending limb of loop of Henle
- Ascending limb of loop of Henle

-Thin ascending limb of loop of Henle

- Thick ascending limb of loop of Henle

iii.Distal convoluted tubule.

Role of the Kidneys

The organs involved in regulation of external acid-base balance are the lungs are the kidneys.
The lungs are important for excretion of carbon dioxide (the respiratory acid) and there is a huge amount of this to be excreted: at least 12,000 to 13,000 mmols/day.
In contrast the kidneys are responsible for excretion of the fixed acids and this is also a critical role even though the amounts involved (70-100 mmols/day) are much smaller.
The main reason for this renal importance is because there is no other way to excrete these acids and it should be appreciated that the amounts involved are still very large when compared to the plasma [H^+] of only 40 nanomoles/litre.
There is a second extremely important role that the kidneys play in acid-base balance, namely the reabsorption of the filtered bicarbonate. Bicarbonate is the predominant extracellular buffer against the fixed acids and it important that its plasma concentration should be defended against renal loss.
In acid-base balance, the kidney is responsible for 2 major activities:

- Reabsorption of filtered bicarbonate: 4,000 to 5,000 mmol/day
- Excretion of the fixed acids (acid anion and associated H^+): about 1 mmol/kg/day.

Both these processes involve secretion of H^+ into the lumen by the renal tubule cells but only the second leads to excretion of H^+ from the body.
The renal mechanisms involved in acid-base balance can be difficult to understand so as a simplification we will consider the processes occurring in the kidney as involving 2 aspects:

- Proximal tubular mechanism
- Distal tubular mechanism

Proximal Tubular Mechanism

The contributions of the proximal tubules to acid-base balance are:

- firstly, reabsorption of bicarbonate which is filtered at the glomerulus
- secondly, the production of ammonium

The next 2 sections explain these roles in more detail.

Bicarbonate Reabsorption

Daily filtered bicarbonate equals the product of the daily glomerular filtration rate (180 l/day) and the plasma bicarbonate concentration (24 mmol/l). This is 180 x 24 = 4320 mmols/day (or usually quoted as between 4000 to 5000 mmols/day).
About 85 to 90% of the filtered bicarbonate is reabsorbed in the proximal tubule and the rest is reabsorbed by theintercalated cells of the distal tubule and collecting ducts.
The reactions that occur are outlined in the diagram. Effectively, H^+ and $HCO3^-$ are formed from CO_2 and H_2O in a reaction catalysed by carbonic anhydrase. The actual reaction involved is probably formation of H^+ and OH^- from water, then reaction of OH^- with CO_2 (catalysed by carbonic anhydrase) to produce $HCO3^-$. Either way, the end result is the same.
The H^+ leaves the proximal tubule cell and enters the PCT lumen by 2 mechanisms:

- Via a Na^+-H^+ antiporter (major route)
- Via H^+-ATPase (proton pump)

Filtered $HCO3^-$ cannot cross the apical membrane of the PCT cell. Instead it combines with the secreted H^+ (under the influence of brush border carbonic anhydrase) to produce CO_2 and H_2O. The CO_2 is lipid soluble and easily crosses into the cytoplasm of the PCT cell. In the cell, it combines with OH^- to produce bicarbonate. The HCO_3^-crosses the basolateral membrane via a Na^+-HCO_3^- symporter. This symporter is electrogenic as it transfers three HCO_3^- for every one Na^+. In comparison, the Na^+-H^+ antiporter in the apical membrane is not electrogenic because an equal amount of charge is transferred in both directions.
The basolateral membrane also has an active Na^+-K^+ ATPase (sodium pump) which transports 3 Na^+ out per 2 K^+ in. This pump is electrogenic in a direction opposite to that of the Na^+-HCO_3^- symporter. Also the sodium pump keeps intracellular Na^+ low which sets up the Na^+ concentration gradient required for the H^+-Na^+ antiport at the apical membrane. The H^+-Na^+ antiport is an example of secondary active transport.
The net effect is the reabsorption of one molecule of HCO_3 and one molecule of Na^+ from the tubular lumen into the blood stream for each molecule of H^+ secreted. This mechanism does not lead to the net excretion of any H^+ from the body as the H^+ is consumed in the reaction with the filtered bicarbonate in the tubular lumen.
[Note: The differences in functional properties of the apical membrane from that of the basolateral membranes should be noted. This difference is maintained by the tight junctions which link adjacent proximal tubule cells. These tight junctions have two extremely important functions:
Gate function: They limit access of luminal solutes to the intercellular space. This resistance can be altered and this paracellular pathway can be more open under some circumstances (ie the 'gate' can be opened a little).
Fence function: The junctions maintain different distributions of some of the integral membrane proteins. For example they act as a 'fence' to keep the Na^+-H^+ antiporter limited to the apical membrane, and keep the Na^+-K^+ ATPase limited to the basolateral membrane. The different distribution of such proteins is absolutely essential for cell function.]
The 4 major factors which control bicarbonate reabsorption are:

- Luminal HCO_3^- concentration

- Luminal flow rate
- Arterial pCO_2
- Angiotensin II (via decrease in cyclic AMP)

An increase in any of these four factors causes an increase in bicarbonate reabsorption. Parathyroid hormone also has an effect: an increase in hormone level increases cAMP and decreases bicarbonate reabsorption.

Outline of Reactions in Proximal Tubule Lumen & Cells

The mechanism for H^+ secretion in the proximal tubule is described as a high capacity, low gradient system:
The high capacity refers to the large amount (4000 to 5000 mmols) of H^+ that is secreted per day. (The actual amount of H^+ secretion is 85% of the filtered load of HCO_3^-).
The low gradient refers to the low pH gradient as tubular pH can be decreased from 7.4 down to 6.7-7.0 only.
Though no net excretion of H^+ from the body occurs, this proximal mechanism is extremely important in acid-base balance. Loss of bicarbonate is equivalent to an acidifying effect and the potential amounts of bicarbonate lost if this mechanism fails are very large.

Ammonium Production

Ammonium (NH_4) is produced predominantly within the proximal tubular cells. The major source is from glutamine which enters the cell from the peritubular capillaries (80%) and the filtrate (20%). Ammonium is produced from glutamine by the action of the enzyme glutaminase. Further ammonium is produced when the glutamate is metabolised to produce alpha-ketoglutarate. This molecule contains 2 negatively-charged carboxylate groups so further metabolism of it in the cell results in the production of 2 HCO_3^- anions. This occurs if it is oxidised to CO_2 or if it is metabolised to glucose.
The pKa for ammonium is so high (about 9.2) that both at extracellular and at intracellular pH, it is present entirely in the acid form NH_4^+. The previous idea that lipid soluble NH_3 is produced in the tubular cell, diffuses into the tubular fluid where it is converted to water soluble NH_4^+ which is now trapped in the tubule fluid is incorrect.
The subsequent situation with ammonium is complex. Most of the ammonium is involved in cycling within the medulla. About 75% of the proximally produced ammonium is removed from the tubular fluid in the medulla so that the amount of ammonium entering the distal tubule is small. The thick ascending limb of the loop of Henle is the important segment for removing ammonium. Some of the interstitial ammonium returns to the late proximal tubule and enters the medulla again (ie recycling occurs).
An overview of the situation so far is that:

- The ammonium level in the DCT fluid is low because of removal in the loop of Henle
- Ammonium levels in the medullary interstitium are high (and are kept high by the recycling process via the thick ascending limb and the late PCT)
- Tubule fluid entering the medullary collecting duct will have a low pH if there is an acid load to be excreted (and the phosphate buffer has been titrated down.

If H^+ secretion continues into the medullary collecting duct this would reduce the pH of the luminal fluid further. A low pH greatly augments transfer of ammonium from the medullary interstitium into the luminal fluid as it passes through the medulla. The lower the urine pH, the higher the ammonium excretion and this ammonium excretion is augmented further if an acidosis is present. This augmentation with acidosis is 'regulatory' as the increased ammonium excretion by the kidney tends to increase extracellular pH towards normal.
If the ammonium returns to the blood stream it is metabolised in the liver to urea (Krebs-Henseleit cycle) with net production of one hydrogen ion per ammonium molecule.

Distal Tubular Mechanism

This is a low capacity, high gradient system which accounts for the excretion of the daily fixed acid load of 70 mmols/day. The maximal capacity of this system is as much as 700 mmols/day but this is still low compared to the capacity of the proximal tubular mechanism to secrete H^+. It can however decrease the pH down to a limiting pH of about 4.5 : this represents a thousand-fold (ie 3 pH units) gradient for H^+ across the distal tubular cell. The maximal capacity of 700 mmols/day takes about 5 days to reach.
The processes involved are:-

- Formation of titratable acidity (TA)
- Addition of ammonium ($NH4^+$) to luminal fluid
- Reabsorption of Remaining Bicarbonate

1. Titratable Acidity

H^+ is produced from CO2 and H2O (as in the proximal tubular cells) and actively transported into the distal tubular lumen via a H^+-ATPase pump. Titratable acidity represents the H^+ which is buffered mostly by phosphate which is present in significant concentration. Creatinine (pKa approx 5.0) may also contribute to TA. At the minimum urinary pH, it will account for some of the titratable acidity. If ketoacids are present, they also contribute to titratable acidity. In severe diabetic ketoacidosis, beta-hydroxybutyrate (pKa 4.8) is the major component of TA.
The TA can be measured in the urine from the amount of sodium hydroxide needed to titrate the urine pH back to 7.4 hence the term ‘titratable acidity’.

2. Addition of Ammonium

Ammonium is predominantly produced by proximal tubular cells. This is advantageous as the proximal cells have access to a high blood flow in the peritubular capillaries and to all of the filtrate and these are the two sources of the glutamine from which the ammonium is produced.
The medullary cycling maintains high medullary interstitial concentrations of ammonium and low concentrations of ammonium in the distal tubule fluid. The lower the urine pH, the more the amount of ammonium that is transferred from the medullary interstitium into the fluid in the lumen of the medullary collecting duct as it passes through the medulla to the renal pelvis. [Note: The medullary collecting duct is different from the distal convoluted tubule.]
The net effect of this is that the majority of the ammonium in the final urine was transferred from the medulla across the distal part of the tubule even though it was produced in the proximal tubule. [Simplistically but erroneously it is sometimes said that the ammonium in the urine is produced in the distal tubule cells.]

Ammonium is not measured as part of the titratable acidity because the high pK of ammonium means no H^+ is removed from $NH4^+$ during titration to a pH of 7.4. Ammonium excretion in severe acidosis can reach 300 mmol/day in humans.
Ammonium excretion is extremely important in increasing acid excretion in systemic acidosis. The titratable acidity is mostly due to phosphate buffering and the amount of phosphate present is limited by the amount filtered (and thus the plasma concentration of phosphate). This cannot increase significantly in the presence of acidosis (though of course some additional phosphate could be released from bone) unless other anions with a suitable pKa are present. Ketoanions can contribute to a significant increase in titratable acidity but only in ketoacidosis when large amounts are present.
In comparison, the amount of ammonium excretion can and does increase markedly in acidosis. The ammonium excretion increases as urine pH falls and also this effect is markedly augmented in acidosis. Formation of ammonium prevents further fall in pH as the pKa of the reaction is so high.

In review

- Titratable acidity is an important part of excretion of fixed acids under normal circumstances but the amount of phosphate available cannot increase very much.
- Also as urine pH falls, the phosphate will be all in the dihyrogen form and buffering by phosphate will be at its maximum.
- A further fall in urine pH cannot increase titratable acidity (unless there are other anions such as keto-anions present in significant quantities)
- The above points mean that titratable acidity cannot increase very much (so cannot be important in acid-base regulation when the ability to increase or decrease renal H^+excretion is required)
- In acidosis, ammonium excretion fills the regulatory role because its excretion can increase very markedly as urine pH falls.

A low urine pH itself cannot directly account for excretion of a significant amount of acid: for example, at the limiting urine pH of about 4.4, $[H^+]$ is a negligible 0.04 mmol/l. This is several orders of magnitude lower than H^+ accounted for by titratable acidity and ammonium excretion. (ie 0.04 mmol/l is insignificant in a net renal acid excretion of 70 mmols or more per day)

3. Reabsorption of Remaining Bicarbonate

On a typical Western diet all of the filtered load of bicarbonare is reabsorbed. The sites and percentages of filtered bicarbonate involved are:

- Proximal tubule 85%
- Thick ascending limb of Loop of Henle 10-15%
- Distal tubule 0-5%

The decrease in volume of the filtrate as further water is removed in the Loop of Henle causes an increase in [HCO3-] in the remaining fluid. The process of HCO3- reabsorption in the thick ascending limb of the Loop of Henle is very similar to that in the proximal tubule (ie apical Na^+-H^+ antiport and basolateral Na^+-HCO3- symport and Na^+-K^+ATPase).

Bicarbonate reabsorption here is stimulated by the presence of luminal frusemide. The cells in this part of the tubule contain carbonic anhydrase.
Any small amount of bicarbonate which enters the distal tubule can also be reabsorbed. The distal tubule has only a limited capacity to reabsorb bicacarbonate so if the filtered load is high and a large amount is delivered distally then there will be net bicarbonate excretion.
The process of bicarbonate reabsorption in the distal tubule is somewhat different from in the proximal tubule:

- H^+ secretion by the intercalated cells in DCT involves a H^+-ATPase (rather than a Na^+-H^+ antiport)
- HCO3- transfer across the basolateral membrane involves a $HCO3^-$-Cl^- exchanger (rather than a Na^+-HCO3- symport)

The net effect of the excretion of one H^+ is the return of one HCO3- and one Na^+ to the blood stream. The HCO3- effectively replaces the acid anion which is excreted in the urine.
The net acid excretion in the urine is equal to the sum of the TA and [$NH4^+$] minus [HCO3] (if present in the urine). The [H^+] accounts for only a very small amount of the H^+ excretion and is not usually considered in the equation (as mentioned earlier).
In metabolic alkalosis, the increased bicarbonate level will result in increased filtration of bicarbonate provided the GFR has not decreased. The kidney is normally extremely efficient at excreting excess bicarbonate but this capacity can be impaired in certain circumstances.

2.4.6 Regulation of Renal H^+ Excretion

The discussion above has described the mechanisms involved in renal acid excretion and mentioned some factors which regulate acid excretion.
The major factors which regulate renal bicarbonate reabsorption and acid excretion are:

1. Extracellular volume

Volume depletion is associated with Na^+ retention and this also enhances HCO3 reabsorption. Conversely, ECF volume expansion results in renal Na^+ excretion and secondary decrease in HCO3 reabsorption.

2. Arterial pCO2

An increase in arterial pCO2 results in increased renal H^+ secretion and increased bicarbonate reabsorption. The converse also applies. Hypercapnia results in an intracellular acidosis and this results in enhanced H^+ secretion. The cellular processes involved have not been clearly delineated. This renal bicarbonate retention is the renal compensation for a chronic respiratory acidosis.

3. Potassium & Chloride Deficiency

Potassium has a role in bicarbonate reabsorption. Low intracellular K^+ levels result in increased HCO_3 reabsorption in the kidney. Chloride deficiency is extremely important in the maintenance of a metabolic alkalosis because it prevents excretion of the excess HCO3 (ie now the bicarbonate instead of chloride is reabsorbed with Na^+ to maintain electroneutrality).

4. Aldosterone & cortisol (hydrocortisone)

Aldosterone at normal levels has no role in renal regulation of acid-base balance. Aldosterone delpetion or excess does have indirect effects. High aldosterone levels result in increased Na^+ reabsorption and increased urinary excretion of H^+ and K^+ resulting in a

metabolic alkalosis. Conversely, it might be thought that hypoaldosteronism would be associated with a metabolic acidosis but this is very uncommon but may occur if there is coexistent significant interstitial renal disease.

5. Phosphate Excretion

Phosphate is the major component of titratable acidity. The amount of phosphate present in the distal tubule does not vary greatly. Consequently, changes in phosphate excretion do not have a significant regulatory role in response to an acid load.

6. Reduction in GFR

It has recently been established that a reduction in GFR is a very important mechanism responsible for the maintenance of a metabolic alkalosis. The filtered load of bicarbonate is reduced proportionately with a reduction in GFR.

7. Ammonium

The kidney responds to an acid load by increasing tubular production and urinary excretion of $NH4^+$. The mechanism involves an acidosis-stimulated enhancement of glutamine utilisation by the kidney resulting in increased production of $NH4^+$ and $HCO3^-$ by the tubule cells. This is very important in increasing renal acid excretion during a chronic metabolic acidosis. There is a lag period: the increase in ammonium excretion takes several days to reach its maximum following an acute acid load. Ammonium excretion can increase up to about 300 mmol/day in a chronic metabolic acidosis so this is important in renal acid-base regulation in this situation. Ammonium excretion increases with decreases in urine pH and this relationship is markedly enhanced with acidosis.

2.4.7 What is the Role of Urinary Ammonium Excretion?

There are different views on the true role of NH_4^+ excretion in urine. How can the renal excretion of ammonium which has a pK of 9.2 represent H^+ excretion from the body?

One school says the production of ammonium from glutamine in the tubule cells results in production of alpha-ketoglutarate which is then metabolised in the tubule cell to 'new' bicarbonate which is returned to the blood. The net effect is the return of one bicarbonate for each ammonium excreted in the urine. By this analysis, the excretion of ammonium is equivalent to the excretion of acid from the body as one plasma H^+ would be neutralised by one renal bicarbonate ion for each ammonium excreted. Thus an increase in ammonium excretion as occurs in metabolic acidosis is an appropriate response to excrete more acid.

The other school says this is not correct. The argument is that metabolism of alpha-ketogluarate in the proximal tubule cells to produce this 'new' HCO3- merely represents regeneration of the HCO3 that was neutralised by the H^+produced when alpha-ketoglutarate was metabolised to glutamate in the liver originally so there can be no direct effect on net H^+ excretion. The key to understanding is said to lie in considering the role of the liver. Consider the following:

Every day protein turnover results in amino acid degradation which results in production of HCO3- and $NH4^+$. For a typical 100g/day protein diet, this is a net production of 1,000mmol/day of HCO3- and 1,000mmol/day of $NH4^+$. (These are produced in equal amounts by neutral amino acids as each contains one carboxylic acid group and one amino group.) The high pK of the ammonium means it cannot dissociate to produce one H^+ to neutralise the HCO3- so consequently amino acid metabolism is powerfully alkalinising to the body. The body now has two major problems:

- How to get rid of 1,000mmol/day of alkali?
- How to get rid of 1,000mmol/day of the highly toxic ammonium?

The solution is to react the two together and get rid of both at once. This process is hepatic urea synthesis (Krebs-Henseleit cycle). The cycle consumes significant energy but solves both problems. Indeed, the cycle in effect acts as a ATP-dependent pump that transfers H^+ from the very weak acid $NH4^+$ to HCO3-. The overall reaction in urea synthesis is:

$2\ NH_4^+ + 2\ HCO_3^- \Rightarrow \text{urea} + CO_2 + 3\ H_2O$

The body has two ways in which it can remove $NH4^+$:

- Urea synthesis in the liver
- Excretion of $NH4^+$ by the kidney

The key thing here is that the acid-base implications of these 2 mechanisms are different. For each ammonium converted to urea in the liver one bicarbonate is consumed. For each ammonium excreted in the urine, there is one bicarbonate that is not neutralised by it (during urea synthesis) in the liver. So overall, urinary excretion of ammonium is equivalent to net bicarbonate production -but by the liver! Indeed in a metabolic acidosis, an increase in urinary ammonium excretion results in an exactly equivalent net amount of hepatic bicarbonate (produced from amino acid degradation) available to the body. So the true role of renal ammonium excretion is to serve as an alternative route for nitrogen elinination that has a different acid-base effect from urea production.
The role of glutamine is to act as the non-toxic transport molecule to carry $NH4^+$ to the kidney. The bicarbonates consumed in the production of glutamine and then released again with renal metabolism of ketoglutarate are not important as there is no net gain of bicarbonate.
Overall: renal $NH4^+$ excretion results indirectly in an equivalent amount of net hepatic HCO_3 production.
Other points are:

- Glutamate metabolism in the proximal tubule converts ADP to ATP and the low availability of ADP limits the maximal rate of NH_4^+ production in the proximal tubule cells. Further as most ATP is consumed in the reabsorption of Na^+, then it is ultimately the amount of Na^+ reabsorbed in the proximal tubule that sets the upper limit for NH_4^+ production.
- The anion that is excreted with the NH_4^+ is also important. Excretion of beta-hydroxybutyrate (instead of chloride) with NH_4^+ in ketoacidosis leads to a loss of bicarbonate as this anion represents a potential bicarbonate.

Finally: The role of urine pH in situations of increased acid secretion is worth noting. The urine pH can fall to a minimum value of 4.4 to 4.6 but as mentioned previously this itself represents only a negligible amount of free H^+.
As pH falls, the 3 factors involved in increased H^+ excretion are:
1. Increased ammonium excretion (increases steadily with decrease in urine pH and this effect is augmented in acidosis) [This is the major and regulatory factor because it can be increased significantly].
2. Increased titratable acidity:

- Increased buffering by phosphate (but negligible further effect on H^+ excretion if pH < 5.5 as too far from pKa so minimal amounts of $HPO4^{-2}$ remaining)

- Increased buffering by other organic acids (if present) may be important at lower pH values as their pKa is lower (eg creatinine, ketoanions)

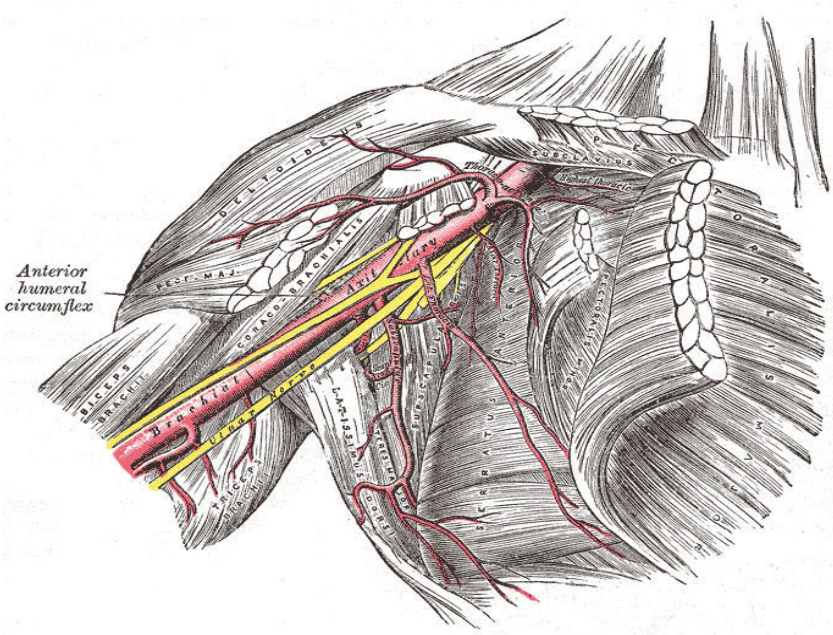
3. Bicarbonate reabsorption is complete at low urinary pH so none is lost in the urine (Such loss would antagonise the effects of an increased TA or ammonium excretion on acid excretion.)

3. Describe the surgical anatomy of axilla. Discuss about the diseases that implicate it. (25)

Answer. The axilla (or armpit, underarm, or oxter) is the area on the human body directly under the joint where the arm connects to the shoulder. It contains the axillary vessels, and the brachial plexus of nerves, with their branches, some branches of the intercostal nerves, and a large number of lymph glands, together with a quantity of fat and loose areolar tissue. The axillary artery and vein, with the brachial plexus of nerves, extend obliquely along the lateral boundary of the axilla, from its apex to its base, and are placed much nearer to the anterior than to the posterior wall, the vein lying to the thoracic side of the artery and partially concealing it. At the forepart of the axilla, in contact with the Pectorales, are the thoracic branches of the axillary artery, and along the lower margin of the Pectoralis minor the lateral thoracic artery extends to the side of the chest. At the back part, in contact with the lower margin of the Subscapularis, are the subscapular vessels and nerves; winding around the lateral border of this muscle are the scapular circumflex vessels; and, close to the neck of the humerus, the posterior humeral circumflex vessels and the axillary nerve curve backward to the shoulder. Along the medial or thoracic side no vessel of any importance exists, the upper part of the space being crossed merely by a few small branches from the highest thoracic artery. There are some important nerves, however, in this situation, viz., the long thoracic nerve, descending on the surface of the Serratus anterior, to which it is distributed; and the intercostobrachial nerve, perforating the upper and anterior part of this wall, and passing across the axilla to the medial side of the arm.

Boundaries

	superiorly: by the outer border of first rib, superior border of scapula, and posterior border of clavicle	
medially: serratus anterior and by the ribcage	anteriorly: by the pectoralis major, minor,and subclavius posteriorly: by the subscapularis above, and teres major and latissimus dorsi below	laterally: by the intertubercular sulcus (coracobrachialis and the short head of the biceps brachii are in the axilla.)
	floor/base: by the skin(visible surface of armpit)	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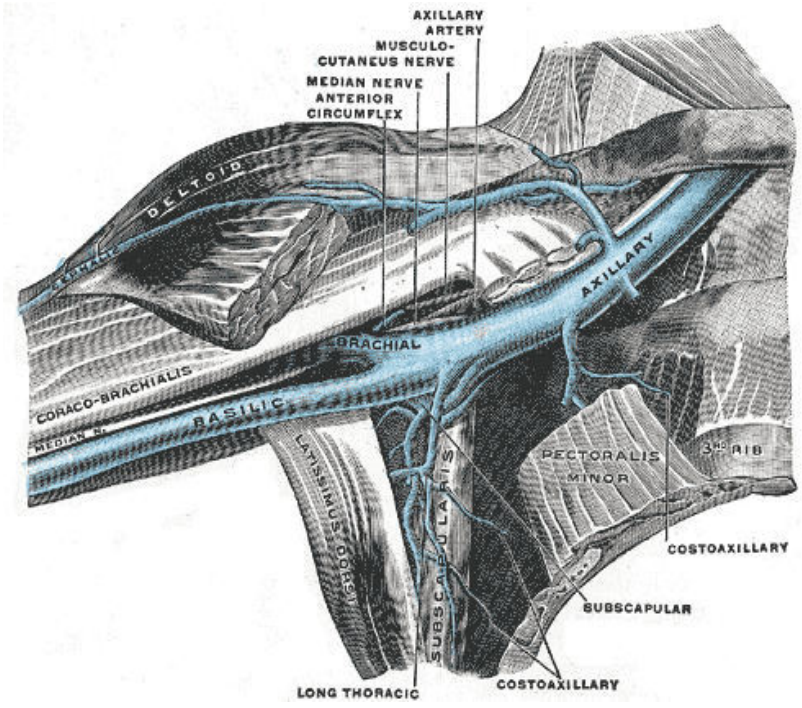
circumflex
DELTOID
PECT. MAJ.
CORACO-BRACHIALIS
BICEPS
BRACHII
Brachial
Ulnar Nerve
TRICEPS
BRACHII
SUBSCAPULARIS
SERRATUS ANTERIOR


AXILLARY
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CIRCUMFLEX
DELTOID
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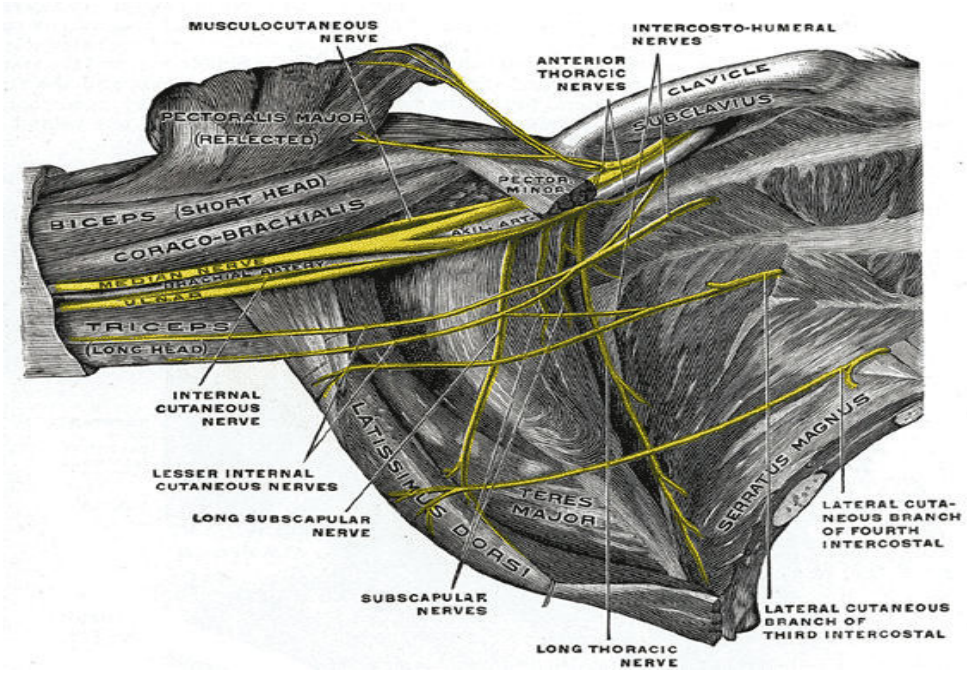
CORACO-BRACHIALIS
BASILIC
MEDIAN N.
PECTORALIS
MINOR
3RD RIB
COSTOAXILLARY
SUBSCAPULARIS
SUBSCAPULAR
LONG THORACIC
COSTOAXILLARY



Diseases implicating axilla:

Lumps in the axilla have various causes. Cysts and infections on the surface of the skin of the armpit may be caused by shaving or use of antiperspirants (as opposed to deodorants). This occurs most frequently in adolescents just beginning to shave. Abscesses under the skin may also produce large, painful lumps in the armpit.

Lumps may occur when lymph nodes are swollen due to bacterial or viral infections, vaccinations, and cancer.

Lymph nodes are filters that can catch infectious organisms or cancerous tumor cells. When they do, lymph nodes increase in size and are easily felt. An armpit lump in a woman should be checked by a health care provider immediately, as it may be a sign of breast cancer.

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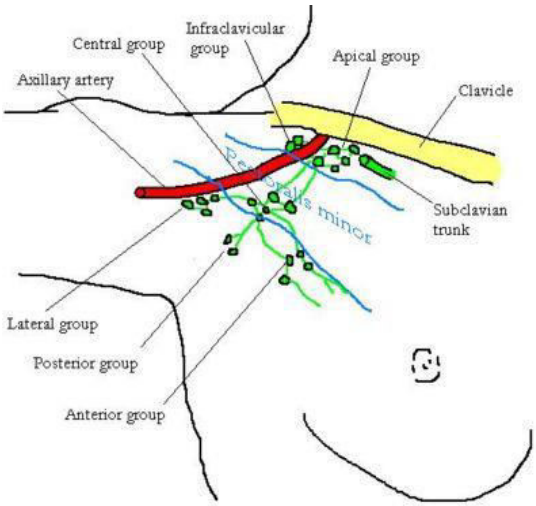
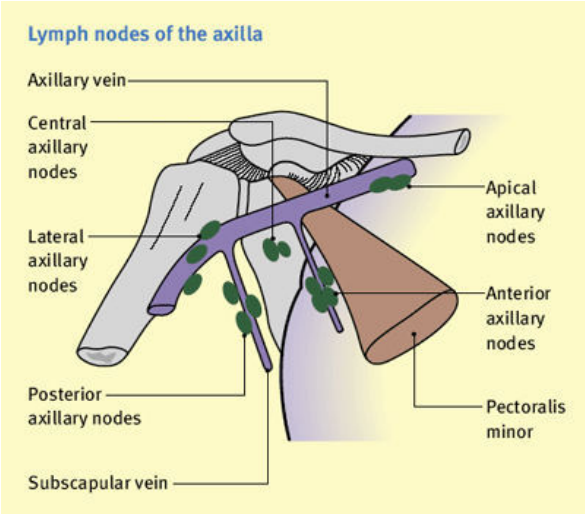




Causes

Infection		Cancer	Other
Bacterial: • Arm or breast infection • Cat scratch disease • Ascending lymphangitis • Lymphadenitis Fun gal: • Sporotrichosis	Viral: • Measles, mumps, rubella vaccine (rare) • Smallpox vaccination • Typhoid vaccine • AIDS • Chickenpox • Infectious mononucleosis • Shingles (herpes zoster)	• Hodgkin's lymphoma • Leukemia • Non-Hodgkin's lymphoma	• Benign (harmless) cyst • Lipomas (harmless fatty growths) • Normal breast tissue (breast tissue extends into the armpit area) • Allergic reaction possibly caused by sulfa drugs, iodine, or penicillin • Vaccinations

4. Give a brief account of rational use of antibiotics in surgical practice. Comment about prevention of abuse of antibiotics. (25)

Answer. Rational Use of Antibiotics:

Antibiotics are the most important weapons in our hands. Each one of them have been invented after spending considerable amount of time, energy and money. Therefore, we cannot afford to lose them. We must exercise considerable restraint in prescribing antibacterials and restrict the use of antibacterials to only certain definite indications.

Indications for antibacterial therapy:

1. Definitive therapy: This is for proven bacterial infections. Antibiotics (read antibacterials) are drugs to tackle bacteria and hence should be restricted for the treatment of bacterial infections only. This may sound silly, but most doctors seem to forget this simple fact! Attempts should be made to confirm the bacterial infection by means of staining of secretions/fluids/exudates, culture and sensitivity, serological tests and other tests. Based on the reports, anarrow spectrum, least toxic, easy-to-administer and cheap drug should be prescribed.
2. Empirical therapy: Empirical antibacterial therapy should be restricted to critical cases, when time is inadequate for identification and isolation of the bacteria and reasonably strong doubt of bacterial infection exists: septicemic shock/ sepsis syndrome, immunocompromised patients with severe systemic infection, hectic temperature, neutrophilic leukocytosis, raised ESR etc. In such situations, drugs that cover the most probable infective agent/s should be used.
3. Prophylactic therapy: Antimicrobial prophylaxis is administered to susceptible patients to prevent specific infections that can cause definite detrimental effect. These include antitubercular prophylaxis, anti rheumatic prophylaxis, anti endocarditis prophylaxis and prophylactic use of antimicrobials in invasive medical procedures etc. In all these situations, only narrow spectrum and specific drugs are used. It should be remembered that there is NO single prophylaxis to 'prevent all' possible bacterial infections.

When choosing an antibiotic for empirical treatment, follow these guidelines:

1. Ensure coverage of the presumed microorganisms involved. This usually means starting broad-spectrum antibiotics, which can then be tailored and narrowed to the specific microorganism isolated. Avoid anaerobic-spectrum antibiotics when possible because this group of bacteria plays an important role in maintaining the gastrointestinal tract microenvironment.

2. Choose an antibiotic that is able to reach the site of the infection. Specifically, for UTI and cholangitis, choose antibiotics with high renal and biliary concentrations, respectively. Consider skin, lung, and central nervous system tissue concentrations for infections at these sites.

3. Consider toxicity, particularly in critically ill patients, in whom bioavailability and the range of therapeutic and toxic levels are harder to predict. Once an antimicrobial with significant toxic side effects is started, closely monitor blood levels and organ function.

4. Whenever an infection that will need antibiotics is identified, aggressively dose the antibiotics. The volume of redistribution of these patients is unpredictable because they have usually undergone aggressive fluid replacements as part of their support or resuscitation.

5. Whenever starting an antibiotic regimen, set a time limit for the period the antibiotic will be given.

The molecular mechanisms by which bacteria acquire resistance to antibiotics can be broadly classified into four categories

1. Decreased intracellular concentration of antibiotic, either by decreased influx or increased efflux. Most antibiotics are susceptible to this mechanism (*Pseudomonas*/Enterobacteriaceae to β-lactams).
2. Neutralization by inactivating enzymes. This is the most common mechanism of antibiotic resistance and affects all β-lactam antibiotics (e.g. β-lactamases from gram-positive and gram-negative bacteria).
3. Alteration of the target at which the antibiotic will act. This category affects all antibiotics and is the main resistance mechanism for some specific bacteria (*Pneumococcus* to penicillin or MRSA to all β-lactam antibiotics).
4. Complete elimination of the target at which the antibiotic will act. Some specific bacteria develop the ability to create new metabolic pathways and completely eliminate a specific target (e.g., VRE).

Prevention of abuse of antibiotics:

Interventions to improve rational use of antibiotics :

- A mandated multi-disciplinary national body to coordinate antibiotic use policies
- Clinical guidelines
- Essential antibiotics list based on treatment choice
- Drug and therapeutics committees in districts and hospitals
- Problem-based learning in pharmacotherapy in undergraduate circulation continuing in-service medical education as a licensure requirement
- Supervision, audit and feedback
- Independent information on public education about antibiotics
- Avoidance of perverse financial incentives
- Appropriate and enforced regulation
- Sufficient government expenditure to ensure availability of antibiotics and staff.

A patient may fail to respond to an antibiotic for a number of reasons which include:

i. The aetiological agent is resistant to the antibiotic
ii. The diagnosis is incorrect
iii. The choice of antibiotic is correct but the dose and/or route of administration is wrong.
iv. The antibiotic cannot reach the site of infection
v. Tthere is a colletion of pus that should be drained surgically or a foreign body/devitalised tissue that should be removed
vi. There is secondary infection
vii. Antibiotic fever
viii. Non-compliance of the host.

- World Health Organisation (WHO) has urged for intensified global commitment to safeguard antibiotics for future generations.

- Antibiotics and other antimicrobial medicines helps to treat conditions that would otherwise be fatal however, antimicrobial resistance which is drug resistance renders these medicines ineffective.
- WHO has urged the governments and stakeholders to implement policies and practices to prevent and counter the emergence of highly resistant microorganisms. Antimicrobial resistance is not a new problem, however it is fast becoming more dangerous.
- Antimicrobial resistance also has enormous social and personal costs. When infections become resistant to first-line antibiotics treatment has to shift to second- and third-line drugs, which are nearly always much more expensive and sometimes more toxic as well.
- The drugs needed to treat multidrug-resistant tuberculosis (MDR-TB) are over 100 times more expensive than the first-line drugs. In some countries the high cost is prohibitive, with the result that some of these cases can no longer be treated. Similarly, the emergence of resistance in HIV to currently effective drugs could destroy the hopes of survival for millions of people living with HIV.
- Discovery, development and distribution of new antibiotics is a long, drawn out and expensive process. After investing millions of dollars and years of research, when a new antibiotic becomes available, its misuse renders it ineffective in a very short time.
- Misuse of antibiotics by prescribers and users have behavioural, educational, ethical and economic dimensions which demand concerted and sustained actions by all sectors of society. Weak pharmaceutical regulatory mechanisms in most developing countries also permit the availability of antibiotics of questionable quality and the unauthorized sale of these antibiotics.

THE WEST BENGAL UNIVER.SITY OF HEALTH SCIENCES

MS (General Surgery) Examination, 2010

PAPER II

Time Allowed: 3 Hours **Full Marks: 100**

Attempt all questions

1. **Discuss the aetiopathology, diagnosis and treatment of soft tissue sarcomas . (4+8+8)**
2. **Discuss the pathology, diagnosis and treatment of cholangiocarcinoma. (4+8+8).**

3. Write short notes on: 5 x 6

- **(a) Primary Lymphoedema**
- **(b) Sjogren Syndrome.**
- **(c) Glucagonoma**
- **(d) Sliding Hernia.**
- **(e) Mesenteric cyst.**

4. Write brief answers: 4 x 7.5

- **(a) Management of aggressive GIST;**
- **(b) Treatment of Venous ulcers;**
- **(c) Short Bowel Syndrome;**
- **(d) Surgical Management of Ulcerative Colitis.**

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES

MS (General Surgery) Examination, 2010

May 2010

PAPER II

Time Allowed: 3 Hours **Full Marks: 100**

Attempt all questions

1. Discuss the aetiopathology, diagnosis and treatment of soft tissue sarcomas . (4+8+8)

Answer.
Aetiopathology:

Predisposing Factors for Sarcomas

Genetic Predisposition

Neurofibromatosis (von Recklinghausen's disease)
Li-Fraumeni syndrome
Retinoblastoma
Gardner's syndrome (familial adenomatous polyposis)

Lymphedema

Postsurgical
Postirradiation status
Parasitic infection (filariasis)

Radiation Exposure

Therapeutic radiation in the orthovoltage and megavoltage range

Chemical

2,3,7,8-Tetrachlorodibenzodioxin (TCDD)
Polyvinyl chloride
Hemochromatosis
Arsenic

Trauma

Postparturition status
Extremity

Genetic alterations that play a role in the development of soft tissue sarcoma segregate into two major types.

i) Sarcomas with specific genetic alterations that result in simple karyotypes, including fusion genes secondary to reciprocal translocations and specific point mutations such as ***KIT*** mutations in gastrointestinal stromal tumors (GISTs) and ***APC***/b-catenin mutations in desmoid tumors.
ii)Sarcomas with nonspecific genetic alterations and typically complex unbalanced karyotypes representing numerous genetic losses and gains.

Histologic type:		
❖ Synovial sarcoma	❖ Dermatofibrosarcoma protuberans	❖ Atypical lipomatous tumor/well-differentiated liposarcoma
❖ Myxoid/round cell liposarcoma	❖ Desmoplastic small round cell tumor	❖ Leiomyosarcoma
❖ Ewing's sarcoma	❖ Clear cell sarcoma	❖ Malignant fibrous histiocytoma .
❖ Alveolar rhabdomyosarcoma	❖ Infantile fibrosarcoma	❖ Malignant peripheral nerve sheath tumor.
❖ Extraskeletal myxoid chondrosarcoma	❖ Alveolar soft part sarcoma	

- **Age is a factor in histopathology:** Embryonal rhabdomyosarcoma is most common in childhood, synovial sarcoma is more likely to be seen in young adults (<35 years), and there is an even distribution of liposarcoma and MFH as the predominant types in the older population.
- Many MFH is currently being reclassified as myofibrosarcoma, pleomorphic sarcoma, or dedifferentiated liposarcoma. Sarcoma histiotype and liposarcoma subtype are generally important determinants of prognosis and a predictor of distinctive patterns of behavior.
  Biologic behavior is currently best predicted on the basis of histologic type, histologic grade, tumor size, and depth.

Management

Diagnosis:

❖ **Imaging:**

- Magnetic resonance imaging (MRI) is a mainstay in the diagnosis, staging, and follow-up of soft tissue sarcomas. MRI provides excellent soft tissue contrast, tissue specificity, and three-dimensional imaging. It allows accurate detection of involvement of bones and neurovascular bundles. It is very sensitive for detection of locoregional recurrence.

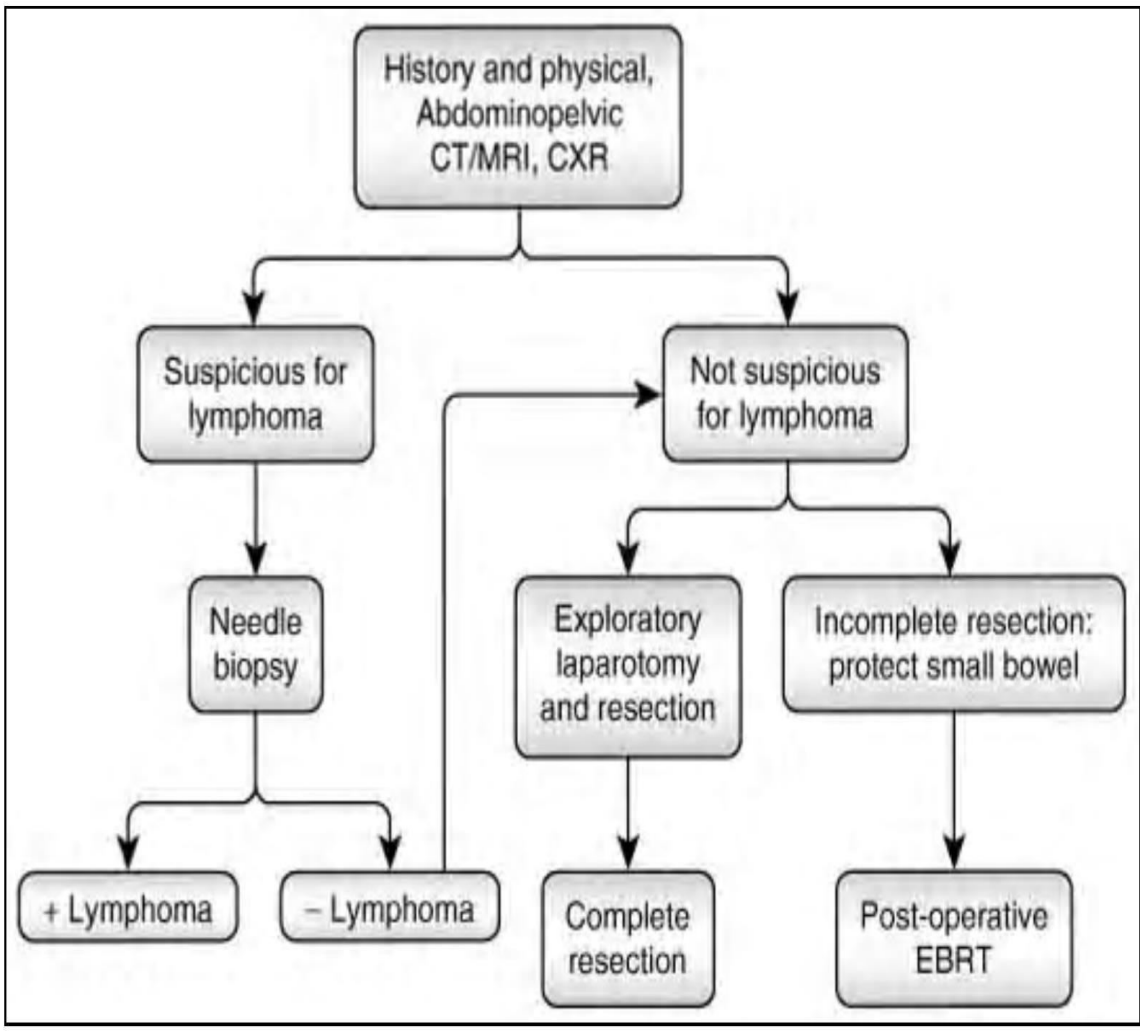
In addition to imaging of the primary, it is also a requisite to survey for metastatic disease.

- Chest x-ray (CXR) and chest computed tomography (CT) scans are important to survey the lungs for hematogenous spread of soft tissue sarcomas.
- Additionally, for sarcomas of the abdomen and pelvis, the liver and peritoneal contents are at risk for metastatic disease and should be surveyed either by MRI or CT.

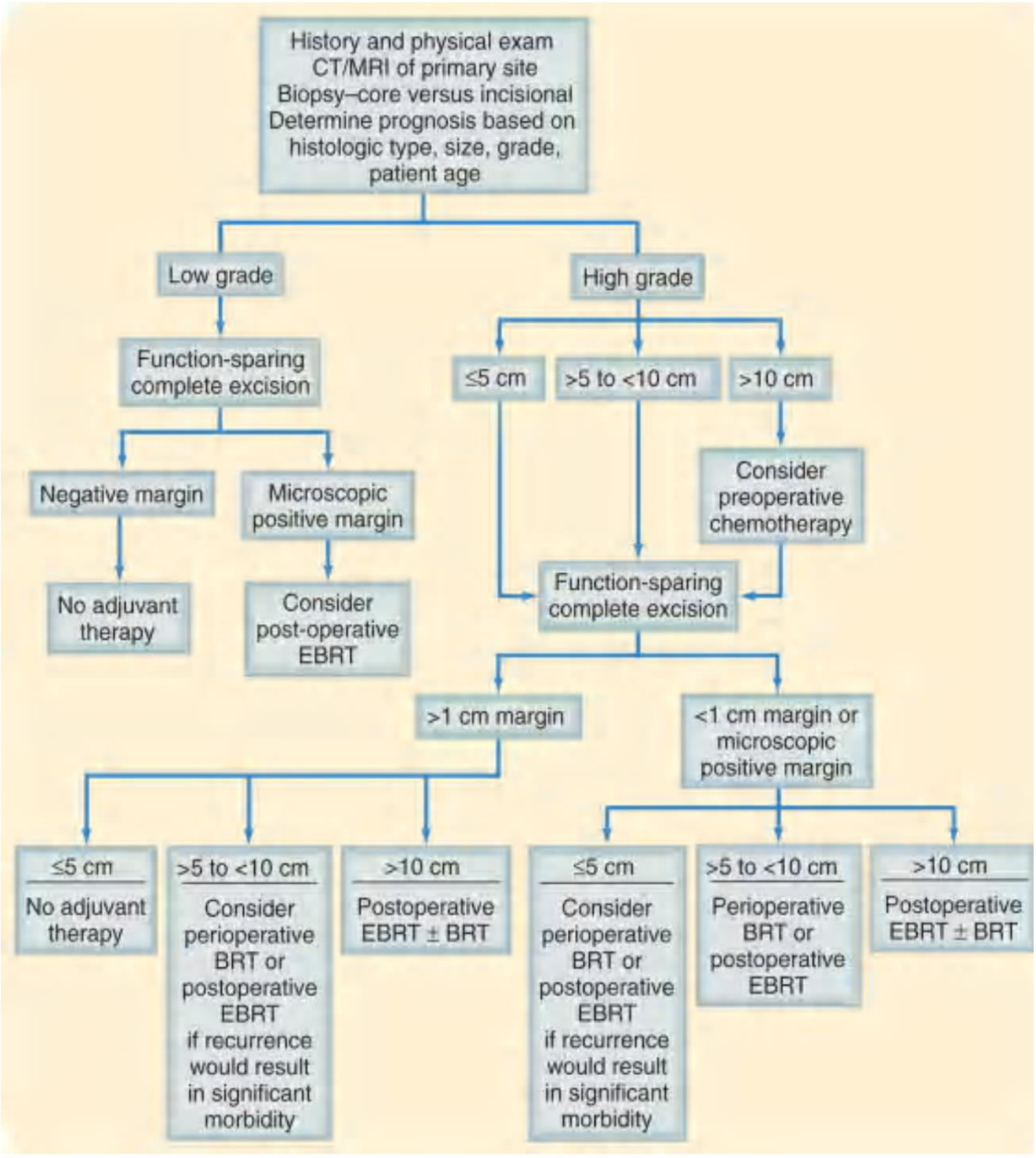
Staging of Soft Tissue Sarcoma:

G, Histologic Grade	**T, Primary Tumor Size**	**N, Regional Nodes**	**M, Distant Metastasis**
GX: Grade cannot be assessed	TX: Primary size cannot be assessed	NX: Regional nodes cannot be assessed	MX: Presence of distant metastasis cannot be assessed
G1: Well differentiated	T0: No evidence of primary tumor	N0: No regional lymph node metastasis	M0: No distant metastasis
G2: Moderately differentiated	T1: Tumor less than 5 cm	N1: Regional lymph node metastasis	M1: Distant metastasis present
G3: Poorly differentiated	T1a: Superficial tumor		
G4: Undifferentiated	T1b: Deep tumor		
	T2: Tumor 5 cm or greater		
	T2a: Superficial tumor		
	T2b: Deep tumor		

Algorithm for the management of primary retroperitoneal or visceral soft tissue sarcomas



Algorithm for the management of Extremity and Superficial Trunk Sarcoma:



2. Discuss the pathology, diagnosis and treatment of cholangiocarcinoma. (4+8+8).

Answer.

Introduction: Most cholangiocarcinomas occur without an apparent underlying etiology or risk factor. Rare patients will have a bile duct adenoma or papillomatosis as a premalignant precursor of bile duct carcinoma. More commonly, chronic inflammation of the bile ducts is implicated as a predisposition for the development of cholangiocarcinoma.

Underlying causes of the ductal irritation include:

- Infection: bacterial infection (***Salmonella typhi*** carrier), parasitic infestation (***Opisthorchis viverrini*** and Clonorchis sinensis in Asia),
- Anatomic anomalies of the biliary tree (choledochal cysts),
- Radiation (thorium dioxide [thorotrast] exposure), and
- Idiopathic causes (primary sclerosing cholangitis).

As with other gastrointestinal cancers, mutations in several proto-oncogenes (K-***ras***, c-***myc***, c-***neu***, c-erb-2 and c-***met***), tumor-suppressor genes (p53, p16, DPC4 and bcl-2), and mismatch repair genes have all been implicated in multiple-step molecular processes leading to malignant transformation.

Pathology: Greater than 90% of bile duct cancers are adenocarcinomas.

Adenocarcinomas of the bile duct are classified as

Sclerosing	Nodular	Papillary
Sclerosing (scirrous) tumors, which comprise over 80% of cholangiocarcinomas, are associated with an intense desmoplastic reaction, tend to be highly invasive, and are associated with low resectability rates.	Nodular tumors have the appearance of constricting annular lesions and are also associated with low resectability rates.	Papillary tumors are rare and present as bulky masses that project into the bile duct lumen.

Because these lesions tend to cause symptomatic obstructive jaundice relatively early in their progression, they are associated with higher resectability rates than sclerosing or

nodular tumors.

Other cancer types include squamous cell carcinoma, small cell carcinoma, and sarcoma.

Diagnosis:

- The majority of extrahepatic bile duct cancer patients present with painless jaundice. Intrahepatic cholangiocarcinomas do not usually cause hyperbilirubinemia.
- Constitutional symptoms (weight loss, anorexia, fatigue, and malaise) and abdominal pain are generally indicators of advanced cancer.
- Serum levels of bilirubin, alkaline phosphatase, and gamma-glutamyltransferase are significantly elevated, whereas serum aminotransferase levels are minimally affected. The glycoprotein marker CA 19-9 has been used to diagnose bile duct cancers but lacks complete sensitivity and specificity, as do other serum tumor markers.

1. Abdominal ultrasonography is usually the first imaging modality done in a patient presenting with jaundice .The bile ducts proximal to the cancer will be dilated, with only intrahepatic dilation with hilar cancer and intrahepatic and extrahepatic distension with distal bile duct cancers. Ultrasonography often will not visualize the primary bile duct tumor, but the site of ductal obstrucsion and the lack of common duct stones often suggest the diagnosis of cholangiocarcinoma.
2. Evidence of vascular involvement by hilar cholangiocarcinomas, including encasement, stenosis, and thrombosis can be detected with duplex ultrasonography.
3. Intrahepatic cholangiocarcinomas appear as malignant solid masses of the liver with no distinguishing features.
4. If the bile duct cancer is unresectable or the patient is not considered an operative candidate due to comorbidities, percutaneous or endoscopic needle biopsy or cytology should be obtained.
5. For intrahepatic tumors and distal malignant bile duct obstruction, a spiral, thin-cut computerized tomography (CT) with intravenous contrast is the imaging study of choice to assess for the extent of the primary tumor, regional nodal involvement, and distant intraabdominal metastasis. In a patient with suspected hilar cholangiocarcinoma, the proximal extent of ductal tumor spread, involvement of hilar blood vessels, and assessment of the parenchyma of both halves of the liver need to be determined.
6. Magnetic resonance imaging (MRI) and magnetic resonance cholangiopancreatography (MRCP) can offer additional information on local invasion, particularly into the porta hepatis. These tests are used selectively if CT findings are equivocal.

7. EUS: useful for diagnosing T stage and taking biopsy.

Cholangiocarcinoma is best classified anatomically into three broad groups:

Intrahepatic	Perihilar	Distal

AJCC Staging System of Extrahepatic Bile Duct Cancers

Primary Tumor (T)	
TX	Primary tumor cannot be assessed
T0	No evidence of primary tumor
Tis	Carcinoma in situ
T1	Tumor confined to the bile duct histologically
T2	Tumor invades beyond the wall of the bile duct
T3	Tumor invades the liver, gallbladder, pancreas, and/or unilateral branches of the portal vein (right or left) or hepatic artery (right or left)
T4	Tumor invades any of the following: main portal vein or its branches bilaterally, common hepatic artery, or other adjacent structures, such as the colon, stomach, duodenum, or abdominal wall
Regional Lymph Nodes (N)	
NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Regional lymph node metastasis (e.g., hilar, celiac, periduodenal, peripancreatic, and superior mesenteric)
Distant Metastasis (M)	
MX	Distant metastasis cannot be assessed
M0	No distant metastasis
M1	Distant metastasis

Stage Grouping			
Stage	**T**	**N**	**M**
Stage 0	Tis	N0	M0

Stage Grouping			
Stage	**T**	**N**	**M**
Stage IA	T1	N0	M0
Stage IB	T2	N0	M0
Stage IIA	T3	N0	M0
Stage IIB	T1-3	N1	M0
Stage III	T4	Any	M0
Stage IV	Any	Any	M1

Classification of Perihilar Bile Duct Cancers According to Anatomic Location. (**The Bismuth-Corlette classification of perihilar cholangiocarcinomas).**

Classification of Perihilar Bile Duct Cancers According to Anatomic Location
Type I: Tumors below the confluence of the left and right hepatic ducts
Type II: Tumors reaching the confluence
Type IIIa/IIIb: Tumors involving common hepatic duct and either the right or the left hepatic duct, respectively
Type IV: Tumors that are multicentric or involve the confluence and both the right and left hepatic ducts

Preoperative preparation:

- The evaluation and correction of concurrent medical problems should be done.
- Clinically jaundiced patients will have reduced stores of fat-soluble vitamins, most importantly for the surgeon, vitamin K. Even if the prothrombin time is normal, a parenteral dose of vitamin K (10 mg) should be administered preoperatively to reduce the risk of perioperative hemorrhage.
- Hepatic insufficiency is one of the most common causes of postoperative morbidity and mortality in patients operated on for a bile duct cancer. There is no indication for preoperative biliary decompression for patients with distal extrahepatic bile duct cancers.
- Despite this risk, when the total bilirubin is greater than 15 mg/dl and a major hepatic resection is planned for a perihilar bile duct cancer, many surgeons recommend transhepatic drainage of the remnant liver to allow for an

improvement in hepatic function. When the projected hepatic remnant appears inadequate for a safe recovery, preoperative portal vein embolization of the tumor-involved side of the liver usually induces sufficient contralateral hepatic hypertrophy to reduce the incidence of postoperative liver insufficiency.

Radiologic Criteria to Suggest Unresectability of Cholangiocarcinoma:

Bilateral hepatic duct involvement up to secondary radicals	Atrophy of one hepatic lobe with contralateral biliary radical involvement
Bilateral hepatic artery involvement	Distant metastasis
Encasement of the portal vein proximal to its bifurcation	

Operative Approach:

Surgical exploration should be undertaken in good-risk patients with no evidence of metastatic or locally unresectable disease.	Selective use of laparoscopy in patients with locally advanced but potentially resectable perihilar cholangiocarcinoma may avoid laparotomy in some patients with metastatic disease.	In patients who are found to have extensive metastatic disease, the preoperatively placed biliary stents should be left in place.
Intraoperatively, more than half of these patients are found to have either peritoneal or hepatic metastases or locally unresectable disease.	In patients with locally advanced unresectable perihilar tumors, several operative approaches are available for palliation, including a Roux-en-Y hepaticojejunostomy to segment III or V.	A cholecystectomy should be performed to avoid the risk of acute cholecystitis, which occurs in patients with long-term indwelling biliary stents.

Distal cholangiocarcinoma	**Intrahepatic cholangiocarcinoma**	**Perihilar cholangiocarcinoma**
Usually treated with pancreaticoduodenectomy.	Treated by hepatic resection, and outcomes depend on disease stage	Bile duct resection alone leads to high local recurrence rates due to early involvement of the

A pylorus-preserving operation is preferable. 5-year survival rates averaging 15% to 25%, but can be as high as 54% in selected patients who undergo complete resection for node-negative disease. If resection is not possible due to vascular encasement, cholecystectomy, Roux-en-Y hepaticojejunostomy proximal to the tumor, and a gastrojejunostomy to prevent gastric outlet obstruction should be performed.	(particularly the status of the lymph nodes) and the ability to achieve negative margins. There is a broad range of long-term outcomes in patients undergoing complete resection (3-year survival rates of 22%-66%).	confluence of the hepatic ducts and the caudate lobe branches. The addition of a modified hepatic resection has improved resectability rates. Surgical treatment depends on the Bismuth-Corlette classification . For type I and II lesions, the procedure is en bloc resection of the extrahepatic bile ducts and gallbladder with 5- to 10-mm bile duct margins, and regional lymphadenectomy with Roux-en-Y hepaticojejunostomy. In addition to the above operations, type II tumors may require hepatic lobectomy. Because type II and III lesions often involve the ducts of the caudate lobe, many surgeons recommend routine caudate lobectomy. Type III and IV tumors are amenable to potentially curative resection in centers with expertise in these procedures. **Aggressive techniques such as hepatectomy and portal vein resection to achieve negative margins are now routine in specialized centers.**

Palliative therapy:

- Patients with cholangiocarcinoma are at high risk for mortality secondary to hepatic failure from chronic biliary obstruction and biliary sepsis, once the bile ducts have been intubated.

- For patients with metastatic or locally advanced tumors not amenable to complete resection, the primary goals of palliation are to: (1) provide biliary drainage to the maximal amount of functioning parenchyma and (2) prevent cholangitis.
- External beam irradiation and catheter-delivered brachytherapy radiation have been recommended for patients with locally advanced, unresectable bile duct cancers, but no large controlled trial has confirmed the benefit of these treatments. At present, there is no recognized efficacious chemotherapy for cholangiocarcinoma.
- For perihilar cholangiocarcinomas, nonoperative biliary decompression is best accomplished with bilateral percutaneous transhepatic catheters placed through the tumor obstruction. Atrophic portions of the liver should not be drained unless they have become infected by prior manipulation. The catheters will need to be changed at least every 3 to 6 months, and often sooner when cholangitis or other signs of malfunction occur.
- Metastatic distal bile duct cancer patients are generally best managed nonoperatively with an endoscopic biliary stent.
- For patients found to have unresectable bile duct cancer at the time of celiotomy, surgical biliary bypass should be undertaken, if this can be achieved without significant morbidity.
- Distal bile duct cancers are usually readily bypassed with a loop or Roux-en-Y choledochojejunostomy or hepaticojejunostomy.
- For perihilar cholangiocarcinomas, the surgical options include a Roux-en-Y cholangiojejunostomy to the segment III duct (this procedure only decompresses the left liver with tumors that have obstructed the hepatic duct confluence) and palliative resection of the tumor with right and left hepaticojejunostomies. Because of an infiltrative growth pattern proximally along the bile ducts, approximately 20% of hilar cholangiocarcinoma resections undertaken with curative intent, even when including a hepatectomy, will not achieve microscopic tumor clearance, most commonly on the proximal duct margin(s). Transhepatic drains should be left in position in these patients and postoperative radiation treatment, with or without chemotherapy as a radiosensitizer, considered. If the liver has been adequately decompressed preoperatively, relying on transhepatic catheter drainage is also a reasonable palliative care option.

Adjuvant therapy:

- No controlled data support the use of chemotherapy or radiation therapy in the management of bile duct cancers. Bile duct cancers are generally radiosensitive, but the risk of toxicity from excessive exposure to radiosensitive organs, especially the liver and bowel, limit its efficacy. Multiple chemotherapy drugs, including 5-fluorouracil, mitomycin-C, doxorubicin, cisplatin, and etoposide, have shown modest

response rates in bile duct cancer patients, but none of the results has generated interest for a major controlled trial of a specific chemotherapy regimen.

- Some clinicians recommend external beam radiation therapy with 5-fluorouracil following curative pancreaticoduodenectomy for distal bile duct tumors, because this treatment is commonly used for other periampullary malignancies. External beam, intraoperative radiation therapy, and brachytherapy delivered by transhepatic catheters have all been evaluated in the management of perihilar cancers.
- No adjuvant therapy is usually recommended for intrahepatic cholangiocarcinomas.

3. Write short notes on: 5 x 6

(a) Primary Lymphoedema
(b) Sjogren Syndrome.
(c) Glucagonoma
(d) Sliding Hernia.
(e) Mesenteric cyst.

Answer.

(a) Primary Lymphoedema

	Congenital hyperplasia(10%)	Distal obliteration(80%)	Proximal obliteration(10%)
Age of onset	Congenital	Puberty(praecox)	Any age
Sex distribution	Male > Female	Female > Male	Male = Female
Extent	Whole leg	Ankle, calf	Whole leg, thigh only
Laterality	Unilateral = bilateral	Often bilateral	Usually unilateral
Family history	Often positive	Often positive	No
Progression	Progressive	Slow	Rapid
Response to compression therapy	Variable	Good	Poor
Comments	Lymphatics are increased in number; although functionally defective, there is usually an increased number of lymph nodes. May have	Absent or reduced distal superficial lymphatics. Also termed aplasia or hypoplasia.	There is obstruction at the level of the aortoiliac or inguinal nodes. If associated with distal dilatation, the patient may benefit from lymphatic

	chylous ascites, chylothorax and protein-losing enteropathy.		bypass operation. Other patients have distal obliteration as well.

(b) Sjogren Syndrome:

Essentials of Diagnosis:

- Women are 90% of patients; the average age is 50 years.
- Dryness of eyes and dry mouth (sicca components) are the most common features; they occur alone or in association with rheumatoid arthritis or other connective tissue disease.
- Rheumatoid factor and other autoantibodies common.
- Increased incidence of lymphoma.

General Considerations:

- Sjögren syndrome is a systemic autoimmune disorder whose clinical presentation is usually dominated by dryness of the eyes and mouth due to immune-mediated dysfunction of the lacrimal and salivary glands.
- The disorder is predominantly seen in women, with a ratio of 9:1; most cases develop between the ages of 40 and 60 years.
- Sjögren syndrome can occur in isolation ("primary" Sjögren syndrome) or in association with another rheumatic disease. Sjögren syndrome is most frequently associated with rheumatoid arthritis but also occurs with SLE, primary biliary cirrhosis, scleroderma, polymyositis, Hashimoto thyroiditis, polyarteritis, and interstitial pulmonary fibrosis.

Symptoms and Signs:

- Keratoconjunctivitis sicca results from inadequate tear production caused by lymphocyte and plasma cell infiltration of the lacrimal glands. Ocular symptoms are usually mild. Burning, itching, and the sensation of having a foreign body or a grain of sand in the eye occur commonly.
- For some patients, the initial manifestation is the inability to tolerate wearing contact lenses. Many patients with more severe ocular dryness notice ropy secretions across their eyes, especially in the morning. Photophobia may signal corneal ulceration resulting from severe dryness.

- For most patients, symptoms of dryness of the mouth (xerostomia) dominate those of dry eyes. Patients frequently complain of a "cotton mouth" sensation and difficulty swallowing foods, especially dry foods like crackers, unless they are washed down with liquids. The persistent oral dryness causes most patients to carry water bottles or other liquid dispensers from which they sip constantly.
- A few patients have such severe xerostomia that they have difficulty speaking. Persistent xerostomia results often in rampant dental carries; carries at the gum line strongly suggest Sjögren syndrome. Some patients are most troubled by loss of taste and smell. Parotid enlargement, which may be chronic or relapsing, develops in one-third of patients. Desiccation may involve the nose, throat, larynx, bronchi, vagina, and skin.
- Systemic manifestations include dysphagia, vasculitis, pleuritis, obstructive lung disease (in the absence of smoking), neuropsychiatric dysfunction (most commonly peripheral neuropathies), and pancreatitis; they may be related to the associated diseases noted above. Renal tubular acidosis (type I, distal) occurs in 20% of patients. Chronic interstitial nephritis, which may result in impaired renal function, may be seen. A glomerular lesion is rarely observed but may occur secondary to associated cryoglobulinemia.

Laboratory Findings:

- Laboratory findings include mild anemia, leukopenia, and eosinophilia. Polyclonal hypergammaglobulinemia, rheumatoid factor positivity (70%), and antinuclear antibodies (95%) are all common findings. Antibodies against the cytoplasmic antigens SS-A and SS-B (also called Ro and La, respectively) are often present in primary Sjögren syndrome and tend to correlate with the presence of extraglandular manifestations. Thyroid-associated autoimmunity is a common finding among patients with Sjögren syndrome.

Useful ocular diagnostic tests include the Schirmer test, which measures the quantity of tears secreted. Lip biopsy, a simple procedure, reveals characteristic lymphoid foci in accessory salivary glands. Biopsy of the parotid gland should be reserved for patients with atypical presentations such as unilateral gland enlargement that suggest a neoplastic process.

Differential Diagnosis:

Isolated complaints of dry mouth are most commonly due to medication side effects. Chronic hepatic C can cause sicca symptoms. Minor salivary gland biopsies reveal lymphocytic infiltrates but not to the extent of Sjögren syndrome, and tests for anti-SS-A

and anti-SS-B are negative. Diffuse infiltration of CD8 T cells producing parotid gland enlargement can develop in HIV-infected individuals. Involvement of the lacrimal or salivary glands, or both in sarcoidosis can mimic Sjögren syndrome; biopsies reveal noncaseating granulomas. Rarely, amyloid deposits in the lacrimal and salivary glands produce sicca symptoms.

Treatment & Prognosis:

Treatment is symptomatic and supportive. Artificial tears applied frequently will relieve ocular symptoms and avert further desiccation. The mouth should be kept well lubricated. Sipping water frequently or using sugar-free gums and hard candies usually relieves dry mouth symptoms. Pilocarpine (5 mg orally four times daily) and the acetylcholine derivative cevimeline (30 mg orally three times daily) may improve xerostomia symptoms. Atropinic drugs and decongestants decrease salivary secretions and should be avoided. A program of oral hygiene, including fluoride treatment, is essential in order to preserve dentition. If there is an associated rheumatic disease, its systemic treatment is not altered by the presence of Sjögren syndrome.

Although Sjögren syndrome may compromise patients' quality of life significantly, the disease is usually consistent with a normal life span. Poor prognoses are influenced mainly by the presence of systemic features associated with underlying disorders, the development in some patients of lymphocytic vasculitis, the occurrence of a painful peripheral neuropathy, and the complication (in a minority of patients) of lymphoma. The patients (3–10% of the total Sjögren population) at greatest risk for developing lymphoma are those with severe exocrine dysfunction, marked parotid gland enlargement, splenomegaly, vasculitis, peripheral neuropathy, anemia, and mixed monoclonal cryoglobulinemia.

(c) Glucagonoma

- It is a tumor of islet alpha cells.
- Glucagonoma causes a syndrome of a characteristic rash, diabetes mellitus, anemia, weight loss, and elevated circulating levels of glucagon.
- The characteristic skin lesion is a necrolytic migrating erythema.
- The syndrome is rare, and most patients are initially recognized by their skin lesions and referred to surgeons by dermatologists.
- Glucagonoma was found to be associated with anemia, glossitis, and most importantly, a low level of amino acids; parenteral administration of amino acids was found to bring about disappearance of the skin lesions.
- Diabetes is usually mild.

- A pseudoglucagonoma syndrome has been described in patients who have necrolytic migratory erythema without a pancreatic tumor; the cause is unknown, but the condition is associated with several chronic illnesses, and only a few patients show elevated levels of glucagon.

Diagnosis:

- The diagnosis of glucagonoma is made from the characteristic skin lesion, elevated levels of glucagon (whose release can be provoked by secretin, if necessary), and a pancreatic tumor. The upper limit of normal for glucagon is 150 to 190 pg/mL; glucagonoma patients have levels of 200 to 2000 pg/mL.
- The islet tumor may be demonstrated by enhanced CT or MRI or by selective angiography.

Localization and treatment:

- Once the diagnosis is made, the patient is prepared by the administration of total parenteral nutrition containing amino acids, along with simultaneous octreotide for symptomatic relief.
- Tumors are best localized by CT, MRI, and intraoperative ultrasonography.
- Because a third of these patients have been reported to have thrombotic complications after surgery, perioperative heparin is indicated.
- Treatment is surgical excision of the tumor, which usually lies in the body or tail of the pancreas. Nearly all glucagonomas are malignant, but an aggressive approach to removal of the primary and metastatic tumor is warranted.
- Even so, the cure rate appears to be only 30%, and long-term chemotherapy has proved disappointing for metastatic disease.
- Symptomatic relief can be achieved with octreotide.

(d) Sliding Hernia. A sliding hernia results due to slipping of the posterior parietal peritoneum on the underlying structures(usually indirect inguinal in location) and a part of the wall of the hernia sac is formed by an intra-abdominal viscus (usually colon – sigmoid colon & its mesentery on the left, the caecum on the right sometimes, on either side by a portion of the bladder bladder).

Incidence:

- Small bowel sliding hernia – 1in 2000 cases.
- Sacless hernia – 1in 8000 cases.

Clinical features:

- Occurs exclusively in male.
- 5 out of 6 are on the left side.
- Bilateral rare.
- Age of the patient is nearly always over 40 years. Incidence rises with age.
- It should be suspected in a very large globular inguinal hernia descending well into the scrotum.
- Occasionally, large intestine is strangulated in a sliding hernia; more often, non-strangulated large intestine is present behind the sac containing strangulated small intestine.

Traetment:

- Impossible to control with truss.
- Operation is indicated.

Principle of operation:

- It is unnecessary to any of the sliding hernial sac provided it is freed completely from the cord and the abdominal wall and that it is replaced deep to the repaired fascia transversalis.
- No attempt should be made to dissect the caecum or colon free from the peritoneum.

Complication of operation:

- Peritonitis.
- Faecal fistula.
- Injury to the vessel of the mesocolon.

(e) Mesenteric cyst.

- Cysts of the mesentery are benign lesions.
- Incidence is less than one in 100,000.
- The etiology of such cysts remains unknown, but several theories regarding their development exist, including degeneration of the mesenteric lymphatics or simply arising as a congenital anomaly.

Cassification:

- Chylolymphatic (commonest) – arises in congenitally misplaced lymphatic tissue that has no efferent communication with the lymphatic system; it arises most frequently in the mesentery of the ileum. It is almost invariably solitary. A Chylolymphatic Cyst has a independent blood supply.Enucleation is possible without resecting gut.
- Simple (mesothelial).
- Enterogenous – derived either from a diverticulum of the mesenteric border of the intestine due to sequestration from the intestinal canal during embryonic life or from a duplication of the intestine.The muscle in the wall of an enteric duplication cyst and the bowel with which it is in contact have a common blood supply; removal of the cyst always entails resection of the related portion of intestine.
- Urogenital remnant.
- Dermoid (teratomatous cyst).

Clinical features:

- Found more frequently in the 2nd decade of life
- .Mesenteric cysts may be asymptomatic or can cause symptoms of a mass lesion.
- Symptoms may be acute or chronic.
- Acute abdominal pain secondary to a mesenteric cyst is generally caused by rupture or torsion of the cyst or from acute hemorrhage into the cyst. There may be infection.
- Mesenteric cysts may also cause chronic intermittent abdominal pain secondary to compression of adjacent structures or spontaneous torsion followed by detorsion of the cyst.
- Mesenteric cysts can be the cause of nonspecific symptoms such as anorexia, nausea, vomiting, fatigue, and weight loss.
- Physical examination may reveal a mass lesion that is mobile only from the patient's right to left or left to right (Tillaux's sign), ie. in a plane at right angles to the attachments of the mesentery. Fluctuant swelling around the cyst.There is a zone of resonance around the cyst.

Imaging studies:

- Computed tomography, abdominal ultrasound, and magnetic resonance imaging all have been used to evaluate patients with mesenteric cysts.Each of the aforementioned imaging modalities reveal a cystic structure without a solid component in the central abdomen.
- These are generally unilocular, but may, on occasion, be multiple or multilocular.

- Irrespective of the imaging method used, it may be difficult to distinguish these cystic masses from rare solid mesenteric tumors with cystic components such as a cystic stromal tumor or mesothelioma.
- Mesenteric cystic lymphangioma may present as numerous, often large cysts in the setting of abdominal pain. These can be difficult to treat and almost invariably recur after excision.

Treatment:

- When symptomatic, simple mesenteric cysts are surgically excised either openly or laparoscopically when feasible.
- Cyst unroofing or marsupialization is not recommended, as mesenteric cysts have a high propensity to recur after drainage alone.
- On rare occasions, adjacent mesentery may be densely adherent to the cyst or mesenteric vessels must be sacrificed in order to achieve complete excision, in which case segmental bowel resection is performed.

4. Write brief answers: 4 x 7.5

(a) Management of aggressive GIST;
(b) Treatment of Venous ulcers;
(c) Short Bowel Syndrome;
(d) Surgical Management of Ulcerative Colitis.

Answer.

(a) Management of aggressive GIST. Gastrointestinal stromal tumors (GIST) are rare malignancies. Although they are the most common sarcoma of the gastrointestinal (GI) tract, they represent only 0.2% of all GI tumors.

KIT and PDGFRA mutations in GIST: KIT and PDGFRA mutations in GIST produce constitutive ligand-independent receptor activation. Response to tyrosine kinase inhibitors correlates with the location of the activating mutation, with best response in patients whose tumors contain mutations in KIT exon 11.

Imaging and Diagnostic Studies:

- The endoscopic appearance of a primary GIST is that of a submucosal lesion, with or without ulceration, present in the upper or lower GI tract. These lesions are visually indistinguishable from other GI tumors of smooth muscle origin. Because of their submucosal location, fine-needle aspiration (FNA) or core biopsy with endoscopic ultrasound guidance is commonly required to obtain tissue for diagnosis.

- CT scans are critical to determine the anatomic extent of a GIST and to assist with operative planning. The CT findings vary with the size of the lesions . Small GIST have sharp margins, an intraluminal growth pattern, and are of homogenous density on both unenhanced and contrast-enhanced scans. In contrast, larger lesions have irregular margins, extraluminal growth patterns, and inhomogeneous density.
- Radiographic signs corresponding to aggressive malignant GIST include calcification, ulceration, necrosis, cystic areas, fistula formation, metastasis, ascites, and signs of infiltration of local tissues. Unfortunately, CT is unable to differentiate between inflammatory adhesions and malignant involvement of adjoining organs. It is also unlikely to identify any peritoneal metastasis smaller than 2 cm in diameter.
- Endoscopic examination of primary gastric GISTis helpful.

GIST commonly present as submucosal tumors in the wall of the GI tract. Central necrosis of high-risk tumors or erosion into blood vessels can produce local inflammation or significant GI bleeding. Positron emission tomography (PET) is a useful adjunct to CT for evaluating GIST, particularly as a means of assessing response to chemotherapy. Metabolically active GIST accumulate 18-fluorodeoxyglucose (^{18}FDG), and blockade of the KIT receptor results in a rapid suppression of this activity. Functional blockade of KIT activation is produced by specific inhibitors of KIT tyrosine kinase activity, such as imatinib mesylate.

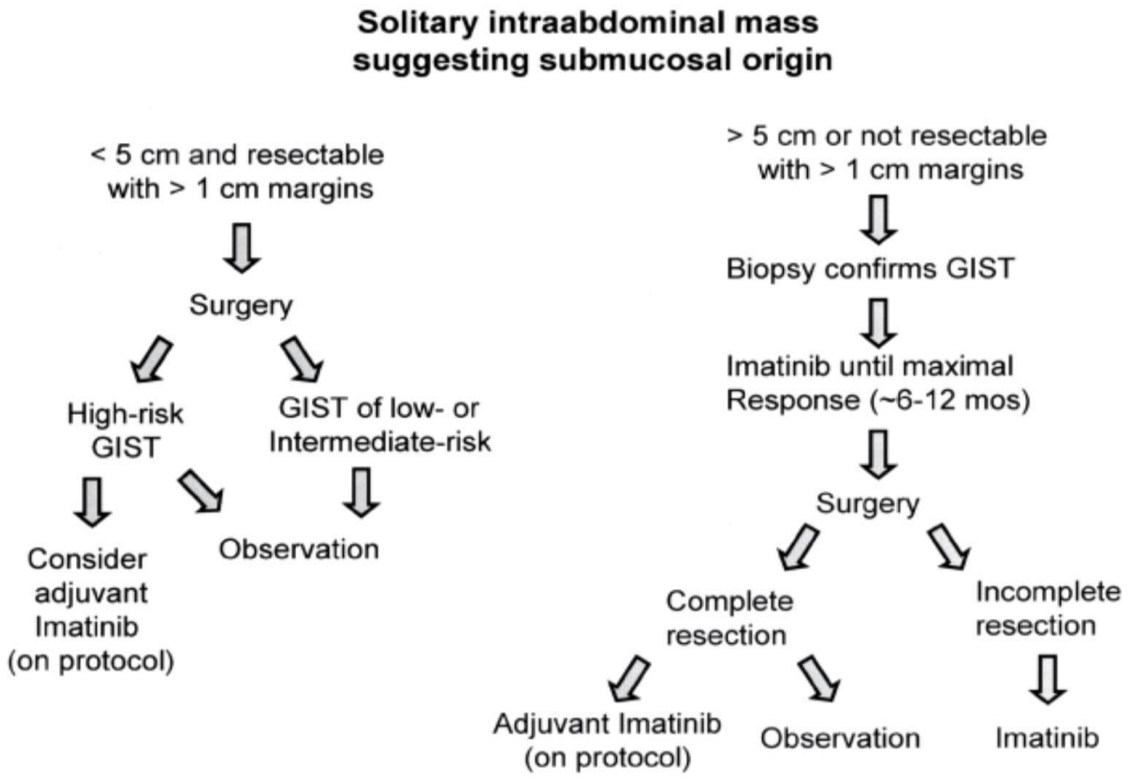
Prognostic Features

Classification of Primary Gastrointestinal Stromal Tumors by Risk of Metastasis

Risk Category	**Size**	**Mitotic Count**
Very Low	<2 cm	<5 per 50 HPFs*
Low	2–5 cm	<5 per 50 HPFs
Intermediate	<5 cm	6–10 per 50 HPFs
	5–10 cm	<5 per 50 HPFs
High	>5 cm	>5 per 50 HPFs
	>10 cm	Any mitotic rate
	Any size	>10 per 50 HPFs

*HPF, high powered field.

Surgical Management of Localized Primary Diseease:



Postsurgical Follow-Up:

The typical sites of tumor recurrence following resection of a GIST with curative intent are the local resection bed, the liver, and the peritoneum. Pulmonary metastases are uncommon. In general, the time to recurrence reflects the original growth pattern of the tumor, and recurrences as early as 3 months following resection have been observed. Most centers use abdominal/pelvic CT scanning for posttreatment follow-up, with PET scanning or MRI reserved for clarification of equivocal CT findings. Because more recurrences occur within the first 5 years after surgery, imaging intervals of 3 to 6 months are standard for patients in the first 5 years of post treatment follow-up, with annual evaluation thereafter. At present there are no specific serum-based markers for the detection of recurrent GIST.

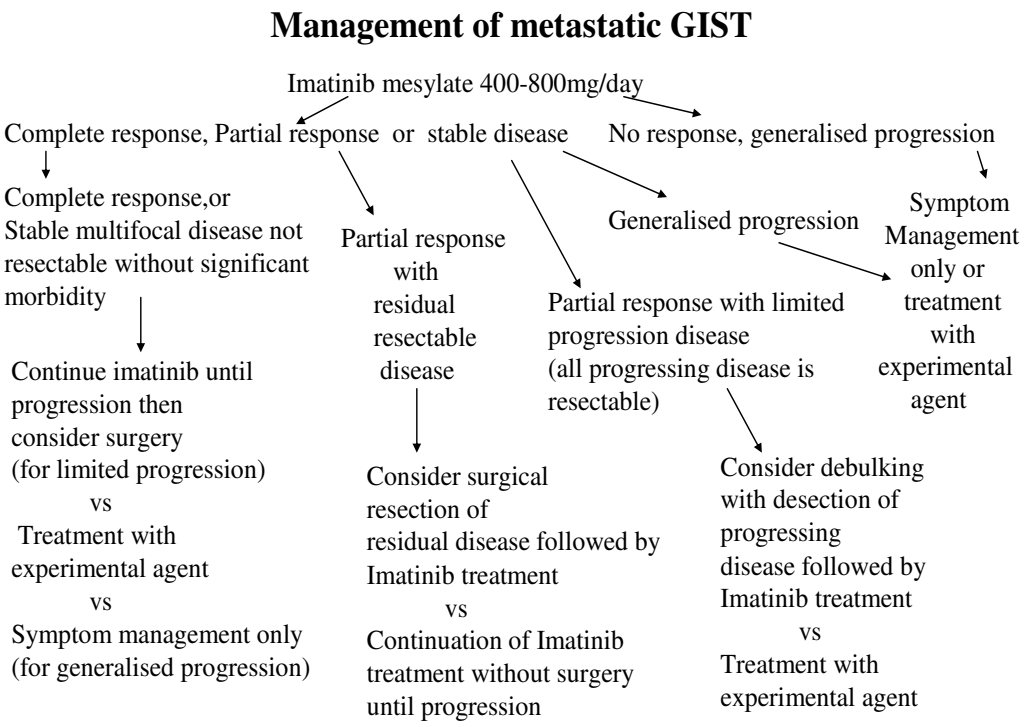
Adjuvant Therapy:

For patients with localized GIST treated with surgery alone, the reported recurrence rate at 5 years varies considerably, with recurrent disease developing in 2–15% of those with low-risk and 70–90% of those with high-risk primary tumors.In the absence of adjuvant therapy, approximately 50% of patients receiving potentially curative surgery will develop either locally recurrent or metastatic disease within 5 years, yielding 5-year

survival rates of 40–55%.The effectiveness of imatinib in managing metastatic disease has led to combined modality treatment of high-risk localized tumors.

Use of Imatinib in Unresectable Primary Disease:

The success of imatinib in managing metastatic disease led to its use as a means of improving the resectability of technically inoperable primary tumors. Surgery is performed when tumor size has decreased to the point at which resection is technically possible, and/or when successive CT scans show no further tumor shrinkage. Generally, this occurs from 9 to 12 months following initiation of chemotherapy. Following surgery, these high-risk patients are routinely maintained on imatinib indefinitely, as long as drug-related toxicity is minimal.



New Therapies for Management of Primary and Secondary Drug Resistance:

A number of different agents are currently under investigation in clinical trials to determine their utility as second-line drugs for the management of imatinib-resistant GIST. These agents have been selected based upon their ability to disrupt KIT tyrosine kinase activity, or to inhibit targets that are downstream of KIT or PDGFRA. At least one

of these, sunitinib, has shown some efficacy in phase II studies. Sunitinib is a tyrosine kinase inhibitor with activity against KIT, PDGFR, FLT3, and VEGFR.

(b) Treatment of Venous ulcers

Nonsurgical Treatment:

A. Infected ulcers

- Necessitate treatment of the infection first.
- Staphylococcus aureus, Streptococcus pyogenes, and Pseudomonas species are responsible for most infections.
- Usually treated with local wound care, wet-to-dry dressings, and oral antibiotics.
- Topical antiseptics should be avoided.
- Severe infections require intravenous antibiotics.

B. Leg elevation
Leg elevation can temporarily decrease edema and should be instituted when swelling occurs. This should be done before a patient is fitted for stockings or boots.

C. Compression therapy
Compression therapy is the primary treatment for CVI.

- Elastic compression stockings
  - Fitted to provide a compression gradient from 30 to 40 mm Hg, with the greatest compression at the ankle.
  - Donned on arising from bed and removed at bedtime.
  - Effective in healing ulcers but can take months to obtain good results.
  - Study of 113 patients treated with initial bedrest, local wound care, and elastic compression stockings demonstrated a 93% ulcer healing rate in a mean of 5.3 months (Surgery 1991;109:575).
  - Stockings do not correct the abnormal venous hemodynamics and must be worn after the ulcer has healed to prevent recurrence.
  - Principal drawback is patient compliance.
  - Recurrence for compliant patients in the same study was 16% at a mean follow-up of 30 months.
- Unna boots
  - Paste gauze compression dressings that contain zinc oxide, calamine, and glycerin.
  - Used to help prevent further skin breakdown.
  - Provide nonelastic compression therapy.

- Changed once or twice a week.
- Healing time for ulcers is less than that of elastic compression alone.

- Pneumatic compression devices
  - Provide dynamic sequential compression.
  - Used primarily in the prevention of deep vein thrombi in hospitalized patients.
  - Also used successfully to treat venous insufficiency.

D. Topical medications

- Largely ineffective as a stand-alone therapy for venous stasis ulcers.
- Topical therapy is directed at absorbing wound drainage and avoiding desiccation of the wound.
- Antiseptics can be counterproductive. Hydrogen peroxide, povidone-iodine, acetic acid, and sodium hypochlorite are toxic to cultured fibroblasts and should be used for the shortest duration necessary to control ulcer infection.

Surgical Therapy:

- Skin grafting
  - Occasionally used to speed healing of large ulcers.
  - Ulcer bed should be dry and free of infection.
  - Fenestrated split-thickness skin graft is preferred to allow for serous drainage.
  - Bedrest is recommended until the ulcer has healed completely.
  - Recurrence is common unless the underlying venous pathology is corrected or conservative support (elastic compression) is initiated and maintained after skin grafting. Cultured skin substitutes are now commonly used to minimize patient discomfort and avoid possible donor-site complications.
  - Stripping the greater saphenous vein.

(d) Short Bowel Syndrome

Introduction: In the adult, the length of the small bowel varies from 300 to 600 cm and correlates directly with body surface area. Several factors determine the severity of short-bowel syndrome, including the extent of resection, the portion of the GI tract removed, the type of disease necessitating the resection, the presence of coexistent disease in the remaining bowel, and the adaptability of the remaining bowel. Generally, resection resulting in less than 120 cm of intact bowel leads to short-bowel syndrome (<150 cm if ending in an ileostomy, <75 cm if colon remains).

Infants may survive resection of up to 85% of bowel owing to the enhanced ability of the bowel to adapt and grow with the child. Because of its specialized absorptive function,

resection of the ileum is usually not well tolerated. However, the entire jejunum can be resected without serious adverse nutritional sequela.

Etiology and pathophysiology:

- Short-bowel syndrome is characterized by dehydration, electrolyte derangements, acidic diarrhea, steatorrhea, malnutrition, and weight loss.
- Congenital anomalies leading to short-bowel syndrome include intestinal atresia, midgut volvulus with intestinal necrosis, and necrotizing enterocolitis.
- In middle-aged adults, inflammatory bowel disease and trauma are the leading causes of massive intestinal resection.
- In the elderly, prominent causes include mesenteric ischemia and strangulated hernia.

Adaptation - The distal small intestine has the greatest adaptive potential and can assume many of the absorptive properties of the proximal GI tract. Cellular hyperplasia and bowel hypertrophy occur over a 2- to 3-year period, increasing the absorptive surface area. Fat absorption is the metabolic process most likely to be permanently impaired; other functions adjust and normalize fairly well.

- Fluid and electrolyte response. Of the 8 to 10 L of fluid presented daily to the small intestine, only 1 to 2 L are delivered into the colon. Significant quantities of electrolytes are absorbed in this process. With short-bowel syndrome, this physiology is altered. Strict intake and output records and close monitoring of serum electrolytes are critical in the early management of patients with short-bowel syndrome.
- Malabsorption and malnutrition
    - **Gastric hypersecretion**, seen early in the postoperative period, can persist for prolonged periods. Increased acid load may injure distal bowel mucosa, leading to hypermotility and impaired absorption. The severity of hypersecretion correlates directly with the extent of bowel resection. This generally is more pronounced after jejunal than after ileal resection. Loss of an intestinal inhibitory hormone has been implicated.
    - **Cholelithiasis**. Altered bilirubin metabolism after ileal resection increases the risk of gallstones secondary to a decreased bile salt pool, which causes a shift in the cholesterol saturation index. Chronic total parenteral nutrition (TPN) also increases risk of cholelithiasis.
    - **Hyperoxaluria and nephrolithiasis**. Excessive fatty acids within the colonic lumen bind intraluminal calcium. Unbound oxalate, normally made insoluble by calcium binding and excreted in the feces, thus is absorbed readily, resulting in hyperoxaluria and calcium oxalate urinary stone formation.

- **Diarrhea and steatorrhea**. Rapid intestinal transit, presence of hyperosmolar enteric contents in the distal bowel, disruption of the enterohepatic bile acid circulation, and bacterial overgrowth all promote diarrhea and steatorrhea. Fat absorption is most severely impaired by ileal resection. The delivery of bile acids into the colon produces a reactive watery diarrhea that may be severe. Unabsorbed fats in the colon further inhibit absorption and stimulate secretion of water and electrolytes.
- **Intestinal microflora**. Loss of the ileocecal valve permits reflux of colonic bacteria into the small bowel. Intestinal dysmotility further promotes bacterial colonization. Bacterial overgrowth and changes in the indigenous microbial population result in pH alteration and deconjugation of bile salts, with resultant malabsorption, fluid loss, and decreased vitamin B_{12} absorption. Infectious diarrhea (bacterial or viral) is a major cause of morbidity.

Acute cases: Acutely, the primary goal is to stabilize the metabolic, respiratory, and cardiovascular parameters related to the fluid shift and sepsis that frequently accompany massive small-bowel resection.

- Deranged motility patterns and changes in intraluminal milieu may produce a prolonged ileus. Parenteral nutrition should be provided until GI function resumes. If ileus persists for an unduly prolonged period, mechanical obstruction or sepsis may be the cause.
- Gastric hypersecretion requires H_2-receptor antagonists or proton-pump inhibitors to reduce the hypersecretion response and protect against peptic ulceration. Antacids neutralize acid on contact and should be administered for nasogastric aspirate pH of less than 5.
- Nutritional support should be instituted early to maintain positive nitrogen balance and to promote wound healing and adaptation of the remaining bowel. Enteral nutrition has a positive trophic effect on the bowel mucosa and should be started as soon as possible. Feeding tubes placed at laparotomy can be very helpful. Even if caloric goals are not met, enteral formula stimulates the remaining intestine and facilitates adaptation. Feeds should initially be low volume, low fat, and isosmotic.

Chronic Treatment:

- Diarrhea has many causes in short-bowel syndrome. Frequently, dietary modification improves symptoms. H_2-receptor antagonists reduce acid production and the volume of enteric contents. Chelating resins, such as cholestyramine, reduce intraluminal bile salts and subsequent diarrhea but affect the available systemic bile salt pools. Antisecretory medications, such as loperamide and

somatostatin analog, may be beneficial. Low-dose oral narcotics, such as diphenoxylate hydrochloride and atropine (Lomotil) or codeine, are efficacious but addictive. Bacterial overgrowth should be evaluated by stool culture and prophylactic antimicrobials administered as needed.

- Nutritional support with supplemental vitamins, trace elements and minerals, and essential fatty acids should be given parenterally until adequate enteral absorption is established. The absorption of fat-soluble vitamins A, D, E, and K is especially likely to be compromised. Vitamin B_{12} and calcium absorption are also affected by altered fat absorption and should be supplemented. If required, chronic TPN can be administered nightly to permit normal daytime activities.

Late complications, mostly secondary to metabolic derangements, are common. Problems include nephrolithiasis, cholelithiasis, nutritional deficiency (e.g., anemia, bone disease, coagulopathy), liver dysfunction, TPN-related complications, and central venous catheter–related problems, for example, sepsis or thrombosis. Anastomotic leak, fistula, stricture, and obstruction can also occur well beyond the early postoperative period. Late obstruction (partial or complete) is fairly common, and reoperative rates are high.

Medical Therapy of Short Bowel Syndrome

Standard Management	
Clinical Issue	**Medical Therapy**
Hypergastrinemia/gastric hypersecretion	H_2 blocker or proton pump inhibitor
Diarrhea	First line of therapy: Loperamide, dyphenoxylate
	Second line of therapy: Codeine, DTO
	Avoid octreotide
Bile salt-induced diarrhea	Cholestyramine (if colon is in continuity and <100 cm ileum)
Electrolyte losses (e.g., K^+, Mg^{++}, Ca^{++})	Replete as needed (may need to give IV if increase in diarrhea with oral repletion)
Vitamin deficiency (e.g., A, D, E, K, B_{12})	Monitor and replete as necessary; may require chronic administration
	Liquid vitamins may be better absorbed than pill form
Mineral deficiency (e.g., zinc, selenium, iron)	Monitor and replete as necessary; may require chronic administration

	Liquid minerals (in a combined multivitamin/mineral preparation) may be better absorbed than pill form
Specialized Treatment	
Clinical Issue	**Medical Therapy**
Bacterial overgrowth	Metronidazole, tetracycline
Large electrolyte and fluid loss not controlled with standard management	Octreotide
Inadequate adaptive response and TPN dependence despite standard management	Consider growth hormone and glutamine
DTO, Deodorized tincture of opium; IV, intravenously; TPN, total parenteral nutrition	

Recommended Diet for Short Bowel Syndrome

Standard Management		
	Colon in Continuity	**No Colon in Continuity**
Protein	20–30% of caloric intake	20–30% of caloric intake
Fat	20–30% of caloric intake	30–40% of caloric intake
Carbohydrate	50–60% of caloric intake	30–40% of caloric intake
Fluid	Isotonic or hypoosmolar	Isotonic, high-sodium oral rehydration solution
Oxalate	Restrict intake	No restrictions
Specialized Treatment		
	Colon in Continuity	**No Colon in Continuity**
Soluble Fiber	5–10 g/day if stool output > 3L	5–10 g/day if stool output > 3L

Surgical therapy: Various surgical procedures have been described for the management of short-bowel syndrome, although they have not been widely adopted. Most important is the prevention of complications by minimizing the extent of initial bowel resection. Bowel transplantation is not widely performed but is an alternative in some patients, especially those with massive resection and virtually no remaining bowel.

A number of surgical strategies have been attempted in patients who are chronically TPN dependent with limited success; these include procedures to delay intestinal transit time, methods to increase absorptive area, and small bowel transplantation.

Methods to delay intestinal transit time include the construction of various valves and sphincters, with inconsistent results reported. Antiperistaltic segments of small intestine have been constructed to slow the transit, thus allowing additional contact time for nutrient and fluid absorption. Moderate successes have been described with this technique.

Other procedures, including colonic interposition, recirculating loops of small bowel, and retrograde electrical pacing, have been tried but were found to be unsuccessful in humans and were largely abandoned. Surgical procedures to increase absorptive area include the intestinal tapering and lengthening procedure originally described by Bianchi. This procedure improves intestinal function by correcting the dilation and ineffective peristalsis of the remaining intestine, as well as by doubling the intestinal length while preserving the mucosal surface area. Although beneficial in selected patients, potential complications can include necrosis of divided segments and anastomotic leaks.

Intestinal transplantation has improved with the introduction of the new immunosuppressive agent tacrolimus (FK506). Intestinal transplantation procedures have included primarily isolated small intestinal grafts and combined liver–small intestinal grafts with a few more extensive cluster grafts in a large series reported from the International Intestinal Transplant Registry.

An alternative to intestinal transplantation is mucosal stem cell transplantation, which involves transplanting enterocytes onto a biomatrix and achieving regeneration of intestinal mucosa. This procedure is, at best, preliminary but has shown some promise in experimental studies.

(d) Surgical Management of Ulcerative Colitis.

Ulcerative Colitis—Indications for Surgery
Intractability
Dysplasia-carcinoma
Massive colonic bleeding
Toxic megacolon

Fulminant Colitis and Toxic Megacolon

Patients with fulminant colitis typically present with high fever, severe abdominal pain, tenderness, tachycardia, and leukocytosis. These patients require hospitalization with intravenous hydration, nasogastric decompression, high-dose intravenous steroids if the patient is steroid dependent, and broad-spectrum antibiotics. Intravenous hyperalimentation may be useful depending on the patient's nutritional status and length of illness before the fulminant episode.

Patients should be closely monitored with serial abdominal exams and leukocyte counts. Deterioration or lack of improvement within 24 to 48 hours of the initiation of medical treatment warrants an urgent procedure because the mortality rate is increased four-fold in patients with colonic perforation.

Toxic megacolon is a serious, life-threatening condition that can occur in patients with ulcerative colitis, in which the bacterial infiltration of the walls of the colon creates a dilation of the colon that progresses to the point of imminent perforation. This decompensation results in a necrotic, thin-walled bowel in which pneumatosis can often be seen radiographically. Although some patients with toxic megacolon have been successfully treated medically, a high rate of recurrence with subsequent urgent operation has been reported.

Aggressive preoperative stabilization is required, using volume resuscitation with crystalloid solutions to prevent dehydration secondary to third-space fluid losses, stress-dose steroids for patients previously on steroid therapy, and broad-spectrum antibiotics.

Restorative proctocolectomy with ileal pouch–anal anastomosis (IPAA) as a single-staged procedure has been reported for toxic megacolon, proctectomy and anastomosis are generally not done in the acutely ill patient with an unprepared bowel. Total proctocolectomy in the urgent setting carries a prohibitively high mortality rate, and the leak rate from a primary anastomosis is unacceptably high. Whereas the goal in elective surgery is to remove all the colonic or dysplastic mucosa, the aim in emergent surgery is to rescue the patient from a life-threatening situation.

A total abdominal colectomy with ileostomy and preservation of the rectum is therefore the preferred operation for this condition. This procedure can be expeditiously performed with relatively low morbidity and mortality, and it serves the main purpose of removing the diseased colon and avoiding a difficult and morbid pelvic dissection.

Preserving the rectum leaves the option of fashioning an ileorectal anastomosis in the future. This is particularly important in patients in whom the diagnosis is unclear and a subsequent ileoanal pouch might be contraindicated (e.g., in Crohn's disease).

Currently, it is distinctly unusual that operative intervention is delayed to such a degree, but the operation may be advantageous in such dire circumstances. The technique consists of performing a skin-level (blow-hole) transverse colostomy (and sometimes the left colon is decompressed with a simultaneous, second sigmoid skin level colostomy). Fashioning a loop ileostomy is an essential component of this operation. The operation was often rewarded with dramatic improvement in the patient's condition. After recovery from the acute illness (usually over a period of several months), the patient could then be treated with restorative proctocolectomy with IPAA, or total proctocolectomy, depending on the circumstances.

Massive Bleeding

Massive hemorrhage from ulcerative colitis is an uncommon event, occurring in less than 5% of patients requiring operation. Patients obviously require resuscitation and stabilization before surgery, with replenishment of extracellular volume and transfusions as needed. Subtotal colectomy is the procedure of choice and will usually suffice. However, if bleeding continues from the remaining rectal mucosa, emergency proctectomy may be required.

Intractability

Colitis with debilitating symptoms refractory to medical therapy is the most common indication for operative therapy. It has been demonstrated that patients' quality of life after surgery for ulcerative colitis is improved, regardless of the procedure performed.

Complications of long-term steroid therapy, such as diabetes mellitus, avascular necrosis of the femoral head, cataracts, psychiatric problems, osteoporosis, and weight gain, are a frequent indication for surgical resection, even though the patient's symptoms may be controlled while on steroids.

Elective surgery should also be considered in patients with significant extracolonic manifestations refractory to nonoperative measures.

Dysplasia or Carcinoma

The finding of dysplastic changes in the colon or carcinoma is an indication for surgical intervention as discussed previously. The presence of cancer may influence the

procedure selected or the sequence of staged procedures. It does not exclude the possibility of performing an ileoanal pouch, but the location and stage of the cancer must be taken into consideration.

Operations

Elective surgical options for ulcerative colitis include total proctocolectomy with ileostomy, restorative proctocolectomy with IPAA, and total proctocolectomy with a continent ileal reservoir (Kock pouch). Segmental colectomy for ulcerative colitis (in contrast to Crohn's disease) has been shown to be an inadequate procedure for controlling disease. For example, in the case of colitis confined to the left side, a proctosigmoidectomy with end descending colostomy or coloanal anastomosis invariably results in the recurrence of disease within the remaining colon within a short time and is contraindicated.

Total Proctocolectomy with End Ileostomy:

Total proctocolectomy has the advantage of removing all diseased mucosa, thereby preventing further inflammation or the potential for progression to dysplasia or carcinoma. The major disadvantage of this procedure is the need for a permanent ileostomy. In addition, despite improvements in bowel preparation, antibiotics, and surgical technique, total proctocolectomy still has a fairly high morbidity rate. Most of the morbidity is related to perineal wound healing, adhesions, the ileostomy, and complications of pelvic dissection. Perineal wound problems may be reduced if an intersphincteric proctectomy is performed. This approach involves a dissection between the internal and external sphincters, preserving the external sphincter and levator ani for a more secure perineal wound closure.

Total proctocolectomy with end ileostomy was one of the earliest operations performed for ulcerative colitis, and despite advances in sphincter-saving procedures, continues to have a role. Elderly patients, those with poor sphincter function, and patients with carcinomas in the distal rectum may be candidates for this procedure. All patients should be marked for an ileostomy preoperatively in the sitting and standing positions. The preferred site of the stoma is within the body of the rectus abdominis muscle at the summit of the infraumbilical fat mound on the right side, away from bony prominences, the umbilicus, and the midline incision.

Total Proctocolectomy with Continent Ileostomy

The continent ileostomy was introduced by Kock in 1969 and became popular in the 1970s because it offered control of evacuations for patients with an ileostomy. A single-

chambered reservoir is fashioned by suturing several limbs of ileum together after the antimesenteric border has been divided. The outflow tract is intussuscepted into the reservoir to create a valve that provides obstruction to the pouch contents (continence). As the pouch distends, pressure over the valve causes it close and retain stool, permitting patients to wear a simple bandage over a skin-level stoma. Between two and four times per day, the patient introduces a tube through the valve to evacuate the pouch.

The major problem with the Kock pouch is the high complication rate necessitating reoperation in up to half of patients. The most common problem is a slipped valve, which occurs when the intussuscepted limb everts, and the "continent" nipple is lost. This leads to either the inability of the pouch to remain continent or the inability to intubate the pouch, leading to spontaneous emptying of the pouch as it overflows. Revision of the nipple valve corrects this problem. Other complications include inflammation of the ileal pouch mucosa ("pouchitis") in 15% to 30% of cases, fistula formation (10%), and stoma stricture (10%).

Since the introduction of restorative proctocolectomy and IPAA, the popularity of the continent ileostomy has declined, and it is seldom used. High complication and reoperation rates have dampened enthusiasm among surgeons for the technique. Although ulcerative colitis patients in whom IPAA is contraindicated may be candidates, realistically, only a very few centers presently offer the operation.

Currently, the most common surgery related to continent ileostomies is revisional surgery. The Kock procedure should not be performed in obese patients, debilitated patients, or any patient with physical or mental handicap that would prohibit safe catheterization of the reservoir. The procedure is contraindicated in patients with Crohn's disease because of the high incidence of recurrence of Crohn's causing failure of the pouch.

Total Proctocolectomy with Ileal Pouch– Anal Anastomosis:

Restorative proctocolectomy with IPAA has become the most common definitive operation for the surgical treatment of ulcerative colitis. The procedure involves a near-total proctocolectomy with preservation of the anal sphincter complex. A single-chambered pouch is fashioned from the distal 30 cm of the ileum and sutured to the anus using a double-stapled technique . Alternatively, a hand-sewn anastomosis may be fashioned between the pouch and the anus after stripping the distal rectal mucosa from the internal anal sphincter (mucosectomy).

The double-stapling technique may leave a small remnant of rectal mucosa at the anastomosis, which, in theory, is at risk for the development of dysplasia and cancer. Mucosectomy has, however, been complicated by cancer arising at the anastomosis and extraluminally in the pelvis, evidently from islands of glands that remained after the mucosa was incompletely removed. Although cancer is exceedingly rare, the mucosectomy technique may conceal retained rectal mucosa in more than 20% of patients.

The double-stapling technique permits surveillance and biopsy of the remaining mucosa. Avoiding the mucosectomy preserves the anal transition zone, which contains nerve endings involved in differentiating liquid and solid stool from gas, and is thus thought to provide superior postoperative continence.

Controversy also exists regarding temporary fecal diversion. The pouch and anastomosis were traditionally protected with a diverting loop ileostomy; however, there are some proponents of the single-stage procedure without diversion. This approach has the advantage of a single operation that avoids the complications that accompany an ileostomy. Disadvantages, however, include an increased risk for pelvic sepsis caused, usually, by anastomotic leak of a pouch suture line or the anal anastomosis. Most surgeons routinely perform a two-stage operation in high-risk patients, particularly those patients taking steroids preoperatively.

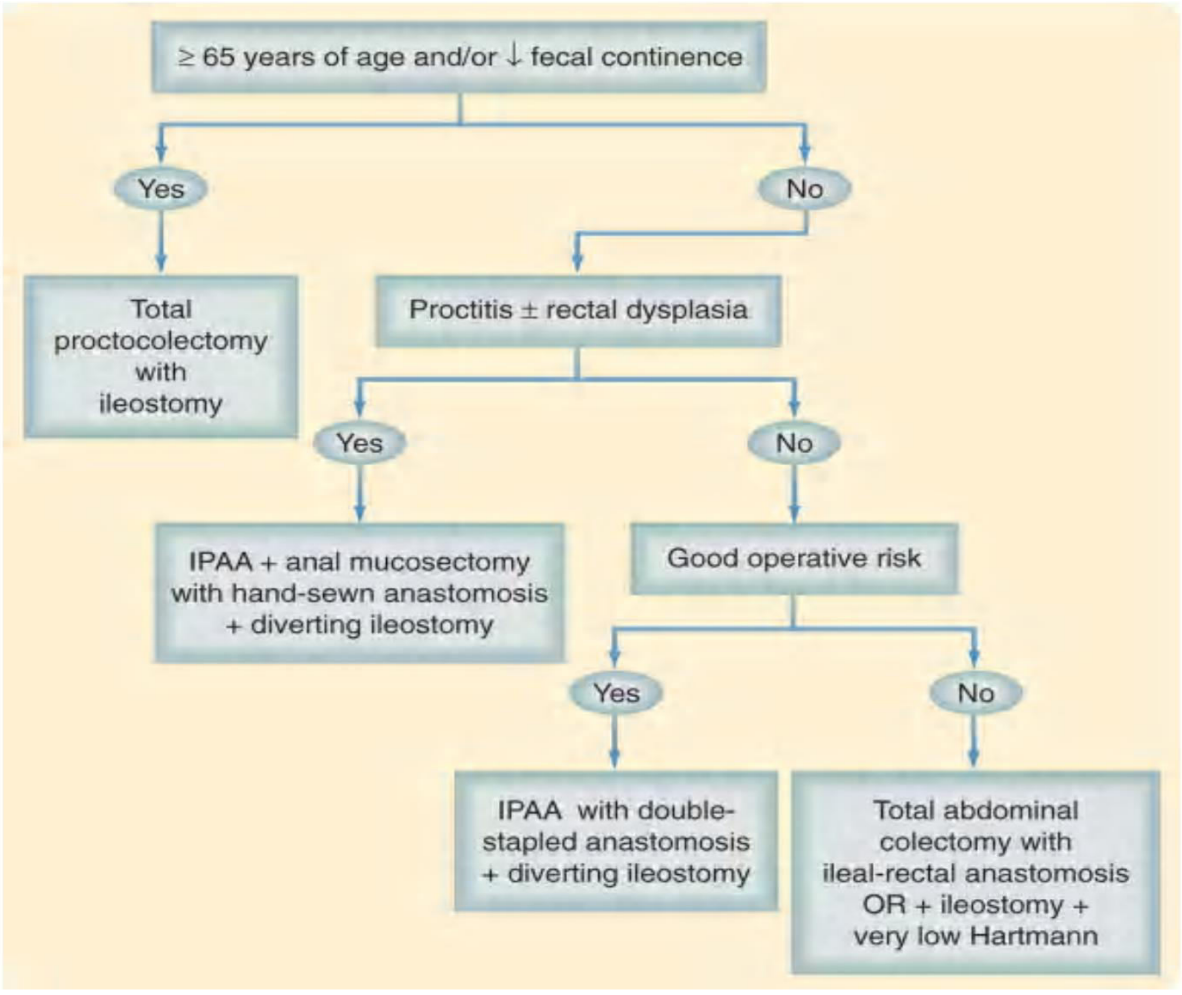
Patients who undergo total proctocolectomy and IPAA typically have between five and seven bowel movements in a 24-hour period. Function continues to improve with time, with numerous studies demonstrating a decrease in the number of daily bowel movements during the ensuing 3 to 24 months after reestablishment of continuity.

Restorative proctocolectomy and IPAA are associated with both early and late complications. A common complication is small bowel obstruction. Bowel obstruction after IPAA tends to be severe and requires surgery in almost half of cases. Another significant complication is pelvic sepsis.

Anastomotic and pouch suture line leaks are devastating complications that can lead to pelvic abscess and seriously threaten the integrity and functionality of the pouch. Treatment of pelvic sepsis secondary to pouch leaks usually requires a diverting ileostomy and drainage of any abscesses. Delayed ileostomy closure after resolution of IPAA complications has no deleterious functional effects. A pouch-vaginal fistula is a specific form of pelvic sepsis that is difficult to manage and occurs in up to 7% of women.

Inflammation of the mucosa of the ileal pouch, or *pouchitis*, occurs in 7% to 33% of patients with ulcerative colitis treated by IPAA. Pouchitis typically presents with increased stool frequency, fever, bleeding, cramps, and dehydration. The cause is unknown but may be related to bacterial overgrowth, mucosal ischemia, or other local factors. Episodes usually respond to rehydration and oral antibiotics (usually metronidazole or ciprofloxacin). Probiotics have recently been reported to provide dramatic resolution in some cases of pouchitis resistant to antibiotic therapy. The diagnosis of Crohn's disease must also be entertained in patients with significant pouchitis that does not respond to medical treatment.

A suggested algorithm for elective operations for patients with intractable mucosal ulcerative colitis is presented below:



Postoperative Care:

- Postoperative care after restorative proctocolectomy with IPAA is similar to other major colorectal procedures. Nasogastric tubes are usually removed at the completion of the procedure, and liquid diets are offered to patients in the early recovery period. Diet is advanced with return of bowel function as evidenced by ileostomy function.
- If a pelvic drain is used, it is typically removed after 48 to 72 hours.
- Bladder catheters are typically left in place for 3 to 4 days depending on the difficulty of pelvic dissection.
- A water-soluble contrast enema is performed about 10 weeks postoperatively to ensure an intact IPAA. If the enema shows a leak, the contrast examination is repeated in 6 weeks; close to 95% of anastomotic leaks heal in the absence of pelvic sepsis. If the radiograph shows no leak, the diverting ileostomy is closed.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES

MS (General Surgery) Examination, 2009

PAPER II

Time Allowed: 3 Hours **Full Marks: 100**

1. Enumerate the causes of upper gastrointestinal tract bleeding. How do you evaluate such a patient coming to the emergency? Outline the management of a case due to portal hypertension. 5+5+10

2. What are the causes of dysphagia? How do you propose to investigate a patient with this problem? Briefly discuss the therapeutic options of a case of squamous cell carcinoma of the oesophagous. 5+8+7

3. Write short notes of the following: 5 x 6

(a) Carcinoid syndrome:

(b) Breast cancer in pregnancy:

(c) Fournier's gangrene:

(d) Sterilization of surgical instruments:

(e) Meckel's diverticulum.

4. Write brief answers: 4 x $7^1/_2$

(a) Pathophysiology of acute pancreatitis .

(b) Chronic limb ischaemia:.

(c) Medullary carcinoma of thyroid:

(d) Polyposis coli.

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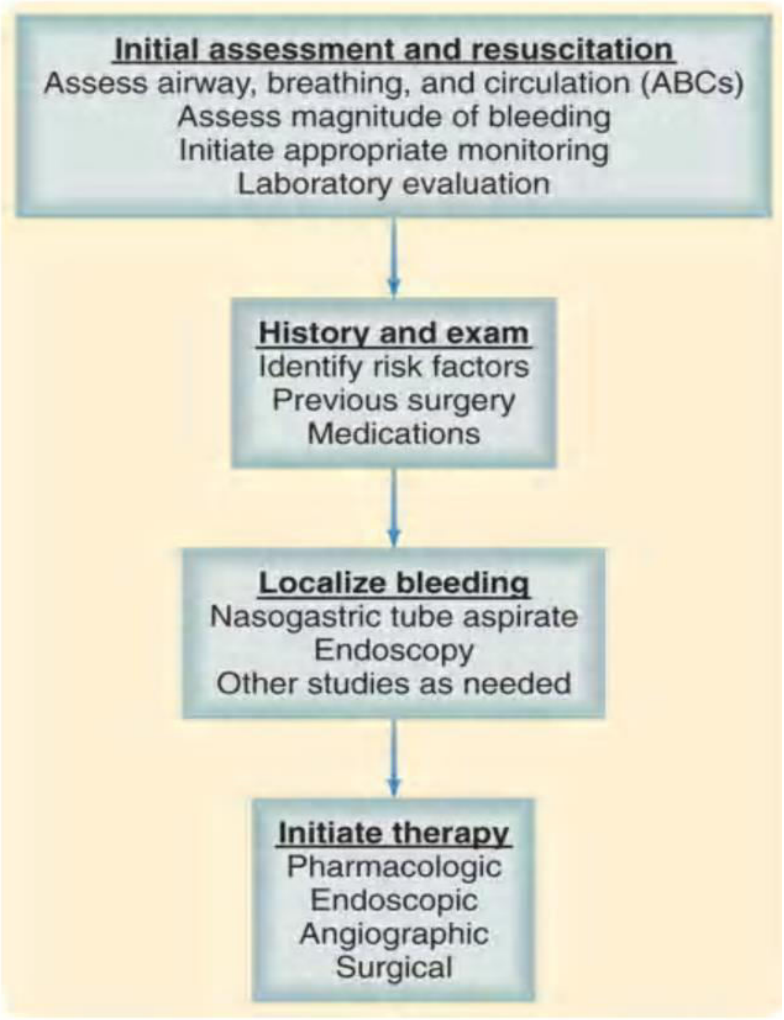
Answer. Common Causes of Upper Gastrointestinal Hemorrhage:

NONVARICEAL BLEEDING	**(80%)**	**PORTAL HYPERTENSIVE BLEEDING**	**(20%)**
Peptic ulcer disease	30-50%	Gastroesophageal varices	>90%
Mallory-Weiss tears	15-20%	Hypertensive portal gastropathy	<5%
Gastritis or duodenitis	10-15%	Isolated gastric varices	Rare
Esophagitis	5-10%		
Arteriovenous malformations	5%		
Tumors	2%		
Other	5%		

General approach to the patient with acute GI hemorrhage:

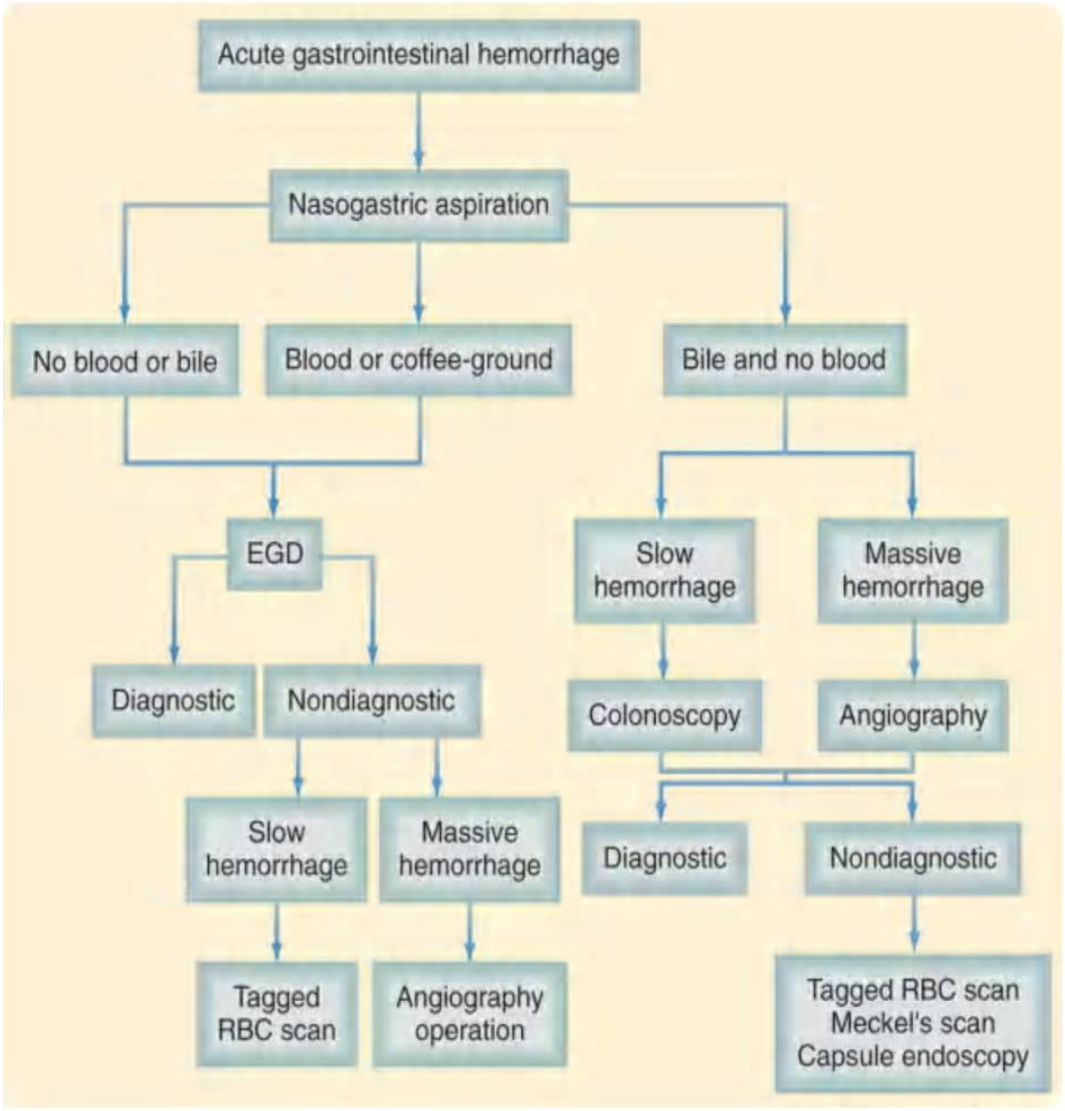
INITIAL MANAGEMENT:



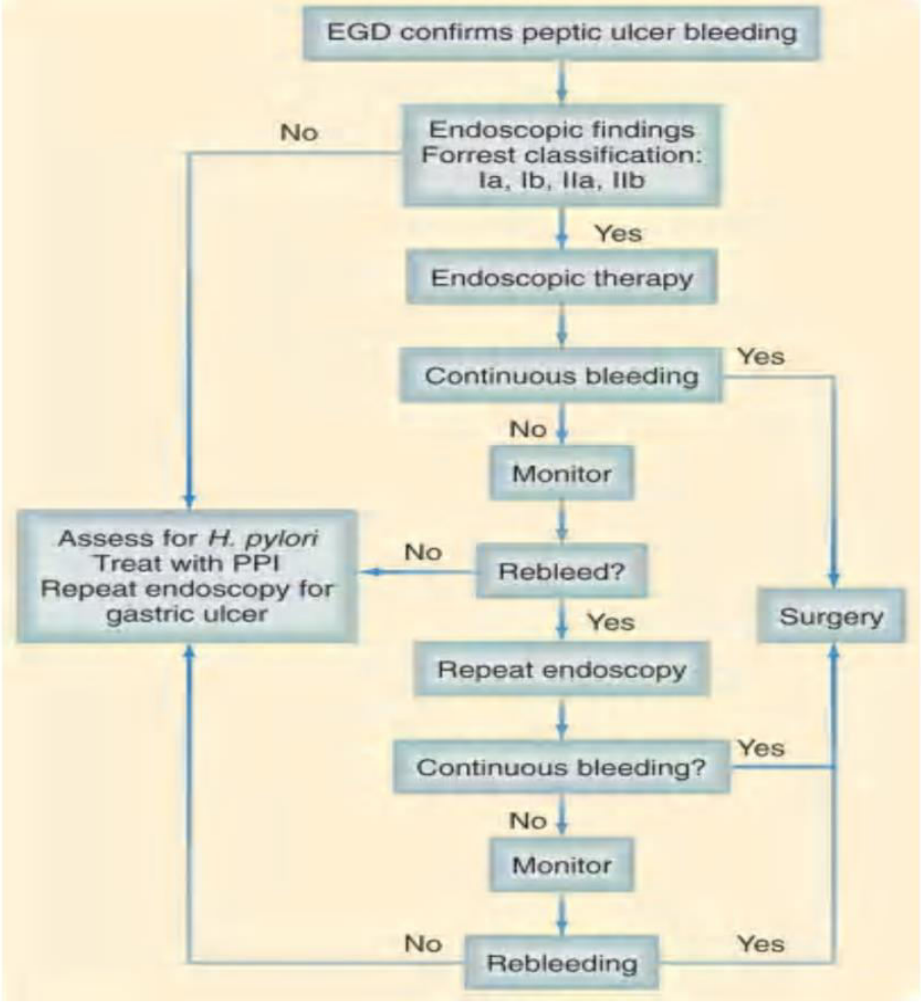
Risk Factors for Morbidity and Mortality in Acute Gastrointestinal Hemorrhage:

Age >60 yr	Comorbid disease: Renal failure Liver disease Respiratory insufficiency Cardiac disease	Magnitude of hemorrhage: Systolic blood pressure <100 mm Hg on presentation	Transfusion requirement: -Persistent or recurrent hemorrhage -Onset of hemorrhage during hospitalization -Need for surgery

ALGORITHM OF MANAGEMENT AFTER INITIAL RESUSCITATION:



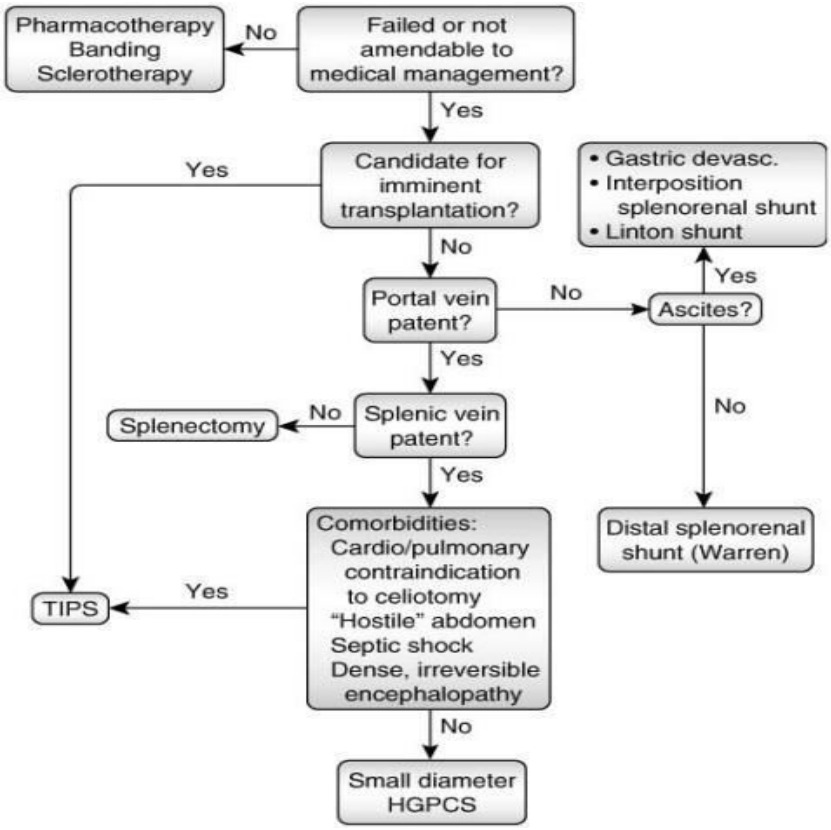
EGD, esophagogastroduodenoscopy; RBC, red blood cell.



Algorithm for the diagnosis and management of nonvariceal upper GI bleeding. EGD, esophagogastroduodenoscopy; PPI, proton pump inhibitor.

Indications for Surgery in Gastrointestinal Hemorrhage:

- Hemodynamic instability despite vigorous resuscitation (>6 units transfusion)
- Failure of endoscopic techniques to arrest hemorrhage
- Recurrent hemorrhage after initial stabilization (with up to two attempts at obtaining endoscopic hemostasis)
- Shock associated with recurrent hemorrhage
- Continued slow bleeding with a transfusion requirement exceeding 3 units/day.



2. What are the causes of dysphagia? How do you propose to investigate a patient with this problem? Briefly discuss the therapeutic options of a case of squamous cell carcinoma of the oesophagous. 5+8+7

Answer. Causes of dysphagia are:

Onset in younger age	Onset in middle age	Onset in late age
1	2	3

Oesophageal web 2 Oesophageal ring 1,2	Achalasia	Myopathy2,3	Multiple sclerosis2,3 Systemic sclerosis2
Cardiomegaly 3	Corrosive Esophagitis 1,2	Medications1,2,3	Radiation therapy2,3
Diverticulum 3 (zenker's – direct compression)	Cerebellar stroke 3	Upper oesophageal sphincter dysfunction3	Candida esophagitis2,3
Peptic stricture1,2,3	Brainstem stroke 3 Bulbar and pseudobulbar palsy	Myasthenia gravis 1,2	Amyotrophic lateral sclerosis2

Oesophageal cancer 3	Mass brain lesion2,3	Peptic esophagitis1,2,3	External esophageal compression1,2,3
Diffuse oesophageal spasm2	Esophageal obstruction by a foreign body2,3	Lead poisoning2,3	Rabies2,3 Tetanus1,2,3
Plummer-Vinson syndrome2	Extrinsic laryngeal carcinoma3	Oral cavity tumor3	Systemic lupus erythematous2

Investigations:

- The 1st investigation is upper GI endoscopy with biopsy from the lesion.
- Chest X-ray: to exclude bronchial carcinoma.
- Barium swallow and meal: to look at mucosal lining and detect achalasia.
- Esophageal manometry is indicated in various clinical situations. Manometry is indicated in the evaluation of patients with dysphagia after a structural obstruction has been excluded.
- Endoluminal ultrasound scan to assess depth of invasionin case of carcinoma oesophagous.
- CT scanning to evaluate local invasion, locoregional lymphadenopathy.
- The gold-standard for diagnosing oropharyngeal dysphagia is Modified Barium Swallow Study or Videofluoroscopic Swallow Study (Fluoroscopy). This is a lateral video X-ray that provides objective information on bolus transport, safest consistency of bolus (honey, nectar, thin, pudding, puree, regular), and possible head positioning and/or maneuvers that may facilitate swallow function depending on each individual's anatomy and physiology.

Therapeutic options for squamous cell carcinoma oesophagous

American Joint Committee on Cancer (AJCC) TNM Classification for Esophageal Carcinoma

T0	No evidence of primary tumor
T1	Invades lamina propria or submucosa
T2	Invades muscularis propria
T3	Invades adventitia
T4	Invades adjacent structures
N0	No regional lymph node metastasis
N1	Regional lymph node metastasis
M0	No distant metastasis

M1	Distant metastasis	
M1a	Invasion of celiac or cervical lymph nodes	
M1b	Other distant metastasis	
Stage 0	Tis N0 M0	
Stage I	T1 N0 M0	
Stage IIA	T2 N0 M0	T3 N0 M0
Stage IIB	T1 N1 M0	T2 N1 M0
Stage III	T3 N1 M0	T4 Any N M0
Stage IV	Any T Any N M1	

Treatment of esophageal carcinoma:

Management

General approaches

- The treatment is determined by the cellular type of cancer (adenocarcinoma or squamous cell carcinoma vs other types), the stage of the disease, the general condition of the patient and other diseases present. Adequate nutrition needs to be assured, and adequate dental care is vital.
- If the patient cannot swallow at all, a stent may be inserted to keep the esophagus patent; stents may also assist in occluding fistulas. Anasogastric tube may be necessary to continue feeding while treatment for the tumor is given, and some patients require a gastrostomy. The latter two are especially important if the patient tends to aspirate food or saliva into the airways, predisposing for aspiration pneumonia.
- Esophagectomy: It is the removal of a segment of the esophagus; as this shortens the length of the remaining esophagus, some other segment of the digestive tract (typically the stomach or part of the Colon or jejunum]) is pulled up to the chest cavity and interposed. If the tumor is unresectable or the patient is not fit for surgery, palliative esophageal stenting can allow the patient to tolerate soft diet.

Types of esophagectomy:

- Thoracoabdominal approach- which opens the abdominal and thoracic cavities together.
- Two stage Ivor Lewis (also called Lewis-Tanner) approach- with an initial laparotomy and construction of a gastric tube, followed by a right thoracotomy to excise the tumor and create an esophagogastric anastomosis.
- Three stage McKeown approach- where a third incision in the neck is made to complete the cervical anastomosis.

Endoscopic Therapy for Localized Disease: The candidates for endoscopic therapy are Stage 1 patients with tumors invading into the lamina propria (T1 mucosal) or submucosa (T1 submucosal) that do not have regional or distant metastasis.

Patients with carcinoma in-situ or high-grade dysplasia can also be treated with endoscopic therapy.

Submucosal cancers with increased risk of nodal metastases may not be as amenable to curative therapy.

- The two forms of endoscopic therapy that have been used for Stage 0 and I disease are endoscopic mucosal resection (EMR) and mucosal ablation using photodynamic therapy, Nd-YAG laser, or argon plasma coagulation.
- EMR Endoscopic Mucosal Resection has been advocated for early cancers. The prognosis after treatment with endoscopic mucosal resection is comparable to surgical resection. This technique can be attempted in patients, without evidence of nodal or distant metastases, with differentiated tumors that are slightly raised and less than 2 cm in diameter, or in differentiated tumors that are ulcerated and less than 1 cm in diameter. The most commonly employed modalities of endoscopic mucosal resection include strip biopsy, double-snare polypectomy, resection with combined use of highly concentrated saline and epinephrine, and resection using a cap.

The strip biopsy method for endoscopic mucosal resection of esophageal cancer is performed with a double-channel endoscope equipped with grasping forceps and snare.

The endoscopic double-snare polypectomy method is indicated for protruding lesions. Using a double-channel scope, the lesion is grasped and lifted by the first snare and strangulated with the second snare for complete resection.

Endoscopic resection with injection of concentrated saline and epinephrine is carried out using a double-channel scope. The lesion borders are marked with a coagulator. Highly concentrated saline and epinephrine are injected (15–20 ml) into the submucosal layer to swell the area containing the lesion and elucidate the markings. The mucosa outside the demarcated border is excised using a high-frequency scalpel to the depth of the submucosal layer. The resected mucosa is lifted and grasped with forceps, trapping and strangulating the lesion with a snare, and then resected by electrocautery.

A fourth method of endoscopic mucosal resection employs the use of a clear cap and prelooped snare inside the cap.

The major complications of endoscopic mucosal resection include postoperative bleeding and perforation and stricture formation.

- Laser therapy is the use of high-intensity light to destroy tumor cells; it affects only the treated area. This is typically done if the cancer cannot be removed by surgery. The relief of a blockage can help to reduce dysphagia and pain. Photodynamic therapy (PDT), a type of laser therapy, involves the use of drugs that are absorbed by cancer cells; when exposed to a special light, the drugs become active and destroy the cancer cells.
- Chemotherapy depends on the tumor type, but tends to be cisplatin-based (or carboplatin or oxaliplatin) every three weeks with fluorouracil (5-FU) either continuously or every three weeks. In more recent studies, addition of epirubicin (ECF) was better than other comparable regimens in advanced nonresectable cancer. Chemotherapy may be given after surgery (adjuvant, i.e. to reduce risk of recurrence), before surgery (neoadjuvant) or if surgery is not possible; in this case, cisplatin and 5-FU are used.
- Radiotherapy is given before, during or after chemotherapy or surgery, and sometimes on its own to control symptoms. In patients with localised disease but contraindications to surgery, "radical radiotherapy" may be used with curative intent.

Follow-up:

Patients are followed up frequently after a treatment regimen has been completed. Frequently, other treatments are necessary to improve symptoms and maximize nutrition.

Prognosis:

In general, the prognosis of esophageal cancer is quite poor, because so many patients present with advanced disease: The overall five-year survival rate (5YSR) is less than 5%. Individualized prognosis depends largely on stage. Those with cancer restricted entirely to the esophageal mucosa have about an 80% 5YSR, but submucosal involvement brings this down to less than 50%. Extension into the muscularis propria (muscular layer of the esophageus) has meant a 20% 5YSR and extension to the structures adjacent to the esophagus results in a 7% 5YSR. Patients with distant metastases (who are not candidates for curative surgery) have a less than 3% 5YSR.

3. Write short notes of the following: 5 x 6

(a) Carcinoid syndrome:
(b) Breast cancer in pregnancy:
(c) Fournier's gangrene:
(d) Sterilization of surgical instruments:
(e) Meckel's diverticulum.

Answer.

(a) Carcinoid syndrome:

Carcinoid syndrome refers to the array of symptoms that occur secondary to carcinoid tumors.

- Carcinoid tumors are discrete, yellow, well-circumscribed tumors that can occur anywhere along the gastrointestinal tract and in the lung. They most commonly affect the appendix, ileum, and rectum. Carcinoids are tumors of neuroendocrine nature, that originate in the cells of the neuroendocrine system and are characterized by production of serotonin (5-hydroxytryptamine; 5-HT). Although quite rare with 15 cases/1,000,000 population, carcinoid tumors account for 75% of gastrointestinal endocrine tumors.

Clinical presentation:

- The carcinoid syndrome occurs in approximately 10% of carcinoid tumors and becomes manifest when vasoactive substances from the tumors enter the systemic circulation escaping hepatic degradation. This is the case when carcinoid tumors metastasize to the liver or they arise for example in the bronchus.
- The most important clinical finding is flushing of the skin, usually of the head and the upper part of thorax. Secretory diarrhea and abdominal cramps are also characteristic features of the syndrome. When the diarrhea is intensive it may lead to electrolyte disturbance and dehydration.
- Other associated symptoms are nausea, and vomiting. Bronchoconstriction, which may be histamine-induced, affects a smaller number of patients and often accompanies flushing.
- About 50% of patients have cardiac abonormalities, caused by serotonin-induced fibrosis of the tricuspid and pulmonary valves.

- Elevated levels of circulating serotonin have been associated with cardiac failure, due to fibrous deposits on the endocardium. These deposits are thought to be responsible for the fibrous degeneration of the valve apparatus. "TIPS" is an acronym for **T**ricuspid **I**nsufficiency, **P**ulmonary **S**tenosis (fibrosis of tricuspid and pulmonary valves).
- Abdominal pain is due to desmoplastic reaction of the mesentery or hepatic metastases.

NOTE: Although the most common site of a carcinoid tumor is Appendix/Terminal ileum, carcinoid syndrome will only occur once metastasized from the gastrointestinal tract to the liver because the serotonin created by the carcinoid tumor released into the blood would only get broken down once it travels from the gastrointestinal tract directly to the liver through the hepatic portal system. However: If the tumor is bronchogenic in origin, then metastasis does not need to occur in order for carcinoid syndrome to occur.

Diagnosis:

- With a certain degree of clinical suspicion, diagnosis is made primarily by measuring plasma levels of the secreted glycoprotein Chromogranin A, supported by measuring the 24 hour urine levels of 5-HIAA (5-hydroxyindoleacetic acid), a breakdown product of serotonin.
- Patients with carcinoid syndrome usually excrete >25 mg of 5-HIAA per day. For localization of both primary lesions and metastasis, the initial imaging method is Octreoscan, where ^{111}Indium labelled somatostatin analogues (octreotide) are used in scintigraphy for detecting tumors expressing somatostatin receptors.
- Median detection rates with octreoscan are about 89%, in contrast to other imaging techniques such as CT scan and MRI with detection rates of about 80%.
- Usually on CT scan, one will note a spider-like/crab like change in the mesentery due to the fibrosis from the release of serotonin. PET scans, which evaluate for increased metabolism of glucose, may also aid in localizing the carcinoid lesion or evaluating for metastases.

Localization of tumour:

Tumour localization may be extremely difficult. Barium swallow and follow-up examination of the intestine may occasionally show the tumour. Capsule video endoscopy has recently been used to localize the tumour. Often laparotomy is the definitive way to localize the tumour.

Treatment:

For symptomatic relief of carcinoid sydrome:

- Octreotide (a somatostatin analogue that neutralizes serotonin and decreases urinary 5-HIAA)
- Methysergide maleate (antiserotonin agent but not used because of serious side effect of retroperitoneal fibrosis)
- Cyproheptadine (an antihistamine drug)

Alternative treatment for qualifying candidates:

- Surgical resection of tumor and chemotherapy (5-FU and doxorubicin)

Prognosis:

- Prognosis varies from individual to individual. It ranges from a 95% 5 year survival for localized disease to an 80%
- 5 year survival for those with liver metastases. The average survival time from the start of octreotide treatment has increased to about 12 years.

(b) Breast cancer in pregnancy:

Incidence: Breast cancer occurs in 1 of every 3000 pregnant women and axillary lymph node metastases are present in up to 75% of these women.

Age: The average age of the pregnant woman with breast cancer is 34 years.

Diagnosis: Less than 25% of the breast nodules developing during pregnancy and lactation will be cancerous.

Ultrasonography and needle biopsy are used in the diagnosis of these nodules.

Open biopsy may be required.

Mammography is rarely indicated because of its decreased sensitivity during pregnancy and lactation and because of the risk of radiation injury to the fetus.

Once a breast cancer is diagnosed, CBC, chest x-ray (with shielding of the abdomen), and liver function studies are performed.

Treatment: Because of the deleterious effects of radiation therapy on the fetus, a modified radical mastectomy is the surgical procedure of choice during the first and second trimesters of pregnancy, even though there is an increased risk of spontaneous abortion following first trimester anesthesia.

During the third trimester, lumpectomy with axillary node dissection is considered if adjuvant radiation therapy is deferred until after delivery.

Lactation is suppressed.

Chemotherapy administered during the first trimester carries a risk of spontaneous abortion and a 12% risk of birth defects.

There is no evidence of teratogenicity resulting from administration of chemotherapeutic agents in the second and third trimesters.

Prognosis: Pregnant women with breast cancer present at a later stage of disease because breast tissue changes that occur in the hormone-rich environment of pregnancy obscure early cancers. However, pregnant women with breast cancer have a prognosis, stage by stage, that is similar to that of nonpregnant women with breast cancer.

(c) Fournier's gangrene:

Introduction: Fournier's gangrene, sometimes called Fournier's disease, is a bacterial infection of the skin that affects the genitals and perineum (i.e., area between the scrotum and anus in men and between the vulva and anus in women). The disease develops after a wound or abrasion becomes infected.

Pathophysiology: A combination of anaerobic (living without oxygen) microorganisms (e.g., staphylococcal) and fungi (e.g., yeast) causes an infection that spreads quickly and causes destruction (necrosis) of skin, tissue under the skin (subcutaneous tissue), and muscle. Staphylococcal bacteria clot the blood, depriving surrounding tissue of oxygen. The anaerobic bacteria thrive in this oxygen-depleted environment and produce molecules that instigate chemical reactions (enzymes) that further the spread of the infection. Fournier's gangrene can be fatal if the infection enters the bloodstream.

Incidence and Prevalence:
Men are ten times more likely than women to develop Fournier's gangrene. Men aged 60-80 with a predisposing condition are most susceptible.

Women who have had a pus-producing bacterial infection (abscess) in the vaginal area, a surgical incision in the vagina and perineum to prevent tearing of skin during delivery of a child (episiotomy), an abortion resulting in fever and an infection of the lining of the uterus (septic abortion), or surgical removal of the uterus (hysterectomy) are susceptible.

Rarely, children may develop Fournier's gangrene as a complication from a burn, circumcision, or an insect bite.

Risk Factors:

Men with alcoholism, diabetes mellitus, leukemia, morbid obesity, and immune system disorders (e.g., HIV, Crohn's disease), and intravenous drug users are at increased risk for developing Fournier's gangrene. The condition also can develop as a complication of surgery.

Causes:

Fournier's gangrene develops when bacteria infect the body through a wound, usually in the perineum, the tube that carries urine outside the body from the bladder (urethra), or the colorectal area. Existing immune system deficiencies help infection to spread quickly, producing a disease that destroys the skin and superficial and deep fascia (membranes that separate muscles and protect nerves and vessels) of the genital area. The chambers in the penis that fill with blood to create an erection (corpora cavernosa), testicles, and urethra are not usually affected.

Signs and Symptoms:

Early physical symptoms of Fournier's gangrene may not indicate the severity of the condition. Pain sometimes diminishes as the disease progresses. Symptoms are progressive and include the following:

- Crepitant ("spongy" to the touch) skin
- Dead and discolored (gray-black) tissue; pus weeping from injury
- Fever and drowsiness (lethargy)
- Increasing genital pain and redness (erythema)
- Odor
- Severe genital pain accompanied by tenderness and swelling of the penis and scrotum

Diagnosis:

Physical examination and blood tests are used to diagnose Fournier's gangrene and the diagnosis is made when examination reveals gangrenous (i.e., spongy, weeping, discolored) skin. Microscopic examination of a tissue specimen (biopsy) may be taken if visible symptoms are insufficient to distinguish between Fournier's and other bacterial infections.

Treatment:

Antibiotics (often double or triple drug therapy) along with aggressive surgical removal of the diseased tissue is required immediately for an optimal outcome.

Without early treatment, bacterial infection enters the bloodstream and can cause delirium, heart attack, respiratory failure, and death.

Complications:
Incomplete debridement (surgical removal of dead tissue) allows wound infection to continue to spread. In this event, follow-up surgery is performed.

(d) Sterilization of surgical instruments:

Answer. Cleaning, Sterilization & Maintenance of Surgical Instruments
1. Rinsing
Immediately after surgery, rinse instruments under warm (not hot) running water. Rinse should remove all blood, body fluids and tissue.
2. Cleaning
(If not done immediately after rinsing, instruments should be submerged in a solution of water and neutral PH(7) detergent.)
A. Ultrasonic Cleaning
For micro and delicate instruments, use Manual cleaning (step C). Instruments should be processed in a cleaner for the full recommended cycle time – usually 5 to 10 minutes. Place instruments in open position into the ultrasonic cleaner. Make sure that "Sharp" (scissors, knives osteotomes, etc.) blades do not touch other instruments. All Instruments have to be fully submerged. Do not place dissimilar metals (stainless, copper, chrome plated, etc.) in the same cleaning cycle. Change solution frequently – at least as often as manufacturer recommends. Rinse instruments after ultrasonic cleaning with water to remove ultrasonic cleaning solution.
B. Automatic Washer Sterilizers
Follow manufacturers recommendations but make sure instruments are lubricated after last rinse cycle and before sterilization cycle.
C. Manual Cleaning

Most instrument manufacturers recommend ultrasonic cleaning as the best and most effective way to clean surgical instruments, particularly those with hinges, locks and other moving parts. If ultra sonic cleaning is not available observe the following steps.

I. Use stiff plastic cleaning brushes (nylon, etc.) Do not use steel wool or wire brushes except specially recommended stainless steel wire brushes for instruments such as bone files, or on stained areas in knurled handles.

II. Use only neutral PH(7) detergents because if not rinsed off properly, low PH detergents will cause breakdown of stainless protective surface and black staining. High PH detergent will cause surface deposit of brown stain, which will also interfere with smooth operation of the instrument.

III. Brush delicate instruments carefully and, if possible, handle them totally separate from general instruments.

IV. Make sure all instrument surfaces are visibly clean and free from stains and tissue. This is a good time to inspect each instrument for proper function and condition.

Check and make sure that :

Scissors blades glide smoothly all the way (they must not be loose when in closed position). Test scissors by cutting into thin gauze. Forceps (pickups) have properly aligned tips.

Hemostats and Needle Holders do not show light between the jaws, lock and unlock easily, joints are not too loose.

Check Needle Holders for wear on jaw surfaces.

Suction tubes are clean inside. Retractors function properly.Cutting instruments and knives have sharp, undamaged blades.

V. After scrubbing, rinse instruments thoroughly under running water. While rinsing, open and close Scissors, Hemostats, Needle Holders and other hinged instruments to make sure the hinge areas are rinsed out, as well as the outside of the instruments.

3. After cleaning

If instruments are to be stored, let them air dry and store them in a clean and dry environment.

4. Autoclaving

If instruments are to be reused or autoclaved:

A. Lubricate all instruments which have any "metal to metal" action such as scissors, hemostats, needle holders, self-retaining retractors, etc.Recommend surgical lubricants such as instrument milk are best. Do not use WD-40, oil or other industrial lubricants.

B. Put instruments up for autoclaving either individually or in sets.

Individual Instruments

Disposable paper or plastic pouches are ideal. Make sure you use a wide enough pouch (4" or wider) for instruments with

ratchet locks such as needle holders and hemostats so the instrument can be sterilized in an open (unlocked) position.

Instrument Sets

Unlock all instruments and sterilize them in an open position. Place heavy instruments on bottom of set (when two layers are

required). Never lock an instrument during autoclaving. It will not be sterile as steam cannot reach the metal to metal surfaces. The instrument will develop cracks in hinge areas because of heat expansion during the autoclave cycle.

Do not overload the autoclave chamber as pockets may form that do not permit steam penetration. Place towel on bottom of pan to absorb excess moisture during autoclaving. This will reduce the chances of getting "Wet packs". Make sure the towels used in sterilization of instruments have no detergent residue and are neutral – PH(7) if immersed in water. This can be a real problem as laundries frequently use inexpensive but high PH(9-13) detergents and do

not properly rinse out or neutralize those detergents in the final wash/rinse cycle. Also, some times bleaches such as Cloroxare added and are not neutralized.
Caution – At the end of the autoclavecycle – before the drying cycle – unlock autoclave door and open it more than a crack (about 3/4"). Then run dry cycle for the period recommended by the autoclave manufacturer. If the autoclave door is
opened fully before the drying cycle, cold room air will rush into the chamber, causing condensation on the instruments. This will result in water stains on instruments and also cause wet packs. If you have any unusual staining on your
instruments during sterilization, contact your local instrument representative.
5. Cold sterilization
Most cold sterilization solutions render instruments, sterile only after a 10 hour immersion. This prolonged chemical action can be more detrimental to the surgical instruments than the usual 20 minute autoclave cycle. If the instruments
need to be "disinfected" only, cold sterilization is okay as disinfection will place in only 10 minutes. But keep in mind the difference between:
Sterile – an absolute term (no living organism survives)and
Disinfected – basically clean. Always use the proper sterilization/cleaning technique to render the instrument in required condition for use. For instruments with Tungsten Carbide inserts (Needle Holders, Scissors, Tissue Forceps), we do not recommend use of solutions containing Benzyl Ammonium Chloride which will destroy the Tungsten Carbide Inserts.

(e) Meckel's diverticulum .

Answer.
Incidence and Etiology:

- Meckel's diverticulum is the most commonly encountered congenital anomaly of the small intestine, occurring in about 2% of the population.
- Meckel's diverticulum is located on the antimesenteric border of the ileum 45 to 60 cm proximal to the ileocecal valve.
- It results from incomplete closure of the omphalomesenteric or vitelline duct.
- An equal incidence is found among men and women.
- Meckel's diverticulum may exist in different forms, ranging from a small bump that may be easily missed to a long projection that communicates with the umbilicus by a persistent fibrous cord or, much less commonly, a patent fistula. The usual manifestation is a relatively wide-mouth diverticulum measuring about 5 cm in length, with a diameter of up to 2 cm .
- Cells lining the vitelline duct are pluripotent; therefore, it is not uncommon to find heterotopic tissue within the Meckel diverticulum, the most common of which is gastric mucosa (present in 50% of all Meckel's diverticula). Pancreatic mucosa is encountered in about 5% of diverticula; less commonly, these diverticula may harbor colonic mucosa.

Clinical Manifestations:

- Most Meckel's diverticula are entirely benign and are incidentally discovered during autopsy, laparotomy, or barium studies .
- The most common clinical presentation of Meckel's diverticulum is gastrointestinal bleeding, which occurs in 25% to 50% of patients who present with complications; hemorrhage is the most common symptomatic presentation in children aged 2 years or

younger. The usual source of the bleeding is a chronic acid-induced ulcer in the ileum adjacent to a Meckel's diverticulum that contains gastric mucosa.

- Diverticulum associated with a fibrotic band attached to the abdominal wall, intussusception, or, rarely, incarceration of the diverticulum in an inguinal hernia (Littre's hernia).
- Volvulus is usually an acute event and, if allowed to progress, may result in strangulation of the involved bowel.
- In intussusception, a broad-based diverticulum invaginates and then is carried forward by peristalsis. This may be ileoileal or ileocolic and present as acute obstruction associated with an urge to defecate, early vomiting, and, occasionally, the passage of the classic currant-jelly stool.
  A palpable mass may be present. Although reduction of an intussusception secondary to Meckel's diverticulum can sometimes be performed by barium enema, the patient should still undergo resection of the diverticulum to negate subsequent recurrence of the condition.
- Diverticulitis accounts for 10% to 20% of symptomatic presentations. This complication is more common in adult patients. Meckel's diverticulitis, which is clinically indistinguishable from appendicitis, should be considered in the differential diagnosis of a patient with right lower quadrant pain. Progression of the diverticulitis may lead to perforation and peritonitis.
- Finally, much rarer complications of Meckel's diverticula include neoplasms, with the most common benign tumors reported as leiomyomas, angiomas, and lipomas. Malignant neoplasms include adenocarcinomas, which commonly originate from the gastric mucosa, sarcoma, and carcinoid tumor.

Diagnostic Studies :

- The diagnosis of Meckel's diverticulum may be difficult.
- Plain abdominal radiographs, CT, and ultrasonography are rarely helpful.
- In children, the single most accurate diagnostic test for Meckel's diverticula is scintigraphy with sodium ^{99m}Tc-pertechnetate. The ^{99m}Tc-pertechnetate is preferentially taken up by the mucus-secreting cells of gastric mucosa and ectopic gastric tissue in the diverticulum .
- In adults, ^{99m}Tc-pertechnetate scanning is less accurate because of the reduced prevalence of ectopic gastric mucosa within the diverticulum. The sensitivity and specificity can be improved by the use of pharmacologic agents such as pentagastrin and glucagon or histamine-2 (H_2)-receptor antagonists (e.g., cimetidine).
- In adult patients, when nuclear medicine findings are normal, barium studies should be performed.
- In patients with acute hemorrhage, angiography is sometimes useful.

Treatment :

- The treatment of a symptomatic Meckel's diverticulum should be prompt surgical intervention with resection of the diverticulum or resection of the segment of ileum bearing the diverticulum.
- Segmental intestinal resection is required for treatment of patients with bleeding because the bleeding site usually is in the ileum adjacent to the diverticulum.

- It is generally recommended that asymptomatic diverticula found in children during laparotomy be resected. The treatment of Meckel's diverticula encountered in the adult patient, however, remains controversial.
- The recommendation is that an incidentally found Meckel's diverticulum should be removed at any age up to 80 years as long as no additional conditions (e.g., peritonitis) made removal hazardous.

4. Write brief answers: 4 x $7^1/_2$

(a) Pathophysiology of acute pancreatitis .
(b) Chronic limb ischaemia:.
(c) Medullary carcinoma of thyroid:
(d) Polyposis coli

(a) Pathophysiology of acute pancreatitis .

Answer. Acute pancreatitis includes a wide spectrum of disease, from one with mild self-limiting symptoms, to fulminant processes with multiorgan failure and high mortality.

Etiologic Factors in Acute Pancreatitis:

METABOLIC	MECHANICAL	VASCULAR	INFECTION
• Alcohol • Hyperlipoproteinemia • Hypercalcemia • Drugs • Genetic • Scorpion venom	• Cholelithiasis • Postoperative • Pancreas divisum • Post-traumatic • Retrograde pancreatography Pancreatic duct obstruction: pancreatic tumor, ascaris infestation • Pancreatic ductal bleeding Duodenal obstruction	• Postoperative (cardiopulmonary bypass) • Periarteritis nodosa • Atheroembolism	• Mumps • Coxsackie • Cytomegalovirus • Cryptococcus

Pancreatitis is characterized by a nonbacterial inflammation of the pancreas. This inflammation is caused by the activation, interstitial liberation, and digestion of the pancreas by its own digestive enzymes. During mild attacks, morphologic changes include pancreatic and peripancreatic edema and fat necrosis. In its severe form, pancreatitis is characterized by pancreatic parenchymal necrosis, extensive pancreatic and peripancreatic fat necrosis, and hemorrhage into and around the pancreas.

Furthermore, ischemia-reperfusion injury is believed to be critical to disease progression. A local inflammatory response in the pancreas is associated with the liberation of oxygen-derived free radicals and cytokines including interleukin 1 (IL-1), IL-6, and IL-8; tumor necrosis factor a (TNF-a); and platelet-activating factor; these mediators play an important role in the transformation from a local inflammatory response to a systemic illness.

(b) Chronic limb ischaemia:

Ans. Chronic upper limb ischaemia

Key facts: Upper limb ischaemia occurs less frequently than lower limb ischaemia.

Causes

- Previous trauma or axillary irradiation leading to arterial stenosis.
- Buerger's disease. Affects small vessels of the hands and feet, principally in smokers, associated with Raynaud's phenomenon. Mostly young men but women may be affected. Presents with digital gangrene/ischaemia and may present with acute limb ischaemia in young people.
- Subclavian steal syndrome. Reversed flow in the vertebral artery/diminished hindbrain perfusion (dizziness/syncope), arm claudication.
- Takayasu's arteritis. Uncommon in Europe; major arch/upper limb vessels affected.
- Thoracic outlet syndrome.
    - Term used to cover a spectrum of symptoms resulting from the compression of the neurovascular bundle as it leaves the chest to enter the upper limb, in an area enclosed by the first rib, clavicle, and scalenus anterior.
    - Presents as a variable combination of neural, arterial, and venous symptoms exacerbated by elevation of the limb, with pain, paraesthesiae, weakness, or arm claudication. 95% are neurogenic and 5% are arterial or venous manifestations.

Clinical features

- Weakness, cramp, or exercise-related pain and digital ischaemia/gangrene.
- Examine bilateral upper limb pulses, BP in both arms (elevated/at sides), wrist Doppler pressures.
- Roos test. Arm abducted to 90Â°, hands up with elbows braced backward, chin elevated, hands serially clenched/opened for 1-2min. Test positive if pain or weakness in hand or forearm.
- Adson's test. Pulse diminishes or absent on elevation/abduction of arm with head turned to contralateral side. Reliability improved by using in conjunction with arterial duplex.
- Allen's test. Assesses integrity of the palmar arch and dominant vessel (radial or ulnar).
- Tinel's test. To exclude carpal tunnel syndrome.

Diagnosis and investigation

- Cervical spine and thoracic outlet X-rays; wrist Doppler pressures.
- CT/MRI to exclude fibrous bands/ribs.
- Arterial duplex or angiography to exclude proximal arterial lesions.

Treatment
Thoracic outlet syndrome

- Mild neurogenic problem: simple analgesia and advice on risk factors.
- Surgery has good results for those with arterial or venous symptoms/complications.
- Excision of the first rib/band will improve symptoms in over 90%.

- Careful evaluation is needed prior to surgery for pure neurological symptoms, e.g. nerve conduction studies.

Cervical sympathectomy in upper limb disease
Indications

- Palmar hyperhidrosis (not axillary).
- Buerger's disease/small vessel disease with digital gangrene.

Approach

- Aim is to de-innervate the second and third thoracic ganglia.
- Approach is almost universally thoracoscopic and open approaches have been largely abandoned.

Complications

- Horner's syndrome.
- Pneumothorax.
- Haemorrhage.
- Compensatory truncal hyperhidrosis.
- Frey's syndrome (gustatory sweating).

Axillary hyperhidrosis

Treatment of choice is now SC botulinum toxin injections to the axillary sweat glands, repeated as necessary.
Anatomy of thoracic outlet
Several structures can compress the neurovascular structures

- Cervical rib: articulates with C7
- Scalene muscle: aberrant anatomy or scarring/swelling from trauma
- Costoclavicular ligament

Chronic lower limb ischaemia:

introduction

- Atherosclerosis is a generalized disease and has a predilection for the coronary, cerebral, and peripheral circulations.
- In the lower limb it may affect the aorto-iliac, femoral or popliteal, and calf vessel levels singly or in combinations.
- Single-level disease usually results in intermittent claudication (IC) and two-level disease in critical limb ischaemia (CLI).

La Fontaine classification of lower limb ischaemia

- I, Asymptomatic
- II, Intermittent claudication
- III, Rest pain
- IV, Ulcers/gangrene

Grades III and IV = critical limb ischaemia (CLI).

Intermittent claudication:
Key facts:

- Affects 7% of men over 50 years of age.
- M:F ratio is 2:1.
- One-third of patients improve, one-third remain stable, and one-third deteriorate.
- 4% require an intervention and 1% result in amputation.

Intermittent claudication: risk factors and associations:

Risk factors	Associations
• Hypertension • Hyperlipidaemia • Diabetes mellitus • Tobacco smoking • Positive family history	• Obesity • Diet • Sedentary lifestyle • Gender • Occupation

Clinical features

- Pain on exercise of the affected limb: worsened with increasing level of exercise; relieved by rest.
- Differential diagnosis.
    - Spinal stenosis. Symptoms caused by drop in distal corda equina blood flow due to exercise leading to neurogenic pain.
    - Osteoarthritis, especially hip joint.
    - Nerve root entrapment.
    - Popliteal artery entrapment due to compression of popliteal artery over medial head of gastrocnemius during exercise. Distal pulses reduced/absent on plantar flexion alone. Treated by surgical release after MRI defines anatomy.

Diagnosis and investigation

- Diagnosis is clinical and not based on imaging.
- Serum fasting glucose, serum cholesterol, BP checked on initial presentation.
- Imaging is only conducted for worsening symptoms, failure to respond to conservative management, consideration of surgery.
    - Angiography (usually digital subtraction (DSA), perhaps CT angiogram, or magnetic resonance angiogram (MRA)).
- Abdominal ultrasound if aneurysm disease suspected.

Treatment
Risk factor modification

- 40% of patients with peripheral vascular disease have coronary or cerebral arterial disease. The mainstay of treatment is aggressive risk factor modification.
    - Stop smoking, oral statin treatment, increased exercise, control of BP and serum glucose.

Endovascular treatment

- Angioplasty ± stent. Excellent results in the aorto-iliac segment (over 90% success) and good results in the superficial femoral segment (90% success and 60-80% patency at 2 years).
    - Usually performed under LA, percutaneously as a day case.
    - Rarely performed for claudication in the popliteal and tibial segments due to high risk of occlusion.

Surgery
Indications for surgery are as follows.

- Failure/unsuitability for endovascular treatment in the aorto-iliac segments. Procedures available are:
    - aortobifemoral graft: 5-year patency of over 90% but carries a 5-8% mortality and a risk of impotence; used for younger patients;
    - femoro-femoral crossover bypass graft: used for isolated unilateral iliac disease. 90% 1-year patency;
    - common femoral endarterectomy: used for isolated common femoral disease; good results and a low complication rate.
- Short distance claudication, severe lifestyle limitation, or failed medical therapy in infrainguinal (superficial femoral, popliteal, and distal) disease. Risks are significant. Procedures available are:
    - femoro-above knee popliteal bypass: 80% 2-year patency with vein or prosthetic graft;
    - femoro-below knee popliteal bypass: 70% 2-year patency with vein graft;
    - femoro-distal (below knee) bypass: 5-year patency < 35%. Usually reserved only for critical ischaemia. **(Oxford Handbook of Clinical Surgery, 3rd Edition).**

(c) Medullary carcinoma of thyroid:

Ans. Medullary carcinoma of thyroid: MTCs account for about 5% of thyroid malignancies and arise from the parafollicular or C cells of the thyroid, which, in turn, are derived from the ultimobranchial bodies. C cells secrete calcitonin, a 32-amino-acid polypeptide that functions to lower serum calcium levels.

Most MTCs occur sporadically. However, approximately 25% occur within the spectrum of several inherited syndromes such as familial medullary thyroid cancer, MEN2A, and MEN2B. All these variants are known to result secondary to germline mutations in the RET proto-oncogene. The syndromes are also characterized by genotype–phenotype correlations, with specific mutations leading to particular clinical manifestations.

Clinical and Genetic Features of Medullary Thyroid Cancer Syndromes

Syndrome	Manifestations	Ret Mutations
MEN2A	MTC, pheochromocytoma, primary hyperparathyroidism, lichen planus, amyloidosis	Exon 10: codons 609, 611, 618, 620
		Exon 11: codon 634 (more commonly associated with pheochromocytoma and primary hyperparathyroidism)
MEN2B	MTC, pheochromocytoma, marfanoid habitus, mucocutaneous ganglioneuromatosis	exon 16: codon 918
Familial MTC	MTC	Codons 609, 611, 618, 620, and 634
		Codons 768, 790, 791, or 804 (rare)
MEN2A and Hirschsprung's disease	MTC, pheochromocytoma, primary hyperparathyroidism, Hirschsprung's disease	Codons 609, 618, 620

Patients with MTC often present with a neck mass that may be associated with palpable cervical lymphadenopathy (15 to 20%). Local pain or aching is more common in patients with these tumors, and local invasion may produce symptoms of dysphagia, dyspnea, or dysphonia. Distant blood-borne metastases to the liver, bone (frequently osteoblastic), and lung occur later in the disease. The female:male ratio is 1.5:1. Most patients present between 50 and 60 years of age, although patients with familial disease present at a younger age. Medullary thyroid tumors secrete not only calcitonin and carcinoembryonic antigen (CEA), but also other peptides such as

calcitonin gene-related peptide (CGRP), histaminadases, prostaglandins E_2 and F_2 , and serotonin. Patients with extensive metastatic disease frequently develop diarrhea, which may result from increased intestinal motility and impaired intestinal water and electrolyte flux. Approximately 2 to 4% of patients develop Cushing's syndrome as a result of ectopic production of adrenocorticotropic hormone (ACTH).

Pathology

MTCs are typically unilateral (80%) in patients with sporadic disease, and multicentric in familial cases, with bilateral tumors occurring in up to 90% of familial patients. Familial cases are also associated with C-cell hyperplasia (Fig. 37-28), which is considered a premalignant lesion. Microscopically, tumors are composed of sheets of infiltrating neoplastic cells separated by collagen and amyloid. Marked heterogeneity is present; cells may be polygonal or spindle-shaped. The presence of amyloid is a diagnostic finding, but immunohistochemistry for calcitonin is more commonly used as a diagnostic tumor marker. These tumors also stain positively for CEA and CGRP.

Diagnosis

The diagnosis of MTC is established by history, physical examination, raised serum calcitonin or CEA levels, and FNA cytology of the thyroid mass. Attention to family history is important because approximately 25% of patients with MTC have familial disease. Because it is not possible to distinguish sporadic from familial disease at initial presentation, all new patients with MTC should be screened for RET point mutations, pheochromocytoma (24-hour urinary levels of VMA, catecholamine, and metanephrine), and hyperparathyroidism (serum calcium). It is important to rule out a coexisting pheochromocytoma to avoid precipitating a hypertensive crisis and death. Screening of patients with familial MTC for RET point mutations has largely replaced using provocation testing with pentagastrin or calcium-stimulated calcitonin levels to make the diagnosis. Calcitonin and CEA are used to identify patients with persistent or recurrent MTC. Calcitonin is a more sensitive tumor marker, but CEA is a better predictor of prognosis.

Treatment

If patients are found to have a pheochromocytoma, this must be operated on first. These tumors are generally (>50%) bilateral. Total thyroidectomy is the treatment of choice for patients with MTC because of the high incidence of multicentricity, the more aggressive course, and ^{131}I therapy is not usually effective. The central compartment nodes are frequently involved early in the disease process, so that a bilateral central neck node dissection should be routinely performed. In patients with palpable cervical nodes or involved central neck nodes, ipsilateral or bilateral, modified radical neck dissection is recommended. Similarly, patients with tumors larger than 1.5 cm should undergo ipsilateral prophylactic modified radical neck dissection, because greater than 60% of these patients have nodal metastases. Approximately 30% of these patients will also have contralateral nodal metastases. In the case of locally recurrent or metastatic disease, tumor debulking is advised, not only to ameliorate symptoms of flushing and diarrhea, but also to decrease risk of death from recurrent central neck or mediastinal disease. External beam radiotherapy is controversial, but is recommended for patients with unresectable residual or recurrent tumor. There is no effective chemotherapy regimen. Radiofrequency ablation done laparoscopically appears promising in the palliative treatment of liver metastases larger than 1.5 cm. Tumors that express c-kit may also respond to tyrosine kinase inhibitors such as Gleevec.

In patients who have hypercalcemia at the time of thyroidectomy, only obviously enlarged parathyroid glands should be removed. The other parathyroid glands should be preserved and marked in patients with normocalcemia as only approximately 20% of patients with MEN2A develop hyperparathyroidism. When a normal parathyroid cannot be maintained on a vascular pedicle, it should be removed, biopsied to confirm that it is a parathyroid, and then autotransplanted to the forearm of the nondominant arm in MEN2A patients and in the sternocleidomastoid muscle in other patients.

Total thyroidectomy is indicated in RET mutation carriers once the mutation is confirmed. The procedure should be performed before age 6 years in MEN2A patients and prior to age 1 year in MEN2B patients.44 Central neck dissection can be avoided in children who are RET positive and calcitonin negative with a normal ultrasound examination. When the calcitonin is increased or the ultrasound suggests a thyroid cancer, a prophylactic central neck dissection is indicated.

Postoperative Follow-Up and Prognosis

Prognosis is related to disease stage. The 10-year survival rate is approximately 80% but decreases to 45% in patients with lymph node involvement. Survival also is significantly influenced by disease type. It is best in patients with non-MEN familial MTC, followed by patients with MEN2A, and then by patients with sporadic disease. Prognosis is the worst (35% at 10 years) in patients with MEN2B. Patients with tumors that stain poorly for calcitonin and with a heterogeneous distribution of calcitonin do worse than patients in whom calcitonin staining is increased and homogeneous. Performing prophylactic surgery in RET oncogene mutation carriers not only improves survival rates, but also renders most patients calcitonin free. **(Schwartz's Surgery > Part II. Specific Considerations > Chapter 37. Thyroid, Parathyroid, and Adrenal).**

(d) Polyposis coli:

Ans. **Hereditary Cancer Syndromes/ Polyposis coli**

	HEREDITARY ADENOMATOUS POLYPOSIS SYNDROMES		**HEREDITARY HAMARTOMATOUS POLYPOSIS SYNDROMES**			**Ruvalcaba-Myhre-Smith Syndrome (Bannayan-Zonana Syndrome)**
HEREDITARY NONPOLYPOSIS COLON CANCER	**Familial Adenomatous Polyposis/Gardner's Syndrome**	**Turcot's Syndrome**	**Cowden's Disease**	**Familial Juvenile Polyposis**	**Peutz-Jeghers Syndrome**	
GI Features						
Small number of colorectal polyps	Hundreds to thousands of colorectal polyps; duodenal adenomas and gastric polyps, usually fundic gland	Colorectal polyps, which may be few or resemble classic familial adenomatous polyposis	Polyps most commonly of colon and stomach	Juvenile polyps mostly in the colon but throughout GI tract Defined by ≥ 10 juvenile polyps	Small number of polyps throughout GI tract but most common in small intestine	Hamartomatous GI polyps, usually lipomas, hemangiomas, or lymphangiomas
Other Clinical Features						
Muir-Torre variant:	Osteomas, desmoid	Brain tumors,	Mucocutaneous lesions,	Congenital abnormaliti	Pigmented	Dysmorphic facial

	HEREDITARY ADENOMATOUS POLYPOSIS SYNDROMES		HEREDITARY HAMARTOMATOUS POLYPOSIS SYNDROMES			Ruvalcaba-Myhre-Smith Syndrome (Bannayan-Zonana Syndrome)
HEREDITARY NONPOLYPOSIS COLON CANCER	**Familial Adenomatous Polyposis/Gardner's Syndrome**	**Turcot's Syndrome**	**Cowden's Disease**	**Familial Juvenile Polyposis**	**Peutz-Jeghers Syndrome**	
sebaceous adenomas, keratoacanthomas, sebaceous epitheliomas, and basal cell epitheliomas	tumors, epidermoid cysts, and congenital hypertrophy of retinal epithelium	including cerebellar medulloblastoma and glioblastomas	thyroid adenomas and goiter, fibroadenomas and fibrocystic disease of the breast, uterine leiomyomas, and macrocephaly	es in at least 20%, including malrotation, hydrocephalus, cardiac lesions, Meckel's diverticulum, and mesenteric lymphangioma	lesions of skin; benign and malignant genital tumors	features, macrocephaly, seizures, intellectual impairment, and pigmented macules of shaft and glans of penis
Malignancy Risk						
70%-80% lifetime risk for colorectal cancer; 30%-60% lifetime risk for endometrial cancer; ↑ risk for ovarian cancer, gastric carcinoma, transitional cell carcinoma of the ureters and renal peivis, small bowel cancer, and sebaceous	Colorectal cancer risk approaches 100%; ↑ risk for periampullary malignancy, thyroid carcinoma, central nervous system tumors, and hepatoblastoma	Colorectal carcinoma and brain tumors	10% risk for thyroid cancer and up to 50% risk for adenocarcinoma of breast in affected women	9% to 25% risk for colorectal cancer; ↑ risk for gastric, duodenal, and pancreatic cancer	↑ Risk for GI malignancy and pancreatic cancer and adenoma malignum of cervix; unknown risk for breast cancer	Malignant GI tumors identified but lifetime risk for malignancy unknown

	HEREDITARY ADENOMATOUS POLYPOSIS SYNDROMES		HEREDITARY HAMARTOMATOUS POLYPOSIS SYNDROMES			Ruvalcaba-Myhre-Smith Syndrome (Bannayan-Zonana Syndrome)
HEREDITARY NONPOLYPOSIS COLON CANCER	**Familial Adenomatous Polyposis/Gardner's Syndrome**	**Turcot's Syndrome**	**Cowden's Disease**	**Familial Juvenile Polyposis**	**Peutz-Jeghers Syndrome**	
carcinomas						
Screening Recommendations						
Colonoscopy at age 20-25 yr; repeat every 1-3 yr Transvaginal ultrasound or endometrial aspirate at age 20-25 yr; repeat annually (expert opinion only)	Flexible proctosigmoidoscopy at age 10-12 yr; repeat every 1-2 yr until age 35; after age 35 repeat every 3 yr Upper GI endoscopy every 1-3 yr starting when polyps first identified	Same as for familial adenomatous polyposis Also consider imaging of the brain	Annual physical exam with special attention to thyroid Mammography at age 30 or 5 yr before earliest breast cancer case in the family Routine colon cancer surveillance (expert opinion only)	Screening by age 12 yr if symptoms have not yet arisen Colonoscopy with multiple random biopsies every several years (expert opinion only)	Upper GI endoscopy, small bowel radiography, and colonoscopy every 2 yr; pancreatic ultrasound and hemoglobin levels annually; gynecologic examination, cervical smear, and pelvic ultrasound annually;	No known published recommendations

	HEREDITARY ADENOMATOUS POLYPOSIS SYNDROMES		HEREDITARY HAMARTOMATOUS POLYPOSIS SYNDROMES			Ruvalcaba-Myhre-Smith Syndrome (Bannayan-Zonana Syndrome)
HEREDITARY NONPOLYPOSIS COLON CANCER	**Familial Adenomatous Polyposis/Gardner's Syndrome**	**Turcot's Syndrome**	**Cowden's Disease**	**Familial Juvenile Polyposis**	**Peutz-Jeghers Syndrome**	
					clinical breast exam and mammography at age 25 yr; clinical testicular exam and testicular ultrasound in males with feminizing features (expert opinion only)	
Genetic Basis						
AD	AD	AD	AD	AD inheritance in some families	AD	AD
MLH1 (chromosome 3p)	APC (chromosome 5q)	APC mutations identified predominantly in	PTEN (chromosome 10q)	Subset of families with mutation in SMAD4	STK11 (chromosome 19p)	PTEN (chromosome 10q) in some
MSH2 (chromosome						

	HEREDITARY ADENOMATOUS POLYPOSIS SYNDROMES		HEREDITARY HAMARTOMATOUS POLYPOSIS SYNDROMES			Ruvalcaba-Myhre-Smith Syndrome (Bannayan-Zonana Syndrome)
HEREDITARY NONPOLYPOSIS COLON CANCER	**Familial Adenomatous Polyposis/Gardner's Syndrome**	**Turcot's Syndrome**	**Cowden's Disease**	**Familial Juvenile Polyposis**	**Peutz-Jeghers Syndrome**	
2p)		families with cerebellar medulloblastoma		(DRC4) (chromosome 10q)		
MSH6/GTMP (chromosome 2p)						
PMS1 (chromosome 2q)		MLH1, PMS2 mutations identified in families with predominance of glioblastomas				
PMS2 (chromosome 7q)						
Genetic Testing						
Clinical testing of MLH1 and MSH2 genes available	Clinical testing of APC gene available	Clinical testing of APC and MLH1 genes available	Research testing of PTEN gene available	Families being collected for research studies only	Research testing of STK11 gene available	Research testing of PTEN gene available

AD, autosomal dominant; GI, gastrointestinal; ↑, increased.

Surgical treatment of patients with FAP is directed at removal of all affected colonic and rectal mucosa. Restorative proctocolectomy with IPAA has become the most commonly recommended operation. The procedure is usually accompanied by a distal rectal mucosectomy to ensure that all premalignant colonic mucosa is removed, and the IPAA is fashioned between the ileal pouch and the dentate line of the anal canal. Patients who undergo this procedure for FAP have a better functional result than patients similarly treated for ulcerative colitis, in that the incidence of inflammation in the ileal pouch (pouchitis) is much lower in patients with FAP than in patients with ulcerative colitis.

An alternative approach, total abdominal colectomy with ileorectal anastomosis, was used extensively before the development of the technique of IPAA and has certain advantages. If an FAP patient has relatively few polyps in the rectum, consideration may be given to this option. The abdominal colon is resected and an anastomosis fashioned between the ileum and rectum. It is technically a simpler operation to perform, and the pelvic dissection is avoided. This eliminates the potential complication of injury to the autonomic nerves that could result in impotence. In addition, there is theoretically less risk for anastomotic leak from the relatively simple ileorectal anastomosis fashioned in the peritoneal cavity, compared with the long suture (or staple) lines required to form the ileal pouch and then fashion the anastomosis between the ileal pouch and the anus.

An additional argument in favor if abdominal colectomy and ileorectal anastomosis is the observation that sulindac and celecoxib have been observed to cause the regression of adenomatous polyps in some patients with FAP. The disadvantages are that the rectum remains at high risk for the formation of new precancerous polyps, a proctoscopic examination is required every 6 months to detect and destroy any new polyps, and there is a definite increased risk for cancer arising in the rectum with the passage of time.

The management of patients with HNPCC is somewhat controversial, but the need for close surveillance in patients known to carry the mutation is obvious. It is usually recommended that a program of surveillance colonoscopy should begin at the age of 20 years. Colonoscopy is repeated every 2 years until the age of 35 years, and then annually thereafter. In women, periodic vacuum curettage is begun at age 25 years, as are pelvic ultrasound and CA-125 levels. Annual tests for occult blood in the urine should also be obtained because of the risk for ureteral and renal pelvic cancer.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES

MS (General Surgery) Examination, 2008

PAPER II

Time Allowed: 3 Hours **Full Marks: 100**

1. **Discuss the aetiopathology, diagnosis and management of chronic pancreatitis. (7+6+7)**
2. **Define early & advanced carcinoma breast. How do you manage impalpable lesions of the breast? (10+10)**

3. Write short notes on: 5 x 6

(a) Duodenal fistula

(b) Closed loop obstruction.

(c) Diabetic ulcer.

(d) Dieulafoy's lesion.

(e) Postoperative fever.

4. Write brief answers: 4 x 7.5

(a) Child's criteria for assessment of hepatic function

(b) Neurogenic bladder

(c) Fistula – in –ano

(d) Strangulated hernia.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES
MS (General Surgery) Examination, 2008

April, 2008

PAPER II

Time Allowed: 3 Hours **Full Marks: 100**

1. Discuss the aetiopathology, diagnosis and management of chronic pancreatitis. (7+6+7)

Answer. Aetiopathology of chronic pancreatitis:

Etiologic Risk Factors Associated With Chronic Pancreatitis: TIGAR-O Classification System

Toxic-metabolic
- Alcoholic
- Tobacco smoking
- Hypercalcemia
- Hyperparathyroidism
- Hyperlipidemia (rare and controversial)
- Chronic renal failure
- Medications
- Phenacetin abuse (possibly from chronic renal insufficiency)
- Toxins - Organotin compounds (e.g., DBTC)

Idiopathic
- Early onset
- Late onset
- Tropical
- Tropical calcific pancreatitis
- Fibrocalculous pancreatic diabetes
- Other

Genetic
- Autosomal dominant
- Cationic trypsinogen (Codon 29 and 122 mutations)
- Autosomal recessive/modifier genes
- CFTR mutations
- SPINK1 mutations
- Cationic trypsinogen (codon 16, 22, 23 mutations)
- a1-Antitrypsin deficiency (possible)

Autoimmune
- Isolated autoimmune chronic pancreatitis

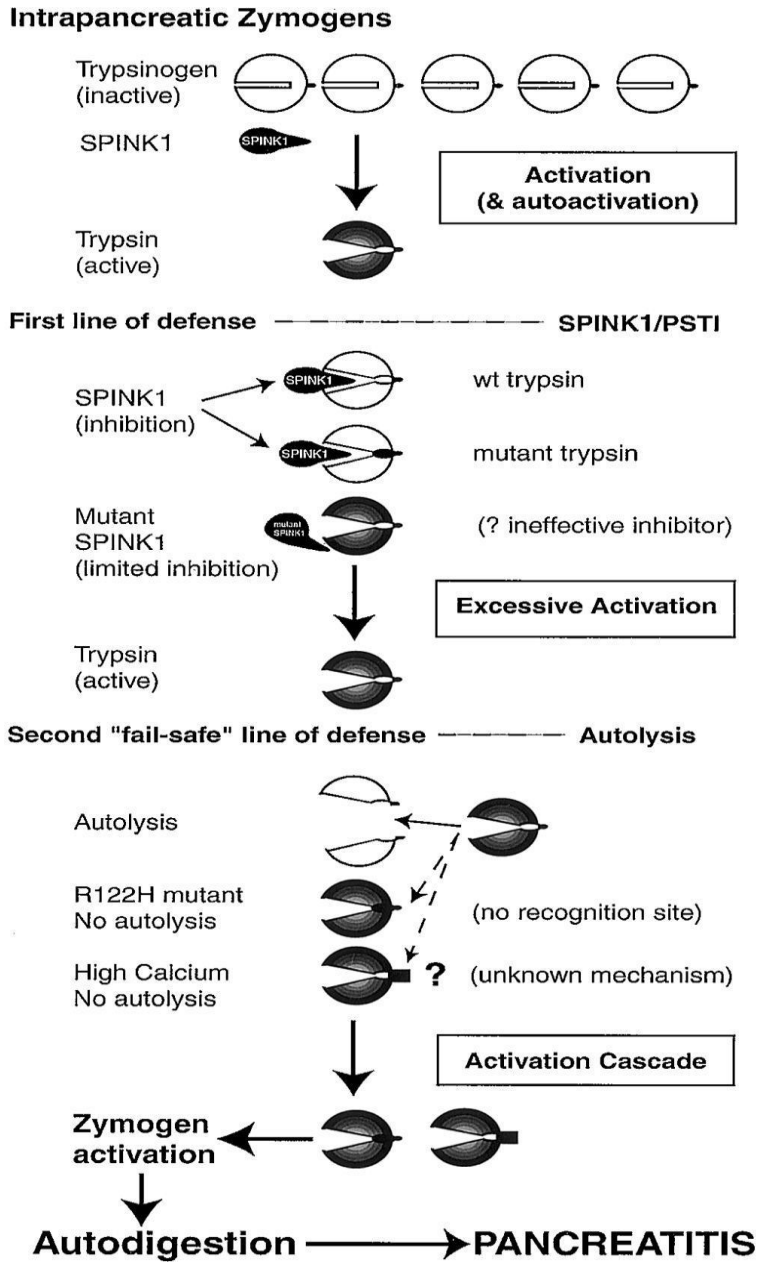
- Syndromic autoimmune chronic pancreatitis
- Sjo¨gren syndrome–associated chronic pancreatitis
- Inflammatory bowel disease–associated chronic pancreatitis
- Primary biliary cirrhosis–associated chronic pancreatitis
- Recurrent and severe acute pancreatitis
- Postnecrotic (severe acute pancreatitis)

Recurrent acute pancreatitis

- Vascular diseases/ischemic
- Post irradiation

Obstructive

- Pancreatic divisum
- Sphincter of Oddi disorders (controversial)
- Duct obstruction (e.g., tumor)
- Preampullary duodenal wall cysts
- Posttraumatic pancreatic duct scars



Pathology:

- Initially the exocrine pancreas is affected.
- Acinar cells are lost and ductal cells eliminated. Remaining Ductal elements are buried connective tissue.
- Islets can be seen disconnected from acinar tissue and are also surrounded by fibrosis.
- The fibrosis that is present includes collagen fibers, fibroblasts, and myofibroblasts.
- Pancreatic stellate cells inthe normal pancreas reside at the base of acinar cells and harbor vitamin A-containing lipid droplets. They represent the main source of

extracellular matrix and collagen in chronic pancreatitis. Ethanol can directly activate stellate cells and may also work through tumor growth factor (TGF)-beta and other proinflammatory cytokines.

- Patients with long standing chronic pancreatitis demonstrate numerous nerves running throughout the fibrotic tissue.These nerves have lost their protective perineurium, which make them more sensitive to various nociceptive substances. Often local inflammatory cells are seen near these nerves, which may be partially responsible for the significant pain.

Diagnosis of chronic pancreatitis:

- There has been considerable confusion concerning the clinical distinction between chronic and acute pancreatitis.

❖ History:

- Patients with chronic pancreatitis may describe prior episodes of pancreatic-type abdominal pain, and 60% to 80% of patients have a long history of alcohol abuse.
- There may be a family history of pancreatitis suggestive of the presence of hereditary pancreatitis or a history of autoimmune diseases, including primary sclerosing cholangitis and Sjögren's syndrome, that raise suspicion of pancreatitis on an autoimmune basis.
- Diabetes mellitus or a history suggestive of malabsorption (i.e., steatorrhea) indicates that significant pancreatic endocrine or exocrine function has been lost, and this is most compatible with the diagnosis of chronic pancreatitis.
- Typically, patients with chronic pancreatitis have upper abdominal pain radiating to the back. It can be constant or episodic and triggered by drinking alcohol or eating.
- Repeated use of heating pads or hot water bottles to treat the chronic pain may result in skin lesions (*erythema ab igne*) that define the distribution of the pain.
- Some patients experience no pain.

❖ Imaging Studies:

- Radiographs or CT scans showing pancreatic calcifications are diagnostic of chronic pancreatitis .Those calcifications reflect the deposition of calcium carbonate in the intraductal protein plugs that frequently, but not invariably, occur in chronic pancreatitis.
- ERCP, CT cholangiopancreatography, or magnetic resonance cholangiopancreatography may be particularly valuable in the diagnosis of chronic pancreatitis.

- Chronic pancreatitis is characterized by irregularities of the pancreatic ducts, ductal strictures, and areas of duct dilation.The major as well as the side-branch ducts may be involved.

- Some patients with chronic pancreatitis develop dilated main pancreatic ducts (large duct disease), whereas others retain ducts of normal or even smaller than normal caliber (small duct disease).
- Some patients with chronic pancreatitis can be shown to have major ducts that have the appearance of a **"chain of lakes"** or a **"string of pearls"** that is the result of segments of dilated duct separated by areas of ductal stricture.
- Transcutaneous and endoscopic ultrasound can also be used to diagnose chronic pancreatitis if duct dilation, calcifications, pseudocysts, or parenchymal fibrosis are seen.
- Ultrasound examination is more operator dependent and perhaps less sensitive than either CT or MRI.

Endoscopic Ultrasound Features of Chronic Pancreatitis

Endoscopic Ultrasound Feature	Implication
Ductal changes	
Duct size >3 mm	Ductal dilation
Tortuous pancreatic duct	Ductal irregularity
Intraductal echogenic foci	Stones or calcification
Echogenic duct wall	Ductal fibrosis
Side-branch ectasia	Periductal fibrosis
Parenchymal changes	
Inhomogeneous echo pattern	Edema
Reduced echogenic foci (1–3 mm)	Edema
Enhanced echogenic foci	Calcifications
Prominent interlobular septae	Fibrosis
Lobular outer gland margin	Fibrosis, glandular atrophy
Large, echo-poor cavities (>5 mm)	Pseudocyst

Cambridge Classification of Chronic Pancreatitis by Endoscopic Retrograde Cholangiopancreatography

Grade	**Main Pancreatic Duct**	**Side Branches**
Normal	Normal	Normal

Suggestive	Normal	<3 Abnormal
Mild	Normal	3 Abnormal
Moderate	Abnormal	>3 Abnormal
Severe	Abnormal plus at least one of the following:	
	Large cavity	
	Duct obstruction	
	Dilation or duct irregularity	
	Intraductal filing defects	

Pancreatic Function Tests
Tubeless Tests Fecal Tests Fat stain 72-hour fat content Chymotrypsin, trypsin, elastase content **Indirect Tests** Bentiromide test Pancreolauryl test Breath tests **Tube Tests** Lundh test meal Secretin or cholecystokinin test

Treatment:

The treatment of chronic pancreatitis requires an interdisciplinary approach.The most common symptom in a patient with chronic pancreatitis is abdominal pain. The etiology of the pain is multifactorial and, in general, is not well understood.

Procedures available to the endoscopist for the treatment of pain include:

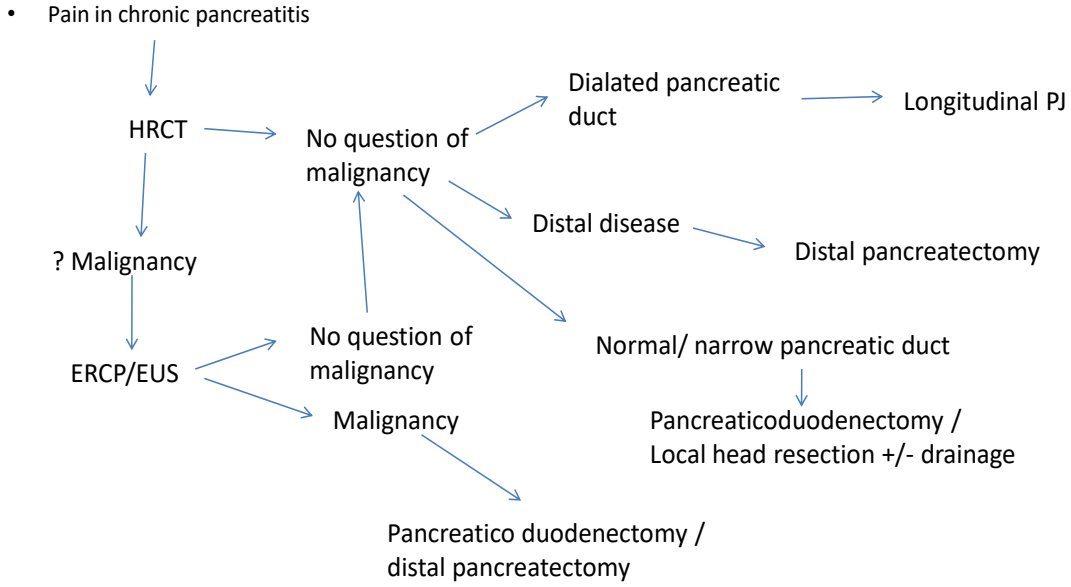
- Pancreatic sphincterotomy,
- Pancreatic ductal stenting,
- Extracorporeal shock wave lithotripsy (eswl) of pancreatic stones,
- endoscopic drainage of pancreatic pseudocysts, and

- Endoscopic celiac nerve block.

- ❖ In addition to pain, patients may present with exocrine or endocrine dysfunction which are managed medically.
- ❖ Diabetes usually requires insulin and dietary restrictions.
- ❖ Malabsorption is treated with pancreatic enzyme replacement in sufficient amount to provide at least 30,000 units of lipase with each meal.
- ❖ Antacid secretory medications also may be useful in patients with acid hypersecretion to avoid denaturation of the replacement enzymes.
- ❖ Rarely, fat intake must also be limited to decrease the bothersome diarrhea that accompanies excessive steatorrhea.
- ❖ Other reasons that a patient may present to a physician for care include various intra-abdominal complications of pancreatitis (e.g., bile duct, colonic or duodenal obstruction, pseudocyst), and the concern for pancreatic cancer.

Indications for Surgery in Chronic Pancreatitis:

Intractable pain
Pancreatic ductal stenosis
Biliary obstruction (Wadsworth syndrome)
Duodenal obstruction
Left sided portal hypertension from splenic vein thrombosis
Colonic obstruction
Pseudocyst
Pancreatic ascites
Pancreatic fistula
Pancreatic carcinoma



- The type of operation depends on the anatomy of the pancreatic ductal system, and whether or not the pancreas is diffusely involved with the disease or it involves one part of the gland more than the others.
- Operations to relieve pain in these patients are designed to either (1) drain a dilated pancreatic ductal system, or (2) resect diseased pancreatic tissue if the duct is not enlarged. The main pancreatic duct normally measures 4 to 5 mm in the head, 3 to 4 mm in the body, and 2 to 3 mm in the tail of the pancreas. The duct is considered dilated when it is at least 7 mm in diameter in the body of the gland. Thus, patients with a dilated pancreatic duct (>7 mm in the body of the gland) are candidates for a drainage operation that decompresses the duct (longitudinal pancreaticojejunostomy, Puestow procedure).
- Those with a duct that is of normal caliber will probably require resection of a part of the pancreas, usually the head of the gland (pancreaticoduodenectomy), Frey or Beger procedure).

Choice of Surgical Procedures for Treatment of Pain in Chronic Pancreatitis

Duct drainage procedure
Lateral pancreaticojejunostomy (Partington-Rochelle modification of Puestow procedure)

Combined duct drainage-resection procedures
Pancreaticoduodenectomy (Kausch-Whipple)
Longitudinal pancreaticojejunostomy with local head resection (Frey)
Duodenum-preserving resection of the head of the pancreas (Beger)
Pure resection procedure
Total pancreatectomy with islet autotransplantation
Neuroablative procedure
Thoracic splanchnicectomy

2. Define early & advanced carcinoma breast. How do you manage impalpable lesions of the breast? (10+10)

Answer. TNM Staging System for Breast Cancer

Primary tumor (T) Definitions for classifying the primary tumor (T) are the same for clinical and for pathologic classification. If the measurement is made by physical examination, the examiner will use the major headings (T1, T2, or T3); if other measurements, such as mammographic or pathologic measurements, are used, the subsets of T1 can be used. Tumors should be measured to the nearest 0.1-cm increment	
TX	Primary tumor cannot be assessed
T0	No evidence of primary tumor
Tis	Carcinoma in situ
Tis(DCIS)	Ductal carcinoma in situ
Tis(LCIS)	Lobular carcinoma in situ
Tis(Paget's)	Paget's disease of the nipple with no tumor (**Note:** Paget's disease associated with a tumor is classified according to the size of the tumor)
T1	Tumor 2 cm or less in greatest dimension
T1mic	Microinvasion 0.1 cm or less in greatest dimension
T1a	Tumor more than 0.1 cm but not more than 0.5 cm in greatest dimension
T1b	Tumor more than 0.5 cm but not more than 1 cm in greatest dimension
T1c	Tumor more than 1 cm but not more than 2 cm in greatest dimension
T2	Tumor more than 2 cm but not more than 5 cm in greatest dimension
T3	Tumor more than 5 cm in greatest dimension
T4	Tumor of any size with direct extension to (a) chest wall or (b) skin,

	only as described below
T4a	Extension to chest wall, not including pectoralis muscle
T4b	Edema (including peau d'orange), or ulceration of the skin of the breast, or satellite skin nodules confined to the same breast
T4c	Both T4a and T4b
T4d	Inflammatory carcinoma
Regional lymph nodes—Clinical (N)	
NX	Regional lymph nodes cannot be assessed (e.g., previously removed)
N0	No regional lymph node metastasis
N1	Metastasis to movable ipsilateral axillary lymph node(s)
N2	Metastases in ipsilateral axillary lymph nodes fixed or matted, or in clinically apparent[a] ipsilateral internal mammary nodes in the absence of clinically evident axillary lymph node metastasis
N2a	Metastasis in ipsilateral axillary lymph nodes fixed to one another (matted) or to other structures
N2b	Metastasis only in clinically apparent[a] ipsilateral internal mammary nodes and in the absence of clinically evident axillary lymph node metastasis
N3	Metastasis in ipsilateral infraclavicular lymph node(s) with or without axillary lymph node involvement, or in clinically apparent [a] ipsilateral internal mammary lymph node(s) and in the presence of clinically evident axillary lymph node metastasis; or metastasis in ipsilateral supraclavicular lymph node(s) with or without axillary or internal mammary lymph node involvement
N3a	Metastasis in ipsilateral infraclavicular lymph node(s)
N3b	Metastasis in ipsilateral internal mammary lymph nodes(s) and axillary lymph node(s)
N3c	Metastasis in ipsilateral supraclavicular lymph node(s)

TNM Stage Groupings

Stage 0	Tis	N0	M0
Stage I	T1[a]	N0	M0
Stage IIA	T0	N1	M0

	T1[a]	N1	M0
	T2	N0	M0
Stage IIB	T2	N1	M0
	T3	N0	M0
Stage IIIA	T0	N2	M0
	T1[a]	N2	M0
	T2	N2	M0
	T3	N1	M0
	T3	N2	M0
Stage IIIB	T4	N0	M0
	T4	N1	M0
	T4	N2	M0
Stage IIIC	Any T	N3	M0
Stage IV	Any T	Any N	M1

Early Invasive Breast Cancer is Stage I, IIa, or IIb.

Advanced Breast Cancer is Stage IIIa or IIIb.

Management of nonpalpable lesions:

- Minimally invasive breast biopsy is the optimal initial tissue acquisition method and procedure of choice for obtaining a pathologic diagnosis of image-detected abnormalities.
- Correlation between pathology results and imaging findings is mandatory.
- Patients with high-risk lesions on image-guided biopsy (ADH, ALH, lobular carcinoma in situ, radial scar) may have malignancy at the same site and should undergo a surgical biopsy.

- **Stereotactic core biopsy** is used for nonpalpable mammographically detected lesions, such as microcalcifications, which cannot be seen with ultrasonography. Contraindications include lesions close to the chest wall or in the axillary tail and thin breasts that may allow needle strikethrough. Superficial lesions and lesions directly beneath the nipple-areola complex are also often not approachable with stereotactic techniques. Nondiagnostic and insufficient specimens should undergo needle-localized excisional biopsy (NLB, see later discussion), as should discordant pathologic findings on core needle stereotactic biopsy.

- **Vacuum-assisted biopsy** is generally used during stereotactic core biopsies and ultrasound-guided core biopsies. Multiple contiguous samples of tissue are collected while the probe remains in the breast. Volumes up to 1 mL can be collected during a single insertion. This is the preferred approach for lesions presenting with microcalcifications without a visible or palpable mass.
- **Ultrasound-guided biopsy** is the preferred method if a lesion can be visualized with ultrasound because it is generally easier to perform than a stereotactic core biopsy. Lesions with a cystic component are better visualized with ultrasound, and ultrasound-guided biopsy can be used to aspirate the cyst as well as to provide core biopsy specimens.
- **Needle localization excisional biopsy(NLB):** A needle and hookwire are placed into the breast adjacent to the concerning lesion under mammographic guidance. The patient is then brought to the operating room for an excisional biopsy. Using localization mammograms as a map, the whole hookwire, breast lesion, and a rim of normal breast tissue are removed en bloc. The specimen is oriented, and a radiograph is performed to confirm the presence of the lesion within the specimen.
- **Magnetic Resonance Imaging:** In the process of evaluating MRI as a means of characterizing mammography abnormalities, additional breast lesions have been detected. However, in the circumstance of both a negative mammogram and a negative physical examination, the probability of a breast cancer being diagnosed by MRI is extremely low. There is current interest in using MRI to screen the breasts of high-risk women and of women with a newly diagnosed breast cancer. In the first case, women with a strong family history of breast cancer or who carry known genetic mutations require screening at an early age, but mammography evaluation is limited because of the increased breast density in younger women.

  Non invasive (in situ) breast cancer: DCIS (ductal carcinoma in situ) or LCIS (lobular carcinoma in situ) are lesions with malignant cells that have not penetrated the basement membrane of the mammary ducts or lobules, respectively.

  - **DCIS:** Surgical excision alone (via partial mastectomy) with margins greater than 10 mm is associated with a local recurrence rate of 14% at 12 years. The addition of adjuvant radiation reduces the local recurrence rate to 2.5%. Approximately half of the recurrences present as invasive ductal carcinomas. Surgical options depend on the extent of disease, grade, margin status, multicentricity of disease, and patient age.
    - Partial mastectomy: For unicentric lesions. Needle localization is required to identify the area to be excised in most cases. Bracket needle localization (two or more wires to map out the extent of disease to be resected) for more extensive lesions is occasionally used.
    - Mastectomy: Total (simple) mastectomy with or without immediate reconstruction is recommended for patients with multicentric lesions, extensive involvement of the breast (disease extent relative to breast size), or persistently positive margins with partial mastectomy.

- Assessment of axillary lymph nodes: Axillary dissection is not performed for pure DCIS.
  - Sentinel lymph node biopsy (SNLB, see later discussion) may be considered when there is a reasonable probability of finding invasive cancer on final pathologic examination (e.g., >4 cm, palpable, or high grade).
  - Some surgeons perform SLNB in all patients with DCIS undergoing mastectomy because SLNB cannot be performed postmastectomy if an occult invasive cancer is found. This is an area of ongoing controversy and research.
  - A positive sentinel node indicates invasive breast cancer and changes the stage of the disease; a completion axillary dissection is then indicated.
- Adjuvant therapy
  - For pure DCIS, there is no added benefit from systemic chemotherapy because the disease is confined to the ducts of the breast. However, in those patients with ER-positive DCIS, adjuvant tamoxifen can reduce the risk of breast cancer recurrence by 37% over 5 years and the risk of developing a new contralateral breast cancer (NSABP B-24 trial). However, there is no survival benefit. Aromatase inhibitors (e.g., anastrazole, exemestane, letrozole), which block the peripheral conversion of androgens into estrogens by inhibiting the enzyme aromatase but does not affect estrogen produced by the ovaries, are sometimes used as an alternative in postmenopausal patients.

Adjuvant radiation should be given to patients with DCIS treated with partial mastectomy to decrease the local recurrence rate (NSABP B-17 trial). This is especially true for younger women with close margins or large tumors. However, there is no survival benefit. For older patients with smaller, widely excised DCIS of low or intermediate grade, the benefit of radiation therapy is so small that adjuvant radiation is not recommended.

LCIS:

- LCIS is not considered a preinvasive lesion but rather an indicator for increased breast cancer risk of approximately 1% per year (~20% to 30% at 15 years) and is not treated as a breast cancer.
- Pleomorphic LCIS is a particularly aggressive subtype of LCIS that is treated more like DCIS; it tends to have less favorable biological markers.
- Treatment options are (1) lifelong close surveillance, (2) bilateral total mastectomies with immediate reconstruction for selected women with a strong family history after appropriate counseling, or (3) prophylaxis with tamoxifen.

Surgical options for stage I and II breast cancer:

- Mastectomy with or without reconstruction.
  - Modified radical mastectomy (MRM) involves total (simple) mastectomy and axillary lymph node dissection. It is indicated for patients with clinically

positive lymph nodes or a Total positive axillary node based on previous SLNB or FNAB.

- (simple) mastectomy with SLNB is for patients with a clinically negative axilla. A skin-sparing mastectomy (preserves skin envelope and inframammary ridge) may be performed with immediate reconstruction, resulting in improved cosmesis: The nipple-areolar complex, a rim of periareolar breast skin, and any previous excisional biopsy or partial mastectomy scars are excised.
- Immediate reconstruction at the time of mastectomy should be offered to eligible patients. Options include latissimus dorsi myocutaneous flaps, transverse rectus abdominis myocutaneous flaps, and inflatable tissue expanders followed by exchange for saline or silicone implants. Immediate reconstruction has been shown not to affect patient outcome adversely. The detection of recurrence is not delayed, and the onset of chemotherapy is not changed.
- Follow-up after mastectomy: physical examination every 3 to 6 months for 3 years, then every 6 to 12 months for the next 2 years, and then annually. Mammography of the contralateral breast should continue yearly. Regular gynecologic follow-up is recommended for all women (tamoxifen increases risk of endometrial cancer).

- Breast conservation therapy (BCT): partial mastectomy and SLNB (or axillary lymph node dissection; see later discussion) followed by breast irradiation.
  - Several trials have demonstrated that BCT with adjuvant radiation therapy has similar survival and recurrence rates to those for MRM.
  - Not every patient is a candidate for BCT. Contraindications for BCT: A patient who may be unreliable with follow-up or radiation therapy (may involve radiation treatment 5 days a week for 5 to 6 weeks); when the extent of disease prevents adequate negative margins; a high tumor-to-breast size ratio, which prevents adequate resection without major deformity; persistently positive margins on re-excision partial mastectomy; and inability to receive adjuvant radiation (e.g., prior radiation to the chest wall; first- and second-trimester pregnancy in which the delay of radiation to the postpartum state is inappropriate; collagen vascular diseases such as scleroderma).
  - For patients with large tumors who desire BCT, neoadjuvant chemotherapy or neoadjuvant hormonal therapy may be offered to attempt to reduce the size of the tumor to make BCT attempt possible.
  - Partial mastectomy incisions should be planned so that they can be incorporated into a mastectomy incision should that prove necessary. Incisions for partial mastectomy and either SLNB or axillary lymph node dissection should be separate.
  - Adjuvant radiotherapy decreases the breast cancer recurrence rate from 30% to less than 7% at 5 years and is a required component of BCT.
  - Follow-up after BCT. Physical examinations are the same as those for mastectomy (see earlier discussion). A posttreatment mammogram of the treated breast is performed to establish a new baseline, no earlier than 6 months after completion of radiation therapy. Mammograms are then

performed every 6 to 12 months after the new baseline mammogram until the surgical changes stabilize and then annually. Contralateral breast mammography remains on an annual basis. Regular gynecologic follow-up is recommended.

- Management of the axilla: Approximately 30% of patients with clinically negative exams will have positive lymph nodes in an axillary lymph node dissection (ALND) specimen. The presence and number of lymph nodes involved affect staging and thus prognosis. However, complications are not infrequent (see later discussion). Thus, sentinel lymph node biopsy was developed to provide sampling of the lymph nodes without needing an ALND.
  - SLNB has been established as a standard of care for predicting axillary involvement in most patients with breast cancer. The procedure requires a multidisciplinary approach, including nuclear medicine, pathology, and radiology.
  - It involves injection of blue dye (either Lymphazurin or methylene blue) in the operating room and/or technetium-labeled sulfur colloid (in the nuclear medicine department, radiology suite, or sometimes by the surgeon). The combination of blue dye and radioisotope provides higher node identification rates and increases the sensitivity of the procedure. The goal is to identify the primary draining lymph node(s) in the axillary nodal basin.
  - A variety of injection techniques are used: intraparenchymal versus intradermal (intradermal methylene blue will cause skin necrosis at the injection site); peritumoral versus periareolar.
  - The SLN is identified by its blue color, and/or by high activity detected by a handheld gamma probe, or by a blue lymphatic seen to enter a nonblue node. Palpable nodes are also sentinel nodes, even if not blue or radioactive.
  - 20% to 30% of the time more than one SLN is identified.
  - Experienced surgeons (those who have performed at least 30 SLNBs, with ALND for confirmation) can identify the SLN in greater than 90% of patients, accurately predicting the patients' remaining axillary lymph node status in greater than 97% of cases.
  - If the SLN is positive for metastasis (micrometastasis 0.2 mm or larger, not isolated tumor cells), a standard ALND is performed. Radiation therapy can be administered to the axilla if the patient refuses ALND.
  - Serial sectioning and immunohistochemical staining of SLNB specimens may improve accuracy in detecting micrometastatic disease.
  - Currently, isolated tumor cells are considered N0 disease, and therapeutic decisions should not be based on finding these.
  - ALND. Patients with clinically positive lymph nodes, with positive SLN, or with positive should undergo ALND for local control. ALND involves the following:
  - Removal of level I and level II nodes and, if grossly involved, possibly level III nodes. Motor and sensory nerves are preserved unless there is direct tumor involvement.
  - An ALND should remove 10 or more nodes. The number of nodes identified is often pathologist dependent.

        - Patients with 4 or more positive lymph nodes should undergo adjuvant radiation to the axilla. Selective patients with 1 to 3 positive nodes may also benefit from radiation therapy to the axilla.
        - Intraoperative complications: potential injury to the axillary vessels and neuropathy secondary to injury to the motor nerves of the axilla (the long thoracic, thoracodorsal, and medial pectoral nerves).
        - Most frequent postoperative complications: wound infections and seromas. Persistent seroma may be treated with repeated aspirations or reinsertion of a drain. Other complications include pain and numbness in the axilla and upper arm, impaired shoulder mobility, and lymphedema.

- Adjuvant chemotherapy is given in appropriate patients after completion of surgery.
    - All node-positive patients should receive adjuvant chemotherapy.
        - Regimens are guided by the tumor biomarkers. Typical regimens comprise four to eight cycles of a combination of cyclophosphamide and an anthracycline, followed by a taxane administered every 2 to 3 weeks.
        - Patients with ER-positive tumors receive adjuvant hormonal therapy for 5 years. Tamoxifen is given to premenopausal women, and aromatase inhibitors are given to postmenopausal women (aromatase inhibitors are not used in premenopausal women).
        - In postmenopausal women older than 70 years, chemotherapy is performed less frequently. In postmenopausal women with tumors with ER or PR positivity, tamoxifen or an aromatase inhibitor is frequently the sole adjuvant medical therapy.
        - In patients with Her2/neu-positive tumors, polychemotherapy is combined with biological therapy targeting the Her2/neu protein: Trastuzumab is a recombinant monoclonal antibody that binds to Her2/neu receptor to prevent cell proliferation. The NSABP trial B-31 and the North Central Cancer Treatment Group trial N9831 showed that adding trastuzumab to a chemotherapy regiment of doxorubicin, cyclophosphamide, and paclitaxel was associated with an increase in the disease-free survival by 12% and a 33% reduction in the risk of death at 3 years. It is usually administered intravenously monthly for 12 months. The most serious toxicity with the regiment was cardiac failure.
    - Node-negative patients may have increased disease-free survival from adjuvant chemotherapy and/or hormonal therapy. An individualized approach is crucial and requires thorough discussion with the patient regarding the risks of recurrence without adjuvant therapy, the cost and toxicities treatment, and the expected benefit in risk reduction and survival.
        - Up to 30% of node-negative women die of breast cancer within 10 years if treated with surgery alone.
        - Node-negative patients who are at high risk and benefit the most from adjuvant chemotherapy include those with tumors greater than 1 cm, higher

tumor grade, Her2/neu expression, aneuploidy, Ki-67 expression, increased percentage in S phase, lymphovascular invasion, and ER/PR-negative tumors.
    - The NSABP B-20 trial and the International Breast Cancer Study Group trial IX showed that polychemotherapy in combination with tamoxifen was superior to tamoxifen alone in increasing disease-free and overall survival, especially in ER-negative patients, regardless of tumor size.
    - The St. Gallen Consensus Panel in 1998 suggested that patients who have node-negative disease and whose tumors are 1 cm or less and ER-positive may be spared adjuvant chemotherapy but still may benefit from tamoxifen.

- Adjuvant radiation
    - Indications for adjuvant radiation to the chest wall and axilla after mastectomy include T3 and T4 tumors, attachment to the pectoral fascia, positive surgical margins, skin involvement, involved internal mammary nodes, inadequate or no axillary dissection, four or more positive lymph nodes, and residual tumor on the axillary vein. Presence of one to three positive axillary nodes is a relative indication.
    - Randomized, prospective trials have shown a significantly decreased recurrence and improved survival in premenopausal women with these indications treated with chemotherapy and radiation therapy. Adjuvant whole-breast radiation after BCT decreases the breast cancer recurrence rate from 30% to less than 7% at 5 years.

Complications: Radiation to the chest wall can cause skin changes. Infrequent complications include interstitial pneumonitis, spontaneous rib fracture, breast fibrosis, pericarditis, pleural effusion, and chest wall myositis. Radiation to the axilla can increase the incidence of lymphedema and axillary fibrosis. Angiosarcoma can occur as a late complication.

3. Write short notes on: 5 x 6
(a) Duodenal fistula
(b) Closed loop obstruction.
(c) Diabetic ulcer.
(d) Dieulafoy's lesion.
(e) Postoperative fever.

Answer.
(a) Duodenal fistula.

Definition: An opening through the duodenal wall and into the peritoneal cavity, into another organ, or through the abdominal wall.

Causes:
- Surgical repair of perforated duodenal ulcer may be followed by external duodenal fistula (EDF).

- Blunt abdominal trauma – duodenal injury.
- Accidental trauma during – biliary surgery, right hemicolectomy, nephrectomy etc.
- Leakage from duodenal stump from gastric resection.

Contributing factors:
- Infection.
- Hematoma formation.
- Impaired blood supply.
- Poor nutrition
- Improperly placed drains.

Types: End type and lateral type.

Effect: This condition is associated with electrolyte imbalance, iron malabsorption, malnutrition, and exposure of tissues to copious amount of enzymes-rich secretions resulting in skin excoriation and sepsis.

Diagnosis: If drain is placed – see the drain fluid – if there is any suspicion (bile) – give methylene blue dye orally – see the colour of drain fluid.
USG – to see intra abdominal collection.
CTscan – to see the site and extent.

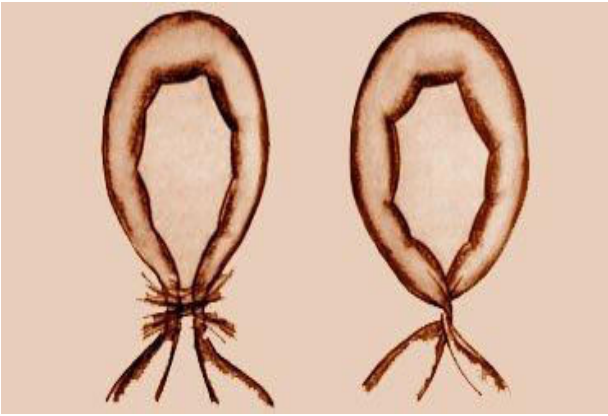
Treatment:
- Surgery in such patients, who are often malnourished and hypercatabolic, is associated with high morbidity and mortality rates.
- Fistula output of >500 mL/day was defined as a high-output fistula. Fistula sites were managed using a variable combination of techniques, including continuous slow suction, ileostomy bag, wound manager and silver paint.
- Serum electrolytes, serum protein and hemoglobin were monitored. All patients should undergo abdominal ultrasonography to look for any intra-abdominal collection. If collections were found, they should be managed by ultrasound-guided pigtail drainage/aspiration or drainage at laparotomy.
- Blood and fluid cultures were obtained and broad-spectrum antibiotics administered according to sensitivity profile of the organisms grown.
- The patients who were initially managed with non-operative treatment must be followed by surgery if required, for failure of fistula closure by conservative treatment. Peritonitis or failure to introduce the Ryle's tube into the jejunum for enteral feeding prompted early surgery.
- TPN.
- Conservative treatment followed by late surgery achieved healing/control of fistula in all patients.An enteral feeding line should be established at the earliest. Failure to establish nasojejunal feeding or peritonitis warrants early surgery.

- The surgical approaches in the management of duodenal fistula include exteriorization, exclusion, closure, or excision of fistula, i.e., Billroth II gastrectomy. The exteriorization procedure converts an uncontrolled duodenal leak into a controlled external duodenal fistula. It is useful in sick, hypoproteinemic and poor-risk patients. Pyloric exclusion, which diverts gastric juice away from the fistula, is indicated in large perforations that are difficult to close. Jejunal serosal patch or Roux-en-Y anastomosis may be other options in these difficult situations.
- Cholecysto-duodenoplasty has also been claimed to be a successful technique in high-output duodenal fistula. It consists of mobilizing the gall bladder and anastomosing its fundus with the duodenum bearing the fistula. Others have used rectus abdominis muscle flap to close large duodenal fistula and have reported good results.
- Gastrectomy (Billroth II) may be recommended in large perforations with or without accompanying bleed in young patients with early peritonitis, however, it is associated with high mortality in the presence of sepsis.

(b) Closed loop obstruction.

Definition: Closed loop obstruction is a specific type of obstruction in which two points along the course of a bowel are obstructed at a single location thus forming a closed loop.

- Usually this is due to adhesions, a twist of the mesentery or internal herniation.
- In the large bowel it is known as a volvulus.
- In the small bowel it is simply known as small bowel closed loop obstruction. Especially in the small bowel the risk of strangulation and bowel infarction is high with a mortality rate of 10-35%.



Small bowel closed loop obstruction: When we have a patient in the ER with what appears to be a small bowel obstruction (SBO), the most important thing we can do, besides making the diagnosis, is to identify the presence or absence of strangulation.

Strangulation is defined as obstruction associated with vascular compromise. The morbidity and mortality rate in the SBO-group is mainly due to bowel infarction and subsequent necrosis.
This is most commonly caused by a closed loop obstruction.

Straight X –ray abdomen – can show multiple air – fluid level.

CT is the imaging procedure of choice in the evaluation of patients suspected of SBO.

The CT-presentation of a closed loop obstruction in the small bowel depends on two things:

- Length of the bowel segment that forms the closed loop
- Orientation of the loop in relation to the imaging plane

Some or all of the following signs may be demonstrated on CT:

- Marked distension of a segment of small bowel
- Radially distributed, c or u-shaped small bowel loops
- "beak sign" : of the tapering bowel loops at the point of obstruction
- "whorl sign" : of the tightly twisted mesentery
- Two adjacent collapsed loops of bowel
- If strangulation is present, signs of bowel ischaemia.

Management:

- Resuscitation with IV fluid , nasogastric suction.
- Operative intervention.

(c) Diabetic ulcer.

Introduction: A diabetic foot is a foot that exhibits any pathology that results directly from diabetes mellitus or any long-term (or "chronic") complication of diabetes mellitus. Presence of several characteristic diabetic foot pathologies is called diabetic foot syndrome. These are thus umbrella terms.

One of the serious foot complications in diabetes is: Diabetic foot ulceration. It occurs in 15% of all patients with diabetes and precedes 84% of all lower leg amputations.

- Diabetics are prone to foot ulcerations due to both neurologic and vascular complications.
- Peripheral neuropathy can cause altered or complete loss of sensation in the foot and /or leg. Similar to the feeling of a "fat lip" after a dentist's anesthetic injection, the diabetic with advanced neuropathy looses all sharp-dull discrimination. Any cuts or trauma to the foot can go completely unnoticed for days or weeks in a patient with neuropathy. It's not uncommon to have a

patient with neuropathy tell you that the ulcer "just appeared" when, in fact, the ulcer has been present for quite some time. There is no known cure for neuropathy, but strict glucose control has been shown to slow the progression of the neuropathy.

- Charcot foot deformity occurs as a result of decreased sensation. People with "normal" feeling in their feet automatically determine when too much pressure is being placed on an area of the foot. Once identified, our bodies instinctively shift position to relieve this stress. A patient with advanced neuropathy looses this important mechanism. As a result, tissue ischemia and necrosis may occur leading to plantar ulcerations. Microfractures in the bones of the foot go unnoticed and untreated, resulting in disfigurement, chronic swelling and additional bony prominences.
- Microvascular disease is a significant problem for diabetics and can lead to ulcerations. It is well known that diabetes is called a small vessel disease. Most of the problems caused by narrowing of the small arteries cannot be resolved surgically. It is critical that diabetics maintain close control on their glucose level, maintain a good body weight and avoid smoking in an attempt to reduce the onset of small vessel disease.

Treatment:

- First, determine the cause of this ulcer. Is it neuropathic, ischemic or a combination?
- Treatment protocol should be based on the etiology of the ulcer.
- Assuming that there is adequate perfusion to heal a plantar ulcer, one should have appropriate shoe modifications made to disperse weight away from the ulcerative area. Absorb any excess discharge and maintain a moist wound environment with appropriate product selection. Keep the wound edges dry. Make sure no sinus tracking occurs. Watch for infection. Debride necrotic debris and the hyperkeratotic rim as they are niduses of for infection.

- Successful treatment of diabetic foot ulcers consists of addressing these three basic issues: debridement, offloading, and infection control.

Debridement:

- Debridement consists of removal of all necrotic tissue, peri-wound callus, and foreign bodies down to viable tissue. Proper debridement is necessary to decrease the risk of infection and reduce peri-wound pressure, which can impede normal wound contraction and healing. After debridement, the wound should be irrigated with saline or cleanser, and a dressing should be applied.

- Dressings should prevent tissue dessication, absorb excess fluid, and protect the wound from contamination. There are hundreds of dressings on the market, including hydrogels, foams, calcium alginates, absorbent polymers, growth factors, and skin replacements. Becaplermin contains the β-chain platelet-derived growth factor and has been shown in double-blind placebo-controlled trials to

significantly increase the incidence of complete wound healing. Its use should be considered for ulcers that are not healing with standard dressings.

- In case of an abscess, incision and drainage are essential, with debridement of all abscessed tissue. Many limbs have been saved by timely incision and drainage procedures; conversely, many limbs have been lost by failure to perform these procedures. Treating a deep abscess with antibiotics alone leads to delayed appropriate therapy and further morbidity and mortality.

Offloading:

- Having patients use a wheelchair or crutches to completely halt weight bearing on the affected foot is the most effective method of offloading to heal a foot ulceration. Total contact casts (TCCs) are difficult and time consuming to apply but significantly reduce pressure on wounds and have been shown to heal between 73 and 100% of all wounds treated with them. Armstrong et al.4 have achieved similar healing rates with an "instant TCC," made by wrapping a removable cast walker with a layer of cohesive bandage or plaster of Paris. Inappropriate application of TCCs may result in new ulcers, and TCCs are contraindicated in deep or draining wounds or for use with noncompliant, blind, morbidly obese, or severely vascularly compromised patients.

- Clinicians often prefer removable cast walkers because they do not have some of the disadvantages of TCCs. Removability is an advantage in that it allows for daily wound inspection, dressing changes, and early detection of infection. But removability is also the greatest disadvantage in that studies have shown that patients wear them only ~ 30% of the time they are walking (usually to and from the doctor's office).

- Postoperative shoes or wedge shoes are also used and must be large enough to accommodate bulky dressings. Proper offloading remains the biggest challenge for clinicians dealing with diabetic foot ulcers.

Infection control:

- Limb-threatening diabetic foot infections are usually polymicrobial. Commonly encountered pathogens include methicillin-resistant *staphylococcus aureus*, β-hemolytic streptococci, enterobacteriaceae, *pseudomonas aeruginosa*, and enterococci. Anaerobes, such as *bacteroides, peptococcus*, and*peptostreptococcus*, are rarely the sole pathogens but are seen in mixed infections with aerobes. Antibiotics selected to treat severe or limb-threatening infections should include coverage of gram-positive and gram-negative organisms and provide both aerobic and anaerobic coverage. Patients with such wounds should be hospitalized and treated with intravenous antibiotics.

- Mild to moderate infections with localized cellulitis can be treated on an outpatient basis with oral antibiotics such as cephalexin, amoxicillin with clavulanate potassium, moxifloxacin, or clindamycin. The antibiotics should be started after initial cultures are taken and changed as necessary.

(d) Dieulafoy's lesion.

- Dieulafoy's lesions are vascular malformations found primarily along the lesser curve of the stomach within 6 cm of the gastroesophageal junction, although they can occur elsewhere in the GI tract.
- They represent rupture of unusually large vessels (1-3 mm) that are found in the gastric submucosa.
- Erosion of the gastric mucosa overlying these vessels leads to hemorrhage. The mucosal defect is usually small (2-5 mm) and may be difficult to identify. Given the large size of the underlying artery, bleeding from a Dieulafoy's lesion can be massive.
- Initial attempts at endoscopic control are often successful.
- Application of thermal or sclerosant therapy is effective in 80% to 100% of cases.
- In cases that fail endoscopic therapy, angiographic coil embolization can be successful.
- If these approaches fail, surgical intervention may be necessary; owing to difficulties in visualization and palpation of these lesions, prior endoscopic tattooing can facilitate the procedure.
- A gastrotomy is performed, and attempts are made at identifying the bleeding source. The lesion can then be oversewn.
- In cases in which the bleeding point is not identified, a partial gastrectomy may be necessary.

(e) Postoperative fever.

Introduction: Postoperative fever is a common condition challenging doctors to find the right diagnosis, because it can be a hallmark of serious underlying conditions. Between 40-50% of surgical patients develop postoperative fever depending on type of surgery but only a small percentage turn out to be due to infection.

Causes: The most common causes have been summarized in a handy mnemonic: the five W's. These tend to occur at specific days after surgery (postoperative days or POD).

Category	Day	Description
Wind	POD1-2	the lungs, i.e. pneumonia, aspiration, and pulmonary embolism. Once attributed to atelectasis, but this has been shown to be inaccurate.
Water	POD3-5	urinary tract infection, related to indwelling catheter (during surgery or currently i.e. Foley catheter)

Wound	POD5-7	surgical site infection, which in obstetrics or gynaecology, may refer to the uterus.
Walking (or VEINS, which then sounds like "Weins")	POD4-6	deep vein thrombosis or pulmonary embolism
Wonder drugs or "What did we do?"	POD7+	drug fever, infections related to intravenous lines

Causes of Fever (think of five W's: Wind, Water, Wound, Walking, Wonderdrug)
Workup: CBC with differential, CXR, 2 blood cultures, UA, Urine Cx

1) Atelectasis (POD 1-2)
    a. Most common cause of post-op fever
    b. Risk factors: prolonged immobilization, smoking hx, lung dz
    c. Sx: low grade fever, rales on auscultation, tachycardia, tachypnea
    d. Tx: bronchoscopy
2) Pneumonia (POD 1-2)
    a. Risk factors:
        i. Respirator use (Pseudomonas, Klebsiella)
            1. Tx: aminoglycosides + penicillin
        ii. Aspiration
            1. Tx: NGT for decompression, oxygenation
    b. Sx: cough, sputum production; Vitals: fever, ↑ RR
    c. Findings: CXR infiltrate, Leukocytes on CBC
3) UTI (POD3+)
    a. 5% chance per each day foley is in
    b. Sx: burning sensation upon urination; no pain if Foley still in
    c. Risk factors: prostate hypertrophy, diabetes, catheters, urinary retention
    d. Microorganisms: Pseudomonas, Serratia, Enterococcus, E.Coli, Proteus, Klebsiella
    e. Findings: leukocyte esterase and nitrite changes on UA; growth on Urine Cx, leukocytes on CBC
    f. Tx: remove foley, increase fluids, Abx
4) Wound (POD 5+)
    a. Sx: >2cm erythema, swelling, warmth, pain around incision site
    b. Microorganisms: Staph most common
    c. GI infections: E. coli, Bacteroides, Enterococcus
    d. GU infections: Pseudomonas, Proteus

    e. Tx: drain and wet-to-dry dressings twice daily
    f. Other complications: hematoma, seroma, dehiscence (fascial opening)
5) DVTs (POD7+)
    a. Sx: leg edema, tender superficial veins, chest pain, tachycardia, tachypnea, SOB
    b. Studies: Doppler, venogram, CT (look for occlusion in pulmonary arteries)
    c. Tx: ambulate, SCDs, systemic heparin, consult cardio
6) Medications – drug rxn can occur anytime
7) Line infections
    a. IV lines need to come out after 72hrs
    b. Sx: fever, leukocytosis on CBC, bacteremia on blood Cx, (+) culture from line tip
    c. Micro: S. aureus, S. epidermidis
    d. Tx: removal of line and +/- IV Abx
8) Peritonitis
    a. Perforation of GI tract → abdominal pain, fever, sepsis
    b. Studies: KUB (check for free air)
    c. Tx: NPO with NGT, Abx, IVF, operate to irrigate abdomen
    d. Complications: abscess formation
        i. Tx: localize with CT scan or Gallium study (xrays while injecting radioactive gallium into bloodstream), drain, Abx

Fever occuring during operation

1) Transfusion reaction
    a. Sx: fever, excess bleeding, urticaria, red urine
    b. Tx: stop transfusion, hydrate with NS, mannitol for osmotic diuresis
2) Intraoperative Septicemia
    a. Release of abscess
    b. Tx: copious irrigation
3) Malignant hyperthermia
    a. Sx: fever, tachycardia, tachypnea, acidosis with hyperkalemia, shock
    b. Causes: halothane, succinylcholine, MAOi + meperidine
    c. Tx:
        i. stop surgery/anesthesia
        ii. dantrolene (muscle relaxant)
        iii. cool patient
        iv. hyperventilate to ↓ acidosis
        v. bicarb + insulin to ↓ hyperkalemia and ↓ acidosis
        vi. mannitol for diuresis
    d. mortality 60%

Fever occuring few hours post-op

1) Thyroid storm
2) Addisonian crisis

    a. Tx: resume steriod therapy for Addison's dz
3) EtOH withdrawal
    a. Sx: low grade fever, tremors, seizures
    b. Wernicke's encephalopathy: confusion, nystagmus, ataxia
    c. Korsakoff's: confabulation, amnesia caused by damage to mamillary bodies
    d. Tx: thiamine
4) Anastomotic leak
5) Clostridium perfringens wound infection

4. Write brief answers: 4 x 7.5

(a) Child's criteria for assessment of hepatic function
(b) Neurogenic bladder
(c) Fistula – in –ano
(d) Strangulated hernia.

Answer.

(a) Child's criteria for assessment of hepatic function .

Introduction: Determination of Functional Liver Reserve is necessary for the patients who are going to have hepatic surgery.

Pugh's Modification of Child's Grading of Cirrhosis			
Measurements	**1 point**	**2 points**	**3 points**
Bilirubin (mg/dL)	1–1.9	2–2.9	>2.9
Prothrombin time prolongation (secs)	1–3	4–6	>6
Albumin (g/dL)	>3.5	2.8–3.4	<2.8
Ascites	none	mild	moderate to severe
Encephalopathy	none	grade 1 or 2	grade 3 or 4

Interpretation : Child's A: 5–6 points; B: 7–9 points; C: 10–15 points.

Functionally well compensated cirrhosis is classified as Pugh-Child's classification grade A; decompensating cirrhosis is grade B; and decompensated cirrhosis is grade C.

Usefulness: Generally, partial hepatectomy is only offered to patients who are Pugh-Child's A and the most favorable class B patients.

In general, Child's class C patients are only offered supportive care, since even nonsurgical ablative methods such as embolization is associated with a procedure related mortality in one-third of patients.

(b) Neurogenic bladder

Introduction: Neurogenic bladder refers to dysfunction of the urinary bladder due to disease of the central nervous system or peripheral nerves involved in the control of micturition (urination).

Causes

- Neurogenic bladder is often associated with spinal cord diseases (such as Syringomyelia/Hydromyelia), injuries, and neural tube defects including spina bifida.
- It may also be caused by brain tumors and other diseases of the brain, and by peripheral nerve diseases.
- It is a common complication of major surgery in the pelvis, such as for removal of sacrococcygeal teratoma and other tumors.
- Other disorders such as syphilis, diabetes mellitus, stroke, ruptured or herniated intervertebral disk.
- Degenerative neurological diseases such as multiple sclerosis and amyotrophic lateral sclerosis.
- Long term effect of alcoholism.

Symptoms:
Symptoms include the following:
- Urinary incontinence, characterized by either involuntary release of large volumes of urine or continuous dribbling of small amounts. Bed-wetting may occur.
- Frequent urination
- Persistent urge to urinate despite recent voiding; a constant feeling that the bladder is not completely empty
- Pain or burning on urination

Diagnosis of Neurogenic Bladder:

- A thorough patient history is essential to record 24-hour urination patterns, including the actual volume of urine voided, how urgent the feeling is to urinate and any factors that aggravate incontinence.
- Physical examination will likely include a rectal, genital, and abdominal exam to check for enlargement of the bladder or other abnormalities. A complete neurological examination is also essential. Tests to measure urine output are conducted.
- To determine whether urine is retained after voiding, the doctor may use an ultrasound-like instrument that estimates the amount left in the bladder or insert a catheter into the bladder.

- In order to detect whether leakage occurs, a full-bladder stress test may be necessary. The bladder is filled to capacity via a catheter and the patient is then asked to bend over, cough, or walk. Urine or blood samples may be taken to look for abnormalities including infection and underlying disorders that might be causing or aggravating the condition.

Treatment of Neurogenic Bladder:

- Neurogenic bladder usually causes difficulty or full inability to pass urine without use of a catheter or other method. Catheterization methods range from intermittent catheterization, which involves no surgery or permanenty attached appliances, to the creation of a stoma, which bypasses the urethra to empty the bladder directly.
- Treatment is aimed at enabling the bladder to empty completely and regularly, preventing infection, controlling incontinence, and preserving kidney function.
- A urinary catheter can be used continuously by patients who have sudden, unexpected bladder contractions. Women usually fare better with such therapy; men are more prone to develop urinary tract infections and complications, including abscess formation.
- Patients suffering from bladder paralysis can be taught to insert a catheter several times a day to drain the bladder completely and so prevent urine retention that may lead to bladder stones and infection. Various medications may help improve bladder muscle control and prevent involuntary muscle contractions. Muscle relaxants, antispasmodics and anticholinergic drugs are also helpful.
- Bethanechol is the most commonly prescribed drug to help stimulate bladder contractions in patients who retain urine. Surgery may be performed to widen the sphincter to decrease resistance in the bladder outlet and thus maximize bladder emptying. In other cases, the sphincter or lower pelvic muscles may be surgically tightened to improve bladder control. In very severe cases, surgery may be done to reroute the flow of urine so that it empties into an externally worn receptacle.
- Function of the stoma may be augmented by periodic injections of botulinum toxin to relax one of the two sphincters involved in normal urination. The effect is longer lasting with botulinum toxin **type A** than with **type B**. This use of botulinum toxin is discussed at length in the French medical literature.

(c) Fistula in Ano.

Introduction: A fistula is an abnormal connection of two epithelial surfaces and the two surfaces joined in fistula in ano are the anorectal lining and the perineal or vaginal skin. Very common, especially in otherwise fit young adults. May occur in the presence of Crohn's disease. Minor association with obesity and diabetes mellitus. Very rarely due to trauma or ulceration of anorectal tumours.

Causes of fistula – in –ano:

Persistent cryptoglandular sepsis	Malignancy :Anal ,Rectal	Foreign body	Pilonidal disease
Anal fissure	Leukemia or lymphoma	Inflammatory bowel disease	Lymphogranuloma venereum
Trauma	Other pelvic malignancy	Diverticulitis	Osteomyelitis
Assault	Radiation injury	Actinomycosis	Urethroperineal fistula
Iatrogenic	Anal intercourse	Hidradenitis suppurativa	AIDS

Commonest cause is sepsis arising in an anal gland that forces its way out through the anal tissues to appear in the perianal or, in women, vaginal skin (cryptoglandular theory of fistula in ano). Often presents initially as an acute perianal abscess. The tissues through which the track pushes determines the classification of fistulas.

Classification of Anorectal Fistulas

Intersphincteric (the most common): The fistula track is confined to the intersphincteric plane.
Trans-sphincteric: The fistula connects the intersphincteric plane with the ischiorectal fossa by perforating the external sphincter.
Suprasphincteric: Similar to trans-sphincteric, but the track loops over the external sphincter and perforates the levator ani.
Extrasphincteric: The track passes from the rectum to perineal skin, completely external to the sphincteric complex.

Clinical features:

- Acute perianal abscess. Rapid onset of severe perianal or perineal pain. Swelling and erythema of the perianal skin with fever and tachycardia.
- Recurrent perianal sepsis. Recurrent intermittent sepsis typified by gradual build-up of pressure sensation and swelling in the perianal skin and eventual discharge of blood-stained purulent fluid.
- Chronic perianal discharge. Persistent low grade sepsis of the track with chronic discharge of seropurulent fluid via a punctum that is usually clearly identified by the patient.

Diagnosis and investigations: Diagnosis and investigation should aim to confirm the presence of a fistula and identify the course of the track to determine the type of fistula.

- Examination of the perineum and rectal examination may reveal a palpable fibrous track.
- Examination under anaesthetic (EUA) with probing of any external opening to aid identification of the course of the track.
- Endoanal ultrasound (sometimes with hydrogen peroxide injected into the track) identifies the course of the track.
- MRI scanning is probably the most sensitive method of determining the course of the track and identifying any occult perianal or pelvic sepsis.
- Flexible sigmoidoscopy if associated colorectal disease, e.g. Crohn's disease, is suspected.

  Goodsall described a means of predicting the offending crypt and internal opening based on the location of the external opening. In brief, an external opening posterior to an imaginary coronal plane bisecting the anus would have an internal opening at the posterior midline, whereas an external opening anterior to this plane would have an internal opening radially directed straight to the nearest anal gland. Goodsall's rule has been shown to be more accurate for women than men and more accurate for posterior than anterior openings. Goodsall's rule has been shown to be misleading for external openings more than 3 cm from the anal verge.

Treatment:

Medical treatment:

- Antibiotics may reduce symptoms from recurrent sepsis but cannot treat the underlying fistula.
- Medical treatment of inflammatory bowel disease may dramatically reduce symptoms from associated fistulas.

Surgical treatment:

Fistulotomy : Single stage Multiple stage	Seton: Cutting Noncutting Draining (Crohn's disease)	York-Mason posterior transsphincteric approach
Anorectal mucosal advancement flap	Fibrin glue filling of tract	Remicade (Crohn's disease)
Dermal island advancement flap		Fistulectomy

Principles of surgical treatment are as follow:

- Drainage of any acute sepsis if present.

- Prevention of recurrent sepsis usually by insertion of a loose seton suture, e.g. silastic sling.
- Low fistula in ano. Lay open track, remove all chronic granulation tissue, and allow to heal spontaneously (fistulotomy). Little risk of impairment of continence due to minimal division of sphincter tissues.
- High fistula in ano.

    - Remove fistula track and close the internal opening (core fistulectomy and endorectal flap advancement).
    - Slowly divide the sphincter tissue between the fistula and the perianal skin (cutting seton): low risk of incontinence.
    - Fill the fistula with fibrin glue.

(d) Strangulated hernia.

Introduction: Strangulated hernias refer to hernias that have their blood supply cut off. When this occurs, any tissue in the hernia, such as intestines, can become ischemic and die quickly. This means that quick surgery is imperative to save the herniated tissue.

Symptoms of a strangulated hernia:

- They are irreducible, are always painful
- Extreme tenderness and redness in the area of the bulge
- Sudden pain that worsens in a short period of time
- Fever
- Tachycardia
- Vomiting , nausea
- Severe infection
- Also, the affected person may feel ill in general

Management:

- Diagnosis should be done on the basis of history and clinical examination. Straight x – ray abdomen can help in the diagnosis.
- Proper resuscitation with aggressive IV fluid, IV antibiotic, nasogastric suction and catheterization should be done.
- Patient should be prepared for emergency operation – on exploration see the viability of the gut – and operative intervention – either resection anastomosis or stoma.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES

MS (General Surgery) Examination, 2007

PAPER II

Time Allowed: 3 Hours **Full Marks:100**

1. Enumerate the premalignant lesions of the oesophagous & discuss pathogenesis of oesophageal carcinoma with reference to staging . Outline the treatment of carcinoma lower third of oesophagous. (20)

2. Classify thyroid tumours. Discuss the management of well differentiated carcinoma of thyroid. (20)

3. Write short notes on: 5 x 6

(a) Raynaud's phenomenon.

(b) Necrotising fascitis.

(c) Melanoma.

(d) Diagnosis and localization of insulinomas.

(e) Solitary rectal ulcer.

4. Answer in brief: 4 x 7.5

(a) Gastrointestinal stromal tumours.

(b) Management of recurrent varicose vein.

(c) Intraoperative radiotherapy for breast cancer.

(d) Approach to obstructive jaundice.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES
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April, 2007

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1. Enumerate the premalignant lesions of the oesophagous & discuss pathogenesis of oesophageal carcinoma with reference to staging . Outline the treatment of carcinoma lower third of oesophagous. (20)

Answer. Premalignant lesions of the oesophagous are:

High Risk:	Intermediate Risk:	Low Risk:
• Tylosis type A. • Plummer – Vinson Syndrome.	• Reflux disease and Barrett's oesophagous • Achalasia • Ectopic gastric mucosa • Previous squamous cell carcinoma of head and neck. • Radiotherapy of Hodgkin's or non – Hodgkin's lymphomas	• Oesophageal diverticula • Corrosive strictures • Celiac disease • Scleroderma.

Pathogenesis:
There are two major forms of this disease, esophageal squamous cell carcinoma (ESSC) and adenocarcinoma.

Other subtypes such as mucoepidermoid, adenosquamous, adenoid cystic, undifferentiated, malignant melanoma, and small cell are also seen with much less frequency.

ESSC	Adenocarcinoma
• Remains the most common form worldwide. • ESSC is thought to originate as an epithelial dysplasia, which initially develops as a mild to severe dysplasia and eventually becomes invasive carcinoma. • Smoking and substantial alcohol intake are the risk factors for the vast majority of ESSC cases. • Only a small proportion being familial. • SC is more prevalent in the upper 2/3 thoracic esophagus, and is associated with chronic mucosal injury.	• The incidence of esophageal adeno carcinoma has risen remarkably over last 20 years. • Adenocarcinoma is linked to chronic gastroesophageal reflux disease (GERD) which can lead to the development of metaplasia (Barrett's esophagus; 5–8% of GERD population). • Progression occurs from metaplasia to dysplasia and ultimately to adenocarcinoma in 0.5% per year. • AD is usually confined to the lower esophagus, including the GE junction.

Factors Associated with Pathogenesis of Esophageal Cancer:

Factor	Squamous Cell Cancer	Adenocarcinoma
Smoking	+++	+
Alcohol consumption	+++	–
Hot beverages	+	–
N-nitroso compounds (e.g., pickled vegetables)	+	–
Betel nut chewing	+	–
Maté drinking	+	–
Deficiencies of green vegetables and vitamins	+	–
Low socioeconomic class	+	–
Fungal toxin or virus	+	–
History of radiation to the mediastinum	+	+
Lye corrosive stricture	+	–
History of aerodigestive malignancy	+++	–
Plummer-Vinson syndrome	+	–
Obesity	–	++
Achalasia	+	–
Gastroesophageal reflux	–	+++
Barrett's esophagus	–	++++

American Joint Committee on Cancer (AJCC) TNM Classification for Esophageal Carcinoma

T0	No evidence of primary tumor
T1	Invades lamina propria or submucosa
T2	Invades muscularis propria
T3	Invades adventitia
T4	Invades adjacent structures
N0	No regional lymph node metastasis
N1	Regional lymph node metastasis
M0	No distant metastasis
M1	Distant metastasis
M1a	Invasion of celiac or cervical lymph nodes
M1b	Other distant metastasis

Stage 0	Tis N0 M0	
Stage I	T1 N0 M0	
Stage IIA	T2 N0 M0	T3 N0 M0
Stage IIB	T1 N1 M0	T2 N1 M0
Stage III	T3 N1 M0	T4 Any N M0
Stage IV	Any T Any N M1	

Treatment of carcinoma lower third of oesophagous:

DIAGNOSIS AND PRETREATMENT EVALUATION

- A ***complete physical examination*** and ***directed laboratory analysis*** should be done in patients suspected of having Carcinoma Oesophagous.
- ***Plain chest radiography*** is often unrewarding unless the esophagus is distended or an air fluid level is observed in the posterior mediastinum.
- Although the diagnosis of Carcinoma Oesophagous can be suspected on a ***barium contrast study***, ***endoscopy*** **is the only modality that provides a tissue diagnosis.** The diagnosis of esophageal cancer is made best from an endoscopic biopsy.

- During endoscopy, it is critical to document the following:

1.	Location of the lesion (with respect to distance from the incisors)
2.	Nature of the lesion (friable, firm, polypoid)
3.	Proximal and distal extent of the lesion
4.	Relationship of the lesion to the cricopharyngeus muscle, the GEJ, and the gastric cardia
5.	Distensibility of the stomach.

- ***Esophagoscopy*** allows direct visualization of mucosal abnormalities and histologic diagnosis from biopsies.

- ***Rigid esophagoscopy*** can be helpful when the lesion is in the cervical esophagus and endoscopic examination of the upper esophageal sphincter is important.

- ***Endoscopic ultrasound (EUS)*** combines conventional endoscopy with ultrasound technology:

- This examination can reveal the depth of mural penetration and invasion of tumor into the periesophageal tissue.
- The accuracy of this technique for assessing depth of tumor penetration (T status) 85% - 90%.
- EUS can also demonstrate extension of tumor into adjacent lymph nodes.

Malignant LN	Benign LN
Hypoechoic lymph nodes Sharply demarcated Larger than 5 mm	Hyperechoic pattern. Not sharply demarcated. Have an elongated bean shape.

EUS is not always able to differentiate between benign and malignant nodes.

- ***Computed tomography (CT)***:
  - Primarily used for preoperative staging.
  - Local extraesophageal extension into adjacent structures such as the aorta, tracheobronchial tree, or mediastinal lymph nodes can be detected at the time of initial presentation.
  - Approximately one third of patients show liver metastasis or abdominal lymphadenopathy.
- ***Magnetic resonance imaging (MRI)***:
  - Not performed routinely and adds to the staging of esophageal cancer in few circumstances.
  - To identify involvement of vascular and neural tissues, MRI is helpful.
  - It can accurately detect T4 lesions and metastatic lesions in the liver.
  - It overstages T and N status with only 74% accuracy.
- ***Positron emission tomography (PET)*** allows the identification of metabolically active areas as evidence of tumor involvement. One potential advantage of PET over CT is the detection of distant metastasis. Because of the possibility of false positive findings, histological confirmation of positive PET or CT findings are often necessary.
- ***Bronchoscopy*** is recommended to exclude tracheal or bronchial invasion in patients with tumors proximal to the carina or in patients with symptoms suggestive of aerodigestive fistula such as recurrent pneumonia or hemoptysis.
- Surgical staging modalities like ***thoracoscopy*** and ***laparoscopy*** have been used to define pretreatment staging but have not gained wide acceptance.

TREATMENT :

When a patient presents with esophageal cancer, the following variables are considered.

1. Histology, location, and local extent (depth of invasion) of the primary tumor
2. Status of the local and regional lymph nodes
3. Presence of distant lymph nodes or systemic disease
4. Overall condition of the patient (including nutritional status and ability to swallow)
5. Intended goal of treatment—curative or palliative

For cancers that are limited to the abdominal esophagus or gastric cardia cancers, an abdominal–right thoracic approach as in a Lewis-Tanner esophagectomy is one option, with the proximal stomach also resected in order to gain an adequate distal resection margin.

A left thoracoabdominal incision through the seventh or eighth rib space also gives excellent exposure of the low mediastinum and upper abdomen. A single left thoracotomy with opening up of the diaphragm is also an option. This gives reasonable exposure to the upper abdomen as well.

However, lymphadenectomy towards the hepatoduodenal ligament is hampered. When a thoracotomy is not desired, opening the hiatus widely by splitting the crura laterally and the diaphragm anteriorly can gain access to the low posterior mediastinum, and distal esophagectomy can be performed with the anastomosis performed from the abdomen without the need for a thoracic incision.

The anastomosis is made easier with a mechanical stapler. When the proximal stomach is involved by tumor, a total gastrectomy with Roux-en-Y reconstruction is preferred by many.

Transthoracic versus **Transhiatal** Resection

Transthoracic	Transhiatal
More thorough lymphadenectomy through a thoracotomy merely improves staging, but does not affect prognosis.	The operating time is shorter and postoperative morbidity is less with the transhiatal approach. Transhiatal resection more suitable for lower esophageal tumors for which much of the mobilization can be performed under vision.

From an oncological standpoint, the philosophy towards lymphadenectomy dictates the surgical approach.

Minimally Invasive Surgery

- Various combinations of minimally invasive approaches including thoracoscopy, laparoscopy, mediastinoscopy, hand-assisted laparoscopy, and open laparotomy and thoracotomy have been explored. The myriad of surgical methods tried implies a lack of consensus on which is superior.
- Potentially serious intraoperative complications can occur. These include bleeding from the azygos vein and from intercostal vessel, and injury to the aorta, tracheobronchial tree, and recurrent laryngeal nerve.The lack of tactile control is probably a contributory factor.
- Except for the few studies mentioned, clear advantages of the minimally invasive methods could not be demonstrated, partly because the number of patients studied generally was too small to have enough statistical power to demonstrate a difference. There are also other reasons why benefits are difficult to confirm. With

modern analgesic methods such as epidural analgesia, postoperative pain control is less critical a problem.

Extent of Resection: ***Axial*** and ***Lateral*** Margin

- An R_0 resection is consistently identified as the most important prognostic factor for long-term survival. An R_0 resection results in total removal of the tumor mass (primary and lymph nodes) with clear proximal, distal, and lateral margins.
- Chance of a histologically positive margin reduces with increasing distance at which the esophagus is transected away from the tumor edge, and that the frequency of anastomotic recurrence is a function of the length of proximal resection margin attained. Taking into account shrinkage of the specimen after resection, as a guide to surgery, an in-situ margin of 10 cm (fresh contracted specimen of approximately 5 cm) should be the goal, to allow a <5% chance of anastomotic recurrence.

❖Intraoperative frozen section is one method to ensure a negative margin.
❖A histologically involved resection margin does not necessarily lead to definite anastomotic recurrence, and a negative margin does not preclude anastomotic recurrence.
❖The occurrence of skip lesions or submucosal spread can be missed, so margins may be falsely negative.
❖Extramural recurrence with infiltration back to the anastomosis may also be indistinguishable from true anastomotic recurrence.
❖Patients who have positive histologic margins are those likely to have more advanced disease, and early recurrences at more distant sites.
❖Microscopic involvement of the lateral margin (macroscopically clear) results in increased chance of local recurrence and worse survival.
❖Obtaining a clear lateral margin is difficult with esophageal cancer because of its anatomic position and adjacent indispensable structures.

Extent of Resection: Lymphadenectomy

For early cancers (m1–m2 tumors involving tissue up to the lamina propria), the prevalence of lymphatic spread is negligible and thus lymph node dissection is not indicated. Instead these are readily treated by EMR.

For m3 tumors that have penetrated the lamina muscularis mucosa, and cancers that have infiltrated further into the esophagus (sm1 and deeper), the chance of lymphatic spread increases substantially, and lymphadenectomy is indicated. The optimal extent of lymphadenectomy is controversial. The ability to perform lymphadenectomy is closely related to the surgical approach utilized, and an open transthoracic approach is necessary, unless a limited lower mediastinal dissection is planned.

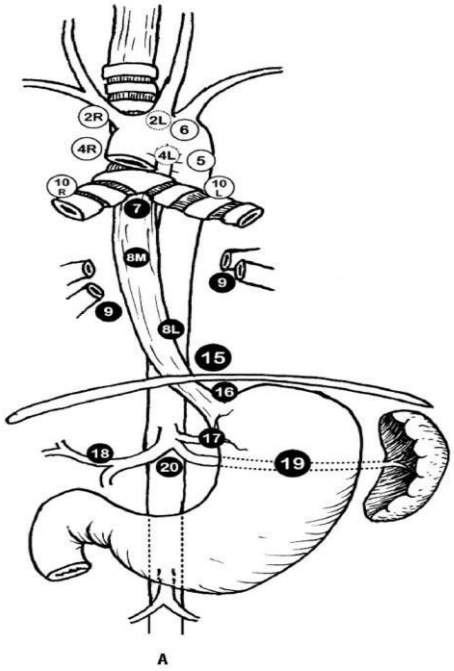
Conventional transthoracic resection usually involves a "standard two-field" lymphadenectomy, which entails removing the nodes and peri-esophageal tissue below the level of the carina, and the lymph node stations around the celiac trifurcation. When superior mediastinal lymph node dissection is performed, it is sometimes known as "extended two-field lymphadenectomy." "Three-field" lymphadenectomy involves additional bilateral cervical lymph node clearance .

The rationale for extensive lymphadenectomy is that lymph node spread occurs widely in esophageal cancer. When preoperative endoscopic injection of technetium-labeled rhenium colloid into the thoracic esophageal wall is performed, radioactivity can be detected to drain via lymphatics to all three fields. However, there is preferential lymphatic flow from the upper and middle esophagus to the neck and upper mediastinum and from the lower esophagus to the abdomen.

For Barrett's adenocarcinomas of the lower esophagus, presumably with less chance of superior mediastinal and cervical lymph node spread, radical lymphadenectomy mostly involves a standard infra-carinal and upper abdominal node dissection.

Removing positive lymph nodes in the neck may not be clinically significant.

Poor prognostic factors include: (1) when all three fields have metastatic nodes; (2) when a lower-third tumor has positive cervical nodes; and (3) when five or more lymph nodes are involved. These situations indicate advanced metastatic disease and three-field lymphadenectomy may not be justified. Replacing three-field lymphadenectomy with neoadjuvant, adjuvant, or intraoperative radiotherapy are alternatives, but their roles remain controversial.



Reconstruction after Esophagectomy

The reconstruction phase of an esophagectomy determines to a significant extent the postoperative morbidity and long-term quality of life. The most commonly used conduit is the gastric tube, and of the many configurations attempted, a tailored iso-peristaltic tube based on the greater curvature with preservation of the right gastric and right gastroepiploic vessels is most reliable.

- Disadvantages of the gastric conduit are that patients who had an intrathoracic stomach often experience postprandial discomfort and early satiety related to loss of normal gastric function such as receptive relaxation.
- Patients may also suffer from acid reflux, possible gastric ulceration, and dysfunctional propulsion.
- Barrett's esophagus has been reported to develop in the esophageal remnant, although the clinical relevance of this finding is at present unknown. These are important considerations, though in our experience serious problems are uncommon.

Patients who have a low intrathoracic anastomosis tend to have more severe reflux and esophagitis compared with the high intrathoracic or cervical anastomosis. Preserving a longer length of esophagus, on the other hand, theoretically may enhance swallowing function, although no conclusive data are available on this point. Inadequate gastric emptying can also be a problem

- There are instances when the stomach cannot be used, such as after previous gastric resection, or tumor involvement of a substantial part of the stomach dictating its removal. In these situations use of the colon is preferred.
- For most, colonic interposition remains an infrequently performed procedure and has the potential for more complications.
- Bowel preparation is required; mobilization of the loop is more complex; its blood supply is less reliable than that of the gastric conduit; three anastomoses are required; and when the colon becomes ischemic, the choice of alternative conduit is restricted.
- It has been suggested that a colon conduit is more durable, and the supposed long-term functional benefits of colon interposition make it the preferred esophageal substitute.
- A colonic conduit seems to have active peristalsis,
- This is cited as an explanation for its superior function as an esophageal substitute compared with a passive gastric conduit.

The jejunum is used most frequently after distal esophagectomy and total gastrectomy for cancer of the lower esophagus and gastric cardia. A Roux-en-Y configuration seems best, as it prevents bile reflux to the esophagus. A jejunal loop used in a modified Merendino procedure to interpose between the esophagus and proximal stomach after limited resection of the distal esophagus and gastroesophageal junction has also been advocated. Excellent postoperative quality of life and function is claimed. A long jejunal loop is sometimes used to reach the neck, but preparation is tedious and the vasculature may not be reliable, and "supercharge" involving microvascular anastomosis to cervical vessels may be required. A free jejunal graft is used for reconstructing the defect after resection of the pharyngo-esophageal segment in the neck.

When palliative resection is carried out for advanced tumor, recurrent tumor could infiltrate into the conduit placed in the posterior mediastinum. However, the site of the obstruction that produced dysphagia was not clearly stated.

Perioperative Care and Postoperative Morbidity and Mortality

With adequate preoperative work-up, serious cardiac events like myocardial infarction should be rare. Atrial arrhythmia is common, affecting about 20% of patients. In itself, atrial fibrillation is benign, but it serves as a marker for more serious underlying pulmonary and septic surgical complications. Occurrence of atrial arrhythmia should prompt a thorough search for a more ominous underlying cause.

Pulmonary complications remain the most common and serious form of postoperative morbidity. Predictive factors include advanced age, supracarinal tumor location, and lengthened operating time. Neoadjuvant therapy did not lead to increased morbidity. Measures to improve respiratory outcome include cessation of smoking preoperatively, chest physiotherapy, avoidance of recurrent laryngeal nerve injury, cautious fluid administration to avoid fluid overload, use of a smaller chest tube, early ambulation, regular bronchoscopy, and early tracheostomy for sputum retention. Epidural analgesia is invaluable in postoperative pain relief and has been shown to improve outcome.

The most common surgical complication after esophagectomy is still anastomotic leak, and the incidence can reach 30%. The actual method of anastomosis is perhaps less important than its proper application. Stapled anastomosis is popular for intrathoracic anastomosis, while the hand-sewn technique is preferred in the neck. The clinical presentation of postoperative anastomotic leak ranges from an asymptomatic radiographic finding to a florid thoracic infection. Early detection of anastomotic leaks is important so that timely intervention can be instituted; sometimes a high index of suspicion is important when other seemingly unrelated complications develop. Treatment principles dictate adequate drainage, whether by radiological, endoscopic, or surgical means. Maintenance of nutritional status is important, preferably via the enteral route, either by a fine-bore nasoduodenal tube placed endoscopically, or by feeding jejunostomy. The mortality of leaks remains high, in spite of recent improvements in perioperative care and intensive care support. Improvements in the management of leak-related sepsis would likely lead to a decrease in morbidity and mortality.

Other surgical complications such as chylothorax and herniation of bowel through the diaphragmatic hiatus are rare, but should be recognized early and both are corrected by surgical re-exploration.

Combined Multimodal Treatment Strategies

The past decade has seen a proliferation of additional treatments for esophageal cancer. The rationale is based on the suboptimal long-term results of surgery or radiotherapy. Both the spatial and synergistic actions of chemotherapeutic agents and radiotherapy are explored in multimodality treatments. How surgical resection and these new combinations should be integrated into treatment programs is an active area of research.

Neoadjuvant Radiotherapy

Trials of neoadjuvant radiotherapy have failed to show increased resection rate or improved survival compared with surgery alone. The European Organization for Research and Treatment of Cancer study suggested improved local disease control but no better long-term outcome.One study, which also involved chemotherapy, suggested a survival advantage imparted by preoperative radiotherapy, but only in the pooled groups of patients receiving radiotherapy. A Cochrane meta-analysis showed that if preoperative radiotherapy regimens do improve survival, then the effect is likely to be modest, with an absolute improvement in 5-year survival of around 3–4%.

Adjuvant Radiotherapy

Postoperative radiotherapy was studied in three randomized trials, and all three demonstrated improved local disease control. The largest study published to date randomized 495 patients with intrathoracic squamous cell cancers. Postoperative radiotherapy of 50–60 Gy was given to 220 patients to the entire mediastinum and bilateral supraclavicular fossa. Per protocol, analysis showed no overall difference in 5-year survival, with 31.7% for the surgery-alone group and 41.3% for the radiotherapy group. A benefit in the radiotherapy group was observed in stage III patients; 5-year survival rates were 13.1% and 35.1%, respectively. In patients with node-positive disease, difference in survival was of borderline significance. The chance of mediastinal, cervical lymph node, and anastomotic recurrences was also reduced.[219] Survival benefit was not demonstrated by the other trials. From these studies it seems reasonable to give postoperative radiotherapy to subgroups of patients, especially those who had palliative resections, to enhance local disease control. Suitable meta-analysis should be carried out to further test the statistical validity of the conclusions.

Neoadjuvant Chemotherapy

Eleven randomized trials studied the role of preoperative chemotherapy. The two largest trials were the Intergroup trial and the MRC trial. The first study randomized patients to undergo surgery alone, or to have three cycles of cisplatin and 5-fluorouracil before surgery, and in those who had stable or responsive disease, two additional postoperative courses. The differences in findings in these two studies are difficult to resolve. There are many differences, including the chemotherapy regimen, distribution of histologic cell types, number of patients undergoing resection, time to resection, type of surgery performed, and number of patients who also had radiotherapy.

A meta-analysis was conducted by the Cochrane group.Altogether 2051 patients were analyzed. Neoadjuvant therapy was found not to alter the rate of resection, rate of complete resection, or postoperative complications. The pooled clinical response was 36%, and pathologically complete response was only 3%. There appears to be a significant survival advantage for chemotherapy. At 3, 4, and 5 years, the increased survival was 21%, 24%, and 44%, respectively, but only reached statistical significance at 5 years. It was estimated that 11 patients needed to be treated to attain one extra survivor at 5 years. It is also worth noting that all trials evaluated patients with squamous cell cancers except the Intergroup and MRC trial. Subgroup analysis did not show any difference between the two cell types.

Adjuvant Chemotherapy

This is the area perhaps least well studied, and trials of pure postoperative chemotherapy are limited. A recent report on 242 patients compared surgical resection with the addition of postoperative cisplatin and 5-fluorouracil showed good result.

Neoadjuvant Chemoradiation

Several groups have explored chemoradiation as neoadjuvant therapy. The radiation dose ranged from 20 Gy to 45.6Gy.

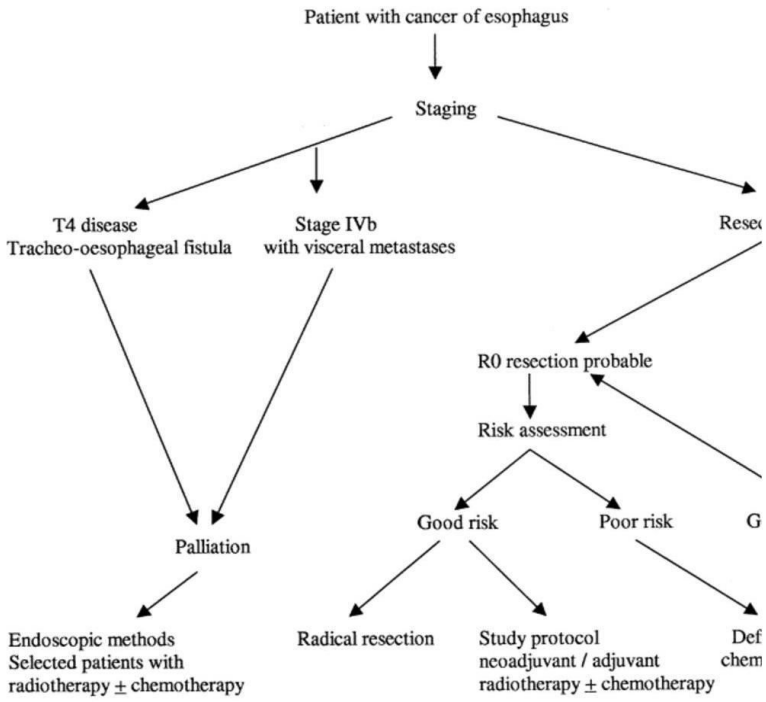
Endoscopic Palliation

Endoscopic palliative treatments for more advanced tumors include

- placement of an esophageal prosthesis,
- laser therapy,
- intralesional injection of various substances, and
- photodynamic therapy.

The two most commonly employed techniques are insertion of a prosthesis and laser therapy. Insertion of self-expanding metallic stents (SEMS) has become the preferred method in many institutions.

Perforation, pneumonia, bleeding, and migration rates were significantly less with metallic stents. Because of the lower morbidity, metallic stents were also more cost-effective despite their higher initial cost. The choice of various metallic stents depends on their individual characteristics in terms of flexibility, tensile force, and degree of shortening on deployment in relation to the site of placement.



2. Classify thyroid tumours. Discuss the management of well differentiated carcinoma of thyroid. (20)

Answer. Classification and Incidence of Thyroid Malignancies

Tumors of Follicular Cell Origin		Tumors of Parafollicular or C-cell Origin	Other
Differentiated	Undifferentiated	Medullary (5%|)	Lymphoma (<1%)
Papillary (75%) Follicular (10%) Hürthle Cell (5%)	Anaplastic (5%)		

Management of well differentiated carcinoma of thyroid:

Investigations:

- Thyroid function test
- USG guided FNAC
- CT/USG of neck.
- Indirect laryngoscopy.

Papillary Cancer:

- Papillary cancer is the most common malignant thyroid tumor, accounting for 70% to 80% of all thyroid malignancies.
- Histologically, papillary carcinoma has characteristic features including psammoma bodies, intranuclear grooves, and cytoplasmic inclusions.

- It is multicentric in 30% to 50% of tumors.
- Papillary cancer tends to spread via the lymphatics with a known propensity for involvement of the mid- and lower-anterior cervical lymph nodes.
- Invasion of adjacent structures and distant metastases at the time of presentation are uncommon.
- Certain subtypes of papillary cancer including tall-cell, columnar cell, and oxyphilic variants tend to be radioiodine resistant, more aggressive, and associated with a poorer prognosis.
- The appropriate extent of surgical therapy for papillary cancer is debated. It is accepted that a lobectomy and isthmusectomy is satisfactory for papillary cancers less than 1.0 cm in size without lymphatic or systemic metastases. These patients have a recurrence rate of less than 5% and a death rate of approximately 0.1%.
- Lobectomy with isthmusectomy is also considered acceptable for patients with papillary cancers that are greater than 1.0 cm when patients are categorized as low risk for recurrence or mortality. Total thyroidectomy is uniformly accepted for treatment of high-risk papillary carcinoma.
- In addition, patients with a history of head and neck irradiation should undergo total thyroidectomy due to the high incidence of carcinoma at sites other than the nodule and their increased lifetime risk of developing thyroid cancer in the remaining thyroid gland.

Low-Versus High-Risk Criteria in Papillary Thyroid Cancer

Low risk	High risk
1. Women < 50 years 2. Men < 40 years 3. Well-or moderately differentiated tumors 4. Tumor < 4 cm in diameter 5. Tumor confined to the thyroid gland 6. No distant metastases	1. Women ≥ 50 yr 2. Men ≥ 40 yr 3. Poorly differentiated tumors, tall-cell, columnar cell, or oxyphilic variants. 4. Tumor ≥ 4 cm in diameter 5. Local invasion 6. Distant metastases

Lobectomy and isthmusectomy	Total thyroidectomy
1. Excellent prognosis for patients with papillary cancer. 2. Lower risk of recurrent laryngeal nerve injury and permanent hypoparathyroidism. 3. > 5% of recurrences occur in the thyroid bed, and that half of local recurrences can be cured surgically.	1. Associated with the lowest incidence of local and regional recurrence and that when combined with postoperative ^{131}I ablation there is improved survival. 2. Performing a total thyroidectomy also allows for the use of serum thyroglobulin (Tg) and radioiodine for early detection and treatment of

4. Tumor multicentricity has little clinical significance.	metastatic disease. 3. It eliminates the 1% risk of anaplastic transformation of cancer that remains in the residual lobe and avoids future reoperative surgery, which is associated with an increased risk of complications.

- Furthermore, the role of lymph node dissection is also debated. Most series in the United States and Europe report a 30% to 40% incidence of cervical lymph node metastases. Prophylactic lymph node dissection is not warranted.
- Enlarged lymph nodes in the central and lateral neck should be removed and submitted for frozen section exam. If positive in the central neck, a central neck dissection should be performed.
- If positive in the lateral neck, a modified radical neck dissection should be undertaken. Modified radical neck dissection, also known as a functional neck dissection, refers to removal of lymph nodes anterior and posterolateral to the internal jugular vein from the mastoid process superiorly to the subclavian vessels inferiorly and laterally to the spinal accessory nerve, levels two through five.
- The internal jugular vein, spinal accessory nerve, and sternocleidomastoid muscle are preserved. Formal lymph node dissection is preferable to "berry-picking" because it is associated with a lower incidence of subsequent recurrent disease and it helps to minimize the need for repeat surgery for recurrent lymph node metastases, which can be difficult in a scarred operative field.

Follicular Cancer:

Follicular cancer accounts for approximately 10% of all thyroid cancer. The female/male ratio is 3:1 and the mean age at presentation is 50 years. It usually presents as a painless solitary nodule or as a rapidly growing nodule within a multinodular goiter. Follicular cancers spread hematogenously with a predilection for the lungs and bones. Multicentricity and lymph node metastases are less common in patients with follicular carcinoma compared with papillary carcinoma. Approximately 10% will have spread to cervical lymph nodes. A follicular carcinoma is distinguished from a follicular adenoma based on the presence of capsular or vascular invasion determined by microscopic examination of a tissue sample. Fine-needle aspiration biopsy, which evaluates cellular features alone, is unable to distinguish a benign from a malignant follicular neoplasm. We counsel patients that there is a 20% likelihood of malignancy when a FNAB is interpreted as consistent with a follicular neoplasm and a serum TSH level is normal or high. Patients with a FNAB that is interpreted as a follicular neoplasm should undergo a lobectomy and isthmusectomy. Frozen section is also unreliable in distinguishing an adenoma from a malignant carcinoma. We recommend a completion thyroidectomy when the permanent section shows a clinically significant follicular cancer.

A lobectomy and isthmusectomy are adequate for minimally invasive follicular cancer because it has an indolent course and patients have a near-normal life expectancy. A minimally invasive tumor is almost indistinguishable from a benign follicular adenoma except for the presence of limited capsular invasion. Invasive follicular carcinoma, defined by major capsular invasion and angioinvasion, is more aggressive, and affected patients are treated with either a near-total or total thyroidectomy. As is the case for papillary cancers, performing a total thyroidectomy facilitates radioiodine ablation, the use of radioiodine for detection and treatment of metastatic disease, and the use of serum Tg as a marker for persistent or recurrent disease. Lymph node dissection is performed only for gross metastatic disease.

HÜRTHLE CELL CANCER

Hürthle cell cancer accounts for approximately 5% of all thyroid cancers. Hürthle cell cancers have many clinicopathologic features that are similar to follicular cancers including difficulty in distinguishing a Hürthle cell carcinoma from a Hürthle cell adenoma and a propensity for vascular invasion. Hürthle cell carcinomas have many features that are distinct from follicular carcinomas including a greater tendency to spread to cervical lymph nodes, a higher incidence of distant metastases, and a failure to concentrate radioiodine. Less than 10% of Hürthle cell cancers take up radioiodine. Hürthle cell cancers have a higher tumor recurrence rate and a higher mortality rate. The mortality for patients with Hürthle cell carcinoma has been reported to be 30% at 10 years.

3. Write short notes on: 5 x 6

(a) Raynaud's phenomenon.
(b) Necrotising fascitis.
(c) Melanoma.
(d) Diagnosis and localization of insulinomas.
(e) Solitary rectal ulcer.

Answer.
(a) Raynaud's phenomenon.
See the Question 3.e of Paper –II of 2015.

(b) Necrotising fasciitis: Necrotizing fasciitis (NF), commonly known as flesh-eating disease or Flesh-eating bacteria syndrome, is a rare infection of the deeper layers of skin and subcutaneous tissues, easily spreading across the fascial plane within the subcutaneous tissue.

Type I describes a polymicrobial infection, whereas Type II describes a monomicrobial infection. Many types of bacteria can cause necrotizing fasciitis (e.g., Group A streptococcus (Streptococcus pyogenes), Staphylococcus aureus, Vibrio vulnificus, Clostridium perfringens, Bacteroides fragilis). Such infections are more likely to occur in people with compromised immune systems.

Historically, Group A streptococcus made up most cases of Type II infections. However, since as early as 2001, another serious form of monomicrobial necrotizing fasciitis has been observed with increasing frequency. In these cases, the bacterium causing it

is methicillin-resistant Staphylococcus aureus (MRSA), a strain of S. aureus that is resistant to methicillin, the antibiotic used in the laboratory that determines the bacterium's sensitivity to flucloxacillin or nafcillin that would be used for treatment clinically.

Several studies demonstrated a link between absorption of non-steroidal anti-inflammatory drugs and flesh-eating disease, without establishing whether the drugs just masked the symptoms or were a cause per se. Signs and symptoms

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Over 70% of cases are recorded in patients with one of the following clinical situations: immunosuppression, diabetes, alcoholism/drugs, malignancies, chronic systemic diseases. Occasionally occurs in people with apparently normal general condition.

The infection begins locally, at a site of trauma, which may be severe (such as the result of surgery), minor, or even non-apparent. Patients usually complain of intense pain that may seem in excess given the external appearance of the skin. With progression of the disease, tissue becomes swollen, often within hours. Diarrhea and vomiting are also common symptoms.

In the early stages, signs of inflammation may not be apparent if the bacteria are deep within the tissue. If they are not deep, signs of inflammation, such as redness and swollen or hot skin, show very quickly. Skin color may progress to violet, and blisters may form, with subsequent necrosis (death) of the subcutaneous tissues.

Patients with necrotizing fasciitis typically have a fever and appear very ill. Mortality rates have been noted as high as 73 percent if left untreated. Without surgery and medical assistance, such as antibiotics, the infection will rapidly progress and will eventually lead to death.

Pathophysiology

"Flesh-eating bacteria" is a misnomer, as the bacteria do not actually "eat" the tissue. They cause the destruction of skin and muscle by releasing toxins (virulence factors), which include streptococcal pyogenic exotoxins. S. pyogenes produces an exotoxin known as a superantigen. This toxin is capable of activating T-cells non-specifically, which causes the overproduction of cytokines and severe systemic illness (Toxic shock syndrome).

Treatment

Patients are typically taken to surgery based on a high index of suspicion, determined by the patient's signs and symptoms. In necrotizing fasciitis, aggressive surgical debridement (removal of infected tissue) is always necessary to keep it from spreading and is the only treatment available. Diagnosis is confirmed by visual examination of the tissues and by tissue samples sent for microscopic evaluation.

Early medical treatment is often presumptive; thus, antibiotics should be started as soon as this condition is suspected. Initial treatment often includes a combination of intravenous antibiotics including penicillin, vancomycin, and clindamycin. Cultures are taken to determine appropriate antibiotic coverage, and antibiotics may be changed when culture results are obtained.

As in other maladies characterized by massive wounds or tissue destruction, hyperbaric oxygen treatment can be a valuable adjunctive therapy but is not widely available. Amputation of the affected organ(s) may be necessary. Repeat explorations

usually need to be done to remove additional necrotic tissue. Typically, this leaves a large open wound, which often requires skin grafting. The associated systemic inflammatory response is usually profound, and most patients will require monitoring in an intensive care unit.

Treatment for necrotizing fasciitis may involve an interdisciplinary care team. For example, in the case of a necrotizing fasciitis involving the head and neck, the team could include otolaryngologists, intensivists, microbiologists and plastic surgeons.

(c) Melanoma.

Ans. Introduction:Melanocytes are cells of neural crest origin that migrate during fetal development to multiple sites in the body, principally the skin. Positioned along the basement membrane at the dermoepidermal junction, these cells are exposed to carcinogenic stimuli that result in malignant transformation to become melanoma.

Epidemiology and Etiology

- The incidence and outcome of melanoma are related to multiple factors. Melanoma is principally a disease of whites.
- The disease occurs much less commonly in Asian and black populations.
- The disease occurs slightly more often in men than women, and the prognosis is slightly better for women when other prognostic factors are taken into account.
- The anatomic distribution of melanoma varies between the two genders. Melanomas arise more commonly on the lower extremity in women and more often on the trunk and head and neck in men.
- Melanoma can occur at any age from birth to advanced age. The median age at diagnosis is in the range of 45 to 55 years. Tumors rarely develop before the age of puberty; however, there is a significant incidence in the third and fourth decades of life.

It is well established that exposure to sunlight, specifically, solar ultraviolet (UV) radiation, increases the risk for development of melanoma in susceptible populations. UVA and UVB cause different patterns of effect in the skin; however, both are considered to be carcinogenic.

It is reported that people incurring severe burns in childhood appear to be at higher risk for the development of melanoma years later. In contrast, those who receive exposure on a regular basis may not be at as high a risk.

The highest-risk population appears to be individuals with a fair complexion who receive intermittent doses of radiation that result in severe sunburns.

Additional factors that increase the risk for development of melanoma include :

- Dysplastic nevus (DN) syndrome,
- Xeroderma pigmentosum,
- A history of nonmelanoma skin cancer (NMSC),
- A family history of melanoma.

The risk for melanoma increases with age; however, the role of aging is not clear. With increasing age there is more opportunity for the initiation of new tumors, either through exposure to carcinogens (UV irradiation) or through the decreasing ability of individual cells to repair DNA damage.

Precursor Lesions and Risk Factors

- Congenital nevi, DNs, Spitz nevi, and familial patterns all raise the risk for development of melanoma. Individuals with congenital nevi have an increased risk that is proportional to the size and number of nevi.
- Small congenital nevi represent a low risk and are therefore observed unless local changes appear.
- Giant congenital nevi are rare (1 in 20,000 newborns) and carry an increased risk for the development of melanoma within the nevi .
- DNs may occur sporadically or in a familial pattern. Individuals with DNs and a family history of melanoma have an extremely high risk for the development of melanoma.
- Patients with DN syndrome (B-K mole syndrome, familial atypical mole–malignant melanoma syndrome) have multiple nevi (>100) that present a great challenge to the patient and physician.
- Spitz nevi (juvenile melanoma, spindle cell melanoma, epithelioid cell melanoma) are rapidly growing, pink or brown benign skin lesions arising most often in children and adolescents, although adult skin lesions may also have spitzoid features.
- In borderline cases, it may be necessary to excise the areas as if a melanoma to ensure adequate treatment. Sentinel lymph node (SLN) biopsy has been proposed as a mechanism to clarify the malignant potential in indeterminate cases.
- If the diagnosis of *melanoma arising within a Spitz nevus* is made, treatment is based on the same criteria as for other types of melanoma.

Familial Melanoma

Approximately 5% to 10% of melanoma patients have a family history of the disease. When compared with patients who have sporadic melanoma, the age of onset is earlier, the incidence of DNs is higher, and multiple primary melanomas are more common. Chromosome mapping studies have shown evidence of linkage and heterogeneity to chromosomes 1p and 9p. Chromosome 1p contributes to both sporadic and familial melanoma, whereas 9p contributes more to sporadic melanoma alone. All reported kindreds are white.

Clinical Features

Cutaneous Melanoma

- Melanoma commonly develops as a changing, pigmented skin lesion.
- Patients typically describe a flat lesion that spreads over the surface of the skin and later becomes elevated. If the lesion is allowed to progress, itching, bleeding, and ulceration will follow.

- In some instances, melanomas arise in preexisting nevi; however, the majority arise de novo. The most important aspect of the history is change.
- The common features of melanoma are summarized in the mnemonic ABCDE: *a*symmetric outline, changing irregular *b*orders, variation in *c*olor, *d*iameter greater than 6 mm, and *e*levation. In early melanoma, the changes may be limited to two or three features.

Unknown Primary Melanoma

Nodal or distant metastasis may be the first evidence of melanoma. This occurs in less than 2% of all melanoma cases and in less than 5% of all patients with metastatic melanoma.

A thorough search for the primary lesion includes a histologic review of all previously removed skin lesions; questions regarding skin lesions that resolved without treatment; and inspection of areas that may have been missed at the initial examination, including the scalp, external auditory canal, oral and nasal mucosa, nail beds, genitalia, anal canal, perianal skin, and the eye. In the case of lymph node metastasis, completion regional lymph node dissection is performed on the assumption that it is a regional node and therefore represents stage III rather than stage IV disease. Such patients may have a better prognosis than those with known primary sites.

The patient is then evaluated for adjuvant therapy, especially for participation in investigational protocols. For metastases at other sites, see Surgical Considerations for Metastases, later in this chapter.

Noncutaneous Melanoma

In embryogenesis, melanocytes arise in the neural crest area and migrate to many sites other than the skin. Less than 10% of melanomas arise in these areas, which include the eye, mucosal surfaces, and unknown primary sites.

Although melanoma has been reported to arise from many tissues and organs throughout the body, there is often the possibility that these lesions are actually metastases from an unknown primary site on the skin. One exception may be in the esophagus, where melanocytic atypia and melanoma in situ have been shown to occur.

Ocular melanoma is the most common malignancy arising in the eye. Within the eye, melanocytes are found in the retina and uveal tract (iris, ciliary body, and choroids).

The most common sites of origin for melanomas arising on the mucous membranes are the head and neck (oral cavity, oropharynx, nasopharynx, and paranasal sinuses), anal canal, rectum, and female genitalia. When compared with melanomas arising on the skin, mucosal melanomas are more advanced and have a poorer prognosis.

<u>Clinical Management</u>

Choice of Biopsy:

- The specific method of biopsy depends on the size of the lesion and its anatomic location. Regardless of the method, biopsy specimens are full thickness into subcutaneous tissue.
- For small lesions, an excisional biopsy is commonly performed that includes a narrow (1-2 mm) margin of surrounding skin.
- The biopsy area is not enlarged to permit a better cosmetic appearance because this may lead to unnecessary expansion of the final wide excision.
- Although shave biopsies are commonly performed for benign-appearing lesions, this technique must not be used when melanoma is suspected.
- In these circumstances the most important prognostic factor, tumor thickness, will not be accurate and could lead to incorrect decisions regarding wide local excision (WLE), SLN biopsy, and adjuvant therapy.
- If a skin lesion reappears at the site of a previously cauterized or frozen skin lesion, excisional biopsy and histopathologic analysis are required.

When performing diagnostic biopsy, orientation of the biopsy closure may affect options for closure of the WLE. For this reason, biopsy excisions on the extremities are closed longitudinally to maximize the possibility for primary wound closure and decrease the need for skin grafts. Larger tissue defects may be closed with local rotational/advancement skin flaps or a skin graft.

Histologic Features of Cutaneous Melanoma

Histologically, melanoma is divided into four major types based on growth pattern and location:

(a) Lentigo maligna melanoma(LMM),
(b) Superficial spreading melanoma (SSM),
(c) Acral lentiginous melanoma (ALM), and
(d) Nodular melanoma (NM).

LMM (~10%) has distinctive clinical and histologic features. It occurs most commonly in older individuals with sun-damaged skin and appears as a flat, darkly pigmented lesion with irregular borders and a history of slow development. Overall, the prognosis of LMM is better than that for other histopathologic types; however, this better prognosis is primarily related to the superficial nature of these lesions.

The most common histologic type is SSM (~70%). It is not necessarily associated with sun-exposed skin. As the name SSM suggests, these lesions initially appear as a flat, pigmented lesion growing in a radial pattern

If left in place, the lA vertical growth pattern develops early in the history of NMs (~15%), and they may be devoid of junctional changes . Melanomas in this group have the worst prognosis because of a higher average tumor thickness lesion begins to thicken as the vertical growth phase develops.

Based on the findings just discussed, a complete pathologic report of cutaneous melanoma includes the following: Breslow thickness, presence or absence of ulceration, Clark level, status of the surgical margins, histologic type, presence or absence of

satellitosis, and presence or absence of regression. The report may also describe tumor-infiltrating lymphocytes, lymphovascular invasion, vertical growth phase, neurotropism, and mitotic rate.

Staging

American Joint Committee on Cancer TNM Melanoma Classification—2002

Primary Tumor (T)	
TX	Primary tumor cannot be assessed (e.g., shave biopsy or regressed melanoma)
T0	No evidence of primary tumor
Tis	Melanoma in situ
T1	Melanoma ≤1.0 mm in thickness, with or without ulceration
T1a	Melanoma ≤1.0 mm in thickness and level II or III, no ulceration
T1b	Melanoma ≤1.0 mm in thickness and level IV or V or with ulceration
T2	Melanoma 1.01-2.0 mm in thickness, with or without ulceration
T2a	Melanoma 1.01-2.0 mm in thickness, no ulceration
T2b	Melanoma 1.01-2.0 mm in thickness, with ulceration
T3	Melanoma 2.01-4.0 mm in thickness, with or without ulceration
T3a	Melanoma 2.01-4.0 mm in thickness, no ulceration
T3b	Melanoma 2.01-4.0 mm in thickness, with ulceration
T4	Melanoma >4.0 mm in thickness, with or without ulceration
T4a	Melanoma >4.0 mm in thickness, no ulceration
T4b	Melanoma >4.0 mm in thickness, with ulceration
Regional Lymph Nodes (N)	
NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in one lymph node
N1a	Clinically occult (microscopic) metastasis
N1b	Clinically apparent (macroscopic) metastasis
N2	Metastasis in two or three regional nodes or intralymphatic regional metastasis without nodal metastases
N2a	Clinically occult (microscopic) metastasis
N2b	Clinically apparent (macroscopic) metastasis
N2c	Satellite or in-transit metastasis without nodal metastasis
N3	Metastasis in four or more regional nodes, matted metastatic nodes, in-transit metastasis, or satellites with metastasis in regional node(s)

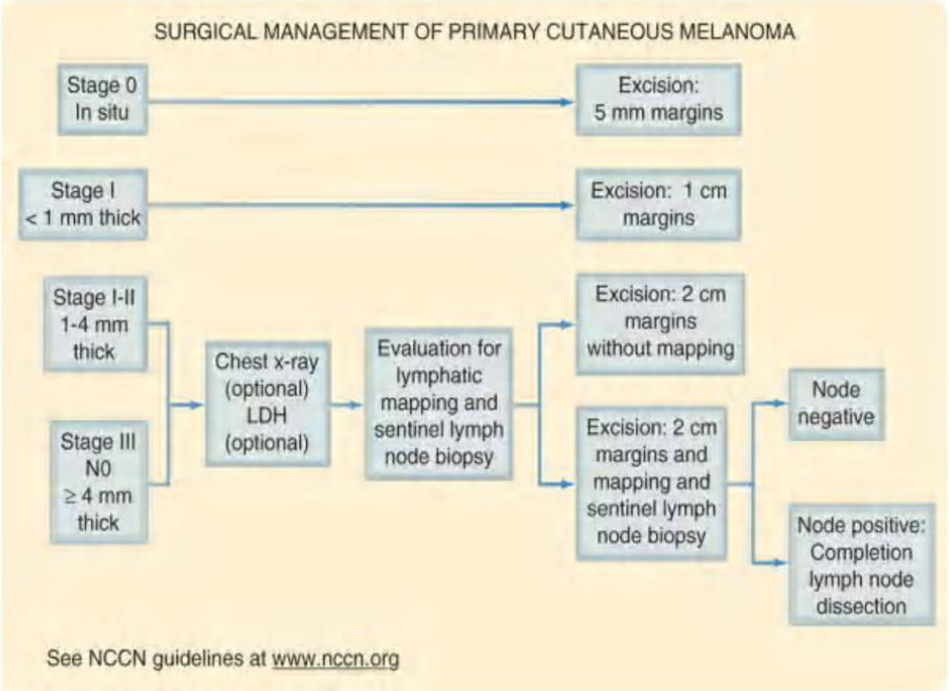
Distant Metastasis (M)	
MX	Distant metastasis cannot be assessed
M0	No distant metastasis
M1	Distant metastasis
M1a	Metastasis to skin, subcutaneous tissue, or distant lymph nodes
M1b	Metastasis to lung
M1c	Metastasis to all other visceral sites or distant metastasis at any site associated with elevated serum lactate dehydrogenase

Surgical Management of the Primary Lesion

The fundamental principle in the management of primary melanoma is to resect the tumor and minimize the risk for local recurrence. Melanomas measuring 1 mm or less in thickness can be resected with a 1-cm margin with a low subsequent risk for local recurrence. Melanomas between 1 and 2 mm in thickness have an equally low risk for local recurrence when a 2-cm margin is used. These margins may be lowered to 1 cm when primary closure of the wound would be facilitated. A narrower margin may result in a small increase in the number of patients in whom local recurrence develops; however, no statistically significant difference in survival has been observed. The factor that most closely correlated with local recurrence was primary tumor ulceration. When melanomas are greater than 4 mm in thickness, recommendations for management are based on retrospective analyses in which there does not appear to be any advantage to extending the resection beyond 2 cm.

Recommended Margins for Surgical Resection of Primary Melanoma:

TUMOR THICKNESS (mm)	MARGIN RADIUS (cm)[*]
In situ	0.5
<1.0	1.0
1-2	1.0-2.0
>2.0	≥2.0



Management of Regional Lymph Nodes

After WLE of the primary tumor, the most common sites of first recurrence are regional (lymph nodes, in-transit metastases, and local recurrences). Nodal metastases generally appear in the basin or basins draining from the primary site. This is a predictable pattern for extremity melanomas; however, truncal and head and neck melanomas may drain to more than one site. This sequence of recurrences led surgeons to conclude that resection of nodal basins containing occult metastases could provide an increase in survival. This procedure, termed *elective lymph node dissection* (ELND), was commonly practiced but was often accompanied by significant morbidity, including lymphedema, muscle weakness, and restricted range of motion.

As prognostic factors became better understood, it was postulated that patients with thin tumors (<1 mm in thickness) would have a low risk of metastases at any site and patients with thick tumors (>4 mm in thickness) would have a high risk of distant as well as regional metastases. In contrast, patients with intermediate-thickness melanoma (1-4 mm) would have an elevated risk for nodal metastases without a high risk for distant disease. Development of the SLN concept ended one debate over ELND, changed clinical management, and opened a new series of questions about the tumor biology of melanoma.

1. By using a combination of isotope lymphatic mapping, an intraoperative hand-held gamma probe, and intraoperative injection of blue dye, the SLN could be identified in more than 95% of cases in the groin and axilla, with identification in the head/neck region being slightly lower (85%).
2. There was great anatomic variation resulting in drainage to multiple or

uncommon sites.

3. Detailed pathologic analysis of the sentinel nodes via step sections enabled detection of micrometastases that could be missed by standard techniques.
4. The probability of finding a positive sentinel node can be predicted by using a nomogram derived from multifactorial analysis.
5. In most cases a positive sentinel node was the only positive node.
6. No prognostic factors were found that accurately identified a subpopulation of SLN-positive patients at zero risk of harboring other positive nodes.

Monitoring of Patients After Surgical Therapy

After primary treatment of melanoma, prediction of the pattern of recurrence can be based on the same factors used to estimate survival (tumor thickness, ulceration, and lymph node status). The risk of the first metastasis being at a distant site increases with thick primary tumors and resected regional positive nodes. Follow-up examinations focus on detection of treatable metastases. The most common sites of initial recurrence are local and regional. Patients are informed about the common symptoms and signs of recurrence so that they can report important changes arising between scheduled examinations. Such changes include local swelling, itching, new lesions in and beneath the skin, enlargement of lymph nodes, central nervous system changes, and pulmonary and gastrointestinal symptoms.

The physical examination is the most important aspect of the return visit. A complete skin examination is performed with inspection and palpation of the primary site and skin surfaces leading to regional nodal basins. In-transit metastases may be palpable but not visible.

The follow-up examination schedule reflects the risk for recurrence. Initially, patients are seen at 3- to 6-month intervals until they have reached the 3-year anniversary. By this time, 75% of patients in whom a metastasis would ever develop would have had that event occur. Annual examinations are scheduled thereafter. Patients with early melanoma, stage IA, are monitored without radiologic or laboratory studies. For asymptomatic patients, a chest radiograph and serum lactate dehydrogenase assay may be performed at 6- to 12-month intervals, although there is no evidence that the routine use of these follow-up tests results in a survival benefit. The routine use of screening computed tomography (CT), magnetic resonance imaging, or positron emission tomography (PET) has not been shown to be cost-effective and remains a subject of investigation. In stage III patients, PET scan results change treatment decisions in up to 20% of cases; however, the rate is very low in patients with a single microscopic nodal metastasis. Scans and other tests may be required for patients participating in clinical protocols.

Surgical Considerations for Metastases

Approximately 80% of patients who are treated for melanoma are cured of their disease. Recurrent disease appears locally, regionally, systemically, or in a combination of these sites.

Regional Nodal Recurrence

Regional nodal metastases are the most common site of first recurrence in patients who undergo WLE alone. When palpable lymph nodes develop, the diagnosis is most rapidly made by fine-needle aspiration (FNA) performed during the office visit. If positive, complete resection of the nodal basin will control regional disease in a large proportion of patients. If FNA is negative or insufficient, excisional biopsy is performed to verify the diagnosis. If nodes are positive, long-term survival is unfortunately low. Even with a single palpable nodal metastasis, the 5-year survival rate is 40% to 50% .

Before complete regional lymphadenectomy, a full metastatic workup is performed, including CT scans of the head, chest, abdomen, and pelvis, although these scans are normal in the majority of patients who are otherwise asymptomatic. The risk for further locoregional recurrence after complete lymph node basin dissection is increased in the presence of multiple positive nodes, especially those containing extracapsular extension. Postoperative irradiation of the involved areas has been advocated in some centers as a way to further reduce recurrences; however, such radiotherapy has not been tested in a prospective, randomized trial.

Local and Regional Recurrences:

- True local recurrence (N2c, stage III) is defined as tumor appearing in skin or subcutaneous tissue within a 5-cm radius of the primary wide excision site. Factors that predict local recurrence are the same as those predicting overall survival. Local recurrence is a poor prognostic sign: less than 20% of patients survive long-term after local recurrence.
- Local recurrence is treated by surgical resection to attain histologically clear margins. WLE guidelines for primary tumors do not apply to local recurrences.
- Amputation for extensive local-regional recurrence is seldom indicated. These patients have a high risk of having other distant metastases, and therefore long-term disease-free survival is not achieved by resection. Occasionally, patients have indolent locoregional disease for which amputation is indicated only after other attempts at locoregional tumor control have been unsuccessful.
- Patients with multiple recurrences on the limbs may be candidates for isolated hyperthermic limb perfusion (IHLP). Interleukin-2, tumor necrosis factor, and multiple other chemotherapeutic agents have also been used. Response rates exceed 80%, and complete responses are seen in 10% to 15% of patients.
- Reperfusion of extremities can be performed in patients who have an excellent initial response. Based on encouraging results with therapeutic IHLP, a randomized trial was designed to test the value of prophylactic perfusion in patients with high-risk melanoma (>1.5 mm in thickness). After more than 6 years' median follow-up, there was no improvement in overall survival.Therefore, IHLP is recommended only for patients with established multiple in-transit metastases.

Distant Metastases

The most common sites of initial distant metastases are the brain, lung, and liver and less commonly the skin, bone, and other gastrointestinal tract sites. The prognosis varies significantly with the site of first metastases . In the majority of cases, metastases

appear at multiple sites simultaneously. In such circumstances, systemic therapy is indicated for palliation. Occasionally, metastases develop that are apparently isolated to a single site. These patients are evaluated for surgical resection because the long-term disease-free survival rate after metastasectomy is reported to range from 10% to 20%.Patients being considered for resection of visceral metastases undergo complete staging, including CT and PET scans. In general, the prognosis for metastases to distant sites is related to the number of metastases and the disease-free interval between primary therapy and recurrent disease. Highly selected patients may undergo excision of multiple intra-abdominal metastases with a favorable outcome.

One-Year Survival Rates for Patients With Distant Metastases

STAGE	METASTATIC SITE(S)	APPROXIMATE 1-YEAR SURVIVAL (%)
M1a	Skin, subcutaneous tissues, lymph nodes	60
M1b	Lung	55
M1c	Other visceral sites	40

Systemic Treatment of Melanoma

- Most of the increase in the incidence of melanoma is attributable to thin melanomas with an excellent prognosis. Unfortunately, the number of deaths from melanoma is also rising. Although melanoma has been reported to metastasize to almost any tissue site, the most common areas are the lung, liver, bone, and brain. The most frequently used drug for systemic therapy is dacarbazine, which has a response rate of 15% to 30%; however, complete responses are rare.
- A doubling of the response rate has been observed with CVD (cisplatin, vinblastine, dacarbazine) combined with interferon alfa, interleukin-2, or a combination of these two biological
- The combination of CVD, interferon, and interleukin-2 (frequently called *biochemotherapy*) has a response rate of 50% and a complete response rate of 15%; however, several trials of this combination have not shown significant prolongation of survival.
- Temozolomide is also being used in combination with other drugs. Current recommendations for chemotherapy are summarized in the National Comprehensive Cancer Network guidelines.Stage IV patients are also candidates for investigational protocols involving the use of immunotherapy. Despite an extensive history of immunotherapy for melanoma, this modality remains investigational. It is postulated that stage IV patients who can undergo resection of all detectable disease will be a group of patients who will benefit from systemic therapies such as immunotherapy.

Adjuvant Systemic Therapy

- Adjuvant systemic therapy has proved to be a distinct advance in the treatment of common cancers such as those arising in the breast and colon. Clinical investigators

have been attempting to identify an effective adjuvant therapy for melanoma for more than 40 years, but no treatment regimen has shown a conclusive benefit. At the present time, stage IIc and III patients are evaluated for and invited to participate in randomized clinical trials of adjuvant therapy, when available.

(d) Diagnosis and localization of insulinomas.

Diagnosis: The diagnostic hallmark of insulinoma is the so-called Whipple triad.

- Symptoms of hypoglycemia (catecholamine release) and
- Low blood glucose (40-50 mg/dl) and
- Relief of symptoms after the iv administration of glucose.

The triad is not entirely diagnostic.

The clinical syndrome of hyperinsulinism may follow one of two patterns or sometimes a combination of both.

The symptom complex may be due to autonomic nervous overactivity, as expressed by fatigue, weakness, fearfulness, hunger, tremor, sweating, and tachycardia, or alternatively, a central nervous system disturbance with apathy (or irritability or anxiety), confusion, excitement, loss of orientation, blurring of vision, delirium, stupor, coma, or convulsions.

The pathognomonic finding is an inappropriately high (>5 mU/mL) level of serum insulin during symptomatic hypoglycemia.

A diagnostic ratio of blood insulin (in microunits per milliliter) to glucose (in milligrams per deciliter) of greater than 0.4 or C peptide levels higher than 2 nmol/L have proved valuable in diagnosis.

As soon as a patient with insulinoma is identified,

- care must be taken to prevent severe hypoglycemia with possible loss of cerebrocytes.
- The diet is modified to include frequent meals, even awakening at night to eat.
- The standard drug is diazoxide, which is helpful in about two thirds of patients but discontinued at least a week before surgery because it may cause intraoperative hypotension.
- The long-acting somatostatin analogue octreotide, though helpful in children with nesidioblastosis, has been effective only rarely in adults.

Localization

- Insulinomas are small (usually <1.5 cm), usually single (only 10% are multiple and those are usually associated with MEN 1 syndrome), usually benign (only 5%-10% are malignant), and generally hard to find.
- **Plain abdominal radiographic** and **ultrasound** studies are rarely helpful, but **contrast-augmented computed tomography (CT)** and **magnetic resonance imaging (MRI)** locate 50% to 60% of tumors.
- Because few insulinomas have many somatostatin receptors, **somatostatin receptor scintigraphy (SRS)** is not highly successful.

- Success in localization by **selective arteriography** varies with the size of the tumor.
- Demonstration of islet tumors by enhanced CT, enhanced MRI, or arteriography depends, of course, on the relatively rich blood supply to islet tumors as compared with the rest of the pancreatic parenchyma. **Selective portovenous sampling** is also helpful for measurement of insulin levels in pancreatic venous tributaries , a method that does not absolutely localize the site of the tumor but, in about 75% of cases, does provide accurate information on the region of the pancreas from which high levels of insulin are released. The method has a relatively high incidence of problems with bleeding into the peritoneal cavity or biliary tree. It is expensive and requires skill.

Calcium is known to release insulin. **Selective intra-arterial injection of calcium** (into the gastroduodenal, superior mesentery, right hepatic, or splenic arteries) and obtaining samples for radioimmunoassay of insulin from the right hepatic vein is useful to localize insulinomas.

(e) Solitary rectal ulcer.

Answer. See the Question 4.c of Paper – II of 2015.

4. Answer in brief: 4 x 7.5
(a) Gastrointestinal stromal tumours.
(b) Management of recurrent varicose vein.
(c) Intraoperative radiotherapy for breast cancer.
(d) Approach to obstructive jaundice.

Answer.
(a) Gastrointestinal stromal tumours.
Introduction: Gastrointestinal stromal tumors (GIST) are rare malignancies. Although they are the most common sarcoma of the gastrointestinal (GI) tract, they represent only 0.2% of all GI tumors.
GIST exhibit heterogeneous histologic features, and are most often composed of long fascicles of bland spindle cells with pale to eosinophilic cytoplasm and rare nuclear pleomorphism. GIST occasionally exhibit epithelioid characteristics, including sheets of round- to oval-shaped cells with abundant eosinophilic cytoplasm and nuclear atypia. Based upon their histologic and immunohistochemical features, GIST are thought to arise from the interstitial cells of Cajal (ICC), which are components of the intestinal autonomic nervous system that serve as pacemakers regulating intestinal peristalsis. Until recently, similarities in histology led to misclassification of many GIST as leiomyomas, leiomyosarcomas, or GI tumors of nerve cell origin.

Signs and symptoms

Patients present with

- trouble swallowing,
- gastrointestinal hemorrhage or metastases (mainly in the liver).
- Intestinal obstruction is rare, due to the tumor's outward pattern of growth.

- Often, there is a history of vague abdominal pain or discomfort, and the tumor has become rather large by time the diagnosis is made.

Generally, the definitive diagnosis is made with a biopsy, which can be obtained endoscopically, percutaneously with CT or ultrasound guidance or at the time of surgery.

Diagnosis

As part of the analysis, blood tests and CT scanning are often undertaken (see the radiology section).

A biopsy sample will be investigated under the microscope.
The histopathologist identifies the characteristics of GISTs (spindle cells in 70-80%, epitheloid aspect in 20-30%). Smaller tumors can usually be found to the muscularis propria layer of the intestinal wall. Large ones grow, mainly outward, from the bowel wall until the point where they outstrip their blood supply and necrose (die) on the inside, forming a cavity that may eventually come to communicate with the bowel lumen.

When GIST is suspected—as opposed to other causes for similar tumors—the pathologist can use immunohistochemistry (specific antibodiesthat stain the molecule CD117 (also known as c-kit) —see below). 95% of all GISTs are CD117-positive (other possible markers include CD34, DOG-1, desmin, vimentin and others). Other cells that show CD117 positivity are mast cells.

If the CD117 stain is negative and suspicion remains that the tumor is a GIST, the newer antibody DOG-1 (Discovered On GIST-1) can be used. Also sequencing of Kit and PDGFRA can be used to prove the diagnosis.

Radiology

Barium fluoroscopic examinations (upper GI series and small bowel series) and CT are commonly used to evaluate the patient with upper abdominal pain. Both are adequate to make the diagnosis of GIST, although small tumors may be missed, especially in cases of a suboptimal examination.

Small GISTs appear as intramural masses. When large (> 5 cm), they most commonly grow outward from the bowel. Internal calcifications may be present. As the tumor outstrips its blood supply, it can necrose internally, creating a central fluid-filled cavity that can eventually ulcerate into the lumen of the bowel or stomach.

The tumor can directly invade adjacent structures in the abdomen. The most common site of spread is to the liver. Spread to the peritoneum may be seen. In distinction to gastric adenocarcinoma or gastric/small bowel lymphoma, malignant adenopathy (swollen lymph nodes) is uncommon (<10%).

Pathophysiology

GISTs are tumors of connective tissue, i.e. sarcomas; unlike most gastrointestinal tumors, they are nonepithelial. About 70% occur in the stomach, 20% in the small intestine and less than 10% in the esophagus. Small tumors are generally benign, especially when cell division rate is slow, but large tumors disseminate to the liver, omentum and peritoneal cavity. They rarely occur in other abdominal organs.

Some tumors of the stomach and small bowel, referred to as leiomyosarcomas (malignant tumor of smooth muscle), would most likely be reclassified as GISTs today on the basis of immunohistochemical staining.

GISTs are thought to arise from interstitial cells of Cajal (ICC), that are normally part of the autonomic nervous system of the intestine. They serve a pacemaker function in controllingmotility.

Most (50-80%) GISTs arise because of a mutation in a gene called c-kit. This gene encodes a transmembrane receptor for a growth factor termed stem cell factor (scf). The c-kitproduct/CD117 is expressed on ICCs and a large number of other cells, mainly bone marrow cells, mast cells, melanocytes and several others. In the gut, however, a mass staining positive for CD117 is likely to be a GIST, arising from ICC cells.

The c-kit molecule comprises a long extracellular domain, a transmembrane segment, and an intracellular part. Mutations generally occur in the DNA encoding the intracellular part (exon 11), which acts as a tyrosine kinase to activate other enzymes. Mutations make c-kit function independent of activation by scf, leading to a high cell division rate and possibly genomic instability. Additional mutations are likely "required" for a cell with a c-kit mutation to develop into a GIST, but the c-kit mutation is probably the first step of this process.

The tyrosine kinase function of c-kit is vital in the therapy for GISTs, as described below.

Genetics

Although some families with hereditary GISTs have been described, most cases are sporadic.

In GIST cells, the c-kit gene is mutated approximately 85% to 90% of the time. 35% of the GIST cells with wildtype (i.e. not mutated) c-kit instead have a mutation in another gene, PDGFR-α (platelet derived growth factor receptor alpha), which is a related tyrosine kinase.

Mutations in the exons 11, 9 and rarely 13 and 17 of the c-kit gene are known to occur in GIST. D816V point mutations in c-kit exon 17 are responsible for resistance to targeted therapy drugs like imatinib mesylate, a tyrosine kinase inhibitor. Mutations in c-kit and PDGFrA are mutually exclusive.

Classification of Primary Gastrointestinal Stromal Tumors by Risk of Metastasis

Risk Category	**Size**	**Mitotic Count**
Very Low	<2 cm	<5 per 50 HPFs*
Low	2–5 cm	<5 per 50 HPFs
Intermediate	<5 cm	6–10 per 50 HPFs
	5–10 cm	<5 per 50 HPFs
High	>5 cm	>5 per 50 HPFs
	>10 cm	Any mitotic rate
	Any size	>10 per 50 HPFs

Therapy

Tumor size, mitotic rate, and location can be used to predict the risk of recurrence in GIST patients. Tumors <2 cm with a mitotic rate of <5/50 HPF have been shown to have lower risk of recurrence than larger or more aggressive tumors. Nevertheless, all GIST tumors should be considered to have malignant potential and no GIST tumor can be correctly classified as "benign."

Surgery is the mainstay of therapy for nonmetastatic GISTs. Lymph node metastases are rare, and routine removal of lymph nodes is typically not necessary. Laparoscopic surgery, a minimally invasive abdominal surgery using telescopes and specialized instruments, has been shown to be effective for removal of these tumors without needing large incisions.

Until recently, GISTs were notorious for being resistant to chemotherapy, with a success rate of <5%. Recently, the c-kit tyrosine kinase inhibitor imatinib (Glivec/Gleevec), a drug initially marketed for chronic myelogenous leukemia, was found to be useful in treating GISTs, leading to a 40-70% response rate in metastatic or inoperable cases.

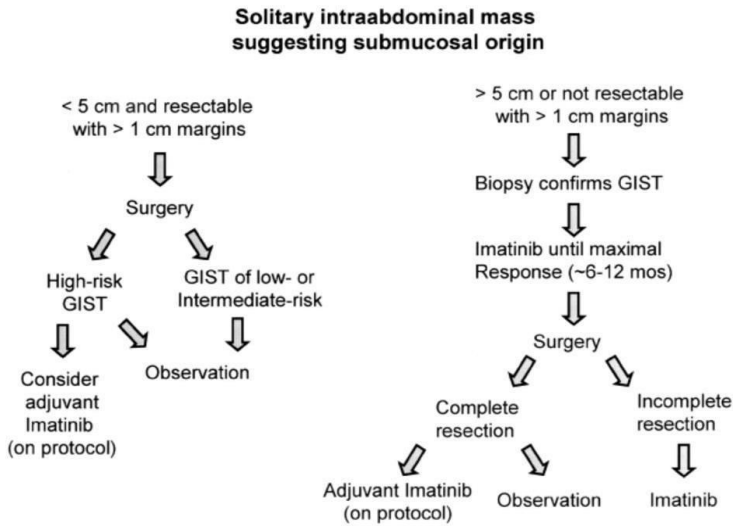
The two year survival of patients with advanced disease has risen to 75–80% following imatinib treatment.

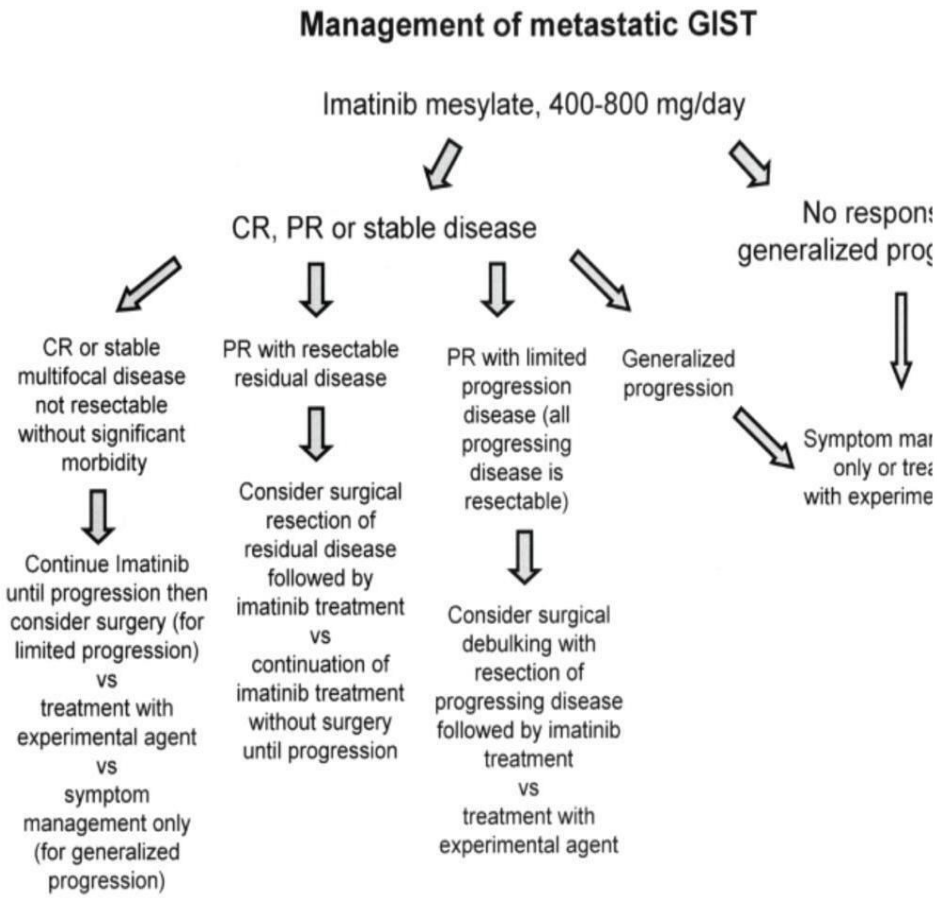
Data presented at the 2007 ASCO meeting showed adjuvant treatment with imatinib following surgical resection of GIST tumors can significantly reduce the risk of disease recurrence (6% recurrence on imatinib vs. 17% without therapy at 12 months). The optimal duration of adjuvant therapy is currently unknown; trials are ongoing evaluating treatment durations of 1, 2, and 3 years.

Patients who develop resistance to imatinib may respond to the multiple tyrosine kinase inhibitor sunitinib.

The effectiveness of imatinib and sunitinib depend on the genotype.

Management of primary localized GIST





(b) Management of recurrent varicose vein.
Recurrence of varicose veins after conventional surgery is common. Recurrence rates of 25–50% are found following SSV surgery after a follow-up period of three to five years. Incomplete dissection, filling by tributaries or residual veins, neovascularization to a network of superficial veins and reflux from another origin may all contribute to varicose vein recurrence.

Recurrent varicose veins occur when you have previously had treatment for varicose veins. Treatment can include injections like sclerotherapy, avulsions, cuts in the groin or cuts behind the knee.

Causes of recurrent varicose veins:

There are several reasons for veins coming back after treatment:

1. Normal veins become varicose.

If you have had varicose veins in the past then you are more likely to develop more varicose veins. For example, you may have had an operation for varicose veins when there has been a cut in the groin. This may have been done if you had varicose veins on the inside of your calf or thigh (long saphenous varicose veins). If your recurrent varicose veins are behind your calf on the same leg then you may have developed new

varicose veins in the area of the short saphenous vein. These short saphenous varicosities were probably normal veins when you had your first operation. Over time and because you have a tendency to have varicose veins, the normal veins have become varicose.

2. Neovascularisation

When a cut has been made in the skin, the wound heals because living tissue has the ability to regenerate. When this happens, new blood vessels grow to provide a means for blood to get to the new skin. The same process is thought to happen in the area where varicose veins have been treated by surgery. The new veins (neo = new, vascularisation = vessel formation), appear to grow from the site of the vein ligation, most commonly the sapheno-femoral junction. The new veins are like weeds growing in the garden. If they connect up to an original main vein, then blood flows through them, runs into the area of varicose veins in the leg. This will cause recurrence of your varicose veins. If the long saphenous vein in your leg was not removed at the original operation then the new veins can connect with it and cause recurrence. People who have this type of recurrence may be suitable for VNUS treatment to remove their veins.

3. Inadequate initial treatment

In the past and rarely now, the surgeon who performed your original varicose vein surgery may not have treated all of the areas of varicose veins. There can be several reasons for this.

4. Pudendal and pelvic varicose veins

Some recurrent varicose veins are caused by connections to veins inside the pelvis. In some people the problem can arise in the veins that run from the testicles or ovaries (gonadal veins). If the blood runs in the wrong direction in these veins (gonadal reflux) then there can be a long column of blood pushing blood out of the pelvis and down the leg.

How are recurrent varicose veins treated?

In much the same way as primary varicose veins (those that have not been operated upon). It is important to know exactly where and how the recurrent veins are formed. To do this, Mr Braithwaite together with most vascular surgeons, arranges an ultrasound scan called a Duplex. This scan, performed by a trained technician, can give Mr Braithwaite an accurate 'map' of the veins in the leg. With this information, Mr Braithwaite can 'tailor' the operation to your requirements.

What does recurrent varicose vein surgery involve?

This depends on the type of recurrence you have.

If you need another cut in the groin, you may be suitable for VNUS.

If you need another cut in the groin, the scarring from the previous can make the surgery more difficult. This means the operation can take longer and there are increased risks. You may be more likely to develop a wound infection, swelling in the groin and rarely permanent swelling of the leg. This can happen if the lymphatics have been damaged by the first operation and the redo or recurrent surgery causes further damage. At worst you could develop lymphoedema.

New techniques like VNUS, EVLT and Varicofoam can avoid these complication.

If you need another cut behind the knee, then you have recurrent short saphenous disease. You may need a much larger cut than before so that the surgeon can carefully identify the blood vessels and nerves behind you knee. With this operation, you may be at increased risks of wound infection and damage to nerves.

It might be worth considering varicofoam therapy if you have recurrent short saphenous vein disease. Mr Braithwaite is performing a study on the techniques.

In20% of the cases, inadequate surgery was considered to be the cause of recurrence and as soon as two weeks after surgery up to 23% of patients showed reflux on duplex scanning.

In the last decade, endovenous techniques, including endovenous laser ablation (EVLA) and radiofrequency ablation (RFA), have been successfully introduced. Various randomized trials have shown that EVLA of the GSV is at least comparable with conventional surgery of primary varicose veins.

(c) Intraoperative radiotherapy for breast cancer.

Intraoperative Radiotherapy is an alternative to conventional post-operative radiotherapy for treatment of early stage invasive breast cancer. With Intraoperative Radiotherapy, women undergoing breast conserving surgery (lumpectomy) may receive their radiotherapy at the time of their breast cancer operation while still under anesthesia, completely replacing the need for a 6-7 week course of radiotherapy normally given after surgery.
Good Candidate for Breast IORT If –
Patients who participated in trials of breast IORT had low-grade, early-stage breast cancer, with no lymph node involvement and clear surgical margins from a lumptectomy. Most participants were women 40 years and older.
Advantages of Intraoperative Radiation Therapy

- Only one radiation treatment needed
- Less patient travel, fewer appointments
- Fewer side effects
- No skin affected by treatment
- Better cosmetic results
- Immediate reconstruction can be done right away
- Adjuvant chemotherapy can be scheduled without conflict

- Faster recovery
- May save cost of treatment

Disadvantages of Intraoperative Radiation Therapy

- Workflow during surgery must be carefully coordinated between surgeons and radiation oncologists
- Small risk of infection
- Difficult to monitor dosage to the radiation site
- Few patients may have access to this technology
- Not yet enough evidence of long-term results

Possible Complications of Breast IORT

- Fat necrosis
- Hematoma
- Mild fibrosis

The local cancer recurrence is1.6%.

(d) Approach to obstructive jaundice.

Common causes of obstructive jaundice are as follows :

Intraluminal abnormalities of bile ducts:	Mural abnormalities of bile ducts:	Extrinsic compression of bile ducts:
○ Choledocholithiasis , gallstones; ○ Blood clot; ○ Parasites (e.g. flukes).	○ Cholangiocarcinoma; ○ Congenital atresia; ○ Sclerosing cholangitis. ○ Biliary cirrhosis (primary (autoimmune) or secondary to sepsis) ○ Traumatic/post surgical stricture	○ Pancreatitis; ○ Tumours, e.g. head of pancreas, ampulla of Vater;lymphadenopathy of porta hepatis nodes.

<u>Diagnosis and investigations</u>:

History -

Common aspects overlooked in the clinical history of jaundiced patients:

- family history of blood disorders;
- recent travel to foreign parts and work (exposure to infective agents);
- recent drugs or changes in medications;
- recent surgery or anaesthesia;
- history of gallstones;
- alcohol intake, cholangitis (pain, fever, rigors), and carcinoma, especially the head of the pancreas.

History and clinical features suggestive of obstructive jaundice:

• Dark coloured urine • Pale stools • Yellow	• Fever, Chills,Rigors • Episodic right upper	• Steatorrhoea • Smelly stools - difficult to flush away due to fat	• Enlarged liver (tender mass under right rib cage)

colouration of skin and eyes • Itchy skin	abdominal pain • Weight loss • Diarrhoea	malabsorption. • Excessive bleeding • Bruising	• Enlarged spleen (tender mass under left side of rib cage) • Night sweats • Anorexia, Malaise

Basic tests:

- Reticulocytosis, abnormal blood film (haemolysis) and Prothrombin time.
- Hepatitis screen (viral titres for hepatitis A, B, C, CMV, EBV).
- Immunology (anti-smooth muscle antibodies (chronic active hepatitis) and anti-mitochondrial antibodies (primary biliary cirrhosis)).
- LFT.

Liver function tests in jaundice

	Haemolytic	Hepatocellular	Obstructive
Unconjugated bilirubin	Increased	Increased	Normal
Alkaline phosphatase	Normal	Normal	Much increased
Î³ glutamyl transferase	Normal	Increased	Much increased
Transanimases	Normal	Increased	Normal
Lactate dehydrogenase	Normal	Increased	Normal

Imaging studies :

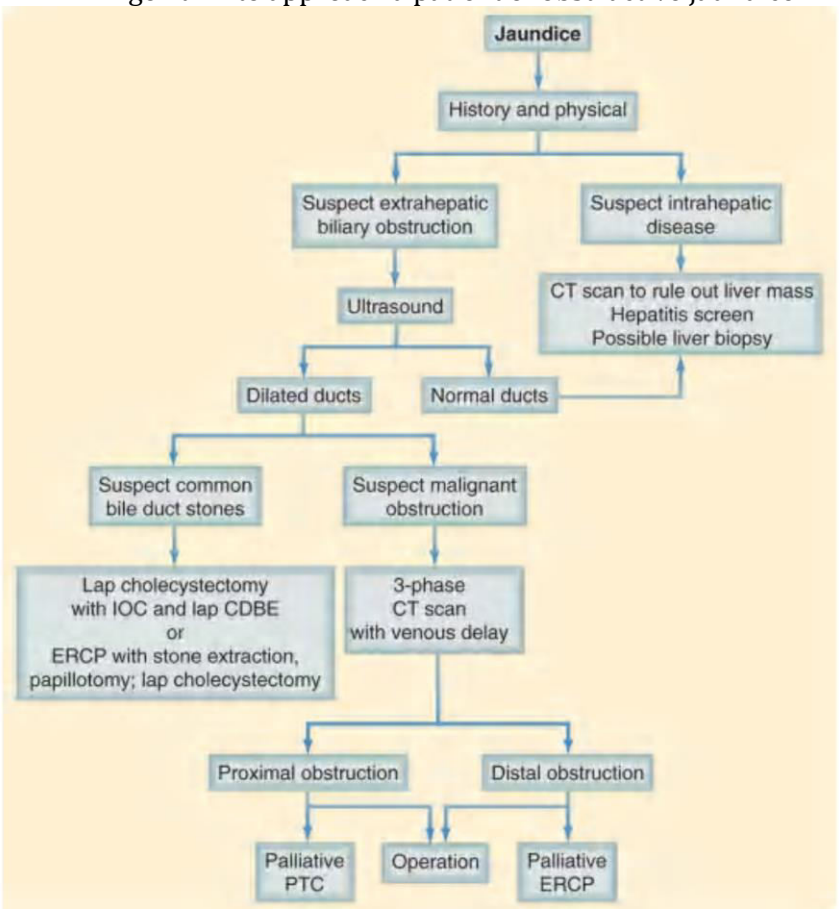
- Ultrasound scan (liver, gallbladder, bile ducts, and pancreas).
  - Excludes the presence of extra-hepatic obstruction (dilated common bile duct).
  - May locate cause of obstruction.
  - Examines hepatic parenchyma in possible hepatitis.
- Magnetic resonance cholangiopancreatography (MRCP) for suspected extrahepatic obstruction with no cause seen on ultrasound.

Complications of obstructive jaundice :

- Renal failure (hepato-renal syndrome). Caused by a combination of infection, dehydration, and a direct effect of high levels of bilirubin and other toxic products of metabolism on the kidney. Mortality is highest in patient of age 65 with an elevated blood urea – prevented by adequate fluid supplementation .
- Biliary infection (cholangitis). Commonest in obstructive jaundice or with previously damaged biliary tree. Commonly due to Gram-negative bacteria (e.g. Escherichia coli, Pseudomonas) – broad spectrum antibiotics to be instituted .
- Disordered coagulation. Due to decreased synthesis of vitamin K dependent clotting factors (III, VII, IX, X) and impaired platelet function – corrected by vit K inj &/ or FFP.

- Relative immunosuppression. Predisposes to systemic infections (e.g. chest infection) and reduces wound healing due to combinations of jaundice, infection, and reduced proteosynthesis – adequate nutrition should be prevented .

Algorithm to approach a patient of obstructive jaundice



THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES

MS (General Surgery) Examination, 2006

PAPER II

Time Allowed: 3 Hours **Full Marks: 100**

1. **Describe the aetiopathology , types , clinical features and management of choledochal cyst . (25)**
2. **Discuss the causes of gastric outlet obstruction in adults. Discuss the aetiopathology, types,clinical features and management of carcinoma stomach . (25)**
3. **Write short notes on: 5 x 10**
   - **(a) Newer classification of inguinal hernia.**
   - **(b) Investigations of Peptic Ulcer Syndrome.**
   - **(c) Lymphatic manifestations of Breast Carcinoma.**
   - **(d) Staging of squamous cell carcinoma**
   - **(e) Complications and Medical treatment of Hydatid Cyst.**
   - **(f) CT Scan.**
   - **(g) ANDI.**
   - **(h) FNAC Scoring of Breast Cancer.**
   - **(i) Classification of Endocrine Tumours of Pancreas.**
   - **(j) Passaro's triangle.**

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES
MS (General Surgery) Examination, 2006

April, 2006

PAPER II

Time Allowed: 3 Hours **Full Marks: 100**

1. **Describe the aetiopathology , types , clinical features and management of choledochal cyst . (25)**

Answer. Introduction: Cystic dialatation of the common bile duct (CBD) is known as choledochal cyst, is a fairly uncommon anomaly of the biliary tract .
Todani et al classified this anomaly into 5 types. Subsequent subtypes are based on cholangiographic findings.
Aetiopathology: The exact cause of choledochal cyst remains obscure. Approximately 20% cases are found in adults.

- The most commonly accepted theory is an **anomalous pancreatic duct–biliary duct junction (APBDJ)**. A high common bile duct–pancreatic duct junction creates a **long common channel (longer than 15 mm)**. This results in reflux of pancreatic fluid into the distal common hepatic duct and results in mucosal injury, chronic inflammation, and weakening of the bile duct wall. This proposed mechanism is supported by elevated levels of amylase in choledochal cysts.
- More recently, abnormal function of the sphincter of Oddi has been reported to predispose to pancreatic reflux into the biliary tree and is therefore associated with choledochal cysts.

Types:

Todani Modification of Alonso-Lej Classification of Choledochal Cysts

Type I: Dilation of the extrahepatic biliary tree.
Type I dilations are further classified according to the shape of the affected segment into

- **Type Ia**: cystic dilation;
- **Type Ib**: focal segmental dilation;
- **Type Ic**: fusiform dilation.

Type II: Diverticular dilation of the extrahepatic biliary tree

Type III: Cystic dilation of the intraduodenal portion of the common bile duct (choledochocele)

Type IVa: Dilation of the extrahepatic and intrahepatic biliary tree

Type IVb: Dilation of multiple sections of the extrahepatic bile ducts

Type V: Dilation confined to the intrahepatic bile ducts (Caroli's disease)

Clinical Features:

- The classic clinical triad associated with choledochal cysts includes right upper quadrant pain, jaundice, and an abdominal mass. Only 10% of patients present with this triad. Adults have a slightly different presentation (abdominal pain and jaundice) than children.
- Cystolithiasis is the most frequent accompanying condition in adults with bile duct cysts.
- Pancreatitis is one of the common presentations of choledochal cysts.

Causes of pancreatitis are:

- **Activation of pancreatic enzymes by bile reflux in association with anomalous pancreaticobiliary junction or**
- **Stone obstruction of the pancreatic duct at the common channel.**

Clinical pancreatitis is present in nearly 30% of patients with choledochal cysts.

Only one third of the patients who develop pancreatitis have normal ducts.

- Severe hepatobiliary complications may result from long-standing biliary cysts if left untreated.
- Portal hypertension may develop as a result of portal vein compression by the adjacent cyst or cirrhosis secondary to long-term biliary obstruction.
- Some patients may present with variceal hemorrhage as an initial manifestation.
- Very rarely, patients may present with bilious ascites and peritonitis as a result of rupture of a choledochal cyst, and pseudocysts may appear surrounding the common bile duct.
- Those patients also have a high incidence of developing sludge, cholelithiasis, or choledocholithiasis and have commonly had a prior cholecystectomy. .
- The incidence of carcinoma (bile duct, hepatic, or gallbladder in origin) in the choledochal cyst ranges from 2.5% to 26% . Many patients have biliary cancer at the time of initial presentation.

Management:

Diagnosis:

- Laboratory evaluation may demonstrate liver dysfunction in 60% of adult patients but is not specific. The diagnosis can be established with ultrasound or CT scan.
- Cholangiography is required to determine the type of choledochal cyst and plan operative treatment.
- ERCP is more useful in defining the distal ductal anatomy and the presence of APBDJ, whereas PTC is useful in defining the proximal ductal anatomy and the presence of intrahepatic disease.

- Most patients with choledochal cysts present with some nonspecific abdominal pain, particularly in the older age groups.
- The very youngest children often have jaundice, and attention is drawn right away to the biliary system. Occasionally, a mass can be palpated in the right upper quadrant.
- Pancreatitis is also a presenting sign in some patients.
- The older patients can have very mild abdominal pain and digestive complaints in the absence of jaundice, which may make a diagnosis difficult, particularly if there is not massive cystic dilation easily identified by CT or ultrasound.

Treatment: The goals of management are to relieve symptoms and prevent long-term complications of biliary cysts such as cholangitis, portal hypertension, cirrhosis, and potential carcinoma.

Treatment Options for Choledochal Cysts :

Type 1	**Type IVa**
Cyst excision with Roux-en-Y hepaticojejunostomy	Extrahepatic cyst
If excision is impossible: choledochojejunostomy and sphincteroplasty or endoscopic sphincterotomy	Excision and Roux-en-Y hepaticojejunostomy
	Lobectomy for localized disease
	Liver transplant (?) for diffuse disease
Type II	**Type IVb**
Excision	Excision and Roux-en-Y hepaticojejunostomy
Primary closure with T-tube placement or Roux-en-Y hepaticojejunostomy	
Type III	**Type V**
Excision	Hepatic resection for localized disease
Sphincteroplasty or endoscopic retrograde cholangiopancreatography+ sphincterotomy	Diffuse disease liver transplant

Follow Up:

- Postoperatively, patients have problems mainly with recurrent cholangitis. There is a small but real potential problem of stenosis of the hepaticojejunostomy, and these patients should be followed for life with serial enzyme determinations and ultrasound.

- If partial obstruction occurs, elevation of alkaline phosphatase may be the only sign for the patient who is otherwise asymptomatic, leading to risk of biliary cirrhosis. Percutaneous or endoscopic dilation of the anastomosis or reoperation would be indicated in this case.
- Recurrent pancreatitis is also a possibility due to inadequate drainage of the pancreatic duct; endoscopic stenting is probably the best choice in these cases if anatomically possible.
- If patients have a choledochocele in the duodenum or pancreas and it is incompletely excised, follow-up is necessary to exclude malignancy.
- In the patient with type IV choledochal cysts, the intrahepatic ducts usually shrink down to near normal size in the early postoperative period. If the ducts remain significantly cystic, particularly those with multiple cysts, careful periodic observation with multiple imaging modalities is indicated to exclude development of malignancy. Treatment with prophylactic antibiotics and ursodiol (Actigall) may be indicated depending on the clinical course.
- In all of these patients, follow-up with serum liver enzymes on an annual basis is indicated to avoid missing development of partial obstruction, which may lead to permanent liver damage.

2. Discuss the causes of gastric outlet obstruction in adults. Discuss the aetiopathology, types,clinical features and management of carcinoma stomach . (25)

Answer. Causes of gastric outlet obstruction:

Benign	**Malignant**
• Peptic ulcer disease. • Infections, such as tuberculosis; and infiltrative diseases, such as amyloidosis. • A rare cause of gastric outlet obstruction is obstruction with a gallstone, also termed *Bouveret's syndrome*.	• Tumours of the stomach, including adenocarcinoma (and its linitis plastica variant), lymphoma, and gastrointestinal stromal tumours; • Malignant lymph node. • Carcinoma stomach:

Aetiopathology

Predisposing factors:

- Diet rich in nitrosamines (smoked or fresh fish, pickled fruit);
- Chronic atrophic gastritis;

- Blood group a;
- Chronic gastric ulceration related to H. Pylori.

Epidemiology

- Gastric cancer is the 14th most common cancer in the world., the incidence of which has been decreasing over the past 70 years.
- Twice as common in men as it is in women, and the incidence is higher among black men than white men.
- The incidence also increases with age, peaking in the seventh decade.
- Studies of migrant populations from areas of high incidence to areas of low incidence suggest that environmental exposure as well as other cultural or genetic factors influence the predisposition to gastric cancer.
- There has been a noticeable shift in the site of gastric cancer from the distal stomach to the more proximal stomach over the past several decades.
- The incidence of adenocarcinoma of the gastric cardia has increased steadily, whereas the incidence of cancer in other anatomic subsites has decreased.
- The increase was most noticeable for white men and is possibly linked to a history of smoking or heavy alcohol use.

Factors Associated With Increased Risk for Developing Stomach Cancer

Nutritional	Environmental	Social	Medical
• Low fat or protein consumption • Salted meat or fish • High nitrate consumption • High complex-carbohydrate consumption	• Poor food preparation (smoked, salted) • Lack of refrigeration • Poor drinking water (well water) • Smoking	• Low social class	• Prior gastric surgery • Helicobacter pylori infection • Gastric atrophy and gastritis • Adenomatous polyps • Male gender

Types

World Health Organization Histologic Typing of Gastric Cancer
Adenocarcinoma
Papillary adenocarcinoma
Tubular adenocarcinoma
Mucinous adenocarcinoma
Signet-ring cell carcinoma
Adenosquamous carcinoma
Squamous cell carcinoma

Small cell carcinoma
Undifferentiated carcinoma
Others

Lauren Classification System

INTESTINAL	DIFFUSE
Environmental	Familial
Gastric atrophy, intestinal metaplasia	Blood type A
Men >women	Women >men
Increasing incidence with age	Younger age group
Gland formation	Poorly differentiated, signet ring cells
Hematogenous spread	Transmural/lymphatic spread
Microsatellite instability APC gene mutations	Decreased E-cadherin
p53, p16 inactivation	p53, p16 inactivation

APC, adenomatous polyposis coli.

TNM Classification of Carcinoma of the Stomach

CATEGORY	CRITERIA
Primary Tumor (T)	
TX	Primary tumor cannot be assessed
T0	No evidence of primary tumor
Tis	Carcinoma in situ: intraepithelial tumor without invasion of the lamina propria
T1	Tumor invades lamina propria or submucosa
T2	Tumor invades muscularis propria or subserosa
T2a	Tumor invades muscularis propria
T2b	Tumor invades subserosa
T3	Tumor penetrates serosa (visceral peritoneum) without invasion of adjacent structures
T4	Tumor invades adjacent structures
Regional Lymph Nodes (N)	

CATEGORY	CRITERIA
NX	Regional lymph node(s) cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in 1 to 6 regional lymph nodes
N2	Metastasis in 7 to 15 regional lymph nodes
N3	Metastasis in more than 15 regional lymph nodes
Distant Metastasis (M)	
MX	Distant metastasis cannot be assessed
M0	No distant metastasis
M1	Distant metastasis

Preoperative Evaluation:

- When gastric cancer is suspected based on history and physical examination, flexible upper endoscopy is the diagnostic modality of choice. Although double-contrast barium upper GI radiography is cost-effective with 90% diagnostic accuracy, the inability to distinguish benign from malignant gastric ulcers makes endoscopy preferable. During endoscopy, multiple biopsy samples (seven or more) should be obtained around the ulcer crater to facilitate histologic diagnosis. The addition of direct brush cytology to multiple biopsy specimens may increase the diagnostic accuracy of the study. Additionally, the size, location, and morphology of the tumor should be noted and other mucosal abnormalities carefully evaluated.
- In select patients with advanced disease EUS can gauge the extent of gastric wall invasion as well as evaluate local nodal status. However, EUS cannot reliably distinguish tumor from fibrosis; therefore, it is not a good modality for evaluating response to therapy.
- When the diagnosis of gastric cancer is confirmed, further studies should include a complete blood count, serum chemistries to include liver function tests, coagulation studies, chest x-ray, and CT scan of the abdomen.
- In women, a pelvic CT scan or ultrasound is also recommended. CT of the chest may be needed for proximal gastric cancers. CT can readily detect the presence of visceral metastatic disease as well as malignant ascites. The major limitations of CT are in the evaluation of early gastric primaries and in the detection of small (<5 mm) metastases in the liver or on peritoneal surfaces.
- Because of the inaccuracy of CT and other modalities for the detection of macrometastases smaller than 5 mm on the peritoneal surface or liver, laparoscopy is recommended as the next step in the evaluation of patients with locoregional disease.
- The addition of laparoscopic ultrasonography may increase the sensitivity of laparoscopic staging in gastric cancer as it has in other abdominal malignancies. However, given the limitations of the available data and the operator-dependent

nature of the technique, further investigation is required to define the role of ultrasound in the staging of gastric cancer.

- Cytologic analysis of peritoneal fluid or of fluid obtained by peritoneal lavage may reveal the presence of free intraperitoneal gastric cancer cells, identifying patients with otherwise occult carcinomatosis. Patients with positive findings on peritoneal cytology have a poor prognosis, similar to that of patients with macroscopic stage IV disease. However, false-positive results can be obtained, and not all studies confirm the prognostic significance of positive findings.
- More sensitive methods of detecting free intraperitoneal gastric cancer cells, such as immunostaining and reverse-transcriptase polymerase chain reaction for carcinoembryonic antigen (CEA) messenger RNA, are under investigation.

Staging

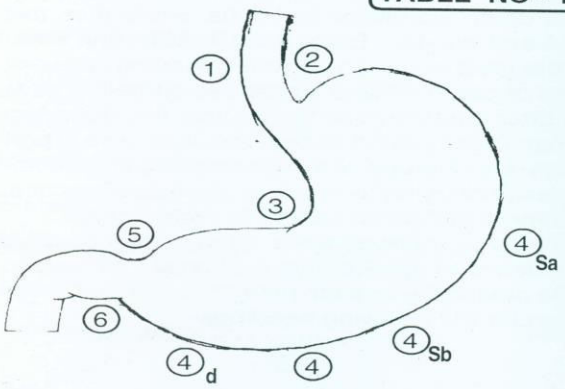
Many staging systems have been proposed for gastric adenocarcinoma. A basic understanding of the older systems is necessary to understand the literature. The pathologic staging system currently in use worldwide is the AJCC TNM staging system. TNM (already mentioned) stands for tumor, nodes, and metastasis and is based on depth of primary tumor invasion through the gastric wall, the number of involved lymph nodes, and the presence or absence of distant metastasis. The TNM system can adequately stratify patients into distinct groups with different risks for tumor-related death. A major revision occurred in the AJCC staging system for gastric cancer in 1997 when nodal status stratification was changed from location of nodes to number of positive nodes. In the current staging system, a minimum of 15 nodes must be evaluated for accurate staging. Nodal staging is then determined by the number of positive nodes, with pN1 reflecting 1 to 6 positive nodes, pN2 designating 7 to 15 positive nodes, and pN3 more than 15 positive nodes. Some data suggest location of the primary (cardia compared with distal tumors) may independently predict survival. However, the current AJCC staging system does not reflect the poorer prognosis for proximal gastric tumors noted in some studies.

The term *R status* was first described by Hermanek in 1994 and is used to describe the tumor status after resection. R0 describes a microscopically margin-negative resection, in which no gross or microscopic tumor remains in the tumor bed. R1 indicates removal of all macroscopic disease, but microscopic margins are positive for tumor. R2 indicates gross residual disease. Because the extent of resection can influence survival, some authors include this R designation to complement the TNM system. Long-term survival can be expected only after an R0 resection; therefore, a significant effort should be made to avoid R1 or R2 resection.

EXTENT OF LYMPHADENECTOMY

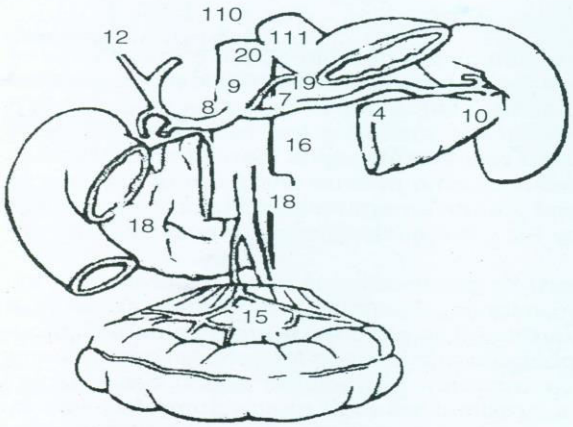
It has been observed that lymphatic spread of the gastric cancer can occur in absence of haematogenous spread and gastric cancer may remain localized disease even when nodes are involved. This underlies the concept of lymphadenectomy as a surgical method for curing gastric cancer.

TABLE NO - 1



1. Right Gastric
2. Left Cardiac
3. Lesser Curvature
4. Greater Curvature
   Sa: Short Gastric
   Sb: Left Gastro-epiploic
   d: Right Gastro-epiploic
5. Supra pyloric
6. Subpyloric

PERIGASTRIC LYMPH NODES



PERIGASTRIC LYMPH NODES

7. Left Gastric Art.
8. Common Hepatic Art.
   a. anterior common
   b. posterior common
9. Coeliac Art.
10. Splenic Hilum
11p. Proximal Splenic
11d. Distal Splenic
12a. Left Hepatoduodenal
12b,p. Post Hepatoduodenal
13. Retropancreatic
14v. Superior Mesenteric Vein
14a. Superior Mesenteric Art.
15. Middle Colic
16. Para-Aortic
19. Infradiaphragmatic
20. Esophageal Hiatus
110. Lower Paraesophageal
111. Supradiaphragmatic

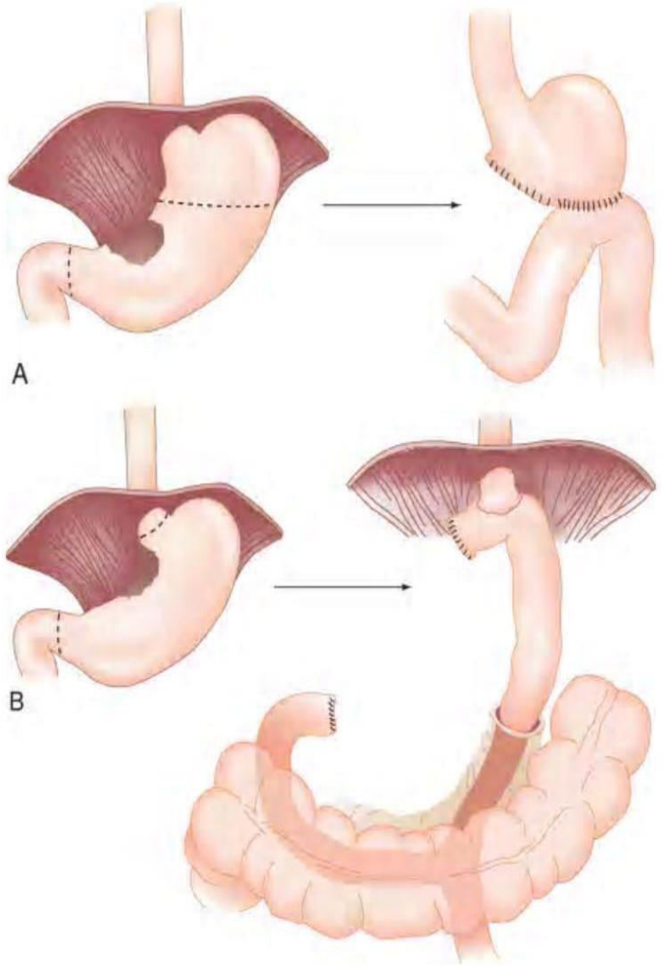
Grouping of Regional Lymph Nodes (Groups 1-3) by Location of Primary Tumor According to the Japanese Classification of Gastric Carcinoma

		LOCATION OF PRIMARY TUMOR IN STOMACH		
LYMPH NODE STATION (NO.)	**DESCRIPTION**	**Upper Third**	**Middle Third**	**Lower Third**
1	Right paracardial	1	1	2

		LOCATION OF PRIMARY TUMOR IN STOMACH		
LYMPH NODE STATION (NO.)	**DESCRIPTION**	**Upper Third**	**Middle Third**	**Lower Third**
2	Left paracardial	1	3	M
3	Lesser curvature	1	1	1
4sa	Short gastric	1	3	M
4sb	Left gastroepiploic	1	1	3
4d	Right gastroepiploic	2	1	1
5	Suprapyloric	3	1	1
6	Infrapyloric	3	1	1
7	Left gastric artery	2	2	2
8a	Anterior comm. hepatic	2	2	2
8p	Posterior comm. hepatic	3	3	3
9	Celiac artery	2	2	2
10	Splenic hilum	2	3	M
11p	Proximal splenic	2	2	2
11d	Distal splenic	2	3	M
12a	Left hepatoduodenal	3	2	2
12b,p	Posterior hepatoduodenal	3	3	3
13	Retropancreatic	M	3	3
14v	Superior mesenteric vein	M	3	2
14a	Superior mesenteric artery	M	M	M
15	Middle colic	M	M	M
16al	Aortic hiatus	3	M	M
16a2,b1	Para-aortic, middle	M	3	3
16b2	Para-aortic, caudal	M	M	M

Surgical Treatment

- The optimal surgical management of gastric cancer must be tailored to the extent and location of disease. In the absence of distant metastatic spread, aggressive surgical resection of the gastric tumor is justified. The extent of gastric resection is determined by the need to obtain a resection margin free of microscopic disease. Because gastric tumors are characterized by extensive intramural spread, a line of resection at least 6 cm from the tumor mass is necessary to ensure a low rate of anastomotic recurrence. The appropriate surgical procedure should be determined by the location of the tumor and the known pattern of spread.
- Tumors of the cardia and proximal stomach account for 35% to 50% of all gastric adenocarcinomas. In general, proximal tumors are more advanced at presentation than more distant tumors, so curative resections are rare. For proximal lesions, either total gastrectomy or proximal gastric resection is necessary to remove the tumor



A, Subtotal gastrectomy with a Billroth II anastomosis. **B,** Total gastrectomy with a Roux-en-Y anastomosis.

- Although there is no evidence that one operation is better than the other for tumor removal, there is abundant evidence that proximal gastric resection results in higher morbidity and mortality than total gastrectomy. Total gastrectomy should be considered the procedure of choice for proximal gastric lesions. Distal tumors account for about 35% of all gastric cancers. Because recent studies have indicated no difference in 5-year survival between patients undergoing potentially curative subtotal versus total gastrectomy, subtotal gastrectomy is appropriate for patients in whom a negative margin resection can be performed. A luminal margin of 5 to 6 cm is recommended with frozen-section analysis when a subtotal gastric resection is performed for adenocarcinoma.

A D1 resection refers to the removal of group 1 lymph nodes, D2 to dissection of group 1 and 2, and D3 resection to a D2 resection plus removal of para-aortic lymph nodes. To effect complete removal of station 10 (parasplenic) and station 11 (parapancreatic), Japanese surgeons perform splenectomy and partial pancreatectomy during D2 resections for primaries whose drainage includes these echelons. Because of the increased morbidity in the patients receiving these adjunctive resections, Western surgeons do not typically resect the spleen or pancreas unless involved by direct extension from a T4 tumor.

Splenectomy is no longer advocated as a routine adjunctive procedure to gastrectomy for cancer. Local organ resection, especially of the spleen, pancreas, or transverse colon, should be performed only when needed to accomplish an R0 resection.

Palliative Treatment

- Because 20% to 30% of gastric cancer patients present with stage IV disease, clinicians must be familiar with different methods of palliative treatment. The goal of palliative treatment is the relief of symptoms with minimal morbidity.
- Surgical palliation of advanced gastric cancer may include resection or bypass alone or in conjunction with percutaneous, endoscopic, or radiotherapy techniques. Complete staging is necessary to determine the appropriate method of palliation for individual patients. In the presence of peritoneal disease, hepatic metastases, diffuse nodal metastases, or ascites, palliation of bleeding or proximal gastric obstruction would preferably be obtained nonoperatively.
- Nonoperative therapies include laser recannulization and endoscopic dilation with or without stent placement. Patients who undergo stent placement for gastric outlet obstruction are frequently able to tolerate solid foods and may not require additional interventions.

Adjuvant Therapy

- Two cycles of 5-fluorouracil and leucovorin with subsequent concurrent chemoradiotherapy, using the same chemotherapeutic agents, as adjuncts following an R0 resection of gastric adenocarcinoma can be used.
- More recently, a British MRC randomized trial (MAGIC) demonstrated improved overall and disease-free survival in patients treated with preoperative or postoperative chemotherapy (epirubicin, cisplatin, and 5-fluorouracil).

- Neoadjuvant chemotherapy is currently under investigation and has yielded some promising results. A few studies have demonstrated acceptable toxicities, with an increased number of patients completing all planned therapy and undergoing R0 resections compared with historic controls. Currently, NCCN guidelines recommend adjuvant chemoradiation with a 5-fluorouracil–based regimen following complete surgical resection for patients with T3, T4, or node-positive cancers. Additionally, patients with microscopically positive resection margins should also have adjuvant therapy.

3. Write short notes on: 5 x 10
(a) Newer classification of inguinal hernia.
(b) Investigations of Peptic Ulcer Syndrome.
(c) Lymphatic manifestations of Breast Carcinoma.
(d) Staging of squamous cell carcinoma
(e) Complications and Medical treatment of Hydatid Cyst.
(f) CT Scan.
(g) ANDI.
(h) FNAC Scoring of Breast Cancer.
(i) Classification of Endocrine Tumours of Pancreas.
(j) Passaro's triangle.

Answers.

(a) Newer classification of inguinal hernia.

Nyhus Classification System	
Type I	Indirect hernia; internal abdominal ring normal; typically in infants, children, small adults
Type II	Indirect hernia; internal ring enlarged without impingement on the floor of the inguinal canal; does not extend to the scrotum
Type IIIA	Direct hernia; size is not taken into account

Type IIIB	Indirect hernia that has enlarged enough to encroach upon the posterior inguinal wall; indirect sliding or scrotal hernias are usually placed in this category because they are commonly associated with extension to the direct space; also includes pantaloon hernias
Type IIIC	Femoral hernia
Type IV	Recurrent hernia; modifiers A–D are sometimes added, which correspond to indirect, direct, femoral, and mixed, respectively

Gilbert Classification System

Type 1	Small, indirect
Type 2	Medium, indirect
Type 3	Large, indirect
Type 4	Entire floor, direct
Type 5	Diverticular, direct
Type 6	Combined (pantaloon)
Type 7	Femoral

(Schwartz's Surgery > Part II. Specific Considerations > Chapter 36. Inguinal Hernias)

(b) Investigations of Peptic Ulcer Syndrome.

Answer.

- History and physical examination are probably of limited value in distinguishing between gastric and duodenal ulceration. Routine laboratory studies include a complete blood count, liver chemistries, serum creatinine, and calcium levels.
- A serum gastrin level should also be obtained in patients with ulcers that are refractory to medical therapy or require surgery.
- An upright chest radiograph is usually performed when ruling out perforation.
- The two principal means of diagnosing peptic ulcers are upper GI radiography and fiberoptic endoscopy.
- Contrast radiography is less expensive, and most (90%) can be diagnosed accurately. However, about 5% of ulcers that appear radiographically benign are malignant. H. pylori testing should also be done in all patients with suspected PUD.

H. pylori Testing:

Diagnostic tests for H. pylori are divided between tests that do or do not require a sample of gastric mucosa. The noninvasive tests available are serology and the carbon-labeled urea breath test. The invasive tests available are the rapid urease test, histology, and culture. Noninvasive tests do not require endoscopy, whereas invasive tests do.

Serology

Because H. pylori infection elicits a local as well as a systemic immunoglobulin G (IgG)-mediated immune response, serology can be used to diagnose H. pylori. There are a variety of enzyme-linked immunosorbent assay (ELISA) laboratory-based tests available as well as some rapid office-based immunoassays. Serology is the diagnostic test of choice when endoscopy is not indicated and has about 90% sensitivity and specificity associated with it. Serology testing, however, is not without its limitations because antibody titers can remain high for a year or more, and consequently, this test cannot be used to assess eradication after therapy.

Urea Breath Test

Another noninvasive test used for diagnosing H. pylori is the carbon-labeled urea breath test. This test is based on the ability of H. pylori to hydrolyze urea. Its sensitivity and specificity are both greater than 95%. The test is performed by having the patient ingest a carbon isotope–labeled urea using either ^{14}C or ^{13}C. If ^{13}C is used, mass spectrometry is required, whereas it is not required for ^{14}C, but ^{14}C is associated with a low level of radiation exposure. After ingestion of the carbon isotope, urea will be metabolized to ammonia and labeled bicarbonate if H. pylori infection is present. The labeled bicarbonate is excreted in the breath as labeled carbon dioxide, which is then quantified. The urea breath test is less expensive than endoscopy and samples the entire stomach. False-negative results can occur if the test is done too soon after treatment, so it is usually best to test 4 weeks after therapy is finished. The urea breath test is the method of choice to document eradication.

Rapid Urease Assay

The method of choice to diagnosis H. pylori if endoscopy is employed is the rapid urease test. This is another test based on the ability of H. pylori to hydrolyze urea. The enzyme urease catalyzes degradation of urea to ammonia and bicarbonate, creating an alkaline environment that can be detected by a pH indicator. Consequently, endoscopy is performed and gastric mucosal tissue biopsied. Mucosal biopsy samples are placed into a liquid or solid medium containing urea and a pH indicator. Sensitivity is about 90% and specificity 98%, and the results are available within hours.

Histology

Endoscopy can also be performed with biopsy samples of gastric mucosa followed by histologic visualization of H. pylori. H. pylori is identified by its appearance and colonization sites with routine hematoxylin and eosin stains or with special stains such as

silver, Giemsa, or Genta, for improved visibility. Sensitivity is about 95% and specificity 99%. This test is widely available and affords the clinician the ability to assess the severity of gastritis as well as to confirm the presence or absence of the organism.

Culture

Culturing of gastric mucosa obtained at endoscopy can also be performed to diagnosis H. pylori. The sensitivity is about 80% and specificity 100%. However, it requires laboratory expertise, it is not widely available, it is relatively expensive, and diagnosis requires up to 3 to 5 days until a diagnosis is made. Nevertheless, it does provide the opportunity to perform antibiotic sensitivity testing on isolates should the need arise.

H. pylori Testing Summary

In summary, it is not necessary to perform endoscopy to diagnose H. pylori. Serology is the test of choice for initial diagnosis when endoscopy is not required. If, however, endoscopy is to be performed, the rapid urease assay and histology are both excellent options, but the cost advantage lies with the rapid urease assay. After treatment, the urea breath test is the method of choice but again should not be performed until 4 weeks after therapy ends. If the breath test is unavailable, endoscopy may be performed in selected patients such as those with bleeding ulcers or other complications of their PUD.

Upper Gastrointestinal Radiography

Diagnosis of peptic ulcer by upper GI radiography requires the demonstration of barium within the ulcer crater, which is usually round or oval, and may or may not be surrounded by edema. This study is useful to determine the location and the depth of penetration of the ulcer as well as the extent of deformation from chronic fibrosis. The ability to detect ulcers on radiography does require the technical skills and abilities of the radiologist but is also dependent on the size and location of the ulcer. With single-contrast radiographic techniques, as many as 50% of duodenal ulcers may be missed, whereas with double-contrast studies, 80% to 90% of ulcer craters can be detected. The location of a gastric ulcer is of little predictive value in establishing malignancy as benign, and malignant ulcers can occur anywhere in the stomach. However, the size of the gastric ulcer may have some predictive value in that larger lesions are more likely to be malignant than smaller ones. In addition, the finding of an ulcer with an associated mass; interrupted, fused, or nodular mucosal folds approaching the margin of the crater; or an ulcer with irregular filling defects in the ulcer crater is suggestive of a malignancy.

Fiberoptic Endoscopy

Endoscopy is the most reliable method of diagnosing a gastric ulcer. When multiple biopsies and brushings for cytology are performed, the probability of diagnosing a malignancy is in excess of 90%. In general, benign ulcers have smoother, more regular, rounded edges with a flat, smooth ulcer base. Malignancy is more often associated with a

mass that may protrude into the lumen or have folds surrounding the ulcer crater that are nodular, clubbed, fused, or stop short of the ulcer margin. Again, multiple biopsy specimens are necessary for any of these ulcers because ruling out a malignancy is mandatory

In addition to providing diagnostic abilities, endoscopy provides the ability to sample tissue for H. pylori testing and may also be used for therapeutic purposes in the setting of GI bleeding.

(c) Lymphatic manifestations of Breast Carcinoma.

Answer.

Introduction: Breast cancer develops when normal cells within the lobules, ducts or nipple of the breast begin to proliferate uncontrollably. In the beginning stages of cancer, the breast cells develop genetic mutations that promote tumor development. As cancer progresses, the cancer cells invade neighboring tissues in a process called metastasis. Breast cancer cells commonly spread to the lymph nodes, a series of structures that make up part of the immune system. Breast cancer spread to the lymph nodes can exhibit a number of symptoms.

Lymphatic manifestations are as follow:

- **Swollen Lymph Nodes in Armpit**

A common symptom initially experienced by patients with breast cancer that has invaded the lymph nodes is lymph node swelling. Under normal conditions, the lymph nodes cannot be felt underneath the skin. When cancer cells invade the lymph nodes, they proliferate uncontrollably within the nodes, leading to an increase in lymph node size. Patients with invasive breast cancer may notice a swelling in the armpit on the same side as the affected breast, corresponding to swollen lymph nodes. As breast cancer progresses, cancer growth within the lymph nodes leads to node fusion and matting, which also causes a noticeable lump.

- **Lymphedema**

Patients with invasive breast cancer may also develop a symptom called lymphedema if their breast cancer spreads to the lymph nodes. Under normal conditions, the lymph nodes allow for circulation of lymphatic fluid. In invasive breast cancer, swelling of the lymph nodes can lead to blockages in the lymphatic system and prevent the circulation and drainage of lymph fluid, called lymphedema. Patients with lymphedema experience swelling of the arm and the hand on the affected side of the body, whch often results in pain and difficulty moving the affected limb. Breast cancer patients who have undergone mastectomy to treat cancer may also suffer from lymphedema, since the removal of lymph nodes during surgery disrupts lymph fluid drainage.

- **Chest Tenderness and Pain**

As the breast cancer progresses, the cancer cells that invaded lymph nodes in the armpit can travel to other lymph nodes throughout the body via the lymphatic system. Breast cancer in the lymph nodes can commonly spread to lymph nodes underneath the

breastbone and around the clavicles. When lymph nodes within the chest cavity enlarge, they put pressure on surrounding healthy tissue, which may lead to chest pain. Breast cancer patients experiencing chest pain should seek immediate medical attention to test the extent of cancer metastasis.

- **Lymph nodal metastasis:** Axillary, internal mammary , infraclavicular and supraclavicular nodes can be involved.

(d) Staging of squamous cell carcinoma .

Answer. American Joint Committee on Cancer System for Classification and Staging of Squamous Cell Carcinoma

Primary Tumor (T)			
TX	Primary tumor cannot be assessed		
T0	No evidence of primary tumor		
Tis	Carcinoma in situ		
T1	Tumor ≤2 cm in greatest dimension		
T2	Tumor >2 cm, but not >5 cm, in greatest dimension		
T3	Tumor >5 cm in greatest dimension		
T4	Tumor invades deep extradermal structures (i.e., cartilage, skeletal muscle, or bone)		
Regional Lymph Nodes (N)			
NX	Regional lymph nodes cannot be assessed		
N0	No regional lymph node metastasis		
N1	Regional lymph node metastasis		
Distant Metastasis (M)			
MX	Distant metastasis cannot be assessed		
M0	No distant metastasis		
M1	Distant metastasis		
Stage Grouping			
Stage 0	Tis	N0	M0
Stage I	T1	N0	M0
Stage II	T2	N0	M0
	T3	N0	M0
Stage III	T4	N0	M0
	Any T	N1	M0

Stage IV	Any T	Any N	M1

(e) Complications and Medical treatment of Hydatid Cyst.

Answer.
Introduction: Echinococcosis (hydatid disease) is a zoonosis caused by the larval stage of *Echinococcus granulosus* (also known as *Taenia echinococcus*). Humans are accidental intermediate hosts, whereas animals can be both intermediate hosts and definitive hosts. The two main types of hydatid disease are caused by *E. granulosus* and *E. multilocularis*. Infection with echinococcal organisms is the most common cause of liver cysts in the world.

Medical therapy for echinococcosis is limited to the benzimidazoles (mebendazole and albendazole) and used alone is only 30% successful. Albendazole is readily absorbed from the intestine and metabolized by the liver to an active form. Mebendazole is poorly absorbed and is inactivated by the liver. Albendazole is thus the drug of choice for medical therapy. Greater success rates may be seen in extrahepatic manifestations of the disease and with the alveolar form caused by *E. multilocularis*. Given for at least 3 months preoperatively, albendazole reduces the recurrence rate when cyst spillage, partial cyst removal, or biliary rupture has occurred. Duration of therapy in these instances is at least 1 month.

Complications:

- Complications from hydatid cysts are seen in one-third of patients.
- Most commonly, the cyst ruptures internally or externally, followed by secondary infection, anaphylactic shock, and liver replacement, in order of decreasing frequency.
- Viable hydatid cysts are space-occupying lesions with a tendency to grow. In confined areas such as the central nervous system, even small cysts can cause severe symptoms. In less confined areas, symptoms depend on the site and size of the cyst. Symptoms result from direct pressure or distortion of neighboring structures or viscera. Compressive atrophy of the surrounding hepatocytes and fibrosis occurs, and these cysts may grow to such an enormous size that they replace an entire lobe.
- As the cysts enlarge, they may also rupture. If rupture of only the endocyst occurs, the content is retained within the pericyst. A communicating rupture is a rupture into the biliary or bronchial tree.Frank intrabiliary rupture is the most common complication of hydatid cysts, and is reported in 5–25% of cases. A free rupture occurs when hydatid contents rupture throughout the peritoneal, pleural, or pericardial cavity. Acute symptomatic rupture into the peritoneal cavity occurs in 1–4% of patients and may precipitate anaphylactic shock

(f) CT Scan.

Answer.

- CT scanning combines special x-ray equipment with sophisticated computers to produce multiple images or pictures of the inside of the body. These cross-sectional images of the area being studied can then be examined on a computer monitor, printed or transferred to a CD.
- CT scans of internal organs, bones, soft tissue and blood vessels provide greater clarity and reveal more details than regular x-ray exams.
- Using specialized equipment and expertise to create and interpret CT scans of the body, radiologists can more easily diagnose problems such as cancers, cardiovascular disease, infectious disease, appendicitis, trauma and musculoskeletal disorders.

Some common uses:

- One of the best and fastest tools for studying the chest, abdomen and pelvis because it provides detailed, cross-sectional views of all types of tissue.
- Often the preferred method for diagnosing many different cancers, including lung, liver and pancreatic cancer, since the image allows a physician to confirm the presence of a tumor and measure its size, precise location and the extent of the tumor's involvement with other nearby tissue.
- An examination that plays a significant role in the detection, diagnosis and treatment of vascular diseases that can lead to stroke, kidney failure or even death. CT is commonly used to assess for pulmonary embolism (a blood clot in the lung vessels) as well as for abdominal aortic aneurysms (AAA).
- Invaluable in diagnosing and treating spinal problems and injuries to the hands, feet and other skeletal structures because it can clearly show even very small bones as well as surrounding tissues such as muscle and blood vessels.

In pediatric patients, CT is rarely used to diagnose tumors of the lung or pancreas as well as abdominal aortic aneurysms.

For children, CT imaging is more often used to evaluate:

Lymphoma	Neuroblastoma	Congenital malformations of blood vessels	The kidneys

Physicians often use the CT examination to:

- Quickly identify injuries to the lungs, heart and vessels, liver, spleen, kidneys, bowel or other internal organs in cases of trauma.
- Guide biopsies and other procedures such as abscess drainages and minimally invasive tumor treatments.
- Plan for and assess the results of surgery, such as organ transplants or gastric bypass.
- Stage, plan and properly administer radiation treatments for tumors as well as monitor response to chemotherapy.
- Measure bone mineral density for the detection of osteoporosis.

In many ways CT scanning works very much like other x-ray examinations. X-rays are a form of radiation—like light or radio waves—that can be directed at the body. Different body parts absorb the x-rays in varying degrees.

- In a conventional x-ray exam, a small burst of radiation is aimed at and passes through the body, recording an image on photographic film or a special image recording plate. Bones appear white on the x-ray; soft tissue shows up in shades of gray and air appears black.
- With CT scanning, numerous x-ray beams and a set of electronic x-ray detectors rotate around you, measuring the amount of radiation being absorbed throughout your body. At the same time, the examination table is moving through the scanner, so that the x-ray beam follows a spiral path. A special computer program processes this large volume of data to create two-dimensional cross-sectional images of your body, which are then displayed on a monitor. This technique is called helical or spiral CT.
- CT imaging is sometimes compared to looking into a loaf of bread by cutting the loaf into thin slices. When the image slices are reassembled by computer software, the result is a very detailed multidimensional view of the body's interior.
- Refinements in detector technology allow new CT scanners to obtain multiple slices in a single rotation. These scanners, called "multislice CT" or "multidetector CT," allow thinner slices to be obtained in a shorter period of time, resulting in more detail and additional view capabilities.
- Modern CT scanners are so fast that they can scan through large sections of the body in just a few seconds. Such speed is beneficial for all patients but especially children, the elderly and critically ill.
- For children, the CT scanner technique will be adjusted to reduce the radiation dose.
- For some CT exams, a contrast material is used to enhance visibility in the area of the body being studied.

Benefits

- CT scanning is painless, noninvasive and accurate.
- A major advantage of CT is its ability to image bone, soft tissue and blood vessels all at the same time.
- Unlike conventional x-rays, CT scanning provides very detailed images of many types of tissue as well as the lungs, bones, and blood vessels.
- CT examinations are fast and simple; in emergency cases, they can reveal internal injuries and bleedingquickly enough to help save lives.
- CT has been shown to be a cost-effective imaging tool for a wide range of clinical problems.
- CT is less sensitive to patient movement than MRI.
- CT can be performed if you have an implanted medical device of any kind, unlike MRI.
- CT imaging provides real-time imaging, making it a good tool for guiding minimally invasive procedures such as needle biopsies and needle aspirations of many areas of the body, particularly the lungs, abdomen, pelvis and bones.
- A diagnosis determined by CT scanning may eliminate the need for exploratory surgery and surgical biopsy.
- No radiation remains in a patient's body after a CT examination.
- X-rays used in CT scans usually have no immediate side effects.

Risks:

- There is always a slight chance of cancer from excessive exposure to radiation. However, the benefit of an accurate diagnosis far outweighs the risk.
- The effective radiation dose for this procedure varies. CT scanning is, in general, not recommended for pregnant women unless medically necessary because of potential risk to the baby.
- Nursing mothers should wait for 24 hours after contrast material injection before resuming breast-feeding.
- The risk of serious allergic reaction to contrast materials that contain iodine is extremely rare, and radiology departments are well-equipped to deal with them.
- Because children are more sensitive to radiation, they should have a CT study only if it is essential for making a diagnosis and should not have repeated CT studies unless absolutely necessary.

Limitations:

Soft-tissue details in areas such as the brain, internal pelvic organs, knee or shoulder can be more readily and clearly seen with magnetic resonance imaging (MRI). The exam is not generally indicated for pregnant women.
A person who is very large may not fit into the opening of a conventional CT scanner or may be over the weight limit for the moving table which is usually about 450 pounds.

(g) ANDI.

Answer. ANDI: Aberrations of Normal Development and Involution

The basic principles underlying the aberrations of normal development and involution (ANDI) classification of benign breast conditions are:

- Benign breast disorders and diseases are related to the normal processes of reproductive life and to involution;
- There is a spectrum of breast conditions that ranges from normal to disorder to disease;
- The ANDI classification encompasses all aspects of the breast condition, including pathogenesis and the degree of abnormality.

ANDI Classification of Benign Breast Disorders			
	Normal	**Disorder**	**Disease**
Early reproductive years (age 15–25)	Lobular development	Fibroadenoma	Giant fibroadenoma
	Stromal development	Adolescent	Gigantomastia

		hypertrophy	
	Nipple eversion	Nipple inversion	Subareolar abscess
			Mammary duct fistula
Later reproductive years (age 25–40)	Cyclical changes of menstruation	Cyclical mastalgia	Incapacitating mastalgia
	Nodularity		
	Epithelial hyperplasia of pregnancy	Bloody nipple discharge	
Involution (age 35–55)	Lobular involution	Macrocysts	
		Sclerosing lesions	
	Duct involution		
	–Dilatation	Duct ectasia	Periductal mastitis
	–Sclerosis	Nipple retraction	
	Epithelial turnover	Epithelial hyperplasia	Epithelial hyperplasia with atypia

(h) FNAC Scoring of Breast Cancer.

Answer. Introduction: Fine needle aspiration cytology (FNAC) of the breast is becoming an increasingly popular investigation in the diagnosis of breast malignancies and their further subtyping. The breast lump and cytological specimen were processed and reported routinely.

After FNAC cytology specimens can be graded by the pathologist, without knowledge of the histology result, in accordance with the NHS Breast screening programme:

- Cl = insufficient cells for cytological analysis, i.e. fewer than five epithelial cell groups.
- C2 = cells present all benign; no suspicious features.
- C3 = cells suspicious but probably benign.
- C4 = cells suspicious but probably malignant.
- C5 = Definitely malignant. Within this unit, this category is used when the degree of diagnostic certainty is such that the pathologist would be happy for the patient to undergo major breast surgery on the basis of the cytology sample alone.

(i) Classification of Endocrine Tumours of Pancreas.

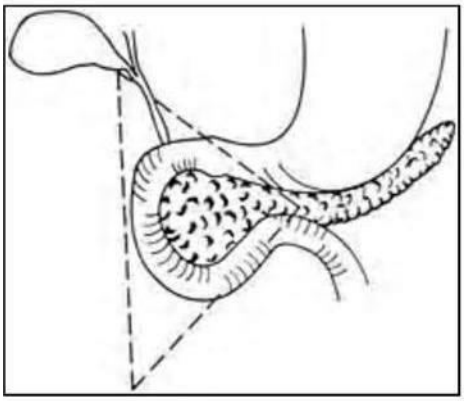
Answer. Classification of Pancreatic Endocrine Tumors

Tumor Name	**Major Hormone(s)**	**Syndromes**	**Eponym**
Insulinoma	Insulin	Hypoglycemic	
Gastrinoma	Gastrin	Ulcerogenic	Zollinger-Ellison

Tumor Name	Major Hormone(s)	Syndromes	Eponym
			syndrome
VIPoma	Vasoactive intestinal polypeptide	Watery diarrhea, hypokalemia, achlorhydria	Verner-Morrison syndrome
Glucagonoma	Glucagon cutaneous	Hyperglycemic	
Nonfunctioning	None	None	
Insulinoma	Insulin	Hypoglycemic	
	Substance P and serotonin		
Tumors (PPomas) are without endocrine symptoms	Pancreatic polypeptide (met-enkephalin, PHI)		
Nonfunctioning	None	None	

(j) Passaro's triangle.

Answer. Passaro's Triangle: In 70% to 90% of patients, the primary gastrinoma is found in Passaro's triangle, an area defined by a triangle with points located at the junction of the cystic duct and common bile duct, the second and third portion of the duodenum, and the neck and body of the pancreas.



THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES
MS (General Surgery) Examination, 2010

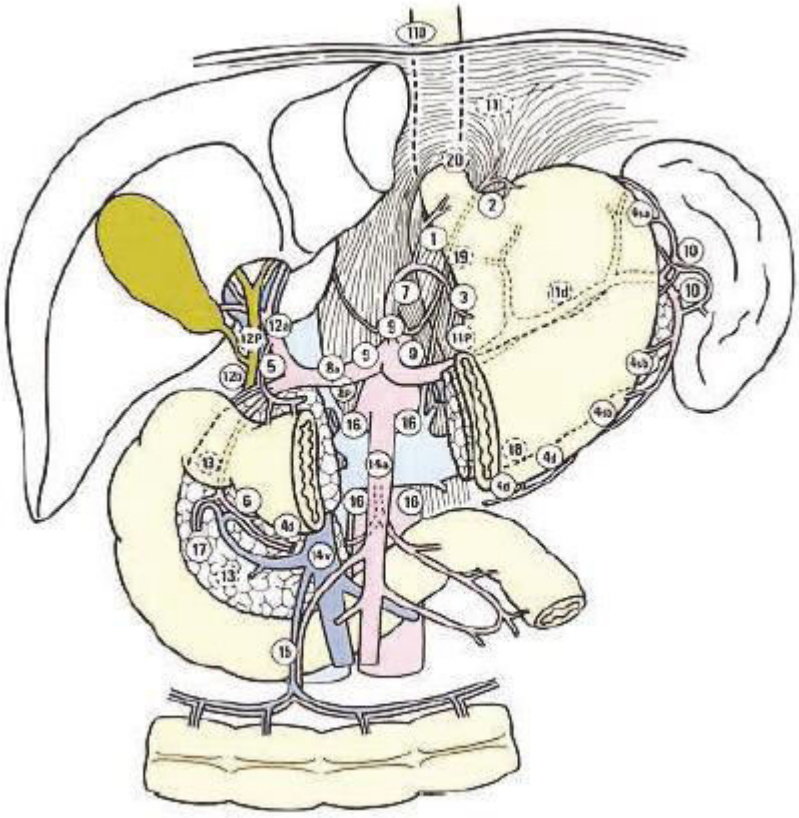
May 2010

PAPER III

Time Allowed: 3 Hours
Full Marks:100

1. **Discuss the evidence based comparison of D1 and D2 dissections for carcinoma of stomach. 20**

Answer. The lymph node stations according to Japanese classification.



No. 1 Right paracardial LN
No. 2 Left paracardial LN
No. 3 LN along the lesser curvature
No. 4sa LN along the short gastric vessels
No. 4sb LN along the left gastroepiploic vessels
No. 4d LN along the right gastroepiploic vessels
No. 5 Suprapyloric LN
No. 6 Infrapyloric LN
No. 7 LN along the left gastric artery
No. 8a LN along the common hepatic artery (Anterosuperior group)
No. 8p LN along the common hepatic artery (Posterior group)
No. 9 LN around the celiac artery

No. 10 LN at the splenic hilum
No. 11p LN along the proximal splenic artery
No. 11d LN along the distal splenic artery
No. 12a LN in the hepatoduodenal
ligament (along the hepatic artery)
No. 12b LN in the hepatoduodenal ligament (along the bile duct)
No. 12p LN in the hepatoduodenal ligament (behind the portal vein)
No. 13 LN on the posterior surface of the pancreatic head
No. 14v LN along the superior mesenteric vein
No. 14a LN along the superior mesenteric artery
No. 15 LN along the middle colic vessels
No. 16a1 LN in the aortic hiatus
No. 16a2 LN around the abdominal aorta (from the upper margin of the celiac trunk to the lower margin of the left renal vein)
No. 16b1 LN around the abdominal aorta (from the lower margin of the left renal vein to the upper margin of the inferior mesenteric artery)
No. 16b2 LN around the abdominal aorta (from the upper margin of the inferior mesenteric artery to the aortic bifurcation)
No. 17 LN on the anterior surface of the pancreatic head
No. 18 LN along the inferior margin of the pancreas
No. 19 Infradiaphragmatic LN
No. 20 LN in the esophageal hiatus of the diaphragm
No. 110 Paraesophageal LN in the lower thorax
No. 111 Supradiaphragmatic LN
No. 112 Posterior mediastinal LN

The Japanese introduced the concept of tiers (level) with lymphatic spread. Nodal stations defined as level (tier) N1, N2 and N3 vary depending on location of tumour.

- level(tier) N1 lymph nodes are generally perigastric nodes within 3 cm of primary tumour
- level(tier) N2 are distant perigastric node and along the main arteries supplying stomach (hepatic and splenic arteries)
- level(tier) N3 are distalmost nodes outside normal lymphatic pathway of stomach

Depending on extent of lymphadenectomy there are three types of curative gastric resection –

a) D1 limited lymphadenectomy – all N1 tier lymph nodes are removed en-block with stomach

b) D2 systemic lymphadenectomy – all level N1 & N2 nodes removed with stomach, if any N2 tier node not removed, then represented as D1/D2 resection.

c) D3 extended lymphadenectomy – more radical en-block resection including third tier nodes.

The standard D2 gastectomy removes the peritoneal layer over pancreas and anterior mesocolon, along with nodes along hepatic and splenic arteries and crural nodes, in addition to the tissue removed in D1 resection.

DI VS D2 GASTRECTOMY:

The ideal lymphadenectomy for gastric cancer (GC) should accurately stage the extent of disease and predict prognosis; in addition it should have the potential to improve survival by selectively and completely removing all metastatic lymph nodes (LN), with minimal morbidity and mortality.
Unfortunately the optimal extent of lymph node dissection (LND) for GC has not been determined and is certainly not standardized.At the time of resection, the macroscopic assessment of nodal metatstases is notoriously unreliable, and the intraoperative assessment of microscopic LN metastases is costly and impractical. Furthermore the pattern of lymph node involvement depends on numerous factors, including location of the primary tumor and depth of tumor invasion.

Opinion over the optimum resection for patients with gastric cancer remains divided, and the literature polarised. The impressive outcomes after D2 gastrectomy published in large retrospective series from Japan (Soga *et al*, 1979; Maruyama *et al*, 1987) have not been reproduced in randomised comparative studies from Europe (Bonenkamp *et al*, 19951999; Cuschieri *et al*, 19961999).

- The two largest randomised studies both report significantly greater operative morbidity and mortality associated with an extended D2 lymphadenectomy when compared with the less aggressive D1 lymphadenectomy, and have failed to demonstrate any survival advantage for a D2 resection.
- Many of the serious complications associated with D2 resections were associated with resections of the pancreas and spleen (Bonenkamp*et al*, 1995; Cuschieri *et al*, 1996), and the best long-term survival was observed in patients undergoing D2 gastrectomy without pancreatico-splenectomy (Cuschieri *et al*, 1999). Although this latter report concluded than a classical D2 resection offered no survival advantage over a D1 resection, the possibility that a modified D2 resection, preserving pancreas and spleen, might be better than a D1 resection was not dismissed (Cuschieri *et al*, 1999).
- Poor outcomes after surgery for gastric cancer in Britain are due to the greater age, comorbidity, advanced stages of disease and greater body mass indices of Western patients when compared with their Japanese counterparts.
- Most oesophagogastric cancer surgery in Britain and much of the West, has by tradition, been performed by general surgeons. Radical lymphadenectomy is a painstaking, technically demanding procedure, which has usually remained within the province of small numbers of specialist upper gastrointestinal surgeons.
- D1 perigastric lymphadenectomy remains the most commonly performed operation for gastric cancer in the West.
- Both MRC ST01 and the Dutch trial have received criticism over the relative inexperience of many different surgeons performing D2 lymphadenectomy. Furthermore, the existence of a relationship between caseload and operative

mortality remains controversial. The specialist surgical unit in Leeds has described a long learning curve during the adoption of D2 gastrectomy, with 10 years elapsing before operative mortality fell to 5%.

- D2 lymphadenectomy can be performed with low morbidity-mortality, and a 5-year survival of more than 50%. The procedure offers benefit in terms of survival for a certain percentage of patients with positive level N2 lymph nodes.
- The rationale for extended lymphadenectomy has been enhanced local control. The clearance of the possible metastatic nodes in the region outside of the perigastric nodes is presumed to impact on overall patient survival rates. The proponents of systemic LND point to numerous anatomical/ lymphographic studies, histopathologic analyses, statistical analyses of LN metastases, clinical prognostic observations, and advances in surgical techniques to support their argument that ELND is the most effective procedure to treat the lymphatic spread to GC.
- Japanese surgeons have been actively performing extended lymphadenectomy (D2, removal of perigastric nodes and nodes along the left gastric, common hepatic, celiac and splenic arteries; or D3, D2 plus removal of nodes in the hepatoduodenal ligament, in the retropancreatic space and along the vessels of the transverse mesocolon). In recent years interest has expanded to superextended lymphadenectomy (D4) of nodes around abdominal aorta (para-aortic lymph nodes from aortic hiatus to aortic bifurcation). Because the therapeutic value of this D4 procedure remains controversial, Maeta et al. initiated a prospective study to compare D3 and D4 lymphadenectomy. Surgical treatment of microscopic disease in grossly normal para-aortic lymph nodes may generate occasional long-term survivors. Selecting appropriate candidates who might benefit from D4 resections needs to be refined.
- In the recently published Dutch trial, the cumulative risk of relapse was lower in patients with spleen preservation than in those with splenectomy, but the aim of the study was to compare D1 and D2 resections.
- The description D dissection for the determination of extent of lymph node dissection arises from the Japanese classification (JRSGC). D1 to D4 dissection correspondences to the anatomical site of dissection of levels N1 to N4. The rationale for extended lymph node dissection is that it achieves a R0 resection due to clearance of the metastatic extraperigastric lymph nodes that can not be removed with a limited D1 node dissection. Thus, it increases the curative resection rate, reduces the locoregional recurrence rate and may improve survival. This hypothesis for improvement of both local control and survival after D2 dissection is supported by a large number of Japanese observational studies that based on historical comparisons. Furthermore, several prospective but non-randomised studies and other observational Western series have shown encouraging long-term results with D2 dissection. However, many surgeons in the West argue against the therapeutic value of D2 dissection and are clearly against the routine use of D2 dissection for Western patients. In their opinion, D2 dissection increases postoperative

morbidity and mortality rates and does not improve long-term survival. This argument has been based on retrospective studies, which have failed to demonstrate any survival benefit in favour of D2 dissection.

- In the Dutch trial, D2 dissection did not improve longterm survival or decrease the risk of relapse. However, the D1 and D2 groups were not well balanced. Resection of the spleen was an independent risk factor for reduced survival but splenectomy and pancreatectomy was significantly more often performed in the D2 than the D1 group.

2. Mention TNM classification of Urinary Bladder Tumour. Discuss management of muscle invasive urinary bladder tumour. 5+15

Answer.

TNM staging of bladder carcinoma

Tx Primary tumour cannot be assessed
T0 No evidence of primary tumour
Ta Non-invasive papillary carcinoma
Tis Carcinoma in situ
T1 Tumour invades subepithelial connective tissue
T2 Tumour invades muscularis propria (detrusor): T2a inner half; T2b outer half
T3 Tumour invades beyond muscularis propria into perivesical fat: T3a = microscopic; T3b = macroscopic
T4a Tumour invades any of: prostate, uterus, vagina, bowel
T4b Tumour invades pelvic or abdominal wall
Nx Regional (iliac and para-aortic) lymph nodes cannot be assessed
N0 No regional lymph node metastasis
N1 Metastasis in a single lymph node <2cm in greatest dimension
N2 Metastasis in a single lymph node 2 - 5cm or multiple nodes <5cm
N3 Metastasis in a single lymph node or multiple nodes >5cm in greatest dimension
Mx Distant metastasis cannot be assessed
M0 No distant metastasis
M1 Distant metastasis present

Diagnosis:

- After a urinary tract infection has been excluded or treated, all patients with microscopic or macroscopic haematuria require investigation of their upper tracts, bladder, and urethra.
- Renal **ultrasound** and **flexible cystoscopy**, performed under local anaesthetic, are first-line investigations.
- If these fail to find a cause, an **IVU** or **CTU** and **urine cytology** are justified second-line investigations.
- Patients with predominantly filling-type LUTS, suprapubic pain, or recurrent UTI/pneumaturia should also be treated with **urine cytology** and **cystoscopy**.
- **CTU** before and after IV contrast is becoming **the first-line radiological investigation of haematuria**.
    - It is faster and more sensitive than ultrasound or IVU in the detection of renal (parenchymal and urothelial) and ureteric tumours.

    - CTU also detects some bladder tumours, but may overcall bladder wall hypertrophy as tumour and will miss flat CIS and urethral pathology.
    - Thus it cannot replace cystoscopy.

If all investigations are normal, consideration should be given to nephrological disorders that may cause haematuria, such as glomerulonephritis. **Cross-referral to a renal physician is advised in patients with persisting microscopic haematuria, <u>especially those with associated proteinuria or hypertension</u>**.

- **Transurethral resection of bladder tumour (TURBT)** usually provides definitive histological diagnosis .
    - This is usually undertaken under general or spinal anaesthesia;
    - Bimanual examination is mandatory before and after bladder tumour resection, to assess size, position, and mobility.
    - The pathologist should report on the tumour type, grade, and stage; in particular, the presence or absence of muscularis propria should be noted, since its absence will preclude reliable T staging.
    - Red patches are biopsied separately; the prostatic urethra is biopsied if radical reconstructive surgery is under consideration.
    - Care is taken in resecting tumours at the dome, since intraperitoneal bladder perforation may occur, especially in women with thin-walled bladders.

Staging investigations are usually reserved for patients with biopsy-proven muscle-invasive bladder cancer unless clinically indicated, since superficial TCC and CIS disease are rarely associated with metastases.

- Pelvic CT or MRI may demonstrate extra-vesical tumour extension or iliac lymphadenopathy, reported if **>8mm** in maximal diameter.
- Chest X-ray
- Isotope bone scan (positive in 5-15% of patients with muscle-invasive TCC) is obtained in cases being considered for radical treatment.
- Staging lymphadenectomy (open or laparoscopic) may be indicated in the presence of CT-detected pelvic lymphadenopathy if radical treatment is under consideration.

<u>Muscle-invasive bladder cancer: surgical management of localized (pT2/3a) disease:</u>

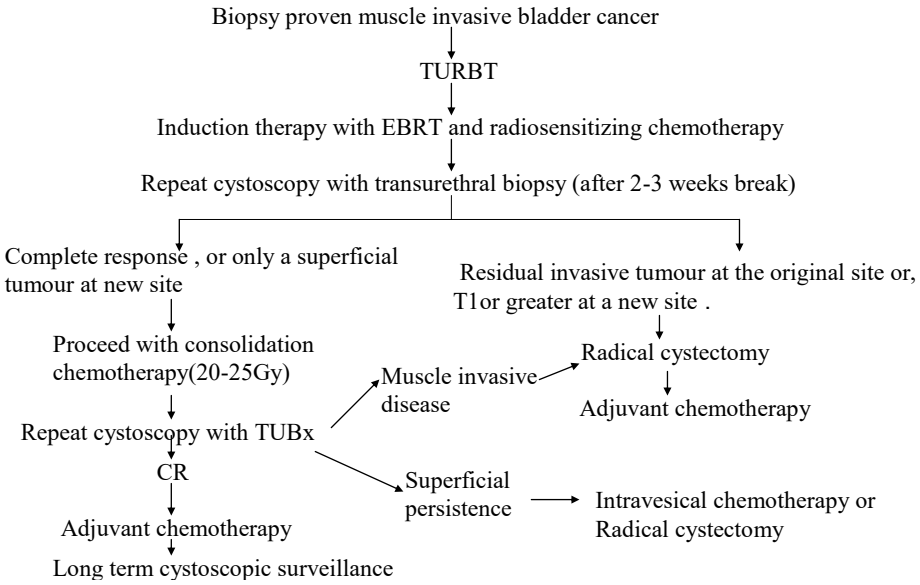
This is a dangerous disease; untreated 5-year survival is 3%. In the absence of prospective randomized trials comparing the surgical and non-surgical treatments, the options for a patient with newly diagnosed confined muscle-invasive bladder cancer are:

Bladder preserving

- Radical transurethral resection of bladder tumour (TURBT) plus systemic chemotherapy: little data, not mainstream
- Palliative TURBT+ palliative radiotherapy (RT): for elderly/unfit patients
- Partial cystectomy ± neoadjuvant systemic chemotherapy

- TURBT plus definitive RT : poor options for SCC and adenocarcinoma as they are seldom radiosensitive

Treatment algorithm for muscle invasive bladder cancer showing bladder sparing approach.



Radical cystectomy with

- Ileal conduit urinary diversion
- Ureterosigmoidostomy urinary diversion
- Continent urinary diversion
- Neoadjuvant chemotherapy: some evidence of benefit
- Neoadjuvant RT: some evidence of benefit.

Post-operative care

- Many patients will spend the first 24h in the high-dependency unit or ITU.
- Daily clinical evaluation, including inspection of the wound (and stoma if present), plus monitoring of blood count and creatinine/electrolytes, is mandatory.
- Broad-spectrum antimicrobial prophylaxis and thromboembolic prophylaxis with TED stockings, pneumatic calf compression, and subcutaneous heparin are standard.
- Mobilization after 24h is ideal.
- Chest physiotherapy and adequate analgesia is especially important in smokers and patients with chest comorbidity.
- Oral intake is restricted until bowel sounds are present; some patients may require parenteral nutrition in the presence of gastrointestinal complications.
- Drains are usually sited in the pelvis and near the uretero-diversion anastomosis, plus ureteric catheters passing from the renal pelves through the

diversion and exiting percutaneously, plus a catheter draining the diversion (except in the case of ileal conduit) exiting urethrally or suprapubically.
- Most patients stay in hospital 10 - 14 days.

Salvage radical cystectomy is technically a more difficult and slightly more morbid procedure. Relatively few patients who have failed primary RT are suitable for this second chance of a cure; fit patients with clinically localized disease.

3. Write short notes on: 6 x 5

(a) Brain death.
(b) Tissue Expansion.
(c) Percutaneous endoscopic gastrostomy.
(d) Surgery for maldescended testis.
(e) Stoppa repair of abdominal hernia.

(a) Brain death.

Answer. The concept of brain death emerged in the 1960s, as the ability to resuscitate individuals and mechanically keep the heart and lungs functioning became prevalent.

Brainstem death is defined by irreversible loss of consciousness,loss of brainstem reflexes and apnoea and these patients are therefore mechanically ventilated in the intensive care setting .

Diagnosis of brainstem death in 3 stages:

- Identification of the cause of irreversible coma.
- Exclusion of irreversible causes of coma.
- Clinical demonstration of absence of brainstem reflexes.

Medical criteria:

A brain-dead individual has no clinical evidence of brain function upon physical examination.

This includes no response to pain and no cranial nerve reflexes.

Reflexes include

- Pupillary reaction to lighi (fixed pupils),
- Vestibulo – occular reflex,
- Corneal reflex,
- Gag reflex,
- Motor response to central pain,
- Apnoea test: apnoea despite a carbon dioxide increase to> 6.65 kPa.

All reflexes must be absent and are tested for twice by two doctors .

It is important to distinguish between brain death and states that may mimic brain death (e.g., barbiturate overdose, alcohol intoxication, sedative overdose, hypothermia, hypoglycemia,coma or chronic vegetative states). Some comatose patients can recover, and some patients with severe irreversible neurological dysfunction will nonetheless retain some lower brain functions such as spontaneous respiration, despite the losses of both cortex and brainstem functionality. Thus, anencephaly, in which there is no higher brain present, is generally not considered brain death, though it is certainly an irreversible condition in which it may be appropriate to withdraw life support.

have been declared dead, and would not have met UK test conditions in any case. This is clearly a case of negligent misdiagnosis.

Organ donation

Brain death may result in legal death, but still with the heart beating, and with mechanical ventilation all other vital organs may be kept completely alive and functional, providing optimal opportunities for organ transplantation.

Most organ donation for organ transplantation is done in the setting of brain death. In some nations (for instance, Belgium, Poland, Portugal and France) everyone is automatically an organ donor, although some jurisdictions (such as Singapore, France, or New Zealand) allow opting out of the system. Elsewhere, consent from family members or next-of-kin is required for organ donation. The non-living donor is kept on ventilator support until the organs have been surgically removed. If a brain-dead individual is not an organ donor, ventilator and drug support is discontinued and cardiac death is allowed to occur.

(b) Tissue Expansion:

Answer. Tissue Expansion: Tissue Expansion is a reconstructive technique that uses an inflatable silicone balloon to serially expand surrounding skin. This expansion adjacent to the wound provides donor tissue of similar color, texture, thickness, and sensation, with minimal scar formation and donor-site morbidity. The technique takes advantage of the skin's ability to accommodate a slowly enlarging mass beneath it by increasing its surface area. The idea is to create and develop donor tissue, harvest it, and leave the original donor site preserved.

A. The advantages

The advantages include lower donor-site morbidity and the provision of donor tissue of similar quality to the recipient tissue. Tissue expansion is a simple and versatile technique that provides robust tissue.

B. The disadvantages

The disadvantages are that it is a staged technique, there is a visible deformity during the period of expansion, it requires frequent visits for expansion, and there is a relatively high rate of complications, including infection and extrusion.

C. Technique

- Preoperative planning involves assessing the defect size, locating matching tissue to be expanded, and deciding where final scars will be.
- Expander placement is usually performed through an incision at the junction of the lesion and the area of proposed expansion. The length of the incision is controversial. Some authors propose one third the length of the expander (it should be big enough to ensure full pocket creation). The filling port can be incorporated into the expander or placed in a separate pocket. In addition, the port may be externalized to minimize anxiety and pain during filling, especially in the pediatric population. Partially filling the expander on initial placement may reduce the duration of the expansion phase and reduce mechanical implant failure due to folding.
- The expansion phase begins 2 to 3 weeks after expander placement. The expander is inflated weekly with saline, using sterile technique. The amount infused with each fill depends on patient comfort, skin tension, and blanching of overlying skin. A rough guide is 10% of expander volume per injection. The duration of the expansion phase can vary from 6 weeks to 3 months. Waiting 2 to 3 weeks after the desired volume is achieved allows the expanded skin to soften, decreasing the contraction at the time of flap transposition.
- Removal of expander is straightforward. However, infection, exposure, or rupture may necessitate early explant.
- The expanded tissue is usually in the form of a random flap (rotation, advancement, or transposition). If more than one flap is necessary using expanded tissue, one must ensure that all flaps have adequate blood supply.

D. The origin of the new tissue

The origin of the new tissue is not completely understood. One potential source is new tissue created in response to the expansion process. Alternatively, tissue may derive from recruitment of adjacent tissues by stretching or creep and by stress relaxation. These possibilities are not mutually exclusive. Studies have shown that expansion gives rise to an increase in the thickness of the epidermis, a decrease in the thickness of the dermis, and atrophy of the underlying muscle and fat .

E. Tissue expanders are indicated

Tissue expanders are indicated for patients who cannot, or choose not to, tolerate the longer operative procedures or rehabilitation associated with more distant flaps. In areas where little suitable tissue is available (e.g., scalp), tissue expansion can be the aesthetically superior option. The patient must be motivated and understand the process. Common indications include burn alopecia, congenital nevi, male pattern baldness, and postmastectomy breast reconstruction.

F. Relative contraindications

Relative contraindications include malignancy or an open wound, active infection, and unwillingness to comply with multiple procedures. Similarly, tissue expanders cannot be placed under burned tissue, scar, skin graft, or a prior incision. In addition, tissue expanders are less effective in areas that will be irradiated because the skin in those areas thickens, scars, and contracts, minimizing the degree of expansion possible.

G. Complications

Complications include pain, seroma, hematoma (rates widely variable), infection (1% to 5%), exposure or extrusion (5% to 10%), and skin necrosis. Less common complications include striae, resorption of underlying bone, and neurapraxia.

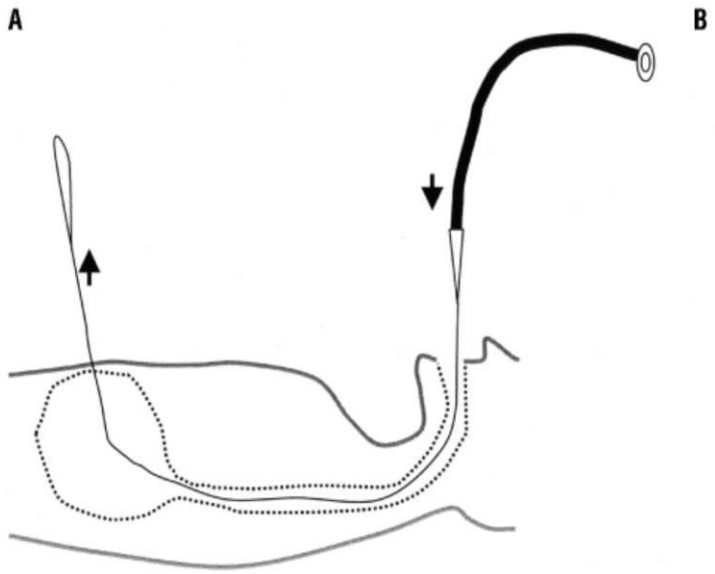
(c) Percutaneous endoscopic gastrostomy:

Answer. PEG:

- Percutaneous endoscopic gastrostomy (PEG) is now the preferred method for long-term feeding in patients who are unable to swallow or who require supplemental nutrition or chronic gastric decompression.
- PEG may be preferable to surgical gastrostomy since it is safe, less expensive, and less invasive.
- PEG and percutaneous endoscopic jejunostomy are contraindicated only in patients with total esophageal obstruction, massive ascites, or intra-abdominal sepsis.

Prior to the procedure, a single dose of prophylactic cephalosporin (or equivalent) should be given intravenously. The patient is placed in the supine or semi-Fowler position, after which the abdomen is prepared and draped using sterile technique. The endoscope is then passed into the stomach, which is distended with air insufflation. The assistant then presses on the abdomen with a finger and the impact against the anterior gastric wall should be noted. Ideally, this point should be 2–3 cm below the left costal margin. It is critical that the assistant's finger be clearly observed to indent the stomach. In patients with thin abdominal walls, light transillumination from within the stomach to the skin surface may aid in identifying a safe landmark.

A polypectomy snare is passed through the endoscope channel. The selected site on the abdominal wall is then infiltrated with local anesthesia. If desired, the fine needle used to anesthetize the skin can be inserted into the abdomen at the intended gastrostomy site; observation of the needle's clean entry into the stomach suggests the position is adequate. After making a small incision (approximately 5 mm) in the skin, the assistant then inserts a 14-gauge intravenous cannula through the incision; the intravenous cannula must be seen to enter the stomach. The snare is then tightened around the cannula and the inner stylet is removed. In the "pull technique," a long looped suture is placed through the cannula, after which the snare is released. The suture is then firmly grasped with the polypectomy snare. The endoscope and the tightened snare are removed together, bringing the suture out of the patient's mouth. The suture is secured to a well-lubricated 20F or 24F gastrostomy tube at its tapered external end. The assistant then pulls on the suture until the attached tube exits the abdominal wall . The endoscope is then reinserted and used to view the tube's inner bolster as the stomach is loosely seated against the abdominal wall and the tube is properly positioned. The endoscope is then removed and the tube is secured in place on the abdominal wall, usually with an external Silastic cross-bar or disc.



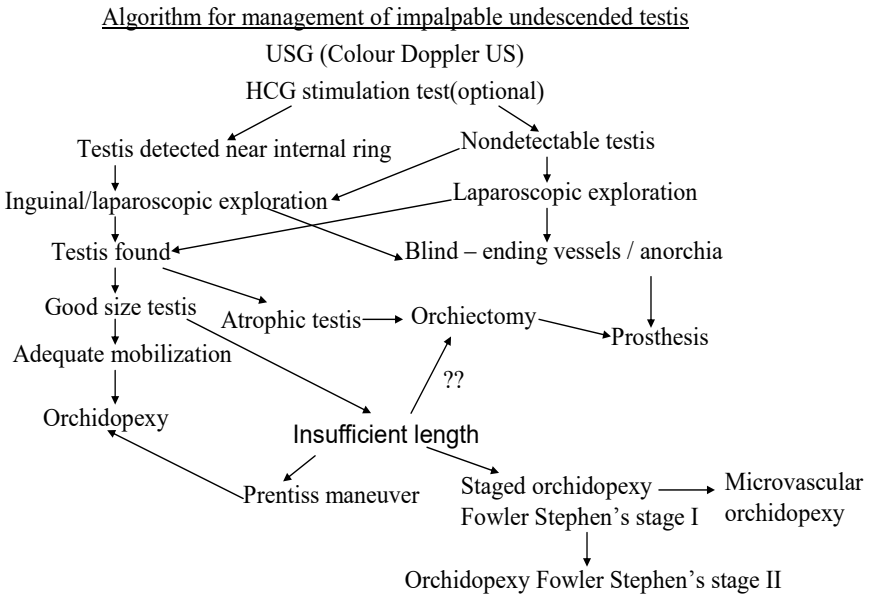
A. After the looped wire is retrieved out of the esophagus and mouth, it is secured to the Silastic (Ponsky) gastrostomy down the esophagus and out of the abdominal wall by the loop wire. B. The tube's bumper is pulled flush against avoid a buttonhole necrosis.

Two other techniques can be used for PEG placement. In the "push technique," in lieu of a suture a guidewire is inserted through the cannula and pulled out the patient's mouth. The gastrostomy tube is then pushed over the wire until it exits the abdominal wall. Finally, in the "introducer technique," the stomach is inflated, the site of insertion selected, and the intravenous cannula introduced into the stomach as described above. A J-tipped guidewire is then passed through the cannula into the stomach after which the cannula is removed. An introducer with a peel-away sheath is then passed over this wire, allowing removal of the wire and introducer. A Foley catheter or other similar gastrostomy tube is then placed through the sheath, its balloon inflated, and the sheath is removed. The catheter is then secured to the abdominal wall.

The PEG procedure can be extended to include jejunostomy (PEG-J) in patients who fail to tolerate gastric feedings due to severe gastroesophageal reflux or gastroparesis. While enteral feeding by PEG-J is intuitively believed to decrease the incidence of aspiration when compared with PEG feedings, most series report aspiration pneumonia to occur in only 0–5% of patients with PEG feedings—even for the neurologically impaired patient.

(d) Surgery for maldescended testis .

Answer.



Methods of orchidopexy;

- Extra-dartos pouch – conventional orchidopexy : high ligation of the sac is done through inguinal approach . Testis is placed in an extra – dartos pouch in the scrotum .
- Prentiss maneuver – if the testis does not reach the scrotum easily then the inferior epigastric artery and vein can be ligated and the testis brought directly through the transversalis fascial floor .
- Transcrotal orchidopexy – it is high ligation of processus vaginalis , dissection of spermatic cord and placement of testis in an ipsilateral subdartos pouch.
- Fowler Stephen's technique – division of the main testicular vessels , thus relies on delicate vessel and cremasteric collaterals for testicular survival and growth.
- Staged Fowler Stephen's technique – it is a 2 staged procedure. In stage 1 ligation of the spermatic vessels to gain length is done to allow the collateral blood supply to develop without mobilizing the testis . After 6 months at 2nd stage the testis is brought into scrotum by inguinal exploration. The testicular blood supply is supported by the artery to the vas.
- Multistage orchiopexy – The testis is mobilized and brought into the inguinal canal as far as possible. The testis and spermatic cord are wrapped with a silicone sheath to prevent adhesions. After 1 year waiting , at 2nd stage , the testis is brought down to the scrotum.
- Microvascular orchiopexy (testicular autotransplantation) – best procedure to avoid testicular atrophy . This involves high mobilization of the testicular vascular pedicle , transfer of testis to the scrotum and immediate revascularization of one arterial and one or two venous anastomoses to the inferior epigastric vessels.

- Refluo technique – consists of full venous drainage by microvascular anastomosis of the testicular vein to the inferior epigastric vein , but relies on the arterial input from the vassal collaterals.
- Qmbredaanne's procedure – testis is placed into the contralateral scrotal sac through the scrotal septum.

(e) Stoppa repair of abdominal hernia.

Answer. The Stoppa repair is a tension free type of hernia repair. It is performed by wrapping the lower part of the parietal peritoneum with prosthetic mesh and placing it at a preperitoneal level over Fruchauds myopectineal orifice. It was first described in 1975 by Rene Stoppa. This operation is also known as giant prosthetic reinforcement of the visceral sac (GPRVS).

This technique has met particular success in the repair of bilateral hernias, large scrotal hernias, and recurrent or rerecurrent hernias in which conventional repair is difficult and carries a high morbidity and failure rate. The most recent reported recurrence rate (involving 230 patients with 420 hernias and a maximum of 8 years follow-up) was 0.71%.

The Laparoscopic transabdominal preperitoneal repair (TAPP) uses exactly the same principles as the Stoppa repair, however it is performed laparoscopically.

Retrorectus mesh repair: Rives-Stoppa technique. This technique utilizes the hernia sac to separate the mesh from the intra-abdominal contents. Superior to the umbilicus, dissection is performed above the posterior rectus fascia and under the rectus muscle. Below the umbilicus, dissection occurs in the preperitoneal space due to the lack of a posterior rectus sheath. A large piece of mesh is placed in the newly formed space, and fixated to the muscle layer above. This repair has decreased recurrences and complications from previous techniques.

In theory, recurrences after Stoppas technique are not possible but they occur in a minority of cases most probably due to technical errors. No other hernioplasty produces better results for recurrent and especially re-recurrent hernias.

Rives-Stoppa repair augmented by laparoscopic components separation is an innovative method for reconstruction of complex abdominal wall defects. Laparoscopic components separation allows fascial closure to be achieved anterior to the mesh in large incisional hernias, which may reduce wound infection rates.

4. Write brief answers: 7.5 x 4

(a) Immediate complications of corrosive acid poisoning.
(b) Principles of surgery for midpenile hypospadius.
(c) Jaw tumours.
(d) Congenital diaphragmatic hernia.

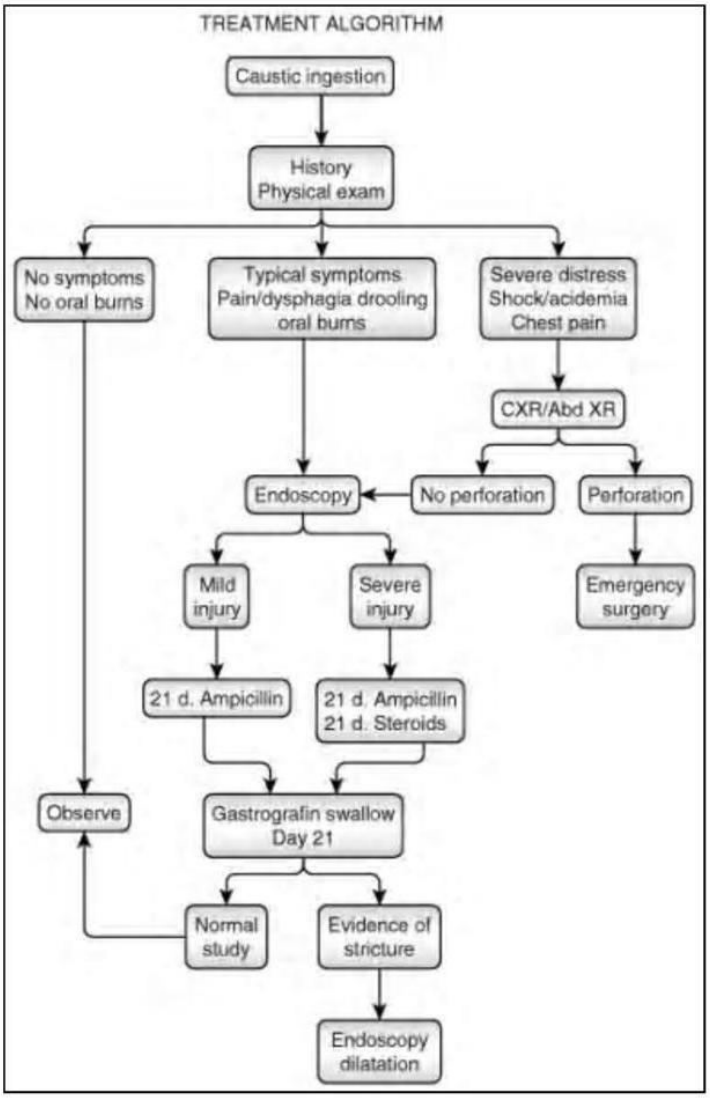
(a) Immediate complications of corrosive acid poisoning.

Answer. **Common Corrosive Agents**

Acids (pH < 7): Corrosives
Hydrochloride acid (pool and metal cleaners)
Hydrofluoric acid (antirust products)
Sulfuric acid (car battery fluid)

Classification of Injury

Mild Injury	
1st degree	Mucosal
	Erythema/edema
2nd degree	Transmucosal
	Sloughing of mucosa/ulcerations
Severe Injury	
2nd degree	Transmucosal
	Circumferential
3rd degree	Transmural periesophageal penetration
	Dark red black tissue exposed



(b) Principles of surgery for midpenile hypospadius.

Answer. Hypospadias can be classified according to the anatomical location of the urethral meatus

- Anterior (or distal): glandular, coronal, and subcoronal (~50%)
- Middle :distal penile, midshaft, and proximal penile (~30%)
- Posterior (or proximal): penoscrotal, scrotal, and perineal (~20%)

Diagnosis

A full clinical examination will make the diagnosis. However, it is also important to seek out associated abnormalities which will need treatment (undescended testes, inguinal hernias, and hydroceles). Patients with absent testes and severe hypospadias should undergo chromosomal and endocrine investigation to exclude intersex conditions.

Treatment

Surgery is indicated where deformity is severe, interferes with voiding, or is predicted to interfere with sexual function. Surgery is now performed between 6 - 12 months of

age. Local application of testosterone for 1 month pre-operatively can help increase tissue size.
Surgery aims to correct penile curvature (orthoplasty), reconstruct a new urethra, and bring the new meatus to the tip of the glans using urethroplasy, glanuloplasty, and meatoplasty techniques.
Severe cases may require staged procedures.
Common operations for anterior hypospadias include meatal advancement and glanuloplasty (MAGPI), meatal-based flaps (Mathieu procedure), and tubularization of the urethral plate. Posterior defects require free grafts (buccal mucosa), onlay grafts, and preputial transfer flaps.
The onlay island flap is a variation of the transverse preputial island flap for hypospadias repair. It is useful in patients without fibrous chordee whose meatus is mid penile or subcoronal. The cosmetic results with the onlay island flap were quite satisfactory and the complication rate was 6 per cent, which was identical to that observed with the Mathieu repair. The onlay island flap is applicable particularly in patients with mid shaft hypospadias without chordee, if the meatus is too proximal for a Mathieu repair and in patients with distal penile hypospadias with deficient ventral skin.

Tabularized incised plate urethroplasty was found to be a successful method for treating distal hypospadias and encouraging results were obtained in mid-penile hypospadias cases.

The Mathieu Righini technique for mid-distal hypospadias repair: This technique employs a meatal flap in which the prepuce is preserved and reconstructed. This technique provides very good cosmetic and functional results: the penis is normal in appearance as well as function. In the author's opinion, the Mathieu-Righini technique is the procedure of choice for mid-distal hypospadias without chordee repair.

(c) Jaw tumours.

Answer. <u>Malignant Jaw Tumours:</u>
Malignancies are usually encountered in the upper jaw. Those in the mandible are rare. The upper jaw malignant tumours are:

1. Columnar cell cancer – Carcinoma Maxillary Antrum
2. Osteogenic sarcoma
3. Squamous cell carcinoma – from epithelium overlying hard palate, tooth socket, gums.
4. Invasion of Maxilla by sarcoma of Ethmoid
5. Lymphoma – Burkitt Tumour

Osteogenic Sarcoma: Seen in women over 40 usually, men are infrequently involved. It is usually of periosteal variety and the anterior part of the bone is usually involved. Later the palatal and nasal walls get affected. Pain, nasal obstruction and epiphora usually occur late in the disease. Histologically it is of round cell variety, but rarely it is highly differentiated fibro-myxochondroma. Treatment is radiotherapy and outcome is fatal.
Carcinoma Maxillary Antrum: This is the commonest jaw malignancy and is seen beyond the age of 40, almost equal in both the sexes. Most of the patients have associated secondary infection of nasal sinuses.

Clinical features – A line diagonal drawn from the inner canthus of the eye to the angle of the mandible divides the maxilla into two parts a lower antro-alveolar and an upper antro-ethmoidal. Tumours in the antro-alveolar segment present with –

- Bulge in lateral nasal wall
- Bulge on the palate
- Pain in teeth, not relieved by regular dental check up
- Foul purulent blood stained discharge from nose
- Free bleeding associated with antral puncture

Tumours originating in the antro-ethmoidal segment present with

- Bulge on the anterior face
- Frog face
- Proptosis and diplopia
- Epiphora
- Intra-cranial extension medially and Infra-temporal extension laterally –
- Trismus - involvement of pterygoid muscles
- Palatal anaesthesia – involvement of greater palatine nerve

Metastasis occurs late in the disease and the jugulo-digastric LN is first to be involved. In 1/3 of the cases, metastasis is seen as the first presenting symptom. Lymph nodes are only involved when the antrum is breached and lymphatic-rich hard palate and cheek get involved.

Biopsy: This is best done by a diagnostic Caldwell-Luc operation. Antral lavage for exfoliative cytology is a poor second choice. Under General Anaesthesia with oral intubation and packing an intra-oral incision 2.5cm long is made in the bucco-labial sulcus centered over the canine fossa. The muco-periosteum is elevated and a 1 to 1.5 cm window is made in the anterior wall of the antrum. Biopsies are taken through this port.

Treatment: A judicious combination of Radiotherapy and Surgery is employed

Step 1. A course of high voltage radiation 6000Rads in 6 weeks or Gamma Ray from teleradium unit.

Step 2. Once the inflammation of Radiotherapy subsides in 6 – 8 weeks surgery is performed. The purpose is to remove any residual growth and take geographical biopsies to exclude any histological evidence of the disease. This is done either by palatal fenestration or by nibbling away the lateral nasal wall with inferior and middle turbinates, a procedure called as Lateral Rhinostomy.

Step 3. This depends on the presence or absence of residual disease in the antrum. In 30% cases there is no residual disease and a functioning dental obturator is provided as well as regular follow up is kept. In the remaining cases in the maxillary obturator is filled with wax in which radium tubes are put for brachy-therapy. In ethmoidal and sphenoidal extensions radium needles are inserted directly into these regions.

Recurrences are treated by total maxillectomy by Waber Ferguson incision or Radical maxillectomy, which includes the eye and cribriform plate by adding a Diffenbach extension and / or bicoronal incision with frontal craniotomy. With the introduction of intratracheal anaesthesia these surgeries now have low mortality and surprisingly very little postoperative deformity. Presence of lymph nodes in the neck demands a radical neck dissection. Cytotoxic drugs are also used for recurrences with limited success.

Malignancies of the Mandible: This is rarely a site for primary or even secondary tumour, though it often gets directly involved in carcinomas originating from the tongue, floor of mouth or gingivo-labial / gingivo-buccal sulcus. Another method of neoplastic involvement is from the involved facial lymph node lying in juxtaposition

to the mandible near the Facial artery groove. The node once involved may invade the mandible. As radiation routinely causes osteo-radio necrosis of the mandible, the treatment remains hemi-mandibulectomy followed by mandibular reconstruction by free iliac bone graft or vascularized fibula.

Benign Jaw Tumours:

A. Fibro-osseous group
   - Fibrous dysplasia
     - Monostotic – Leontiasis ossea
     - Polyostotic
   - Cherubism
   - Paget's Disease of Jaw

B. Giant cell tumours
   - Giant cell reparative granuloma
   - Aneurysmal bone cyst
   - Osteoclastoma
   - Brown Tumour of Hyper-parathyroidism

Fibrous dysplasia of the bone is a lesion of unknown aetiology, uncertain pathology, diverse histology, which although is not strictly a neoplasm but behaves like one. It is a developmental derangement of bones caused by an aberrant activity of bone forming mesenchymal tissue resulting in abnormal proliferation of undifferentiated mesenchymal bone forming cells. The bony lesion exhibits general histological features of fibrosis with varying degree of simultaneous resorption and repair. The disease is either Monostotic or Polyostotic. The latter is further classified into:

1. Fibrous dysplasia involving variable number of bones although most of the skeleton is normal and accompanied by pigmented lesions or café-au-lait spots – Jaffe type
2. A more severe disease involving nearly all he bones of the skeleton, accompanied by pigmented skin lesions and endocrine disturbances of various types – Albright's syndrome

Monostotic fibrous dysplasia is a totally different disease altogether and will not march on to become polyostotic type. It also does not manifest extraskeletal lesions as seen in the polyostotic variety.

Treatment of Polyostotic fibrous dysplasia is aimed at achieving facial symmetry and not at total extirpation of tumour. Monostotic lesions in the mandible are excised en-block and the mandible is reconstructed by vascularized bone graft.

Paget's Disease of the Jaws: This may be a part of generalized Paget's disease or confined mainly to the jaws. A differential diagnosis of Polyostotic Fibrous dysplasia, but it occurs in older people, it is painful, and radiological appearance of teeth, if they are any, show apical club shaped swellings – cementosis. Treatment is aimed at achieving facial symmetry by bone contouring. Sarcomas develop more often in the jawbones affected by Paget's disease.

Giant-cell reparative granuloma: Seen more frequently in females than in males, and nearly always between the age of 10 and 25, there is usually a history of trivial trauma and a painless swelling in the jaw, usually the mandible. X ray reveals expansion of both tables with a uni-locular or multi-locular cystic appearance. Other giant cell tumours are always in the differential diagnosis along with ameloblastoma. The lesion is not locally malignant and so if the inner table is thick, we can treat it by curettage and bone chip filling. Should the condition recur, hyper-parathyroidism should be suspected, as even histologically it is very difficult to distinguish it from Brown Tumour of Hyper-parathyroidism.

(d) Congenital diaphragmatic hernia.

Answer. Congenital diaphragmatic hernia (CDH)

Aetiology:

- Incomplete diaphragm development at 8 weeks' gestation results in herniation of abdominal organs into the chest, preventing normal lung development.
- Left leaflet occurs in 90% of cases, and right leaflet in 10%.
- Bochdalek (posterolateral defect; 85% of cases); Morgagni (anterior, parasternal; fewer pulmonary and systemic complications).
- A 1:1 male-to-female ratio.

- Diagnosis
    - Antenatal ultrasound and maternal-fetal magnetic resonance imaging (MRI). Polyhydramnios is detected in up to 80% of cases.
    - Physical exam
        - Cardiorespiratory distress.
        - Asymmetric “funnel” chest.
        - Reduced breath sounds on the affected side.
        - Scaphoid abdomen.
    - Chest x-ray
        - Herniated abdominal viscera within the chest.
        - Mediastinal shift.
- Mortality
    - Approximately 35%.
    - May be higher in patients with severe pulmonary hypoplasia and hypertension or in the presence of associated congenital anomalies.
- Management
    - Immediate postnatal care
        - Supplemental oxygen.
        - Endotracheal intubation is indicated if significant respiratory distress is present. Avoid bag-mask ventilation, which exacerbates gastrointestinal (GI) distention and further impedes lung ventilation.
        - Intravenous access is necessary for the administration of fluids to maintain organ perfusion.
        - GI decompression by orogastric or nasogastric intubation reduces distention of the stomach and within the thoracic cavity.
    - Intensive care
        - Conventional ventilation utilizing permissive hypercapnia to minimize barotrauma.
            - Once stabilized, wean fraction of inspired oxygen (FIO_2) for preductal oxygen saturation greater than 90%.
            - Arterial carbon dioxide tension of 60 to 70 mm Hg is acceptable.
            - Maintain pH greater than 7.20.

        - Peak airway pressures less than 25 cm H_2O.
        - Mean airway pressures less than 12 cm H_2O.
    - High-frequency oscillating ventilator (HFOV)
        - May be used if conventional ventilation failed.
        - Possibly limit barotrauma.
    - Inhaled nitric oxide may decrease severity of pulmonary hypertension.
    - Extracorporeal membrane oxygenation (ECMO) is considered for patients with severe preductal hypoxemia or right-to-left shunting due to high pulmonary hypertension. Patients with overwhelming pulmonary hypoplasia may not be not candidates for ECMO.
- Operative intervention
    - Usually deferred until the patient's pulmonary status is stable.
    - A subcostal incision on the affected side allows the herniated abdominal contents to be replaced in the peritoneal cavity.
    - Repaired primarily or with a synthetic patch, depending on the size of the defect.
- Complications: long-term respiratory insufficiency, GI reflux, neurologic sequelae from prolonged hypoxia, recurrence of hernia.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES
MS (General Surgery) Examination, 2009

May 2009

PAPER III

Time Allowed: 3 Hours **Full Marks:100**

1. What is morbid obesity? Discuss the ill effects of this condition. What are the surgical options for this condition ? 6+7+7

Answer. Morbid obesity is defined as being either 100 pounds above ideal body weight, or twice ideal body weight, or having a BMI(Body mass index) > 40 in Western population and >37.5 in Asian population.

Ill effects of morbid obesity:

<table>
<tr><th>Medical</th><th>Psychological</th><th>Economic</th><th>Social</th></tr>
<tr><td rowspan="2">Coronary heart disease - Prevalence 6 times higher</td><td rowspan="2">Depression</td><td rowspan="3">Cost of futile weight loss treatment</td><td>Social isolation</td></tr>
<tr><td rowspan="2">Daily prejudice</td></tr>
<tr><td rowspan="3">Non insulin dependent diabetes mellitus – twice as high</td><td rowspan="4">Insomnia</td></tr>
<tr><td rowspan="2">Cost of treating various medical conditions</td></tr>
<tr><td>Dyslipidemias</td><td>Physical abuse</td></tr>
<tr><td>Hypertension</td><td rowspan="4">Inability to obtain insurance coverage or increased premium</td><td>Sexual abuse</td></tr>
<tr><td>Gastro oesophageal reflux disorder</td><td rowspan="3">Suicide</td><td>Limitations in performing daily activities</td></tr>
<tr><td rowspan="2">Gall stones</td><td>Difficulty with hygiene</td></tr>
<tr><td rowspan="2">Osteoarthritis</td><td rowspan="3">Neurotic disorders</td><td>Cost of special clothing</td><td>Clothing limitations</td></tr>
<tr><td rowspan="2">Sleep apnoea</td><td rowspan="3">Difficulty obtaining good job or promotion</td><td rowspan="2">Limited mobility</td></tr>
<tr><td rowspan="2">Malignancy</td><td rowspan="2">Self-hate and feeling of guilt</td></tr>
<tr><td>Limited access to seats, passageways, etc.</td></tr>
</table>

Indications for Bariatric Surgery: Patients must meet the following criteria for consideration for bariatric surgery:

1. BMI >40 kg/m^2 or BMI >35 kg/m^2 with an associated medical comorbidity worsened by obesity

2. Failed dietary therapy
3. Psychiatrically stable without alcohol dependence or illegal drug use
4. Knowledgeable about the operation and its sequelae
5. Motivated individual
6. Medical problems not precluding probable survival from surgery

Bariatric Operations: Mechanism of Action

Restrictive: Vertical banded gastroplasty (VBG) (historic purposes only)
Laparoscopic adjustable gastric banding (LAGB)

Largely Restrictive/Mildly Malabsorptive: Roux-en-Y gastric bypass (RYGB)

Largely Malabsorptive/Mildly Restrictive: Biliopancreatic diversion (BPD)
Duodenal switch (DS)

Vertical Banded Gastroplasty: This procedure has now largely been abandoned.

Causes are:

- Poor long-term weight loss,
- A high rate of late stenosis of the gastric outlet, and
- A tendency for patients to adopt a high-calorie liquid diet, thereby leading to regain of weight.
- Weight regain several years after surgery is typically due to a breakdown of the vertical staple line and patients report new dietary freedom. Revisional surgery and conversion to a gastric bypass usually result in durable weight loss but can be associated with a higher rate of perioperative complications.

Adjustable Gastric Banding

- The AGB procedure may be performed with any of three types of adjustable bands.
- They all work on the principle of restriction of oral intake by limiting the volume of the proximal part of the stomach.
- Their advantage over the traditional vertical banded gastroplasty is adjustability.
- Gastric banding is a least-invasive operation with 50-60% excess weight loss over 5 years but long term results are unknown and the complication rate may be cumulative.

Roux-en-Y Gastric Bypass

Essential Components of Roux-en-Y Gastric Bypass
Small proximal gastric pouch

Gastric pouch constructed from the cardia of the stomach to prevent dilation and minimize acid production
Gastric pouch divided from the distal part of the stomach
Roux limb at least 75 cm in length
Enteroenterostomy constructed to avoid stenosis or obstruction
Closure of all potential spaces for internal hernias

Results of Roux-en-Y Gastric Bypass

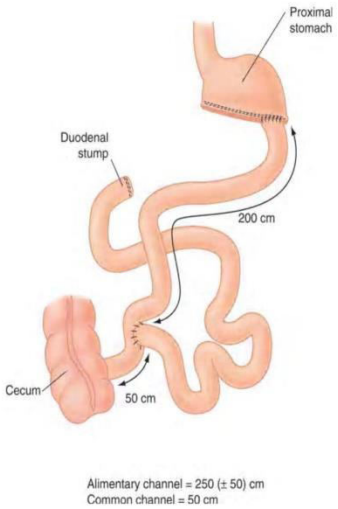
- RYGB has an established track record longer than that of any other operation. Its Recovery after RYGB is improved after a laparoscopic approach.
- This improvement is largely related to the decrease in postoperative pain experienced by patients after laparoscopic RYGB versus open RYGB.
- Weight loss with both approaches was comparable (68% loss of excess weight for laparoscopic RYGB versus 62% loss of excess weight for open RYGB).
- Another important advantage of the laparosCopic approach for RYGB is a decrease in the incidence of wound complications and incisional hernia seen after RYGB.
- There is no difference in the rate resolution of comorbid conditions or weight loss between the two procedures. after RYGB.
- Resolution of comorbid conditions after open and laparoscopic RYGB has generally been excellent.
- Rate of resolution and improvement of diabetes, hypertension and obstructive sllep apnoea is significant.
- Metabolic syndrome is cured or ameliorated with gastric bypass.
- Hyperlipidemia, hypercholesterolemia, and hypertriglyceridemia has been improved significantly in patients undergoing RYGB.
- RYGB has also been shown to resolve the symptoms of pseudotumor cerebri, as well as cure the difficult problem of venous stasis ulcers.
- Immediate resolution of symptoms of GERD occurs in more than 90% of cases.

Biliopancreatic Diversion

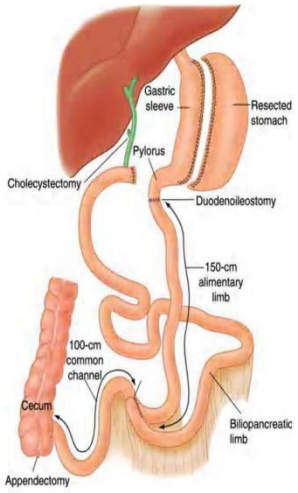
- BPD produces weight loss based primarily on malabsorption, but it does have a mild restrictive component.
- The intestinal tract is reconstructed to allow only a short so-called common channel of the distal 50-cm of terminal ileum for absorption of fat and protein.
- The alimentary tract beyond the proximal part of the stomach is rearranged to include only the distal 200 cm of ileum, including the common channel.
- The proximal end of this ileum is anastomosed to the proximal end of the stomach after performing a distal hemigastrectomy.
- The ileum proximal to the end that is anastomosed to the stomach is in turn anastomosed to the terminal ileum within the 50- to 100-cm distance from the ileocecal valve, depending on the surgeon' preference and the patient's size.

Duodenal Switch

- This modification was developed to help lessen the high incidence of marginal ulcers after BPD.
- The mechanism of weight loss is similar to that of BPD.
- An appendectomy is followed by measurement of the terminal ileum. Notably in the DS procedure, the common channel is 100 cm and the entire alimentary tract is 250 cm.
- However, the major difference between DS and BPD is the gastrectomy and the proximal anatomy. Instead of a distal hemigastrectomy, a sleeve gastrectomy of the greater curvature of the stomach is performed. This procedure is done as the initial part of the operation because if the patient exhibits any intraoperative instability, the operation can be discontinued after the sleeve gastrectomy alone.
- A two-stage DS has been used in patients who have an extremely high BMI and are high operative risks.
- The goal is to produce a lesser curvature gastric sleeve with a volume of 150 to 200 mL.
- After sleeve gastrectomy, or preceding it in smaller patients, the duodenum is divided with the stapler approximately 2 cm beyond the pylorus.
- The distal connections are performed as for BPD. The distal anastomosis is created at the 100-cm point proximal to the ileocecal valve.
- The proximal anastomosis is created between the proximal end of the 250 cm of terminal ileum and the first portion of the duodenum. The duodenoileostomy is an antecolic end-to-side duodenoenterostomy.



Scematic diagram of Biliopancreatic Diversion



Scematic diagram of Duodenal Switch

Results of Biliopancreatic Diversion/Duodenal Switch

- Excess weight loss (EWL)after BPD/DS is the highest of all the bariatric operations
- In a recent study comparing morbidly obese patients with a BMI greater than 50 kg/m^2, there was significantly more EWL at 12, 18, and 24 months postoperatively after DS than after RYGB.
- Thus, some surgeons argue that super-obese patients fare better and maintain weight loss better in the long term after undergoing DS than after other bariatric operations.
- BPD/DS has also been highly effective in treating comorbid conditions, including hypertension, diabetes, lipid disorders, and obstructive sleep apnea.
- Lipid disorders and type 2 diabetes are almost uniformly resolved after BPD/DS.
- Hypertension is cured in 83.4% and obstructive sleep apnea resolves in 91.9% of patients.
- After BPD, patients typically have between two and four bowel movements per day. Excessive flatulence and foul-smelling stools are the rule.
- Relatively selective malabsorption of starch and fat provides the major mechanism of weight loss, although the partial gastric resection does contribute a restrictive component to the operation.
- When protein malnutrition does occur, the common channel may need to be lengthened with a reoperation.
- Major considerations for achieving excellent results in patients offered BPD/DS include the ability to reliably monitor these patients, as well as confirm that they are being compliant with the recommendations to take appropriate vitamin supplements.
- Supplements include multivitamins, as well as at least 2 g of oral calcium per day. Supplemental fat-soluble vitamins, including D, K, and A, are indicated monthly as well.
- Because of a high incidence of morbidity and mortality in patients with a BMI greater than 60 kg/m^2 undergoing laparoscopic DS, surgeons developed the two-stage DS, with sleeve gastrectomy alone performed as the first stage to decrease morbidity in this super-obese patient population.

Comments:

- Cultural factors, surgeon and patient preference drive choice of operation which should be based on the balance of risk and benefit.
- Bariatric surgery pays for itself within 3-4 years after surgery.
- Bariatric surgery patients have better long-term survival than obese controls.
- A number of procedures have been investigated for weight loss surgery but have not been totally accepted by the surgical community. Several surgeons have proposed a two-stage procedure for a super-obese patient, who often has a large liver that precludes safe retraction for gastric bypass, because of the recognition

that the DS procedure has been associated with much higher mortality and morbidity rates in the super-obese (BMI >60 kg/m^2).

- Now, reports of vertical sleeve gastrectomy (VSG) alone suggest that weight loss is sufficient to preclude conversion to gastric bypass or DS.
- Gastric pacing has been performed in several trials but has not gained widespread acceptance. The concept is to stimulate gastric smooth muscle by implanting a pacemaker in the body of the stomach to induce early satiety, which reduces caloric intake and therefore results in weight loss.
- Increasingly, surgeons are observing effects of bariatric operations not just on the physical reduction of caloric intake or malabsorption. Alteration in comorbid conditions caused by metabolic processes may prove equally as important. For example, bariatric operations may have important metabolic components that alter the hormonal/cytokine/metabolic rate of patients.

2. Classify mediastinal tumours .How do you investigate such a tumour? Discuss the clinical features & management of such a patient. (3+3+7+7)

Answer. Classification of Primary Mediastinal Tumors and Cysts

Neurogenic Tumors
Neurofibroma
Neurilemoma
Paraganglioma
Ganglioneuroma
Neuroblastoma
Chemodectoma
Neurosarcoma
Thymoma
Benign
Malignant
Lymphoma
Hodgkin's disease
Lymphoblastic lymphoma
Large cell lymphoma
Germ Cell Tumors
Teratodermoid

Benign Malignant Seminoma Nonseminoma Embryonal Choriocarcinoma Endodermal
Primary Carcinomas
Mesenchymal Tumors Fibroma/fibrosarcoma Lipoma/liposarcoma Leiomyoma/leiomyosarcoma Rhabdosarcoma Xanthogranuloma Myxoma Mesothelioma Hemangioma Hemangioendothelioma Hemangiopericytoma Lymphangioma Lymphangiomyoma Lymphangiopericytoma
Endocrine Tumors Intrathoracic thyroid Parathyroid adenoma/carcinoma Carcinoid
Cysts Bronchogenic Pericardial Enteric

Thymic Thoracic duct Nonspecific
Giant Lymph Node Hyperplasia Castleman's disease
Chondroma
Extramedullary Hematopoiesis

Tumours of anterior mediastinum:

- Thymic Tumors: Thymomas represent the most common mediastinal neoplasm, as well as the most common anterior compartment neoplasm, constituting approximately 40% to 50% of the anterior compartment masses and 15% of all mediastinal masses.
- Lymphoma: Lymphomas constitute the second most common anterior compartment neoplasm in adults and the most common neoplasm in the pediatric population.
- Germ-Cell Tumor: Benign teratomas, seminomas, and nonseminomatous germ-cell tumors are a heterogeneous group of benign and malignant neoplasms thought to originate from primordial germ cells that fail to complete migration from the urogenital ridge during embryogenesis.
- Thyroid and Parathyroid tumours and cysts.

Tumours of middle mediastinum:

- Lymphomas.
- Cystic masses: Bronchogenic cysts, neuroenteric cysts, pericardial cysts.
- Mesenchymal tumours.

Tumours of posterior mediastinum:

- Neurogenic tumours: 1.Originating from peripheral nerves - Schwannoma (or neurilemoma), neurofibroma, and malignant nerve sheath tumours.
  2. Originating from sympathetic ganglia - ganglioneuroma, ganglioneuroblastoma, and neuroblastoma.
  3.Originating from the paraganglionic cell - Pheochromocytomas and chemodectomas.
- Cystic masses.
- Mesenchymal tumours.

Tumours of superior mediastinum:

- Lymphomas.
- Thyroid and Parathyroid tumours and cysts.

Investgations:

Anterior mediastinal tumours:

- CT scan of the chest - An anterior compartment mass in a patient with a history of myasthenia gravis can almost be assumed to represent a thymoma. On CT scan mediastinal goiters are well-encapsulated, lobulated, and heterogeneous tumors. Coarse or punctate calcifications are common and goiters often demonstrate intense and sustained contrast enhancement. The continuity between the cervical and mediastinal components of the thyroid gland on CT scan is usually diagnostic without the need for biopsy. Parathyroid tumors are usually located on the posterior capsule of the thyroid but may be in ectopic positions.
- Parathyroid hormone (PTH) levels can localize metabolically active parathyroid adenomas to the anterior mediastinal compartment.
- Tumour markers: For any young adult male presenting with a mass in the anterior compartment, simply obtaining serum tumor marker (STM) levels, which include β-human chorionic gonadotropin (β-hCG) and α-fetoprotein (AFP) is essential. A significant elevation of either STM is virtually diagnostic of a nonseminomatous germ-cell tumor primarily arising in the mediastinum (PMNSGCT).
- Following initial clinical and CT evaluation, biopsy of the anterior compartment mass may be indicated. The least invasive technique is CT-guided fine-needle aspiration (FNA). CT-guided core needle biopsy can safely be performed if the mass abuts a significant area of the anterior chest wall and therefore can be accessed without traversing lung parenchyma with a large-bore needle.
- Video-assisted thoracic surgery (VATS) does provide excellent exposure to the anterior compartment for biopsy purposes; however, the pleural space is traversed and selective lung ventilation with a double-lumen endobronchial tube is required. Moreover if thymoma is a diagnostic possibility, pleural space seeding can potentially occur during VATS biopsy efforts.

Middle mediastinal tumours:

- Chest X-ray and CT chest should be done first.
- Because a wide variety of conditions may cause lymphadenopathy in this compartment, a thorough history and physical examination, in addition to a complete chest and upper abdominal CT scan, are essential for the initial evaluation. Occasionally, elevated serum levels of angiotensin-converting enzyme (ACE) can be indicative of sarcoidosis. Elevated serum histoplasmosis or coccidioidomycosis antigen or antibody titers can be suggestive of the etiology of mediastinal lymphadenopathy but should not be totally relied on for a definitive diagnosis. Extensive paratracheal and bilateral hilar lymphadenopathy in an otherwise asymptomatic patient is often a result of sarcoidosis. In contrast, extensive paratracheal lymphadenopathy in a patient who presents with a new onset of a cough, weight loss, shortness of breath, and a smoking history would be suspicious for metastases from either small-cell or inoperable non-small-cell lung cancer.
- As CT-guided FNA biopsy cannot safely be performed on most masses in the middle mediastinum, other diagnostic procedures are required. Biopsy of upper and lower paratracheal masses is best performed with transcervical mediastinoscopy.

- If a diagnosis cannot be established by cytology, biopsy either by a VATS or a limited right thoracotomy approach would be recommended.

Posterior mediastinal tumours:

- Typically benign nerve sheath tumors present in adult patients with symptoms such as minor back discomfort or asymptomatic masses found on incidental chest radiograph or CT scan. Chest CT or MRI should be used to initially define the characteristics and location. A well-circumscribed, smooth-walled mass in the paravertebral sulcus is a typical finding for a benign nerve sheath tumor.
- CT scan or MRI will also assess the relationship between the mass and potentially adjacent structures such as the esophagus, aorta, and spinal cord. If the CT scan demonstrates the tumor to abut the costovertebral angle, MRI is indicated to rule out extension into the neural foramen.
- CT-guided FNA biopsy can be confirmatory, although surgical extirpation is usually recommended for both diagnostic and therapeutic purposes.
- Transesophageal ultrasound can also differentiate between solid and cystic intraluminal esophageal masses, which are often not discernible by CT scan.
- Typically, pericardial cysts are observed radiologically, reserving surgical intervention if a foregut cyst or cystic neoplasm cannot be ruled out.

Mediastinal tumour	Clinical features	Treatment
Thymoma	Thymomas usually present in the fourth and fifth decades of life, although cases have been reported from childhood well into the ninth decade of life. There is no clear sex predisposition. One third to one half of patients present with an asymptomatic anterior mediastinal mass on chest radiograph or computed tomography (CT), one third present with local symptoms (e.g., cough, chest pain, phrenic nerve palsy, superior vena cava syndrome, dysphagia), and one third are detected during the evaluation of myasthenia gravis. Distant metastases are uncommon at initial presentation. However, when present, the most common metastatic site is the pleura. Metastasis to the kidney, bone, liver, and brain is rarely seen. **Paraneoplastic Syndromes Associated with Thymoma** **Cardiac Disorders** Acute pericarditis	**SURGICAL RESECTION** The basic principles underlying successful surgical therapy are: (1) median sternotomy; (2) complete wide resection of the tumor; (3) resection of all normal thymic tissue; (4) extended en bloc resection of invasive tumors (Masaoka stage III), including, if necessary, pericardium, lung, brachiocephalic vein, or superior vena cava; and (5) excision of all pleural implants or lung metastases. For tumors confined to the thymus gland, wide excision should still be performed, extending from phrenic nerve to phrenic nerve and from the superior thymic poles to the diaphragm. Lesser resection even for small and seemingly encapsulated tumors

	Myocarditis **Collagen Disorders** Polymyositis Rheumatoid arthritis Scleroderma Systemic lupus erythematosus **Dermatologic Disorders** Alopecia areata Pemphigus (vulgaris, erythematosus) **Endocrine Disorders** Addison's disease Cushing's syndrome Panhypopituitarism Thyroiditis **Gastrointestinal Disorders** Ulcerative colitis **Hematologic Disorders** Agranulocytosis Erythrocytosis Hemolytic anemia Pancytopenia Pernicious anemia Red cell aplasia **Immune Deficiency Syndromes** Hypogammaglobulinemia DiGeorge syndrome **Neuromuscular Syndromes** Myasthenia gravis Myositis Limbic encephalopathy Myotonic dystrophia Stiff-person syndrome **Renal Disorders** Minimal change nephropathy Nephrotic syndrome	is not appropriate **INDUCTION (NEOADJUVANT) THERAPY** With cisplatin, epirubicin, and etoposide before and after resection has resulted in complete or partial responses in most patients, with dramatic improvements in survival for higher stage. Patient selection for induction therapy is generally based on the size of the tumor, because tumors greater than 10 cm appear to be invasive in most cases and associated with poorer outcome.

Lymphoma	Cough, chest pain, fever, and weight loss.	Combination chemotherapy
Germ-Cell Tumors	Chest pain, cough, and hoarseness.	Seminoma – surgical resection+ post operative radiotherapy.
Neurogenic tumours	Located in the paravertebral gutter. 10-20% are malignant. Chest pain is the commonest symptom. May cause spinal cord compression.	Surgical excision. In malignant tumours postoperative radiotherapy is helpful.
Pericardial cyst	Usually asymptomatic.	Surgery is usually done as a diagnostic procedure to identify the lesion.

3. Write short notes of the following : 6 x 5

(a) Flail chest.
(b) Glasgow Coma Scale.
(c) Biliary atresia.
(d) Meningocoele.
(e) Osteomyelitis

Answer. a) Flail chest.

- Multiple injuries to multiple ribs can result in an island of chest wall that is not fixed to the surrounding thoracic cage.
- Results in paradoxical motion of the chest wall. Hypoxia is caused by restricted chest wall movement and underlying lung contusion.
- If the segment is small and respiration is not compromised, nurse patient in HDU with adequate analgesia. Encourage early ambulation and vigorous physiotherapy. Regular blood gas analysis is needed.
- In more severe cases, endotracheal intubation with positive-pressure ventilation is required.
- The management of flail chest should focus on the size and degree of anticipated pulmonary contusion. The contusion is often underestimated initially because it does not appear on x-ray film until 24 hours later.
- Small flail segments can usually be managed conservatively with pain control. Larger flail segments hinge on pain control and management of underlying pulmonary

contusion. As the pulmonary contusion improves, the respiratory mechanics related to the area of flail will improve.

- Surgical stabilization of severe flail injuries remains controversial. Surgical devices such as wires, compression plates, absorbable surgical ties, straps, and fixators have been described and proponents claim shorter time on the ventilator with less pain. These procedures are often performed via an extrapleural approach. Thoracotomy solely to perform open reduction and internal fixation of flail segments has generally not been recommended. Operative fixation of the flail chest remains an extremely uncommon procedure. Some authors have adapted the approach that they may attempt fixation of ribs in a patient who is undergoing thoracotomy for another reason.

(b) Glasgow Coma Scale.

Answer. Initial management of head injury focuses on hemodynamic stabilization through establishment of an adequate airway, ventilation, and support of circulation, followed by the rapid diagnosis and treatment of intracranial injuries.

A rapid but systematic neurologic examination is performed on the scene and is repeated frequently during transport and on initial presentation to the emergency room. Examination focuses on the three components of the Glasgow Coma Scale (GCS): eye opening, verbal response, and motor response.This score indicates injury severity and measures changes in the impairment of consciousness.

Glasgow Coma Scale

Component	Points
Eye opening	
Spontaneous	4
To voice	3
To stimulation	2
None	1
Motor response	
To command	6
Localizes	5
Withdraws	4
Abnormal flexion	3
Extension	2
None	1
Verbal response	
Oriented	5
Confused but comprehensible	4
Inappropriate or incoherent	3
Incomprehensible (no words)	2
None	1

Glasgow Coma Score = best eye opening + best motor response + best verbal response. If patient is intubated, the verbal score is omitted and an addendum of

"T" is given to the best eye opening + best motor response score.
Mild injury GCS 14–15.Moderate injury GCS 9–13.Severe injury GCS 3–8.

(c) Biliary atresia.

Answer. Biliary atresia:

- Most common cause of infantile jaundice that requires surgical correction.
- Etiology is unknown.
- Disease characteristics
  - Progressive obliteration and sclerosis of the biliary tree.
  - With age, obliteration of the extrahepatic bile ducts, proliferation of the intrahepatic bile ducts, and liver fibrosis progress at an unpredictable rate.

There are variants of BA ranging from fibrosis of the distal bile ducts with i. proximal patency (5%, considered correctable form), ii. fibrosis of the proximal bile ducts with distal patency (15%), or iii. fibrosis of both proximal and distal bile ducts (80%).

- Diagnosis
  - Clinical and laboratory information is often nonspecific.
  - Jaundice.
  - Alcoholic stools.
  - Dark urine.
  - Hepatomegaly.

A serum direct bilirubin level of more than 2.0 mg/dL or greater than 15% of the total bilirubin level defines cholestasis.

In addition to a careful history and physical examination the following investigations are done:

- Blood and urine are obtained for bacterial and viral cultures.
- Reducing substances in the urine to rule out galactosemia
- Serum immunoglobulin M (IgM) titers for syphilis
- Cytomegalovirus, herpes, and hepatitis B,
- Serum a_1-antitrypsin level and phenotype,
- Serum thyroxine level, and
- Sweat chloride test done to exclude cystic fibrosis.
- Percutaneous liver biopsy. Biopsy results range from classic biliary tree fibrosis to those unable to be differentiated from α1-antitrypsin deficiency or neonatal hepatitis.
- Technetium-99m iminodiacetic acid hepatobiliary imaging aids in differentiation between liver parenchymal disease and biliary obstructive disease.

In biliary atresia, the liver readily takes up this tracer molecule, but no excretion into the extrahepatic biliary system or duodenum is seen.

- Ultrasonography. Shrunken extrahepatic ducts and a noncontractile or absent gallbladder.

- Management
  - Open liver biopsy and cholangiogram
  - The common bile duct is visualized by cholangiography in only 25% of patients with biliary atresia.
  - Cholangiography in the remaining 75% of patients demonstrates an atretic biliary tree.
  - Operative intervention
  - Kasai procedure:
  - "Hepatoportoenterostomy."
  - Excision of obliterated extrahepatic ducts with hepaticojejunostomy.
  - When the distal common bile duct is patent, a choledochojejunostomy is constructed.

The overall results have shown that in children treated with a Kasai procedure, one third show long-term improvement, one third obtain temporary benefit, and one third experience treatment failure.

Postoperative Management:

- Postoperatively, the use of oral choleretic bile salts such as ursodeoxycholic acid may facilitate bile flow.
- In addition, methylprednisolone is employed as an anti-inflammatory agent.
- Trimethoprim sulfamethoxazole is administered for long-term antimicrobial prophylaxis.
- Cholangitis is a serious but common problem after hepatoportoenterostomy. Episodes of cholangitis are managed by hospitalization, rehydration, broad-spectrum IV antibiotics, steroids, and occasionally surgical exploration of the portoenterostomy.

(d) Meningocoele.

Answer. Meningocele: The least common form of spina bifida is a posterior meningocele (or meningeal cyst). In a posterior meningocele, the vertebrae develop normally, however the meninges are forced into the gaps between the vertebrae. As the nervous system remains undamaged, individuals with meningocele are unlikely to suffer long-term health problems, although there are reports of tethered cord.

Causes of meningocele include

- **Teratoma** and other tumors of the sacrococcyx and of the presacral space, and
- **Currarino syndrome**, Bony defect with outpouching of meninges.

Symptoms of Meningocele			
Asymptomatic	Bowel dysfunction	Hydrocephalus	Bladder dysfunction
Spinal defect	Paralyzed legs	Spinal mass	Pain

DIAGNOSIS: In most cases, a meningocele diagnosis is made before birth.

Investigations:

Second-trimester maternal serum alpha-fetoprotein screening

Fetal ultrasound Multiple-marker screens Amniocentesis.

Meningocele repair

Meningocele repair (also known as myelomeningocele repair) is surgery to repair birth defects of the spine and spinal membranes. Meningocele and myelomeningocele are types of spina bifida.

Description

A meningocele repair is a surgical procedure performed to repair an abnormal opening in the spinal column (called spina bifida) by draining excess fluid and closing the opening.

For both meningoceles and myelomeningoceles, the surgeon will close the opening in the back.

After birth, the defect is covered by a sterile dressing. The child may then be transferred to a neonatal intensive care unit (NICU) and cared for by a medical team experienced in caring for children with spina bifida.

Baby will likely have an MRI (magnetic resonance imagining) or ultrasound of the back. An MRI or ultrasound of the brain may be done to look for hydrocephalus(extra fluid in the brain).

If the myelomeningocele is not covered by skin or a membrane when your child is born, surgery will happen within 24 to 48 hours after birth to prevent infection. If the child has hydrocephalus, the doctor may put a shunt (plastic tube) in their brain to drain the extra fluid to the stomach. This prevents pressure that could damage the baby's brain.

The child should not be exposed to latex before, during, and after surgery.

Many of these children have very bad allergies to latex.

Risks

Risks for any anesthesia are:

- Breathing problems
- Reactions to medications

Risks for any surgery are:

- Infection
- Bleeding

Risks for this surgery are:

- Fluid build up and pressure in the brain (hydrocephalus)
- Increased chance of urinary tract infection and bowel problems
- Infection or inflammation of the spinal cord
- Paralysis, weakness, or sensation changes due to loss of nerve function

(e) Osteomyelitis.

Answer. Acute haematogenous osteomyelitis:This is a disease of growing bones. It is common in infants and children but rare in adults unless they are immunocompromised or diabetic.

Aetiology:

- Infants: Staphylococcus aureus, Streptococcus, and Escherichia coli.
- Children: S. aureus, Streptococcus, Haemophilus influenzae B.
- Adults: S. aureus.
- Sickle-cell patients: Salmonella sp.
- Rare causes: Brucella, TB, spirochetes, and fungi.

Pathological features: The organisms settle near the metaphysis at the growing end of a long bone. The following stages typically occur.

- Inflammation. Acute inflammation with venous congestion.
- Suppuration. After 2-3 days pus forms in the medulla and forces its way out to the periosteum.
- Necrosis. After 7 days blood supply is compromised and infective thrombosis leads to necrosis and formation of a pocket of dead tissue (sequestrum).
- Repair. At around 10-14 days new bone is formed from the subperiosteal layer that was stripped with the swelling (involucrum).
- Discharge. Involucrum can develop defects (cloacae) allowing discharge of pus and sequestrum to allow resolution. This can also be achieved by surgical release and debridement.

Clinical features

- Usually a child with a preceding history of trauma or infection (skin or respiratory).

- Fever, pain, and malaise develop after a few days.
- The child may be limping or refusing to weight bear.
- On examination there may be localized swelling or redness of a long bone.
- Infants may present with a failure to thrive, drowsiness, or irritability.
- Neonates may present with life-threatening septicaemia in which obvious inflammation of a long bone develops, or a more benign form in which the symptoms are slow to develop but bone changes are extensive and often multiple.

Investigation

- Plain X-rays may be normal for the first 10 days. Do not be reassured!
- ^{99}Technetium bone scan is usually positive in the first 24-48h and is effective in confirming diagnosis early.
- ^{67}Gallium bone scan and ^{111}Indium-labelled white cell scans are more specific.
- MRI is very sensitive but not specific and difficult for children.
- CT scanning can define extent of bone sequestration and cavitation.

X-ray features

- Soft tissue swelling is an early sign; look for displacement of fat planes.
- Patchy lucencies develop in the metaphysis at around 10 days.
- Periosteal new bone may be seen.
- Involucrum formation is only apparent at around 3 weeks.
- Sequestrum appears radiodense compared to the surrounding bone, which is osteopenic.
- Normal bone density occurs with healing.

Laboratory tests results

- PBC , normally with a raised neutrophil count.
- ESR increased.
- CRP increased.
- Blood cultures positive in 50% of cases (use to inform and adjust antibiotic therapy).
- Perform Urea & Creatinine, LFTs, and glucose.

Treatment

- Pain relief by bed rest, splintage, and analgesics.
- Give IV antibiotics according to local guidelines (after blood cultures and pus swab samples taken), e.g. flucloxacillin IV then PO qds for up to 6 weeks dose-adjusted according to age, clindamycin if penicillin-allergic, vancomycin if MRSA, ampicillin for Haemophilus.

- Surgical drainage of mature subperiosteal abscess with debridement of all necrotic tissue, obliteration of dead spaces, adequate soft-tissue coverage, and restoration of an effective blood supply.

Complications

- Disseminated systemic infection, e.g. septicaemia, cerebral abscess.
- Chronic osteomyelitis.
- Septic arthritis.
- Deformity due to epiphyseal involvement.

Chronic osteomyelitis

Causes

- Occasionally following acute haematogenous osteomyelitis.
- Most common following contaminated trauma and open fractures.
- After joint replacement surgery.
- Primary chronic infections of bone.
- Secondary to acute osteomyelitis.

Features

- Sinus formation due to sequestra or resistant bacteria.
- Prevented by adequate treatment of the initial acute attack.

Treatment

- Conservative (simple dressings) may be appropriate (elderly). Recurrent attacks with spontaneous recovery may occur and surgery should be reserved for cases where an abscess forms.
- Chronic abscess. May require drainage, debridement of all necrotic tissue, and obliteration of dead spaces. May involve plastic surgery to achieve soft-tissue cover and restoration of an effective blood supply.
- Closed suction drainage/irrigation systems can be effective, especially if irrigation fluid contains antibiotics. The disadvantage is that early blockage of the system can occur.
- Antibiotic (gentamicin) impregnated beads or sponges deliver high local levels and may be beneficial in areas of poor blood supply, hence systemic antibiotic penetration.
- Unresolving cases may require amputation.

Secondary to trauma (open fractures)

- Prevention by early aggressive approach to compound fractures with debridement and lavage of contaminated tissue.

    - Excise all dead tissue widely and remove all devitalized bone fragments, i.e. with no soft tissue connections.
    - Copious lavage is necessary.
    - Skeletal stabilization is mandatory.
    - IV antibiotics, e.g. IV cefuroxime ± metronidazole if anaerobes may be involved (soil).
- Treat established chronic infection as above with removal of internal foreign bodies, e.g. metalwork, and possible application of external fixation.

Secondary to joint replacement surgery

- Rare, but is often a disaster for the elective patient.
- Prevention is better than cure. Dedicated laminar flow theatres, strict theatre discipline, and prophylactic IV antibiotics are mandatory.
- 50% will require surgical intervention.
    - Initial joint irrigation, debridement, and tissue sampling can be attempted if the prosthesis is still solid and not loose.
    - If grossly infected, the prosthesis must be removed, the surfaces debrided and an antibiotic cement spacer placed on the raw bone ends to allow the soft tissue envelope to settle.
    - Once inflammatory markers have settled (CRP is the best) and the clinical infection has resolved, second stage replacement of the spacer with a new prosthetic joint can go ahead. This may take 12 months and may not be possible.

Chronic osteomyelitis as an initial presentation

- Brodie's abscess

- An isolated well-contained chronic abscess.
- Treatment: operative drainage with excision of the abscess wall and antibiotics.

- Tuberculosis

- Usually associated with other systemic features of the disease.
- May present acutely.
- Muscle atrophy develops and spontaneous discharge of a cold abscess may lead to sinus formation and destruction of bone.
- Spinal TB may cause vertebral collapse leading to acute neurology Pott's paraplegia.

- Syphilitic osteomyelitis

- Associated with advanced, tertiary disease in adults. Features diffuse periostitis (with sabre tibia) or localized gummata with sequestra, sinus formation, and pathological fractures. X-rays show periosteal thickening with punched out areas in sclerotic bone.
- Infants with congenital disease have epiphysitis and metaphysitis. X-rays show areas of sclerosis near the growth plate separated by areas of rarefication.

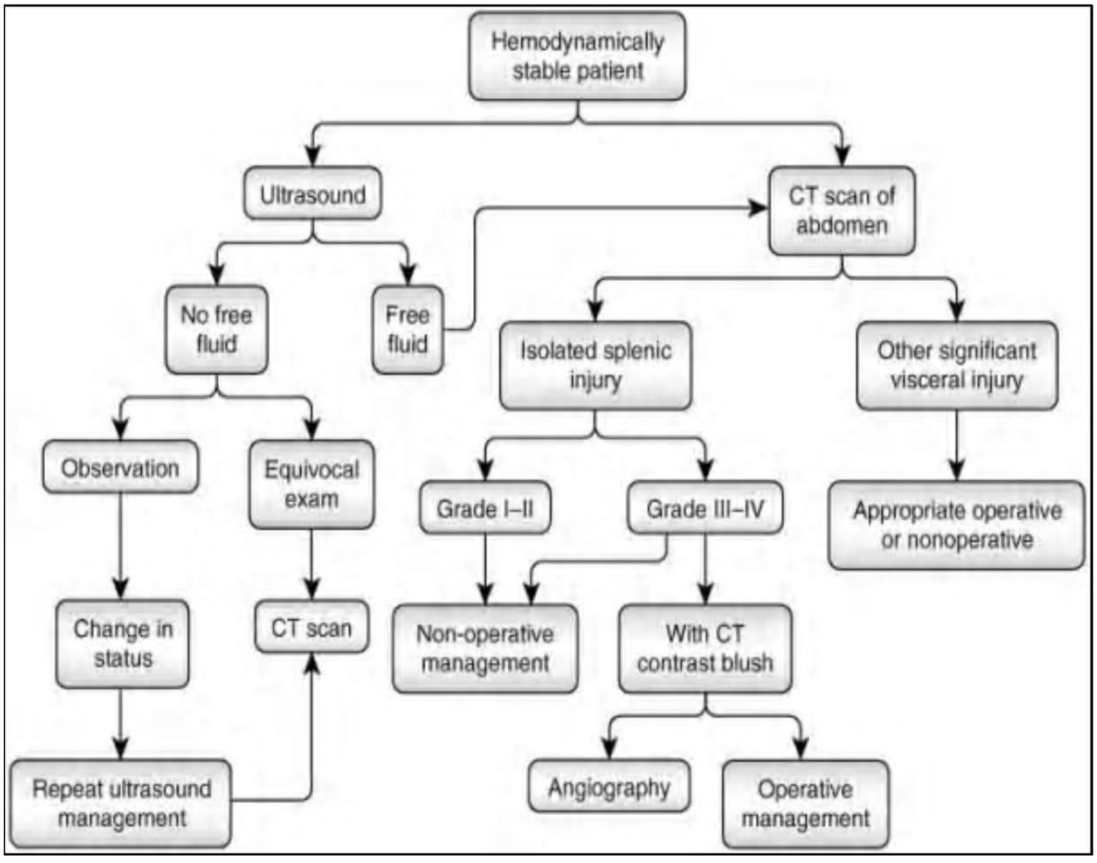
➢ Mycotic osteomyelitis

- Typically occurs in immunocompromised patients.
- Bone granulomas, necrosis, and suppuration present without worsening acute illness.
- Usually occurs as spread from primary lung infections such as coccidiomycosis, cryptococcosis, blastomycosis, and histoplasmosis.
- Treatment: amphotericin B and/or surgical excision.

4. Answer briefly on the followings: 4 x 7.5
(a) Splenic preservation.
(b) Urethral injury.
(c) Diabetic foot.
(d) Surgical Site Infection.

(a) Splenic preservation.

Answer. An algorithm for the diagnostic imaging of patients with suspected blunt splenic trauma



All hemodynamically normal patients are candidates for nonoperative management. In addition, those patients who are initially hemodynamically labile but respond to crystalloid without further episodes of instability may also be considered for nonoperative management. The only true contraindication to nonoperative management in properly

monitored patients is hemodynamic instability, or another indication mandating laparotomy. Nonoperative management should not be entertained at hospitals where surgeons, operative capabilities, and intensive care resources are not immediately available at all times.

Criteria for Nonoperative Management of Splenic Injury:

• No indications for laparotomy (physical examination signs/symptoms or other injuries)
• Hemodynamically normal after resuscitation with crystalloid
• No injuries that preclude physical examination of the abdomen (e.g., CHI, spinal cord injury)
• No transfusion requirements (PRBC)
• Constant availability of surgical and critical care resources

Criteria for Failure of Nonoperative Management:

• Increasing fluid requirements to maintain normal hemodynamic status
• Failed angio embolization of A-V fistulae/pseudoaneurysm
• Transfusion requirements to maintain Hct/Hgb and normal hemodynamic status
• Increasing hemoperitoneum associated with hemodynamic liability
• Peritoneal signs/rebound tenderness

- Splenectomy should be considered in all adult patients with grades IV and V injuries. Lesser injury grades can often be managed with splenorrhaphy.
- Pediatric patients should have every attempt at splenic salvage.
- **Techniques for Splenorrhaphy/Spllenic conservation surgery:**

Topical Agents
Absorbable gelatin sponge
Oxidized regenerated cellulose
Microfibrillar collagen
Fibrin sealant
Coagulation
Electrocautery
Argon beam coagulator
Mesh
Polyglactin

Polyglycolic acid
Partial/Hemisplenectomy
Segmental vessel ligation, resection, mattress sutures
Stapler
Ultrasonic surgical aspirator

(b) Urethral injury.

Answer. Urethral Injuries

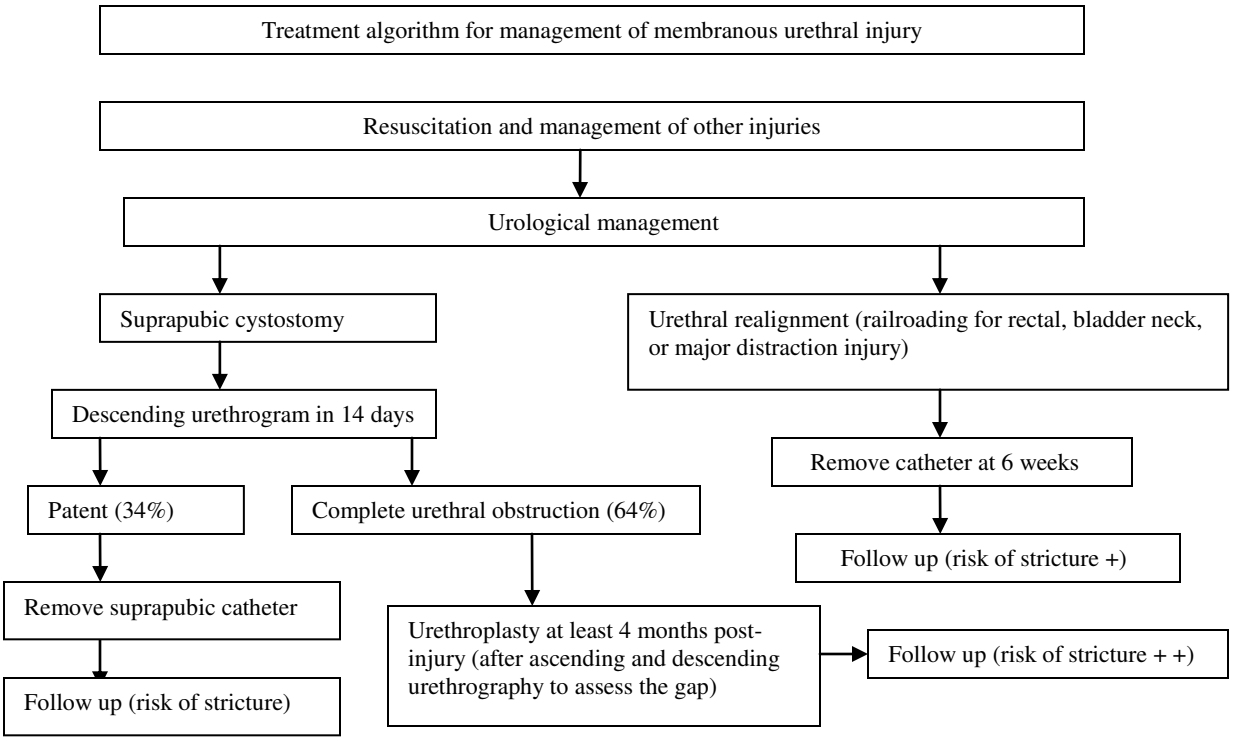
Urethral injuries are associated with 4% to 14% of all pelvic fractures and are more common in cases of bilateral pelvic injuries. Diagnosis of urethral injury is made by a high index of suspicion in the presence of blood at the urethral meatus, inability to urinate, or a palpable full bladder on abdominal examination. When blood is present at the meatus, retrograde urethrography aids in the diagnosis of urethral injury. In the presence of minor urethral injury, a catheter can be placed by an experienced urologist with or without the aid of a cystoscope.

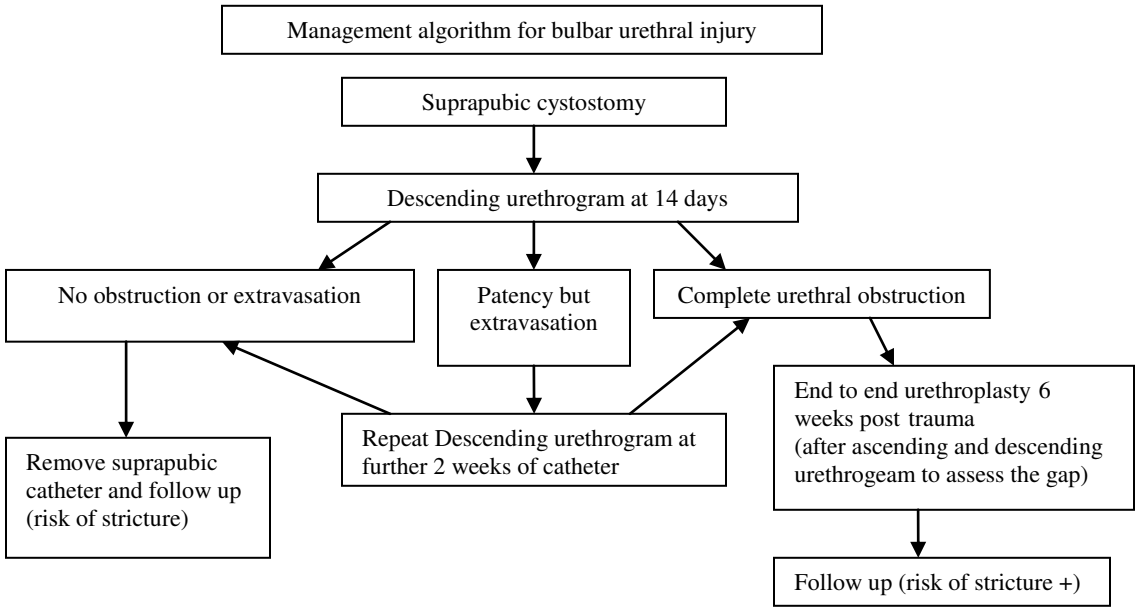
Urethral injuries are classified as those confined to the posterior urethra (above the urogenital diaphragm) or the anterior urethra (below the urogenital diaphragm).

Posterior urethral injuries are further subclassified as type I (urethral stretch), type II (urethral disruption proximal to the urogenital diaphragm), or type III (proximal and distal disruption of the urogenital diaphragm).

For treatment of posterior urethral injuries, early endoscopic realignment has become more accepted as an excellent initial option. Realignment of the damaged urethra with a stented Foley catheter can lead to either complete healing of the urethral injury or need for future endoscopic treatment of developed urethral strictures. If realignment of the damaged urethra cannot be achieved, then suprapubic catheterization, followed by delayed combined antegrade and retrograde endoscopic repair or open surgical repair, is undertaken.

In contrast to posterior urethral injuries, which are often associated with many other pelvic injuries, anterior urethral injuries are often isolated and associated with straddle injuries. The bulbar urethra is typically the site of injury. The best initial treatment modality for anterior urethral injuries is not well defined; however, most would agree that primary realignment with a Foley catheter is the best initial treatment. In cases of severe anterior urethral injury, a suprapubic catheter may be required, followed by delayed open surgical repair.





(c) Diabetic foot.

Answer. Surgeons are frequently called upon to evaluate and treat the complications of diabetic foot disease. In the diabetic population, foot disease is the leading cause of hospitalization.

Pathophysiology:

Diabetic foot disease involves a complex sequence of changes that lead to the destruction of the foot's function and structure. Peripheral neuropathy, arthropathy, arterial insufficiency, and infection are the major derangements underlying diabetic foot disease. The final common pathway is often one of ulceration, gangrene, and limb loss.

Peripheral Neuropathy

Peripheral neuropathy is common among diabetics and is the most difficult component of diabetic foot disease to treat.. Its severity ranges from minimal somatosensory changes to total anesthesia with the complete absence of sweating. Diabetic neuropathy affects the motor, sensory, and autonomic nervous systems, which leads to multiple pathologic conditions.

Destruction of myelinated motor fibers produces a motor polyneuropathy that tends to be bilateral and symmetric. Attenuation of the Achilles reflex is an early sign of motor neuropathy. Progression leads to loss of function and atrophy of the lumbricals and intrinsic muscles of the foot. Unopposed, the extrinsic muscles distort the foot, leading to depression of the metatarsal heads, digital contractures, "clawing" of the toes, and a varus malformation. These alterations allow pressure points and ulcers to develop over the proximal interphalangeal joints and beneath the metatarsal heads.

Sensory neuropathy involving type A myelinated fibers leads to loss of proprioception, light tough, pressure, and vibration and is noted to develop early. Compromise of these nerves leads to an ataxic gate and pressure ulceration. Loss of type C sensory fibers limits a patient's ability to detect painful or noxious stimuli, which contributes to neuroarthropathy, injuries, and the formation of ulcers. C fibers also participate in nociceptive or neuroinflammatory responses that blunt the signs of trauma and infection in diabetic feet. Sensory neuropathy can also paradoxically produce pain and paresthesias. Neuropathic pain can be severe and quite difficult to differentiate from other foot problems. Diabetic paresthesias are characterized by hyperesthesia, burning, or tingling sensations and tend to resolve over time.

Autonomic neuropathy produces abnormal vasomotor responses. Autosympathectomy increases arteriovenous shunting, which leads to osteopenia. Hypohidrosis results in dry, cracked skin, which compromises the skin barrier and increases the susceptibility to infections.

Neuroarthropathy

Neuroarthropathy or Charcot's foot is a condition in which extensive destruction of the joints occurs within the foot consequent to a loss in muscular stability, osteopenia, and insensitivity to pain. It is present in many disorders of peripheral nerves but is most common in diabetics. Sensory neuropathy permits mechanical insults to go unrecognized

by the patient, which produce severe stresses on osteopenic bone. Bony dissolution and loss of structural integrity follow, leading to collapse of the midfoot arch.

Minor foot trauma (e.g., minor fracture, sprain, and contusion) often catalyzes neuropathic skeletal changes. Localized warmth and swelling at the site occur initially; this is usually nontender and out of proportion to the injury. The warmth and swelling of this inflammatory stage of neuropathic osteoarthropathy can be difficult to distinguish from infection. Chronically, skeletal encroachment occurs in the tarsometatarsal and metatarsophalangeal joints followed by collapse of the arch. Subluxation or even lateral dislocation of the forefoot occurs, producing a bowed, "rocker-bottom" foot, which is extremely susceptible to ulceration.

Arterial Insufficiency

Altered glucose metabolism in diabetics leads to changes in response to endothelial injury, dyslipidemia, enhanced platelet activity, and increased blood viscosity. These factors contribute to an increased incidence of atherosclerotic occlusive disease and hypercoagulability in diabetic patients. Arterial insufficiency contributes to the development of neuropathy, limits a patient's ability to heal ulcers and traumatic injuries, and in the worst case, produces ischemic ulcers. The distribution of atherosclerotic disease in diabetics typically involves the femoropopliteal and tibial vessels with relative sparing of aortoiliac and pedal vessels. Consequently, femoral to distal tibial or pedal bypasses are feasible for limb salvage in many diabetics with critical ischemia.

Infection

Diabetic foot infections range from superficial cellulitis to wet gangrene and abscess formation. Peripheral neuropathy and neglected skin lesions or ulcers often initiate deep-space infections. Fungal infections contribute to ulceration and secondary bacterial infection either by direct microscopic breaks in the skin or through toenail loss. Loss of proprioception facilitates traumatic injury. Arterial insufficiency and the diminished inflammatory response allow for rapid progression of an infection. The hallmark of diabetic foot infections is a polymicrobial infection. Gram-positive species, including streptococci, staphylococci, enterococci, and gram-negative anaerobes are present.

Assessment:

Assessment of the patient with diabetic foot disease begins with a thorough history with attention given to the presence of cardiac disease, renal disease, peripheral vascular disease, smoking history, and habits of foot care. Laboratory studies include a basic chemistry, a complete blood cell count, a hemoglobin A_{1c} concentration, and a urinalysis. A comprehensive physical examination with emphasis on the neurologic and vascular function of the lower extremities is essential.

The feet should be carefully examined for capillary refill, nail thickening, ulcers or ischemic lesions, signs of infection, intrinsic muscle wasting, bony prominences, and

evidence of tenderness. Ischemic ulcers tend to occur in the toes, whereas neuropathic ulcers present over bony prominences and the heel in patients who are bed bound. Gentle probing of ulcers to determine whether there is palpable bone at the base is encouraged as a highly specific and accurate test for the detection of osteomyelitis. Superficial wound cultures are of little utility. Pulse assessment of the femoral, popliteal, dorsalis pedis, and posterior tibial arteries should be performed. In the absence of palpable pulses, a continuous-wave Doppler probe should be used to assess for the presence of signals.

Neurologic examination begins with examination of vibratory sense in the toes and ankle and proceeds to the deep-tendon reflexes of the Achilles and patellar tendons. Finally, sensation of pain, light touch, and temperature in the feet should be evaluated. Inability to feel the pressure from a Semmes-Weinstein 5.07 nylon monofilament applied to multiple points on the plantar surface is a sensitive screening test for the detection of neuropathy.

Noninvasive laboratory studies are often helpful in assessing the degree of vascular insufficiency and predicting healing potential. Studies may include segmental limb pressures with Doppler waveforms, calculation of ankle-brachial indices, and toe pressures. Any gradient greater than 30 mm Hg between any level indicates the presence of a high-grade stenosis or occlusion. Calcification within the arterial wall often renders diabetic vessels incompressible and waveform interpretation must be used to identify disease. In diabetics, toe or partial foot amputations require ankle pressures of at least 55 mm Hg and toe pressures of at least 30 mm Hg to heal. Transcutaneous oxygen measurements may also predict healing if there is at least a pressure of 30 mm Hg.

Imaging

- Plain radiographs are useful in the assessment of the diabetic foot. They can identify soft tissue calcifications, gas from bacterial infections, the presence of foreign bodies, and fractures. Plain radiographs can detect osteomyelitis but are most sensitive in the later stages when bony destruction has occurred. Three-phase bone scans are highly sensitive for osteomyelitis except in the presence of severe vascular disease.
- Arteriography is required when foot perfusion appears inadequate and arterial reconstruction is planned. The location and degree of occlusive disease are readily assessed with arteriography. Assessment of the outflow vessels and, in particular, communication of the pedal vessels with the pedal arch is also important. Magnetic resonance angiography (MRA) is highly accurate in the detection of large-vessel disease and is useful in patients who are intolerant of contrast dyes.
- Magnetic resonance imaging (MRI) of the diabetic foot provides unparalleled imaging of the soft tissues. Contrast-enhanced sequences can be used to determine the extent of osseous and soft tissue infection and can delineate areas of ischemia or necrosis. Cellulitis and deep infection appear as areas of bright enhancement on contrast-enhanced images. Diffuse soft tissue edema is very common in the diabetic foot and is differentiated from cellulitis by the presence of minimal contrast enhancement. Bone infarction appears as areas of a longitudinally oriented signal abnormality in the central medullary cavity, with a sharp well-defined margin. MRI is increasingly becoming the preferred imaging test for osteomyelitis. Osteomyelitis is characterized by an altered

bone marrow signal. Sensitivity of MRI for osteomyelitis ranges from 77% to 100%, with a specificity from 79% to 100%.

Management: Optimal treatment of diabetic patients requires a comprehensive program involving control of hyperglycemia, foot protection, and the timely treatment of ulcers, osteoarthropathy, and infections. Diabetics with high-risk foot conditions benefit from education regarding risk factors and their appropriate management.

Prevention

Prevention of ulceration is imperative to prevent limb loss. Every diabetic patient should have an annual foot examination by a physician and, if the patient has neuropathy, at every visit with a health care professional. Self-examinations should occur daily. Hyperglycemia should be controlled to slow the development of neuropathy. The risk of ulceration is increased in diabetic patients with longstanding diabetes, male gender, poor glucose control, neuropathy, or the presence of vascular, renal, or cardiac disease complications of diabetes. Patients with signs of increased plantar pressure such as erythema, warmth, or callus formation should use protective footwear that cushions and redistributes weight appropriately. Nonambulatory or bed-bound patients should also have their feet protected; the Rooke boot is a sheepskin and foam boot that is ideal for this purpose (Osbourne Medical, Rochester, Minn.). Calluses should be débrided by a specialist. Dryness and fungal infections should be treated appropriately with petroleum-based products (e.g., Aquaphor ointment) and topical antifungals, respectively.

Neuropathic Ulcers

Diabetic foot ulcerations fall into two main categories, ischemic and neuropathic, with some overlap between. In about 30% of patients with plantar neuropathic ulcerations, ischemia will prolong healing times. Vascular assessment is always required to ascertain the presence of coexisting occlusive disease. However, even with adequate arterial inflow, neuropathic ulcers may remain unchanged for months secondary to impaired inflammatory and healing responses. These ulcers are subject to the constant risk of infection, and prolonged ulcer healing lengthens the exposure to this risk. Infection requires prompt management when it arises.

The extent and anatomic site of neuropathic ulcers direct their management. They are most commonly located beneath one of the metatarsal heads or the interphalangeal joint of the hallux. Aggressive débridement is essential in establishing the depth of the ulcer. This requires removal of excessive callus and all necrotic debris. Daily wound care minimizes the risk of infection.

Protecting the weightbearing surface of the foot and offloading mechanical stress is paramount in the treatment of neuropathic ulcers not associated with ischemia. Nonweightbearing for 6 to 12 weeks is an ideal, though poorly tolerated, method to promote healing. An alternative treatment is total contact casting (TCC), which uses a protective short leg cast to mechanically offload the foot at the ulcer site and control edema

while allowing some degree of ambulation. TCC results in healing of 70% to 100% of neuropathic plantar ulcers within 5 to 8 weeks. Neuropathic ulcers 3 cm or less in diameter can be treated with TCC unless severe ischemia, deep ulceration, infection, or significant edema is present. TCC is generally well tolerated and complications infrequently occur from inadequate padding of bony prominences, cast friction, or skin irritation. Specialized shoes with arch bars or formed orthotic insoles are another method to redistribute pressure evenly across the plantar foot.

When casting or specialized footwear alone is inadequate, surgical correction of architectural deformities may be necessary. Correction of clawed toes at the metatarsophalangeal or proximal interphalangeal joints, arthrodesis of the interphalangeal joint of the hallux, resection of the head of one or more metatarsals, or even lengthening of the calcaneus tendon may be needed to maintain closure of an ulcer. Adequate perfusion must be confirmed before any surgical procedure is performed.

Once the ulcer is healed, the foot must be continually protected in appropriate footwear. In the diabetic foot, all calluses should be considered preulcerative and should not be mistaken as "benign." Calluses signify areas of repetitive trauma over bony prominences due to vertical, shear, and compressive forces. Calluses should be reduced, bony prominences padded or relieved, and skin lotion used after bathing the feet once or twice daily, drying well between the toes. Antibiotics are used in neuropathic and ischemic foot ulcers only in the presence of cellulitis, abscess, osteomyelitis, or pyarthrosis.

Ischemic Ulcers and Gangrene

Gangrene and ischemic ulcers are common complications with which surgeons are confronted. Patients with dry gangrene—for example, a black, "mummified" toe—without signs of infection should undergo a careful vascular evaluation to determine healing potential. If the healing potential is inadequate, evaluation for revascularization should be undertaken with amputations delayed until this can be safely performed. Dry gangrene in this setting is almost never the cause of systemic infection.

- Aggressive arterial reconstruction has been paramount in reducing amputation rates over the past several decades.
- With current techniques, 90% of diabetics presenting with ischemic ulceration or gangrene are found to have surgically correctable occlusive disease.
- Vascular reconstruction in diabetic patients typically involves bypass from the femoral or popliteal arteries to tibial, peroneal, or pedal vessels.
- In the absence of continuous tibial vessels, a bypass to a patent pedal vessel or a peroneal artery with good ankle collaterals is a reasonable option.
- Autologous vein should be used whenever possible because synthetic grafting to infrageniculate vessels has poor long-term patency.
- Primary patency rates of 80% at 1 year and limb-salvage rates of 92% at 3 years are achievable.
- Although endovascular treatment of flow-limiting arterial stenoses and occlusions has not achieved the long-term patency of distal bypass with autologous vein grafts, this

modality is effective in short-term improvements in circulation. Endovascular therapy may be preferable in patients unable or unwilling to undergo traditional bypass.

Invasive Foot Infection:

Infection often originates in areas of recent trauma or chronic ulceration and may progress rapidly to acute foot sepsis. Pain and tenderness, for instance, may be absent depending on the degree of sensory neuropathy. Infection alters glucose metabolism further in the diabetic, and extreme hyperglycemia or even ketoacidosis may occur. The initial management is directed at resuscitation and control of the foot infection.

- Drainage and débridement of necrotic tissues are the primary goals in invasive diabetic foot infections with revascularization delayed until the infection has been controlled.
- Diabetic foot infections are often much more extensive than assumed given the propensity of infection to track along the tendon and fascial sheaths.
- Wound and sinus tracts should be opened to permit free drainage; involved bone or joints should be resected.
- Subsequent procedures may be necessary to ensure adequate drainage.
- Deep cultures of the wound should be obtained.
- Initial broad-spectrum antibiotics should be administered and refined based on culture and sensitivity data.
- Partial foot amputations may be warranted to control sepsis.
- These amputations are left open, and are designed to remove large areas of devitalized tissue, destroyed bones, and septic joints.
- Careful consideration must be given to preserve as much functional tissue as possible.
- Guillotine amputation at or above the ankle is required when infection extends proximal to the midfoot and is associated with hindfoot ischemia.
- Definitive amputation follows when infection has been controlled and metabolic derangements improve.

Amputation

- Amputations are indicated for removal of gangrenous or necrotic tissue.
- The level of amputation depends on the proximal extent of the disease and the degree of vascular insufficiency.
- With normal circulation, amputation can usually be performed just proximal to the extent of the necrotic tissue.
- Distal toe ulcers will often slough once revascularization has occurred and should be followed for several months before amputation is offered.
- Although some allow dry gangrenous digits to autoamputate, button or ray amputations are preferable to spare the discomfort and distress autoamputations can cause patients.
- Resection of the distal metatarsal head and removal of all cartilage and sesamoid bones are essential maneuvers in the performance of ray amputations.
- A transmetatarsal amputation is considered if multiple toes or the distal forefoot is involved. A transmetatarsal amputation is inadequate in the presence of the midplantar foot ischemia, especially if the medial two or lateral three rays must be sacrificed. In

this scenario, midfoot or hindfoot amputations (e.g., Chopart's) along with the appropriate tenotomies may be considered.

- Even if successful, however, hindfoot amputations leave patients with a "peg leg," which may not be as functional as a below-knee amputation.If ischemic disease or infection involves the proximal foot, a below-knee amputation is indicated in the ambulatory patient.
- Furthermore, in the presence of extensive neuropathic osteoarthropathy involving the tarsal bones with total collapse of the midfoot, a below-knee amputation may be preferable to extensive attempts at reconstruction.
- Above-knee amputations may be appropriate in chronically nonambulatory patients, patients with fixed knee contractures, or patients with marginal, unreconstructible, distal circulation.

Adjunctive Therapies

Despite good wound care, adequate offloading, and treatment of infection, diabetic foot ulcers will occasionally be recalcitrant to standard care. Several therapies exist to improve wound healing, including hyperbaric oxygen therapy, platelet-derived growth factors, bioengineered skin and dermal substitutes, granulocyte colony-stimulating factor, and vacuum-assisted dressings. Vacuum-assisted dressings (VAC; Kinetic Concepts, Inc., San Antonio, Texas) have been particularly useful in stimulating reepithelialization and granulation tissue formation and are well tolerated by patients.

(d) Surgical Site Infection

Answer. Surgical site infections (SSIs) are infections present in any location along the surgical tract after a surgical procedure.
SSIs are the most common nosocomial infection in our population and constitute 38% of all infections in surgical patients. By definition, they can occur anytime from 0 to 30 days after the operation or up to 1 year after a procedure that has involved the implantation of a foreign material (mesh, vascular graft, prosthetic joint, and so on). Incisional infections are the most common; they account for 60% to 80% of all SSIs and have a better prognosis than organ/space-related SSIs do.
Staphylococcus aureus remains the most common pathogen in SSIs, followed by coagulase-negative staphylococci, enterococci, and ***Escherichia coli***. However, for clean-contaminated and contaminated procedures, ***E. coli*** and other Enterobacteriaceae are the most common cause of SSI. In addition, some emerging organisms have become more common in recent years. Vancomycin-resistant enterococci (VRE) and gram-negative bacilli with unusual patterns of resistance have been isolated more frequently. Of particular interest is the growing frequency of ***Candida*** species as a cause of SSI and surgical infections in general.

Risk Factors for Surgical Site Infection According to the Three Main Determinants of Such Infection

MICROORGANISM	LOCAL WOUND	PATIENT

MICROORGANISM	LOCAL WOUND	PATIENT
Remote site infection	Surgical technique:	Age
Long-term care facility	Hematoma/seroma	Immunosuppression
Recent hospitalization	Necrosis	Steroids
Duration of the procedure	Sutures	Malignancy
Wound class	Drains	Obesity
Intensive care unit patient	Foreign bodies	Diabetes
Previous antibiotic therapy		Malnutrition
Preoperative shaving		Multiple comorbid conditions
Bacterial number, virulence, and antimicrobial resistance		Transfusions
		Cigarette smoking
		Oxygen
		Temperature
		Glucose control

Bacterial factors include virulence and bacterial load in the surgical site. The development of infection is affected by the toxins produced by the microorganism and the microorganism's ability to resist phagocytes and intracellular destruction. Several bacterial species have surface components that contribute to their pathogenicity by inhibiting phagocytosis (e.g., the capsules of ***Klebsiella*** and ***Streptococcus pneumoniae,*** the slime of coagulase-negative staphylococci). Gram-negative bacteria have surface components (endotoxin or lipopolysaccharide) that are toxic, and others, such as certain strains of clostridia and streptococci, produce powerful exotoxins that enable them to establish invasive infection after smaller inocula than needed for other pathogens and to evolve much more rapidly. Thus, although most wound infections do not become clinically evident for 5 days or longer after the operation, streptococcal or clostridial infections may become severe within 24 hours.

Preventive Measures for Surgical Site Infection

TIMING OF ACTION	DETERMINANT IN WHICH THE PREVENTIVE MEASURE ACTS		
	Microorganism	**Local**	**Patient**
Preoperative	Shorten preoperative stay Antiseptic shower	Appropriate preoperative hair removal or no hair removal	Optimize nutrition Preoperative

TIMING OF ACTION	DETERMINANT IN WHICH THE PREVENTIVE MEASURE ACTS		
	Microorganism	**Local**	**Patient**
	preoperatively Appropriate preoperative hair removal or no hair removal Avoid or treat remote site infections Antimicrobial prophylaxis		warming Tight glucose control (insulin drip) Stop smoking
Intraoperative	Asepsis and antisepsis Avoid spillage in gastrointestinal cases	Surgical technique: Hematoma/seroma Good perfusion Complete débridement Dead spaces Monofilament sutures Justified drain use (closed) Limit use of sutures/foreign bodies Delayed primary closure when indicated	Supplemental oxygen Intraoperative warming Adequate fluid resuscitation Tight glucose control (insulin drip)
Postoperative	Protect incision for 48-72 hours Remove drains as soon as possible Avoid postoperative bacteremia	Postoperative dressing for 48-72 hours	Early enteral nutrition Supplemental oxygen Tight glucose control (insulin drip) Surveillance

TIMING OF ACTION	DETERMINANT IN WHICH THE PREVENTIVE MEASURE ACTS		
	Microorganism	**Local**	**Patient**
			programs

Microorganism Related :

Microorganisms causing SSI can be either exogenous or endogenous. Exogenous microorganisms come from the operating team or from the environment around the surgical site (OR, equipment, air, water, and so on). Endogenous microorganisms come either from the bacteria present in the patient at the surgical site or from bacteria present at a different location (e.g., remote site infection, nasal colonization). Two primary measures exist to control the bacterial load in the surgical site: aseptic and antiseptic methods and antimicrobial prophylaxis.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES
MS (General Surgery) Examination, 2008

April, 2008

PAPER III

Time Allowed: 3 Hours **Full Marks:100**

1. Describe the preoperative preparation, operative steps & complications of transverse colostomy. (5+7+8)

Answer.

Indications of Transverse loop colostomy:

- Obstruction of the distal (left) colon or rectum where primary resection and anastomosis of the obstructing lesion is unsafe or impossible.
- To protect a low anastomosis of the rectum.

Preoperative preparation:

In obstructed patients, fluid and electrolyte imbalances should be corrected to the extent possible in an expeditious manner prior to surgery.

- The amount of time available to do this depends on factors such as:
- Degree of dilation of the proximal colon.
- Risk of perforation.

In general, it is desirable to verify the presence of mechanical obstruction and the site of obstruction using CT scanning with rectal contrast, water-soluble contrast enema, or endoscopy.

- A site for the colostomy should be chosen over the rectus sheath and away from all bony prominences to allow for placement of the ostomy base plate.
- Preoperative prophylactic antibiotics**(Cefuroxime + Metrogyl)** are given 30 minutes prior to incision.
- Deep venous thrombosis prophylaxis with sequential compression devices or subcutaneous heparin is employed according to the patient's risk factors.

Procedure

Step 1. The patient is positioned supine and general anesthesia is used to allow for adequate exploration. Draping exposes the area from xiphoid to umbilicus and just lateral to rectus sheath edges.

Step 2. A midline incision can be made or a transverse incision over the right rectus muscle. If the colostomy is intended to be permanent, placement of the stoma in the left transverse colon through a midline or left rectus incision may help to prevent subsequent prolapse. The incision in any case must be large enough to permit safe manipulation and delivery of the dilated colon segment.

Step 3. The dilated transverse colon is identified by following the omentum down to its colonic attachment and the omentum and dilated segment of colon are delivered through the abdominal wall up into the operative field.

Step 4. The omentum is separated from the colon over a short distance by sharp or cautery dissection through the avascular plane and the omentum then transected at right angles to the colon between clamps, allowing it to remain attached to the colon but fall back into the peritoneal cavity out of the way.

Step 5. If the colon is extremely tense or difficult to handle, it is possible to decompress it with an aspirating needle and close the opening with a suture, incorporating the hole into the site of the eventual colostomy.
Step 6. A short segment on the mesenteric border of the colon is cleared to allow passage of a small Penrose drain. The Penrose is then used as a handle to pull up the colon segment. It is important to bring enough colon out of the abdominal cavity to eliminate tension once the bridge has been placed under the loop.
Step 7. The fascia is closed with 0 absorbable suture loosely enough to allow for easy passage of a finger between the colon and the fascia. A few of the epiploic fat attachments and/or the colon serosa are tacked to the fascia with Vicryl suture to prevent herniation.
Step 8. The subcutaneous layer is closed with 3-0 absorbable suture and the skin with 3-0 simple sutures. The skin closure should extend close to the colon but be loose enough to allow passage of a finger around the edge of the wound next to the colon.
Step 9. The Penrose drain is withdrawn while simultaneously replacing it with a supporting rod or bridge. The rod is fastened to the skin with nylon suture. The exact nature of fixation to the skin will vary between different devices.
Step 10. The colon loop is opened along one of the tenia colae for approximately one-third of its length, incorporating any previous decompression site. The edges of the opened bowel are everted and gently approximated to the dermis, except at the point where the rod or bridge passes behind the loop, or the opening may be left alone to evert over time.
Step 11. An opening is fashioned in the ostomy disc just large enough to fit around the ostomy flush against the surface, and the disc is placed on the skin.

Postoperative care:

- The ostomy can be expected to become edematous while the rod is in place.
- The absorbable sutures in the derma should be removed at about 10 days if they have not dissolved.
- The initial size of the opening in the colon should take this into account to allow for function in spite of the edema. The rod or bridge is left in place 6-10 days and then removed, allowing for shrinkage of the stoma and a reduction in the size of the base plate opening.
- The wound and surrounding skin should be checked for any signs of infection as part of the follow-up care.
- A good seal is mandatory each time the bag is changed, although this can be hard to accomplish until the bridge is removed.
- The absorbable sutures in the derma should be removed at about 10 days.

Complications:

Early

- Bleeding may occur from the stoma site.
- Retraction of stoma.
- Necrosis of the stoma may occur if it is brought up under too much tension.

Late

- Either the proximal or distal end of the stoma may prolapse.
- Herniation of small bowel through the incision opening may occur if the fascial closure is not snug enough.

Follow-Up:

- In time, the size of the bag is decreased gradually to fit the smaller stoma.
- The patient should in general be referred to an enterostomal therapist if they did not see one preoperatively.
- Closure of the ostomy is planned when the reason for its initiation is no longer in existence
- Contrast studies through the stoma and through the rectum are performed prior to colostomy closure.

2. Discuss the preventive measure, diagnosis and complications of total thyroidectomy. (5+5+10)

Answer. Postoperative complications:

- ***Haemorrhage*** A tension haematoma deep to the cervical fascia is usually due to reactionary haemorrhage from one of the thyroid arteries; occasionally, haemorrhage from a thyroid remnant or a thyroid vein may be responsible. This is a rare but desperate emergencyIf releasing the tension haematoma does not immediately relieve airway obstruction, the trachea should be intubated at once. An endotracheal tube can be left in place for several days; steroids are given to reduce oedema and a tracheostomy is rarely necessary. Intubation in the presence of laryngeal oedema may be very difficult and should be carried out by an experienced anaesthetist. Repeated unsuccessful attempts may aggravate the problem and, in a crisis, it is safer to perform a needle tracheostomy as a temporary measure; a large bore 12G intravenous cannula (diameter 2.3 mm) is satisfactory.
- ***Recurrent laryngeal nerve paralysis and voice change*** RLN injury may be unilateral or bilateral, transient or permanent. Early routine postoperative laryngoscopy reveals a much higher incidence of transient cord paralysis than is detectable by simple assessment of the integrity of the voice and cough. Such temporary dysfunction is not clinically important, however, but voice and cord function should be assessed at the first follow-up 4 weeks postoperatively.
  Permanent paralysis is rare if the nerve has been identified at operation.
  Injury to the external branch of the superior laryngeal nerve is more common because of its proximity to the superior thyroid artery. This leads to loss of tension in the vocal cord with diminished power and range in the voice. Patients, particularly those who use their voices professionally, must be advised that any thyroid operation will result in change to the voice even in the absence of nerve trauma. Fortunately, for most patients the changes are subtle and only demonstrable on formal voice assessment.
- ***Thyroid insufficiency*** Following subtotal thyroidectomy this usually occurs within 2 years; however, there is a small but progressive annual incidence over many years, which is often insidious and difficult to recognise. The incidence is considerably higher than was previously thought and rates of 20–45% at 10 years have been reported. This results from a change in the autoimmune response, from stimulation to destruction of the thyroid cells. There is a definite relationship between the estimated weight of the thyroid remnant and the development of thyroid failure after subtotal

thyroidectomy for Graves' disease. Thyroid insufficiency is rare after surgery for a toxic adenoma because there is no autoimmune disease present.

- ***Parathyroid insufficiency*** This is due to removal of the parathyroid glands or to infarction through damage to the parathyroid end artery; often, both factors occur together. Vascular injury is probably far more important than inadvertent removal. The incidence of permanent hypoparathyroidism should be less than 1% and most cases present dramatically 2–5 days after operation; however, very rarely the onset is delayed for 2–3 weeks or a patient with marked hypocalcaemia is asymptomatic.
- ***Thyrotoxic crisis (storm)*** This is an acute exacerbation of hyperthyroidism. It occurs if a thyrotoxic patient has been inadequately prepared for thyroidectomy and is now extremely rare. Very rarely, a thyrotoxic patient presents in a crisis and this may follow an unrelated operation. Symptomatic and supportive treatment is for dehydration, hyperpyrexia and restlessness. This requires the administration of intravenous fluids, cooling the requiring urgent decompression by opening the layers of the wound, not simply the skin closure, to relieve tension before urgent transfer to theatre to secure the bleeding vessel. A subcutaneous haematoma or collection of serum may form under the skin flaps and require evacuation in the following 48 hours. This should not be confused with the potentially lifethreatening deep tension haematoma.
- ***Respiratory obstruction*** This is very rarely due to collapse or kinking of the trachea (tracheomalacia). Most cases are caused by laryngeal oedema. The most important cause of laryngeal oedema is a tension haematoma. However, trauma to the larynx by anaesthetic intubation and surgical manipulation are important contributory factors, particularly if the goitre is very vascular, and may cause laryngeal oedema without a tension haematoma. Unilateral or bilateral recurrent nerve paralysis will not cause immediate postoperative respiratory obstruction unless laryngeal oedema is also present but it will aggravate the obstruction. patient with ice packs, the administration of oxygen, diuretics for cardiac failure, digoxin for uncontrolled atrial fibrillation, sedation and intravenous hydrocortisone. Specific treatment is with carbimazole 10–20 mg 6-hourly, Lugol's iodine 10 drops 8-hourly by mouth or sodium iodide 1 g intravenously. Propranolol intravenously (1–2 mg) or orally (40 mg 6-hourly) will block β-adrenergic effects.
- ***Wound infection*** Cellulitis requiring prescription of antibiotics,often by the general practitioner, is more common than most surgeons appreciate. A significant subcutaneous or deep cervical abscess is exceptionally rare and should be drained.
- ***Hypertrophic or keloid scar*** This is more likely to form if the incision overlies the sternum and in dark-skinned individuals. Intradermal injections of corticosteroid should be given at once and repeated monthly if necessary. Scar revision rarely results in significant long-term improvement.
- ***Stitch granuloma*** This may occur with or without sinus formation and is seen after the use of non-absorbable, particularly silk, suture material. Absorbable ligatures and sutures must be used throughout thyroid surgery. Some surgeons use a subcuticular absorbable skin suture rather than the traditional skin clips or staples. Skin staples, if used, can be removed safely in less than 48 hours because the skin closure is supported by the platysma stitch.

Postoperative care: If the voice is normal and the cough occlusive it is not essential to carry out laryngoscopy before leaving hospital. Transient cord palsies are probably more common than generally appreciated. Even if routine cord visualisation is not part of the normal postoperative review, persisting voice change requires visualisation of the cords. Hypocalcaemia depends not only on the identification but also on the preservation of the parathyroid glands with an intact blood supply. In addition, metabolic bone disease, 'hungry bones syndrome', results in the rapid influx of serum calcium into bones, particularly if preoperative preparation has been with β-blockade rather than normalisation of the serum T4. Hypocalcaemia is more common after total than subtotal thyroidectomy and reflects the increased trauma to the parathyroids. About 25% of patients develop transient hypocalcaemia and oral calcium may be necessary (1 g three or four times daily). If associated symptoms are severe and the serum calcium less, 10 ml of 10% calcium gluconate (equivalent to 8.4 mg or 2.3 mmol calcium) should be given. To screen for parathyroid insufficiency the serum calcium should be measured at the first review attendance 4–6 weeks after operation. After subtotal resection, stability in terms of thyroid function takes time. It is important that biochemical (subclinical) thyroid failure should not be an indication for treatment during the first year, as the majority of patients with early subclinical failure, which is common, ultimately regain normality. Even when there are clinical features of failure, thyroxine should be withheld if possible during the first 6 months. Most patients who develop thyroid failure do so within the first 2 years but there is a continuing incidence thereafter. Recurrent thyrotoxicosis may occur atany time after operation and follow-up should therefore be for life. Once a stable situation has been achieved, follow-up after thyroid surgery should be carried out by an automated computerized system, which dramatically reduces the number of patient attendances at the thyroid clinic.

3. Write short notes on: 5 x 6
(a) Circumcision.
(b) Percutaneous tracheostomy.
(c) Repair of meningomyelocoele.
(d) Surgical site infection.
(e) Intra operative radiotherapy.

(a) Circumcision.

Answer. Male circumcision is the removal of some or all of the foreskin (prepuce) from the penis.
Indications: The prevalence of circumcision varies mostly with religious affiliation, and sometimes culture. Most circumcisions are performed during adolescence for cultural or religious reasons; in some countries they are more commonly performed during infancy. Circumcision is also used therapeutically, as one of the treatment options for

Balanitis Xerotica Obliterans, Paraphimosis, Balanitis,	Posthitis, Balanoposthitis, Urinary Tract Infections

Benefits:1 Many older men, who have bladder or prostate gland problems, also develop difficulties with their foreskins due to their surgeon's handling, cleaning, and using instruments. Some of these patients will need circumcising. Afterwards it is often astonishing to find some who have never ever seen their glans (knob) exposed before!

2 Some older men develop cancer of the penis - about 1 in 1000 - fairly rare, but tragic if you or your son are in that small statistic. Infant circumcision gives almost 100% protection, and young adult circumcision also gives a large degree of protection.

3 Cancer of the cervix in women is due to the Human Papilloma Virus. It thrives under and on the foreskin from where it can be transmitted during intercourse. An article in the *British Medical Journal* in April 2002 suggested that at least 20% of cancer of the cervix would be avoided if all men were circumcised. Surely that alone makes it worth doing?

4 Protection against HIV and AIDS. Another *British Medical Journal* article in May 2000 suggested that circumcised men are 8 times less likely to contract the HIV virus. (It is very important here to say that the risk is still far too high and that condoms and safe sex must be used - this applies also to preventing cancer of the cervix in women who have several partners.)

A *BBC* television programme in November 2000 showed two Ugandan tribes across the valley from one another. One practised circumcision and had very little AIDS, whereas, it was common in the other tribe, who then also started circumcising. This programme showed how the infection thrived in the lining of the foreskin, making it much easier to pass on.

5 As with HIV, so some protection exists against other sexually transmitted infections. Accordingly, if a condom splits or comes off, there is some protection for the couple. However, the only safe sex is to stick to one partner or abstain.

6 Lots of men, and their partners, prefer the appearance of their penis after circumcision, It is odour-free, it feels cleaner, and they enjoy better sex. Awareness of a good body image is a very important factor in building self confidence.

7 Balanitis is an unpleasant, often recurring, inflammation of the glans. It is quite common and can be prevented by circumcision.

8 Urinary tract infections sometimes occur in babies and can be quite serious. Circumcision in infancy makes it 10 times less likely.

Immediate Complications

- Infection
  - Infections are usually minor and local, but sometimes they have led to urinary tract infection, life-threatening systemic infections, meningitis or death.

Staphylococcal infections are a growing problem in hospitals for any operation, and MSSA (methicillin susceptible) strains of *s.aureus* have affected neonatal nurseries.

- Herpes

- Hemorrhage: Bleeding after circumcision is usually minor and easily controlled, but on rare occasions it has led to shock from blood loss (hypovolemic shock) or death (exsanguination). Coagulation disorders affect from 2 to 4 per cent of the population and the condition is underdiagnosed. Severe bleeding following circumcision may be a sign of hemophilia.
- Surgical mishap:Mistakes can happen with any surgery. Surgical mistakes from circumcision include documented cases of penile denudation, cutting off part or all of the glans penis, urethral fistula, several types of injury associated with certain types of circumcision clamps usedand penile necrosis which results in loss of the entire penis.
- Anesthetic risk:Anesthetic risk includes Methemoglobinemia.

Delayed Complications

- Meatal stenosis may be a common longer-term complication from circumcision.
- The opening to the urethra (meatus) may also be affected, leading to inflammation and meatal ulceration.
- Urinary retention
- Venous stasis, the slowing down of venous blood flow
- Concealed penis
- Adhesions
- Skin bridges, when the cut skin attaches to the glans penis. Skin bridges do not commonly require surgical correction; rather, a brief, simple office procedure may be performed.
- Painful erections

(b) Percutaneous tracheostomy.

Answer. Background: Percutaneous tracheostomy (PT) has gained an increasing acceptance as an alternative to the conventional surgical tracheostomy (ST). In experienced hands, and with proper patient selection, it is safe, easy and quick. The advantages of PT are that it is a simple, fast, and minimally invasive bedside procedure leading to less stress to the patient compared with surgical tracheostomy (ST).

Advantages of percutaneous tracheostomy over surgical tracheostomy:

- It is a relatively simple technique suitable for trained staff in the critical care setting.
- It does not require an operating theatre and the procedure is usually performed under local anaesthetic, sedation and neuromuscular blockade as appropriate.

- It forms a stoma between tracheal rings, resulting in reduced blood loss as there is usually no disruption of blood vessels. Moreover, the tracheostomy tube is fitted snugly in the stoma thereby minimising any tendency to bleeding after the procedure.
- Infection rates for percutaneous tracheostomy range from 0 to 3.3%, whereas those for open tracheostomy have been reported to be as high as 36%.
- Stenosis rates for percutaneous tracheostomy range from 0 to 9%. The reported incidence of late complications resulting from open tracheostomy such as tracheal stenosis, tracheomalacia, fistula and scarring varies widely.
- Small and neat stoma of dilatational tracheostomy generally results in a more cosmetic scar.

Indications:

- Facilitate weaning from positive pressure ventilation and sedation
- Bypass an obstruction of the upper respiratory tract.
- Prevent aspiration from the pharynx or gastrointestinal tract.
- Facilitate removal of secretion by aspiration.
- Facilitate long-term airway management.

Techniques: Ciaglia's method is the most commonly applied, but no study has shown superiority of any of the percutaneous techniques described. A variant of the original Ciaglia technique using a single tapered dilator known as a "blue rhino" is the most commonly used of these newer techniques and has largely taken over from the early multiple dilator technique.

Complications: Perioperative complications are comparable with those of ST and these are mostly minor. An important advantage of PT over ST is that there is no need to move a critically ill patient to the operating room and the rate of stomal infection is very low. Although data on late complications of PT are not yet sufficient, available reports show a favourable result.

Complications of insertion

Early

- During the procedure, the patient may develop hypoxia due to failure of ventilation. Furthermore, ventilation of the patient may also be difficult if the cuff of the endotracheal tube is inadvertently punctured. If any difficulties are encountered on insertion of the tracheostomy tube, the existing endotracheal tube should be advanced beyond the incision in the trachea and ventilation recommenced until the patient is stable enough to resume the procedure.
- The patient may develop pneumothorax, pneumomediastinum or creation of a false passage and subcutaneous emphysema due to the placement of the tracheostomy tube in the paratracheal space.
- Damage or injury to the posterior tracheal wall may lead to tracheo-oesophageal fistula.

- Major bleeding is unusual. Minor bleeding can usually be controlled by pressure or occasionally a suture. Haemorrhage into the airway is potentially dangerous as it may result in a blood clot obstructing the airway.
- Needle puncture on the lateral wall of trachea may subsequently lead to stenosis.
- Dislodgement of the tracheostomy tube soon after the procedure may be hazardous as the entry to the trachea is small and deep, hence replacement of the tube may be impossible. The percutaneous tracheostomy tube should not be pushed blindly back in but replaced after proper dilation of the track following orotracheal reintubation.
- Secondary haemorrhage may occur either from infection or erosion of vessels.

Late: The incidence of clinically significant subglottic stenosis is low in percutaneous tracheostomy. The reasons behind the development of subglottic stenosis include laryngeal oedema, damage to the tracheal mucosa, high pressure exerted by the endotracheal cuff and prolonged translaryngeal intubation.

(c) Repair of meningomyelocoele.

Answer. Repair of meningomyelocoele : Spina bifida may be associated with a wide range of meningeal and neural herniations that varies from a small outpouching of the meninges to complete rachischisis. Meningoceles and myelomeningoceles (Fig. 1) pre sent with a great variety of locations, forms, sizes, and coverings. Neurological function varies from complete paralysis below the level of the lesion to patients being neurologically intact. The most common location for these congenital anomalies is over the lumbo sacral region. These children may also have many other congenital abnormalities.

In most instances, myelodysplasia should be treated surgically as soon after birth as possible, irrespective of whether the meningocele has ruptured. The goals of surgery are to protect the neural elements, to remove excess skin tissue, and to obtain a watertight dural closure to prevent infection without exacerbating neurological deficits.

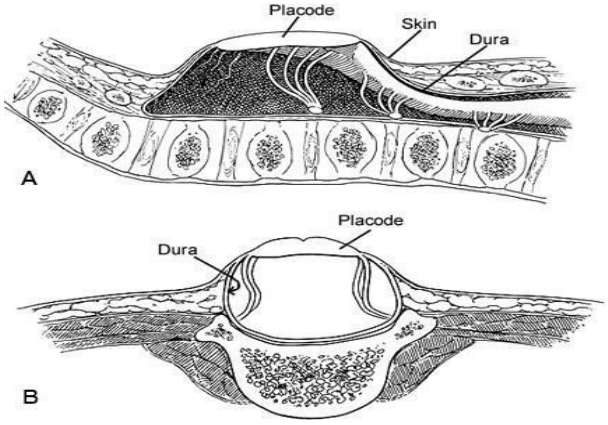


Figure 1. (A) Midsagittal section (B) cross-section of a lumbar myelomeningocele.

For both meningoceles and myelomeningoceles, the surgeon will close the opening in the back.

After birth, the defect is covered by a sterile dressing. Your child may then be transferred to a neonatal intensive care unit (NICU) and cared for by a medical team experienced in caring for children with spina bifida.

Your baby will likely have an MRI (magnetic resonance imagining) or ultrasound of the back. An MRI or ultrasound of the brain may be done to look for hydrocephalus (extra fluid in the brain).

If the myelomeningocele is not covered by skin or a membrane when your child is born, surgery will happen within 24 to 48 hours after birth to prevent infection.

If your child has hydrocephalus, the doctor may put a shunt (plastic tube) in their brain to drain the extra fluid to the stomach. This prevents pressure that could damage the baby's brain. See also: Ventriculoperitoneal shunt

Your child should not be exposed to latex before, during, and after surgery. Many of these children have very bad allergies to latex.

Why the Procedure is Performed

Repair of a meningocele or myelomeningocele is needed to prevent infection and further injury to the child's spinal cord and nerves. Surgery cannot correct the defects in the spinal cord or nerves.

Risks: Risks for any anesthesia are:

- Breathing problems
- Reactions to medications

Risks for any surgery are:

- Bleeding
- Infection

Risks for this surgery are:

- Fluid build up and pressure in the brain (hydrocephalus)
- Increased chance of urinary tract infection and bowel problems
- Infection or inflammation of the spinal cord
- Paralysis, weakness, or sensation changes due to loss of nerve function

Before the Procedure

A health care provider often will find these defects before birth using fetal ultrasound. The doctor will follow the fetus very closely until birth. It is better if you're infant is carried to full term. Your doctor will want to do a cesarean section (C-section). This will prevent further damage to the sac or exposed spinal tissue.

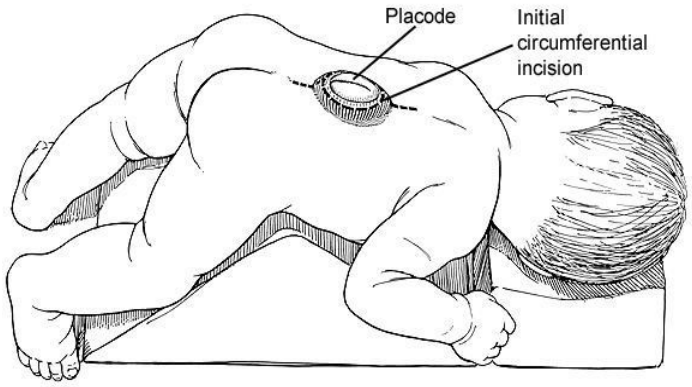


Figure 2. Operative position of the patient. The lumbar area is elevated with respect to the head to reduce the loss of cerebrospinal fluid. The circumferential incision preserves as much healthy skin as possible.

Technique: After general anesthesia has been induced, the baby is placed prone on the operating table with the head slightly lower than the back so that the cerebrospinal fluid will not be replaced by air (Fig. 2). The operative site is cleaned appropriately. The myelomeningocele is irrigated well with warm sterile saline, and the surrounding skin is cleansed with Betadine (Purdue Frederick Co., Norwalk, CT) or Hibiclens (Zeneca Pharmaceuticals, Wilmington, DE). Betadine and Hibiclens are neurotoxic and should not be placed directly on the exposed neural placode. The anus is sealed off from the operative field by proper draping. The drapes are applied with a generous area exposed so that extensive skin flaps can be mobilized.

The skin incision is sketched on the baby's back. Vertical extensions are planned to undermine the skin and to achieve a midline closure, if possible. The skin is incised immediately adjacent to the exposed meninges (Fig. 3). Even if the skin extends well on to the dome of the lesion, it should be preserved. Redundant skin is excised later. The incision is carried down and often into the meningeal sac, initially in an area free of neural elements. The skin edges are retracted laterally. In the wall of the sac, nerve roots that course back into the spinal canal are mobilized. Some neural elements are atretic and terminate in the sac itself and may be sacrificed. The edges of the neural placode are then folded and sutured with interrupted 6-0 monofilament nylon (Fig. 4) to reanimate (restore) the configuration of the spinal cord. Within the spinal canal, the filum terminale can often be identified. It should be cut sharply to release the associated tethering of the spinal cord.

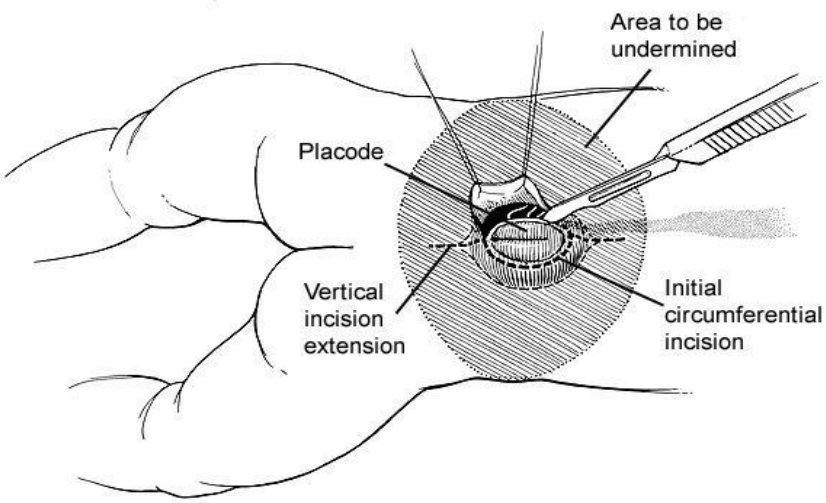


Figure 3. Operative field as viewed by the surgeon. Dashed lines indicate circumferential incision at the edge of the placode and its rostral and caudal extensions. Dotted lines indicate the extent of the undermined skin needed to accomplish closure.

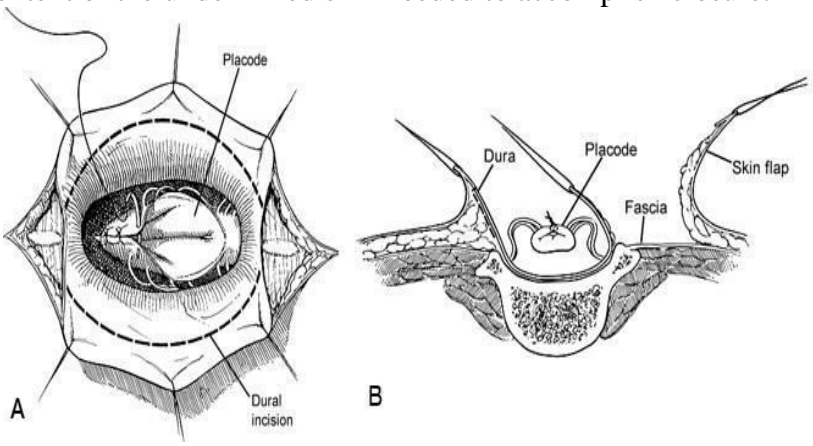


Figure 4. (A) Surgeon's view of the neural placode during closure. Dashed lines indicate dural incision.Figure 4. (B) Cross-section of the placode sutured closed. The dura is incised and mobilized. The skin flap is dissected subcutaneously from the fascia, beginning at the edge of the bony defect.

The dura is then dissected from the subcutaneous tissue and lumbosacral fascia (Fig. 4). A watertight closure is essential and can be verified by the anesthesiologist performing a Valsalva maneuver on the patient. To reinforce the dural suture line, the paravertebral muscles and fascia are mobilized to close in the midline, if possible, and to reestablish their proper dorsal position relative to the vertebral elements (Fig. 5). Alternatively, semilunar flaps of lumbosacral fascia can be swung across the midline and sutured to the base of the opposite side (Fig. 6). The skin margins are undermined to the extent that the wound can be reapproximated without tension, often far out to the lateral flank areas bilaterally. Placement of small subcutaneous drains bilaterally along the flank is optional and helps prevent seromas that might be confused with cerebrospinal fluid leakage from

the spinal column (1 to 2 days of drainage is adequate). A layer of absorbable, undyed suture is used to close the subcutaneous dissection.

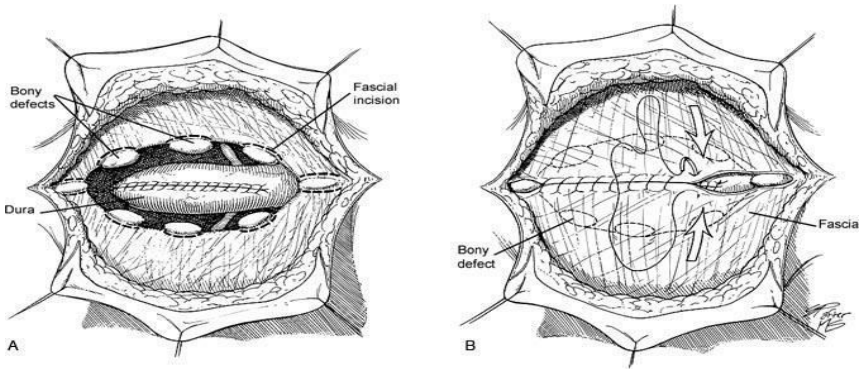


Figure 5. (A) Dural closure and fascial incision (dashed lines). (B) Closure of the fascia and paravertebral muscles along the midline. The bony defect is ghosted below the closure.

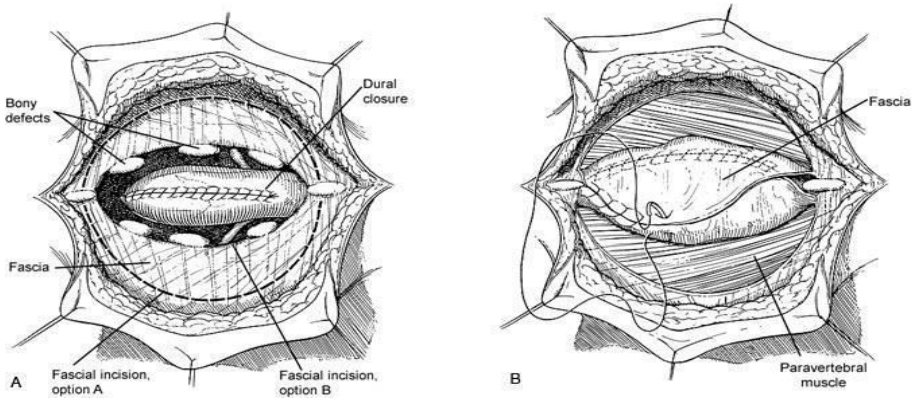


Figure 6. (A) Alternate fascial closure using semilunar flaps. The dashed lines indicate the fascial incision. (B) Each semilunar flap is sutured across the midline to the other fascial flap.

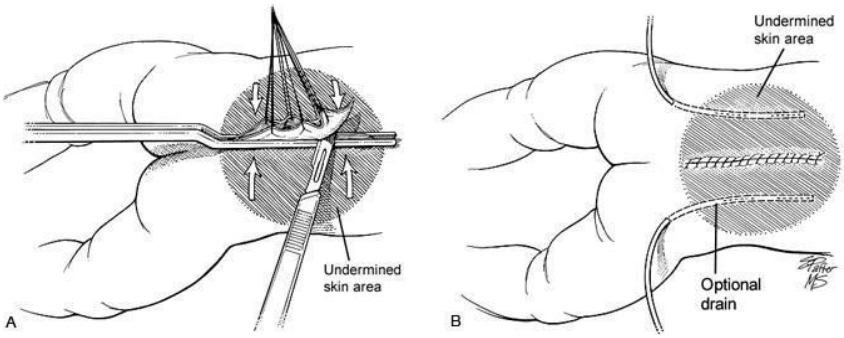


Figure 7. (A) The skin is approximated and trimmed. The subcutaneous undermining is indicated by the shaded area. (B) The skin is closed with a continuous suture. Optional subcutaneous epifascial drains are shown.

Nylon is used to suture the skin and is left in place about 10 days. The dressing must remain dry and the wound isolated from feces. The child is nurtured prone or lateral 3 to 4 days after surgery to avoid undue pressure on the fresh surgical wound and to allow dependent drainage of urine and feces.

After the Procedure:

- Child will usually need to spend about 2 weeks in the hospital after surgery. The child must lay flat without touching the wound area. After surgery, your child will receive antibiotics to prevent infection.
- MRI or ultrasound of the brain is repeated after surgery to see if hydrocephalus develops once the defect in the back is repaired.
- Child may need physical, occupational, and speech therapy. Many children with these problems have gross (large) and fine (small) motor disabilities, and swallowing problems, early in life.
- The child may need to see a team of medical experts in spina bifida often after they are discharged from the hospital.

Outlook (Prognosis)

How well a child does depends on the initial condition of their spinal cord and nerves. After a meningocele repair, children often do very well and have no further brain, nerve, or muscle problems.

Children born with myelomeningocele usually have paralysis or weakness of the muscles below the level of their spine where the defect is. They also may not be able to control their bladder or bowels. They will likely need medical and educational support for many years.

The ability to walk and control bowel and bladder function depends where the birth defect was on the spine. Defects lower down on the spinal cord may have a better outcome.

(d) Surgical site infection.

Answer. See answer. of 4(d) of Paper – III, 2009.

(e) Intra operative radiotherapy.

Answer. Intraoperative Radiation Therapy (IORT)
Intraoperative radiation therapy (IORT) is an intensive radiation treatment that delivers a concentrated beam of radiation to tumors as they are located during surgery. IORT allows direct radiation to the tumor while sparing normal surrounding tissue.

Conditions treated with IORT
IORT is used for cancer that cannot be cured with surgery alone and for tumors that are either attached or close to vital tissues.

IORT is used to treat:

- Recurrent rectal cancer
- Cervical, uterine and ovarian cancers
- Breast cancer
- Prostate, bladder and kidney cancer
- Stomach, pancreatic, colon, rectal and anal cancer
- Soft tissue sarcoma.

For people who have recurring cancer and people who have locally advanced cancers, IORT can be an
effective therapy in achieving long-term tumor control.

How it works
IORT is almost always used along with external radiation prior to surgery. During surgery, after the surgeon removes as much of the tumor as possible, a machine called a linear accelerator delivers a concentrated beam of electron radiation directly to the tumors. The surgeon moves healthy organs out of the radiation field to prevent damage, and special tubes are used to focus the beams safely on the tumour. IORT also may be used when the remaining tumor is too small to see.
Advantages of IORT
IORT allows doctors to administer high doses of radiation to tumors without exposing nearby healthy organs to radiation. A single dose of intraoperative radiation may have as much effect on the tumor as 10 to 20 daily radiation treatments. IORT offers the following benefits:
· Higher radiation dosage to cancer cells
· Faster radiation delivery during surgery
· Decreased radiation exposure to normal surrounding tissue
· Safer application compared with traditional radiation treatments

4. Answer briefly of the following: 4 x 7.5

(a) Investigations for intra abdominal infection.
(b) Management of acute oesophageal injuries due to corrosive ingestion.
(c) Live related liver transplant.
(d) Management of urethral injury.
Answer.
(a) Investigations for intra abdominal infection.

Definition:Infections that spread beyond the hollow viscus of origin into the peritoneal space and are associated with:

- Abscess formation or
- Peritonitis
  - Primary = spontaneous bacterial peritonitis : Arises without a breach in the peritoneal cavity or gi tract
  - Secondary

        - Occurs as a result of spillage of gut organisms through a physical hole in the GI tract or through a necrotic gut wall
        - May be community acquired or healthcare associated
    - Tertiary
        - Peritonitis in a critically ill patient which persists or recurs at least 48 h after apparently adequate management of primary or secondary peritonitis

Microbiology: Abscesses or secondary peritonitis

- Health care associated intra-abdominal infection usually due to nosocomial organisms particular to the site of the operation and specific hospital and unit
- Community acquired infections
    - Infections derived from stomach, duodenum, biliary system and proximal small bowel:
        - Gram positive and gram negative aerobic and facultative bacteria
    - Distal small bowel:
        - Gram negative facultative and aerobic bacteria
        - Anaerobes
    - Large bowel:
        - Facultative and obligate anaerobic bacteria
        - Streptococci and enterococci commonly present

Tertiary peritonitis

- Coagulase negative staphylococci
- *Pseudomonas*
- *Candida*
- Enterococci

Clinical features

- Difficult to diagnose in the critically ill patient because history is usually unobtainable and physical signs usually masked by decreased conscious level
- Consider diagnosis in the appropriate clinical setting in patients with otherwise unexplained signs of sepsis or organ dysfunction:
    - Recent abdominal surgery
    - Source of arterial emboli
    - Peripheral vascular disease
    - Thrombotic disorder
    - Recent arteriography
    - History of reduced splanchnic blood flow (eg use of vasopressors or prolonged shock)
- Unexpected shortness of breath or supraventricular tachycardia occurring 3-4 days after an abdominal operation, new onset renal dysfunction or elevated bilirubin or transaminases should all raise suspicion of intra-abdominal infection

Investigations (answer proper)

Microbiological

- Blood cultures
  - Often negative
  - Polymicrobial or anaerobic bacteraemia should raise possibility of anaerobic infection
- Community acquired infections: gram stain of no value
- Healthcare associated infections: gram stain may be valuable in defining need for specific therapy *s.aureus* or *enterococcus* spp.

Radiological

- Definitive diagnostic approach to intra-abdominal infection

Abdomainal x-ray:

- Look for free gas, bowel obstruction, or subtle signs of intestinal ischaemia
- Water-soluble contrast studies can show leaks
- Injection of contrast into drains, fistulae or sinus tracts may help demonstrate anatomy of complex infectios and help monitor adequacy of abscess drainage

Ultrasound

- Advantage of being portable and almost risk-free
- Useful for:
  - Identifying abscesses and fluid collections
  - Guidance of percutaneous drainage procedures
  - Detection of free fluid
  - Evaluation of biliary tree
- Disadvantages:
  - Operator dependent
  - Difficult to perform in patients who have abdominal dressings or paralytic ileus

Ct abdomen

- With use of iv and oral or rectal contrast most causes of secondary peritonitis can be readily diagnosed
- Requires movement of potentially unstable patient out of icu
- Relative contraindications:
  - Renal dysfunction: contrast may aggravate renal dysfunction
  - Paralytic ileus

- A negative ct generally indicates a very low probability of a process that can be reversed by surgical intervention, however bowel ischaemia cannot be excluded, particularly in the early stages

Invasive investigations in icu

- Judicious probing of surgical wounds with sterile culture swab or gloved finger can often identify collections of infected material immediately adjacent to incision
- Diagnostic peritoneal lavage
    - May reveal bacteria, white cells, bile or intestinal contents
    - Bloody lavage return suggests acute intestinal ischaemia
- Bedside laparoscopy
    - Difficult
    - Experience in critically ill patients largely anecdotal

Management

- Physiological resuscitation
- Systemic antibiotics
- Source control

Antibacterials

Should be administered as soon as infection is suspected and preferably before surgical intervention (to minimize the risk of surgical wound infection.

Choice of antibacterials for community acquired infection

- Should be active against enteric gram negative aerobic and facultative bacilli and ß-lactam susceptible gram positive cocci
- For distal small bowel and colon-derived infections antibacterials should cover anaerobes. Same recommendation also applies to more proximal gi perforations when obstruction is present
- Avoid agents used to treat nosocomial infection in the icu, except for high risk patients
- Inclusion of antibacterials that cover enterococcal infections provides no benefit in terms of outcome for patients with community acquired infections
- Suitable regimes include:
    - Cefazolin or cefuroxime plus metronidazole
    - Metronidazole plus quinolone (eg moxifloxacin or gatifloxacin)
- High risk patients should be given antibacterials with a wider spectrum of activity
    - Risk factors:
        - Higher apache ii
        - Poor nutritional status
        - Significant cardiovascular disease
        - Inability to obtain adequate source control

        - Immunosuppression
    - Suitable regimes include:
        - piperacillin/tazobactam
        - imipenem/cilastin, meropenem
        - 3rd or 4th generation cephalosporin plus metronidazole
        - ciprofloxacin plus metronidazole
        - Aztreonam plus metronidazole

Choice of antibacterials for healthcare associated infection

- More resistant flora routinely encountered
- Organisms seen are similar to those seen in other nosocomial infections
- Treatment should be based on knowledge of local nosocomial flora and their resistance patterns
- Agents which cover enterococci should be used when enterococci are isolated from patients with healthcare associated infections
- Role of antibiotics in tertiary peritonitis is poorly defined
    - Little evidence that they significantly alter outcome
    - Some recommend the use of narrow spectrum agents based on results of culture and sensitivity and avoidance of agents with anti-anaerobic activity
        - There are some data which suggest that use of antibiotics with anti-anaerobic activity increases gut colonization with *candida* and vancomycin resistant *enterococci*.

Duration of therapy

- No more that 24 h for:
    - Bowel injuries due to penetrating, blunt or iatrogenic trauma that are repaired within 12 h
    - Intraoperative contamination of operative field by enteric contents
    - Acute perforations of stomach, duodenum and proximal jejunum in absence of antacid therapy or malignancy
- For patients with established infections:
    - Until resolution of signs of infection occurs. This assessment should be based on signs of sepsis and return of gi function
    - If source control is adequate the role of antibiotics is largely adjuvant and the course can usually be restricted to 5-7 days
    - Further investigation is indicated or patients with persistent or recurrent clinical evidence of intra-abdominal infection after 5-7 days of therapy

Source control

Physical measures to eradicated focus of infection, prevent on-going contamination and ultimately to restore optimal anatomy and function

- Drainage

- Debridement
- Definitive management

Successful source control and antibiotic management is associated with resolution of clinical features of systemic inflammation and reversal of organ dysfunction. Progression or failure of resolution of organ dysfunction suggests persistence of the disease and the need for further intervention

Drainage

- Formation of an abscess isolates infection from surrounding sterile tissues but has disadvantage of preventing influx of host immune cells and antibiotics
- Drainage converts to a controlled sinus or fistula
- Percutaneous ultrasound or ct guided drainage is initial intervention of choice for management of localized, radiologically defined infectious foci
- Can also be used as a temporizing measure eg to decompress infected retroperitoneal collections in patients with necrotizing pancreatitis so operative intervention can be delayed until it is safer
- Indications for surgical drainage:
    - Failure of percutaneous drainage
    - Collections with a significant solid tissue component requiring debridement
    - Simultaneous managment of a source of ongoing contamination
    - When local peritoneal defences have not contained the infectious focus, resulting in generalized peritonitis

Debridement

- In contrast to drainage which removes the liquid component of an infection, debridement is the physical removal of infected or necrotic solid removal
- Debridement less frequently required in patients with intra-abdominal infection
- Main indications in this setting:
    - Intestinal infarction
    - Infected peripancreatic necrosis
- Decision of when to operate relates to relative risks and benefits. Thus the benefit of early excision of necrotic bowel vastly outweighs the risks. In contrast, the bacterial burden in infected retroperitoneal necrosis is lower and the organisms sequestered in the necrotic tissue are less able to gain access to the circulation. In addition early exploration is difficult because of poor demarcation between viable and non viable tissue. As a result there is a trend to delayed rather than immediate intervention.

(b) Management of acute oesophageal injuries due to corrosive ingestion.

Answer. Caustic Injury: Caustic injuries of the esophagus can have devastating consequences, and the best cure for this condition is an ounce of prevention. In

children, ingestion of caustic materials is accidental and tends to be in small quantities. There are both acute and chronic phases to caustic esophageal injuries. The acute phase is dependent on the severity and location of the injury and on the type of substance ingested (acid versus alkali), the form of the substance (liquid versus solid), the quantity and concentration of the substance ingested, the amount of residual food in the stomach, and the duration of tissue contact.

Alkali Ingestion :Alkaline substances dissolve tissues by liquefactive necrosis, deeply penetrating the tissues they touch. There are three phases of tissue injury from alkali ingestion.

1. *Phase 1:* The acute necrotic phase lasts 1 to 4 days after injury, during which coagulation of intracellular proteins results in cell necrosis. The surrounding tissues develop an intense inflammatory reaction.
2. *Phase 2:* The ulceration and granulation phase is next and starts 3 to 5 days after injury and lasts about 3 to 12 days. It is during this time that the tissues slough and granulation tissue begins to fill in the ulcerated base left behind. The esophagus is at its weakest point during this second phase.
3. *Phase 3:* In the third phase, cicatrization and scarring begin, and the newly formed connective tissue begins to contract, resulting in esophageal narrowing. This occurs 3 weeks after the initial injury. Adhesions form between areas of granulation resulting in bands that significantly constrict the esophagus. During this time, efforts are aimed at reducing stricture formation.

Three Phases of Tissue Injury From Alkali Ingestion

PHASE	TISSUE INJURY	ONSET	DURATION	INFLAMMATORY RESPONSE
1	Acute necrosis	1-4 days	1-4 days	Coagulation of intracellular proteins
				Inflammation
2	Ulceration and granulation	3-5 days	3-12 days	Tissue sloughing
				Granulation of ulcerated tissue bed
3	Cicatrization and scarring	3 weeks	1-6 months	Adhesion formation
				Scarring

Acid Ingestion : Ingestion of acid is difficult because it gives an immediate burning in the mouth. When compared with lye ingestions, the quantity and concentrations are quite modest. Acid substances cause coagulative necrosis, forming an eschar that limits tissue penetration. In some cases, acid burns result in full-thickness injury, although in most, it is limited. Within 48 hours, the extent to which the acid will injure the esophagus is

already determined. These injuries tend to be less severe and relatively spare the esophagus over the stomach.

Symptoms and Diagnosis

Symptoms of caustic burns to the esophagus are determined by the severity of the burn and parallel the stages of tissue injury. During phase one, patients may complain of oral and substernal pain, hypersalivation, odynophagia and dysphagia, hematemesis, and vomiting. During stage two, these symptoms may disappear only to see dysphagia reappear as fibrosis and scarring begin to narrow the esophagus throughout stage three. A fever is usually an indicator that esophageal injury is present. Symptoms of respiratory distress, such as hoarseness, stridor, and dyspnea, suggest upper airway edema and are usually worse with acid ingestion. Pain in the back and chest may indicate a perforation of the mediastinal esophagus, whereas abdominal pain may indicate abdominal visceral perforation. Diagnosis is initiated with a physical exam specifically evaluating the mouth, airway, chest, and abdomen. Careful inspection of the lips, palate, pharynx, and larynx is done. Auscultation of the lungs is critical to determining the degree of upper airway involvement. The abdomen is examined for signs of perforation. Early endoscopy is recommended 12 to 24 hours after ingestion to identify the grade of the burn. Radiographic examination in adults is not a useful tool at initial presentation but is helpful in later stages to assess stricture formation. Serial chest and abdominal radiographs are indicated to follow patients with questionable chest and abdominal exams. A CT scan is indicated in a patient with an equivocal endoscopic exam in whom there is a strong index of suspicion for a perforation.

Endoscopic Grading and Treatment of Corrosive Esophageal and Gastric Burns

DEGREE OF BURN	ENDOSCOPIC EVALUATION	TREATMENT
First degree	Mucosal hyperemia	48-hr observation
	Edema	Acid suppression
Second degree	Limited hemorrhage	Aggressive IV resuscitation
	Exudates	IV antibiotics
	Ulceration	Acid suppression
	Pseudomembrane formation	
Third degree	Mucosal sloughing	Inhaled steroids
	Deep ulcerations	Fiberopticintubation(ifneeded)
	Massive hemorrhage	
	Complete luminal obstruction	
	Charring	
	Perforation	

Treatment

The treatment of caustic lesions of the esophagus is determined by the extent of the injury and addresses the injuries that occur both in the acute and chronic phase.

Acute Phase: Management of the acute phase is aimed at limiting and identifying the extent of the injury. It begins with neutralization of the ingested substance. If a patient presents within the first hour of ingestion, neutralization is attempted. Alkalis (including lye) are neutralized with half-strength vinegar or citrus juice. Acids are neutralized with milk, egg whites, or antacids. Emetics and sodium bicarbonate need to be avoided. Further treatment is guided by the extent of injury endoscopically identified and the patient's underlying condition.

No Evidence of Burn

Early observation is safe in those asymptomatic patients whose physical exam and initial endoscopy are negative. Oral nutrition may be resumed when a patient can painlessly swallow saliva.

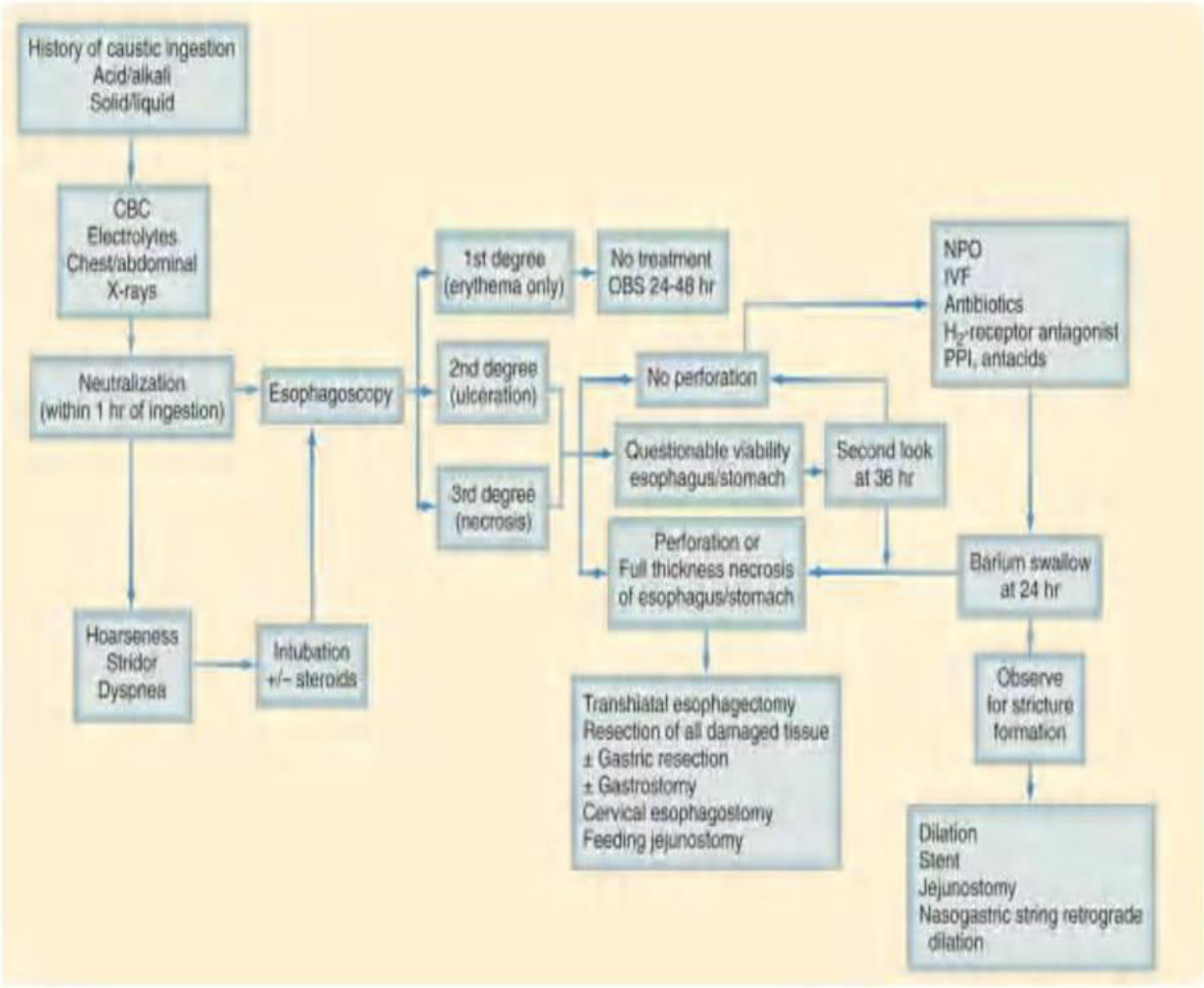
First-Degree Burn : In patients with endoscopically identified first-degree burns, 48 hours of observation is indicated. Oral nutrition can be resumed when a patient can painlessly swallow saliva. A repeat endoscopy and barium esophagram are done in follow-up at intervals of 1, 2, and 8 months, at which time 60%, 80%, and nearly 100% of strictures will have developed, respectively.

Second- and Third-Degree Burns : Patients with second- and third-degree burns to the esophagus are triaged similar to burn patients. Massive fluid shifts, renal failure, and sepsis can occur rapidly, and underestimation of extent of injury can lead to fatal outcomes. Resuscitation is aggressively pursued. The patient is monitored in the intensive care unit (ICU) and kept nil per oral (NPO) with IV fluids. IV antibiotics and a proton pump inhibitor are started. In patients with evidence of acute airway involvement, aerosolized steroids may be used to relieve airway obstruction. Fiberoptic intubation may be needed and must be available. The use of steroids to prevent stricture formation is controversial.

The management of second- and third-degree burns of the esophagus is multifaceted and has several acceptable options. Aggressive resuscitation and placement of an esophageal stent are one option. Oral nutrition is resumed when a patient can painlessly swallow saliva. Alternatively, a feeding tube or central venous catheter is placed, and the patient is kept NPO until oral pain subsides. If the diagnosis is not secured with endoscopy, an exploratory laparoscopy (in stable patients) or laparotomy (in unstable patients) is performed. A viable stomach and esophagus are left in situ, a feeding jejunostomy tube is placed, and an esophageal stent is placed endoscopically in the operating room. A questionable esophagus and stomach are left in situ, and a second-look operation is planned for 36 hours. Management at 36 hours is dictated by the findings at that time. If full-thickness necrosis or perforation of the esophagus or stomach is found at any time, an emergent exploratory laparotomy is indicated. The esophagus and stomach and all affected surrounding organs and tissues are resected, an end-cervical esophagostomy is

performed, and a feeding jejunostomy is placed. Postoperatively, the patient is monitored in an ICU and aggressively managed.

Management of caustic injury of the esophagus: acute phase. IVF, intravenous fluids; OBS, observe; PPI, proton pump inhibitor:



(c) Live related liver transplant.

Answer. Liver transplantation or **hepatic transplantation** is the replacement of a diseased liver with a healthy liver allograft. The most commonly used technique is orthotopic transplantation, in which the native liver is removed and replaced by the donor organ in the same anatomic location as the original liver. Liver transplantation nowadays is a well accepted treatment option for end-stage liver disease and acute liver failure. It is also one of the most expensive treatments in modern medicine. Typically 3 surgeons and 1 anesthesiologist are involved, with up to 4 supporting nurses.

The first human liver transplant was performed in 1963 by a surgical team led by Dr. Thomas Starzl of Denver, Colorado, United States.

Living donor liver transplantation (LDLT) has emerged in recent decades as a critical surgical option for patients with end stage liver disease, such as cirrhosis and/or hepatocellular carcinoma often attributable to one or more of the following: long-term alcohol abuse, long-term untreated hepatitis C infection, long-term untreated hepatitis B infection. The concept of LDLT is based on (1) the remarkable regenerative capacities of the human liver and (2) the widespread shortage of cadaveric livers for patients awaiting transplant. In LDLT, a piece of healthy liver is surgically removed from a living person and transplanted into a recipient, immediately after the recipient's diseased liver has been entirely removed.

Historically, LDLT began as a means for parents of children with severe liver disease to donate a portion of their healthy liver to replace their child's entire damaged liver. The first report of successful LDLT was by Dr. Christoph Broelsch at the University of Chicago Medical Center in November 1989, when two-year-old Alyssa Smith received a portion of her mother's liver. Surgeons eventually realized that adult-to-adult LDLT was also possible, and now the practice is common in a few reputable medical institutes. It is considered more technically demanding than even standard, cadaveric donor liver transplantation, and also poses the ethical problems underlying the indication of a major surgical operation (hepatectomy) on a healthy human being. In various case series, the risk of complications in the donor is around 10%, and very occasionally a second operation is needed. Common problems are biliary fistula, gastric stasis and infections; they are more common after removal of the right lobe of the liver. Death after LDLT has been reported at 0% (Japan), 0.3% (USA) and <1% (Europe), with risks likely to decrease further as surgeons gain more experience in this procedure.

In a typical adult recipient LDLT, 55 to 70% of the liver (the right lobe) is removed from a healthy living donor. The donor's liver will regenerate approaching 100% function within 4–6 weeks, and will almost reach full volumetric size with recapitulation of the normal structure soon thereafter. It may be possible to remove up to 70% of the liver from a healthy living donor without harm in most cases. The transplanted portion will reach full function and the appropriate size in the recipient as well, although it will take longer than for the donor.

Living donors are faced with risks and/or complications after the surgery. Blood clots and biliary problems have the possibility of arising in the donor post-op, but these issues are remedied fairly easily. Although death is a risk that a living donor must be willing to accept prior to the surgery, the mortality rate of living donors in the United States is low. The LDLT donor's immune system does diminish as a result of the liver regenerating, so certain foods which would normally cause an upset stomach could cause serious illness.

Liver donor requirements

Any member of the family, parent, sibling, child, spouse or a volunteer can donate their liver. The criteria for a liver donation include:

- Being in good health
- Having a blood type that matches or is compatible with the recipient's

- Having a charitable desire of donation without financial motivation
- Being between 18 and 60 years old
- Being of similar or bigger size than the recipient
- Before one becomes a living donor, the donor must undergo testing to ensure that the individual is physically fit. Sometimes CT scans or MRIs are done to image the liver. In most cases, the work up is done in 2–3 weeks

Complications: Living donor surgery is done at a major center. Very few individuals require any blood transfusions during or after surgery. Even though the procedure is very safe, all potential donors should know there is a 0.5 to 1.0 percent chance of death. Other risks of donating a liver include bleeding, infection, painful incision, possibility of blood clots and a prolonged recovery. The vast majority of donors enjoy complete and full recovery within 2–3 months.

Pediatric transplantation: In children, living liver donor transplantations have become very accepted. The accessibility of adult parents who want to donate a piece of the liver for their children/infants has reduced the number of children who would have otherwise died waiting for a transplant. Having a parent as a donor also has made it a lot easier for children - because both patients are in the same hospital and can help boost each other's morale.

Benefits: There are several advantages of living liver donor transplantation over cadaveric donor transplantation, including:

- Transplant can be done on an elective basis because the donor is readily available
- There are fewer possibilities for complications and death while waiting for a cadaveric organ donor.
- Because of donor shortages, UNOS has limited the cadaveric organ allocation to foreigners who seek medical help in the USA. However, with the availability of living donor transplantation, this will now allow foreigners a new opportunity to seek medical care in the USA

Screening for donors:

Living donor transplantation is a multidisciplinary approach. All living liver donors undergo medical evaluation. Every hospital which performs transplant have dedicated nurses that provide specific information about the procedure and answer questions that families may have. During the evaluation process, confidentially is assured on the potential donor. Every effort is made to ensure that organ donation is not made by coercion from other family members. The transplant team provides both the donor and family thorough counseling and support which continues until full recovery is made.

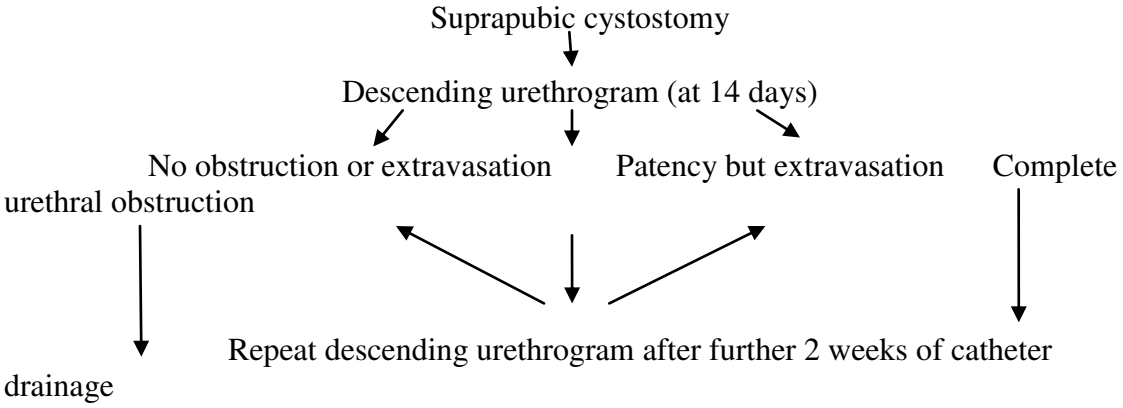
All donors are assessed medically to ensure that they can undergo the surgery. Blood type of the donor and recipient must be compatible but not always identical. Other things assessed prior to surgery include the anatomy of the donor liver. However, even with mild variations in blood vessels and bile duct, surgeons today are able to perform transplantation without problems. The most important criterion for a living liver donor is to be in excellent health.[13]

Economic aspect

Typical expenses during the first year (everything included from surgey, hospitalization, lab testing, medications) are up to $315,000, excluding insurance or government assistance. The cost is considerably lower in countries like India where a living donor liver transplant typically costs about $50,000/-. The high volume of such procedures in a few centers in India results in excellent outcomes comparable to the best centers in the world

(d) Management of urethral injury.

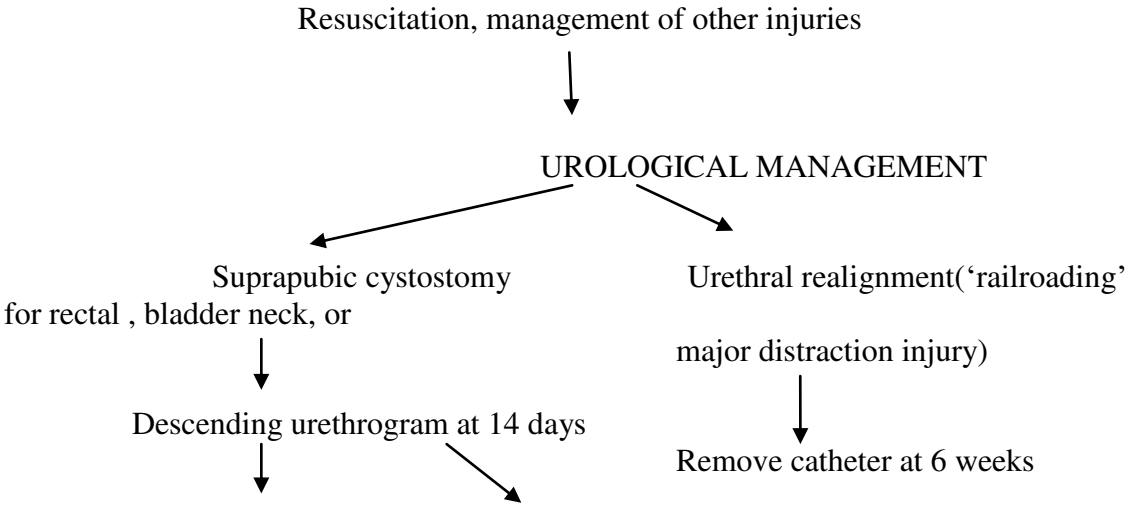
Answer. Management of bulbar urethral injury:

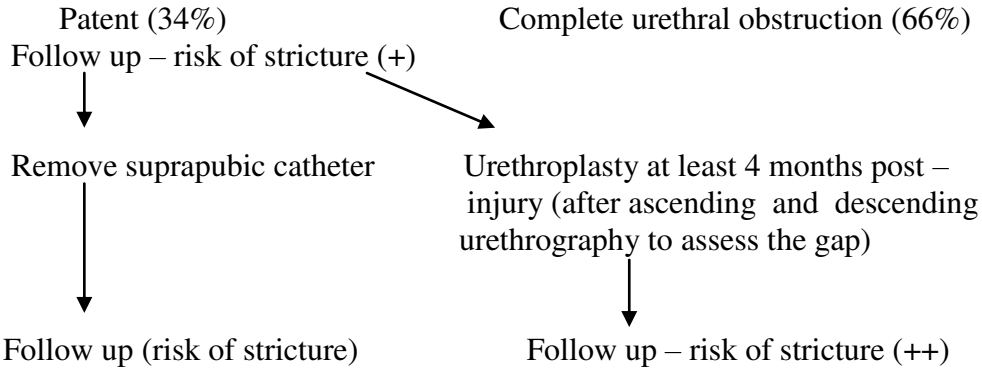


Remove suprapubic catheter and follow up (risk of stricture End – to – end urethroplasty 6 weeks post-trauma (after ascending & descendingurethrogram to assess the gap)

Follow up (risk of stricture +)

Management of membranous urethral injury:





Principles of end – to – end urethroplasty:

- Adequate perineal exposure.
- Freeing of corpus spongiosum proximal and distal to injury for tension free anastomosis.
- Excision back to normal urethra.
- Spatulation of urethral ends.
- Anastomosis with interrupted sutures.
- Transanastomotis silastic catheter plus suprapubic cystostomy.
- Perineal drain.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES
MS (General Surgery) Examination, 2007

April, 2007

PAPER III

Time Allowed: 3 Hours **Full Marks: 100**

1. Classify tumors of the kidney. Discuss the management of a case of renal cell carcinoma with particular reference to different surgical approaches and their steps appropriate for such a patient. (20)

Answer. WHO Classification of kidney tumours:

Familial renal cancer
- Renal cell tumors
  - Malignant
    - Clear cell renal cell carcinoma
    - Multilocular clear cell renal cell carcinoma
    - Papillary renal cell carcinoma
    - Chromophobe renal cell carcinoma
    - Carcinoma of the collecting ducts of Bellini
    - Renal medulary carcinoma
    - Xp11 translocation carcinomas
    - Carcinoma associated with neuroblastoma
    - Mucinous tubular and spindle cell carcinoma
    - Renal cell carcinoma unclassified
  - Benign
    - Papillary adenoma
    - Oncocytoma
- Metanephric tumors
  - Metanephric adenoma
  - Metanephric adenofibroma
  - Metanephric stromal tumors
- Mixed mesenchymal and epithelial tumors
  - Cystic nephroma
  - Mixed epithelial and stromal tumor
  - Synovial sarcoma
- Nephroblastic tumors
  - Nephrogenic rests
  - Nephroblastoma
  - Cystic partially differentiated nephroblastoma
- Neuroendocrine tumors
  - Carcinoid
  - Neuroendocrine carcinoma

Primitive neuroectodermal tumor
Neuroblastoma
Phaeochromocytoma

Other tumors
Mesenchymal tumors
Haematopoietic and lymphoid tumors
Germ cell tumors
Metastatic tumors

Paediatric patients – Wilm's tumour.

Laboratory Findings

- Microscopic urinalysis reveals hematuria in most patients.
- The erythrocyte sedimentation rate may be elevated but is nonspecific.
- Elevation of the hematocrit and levels of serum calcium, alkaline phosphatase, and aminotransferases occur in less than 10% of patients.
- These findings nearly always resolve with curative nephrectomy and thus are not usually signs of metastases.
- Anemia unrelated to blood loss occurs in 20% to 40% of patients, particularly those with advanced disease.

Imaging Studies

- The diagnosis of renal cell carcinoma is often made by CT (and, less frequently, by intravenous urography) performed as an initial step in the workup of hematuria, an enigmatic metastatic lesion, or suspicious laboratory findings.
- Ultrasonography and CT scan often reveal incidental renal masses, which now account for 50% of the initial diagnoses of renal cancer in patients without manifestations of renal disease.
- Plain abdominal x-rays may reveal a calcified renal mass, but only 20% of renal masses contain demonstrable calcification. (Twenty percent of masses with peripheral calcification are malignant; over 80% with central calcification are malignant.)
- The initial technique for workup of hematuria is currently CT urography; intravenous urography alone defines only 75% of renal mass lesions.
- Differentiation of the most common renal mass (ie, a simple benign cyst) can be made by the finding of a radiolucent center with a thin wall and a sharp interface between the mass and the renal cortex (the typical "beak sign" of a cortical cyst).

Ultrasonography

Further definition of all renal masses seen on intravenous urography is required. Occasionally, some masses detected on CT require further characterization by ultrasound. Abdominal ultrasound can also identify a vena caval tumor thrombus and its cephalad extent in the cava.

Isotope Scanning

Occasionally, a renal mass is suspected on intravenous urography but is equivocal or not seen on ultrasound. In these cases, a renal cortical isotope scanning agent such as technetium-99m DMSA is helpful. Isotope scans of a renal tumor or cyst show an area of decreased uptake, whereas an area of increased uptake indicates a renal "pseudotumor" or a hypertrophied column of Bertin.

CT Scan

CT scan is the diagnostic procedure of choice when a solid renal mass is noted on ultrasound. CT scan accurately delineates renal cell carcinoma in over 95% of cases. Over 80% of tumors are enhanced by iodinated contrast medium, reflecting their high vascularity.

CT scan is also helpful in local staging and can reveal tumor penetration of perinephric fat; enlargement of local hilar lymph nodes, indicating metastases; or tumor thrombi in the renal vein or inferior vena cava. CT angiography can delineate the renal vasculature, which is helpful in surgical planning for partial nephrectomies.

MRI

MRI is not more accurate than CT and is much more expensive. It is, however, the most accurate noninvasive means of detecting renal vein or vena caval thrombi. With the further refinement of pulse sequencing and the use of paramagnetic contrast agents, MRI has become one of the primary techniques for staging solid renal masses. Magnetic resonance angiography (MRA) has become particularly useful for mapping the blood supply and the relationship to adjacent structures in candidates for partial nephrectomy.

Other Diagnostic or Staging Techniques

Isotopic bone scanning is useful in patients with bone pain, elevated alkaline phosphatase, or known metastases. Chest x-ray is sufficient if negative, but if equivocal, then CT scan of the chest can be used to detect metastases. There are currently no tumor markers specific for renal cell carcinoma. Occasionally, aspiration cytology of the mass can be useful in an enigmatic case. Previously, such procedures were discouraged because of fear of disseminating the tumor along the needle tract, but this has proved to be rare, and the technique is safe. The diagnosis is most often made by noninvasive means, and needle aspiration is required only in indeterminate cases (< 10%).

Treatment

Staging is the key to designing the treatment plan. Patients with disease confined within the renal fascia (Gerota's fascia) or limited to nonadherent renal vein or vena caval tumor thrombi (stages T1, T2, and T3a) are best treated by radical nephrectomy. This involves en bloc removal of the kidney and surrounding Gerota fascia (including the ipsilateral adrenal), the renal hilar lymph nodes, and the proximal half of the ureter. Para-aortic node dissection has not been proven beneficial and is not routinely performed. In patients

with very large tumors and a normal contralateral kidney, radical nephrectomy is recommended. Recent reports have advocated including the ipsilateral adrenal gland in the resection only in cases where the mass is large or involving the upper pole of the kidney. Patients with tumors in solitary kidneys, those with diabetes mellitus or renal insufficiency, and those with tumors under 4 cm (even with a normal opposite kidney) should be considered for partial nephrectomy because the prognosis in such cases (if negative surgical margins are obtained) is the same as that of radical nephrectomy. Laparoscopic radical or partial nephrectomy has been advocated as a method equal to the open approach with the advantages of less blood loss, shorter hospitalization, and earlier return to normal function. It is the gold standard in institutions with appropriate expertise. Laparoscopic or percutaneous cryoablation of renal cancer has also shown considerable promise. Alternatively, radiofrequency ablation has been utilized for small renal tumors, but this procedure requires more definitive study and longer follow-up in treated patients.

TNM Staging Classification and Prognosis of Renal Cell Cancer.

Robson Stage	**T**	**N**	**M**	**5-Year Survival (%)**
I. Tumor confined by renal capsule	T1 (< 7.0 cm tumor)	N0 (nodes negative)	M0 (no distant metastases)	80–100
	T2 (> 7.0 cm tumor)			
II. Tumor extension to perirenal fat or ipsilateral adrenal but confined by Gerota's fascia	T3a	N0	M0	50–60
IIIa. Renal vein or inferior vena cava involvement	T3b (renal vein involvement)	N0	M0	50–60 (renal vein)
	T3c (renal vein and caval involvement below the diaphragm)			25–35 (vena cava)
	T4b (caval involvement above the diaphragm)			
IIIb. Lymphatic involvement	T1–3	N1 (single regional node involved)	M0	15–35
		N2 (multiple regional		

		contralateral, or bilateral nodes involved)		
IIIc. Combination of IIIa and IIIb	T3–4	N1–2	M0	15–35
IVa. Spread to contiguous organs except ipsilateral adrenal	T4	N1–2	M0	0–5
IVb. Distant metastases	T1–4	N0–2	M1	0–5

- Nephrectomy has not been associated with improved survival rates in patients with multiple distant metastases (stage IV), and the procedure is not recommended unless patients are symptomatic or a promising therapeutic protocol is being studied.
- Patients with solitary pulmonary metastases have benefited from joint surgical removal of both the primary lesion and the metastatic lesion (30% survival at 5 years).
- Preoperative arterial embolization in patients with or without metastases does not improve survival rates, though it may be helpful as a single treatment measure in patients with symptomatic but nonresectable primary lesions.
- Radiation therapy is of little benefit except as treatment for symptomatic bone metastases.
- Medroxyprogesterone for metastatic renal cell carcinoma has given an equivocal 5% to 10% response rate of short duration.
- Vinblastine has also had a response rate of approximately 20%, again of minimal duration. There are no other cytotoxic chemotherapeutic agents of benefit.
- Immunotherapy with interferon alfa has had a 15% to 20% response rate. Other interferons, alone (interferon beta, interferon gamma) or in combination with chemotherapeutic agents, have been less effective than interferon alfa.
- Adoptive immunotherapy—using lymphocytes (lymphokine-activated killer cells) from exposure of the patient's own peripheral blood lymphocytes to interleukin-2 (IL-2) in vitro followed by reinfusion into the patient along with systemic High-dose intravenous IL-2 causes a profound capillary leak syndrome and substantial toxicity.

  - Recent advances in research on the von Hippel-Lindau tumor suppressor gene has led to identification of growth factors including vascular endothelial growth factor (VEGF) and platelet-derived growth factor as molecular targets in treating advanced renal cancer.
  - Initial studies using bevacizumab, an anti–vascular endothelial growth factor antibody, have shown promising results.
  - Sorafenib, a tyrosine kinase inhibitor that blocks the pathway leading to the production of several growth factors, has been studied in patients with metastatic renal cancer and shown longer median progression-free survival than placebo (24 weeks vs 6 weeks).

- ❖ Sunitinib, another tyrosine kinase inhibitor, has shown longer progression-free survival and higher response rates than interferon alfa in patients with metastatic renal cancer. These oral agents are currently used as first-line therapy in this group of patients.
- ❖ Temsirolimus is another targeted agent that is a specific inhibitor of the mammalian target of rapamycin kinase (mTOR inhibitor) and has shown promising results. It is now used as first-line therapy in poor prognosis patients. Many other agents are currently being studied.

Prognosis

Patients with localized renal cancer (stages T1, T2, and T3a) treated surgically have 5-year survival rates of approximately 70% to 80%, whereas rates for those with local nodal extension or distant metastases are 15% to 25% and less than 10%, respectively. Most patients who present with multiple distant metastases succumb to disease within 15 months . The advent of new agents for renal cancer may improve the outcome in these patients.

Different surgical approaches:

Most common operation for renal cell carcinoma is nephrectomy.

- ➢ The transperitoneal abdominal approach is usually considered superior in renal malignancy for the better access it gives to the great vessels.
- ➢ For very large upper renal tumours, or for retroperitoneal sarcomas, a thoracoabdominal approach, with or without liver mobilization provides very extensive access.
- ➢ Alternatively retroperitoneal loin approach may also be preferable.
- ➢ Nephrectomy may involve removing either an entire organ and surrounding tissue or a small part of the kidney.

    - Partial Nephrectomy: In partial nephrectomy, which is also referred to as kidney-sparing surgery, only the diseased or infected portion of the kidney is removed. The healthy kidney tissue is left in place as much as possible.
    - Radical Nephrectomy: Radical nephrectomy involves the removal of the entire kidney, the adrenal gland, the ureter and the fatty tissue that surrounds the kidney.
    - Simple Nephrectomy:Simple Nephrectomy is done for a living donor transplant purposes that requires the removal of the kidney and a section of the attached ureter.
    - Open Nephrectomy: This is the traditional surgery and the donor or patient may be under general anesthesia. A 10 to 20-inch incision is done in the side or abdomen and a lower rib may be removed.
    - Laparoscopic Nephrectomy: This is a minimally invasive surgery.This type of nephrectomy may last longer than the open nephrectomy, but studies have shown that it promotes faster recovery time and less post-operative pain.

Nephrectomy:

- Abdomen is opened through midline incision.
- The gonodal vein runs superiorly between the transversalis fascia and psoas major. The ureter runs parallel with the gonodal vein, but straight to the hilum of the kidney. It can be seen running superiorly inside the renal fascia, on the surface of paranephric fat.

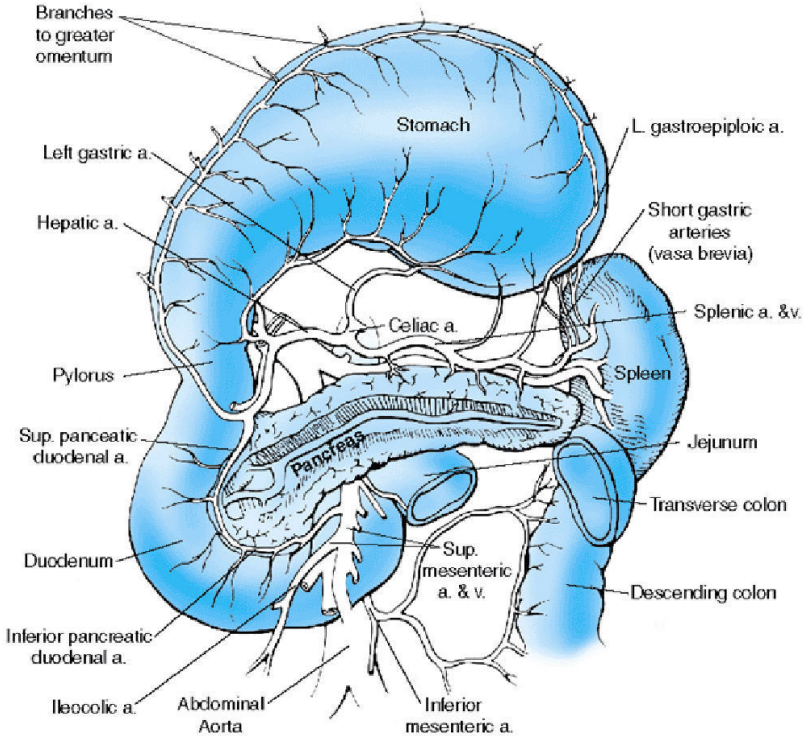
Step 1 – Identify the gonodal vein and the ureter. Visually identify the gonodal vein running inferiorly on psoas major. Visually identify the ureter inferiorly of the inferior pole and verify that it is the ureter by prodding it with the ultrasonic dissector and observing wormlike movement.
Step 2 – Free the ureter.
Step 3 – Lligate and cut the ureter. Dissect the hilum. The kidney is connected to the vena cava and the abdominal aorta by the renal vein and renal artery. These vessels runs medially inside the medial perinephric fat and attach to the kidney at the hilum.
Clear vessel, push the kidney anterior keep fatty tissue in the hilum under tension.
Dissect surface layer of tense fatty tissue in hilum region using the ultrasonic dissector until vessel structure becomes apparent in tissue. Ligate and cut vessel.
Freeing the superior pole and peritoneum:The renal hilum is now dissected; the kidney is still connected by binding tissue to the diaphragm, the abdominal wall and the peritoneum.
Freeing the superior pole.
Freeing part of the peritoneum.
Take out the kidney.

2. Discuss the blood supply & lymphatic drainage of stomach & outline rationale of D2 gastrectomy in carcinoma stomach. Mention different types of reconstructions following such procedure. (20)

Answer. Arterial supply and Venous drainage of stomach:

The stomach is the most richly vascularized portion of the alimentary canal. Both the quantity of blood delivered to the stomach and the richness of the intramural gastric vascular anastomotic network are impressive. The large majority of the gastric blood supply is from the celiac axis via four named arteries. The left and right gastric arteries form an anastomotic arcade along the lesser curvature, and the right and left gastroepiploic arteries form an arcade along the greater gastric curvature. The consistently largest artery to the stomach is the left gastric artery, which usually arises directly from the celiac trunk and divides into an ascending and descending branch along the lesser gastric curvature. Approximately 15% of the time, the left gastric artery supplies an aberrant vessel which travels in the gastrohepatic ligament (lesser omentum) to the left side of the liver. Rarely, this is the only arterial blood supply to this part of the liver, and inadvertent ligation may lead to clinically significant hepatic ischemia. The second largest artery to the stomach is usually the right gastroepiploic artery, which arises fairly consistently from the gastroduodenal artery behind the first portion of the

duodenum. The left gastroepiploic artery arises from the splenic artery, and together with the right gastroepiploic artery, forms the rich gastroepiploic arcade along the greater curvature. The right gastric artery usually arises from the hepatic artery near the pylorus and hepatoduodenal ligament, and runs proximally along the distal stomach. In the fundus along the proximal greater curvature, the short gastric arteries and veins arise from the splenic circulation. There also may be vascular branches to the proximal stomach from the phrenic circulation.

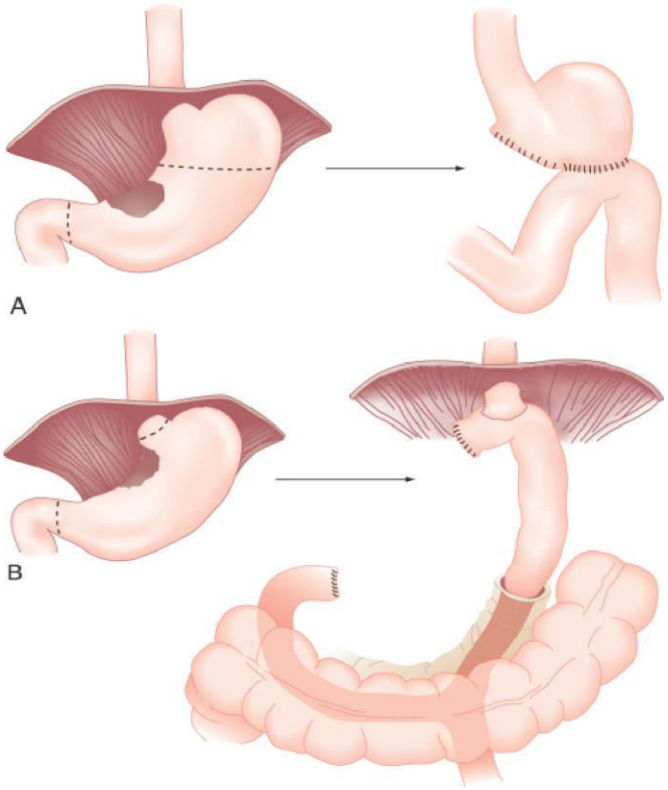


See Q. No 1 of Paper III of May, 2010 for lymphatic drainage of stomach and also for rationale of D2 gastrectomy.

Different reconstruction after D2 gastrectomy:

Subtotal gastrectomy , Billroth Ⅰ	Procedure: digestive tract reconstruction Subtotal gastrectomy with Billroth Ⅰ or Billroth Ⅱ, total gastrectomy with jejunal interposition or Roux-en-Y reconstruction
Subtotal gastrectomy , Billroth Ⅱ	Procedure: digestive tract reconstruction Subtotal gastrectomy with Billroth Ⅰ or Billroth Ⅱ, total gastrectomy with jejunal interposition or Roux-en-Y reconstruction

total gastrectomy, jejunal interposition	Procedure: digestive tract reconstruction Subtotal gastrectomy with Billroth Ⅰ or Billroth Ⅱ, total gastrectomy with jejunal interposition or Roux-en-Y reconstruction
total gastrectomy , Roux-en-Y	Procedure: digestive tract reconstruction Subtotal gastrectomy with Billroth Ⅰ or Billroth Ⅱ, total gastrectomy with jejunal interposition or Roux-en-Y reconstruction



A, Subtotal gastrectomy with a Billroth II anastomosis. **B,** Total gastrectomy with a Roux-en-Y anastomosis.

3. Write short notes on: 5 x 6
(a) Subdural haematoma.
(b) Haemopneumothorax.
(c) Extra anatomic by pass for leg ischaemia.
(d) Management of cardiac arrest during surgery.

(e) Flaps used in surgery.

(a) Subdural haematoma.

Answer. A subdural hematoma or subdural haematoma (British spelling), also known as a subdural hemorrhage (SDH), is a type ofhematoma, a form of traumatic brain injury. Blood gathers within the outermost meningeal layer, between the dura mater, which adheres to the skull, and the arachnoid mater, which envelops the brain. Usually resulting from tears in bridging veins which cross the subdural space, subdural hemorrhages may cause an increase in intracranial pressure (ICP), which can cause compression of and damage to delicate brain tissue. Subdural hematomas are often life-threatening when acute. Chronic subdural hematomas, however, have better prognosis if properly managed.

Classification: Subdural hematomas are divided into acute, subacute, and chronic, depending on their speed of onset.

- **Acute subdural hematomas** that are due to trauma are the most lethal of all head injuries and have a high mortality rate if they are not rapidly treated with surgical decompression.
- Though much faster than chronic subdural bleeds, acute subdural bleeding is usually venous and therefore slower than the usually arterial bleeding of an epidural hemorrhage.
- The mortality rate associated with acute subdural hematoma is around 60 to 80%.
- **Chronic subdural bleeds** develop over the period of days to weeks, often after minor head trauma, though such a cause is not identifiable in 50% of patients.
- They may not be discovered until they present clinically months or years after a head injury. The bleeding from a chronic bleed is slow, probably from repeated minor bleeds, and usually stops by itself. Since these bleeds progress slowly, they present the chance of being stopped before they cause significant damage.

Signs and symptoms:

- Chronic subdural hematomas are common in the elderly.

Hematoma type	Epidural	Subdural
Location	Between the skull and the dura	Between the dura and the arachnoid
Involved vessel	Temperoparietal locus (most likely) - Middle meningeal artery Frontal locus - anterior ethmoidal artery Occipital locus - transverse or sigmoid sinuses Vertex locus - superior sagittal sinus	Bridging veins
Symptoms	Lucid interval followed by unconsciousness	Gradually increasing headache and confusion
Appearance on CT	Biconvex lens	Crescent-shaped

Other signs and symptoms of subdural hematoma can include any combination of the following:

A history of recent head injury	Numbness	Loss of appetite	Disorientation
Loss of consciousness or fluctuating levels of consciousness	Nausea or vomiting	Personality changes	Amnesia
Irritability	Hearing loss or hearing ringing (tinnitus)	Inability to speak or slurred speech	Weakness or lethargy
Seizures	Headache (either constant or fluctuating)	Ataxia, or difficulty walking	Blurred Vision
Pain	Dizziness	Altered breathing patterns	Deviated gaze, or abnormal movement of the eyes.

Causes:

- Subdural hematomas are most often caused by head injury, when rapidly changing velocities within the skull may stretch and tear small bridging veins. Subdural hematomas due to head injury are described as traumatic.
- Much more common than epidural hemorrhages, subdural hemorrhages generally result from shearing injuries due to various rotational or linear forces.
- Subdural hemorrhage is a classic finding in shaken baby syndrome, in which similar shearing forces classically cause intra- and pre-retinal hemorrhages.

- Subdural hematoma is also commonly seen in the elderly and in alcoholics, who have evidence of cerebral atrophy.
- It is also more common in patients on anticoagulants, especially aspirin and warfarin.

Risk factors:

Factors increasing the risk of a subdural hematoma include very young or very old age. In juveniles, an arachnoid cyst is a risk factor for a subdural hematoma.

Other risk factors for subdural bleeds include taking blood thinners (anticoagulants), long-term alcohol abuse, and dementia.

Pathophysiology

Collected blood from the subdural bleed may draw in water due to osmosis, causing it to expand, which may compress brain tissue and cause new bleeds by tearing other blood vessels. The collected blood may even develop its own membrane.

In some subdural bleeds, the arachnoid layer of the meninges is torn, and cerebrospinal fluid (CSF) and blood both expand in the intracranial space, increasing pressure.

Substances that cause vasoconstriction may be released from the collected material in a subdural hematoma, causing further ischemia under the site by restricting blood flow to the brain. When the brain is denied adequate blood flow, a biochemical cascade known as the ischemic cascade is unleashed, and may ultimately lead to brain cell death.

The body gradually reabsorbs the clot and replaces it with granulation tissue.

Diagnosis

On a CT scan, subdural hematomas are classically crescent-shaped, with a concave surface away from the skull. However, they can have a convex appearance, especially in the early stage of bleeding. This may cause difficulty in distinguishing between subdural and epidural hemorrhages. A more reliable indicator of subdural hemorrhage is its involvement of a larger portion of the cerebral hemisphere since it can cross suture lines, unlike an epidural hemorrhage. Subdural blood can also be seen as a layering density along the tentorium cerebelli. This can be a chronic, stable process, since the feeding system is low-pressure. In such cases, subtle signs of bleeding such as effacement of sulci or medial displacement of the junction between gray matter and white matter may be apparent. A chronic bleed can be the same density as brain tissue (called isodense to brain), meaning that it will show up on CT scan as the same shade as brain tissue, potentially obscuring the finding.

Treatment

It is important that a patient receive medical assessment, including a complete neurological examination, after any head trauma. A CT scan or MRI scan will usually detect significant subdural hematomas.

Treatment of a subdural hematoma depends on its size and rate of growth. Some small subdural hematomas can be managed by careful monitoring until the body heals itself. Other small subdural hematomas can be managed by inserting a temporary small catheter through a hole drilled through the skull and sucking out the hematoma; this procedure can

be done at the bedside. Large or symptomatic hematomas require a craniotomy, the surgical opening of the skull. A surgeon then opens the dura, removes the blood clot with suction or irrigation, and identifies and controls sites of bleeding. Postoperative complications include increased intracranial pressure, brain edema, new or recurrent bleeding, infection, and seizure. The injured vessels must be repaired.

It is also worth noting, that dependent on the size and deterioration, age of the patient and anaesthetic risk posed, some subdurals will be inoperable and palliative management is the suitable treatment option.

(b) Haemopneumothorax.

Answer. Hemopneumothorax, or haemopneumothorax, is a medical term describing the combination of two conditions: pneumothorax, or air in the chest cavity, and haemothorax (also called haemothorax), or blood in the chest cavity.

A haemothorax, pneumothorax or both can occur if the chest wall is punctured. To understand the ramifications of this it is important to have an understanding of the role of the pleural space. The pleural space is located anatomically between the visceral membrane, which is firmly attached to the lungs, and the parietal membrane which is firmly attached to the chest wall (ribcage and intercostal muscles, muscles between the ribs). The pleural space contains pleural fluid. This fluid holds the two membranes together by surface tension, much as a drop of water between two sheets of glass prevents them from separating. Because of this, when the intercostal muscles move the ribcage outward, the lungs are pulled out as well, dropping the pressure in the lungs and pulling air into the bronchi, when we 'breathe in'. The pleural space is maintained in a constant state of negative pressure (in comparison to atmospheric pressure).

If the chest wall, and thus the pleural space, is punctured, blood, air or both can enter the pleural space. Air/blood rushes into the space in order to equalise the pressure with that of the atmosphere. As a result the fluid is disrupted and the two membranes no longer adhere to each other. When the rib cage moves out, it no longer pulls the lungs with it. Thus the lungs cannot expand, the pressure in the lungs never drops and no air is pulled into the bronchi. Respiration is not possible. The affected lung, which has a great deal of elastic tissue, shrivels in what is referred to as a collapsed lung.

Treatment

Treatment for this condition is the same as for hemothorax and pneumothorax independently: by tube thoracostomy, the insertion of a chest drain through an incision made between the ribs, into the intercostal space. A chest tube must be inserted to drain blood and air from the pleural space so it can return to a state of negative pressure and function normally.

Commonly, surgery is needed to close off whatever injuries caused the blood and air to enter the cavity (e.g. stabbing, broken ribs).

(c) Extra anatomic by pass for leg ischaemia.

Answer. Chronic critical limb ischemia is manifested by pain at rest, nonhealing wounds and gangrene. Ischemic rest pain is typically described as a burning pain in the arch or distal foot that occurs while the patient is recumbent but is relieved when the patient returns to a position in which the feet are dependent. Objective hemodynamic parameters that support the diagnosis of critical limb ischemia include an ankle-brachial index of 0.4 or less, an ankle systolic pressure of 50 mm Hg or less, or a toe systolic pressure of 30 mm Hg or less. Intervention may include conservative therapy, revascularization or amputation. Progressive gangrene, rapidly enlarging wounds or continuous ischemic rest pain can signify a threat to the limb and suggest the need for revascularization in patients without prohibitive operative risks. Bypass grafts are usually required because of the multilevel and distal nature of the arterial narrowing in critical limb ischemia. Patients with diabetes are more likely than other patients to have distal disease that is less amenable to bypass grafting. Compared with amputation, revascularization is more cost-effective and is associated with better perioperative morbidity and mortality. Limb preservation should be the goal in most patients with critical limb ischemia.

As with all occlusive arterial disease it is important to identify and treat any risk factors that may be accelerating or aggravating the condition. In CLI the collateral arteries have reached the limit of their ability to compensate and spontaneous improvement is unlikely. CLI is strongly associated with arterial disease elsewhere, particularly in the heart, and this increases the risk of serious complications. In general, unless some action is taken, a patient with CLI has a significant chance of developing gangrene in the affected leg and requiring an amputation. The most effective treatment is an operation to bypass the blocked arteries and to restore a good blood flow to the leg and foot. This is a major operation and can only be offered if the patient is well enough to undergo an anaesthetic. Occasionally it is possible to do a lesser operation which does not completely relieve all the occlusions but improves the blood supply sufficiently to relieve the worst symptoms and avoid an amputation. In some cases it is possible to improve the blood supply by angioplasty. In order to decide which form of treatment is possible your vascular will need to get an x-ray of the arteries (angiogram). With this information your vascular surgeon can discuss the possible treatment options.

How do bypass operations work?

The essential problem in all patients undergoing bypass surgery to the lower legs is that insufficient blood and hence oxygen is able to reach the tissues of the leg and foot. The tissues are starved of oxygen and this is what leads to pain, gangrene and ulceration.

Bypass operations work by improving the blood supply and hence the oxygen supply to the tissues that have been starved of oxygen. To do this the surgeon must take blood from a good artery above the area where the artery is blocked, and take the blood along a tube (the conduit) to a good artery below the blocked segments.

There are variations on this principle when blood can be taken from one side of the body to the other (femoro-femoral crossover) or blood is diverted from arteries to the arms down to the legs (axillo-bifemoral grafting).

Extra anatomic bypass are as follows:

- Infra- inguinal bypass: above knee and below knee femoropopliteal bypass grafting has been used using saphenous vein and PTFE as the graft material. Comparative studies have shown that while saphenous is superior to PTFE in graft patency at 2 years for below knee level, the results are equivocal for above knee level but favour the use of vein.
- Supra – inguinal bypass: Aorto-bifemoral bypass has>90% patency at 1 year but higher mortality.

Specific risks for aortobifemoral bypass surgery include:

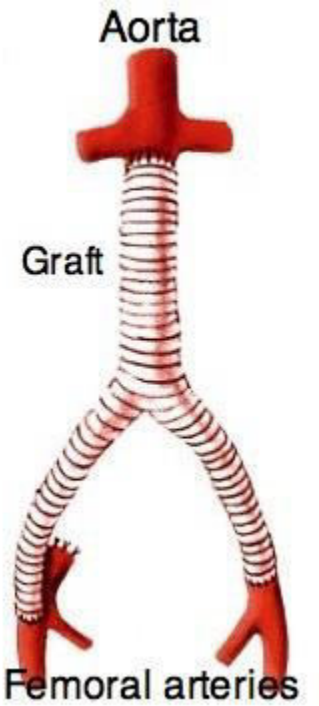
- Leg swelling.
- Failed or blocked grafts.
- Sexual dysfunction caused by nerve damage in the pelvis.

Cross femoral or ilio-femoral by pass have similar success rates for unilateral disaease with lower mortality rates. Axillo-bifemoral grafts have a lower patency rate and are not justifiable for claudication.

They can also be classified as follows:

Inflow operations

Inflow operations are performed to restore blood flow to the top of the leg. When the blood flow is normal to the legs, femoral pulses can be felt around the groin area. If major blood vessels (aorta or iliac arteries) are blocked then the blood flowing into the leg is severely reduced. An inflow operation is required to restore this flow to normal.



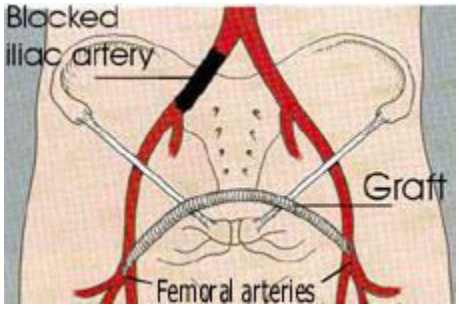
There are many operations to restore inflow to the legs, but the commonest are:

Aorto bifemoral grafting - these grafts originate on the aorta (main artery in the abdomen) and take blood to the femoral arteries at the groins. These are major operations with an approximately 5% (1 in 20) risk of dying. Aorto-bifemoral grafting is the most successful type of inflow operation, but is also the most traumatic. A Dacron graft (usually) is stitched to the aorta in the abdomen, just below the level of the kidneys. The graft has two legs which are then tunnelled under the abdominal muscles into the groins

to be attached to the femoral arteries. A diagram of an aorto-bifemoral graft is shown on the left.

Axillobifemoral grafting - these grafts originate from the axillary arteries (supplying the arms) and take blood to the femoral arteries. This is sometimes the best option in very elderly or very unfit patients as it is a less traumatic alternative to aorto-bifemoral grafting.

Femoro-femoral cross-over grafting - these grafts originate from a normal femoral artery in the groin on one leg and take blood to the femoral artery in the groin on the opposite leg. In the example shown below right a graft is running from the left femoral artery in the patient to the right femoral artery. At least one of the arteries running to the legs must be flowing normally in order to perform this operation.



Ilio-femoral bypass grafting - these grafts originate from the iliac arteries in the pelvis and take blood to the femoral arteries in the groins. These can be useful if there is enough normal iliac artery in the pelvis

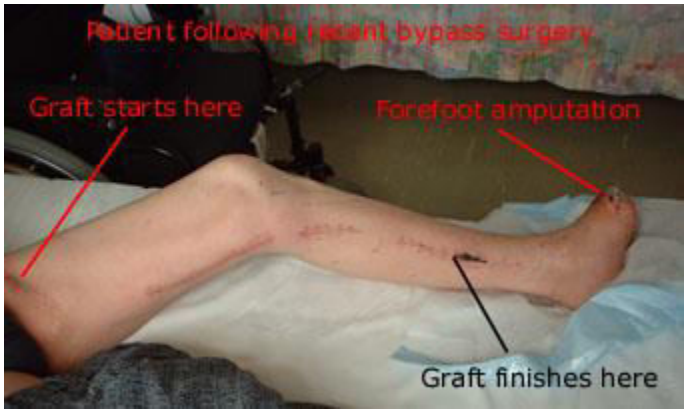
The common theme with these operations is that they restore good blood flow to the femoral artery at the top of the leg in the groin region. Restoring the inflow to normal levels is often all that is required in many patients, even if there is disease in the arteries further down the leg. The improvement in inflow alone can be enough to permit healing or reduce symptoms to a more bearable level.

Outflow operations

If the arterial flow to the femoral artery is good and there are good femoral pulses in the groin, an inflow operation is not required. If the patient has a shortage of blood to the leg and good femoral pulses then the arterial problem must lie at a lower level in the thigh and/or the calf arteries. These operations are frequently prolonged and complex procedures. There are many operations to restore blood flow to the legs, but the commonest are:

Femoro-popliteal bypass - these grafts originate from the femoral artery in the groin and take blood to the popliteal artery either just above or below the knee. The video opposite shows the main stages of femoro-popliteal bypass. If you are squeamish it may be better not to watch this video.

Femoro-distal or femoro-crural bypass - these grafts originate from the femoral artery at the groin and take blood to one of the 3 calf blood vessels (anterior and posterior tibial arteries and peroneal artery). The picture below shows the appearance of the leg following a femoro-peroneal bypass. This patient also required amputation of toes.



Pedal bypasses - these grafts can originate from any artery in the leg (femoral, popliteal and rarely tibial) and take blood to arteries below the level of the ankle joint (dorsalis pedis artery and posterior tibial artery or their branches). For pedal and distal bypasses the arteries are small and difficult to stitch with the naked eye so magnifying glasses are required to perform these operations reliably.

Femoral endarterectomy is a similar operation to carotid endarterectomy in which the diseased lining of the artery can be removed and blood flow restored.

General graft principles: For inflow operations it is better to use artificial grafts. They perform well when the arterial flow is high and have been shown over years to provide durable results. The commonest material used for inflow operations is Dacron.

For outflow operations it is generally better not to use artificial materials. Sometimes artificial materials have to be used as no vein is available and in these circumstances Goretex (PTFE) is the commonest material in use, although there is some reasonable evidence that for femoro-popliteal bypasses that Dacron impregnated with heparin can perform better.

In general with any graft it is better to make the grafts as short as possible and to avoid crossing joints, but quite frequently, grafts have to be long and cross joints to salvage the leg and foot.

The grafts are stitched to the arteries using fine polypropylene stitches (similar to nylon). Permanent stitches are used because the joins between the grafts and the native artery require lifelong support. This is especially important when using artificial grafts.

Complications of any operation can be divided into local and general.

Local complications

Graft failure - the bypass graft may block and the blood supply to the leg and foot will be reduced to the levels that were present before the bypass surgery. In fact on many occasions when a bypass graft blocks, especially in the early period (hours to weeks)

after surgery, the blood supply can be worse than before the bypass surgery. In these circumstances amputation can be inevitable unless the graft can be salvaged and the blood supply restored.

Wound infection - wound infections are not uncommon in vascular bypass surgery in the lower leg. This is because the incisions are long, the operations are prolonged and the tissues of the leg are often already damaged and frequently swollen. Superficial wound infections are usually treatable by a combination of dressings, antibiotics and sometimes drainage of infection.

Graft infection - if the infection involves the graft, especially if the graft is PTFE or Dacron, the situation is very much more serious. It is frequently impossible to eradicate infection from artificial materials and they need to be removed. When this happens the blood flow down the bypass graft is also removed. Unless an alternative route for a bypass graft can be found, this situation will frequently end in amputation. The presence of infection around a vein graft is more controllable and less likely to end in serious consequences.

Leg swelling - this is a very common event after lower limb bypass surgery. It is due to a combination of factors. Much of it may resolve, but frequently persistent swelling is the price a patient pays to avoid amputation.

General complications

Chest infections, heart attacks and strokes - because bypass surgery is prolonged and frequently takes place in elderly patients with other significant problems, these complications are not uncommon. However, they are just as common, if not more so, in patients undergoing amputation which is frequently the only alternative.

Maintenance and follow up:

All patients with hardening of the arteries should take a small dose of aspirin every day. As little as 75 milligrammes of aspirin per day is beneficial and should be continued after your operation. Aspirin interferes with the function of platelets in the blood. Platelets are important in blood clotting and aspirin "thins" the blood making clots less likely to form. A new medication - clopidogrel - is being used more frequently. It has little advantage over aspirin alone but there is increasing evidence that the combination of aspirin and clopidogrel may be more beneficial than either alone.

In some patients aspirin alone is not considered a strong enough treatment to prevent blood clotting. In these patients warfarin can be used either alone or with aspirin to reduce the chances of clots.There is good evidence that use of warfarin prolongs the patency of bypass grafts in the legs but for various reasons it is not used as frequently as it probably should.

In addition to aspirin the vascular risk factors should be treated.

Duplex surveillance

In about one third of grafts performed with veins a narrowing develops after the operation. To identify patients who develop narrowing, Duplex scanning of the graft is usually recommended at frequent intervals over the first two years. This enables problems to be identified at an early stage when they can be corrected before the graft blocks. Once a graft blocks it is usually more difficult to rescue the situation and restore normal blood flow. This sort of scanning is most useful in patients with vein grafts.

Only a few patients are unsuitable for a bypass operation or angioplasty, and in for these unfortunate cases the treatment options are limited. There are no drugs or other treatments that have been shown to be very effective. Often strong pain killers are the only option to control the symptoms.

If the pain is uncontrollable or gangrene and infection become established then an amputation may be necessary as a life protecting operation because without it there is a risk of the infection spreading and causing blood poisoning.

(d) Management of cardiac arrest during surgery.

Answer. Perioperative Cardiac Arrest- causes and Management

A sudden unsuspected cessation of effective cardiac pump function is termed cardiac arrest (CA). In the perioperative period it occurs more frequently during GA (5.5/10,000) than regional anaesthesia (1.5/10,000).
Peri-operative cardiac arrest may be due to

1. Medication related (B blocker, antiarrthymic drugs , anticholinesterase)
2. Airway or ventilation(esophageal intubtion, hypoxia,N2O without O2)
3. Electrolyte and metabolic disturbance(potassium, calcium etc)
4. Hypovolumia ,blood loss.

In general there are four basic mechanisms of cardiac arrest

1. Ventricular fibrillation(VF)
2. Ventricular tachycardia leading to pulseless (VT)
3. Asystole
4. Pulseless electrical activity(PEA)

Majority of in hospital CA are VF/VT but peri-operative cardiac arrest are mainly asystole. usually asystole have poor prognosis in CA but in the peri-operative period prognosis is better as majority are due to temporary increased parasympathetic activity(peritoneal pull, anticholinesterase without atropine).

Perioperative cardiac arrest management:

1. **Basic life support(BLS)**
2. **Advanced life support(ALS)**

An ultra quick rapid assessment to conform collapse, unconscious state is due to CA is made. It includes carotid ,femoral pulses pallor, cyanosis,unconsciousness (if not in GA) and presense of any secretion in upper airway.

Once confirmed, CALL for HELP.
Begin BLS immediately.
BASIC LIFE SUPPORT includes three components

1. Airway patency (jaw thust- chin lift, oropharyngeal airway, tracheal intubation)
2. Ventilation by self inflating bag or intubation. Lung should be inflated 8-10 times/ min with tidal volume of 7ml/kg with adequqte spacing.
3. Chest compression with heel of dominant hand in center of sternum. Rate of 100/minute.

CONTINUE CPR till defibrillator available and personnel trained in ALS are present

Advance life support

Starts with intubation and ventilation & use of defibrillator. Peripheral Venous access is made and various vasopressors and antiarrthmics are administered. In case of asystole and PEA transvenous pacing may be required.if hypovolumic administer fluid(RL/NS). CPR is continued throughout the process.

Drugs used

1. **Vasopressor** :epinephrine, vasopressin

2. **Antiarrthymics :** atropine , lidocaine ,amioderone,magnesium

3. **Others:** calcium gluconate, sodium bicarbonate, potassium chloride, calcium chloride.

Monitoring

1. arterial pulse and pressure
2. coronary perfusion pressure
3. end tidal CO2
4. venous blood gas
5. ECG

How long to resuscitate : 15 to 30 minutes

(e) Flaps used in surgery.

Answer. See ans. of Q. 3(d) of Paper-IV of May, 2009.

4. Answer in brief. 4 x 7.5

(a) Types of neck node dissection & their indications.
(b) Surgical treatment of portal hypertension.
(c) Risk factors for surgical infection.
(d) Thoracic outlet syndrome.

Answers.

(a) Types of neck node dissection & their indications.

Classification of Different Types of Neck Dissection with Clinical Indications

Comprehensive	Nodal Levels Removed	Structures Preserved	Indications
Radical neck dissection	Levels I–V	None	N+ neck for SCC where SAN involved
Modified radical neck dissection type type I	Levels I–V	SAN	N+ neck for SCC where SAN free of disease
Modified radical neck dissection type II	Levels I–V	SAN, SCM	N+ neck for SCC where IJV involved but SAN free of disease
Modified radical neck dissection type III	Levels I–V	SAN, SCM, IJV	Metastatic differentiated thyroid carcinoma
Selective			
Supraomohyoid neck dissection	Levels I–III	SAN, SCM, IJV	N0 neck for SCC of oral cavity and oropharynx (include level 4); N0 neck maliganant melanoma where primary site is anterior to ear (include parotidectomy for face and scalp)
Extended supraomohyoid neck dissection	Levels I–IV	SAN, SCM, IJV	N0 neck for SCC of lateral tongue
Lateral neck dissection	Levels II–IV	SAN, SCM, IJV	N0 neck for SCC of larynx and hypopharynx
Posterolateral neck dissection	Levels II–V, subocciptial, retroauricular nodes	SAN, SCM, IJV	N0 neck malignant melanoma where primary site is posterior to ear
SAN, spinal accessory nerve; SCM, sternocleidomastoid muscle; IJV, internal jugular vein.			

The cervical lymphatic nodal basins contain between 50 and 70 lymph nodes per side and are divided into seven levels.

1. Level I is subdivided.

Level IA is bounded by the anterior belly of the digastric muscle, the hyoid bone, and the midline.

Level IB is bounded by the anterior and posterior bellies of the digastric muscle and the inferior border of the mandible. Level IB contains the submandibular gland.

2. **Level II is bounded superiorly by the skull base, anteriorly by the stylohyoid muscle, inferiorly by a horizontal plane extending posteriorly from the hyoid bone, and posteriorly by the posterior edge of the sternocleidomastoid muscle. Level II is further subdivided.**

   Level IIA is anterior to the spinal accessory nerve.

   Level IIB, or the so-called submuscular triangle, is posterior to the nerve.

3. **Level III begins at the inferior edge of level II and is bounded by the laryngeal strap muscles anteriorly, by the posterior border of the sternocleidomastoid muscle posteriorly, and by a horizontal plane extending posteriorly from the inferior border of the cricoid cartilage.**
4. **Level IV begins at the inferior border of level III and is bounded anteriorly by the strap muscles, posteriorly by the posterior edge of the sternocleidomastoid muscle, and inferiorly by the clavicle.**
5. **Level V is posterior to the posterior edge of the sternocleidomastoid muscle, anterior to the trapezius muscle, superior to the clavicle, and inferior to the base of skull.**
6. **Level VI is bounded by the hyoid bone superiorly, the common carotid arteries laterally, and the sternum inferiorly. Although level VI is large in area, the few lymph nodes that it contains are mostly in the paratracheal regions near the thyroid gland.**
7. **Level VII (superior mediastinum) lies between the common carotid arteries and is superior to the aortic arch and inferior to the upper border of the sternum.**

(b) Surgical treatment of portal hypertension.

Answer.

Causes of Portal Hypertension.
I. Increased resistance to flow
A. Prehepatic (portal vein obstruction)
1. Congenital atresia or stenosis
2. Thrombosis of portal vein

3. Thrombosis of splenic vein
4. Extrinsic compression (eg, tumors)
B. Hepatic
1. Cirrhosis
a. Portal cirrhosis (nutritional, alcoholic, Laënnec)
b. Postnecrotic cirrhosis
c. Biliary cirrhosis
d. Others (Wilson disease, hemochromatosis)
2. Acute alcoholic liver disease
3. Chronic active hepatitis
4. Congenital hepatic fibrosis
5. Idiopathic portal hypertension (hepatoportal sclerosis)
6. Schistosomiasis
7. Sarcoidosis
C. Posthepatic
1. Budd-Chiari syndrome (hepatic vein thrombosis)
2. Veno-occlusive disease
3. Cardiac disease
a. Constrictive pericarditis
b. Valvular heart disease
c. Right heart failure
II. Increased portal blood flow
A. Arterial-portal venous fistula
B. Increased splenic flow
1. Banti syndrome
2. Splenomegaly (eg, tropical splenomegaly, myeloid metaplasia)

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Surgical Procedures for Oesophageal varices.
A. Direct variceal obliteration
1. Variceal suture ligation
a. Transthoracic

b. Transabdominal
2. Esophageal transection and reanastomosis
a. Suture technique
b. Staple technique
3. Variceal sclerosis
a. Esophagoscopic
b. Transhepatic
4. Variceal resection
a. Esophagogastrectomy
b. Subtotal esophagectomy
B. Reduction of variceal blood flow and pressure
1. Portasystemic shunts
a. End-to-side
b. Side-to-side
1. Side-to-side portacaval
2. Mesocaval
3. Central splenorenal
4. Renosplenic
2. Selective shunts
a. Distal splenorenal (Warren)
b. Left gastric vena caval (Inokuchi)
3. Reduction of portal blood flow
a. Splenectomy
b. Splenic artery ligation
4. Reduction of proximal gastric blood flow
a. Esophagogastric devascularization
b. Gastric transection and reanastomosis (Tanner)
5. Stimulation of additional portasystemic venous collaterals
a. Omentopexy
b. Splenic transposition
C. Measures to preserve hepatic blood flow after portacaval shunt
1. Arterialization of portal vein stump

Liver Transplantation

Any relatively young patient with cirrhosis who has survived an episode of variceal hemorrhage should be considered a candidate for liver transplantation, since any other form of therapy carries a much higher (about 80%) mortality rate within the subsequent 1–2 years as a result of repeat bleeding or complications of hepatic failure. Obviously, continued alcohol use is a contraindication to transplantation in most patients. The good transplantation candidates, however, should not be subjected to portasystemic shunts or other procedures if it appears that they will come to transplantation in the near future. In general, Child-Pugh A patients are candidates for portal decompression; Child-Pugh C patients are candidates for a transplant. A transjugular intrahepatic shunt (see previous section) is an excellent way to control bleeding while the patient is being prepared for a transplant.

Portasystemic Shunts

- The advent of TIPS has resulted in a marked decline in the number of shunt operations performed. However, surgical shunts are much more durable than TIPS, and good risk patients appear to benefit from theses procedures.
- Portasystemic shunts can be grouped into those that shunt the entire portal system (total shunts) and those that selectively shunt blood from the gastrosplenic region while preserving the pressure-flow relationships in the rest of the portal bed (selective shunts). All of the shunt operations commonly used today reduce the incidence of rebleeding to less than 10%, compared with about 75% in unshunted patients.
- The choice of shunt has been the subject of much debate and several randomized trials. The principal question in recent years has been whether encephalopathy and survival are better with a selective shunt (eg, a distal splenorenal shunt) than with a total shunt (eg, a mesocaval or an end-to-side portacaval shunt). The results are conflicting, but in general they support the contention that there is about half as much severe encephalopathy following selective shunts. None of the trials have shown any particular shunt to be associated with longer survival.

Severity of Hepatic Disease and Operative Risk

The immediate death rate of an elective shunt procedure can be predicted from the patient's hepatic function as reflected by the Child-Pugh classification. In addition to operative death rate, the figures also correlate with the death rate in the first postshunt year. Thereafter, survival curves of the different risk classes become reasonably parallel.

The severity of histopathologic changes in liver biopsies correlates with the immediate surgical death rate, the most ominous findings being hepatocellular necrosis, polymorphonuclear leukocyte infiltration, and the presence of Mallory bodies. The extent of histologic change also correlates with the more easily obtained data in the Child-Pugh classification (ie, severe changes occur in class C patients), so results of biopsies have no independent predictive value.

<u>Types of Portasystemic Shunts</u>

Total Shunts

- The end-to-side shunt completely disconnects the liver from the portal system. The portal vein is transected near its bifurcation in the liver hilum and anastomosed to the side of the inferior vena cava. The hepatic stump of the vein is oversewn.
- Postoperatively, the WHVP (sinusoidal pressure) drops slightly, reflecting the inability of the hepatic artery to compensate fully for the loss of portal inflow.
- The side-to-side portacaval, mesocaval, mesorenal, and central splenorenal shunts are all physiologically similar, since the shunt preserves continuity between the hepatic limb of the portal vein, the portal system, and the anastomosis. Flow through the hepatic limb of the standard side-to-side shunt is nearly always away from the liver and toward the anastomosis.
- The end-to-side portacaval shunt gives immediate and permanent protection from variceal bleeding and is somewhat easier to perform than a side-to-side portacaval or central splenorenal shunt.
- Encephalopathy may be slightly more common after side-to-side than end-to-side portacaval shunts. Side-to-side shunts are required in patients with Budd-Chiari syndrome or refractory ascites (when the latter is treated by a portasystemic shunt).
- **The mesocaval shunt** interposes a segment of prosthetic graft or internal jugular vein between the inferior vena cava and the superior mesenteric vein where the latter passes in front of the uncinate process of the pancreas.
- The mesocaval shunt is particularly useful in the presence of severe scarring in the right upper quadrant or portal vein thrombosis, and in some cases it may be technically easier than a conventional side-to-side portacaval shunt if a side-to-side type of shunt is necessary. In most cases, portal flow to the liver is lost after this shunt.

Selective Shunts

- Selective shunts lower pressure in the gastroesophageal venous plexus while preserving blood flow through the liver via the portal vein.
- **The distal splenorenal (Warren) shunt** involves anastomosing the distal (splenic) end of the transected splenic vein to the side of the left renal vein, plus ligation of the major collaterals between the remaining portal and isolated gastrosplenic venous system. The latter step involves division of the gastric vein, the right gastroepiploic vein, and the vessels in the splenocolic ligament. The operation is more difficult and time consuming than conventional shunts and except for the experienced operator is probably too complex for emergency portal decompression. If mobilization of the splenic vein is hazardous, the renal vein may be transected and its caval end joined to the side of the undisturbed splenic vein. The segment of splenic vein between the anastomosis and the portal vein is then ligated. Surprisingly, this seems to have little permanent effect on renal function as long as the remaining tributaries are preserved on the oversewn renal vein stump.
- In contrast to total shunts, the Warren shunt does not improve ascites and should not be performed in patients whose ascites has been difficult to control. Preoperative angiography should be performed to determine if the splenic vein and left renal vein

are large enough and close enough together for performance of this shunt. Recent pancreatitis may preclude safe dissection of the splenic vein from the undersurface of the pancreas.

- **Another type of selective shunt (Inokuchi shunt)** consists of joining the left gastric vein to the inferior vena cava by a short segment of autogenous saphenous vein. The procedure has not become popular, perhaps because of its technical complexity.

    - Selective shunts tend to become less selective over several years as new collaterals develop between the high-pressure and low-pressure regions of the portal system. This is accompanied by a gradual decrease in portal pressure (measured by WHVP) and evolution of the procedure into a version of side-to-side total shunt. The enlargement postoperatively of small venous tributaries entering the distal splenic vein from the pancreas suggests that this is the path by which nonselectivity develops. It is possible that this can be avoided by mobilizing the splenic vein all the way to the hilum (dividing these small vessels) before performing the splenorenal anastomosis.

Choice of Shunt

A reasonable approach to shunt selection is as follows:

- The distal splenorenal shunt is the first choice for elective portal decompression. If ascites is present or the anatomy is unfavorable, an end-to-side portacaval shunt is preferred. Side-to-side shunts would be done for patients with severe ascites or Budd-Chiari syndrome. The H-mesocaval and central splenorenal shunts are reserved for special anatomic situations in which the above operations are unsuitable. An end-to-side shunt or H-mesocaval shunt is performed for emergency decompression.
- Portacaval and distal splenorenal shunts are often followed by a rise in platelet count in patients with secondary hypersplenism. The response is unpredictable, however, and hypersplenism need not necessarily dictate the type of shunt since it rarely produces clinical manifestations. A central splenorenal shunt, in which splenectomy is performed, should not be considered preferable to other kinds of shunt just because the patient has a low platelet count.

Devascularization Operations: The objective of devascularization is to destroy the venous collaterals that transport blood from the high-pressure portal system into the veins in the submucosa of the esophagus.

Miscellaneous Operations:

- Attempts have also been made to decrease portal pressure by decreasing splanchnic inflow through splenectomy or splenic artery ligation. Diseases characterized by marked splenomegaly may rarely be associated with portal hypertension as a consequence of increased splenic blood flow, which has been known to reach levels as high as 1000 mL/min.

- Splenic blood flow may occasionally be increased enough in patients with cirrhosis to contribute significantly to the portal hypertension. However, splenectomy or splenic artery ligation in cirrhosis most often gives only a transient decrease in portal pressure, and over half of patients having these operations bleed again. Some workers have suggested that the absolute size of the splenic artery (a crude index of splenic flow) correlates with the clinical effectiveness of splenic artery ligation, a good result being predictable if the diameter of the artery is 1 cm or greater.

(c) Risk factors for surgical infection.
Answer. Risk Factors for Surgical Site Infection According to the Three Main Determinants of Such Infection

MICROORGANISM	LOCAL WOUND	PATIENT
Remote site infection	Surgical technique:	Age
Long-term care facility	Hematoma/seroma	Immunosuppression
Recent hospitalization	Necrosis	Steroids
Duration of the procedure	Sutures	Malignancy
Wound class	Drains	Obesity
Intensive care unit patient	Foreign bodies	Diabetes
Previous antibiotic therapy		Malnutrition
Preoperative shaving		Multiple comorbid conditions
Bacterial number, virulence, and antimicrobial resistance		Transfusions
		Cigarette smoking
		Oxygen
		Temperature
		Glucose control

(d) Thoracic outlet syndrome.

Answer. The term thoracic outlet syndrome (TOS) describes a complex of signs and symptoms caused by compression of the neurovascular structures of the arm as they exit the thoracic outlet. The neurovascular bundle has three components: nerve, artery, and vein, each of which can be individually compressed, producing a distinct symptom complex different from the others. Specifically, TOS is categorized as neurogenic, arterial, or venous in origin, with the neurogenic form constituting the most common form (greater than 95%) followed by the venous (2 to 3%) and the arterial forms (1%).
Aetiology:

- **Neurogenic Thoracic Outlet Syndrome:** Compression of elements of the brachial plexus most often results from variations in scalene muscle anatomy as they insert on the first rib.

A history of neck trauma can be identified in approximately 80% of neurogenic TOS cases.

The most common type is hyperextension trauma (e.g., a whiplash injury from an auto accident) that stretches the scalene muscles causing hemorrhage and edema. These swollen and scarred muscle fibers can compress the adjacent brachial plexus leading to irritation and resultant neurologic symptoms.

Scalene trauma can also result from repetitive stress injury such as is seen in jobs requiring repeated, frequent movements of the neck and upper extremity (e.g., assembly line worker or shelf stocker) or prolonged neck hyperextension (e.g., keyboard operator). Other predisposing factors include osseous abnormalities (e.g., cervical ribs) and congenital bands and ligaments narrowing the thoracic outlet, and less commonly, sagging shoulders from poor posture and significant macromastia.

- **Arterial Thoracic Outlet Syndrome:** Arterial compression is almost universally associated with a bony abnormality of the thoracic outlet.

  - Cervical ribs, which occur in 0.5% to 1.5% of the normal population, are present in more than 95% of patients with arterial TOS.
  - Other abnormalities include elongated transverse processes of C7, anomalies of the first rib (anterior agenesis, exostoses), and scarring associated with clavicular fractures.
  - These osseous structures are usually associated with anomalous myofascial bands that elevate and "pinch" the subclavian artery with abduction of the arm. Repetitive compression and trauma to the subclavian artery lead to intimal injury/ulceration at the site of compression or poststenotic dilation/aneurysmal degeneration of the artery just distal to the point of compression. Mural thrombus forming at the site of the damaged intima or within an aneurysm can then embolize down the arm, leading to ischemic symptomatology.

- **Venous Thoracic Outlet Syndrome:** Compression of the subclavian vein usually occurs at the junction of the clavicle and first rib. During strenuous hyperabduction of the arm, the vein can be crushed between the overlying clavicle (and subclavius muscle/tendon) and the first rib. Repetitive trauma at this location produces intimal injury and perivenous scarring eventually followed by clot formation. The result is primary subclavian-axillary vein thrombosis, also known as Paget-Schroetter syndrome or effort thrombosis because it often occurs after exertion of the affected extremity. Secondary subclavian-axillary vein thrombosis is usually the result of direct subclavian trauma (e.g., from an indwelling central venous catheter) and is much more common and better tolerated than primary thrombosis.

Diagnosis

Neurogenic Thoracic Outlet Syndrome:

- The major symptoms of neurogenic TOS are upper extremity pain and paresthesias. The most common pattern is irritation of all the nerve roots, followed by the lower plexus (ulnar nerve distribution), and lastly the upper plexus (median nerve distribution).
- Pain can also occur in the neck (from scalene muscle spasm), shoulder, parascapular area, and anterior chest wall; it usually radiates down the arm into the hand with paresthesias and numbness in the fingertips.
- Occipital headache is a common complaint and probably represents referred pain from scalene muscle spasm.
- Women typically outnumber men, and most patients are in their thirties or forties.
- In patients with a history of neck trauma, symptoms typically begin within a few days of the injury; in patients with work-related overactivity, the onset of symptoms is more gradual.
- Symptoms can be precipitated (or aggravated) by activities that stretch the plexus (e.g., hair combing, reaching up to a shelf, or even grasping a steering wheel).

Physical examination:

- Often reveals tenderness over the scalene muscles or supraclavicular fossa on the involved side.
- Radiating pain or paresthesias in the affected arm can often be elicited by pressing on the scalene muscles, tapping over the brachial plexus, or by tilting the head to the opposite side.
- Abducting the arms to 90 degrees in external rotation (the so-called "stick-em-up" position) can also elicit symptoms.
- Decreased grip strength and loss of light touch in the affected hand is common.
- Atrophy of the ulnar innervated intrinsic musculature of the hand is an uncommon and late finding.
- Much has been made of the loss of radial pulse with provocative positioning. Adson's sign (loss of radial pulse produced by head rotation to the affected side with deep inspiration) and other tests, however, are positive in up to 50% of normal individuals and hence have no diagnostic utility in neurogenic TOS.

Investigations

- Nerve compression is usually not severe enough to cause neurophysiologic abnormalities.
- Electromyography and nerve conduction studies are therefore of little help in patients with neurogenic TOS.
- Cervical spine films should be obtained to determine the presence of osseous abnormalities.
- Cross-sectional imaging with CT (computed tomography) or MRI (magnetic resonance imaging) can be helpful to rule out cervical spine disease.

- Other conditions that can mimic the neurogenic TOS include carpal tunnel syndrome and shoulder tendonitis.

Arterial Thoracic Outlet Syndrome

- Patients with symptomatic arterial TOS most commonly present with symptoms of digital ischemia related to microembolization of platelet aggregates formed on intimal lesions or mural thrombus from poststenotic aneurysms.
- Early symptoms may include unilateral Raynaud's syndrome, or complaints of digital cyanosis, pallor, coldness, pain, and paresthesias. Findings are most common in the thumb and index finger because of the direct pathway of the radial artery.
- If the arterial lesion is not diagnosed and corrected, repeated embolization will lead to obliteration of the distal arterial bed, with worsening ischemia leading to digital ulceration and gangrene.
- Macroembolization to the larger arteries of the arm presents as acute limb ischemia, the severity of symptoms depending on the patency of the distal arterial tree.
- Thrombotic occlusion of the subclavian artery can also occur.

Physical examination

- May be normal or reveal signs of ischemia of varying degrees and extents.
- A supraclavicular bruit or pulsatile mass may occasionally be detected.
- Concomitant symptoms of neurogenic TOS may be present but are often overshadowed by ischemic symptomatology.

Investigations

- Because the most common etiologic agent is a cervical rib, cervical spine films are extremely helpful.
- Recognition of arterial TOS usually requires a high index of suspicion.
- Patients with Raynaud's symptoms or subtle signs of digital ischemia should always undergo segmental pressure testing including the digital waveforms to rule out an occlusive process.
- In the absence of a documented collagen vascular disorder or severe arteriosclerosis, the presence of small artery (palmar/digital) occlusive disease should always lead to arteriography.
- Patients with macroemboli and no obvious cardiac source should also undergo arteriography to exclude a proximal arterial source. Arteriography should include complete views from the aortic arch to the fingertips.
- If the subclavian artery appears normal in the neutral position, repeat view should be obtained with the arm abducted.
- Magnified views of the hand following vasodilator injection are helpful in distinguishing embolic occlusions from intrinsic disease.

- Arteriographic findings can be subtle, ranging from minor wall irregularities and minimal poststenotic dilation to frank aneurysms.
- Duplex scanning may be useful in patients who present with pulsatile supraclavicular masses or questionable findings on arteriography.

Venous Thoracic Outlet Syndrome

- Primary axillosubclavian venous thrombosis is characterized by the sudden development of swelling and pain in the affected extremity.
- Edema is typically nonpitting and involves the entire arm. Additional signs can include prominence of superficial veins over the shoulder and anterior chest wall and varying degrees of cyanosis.
- Symptoms are worsened by use of the affected extremity.
- Patients are typically young, healthy adults (males outnumber females 2:1) with a history of strenuous or repetitive activity (e.g., weightlifting) before symptom onset.

Investigations

- Patients with suspected axillosubclavian venous thrombosis should undergo duplex ultrasonography to confirm the diagnosis.
- Phlebography remains the "gold standard" testing modality and should be used whenever intervention is contemplated or the diagnosis is in doubt following duplex scanning.
- In the absence of thrombus, additional views should be obtained with the arm fully abducted or held behind the head to exclude extrinsic compression.
- Special attention should be focused on the subclavian vein at the costoclavicular junction where intrinsic lesions are most often found.

Treatment

Neurogenic Thoracic Outlet Syndrome

- Initial treatment for neurogenic TOS should almost always be conservative, except in rare cases when patients present with longstanding symptoms and evidence of distal muscle atrophy.
- Start patients on an aggressive home physical therapy regimen consisting of neck stretching exercises, posture improvement, and avoidance of aggravating activities.
- Patients are given modest doses of analgesics and muscle relaxants.
- Majority of patients improve significantly on this regimen.
- Operative intervention is offered only to stable patients whose symptoms remain significantly disabling after a minimum of 6 to 12 months of this conservative therapy.
- The goal of surgery is to decompress the brachial plexus.
- The traditional operative approach calls for resection of all of the rigid scalene triangle structures that may be compressing the plexus—the first rib and any other

osseous abnormalities (e.g., cervical ribs) inferiorly, the anterior scalene muscle anteriorly, and the middle scalene posteriorly.

- Thoracic outlet decompression with first rib resection is associated with greater morbidity and greater length of stay than when the first rib is spared, with no difference in neurologic outcome.
- Which approach (supraclavicular versus transaxillary) to use for surgical decompression of the brachial plexus remains controversial.
- A supraclavicular approach for neurogenic TOS provides the best exposure of the brachial plexus and associated myofascial/bony pathology.
- A transaxillary approach does provide good exposure for first rib resection, but identification of offending soft tissue pathology and complete brachial plexus neurolysis is difficult if not impossible through this approach.

Arterial Thoracic Outlet Syndrome

- Therapy for arterial TOS must address not only the pathologic lesion in the involved subclavian artery but also any clot that may have embolized into the distal arterial tree.
- The timing and sequence of intervention depend on the severity and acuteness of the patient's ischemic symptoms.
- Patients with arterial TOS most commonly present with symptoms of chronic ischemia in which limb viability is not immediately threatened. In this situation, treat the subclavian artery pathology first; postponing any attempt at distal vascular reconstruction (i.e., bypass surgery) until after the proximal emboligenic site has been eliminated.
- In situations of acute upper extremity ischemia, however, distal thromboemboli should be cleared by standard techniques before treating the subclavian artery.
- Although in some situations both lesions can be treated at the same operation, postponing surgery on the thoracic outlet at least several days may be safer.
- Arterial vasospasm at the site of distal thromboembolectomy and residual surgically inaccessible clot can compromise the outcome of the subclavian repair. Treatment with heparin anticoagulation allows time for resolution of distal arterial vasospasm and maximizes the chances for endogenous lysis of remaining thrombus.
- Catheter-directed thrombolysis has been a useful modality for treating some patients with acute limb ischemia.
- Thoracic outlet decompression for arterial TOS requires elimination of the causative compressive pathology as well as correction of the resulting arterial pathology.
- As discussed earlier, the overwhelming majority of patients have a bony anomaly, most commonly a cervical rib. Isolated myofascial bands and scalene anomalies have been described with arterial TOS but are exceedingly rare. Identification of the causative pathology is usually straightforward during exposure of the subclavian artery. Resection of these compressive elements proceeds before arterial reconstruction, taking care to avoid undue manipulation of the artery with the potential for further distal embolism. In rare situations with clavicular pathology (e.g., hypertrophic callus from a previous fracture), medial claviculectomy may be required.

- If there is any concern about significant ulceration or residual mural thrombus, the involved arterial segment must be opened and the culprit lesion removed.
- A limited superficial thromboendarterectomy (intimectomy) of a focal lesion can usually be performed through a longitudinal arteriotomy.
- The approach to patients with poststenotic dilation or aneurysmal changes without intraluminal thrombus/ulceration must be tailored to the specific lesion.
- In cases of minimal poststenotic dilation (only 1 or 2 mm larger than the adjacent normal artery), treatment is decompression alone without arterial reconstruction. With more significant degrees of dilation, arterial placation can be performed; When the dilated segment approaches twice the normal arterial diameter, arterial resection may be the only choice. In these situations, it necessary to place an interposition graft; saphenous vein graft is preferred.
- If concomitant first rib resection is required, there is often enough arterial redundancy to allow a primary end-to-end anastomosis.
- In patients with severely compromised distal circulation, we always consider concomitant cervicodorsal sympathectomy at the time of thoracic outlet decompression.

Venous Thoracic Outlet Syndrome

- Traditional management of primary axillosubclavian vein thrombosis has been conservative—systemic anticoagulation with rest and elevation of the affected limb.
- Current management of primary subclavian vein thrombosis has evolved in an attempt to avoid this outcome. Modern therapy is designed to restore venous patency, relieve extrinsic compression on the vein, and eliminate any venous scarring/stenosis that may be impeding flow.
- When thrombosis is confirmed, catheter-directed thrombolysis is initiated using recombinant tissue plasminogen activator or urokinase according to institutional preference/protocol. It is rarely necessary to continue thrombolysis beyond 36 to 48 hours.
- Following successful thrombolysis, careful phlebographic assessment of the subclavian vein is performed (using arm elevation if necessary) to demonstrate extrinsic compression or intrinsic stenosis.
- Narrowing of the vein at the costoclavicular junction is almost universally present. Relief of this narrowing is necessary to prevent recurrent thrombosis or, in the absence of thrombosis, activity-induced upper extremity venous congestion.
- Angioplasty with or without stenting has been advocated as a good technique for treating this lesion, although recent experience suggests a high failure rate for this approach without concomitant first rib resection.
- Optimal timing of operative intervention remains controversial, with opinions ranging from treating patients urgently following successful thrombolysis to placing them on anticoagulation for several weeks to allow time for the venous endothelium to heal and the surrounding inflammatory response to subside.
- Prolonged waiting, however, risks recurrent thrombosis and delays return to normal activity for these young, healthy patients. In our practice, patients are anticoagulated

following thrombolysis and undergo operation electively during the same hospitalization.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES
MS (General Surgery) Examination, 2006

April, 2006

PAPER III

Time Allowed: 3 Hours **Full Marks:100**

1. Discuss the current status of diagnosis & management of oesophageal carcinoma. (20)

Answer. Diagnosis and pretreatment evaluation

- A complete physical examination and directed lab analysis should be part of the workup of patients suspected of having CE.
- Plain chest radiography is often unrewarding unless the esophagus is distended or an air fluid level is observed in the posterior mediastinum.
- Although the diagnosis of CE can be suspected on a barium contrast study, endoscopy is the only modality that provides a tissue diagnosis.
- Esophagoscopy allows direct visualization of mucosal abnormalities and histologic diagnosis from biopsies. It will also determine if there are changes consistent with BE, because this may impact the extent of subsequent esophageal resection.
- Rigid esophagoscopy can be helpful when the lesion is in the cervical esophagus and endoscopic examination of the upper esophageal sphincter is important.
- Endoscopic ultrasound (EUS) combines conventional endoscopy with ultrasound technology.

    - Esophageal carcinoma presents as a hypoechoic mass that disrupts the normal anatomy of the esophagus on endoluminal sonography.
    - This examination can reveal the depth of mural penetration and invasion of tumor into the periesophageal tissue.
    - The reported accuracy of this technique for assessing depth of tumor penetration (T status) ranges from 85% to 90%.
    - EUS can also demonstrate extension of tumor into adjacent lymph nodes. Hypoechoic lymph nodes sharply demarcated and larger than 5 mm are more likely malignant, whereas lymph nodes that are not sharply demarcated and have an elongated bean shape with hyperechoic pattern are more likely benign.
    - EUS is not always able to differentiate between benign and malignant nodes, and the overall N staging accuracy is approximately 77%. EUS-guided fine needle aspiration of periesophageal lymph nodes has a positive predictive value of 92% and a negative predictive value of 89% and an overall accuracy of 91%.
    - Computed tomography (CT) is primarily used for preoperative staging. The overall accuracy of CT in staging esophageal cancer is approximately 80% to 90%. Local extraesophageal extension into adjacent structures such as the aorta, tracheobronchial tree, or mediastinal lymph nodes are detected in 76% to 86% of

cases at the time of initial presentation. Approximately one third of patients show liver metastasis or abdominal lymphadenopathy.

- Positron emission tomography (PET) allows the identification of metabolically active areas as evidence of tumor involvement. One potential advantage of PET over CT is the detection of distant metastasis.
- Bronchoscopy is recommended to exclude tracheal or bronchial invasion in patients with tumors proximal to the carina or in patients with symptoms suggestive of aerodigestive fistula such as recurrent pneumonia or hemoptysis. Surgical staging modalities like thoracoscopy and laparoscopy have been used to define pretreatment staging but have not gained wide acceptance.

The esophageal cancer staging system is based on the American Joint Committee on Cancer (AJCC) TNM system.

American Joint Committee on Cancer (AJCC) TNM Classification for Esophageal Carcinoma

T0	No evidence of primary tumor	
T1	Invades lamina propria or submucosa	
T2	Invades muscularis propria	
T3	Invades adventitia	
T4	Invades adjacent structures	
N0	No regional lymph node metastasis	
N1	Regional lymph node metastasis	
M0	No distant metastasis	
M1	Distant metastasis	
M1a	Invasion of celiac or cervical lymph nodes	
M1b	Other distant metastasis	
Stage 0	Tis N0 M0	
Stage I	T1 N0 M0	
Stage IIA	T2 N0 M0	T3 N0 M0
Stage IIB	T1 N1 M0	T2 N1 M0
Stage III	T3 N1 M0	T4 Any N M0
Stage IV	Any T Any N M1	

Imaging: Several different imaging modalities are used to stage newly diagnosed esophageal cancer. Although either a barium study or an esophagogastroduodenoscopy may initially raise suspicion of esophageal cancer, the definitive histologic diagnosis is made with esophagogastroduodenoscopy and biopsy. Chromoendoscopy can help highlight the area of the esophagus to be biopsied and determine the extent of disease. Many new endoscopic techniques are being studied with the advent of increased adenocarcinoma prevalence and increased understanding of Barrett's esophagus. These

new techniques may help diagnose early disease and include trimodality endoscopy providing high-resolution white-light endoscopy, autofluorescence and narrow-band imaging, confocal fluorescence microscopy, elastic scattering spectroscopy, and optical coherence tomography.

- Once the diagnosis is made, endoscopic ultrasound is the modality of choice to stage local disease, that is, depth of tumor invasion and presence of regional lymph nodes . A biopsy or fine-needle aspirate of regional lymph nodes, as well as of ascites and regional liver or adrenal metastases, can also be performed at the time of endoscopic ultrasound.
- The American Joint Committee on Cancer (AJCC) staging now delineates regional nodes as N1-3 depending on the number of involved nodes.
- Although technically celiac nodes for a gastroesophageal junction cancer are regional, their involvement portends a poor prognosis, and immediate surgery in such patients is usually deferred with the delivery of neoadjuvant therapy given first to generate a response.
- Lymph nodes can be pathologically staged by using endoscopic ultrasound with fine-needle aspiration. Similarly, distal esophageal tumors with evidence of high mediastinal or paratracheal nodes, although regional according to the new AJCC staging, may represent metastatic disease, which would not be treated with primary surgical intervention or radiotherapy.
- Endoscopic ultrasound has consistently proved superior to computed tomography (CT) scanning, magnetic resonance imaging, and positron emission tomography (PET) scanning in providing regional tumor/node/metastasis (TNM) staging. With a stenosing tumor that does not allow passage of the endoscope (around 30% of cases), the role of endoscopic ultrasound is limited. However, in these cases the tumor is likely to be at least T3, and CT or PET scanning can be used to assess the presence of regional or distant nodal disease.
- There is currently no consensus on the role of staging laparoscopy or thoracoscopy in the era of endoscopic ultrasound and PET scanning. Noninvasive staging with PET and endoscopic ultrasound- fine-needle aspirate has become the preferred algorithm. Preoperative bronchoscopy is reserved for patients with potentially resectable disease located above the carina, usually squamous cancers, to rule out airway invasion.

Treatment Option Overview:

- The prevalence of Barrett metaplasia in adenocarcinoma of the esophagus suggests that Barrett esophagus is a premalignant condition. Strong consideration should be given to resection in patients with high-grade dysplasia in the setting of Barrett metaplasia.
- Endoscopic surveillance of patients with Barrett metaplasia may detect adenocarcinoma at an earlier stage more amenable to curative resection.The survival rate of patients with esophageal cancer is poor. Asymptomatic small tumors confined to the esophageal mucosa or submucosa are detected only by chance. Surgery is the treatment of choice for these small tumors. Once symptoms are present (e.g.,

dysphagia, in most cases), esophageal cancers have usually invaded the muscularis propria or beyond and may have metastasized to lymph nodes or other organs.

- In the presence of complete esophageal obstruction without clinical evidence of systemic metastasis, surgical excision of the tumor with mobilization of the stomach to replace the esophagus has been the traditional means of relieving the dysphagia.
- The optimal surgical procedure is controversial. One approach advocates transhiatal esophagectomy with anastomosis of the stomach to the cervical esophagus. A second approach advocates abdominal mobilization of the stomach and transthoracic excision of the esophagus with anastomosis of the stomach to the upper thoracic esophagus or the cervical esophagus. One study concluded that transhiatal esophagectomy was associated with lower morbidity than transthoracic esophagectomy with extended en bloc lymphadenectomy; however, median overall disease-free and quality-adjusted survival did not differ significantly.
- Surgical treatment of resectable esophageal cancers results in 5-year survival rates of 5% to 30%, with higher survival rates in patients with early-stage cancers. This is associated with a less than 10% operative mortality rate.

Stage 0 squamous esophageal cancer is rarely seen, but surgery has been used for this stage of cancer.

Stage 0: A stage 0 tumor is not true cancer. It contains abnormal cells called *high-grade dysplasia* and is really a type of pre-cancer. The abnormal cells look like cancer cells, but they are only found in the inner layer of cells lining of the esophagus (the epithelium). They have not grown into deeper layers of the esophagus. This stage is often diagnosed when a routine biopsy is done in someone with Barrett's esophagus.

Options for treatment may include endoscopic treatments such as photodynamic therapy, radiofrequency ablation, or endoscopic mucosal resection (EMR). Still, many doctors feel that surgery to remove the esophagus remains the standard treatment.

Stage I Esophageal Cancer - Standard treatment option: Surgery.

Stage I: In this stage the cancer has grown into some of the deeper layers of the esophagus (past the innermost layer of cells) but has not reached the lymph nodes or other organs.

Some very early stage I cancers that involve only a small area of the mucosa and haven't grown into the submucosa may be treated with EMR. This may be followed by some type of endoscopic procedure to destroy any remaining abnormal areas in the esophagus lining.

But most patients who are healthy enough have surgery (esophagectomy) to remove the part of their esophagus that contains the cancer. Some doctors may recommend treatment with chemotherapy and radiation therapy (chemoradiation) after surgery, especially if there are signs that all of the cancer may not have been removed.

If the cancer is in the upper part of the esophagus (in the neck), chemoradiation may be recommended as the main treatment instead of surgery. This may cure the cancer in some patients. Close follow-up with endoscopy is very important to look for possible signs of cancer returning.

Patients who cannot have surgery because they have other serious health problems may be treated with chemotherapy, radiation therapy, or both together.

Stage II Esophageal Cancer

Standard treatment options:

1. Surgery.
2. Chemoradiation.

Stage II: Stage II includes cancers that have grown into the main muscle layer of the esophagus or through the muscle layer and into the connective tissue on the outside of the esophagus. This stage also includes some cancers that have spread to 1 or 2 nearby lymph nodes.

For people who are healthy enough, treatment options for this stage include surgery, chemotherapy followed by surgery, or chemoradiation followed by surgery. Patients with adenocarcinoma at the place where the stomach and esophagus meet (the gastroesophageal junction) are often treated with chemotherapy (possibly including the targeted drug Herceptin) followed by surgery.

If surgery is the initial treatment, chemoradiation may be recommended afterward, especially if the cancer is an adenocarcinoma or if there are signs that some cancer may have been left behind.

In some instances (especially for cancers in the upper part of the esophagus), chemoradiation may be recommended as the main treatment instead of surgery. Patients who do not have surgery need close follow-up with endoscopy to look for possible signs of remaining cancer. Unfortunately, even when cancer cannot be seen, it can still be present below the inner lining of the esophagus, so close follow-up is very important.

Patients who cannot have surgery because they have other serious health problems are usually treated with chemotherapy and radiation therapy.

Stage III Esophageal Cancer

Standard treatment options:

1. Surgical resection of T3 lesions.
2. Chemoradiation.

Stage III

Stage III includes some cancers that have grown through the wall of the esophagus to the outer layer, as well as cancers that have grown into nearby organs or tissues. It also includes most cancers that have spread to nearby lymph nodes.

These cancers are treated much like stage II cancers.

For people who are healthy enough, treatment options for this stage may include surgery, chemotherapy followed by surgery, or chemoradiation followed by surgery. Patients with adenocarcinoma at the place where the stomach and esophagus meet (the gastroesophageal junction) are often treated with chemotherapy (possibly including the targeted drug Herceptin) followed by surgery.

If surgery is the initial treatment, chemoradiation may be recommended afterward, especially if the cancer is an adenocarcinoma or if there are signs that some cancer may have been left behind.

In some instances (especially for cancers in the upper part of the esophagus), chemoradiation may be recommended as the main treatment instead of surgery. Patients who do not have surgery need close follow-up with endoscopy to look for possible signs of remaining cancer. Unfortunately, even when cancer cannot be seen, it can still be present below the inner lining of the esophagus, so close follow-up is very important.

Patients who cannot have surgery because they have other serious health problems are usually treated with chemotherapy and radiation therapy.

Stage IV Esophageal Cancer

At diagnosis, approximately 50% of patients with esophageal cancer will have metastatic disease and will be candidates for palliative therapy.

Standard treatment options:

1. Endoscopic-placed stents to provide palliation of dysphagia.
2. Radiation therapy with or without intraluminal intubation and dilation.
3. Intraluminal brachytherapy to provide palliation of dysphagia.
4. Nd:YAG endoluminal tumor destruction or electrocoagulation.
5. Chemotherapy has provided partial responses for patients with metastatic distal esophageal adenocarcinomas.

Stage IV

Stage IV esophageal cancer has spread to distant lymph nodes or to other distant organs.

In general, these cancers are very hard to get rid of completely, so surgery to try to cure the cancer is usually not a good option. Treatment is used mainly to help keep the cancer under control for as long as possible and to relieve any symptoms it is causing.

Chemotherapy may be given to try to help patients feel better and live longer, but the benefit of giving chemo is not clear. Radiation therapy or other treatments may be used to help with pain or trouble swallowing. Some people prefer not to have treatments that have serious side effects and choose to receive only those treatments that will keep them comfortable and add to their quality of life. For more information on treatments that may be helpful, see the section, "Palliative therapy for esophagus cancer."

2. Write short notes on: 10 x 3

a) Prognostic factors in Colo-rectal cancer.

b) Management of burst abdomen.

c) Tumour markers

(a) Prognostic factors in Colo-rectal cancer.

Answer. There are multiple factors that influence the possible significance of outcome in colorectal carcinoma. Accordingly, the following categories of prognostic factors can be defined.

Category I: includes factors definitively proven to be of prognostic import based on evidence from multiple statistically robust published trials and generally used in patient management.

Category IIA: includes factors extensively studied biologically and/or clinically and repeatedly shown to have prognostic value for outcome and/or predictive value for therapy that is of sufficient import to be included in the pathology report but that remains to be validated in statistically robust studies.

Category IIB: includes factors shown to be promising in multiple studies but lacking sufficient data for inclusion in category I or IIA.

Category III: includes factors not yet sufficiently studied to determine their prognostic value.

Category IV: includes factors well studied and shown to have no prognostic significance.

Metastasis to regional lymph nodes as determined by pathologic assessment is, among the factors that most strongly predict outcome following surgical resection, second only to distant metastatic disease in importance.

- Factors in category I are as follows:
  - ❖ The local extent of tumor assessed pathologically (the pT category of the TNM staging system of the American Joint Committee on Cancer and the Union Internationale Contre le CancerAJCC/UICC]); regional lymph node metastasis (the pN categoryof theTNM staging system);
  - ❖ Blood or lymphatic vessel invasion; residual tumor following surgery with curative intent
  - ❖ (the R classification of the AJCC/UICC staging system), especially as it relates to positive surgical margins;
  - ❖ Preoperative elevation of carcinoembryonic antigen elevation (a factor established by laboratory medicine methods rather than anatomic pathology)- The CEA blood test is not reliable for diagnosing cancer or as a screening test for early detection of cancer. Most types of cancer do not produce a high CEA. Elevated CEA levels should return to normal after successful surgical resection, or within 6 weeks of starting treatment if cancer treatment is successful.
- Factors in category IIA included the following:
  - ❖ Tumor grade, radial margin status (for resection specimens with nonperitonealized surfaces),
  - ❖ Residual tumor in the resection specimen following neoadjuvant therapy (the ypTNM category of the TNM staging system of the AJCC/UICC).

- Factors in category IIB included the following:
  - Histologic type,
  - Histologic features associated with microsatellite instability (msi) (ie, host lymphoid response to tumor and medullary or mucinous histologic type),
  - High degree of msi (msi-h),
  - Loss of heterozygosity at 18q (dcc gene allelic loss), and
  - tumor border configuration (infiltrating vs pushing border).
- Factors grouped in category III included the following:
  - DNA content, all other molecular markers except loss of heterozygosity 18q/DCC and MSI-H,
  - Perineural invasion,
  - Microvessel density,
  - Tumor cell–associated proteins or carbohydrates

Among the numerous cell proteins and carbohydrate markers that have been reported in colorectal cancer, none have been extensively studied in clinical trials.
This class of tumor markers includes all the following substances:
Class I HLA molecules
Class II HLA molecules
CA 19–9
CA 72–4
Sialyl Lex
Sialosyl-Tn
Urokinase-type plasminogen activator
Plasminogen activator inhibitor 2
Glycoprotein 72
P-glycoprotein (multidrug resistance gene product)
MUC-1 mucin
E-cadherin
a-Catenin
Integrins
Type IV collagen
Gelatinase B (metalloproteinase-9)
Laminin
Tenascin
Autocrine mobility factor receptor (gp78)
Phospholipase C
Secretory component of immunoglobulin A
Metallothionein
EGF-R
Gastrin receptor
Somatostatin receptors
Sucrase-isomaltase
Cathepsin B, L, and D (cysteine/aspartyl proteases)
Ferritin
CD44
Vitamin D receptor protein

Cytokeratin 20

- ❖ Peritumoral fibrosis,
- ❖ Inflammatory response,
- ❖ focal neuroendocrine differentiation,
- ❖ Nuclear organizing regions,
- ❖ Proliferation indices.

- Category IV factors includes
  - ❖ Tumor size
  - ❖ Gross tumor configuration.
  - ❖ Molecular markers: Tumor suppressor genes (*LOH 1p/p53*, *LOH 8p*, *LOH 1p*, *LOH 5q*)

    Cyclin-dependent kinase inhibitor genes (*p27*, *p21*)
    Oncogenes (K-*ras*, c-*myc*)
    Apoptosis and cell suicide-related genes (*bcl*-2; *BAX*)
    DNA synthesis-related genes (thymidylate synthase; thymidine phosphatase)
    Transforming growth factors (TGF) and epidermal growth factor receptor (EGF- R) genes (*TGF*-a, *TGF*-b, c*erb*-b*/her2/neu*, *EGF-R*)
    Angiogenesis-related genes (vascular endothelial growth factor)
    Adhesion molecule and glycoprotein genes (CD44, Ecadherin, sialo-Tn antigen)
    Matrix metalloproteases and inhibitors (urokinase-type plasminogen activator)
    Metastasis suppressor genes (*nm23-H1*)

(b) Management of burst abdomen.

Answer. Burst abdomen describes partial or complete postoperative separation of abdominal wound closure.

Most bursts occur between the 6^{th} and 9^{th} post operative day.

Diagnosis : burst abdomen occurs mostly in patients undergoing emergency operations. A pink serosanguinous discharge from the laparotomy wound and a sensation of somethinh giving way in abdomen are indicators for burst abdomen.

Risk factors: Preoperative:

Male sex	Obesity	Jaundice	Smoking
Age >50 years	Diabetes	Malnutrition	COPD
Emergency operations	Renal failure	Corticosteroid use	

Operative:

- Surgical technique: cut through of the suture is the most common cause.
- Incision type: no significant difference in paramedian versus median and upper abdomen versus lower abdomen incisions.
- Closure: mass closure showed low incidence of hernia and dehiscence.
- Interrupted versus continuous: continuous suturing technique gives good results in elective setting. However, interrupted sutures are better in emergency surgery.
- Suture type: there is no significant difference between slowly absorbable or non-absorbable suture material but absorbable sutures are associated with less incidence of suture pain and sinus formation.

- Suture length to wound length ratio: Jenkins rule states that ratio of suture length to wound length should be 4:1, if the interval between stitches is 1cm and tissue bite is 1cm from the edge of incision.

Postoperative factors:

- Elevation of intra-abdominal pressure: Sudden forceful acts like vomiting, coughing, sneezing, retching, straining, distension due to ileus, urinary retention, ascites , fecal impaction, UGI endoscopy will lead to elevation of IAP.
- Radiation therapy: delayed wound healing occurs in patients who underwent radiotherapy as there is decreased collagen synthesis. Peri-operative regional radiotherapy does not affect fascial healing, if given 2 weeks before or after the operation.
- Anti-neoplastic agents: recent studies have shown no effect of chemotherapeutic drugs on wound healing.
- Wound infection: infection is considered the biggest enemy of wound healing and a major cause of wound dehiscence.

Management of burst abdomen: The basic treatment principle for repair of the disrupted wound is re-suturing of wound edges.

- Non-operative management: it is utilized for unstable patients, or where there is no evisceration, or fascial closure is not possible because of bowel oedema.
  - Abdominal binder to support disrupted abdominal wound.
  - Gauge packing/ covering the gut with a sterile moist occlusive dressing. This allows abdominal wall to granulate which may be skin grafted later. A ventral develops which is treated after 6 months, when the inflammatory process subsides facilitating entry into the abdomen.
- Operative treatment: Heavy non-absorbable sutures like Nylon or Polypropylene no.1 is used.Wide interrupted sutures with bites of 3cm from edge at a distance of 3cm or less are taken.External sutures are removed after 3 weeks.

The unclosable abdomen: sometimes it is difficult to close the abdomen because of

- Gross abdominal sepsis.
- Major abdominal trauma.
- Retro-peritoneal heamatoma.
- Loss of abdominal wall due to necrotizing fasciitis.

Various techniques of temporary abdominal closures (TAC) are used.

- Skin only closure: this is done when patient needs re-exploration within 24 hours.
- Bogota bag:This is made by gas sterilized urobag which is cut in oval shape and sutured to skin or rectus sheath. The advantage of this method of closure are easy availability, low cost, nonadherence to gut, prevention of evisceration and the ease of application.

Disadvantages of the technique are tearing of the skin, adherence of bowel to abdominal wall and no control of third space fluid loss which leaks out and produces hypothermia and hypovolemia.

- Mesh closure: its advantages include use of ploacement and facilitation of reexploration. There are three types of meshes in use
  - Absorbable mesh: it is used for temporary closure and good for infected abdomen but incisional hernia is inevitable.

- ❖ Polypropylene mesh: it is a nonabsorbable mesh with risk of erosion to bowel and fistula formation. It resists infection and forms dense adhesions.
- ❖ PTFE: the main advantage of PTFE is relativefreedom from adhesion formation. It is soft and pliable and conforms to the defect very well. The disadvantages are poor tolerance to infection, inability to skin graft and high cost.

- ➢ Witmann patch: this allows re-exploration and facilitates closure of abdomen with serial narrowing and trimming of mesh. The incidence of IAH and ACS are high with Witmann patch.
- ➢ Vacuum pack: it is a modification of Bogota bag. A nonadherent polyethylene sheet with fenestrations is placed over the viscera and covered with moist towels. Two silicone drains are placed over the towels and wound issealed with iodoform impregnated adhesive dressings. Continuous wall suction is applied at 100-150 mm Hg. Patients are serially re-explored at the time of change of dressings.
- ➢ Vacuum assisted closure (VAC) therapy: here primary closure of abdominal wall can be achieved.
- ➢ Sure closure skin stretching system application.
- ➢ Delayed primary fascial closure: this can be done within 7-10 days, if abdominal cavity can be closed without significant rise in intra-abdominal pressure.
- ➢ Fabian protocol: it provides a staged care of the burst abdominal wound. The patients are then brought after 6-12 months for abdominal wall reconstruction.
  - o Stage I: Mesh placement(Vicryl/Dacron)
  - o Stage II: Mesh removal or allowance of granulation tissue to grow.
  - o Stage III: Split thickness skin graft if granulation tissue is adequate.
  - o Stage IV: Definite abdominal reconstruction.

(c) Tumour markers. Answer. See ans. of Q.3(e) of Paper-I of May,2010.

3. Write short notes on: 10 x 3

(a) Port site recurrences in Laparoscopic Surgery.
(b) Ultrasonic dissection in Surgery – Technique, advantages and disadvantages.
(c) Gene therapy for cancer.

a) Port site recurrences in Laparoscopic Surgery.

Answer. Port site recurrence

Background: The advent of laparoscopic surgery and the increasing use in intraabdominal and thoracic malignancy has brought forward an un common problem of port site recurrence of the primary tumor.The biology,etiology,pathogenesis,treatment is discussed below.

The diagnosis must ascertain

1.Histologically proven tumor deposit

2. A localized circumscribed subcutaneous tumor growth of the abdominal or thoracic wall occurs within the fibrous tissue of the scar as the consequence of a trocar or other sites after laparoscopy,thoracoscopy or mediastinoscopy.

3.Early tumor recurrence(<200 days)

4.Port site metastasis is not identical to peritoneal metastasis,serosal invasion,skin mets etc.

Etiology & Pathogenesis:

The deposition of metastasis in port site can be attributed due to

1.Increased circulation to parieties in laparoscopic surgery

2.Chimney effect: At all times a exodus of the insufflation gas tends to spread the tumor to port site via aerosols.

3.Contamination of port by tumor seedling(though not conforming to definition)

Presentation

The revival of the same histologic nature of tumor along the site of extraction of the specimen/unrelated to extraction site may occur in early recurrence. Late recurrence is un common.

Diagnosis: As this is diagnosed during follow up of epithelial cancer the diagnosis is mostly clinical followed by cross sectional imaging. An excisional biopsy proves the diagnosis.

Treatment: Surgical excision of the port site recurrence with suitable margin (at least 1cm) along with removal of any intraabdominal recurrence if any.

Prognosis: With a few reports available only port site metastasis does not have a favorable prognosis fpr epithelial cancers. Though they behave differently from recurrence and thus the longevity is much more than recurrence.

Prevention of Port site recurrence:

Before resection:
Proper patient selection (exclude patients with very large lesions or lesions invading adjacent organs)
Adequate training and experience of surgeon and assistants
Careful tumor localization (tattoo, barium enema, etc.)
Proper port placement (position, angle, incision size, etc.)
Anchor all ports (grips, threaded ports, or skin sutures)
During resection:
Avoid gas leaks
Identify tumor-bearing segment (intraoperative colonoscopy if needed)
Avoidance of direct tumor handling or manipulation
Sound surgical techniques
Respect of oncologic principles (closed resection, en bloc resection with lymphatic drainage, proximal vessel control, clearance of resection margin)
Thorough irrigation and suctioning of abdomen
After resection:
Adequate-sized incision for removal of specimen
Wound protector or specimen bag
Macroscopic and, if need be, microscopic analysis of resected specimen
Thorough irrigation of ports and abdomen before closure (cytotoxic solution or saline)
Fascial and peritoneal wound closure (for ports 10 mm or larger)

(b) Ultrasonic dissection in Surgery – Technique, advantages and disadvantages.

Answer. Ultrasonic dissectors are of two types: low power which cleaves water containing tissues by cavitations leaving organized structures with low water content intact, e.g. blood vessels, bile ducts etc.; and high power systems which cleave loose areolar tissues by frictional heating and thus cut and coagulate the edges at the same time. Thus low power systems are used for liver surgery (Cusa, Selector) and do not coagulate vessels. High power systems (Autosonix, Ultracision) are used extensively especially in Fundoplication and laparoscopic colon surgery. It is important to remember that high power ultrasonic dissection systems may cause collateral damage by excessive heating and this is well documented in clinical practice.

Ultrasonic surgical dissection allows coagulation and cutting with less instrument traffic (reduction in operating time), less smoke and no electrical current.

- Mechanical energy at 55,500 vibrations / sec.
- Disrupts hydrogen bonds & forms a Coagulum
- Temperature by Harmonic Scalpel - 80 - 100 ° C
- Temperature through Electro coagulation - 200 - 300 ° C
- Collateral damage
- Tissue necrosis

The ultrasonic shears (harmonic scalpel) is ideal for dividing and simultaneously sealing small and medium vessels by tamponade and heat. However, larger vessels, greater than 2mm in diameter, need additional measures (clips, tie or staple) to control bleeding. Other disadvantages of the harmonic scalpel include lack of tissue selectivity and relatively expensive. Ultrasonic dissecting applicators are also designed in hook, spatula or ball coagulator shapes.

Harmonic scalpel: The Cavitational Ultrasonic Aspirator has the advantage of removing debris and is tissue selective e.g. divides liver but spares bile ducts and vessels. It affords safe, rapid dissection with reduction in tissue damage and blood loss compared to the harmonic scalpel.

The problems associated with its use are evacuation of the pneumoperitoneum together with vibration and irrigation which cloud the telescope lens necessitating frequent cleaning.

(c) Gene therapy for cancer.

Answer. Advances in understanding and manipulating genes have set the stage for scientists to alter a person's genetic material to fight or prevent disease. Gene therapy is an experimental treatment that involves introducing genetic material (DNA or RNA) into a person's cells to fight disease. Gene therapy is being studied in clinical trials (research studies with people) for many different types of cancer and for other diseases. It is not currently available outside a clinical trial.

Strategies of gene therapy for cancer

1. Enhancing the immunogenicity of the tumor, for example by introducing genes that encode foreign antigens.
2. Enhancing immune cells to increase anti-tumor activity, for example by introducing genes that encode cytokines.
3. Inserting a "sensitivity" or suicide' gene into the tumor, for example by introducing the gene that encodes HSVtk.
4. Blocking the expression of oncogenies, for example by introducing the gene that encodes antisense K-RAS message.
5. Inserting a wild-type tumor suppressor gene, for example P53 or the gene involved in Wilm' tumor.
6. Protecting stem cells from the toxic effects of chemotherapy, for example by introducing the gene that confers MDR-1.
7. Blocking the mechanisms by which tumors evade immunological destruction, for example by introducing the gene that encodes antisense IGF-1 message.
8. Killing tumor cells by inserting toxin genes under the control of a tumor-specific promoter, for example the gene that encodes diphtheria A chain.

Approaches to ex vivo gene transfer

(1) Genetically engineered tumor cells

Various groups are investigating the production of autologous cellular vaccines for the treatment and prevention of cancer. This is most commonly attempted by surgically removing tumor cells from the patient, growing them in tissue culture and inserting immunostimulatory genes in vitro. These cells are then reinjected into the patient in an effort to induce a significant systemic immune response that will both destroy tumor cells and protect the patient against a recurrence of the tumor. Treating cells that produce cytokines has been shown to result in systemic immunity in mice. Alteration of syngeneic tumors with the genes that encode IL-1 b, IL-2, IL-4, IL-6, TNFa, GM-CSF or r-interferon results in immunological destruction of the tumor cells in vivo.

In human gene therapy trials, patients are injected with either autologous or allogeneic genetically modified tumor cells. These trials involved the insertion of retroviral vectors carrying the gene that encodes either IL-2, TNFa or GM-CSF into melanoma, colorectal renal cell carcinoma, neuroblastoma or breast cancer cells in vitro. One modification of this technique is the insertion of the gene for either IL-2 or IL-4 into autologous fibroblasts, which are then mixed with irradiated tumor cells from the patient and reinjected. This approach has the advantage that growing fibroblasts in vitro is much easier than culturing tumor cells from a large number of individuals. Besides modifying tumor cells to produce immune activating cytokines, another strategy is to block the production of insulin-like growth factor-1 (IGF-1). Many tumors such as breast cancer produce high levels of IGF-1. Insertion of an antisense gene that stops production of IGF-1 in the tumor allows immunological rejection of the genetically altered tumor after

reimplantation. Destruction of the tumor is mediated by cytotoxic T lymphocytes. The precise mechanism by which IGF-1 mediates tumor protection in vivo remains unclear.

(2) Genetically engineered T lymphocytes

T lymphocytes have the capacity to hone in on tumor tissue. This property has been used to deliver cytokines directly to tumor masses for human gene therapy. The secretion of cytokines locally at the tumor site by the effector T lymphocytes will enhance their anti-tumor activity and avoid the side-effects that result from the systemic administration of cytokines. For the trial of TNF-modified tumor infiltrating T lymphocytes, T lymphocytes are difficult to transduce with retroviral vectors and tend to downregulate expression of the cytokine gene carried by the vector.

These two problems of poor gene transfer efficiency and poor cytokine expression have so far limited the application of this approach, and have shifted the emphasis from modification of T lymphocytes toward the genetic alteration of tumor cells, which are much easier to grow in culture and more readily engineered.

(3) Insertion of a sensitivity gene

Gene therapy uses the genes to activate a relatively nontoxic pro-drug to form a highly toxic agent. The most widely studied system uses the thymidine kinase gene of the Herpes simplex virus (HSVtk). The HSVtk gene confers sensitivity to the anti-herpes drug, ganciclovir (GCV), by phosphorylating GCV to a monophosphate form (GCV-MP). Phosphorylation to the triphosphate form(GCV-TP) by cellular kinases results in inhibition of DNA polymerase, and leads to cell death. In this procedure, GCV kills tumor cells which express KSVtk, and the adjacent cells that lack the gene are also destroyed. This is termed the bystander effect phenomenon. To use the bystander effect to kill human cancer in vivo, the irradiated ovarian tumor cells that contain the HSVtk gene will be injected into the peritoneal cavity of patients, who will be given GCV. These HSVtk-expressing cells will destroy bystander tumor cells in vivo.

(4) Protection of hematopoietic stem cells

Protection of hematopoietic stem cells (HSCs) from the toxic effects of chemotherapy by using the gene that confers multiple drug resistance type 1(MDR-1) is another possible strategy for human cancer therapy. The MDR-1 gene will be isolated from tumor cells, where it functions to pump chemotherapy drugs (including daunorubicin, doxorubicin, vincristine, vinblastine, VP-16, VM-26, taxol and actinomycin-D) from within the cell. Transfer of a retroviral vector carrying the MDR-1 gene into bone marrow stem cells and their subsequent reintroduction will protect stem cells in vivo from the effects of large doses of taxol.

Genetic alteration of cancer cells in situ

(1) Liposome-mediated gene transfer

The genetical modification of tumors in situ involves the direct injection of liposomes containing an allogene that encoded HLA-B7, a foreign antigen that is transiently expressed on the cell surface and includes an immune reaction against the altered tumor cells. Anti-tumor immune response is significantly increased when some of the tumor cells express foreign antigens on their cell surface. The transient expression of immunostimulatory genes in tumors might have potential as a treatment and as a vaccination against certain malignancies.

(2) Retrovirus-mediated gene transfer

In vivo gene transfer using murine retroviral vectors has been applied to the treatment of brain tumors.In this process, murine fibroblasts that are actively producing retroviral vectors, so-called retro viral vector producer cells or VPCs, are implanted directly into growing tumors. The gene transferred by the retroviral vectors into the surrounding tumor cells is the HSVtk gene. The HSVtk gene should integrate only into the proliferating tumor cells because retrovirus-mediated gene transfer is limited to mitotically active cells. This technique resulted in transfer of the gene for HSVtk into 30-60% of brain tumor cells and was capable of mediating complete tumor destruction in 80% of patients.

More than 50% of the cancers can be eliminated completely, At least 10% of cells in a tumor contain HSVtk, adjacent tumor cells that do not contain HSVtk are destroyed through the bystander effect. No associated systemic toxicity or evidence of systemic spread of the retroviral vectors is seen with this form of in vivo gene transfer. So far, however, it is not clear whether this gene delivery system will suffice to eradicate the larger, infiltrative human tumors.

Two protocols for in vivo gene transfer for cancer therapy have been approved for clinical trials. Both entail the direct injection of a supernatant containing a retroviral vector (RV) into tumor deposits. One group will inject two different RVs into endobronchial non-small-cell lung cancers.The vectors will carry genes that target the genetic mechanisms responsible for the malignancy: for example, if the lung tumors are deficient in expression of the P53 tumor suppressor gene, this gene will be used. In lung cancers that overexpress the K-RAS oncogene, a vector containing an antisense K-RAS gene will be used. Experiments in vitro have demonstrated that the introduction of both such vectors can result in decreased tumorigenicity. Another group will inject a RV containing a vector that encodes r-interferon directly into melanoma deposits.

Some uses:

1. Gene Therapy Alters Immune Cells for Treatment of Advanced Melanoma.
2. Gene therapy is also used for human breast cancer.
3. Can also be used to treat prostate cancer.
4. Used in oral cancers.
5. HLA-B7 is the most common gene used in bladder cancer.
6. Novel Suicide Gene Therapy Used to Treat Malignant Brain Tumors.
7. Gene therapy-based sFLT-1 approach for carcinoma of the ovary.

8. It is also used in liver cancer.
9. Mesothelioma may be particularly well suited for gene therapy treatment owing to its accessibility, allowing both intrapleural and intratumoral gene delivery.
10. It is also used in blood cancers.

4. Discuss the current evidence based management of locally advanced breast cancer. (20)

Answer. Advanced Locoregional Regional Breast Cancer (Stage IIIa or IIIb):

Women with stages IIIa and IIIb breast cancer have advanced locoregional breast cancer. Stage IIIa patients are divided into those who have operable disease and those who have inoperable disease. Surgical therapy for women with operable stage IIIa disease is usually a modified radical mastectomy, followed by adjuvant chemotherapy, followed by adjuvant radiation therapy. Adjuvant chemotherapy is used to maximize distant disease-free survival, while radiation therapy is used to maximize locoregional disease-free survival. In selected stage IIIa patients, initial (neoadjuvant) chemotherapy is used to reduce the size of the primary cancer and permit conservation surgery. For inoperable stage IIIa and for stage IIIb breast cancer, neoadjuvant chemotherapy is used to decrease the locoregional cancer burden and may permit subsequent surgery to establish locoregional control. In this setting, surgery is followed by adjuvant chemotherapy and adjuvant radiation therapy.

Treatment for locally advanced breast cancer will usually involve a combination of treatments:

- Chemotherapy
- Breast surgery
- Radiotherapy
- Targeted therapies
- Hormonal therapies.

In addition, some women choose to use complementary therapies or may be involved in a clinical trial.

Chemotherapy: Treatment for locally advanced breast cancer often starts with chemotherapy. The aim of giving chemotherapy first is to make the breast cancer smaller, and to destroy any cancer cells that may be elsewhere in the body, but cannot be detected using routine tests.

Breast surgery: Breast surgery may be recommended for some but not all women with locally advanced breast cancer. Most women who do have surgery will have the breast removed completely (mastectomy).

Breast reconstruction may be possible once all initial treatment has been completed but this should be discussed with the treating doctors.

Radiotherapy: Radiotherapy may be used before or after surgery for locally advanced breast cancer to destroy cancer cells in the breast, armpit, neck or surrounding areas.

Targeted therapies: Targeted therapies (also known as targeted therapies) are drugs that stop the growth of particular types of cancer cells. Targeted therapies are only suitable for some women. They may be used with other breast cancer treatments for locally advanced breast cancer.

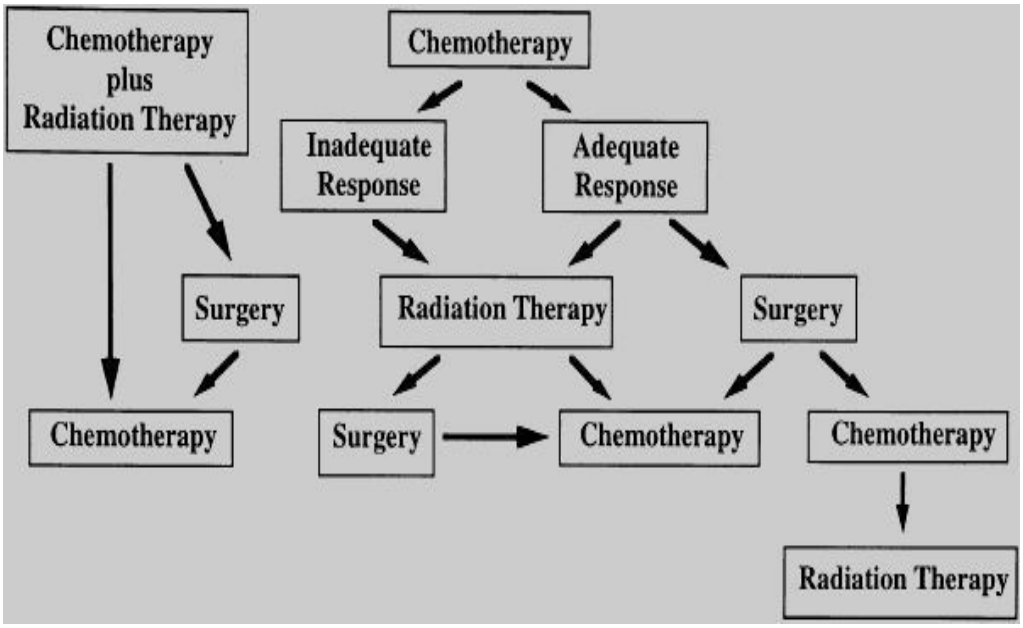
Hormonal therapies: Hormonal therapies are drugs used to treat women who have hormone receptors on their breast cancer cells. Hormonal therapies may be used alone or with other breast cancer treatments for locally advanced breast cancer.

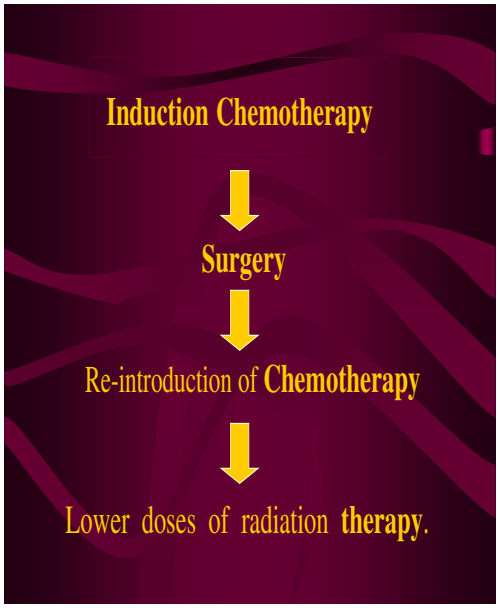
The treatment of locally advanced breast cancer (LABC) must include two major goals: control of locoregional disease and eradication of occult systemic metastases. The patterns and risk of locoregional recurrence after mastectomy are functions of the size of the primary tumor, the degree of regional nodal involvement, the presence or absence of skin or chest wall involvement,and the type of surgical procedure performed. Patients with LABC have historically had a poor prognosis, and some are initially inoperable. They include patients with evidence of multiple (≥4) or matted axillary lymph nodes or involvement of the second-echelon nodal basins of the infraclavicular , supraclavicular , and internal mammary lymph nodes (IMN). A clinically distinct but similarly high-risk type of LABC is inflammatory breast cancer. Overall, LABC is very heterogeneous, with highly variable tumor sizes and nodal status.

- Breast imaging is important to determine the extent of primary disease and to evaluate for multifocal, multicentric, or contralateral breast cancer. A bilateral diagnostic mammogram with compression or magnifications views if needed is essential for all breast cancer patients.
- Ultrasound (US) may provide additional information regarding breast malignancy and may also be used to evaluate the axilla. US-guided biopsy may be performed for enlarged lymph nodes or lymph nodes demonstrating architectural distortion. The sensitivity of US is low, and therefore patients with negative axillary lymph nodes by US will still require surgical evaluation with sentinel lymph node biopsy or axillary lymph node dissection.
- Magnetic resonance imaging (MRI) has been increasingly used and recognized as an important tool in evaluating the extent of disease for LABC. It is useful for detecting abnormal lymph nodes and contralateral disease, and it may aid in determining if a mastectomy is feasible without neoadjuvant therapy. MRI can also be used for evaluating response to neoadjuvant chemotherapy.
- Because of the high probability of, metastatic disease in patients with LABC, imaging studies including bone scan and computed tomography (CT) of the upper abdomen and chest are useful.
- Positron emission tomography (PET) is sometimes used in lieu of CT of the chest and abdomen, and sometimes bone scan, although there is not universal agreement on which of these modalities may be preferred.

For operable patients undergoing mastectomy without irradiation, certain subgroups at higher risk for recurrence were identified. The clinical and pathologic status of axillary nodes was found to be an important indicator of preoperative RT was able to convert over 80% of patients to operable status and allow them to undergo mastectomy.

A randomized trial in stage III breast cancer patients from Helsinki has clearly shown the efficacy of combining all three therapeutic modalities of surgery, chemotherapy, and RT. In this trial, 120 patients with stage IIIA breast cancer were randomized to one of three arms after modified radical mastectomy: locoregional irradiation alone, systemic VAC (vincristine, adriamycin, cyclophosphamide) chemotherapy (with or without levamisole), or both VAC and irradiation. At both 3 and 5 years, RT reduced local failures relative to the chemotherapy arm, whereas VAC reduced the number of distant failures. The best DFS and local control rates were seen in the combined-modality arm.





Factors to Consider in Evaluating Patients for Treatment with Breast-Conserving Therapy*

Histopathologic Factors	Clinical Factors	Patient Factors
Presence and extent of microscopic involvement of resection margins	Tumor size in relation to breast size	Pregnancy
Histologic features of the tumor	Presence of nipple discharge	Patient preference
Tumor location	Age	
Presence of multiple lesions on palpation	Collagen vascular disease	
Radiological evidence of diffuse or multi centric disease .)	Other medical conditions	

- Although screening mammography has resulted in proportionally fewer cases, locally advanced breast cancer remains a difficult clinical problem due to the high rate of relapse and low rate of overall survival.
- The range of disease encompassed by the term locally advanced is wide and includes patients with large, but operable, primary tumors to patients with rapidly progressing inflammatory carcinomas, who have very different prognoses.
- Thus, treatment decisions must be tailored to the individual patient. Of critical importance in all cases of locally advanced breast tumors is coordination of care among medical oncology, surgical oncology, and radiation oncology, as the vast majority of patients need chemotherapy, surgery, and radiotherapy.
- With continued support of basic and clinical research, further advances in the treatment and prognosis of women with locally advanced breast cancer will occur.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES

MS (General Surgery) Examination, 2010

PAPER IV

Time Allowed: 3 Hours **Full Marks: 100**

Attempt all questions

1. **Discuss the current status of different types of bariatric surgery. 20**

2. **Discuss laparoscopic management of acute abdomen. 20**

3. **Write short notes on the following: 6 x 5**
   a) **Drains in Gastrointestinal surgery.**
   b) **Acute acalculous cholecystitis.**
   c) **Sterilisation of Endoscopes.**
   d) **Carcinoid syndrome.**
   e) **Small bowel enema.**

4. **Write brief answers on: 4 x $7^1/_2$**
   (a) **Management of skin loss.**
   (b) **Complications of chest injury.**
   (c) **Breast ultrasound.**
   (d) **Intra-abdominal sepsis.**

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES
MS (General Surgery) Examination, 2010

May 2010

PAPER IV

Time Allowed: 3 Hours **Full Marks: 100**

Attempt all questions

1. **Discuss the current status of different types of bariatric surgery. 20**

Answer. Eligibility Criteria for bariatric Surgery

▪ BMI > 40; or BMI > 35, with obesity-related comorbidity
▪ Failed previous attempts at nonsurgical weight reduction
▪ No active history of alcohol or substance abuse
▪ Realistic expectations of outcomes and commitment to long-term follow-up
▪ Acceptable medical risk for surgery

Bariatric Operations: Mechanism of Action
Restrictive
Vertical banded gastroplasty (VBG) (historic purposes only)
Laparoscopic adjustable gastric banding (LAGB)
Largely Restrictive/Mildly Malabsorptive
Roux-en-Y gastric bypass (RYGB)
Largely Malabsorptive/Mildly Restrictive
Biliopancreatic diversion (BPD)
Duodenal switch (DS)

Vertical Banded Gastroplasty

This procedure has now largely been abandoned.

Causes are:

- Poor long-term weight loss,
- A high rate of late stenosis of the gastric outlet, and
- A tendency for patients to adopt a high-calorie liquid diet, thereby leading to regain of weight.
- Weight regain several years after surgery is typically due to a breakdown of the vertical staple line and patients report new dietary freedom. Revisional surgery and

conversion to a gastric bypass usually result in durable weight loss but can be associated with a higher rate of perioperative complications.

Adjustable Gastric Banding

- The AGB procedure may be performed with any of three types of adjustable bands.
- They all work on the principle of restriction of oral intake by limiting the volume of the proximal part of the stomach.
- Their advantage over the traditional vertical banded gastroplasty is adjustability.
- Gastric banding is a least-invasive operation with 50-60% excess weight loss over 5 years but long term results are unknown and the complication rate may be cumulative.

Roux-en-Y Gastric Bypass

Essential Components of Roux-en-Y Gastric Bypass
Small proximal gastric pouch
Gastric pouch constructed from the cardia of the stomach to prevent dilation and minimize acid production
Gastric pouch divided from the distal part of the stomach
Roux limb at least 75 cm in length
Enteroenterostomy constructed to avoid stenosis or obstruction
Closure of all potential spaces for internal hernias

Results of Roux-en-Y Gastric Bypass

- RYGB has an established track record longer than that of any other operation. Its Recovery after RYGB is improved after a laparoscopic approach.
- This improvement is largely related to the decrease in postoperative pain experienced by patients after laparoscopic RYGB versus open RYGB.
- Weight loss with both approaches was comparable (68% loss of excess weight for laparoscopic RYGB versus 62% loss of excess weight for open RYGB).
- Another important advantage of the laparosCopic approach for RYGB is a decrease in the incidence of wound complications and incisional hernia seen after RYGB.
- There is no difference in the rate resolution of comorbid conditions or weight loss between the two procedures. after RYGB.
- Resolution of comorbid conditions after open and laparoscopic RYGB has generally been excellent.
- Rate of resolution and improvement of diabetes, hypertension and obstructive sllep apnoea is significant.
- Metabolic syndrome is cured or ameliorated with gastric bypass.

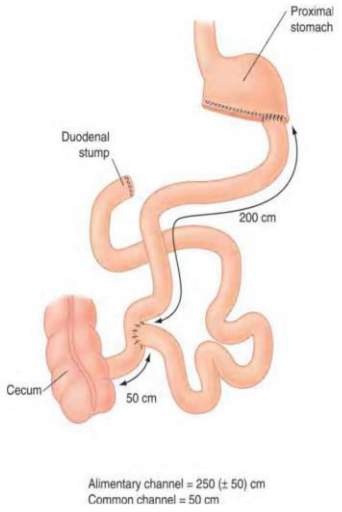
- Hyperlipidemia, hypercholesterolemia, and hypertriglyceridemia has been improved significantly in patients undergoing RYGB.
- RYGB has also been shown to resolve the symptoms of pseudotumor cerebri, as well as cure the difficult problem of venous stasis ulcers.
- Immediate resolution of symptoms of GERD occurs in more than 90% of cases.

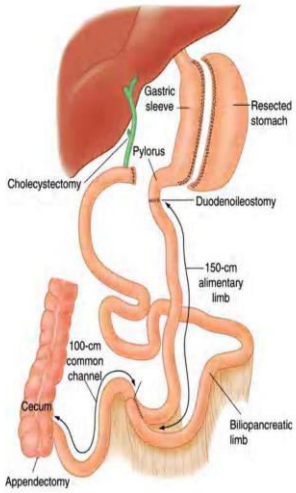
Biliopancreatic Diversion

- BPD produces weight loss based primarily on malabsorption, but it does have a mild restrictive component.
- The intestinal tract is reconstructed to allow only a short so-called common channel of the distal 50-cm of terminal ileum for absorption of fat and protein.
- The alimentary tract beyond the proximal part of the stomach is rearranged to include only the distal 200 cm of ileum, including the common channel.
- The proximal end of this ileum is anastomosed to the proximal end of the stomach after performing a distal hemigastrectomy.
- The ileum proximal to the end that is anastomosed to the stomach is in turn anastomosed to the terminal ileum within the 50- to 100-cm distance from the ileocecal valve, depending on the surgeon's preference and the patient's size.

Duodenal Switch

- This modification was developed to help lessen the high incidence of marginal ulcers after BPD.
- The mechanism of weight loss is similar to that of BPD.
- An appendectomy is followed by measurement of the terminal ileum. Notably in the DS procedure, the common channel is 100 cm and the entire alimentary tract is 250 cm.
- However, the major difference between DS and BPD is the gastrectomy and the proximal anatomy. Instead of a distal hemigastrectomy, a sleeve gastrectomy of the greater curvature of the stomach is performed. This procedure is done as the initial part of the operation because if the patient exhibits any intraoperative instability, the operation can be discontinued after the sleeve gastrectomy alone.
- A two-stage DS has been used in patients who have an extremely high BMI and are high operative risks.
- The goal is to produce a lesser curvature gastric sleeve with a volume of 150 to 200 mL.
- After sleeve gastrectomy, or preceding it in smaller patients, the duodenum is divided with the stapler approximately 2 cm beyond the pylorus.
- The distal connections are performed as for BPD. The distal anastomosis is created at the 100-cm point proximal to the ileocecal valve.
- The proximal anastomosis is created between the proximal end of the 250 cm of terminal ileum and the first portion of the duodenum. The duodenoileostomy is an antecolic end-to-side duodenoenterostomy.





Scematic diagram of Biliopancreatic Diversion **Scematic diagram of Duodenal Switch**

Results of Biliopancreatic Diversion/Duodenal Switch

- Excess weight loss (EWL)after BPD/DS is the highest of all the bariatric operations
- In a recent study comparing morbidly obese patients with a BMI greater than 50 kg/m^2, there was significantly more EWL at 12, 18, and 24 months postoperatively after DS than after RYGB.
- Thus, some surgeons argue that super-obese patients fare better and maintain weight loss better in the long term after undergoing DS than after other bariatric operations.
- BPD/DS has also been highly effective in treating comorbid conditions, including hypertension, diabetes, lipid disorders, and obstructive sleep apnea.
- Lipid disorders and type 2 diabetes are almost uniformly resolved after BPD/DS.
- Hypertension is cured in 83.4% and obstructive sleep apnea resolves in 91.9% of patients.
- After BPD, patients typically have between two and four bowel movements per day. Excessive flatulence and foul-smelling stools are the rule.
- Relatively selective malabsorption of starch and fat provides the major mechanism of weight loss, although the partial gastric resection does contribute a restrictive component to the operation.
- When protein malnutrition does occur, the common channel may need to be lengthened with a reoperation.
- Major considerations for achieving excellent results in patients offered BPD/DS include the ability to reliably monitor these patients, as well as confirm that they are being compliant with the recommendations to take appropriate vitamin supplements.
- Supplements include multivitamins, as well as at least 2 g of oral calcium per day. Supplemental fat-soluble vitamins, including D, K, and A, are indicated monthly as well.

- Because of a high incidence of morbidity and mortality in patients with a BMI greater than 60 kg/m^2 undergoing laparoscopic DS, surgeons developed the two-stage DS, with sleeve gastrectomy alone performed as the first stage to decrease morbidity in this super-obese patient population.

Other procedures:

- Endoluminal sleeve
- Jejunoileal bypass

Comments:

- Cultural factors, surgeon and patient preference drive choice of operation which should be based on the balance of risk and benefit.
- Bariatric surgery pays for itself within 3-4 years after surgery.
- Bariatric surgery patients have better long-term survival than obese controls.
- A number of procedures have been investigated for weight loss surgery but have not been totally accepted by the surgical community. Several surgeons have proposed a two-stage procedure for a super-obese patient, who often has a large liver that precludes safe retraction for gastric bypass, because of the recognition that the DS procedure has been associated with much higher mortality and morbidity rates in the super-obese (BMI >60 kg/m^2).
- Now, reports of vertical sleeve gastrectomy (VSG) alone suggest that weight loss is sufficient to preclude conversion to gastric bypass or DS.
- Gastric pacing has been performed in several trials but has not gained widespread acceptance. The concept is to stimulate gastric smooth muscle by implanting a pacemaker in the body of the stomach to induce early satiety, which reduces caloric intake and therefore results in weight loss.
- Increasingly, surgeons are observing effects of bariatric operations not just on the physical reduction of caloric intake or malabsorption. Alteration in comorbid conditions caused by metabolic processes may prove equally as important. For example, bariatric operations may have important metabolic components that alter the hormonal/cytokine/metabolic rate of patients.

2. Discuss laparoscopic management of acute abdomen. 20

Answer. The acute abdomen is characterized by the sudden appearance of abdominal complaints that oblige the surgeon to decide promptly whether to operate immediately,to treat conservatively or to observe the patient.

Possibilities for initial diagnosis of the acute abdomen:

Localized peritonitis

- Acute cholecystitis
- Acute appendicitis (right lower abdomen)

- Acute diverticulitis

Generalized peritonitis of unknown origin

Peritonitis by perforation
- Perforated gastroduodenal ulcer
- Perforated acute diverticulitis (colon sigmoid)
- Small intestinal perforation (typhoid, trauma)

Intestinal obstruction

Role of laparoscopic procedure in different causes of acute abdomen;
- Percutaneous transhepatic cholecystostomy is a valuable and effective procedure in a high – risk group of patients with acute cholecystitis.
- Early laparoscopic cholecystectomy is the treatment of choice in patients with acute cholecystitis with acceptable risk.
- Helical CT or an USG will resolve the diagnosis in many patients with right iliac fossa.
- All patients suspected of acute appendicitis should have an USG. Patients will be clinically divided in equivocal and unequivocal appendicitis.
- Unequivocal clinical diagnosis plus positive USG will require diagnostic laparoscopy followed by laparoscopic appendicectomy.
- Unequivocal clinical diagnosis plus negative USG will lead to clinical observation. Helical CT should be done to exclude other diagnoses. If the clinical condition worsens, a diagnostic laparoscopy should be performed.
- An equivocal clinical diagnosis will lead to clinical observation. If local peritonitis develop, a diagnostic laparoscopy should be performed.
- Diagnostic laparoscopy should be performed in all patients with signs of generalized acute abdomen in whom no clear cause is suspected after clinical, radiological and laboratory investigation. A correct diagnosis will dictate the right surgical approach.
- Laparoscopic closure of perforated duodenal ulcer, in experienced hands, confers superior short – term benefits in terms of postoperative pain and wound morbidity.
- Laparoscopic lavage and drainage in the acute management of perforated acute diverticulitis is a promising alternative to more radical procedures.
- Laparoscopy is an effective procedure for the treatment of acute small bowel obstruction with acceptable risk of morbidity and early recurrence, but conversion rates of 30-40% should be expected.

3. Write short notes on the following: 6 x 5

a) Drains in Gastrointestinal surgery.
b) Acute acalculous cholecystitis.
c) Sterilisation of Endoscopes.
d) Carcinoid syndrome.
e) Small bowel enema.

Answers.

(a) Drains in Gastrointestinal surgery.

Types of drains – based on:

- Rationale: Prophylactic and therapeutic drains.
- Mechanism: Open and closed drains.
    - Suction drains.
    - Sump suction drains.
- Drain materials: Polyurethane, Silicone, PTFE etc.

Reasons for the insertion of a drain:

- To remove unwanted fluid/exudates/pus/gas.
- To allow monitoring of fluid volume and quality.
- To promote tissue apposition.
- To allow diversion of body fluids.
- To facilitate subsequent access to a body space or cavity.
- To allow the injection of dye or contrast to provide diagnostic information about an underlying cavity or fistula.

Things to watch for in a patient with drain:

- Is the patient well?
- Has the drain been secured and is still secure?
- Are the signs of infection, excoriation or peritubal leakage at the skin exit site?
- Is the tube kinked or damaged?
- Is the drain connected properly?
- What is draining? – volume, colour, nature, smell.
- Has there been any change in the nature or volume of the effluent?
  - little or no output indicates that the drain is either blocked or that it has served its function. – excessive drainage may indicate formation of a fistula or tissue irritation.
- Is the vacuum for suction drains working?

Common complications of drains:

- ✓ Tissue damage during insertion and removal.
- ✓ Erosion into adjacent structures – blood vessels, bowel, anastomoses.
- ✓ Skin flap necrosis.
- ✓ Visceral herniation through the drain tract.
- ✓ Bacterial colonization and infection.
- ✓ Loss of fluid and electrolytes(e.g.through a T- tube).
- ✓ Pain.
- ✓ Migration or displacement of the drain(into the body cavity or extrusion).
- ✓ Accidental removal.
- ✓ Blockage.
- ✓ Fracture of the drain.
- ✓ Failure of vacuum in suction drains.
- ✓ Restricted patient mobility.

Key points:

- Use of drain after routine colorectal surgery cannot be recommended.
- The placement of a prophylactic drain following appendicectomy is not indicated, regardless of the severity of appendicitis.
- Prophylactic drain placement can not be advocated following simple cholecystectomy, either laparoscopic or open.
- Prophylactic drain placement following liver resection is not beneficial and may even be detrimental.
- In upper gastrointestinal and pancreatic surgery there is little evidence to support the use of drains.
- Currently, the placement of drains following incisional hernia repair has to be the discretion of the operating surgeon.
- Percutaneous drainage is the recommended treatment for symptomatic postoperative collections and when there are signs of infection**.(Recent**

(b) Acute acalculous cholecystitis.

Introduction: Acute acalculous cholecystitis (AAC) often complicates the course of ICU patients and those recovering from trauma or nonbiliary tract surgery. This disorder has a more fulminant course and is often associated with gangrene, perforation, and empyema. If left untreated, it has a mortality rate as high as 70%.

Sign/symptoms:

- Classic signs and symptoms of abdominal pain, right upper quadrant pain, and fever may be missing in critically ill patients.
- Laboratory tests such as bilirubin and leukocytosis may be elevated for different reasons. Unexplained fever and sepsis may be the only manifestation of acalculous cholecystitis.

Risk factors for AAC:

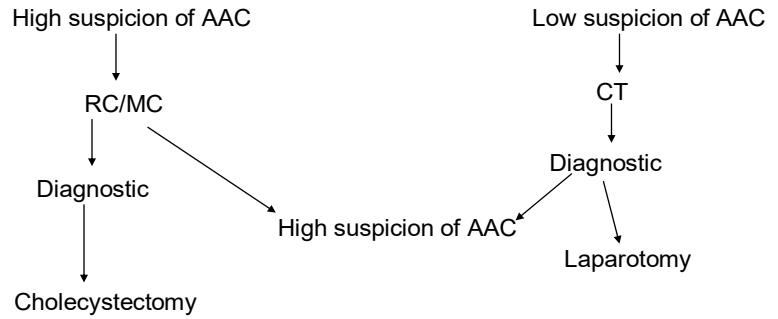
- Previous surgery and trauma,
- Visceral hypoperfusion,
- Mechanical ventilation,
- Biliary stasis and gallbladder dysmotility secondary to prolonged fasting,
- Total parenteral nutrition (TPN).
- Narcotics may play a role by causing spasm of the sphincter of Oddi.
- AAC can be caused by infection with cytomegalovirus or Cryptosporidium in patients infected with human immunodeficiency virus.
- Ischemia/hypoperfusion may play a role also in pathogenesis of AAC.
- Studies have shown a significant association between the duration of cardiopulmonary bypass and the incidence of AAC.

Diagnosis: There are various diagnostic modalities available.

- Radionuclide cholescintigraphy with intravenous morphine cholescintigraphy (RC/MC) has the highest sensitivity (90%), followed by CT scan (67%) and ultrasound (29%).
- If the suspicion for biliary sepsis and AAC is high, the initial test should be RC/MC and follow-up with CT to look at the remainder of the abdomen if RC/MC is normal. CT abnormalities include a dilated gallbladder with thickened walls, sludge in the gallbladder, and pericholecystic fluid outside of the gallbladder.
- If suspicion for AAC is low, CT is used to search for abdominal pathology. If CT is suggestive of AAC, follow with RC/MC.

Algorithm for evaluation of patients for suspected AAC:

AAC = Acute acalculous cholecystitis
RC/MC = Radionuclide cholescintigraphy/morphinechole scintigraphy
CT = Computed tomography



Treatment:

- Treatment is open or laparoscopic cholecystectomy. In critically ill patients, ultrasound-guided percutaneous cholecystotomy tube placement can provide temporary relief. The usual organisms include gram-negative bacteria.
- Antibiotics are used pending the result of cultures. Adequate coverage can be provided with ampicillin and gentamicin, ticarcillin/clavulanate, piperacillin/tazobactam, or ampicillin/sulbactam. If the patient is penicillin allergic, a combination of cefotetan or cefoxitin with another antibiotic that covers pseudomonas and anaerobes is adequate.

(c) Sterilisation of Endoscopes.

Answer. Introduction: The infection associated with endoscopic surgery – routine SSIs and Nontuberculous mycobacteria (NTMs).

Features to consider when purchasing a machine:

- The number of scopes which can be processed simultaneously.
- A cycle counter and fault indicator.
- A water treatment system which prevents recontamination of processed instruments during rinsing. Filtration using bacteria retaining filters with a pore size of 0.2 to 0.45u is satisfactory.
- A reliable, effective and simple machine disinfection system.
- An air-drying facility to expel fluids and dry the channels of the cope at the end of a cycle.
- A facility to irrigate the channels of the cope with alcohol before storage.
- A leak test facility.
- A print-out, of cycle parameters, which can be retained for quality assurance, records.

Cleaning and disinfection – practical recommendations

At the start of the day:

1. Instruments to be used should be checked.
2. If instruments have been thoroughly cleaned and disinfected at the end of the previous day, they should be put through an automated cleaning and disinfection process.
3. All channels be flushed with disinfectant.
4. The instrument should be fully immersed in disinfectant for the correct contact time.
5. Requires flushing manually using a 2 ml syringe and a channel adapter.
6. The valves that will be used during the list, ideally one set per case, should be disinfected in the same way.
7. After disinfection, copes and valves should be rinsed in bacteria free water ensuring all traces of disinfectant are removed from the channels, control body and eyepiece.
8. The instrument should then be plugged into the light source and connected to the suction pump. Air should be blown through all the channels to expel excess fluid.
9. The instrument should then be ready for use.

Cleaning and disinfection of endoscopes between cases:

- Before detaching the instrument the air/water channels should be flushed with water for min 15 seconds.
- After thorough flushing instrument should be checked for any leaks and other faults.
- Perform all the steps from 2-8 as mentioned before.

- All valves should be removed and cleaned individually with a cotton wood bud or small brush.
- The suction / biopsy channel should be cleaned properly prior to reinsertion.
- When the channels have been cleaned the suction and air/water ports must be cleaned.
- The instrument must be immersed in disinfectant for the correct contact time.
- The relevant work surfaces must be wiped clean.

Cleaning and disinfection of endoscopes after the last case:

- Leak test the cope, clean and disinfect with Cidex OPA (Orthophthaldehyde) / 2% Glutaraldehyde/Peracetic acid/Chlorinedioxide performing steps 2-8 as mentioned.
- Copes should be stored hanging vertically in designated ventilated cupboard, not in their transit cases.
- All valves used during the list should, after disinfection and rinsing, be dried with a cotton wool bud and lubricated with silicone oil. They should not be replaced in the endoscope case for storage.

Cleaning and disinfection of accessories is also essential.

Disinfectants used for endoscopes:

- Orthophthaldehyde.
- Formalin cabinets.
- Hydrogen peroxide gas plasma method.

(d) Carcinoid syndrome.

Answer. Introduction: The carcinoid syndrome is a relatively rare disease, occurring in fewer than 10% of patients with carcinoid tumors. The syndrome is most commonly associated with carcinoid tumors of the gastrointestinal tract, particularly from the small bowel, but carcinoids in other locations, such as the bronchus, pancreas, ovary, and testes, have also been described in association with the syndrome.

Sign/Symptoms:**The classic description of the carcinoid syndrome typically includes vasomotor, cardiac, and gastrointestinal manifestations.**

- A number of humoral factors are produced by carcinoid tumors, but those considered to contribute to the carcinoid syndrome include serotonin, 5-hydroxytryptophan (a precursor of serotonin synthesis), histamine, dopamine, kallikrein, substance P, prostaglandin, and neuropeptide K.
- Most patients who exhibit malignant carcinoid syndrome have massive hepatic replacement by metastatic disease. However, tumors that bypass the liver, specifically ovarian and retroperitoneal carcinoids, may produce the syndrome in the absence of liver metastasis.

- Common symptoms and signs include cutaneous flushing (80%); diarrhea (76%); hepatomegaly (71%); cardiac lesions, most commonly right heart valvular disease (41%-70%); and asthma (25%).
- Cutaneous flushing in the carcinoid syndrome may be of four varieties: diffuse erythematous, which is short lived and normally affects the face, neck, and upper chest; violaceous, which is similar to diffuse erythematous flush except that the attacks may be longer and patients may develop a permanent cyanotic flush with watery eyes and injected conjunctivae; prolonged flushes, which may last up to 2 to 3 days and involve the entire body and be associated with profuse lacrimation, hypotension, and facial edema; and a bright-red patchy flushing, which is typically seen with gastric carcinoids.
- The diarrhea associated with carcinoid syndrome is episodic (usually occurring after meals), watery, and often explosive. Increased circulating serotonin levels are thought to be the cause of the diarrhea because the serotonin antagonist methysergide effectively controls the symptom.

Treatment:

- Medical therapy for patients with malignant carcinoid syndrome is primarily directed toward the relief of symptoms caused by the excess production of humoral factors.Various long-acting analogues of somatostatin, such as octreotide (Sandostatin), relieve symptoms (diarrhea and flushing) of the carcinoid syndrome in most patients.
- In addition to the relief of symptoms using octreotide, tumor regression has been reported in some patients. There is no doubt of the important role of somatostatin analogues in the control of symptoms; however, their potential role in tumor inhibition has not been resolved.
- Interferon-α has also been shown to provide symptomatic relief in patients with carcinoid syndrome. High incidence of various side effects (e.g., fever, fatigue, anorexia, and weight loss) precludes the widespread use of this drug.
- Serotonin receptor antagonists have been used with limited success. Methysergide is no longer used owing to the incidence of retroperitoneal fibrosis. Ketanserin and cyproheptadine have been shown to provide some control of symptoms, and other antagonists, such as ondansetron, may be even more effective.
- Cytotoxic chemotherapy has had only limited success. The role of chemotherapy is confined predominantly to patients with metastatic disease who are symptomatic and unresponsive to other therapies.

    - The most frequent combination used is streptozotocin and 5-fluorouracil or cyclophosphamide, which may result in some tumor regression in up to one third of the patients. The duration of response, however, is short lived.
    - The use of cisplatin and etoposide has shown some promise only in patients with well-differentiated carcinoids.
    - Results using dacarbazine (DTIC) are conflicting.

(e) Small bowel enema.

Answer. Small Bowel Enema or enteroclysis:

Indications:

- Suspected inflammatory bowel disease, including exclusion of small bowel disease in Crohn's colitis.
- Intermittent or partial small bowel obstruction.

Preparation - One day before the test - Have a normal breakfast, clear fluids only for lunch (no solid foods or dairy products). After lunch take 2 Dulcolax tablets. Drink 1 glass of clear liquid every hour until bed. At 6 p.m. take 1 bottle of Citromag followed by a glass of water. On the day of the test, have nothing to eat or drink.

Time to perform - 60 to 90 minutes.

Procedure: This procedure involves inserting a thin tube through the mouth, esophagus and past the stomach to inject barium,methylcellulose and water into the small bowel. This allows for better visualization of the small bowel than can be seen during a small bowel follow-through. The back of the throat is 'frozen' to allow easier passage of the tube.

Problem:

- Greater discomfort to the patient, a higher radiation dose and possibility that gastroduodenal disease may not be seen.
- The investigation requires more experienced radiologists.
- Some radiologists advocate that small bowel enema with careful fluoroscopy and vigorous manual palpation can produce acceptable results with less cost, radiation exposure and discomfort to the patient.

4. Write brief answers on: 4 x $7^{1}/_{2}$

(a) Management of skin loss.
(b) Complications of chest injury.
(c) Breast ultrasound.
(d) Intra-abdominal sepsis.

Answer.

(a) Management of skin loss. <u>The Reconstructive Ladder:</u>

The reconstructive ladder of soft-tissue coverage begins with consideration of the simplest approach (healing by secondary intention) and culminates with the most complex (free tissue transfer), maximizing opportunities for success.

- Healing by secondary intention is the simplest approach but is not always feasible. Absolute contraindications include exposed vessels, nerves, tendons, viscera, or bone. Relative contraindications include a large or poorly vascularized wound with a prolonged (>3 weeks) anticipated period of healing and undesirable aesthetic consequences.
- Primary closure may provide the most aesthetically pleasing result, but excessive tension on the skin may cause displacement of neighboring structures (e.g., lower eyelid) or necrosis of the skin flaps.
- Skin grafting is the most common method of large-wound closure. Skin grafts require a healthy, uninfected bed, protected from shear forces, to survive. Wound surfaces such as bare tendon, dessicated bone or cartilage, or infected tissue beds will not support skin graft survival. In addition, exposed vessels, nerves, or viscera are relative contraindications for skin grafting.
- Local tissue transfers of skin, fascia, and muscle may be used in regions with healthy adjacent tissue. If the adjacent tissue cannot be adequately mobilized or the wound requires more bulk than is locally available, the sole use of local flaps may not be adequate.
- Distant tissue transfers were the mainstay of difficult wound closure until the advent of free tissue transfer. This involves transferring healthy tissue into the wound bed while leaving it attached to its native blood supply. The pedicle is divided in a subsequent procedure. Inherent disadvantages of this technique include multiple operations, prolonged wound healing, immobilization for at least 3 weeks, and a limited choice of donor sites.
- Free tissue transfer is the most technically demanding approach to wound closure but has several potential advantages, including single-stage wound closure, a relatively wide variety of flaps to ensure closure specifically tailored to coverage needs, and, in many cases, an aesthetically pleasing outcome.
- Vacuum-assisted closure has altered wound management by decreasing bacterial load and accelerating granulation. Wounds may be treated adequately with vacuum-assisted closure that would not traditionally be candidates for healing by secondary intention. Furthermore, it may convert a wound that would otherwise need adjacent or free tissue transfer into a wound that needs only split-thickness skin grafting.

(b) Complications of chest injury.

Answer. 1. Early after primary survey:

Open pneumothorax	Flail chest	Massive haemothorax	Cardiac tamponade
• Occlude with a three-sided dressing. • Follow by immediate insertion of an intercostal drain through a separate	• Results in paradoxical motion of the chest wall. Hypoxia is caused by restricted chest wall movement and underlying	• Accumulation of more than 1500mL of blood in pleural cavity. • Suspect when shock is associated with dull percussion note and absent breath	• Most commonly results from penetrating injuries but blood can also accumulate in pericardial sac after blunt trauma. • Recognize by

incision.	lung contusion. • If the segment is small and respiration is not compromised, nurse patient in HDU with adequate analgesia. Encourage early ambulation and vigorous physiotherapy. Do regular blood gas analysis. • In more severe cases, endotracheal intubation with positive-pressure ventilation is required.	sounds on one side of chest. • Simultaneously restore blood volume and carry out decompression by inserting a wide bore chest drain. • Consider need for urgent thoracotomy to control bleeding if there is continued brisk bleeding and need for persistent blood transfusion. Consult with a regional thoracic centre.	haemodynamic instability: hypotension, tachycardia, raised jugular venous pressure, pulsus paradoxus, and faint heart sounds. • If critically ill with suspected tamponade perform blind pericardiocentesis and call cardiothoracic or general surgeons to consider emergency thoracotomy. • If unwell but responding to treatment arrange urgent transthoracic echo or focused abdominal ultrasound.

2. After secomdary survey:

- ❖ Simple pneumo-haemothorax:Treat with a chest drain if large or symptomatic or in any patient likely to undergo a general anaesthetic.
- ❖ Pulmonary contusion:Most common potentially lethal chest injury. Risk of worsening associated consolidation and local pulmonary oedema. Treat with analgesia, physiotherapy, and oxygenation. Consider respiratory support for a patient with significant hypoxia.
- ❖ Tracheobronchial rupture:
  - Suspect when there is persistent large air leak after chest drain insertion. Seek immediate (cardiothoracic) surgical consultation.
  - Thoracic CT scan usually diagnostic.
- ❖ Blunt cardiac injury (myocardial contusion/traumatic infarction): Suspect when there are significant abnormalities on ECG or echocardiography. Seek cardiological/cardiothoracic surgical advice.
- ❖ Aortic disruption:
  - Patients survive immediate death because the haematoma is contained.
  - Suspect when history of decelerating force and where there is widened mediastinum on chest X-ray.
  - Thoracic CT scan is diagnostic.

- Consider cardiothoracic surgical referral.

❖ Diaphragmatic rupture:
- Usually secondary to blunt trauma in restrained car passengers (seat belt compression causes burst injury commonly on the left side).
- Suspect in patient with a suitable history and a raised left hemidiaphragm on CXR.
- Penetrating trauma below the fifth intercostal space can produce a perforation.
- Thoracoabdominal CT scan usually diagnostic.

(c) Breast ultrasound.

Answer.

- Ultrasonography is used to characterize a breast lesion identified by physical examination or mammography.
- It can determine whether a lesion is solid or cystic and can define the size, contour, or internal texture of the lesion.
- Although not a useful screening modality by itself due to significant false-positive rates, when used as an adjunct with mammography, ultrasonography may improve diagnostic sensitivity of benign findings to greater than 90%, especially among younger patients for whom mammographic sensitivity is lower due to denser breast tissue.
- In those patients with a known cancer, ultrasound is sometimes used to detect additional suspicious lesions and/or to map the extent of disease.
- Benign breast masses usually show smooth contours, round or oval shapes, weak internal echoes, and well-defined anterior and posterior margins.
  Breast cancer characteristically has irregular walls, but may have smooth margins with acoustic enhancement.
- Ultrasonography is used to guide fine-needle aspiration biopsy, core-needle biopsy, and needle localization of breast lesions. It is highly reproducible and has a high patient acceptance rate, but does not reliably detect lesions that are 1 cm or less in diameter.
  Uses:

  - **Determining the Nature of a Breast Abnormality**
    The primary use of breast ultrasound today is to help diagnose breast abnormalities detected by a physician during a physical exam (such as a lump or bloody or spontaneous clear nipple discharge) and to characterize potential abnormalities seen on mammography or breast magnetic resonance imaging (MRI).

    Ultrasound imaging can help to determine if an abnormality is solid (which may be a non-cancerous lump of tissue or a cancerous tumor) or fluid-filled (such as a benign cyst) or both cystic and solid. Ultrasound can also help show additional features of the abnormal area.

    Doppler ultrasound is used to assess blood supply in breast lesions.

- **Supplemental Breast Cancer Screening**
  Mammography is the only screening tool for breast cancer that is known to reduce deaths due to breast cancer through early detection. Even so, mammograms do not detect all breast cancers. Some breast lesions and abnormalities are not visible or are difficult to interpret on mammograms. In breasts that are dense, meaning there is a lot of ducts, glands, fibrous tissue and less fat, many cancers can be hard to see on mammography.

  Ultrasound can be offered as a screening tool for women who:

  - Are at high risk for breast cancer and unable to undergo an MRI examination.
  - Are pregnant or should not be exposed to x-rays (which is necessary for a mammogram).

- **Ultrasound-guided Breast Biopsy**
  When an ultrasound examination reveals a suspicious breast abnormality, a physician may choose to perform an ultrasound-guided biopsy. Because ultrasound provides real-time images, it is often used to guide biopsy procedures. An ultrasound exam will usually need to be performed before the biopsy in order to plan the procedure and to determine if this method of biopsy can be used.

(d) Intra-abdominal sepsis.

Answer. Introduction: Intra-abdominal sepsis may occur in – i. the peritoneal cavity.
ii. the retroperitoneum
iii. an organ.
An intra-abdominal sepsis may lead to septicemia, septic shock and multiple organ dysfunction syndrome.

Causes of Intra-abdominal sepsis:

Surgical causes:

- Acute infections like acute appendicitis, acute cholecystitis, acute tubo-ovarian infection, acute diverticulitis,etc.
- Acute infections supervening on acute inflammation, e.g. acute necrotizing pancreatitis followed by infection,acute necrotizing enterocolitis,etc.
- Perforations, e.g. appendicular, peptic, tuberculous and enteric perforations and diverticulitis with perforation.Patients on steroid therapy and NSAIDS are vulnerable.
- Hollow visceral injury by accidental trauma, or during an operation, endoscopy or an interventional radiologic procedure.
- Strangulation, infarction or ischaemic gangrene of bowel with or without perforation.
- Anastomotic dehiscence.

- Postoperative sepsis due to breakdown of intraoperative asepsis especially in a patient who is diabetic, malnourished, immunocompromised or neutropenic, or in a patient who is on steroids or chemotherapy.

Medical causes:

- Spontaneous bacterial peritonitis in preexisting liver disease.
- Nephrotic syndrome.
- SLE.
- Malignancy.

Bacteriology: May be monobacterial – due to pneumococci or B haemolytic streptococci or staphylococcus aureus.
May be polybacterial – Gram (-) ve bacilli – E.coli being the most common.
Anaerobes like bacteroides fragilis, anaerobic cocci and clostridia are frequently encountered.
Fungus is isolated with immunocompromised patients.

Clinical features:

- Pain.
- Tenderness, guarding, abdominal distension, absent bowel sounds.
- Restricted abdominal movement with respiration.
- Tachycardia, pyrexia, tachypnoea.
- May be features of shock.

Laboratory studies:

- Leucocytosis, haemoconcentration and electrolyte disturbances.
- Liver functions, renal profile or coagulation parameters may be deranged.
- Blood culture may be helpful.
- Procalcitonin level has some help.

Imaging studies:

- X-Ray chest – pneumonic patch, collapse, pleural effusion, pneumoperitoneum.
- X-Ray abdomen – air fluid level
- USG – can detect intraabdominal abscess, distended bowel loops and interloop collections.
- CT and CECT – best diagnostic tool to diagnose pancreatic and peripancreatic abscess.Extraluminal air is diagnostic of infection in acute necrotizing pancreatitis.
- MRI – for detecting complications of gallstone disease, pancreatitis etc.
- Doppler USC,CECT with angiography and MRA – help in evaluation of vascular pathology related to intra-abdominal sepsis.
- USG/CT guided paracentesis or laparoscopy – useful in diagnostic dilemma.

Local treatment:

- In de-novo intra-abdominal sepsis,after initial resuscitation, an aggressive prompt surgical intervention should be in stituted.

- Patients who have been operated earlier for intra-abdominal sepsis are treated by percutaneous CT guided drainage with, wide multiple pigtail drains or sump drains.

Contraindications to conservative approach:

- Multiple pockets of pus.
- Very thick pus.
- Considerable necrosis.
- Absence of a “safe-window”.
- Overwhelming septicaemia.

Surgery is essential in:

- Intra-abdominal sepsis where percutaneous drainage is not possible or has failed.
- In patients with infected pancreatic necrosis after delaying surgery as much as possible.
- All other patients with intra-abdominal sepsis.

Guidelines for surgery:

- Once the decision of surgery is made, it should be carried out without any delay.
- The perforation should be closed if possible. The septic focus is eradicated totally or eradicated as much as possible.
- If the infective focus is localized and cannot be eradicated, a diversion stoma is fashioned.
- Thorough copious irrigation of peritoneal cavity is essential.
- In selected cases with severe sepsis and considerable local necrosis with large number of pockets of pus laparostoma may have to be resorted to.

General treatment:

- Fluid and electrolyte administration.
- Inotropic support if needed.
- Appropriate antibiotic therapy with broad spectrum coverage.
- Chest physiotherapy.
- Nutritional support.
- Organ support.

Newer aspects:

- To prevent multiple organ failure .
- To prevent thrombosis in microcirculation one can use – antithrombin IV, tissue factor pathway inhibitor and activated protein C.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES

MS (General Surgery) Examination, 2009

PAPER IV

Time Allowed: 3 Hours **Full Marks: 100**

1. **Discuss the pathogenesis of urinary stones. What are the features of stones in the renal pelvis? How do you manage such a patient? (7+5+8)**
2. **What are the causes of enterocutaneous fistula? Classify them & give an account on outline of their management. (5+5+10)**

3. Write short notes of the following: 5 x 6

a) Epidural haematoma of the head.

b) Pouch Surgery.

c) Vascular problem of supracondylar fracture of humerus.

d) Flaps in Surgery.

e) Small bowel enema.

4. **Answer briefly on the following: 4 x 7.5**

a) Management of hydatid cyst.

b) Live donor liver Transplantation.

c) Acute mesenteric ischaemia.

d) Fast track Surgery.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES
MS (General Surgery) Examination, 2009

May 2009

PAPER IV

Time Allowed: 3 Hours **Full Marks: 100**

1. Discuss the pathogenesis of urinary stones. What are the features of stones in the renal pelvis? How do you manage such a patient? (7+5+8)

Answer. Formation of kidney stones are the result of a combination of factors. A stone is created when the urine does not have the correct balance of fluid and a combination of minerals and acids. When the urine contains more crystal-forming substances than the fluid can dilute, crystals can form. Normally the urine contains components that prevent these crystals from attaching to each other. When these substances fall below their normal proportions, stones can form out of an accumulation of crystals.

Aetiology:

- Dietetic: Deficiency of **vitamin A** causes desquamation of epithelium. The cells form a nidus on which a stone is deposited. It is uncertain whether this mechanism is of importance other than in the formation of bladder calculi.
- Altered urinary solutes and colloids: Dehydration increases the concentration of urinary solutes until they are liable to precipitate. Reduction of urinary colloids, which adsorb solutes, or mucoproteins, which chelate calcium, might also result in a tendency for crystal and stone formation.
- Decreased urinary citrate:The presence of citrate in urine, 300–900 mg 24 h–1 (1.6–4.7 mmol 24 h–1) as citric acid, tends to keep otherwise relatively insoluble calcium phosphate and citrate in solution. The urinary excretion of citrate is under hormonal control and decreases during menstruation.
- Renal infection: Infection favours the formation of urinary calculi. Clinical and experimental stone formation are common when urine is infected with urea-splitting streptococci, staphylococci and especially Proteus spp. The predominant bacteria found in the nuclei of urinary stones are staphylococci and Escherichia coli. Inadequate urinary drainage and urinary stasis Stones are liable to form when urine does not pass freely. Prolonged immobilisation
- Immobilisation from any cause, e.g. paraplegia, is liable to result in skeletal decalcification and an increase in urinary calcium favouring the formation of calcium phosphate calculi.
- Hyperparathyroidism: Hyperparathyroidism leading to hypercalcaemia and hypercalciuria is found in 5% or less of those who present with radioopaque calculi. In cases of recurrent or multiple stones, this cause should be eliminated by appropriate investigations. Hyperparathyroidism results in a great increase in the elimination of calcium in the urine. These patients ‘pass their skeletons in their urine’. A parathyroid adenoma should be removed before definitive treatment for the urinary calculi.

  Clinical features:

- **Pain:** Pain is the leading symptom in 75% of people with urinary stones. Fixed renal pain is located posteriorly in the renal angle), anteriorly in the hypochondrium, or in both. It may be worse on movement, particularly on climbing stairs.
- **Haematuria:** Haematuria is sometimes a leading symptom of stone disease and occasionally the only one. As a rule, the amount of bleeding is small.
- **Pyuria:** Infection is likely in the presence of stones and is particularly dangerous when the kidney is obstructed. As pressure builds in the dilated collecting system, organisms are injected into the circulation and a life-threatening septicaemia can quickly develop. The mechanical effect of stones irritating the urothelium may cause pyuria even in the absence of infection.

Investigation of suspected urinary stone disease

- **Radiography:** The 'KUB' film shows the kidney, ureters and bladder. When a renal calculus is branched, there is no doubt about the diagnosis. An opacity that maintains its position relative to the urinary tract during respiration is likely to be a calculus. Calcified mesenteric nodes and opacities within the alimentary tract can sometimes be shown to be anterior to the vertebral bodies on a lateral radiograph and hence outside the urinary tract.

Opacities on a plain abdominal radiograph that may be confused with renal calculus

■ Calcified mesenteric lymph node
■ Gallstones or concretion in the appendix
■ Tablets or foreign bodies in the alimentary canal [e.g. cyclopenthiazide (Navidrex-K)]
■ Phleboliths – calcification in the walls of veins, especially in the pelvis
■ Ossified tip of the 12th rib
■ Calcified tuberculous lesion in the kidney
■ Calcified adrenal gland

- **Contrast-enhanced computerised tomography:** CT, preferably spiral, has become the mainstay of investigation for acute ureteric colic.
- **Excretion urography:** Urography will establish the presence and anatomical site of a calculus. It also gives some important information about the function of the other kidney.
- **Ultrasound scanning:** Ultrasound scanning is of most value in locating stones for treatment by extracorporeal shock wave lithotripsy (ESWL).

Treatment:

Conservative management: Calculi smaller than 0.5 cm pass spontaneously unless they are impacted. Any surgical intervention carries the risk of complications and needless intervention should be avoided. Small renal calculi may cause symptoms by obstructing a calyx or acting as a focus for secondary infection. However, most can be safely observed until they pass.

Management of small stones

- **Most small urinary calculi will pass spontaneously and can be treated conservatively**
- **The presence of infection in an upper urinary tract obstructed by stone is dangerous and is an indication for urgent surgical intervention**

Preoperative treatment: If urinary infection is present, appropriate antibiotic treatment is started and continued during and after surgery as necessary.

Operation for stone: In developed countries, most stones are treated by urologists using minimal access and minimally invasive techniques. Open operations are still needed when appropriate expertise is not available or newer techniques have failed to clear the calculus.
.

Modern methods of stone removal:

Kidney stones

Kidney stone removal

■ Most urinary calculi can be treated by minimal access techniques.

Percutaneous nephrolithotomy:

- This involves the placement of a hollow needle into the renal collecting system through the soft tissue of the loin and the renal parenchyma.
  - ❖ A wire inserted through the needle is used to guide the passage of a series of dilators, which expand the track into the kidney until it is large enough to take the nephroscope used to visualise the stone.
  - ❖ Small stones may be grasped under vision and extracted whole.
  - ❖ Larger stones must be fragmented by an ultrasound, laser or electrohydraulic probe and removed in pieces.
  - ❖ The aim is to remove all fragments if possible, and this may take some time if the calculus is large.
  - ❖ When the operation is over, a nephrostomy drain is left in the system. This decompresses the kidney and allows repeated access if stone particles remain.
  - ❖ Percutaneous nephrolithotomy is sometimes combined with ESWL in the treatment of complex (stag-horn) calculi. The surgeon removes the central part of the stone percutaneously and the more peripheral fragments are treated by ESWL.

Complications of percutaneous nephrolithotomy include :
(1) haemorrhage from the punctured renal parenchyma – this may be profuse and difficult to control;
(2) perforation of the collecting system with extravasation of saline irrigant;
(3) perforation of the colon or pleural cavity during placement of the percutaneous track.

Extracorporeal shock wave lithotripsy: A urinary calculus has a crystalline structure. Bombarded with shock waves of sufficient energy it disintegrates into fragments. The principle is seen at its simplest in the original Dornier machine, in which shock waves were generated by an electrical discharge placed at one focus of an ellipsoid mirror.
When ESWL is successful, the stone fragments must pass down the ureter. Ureteric colic is common after ESWL, and the patient needs analgesia, usually in the form of a non-steroidal anti-inflammatory drug such as diclofenac. The bulky fragments of a large stone may impact in the ureter, causing obstruction. To avoid this, a stent should be placed in the ureter so that the

kidney can drain while the pieces of stone pass. Occasionally, impacted fragments have to be removed ureteroscopically
The principal complication of ESWL is infection. Many calculi contain bacteria, which are released from the broken stone. It is wise to give prophylactic antibiotics before ESWL, and an obstructed system should be decompressed by the insertion of a ureteric stent or percutaneous nephrostomy before treatment. The clearance of stone from the kidney will depend upon the consistency of the stone and its site. Most oxalate and phosphate stones fragment well and, if lying in the renal pelvis, will clear within days. The results with harder stones, especially cystine stones, are less satisfactory. When treating calyceal stones, the patients should be warned that the clearance of fragments may take months.
There is currently great interest in the long-term outcome of patients treated by ESWL. Certainly, some stones recur, especially if small fragments remain after treatment.

Open surgery for renal calculi: Operations for kidney stone are usually performed via a loin or lumbar approach. All of the procedures are difficult unless thekidney is fully mobilised and its vascular pedicle controlled. A sling should be placed around the upper ureter to stop stones migrating downwards.

Indications:

- Complex stone burden (projection of stone into multiple calyces, such that multiple PCNL tracks would be required to gain access to all the stone)
- Failure of endoscopic treatment (technical difficulty gaining access to the collecting system of the kidney)
- Anatomic abnormality that precludes endoscopic surgery (e.g. retrorenal colon)
- Body habitus that precludes endoscopic surgery (e.g. gross obesity, kyphoscoliosis open stone surgery can be difficult)
- Patient request for a single procedure where multiple PCNLs might be required for stone clearance
- Non-functioning kidney.

Pyelolithotomy - Pyelolithotomy is indicated for stones in the renal pelvis. When the wall of the renal pelvis has been dissected free from its surrounding fat, an incision is made in its long axis directly on to the stone. The stone is removed with gallstone forceps,taking care not to break it because fragments may be difficult to retrieve. Stone fragments in peripheral calyces may be detected by direct palpation or by intraoperative radiography or nephroscopy. If there is no infection, the pelvic incision is closed with interrupted absorbable sutures. If there is gross sepsis, a nephrostomy is essential to drain the system.
Extended pyelolithotomy - The plane between the renal sinus and the wall of the collecting system is developed on the posterior surface of the kidney. This avoids major vessels and allows incisions to be made into the calyces so that even large staghorn stones can be removed intact.
Nephrolithotomy - If there is a complex calculus branching into the most peripheral calyces, it may be necessary to make incisions into the renal parenchyma to clear the kidney. Nephrolithotomy may also be necessary when the adhesions resulting from previous surgery make access to the renal pelvis difficult.

Treatment of bilateral renal stones: Usually the kidney with better function is treated first unless the other kidney is more painful or there is pyonephrosis, which needs urgent decompression. Silent bilateral staghorn calculi in the elderly and infirm may be treated conservatively. The patient should be encouraged to maintain a high fluid intake.

Prevention of recurrence

Ideally, all stone formers should be investigated to exclude metabolic factors, although the diagnostic yield is low in patients with a single small stone. The urine of all patients with stones should be screened for infection. The following investigations are appropriate in bilateral and recurrent stone formers:

• Serum calcium, measured fasting on three occasions to exclude hyperparathyroidism;
• Serum uric acid;
• Urinary urate, calcium and phosphate in a 24-hour collection; the urine should also be screened for cystine;
•Analysis of any stone passed.

Dietary advice is not usually helpful in avoiding stone recurrence in people who have a balanced diet. The enthusiast for excessive amounts of milk products (calcium stones), rhubarb, strawberries, plums, spinach and asparagus (calcium oxalate stones) should be advised to be more moderate. Patients with hyperuricaemia should avoid red meats, offal and fish, which are rich in purines, and should be treated with allopurinol. Eggs, meat and fish are high in sulphur-containing proteins and should be restricted in cystinuria. Stone sufferers should drink plenty to keep their urine dilute. Fluid intake should be increased appropriately to take account of increased losses. Drug treatment is largely ineffective except in those few patients who are shown to have idiopathic hypercalciuria. Bendroflumethiazide (5 mg) and a calcium-restricted diet reduce urinary calcium.

2. **What are the causes of enterocutaneous fistula? Classify them & give an account on outline of their management. (5+5+10)**

Answer. Enterocutaneous fistula represents a second group of complex intraperitoneal infectious processes.

- Mortality remains high, between 10 - 30%, largely due to the frequent complications of sepsis and malnutrition.
- Electrolyte imbalances is a key factor leading to mortality. Key to the management of these complex patients is an understanding of the pathophysiology and a sound, multidisciplinary team approach to diagnosis, physiologic support, and reconstruction of these fistula.

Etiology: Enterocutaneous fistula results from several processes:

- Diseased bowel extending to surrounding structures;
- Extraintestinal disease involving otherwise normal bowel;
- Trauma to normal bowel including inadvertent or missed enterotomies; or
- Anastomotic disruption following surgery for a variety of conditions.

- Approximately three-quarters of fistulas occur following an operation, most commonly subsequent to procedures performed for malignancy, inflammatory bowel disease, or adhesions.

- Patient factors that increase the likelihood of developing a postoperative fistula include malnutrition, infection, and emergency operations with concomitant hypotension, anemia, hypothermia, and poor oxygen delivery.
- The spontaneous fistula often develop in patients with cancer or following radiation therapy.
- A second major group of patients with spontaneous fistula are those with inflammatory conditions such as inflammatory bowel disease, diverticular disease, perforated ulcer disease, or ischemic bowel.

Anatomic Classification: Fistula may communicate with the skin (external) or other intraperitoneal or intrathoracic organs (internal). Internal fistula that by pass only short segments of bowel may not be symptomatic; however, internal fistula of bowel that bypass significant length of bowel or that communicate with either the bladder or vagina typically cause symptoms and become clinically evident. The identification and management of internal fistula is beyond the scope of this review, but in general, internal fistulas should be resected if they are symptomatic or cause physiologic or metabolic complications.

Identification of the anatomic site of origin of external fistula

- Oral, Pharyngeal, and Esophageal fistula
- Gastric fistula : Spontaneous gastrocutaneous fistula are uncommon, but can result from inflammation, ischemia, cancer, and radiation.
- Duodenal fistula
- Small Bowel fistula: The majority of gastrointestinal fistula arise from the small intestine. Seventy to ninety percent of enterocutaneous fistulas occur in the postoperative period. Postoperative small bowel fistulas result from either disruption of anastomoses or injury to the bowel during dissection or closure of the abdomen. Crohn's disease is the most common cause of spontaneous small bowel fistula.
- Colonic fistula: Spontaneous fistulas of the colon result from diverticulitis, malignancy, inflammatory bowel disease, appendicitis, and pancreatitis, while treatment of these conditions accounts for the majority of postoperative colocutaneous fistulas. Radiation therapy contributes to both spontaneous and postoperative colocutaneous fistula.

Physiologic Classification: Fistula may be divided into high-output (>500 mL per day), moderate-output (200–500 mL/day), and low-output (<200 mL/day) groups.

Prevention: Proper preoperative patient preparation and meticulous surgical technique will lessen the risk of postoperative fistula formation. In the elective setting, operation may be delayed to allow for normalization of nutritional parameters, thus optimizing wound healing and immune function. Several nutritional characteristics have been suggested to increase the risk of anastomotic breakdown:

1. Weight loss of 10–15% of total body weight over 3–4 months;
2. Serum albumin less than 3 mg/dL;
3. Serum transferrin less than 220 mg/dL;
4. Anergy to recall antigens; or
5. Inability to perform activities of daily living due to weakness or fatigue.[59]

Delay of elective procedures to allow for nutritional support to address these abnormalities may decrease the risk of anastomotic dehiscence and formation of postoperative fistula.

Predictive Factors for Spontaneous Closure and/or Mortality		
Factor	**Favorable**	**Unfavorable**
Organ of origin	Oropharyngeal	Gastric
	Esophageal	Lateral duodenal
	Duodenal stump	Ligament of Treitz
	Pancreaticobiliary	Ileal
	Jejunal	
	Colonic	
Etiology	Postoperative	Malignancy
	Appendicitis	Inflammatory bowel disease
	Diverticulitis	
Output	Low (<200–500 mL/day)	High (>500 mL/day)
Nutritional status	Well-nourished	Malnourished
	Transferrin >200 mg/dL	Transferrin <200 mg/dL
Sepsis	Absent	Present
State of bowel	Healthy adjacent tissue	Diseased adjacent bowel
	Intestinal continuity	Distal obstruction
	Absence of obstruction	Large abscess
		Bowel discontinuty
		Previous irradiation
Fistula characteristics	Tract >2 cm	Tract <1 cm
	Bowel wall defect <1 cm^2	Defect >1 cm^2
		Epithelialization
		Foreign body
Miscellaneous	Original operation performed at same	Referred from outside

	institution	institution

Management Phases of fistula		
Phase	**Goals**	**Time Course**
Recognition/stabilization	Resuscitation with crystalloid, colloid, or blood	24–48 hours
	Control of sepsis with percutaneous or open drainage and antibiotics	
	Electrolyte repletion	
	Provision of nutrition	
	Control of fistula drainage	
	Commencement of local skin care and protection	
Investigation	Fistulogram to define anatomy and characteristics of fistula. CT scan .	7–10 days
Decision	Evaluate the likelihood of spontaneous closure	10 days to 6 weeks
	Decide duration of trial of nonoperative management	
Definitive management	Plan operative approach	When closure unlikely or after 4–6 weeks
	Refunctionalization of entire bowel	
	Resection of fistula with end-to-end anastomosis	
	Secure abdominal closure	
	Gastrostomy and jejunostomy	
Healing	Continue nutritional support until full oral nutrition achieved	5–10 days after closure until full oral nutrition
	Zinc supplementation	
	Psychological and emotional support	

3. Write short notes of the following: 5 x 6

a) Epidural haematoma of the head.

b) Pouch Surgery.

c) Vascular problem of supracondylar fracture of humerus.

d) Flaps in Surgery.
e) Small bowel enema.

Answer.
(a) Epidural haematoma of the head: Epidural or extradural hematoma (haematoma) is a type of traumatic brain injury (TBI) in which a buildup of blood occurs between thedura mater (the tough outer membrane of the central nervous system) and the skull. The dura mater also covers the spine, so epidural bleeds may also occur in the spinal column. Often due to trauma, the condition is potentially deadly because the buildup of blood may increasepressure in the intracranial space and compress delicate brain tissue. The condition is present in one to three percent of head injuries. Between 15 and 20% of patients with epidural hematomas die of the injury.
Signs and symptoms:

Epidural bleeds, like subdural and subarachnoid hemorrhages, are extra-axial bleeds, occurring outside of the brain tissue, while intra-axial hemorrhages,
including intraparenchymal and intraventricular hemorrhages, occur within it.

Epidural bleeding is rapid because it is usually from arteries, which are high pressure. Epidural bleeds from arteries can grow until they reach their peak size at six to eight hours post injury, spilling from 25 to 75 cubic centimeters of blood into the intracranial space. As the hematoma expands, it strips the dura from the inside of the skull, causing an intense headache.

Epidural bleeds can become large and raise intracranial pressure, causing the brain to shift, lose blood supply, or be crushed against the skull. Larger hematomas cause more damage. Epidural bleeds can quickly expand and compress the brain stem, causingunconsciousness, abnormal posturing, and abnormal pupil responses to light.

On images produced by CT scans and MRIs, epidural hematomas usually appear convex in shape because their expansion stops at skull'ssutures, where the dura mater is tightly attached to the skull. Thus they expand inward toward the brain rather than along the inside of the skull, as occurs in subdural hematoma. The lens like shape of the hematoma leads the appearance of these bleeds to be called "lentiform".

Epidural hematomas may occur in combination with subdural hematomas, or either may occur alone. CT scans reveal subdural or epidural hematomas in 20% of unconscious patients.

In the hallmark of epidural hematoma, patients may regain consciousness during what is called a lucid interval, only to descend suddenly and rapidly into unconsciousness later. The lucid interval, which depends on the extent of the injury, is a key to diagnosing epidural hemorrhage. If the patient is not treated with prompt surgical intervention, death is likely to follow.

Causes: The interior of the skull has sharp ridges by which a moving brain can be injured.

The main cause of epidural hematoma is usually traumatic, although spontaneous hemorrhage is known to occur. Hemorrhages commonly result from acceleration-deceleration trauma and transverse forces. 10% of epidural bleeds may be venous, due to shearing injury fromrotational forces. Epidural hematoma commonly results from a blow to the side of the head. The pterion region which overlies the middle meningeal artery is relatively weak and prone to injury. Thus only 20 to 30% of epidural hematomas occur outside the region of the temporal bone.

The brain may be injured by prominences on the inside of the skull as it scrapes past them.

Epidural hematoma is usually found on the same side of the brain that was impacted by the blow, but on very rare occasions it can be due to a contrecoup injury.

Treatment

As with other types of intracranial hematomas, the blood may be aspirated surgically to remove the mass and reduce the pressure it puts on the brain. The hematoma is evacuated through a burr hole or craniotomy. If transfer to a facility with neurosurgery is prolonged trephination may be performed in the emergency department.

Prognosis

In TBI patients with epidural hematomas, prognosis is better if there was a lucid interval (a period of consciousness before coma returns) than if the patient was comatose from the time of injury. Unlike most forms of TBI, people with epidural hematoma and a Glasgow Coma Score of 3 (the lowest score) are expected to make a good outcome if they can receive surgery quickly.

(b) Pouch Surgery.
Answer. The ileal pouch-anal anastomosis (IPAA), also known as an ileo-anal pouch, restorative proctocolectomy, ileal-anal pullthrough, or sometimes referred to as a j-pouch, s-pouch, w-pouch or an internal pouch, is an internal reservoir; usually situated where the rectum would normally be. It is formed by folding loops of small intestine (the ileum) back on themselves and stitching or stapling them together. The internal walls are then removed thus forming a reservoir. The reservoir is then stitched or stapled into the perineum where the rectum was.

Ileo-anal pouches are constructed for people who have had their large intestine surgically removed due to disease or injury. Diseases and conditions of the large intestine which may require surgical removal include:

- Ulcerative colitis
- Crohn's disease
- Familial adenomatous polyposis
- Colon cancer
- Toxic megacolon

There is debate about whether patients suffering from Crohn's disease are suitable candidates for an ileo-anal pouch due to the risk of the disease occurring in the pouch, which could make matters even worse. An alternative to an ileo-anal pouch is an ileostomy.

In some cases where the pouch was formed to manage colitis, inflammation can return to the pouch in a similar way to the original inflammation in the colon. This is known as pouchitis.

Surgical Procedure

The entire procedure can be performed in one operation, but is usually split into two or three. When done as a two-step, the first operation (step one) involves a colectomy (removal of the

large intestine), and fashioning of the pouch. The patient is given a temporary defunctioning ileostomy (also known as a "loop ileostomy"). After a period of usually 6-8 weeks the second step (sometimes called the "takedown") is performed, in which the ileostomy is reversed. The reason for the temporary ileostomy is to allow the newly constructed pouch to fully heal without waste passing through it, thus avoiding infection.

Some surgeons prefer to perform a subtotal colectomy (removing all the colon except the rectum), since removal of the rectum can lead to complications with the anal sphincters. When a colectomy is performed as an emergency (which can arise from Toxic megacolon and other complications), and/or when the patient is extremely ill, the colectomy and pouch construction are performed in separate stages resulting in a three-part surgery.

Pouch behavior

Immediately after the surgery is complete, the patient tends to pass liquid stool with frequent urgency, and he or she may have 8 to 15 bowel movements per day, but this eventually decreases with time. Because the ileo-anal pouch is considerably smaller a reservoir than the colon, patients tend to have more frequent bowel motions; typically 6-8 times a day. Also because the ileum does not absorb as much water as the colon, the stools tend to be less formed, and sometimes fluid.

Because the ileum does not absorb as much of the gastric acid produced by the stomach as the colon did, pouch output also tends to burn the anal region slightly, and many patients find it helpful to wash the area regularly, sometimes using protective barrier cream.

Diet: Because more water is lost through pouch output, patients can get dehydrated easily and can also suffer salt deficiency. For this reason, some are encouraged to add extra salt to meals. Persistent dehydration is often supplemented with an electrolyte mix drink.

Many patients choose to eat more white carbohydrates, because this thickens the pouch output and reduces the risk of dehydration or the aforementioned burning of the anal region. It is also common among pouch-owners to eat little and often, or "graze", rather than having three large meals a day. Some patients avoid eating much after 6-7pm to avoid having to get up during the night.

Immediately after surgery, patients are encouraged to eat low fiber, high protein/carbohydrate meals, but after the pouch function has settled, most are able to reintroduce a fully varied diet. There are some foods that are known to irritate the pouch, however, and though they may be introduced carefully, are best avoided immediately following surgery.

- Increased stool output can be caused by fibrous foods (such as pulses, green leaves, raw vegetables etc.) and also by spicy foods, alcohol and caffeine.
- Anal irritation can be caused by nuts, seeds, citric acid, raw fruit and spicy food.
- Increased gas can be caused by fizzy drinks, milk, beer, broccoli, cauliflower, sprouts, cabbage etc.
- Increased odor can be caused by foods such as fish, onions, garlic and eggs.
- Pouchitis is inflammation of the ileal pouch, which is created in the management of patients with ulcerative colitis, indeterminate colitis, or, rarely, other colitides.

- Patients with pouchitis typically present with bloody diarrhea, urgency in passing stools, or discomfort while passing stools. The loss of blood and/or dehydration resulting from the frequent stools will frequently result in nausea. In fewer cases, pain can occur with pouchitis.
- Endoscopy in patients with pouchitis usually reveals erythematous pouch mucosa, loss of pseudocolonic vaculature or other architecture, and friability of the mucosa. Biopsies show evidence of inflammatory cells or red blood cells in the lamina propria.
- Pouchitis is usually treated with antibiotics, and specifically usually with ciprofloxacin and metronidazole. In refractory cases, probiotics such as VSL-3 and Bio-K+ may be useful.

(c) Vascular problem of supracondylar fracture of humerus.

Answer. Supracondylar fractures of the humerus in children remain a challenging problem. The possibility of Volkmann's ischemic contracture is rare but devastating. The role of arteriography and indications for operative repair of the brachial artery remain controversial. In addition, the treatment of a pink yet pulseless hand is also unclear.

- Vascular compromise occurs in about 5-20% of children with supracondylar fracture. less than 1% will be complicated by compartment syndrome and the resulting sequelae of Volkmann's contracture;
- A median nerve palsy, may mask a pending compartment syndrome (because there will be no pain);
- Fracturess w/ posterolateral displacement of distal fragment are more susceptible to vascular injury, since the medial spike of the proximal humerus can tether the brachial artery.
- Tenting of the artery from the biciptal aponeurosis.
- The bicipital aponeurosis may be involved in the anatomical etiology of arterial compromise after swelling in supracondylar fracture.

Treatment:

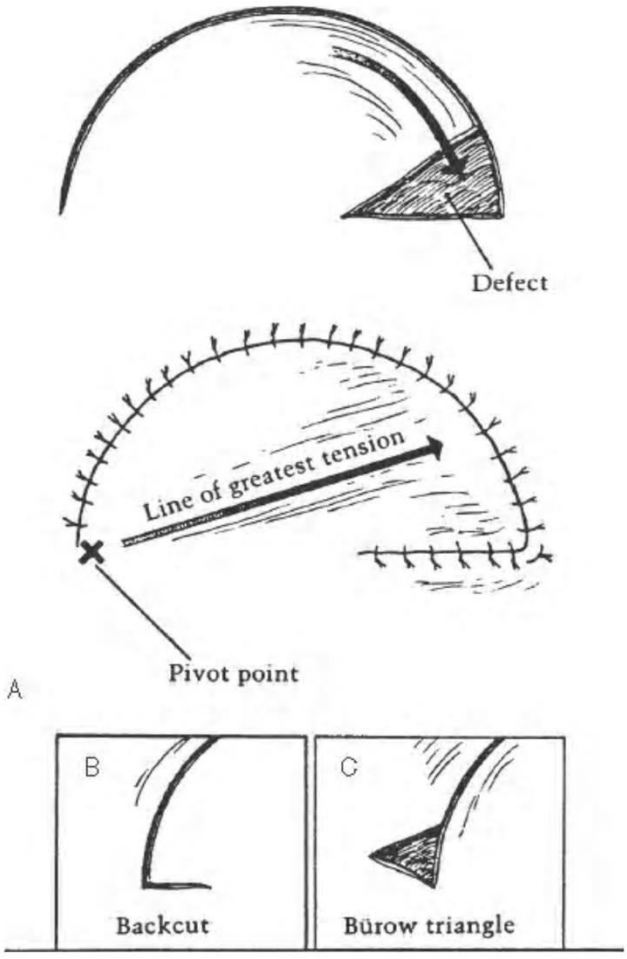
- While patient is being worked up, consider applying a continuous pulse ox so nurses can follow an objective measurement of perfusion;
- Arteriography: fracture reduction usually restores the pulses, and therefore reduction should not be delayed by waiting for an angiographic study; Arteriography is only indicated if circulation is not restored following reduction.
- If circulation is not restored by closed reduction, immediate open reduction and surgical exploration of the artery is indicated;
- Arteriography is not indicated since it provides little additional information and only delays restoration of circulation to the extremity;
- Morbidity associated with surgical exposure of the artery is low.
- Procrastination will only increase patient morbidity;
- Possible compartment syndrome will need fasciotomy;

(d) Flaps in Surgery.

Answer. Definition: A flap is a unit of tissue that is transferred from one site (donor site) to another (recipient site) while maintaining its own blood supply.

A. Classification based on blood supply

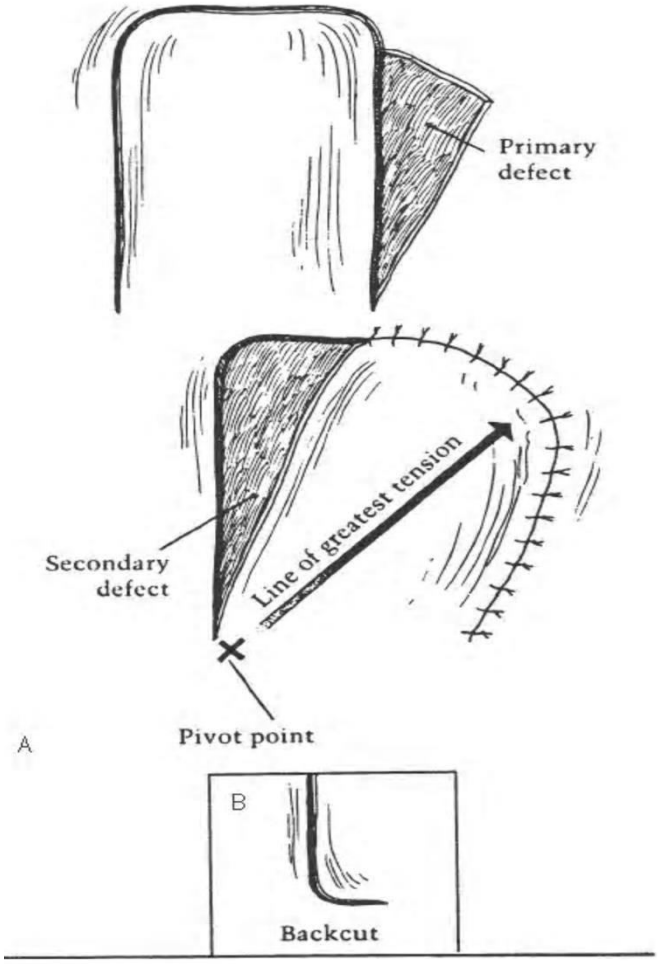
- Random cutaneous flaps have a blood supply from the dermal and subdermal plexus without a single dominant artery. They generally have a limited length-to-width ratio , although this varies by anatomic region (e.g., the face has a ratio of up to 5:1). These flaps are usually used locally to cover adjacent tissue defects but can be transferred to a distant site by use of a staged procedure.
  Depending on the size of the defect to be covered, moving a local tissue flap can create a donor defect, which which may require skin grafting. All local flaps are comparatively easier to use with the loose skin of the elderly.



Rotation flap. A: The edge of the flap is four to five times the length of the base of the defect triangle. B, C: A backcut or Burow triangle can be useful if the flap is under tension.

- Flaps that rotate around a pivot point include rotation flaps and transposition flaps. Planning for shortening of the effective length through the arc of rotation is important when designing these flaps. More complex rotation flaps include bilobed flaps and rhomboid flaps.
- Advancement of skin directly into a defect without rotation can be accomplished with a simple advancement, a V-Y advancement, or a bipedicle advancement flap.

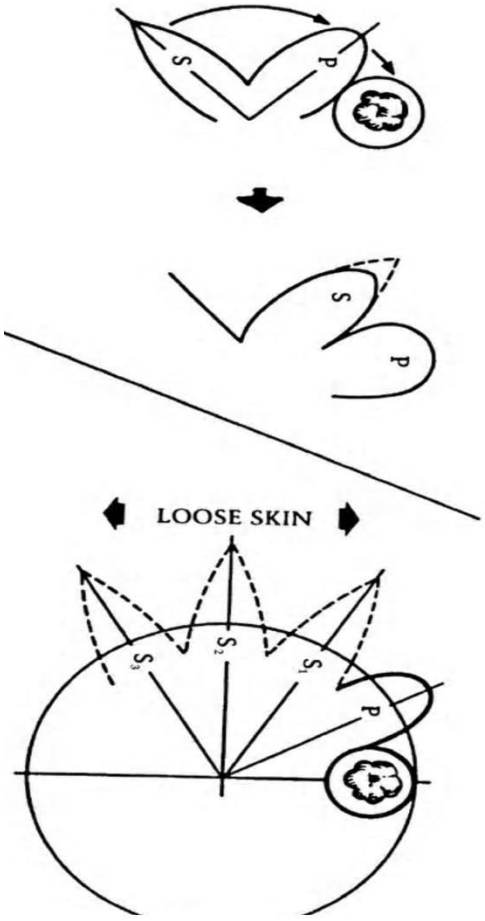
- Axial cutaneous flaps contain a single dominant arteriovenous system. This results in a potentially greater length-to-width ratio.
  - Peninsular flaps are those in which the skin and vessels are moved together as a unit.
  - Island flaps are those in which the skin is divided from all surrounding tissue but maintained on an isolated, intact vascular pedicle.
  - Free flaps are those in which the vascular pedicle is isolated and divided. The flap and its pedicle are then moved to a new location and microsurgically anastomosed to vessels at the recipient site, allowing for long-distance transfer of tissue.



A: Transposition flap. The secondary defect is typically covered with a skin graft.
B: A backcut may be added to reduce tension at the pivot point.

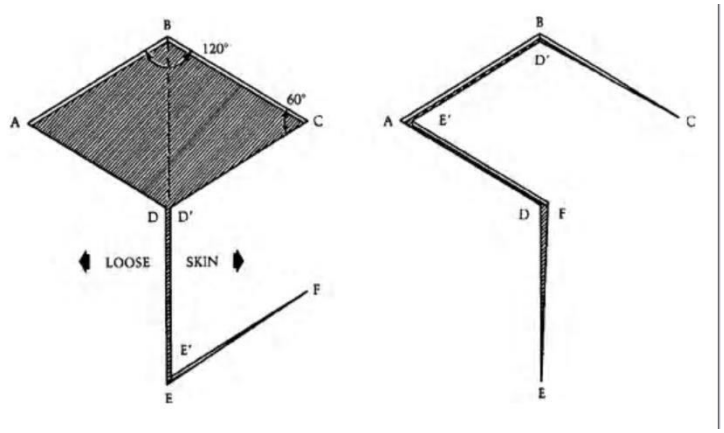
B. Classification based on tissue type:

- Cutaneous flaps include the skin and subcutaneous fat. These are generally random flaps because the axial blood supply is deep to the fat.
- Fasciocutaneous flaps are axial flaps with a single dominant blood supply contained in the deep fascia along with the overlying fat and skin. A wide variety of fasciocutaneous flaps have been described, but those commonly used include radial forearm, parascapular, lateral arm, and groin flaps. These flaps are often utilized for coverage of mobile structures such as tendons.
- Muscle flaps use the specific axial blood supply of a muscle to provide well-vascularized soft-tissue bulk. These flaps can often be transferred with the overlying skin as a myocutaneous flap. Alternatively, they may be transferred without the overlying skin to fill a cavity or may be covered with a skin graft.



Bilobed flap. After the lesion is excised, the primary flap (P) is transposed into the initial defect, and the secondary flap (S) is moved to the site vacated by the primary flap. The bed of the secondary flap is then closed primarily. The primary flap is slightly narrower than the initial defect, whereas the secondary flap is half the width of the primary flap. To be

effective, this must be planned in an area where loose skin surrounds the secondary flap site. Three choices for the secondary flap are shown (S_1, S_2, S_3).



- Rhomboid or Limberg flap. The rhomboid defect must have 60- and 120-degree angles so that the length of the short diagonal is the same as the length of the sides. The short diagonal is extended by its own length to point E. The line EF is parallel to CD, and they are equal in length. There are four possible Limberg flaps for any rhomboid defect; the flap should be planned in an area where loose skin is available to close the donor defect primarily.

- Considerations in the transfer of vascularized muscle include the pattern of circulation, arc of rotation, donor-site contour, and donor-site functional defects. Commonly used muscle flaps include the latissimus dorsi, pectoralis major, rectus abdominis, gastrocnemius, soleus, gracilis, tensor fascia lata, trapezius, and gluteus maximus, but any muscle can potentially be transferred as a flap.
- A musculocutaneous flap involves transfer of a muscle with the overlying skin and subcutaneous tissue. The skin is vascularized via myocutaneous or septocutaneous perforating vessels.

C. Specialized flaps

- Fascial flaps are used when thin, well-vascularized coverage is needed (e.g., for coverage of ear cartilage or the dorsum of the hand or foot). The temporoparietal fascia flap is a classic example, but other fasciocutaneous flaps can be transferred without the overlying skin.
- Vascularized bone flaps are designed to meet specific reconstructive needs, as dictated by loss of bony structure. Because they must be transferred to a specific location, they are generally transferred as free flaps. They may or may not include muscle and/or overlying skin. Commonly used bone flaps include free fibula, scapular spine, iliac (with overlying internal oblique muscle), and rib (with pectoralis major or intercostal muscle).

- Functional muscle may be transferred with its accompanying dominant nerve. Common functional muscle transfers include transfer of gracilis for restoration of facial movement or latissimus for replacement of biceps function.
- Segmental muscle flaps can be used when multiple sources provide blood supply to the muscle. A portion of the muscle is used as a flap, leaving behind a vascularized, innervated, functional muscle. This technique minimizes donor-site functional loss. Portions of the serratus anterior and gluteus maximus can be transferred as segmental flaps.

V-Y advancement. The skin to the sides of the V is advanced.

(e) Small bowel enema.

Answer. See the answer of Q.No.3(e) of Paper – IV of 2010.

4. Answer briefly on the following: 4 x 7.5
a) Management of hydatid cyst.
b) Live donor liver Transplantation.
c) Acute mesenteric ischaemia.
d) Fast track Surgery.

Answer.
(a) Management of hydatid - cyst of liver.
Hepatic echinococcosis is endemic in regions where dogs, the primary host for this intestinal tapeworm, are in contact with sheep, elk, or caribou, the intermediate host. Hydatid disease of the liver occurs most commonly in the Mediterranean region, the Middle East, and South America.

Workup: Various laboratory tests have been used to establish the diagnosis, but none is definitive. Traditionally, the Casoni and Weinberg skin tests have been employed to aid in the diagnosis. However, their sensitivity is so low that they are no longer used. The enzyme-linked immunosorbent assay (ELISA) to identify specific antigens and immune complexes has up to a 90% sensitivity depending on the antigen preparation. This assay is clinically useful because the results can be obtained within hours as opposed to days with more sensitive tests such as Western blotting. Complement fixation and indirect hemagglutination test results are positive in 85% to 90% of active cysts. Eosinophilia is present in approximately 40% of patients but is not diagnostic.

- Gharbi published an ultrasound classification of hepatic echinococcal cysts that has become even more relevant as less invasive therapies have become popular.

- ❖ Gharbi's type I cysts have pure fluid similar to a simple cysts.
- ❖ Type II cysts have a fluid collection with a split-wall floating membrane.
- ❖ Type III cysts have a potentially drainable fluid collection with septa, daughter cysts, or a honeycomb image.
- ❖ Type IV cysts have a heterogeneous echographic pattern.
- ❖ Type V cysts have reflecting thick walls. This same classification may be applied to computed tomography (CT) or magnetic resonance imaging (MRI) scans.

- ➢ Ultrasonography (US) and CT scans are the most widely used imaging studies to diagnose liver cysts. Both studies will show position, size, number of cysts, their proximity to vascular structures, and evidence of extrahepatic cysts. The classic findings for hydatid cysts are thick walls, often with calcifications, and many have daughter cysts.
- ➢ CT scanning may give better information about the location and depth of the cyst than ultrasound.
- ➢ MRI scans also demonstrate cyst characteristics and their relation to vascular and biliary structures. However, MRI does not give additional information and may not be cost effective.
- ➢ Some centers advocate the use of endoscopic retrograde cholangiopancreatography (ERCP) in the workup of hydatid cysts when there is a complication such as cholangitis or jaundice. In these situations, ERCP can show communication between the cysts and bile ducts and can be used to drain the biliary tree before surgery. Routine use of ERCP can completely define the bile duct anatomy and to visualize any clinically silent connections between the bile ducts and cysts.
- ➢ Intraoperative ultrasound also should be used when operative treatment for hydatid disease is undertaken.
- ➢ Operative ultrasound can show the relationship of bile ducts and vessels to the cyst with greater resolution than a transabdominal probe.

Treatment:

Principles and Options:

Although most echinococcal cysts are asymptomatic on presentation, their potential complications such as cholangitis, cyst rupture with anaphylaxis, or pulmonary infection require that all cysts be considered for treatment. However, because most cysts are asymptomatic, a careful well-thought-out plan of treatment should be devised, potentially using a number of modalities. Depending on the patient's state of health and the complicated nature of the cysts, medical, surgical, and percutaneous approaches to the disease should be considered. The principles of treatment are to eradicate the parasite within the cysts, to protect the host against spillage of scolices, and to manage complications.

Medical Therapy:

Medical therapy has been limited to the benzimidazoles (mebendazole and albendazole). Albendazole is more readily absorbed from the intestine and is metabolized by the liver to its active form, whereas mebendazole is poorly absorbed and is inactivated by the liver. Therefore

albendazole is the drug of choice for medical therapy. However, medical therapy alone for echinococcal cysts has a less than 30% success rate. The response has been shown to be higher in extrahepatic manifestations of the disease and with the alveolar form caused by E. multilocularis. Preoperative treatment with albendazole for at least 3 months has been shown to reduce the recurrence when cyst spillage, partial cyst removal, or biliary rupture has occurred. The length of therapy in these circumstances should be at least 1 month.

Surgical Therapy:

Scolecoidal Agents:

- Considerable controversy exists regarding the use of scolecoidal agents. During the early experience with surgical management by cyst evacuation, a high rate of peritoneal implantation occurred.
- As a result, various scolecoidal agents were tried to inject into the cyst before evacuation, to place into the adjacent peritoneal cavity, and to place into the cyst after evaluation.
- Formalin was one of the first scolecoidal agents, but formalin can cause sclerosing cholangitis when it enters the biliary tract. Therefore formalin should never be used for this purpose.
- Hypertonic saline also has been widely used, but care should be taken to avoid biliary injection and overuse with resultant hypernatremia.
- Some authorities recommend cetrimide or chlorhexidine, but the safety of any agent entering the biliary tree has not been established.
- In addition, preevacuation injection should be avoided because intracyst pressure is already high. Thus many surgeons prefer a meticulous surgical technique rather than overeliance on scolecoidal agents.

Open Cyst Evacuation: Cysts on the periphery of the liver are easily treatable by open cyst evacuation, which is the safest surgical approach. Anterior cysts are best treated through an abdominal approach, whereas cysts in segments VI and VII may be best approached through a lateral flank approach. Before entering the cyst, the field is lined with hypertonic saline (20%) soaked gauze in the event of spillage.The cyst cavity is then opened, and the contents are aspirated with a large suction device that can generate high negative pressure. Once the contents are aspirated, the cyst can be opened completely, and any remaining debris can be meticulously cleared. The cyst may then be irrigated with a scolecoidal agent, as described above.

If the cyst fluid is bile stained or a connection with the bile ducts was shown on preoperative ERCP, intracavity scolecoidal agents should be avoided. If a connection with the biliary tree is identified, simple closure of the bile duct should be performed using absorbable sutures, and the cyst cavity should be filled with omentum. If the communication cannot be easily closed, external drainage with a closed suction drain or internal drainage with a cystojejunostomy may be warranted.

Laparoscopic Cyst Evacuation: Several recent reports suggest that carefully selected patients with peripherally located echinococcal hepatic cysts may be safely managed by laparoscopic

cyst evacuation. The laparoscopic approach is best suited for anterior cysts without thick calcified walls, but cysts in segments VI and VII may be managed by a right lateral approach.

In properly selected patients with uncomplicated cysts, the conversion rate should be less than 5%. Operative times of less than 90 minutes have been reported, and complication rates have been low. A major disadvantage of the laparoscopic approach is the relative inability to avoid peritoneal spillage, especially with high intraabdominal pressures due to the pneumoperitoneum. Advocates of this approach recommend oral albendazole for 10 days preoperatively and for 3 months postoperatively. Obvious advantages for the laparoscopic approach, on the other hand, are reduced hospital stay, reduced hospital cost, and earlier return to productive activity.

Pericystectomy: Pericystectomy involves complete resection of the cyst wall without entering the cyst cavity itself. This procedure can be done either along a plane outside the pericyst or along the cyst wall itself. Pericystectomy, like cyst evacuation, is best performed on cysts that are accessible along the periphery of the liver. The use of intraoperative ultrasound also is very helpful in identifying and avoiding key structures. The advantage of this procedure over simple cyst drainage is that it decreases the risk of content spillage into the peritoneal cavity, which can cause anaphylaxis, as well as the risk of recurrence. The disadvantage of pericystectomy is an increased risk of bleeding or damage to bile ducts in proximity to the cyst wall.

Liver Resection/Transplantation:

Some experts believe that formal liver resection for benign disease is excessive, whereas others argue that hepatic resection is now very safe. Liver resection should be considered for multiple cysts within proximity to one another or major hepatic blood supply or when the resection would be relatively safe such as cysts confined to segments II or III. A formal resection of the liver should be initiated only if complete excision of all cysts is possible. E. multilocularis can produce a more complicated form of the disease known as alveolar echinococcosis. Multiple cysts from E. multilocularis infection can lead to fulminant liver failure from sclerosing cholangitis, Budd-Chiari syndrome, or biliary sclerosis. In these rare situations in which the patient's life is threatened, orthotopic liver transplantation may be necessary for the treatment of this rare form of echinococcal liver disease.

Percutaneous (PAIR)Therapy:

Although surgical therapy remains the gold standard, percutaneous drainage has become acceptable for carefully selected patients. Albendazole was administered before the procedure for 10 days to those undergoing percutaneous drainage. In the percutaneously managed patients, the cyst was rapidly aspirated with a 5-Fr catheter. If daughter cysts were present, they were aspirated with a 20-cm long cholangiography needle. Then the cyst was filled with 20% saline for 20 minutes. The cyst was then aspirated completely, irrigated with 0.9% saline, and partially filled with 0.9% saline. The patients were then monitored and discharged from the hospital on average within 48 hours.

(b) Live donor liver Transplantation.

Answer. Liver transplantation or hepatic transplantation is the replacement of a diseased liver with a healthy liver allograft. The most commonly used technique is orthotopic transplantation, in which the native liver is removed and replaced by the donor organ in the same anatomic location as the original liver. Liver transplantation nowadays is a well accepted treatment option for end-stage liver disease and acute liver failure. It is also one of the most expensive treatments in modern medicine.

Indications:

- Liver transplantation is potentially applicable to any acute or chronic condition resulting in irreversible liver dysfunction, provided that the recipient does not have other conditions that will preclude a successful transplant.
- Uncontrolled metastatic cancer outside liver, active drug or alcohol abuse and active septic infections are absolute contraindications.
- While infection with HIV was once considered an absolute contraindication, this has been changing recently.
- Advanced age and serious heart, pulmonary or other disease may also prevent transplantation (relative contraindications).
- Most liver transplants are performed for chronic liver diseases that lead to irreversible scarring of the liver, or cirrhosis of the liver. Another cause is cryptogenic liver disease.

Living donor transplantation

Living donor liver transplantation (LDLT) has emerged in recent decades as a critical surgical option for patients with end stage liver disease,such as cirrhosis and/or HCC often attributable to one or more of the following: long-term alcohol abuse, long-term untreated hepatitis C infection, long-term untreated hepatitis B infection. The concept of LDLT is based on (1) the remarkable regenerative capacities of the human liver and (2) the widespread shortage of cadaveric livers for patients awaiting transplant. In LDLT, a piece of healthy liver is surgically removed from a living person and transplanted into a recipient, immediately after the recipient's diseased liver has been entirely removed.

- Historically, LDLT began as a means for parents of children with severe liver disease to donate a portion of their healthy liver to replace their child's entire damaged liver.
- The first report of successful LDLT was by Dr. Christoph Broelsch at the University of Chicago Medical Center in November 1989.
- In a typical adult recipient LDLT, 55 to 70% of the liver (the right lobe) is removed from a healthy living donor. The donor's liver will regenerate approaching 100% function within 4–6 weeks, and will almost reach full volumetric size with recapitulation of the normal structure soon thereafter. It may be possible to remove up to 70% of the liver from a healthy living donor without harm in most cases. The transplanted portion will reach full function and the appropriate size in the recipient as well, although it will take longer than for the donor.

Living donors are faced with risks and/or complications after the surgery. Blood clots and biliary problems have the possibility of arising in the donor post-op, but these issues are

remedied fairly easily. Although death is a risk that a living donor must be willing to accept prior to the surgery, the mortality rate of living donors in the United States is low. The LDLT donor's immune system does diminish as a result of the liver regenerating, so certain foods which would normally cause an upset stomach could cause serious illness.

Liver donor requirements:

Any member of the family, parent, sibling, child, spouse or a volunteer can donate their liver. The criteria for a liver donation include:

- Being in good health
- Having a blood type that matches or is compatible with the recipient's
- Having a charitable desire of donation without financial motivation
- Being between 18 and 60 years old
- Being of similar or bigger size than the recipient
- Before one becomes a living donor, the donor must undergo testing to ensure that the individual is physically fit. Sometimes CT scans or MRIs are done to image the liver. In most cases, the work up is done in 2–3 weeks.

Complications:

Living donor surgery is done at a major center. Very few individuals require any blood transfusions during or after surgery. Even though the procedure is very safe, all potential donors should know there is a 0.5 to 1.0 percent chance of death. Other risks of donating a liver include bleeding, infection, painful incision, possibility of blood clots and a prolonged recovery. The vast majority of donors enjoy complete and full recovery within 2–3 months.

(c) Acute mesenteric ischaemia.

Answer. Acute Mesenteric Ischemia:

Presentation:

- The classic description of patients who present with embolic disease of the mesenteric vessels is of sudden-onset midabdominal pain described as pain out of proportion to physical examination findings and is associated with immediate bowel evacuation.
- However, only approximately one third of patients present with the triad of abdominal pain, fever, and heme-positive stools.
- A recent history of a myocardial infarction, atrial fibrillation, mural thrombus, mitral valve disease, left ventricular aneurysm, or embolic disease should suggest the diagnosis.
- If the etiology of the acute mesenteric ischemia is secondary to thrombotic occlusive disease, before the acute presentation, these patients typically describe symptoms of chronic abdominal pain following meals and will usually have experienced significant weight loss.
- These patients typically have other manifestations of diffuse atherosclerotic disease, such as coronary artery disease, peripheral artery disease, and carotid stenosis.
- Patients who present with nonocclusive mesenteric ischemia (NOMI) present in a manner slightly different from patients with embolic or thrombotic etiologies.

- It usually occurs during severe low-flow states and represents extreme mesenteric vasoconstriction.
- It is much more common among severely ill patients in an intensive care setting who require vasopressors.
- The symptoms are typically less acute and more diffuse and may wax and wane throughout its course.

Diagnosis :

- Once suspected by history and physical examination results, noninvasive or invasive studies can confirm the diagnosis of mesenteric ischemia. Abdominal plain x-ray films are nondiagnostic. They may reveal edematous bowel with thumbprinting. In severe cases, the x-ray films may reveal gas in the bowel wall and the portal vein. However, more commonly plain films reveal a pattern consistent with an ileus or are completely unremarkable.
- Computed tomography (CT) and magnetic resonance angiography (MRA) are the most commonly used noninvasive studies to confirm the diagnosis of mesenteric ischemia. Both technologies have undergone significant advances over the past decade. Traditional CT scan technology could evaluate arterial patency, anatomy, calcifications, and aneurysms. Furthermore, it could evaluate the status of the bowel and help identify other causes of abdominal pain.
- However, not until the advent of helical CT scan technology and the more recently developed multislice, multiarray helical CT scan technology with maximum intensity projection (MIP) could visceral arterial anatomy be visualized with three-dimensional spatial resolution.

  - Although the technology has become much more sophisticated and the image clarity and definition have improved, limitations of what can be determined from CT technology remain. The origin of the celiac artery and the SMA can be well visualized, but secondary, tertiary, and smaller branches are less well defined. For this, contrast-enhanced angiography remains the gold standard.
  - Another limitation of CT scan technology is the administration of intravenous contrast, which can be nephrotoxic or become problematic for patients with contrast allergies. CT scan technology also tends to overestimate the degree of critical stenosis compared to conventional angiography. However, with the advent of multiarray or multidetector technology, this limitation appears to be less of an issue because the images obtained are more sensitive for the detection of arterial stenotic lesions.
  - Finally, the amount of calcification present at the origin of the vessel can make it difficult to determine the true degree of stenosis with CT scan technology.

- Recent advances in magnetic resonance technology with contrast-enhanced three-dimensional MRA have made MRA imaging of visceral vessels much more practical than earlier studies. Fast imaging techniques using intravenously administered gadolinium over a single breath-hold can provide high-quality three-dimensional images. Images can be obtained in axial, sagittal, and oblique planes to align the plane of the vessel.
- One benefit of MRA technology over CT scan is the use of gadolinium. Gadolinium is significantly less nephrotoxic compared to the intravenous contrast used for CT scans. A

well-known limitation of MRA technology, similar to CT scan, is that it will adequately assess the distal branches of the mesenteric vessels.

- Contrast angiography has long been considered the gold standard for imaging of the visceral vessels. It is performed in a transfemoral manner using the Seldinger technique.
- Arteriography should include both anteroposterior and lateral views of the celiac artery, the SMA, and the inferior mesenteric artery (IMA). The origins of the celiac artery and the SMA are best visualized on the lateral view, whereas the middle and distal SMA and IMA are best visualized on the anteroposterior view. Delayed views are useful to evaluate for NOMI.

    - A classic angiographic pattern is associated with mesenteric ischemia of embolic versus thrombotic disease.
    - Of all three mesenteric vessels, emboli typically lodge in the SMA given the less acute angle of the origin of this vessel from the main axis of the aorta.
    - The celiac artery and IMA arise off the aorta at more perpendicular angles.
    - When emboli lodge in the SMA, they usually lodge distal to the middle colic branch and the jejunal branch.
    - With thrombotic disease, the thrombus usually forms at the atherosclerotic plaque. For most patients, this region is usually at the origin of the mesenteric vessel.
    - Therefore the angiogram typically demonstrates complete absence of flow in the mesenteric vessel, often making it difficult to ascertain the location of the origin of the mesenteric vessel.

In addition to the superior image quality of contrast-enhanced angiography, another benefit of angiography is the ability to perform selective injections of each mesenteric vessel. Furthermore, with NOMI, the SMA can be selectively catheterized to instill papaverine. In the case of chronic mesenteric ischemia with stenotic lesions of the mesenteric vessels, this would allow for percutaneous balloon angioplasty and/or stent placement. Therefore not only is angiography the gold standard for diagnostic imaging, but it also provides important therapeutic options.

Intraoperative Evaluation： Often, the diagnosis of mesenteric ischemia is considered in the operating room when a patient is explored for abdominal pain and necrotic bowel is encountered. In this case, the root of the mesentery should be palpated for a pulse. Alternatively, a handheld Doppler can be used to listen to the quality and character of the arterial signal. Intraoperative angiography can also be performed, but it is often difficult to perform in the operating room if not adequately prepared and set up ahead of time, and the available technology may limit the usefulness of such endeavors.

Assessment of Bowel Viability：

- Multiple techniques have been described to assess bowel viability intraoperatively following revascularization.
- After restoration of flow, the bowel may contain frankly necrotic segments in need of resection, areas of normal bowel, and areas of marginally perfused bowel.
- It is this latter category that often provides a challenge to the surgeon.
- Many diagnostic options are available to the clinician.

- These range from determination of the pulse quality and character, use of a handheld intraoperative Doppler, intravenous fluorescein, and transcutaneous oxygen measurements (TCO_2) to a second-look exploratory laparotomy.
- All of these methods are relatively simple to perform, but each has its limitations. Obviously, clearly necrotic or nonviable bowel must be resected at the time of the operation.
- Determination of the viability of marginally perfused bowel is more difficult.
- Intraoperatively, the color, motility, and integrity of the bowel should be evaluated.
- The appearance of ischemic intestine includes loss of the normal sheen, dull-gray discoloration, and lack of peristalsis.
- Determination of the character and quality of the pulsation in the antimesenteric border and the mesenteric arcades may help determine which bowel will remain viable once revascularization has been performed.
- Intraoperative Doppler assessment of the perfusion of the bowel can be performed using a sterilized, continuous-wave Doppler ultrasound flow detector.
- The probe is placed on the antimesenteric border of the intestine to detect pulsatile Doppler signals. However, even in the best of hands, this technique remains unreliable in predicting which bowel will remain viable.
- Second-look laparotomy is essential in the management of acute mesenteric ischemia. No matter which adjunctive method is used intraoperatively to assess bowel perfusion and viability, second-look laparotomy is the most reliable means of determining viability of marginally perfused bowel once revascularization has been performed. The decision to perform a second-look laparotomy should be determined during the first operation and adhered to no matter what the condition of the patient is 24 to 48 hours later.

Management:

Once the diagnosis of acute mesenteric ischemia has been suspected or proven, prompt therapeutic intervention is necessary, regardless of the etiology of the mesenteric ischemia. The patient should be placed on appropriate cardiac monitors, and intravenous lines should be instituted for aggressive fluid resuscitation. Acidosis should be corrected. Pain should be controlled with analgesics. Intravenous heparinization and antibiotics should be administered promptly. A Foley catheter is inserted to monitor urine output. Any precipitating event should be sought out and corrected.

Nonocclusive Mesenteric Ischemia

- Management of NOMI is largely nonoperative. Once the diagnosis has been established with angiography,the treatment of the underlying precipitating cause is the most important therapeutic intervention.
- Methods such as optimizing fluid resuscitation, improving cardiac output, and eliminating vasopressor agents will have the greatest impact on outcome.
- An adjunctive therapy is selective catheterization of the SMA with direct intraarterial infusion of papaverine (30 to 60 mg/hr).
- The infusion is continued for at least 24 hours. Repeated angiography at regular intervals is required to determine the effectiveness of this therapy. If the patient presents with peritoneal signs on physical examination, an exploratory laparotomy will be required to resect frankly necrotic or gangrenous bowel.

- If an intraarterial infusion of papaverine has been initiated, it is important to continue this throughout the exploratory laparotomy. It is also imperative to perform a second-look laparotomy in this cohort of patients given the waxing and waning of this disease process.

Mesenteric Embolic Disease:

- The goal in the surgical treatment of acute mesenteric ischemia is to restore normal pulsatile flow to the SMA and resect nonviable intestine.
- In general, revascularization precedes resection.
- Depending on the etiology, the therapeutic approach varies. For embolic disease of the SMA, a surgical embolectomy is performed.
- Percutaneous interventional treatment of acute SMA occlusion has been described in the literature. However, its application is limited because most patients present with symptoms that warrant an exploratory laparotomy to evaluate intestinal viability.
- Even if thrombolytic therapy could restore blood flow to the ischemic intestine, most patients will experience pain sufficient to necessitate exploration.
- Furthermore, physical examination alone is not adequate to predict the extent and degree of intestinal ischemia.

Mesenteric Thrombotic Disease:

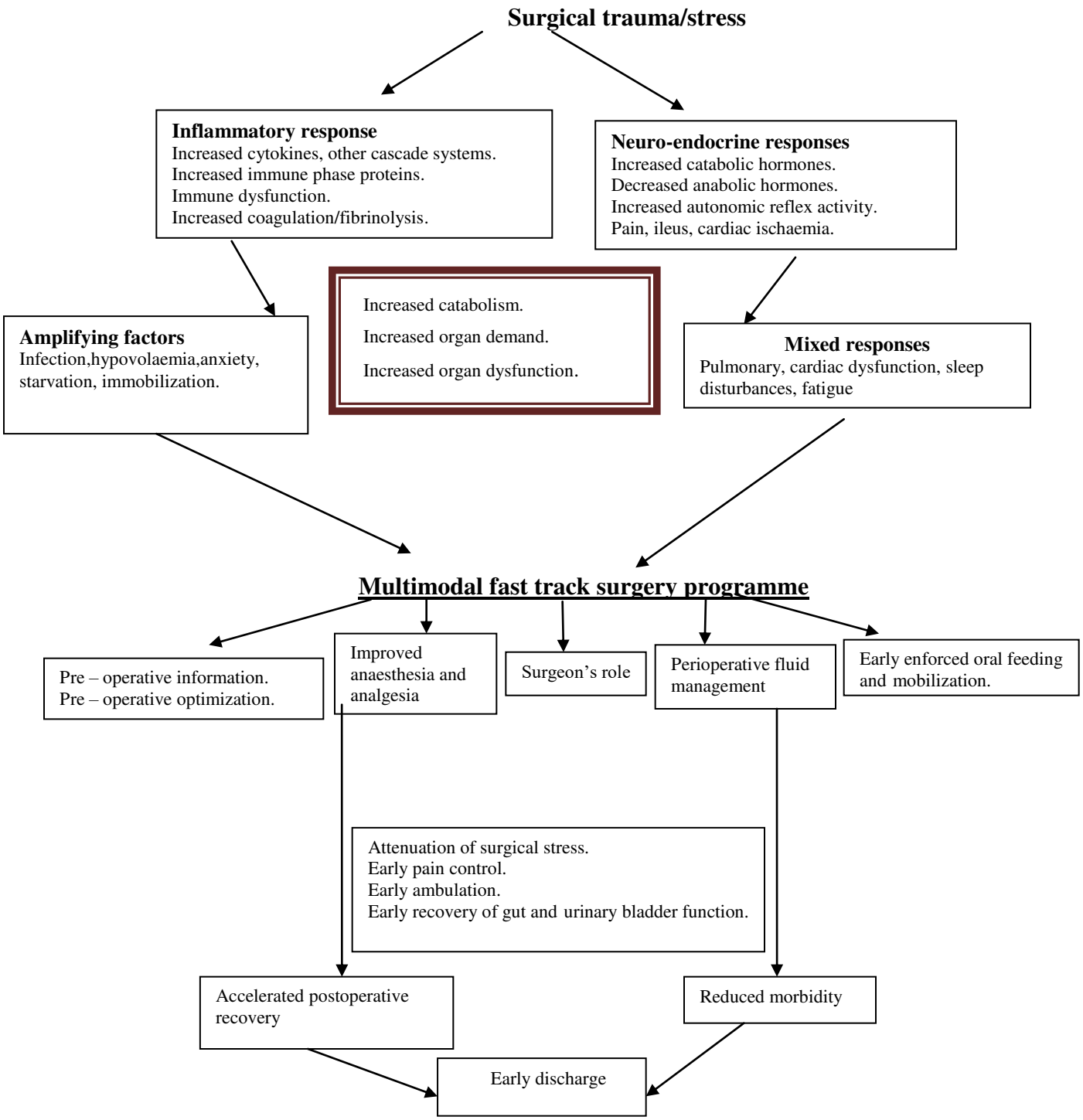
- ❖ Acute mesenteric ischemia secondary to thrombotic disease occurs in patients with longstanding atherosclerotic disease of the mesenteric vessels. When this occurs, usually the entire midgut is involved.
- ❖ Surgical treatment consists of a surgical bypass, either in an antegrade or a retrograde manner. The conduit of choice for the treatment of acute mesenteric ischemia is autologous greater saphenous vein in a reversed fashion. Synthetic graft material should be avoided in the setting of acute bowel ischemia, given the high risk of transmural bowel infarction and perforation.
- ❖ During acute mesenteric ischemia in which time is of the essence and prompt revascularization of the bowel is required, it is often more simple to perform a retrograde bypass from the infrarenal aorta or iliac artery because the exposure is much more simple.
- ❖ Although the management of acute mesenteric ischemia is initially surgical, the morbidity and mortality from this disease process are due to the ischemia/reperfusion insult and remain difficult to manage.

(d) Fast track Surgery.
Answer.

- Fast track Surgery is a multimodal, evidence – based comprehensive programme aimed at enhancing post operative recovery and outcome by attenuating stress – related organ dysfunction.
- It is important to understand that the discharge criteria (mobile patient,pain free on oral analgesia, complete recovery of gut and bladder functions) in fast – track surgery remain unaltered but achieved sooner.

- The modern concepts of patient education, pre – optimisation, improved analgesia and surgical techniques and better understanding of peri-operative care principles with enforced early oral feeding and ambulation have led to the success of fast – track surgery.
- Published evidence confirms the benefits of fast-track surgery including reduced hospitalization, low morbidity, cost – effectiveness, patient satisfaction and safety, with very low re-admission rates.
- Organization and team delivery with strict attention to the protocols are vital to the suces of a fast-track surgery programme.
- An analysis of outcome is essential to success and a database should be maimtained to aid this assessment

Mediators and modulators of catabolic pathways after surgery



THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES

MS (General Surgery) Examination, 2008

PAPER IV

Time Allowed: 3 Hours **Full Marks:100**

Attempt all questions

1. **Evaluate the place of pathological evaluation in colorectal cancer with special reference to TME. (20)**

2. Define morbid obesity and outline the various surgical options and their rationale. 5+15

3. Write short notes on: 5 x 6

a) **Thoracoscopic sympathectomy.**
b) **PET Scan.**
c) **TIPS.**
d) **Stapled haemorrhoidectomy.**
e) **Incentive spirometry.**

4. Answer briefly of the following: 4 x $7^1/_2$

a) **Day care surgery.**
b) **Surgical audit.**
c) **Robotics in Surgery.**
d) **Gastric varices.**

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES
MS (General Surgery) Examination, 2008

May 2008

PAPER IV

Time Allowed: 3 Hours **Full Marks:100**

Attempt all questions

1. **Evaluate the place of pathological evaluation in colorectal cancer with special reference to TME. (20)**

Answer.
Introduction: Total mesorectal excision (or TME) is a standard technique for treatment of colorectal cancer, devised some 20 years ago by Professor Bill Heald at the UK's Basingstoke District Hospital For patients with advanced rectal cancers, surgery that includes total mesorectal excision (TME) often provides the best possible patient outcomes and survival.

- The mesorectum is a fatty tissue directly adjacent to the rectum that contains blood vessels and lymph nodes. When rectal cancers recur, it is often in these lymph nodes.

- Patients who have rectal cancer that is confined to the lower two-third of the rectum are generally considered good candidates for TME surgery. However, many factors can determine whether TME is right for you, and your Cancer Center physician will ultimately help you choose the most appropriate treatment for your individual situation.

Principles of TME: Although TME has been modified over time, the basic principle of excising tumor and the mesorectum en bloc remains its foundation. This principle is based on the original observations of Moynihan19 in 1908 regarding potential pathways for lymphatic spread and also on the hypothesis of Heald that the mesorectum represents embryological advantages conferring pr tection against tumor dissemination until the terminal stages. Lymphoscintigraphy further demonstrated this in an anatomical study of the lymphatics that drain the rectum.

- Extent and technique of TME: The sigmoidal vessels are isolated. There is a clear space on either side of the vessels. Care is taken to preserve the hypogastric nerves. The sigmoid is divided. The point of vessel ligation is at the level of the left colic artery. The colon is divided and packed cephalad, out of the field. The left colon is mobilized by incising the white line of Toldt and freeing the splenic flexure attachments.
- TME begins by pulling the rectum up and incising the peritoneum on both sides down to meet anteriorly at the deepest point in the cul de sac. By retracting the rectum anteriorly, a loose areolar plane of avascular tissue is visible. Dissection in this plane prevents entry into presacral veins. The nerves are visible and kept posterior to the

plane of dissection. Both ureters should be visualized and retracted laterally. The tumor should be removed en bloc to include organs directly involved with tumor extension, such as the ovaries, uterus, bladder wall, and vagina. The presacral fascia is incised down to the rectosacral (Waldeyer's) fascia, a thickened band attaching the rectum to the endopelvic fascia at the S4 level. Divide this fascia and continue dissection to the coccyx.

- Continue the dissection laterally down to the lateral ligaments or "stalks," which may contain the middle rectal vessels. In fact, only 25% of patients have distinct branches of the middle rectal vessels in these ligaments. The lateral dissection ends at the levator muscles, the caudad boundary of the pelvic cavity.
- The anterior dissection is the most difficult because the planes are less distinct and the fat of the mesorectum is thin. This dissection is continued parallel to the plane of Denonvilliers' fascia between the rectum and the posterior wall of the vagina or the seminal vesicles. These structures are elevated anteriorly. Small vessels that traverse this area have a propensity for nuisance bleeding. The entire mesorectum should be contained within the fascia propria of the extraperitoneal rectum.

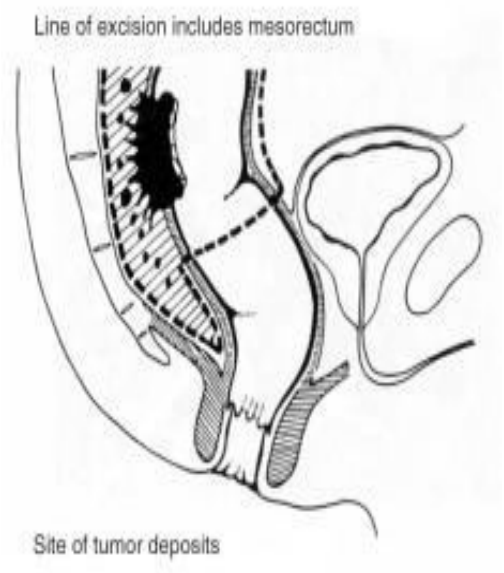
- The point of transection is chosen distal to the tumor. Multiple studies comparing distal cancer spread have shown that a 2-cm margin is necessary for low rectal excisions to perform an adequate oncologic resection.
- Less than 2% to 4% of tumors will have intraluminal implantation beyond 2 cm distally. Rigid sigmoidoscopy may be used to identify a point at least 2 cm distal to the carcinoma.
- Depending on the depth of the pelvis and therefore the diameter of the pelvis, a linear stapler of 30, 45, or 60 mm length is used for the first staple line in the double-stapling technique.
- The bowel is clamped just proximal to this point, the stapler is fired, and the rectum sharply transected and handed off the field. The operating field is secondarily isolated with towels while the open colon is manipulated.
- The proximal end of the colon is prepared by resecting off residual fat where the anastomosis is to be constructed. The staple line is sharply excised.
- A purse-string suture of 2-0 or 3-0 polypropylene is placed using full-thickness bites at 2-mm intervals.
- The anvil of the selected circular stapling device is inserted and the purse-string suture is tied around the shaft.
- The stapler is inserted transanally up to the previous staple line where the rectum was cut across.
- The trocar is introduced through or adjacent to the staple line. The anvil and the stapler are joined.
- The colon is inspected to verify that no adjacent tissue is entrapped and that the bowel is not twisted. The stapler is fired, opened slightly, and then removed. This is the second staple line in the double-stapling technique.
- The tissue removed in the stapler is checked to see that the tissue from the proximal and distal bowel is intact in two rings, the so-called "doughnuts."

- Check the anastomosis by clamping the bowel proximally, filling the pelvis with saline, and insufflating air under direct vision with a rigid proctoscope to see if bubbles can be detected coming through the anastomosis from the abdominal view.
- The rigid proctoscope permits assessment of the anastomosis for bleeding or saline entering the lumen from the abdomen.

- For the low coloanal anastomosis and for the preoperatively radiated pelvis, we routinely perform a diverting loop ileostomy at a premarked site.
- The site is kept away from bony prominences, the umbilicus, scars, and creases and is determined with the patient sitting, standing, and lying supine.
- This can usually be closed within 12 weeks of surgery.
- A 2 cm in diameter skin opening is made; dissection is carried down to the anterior rectus sheath.
- The fascial sheath is opened in a cruciate fashion, the muscle split bluntly, and the posterior peritoneum opened.
- The ileum is brought through the rectus muscle, identifying which end is proximal. The ostomy is matured following skin closure and dressing placement.
- The ileum is incised transversely 1 cm above the skin on the distal limb of the loop.
- As these cut edges of the ileum are sewn to the skin, the proximal ileum is everted to create an elevated tip.

- Macroscopic examination of the mesorectal surface helps us evaluate the quality of the surgical specimen.

Mesorectal defects are classified into three categories:
(a) Complete: mesorectum is intact, smooth with only minor irregularities without defect > 5 mm;
(b) Moderate: moderate bulk to mesorectum but irregularity of the mesorectal surface; muscularis propria is not visible with the exception of the area of insertion of levator muscles, and
(c) Incomplete: little bulk to mesorectum with defects down into muscularis propria.



Circumferential Resection Margin:

- After the introduction of TME, the CRM on the nonperitonealized surface of the resected rectal specimen isone of the most important predictors of local recurrence. Quirke et al.were the first to show that the radial or CRM is an important factor for local recurrence. The CRM can be involved by the tumor in various ways, such as direct spread, by tumor emboli in veins, lymphatics or lymph nodes or by tumor deposits discontinuous from the main growth.
- The rectum is covered anteriorly by peritoneum and only in the area below the peritoneal reflection is there a risk of the CRM being involved. The posterior area, which continues up to the sigmoid mesocolon, is at risk of being involved by the tumor not only by direct spread but also with metastatic deposits in lymph nodes. The mesorectum is thinner anteriorly than posteriorly and the risk of a positive resection margin is higher for anteriorly located tumors than in posterior ones.
- Microscopic tumor nodules can be observed in the mesorectum. Studies have shown that the presence of widespread microscopic nodules in the mesorectum was present in up to 38.7%.
- Other studies proved that lymph nodes <5 mm in diameter are more often involved by the tumor than bigger ones. Such findings pinpoint the importance of TME.
- The pathologist must differentiate the peritonealized from nonIf a tumor is detected on the non-peritonealized surface, adjuvant radiotherapy may be appropriate without the dimensions of the tumor to be considered.

  - The macroscopic CRM is measured with a ruler; the microscopic CRM measurement is done better by using a sheet of graph paper that is photocopied onto a sheet of acetate and cut to size than to using the Vernier scale .
  - The CRM is divided into two categories: an involved (positive) CRM when the tumor extends to within 1 mm of the circumferential margin and an uninvolved (negative) CRM when the distance between the tumor and CRM is >1 mm.
  - All the studies in the literature consider a positive CRM to be a distance between tumor and margin of <1 mm.
  - Several studies also indicated that the frequency of a positive CRM is higher in abdominoperineal resection than in low anterior resection.

TME With or Without Radiotherapy:

- The initial report by Heald expounded the virtue of TME as an adjunctive therapy in its own right but shied away from suggesting TME as a replacement for chemoradiotherapies of the time.
- Adjunctive combination therapies came to the fore with the positive findings of Krook and colleagues,published in 1991.
- A National Institutes of Health consensus statement followed shortly afterward, which cemented the acute change in postoperative management of rectal tumors.
- Now patients were routinely receiving chemoradiotherapy, and trials regarding preoperative timing of adjuvant radiotherapy were coming to fruition. Preoperative radiotherapy provides a number of potential benefits: the disease may be “down-

staged" (particularly with long-course radiotherapy), small extramural deposits may be cleared, techniques to exclude the small intestine from the field of therapy are more effective, the field should be relatively better oxygenated, and there is no new anastomosis.

- Intuitively, the oncologic locoregional benefits of adjunctive radiotherapy would marry well with augmented clearance by TME. The combination of techniques would be expected to convey low local failure rates.

The pathological assessment of mesorectal excision:

- Careful macroscopic evaluation of the resection specimen should be standardized. This may be supplemented by stain marking after postoperative filling the inferior mesenteric or superior rectal artery with ink or methylene blue solution. Photodocumentation is highly desirable. The pathological assessment of adequacy of mesorectal excision should be taken into account in selection for adjuvant radiotherapy.
- Objective macro- and microscopic assessment of mesorectal excision by pathologists is essential for quality management throughout patient care and in clinical trials.

2. Define morbid obesity and outline the various surgical options and their rationale. 5+15

Answer. See the answer of Q. No.2 of Paper – IV of 2013, Q. No.1 of Paper –IV of 2010 and Q.No.1 of Paper –III, 2009

3. Write short notes on: 5 x 6

a) Thoracoscopic sympathectomy.
b) PET Scan.
c) TIPS.
d) Stapled haemorrhoidectomy.
e) Incentive spirometry.

Answer.
(a) Thoracoscopic sympathectomy.

- Thoracoscopic sympathectomy resolves primary hyperhidrosis, Raynaud's phenomenon and excessive facial blushing.
- During the procedure, a surgeon partially disables the nerve that controls the sweat glands as well as the dilation of blood vessels near the surface of the skin of the face and fingers.
- Performed as a minimally invasive procedure, thoracoscopic sympathectomy has few risks, but people who undergo the surgery often experience later sweating problems in other parts of their bodies.

Procedure: During a thoracoscopic sympathectomy, a surgeon severs and seals the right-hand side of the sympathetic nerve, which runs along the inside of the spinal

column. The nerve gets cut at a point parallel to the space between the second and third ribs. Most thoracoscopic sympathectomies are now performed as endoscopic procedures, in which the surgeon makes a small incision below the right armpit to access the sympathetic nerve and uses a flexible camera cable to guide the application of surgical instruments. Patients undergoing endoscopic thoracoscopic sympathectomy receive general anesthesia and must have their right lung deflated during the surgery, but patients generally do not need to stay in the hospital overnight. Full recovery occurs within a few days.

Risks and Complications:

- Patients can expect some pain following a thoracoscopic sympathectomy.
- Also, an initial thoracoscopic sympathectomy does not always resolve a patient's problem, so a second procedure may be necessary.
- Rarer risks from the procedure include a collapsed lung caused by the collection of air in the chest cavity during surgery and inadvertent damage to other nerves near the sympathetic nerve.
- Some patients have developed Homer's syndrome following thoracoscopic sympathectomy, experiencing drooping eyelids, constricted pupils and reduced facial sweating.

(b) PET Scan.

Answer. Positron emission tomography (PET) is a nuclear medicine imaging technique which produces a three-dimensional image or picture of functional processes in the body. The system detects pairs of gamma rays emitted indirectly by a positron-emitting radionuclide (tracer), which is introduced into the body on a biologically active molecule. Images of tracer concentration in 3- dimensional or 4-dimensional space (the 4th dimension being time) within the body are then reconstructed by computer analysis. In modern scanners, this reconstruction is often
accomplished with the aid of a CT X-ray scan performed on the patient during the same session, in the same machine.
If the biologically active molecule chosen for PET is FDG, an analogue of glucose, the concentrations of tracer imaged then give tissue metabolic activity, in terms of regional glucose uptake. Although use of this tracer results in the most common type of PET scan, other tracer molecules are used in PET to image the tissue concentration of many other types of molecules of interest.

Operation: To conduct the scan, a short-lived radioactive tracer isotope is injected into the living subject (usually into blood circulation). The tracer is chemically incorporated into a biologically active molecule. There is a waiting period while the active molecule becomes concentrated in tissues of interest; then the research subject or patient is placed in the imaging scanner. The molecule most commonly used for this purpose isfluorodeoxyglucose (FDG), a sugar, for which the waiting period is typically an hour. During the scan a record of tissue concentration is made as the tracer decays. As the

radioisotope undergoes positron emission decay (also known as positive beta decay), it emits a positron, an antiparticle of the electronwith opposite charge. After travelling up to a few millimeters[quantify] the positron encounters an electron. The encounter annihilates them both, producing a pair of annihilation (gamma) photons moving in opposite directions. These are detected when they reach a scintillator in the scanning device, creating a burst of light which is detected by photomultiplier tubes or silicon avalanche photodiodes (Si APD). The technique depends on simultaneous or coincident detection of the pair of photons moving in approximately opposite direction (it would be exactly opposite in their center of mass frame, but the scanner has no way to know this, and so has a built-in slight direction-error tolerance). Photons that do not arrive in temporal "pairs" (i.e. within a timing-window of few nanoseconds) are ignored.

Localization of the positron annihilation event: The most significant fraction of electron-positron decays result in two 511 keV gamma photons being emitted at almost 180 degrees to each other; hence it is possible to localize their source along a straight line of coincidence (also called formally the line of response or LOR). In practice the LOR has a finite width as the emitted photons are not exactly 180 degrees apart. If the resolving time of the detectors is less than 500 picoseconds rather than about 10 nanoseconds, it is possible to localize the event to a segment of a chord, whose length is determined by the detector timing resolution. As the timing resolution improves, the signal-to-noise ratio (SNR) of the image will improve, requiring fewer events to achieve the same image quality. This technology is not yet common, but it is available on some new systems.

Image reconstruction using coincidence statistics:
More commonly, a technique much like the reconstruction of computed tomography (CT) and single photon emission computed tomography (SPECT) data is used, although the data set collected in PET is much poorer than CT, so reconstruction techniques are more difficult. Using statistics collected from tens-of-thousands of coincidence events, a set of simultaneous equations for the total activity of each parcel of tissue along many LORs can be solved by a number of techniques, and thus a map of radioactivities as a function of location for parcels or bits of tissue (also called voxels), may be constructed and plotted. The resulting map shows the tissues in which the molecular probe has become concentrated, and can be interpreted by a nuclear medicine physician or radiologist in the context of the patient's diagnosis and treatment plan.

Combination of PET with CT and MRI:
PET scans are increasingly read alongside CT or magnetic resonance imaging (MRI) scans, the combination ("co-registration") giving both anatomic and metabolic information (i.e., what the structure is, and what it is doing biochemically). Because PET imaging is most useful in combination with anatomical imaging, such as CT, modern PET scanners are now available with integrated high-end multi-detector-row CT scanners. Because the two scans can be performed in immediate sequence during the same session, with the patient not changing position between the two types of scans, the two sets of images are more-precisely registered, so that areas of abnormality on the PET imaging can be more perfectly correlated with anatomy on the CT images. This is very

useful in showing detailed views of moving organs or structures with higher anatomical variation, which is more common outside the brain.
PET-MRI: Presently, only the head and brain can be imaged at these high magnetic field strengths.

Radionuclides: Radionuclides used in PET scanning are typically isotopes with short half lives such as carbon- 11 (~20 min), nitrogen-13 (~10 min), oxygen-15 (~2 min), and fluorine-18 (~110 min).
It is important to recognize that PET technology can be used to trace the biologic pathway of any compound in living humans (and many other species as well), provided it can be radiolabeled with a PET isotope. Thus the specific processes that can be probed with PET are virtually limitless, and radiotracers for new target molecules and processes are being synthesized all the time; as of this writing there are already dozens in clinical use and hundreds applied in research. Presently, however, by far the most commonly used nuclide in clinical PET scanning is fluorine-18 in the form of FDG. The half life of fluorine-18 is long enough such that fluorine-18 labeled radiotracers can be manufactured commercially at an offsite location.

Limitations:

- The minimization of radiation dose to the subject is an attractive feature of the use of short-lived radionuclides. Besides its established role as a diagnostic technique, PET has an expanding role as a method to assess the response to therapy, in particular, cancer therapy, where the risk to the patient from lack of knowledge about disease progress is much greater than the risk from the test radiation.
- Limitations to the widespread use of PET arise from the high costs of cyclotrons needed to produce the short-lived radionuclides for PET scanning and the need for specially adapted on-site chemical synthesis apparatus to produce the radiopharmaceuticals. This limitation restricts clinical PET primarily to the use of tracers labelled with fluorine-18, which has a half life of 110 minutes and can be transported a reasonable distance before use, or to rubidium-82, which can be created in a portable generator and is used for myocardial perfusion studies.
- The presence of the small on-site cyclotron promises to expand in the future as the cyclotrons shrink in response to the high cost of isotope transportation to remote PET machines Because the half-life of fluorine-18 is about two hours, the prepared dose of a radiopharmaceutical bearing this radionuclide will undergo multiple half-lives of decay during the working day. This necessitates frequent recalibration of the remaining dose (determination of activity per unit volume) and careful planning with respect to patient scheduling.

PET is a valuable technique for some diseases and disorders, because it is possible to target the radio-chemicals used for particular bodily functions.

Oncology:

1. PET scanning with the tracer fluorine-18 (F-18) fluorodeoxyglucose (FDG), called FDG-PET, is widely used in clinical oncology. This tracer is a glucose analog that is taken up by glucose-using cells and phosphorylated by hexokinase (whose mitochondrial form is greatly elevated in rapidly growing malignant tumours).

2. A typical dose of FDG used in an oncological scan is 200- 400 mBq for an adult human. Because the oxygen atom which is replaced by F-18 to generate FDG is required for the next step in glucose metabolism in all cells, no further reactions occur in FDG.
3. FDG-PET can be used for diagnosis, staging, and monitoring treatment of cancers, particularly in Hodgkin's lymphoma, non-Hodgkin lymphoma, and lung cancer.
4. Many other types of solid tumors will be found to be very highly labeled on a case-by case basis—a fact which becomes especially useful in searching for tumor metastasis, or for recurrence after a known highly active primary tumor is removed.
5. Because individual PET scans are more expensive than "conventional" imaging with computed tomography (CT) and magnetic resonance imaging (MRI), expansion of FDG-PET in cost-constrained health services will depend on proper health technology assessment; this problem is a difficult one because structural and functional imaging often cannot be directly compared, as they provide different information. Oncology scans using FDG make up over 90% of all PET scans in current practice.

Neurology:

1. PET neuroimaging is based on an assumption that areas of high radioactivityare associated with brain activity. Early diagnosis of Alzheimer's disease can be done.
2. Cardiology, atherosclerosis and vascular disease study: In clinical cardiology, FDG-PET can identify so-called "hibernating myocardium", but its cost-effectiveness in this role versus SPECT is unclear.
3. Neuropsychology / Cognitive neuroscience: To examine links between specific psychological processes or disorders and brain activity.

Psychiatry:

1. Numerous compounds that bind selectively to neuroreceptors of interest in biological psychiatry have been radiolabeled with C-11 or F-18. Radioligands that bind todopamine receptors (D1,D2, reuptake transporter), serotonin receptors (5HT1A, 5HT2A, reuptake transporter) opioid receptors (mu) and other sites have been used successfully in studies with human subjects.
2. Studies have been performed examining the state of these receptors in patients compared to healthy controls in schizophrenia, substance abuse,mood disorders and other psychiatric conditions.

Pharmacology: In pre-clinical trials, it is possible to radiolabel a new drug and inject it into animals.

Pulse Shape Discrimination: The pulse Shape Discrimination (PSD) is a technique used to define which pulse is related to each crystal. Different Techniques were introduced to discriminate between two-types of pulses according to its shape (indeed due to the decay time).

- **Safety:** PET scanning is non-invasive, but it does involve exposure to ionizing radiation. The total dose of radiation is not insignificant, usually around 11 mSv. When compared to the classification level for radiation workers in the UK, of 6 mSv it can be seen that PET scans need proper justification. This can also be compared to 2.2 mSv average annual background radiation in the UK, 0.02 mSv for a chest x-ray

and 6.5 - 8 mSv for a CT scan of the chest, according to the Chest Journal and ICRP. A policy change suggested by the IFALPA member associations in year 1999 mentioned that an aircrew member is likely to receive a radiation dose of 4–9 mSv per year.

(c) TIPS.

Answer. Transjugular Intrahepatic Portosystemic Shunt

Indications for TIPS include

(1) Patients with esophageal varices hemorrhage who are refractory to conventional medical therapy (i.e., medications administered to reduce portal pressure);

(2) Patients who have had maximized endoscopic banding and continue to bleed;

(3) Intractable ascites refractory to medical therapy;

(4) Patients who are not surgical candidates for conventional portosystemic shunts;

(5) A bridge to hepatic transplantation;

(6) Patients with recurrent hydrothorax due to cirrhosis; and

(7) Patients with portal hypertension due to Budd-Chiari syndrome. The application of TIPS in patients with portal hypertensive gastropathy and hepatorenal syndrome remains controversial.

Contraindications to the TIPS procedure include

(1) Patients with advanced hepatic encephalopathy;

(2) Patients with significant cardiac disease (e.g., patients with marked valvular insufficiency, global cardiac dysfunction, significant pulmonary hypertension, constrictive pericarditis);

(3) Patients with any polycystic liver disease in which the path of the shunt would traverse liver cysts;

(4) Patients with active infection (peritoneal, intrahepatic, systemic, etc.);

and

(5) Patients with hypervascular primary or metastatic hepatic tumors in the path of the proposed TIPS.

Relative contraindications to TIPS include

(1) Those patients with portal vein occlusion (in this setting, aggressive percutaneous interventional therapy may result in successful recanalization of the portal vein; such procedures may require percutaneous angioplasty and stenting of the portal vein prior to creation of TIPS);

and

(2) Patients with impending liver failure.

Technique of TIPS: Prior to the TIPS procedure, diagnostic imaging to confirm patency of the portal vein and to define hepatic configuration is essential. Portal vein patency may be assessed using color-flow Doppler ultrasound, computed tomography (CT) with intravenous contrast, or magnetic resonance imaging (MRI). Though more invasive arterial portography may also define vascular anatomy (e.g., delayed imaging obtained during the venous phases of selective celiac or super mesenteric artery [SMA] injections). However, in those patients with severe portal hypertension and hepatofugal flow, arterial portography may not accurately define anatomy of the portal vein (e.g., patency). In addition, use of iodinated contrast in patients with renal dysfunction may cause further deterioration with progressive elevation of creatinine.

Patients with advanced liver disease may be hypovolemic due to (1) recent life-threatening variceal hemorrhage; (2) aggressive medical treatment (e.g., diuretic therapy) used for managing ascites; or (3) hypovolemia due to large-volume paracenteses. Preprocedure planning and noninvasive imaging are important for determining the approach (e.g., creating the TIPS from the right hepatic to right portal veins or middle hepatic to left portal veins).

TIPS has been approved by the U.S. Food and Drug Administration (FDA) as a minimally invasive alternative in the management of patients with portal hypertension.

For the standard TIPS procedure, percutaneous puncture into the right internal jugular vein is made. A straight intravascular sheath is advanced to the level of the superior vena cava-right atrial junction. A multipurpose catheter (usually 6–7 Fr) and a standard angiographic guidewire are advanced through the sheath and used to selectively catheterize a hepatic vein. Free and wedged hepatic venography is generally performed, and the largest hepatic vein with the most favorable angle to the portal vein is selected. Often this is the right hepatic vein. Contrast injected with the catheter wedged in a peripheral hepatic vein may document patency of the portal vein and demonstrate hepatofugal flow. Carbon dioxide may also be used.

With the hepatic vein selected, the multipurpose catheter is exchanged for an angled 9F guiding catheter. Through this, a 16-gauge angulated needle is directed in a manner similar to that used for transjugular liver biopsies. Technique modifications have included the development of smaller-gauge needles to probe through the cirrhotic liver.

The transjugular hepatic biopsy needle is advanced through the angulated sheath. With the tip of the sheath in the orifice of the hepatic vein, the needle exits the distal end of the sheath and is directed into the hepatic vein wall. A puncture through the hepatic vein wall and hepatic parenchyma is made and the needle is advanced toward the portal vein (avoiding the bifurcation). Needle passes are generally "blind," although some interventional radiologists monitor needle passes under imaging guidance (e.g., real-time ultrasound).If the portal vein has been entered, venous blood is aspirated. Contrast is injected to confirm portal vein entry.

When the needle enters a main right or left portal vein branch and position is confirmed by contrast injection, a floppy-tipped guidewire is directed through the needle into the main portal vein. The 9F sheath is advanced through the tract. The needle is removed, and a catheter is directed over the guidewire into the portal vein. The guidewire is removed, and direct portal pressures are measured and recorded. The right atrial pressure is also recorded through the jugular vein sheath. The gradient between the portal vein and right atrium (i.e., central venous pressure) is calculated.

A portal venogram using iodinated contrast, or alternatively carbon dioxide, is performed to define anatomy. A stiff guidewire is reintroduced through the 5F catheter and an angioplasty balloon is used to dilate the vein walls and parenchymal tract (i.e., between hepatic vein and portal vein). During dilation, a waist on the balloon is typically identified at the hepatic vein and portal vein entry sites. After complete tract dilation, the angioplasty balloon is removed over the guidewire. Unless a stent is placed in the newly created parenchymal tract, the tract will close. At the authors' institution (George Washington University Medical Center), a self-expanding metallic stent is directed over the guidewire and positioned such that complete coverage of the parenchymal tract will be achieved when fully expanded. A small segment of stent extends into both the main portal and hepatic veins. Gaps, if present, may result in premature shunt thrombosis or restenosis. Therefore it is essential that the entire course of the parenchymal tract be paved with stent material. If necessary, stents are overlapped to prevent gaps in coverage. Once the self-expanding metallic stents are deployed, repeat balloon dilation further expands the stent. The stent's struts are embedded in parenchymal tissue and the stent maintains patency of the percutaneously created tract between hepatic and portal veins. Care is taken to avoid placing the stents into the inferior vena cava or main portal vein because this may jeopardize possible future hepatic transplantation.

Pressure measurements are obtained after the TIPS procedure, and the shunt is dilated again, if necessary, to increase its diameter and further reduce the gradient. We have found that a 10- to 12-mm diameter stent is generally sufficient to decompress esophageal varices without completely shunting blood through the liver. The recommended reduction in pressure gradient remains controversial; however, most investigators attempt to reduce the gradient to approximately 10 mm Hg. (This is based on retrospective data analysis in patients with portal hypertension undergoing surgically created portosystemic shunts. In such patients, the relationship between the presence of bleeding esophageal varices and the portal-hepatic vein gradient was evaluated). To date, the optimal gradient has not been prospectively determined.

If the indication for the TIPS procedure is active bleeding from esophageal varices, the left gastric vein (i.e., coronary vein) may be occluded using transcatheter embolotherapy techniques. Gelfoam (Upjohn, Kalamazoo, Mich.), Gianturco occlusion spring coils (Cook, Inc., Bloomington, Ind.), and/or sclerosing agents may be used to occlude the left gastric (coronary) vein and its varicoid tributaries.

After the TIPS procedure, the patient is observed in the recovery room for any signs of intraperitoneal hemorrhage, and so on. These patients are usually transferred to a "monitored bed."

The application of TIPS in a patient with chronic Budd-Chiari syndrome provides perhaps the greatest technical challenge. In such patients, there is often a small occluded hepatic vein orifice to attempt placement of the shunt. The shunt itself is long, and multiple stents are overlapped. In such patients, extreme care must be taken to avoid placing stents into the right atrium or too far into the portal vein.

As further experience accumulates, the TIPS procedure plays an increasing role in the management of patients with portal hypertension. Although restenosis and shunt thrombosis continue to be problematic.

(d) Stapled haemorrhoidectomy.

Answer. Severe cases of hemorrhoidal prolapse – 3rd and 4th Degree – will normally require surgery. Traditional hemorrhoidectomy is notorious for the level of post operative pain the patient must endure, coupled with a long recuperation period.

Newer surgical procedures include stapled transanal rectal resection (STARR) and procedure for prolapse and hemorrhoids (PPH). Both STARR and PPH are contraindicated in persons with either enterocele or anismus.

Procedure for Prolapse and Hemorrhoids (PPH)

PPH uses a circular stapler to reduce the degree of prolapse. The procedure avoids the need for wounds in the sensitive perianal area thus reducing post-operative pain considerably, and facilitates a speedier return to normal activities.[2][3][4]

Development

This procedure was first described by an Italian surgeon – Dr. Antonio Longo, Department of Surgery, University of Palermo – in 1993 and since then has been widely adopted through Europe.

This procedure avoids the need for wounds in the sensitive perianal area and, as a result, has the advantage of significantly reducing the patient's post operative pain. Follow-up on relief of symptoms indicate a similar success rate to that achieved by conventional haemorrhoidectomy.

Since PPH was first introduced it has been the subject of numerous clinical trials and in 2003 the National Institute of Clinical Evidence (NICE) in the UK issued full guidance on the procedure stating it was safe and efficacious.

How the Procedure works

Prolapse:

PPH employs a unique circular stapler which reduces the degree of prolapse by excising a circumferential strip of mucosa from the proximal anal canal. This has the effect of pulling the hemorrhoidal cushions back up into their normal anatomical position.

Bleeding:

In addition to correcting the symptoms associated with the prolapse, problems with bleeding from the piles are also resolved by this excision. Although the cushions may be totally or partially preserved, the blood supply in interrupted or venous drainage is improved by the repositioning. Any external component which remains will usually regress over a period of 3–6 months. Prominent skin tags may, on occasion, be removed during the operation, but this has not been associated with any significant increase in pain.

Indications:

PPH is generally indicated for the more severe cases of hemorrhoidal prolapse (3rd and 4th degree) where surgery would normally be indicated. It may also be indicated for patients with minor degree haemorrhoids who have failed to respond to conservative treatments. The procedure may be contra-indicated when only one cushion is prolapsed or in severe cases of fibrotic piles which cannot be physically repositioned.

- The Procedure: Usually the patient will be under general anesthetic, but only for 20–30 minutes. Many cases have been successfully performed under local or regional anesthesia and the procedure is suited to day case treatment.
- Post Operative Course: Due to the low level of post-operative pain and reduced analgesic use, patients will usually be discharged either the same day or on the day following surgery.

Most patients can resume normal activities after a few days when they should be fit for work. The first bowel motion is usually on day two and should not cause any great discomfort. Staples may be passed from time to time during defecation. This is normal and should not be a cause for concern.

- Post-operative complications: Urinary retention in the immediate post-operative period appears to be the most common complication.

Should there be some post-operative bleeding, this can be dealt with either by the district nurse or in the GP surgery. If it is still a concern, refer the patient back to the hospital.

A condition called post PPH syndrome is also possible. This occurs when the staples are placed slightly low and it can cause the internal sphincter muscle to become inflamed. Treatment includes anti-inflammatory oral medications and suppositories.

(e) Incentive spirometry.

Answer. An incentive spirometer is a medical device used to help patients improve the functioning of their lungs. It is provided to patients who have had any surgery that might

jeopardize respiratory function, particularly surgery to the lungs themselves, but also commonly to patients recovering from cardiac or other surgery involving extended time under anesthesia and prolonged in-bed recovery. The incentive spirometer is also issued to patients recovering from rib damage to help minimize the chance of fluid build-up in the lungs.

The patient breathes in from the device as slowly and as deeply as possible. An indicator provides a gauge of how well the patient's lung or lungs are functioning, by indicating sustained inhalation vacuum. The patient is generally asked to do many repetitions a day while measuring his or her progress by way of the gauge.

How to use:

- Sit or lie upright in a comfortable position.
- Hold the incentive spirometer upright, with both hands.
- Slide the indicator (located in the left-hand column when you are facing the spirometer) to the desired level. For example, start at 1250 milliliters and slowly increase as your treatment progresses.
- Place the mouthpiece into your mouth and tightly seal your lips around it.
- With your lips tightly sealed around the mouthpiece, breathe in slowly and as deeply as possible. The piston that is resting below the indicator should now rise toward the top of the column.
- Hold your breath for at least 3 seconds and allow the piston to fall back to the bottom of the column.
- After each set of deep breathing, cough to help clear your airways of mucus.
- Rest for a few seconds and repeat steps two through eight, 10 times each hour while you are awake.

Incentive spirometry: a method of encouraging voluntary deep breathing by providing visual feedback about inspiratory volume. Using a specially designed spirometer, the patient inhales until a preset volume is reached, then sustains the inspiratory volume by holding his or her breath for 3 to 5 seconds. Incentive spirometry reduces the risk of atelectasis and pulmonary consolidation.

Note: Incentive spirometry does not clearly help reduce pulmonary complications during the perioperative care after coronary artery bypass grafting according to ameta-analysis of randomized controlled trials by the Cochrane Collaboration.

It is not clear that incentive spirometry is better than supervised deep breathing according to a randomized controlled trial.

4. Answer briefly of the following: 4 x $7^1/_2$

a) Day care surgery.
b) Surgical audit.
c) Robotics in Surgery.
d) Gastric varices.

(a) Day care surgery.

Answer.

Introduction: In the modern day hospitals, there is an established belief of ambulating the surgical patient as early as possible. This philosophy has gone a step further by sending the post-operative patient home as soon as the critical period or immediate post-operative nursing needs have been met with. This has led to the concept of day care surgery, which is evidenced by the establishment of plenty of day care surgical centres in the West, followed by the hospitals of developing countries as well.

The first report of operations being performed under general anaesthesia on ambulatory basis in the Western Hemisphere was that of Dr RM Waters of Sioux City, Iowa in 1918. The first successful freestanding outpatient surgery centre was opened by Charles Hill and four associates called as Dudley Street Ambulatory Surgical Centre in Providence, Rhode Island, in 1968.
It would be pertinent to mention that day care surgery is different from outpatient surgery in that the patients of day care surgery need some degree of post-operative specialised nursing care necessitating post-op observation for a few hours. All day care surgical patients essentially come early in the morning and after the surgery is over and the post-operative observation is uneventful, the patient is discharged home the same day. Facilities and standards of care in the day care surgical unit must be comparable to those provided for in-patient. The patient is to be formally admitted to day care unit, full work up and documentation is done and there should be facilities to observe the patient for 12 hours and with the possibility of admission.
Advantages of day care surgery:

Advantages to the patient		Advantages to the purchaser/ hospital:
• Pre-booked date and less likely to be cancelled • Shorter waiting lists and lesser uncertainty of a long wait • Easier domestic arrangements	• Reduced risk of cross-infection • Avoidance of disruptive nights in hospital wards • Less loss of time at work • Less psychological disturbances in children	• Reduced cost • High patient satisfaction • High volume, low risk surgery • Lesser incidence of hospital acquired infections • Cost effective

• Earlier mobilization • Minimal disruption of patient's personal life • Earlier return to normal environment	• Lesser chances of cancellation due to pressures of emergency surgeries in a dedicated day care facility.	

Disadvantages of day care surgery:

- The need for a responsible person to oversee the day care patient at home for the first 24-48 hours.
- The restriction of day case surgery to experienced senior staff; little opportunity for junior staff to practice.
- Extra work for the general practitioner in the postoperative period; patients often ring them for advice or treatment.
- The cost-effectiveness of the unit is reduced when less complex cases are dealt with on a day basis.

Contra-indications to day surgery;

- Medical: Angina at rest, myocardial infarct in last six months, hypertensives – diastolic greater than 105 mmHg, cardiac failure, acute respiratory infection, asthma - moderate to severe require increased observation, chronic bronchitis , emphysema, goss obesity: body mass index > 35 ,Insulin dependent diabetics.
- Psychological: Psychologically unstable, e.g. pyschosis, concept of day surgery is unacceptable to the patient
- Social: Lives over one hour away from the centre. No reliable person to drive patient home after surgery and look after them for the first 24-48 hours postoperatively, At home, no access to a lift, telephone or indoor toilet and bathroom.

Selection of procedures: The procedures that can be undertaken as day care surgical procedures are as per the BADS or Audit Commission of UK which came up with 'Basket of 20' surgical procedures to provide a more consistent measure of performance. The 25 procedures included as found updated in the report of the Audit Commission of UK, 2000 are as follows: (Updated report, 2001)

1. Orchidopexy	6.Haemorrhoidectomy	11. Carpal tunnel decompression	16. Extraction of cataract with/without implant	21. Reduction of nasal fracture
2. Circumcision	7. Laparoscopic cholecystectomy	12. Excision of ganglion	17. Correction of squint	22. Operation for bat ears
3. Inguinal hernia repair	8. Varicose vein stripping or ligation	13. Arthroscopy	18. Myringotomy	23. Dilatation and curettage/hysteroscopy
4. Excision of breast lump	9. Transurethral resection of bladder tumour	14. Bunion operations	19. Tonsillectomy	24. Laparoscopy
5. Anal fissure dilatation or excision	10. Excision of dupuytren's contracture	15. Removal of metalware	20. Sub Mucous resection	25. Termination of Pregnancy

The British Association of Day Surgery (BADS) has recommended inclusion of another fifty procedures under the name of Trolley of procedures.
Types of Day Care Surgical Centres:
James E Davis and Wallace A Reed described that Day Care Surgery can take\ place in various settings which are basically four types in use .

Hospital Integrated Unit : This unit provides a designated area to which patients are admitted and from which they are discharged home and in which preoperative evaluation and preparation are carried out. The hospital operating rooms and recovery rooms are used for both inpatients and Day Care Surgery patients. There is only one advantage in this type of unit, i.e. having to make little capital investment, but the disadvantages are; it is difficult to blend inpatients and Day Care Surgery patients in the same operating schedule.

Hospital Autonomous Unit: This unit is totally self-sufficient. This type of unit is located within the hospital or on the grounds of the hospital, but operates totally independent of other portions of the hospital. A tailor- made area is available for Day Care Surgery,to provide the best patient care from the point of view of the physical facilities. There is greater satisfaction on the part of all personnel, patients and surgeons since everything is tailor-made. The major disadvantage in this unit is loss of flexibility because if the unit is not successful or sufficiently utilised it is unlikely that the space can be utilised for alternative services of the hospital without any additional capital investment.

Hospital Satellite Unit: This is an autonomous facility which is sponsored and/or operated by the hospital but located away from the campus of the hospital.

Free-Standing Unit: This is an autonomous unit which is not geographically or administratively part of any other health care facility. The advantage of this type of unit is it meets the needs of a particular community as far as their Day Care Surgery requirements are concerned. The disadvantages of this type of unit are that there is a possibility of increase in the net community cost and also a greater distance from the hospital's emergency backup facilities.

Complications: The complication rates in day surgery, resulting in unexpected re-admissions are equally divided between problems with the surgery and anaesthesia: haemorrhage, pain in 50% Nausea and vomiting, dizziness in 50%

Conclusion: Day care surgery has now come to be an accepted modality of treatment for most surgical patients and it has multiple advantages to all the stakeholders as well as the patients, besides being an economically better option for the health sector which is beleaguered with scarce resources. Therefore, creating more day care centres should be in the better interest of the patients and the nation as a whole.

(b) Surgical audit.

Answer.

Introduction: Clinical audit is one of the “keystones” of clinical governance. A surgical department that subjects itself to regular and comprehensive audit should be able to

provide data to current and prospective patients about the quality of the services it provides, as well as reassurance to those who pay for and regulate health care. Well-organized audit should also enable the clinicians providing services to continually improve the quality of care they deliver. There are many similarities between audit and research, but historically audit has often been seen as the "poor relation." For audit to be meaningful and useful, it must, like research, be methodologically robust and have sufficient "power" to make useful observations; it would be easy to gain false reassurance about the quality of care by looking at outcomes in a small or "cherry-picked" group of straightforward cases. Audit can be conducted retrospectively or prospectively and, again like research, prospective audit has the potential to provide the most useful data, and routine prospective audit provides excellent opportunities for patient benefit.

Much of the experience we draw on comes from cardiac surgery, where there is a long history of structured data collection, both in the United States and in the United Kingdom. This was initially driven by clinicians, but more recently has been influenced by politicians and the media. Cardiac surgery is regarded as an easy specialty to audit in view of the high volume and proportion of a single operation coronary artery bypass graft (CABG) in most surgeons practice set against a small but significant hard measurement end point of mortality (which is typically approximately 2%).
Possible reasons for conducting clinical audit:

- As a result of local clinical interests
- As a result of clinical incident reporting
- To comply with regional or national initiatives
- To inform patients about surgical results
- To drive continuous quality improvement
- For health care regulation.

What data can be used for audit?
Routine hospital data:

- Ideally, hospitals should have clearly defined systems in place to use the data: for example, they should regularly compare their outcomes for chosen procedures against an appropriately selected group of other hospitals. Significant\ "good" practice should be celebrated and shared with others inside and outside the organization, and bad outcomes should be investigated.
- It is not infrequent that high mortality or other clinical indictor rates may have a clear explanation other than that of "bad" clinical practice. The data may be incorrect, or there may be issues about classification or attribution that explain away an apparent alert, but structured investigation should improve the organization's and the clinician's knowledge about their data systems and may lead to impressions that necessitate improvements in patient care.

Specialty-specific multicenter data: These are usually clinically driven and have benefits above routine hospital data in that a more useful dataset can be designed for specific purposes and in particular can look in more detail at subtleties of casemix and specific clinical outcomes in a way that is more robust and sensitive than that derived from routine hospital administration systems.

Locally derived data: Individual hospital departments will often decide to audit a specific theme that may be chosen because of clinical risk management issues, subspecialist interest, or other concerns.

To maximize the chances of improving care as a result of audit, the following should be considered.

- Will the sample size be big enough to be useful? What dataset is needed? Will that data be accessible from existing hospital case notes or will prospective data collection be necessary?
- Is there an existing robust benchmark to which the results of the audit can be compared? How will the "significance" of the results be analyzed?
- Does conducting the audit have financial implications?
- Will the potential results of the audit have financial implications?
- Are all stakeholders who may need to change their behavior as a result of the audit involved in the process?

Good practice in audit: A clinical department should benefit from a clear forward plan about its audit activity that should be developed by the multidisciplinary team in conjunction with patients and their carers. The audit activity should include an appropriate mix of national, local, and risk management driven issues, and the specifics should depend on the configuration of services and local preferences. The plan should include thoughts about dissemination of results to users and potential users of the services. The multidisciplinary team should include doctors, professionals allied to medicine, and administration staff. Adherence to the audit plan should be monitored through the departmental operational management structures. For the department to be successful in improving care as a result of audit, there should be clear understanding of effective techniques of change management.

Arguments against audit: In the United Kingdom, audit has been an essential part of all doctors' job plans for a number of years, but audit activity remains sporadic. In some high-profile specialties such as cardiac surgery, comprehensive audit has been led by clinicians and driven by politicians and the media. In other areas there has been little or no coordinated national audit activity. This may be due to a perceived lack of benefits from audit from clinicians along with failure to meet challenges in gaining consensus or difficulties in securing adequate resource. The experience from cardiac surgery is that structured national audit improves the quality of mortality outcomes. It is likely that other issues such as complication rates are also reduced with associated costs savings, and as such effective audit may well pay for itself.

Summary; In modern health care, patients are increasingly looking to be reassured about the quality of care they receive and doctors are being driven toward demonstrating their competence, rather than this being assumed. Hospital departments should have a robust clinical governance strategy that should include "joined-up" clinical risk management and audit activity. There are strong arguments that structured audit activity improves the

quality of outcomes and for these benefits to be maximized there should be involvement of multidisciplinary teams supported by high-quality operational management.

(c) Robotics in Surgery.

Answer. See the answer of question 4.a of Paper – IV of 2013

(d) Gastric varices.

Answer.

- Gastric varices are dilated submucosal veins in the stomach, which can be a life-threatening cause of upper gastrointestinal hemorrhage. They are most commonly found in patients with portal hypertension, or elevated pressure in the portal vein system, which may be a complication of cirrhosis.
- Gastric varices may also be found in patients with thrombosis of the splenic vein, into which the short gastric veins which drain the fundus of the stomach flow. The latter may be a complication of acute pancreatitis, pancreatic cancer, or other abdominal tumours. Patients with bleeding gastric varices can present with bloody vomiting (hematemesis), dark, tarry stools (melena), or frank rectal bleeding.
- The bleeding may be brisk, and patients may soon develop shock.
- Treatment of gastric varices can include injection of the varices with cyanoacrylate glue, or a radiological procedure to decrease the pressure in the portal vein, termed transjugular intrahepatic portosystemic shunt or TIPS. Treatment with intravenous octreotide is also useful to shunt blood flow away from the stomach's circulation. More aggressive treatment including splenectomy (or surgical removal of the spleen) or liver transplantation may be required in some cases.

Clinical presentation

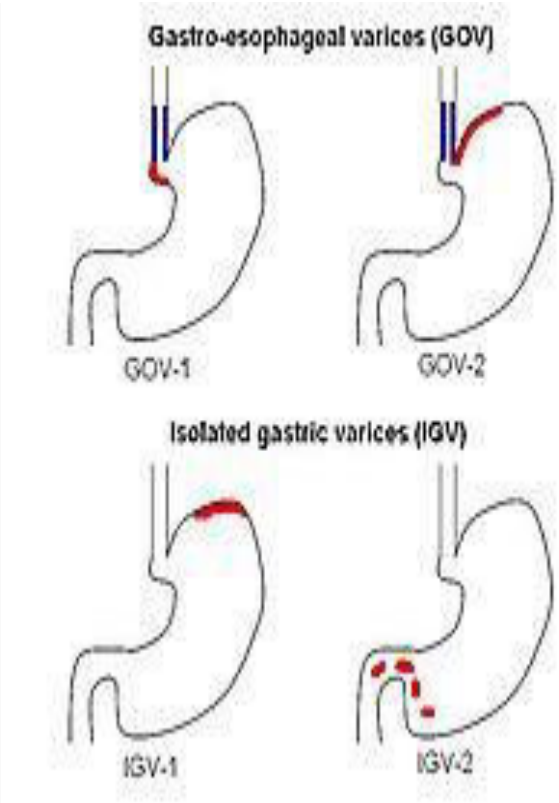
Gastric varices can present in two major ways. First, patients with cirrhosis may be enrolled in screening gastroscopy programs to detect esophageal varices. These evaluations may detect gastric varices that are asymptomatic. When gastric varices are symptomatic, however, they usually present acutely and dramatically with upper GI hemorrhage. The symptoms can include hematemesis, or vomiting blood; melena, passing black, tarry stools; or passing maroon stools or frank blood in the stools. Many patients with bleeding gastric varices present in shock due to the profound loss of blood.

Secondly, patients with acute pancreatitis may present with gastric varices as a complication of thrombosis of the splenic vein. The splenic vein sits over the pancreas anatomically and inflammation or cancers of the pancreas may result in thrombosis, or clotting of the splenic vein. As the short gastric veins of the fundus of the stomach drain into the splenic vein, thrombosis of the splenic vein will result in increased pressure and engorgement of the short veins, leading to varices in the fundus of the stomach.

Laboratory testing usually shows anemia and often thrombocytopenia (a low platelet count). If cirrhosis is present, there may be coagulopathy manifested by a prolonged INR; both of these may worsen the hemorrhage from gastric varices.

In very rare cases, gastric varices are caused by splenic vein occlusion as a result of the mass effect of slow-growing pancreatic neuroendocrine tumors.

Diagnosis and classification



The Sarin classification of gastric varices identifies two types of **gastroesophageal varices**, where esophageal varices are found concurrently, and two types of **isolated gastric varices**, found in the absence of esophageal varices.
Antral varices, of Sarin classification IGV-2, an unusual class of **gastric varices**.

Diagnosis of gastric varices is often made at the time of upper endoscopy.

The Sarin classification of gastric varices identifies four different anatomical types of gastric varices, which differ in terms of treatment modalities.

Treatment

Initial treatment of bleeding from gastric varices focuses on resuscitation, much as with esophageal varices. This includes administration of fluids, blood products, and antibiotics.

The results from the only two randomized trials comparing band ligation vs cyanocarylate suggests that endoscopic injection ofcyanoacrylate, known as gastric variceal obliteration or GVO is superior to band ligation in preventing rebleeding rates.

Cyanoacrylate, a common component in 'super glue' is often mixed 1:1 with lipiodol to prevent polymerization in the endoscopy delivery optics, and to show on radiographic imaging. GVO is usually performed is specialized therapeutic endoscopy centers. Complications include sepsis, embolization of glue, and obstruction from polymerization in the lumen of the stomach.

Other techniques for refractory bleeding include:

- Transjugular intrahepatic portosystemic shunts (TIPS)
- Balloon occluded retrograde transvenous obliteration techniques (BORTO)
- Gastric variceal ligation, although this modality is falling out of favour
- Intra-gastric balloon tamponade as a bridge to further therapy: A caveat is that a larger balloon is required to occupy the fundus of the stomach where gastric varices commonly occur
- Liver transplantation.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES

MS (General Surgery) Examination, 2007

April, 2007

PAPER IV

Time Allowed: 3 Hours **Full Marks: 100**

1. **Mention the different morbidity of obesity. What is morbidly obese? Discuss the management of morbid obesity. (20)**
2. **Discuss the recent trends in management of acute pancreatitis. 20**
3. **Write short notes on: 5 x 6**

a) **Radiofrequency thermal ablation of malignant tumours.**
b) **PAIR therapy for hydatid cyst.**
c) **Oncogene.**
d) **Abdominal compartment syndrome.**
e) **Levels of evidence.**

4. Answer in brief: 4 x 7.5

a) **Outline the management of pancreaticoduodenal injury.**
b) **Use of nuclear medicines in surgery.**
c) **Biologic therapy for inflammatory bowel disease.**
d) **Strategies of breast cancer prevention.**

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES
MS (General Surgery) Examination, 2007

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Time Allowed: 3 Hours **Full Marks: 100**

1. Mention the different morbidity of obesity. What is morbidly obese? Discuss the management of morbid obesity. (20)

Answer.

See the ans. of Q.No.1 of Paper – III of May, 2009 and Q.No.1 of Paper – IVof May, 2010

Medical therapy for severe obesity has limited short-term success and almost nonexistent long-term success. Once severely obese, the likelihood that a person will lose enough weight by dietary means alone and remain at a BMI below 35 kg/m^2 is estimated at 3% or less. A 10% weight loss attained over a period of months at a rate of 0.5 to 2 lb/wk is the initial goal of medical therapy. Very low-calorie diets fall into two categories: those that primarily restrict fat intake and those that primarily restrict carbohydrate intake. Both diets produce weight loss that is insufficient to affect any major change in health status. Two medications are used. Sibutramine blocks presynaptic receptor uptake of both norepinephrine and serotonin, thereby potentiating their anorexic effect in the central nervous system. Orlistat inhibits pancreatic lipase and thereby reduces absorption of up to 30% of ingested dietary fat. A maximum weight loss of up to 10% of body weight has been noted in unselected individuals taking either or both drugs; however, weight is regained within 12 to 18 months. For a severely obese individual, neither drug has proved to be effective therapy alone.

2. Discuss the recent trends in management of acute pancreatitis. 20

Answer.

Investigations and diagnosis

Blood Investigations - Full blood count, Renal function tests, Liver Function, serum calcium, serum amylase and lipase, Arterial blood gas.

Imaging - Chest Xray (for exclusion of perforated viscus), Abdominal Xrays (for detection of "sentinel loop" dilated duodenum sign, and gallstones which are radioopaque in 10%) and CT abdomen

Amylase and lipase

Elevated serum amylase and lipase levels, in combination with severe abdominal pain, often trigger the initial diagnosis of acute pancreatitis.

Serum lipase rises 4 to 8 hours from the onset of symptoms and normalizes within 7 to 14 days after treatment.

Serum amylase may be normal (in 10% of cases) for cases of acute or chronic pancreatitis (depleted acinar cell mass) and hypertriglyceridemia.

Reasons for false positive elevated serum amylase include salivary gland disease (elevated salivary amylase) and macroamylasemia.

If the lipase level is about 2.5 to 3 times that of amylase, it is an indication of pancreatitis due to alcohol.[6]

Regarding selection on these tests, two practice guidelines state:

> "It is usually not necessary to measure both serum amylase and lipase. Serum lipase may be preferable because it remains normal in some nonpancreatic conditions that increase serum amylase including macroamylasemia, parotitis, and some carcinomas. In general, serum lipase is thought to be more sensitive and specific than serum amylase in the diagnosis of acute pancreatitis"
>
> "Although amylase is widely available and provides acceptable accuracy of diagnosis, where lipase is available it is preferred for the diagnosis of acute pancreatitis (recommendation grade A)

Computed tomography

Regarding the need for computed tomography, practice guidelines state:

"Many patients with acute pancreatitis do not require a CT scan at admission or at any time during the hospitalization. For example, a CT scan is usually not essential in patients with recurrent mild pancreatitis caused by alcohol. A reasonable indication for a CT scan at admission (but not necessarily a CT with IV contrast) is to distinguish acute pancreatitis from another serious intra-abdominal condition, such as a perforated ulcer."

"Patients with persisting organ failure, signs of sepsis, or deterioration in clinical status 6–10 days after admission will require CT (recommendation grade B)."

CT abdomen should not be performed before the 1st 48 hours of onset of symptoms as early CT (<48 h) may result in equivocal or normal findings.

CT Findings can be classified into the following categories for easy recall :

Intrapancreatic - diffuse or segmental enlargement, edema, gas bubbles, pancreatic pseudocysts and phlegmons/abscesses (which present 4 to 6 wks after initial onset)

Peripancreatic / extrapancreatic - irregular pancreatic outline, obliterated peripancreatic fat, retroperitoneal edema, fluid in the lessar sac, fluid in the left anterior pararenal space

Locoregional - Gerota's fascia sign (thickening of inflamed Gerota's fascia, which becomes visible), pancreatic ascites, pleural effusion (seen on basal cuts of the pleural cavity), adynamic ileus, etc.

Magnetic resonance imaging

While computed tomography is considered the gold standard in diagnostic imaging for acute pancreatitis, magnetic resonance imaging (MRI) has become increasingly valuable as a tool for the visualization of the pancreas, particularly of pancreatic fluid collections and necrotized debris.Additional utility of MRI includes its indication for imaging of patients with an allergy to CT's contrast material, and an overall greater sensitivity to hemorrhage, vascular complications, pseudoaneurysms, and venous thrombosis.

Another advantage of MRI is its utilization of magnetic resonance cholangiopancreatography (MRCP) sequences. MRCP provides useful information regarding the etiology of acute pancreatitis, i.e., the presence of tiny biliary stones (choledocholithiasis or cholelithiasis) and duct anomalies. Clinical trials indicate that MRCP can be as effective a diagnostic tool for acute pancreatitis with biliary etiology as endoscopic retrograde cholangiopancreatography, but with the benefits of being less invasive and causing fewer complications.

Classification by severity

Progression of pathophysiology

Acute pancreatitis can be further divided into mild and severe pancreatitis. Mostly the Atlanta classification (1992) is used. In severe pancreatitis serious amount of necrosis determine the further clinical outcome. About 20% of the acute pancreatitis are severe with a mortality of about 20%. This is an important classification as severe pancreatitis will need intensive care therapy whereas mild pancreatitis can be treated on the common ward.

Necrosis will be followed by a systemic inflammatory response syndrome (SIRS) and will determine the immediate clinical course. The further clinical course is then determined by bacterial infection. SIRS is the cause of bacterial (Gram negative) translocation from the patients colon.

There are several ways to help distinguish between these two forms. One is the above mentioned Ranson Score.

Prognostic indices

In predicting the prognosis, there are several scoring indices that have been used as predictors of survival. Two such scoring systems are the Ranson criteria and APACHE II (Acute Physiology, Age and Chronic Health Evaluation) indices. Most, but not all studies report that the Apache score may be more accurate. In the negative study of the Apache II, the Apache II 24 hr score was used rather than the 48 hour score. In addition, all patients in the study received an ultrasound twice which may have influenced allocation of co-interventions. Regardless, only the Apache II can be fully calculated upon admission. As the Apache II is more cumbersome to calculate, presumably patients whose only laboratory abnormality is an elevated lipase or amylase do not need prognostication with the Apache II; however, this approach is not studied. The Apache II score can be calculated .

Pracactice guidelines state:

"The two tests that are most helpful at admission in distinguishing mild from severe acute pancreatitis are APACHE-II score and serum hematocrit. It is recommended that APACHE-II scores be generated during the first 3 days of hospitalization and thereafter as needed to help in this distinction. It is also recommended that serum hematocrit be obtained at admission, 12 h after admission, and 24 h after admission to help gauge adequacy of fluid resuscitation."

"Immediate assessment should include clinical evaluation, particularly of any cardiovascular, respiratory, and renal compromise, body mass index, chest x ray, and APACHE II score".

Ranson criteria is a clinical prediction rule for predicting the severity of acute pancreatitis. It was introduced in 1974.

At admission

- Age in years > 55 years
- White blood cell count > 16000 cells/mm3
- Blood glucose > 10 mmol/l (> 200 mg/dl)
- Serum AST > 250 iu/l
- Serum LDH > 700 iu/l

At 48 hours

- Calcium (serum calcium < 2.0 mmol/L (< 8.0 mg/dL)
- Hematocrit fall > 10%
- Oxygen (hypoxemia PO2 < 60 mmHg)
- BUN increased by 1.8 or more mmol/L (5 or more mg/dL) after IV fluid hydration
- Base deficit (negative base excess) > 4 mEq/L
- Sequestration of fluids > 6 L

The criteria for point assignment is that a certain breakpoint be met at anytime during that 48 hour period, so that in some situations it can be calculated shortly after admission. It is applicable to both gallstone and alcoholic pancreatitis.

Alternatively, pancreatitis can be diagnosed by meeting any of the following:

APACHE II score

- Apache score of ≥ 8 Organ failure Substantial pancreatic necrosis (at least 30% glandular necrosis according to contrast-enhanced CT)
- Interpretation If the score ≥ 3, severe pancreatitis likely. If the score < 3, severe pancreatitis is unlikely Or
- Score 0 to 2:2% mortality Score 3 to 4 : 15% mortality Score 5 to 6 : 40% mortality Score 7 to 8 : 100% mortality
- "Acute Physiology And Chronic Health Evaluation" (APACHE II) score > 8 points predicts 11% to 18% mortality .

Balthazar scoring:

Developed in the early 1990s by Emil J. Balthazar et al., the Computed Tomography Severity Index (CTSI) is a grading system used to determine the severity of acute pancreatitis. The numerical CTSI has a maximum of ten points, and is the sum of the Balthazar grade points and pancreatic necrosis grade points:

Balthazar Grade

Balthazar Grade	Appearance on CT	CT Grade Points
Grade A	Normal CT	0 points
Grade B	Focal or diffuse enlargement of the pancreas	1 point
Grade C	Pancreatic gland abnormalities and peripancreatic inflammation	2 points
Grade D	Fluid collection in a single location	3 points
Grade E	Two or more fluid collections and / or gas bubbles in or adjacent to pancreas	0 points

Necrosis Score

Necrosis Percentage	Points
No necrosis	0 points
0 to 30% necrosis	2 points
30 to 50% necrosis	4 points
Over 50% necrosis	6 points

CTSI's staging of acute pancreatitis severity has been shown by a number of studies to provide more accurate assessment than APACHE II, Ranson, and C-reactive protein (CRP) level. However, a few studies indicate that CTSI is not significantly associated with the prognosis of hospitalization in patients with pancreatic necrosis, nor is it an accurate predictor of AP severity.
Glasgow Imrie criteria
3 or more positive criteria within 48h of admission = severe attack

- Age > 55y
- WCC > 15 000 Ã— 10^9/L
- Glucose > 7mmol/L
- Blood urea > 7mmol/L
- Albumin < 35g/L
- Corrected calcium < 2mmol/L
- PaO_2 < 10kPa

Treatment:

- Pain control
- Bowel rest: In the management of acute pancreatitis, the treatment is to stop feeding the patient, giving him or her nothing by mouth, giving intravenous fluids to prevent dehydration, and sufficient pain control. As the pancreas is stimulated to secrete enzymes by the presence of food in the stomach, having no food pass through the system allows the pancreas to rest.
- Nutritional support: Recently, there has been a shift in the management paradigm from TPN (total parenteral nutrition) to early, post-pyloric enteral feeding (in which a feeding tube is endoscopically or radiographically introduced to the third portion of the duodenum). The advantage of enteral feeding is that it is more physiological, prevents gut mucosal atrophy, and is free from the side effects of TPN (such as fungemia). The additional advantages of post-pyloric feeding are the inverse relationship of pancreatic exocrine secretions and distance of nutrient delivery from the pylorus, as well as reduced risk of aspiration.

Disadvantages of a naso-enteric feeding tube include increased risk of sinusitis (especially if the tube remains in place greater than two weeks) and a still-present risk of accidentally intubating the bronchus even in intubated patients.

- Antibiotics: Carbapenems

An early randomized controlled trial of imipenem 0.5 gram intravenously every eight hours for two weeks showed a reduction in from pancreatic sepsis from 30% to 12%.

Another randomized controlled trial with patients who had at least 50% pancreatic necrosis found a benefit from imipenem compared to pefloxacin with a reduction in infected necrosis .

The role of antibiotics is controversial. One recent expert opinion suggested the use of imipenem if CT scan showed more than 30% necrosis of the pancreas.

- ERCP: Early ERCP (endoscopic retrograde cholangiopancreatography), performed within 24 to 72 hours of presentation, is known to reduce morbidity and mortality.

- ❖ The indications for early ERCP are as follows :

- ➢ Clinical deterioration or lack of improvement after 24 hours
- ➢ Detection of common bile duct stones or dilated intrahepatic or extrahepatic ducts on CT abdomen

The disadvantages of ERCP are as follows :

- o ERCP precipitates pancreatitis, and can introduce infection to sterile pancreatitis
- o The inherent risks of ERCP i.e. bleeding
- o It is worth noting that ERCP itself can be a cause of pancreatitis.

Surgery: Surgery is indicated for (i) infected pancreatic necrosis and (ii) diagnostic uncertainty and (iii) complications.

The most common cause of death in acute pancreatitis is secondary infection.

Infection is diagnosed based on 2 criteria:

- ➢ Gas bubbles on CT scan (present in 20 to 50% of infected necrosis)
- ➢ Positive bacterial culture on FNA (fine needle aspiration, usually CT or US guided) of the pancreas.

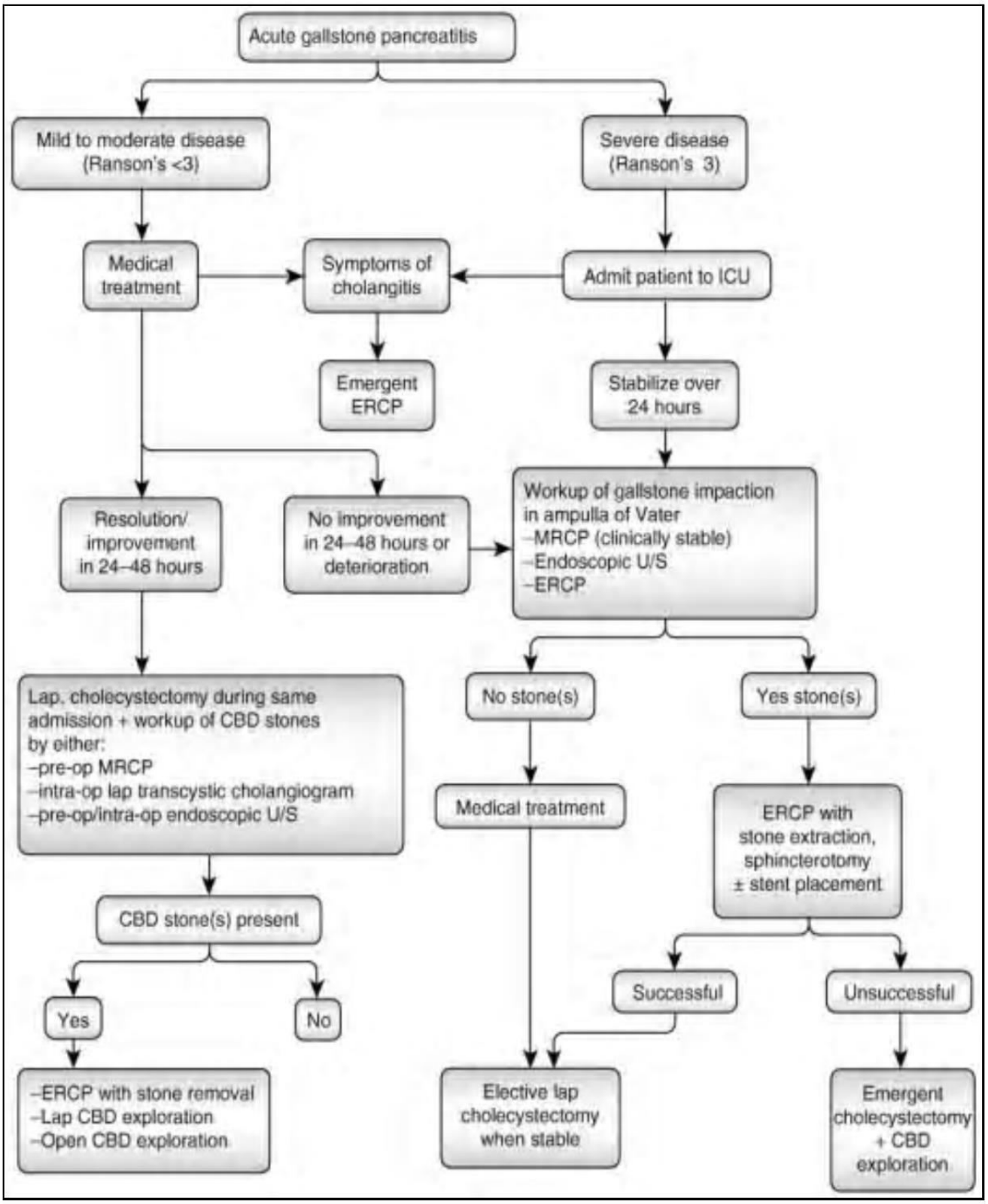
Surgical options for infected necrosis include:

- o Minimally invasive management - necrosectomy through small incision in skin (left flank) or stomach
- o Conventional management - necrosectomy with simple drainage
- o Closed management - necrosectomy with closed continuous postoperative lavage
- o Open management - necrosectomy with planned staged reoperations at definite intervals (up to 20+ reoperations in some cases)

Other measures: Pancreatic enzyme inhibitors are not proven to work.

The use of octreotide has not been shown to improve outcome.

a management algorithm for both mild and severe pancreatitis.



The Complications of Acute Pancreatitis

Local	Fluid collections
	Pancreatic ascites/pleural effusion
	Pancreatic pseudocyst
	Pancreatic necrosis
	Infected pancreatic abscess
	Hemorrhage/pseudoaneurysm
Regional	Venous thrombosis
	Paralytic ileus
	Intestinal obstruction
	Intestinal ischemia/necrosis
	Cholestasis
Systemic	Systemic inflammatory response syndrome
	Multiple-organ-dysfunction syndrome
	ARDS/pulmonary failure
	Renal failure
	Cardiovascular complications
	Hypocalcemia
	Hyperglycemia
	Disseminated intravascular coagulopathy
	Protein calorie malnutrition
	Encephalopathy

Complications are treated accordingly

3. Write short notes on: 5 x 6

a) Radiofrequency thermal ablation of malignant tumours.
b) PAIR therapy for hydatid cyst.
c) Oncogene.
d) Abdominal compartment syndrome.
e) Levels of evidence.

Answer.

a) Introduction: In 1868, the French scientist Jaques Arsen D'Arsonval described the basic principle of RFA demonstrating that an alternating electric current greater than 10 kHz could pass through living tissue without neuromuscular effect.

Since then, more studies have proved the utility and essential efficacy of RFA in treating discrete tumours in humans.

Many different ablation systems have been explored, including monopolar, bipolar and multipolar electrodes, cooled tip electrodes and expandable electrodes with multiple tines, with aim of improving local thermal efficacy and, thereby, oncological results.

In the last decade, this technique has been increasingly used as an alternative, or as an adjunct or bridge, to surgery and/or transplant in the treatment of liver tumours. More recently, RFA has confirmed its usefulness in the treatment of renal, adrenal, lung and other focal cancers.

Principle: Radiofrequency ablation works by converting electromagnetic energy into heat but its effectiveness is influenced by thermal conductivity and perfusion of the target tissue.

RFA can be performed via a percutaneous approach, laparoscopically or during open laparotomy. For liver and renal tumours, RFA is usually performed under ultrasound guidance. For lung tumours, the procedure is necessarily performed under CT guidance.

Use: 1.In liver: The ideal lesion for RFA using current technology is around 3cm in diameter, not superficial and distant from large bowel and large vascular structures.Caution should be taken when treating lesions near the proximal bile ducts, large vessels, diaphragm, and gut to avoid possible complications such as bleeding, infection, bilomas, biliary strictures, haemothorax, and colonic perforation due to heat transmission.

Radical surgical resection is the best treatment for discrete, non-transplantable HCC in favourable patients. RFA plays an important role as an alternative treatment for primary lesion when surgery is not possible and has been shown to be more effective than other local therapies such as percutaneous ethanol injection and cryotherapy.

2. In colorectal liver metastases: RFA plays an important role in the treatment of non-resctable liver colorectal metastases and as a complement to surgical resection.

The fibrotic nature of the colorectal liver metastases, surrounded by normal parenchyma may compromise the efficacy of RFA. Lesions>3cm in diameter are considered at significantly higher risk of recurrence.

3. In metastatic neuroendocrine tumours: RFA alone or in combination with surgery appears to be a useful 'cytoreductive' tool which can improve neuroendocrine symptomatology from metastatic neuroendocrine tumours.

4. In renal tumours: Minimally invasive therapy can be employed for the treatment of small volume (< 4 cm), T1a disease renal malignancy. RFA under imaging guidance appears well suited to this task.

5. In lung malignancies: RFA has shown to be feasible, effective and safe in the treatment of poorly resectable primary and secondary lung malignancies.

b) PAIR – Percutaneous therapy by puncture, aspiration, injection and reaspiration combined with albendazole is a good method for the treatment of hydatid cyst.

Scolicidal agents used in PAIR:

- 95% sterile ethanol.
- Absolute alcohol.
- Mebendazole 2.4ug/ml.

Indications for PAIR:	Contraindications to PAIR:
• Refusal of surgery. • Inoperable cases. • Type I, II and III cysts. • Cystic lesion>= 5cm in diameter. • Multiple cysts >= 5cm in diameter in different liver segments. • Relapse post surgery. • Lack of response to chemotherapy.	• Inaccesible or hazardous location of cyst. • Multiple septal divisions. • Dead or inactive cysts. • Communicating cysts. • Lung or bone cysts.

Complications of PAIR:

- Urticaria.
- Anaphylaxis.
- Subcapsular haematoma.
- Fever.
- Biliary fistula.
- Secondary infection of cyst cavity.
- Hypotension/hypotensive shock.

c) An oncogene is a gene that has the potential to cause cancer. In tumour cells, they are often mutated or expressed at high levels. Many abnormal cells normally undergo a programmed form of death (apoptosis). Activated oncogenes can cause those cells to survive and proliferate instead. Most oncogenes require an additional step, such as mutations in another gene, or

environmental factors, such as viral infection, to cause cancer. Since the 1970s, dozens of oncogenes have been identified in human cancer. Many cancer drugs target those DNA sequences and their products.

A proto-oncogene is a normal gene that can become an oncogene due to mutations or increased expression**.**

Activation

The proto-oncogene can become an oncogene by a relatively small modification of its original function. There are three basic activation types:

- A mutation within a proto-oncogene can cause a change in the protein structure, causing
  - an increase in protein (enzyme) activity
  - a loss of regulation
- An increase in protein concentration, caused by
  - an increase of protein expression (through misregulation)
  - an increase of protein (mRNA) stability, prolonging its existence and thus its activity in the cell
  - a gene duplication (one type of chromosome abnormality), resulting in an increased amount of protein in the cell
- A chromosomal translocation (another type of chromosome abnormality), causing
  - an increased gene expression in the wrong cell type or at wrong times
  - the expression of a constitutively active *hybrid protein*. This type of aberration in a dividing stem cell in the bone marrow leads to adult leukemia

The expression of oncogenes can be regulated by microRNAs (miRNAs), small RNAs 21-25 nucleotides in length that control gene expression by downregulating them. Mutations in such microRNAs (known as oncomirs) can lead to activation of oncogenes. Antisense messenger RNAs could theoretically be used to block the effects of oncogenes.

Classification

There are several systems for classifying oncogenes, but there is not yet a widely accepted standard. They are sometimes grouped both spatially (moving from outside the cell inwards) and chronologically (parallelling the "normal" process of signal transduction). There are several categories that are commonly used:

Category	**Examples**	**Description**

Growth factors, or mitogens	c-Sis	Usually secreted by specialized cells to induce cell proliferation in themselves, nearby cells, or distant cells. An oncogene may cause a cell to secrete growth factors even though it does not normally do so. It will thereby induce its own uncontrolled proliferation (*autocrine loop*), and proliferation of neighboring cells. It may also cause production of growth hormones in other parts of the body.
Receptor tyrosine kinases	epidermal growth factor receptor (EGFR),platelet-derived growth factor receptor(PDGFR), and vascular endothelial growth factor receptor (VEGFR),HER2/neu	Kinases add phosphate groups to other proteins to turn them on or off. Receptor kinases add phosphate groups to receptor proteins at the surface of the cell (which receive protein signals from outside the cell and transmit them to the inside of the cell). Tyrosine kinases add phosphate groups to the amino acid tyrosine in the target protein. They can cause cancer by turning the receptor permanently on (constitutively), even without signals from outside the cell.
Cytoplasmictyrosine kinases	Src-family, Syk-ZAP-70 family, and BTKfamily of tyrosine kinases, the Abl gene in CML - Philadelphia chromosome	-

CytoplasmicSerine/threonine kinases and their regulatory subunits	Raf kinase, and cyclin-dependent kinases (through overexpression).	-
RegulatoryGTPases	Ras protein	Ras is a small GTPase which hydrolyses GTP into GDP and phosphate. Ras is activated by growth factor signaling (ie. EGF, TGFalpha) and acting like a binary switch (on/off) in growth signaling pathways. Downstream effectors of Ras include Raf, MEK, MEKK, MAPK, ERK, most of which in turn regulate genes that mediate cell proliferation.
Transcription factors	myc gene	-They regulate transcription of genes that induce cell proliferation.

Conversion of proto-oncogenes:

There are two mechanisms by which proto-oncogenes can be converted to cellular oncogenes:

Quantitative: Tumor formation is induced by an increase in the absolute number of proto-oncogene products or by its production in inappropriate cell types.

Qualitative: Conversion from proto-oncogene to transforming gene (c-onc) with changes in the nucleotide sequence which are responsible for the acquisition of the new properties.

d) Introduction: Abdominal compartment syndrome (ACS) is a constellation of physiologic derangements associated with intraabdominal hypertension (IAH) or intraabdominal pressure (IAP) elevation. A variety of clinical situations may cause IAH.

Pathophysiology: Increased IAP above 10 mm Hg leads to diminished venous return, stroke volume, and cardiac output, despite a normal arterial pressure. The central venous pressure and pulmonary capillary wedge pressure may be spuriously elevated, *despite a low intravascular volume*. Respiratory failure is typical with excessive IAH and is characterized by high inspiratory

pressures, hypoxia, hypercarbia, and a reduction in static and dynamic pulmonary compliance. Pulmonary vascular resistance increases. Total lung capacity and functional residual capacity (FRC) decrease and V-Q mismatch begins. A progressive difficulty in maintaining oxygenation and ventilation is the net result. Oliguria progressing to anuria from IAH may be related to a decrease in renal blood flow, glomerular filtration rate, and glucose reabsorption and is associated with an increase in plasma renin and aldosterone levels. Direct compression of the renal vein, cortical arterioles, or parenchyma may result in an oliguria unresponsive to volume loading. The renal effects of IAH can be seen in postoperative patients and in cirrhotic patients with ascites. The adverse effects of IAH on visceral blood flow are profound and include a decline in the mesenteric and mucosal blood flow with an IAP higher than 20 mm Hg. Corresponding to these changes, the intestinal mucosa, as studied by tonometer, becomes severely acidotic. In anesthetized pigs, an IAP of 10 mm Hg causes a significant decrease in hepatic arterial blood flow and hepatic microvascular blood flow. The decreases are exaggerated with higher levels of IAP. The exact mechanism of the diminished splanchnic perfusion is not known but may involve a direct effect of increased IAP on mesenteric arterial resistance, humoral factors, or a combination of the two. Hypovolemic shock and resuscitation prior to IAH may amplify the ill effects of IAH or lower the critical level of IAH, the typical clinical scenario in trauma. In animals subjected to a 20% hemorrhage followed by resuscitation prior to an increase in the IAP to 10 and 20 mm Hg, the Pao_2/Fio_2 ratios were significantly lower than in a control group of animals without prior shock and resuscitation. In the same model, a similar synergistic adverse effect on superior mesenteric artery flow is noted with ischemia reperfusion followed by IAH.

Identification of Patients at Risk and Prevention of Intraabdominal Hypertension and Abdominal Compartment Syndrome:

IAH may be predicted by the following risk factors: (1) preoperative hypovolemic shock and massive fluid resuscitation, for example, burns, peritonitis, pancreatitis, ruptured abdominal aortic aneurysm (AAA), gastrointestinal (GI) tract hemorrhage, and multiple or multisystem injuries; (2) increased intraabdominal fluid accumulation, for example, ascites, excessive fluid resuscitation, coagulopathy and abnormal bleeding, peritonitis, ruptured AAA, pelvic and retroperitoneal hematomas, and intestinal obstruction; (3) mechanical increase in abdominal pressure, for example, "damage-control" surgery with intraabdominal packing, sudden intraabdominal reduction of longstanding hernial contents (loss of right of domain), tension pneumothorax, massive hemothorax, and "chronic" ACS from morbid obesity or intestinal obstruction.

Prophylaxis against Abdominal Compartment Syndrome

It is important to anticipate the complication of IAH in these clinical situations and attempt its prevention by "not closing the abdomen" ("open abdomen") at the initial operation. The "open abdomen" approach offers several advantages. First, it provides a rapid method of abbreviating the laparotomy and transporting the critical patient to the intensive care unit for resuscitation. Second, in a significant number of patients, it may actually prevent IAH. Two recent reports documented a significant reduction in IAH by an absorbable mesh closure of the abdomen at the initial celiotomy.

The other important aspect of prevention of IAH is to monitor IAP in high-risk patients and to intervene if IAH occurs. The most widely used method to monitor IAP is to use bladder pressure as a surrogate. About 50 ml of saline is introduced into the urinary bladder through the urethral catheter. The tubing of the collecting bag is clamped, and a needle is inserted into the specimen-collecting port of the tubing proximal to the clamp and is attached to a manometer. Bladder pressure, measured in centimeters of water, is the height at which the level of the saline column stabilizes with the symphysis pubis as the zero point. The catheter may be connected to bedside monitors through a transducer and the bladder pressure may be monitored continuously in millimeters of mercury, where 1 mm Hg equals 1.36 cm H_2O.

When to Consider Treatment: What Is the Critical Level of Intraabdominal Pressure?

Burch et al. described a grading system of elevated IAP: grade I (10 to 15 cm H_2O), grade II (15 to 25 cm H_2O), grade III (25 to 35 cm H_2O), and grade IV (>35 cm H_2O) and suggested that most of the patients with grade III and all of the patients with grade IV elevations in IAP should undergo abdominal decompression. Even at lower pressures, the onset of the syndrome of ACS should prompt abdominal decompression. Our practice is to consider a persistent elevation of IAP beyond 20 to 25 cm H_2O (15 to 19 mm Hg) as IAH and institute therapy, even in the absence of established signs of ACS. A recent concept is to calculate abdominal perfusion pressure (APP) as the difference between mean arterial pressure and intraabdominal pressure, with an APP of 50 mm Hg as the optimal level. This concept needs to be validated in prospective studies before it can be applied widely in clinical practice. Obviously, any of the features of ACS should trigger abdominal decompression.

Treatment Approaches for Intraabdominal Hypertension:

The first step in the evaluation of an increased IAP, especially in the presence of agitation and restlessness, is to sedate and, if necessary, chemically paralyze the patient. If the bladder pressures are still high and/or systemic manifestations of IAH (as described above) are evident, the appropriate treatment, in most instances, is abdominal decompression by laparotomy.

If the abdomen is already "open," this may involve the opening of the temporary closure (mesh, plastic, or towel clips, etc.); evacuation of the abdomen of fluid and blood; removal of abdominal packs (with repacking, if necessary); and enlargement of the abdominal space to accommodate the edematous and swollen contents. Some centers have started evacuation of ascitic fluid as a temporary measure of abdominal decompression after secondary ACS, especially in burn patients. Evacuation of hemoperitoneum by ultrasound (US)-guided paracentesis may be employed with success in patients with nonoperative management of hepatic and splenic injuries. If the abdomen was previously closed by fascial sutures, there was no previous laparotomy, or bedside decompression is not efficacious or possible, a formal operating room celiotomy should be performed.

At celiotomy, the abdomen is decompressed, abdominal fluid and/or blood evacuated, and hemostasis obtained or ensured. The results of abdominal decompression are usually rather dramatic, with a decreased inspiratory pressure, improved gas exchange, lessened systemic vascular resistance, and increased cardiac index and oxygen delivery. The gut appearance will

improve, and, if measured, oxygenation of the gut can be shown to have improved. A brisk increase in urinary output occurs.

Once abdominal decompression is achieved, it is the usual practice to leave the abdominal fascia and skin "open" with some type of foreign material at the skin level to prevent evisceration. These materials include various absorbable or nonabsorbable types of mesh, an artificial burr device, sterilized saline bags with or without a zipper (the Bogota bag), dressings with moist gauze or Vi-drapes, "vacuum-pack" etc. All have in common the goals of preventing evisceration, allowing enlargement of the abdominal cavity, keeping the IAP low, and preventing reoccurrence of IAH and ACS.

e) Levels of evidence:

Level	Description
1a	Systematic Review (or Metanalysis) (with homogeneity) of Randomized Controlled Trials (RCT)
1b	Individual RCT (with narrow Confidence Interval)
1c	All or none
2a	Systematic Review (with homogeneity) of cohort studies
2b	Individual cohort study (including low quality RCT; e.g., <80% follow-up)
2c	"Outcomes" Research; Ecological studies
3a	SR (with homogeneity) of case-control studies
3b	Individual Case-Control Study
4	Case-series (and poor quality cohort and case-control studies)
5	Expert opinion without explicit critical appraisal, or based on physiology, bench research or "first principles"

What's homogeneity/heterogeneity? - In a systematic review when results from different studies when there are worrsiome variations in the direction and degree of results between individual studies it is called heterogeneity. Studies without significant variations are 'homogeneous'.
· When there is either a single result with a wide confidence interval or a systematic review with troublesome heterogeneity add a minus "-" sign. The grade of recommendation then becomes D.
· All or none studies: when all patients died before the Rx became available, but some now survive on it; or when some patients died before the Rx became available, but none now die on it.

Grades of Recommendations:

Grade	Description
A	consistent level 1 studies
B	consistent level 2 or 3 studies or extrapolations from level 1 studies
C	level 4 studies or extrapolations from level 2 or 3 studies
D	level 5 evidence or troublingly inconsistent or inconclusive studies of any level

It is important to remember, however, that evidence-based-practice is not just blindly following published evidence - it has to be combined with 2 other important elements - clinical expertise and patient wishes and expectations.

4. Answer in brief: 4 x 7.5

a) Outline the management of pancreaticoduodenal injury.
b) Use of nuclear medicines in surgery.
c) Biologic therapy for inflammatory bowel disease.
d) Strategies of breast cancer prevention.

Answers.

a) Introduction: Detection and surgical management of duodenal and pancreatic injuries pose a difficult challenge to the trauma surgeon. Multiple factors contribute to the difficulty in diagnosis and surgical management of these injuries. Delay in diagnosis is common in part because of nonspecific clinical findings and high incidence of associated injuries. Moreover, the low incidence of injuries to the duodenum and pancreas prevents any one center from acquiring a large clinical experience often resulting in a low index of suspicion.

Diagnosis :

- The approach to pancreatic and duodenal injuries begins with the evaluation of the injured patient.
- Management of hemodynamic instability and penetrating abdominal injuries is straightforward and requires little preoperative evaluation. Diagnosis of blunt injuries in hemodynamically stable patients can be more challenging.
- Common mechanisms include deceleration injuries with blunt force to the epigastrium and crush injuries that appose the anterior abdominal wall with the spinal column.
- Initial physical examination findings may be subtle and include persistent abdominal pain and tenderness with or without peritonitis, or may be overshadowed by findings associated with major chest, abdominal, and pelvic injuries.
- Duodenal injuries missed on initial evaluation often develop into peritonitis and shock within a few days of injury. Plasma amylase or lipase activity should be measured in all patients with suspected pancreatic and duodenal injuries. Although a single elevated value is not diagnostic for pancreatic injury, serial tests showing elevated amylase or lipase activity with peritonitis demands further evaluation or exploration.

Initial plain radiographs are of little use in the diagnosis of pancreatic or duodenal injuries. Signs such as retroperitoneal air, obliteration of the right psoas shadow, and mild spine scoliosis are subtle or absent.

Similarly, although a Focused Abdominal Sonography for Trauma (FAST) exam is helpful in identifying hemoperitoneum in hemodynamically unstable patients, it is not helpful in locating the source of bleeding or in diagnosing retroperitoneal injuries. An early suspicion of retroperitoneal duodenal injury is best evaluated by computed tomography (CT) scan with intravenous (IV) and oral contrast. Contrast must be visible at some time within all portions of the duodenum and pass with no obstruction. Extraluminal gas or contrast extravasation is diagnostic for duodenal perforation. False-negative studies occur and the CT scan must be

evaluated with the possibility of duodenal injury in mind. Equivocal studies should be further evaluated by duodenography first using water-soluble contrast followed by barium. Although the specificity of duodenography is 98%, sensitivity is poor.

Preoperative identification of blunt pancreatic injuries in the absence of clear indications for laparotomy is difficult. Delayed presentation of symptoms is in part because of the requirement of pancreatic secretions to be activated by infection or small bowel contents. CT findings may be normal or show pancreatic transection, hemorrhage, phlegmon, and peripancreatic fluid. Initial CT scans may not define pancreatic injuries and should be repeated for patients with persistent abdominal pain, fever, or chemical evidence of pancreatitis.

Endoscopic retrograde cholangiopancreatography (ERCP) is used in selected patients with suspected pancreatic injuries for which other diagnostic maneuvers are equivocal. The value of ERCP is to exclude main pancreatic duct injury in the patient with CT and chemical evidence of pancreatitis but with minimal clinical signs and symptoms. In such cases, observation is often sufficient therapy. Pancreatic duct injuries identified by ERCP are generally treated surgically. Treatment of pancreatic duct injury by ERCP-placed stents in select patients with a prohibitive operative risk has been reported, but the technical success rate is low and long-term results not available.

Pancreatic and duodenal injuries are often discovered at the time of laparotomy. In such cases, the principles of trauma surgery priority pertain, with control of life-threatening hemorrhage and control of enteric spillage. Damage control maneuvers should be considered early and abbreviated laparotomy performed to avoid acidosis, hypothermia, and coagulopathy. As in any abdominal exploration for trauma, a systematic examination of the abdomen is performed. Complete evaluation of the duodenum and pancreas is indicated for the presence of upper or central retroperitoneal hematoma, air, or bile staining. Intraoperative indications of pancreatic injury include peripancreatic fluid, subcapsular hematoma, hemorrhage, parenchymal disruption, pancreatic fluid leak, and fat necrosis.

Intraoperative evaluation of the duodenum requires examination of anterior and retroperitoneal surfaces from the pylorus to the mesenteric vessels and inspection of the fourth portion of the duodenum to the left of the mesenteric vessels. Complete evaluation of the pancreas requires visualization of the head, body, and tail. Because the duodenum and pancreas are intimately associated, examination of both structures proceeds simultaneously. The hepatic flexure of the colon is taken down and duodenum then mobilized by performance of a Kocher maneuver to the midline. This exposes the anterior and posterior surfaces of the second and third portions of the duodenum as well as the head and uncinate process of the pancreas. Visualization of the fourth portion of the duodenum is achieved by dividing the ligament of Treitz, taking care to avoid the superior mesenteric vessels, and reflecting the duodenum from left to right. This, along with the Kocher maneuver, allows complete evaluation of the duodenum. Examination of the body and tail of the pancreas is accomplished by division of the gastrocolic ligament and reflection of the stomach cephalad. This allows full inspection of the anterior surface of the pancreas from head to tail and from superior to inferior border. The splenic artery and vein can also be exposed along the superior border of the pancreas. In cases of injury to the splenic, superior mesenteric, or portal vein behind the neck of the pancreas, the pancreas should be divided at the neck without

hesitation. The presence of penetrating injury to the anterior surface of the pancreas requires assessment of the main pancreatic duct. This requires exposure of the posterior surface of the body and tail and is achieved by division of the retroperitoneal attachments along the inferior border of the pancreas and retraction of the pancreas cephalad. Additional mobilization of the spleen and reflection at the spleen and tail of the pancreas from the left to right exposes the posterior surface of the tail at the spleen. Direct exploration identifies injury in most cases of penetrating trauma. However, the integrity of the pancreatic duct occasionally remains unclear despite extensive pancreatic mobilization. It is crucial to either prove or disprove the presence of a main pancreatic duct injury in such cases of penetrating trauma. Our approach is to first squeeze the gallbladder and look for bile extravasation. If none is seen, we then perform a duodenotomy, identify the major papilla, and cannulate the main pancreatic duct with a blunt-tipped probe. The probe can often be seen in the area of the pancreatic injury. If not, we then cannulate the major papilla and perform intraoperative pancreatography. Intraoperative pancreatography via transecting the tail of the pancreas and cannulation of the distal pancreatic duct has been proposed. However, identification of the distal pancreatic duct and unnecessary pancreatic resection with associated complications are obvious concerns with this procedure. Although technically possible, intraoperative ERCP is often prohibited by time constraints and inability to cannulate the major papilla endoscopically.

Modified Lucas classification of pancreatic injury:

Class I: Simple superficial contusion or peripheral laceration with minimal parenchymal damage. Any portion of the pancreas can be affected, but the main pancreatic duct is intact.

Class II: Deep laceration, perforation or transaction of the neck, body or tail of the pancreas, with or without pancreatic duct injury.

Class III: Severe crush, perforation or transaction of the head of the pancreas, with or without pancreatic duct injury.

Class IV: Combined pancreaticoduodenal injuries, subdivided into;

- (i) Minor pancreatic injury.
- (ii) Severe pancreatic injury and duct disruption.

Type of injury	Treatment
Class I	Control of bleeding and simple external drainage without repair of capsular lacerations + put a closed suction drain.
Class II	Distal pancreatectomy + spleenectomy, some surgeons prefer additional Roux – en – Y pancreaticojejunostomy to prevent pancreatic fistula , but a visible pancreatic duct is best managed by ligation with a transfixing suture. Oversewing or stapling at the end of the pancreas and using simple buttress or seal the cut margin are sufficient.
Class III	Simple external drainage, even if there is a suspected isolated pancreatic duct injury , provided there is no devitalisation and ampulla is intact.
Class IV	Define the integrity of the CBD, pancreatic duct and the ampulla and the viability of the

	duodenum. If the CBD and ampulla are shown to be intact , the duodenal laceration is repaired and the pancreatic injury is treated according to the site of the injury. Localized ischemia at the site of the duodenal injury should be debrided before primary duodenal closure. With a severe injury to the duodenum – diversion of gastric and biliary contents from the duodenal repair is advised by some- like duodenal "diverticulisation" procedure with primary closure of the duodenal wound, a vagotomy, an antectomy with an end to side gastrojejunostomy, a T- tube CBD drainage and a tube duodenostomy. Another procedure is pyloric exclusion . Pancreatico duodenectomy is needed when: • Extensive devitalisation of the head of the pancreas and duodenum so that reconstruction is not possible. • Ductal disruption of the pancreatic head in association with injuries to the duodenum and distal CBD. • Injury to the ampulla of Vater, with disruption of the MPD from the duodenum. • Uncontrollable bleeding from vessels in the head of the pancreas. • Inaccessible exsanguinating retropancreatic portal or SMV injury.

Complications :

Exsanguination is the most common cause of death associated with pancreatic and duodenal injuries. Early consideration of damage control maneuvers is central to successful management of coagulopathy, acidosis, and hypothermia that accompanies massive resuscitation. Other causes of death include multiple organ failure and sepsis. Mortality from combined pancreatic and duodenal injury approaches 30%, twice that observed in either injury alone. Both pancreatic and duodenal fistula are often encountered following injury. Management includes correction of fluid and electrolyte abnormalities and protection of skin with topical agents. Lateral duodenal fistulas are difficult to manage and rarely close spontaneously. Control of infection requires drainage of associated intraabdominal abscesses and antibiotic treatment. Persistent fistulas can be successfully treated with pyloric exclusion. Pancreatic fistulas occur more often than duodenal fistulas but are more likely to close spontaneously. Somatostatin analogs decrease the amount of fistula drainage but do not decrease the duration of fistula or increase the rate of spontaneous closure. Persistent pancreatic fistulas may require distal pancreatectomy or Roux-en-Y pancreaticojejunostomy depending on the location of the duct injury.

b) Nuclear medicine

Nuclear medicine is a branch of medical imaging that uses small amounts of radioactive material to diagnose or treat a variety of diseases, including many types of
cancers, heart disease and certain other abnormalities within the body. Nuclear medicine or radionuclide imaging procedures are noninvasive and, with the
exception of intravenous injections, are usually painless medical tests that help physicians diagnose medical conditions. These imaging scans use radioactive materials called radiopharmaceuticals or radiotracers. Depending on the type of nuclear medicine exam you are undergoing, the radiotracer is
either injected into a vein, swallowed or inhaled as a gas and eventually accumulates in the organ or area of your body being examined, where it gives off energy in the form of gamma rays. This

energy is detected by a device called a gamma camera, a (positron emission tomography) PET scanner and/or probe. These devices work together with a computer to measure the amount of radiotracer absorbed by your body and to produce special pictures offering details on both the structure and function of organs and tissues.
In some centers, nuclear medicine images can be superimposed with computed tomography (CT) or magnetic resonance imaging (MRI) to produce special views, a
practice known as image fusion or co-registration. These views allow the information from two different studies to be correlated and interpreted on one image, leading to more precise information and accurate diagnoses. In addition, manufacturers are now making single photon emission computed tomography/computed tomography (SPECT/CT) and positron emission tomography/computed tomography (PET/CT) units that are able to perform both imaging studies at the same time. Nuclear medicine also offers therapeutic procedures such as radioactive iodine (I-131) therapy that uses radioactivematerial to treat cancer and other medical conditions affecting the thyroid gland.

What are some common uses of the procedure?

Physicians use radionuclide imaging procedures to visualize the structure and function of an organ, tissue, bone or system of the body.
Nuclear medicine imaging scans are performed to:

- Analyze kidney function.
- Visualize heart blood flow and function (such as a myocardial perfusion scan).
- Scan lungs for respiratory and blood flow problems.
- Identify inflammation in the gallbladder.
- Evaluate bones for fractures, infection, arthritis and tumors.
- Determine the presence or spread of cancer in various parts of the body.
- Identify bleeding into the bowel.
- Locate the presence of infection.
- Measure thyroid function to detect an overactive or underactive thyroid.
- Investigate abnormalities in the brain, such as seizures, memory loss and abnormalities in blood flow.
- Localize the lymph nodes before surgery in patients with breast cancer or melanoma.

In children, nuclear medicine is also used to:

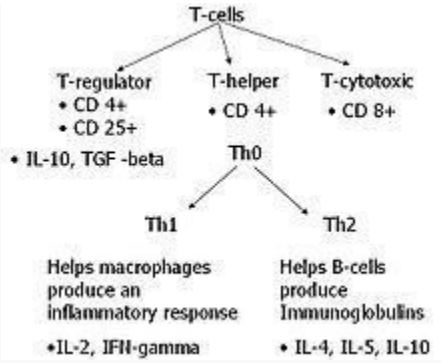
- Investigate abnormalities in the esophagus, kidneys and intestines.
- Evaluate the openness of tear ducts and shunts in the brain and heart.

Nuclear medicine therapies include:

- Radioactive iodine (I-131) therapy used to treat hyperthyroidism (overactive thyroid gland, for example, Graves' disease) and thyroid cancer.
- Radioactive antibodies used to treat certain forms of lymphoma (cancer of the lymphatic system).
- Radioactive phosphorus (P-32) used to treat certain blood disorders.
- Radioactive materials used to treat painful tumor metastases to the bones.
- I-131 MIBG (radioactive iodine laced with metaiodobenzylguanidine) used to treat adrenal gland tumors in adults and nerve tissue tumors in children.

c)

Rationale for biological therapy



Cytokines involved in IBD

Prior to the development of biological therapy as a modality to treat IBD, other medications that modulate the immune system—including 5-aminosalicylates, steroids, azathioprine, and other immunosuppressants—were primarily used in treatment. Patients with Crohn's disease that developed complications, including fistulae (= abnormal connections to the bowel) were treated with surgery. Patients with ulcerative colitis who do not respond to medications are still treated with colectomy (= removal of the colon).

However, basic science research showed that there were many cytokines that were elevated in both Crohn's disease and ulcerative colitis. In Crohn's disease cytokines, are of the type 1 (*Th1*) cytokines which include TNF-α, interleukin-2, and interferon γ. Ulcerative colitis was less conclusively linked to the production of *Th2* cytokines.

Infliximab monoclonal antibody infliximab is a mouse-human chimeric antibody to TNF-α. It first was used in the treatment of rheumatoid arthritis, and was the first biological agent used in the treatment of IBD. It is also used in the treatment of psoriasis and ankylosing spondylitis. Infliximab has shown significant success in treating Crohn's disease.

Other monoclonal antibodies

Other biological therapy agents and monoclonal antibodies have not showed as much efficacy in the treatment of IBD. These include etanercept (which is the soluble receptor for TNF. Adalimumab (which is a humanized recombinant antibody to TNF) showed effectiveness in patients with moderate-to-severe Crohn's disease, but less than that of infliximab. It however conveys an advantage in that it is given by subcutaneous injection as opposed to infliximab, which is given by intravenous infusion.

In 2005, two other recombinant medications were reported to have benefit in moderate to severe Crohn's disease. Certolizumab is a Fab fragment of a humanized anti-TNF alpha monoclonal antibody that is attached to polyethylene glycol to increase its half-life in circulation. It was found to have efficacy over placebo medications for 10 weeks in the treatment of moderate to

severe Crohn's disease in one large trial. Natalizumab is an anti-integrin monoclonal antibody that shown utility as induction and maintenance treatment for moderate to severe Crohn's disease. However, it has been associated with progressive multifocal leukoencephalopathy, a usually fatal viral infection of the brain, that may limit its use.

Biologic therapy for Ulcerative Colitis (UC):

A range of biological drugs have been evaluated recently for their therapeutic effect in treating patients with UC. Positive clinical trials include antitumour necrosis factor (anti-TNF)-α antibodies, anti-CD3 antibodies, and anti adhesion molecules.

Infliximab

Infliximab, a chimeric monoclonal antibody to TNF-α, has been used for the treatment of UC. Infliximab has a proven role in patients with acute severe UC.

Adalimumab

Adalimumab is a humanized anti-TNF agent administered subcutaneously. Current trials are in progress to evaluate the efficacy in the treatment of UC.

Visilizumab (Anti-CD3 Antibody)

Visilizumab is a humanized immunoglobulin G2 monoclonal antibody that binds specifically to human CD3 expressed on T cells, thereby inducing T-cell apoptosis. Visilizumab demonstrated clinical benefit in patients with severe UC refractory to intravenous steroids.

Alpha-4-integrin Blockade

Alpha-4 integrins facilitate leucocyte migration across vascular endothelium at the sites of inflammation. The development of monoclonal antibodies that inhibit or block alpha-4 integrins permitted the targeting of lymphocyte trafficking into the intestine as a novel therapeutic intervention. Natalizumab is available for use under a restricted license in the US, for patients with CD who are resistant to other therapies. A more gut-selective humanized antibody to the alpha-4 beta7 integrin (MLN02; Millennium Pharmaceuticals, Cambridge, MA, USA) has been evaluated in a multi-centre, double-blind, placebo-controlled trial. Short-term results are promising but long-term data are required for its use in UC.

Anti-interleukin IL-2 Receptor Antibody (Daclizumab)

An uncontrolled pilot study demonstrated that daclizumab, a humanized monoclonal antibody to the IL-2 receptor (CD25), may be effective for the treatment of active UC.

d)

Risk Factors for Breast Cancer
Factors Important in Populations
Age at menarche and menopause
Parity
Age at first birth
Breast-feeding

Exogenous hormone use or exposure
Alcohol consumption
Factors Important in Individual Patients
Gender (female >> male)
Age (steady increase with age)
Family history (mothers, sisters, daughters)
History of previous breast cancer (noninvasive or invasive, ipsilateral or contralateral)
Histologic Risk Factors
Proliferative breast disease
Atypical ductal hyperplasia (ADH)
Atypical lobular hyperplasia (ALH)
Lobular carcinoma in situ (LCIS)

Management of High-Risk Patients

In practice, clinicians prioritize risk factors and consider those that are important to individual patients in making recommendations about screening and intervention. For instance, as discussed earlier, reproductive factors are important for breast cancer incidence in populations but generally insufficient to base a recommendation for medical or surgical intervention.

Close Surveillance

Surveillance guidelines for individuals at high risk for breast cancer were established in 2002 by the National Comprehensive Cancer Network and the Cancer Genetics Studies Consortium. These guidelines are based primarily on expert opinion; screening guidelines for high-risk individuals are not established by prospective trials.

Recommendations for women in a family with a breast and ovarian cancer syndrome include monthly breast self-examination beginning at 18 to 20 years of age, semiannual clinical breast examination beginning at age 25, and annual mammography beginning at age 25, or 10 years before the earliest age at onset of breast cancer in a family member. Nonetheless, studies of women with known BRCA1 or BRCA2 mutations find that half the detected breast cancers were diagnosed as interval cancers; that is, they occurred between screening episodes and not during the course of routine screening. This observation has prompted many groups to add annual screening MRI to mammography, with some doing both simultaneously and others staggering the two examinations. If not done previously, genetic counseling is offered to those with a strong family history of early-onset breast and ovarian cancer, including a discussion of genetic testing for BRCA1 and BRCA2 mutations.

Chemoprevention for Breast Cancer

The only drug currently approved for reducing breast cancer risk is tamoxifen. Tamoxifen is an estrogen antagonist with proven benefit for the treatment of estrogen receptor (ER)-positive breast cancer. Furthermore, tamoxifen reduces the incidence of a second primary breast cancer in the contralateral breast of women who received the drug as adjuvant therapy for a first breast cancer. In the Early Breast Cancer Trialists' Collaborative Group (EBCTCG), adjuvant tamoxifen reduced the risk for a second breast cancer in the unaffected breast by 47%. Four prospective, randomized trials of preventive tamoxifen were initiated in healthy women, and the National Cancer Institute (NCI) and the National Surgical Adjuvant Breast and Bowel Project (NSABP) recently announced findings of the STAR trial of Tamoxifen treatment for 5 years was not devoid of complications. In the tamoxifen treatment arm, endometrial cancers resulting from estrogen-like effects of the drug on the endometrium were increased. Pulmonary embolism and deep venous thrombosis were also more common. Data on the efficacy of tamoxifen for reduction of breast cancer risk in BRCA1 and BRCA2 mutation carriers are currently too limited to quantify.

The second prevention trial conducted by the NSABP is the STAR trial, in which tamoxifen was compared with raloxifene. This comparison was based on analysis of more than 10,000 women who participated in placebo-controlled trials to evaluate the efficacy of raloxifene for prevention and treatment of osteoporosis. In favor of raloxifene, the number of uterine cancers was reduced by 36%, women taking raloxifene had 29% fewer episodes of venous thrombosis, and pulmonary embolism developed in fewer women .

Prophylactic Mastectomy

To summarize the accumulating evidence, prophylactic mastectomy probably reduces the chance of contracting breast cancer in high-risk women by 90%. However, women who are screened by mammograms annually have an overall 80% chance of surviving the occurrence of breast cancer. Coupled with penetrance figures in the range of 50% to 60% for mutation carriers, the chance of dying of breast cancer for carriers of BRCA1 or BRCA2 mutations is approximately 10% without undergoing preventive mastectomy.

Summary: Risk Assessment and Management

Risk factors for disease provide clues to pathogenesis and identify patients likely to benefit from surveillance and risk reduction interventions. Although breast cancer can develop in both sexes, women are at greatly increased risk and breast cancer in males is uncommon. Age is a useful risk factor applied everyday in clinical practice; breast cancer is rare in women younger than 30 and very common in women older than 60. Family history is most significant when breast cancer affects young first-degree relatives (mothers, sisters, and daughters) and when cases of ovarian cancer are found within the same side of the family. Histologic risk factors most concerning are ADH, ALH, and LCIS. A personal history of breast cancer predisposes to contralateral breast cancer in women undergoing mastectomy and to bilateral breast cancer in women undergoing breast conservation with wide excision and radiation.

Significant Risk Factors for Breast Cancer in Women: Assessment and Recommendations

RISK FACTORS	MAMMOGRAPHY SCREENING RECOMMENDATIONS	PREVENTIVE OPTIONS
Factors Conferring Moderate to High Risk		
Age >60 yr	Annual	Not usually recommended
Atypical hyperplasia (ductal or lobular)	Annual after diagnosis	Tamoxifen, 20 mg/day × 5 yr
LCIS	Annual after diagnosis	Tamoxifen, 20 mg/day × 5 yr
Personal history of either DCIS or invasive cancer, age >40 yr	Annual after diagnosis	No specific preventive recommended
Family history of breast cancer (1st-degree relative, age <50 yr; two relatives on same side of family)	Annual after age 40	Referral for genetic counseling
Factors Conferring Very High Risk		
Therapeutic thoracic radiation (age <30 yr)	Annual at 10 yr after radiotherapy	No specific preventive recommended
Personal history of DCIS or invasive cancer, age <40 yr	Annual after diagnosis	No specific preventive recommended
Family history of breast cancer (two 1st-degree relatives, age <50)	Annual after age 35-40	Referral for genetic counseling
Family history of breast and ovarian cancer (1st-degree relatives)	Annual after age 35-40	Referral for genetic counseling
Known carrier of a mutation in BRCA1 or BRCA2 or a 1st-degree relative with a mutation	Annual after age 25; consider annual MRI	Genetic testing for relatives; discuss prophylactic mastectomy or oophorectomy for carriers

DCIS, ductal carcinoma in situ; LCIS, lobular carcinoma in situ; MRI, magnetic resonance imaging.

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES

MS (General Surgery) Examination, 2006

April, 2006

PAPER IV

Time Allowed: 3 Hours **Full Marks: 100**

1. **Write about the following. 10 x 10**
    a) **Imperforate anus.**
    b) **Extra-hepatic biliary atresia.**
    c) **Regional anaesthesia.**
    d) **TRUS.**
    e) **Hydrocephalus.**
    f) **Pathogenesis of oral cancer.**
    g) **Vesico Vaginal Fistula.**
    h) **PCNL.**
    i) **Role of Radiotherapy in Colo-Rectal cancer.**
    j) **Empyema Thoracis.**

THE WEST BENGAL UNIVERSITY OF HEALTH SCIENCES
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PAPER IV

Time Allowed: 3 Hours **Full Marks: 100**

Answers.

(a) Imperforate anus.

Answer. Imperforate Anus:

- Incidence ranges from 1 in 4000 to 5000 live births
- Slightly more common in boys.
- The most common defect is an imperforate anus with a fistula between the distal colon and the urethra in boys or the vestibule of the vagina in girls.

Anorectal Embryology : By 6 weeks of gestation, the urorectal septum moves caudally to divide the cloaca into the anterior urogenital sinus and posterior anorectal canal. Failure of this septum to form results in a fistula between the bowel and urinary tract (in boys) or the vagina (in girls). Complete or partial failure of the anal membrane to resorb results in an anal membrane or stenosis. The perineum also contributes to development of the external anal opening and genitalia by formation of cloacal folds, which extend from the anterior genital tubercle to the anus. The perineal body is formed by fusion of the cloacal folds between the anal and urogenital membranes. Breakdown of the cloacal membrane anywhere along its course results in the external anal opening being anterior to the external sphincter (i.e., anteriorly displaced anus).

Classification of Congenital Anomalies of the Anorectum

	Female	**Male**
High	○ Anorectal agenesis with or without rectovaginal fistula. ○ Rectal atresia.	○ Anorectal agenesis with or without rectoprostatic urethral fistula. ○ Rectal atresia

Intermediate	○ Anorectal agenesis with or without rectovaginal fistula. ○ Anal agenesis.	○ Anorectal agenesis with or without rectobulbar urethral fistula. ○ Anal agenesis
Low	○ Anovestibular or anocutaneous fistula (anteriorly displaced anus). ○ Anal stenosis.	○ Anocutaneous fistula (anteriorly displaced anus). ○ Anal stenosis.
Cloaca		

Rectal atresia refers to an unusual lesion in which the lumen of the rectum is either completely or partially interrupted, with the upper rectum being dilated and the lower rectum consisting of a small anal canal. A persistent cloaca is defined as a defect in which the rectum, vagina, and urethra all meet and fuse to form a single, common channel. In girls, the type of defect may be determined by the number of orifices at the perineum. A single orifice would be consistent with a cloaca. If two orifices are seen (i.e., urethra and vagina), the defect represents either a high imperforate anus or, less commonly, a persistent urogenital sinus comprising one orifice and a normal anus as the other orifice.

Associated Anomalies:

Congenital anorectal anomalies often coexist with other lesions, and the VATER or VACTERL association must be considered. Bony abnormalities of the sacrum and spine occur in about one third of patients and consist of absent, accessory, or hemivertebrae or an asymmetric or short sacrum. Absence of two or more vertebrae is associated with a poor prognosis for bowel and bladder continence. Occult dysraphism of the spinal cord also may be present and consists of tethered cord, lipomeningocele, or fat within the filum terminale.

Preoperative Evaluation:

- Clinical evaluation includes plain radiographs of the spine as well as an ultrasound of the spinal cord.
- Genitourinary abnormalities other than the rectourinary fistula occur in 26% to 59% of patients.
- Vesicoureteral reflux and hydronephrosis are the most common, but other findings such as horseshoe, dysplastic, or absent kidney, as well as hypospadias or cryptorchidism, also must be considered.
- In general, the higher the anorectal malformation, the greater the frequency of associated urologic abnormalities.
- Radiographic evaluation of the urinary tract includes renal ultrasonography and voiding cystourethrography; a rectourinary fistula (if present) likely will be demonstrated by the latter procedure.

- In addition to the other tests described previously, a plain chest radiograph and careful clinical evaluation of the heart are conducted. If a cardiac defect is suspected, echocardiography is performed before any surgical procedure.
- Before feeding, a nasogastric tube is placed and its presence within the stomach confirmed to exclude esophageal atresia.

Low Lesions: The newborn infant with a low lesion can have a primary, single-stage repair procedure without need for a colostomy.

- Three basic approaches may be used. For anal stenosis in which the anal opening is in a normal location, serial dilation alone is usually curative.
  - Dilations are performed daily by the caretaker, and the size of the dilator is increased progressively (beginning with 8-9 French and increased slowly to 14 to 16 French).
  - If the anal opening is anterior to the external sphincter (i.e., anteriorly displaced anus) with a small distance between the opening and the center of the external sphincter, and the perineal body is intact, a cutback anoplasty is performed.
  - This consists of an incision extending from the ectopic anal orifice to the central part of the anal sphincter, thus enlarging the anal opening.
  - Alternatively, if there is a large distance between the anal opening and the central portion of the external anal sphincter, a transposition anoplasty is performed in which the aberrant anal opening is transposed to the normal position within the center of the sphincter muscles, and the perineal body is reconstructed.

High or Intermediate Lesions: Infants with intermediate or high lesions traditionally require a colostomy as the first part of a three-stage reconstruction.

- The colon is completely divided in the sigmoid region, with the proximal bowel as the colostomy and the distal bowel as a mucous fistula.
- Complete division of the bowel minimizes fecal contamination into the area of a rectourinary fistula, and it may lessen the risk for urosepsis.
- Furthermore, the distal bowel can be evaluated radiographically to determine the location of the rectourinary fistula.
- The second-stage procedure usually is performed 3 to 6 months later and consists of surgically dividing the rectourinary or rectovaginal fistula with pull-through of the terminal rectal pouch into the normal anal position.
- A posterior sagittal approach is the procedure most frequently performed. This consists of determination of the location of the central position of the anal sphincter by electrical stimulation of the perineum.
- An incision is then made in the midline extending from the coccyx to the anterior perineum and through the sphincter and levator musculature until the rectum is identified. The fistula from the rectum to the vagina or urinary tract is divided.
- The rectum is then mobilized and the perineal musculature reconstructed. The third and final stage is closure of the colostomy, which is performed a few months later. Anal dilations are begun 2 weeks after the pull-through procedure and continue for several months after the colostomy closure.

More recently, a single-stage procedure using a transabdominal laparoscopic approach has been described for treatment of intermediate and high imperforate anus anomalies.

This technique offers the theoretic advantages of placement of the neorectum within the central position of the sphincter and levator muscle complex under direct vision and avoids the need to cut across these structures. The long-term outcome of this new approach when compared with the standard posterior sagittal method is presently unknown.

- Most of the morbidity in patients with anorectal malformations is related to the presence of associated anomalies.
- Fecal continence is the major goal regarding correction of the defect.
- Prognostic factors for continence include the level of the pouch and whether the sacrum is normal.
- Constipation is the most common sequela.
- A bowel management program consisting of daily enemas is an important postoperative plan to reduce the frequency of soilage and improve the quality of life for these patients.

(b) Extra-hepatic biliary atresia. Answer. See the answer of question no.3(c) of Paper – III of 2006.

(c) Regional anaesthesia.

Answer. Regional anaesthesia is anaesthesia affecting only a large part of the body, such as a limb. Regional anaesthetic techniques can be divided into central and peripheral techniques. The central techniques include so called neuroaxial blocks (epidural anaesthesia, spinal anaesthesia). The peripheral techniques can be further divided into plexus blocks such as brachial plexus blocks, and single nerve blocks. Regional anaesthesia may be performed as a single shot or with a continuous catheter through which medication is given over a prolonged period of time, e.g. continuous peripheral nerve block. Last but not least, regional anaesthesia can be provided by injecting local anaesthetics directly into the veins of an arm (provided the venous flow is impeded by a tourniquette.) This is called intravenous regional techniques (Bier block). Another example of regional anaesthesia is called tumescent anaesthesia. In tumescent anaesthesia a dilute solution of local anaesthetic is pumped into the subcutaneous tissues of the region requiring surgery.

Regional anaesthesia generally involves the introduction of local anaesthetics to block the nerve supply to a specific part of the body, such as a limb, so patients cannot feel pain.

Indications and applications

Regional anaesthesia may provide anaesthesia (absence of feeling, including pain) to allow a surgical operation, or provide post-operative pain relief. Various brachial plexus

blocksexist for shoulder and arm procedures. Methods similar to routine regional anaesthetic techniques are also often used for treating chronic pain.

In labour and childbirth, epidural or combined spinal epidurals provide effective pain relief. Regional anaesthesia is now more common than general anaesthesia for caesarian sectionprocedures.

Nerve blocks are widely used in veterinary medicine to diagnose lameness. A very common application is the diagnosis of navicular disease in horses.

Relationship to other anaesthetic techniques

Unlike general anaesthesia, patients may remain awake during the procedure, resulting in reduced side-effects and enabling the surgeon to converse with the patient during the procedure if required. However, many patients prefer to receive sedation either during the block, the procedure, or both.

There is a spectrum of complexity between simple local anaesthetic infiltration and major regional blocks, such as the 'central neuraxial blocks' (spinal and epidural), with nerve blocks lying in the middle. Nerve blocks affecting major peripheral nerves such as the femoral nerve and sciatic nerve are also sometimes viewed as regional anaesthetic techniques.

Complications

Unlike a minor local anaesthetic infiltration to allow a wound to be sutured, or a skin lesion to be excised, regional anaesthesia may involve large doses of local anaesthetic, or administration of the local anaesthetic very close to, or directly into the central nervous system. Therefore there is a risk of complications from local anaesthetic toxicity (such as seizures and cardiac arrest) and for a syndrome similar to spinal shock.

Most regional anaesthetic techniques, even in expert hands, have a failure rate of 1–10%. Therefore general anaesthesia may become necessary even when a procedure was initially planned to be conducted under a regional technique.

For these reasons, regional anaesthesia is only ever conducted in an environment that is fully equipped and staffed to provide safe general anaesthesia should this be needed.

(d) TRUS.

Answer. TRUS (transrectal ultrasound): A transrectal ultrasound (TRUS) is an ultrasound technique that is used to view a man’s prostate and surrounding tissues. The ultrasound transducer (probe) sends sound waves through the wall of the rectum into the prostate gland, which is located directly in front of the rectum. TRUS may also be called prostate sonogram or endorectal ultrasound.

Indications:

- Measurement of prostate size (where gross prostatic enlargement is suspected on the basis of a DRE, and surgery, in the form of open prostatectomy, is contemplated)
- To assist prostate biopsy (allows biopsy of hypoechoic or hyperechoic lesions)
- Investigation of azoospermia (can establish the presence of ejaculatory duct obstruction)
- Help to guide during treatments for prostate cancer

    - TRUS is used to guide the placement of implants during brachytherapy.
    - It may help the doctor perform cryosurgery.
    - TRUS may be used to help deliver high-intensity focused ultrasound (HIFU).

A TRUS is usually done as on outpatient procedure in a doctor's office, clinic or hospital. The test usually takes 15–30 minutes.

- ❖ Some special preparation is needed before a TRUS.

    - ➢ Avoid taking Aspirin and other medications that may thin the blood for 7–10 days before the test.
    - ➢ An enema is used 1–4 hours before the procedure to help clean out the colon.
    - ➢ The man may be told to urinate to empty his bladder just before the procedure.

(e) Hydrocephalus.

Answer. Hydrocephalus, also known as "water on the brain", is a medical condition in which there is an abnormal accumulation of cerebrospinal fluid (CSF) in the ventricles, or cavities, of the brain. This may cause increased intracranial pressure inside the skull and progressive enlargement of the head, convulsion, and mental disability. Hydrocephalus can also cause death.

Signs and Symptoms: Symptoms of increased intracranial pressure may include headaches, vomiting, nausea, papilledema, sleepiness, or coma.
Elevated intracranial pressure may result in uncal and/orcerebellar tonsill herniation, with resulting life threatening brain stem compression. For details on other manifestations of increased intracranial pressure:

- ➢ The triad (Hakim triad) of gait instability, urinary incontinence and dementia is a relatively typical manifestation of the distinct entity normal pressure hydrocephalus (NPH). Focal neurological deficits may also occur, such as abducens nerve palsy and vertical gaze palsy (Parinaud syndrome due to compression of

the quadrigeminal plate, where the neural centers coordinating the conjugated vertical eye movement are located).

Normal pressure hydrocephalus: The symptoms depend on the cause of the blockage, the person's age, and how much brain tissue has been damaged by the swelling.

In infants with hydrocephalus, CSF fluid builds up in the central nervous system, causing the fontanelle (soft spot) to bulge and the head to be larger than expected.

Early symptoms may also include:	Symptoms that may occur in older children can include:	
• Eyes that appear to gaze downward • Irritability • Seizures • Separated sutures • Sleepiness • Vomiting	• Brief, shrill, high-pitched cry • Changes in personality, memory, or the ability to reason or think • Changes in facial appearance and eye spacing • Crossed eyes or uncontrolled eye movements • Difficulty feeding • Excessive sleepiness • Headache • Irritability, poor temper control	• Loss of bladder control (urinary incontinence) • Loss of coordination and trouble walking • Muscle spasticity (spasm) • Slow growth (child 0-5 years) • Slow or restricted movement • Vomiting.

Epidemiology: Hydrocephalus affects both pediatric and adult patients. According to the NIH website, there are an estimated 700,000 children and adults living with hydrocephalus.

Pediatric hydrocephalus affects one in every 500 live births, making it one of the most common developmental disabilities, more common than Down syndrome or deafness. It is the leading cause of brain surgery for children in the United States. There are over 180 different causes of the condition, one of the most common acquired etiologies being brain hemorrhage associated with premature birth. Pediatric hydrocephalus may also be a

heritable condition, and mainly affects males. Hydrocephalus may be detectable during prenatal ultrasound examinations.

One of the most performed treatments for hydrocephalus, the cerebral shunt, was first developed in 1960. The shunt must be implanted through neurosurgery into the patient's brain, a procedure which itself may cause brain damage.

Hydrocephalus also causes infection in the cerebrospinal fluid. It can also result from maternal infection during birth.

Pathology

Hydrocephalus is usually due to blockage of cerebrospinal fluid (CSF) outflow in the ventricles or in the subarachnoid space over the brain. In a person without hydrocephalus, CSF continuously circulates through the brain, its ventricles and the spinal cord and is continuously drained away into the circulatory system. Alternatively, the condition may result from an overproduction of the CSF fluid, from a congenital malformation blocking normal drainage of the fluid, or from complications of head injuries or infections.

Compression of the brain by the accumulating fluid eventually may cause convulsions and mental retardation. These signs occur sooner in adults, whose skulls no longer are able to expand to accommodate the increasing fluid volume within. Fetuses, infants, and young children with hydrocephalus typically have an abnormally large head, excluding the face, because the pressure of the fluid causes the individual skull bones — which have yet to fuse — to bulge outward at their juncture points. Another medical sign, in infants, is a characteristic fixed downward gaze with whites of the eyes showing above the iris, as though the infant were trying to examine its own lower eyelids.

The elevated intracranial pressure may cause compression of the brain, leading to brain damage and other complications. Conditions among affected individuals vary widely. Children who have had hydrocephalus may have very small ventricles, and presented as the "normal case".

If the foramina of the fourth ventricle or the cerebral aqueduct are blocked, cereobrospinal fluid (CSF) can accumulate within the ventricles. This condition is called **internal hydrocephalus** and it results in increased CSF pressure. The production of CSF continues, even when the passages that normally allow it to exit the brain are

blocked. Consequently, fluid builds inside the brain causing pressure that compresses the nervous tissue and dilates the ventricles. Compression of the nervous tissue usually results in irreversible brain damage. If the skull bones are not completely ossified when the hydrocephalus occurs, the pressure may also severely enlarge the head. The cerebral aqueduct may be blocked at the time of birth or may become blocked later in life because of a tumor growing in the brainstem.

Internal hydrocephalus can be successfully treated by placing a drainage tube (shunt) between the brain ventricles and abdominal cavity to eliminate the high internal pressures. There is some risk of infection being introduced into the brain through these shunts, however, and the shunts must be replaced as the person grows. A subarachnoid hemorrhage may block the return of CSF to the circulation. If CSF accumulates in the subarachnoid space, the condition is called **external hydrocephalus**. In this condition, pressure is applied to the brain externally, compressing neural tissues and causing brain damage. Thus resulting in further damage of the brain tissue and leading to necrotization.

Classification

- Hydrocephalus can be caused by impaired cerebrospinal fluid (CSF) flow, reabsorption, or excessive CSF production.
- The most common cause of hydrocephalus is CSF flow obstruction, hindering the free passage of cerebrospinal fluid through the ventricular system and subarachnoid space (e.g.,stenosis of the cerebral aqueduct or obstruction of the interventricular foramina - **foramina of Monro** secondary to tumours, haemorrhages, infections or congenital malformations.
- Hydrocephalus can also be caused by overproduction of cerebrospinal fluid (relative obstruction) (e.g., papilloma of choroid plexus).
- Based on its underlying mechanisms, hydrocephalus can be classified into **communicating** and **non-communicating** (obstructive). Both forms can be either **congenital** or **acquired**.

Communicating: Communicating hydrocephalus, also known as non-obstructive hydrocephalus, is caused by impaired cerebrospinal fluid resorption in the absence of any CSF-flow obstruction between the ventricles and subarachnoid space. It has been theorized that this is due to functional impairment of the arachnoid granulations, which are located along the superior sagittal sinus and is the site of cerebrospinal fluid resorption back into the venous system. Various neurologic conditions may result in communicating hydrocephalus, including subarachnoid/intraventricular haemorrhage, meningitis, Chiari malformation, and congenital absence of arachnoidal granulations

(Pacchioni's granulations). Scarring and fibrosis of the subarachnoid space following infectious, inflammatory, or hemorrhagic events can also prevent resorption of CSF, causing diffuse ventricular dilatation.

Normal pressure hydrocephalus (NPH): is a particular form of communicating hydrocephalus, characterized by enlarged cerebral ventricles, with only intermittently elevated cerebrospinal fluid pressure. The diagnosis of NPH can be established only with the help of continuous intraventricular pressure recordings (over 24 hours or even longer), since more often than not instant measurements yield normal pressure values. Dynamic compliance studies may be also helpful. Altered compliance (elasticity) of the ventricular walls, as well as increased viscosity of the cerebrospinal fluid, may play a role in the pathogenesis of normal pressure hydrocephalus.

Hydrocephalus ex vacuo also refers to an enlargement of cerebral ventricles and subarachnoid spaces, and is usually due to brain atrophy (as it occurs in dementias), post-traumatic brain injuries and even in some psychiatric disorders, such as schizophrenia. As opposed to hydrocephalus, this is a compensatory enlargement of the CSF-spaces in response to brain parenchyma loss - it is not the result of increased CSF pressure.

Non-communicating: Non-communicating hydrocephalus, or obstructive hydrocephalus, is caused by a CSF-flow obstruction ultimately preventing CSF from flowing into the subarachnoid space (either due to external compression or intraventricular mass lesions).

- **Foramen of Monro obstruction** may lead to dilation of one or, if large enough (e.g., in Colloid cyst), both lateral ventricles.
- **The aqueduct of Sylvius,** normally narrow to begin with, may be obstructed by a number of genetically or acquired lesions (e.g., atresia, ependymitis, hemorrhage, tumor) and lead to dilation of both lateral ventricles as well as the third ventricle.
- **Fourth ventricle** obstruction will lead to dilatation of the aqueduct as well as the lateral and third ventricles.
- **The foramina of Luschka and foramen of Magendie** may be obstructed due to congenital failure of opening (e.g., Dandy-Walker malformation).

Congenital: The cranial bones fuse by the end of the third year of life. For head enlargement to occur, hydrocephalus must occur before then. The causes are usually genetic but can also be acquired and usually occur within the first few months of life, which include 1) intraventricular matrix hemorrhages in premature infants, 2) infections, 3) type II Arnold-Chiari malformation, 4) aqueduct atresia and stenosis, and 5) Dandy-Walker malformation.

In newborns and toddlers with hydrocephalus, the head circumference is enlarged rapidly and soon surpasses the 97th percentile. Since the skull bones have not yet firmly joined together, bulging, firm anterior and posterior fontanelles may be present even when the patient is in an upright position.

The infant exhibits fretfulness, poor feeding, and frequent vomiting. As the hydrocephalus progresses, torpor sets in, and the infant shows lack of interest in his surroundings. Later on, the upper eyelids become retracted and the eyes are turned downwards (due to hydrocephalic pressure on the mesencephalic tegmentum and paralysis of upward gaze). Movements become weak and the arms may become tremulous. Papilledema is absent but there may be reduction of vision. The head becomes so enlarged that the child may eventually be bedridden. About 80-90% of fetuses or newborn infants with spina bifida—often associated with meningocele or myelomeningocele—develop hydrocephalus.

Acquired: This condition is acquired as a consequence of CNS infections, meningitis, brain tumours, head trauma, intracranial hemorrhage (subarachnoid or intraparenchymal) and is usually extremely painful.

Effects

Because hydrocephalus can injure the brain, thought and behavior may be adversely affected. Learning disabilities including short-term memory loss are common among those with hydrocephalus, who tend to score better on verbal IQ than on performance IQ, which is thought to reflect the distribution of nerve damage to the brain. However the severity of hydrocephalus can differ considerably between individuals and some are of average or above-average intelligence. Someone with hydrocephalus may have motion and visual problems, problems with coordination, or may be clumsy. They may reach puberty earlier than the average child . About one in four develops epilepsy.

Treatment

Hydrocephalus treatment is surgical. It involves the placement of a ventricular catheter (a tube made of silastic), into the cerebral ventricles to bypass the flow obstruction/malfunctioningarachnoidal granulations and drain the excess fluid into other body cavities, from where it can be resorbed. Most shunts drain the fluid into the peritoneal cavity (ventriculo-peritoneal shunt), but alternative sites include the right atrium (ventriculo-atrial shunt), pleural cavity (ventriculo-pleural shunt), and gallbladder. A shunt system can also be placed in the lumbar space of the spine and have the CSF redirected to the peritoneal cavity (Lumbar-peritoneal shunt). An alternative treatment for obstructive hydrocephalus in selected patients is theendoscopic third ventriculostomy (ETV), whereby a surgically created opening in the floor of the third ventricle allows the CSF to flow directly to the basal cisterns, thereby shortcutting any obstruction, as in aqueductal stenosis. This may or may not be appropriate based on individual anatomy.

(f) Pathogenesis of oral cancer.

Answer. Risk factors for oral cancer

- **Tobacco:** Oral neoplasia has been associated with chewing of tobacco with betel quid (BQ) in India and other asian countries, whereas in western countries, cigarette smoking and heavy alcohol consumption are the main risk factors. Chewing of tobacco with BQ increases exposure to carcinogenic tobacco-specific nitrosamines (TSNA) and to nitrosamines derived from areca nut alkaloids. Furthermore, reactive oxygen species (ROS) implicated in multistage carcinogenesis, are also generated in substantial amounts in the oral cavity during chewing.
- **Betel Quid and Areca Nut:** Betel chewing is reported to be the most important etiological factor in oral submucous fibrosis. The use of betel quid, containing both areca nut and tobacco, is associated with a much higher relative risk of oral cancer, between 8-15 times as compared to that of 1-4 times, associated with using the quid, without tobacco.
- **Alcohol:** Alcohol, acting both independently as well as synergistically with smoking, has been implicated in oral carcinogenesis.
- **Viruses:** Another risk factor is human papillomavirus (HPV), which is also closely associated with benign and malignant oral lesions. This virus is detected in condylomas, focal epithelial hyperplasia, squamous cell papilloma and malignant oral lesions. HPV positivity is higher in tumors from the oral cavity (59%), pharynx (43%) and larynx (33%).
- **Diet:** The importance of diet and nutrition in oral neoplasia has been indicated in several epidemiological studies. Fruits and vegetables (high in vitamins A and C) are described as protective in oral neoplasia, whereas meat and red chilli powder are thought to be risk factors. Although the individual micronutrients responsible have not been formally identified, vegetables and fruits that protect against oral cancer and precancer, are rich in b-carotene, vitamin C and vitamin E, with anti-oxidant properties. Iron deficiency, resulting in oral epithelial atrophy and the Plummer-Vinson (Patterson Brown Kelly) syndrome, is associated with cancer of upper air and food passages and dietary iron may play a protective role in maintaining the thickness of the epithelium.
- **Family History of Head and Neck Squamous Cell Carcinoma (HNSCC) :** Epidemiological evidence from case-control studies of HNSCC, indicates that a family history of head and neck cancer is a risk factor. The ability to repair DNA damaged by tobacco carcinogens, such as benzo-[a]-pyrene diol epoxide, is defective in some patients with head and neck cancer. Head and neck cancer patients show an increased susceptibility to chromosome damage by mutagens.

- **Immune Deficiency:** A defective immune response, as seen in a human immunodeficiency virus (HIV)-infected individual, may predispose to cancer. The commonest oral malignancy in HIV-infected patients is Kaposi's sarcoma and the Human Herpes virus type 8 (HHV-8) has been implicated as the aetiological agent. Lymphoma, mostly non-Hodgkin B cell lymphoma in HIV-infected individuals, or other immunosuppressed states, is commonly associated with Epstein-

Barr virus and may occur in the head and neck. Oral squamous cell carcinomas of the lip are more common in transplant recipients receiving immunosuppressive therapy, but HIV infection does not predispose to intra-oral squamous cell carcinoma.

Candida: Candida albicans can induce epithelial proliferation and can produce carcinogens from procarcinogens in vitro . Chronic hyperplastic candidosis presents as nodular or speckled-white mucosal plaques. They are potentially malignant oral epithelial lesions.

Molecular Changes in Oral Cancer:

Cancer occurs through multiple steps, each characterized by the sequential stimulation of additional genetic defects, followed by clonal expansion. The genetic alterations observed in head and neck cancer are mainly due to oncogene activation and tumor suppressor gene inactivation, leading to de-regulation of cell proliferation and death. These genetic alterations, include gene amplification and overexpression of oncogenes such as myc, erbB-2, Epidermal Growth Factor Receptor (EGFR), cyclin D1 and mutations, deletions and hypermethylation leading to p16 and p53 tumor suppressor gene inactivation.

Tumor Suppressor Genes (TSGs) and Growth Regulators:
Growth regulators and TSGs act as transducers of negative growth signals. Genetic alterations involving the tumor suppressor genes *p16* and *p53,* are frequently observed in head and neck tumors. Genetic abnormalities inactivating the *p16* gene might confer cell growth defects, contributing to the tumorigenic process. These genes are involved in cell cycle regulation, including cell cycle arrest and apoptosis. Alteration in both alleles of a gene is required for the loss of function.

Genomic instability

The evaluation of the genomic stability can be done using techniques such as Loss of heterozygosity (LOH) screening and comparative genomic hybridization (CGH). Loss of heterozygosity of the *p53* allele has been reported in 20% of OSCCs, as well as in 22% of premalignant oral lesions.[33] The risk of progression from premalignancy to cancer is low, when no genetic changes were seen, intermediate, if there is genetic loss on the short arms of chromosomes 3 and 9 (3p and 9p) and high, if there is 3p and 9p loss accompanied by genetic loss on additional chromosome arms, including 4q, 8p, 11q, 13q and 17p. The LOH of the H-ras allele may encompass a tumor suppressor gene in the vicinity of the oncogene. Loss of chromosome 11 alleles has been reported in a number of tumors.

Role of deregulated apoptosis in the pathogenesis of oral cancer

The deregulation of apoptosis-related genes, aids in successful carcinogenesis. The relative contribution of apoptosis and proliferation to disease progression in the oral mucosa, was examined using terminal deoxynucleotidyl transferase nick end-labeling (TUNEL) assay and Ki-67 staining. Further apoptosis related cell cycle regulators, namely Retinoblastoma (Rb), cyclin D1 and Fragile histidine triad gene (FHIT) were

analyzed for Loss of heterozygosity (LOH), gene amplification and aberrant transcripts, respectively in oral cancer samples. Status of *p53* , bcl-2 and bax, members of the *p53* dependent apoptotic pathway, were evaluated in oral cancers/oral lesions by immunohistochemistry. Frequent overexpression of apoptosis regulators p53, bcl-2 and bax, was observed in oral cancers and in a subset of oral lesions. It was further revealed that there is overexpression of anti-apoptotic members of the bcl-2 family namely, bclxL and Mcl-1, in oral cancer cell lines. These studies thus indicate, that evasion of apoptosis via abnormal expression of bcl-2, bclxL, mcl-1 and *p53,* may contribute to oral cancer pathogenesis.

Enhanced telomerase activity

The structures at the ends of eukaryotic chromosomes are termed "Telomeres". As telomeres are lost during cell divisions, the chromosomal ends are no longer protected, which leads to the fusion of the chromosomes and karyotypic abnormalities, that eventually cause cell death. The ribonucleoprotein enzyme telomerase extends the telomeric repeat sequences at the chromosomal ends; and it is active in a majority (90%) of human neoplasia, but inactive in most normal cells.

Neovascularization:
Angiogenesis, defined as the growth of new blood vessels (neovascularization) from pre-existing ones, is a complex process, absolutely needed for the continued growth and survival of solid neoplasia. This process of angiogenesis is in itself a multi-step process, that appears to be regulated by both stimulatory and inhibitory factors.The steps critical to successful angiogenesis, include the degradation of the extracellular matrix, endothelial cell proliferation, migration and assembly of endothelial cells into higher order structures. In the majority of cancers, highly vascularized tumors showed a poor prognosis and the influence of tumor angiogenesis proved to be independent of conventional prognostic indicators.

Cytokine profile

Suppression of *Th1* cytokine genes, was reported due to increase in tumor load and lymph node invasion, which skewed it towards a *Th2* -like cytokine response. HNSCCs, express Interleukin-4 receptors *in situ* and Interleukin-13 was secreted by HNSCCs, but does not modulate their growth *in vitro* . It has been postulated that these cytokines, in addition to others, were responsible for the growth pattern of these tumors and could be responsible for their active spread.

(g) Vesico Vaginal Fistula.

Answer. Vesicovaginal Fistula

Introduction

Vesicovaginal fistula (VVF) is a subtype of female urogenital fistula (UGF). VVF is an abnormal fistulous tract extending between the bladder and the vagina that allows the continuous involuntary discharge of urine into the vaginal vault.

Etiology

Developing countries

- Numerous factors contribute to the development of VVF in developing countries.
- Marriage and conception at a young age, often before full pelvic growth has been achieved.
- Chronic malnutrition increases the risk of cephalopelvic disproportion and malpresentation.
- Few women are attended by qualified health care professionals or have access to medical facilities during childbirth; their obstructed labor may be protracted for days or weeks.
- Other cultural factors that increase the likelihood of obstetrical UGFs include outlet obstruction due to female circumcision and the practice of harmful traditional medical practices such as Gishiri incisions (anterior vaginal wall incisions) and the insertion of caustic substances into the vagina with the intent to treat a gynecologic condition or to help the vagina to return to its nulliparous state.

Developed countries

- VVFs in developing countries are attributed predominantly to inadvertent bladder injury during pelvic surgery (90%). Such injuries include unrecognized intraoperative laceration of the bladder, bladder wall injury from electrocautery or mechanical crushing, and the dissection of the bladder into an incorrect plane, causing avascular necrosis.
- Suture placement through the bladder wall in itself may not play a significant role in VVF development. However, the risk of formation of a hematoma or avascular necrosis after a suture is placed through the bladder wall can lead to infection, abscess, and subsequent suture erosion through the bladder wall. Gynecologic procedures are the most common iatrogenic factor.
- Also occurred after radiotherapy.
- Obstetric UGFs in modern centers include vaginal lacerations from forceps rotations, cesarean delivery, hysterectomy, and ruptured uterus.
- Other types of pelvic surgery (eg, urologic, gastrointestinal surgery) also contribute to the incidence of VVFs; such surgeries include suburethral sling procedures, surgical repair of urethral diverticulum, electrocautery of bladder papilloma, and surgery for pelvic carcinomas.
- Other less common causes of VVFs include pelvic infections (eg, tuberculosis, syphilis, lymphogranuloma venereum), vaginal trauma, and vaginal erosion with

foreign objects (eg, neglected pessary). Lastly, a congenital urogenital abnormality may exist that includes a VVF.

- Risk factors that predispose to VVFs include prior pelvic or vaginal surgery, previous PID, ischemia, diabetes, arteriosclerosis, carcinoma, endometriosis, anatomic distortion by uterine myomas, and infection, particularly postoperative cuff abscess.

Presentation

The uncontrolled leakage of urine into the vagina is the hallmark symptom of patients with UGFs. Patients may complain of urinary incontinence or an increase in vaginal discharge following pelvic surgery or pelvic radiotherapy with or without antecedent surgery. The drainage may be continuous; however, in the presence of a very small UGF, it may be intermittent. Increased postoperative abdominal, pelvic, or flank pain; prolonged ileus; and fever should alert the physician to possible urinoma or urine ascites and mandates expeditious evaluation. Recurrent cystitis or pyelonephritis, abnormal urinary stream, and hematuria also should initiate a workup for UGF.

The time from initial insult to clinical presentation depends on the etiology of the VVF. A VVF secondary to a bladder laceration typically presents immediately. Approximately 90% of genitourinary fistulas associated with pelvic surgery are symptomatic within 7-30 days postoperatively.

Patients with radiation-induced VVFs initially present with symptoms of radiation cystitis, hematuria, and bladder contracture.

Indications

Symptomatic VVF merits appropriate treatment. Further details regarding the indications for a specific procedure are described in Surgical objectives or principles.

Contraindications

In general, no absolute contraindications exist for the attempted correction of a VVF in patients who can medically tolerate a surgical procedure.

Workup

Laboratory Studies

- Upon examination of the vaginal vault, any fluid collection noted can be tested for urea, creatinine, or potassium concentration to determine the likelihood of a diagnosis of VVF as opposed to a possible diagnosis of vaginitis.
- Indigo carmine dye can be given intravenously and if the dye appears in the vagina, a fistula is confirmed.
- Once the diagnosis of urine discharge is made, the physician must identify its source.
- Cystourethroscopy may be performed, and the fistula(s) may be identified.
- If ureter involvement is suspected then IVP can be performed.

- The differential diagnosis for the discharge of urine into the vagina includes single or multiple vesicovaginal, urethrovaginal, or ureterovaginal fistulas and fistula formation between the urinary tract and the cervix, uterus, vagina, vaginal cuff, or (rarely) ureteral fistula to a fallopian tube.
- A full vaginal inspection is essential and should include assessment of tissue mobility; accessibility of the fistula to vaginal repair; determination of the degree of tissue inflammation, edema, and infection; and possible association of a rectovaginal fistula.
- Urine should be collected for culture and sensitivity, and patients with positive results should be treated prior to surgery.
- In patients with a history of local malignancy, a biopsy of the fistula tract and microscopic evaluation of the urine is warranted.

Imaging Studies

- Radiologic studies should be employed prior to surgical repair of a VVF.
  - An intravenous urogram (IVU) is necessary to exclude ureteral injury or fistula because 10% of VVFs have associated ureteral fistulas.
  - If suspicion is high for a ureteral injury or fistula and the IVU findings are negative, retrograde ureteropyelography should be performed at the time of cystoscopy and examination under anesthesia.
  - A Tratner catheter can be used to assist in evaluation of a urethrovaginal fistula.
- Fibrin occlusion therapy is used for the treatment of a variety of fistulas, such as enterocutaneous, anorectal, bronchopleural, ureterocutaneous, and, more recently, VVFs. Fistulograms are a valuable adjunct to fibrin occlusion therapy.

Diagnostic Procedures

- Intraoperative assessment for bladder or ureteral injury may be performed by administering indigo carmine intravenously and closely observing for any subsequent extravasation of dye into the pelvis.
  - Cystourethroscopy to assure bilateral ureteral patency and absence of suture placement in the bladder or urethra has been advocated by some authors as a standard for all pelvic surgery.
  - Alternatively, intraoperative back-filling of the bladder with methylene blue or sterile milk before completing abdominal or vaginal surgery also may help detect a bladder laceration.
  - Retrograde filling of the bladder also can be used during surgery to better define the bladder base in more difficult dissections.
- The evaluation should include a complete physical examination and detailed review of systems. A cystoscopic examination with a small scope (eg, 19F) may be used to identify VVF in the bladder or urethra, to determine the number and location and proximity to ureteric orifices, and to identify and remove abnormal entities such as calculi or sutures in the bladder.

- In the office, as with the operating room setting, the bladder can be filled with sterile milk or methylene blue in retrograde fashion using a small transurethral catheter.
    - Placement of tampons in tandem in the vaginal vault and observation for staining of the tampons by methylene blue may help to identify and locate fistulas.
    - Staining of the apical tampon would implicate the vaginal apex or cervix/uterus/fallopian tube; staining of a distal tampon raises suspicion of a urethral fistula.
    - If the tampons are wet but not stained, oral phenazopyridine (Pyridium) or intravenous indigo carmine then can be used to rule out a ureterovaginal, ureterouterine, or ureterocervical fistula.
    - Evidence of staining or wetting of a tampon should then prompt the physician to proceed with additional diagnostic testing prior to proceeding with definitive management.
- Water cystoscopy may be inadequate in the face of large or multiple fistulas.
- A cystoscopic examination using carbon dioxide gas may be used with the patient in the genupectoral position. With the vagina filled with water or isotonic sodium chloride solution, the infusion of gas through the urethra with a cystoscope produces air bubbles in the vaginal fluid at the site(s) of a UGF (flat tire sign).
- Combined vaginoscopy-cystoscopy: Andreoni et al describe their technique of simultaneously viewing 2 images on the monitor screen (both cystoscopic and vaginal examinations).[14] They use a laparoscope and clear speculum in the vagina and they use regular cystoscope in the bladder to enhance visualization and identification of VVFs. Transillumination of the bladder or vagina by turning off the vaginal or bladder light source allows for easier identification of the fistula in the more difficult cases.
- Color Doppler ultrasonography with contrast media of the urinary bladder may be considered in cases where cystoscopic evaluation is suboptimal, such as in those patients with severe bladder wall changes like bullous edema or diverticula. Color Doppler ultrasonography demonstrated a VVF in 92% of the patients studied by Volkmer and colleagues using diluted contrast media and observing jet phenomenon through the bladder wall toward the vagina.

Treatment

Medical Therapy

Conservative management

If VVF is diagnosed within the first few days of surgery, a transurethral or suprapubic catheter should be placed and maintained for up to 30 days. Small fistulas (<1 cm) may resolve or decrease during this period if caution is used to ensure proper continuous drainage of the catheter.

Surgical Therapy

Surgical objectives or principles

Adequate exposure of the operative field should be obtained to avoid inadvertent organ injury and to ensure prompt identification of any injury incurred.

Minimize bleeding and hematoma formation. The closure of dead space at the anterior vaginal wall upon completion of an anterior colporrhaphy will prevent hematoma formation. This technique employs intermittently incorporating pubocervicovaginal fascia with the vaginal mucosal layer as the vaginal wall is sutured.

Widely mobilize the bladder from the vagina during hysterectomy to diminish the risk of suture placement into the bladder wall. A minimum of a 1- to 2-cm margin of dissection of the bladder from the vaginal cuff should be developed prior to cuff closure.

Dissect the pubocervicovaginal endopelvic fascia between the vagina and the bladder in the appropriate plane. Dissection may be easier with a sharp technique compared to a blunt technique; the key is to prevent trauma and separation of bladder wall fibers as the bladder is mobilized off the anterior vaginal wall. The principle of traction and countertraction of the bladder and uterus works well to effect a bloodless dissection at the areolar pubocervicovaginal fascial plane.

If scarring is present at the pubocervicovaginal fascia and dissection is difficult, consider performing an intentional anterior extraperitoneal cystotomy. This technique enables the surgeon to assess the anatomic boundaries of the bladder wall with digital palpation. If scarring is present at the pubocervicovaginal fascia and dissection is difficult, consider employing an intrafascial technique of hysterectomy to best dissect the endopelvic fascial plane.

Intraoperative retrograde filling and emptying of the bladder or mild traction on a temporarily placed small Foley catheter inserted into the fistula itself are helpful to optimally identify anatomical planes and reveal intraoperative bladder lacerations.

Consider supracervical abdominal hysterectomy instead of TAH. The incidence of UGF formation is lower for supracervical versus total hysterectomy.

If an intraoperative bladder injury does occur, Tancer argues strongly for widely mobilizing the bladder from the underlying structures (fascia and vagina, cervix, or uterus). In doing so, the surgeon can effect a VVF closure under no tension.

For repairing a cystotomy at the trigonal area, a transverse closure is preferable over a vertical one. Vertical closure would be more likely to produce ureteral obstruction because the ureteral orifices would be drawn inward toward each other. Ureteral catheters should be considered in repair of a cystotomy involving or encroaching on ureteric orifices.

Consider performing cystourethroscopy when performing pelvic surgery. Cystourethroscopy to assure bilateral ureteral patency and the absence of suture placement in the bladder or the urethra has been advocated by some authors as a standard for all pelvic surgery.

(h) PCNL.
Answer. Percutaneous nephrolithotomy (PCNL)
Indications for PCNL:

PCNL is generally recommended for

- Stones >3cm in diameter,
- Those that have failed eswl and/or an attempt at flexible ureteroscopy and laser treatment.
- It is the first-line option for staghorn calculi,with ESWL and/or repeat PCNL being used for residual stone fragments.

For stones 2-3cm in diameter, options include ESWL (with a JJ stent in situ), flexible ureteroscopy and laser treatment, and PCNL. PCNL gives the best chance of complete stone clearance with a single procedure, but this is achieved at a higher risk of morbidity. Some patients will opt for several sessions of ESWL or flexible ureteroscopy/laser treatment and the possible risk of ultimately requiring PCNL because of failure of ESWL or laser treatment, rather than proceeding with PCNL up front ~50% of stones >2cm in diameter will be fragmented by flexible ureteroscopy and laser treatment.

Technique: PCNL is the removal of a kidney stone via a track developed between the surface of the skin and the collecting system of the kidney. The first step requires inflation of the renal collecting system (pelvis and calyces) with fluid or air instilled via a ureteric catheter inserted cystoscopically. This makes subsequent percutaneous puncture of a renal calyx with a nephrostomy needle easier. Once the nephrostomy needle is in the calyx, a guidewire is inserted into the renal pelvis to act as a guide over which the track is dilated. An access sheath is passed down the track and into the calyx, and through this a nephroscope can be advanced into the kidney. An ultrasonic lithotripsy probe is used to fragment the stone and remove the debris.
A posterior approach is most commonly used; below the 12th rib (to avoid the pleura and far enough away from the rib to avoid the intercostals, vessels, and nerve). The preferred approach is through a posterior calyx, rather than into the renal pelvis, because this avoids damage to posterior branches of the renal artery which are closely associated with the renal pelvis. General anaesthesia is usual, though regional or even local anaesthesia (with sedation) can be used.

Outcomes of PCNL:

For small stones, the stone-free rate after PCNL is in the order of 90â€“95%. For staghorn stones, the stone-free rate of PCNL, when combined with post-operative ESWL for residual stone fragments, is in the order of 80-85%.

(i) Role of Radiotherapy in Colo-Rectal cancer.

Answer. Colorectal cancer is the third most common malignancy in the United States.Worldwide, there were approximately 1 million new cases and 500,000 deaths secondary to colorectal cancer. Approximately 80% of patients with colorectal cancer present with localized disease; however, despite surgery with curative intent, patients still face a significant risk of local and distant recurrence and cancer-related mortality.

Surgical resection of the primary tumor with acceptable margins and concurrent, en bloc lymph node dissection remains the standard of care for patients who present with localized disease and remains the most effective modality of cure of colorectal cancer. The likelihood of cure is directly related to the depth of tumor penetration through the bowel wall and lymph node metastasis. While patients with lymph node involvement can be cured with surgery alone, up to 40% to 60% of these patients will have recurrence or will die of metastatic disease.

Rationale for adjuvant therapy

Patients with colorectal cancer who undergo surgery with curative intent, in whom no gross postoperative disease is detectable, may still develop either local recurrence or distant metastasis. This is due to the existence of micrometastatic disease present at the time of surgery that remains in the tumor bed after resection or by tumor cells that spread by lymphatic or blood vessels to distant sites. The main goal of adjuvant therapy is to eliminate these micrometastases. This goal should be achieved while maintaining the best possible quality of life for the patient without compromising the ability to cure the malignancy. The identification of patients with a sufficiently high risk of recurrence to warrant the risks of adjuvant chemotherapy and possible radiation therapy depends on appropriate staging.

The treatment of rectal cancer has changed significantly during the past 20 years, and there is considerable controversy today concerning the precise role of surgery, radiation therapy, and chemotherapy, and the ideal timing of each modality with relation to the others. Although information from clinical trials has provided data supporting the multimodality treatment of rectal cancer, the criteria for patient selection remains controversial. However, some generalities can be made at the present time:

RT offers significant benefit to many patients with rectal cancer, and preoperative radiation is superior to postoperative radiation. Until recently, preoperative radiation (combined with chemotherapy) has generally been reserved for locally advanced distal rectal cancers (within 10 cm of the anal verge, stage II or higher), but a recent analysis based on a cooperative 7-year trial of the National Research Council (NRC) of the United Kingdom and the National Cancer Institute of Canada (NCIC) has shown that short-term preoperative radiation (25Gy over 5 days) results in a significant reduction in the local recurrence rate and improved disease-free survival for all stages of rectal cancer.

Current Recommendations for Chemoradiation in Rectal Cancer Patients after Radical Resection

Stage I	No adjuvant therapy
Stage II or III	Neoadjuvant chemoradiation for 5 weeks
Low/mid	5-FU based chemotherapy with XRT (180 cGy 5 days/week x 5

lesion	weeks)
	Rest for 6 weeks
	Total mesorectal excision
	Rest for 4 weeks
	Continue 5-FU–based chemotherapy for 8 weeks
High lesion	Preop or postop chemotherapy
	Total mesorectal excision
Stage IV	LAR or APR for palliation/prevention of obstruction or bleeding
	Adjuvant chemotherapy
	5-FU + leucovorin ± irinotecan or oxaliplatin with individualized XRT

(j) Empyema Thoracis

Answer.

Introduction: Empyema thoracis is the condition of pus formation in the pleural space.

Aetiology:

- The pleural effusion which accompanies infective conditions of the lung may itself become infected and form pus.
- A lung abscess may burst into the pleural space.
- Haemothorax may become infected.

Clinical features:

- The symptoms which the condition produces depend on the amount of pus present, and the degree of compression of, or disease in, the underlying lungs.
- There is some embarrassment of respiration, and 'swinging' fever, typically present whenever pus has accumulated in the body.
- Fever may sometimes be absent, especially if the patient is being treated with antibiotics.
- The patient with an empyema is severely toxic, looks ill, loses weight rapidly and becomes severely anaemic.
- If the pus is sufficiently thin to be removed through aneedle, then the treatment is entirely by aspiration.

Treatment:

- Penicillin or other antibiotic solution may be injected into the pleural space after the withdrawal of the pus.
- Aspiration needs to be repeated daily until lung expansion is adequate.

- If the pus becomes too thick for aspiration, the empyema is treated by one of two surgical methods:

(a) A small intercostal incision is made and a large self- retaining catheter placed into the pleural space to allow the escape of pus. The catheter is connected to an under-water seal. This method of intercostal drainage is rarely used but it is sometimes suitable for children.

(b) Rib resection and drainage by a wide-bore tube. Part of one rib is removed and the pleural space opened through itsperiosteum. This tube may be left open at its outer end or may be attached to an under-water seal. One type of tube is the 'Tudor- Edward' empyema tube which has an additional small rubber side tube through which the empyema cavity can be irrigated.