جامعة الملك سعود
King Saud University

Summary of

(Davidson's principles and practice of surgery)

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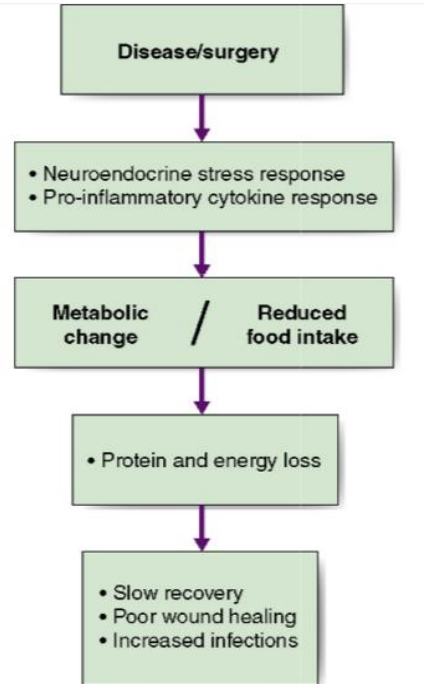
L1-Nutritional support in surgical patients



Introduction:

Nutritional disorders in surgical practice have two principal components. **First is** reduced food intake or problem in digestion like anorexia or short bowel syndrome. **The second is** metabolic change like in surgical and traumatic patients (increase demand).

Assessment of nutritional status

The main energy reserves in the body are found in subcutaneous and intraabdominal fat. The three key elements of nutritional assessment are: current food intake, levels of energy and protein reserves, and the patient's likely clinical course. This assessment is done to know if the patient needs more nutritional support.

SUMMARY BOX 3.2
Nutritional status

- Nutritional status in surgical patients may be adversely affected by starvation (effects of disease such as oesophageal cancer, restricted intake), the effects of inflammation (increased catabolism) and the effects of the operation itself (stress/inflammatory response)
- Nutritional status is assessed by current food intake, levels of reserves and likely clinical course.

Assessment of nutritional requirements:

The most common method for assessing protein/nitrogen requirement is based on body weight. Enteral diets will usually provide protein whereas parenteral nutrition provides the nitrogen (N) in the form of amino acids. Most patients will require 1gN per 200 kcal (typically 10gN but may reach 20g in critically ill patients).

TABLE 3.1 Estimation of energy and protein requirements in adult surgical patients

	Uncomplicated	Complicated/stressed
Energy (kcal/kg/day)	25	30–35
Protein (g/kg/day)*	1.0	1.3–1.5

Causes of inadequate intake:

Causes of inadequate intake are either problem in the food intake or digestion and absorption like 'intestinal failure'. Intestinal failure could be acute or chronic. Acute intestinal failure usually comes after abdominal surgery as a complication like intestinal

obstruction and paralytic ileus. Chronic intestinal failure may result from short bowel syndrome, following extensive small bowel resection or extensive small bowel disease, such as Crohn's disease. Patients with intestinal failure need more energy requirements and they have impaired ability to utilize administered nutrients.

Methods of providing nutritional support

Nutrients can be given via the gastrointestinal tract, i.e. enteral nutrition, or intravenously, i.e. parenteral nutrition. Parenteral nutrition is indicated only when enteral feeding is not feasible.

1-Enteral nutrition: Oral, nasogastric, nasojejunal tubes, gastrostomy or jejunostomy.

Many patients suffer from early satiety so oral supplements containing the energy requirements can be used (like protein supplements). If the patient is unconscious or cannot eat for any reason can be fed by nasogastric or nasojejunal tube. A patient with pseudobulbar palsy or an oesophageal fistula can be fed through a gastrostomy, and a patient with a gastric or duodenal fistula can be fed through a jejunostomy.

Complications of enteral nutrition:

Diarrhea is common with nasogastric feeding. Intraperitoneal sepsis could result from early accidental removal of a jejunostomy tube, with intraperitoneal leakage.

2-Parenteral nutrition:

Parenteral nutrition can provide the patient's total needs for protein, energy, electrolytes, trace metals and vitamins, i.e. total parenteral nutrition (TPN). The chief indication for TPN is intestinal failure. TPN should continue until intestinal function has recovered .The solutions contain fixed amounts of energy and nitrogen, and typically provide 1400–2400 kcal (50% glucose, 50% lipid) and 10–14 g nitrogen.

TPN solutions are typically very hypertonic and acidic so should be infused slowly using a vein with a high blood flow (like subclavian or jugular) to prevent thrombophlebitis and venous thrombosis or it could be infused indirectly via a peripherally inserted central (PIC) line.

For longer-term feeding, catheters are tunnelled subcutaneously to reduce the risk of infection. For very long term (including home) parenteral feeding a Hickman catheter is used.

Complications of TPN:

-Catheter problems: Percutaneous insertion of a catheter can cause pneumothorax, air embolus and haematoma.

- Thrombophlebitis: The signs are redness and tenderness over the cannulated vein. Occasionally, superior mediastinal syndrome develops in patients with superior vena cava thrombosis. If major vessel occlusion is suspected, the diagnosis is confirmed by venography and anticoagulation is commenced with heparin. Sometimes thrombolytics are used.

-Infection: The usual offending organisms are coagulase-negative staphylococci, Staphylococcus aureus and coliforms. The triad of central line, TPN and broad spectrum antibiotics predispose to systemic fungal infection.in addition, immunocompromized patients are more prone to fungal infections.

- Metabolic complications: overhydration in patients with co-existing medical conditions (e.g. cardiac failure). Hyperglycemia affects the liver function. Excessive administration of glucose may also aggravate respiratory failure. Hypokalaemia and hypophosphataemia are common when severely malnourished patients are re-fed after a long period of starvation which might lead to cardiac arrhythmia.

Monitoring of nutritional support:

Patients receiving nutritional support are monitored to detect deficiency states. Pulse rate, blood pressure and temperature are recorded regularly, urine is checked daily for glycosuria. Body weight is measured twice weekly. Serum urea and electrolytes are measured daily. Full blood count, liver function tests, and serum albumin, calcium, magnesium and phosphate are monitored once or twice weekly.

SUMMARY BOX 3.3

Enteral nutrition

- If patients cannot eat adequate amounts of food, they should be reviewed by the ward dietitian
- If oral supplements fail, a fine-bore tube can be used for supplemental or total enteral nutrition
- Most patients tolerate a whole-protein feed (1 kcal/ml), which can be escalated to 100 ml/hour and thus supply about 2400 kcal/day and 14 g nitrogen/day
- If a tube cannot be passed down the oesophagus, gastrostomy and jejunostomy feeding should be considered
- The main complications of enteral feeding relate to patient tolerance (nausea, vomiting and diarrhoea) and to the insertion site (gastrostomy or jejunostomy).

TABLE 3.4 Detection and treatment of catheter related sepsis

If a pyrexia > 38°C develops, or there is a further rise in temperature if already pyrexial
• Stop parenteral nutrition and check for other sources of pyrexia (e.g. chest or urinary tract infection) • Take peripheral and central line blood cultures • Administer intravenous fluids • Heparinize catheter • Consult senior medical staff
If blood culture is negative
• Restart parenteral nutrition and continue to monitor for signs of sepsis
If blood culture is positive
• Remove catheter and send tip for bacteriological analysis • Administer appropriate antibiotic therapy • If necessary, replace catheter and restart parenteral nutrition within 24–48 hours
Where central access must be preserved
• Seek specialist advice from hospital nutrition team

SUMMARY BOX 3.4

Parenteral nutrition

- Parenteral feeding is indicated if the patient cannot be fed adequately by the oral or enteral route
- The need to restrict volume when using total parenteral nutrition (TPN) means that concentrated solutions are used, which may be irritant and thrombogenic. TPN is therefore infused through a catheter in a high-flow vein (e.g. superior vena cava)
- TPN is usually given in an 'all-in-one' bag with a mixture of glucose, fat and L-amino acids combined with fluid, electrolytes, vitamins, minerals and trace elements
- The major complications with TPN can be classed as catheter-related, septic or metabolic. A multidisciplinary approach to the management of TPN patients by a nutrition team will minimize such complications.

Adrenal gland:

- **Surgical anatomy and development:**
  - Adrenal gland weighs approximately **4g** and lies immediately **above and medial to the kidneys.**
  - Right adrenal drains into **inferior vena cava.**
  - Left adrenal vein drains into the **left renal vein**
  - The glands are supplied by small vessels that arise from the **aorta, renal and inferior phrenic arteries.**
  - Each gland has an **outer cortex** and **inner medulla.**
  - Cortex is derived from **mesoderm** and medulla is derived from the **chromaffin ectodermal cells of the neural crest.**
  - The medulla is part of the sympathetic nervous system. Its APUD cells secrete the **catecholamines**, adrenaline (epinephrine), noradrenaline (norepinephrine) and dopamine, and are supplied by **preganglionic sympathetic nerves.**

❖ Adrenal Cortex:

- **Cortical function:**
  - Adrenal cortex has three zones:
  1. The outer **zona glomerulosa** secretes **mineralocorticoid, aldosterone.**
  2. The **zona fasciculata and zona reticularis** act as a functional unit and secrete:
     - **Glucocorticoids (cortisol and corticosterone)**
     - **Androgenic steroids (androstenedione, 11-hydroxy-androstenedione and testosterone)**
     - **Inactive androgen and oestrogen precursor, dehydroepiandrosterone sulphate (DHA-S).**
  - The hormones are circulate either **free (5%) or bound to α-globulin.**

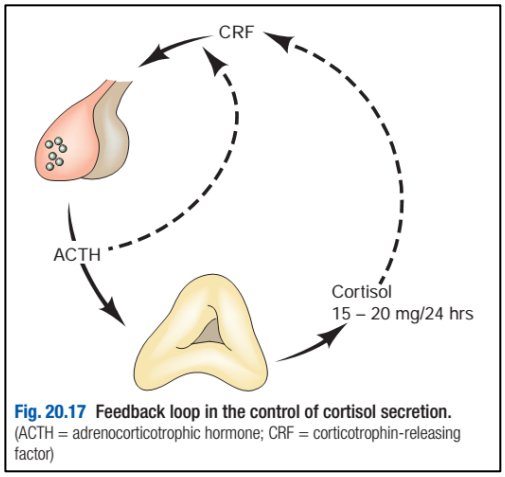


Fig. 20.17 Feedback loop in the control of cortisol secretion. (ACTH = adrenocorticotrophic hormone; CRF = corticotrophin-releasing factor)

SUMMARY BOX 20.5

Adrenocortical hormones

- Cortisol secretion is controlled by pituitary adrenocorticotrophic hormone (ACTH). Cortisol protects against stress, maintains blood pressure and aids recovery from injury/shock. Its metabolic activities include protein breakdown, increased gluconeogenesis, reduced glucose utilization and mobilization/redistribution of fat and water
- In excess, cortisol has mineralocorticoid activity, can cause psychosis, and has anti-inflammatory effects (used in transplantation immunosuppression)
- Aldosterone secretion is controlled mainly by angiotensin levels (and thus by renin release from the juxtaglomerular apparatus during decreased renal perfusion)
- Aldosterone conserves sodium (by facilitating its exchange for potassium and hydrogen ions in the kidney) and is a major determinant of extracellular fluid conservation
- Androgenic steroids and dehydroepiandrosterone sulphate (DHA-S) are also secreted by the adrenal cortex. DHA-S is converted to testosterone and oestrogen in fat and liver, and this peripheral aromatization is the main source of oestrogen in postmenopausal women.

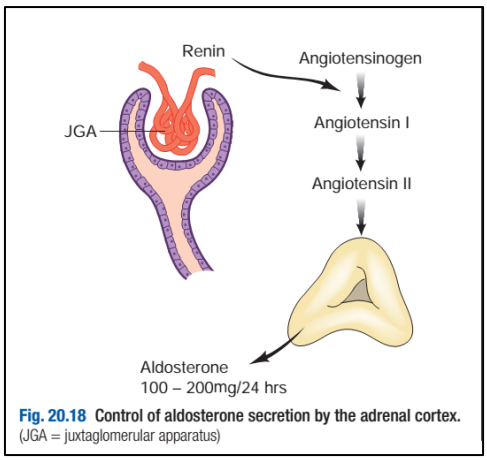


Fig. 20.18 Control of aldosterone secretion by the adrenal cortex. (JGA = juxtaglomerular apparatus)

- ## Cushing's syndrome:

It results from **any prolonged and inappropriate exposure to cortisol**

Causes:

- **Tumours of the adrenal cortex (20%):**

1 - Benign adenoma: (the most common adrenal cause of Cushing's syndrome)

- Unilateral and is more common in **females.**
- the tumour contains **clear cells** or **compact cells**
- Autonomous cortisol secretion **inhibits ACTH production**, so that the **contralateral gland becomes atrophic and ceases to function.**

2- Adrenal carcinoma: (is a rare cause of Cushing's syndrome)

- More frequently in **young adults and children.**
- The tumour grows to a **large size** and has **frequently metastasized** by the time of presentation.

- **Pituitary disease (80%):**
  - They usually **basophil** or **chromophobe** adenomas of **ACTH-secreting cells.**
  - Because of the continued ACTH secretion, both adrenals become **hyperplastic.**
  - When Cushing's syndrome is caused by a pituitary tumour, it is referred to as **Cushing's disease.**
- **Ectopic ACTH production:**

Inappropriate secretion of **ACTH-like peptide** by tumours of **non-pituitary origin** (e.g. pancreas, bronchus, thymus) is a rare cause.

- **Iatrogenic:**
  - Side effect of **therapeutic steroid use.**
  - **Adrenal atrophy** occurs if the steroid dosage is supraphysiological (**> 20 mg equivalent of prednisone per day**)

Clinical features:

The main clinical features are: truncal obesity, buffalo hump, mooning of the face ('tomato head, potato body,
four matchsticks as limbs'), thinning of the skin, livid striae and proximal muscle weakness

Investigations:

Before proceeding to **adrenalectomy,** the surgeon must be convinced that:

- **Cortisol secretion is beyond normal control:**
  1. Plasma cortisol levels are high
  2. Diurnal variation is lost
  3. Secretion is not suppressed by low-dose dexamethasone
- **The primary problem is in the adrenal:**
  1. ACTH cannot be detected in the plasma
  2. Urinary excretion of cortisol is not suppressed by high-dose dexamethasone
- **Pituitary and ectopic sources of excessive ACTH production have been excluded:**
  1. In pituitary adenoma, plasma ACTH levels are inappropriately

2. Urinary cortisol excretion is suppressed by dexamethasone.
3. In ectopic ACTH syndrome, the ACTH levels are often exceedingly high and there is an associated electrolyte disturbance.

- **Attempts have been made to localize the lesion by techniques such as CT or MRI.**

Management:

Adrenal adenoma:

An adrenal tumour is usually treated by **unilateral adrenalectomy**, but **cortisone replacement** is needed until
the suppressed contralateral adrenal recovers

Adrenal carcinoma

1. Adrenal carcinomas should be **completely removed.**
2. **Chemotherapy with mitotane or p-pDDD** may be tried, but this is a toxic drug and is often poorly

Pituitary disease

Pituitary disease is best treated by **pituitary surgery (or irradiation)** rather than by bilateral adrenalectomy. This
avoids continued growth of the pituitary tumour, problems due to ACTH and MSH production (pigmentation, Nelson's syndrome), and the risks of adrenalectomy.

• Hyperaldosteronism

Primary hyperaldosteronism (Conn's syndrome):

- Usually due to a benign adenoma and is most common in **young or middle-aged women.**
- The adenoma is:
  - small, single, canary yellow on bisection
  - Composed of **cells of the glomerulosa type.**

- The high circulating levels of aldosterone suppress renin secretion – a helpful biochemical diagnostic observation.

Clinical features

1. **Retention of sodium leads to:**

Hypertension, headaches and visual disturbance (although serious retinopathy is uncommon).

2. **Potassium loss leads to:**

Worsening hypokalaemia, episodes of muscle weakness and nocturnal polyuria.

Diagnosis

- **Low serum potassium in a hypertensive patient** should signal the possibility of hyperaldosteronism.
- **Confirm hypokalaemia:**
  By **repeated blood sampling** and **24-hour urine collections** usually show increased potassium excretion.
- **Demonstrate hypersecretion of aldosterone:**
  1. Plasma and/ or urinary aldosterone levels are measured at 4-hourly intervals to allow for diurnal variations.
  2. Giving the aldosterone antagonist, **spironolactone**, **should reduce blood pressure and reverse hypokalaemia**.
- **Exclude secondary hyperaldosteronism:**
  1. Renin levels are increased in secondary hyperaldosteronism but undetectable in the primary disease.
  2. Spironolactone causes further increases in renin levels in secondary hyperaldosteronism.
- **Localize the adenoma:**
  To localize the adenoma we have to use **CT or MRI,** failure to 'see' an adenoma mean there is no discrete tumour and that the patient has bilateral cortical hyperplasia.

Management

In Adenoma: removal of the affected gland after correcting the hypokalaemia with **oral potassium and spironolactone**.
In Adrenal hyperplasia: long-term drug treatment.

Secondary hyperaldosteronism:

Hyperaldosteronism is most commonly secondary to excessive renin secretion (and stimulation of the zona glomerulosa by angiotensin) in chronic liver, renal or cardiac disease

• Adrenogenital syndrome (adrenal virilism):

Pathophysiology

This syndrome is due to one of a number of genetically determined **enzyme defects that impair cortisol synthesis.**
The resultant **increase in pituitary ACTH production causes adrenal hyperplasia and inappropriate adrenal androgen secretion.**

Clinical features

The effects depend on the patient's sex and age:

- Female infants show **enlargement of the clitoris** and **varying fusion of the labial folds**.
- Young boys have **precocious isosexual puberty.**
- In both sexes, growth is at first rapid, but the **epiphyses fuse early so that the final height is stunted**.
- **Excess muscle growth** produces an **'infant Hercules' appearance.**
- Milder forms of the disease may affect older girls and cause **hirsutism and acne.**

Management

1. The patient is given **cortisol for replacement purposes** and **to suppress ACTH production**.
2. Surgical correction of the genital abnormality may be needed.

- ## Adrenal feminization

Exceptionally, a tumour of the adrenal cortex may secrete **oestrogens**. Such tumours are usually large and malignant.

- In the female, there is **sexual precocity**
- in the male, there is feminization, with **gynaecomastia, decreased libido and testicular atrophy.**

Treatment consists of **removing the tumour**, although recurrence and metastatic spread are common.

❖ Adrenal medulla

Pathophysiology

- The normal adrenal medulla secretes **catecholamines in the ratio 80% adrenaline to 20% noradrenaline.**
- It also secretes the **noradrenaline precursor, dopamine.**
- Small amounts of catecholamines are excreted in the urine in **free and conjugated form.**
- Larger amounts are excreted as **metnoradrenaline and 3-methoxy-4-hydroxymandelic acid (VMA).**

- ## Phaeochromocytoma

Pathology:

- Benign tumour of the adrenal medulla (80%) but 20% arise in extra-adrenal paraganglionic tissue.
- 10% are multiple and 10% are malignant
- May be associated with neurofibromatosis, medullary carcinoma of the thyroid (MEN II), Von Hipple–Lindau disease, duodenal ulcer and renal artery stenosis

Clinical features

Usually presents clinically with **paroxysmal hypertension**, which is often, and with metabolic effects such as
diabetes mellitus

Investigations

1. **All hypertensive patients < 40 years old** should be screened for phaeochromocytoma
2. Overnight or 24 hour urinary and plasma **metadrenaline and normetadrenaline levels**
3. Location is best defined by **CT and radiolabeled metaiodobenzylguanidine (MIBG) scanning**

Management

Treatment consists of **adrenalectomy** after careful preparation **to control blood pressure and heart rate and**
to re-expand blood volume (by α-adrenergic blockade with β-blockade).

❖ Non-endocrine adrenal medullary tumours

Ganglioneuromas

- **Pathology:** benign, firm, well-encapsulated tumours of **ganglion cells.**
- **Symptoms**: diarrhea
- **Treatment:** Surgical excision

Neuroblastomas

- **Pathology:** highly malignant tumours arising from **sympathetic nervous tissue.**
- **Epidemiology:** They are one of the most common malignant tumours of infancy and childhood
- **Treatment:** radical excision, radiotherapy and chemotherapy

Adrenal 'incidentaloma'

The increasing use of imaging modalities such as CT or MRI has led to adrenal tumours being discovered:

- If there is **no hyperfunction and the swelling is < 3.5cm in diameter** no further investigation needed.
  The lesion is likely to be a **benign non-functioning cortical adenoma.**
- Endocrine **hyperfunction, a swelling > 3.5cm** or the suspicion of malignancy is an indication for further assessment.

Adrenalectomy

Technique

- For benign tumors: **laparoscopically by the transperitoneal or posterior route.**
- For large malignant tumors: **flank incision, after removing a rib to allow access**.
- The open posterior approach through the bed of the 11th or 12th rib is technically more difficult, but has low morbidity
- The usual route is anteriorly, beneath the costal margins, transperitoneally with reflection of liver on the right and spleen, pancreas and colon on the left.
- The adrenal vein can often be divided early in laparoscopic surgery which, in phaeochromocytomas, prevents
  catecholamines from circulating, thereby reducing blood pressure swings following manipulation of the tumour.

Replacement therapy

- **After bilateral total adrenalectomy:** Corticosteroid replacement is needed for life.
- **After unilateral adrenalectomy:** may not be needed permanently
- **Replacement is best achieved by a combination of: oral hydrocortisone (30 mg daily in divided doses) and the mineralocorticoid fludrocortisone acetate (0.1 mg daily).**

What If both adrenals are removed and patient undergo surgery?

The operation must be covered by commencing steroid replacement at the time of surgery.
Adequacy of replacement is assessed by **serum levels and response to the dexamethasone test**.
Doses of hydrocortisone are given intravenously until the patient can take oral steroid.
If hypotension occurs, 100mg hydrocortisone is given immediately by intravenous injection, followed by 100mg every 6–8 hours All adrenalectomized patients must be warned to increase the dose of steroid if stress or infection occurs.

What if replacement fails?

Patient may precipitate **an 'adrenal crisis', with acute hypotension and collapse**. Such patients should carry **a 'steroid card'** giving details of dosage and possible complications, and should be able to recognize the symptoms of adrenal insufficiency (i.e. **loss of appetite, nausea, cramps, muscle pains and malaise**).
If such symptoms occur, the patient should take an **extra two tablets of hydrocortisone** and seek urgent medical help.

SUMMARY BOX **20.9**

Management of adrenal pathologies

- Hyperfunctioning benign adrenal masses should be removed surgically (phaeochromocytoma, cortisol secreting adenomas and Conn's syndrome). The laparoscopic approach is the preferred one
- Careful preoperative assessment to exclude multiple hormone secretions from a single or bilateral adrenal masses must be undertaken (e.g. a phaeochromocytoma that is also secreting cortisol)
- In primary hyperaldosteronism (Conn's syndrome) preoperative selective venous sampling from both adrenals should be considered to confirm the site of maximal secretion. It can be misleading to assume this will be the side with the mass lesion in it
- Incidentally found adrenal masses must be investigated for possible hypersecretion of all adrenal hormones prior to their removal or a decision to leave them in situ and follow up.
- Have a low threshold to remove non-functioning incidentalomas > 3.5 cm in size, as the incidence of adrenocortical carcinomas increases significantly above this size
- Only biopsy an adrenal mass if you think it is due to a metastasis from a previous known malignancy. Always exclude a phaeochromocytoma before biopsy of any adrenal mass.

British Association of Endocrine and Thyroid Surgeons' Guidelines at www.BAETS.ORG.UK

The Biology of Cancer:

- Transferred cells that don't respond in a normal way to growth regulatory system. (I.e. **imbalance between cell replication and cell death**)
- Cellular insult → DNA mutation → Cancerous cell.
- Changes within the cellular genome occurs frequently and don't necessarily develop cancer due to protective mechanisms.
- **Two types of cancer:**
  A) **Benign** → non-invasive.
  B) **Malignant** → It is invasive because of accumulation of mutations and its cells are pluripotent (i.e. has the ability to give rise to more than one cell type)
     Pluripotent cells **form**:
     I) Epithelium.
     II) Stroma.
     III) Vascular components.
- **Invasion**: replacement of normal tissue, destroying supporting structures & disrupt the function.
  The mechanism that controls invasion:
  1) Increase local pressure from the expanding tumor.
  2) Increase motility of cancer cells. (**Local invasion**)
  3) Malignant cells release MMPs to degrade the extracellular matrix.
- **Metastasis:** The spread of the cancerous cells to different distant tissue.
  Metastasis Signature: Further mutations of the cancer cells to occur.
  Some tumors metastasize earlier than others & this depends on the tissue of origin, genotype, phenotype.
  Clumps of cancer cells can travel to distant tissues, and settle to those organs with a fine capillary bed (e.g. Liver, Lungs).
- Cancer cells can spread **prostaglandins** to enhance the deposition on bones.
- **The survival of metastatic deposits depends on angiogenesis**:
  A) Angiogenesis natural inhibitors → Angiostatin, Endostatin.
  B) Angiogenesis natural enhancers → VEGF, Fibroblast Growth Factor.
- ¾ of the lifespan of the tumor is spent pre-clinically, and the clinical manifestation of the disease is limited in the last ¼.
- Total cure and eradication of a tumor is rare.
- **Evidence of cure is usually enough which is normal duration of life of a patient without further clinical evidence.**
- The management of patient with cancer has the following goals:
  A) Prevention.
  B) Cure. (Locally advance disease, or metastasize decrease the chance of cure)
  C) Palliation → Manage the symptoms if there no chance of survival.

- **Screening:** **Detecting being lesion with malignant potential, pre-invasive cancer & invasive malignancy before it becomes symptomatic.**

  For the screening to be more effective:

  A) Be sensitivity.
  B) Be specificity.
  C) Be acceptable.
  D) Detect cancer at a stage when early treatment is beneficial.
  E) Be cost-effective.

  It is appreciable to screen for inherited diseases.

The Surgical Management of a cancer patient: Radical Surgery for curing, or palliative to relieve distressing symptoms.

- **Symptoms of cancer:**

A) **Local effects:** Depends on the organ & the behavior of a tumor. Also it could mimic the pain of a benign disease (e.g. peptic ulcer)

B) **Systemic effects:**

I) Weight loss.

II) **Cancer cachexia** → patients appear to die of starvation and clinically characterized by anorexia, severe weight loss, lethargy, anemia and edema.

III) Some cancers (neuroendocrine) produce ectopic secretions and give endocrine syndrome that disrupt the hormonal system.

IV) **Paraneoplastic syndrome:** signs or symptoms occurring as result of antibodies by the cancer and are not due directly to local effects of the cancer cells. Example is myasthenia gravis secondary to a tumor of the thymus.

- **Investigations serve two main purposes:**

1) Confirmation by histopathology or cytology.
2) Assessment of the local invasion and metastasis.

Diagnostic Investigations:

1) Blood sample (tumor markers)
2) Imaging.
3) Histopathology.

In general, a **treatment plan** for a cancer patient **cannot be done** until confirmation by histopathology **(best modality of staging)**. One of the exceptions is pancreatic cancer. Staging depends on the **site** of the primary cancer and the relevant common sites of **metastases**.

Local invasion can be assessed by:

A) Endoscopy.
B) CT.
C) MRI.

Metastatic spread assessed by:

A) CT.
B) PET.
C) Laparoscopy.

- **Staging:** **The aim of staging is assessing therapeutic plan and prognosis.**

TNM is the system of staging in which; T is the extent of the tumor; N lymph node involvement; M metastasis.

- **Management:**
  A) **Benign:** local excision with sufficient surrounding tissue.
  B) **Malignant:** A radical surgery; complete removal of the tumor with the surrounding normal tissue.

  En Bloc Resection: Local resection of the tumor with local lymph nodes.
  Sometimes lymph nodes are resected to identify the stage of the tumor.
  # Complete Radical Excision when there's no lymph node involvement carries a high chance of curability.
  During any operation of cancer, try to **avoid any spillage of malignant cells** which may cause cancer recurrence, so in some sites it is usual to ligate the main vessels draining the area before tumor is mobilized.
- **Adjuvant Therapy:** Used when it is not possible to remove all the local disease & there was a metastasis. And it provides local and systemic control.
  Chemotherapy: may help to prevent local recurrence & distance metastasis. It mostly used in breast & colorectal cancer with lymph node involvement.
- Sometimes, radical operation is impossible and the surgical reduction of the tumor is a help for the systemic therapy, which is aimed to control the disease as a whole.
- In general, drugs are given in combination over a **period of 6-12 months.**
- Toxicity →Diarrhea, mouth ulcers, weakness, alopecia.

- **Radiotherapy:** is localized so it tries to prevent local recurrence.
- Could be given prior to surgery to shrink the tumor down.
- Could be given postoperatively in whom the chance of recurrence is high.
- Can reduce the need for radical surgery in those with radiosensitive tumors.
- The impact of radio- chemotherapy on the growth of the children can be significant which has to be balance with the potential of cure.

- **Neoadjuvant Therapy:** Use of therapeutic modalities such as chemotherapy, hormonal or immune prior to definitive treatment of the locally advanced disease by surgery or radiotherapy.
- Its aim is to reduce tumor size, extent and burden. Also to allow a more curative excision with disease-free margins.
- Patients with metastases can be offered surgical treatment such as liver or lung resection followed by second- and third-line chemotherapy.
- Follow-ups for the next months after surgery is extensive to check if there's any recurrence & the patient is asymptomatic.
- Palliation of advanced disease:
- **The management of patients with incurable disease involves the relief of distressing symptoms.**
- Effective palliation therapy can be achieved by local and systemic adjuvant therapy.
- Medical treatments are used to treat pain, nausea, depression, infection etc.
- **The aim of pain management is to achieve complete analgesia without disturbing mental clarity.**

Peripheral nerve lesions

Lesions of the peripheral nerves can be classified as: traumatic, compressive (common), metabolic, inflammatory, autoimmune, neoplastic and genetic. The common compressive neuropathies are carpal tunnel syndrome, ulnar nerve compression at the elbow and meralgia paraesthetica.

Carpal tunnel syndrome

The syndrome consists of symptoms of pain and numbness in the distribution of the median nerve in the hand. It is more common in patients with diabetes, hypothyroidism, acromegaly and pregnancy. Symptoms may be intermittent, are usually worst at night, and may be relieved by shaking the hand while holding it in a dependent position. The symptoms are often provoked by wrist flexion. On examination, there are usually no signs. Occasionally, there may be wasting of the thenar eminence, weakness of the abductor pollicis brevis, and diminished or altered sensation in the median nerve distribution. Tapping over the nerve in the carpal tunnel may elicit paraesthesia in the median nerve distribution (Tinel's sign). Phalen's test involves acutely flexing the wrist and holding it in this position. This may precipitate paraesthesia or numbness, and this is abnormal if it occurs within 1 minute. The diagnosis can be confirmed using electrophysiology to measure nerve conduction velocity and distal motor latency. Treatment depends on severity of symptoms. Splinting the wrist or injections of steroid into the carpal tunnel provide relief in a third of cases. If this fails, the transverse carpal ligament can be divided surgically, and in many cases this can be performed as a day case under local anaesthetic.

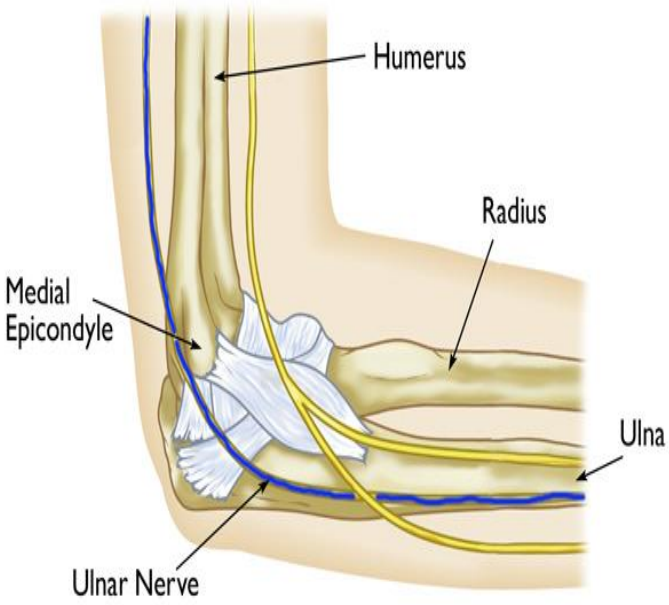
Carpal tunnel syndrome

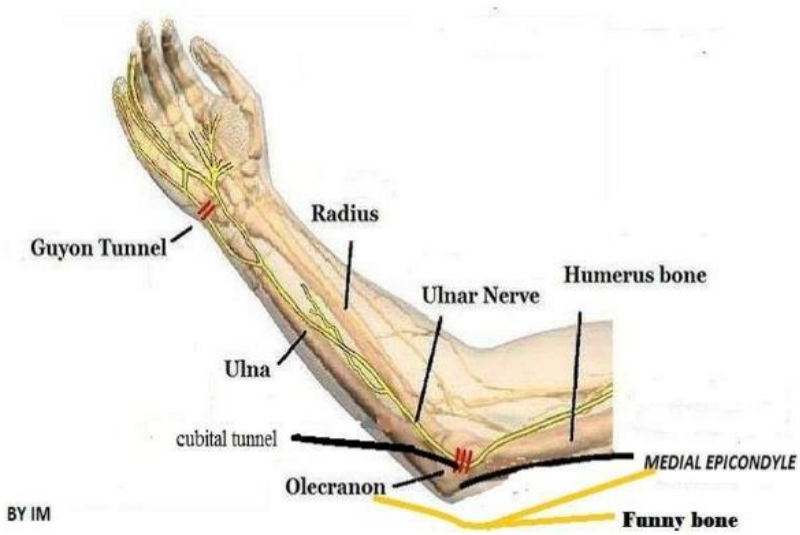
Ulnar nerve compression at the elbow

This is usually due to acute and chronic trauma, osteoarthritis or rheumatoid arthritis. The nerve may suffer repeated dislocation over the medial epicondyle on flexion of the elbow. Sometimes, the nerve may be compressed by the aponeurosis between the two heads of flexor carpi ulnaris. There is pain in the forearm and wasting of the small muscles of the hand, leading in the worst cases to an ulnar 'claw' hand. There may be reduced sensation in the ulnar distribution of the hand. The diagnosis may be made clinically, but electrophysiology is recommended to confirm the diagnosis. Treatment consists of surgically releasing and decompressing the nerve.

Meralgia paraesthetica

This is numbness and painful paraesthesia in the lateral thigh caused by compression or injury of the L2/3 sensory lateral cutaneous nerve. The nerve emerges from the lateral border of the psoas muscle just above the iliac crest and crosses the iliacus to pass beneath or through the inguinal ligament, 1 cm medial to the anterior superior iliac spine, to pass into the thigh. Seat belts, pregnancy, trauma and postsurgical scar tissue, to name but a few, can cause mechanical compression. Diabetes is present in up to 10% of cases. The clinical diagnosis can be confirmed by injecting local anaesthetic into the inguinal region 1 cm medial to the anterior superior iliac spine. Treatment includes weight loss, the removal of constricting clothes and belts, nonsteroidal anti-inflammatory drugs, ice packs and injections of corticosteroid. Most cases will settle within 2 years. Surgical decompression is reserved for those that do not.





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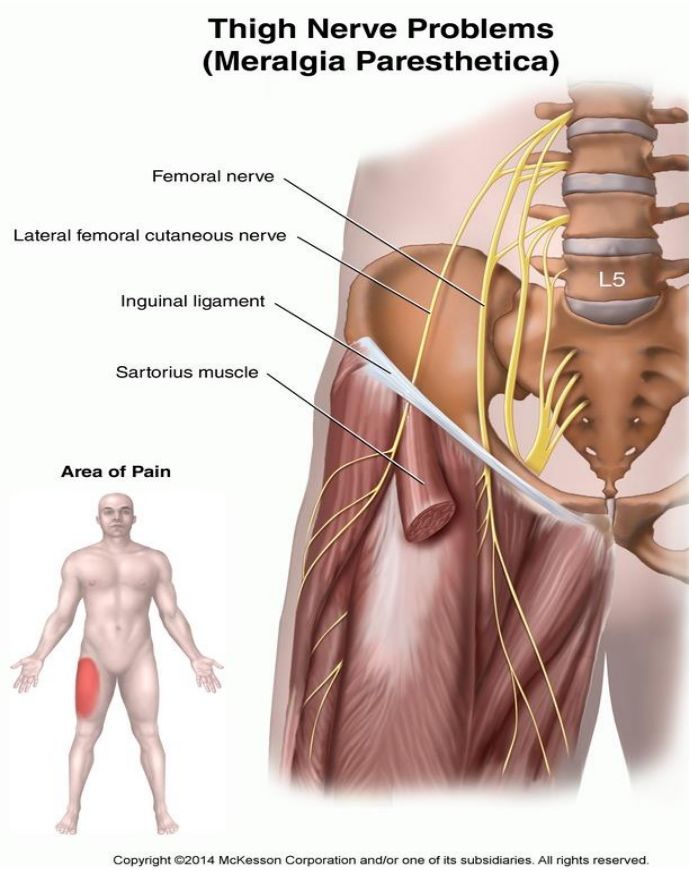
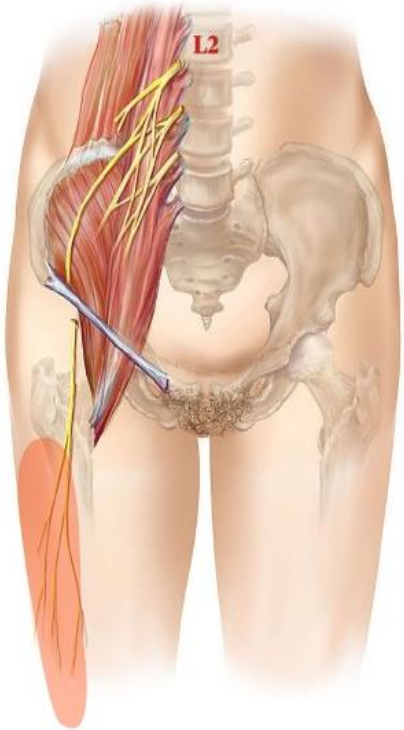




Lateral femoral cutaneous nerve innervation (L2,3)

- Sensory innervation to anterolateral thigh
- Injury may result in a severe burning sensation along the anterolateral aspect of the thigh

Skin and Soft Tissue Tumors:

First) Skin Tumors: Arise form epidermal OR Dermal layers.

1- Benign Lesions.

(A) Papilloma	**(I) Infective warts:** arise from basal cells & caused by viral infection. Common in immunocompromised patients. - **Site:** Hands & Fingers. - **Shape:** Greyish-brown, round elevated lesion with filiform surface and keratinized projections. - **Management:** Acetic acid OR freezing (liquid nitrogen or CO2 snow) - **Verruca Plantaris:** Plantar warts, acquired in swimming pools & showers. Found under the heel OR metatarsal head. They are Painful and managed with Curettage OR freezing. - Infective warts in the perineum & on penis are due to sexual intercourse & associated with STDs.
	(II) Senile warts (**Seborrheic Keratosis**: Basal cell papilloma, common in elderly. - **Site:** Upper back or trunk. - **Shape:** Yellowish-brown or black greasy plaque with cracked surface. - **Management:** Curettage.
	(III) Pedunculated Papilloma: Non-infective. - **Shape:** Fresh-colored spherical warty mass. - **Management:** **Small** → Grasped by forceps and cutting with scissors. **Large** → Resection of the papilloma and its pedicles.
(B) Dermatofibroma	- Also called histocytoma OR Sclerosing Hemangioma. - **Site:** Hands and feet. - **Shape:** Firm to hard nodules, brownish in color with rough surface. - **Managements:** Surgical Excision.
(C) Keratoacanthoma (Molluscom Sebaceum)	- Found in those over 50 years. Histologically, it resembles Squamous Cell Carcinoma (the distinction is by history, Squamous cell carcinoma grows slower than keratoachanthoma.) - Grows rapidly over 4-6 weeks & then **involutes.** - **Site:** Face. - **Shape:** Hemispherical nodule with red center crusted with keratin. - **Management:** Healed after shedding its center core OR it can be eradicated by curettage.
(D) Dermatofibrosarcoma Protruberans	- Rare, rarely metastatic, slow, locally invasive of the dermis. Arising from fibroblasts. - **Site:** Trunk. - **Shape:** Solitary multinodular mass which is firm, painless & red or bluish in color. - **Management:** Wide excisional + skin cover + radiotherapy.
(E) Benign Pigmented Mole	- **Defined as:** Migration of mixture of melanocytes to the dermis or epidermis. - The naevus cells cause pigmented spots or swellings (**naevi**) based on their sites or activity. - Moles showing at the junction of dermis and epidermis (junctional change) are common in children. **All mole of palms and soles are of this type.** - **Dermal naevus:** Migration of naevus cells to the dermis. - **Compound naevus:** migration of naevus cells to dermis and epidermis.
	(I) Common moles: Active growth in childhood because of junctional activity. - **Shape:** flat or slightly elevated brown black lesion covered by normal layer of epidermis. - **Management**: symptomatic, or growth after puberty requires removal. **(II) Giant Hairy Naevus:** Present at birth. Keep under observation for malignancy. - **Site:** trunk or face. - **Management:** Excision for cosmetic indications. **(III) Blue naevus:** Intradermal lesion. **(IV) Halo naevus:** Surrounded by a white circle of depigmentation with lymphocytic infiltration.

2- Premalignant lesions:

A- Actinic (solar) kartosis:	B- Bowen's Disease:	C- Erythroplasia of Querat:	D- Marjolin's Ulcer:
- **Site:** face, back of the neck, hands. - **Shape:** small, single or multiple, firm warty spots. - Common in elderly. - The scaly lesion drops off periodically to leave a premalignant ulcer. - Biopsy to exclude malignancy. - **Management:** Freezing.	- **Site:** Exposed areas. - **Shape:** elevated red scaling plaque, single or multiple. - They enlarge, & thicken & may transform into squamous cell carcinoma. - Biopsy is needed. - **Management**: Excision, curettage & cautery, cryotherapy, radiotherapy or topical 5-flurouracil.	- **Site: It's Bowen's disease** affecting the glans of penis or vulva. - **Shape:** Red, velvety patch. - **Management:** Excision. - If left untreated it'll cause an invasive carcinoma.	- **Site:** Develops in chronic non-healing burn. - Nerves are destroyed, due to extensive scarring, making this lesion painful. - **Management:** excision with wide margins.

3- Malignant lesions:

(A) Cancer of epidermis	- Occurs primarily in exposed areas. - Risk factors: Albinos & patients with Xeroderma Pigmentosa, radiation, irritative chemicals, chronic ulceration.
	(I) Basal Cell Carcinoma (Rodent Ulcer): extremely radiosensitive. - **Most common malignancy arising from the skin and seen in the elderly.** - Slow growing, locally invasive & **never metastasize**; possibly because of the large malignant cells. - **Site:** middle third of the face, node, eyelids, forehead, and inner canthus of the eye. - **Early shape:** Hard pearly nodule, dimpled in its center, & covered by telangiectastic skin, and the ulcer has rolled edges. - Clinical types are: cystic, nodular, sclerosing, morphoeic, centrally healing and 'field fire'. - The rodent ulcer repeatedly scales over & breaks down. - Biopsy is needed. - **Management:** 1) Surgical excision with adequate margins or radiotherapy. (the latter is contraindicated iif the lesion was close to the eye or a cartilage) 2) Small lesions could be managed by cryosurgery, curettage or cautery. 3) Morph's micrographic surgery is useful in excising lesions which are recurrent, or close to important structures. 4) Complex reconstructive surgery may be needed to restore structure or function for patients who present late.
	(II) Squamous Cell Carcinoma: Arise from Stratum Spinosum of the epidermis. - Less common but more aggressive. - **Site:** commonly in areas of epithelial hyperplasia or keratosis. - **Shape:** Hard, erythematous nodule, proliferates to form cauliflower-like growth or ulcerates to form malignant ulcer. - Regional lymph nodes can be involved. - **Management:** 1) Surgery or radiotherapy, depends on the tumor site, size and aggressiveness. 2) Palpable lymph nodes require regional lymphadenectomy by block dissection. 3) Adjuvant radiotherapy may be require if histopathology shows extracapsular spread.

(B) **Malignant Melanoma**	- **The most lethal of all skin cancers.** - Usually affects the depigmented areas; soles, palms and mucosa. - Risk factors: Sunlight exposure, females, increase the number of naevi in the body (average number is 14 for any individual, the higher the number the higher the risk.) - The essential feature of melanoma is the invasion of the dermis by proliferating melanocytes with large nuclei and prominent nucleoli and frequent mitosis.
	(I) Hutchson's Melanotic Freckle (Lentigo Maligna): Vertical extension of melanocytes into the dermis. - Arise from **senile freckle.** - **Site:** Face. - **Shape:** brown-red patch (1st sign of malignancy), serrated edge with abrupt margins with normal skin with kaleidoscopic pigmentation of the surface. - Premalignant phase lasts (10-15) years.
	(II) Superficial Spreading Melanoma: - **The most common type of malignant melanoma.** - **Site:** Trunk and exposed parts. - Most common in middle age. - **Horizontal Growth Phase in The Epidermis.** Pre-invasive phase (1-2) years - Shape: elevated surface, indistinct outlines with patchy pigmentations & wide range of colors. - **Vertical Growth Phase; invasion of the dermis**, the lesion produces indurated nodule, which soon ulcerates & bleeds.
	(III) Nodular Melanoma: - **Site:** More common in legs, or pre-existing benign naevus. - **Shape:** Elevated, deeply pigmented melanoma. - **Invade vertically from the start.** - No surrounding intra-epidermal macule. - Destruction of normal skin line → spread. - Active lesion darkens the overlying skin and becomes jet black and glossy. - Neglected lesion → satellite nodules around it. - Crusting, scab formation, itching, irritation and ulceration are typical.
	(IV) Amelanotic Melanomas: Pale pink lesion that grows rapidly.
	(V) Acral Lentiginous Melanoma: Occurs in Soles and Palms.
	(VI) Subungal Melanoma: - **Site:** Thumb or great toe. - Causes chronic inflammation beneath the nails. - Could be misdiagnosed with paronychia or ingrowing toenail.

Spread of Malignant melanoma: Through lymphatics or bloodstream.

- **Transient Metastases** → formation of painless, discolored nodules in the line of lymphatics between the primary & the regional lymph nodes.
- **Lymph node metastases** → Firm enlargement.
- Blood-borne metastases → brain, liver, lungs, skin and subcutaneous tissue.

Table 18.6 Prognosis in relation to the stage and depth of malignant melanoma

Clinical stage	5-year survival rate (%)
I Primary lesion only	
Breslow depth (mm)	70
< 1.5	93
1.5–3.5	60
> 3.5	48
II Primary lesion + regional lymph node or satellite deposit	30
III Metastatic disease	0

Clinical and pathological staging:

- For **stage I** (look at the 1st table on the right) **the most accurate prognostic factor is the depth of the lesion**.
- Measuring of the depth:
  1) **Clark:** by reference to the normal layers of the skin.
  2) **Breslow:** by micrometer gauge. (preferred)
- Melatonic freckles & superficial spread melanoma have better prognosis than nodular melanoma.

Management of Melanoma:

- Excision biopsy for diagnosis.
- The depth and the stage are assessed to define the treatment plan.
- Small pigmented lesions are excised with 3mm of margins of normal skin.
- Surgical excision for Stage I.
- Small defects are closed with primarily.
- Large defects need split-skin graft, or flap.
- Block dissection of the **nodes** is preferred if they are involved.
- Recurrence disease in a single limb → isolated limb profusion with cytotoxic drugs.
- Positive sentinel **node**? → Radical node dissection.

Melanoma TNM Classification

T classification	Thickness	Ulceration Status/Mitoses
Tis	N/A	N/A
T1	≤ 1.0 mm	a: w/o ulceration and mitosis <1/mm²
		b: with ulceration or mitoses ≥ 1/mm²
T2	1.01 - 2.0 mm	a: w/o ulceration
		b: with ulceration
T3	2.01 - 4.0 mm	a: w/o ulceration
		b: with ulceration
T4	> 4.0 mm	a: w/o ulceration
		b: with ulceration
N classification	**# of Metastatic Nodes**	**Nodal Metastatic Mass**
N0	0 nodes	N/A
N1	1 node	a: micrometastasis*
		b: macrometastasis**
N2	2-3 nodes	a: micrometastasis*
		b: macrometastasis**
		c: in-transit met(s)/satellite(s) without metastatic nodes
N3	4 or more metastatic nodes, or matted nodes, or in-transit met(s)/satellite(s) with metastatic node(s)	
M classification	**Site**	**Serum LDH**
M0	0 sites	N/A
M1a	Distant skin, subcutaneous, or nodal mets	Normal
M1b	Lung metastases	Normal
M1c	All other visceral metastases	Normal
	Any distant metastasis	Elevated

*Micrometastases are diagnosed after sentinel lymph node biopsy and completion lymphadenectomy (if performed).

**Macrometastases are defined as clinically detectable nodal metastases confirmed by therapeutic lymphadenectomy or when nodal metastasis exhibits gross extracapsular extension.

Second) Vascular Neoplasm. (Hemangiomas)

- **They are based on their clinical behavior into:**

<table>
<tr><td>1) Involuting Hemangiomas</td><td>2) Non-involuting Hemangiomas: Due to abnormal blood vessels formation.</td></tr>
<tr><td>- Arise from endothelial cells.
- Appear within weeks or at birth.
- Site: Affect the neck or head.
- Shape: Superficial Involuting hemangiomas form a bright-red elevated mass with irregular bosselated surface (Strawberry naevus).
Deeper lesions form a blue-black tumor.
- Active growth for 6 months, static until 2-3 years then starts to shrink & lose its color.
- Disappear before the age of 7.
- Complications: surface ulceration leads to bleeding, and Kasabach-Merritt Syndrome; consumption of coagulopathy through sequestration of platelets and fibrinogen.)
- Salmon Patch (Stork bites) present at birth over the forehead in the midline & the back of the neck. Involute at the age of 7.
- Management: left alone unless it involves periorbital (amblyopia, anisometropia).
Laser photocoagulation + intralesional steroids.</td><td>- Site: Overlies the distribution of peripheral nerves of the face of scalp.
- Shape: Bright-red to deep crimson, thick surface with polypoidal outgrowth.
- Management: CO2 laser therapy. Only for cosmetic reasons because it doesn’t overgrowth or involute.

Cavernous Hemangiomas:
- Shape: Elevated bluish-purple in children.
- Empties on pressure and refill.
- Histologically → mature vein-like structure.
- Management: Excision.

- Cirsoid aneurysm is a rare lesion in which it fed directly by arterial blood and becomes tortuous, dilated and pulsating. Managed with angiographic embolization, ligation of the feeding vessels and excision of this lesion.</td></tr>
<tr><td colspan="2">3) Other vascular lesions of the skin:
- Angiokeratoma → Subepidermal vascular malformation covered by hyperkeratotic epidermis. Appears as a red papule.
- Senile angioma → Cherry angioma, smooth, doom shaped, cherry red to purple lesion. Doesn’t require any treatment.
- Glomus Cell Tumor → benign tumor arising from glomus cells, causes severe pain and requires removal.</td></tr>
</table>

Third) Tumors of the Nerves:

1) Neurilemmoma	2) Neurofibroma: Hematoma of nerve tissue.
- **Site:** Nick & Limbs. - **Shape:** Encapsulated solitary benign tumor arise from the schwann cells & form subcutaneous swellings in the nerve course. - **Causes radiating pain.** - No malignant potential. - **Management:** Excision, but can result in loss of function.	- Solitary, but multiple in **Von Recklinghausen’s** disease (neurofibromatosis). - **Café au leit spots:** multiple dermal and subcutaneous nodules arise from peripheral nerves. - Can cause bony deformities, especially in the spine. - Could transform into neurofibrosarcoma.

Fourth) Tumors of Muscles and Connective tissue:

1) Lipoma: benign slow growing of fatty tissue.

- **Site:** arise betwee the skin and deep fascia, or fat in the intramuscular septa which becomes more prominent when the related muscle contracts.
- **Shape:** lobulated, soft mass, enclosed by a thin fibrous capsule.
- **Management:** Large or symptomatic → excision or liposuction.

2) Liposarcoma: the most common sarcoma in middle age.

- **Site:** Retroperitoneum and legs.
- **Management:** wide surgical excision.

3) Fibrosarcoma:

- **Site:** Lower limb & buttocks.
- **Shape:** large, deep firm mass.
- **Management:** wide surgical excision (initially). If recurred start radiotherapy.

4) Rhabdomyosarcoma: Highly malignant, more common in children

- **Site:** Striated muscle.
- **Shape:** Greyish-pink, soft, fleshy lobulated.
- **Management:** Radical excision with radiotherapy or limb amputation.

5) Kaposi's Sarcoma: tumor of the capillary & perivascular connective tissue.

- **Two clinical subtypes:**
  A) Endemic (slow growing)
  B) Association with aids or immunocompromised patients (painful, fast growing)
- **Site:** The legs (mostly), if aggressive it'll involve hands, ears and nose.
- **Shape:** Mauve or purplish-red nodules and plaques & brownish macules.
- **Management:** Radiotherapy and systemic immunotherapy for metastatic and multifocal.

Superfascial Lumps

Benign skin tumours

1. Papilloma (wart):

- Finger like projection, Painful.
- Usually infective (papilloma virus).
- Plantar warts (verruca plantaris) are particularly troublesome infective warts acquired in swimming pools and showers. They are found under the heel and metatarsal heads. They are flush with the surface and may be intensely painful.
- Rx: Cauterization (small or multiple), Excision (large or sessile)

2. Hyper trophic scar	3. Keloid
- Excessive fibrous tissue in a scar. - Confined to the scar. - No neovascularization. - Clinically: non-tender swelling with no itching. - It may regress gradually in six months. - Resolution can be hastened by elastic pressure garments, steroid injections or the application of silicone gel. These scars should not be excised.	-Excessive fibrous and collagen tissue with neovascular proliferation in a scar. - usually extends beyond the original scar -Initially raised, pink, tender, itchy and may ulcerate. - They are most likely to occur across the upper chest, shoulders and earlobes, and are common in black patients. -Rx: Injection (hyaluronidase, steroids etc.) Excision (Keloids can recur after excision) & grafting.

4. Pyogenic granuloma

-Excessive granulation tissue growth **in ulcers**.
-Red selling that bleed on touch, (Recurrent bleeding when exposed to Trauma).
-Rx: Cauterization (if small), Excision (if large).

5. Haemangioma

- Developmental malformation of blood vessels rather than a tumor.
- It commonly occurs in skin & sub cutaneous tissue but other organs e.g. lips, tongue, liver, brain may be affected.

Malignant skin tumors

1. Basal cell carcinoma (BCC)	2. Squamous cell carcinoma (Epithelioma)
- Middle aged white tropical males (high UV light exposure). - Common in the face. (Triangle of face: nose, forehead, and eyelids) -Slowly growing tumor. Clinically: - Rolled-in edges (inverted) with attempts of healing (shows unhealthy granulation). -Spreads locally (usually no Lymph Nodes metastases). -Rx: Radiotherapy & surgery	-Arise from squamous cell layer of skin or mucus membrane; it may arise from metaplasia of columnar epithelium (Due to chronic irritation). -Male>Female. - More malignant and rapidly growing than BCC. Clinically: -Edges are rolled out (everted) -Spreads: Locally, L.N, and blood. -Rx: Radiotherapy & Surgery

3. Naevus (mole)

- Includes moles & birth marks, they may present at birth, or even later.
- **Evidences of malignant change:** Increase in size, Change to irregular edge, Change in thickness , Change in color , Change in surrounding tissue , Symptoms e.g.: itching, bleeding discharge , Lymphadenopathy and microscopic evidence.

4. Marjolin ulcer

- It is a low grade squamous cell carcinoma.
- **Arising in chronically inflammed ulcers or scars (long standing scar).**
- Rx: Radiotherapy & Surgery

5. Malignant Melanoma

- It a rare but **most rapidly infiltrating skin tumor.**
- De-novo (10 %) , Pre-existing naevus (90 %).
- Metastasis: ● Local & satellite nodules. ●Lymphatic. (early metastasis to LN). ●Blood (liver, lung, bone etc.).

▪ Skin Cysts

Implantation Dermoid	Sebaceous Cyst
-It is a post traumatic dermoid (found at sites of injury). -Commonly in fingers and hands of farmers & taylors. -Clinically: -Tense, may be hard tender swelling, attached to skin. -Contains desquamated epithelial cells. -Rx: Excision is curative.	-It is a retention cyst due to blockage of its duct. -Lined by squamous epithelium and contains sebum and Spherical, attached to skin with punctum (very diagnostic). that may discharge sebum upon squeezing. - Contain cheesy white epithelial debris and sebum. - Transillumination test is negative. -Commonly in scalp, Face, scrotum and vulva (never in palm & sole). - Rx : ● Excision→(un infected cyst) ● Drainage followed by excision→(infected s/c).

▪ Subcutaneous Lumps (Cystic swellings)

1. Dermoid cyst: Clinically four varaieties: Sequestration dermoid, Implantation drmoid, Tubulo-dermoid and Terato-dermoid.

▪ Sequestration dermoid	▪ Tubulo-dermoid	▪ Teratomatous dermoid
-It is a true congenital cyst. -Ectodermal tissue buried in mesoderm forming a cyst, contains paste-like desquamated epith. -Common at lines of Embryonic fusion sites: 1.Midline: neck & root of nose 2.Scalp. 3.Inner or outer angles of eyes. Clinically: -Painless, not attached to skin, no punctum, not compressible , Cough impulse and bone indentation (scalp) and transillumination test is negative. - External angular dermoid is the most common congenital dermoid cyst and lies at the junction of the outer and upper margins of the orbit, in the line of fusion of the maxilla and frontal bones.	-E.g: Thyrpglossal cyst (remnant of thyroglossl duct). ▪ Most common midline neck swelling and usually presents as a painless, rounded cystic lump, which moves on swallowing or protruding the tongue.	- Cystic swelling arising from the totipotent cells . -They usually contain derivatives of mesoderm (cartilage, bone, hair, cheasy material).

2. Cystic hygroma

- A congenital malformation affecting lymphatic channels.

Clinically:

-Appears early, multilocular, **filled with clear fluid (transillumination +ve)**

Clinically:

- Common in: neck, axilla, groin, medistinum and tongue.

3. Branchial cyst

- A congenital cyst in persistent cervical sinus (located below angle of mandible, behind mid sternocleomastoid muscle).

Clinically:

-Tense, distinct edges, +ve flyctuation and –ve transillumination.

- **Contains cholestrol crystals (diagnostic).**

4. Ganglion

-It a cystic swelling **of synovial membrane of tendon or capsule in small joints**.
- Myxomatous degeneration.
- Common sites: Dorsum of wrist, dorsum of foot and ankle, palmar aspect of wrist & fingers.

Clinically:
- Slowly growing lump, Mobile across tendon axis but limited along longitudinal axis.
- Common in females.
- Rx: excision.

▪ Subcutaneous Lumps (solid swellings)

Lipoma (The most common benign tumor in subcutaneous tissue)	**Neurofibroma**
-Benign tumor of adipose tissue. -May be mixed e.g: fibrolipoma , neurolipoma (with neural tissue) , haemangioma-lipoma (with vascular tissue). -Dercum's diseas (multiple lipomatosis). Clinically: -Painless, soft and lobulated lump. -Well-defined edges and skin is free, Slipping sign positive, Fluctuation and tranillumination tests are negative. Treatment: -Small asymptomatic→ re-assurance -Symptomatic: surgical excision (if encapsulated), Liposuction (if diffuse).	- Tumour of nerve connective tissue (not neurons) - Types: 1.Localised or solitary NF. 2.Generalized multiple neurofibromatosis type 1 (VonRecklinghausen"s disease)*. etc. Clinically: -Smooth, firm with well defined edges -Tenderness and parasthesia may be present (nerve compression). -Mobility may be diminished along nerve-axis - Rx: excision.

* **Multiple neurofibromatosis type 1 (VonRecklinghausen"s disease)**

- This autosomal dominant disorder is present at birth or becomes apparent in early childhood. Multiple dermal and subcutaneous nodules arise from peripheral nerves in association with patches of dermal pigmentation ('café au lait' spots).

Postoperative care and complications

Immediate Postoperative Care.

Shallow breathing may mean that the patient is still partially paralyzed. A dose of neostigmine can reverse the residual effects of curariform agents. **Cyanosis is an ominous sign indicating hypoxaemia** due to inadequate oxygenation, and may be due to airway obstruction or impaired ventilation.

Respiratory depression later on in the postoperative period is usually caused by over-sedation with opioid analgesic agents

Optimal postoperative care:

Optimal postoperative care requires clinical assessment and monitoring; respiratory management; cardiovascular management; fluid, electrolyte and renal management; control of sepsis; and nutrition

Airway obstruction

The main causes of airway obstruction are as follows:

• **Obstruction by the tongue** may occur with a depressed level of consciousness. Loss of muscle tone causes the tongue to fall back against the posterior pharyngeal wall, and may be aggravated by masseter spasm during emergence from anaesthesia.

• **Obstruction by foreign bodies**.

• **Laryngeal spasm** can occur at light levels of unconsciousness and is aggravated by stimulation.

• **Laryngeal oedema** may occur in small children after traumatic attempts at intubation, or when there is infection (epiglottitis).

• **Tracheal compression** may follow operations in the neck, and compression by haemorrhage is a particular anxiety after thyroidectomy.

• **Bronchospasm or bronchial obstruction**

Airway maintenance techniques include the chin-lift or jaw thrust manoeuvres, which lift the mandible anteriorly and displace the tongue forward

The pharynx is then sucked out, an oropharyngeal airway is inserted to maintain the airway, and supplemental oxygen is administered. **If cyanosis does not improve or if stridor persists, reintubation may be necessary**.

Haemorrhage

Reactive bleeding is usually caused by a slipped ligature or dislodgement of a diathermy coagulum as the blood pressure recovers from the operation.

patients who have undergone neck surgery must be observed for the accumulation of blood in the wound. If necessary, the wound can be reopened in the recovery room to prevent airway compression and asphyxia.

Late secondary haemorrhage typically occurs 7–10 days after an operation and is due to infection eroding a blood vessel. Rigid drain tubes may also occasionally erode a large vessel and cause dramatic late postoperative bleeding

Surgical Ward Care General Care

The return of bowel sounds and the free passage of flatus reflect →recovery of gut peristalsis.

Tubes, drains and catheters

Nasogastric tubes are removed once the volume of aspirate diminishes

Surgical drains are generally removed when the volume of effluent diminishes. If a urinary catheter has been placed, it should be removed once the patient is mobile.

Fluid balance

Fluid balance is reviewed regularly. The standard intravenous fluid requirement for an adult is 3 litres/day, of which 1 litre should ordinarily be normal (isotonic) saline and 2 litres should be 5% dextrose. In the first 24 hours after surgery, normal saline can be omitted and replaced by 5% dextrose due to sodium conservation as a result of metabolic response. However, this should be judged according to the patient's general circulatory status, it is not necessary to replace potassium within the first 24–48 hours after surgery, as potassium is released from injured cells and tissues at the surgical site in sufficient quantity. Potassium supplements (60–80 mmol daily) can subsequently be added to intravenous fluids, provided urinary output is adequate. Intravenous fluid therapy is discontinued once oral fluid intake has been established.

Blood transfusion

Haemoglobin measurement will be a guide to the need for postoperative blood transfusion. A full blood count should be undertaken within 24 hours of surgery and, as a general rule, blood is administered if the Hb is less than 80 g/l. Above this level, patients can be prescribed oral iron, **unless they have cardiovascular instability or are symptomatic from their anaemia.**

complications of blood transfusion are hypothermia (if the blood has not been adequately warmed), hyperkalaemia (due to leakage of potassium from the red blood cells), acidosis (if the blood has been stored for a long period) and coagulation abnormalities (as stored blood is deficient in clotting factors).

Nutrition

Enteral nutrition is preferred, as it is associated with fewer complications and is believed to augment gut barrier function. If a prolonged period of starvation is anticipated in the postoperative period, a feeding jejunostomy tube can be inserted at the time of abdominal surgery. Alternatively, a fine-bore nasogastric or nasojejunal feeding tube can be passed .If the enteral route cannot be used, total parenteral nutrition can be prescribed..

Complications Of Anaesthesia And Surgery

A) General complications

Transient hiccups in the first few postoperative days are usually no more than a nuisance. Persistent hiccups can be a serious complication, exhausting the patient and interfering with sleep, and may be due to diaphragmatic irritation, gastric distension or metabolic causes, such as renal failure. If no precipitating cause can be found, small doses of chlorpromazine may be helpful.

Spinal anaesthesia may cause headache as a result of leakage of cerebrospinal fluid, and patients should remain recumbent for 12 hours after this form of anaesthesia. If headache persists, it may be necessary to seal the injection site in the dura-arachnoid with a 'blood patch' (i.e. an extradural injection of the patient's blood, which clots and so seals the leak).

Myalgia affecting the chest, abdomen and neck is a specific complication of suxamethonium administration, and may last for up to a week.

Arterial cannulae and needle punctures are the most common cause of arterial injury, and may rarely lead to arterial occlusion and gangrene.

B)Pulmonary complications

Once a patient has fully recovered from anaesthesia, the main respiratory problems are pulmonary collapse and pulmonary infection. Pleural effusion and pneumothorax occur less commonly. Pulmonary embolism is a major complication of deep venous thrombosis, which is considered later.

Pulmonary collapse

Inability to breathe deeply and cough up bronchial secretions is the primary cause of pulmonary collapse after surgery.

The clinical signs of pulmonary collapse include rapid respiration, tachycardia and mild pyrexia, with diminished breath sounds and dullness to percussion over the affected segment. Arterial PaO2 is low and the chest X-ray shows areas of increased opacification.

Preoperative measures to reduce the risk of pulmonary collapse following surgery include stopping smoking before the operation, physiotherapy for patients with COPD, **and deferring elective surgery for at least 2 weeks in patients with a chest infection**. Practising with an incentive spirometer preoperatively will help.

Pulmonary infection

Pulmonary infection commonly follows pulmonary collapse or the aspiration of gastric secretions. Pyrexia, tachypnoea and green sputum are typical.

The chest signs are those of collapse with absent or diminished breath sounds, often in association with bronchial breathing and coarse crepitations from surrounding areas of partial bronchial occlusion

Most pulmonary infections are caused by the respiratory commensals, Streptococcus pneumoniae and Haemophilus influenzae, but many postoperative pulmonary infections are caused by Gram-negative bacilli acquired by aspiration of oropharyngeal secretions.

Respiratory failure

Respiratory failure is defined as an inability to maintain normal partial pressures of oxygen and carbon dioxide (PaO2and PaCO2) in arterial blood.

Severe hypoxaemia may result in visible central cyanosis. **In type 1 respiratory failure there is** hypoxia and **in type 2 there is** hypercarbia with hypoxia.

Acute respiratory distress syndrome (ARDS)

ARDS is characterized by impaired oxygenation, diffuse lung opacification on chest X-ray and an increasing 'stiffness' of the lungs (decreased compliance). It may result from pulmonary or systemic sepsis, following massive blood transfusion, or as a consequence of aspiration of gastric contents.

The pathophysiology is unclear, but endotoxin-activated leucocytes are thought to be deposited in the pulmonary capillaries, releasing oxygen-derived free radicals, cytokines and other chemical mediators. Damage to the vascular endothelium results in increased

capillary permeability and leakage of fluid, causing widespread interstitial and alveolar oedema. **This is seen as bilateral diffuse fluffy opacities on chest X-ray .**

Management includes supportive measures in the form of ventilation with positive end-expiratory pressure (PEEP) and treatment of the underlying condition. The mortality rate of severe ARDS is approximately 50%.

Pleural effusion

The appearance of a pleural effusion 2–3 weeks after an abdominal operation may suggest the presence of a subphrenic abscess. Small effusions may be left alone to reabsorb if they do not interfere with respiration. Alternatively, pleural aspiration is performed and the fluid sent for bacteriological culture.

Pneumothorax

The most common cause of postoperative pneumothorax **is the insertion of a central venous line,** and a chest X-ray is necessary after this procedure to exclude this potential complication.

C)Cardiac complications

Valvular disease, especially aortic stenosis, impairs the ability of the heart to respond to the increased demand of the postoperative period.

The administration of fluids to patients with severe aortic or mitral valve disease should be carefully monitored.

Myocardial ischaemia/infarction

The absence of symptoms after operation is thought to be due to the residual effects of anaesthesia and to the administration of postoperative analgesia. If ischaemia is suspected, an **ECG** is performed urgently and arrangements are made for cardiac monitoring**. A sample of blood is withdrawn to estimate concentrations of cardiac enzymes.** One-third of postoperative myocardial infarctions are fatal.

Cardiac failure

patients with ischaemic or valvular heart disease, arrhythmias or major surgical insult can also go into failure in the subsequent recovery period.

Clinical manifestations are progressive dyspnoea, hypoxaemia and diffuse congestion on chest X-ray. **Excessive administration of fluid in the early postoperative period in patients with limited myocardial reserve is a common cause,** which can be avoided by monitoring CVP**. Treatment** consists of avoiding further fluid overload, and the administration of diuretics and cardiac inotropes.

Arrhythmias

Sinus tachycardia is common and may be a **physiological response to hypovolaemia or hypotension**. It is also caused by pain, fever, shivering or restlessness.

Sinus bradycardia may be due to vagal stimulation by neostigmine, pharyngeal irritation during suction, or the residual effects of anaesthetic agents.

Atrial fibrillation is the most common postoperative arrhythmia. Fast atrial fibrillation may result in haemodynamic disturbances and may require pharmacological intervention. Refractory cases may require cardioversion.

Postoperative shock

Shock is defined as a failure to maintain adequate tissue perfusion. **The three main types are hypovolaemic, cardiogenic and septic shock**. **Hypovolaemic** shock may be caused by inadequate replacement of pre- or perioperative fluid losses or postoperative haemorrhage, whereas **cardiogenic shock** is usually secondary to acute myocardial ischaemia/infarction or an arrhythmia. **Hypovolaemic** and **cardiogenic shock** are characterized by tachycardia, hypotension, sweating, pallor and vasoconstriction.

Septic shock is characterized in the **early stages** by **a hyperdynamic circulation with fever, rigors, a warm vasodilated periphery and a bounding pulse.**

Later features include hypotension and peripheral vasoconstriction.

D)Urinary complications

Postoperative urinary retention

Inability to void postoperatively is common**, especially** after groin, pelvic or perineal operations, or operations under spinal/epidural anaesthesia.

Males tend to be more commonly affected than females. When its normal capacity of approximately 500 ml is exceeded, the bladder may be unable to contract and empty itself. Frequent dribbling or the passage of small volumes of urine may indicate overflow incontinence, and examination may reveal a distended bladder. **The management of acute urinary retention is** catheterization of the bladder, with removal of the catheter after 2–3 days

Urinary tract infection

Urinary tract infections are most common after urological or gynaecological operations. Pre-existing contamination of the urinary tract, urinary retention and instrumentation are the principal factors contributing to postoperative urinary infection. **Cystitis is manifested by** frequency, dysuria and mild fever, and **pyelonephritis by** high fever and

flank tenderness. **Treatment** involves adequate hydration, proper drainage of the bladder and appropriate antibiotics.

Renal failure

Acute renal failure after surgery results from protracted inadequate perfusion of the kidneys. The most common cause of postoperative oliguria is pre-renal vascular insufficiency from hypovolaemia, water depletion or extracellular fluid depletion. **Hypoperfusion of the kidney may be aggravated by** hypoxia, sepsis and nephrotoxic drugs. Patients with pre-existing renal disease and jaundice are particularly susceptible to hypoperfusion, and are more likely to develop acute renal failure.

The complication can largely be prevented by adequate fluid replacement before, during and after surgery, so that urine output is maintained at 0.5 ml/kg/hr or more. The importance of monitoring hourly urine output means that bladder catheterization is needed in all patients undergoing major surgery, and in those at risk of renal failure.

Urine output below 700 ml in 24 hours (or less than 0.5 ml/kg/hr for several hours on catheter drainage) should be considered pathological oliguria.

Diuretics may be administered only if the patient is well hydrated; however, they should not be continually prescribed if the patient remains oliguric.

Acute postoperative renal failure occurs when the reversible stage of acute renal insufficiency progresses to acute tubular necrosis. Volume loading becomes potentially dangerous with established renal failure, and the mainstays of treatment at this stage are the replacement of observed fluid loss, plus an allowance of approximately 500 ml/day for insensible loss, and **restriction of dietary protein intake to less than 20 g/day.**

Hyperkalaemia can be treated by intravenous administration of insulin and glucose, or cation exchange resins. Haemofiltration or haemodialysis may be indicated if conservative measures fail to prevent rapid rises in serum concentrations of urea and potassium. Recovery from acute tubular necrosis can be anticipated in survivors after 2–4 weeks. The patient will then enter a polyuric phase, in which fluid and electrolyte balance requires careful monitoring.

The mortality rate in patients who develop postoperative renal failure is 50%.

E)Cerebral complications

Cerebrovascular accidents (CVA)

These are usually precipitated by sudden hypotension during or after surgery in elderly hypertensive patients with severe atherosclerosis.

Neuropsychiatric disturbances

The most common is mental confusion with agitation, restlessness and disorientation, and is known as delirium.

Delirium tremens (acute alcohol withdrawal syndrome)

Delirium tremens occurs in alcoholics who **stop** drinking suddenly. In most instances, this can be predicted from a detailed history. **Prodromal symptoms include** personality changes, anxiety and tremors. The fully developed condition is characterized by extreme agitation, visual hallucinations, restlessness, confusion and, rarely, convulsions and hyperthermia. If symptoms are mild, treatment involves the prescription of oral diazepam and vitamin B (thiamine). Control of extreme agitation may require intravenous administration of diazepam, or haloperidol.

F)Venous thrombosis and pulmonary embolism

Deep venous thrombosis (DVT)

The pathogenesis of venous thrombosis involves stasis, increased blood coagulability and damage to the blood vessel wall (Virchow's triad). **the associated risk factors, which include** increasing age, obesity, prolonged operations, pelvic and hip surgery, malignant disease, previous DVT or pulmonary embolism (PE), varicose veins, pregnancy, and use of the oral contraceptive pill.

Measures to prevent DVT include taking care to avoid prolonged compression of the leg veins during and after the operation; the use of graded compression support stockings (TED stockings); mechanical or electrical compression of the calf muscles during surgery; and low molecular weight heparin (LMWH).

Duplex ultrasonography is now the investigation of choice for diagnosing DVT.**Nowadays, most DVTs are treated with LMWH injected subcutaneously once daily**. Heparin therapy is stopped once the patient is fully anticoagulated with warfarin, which is then normally continued for 3–6 months. The dose of warfarin is adjusted to maintain an international normalized ratio (INR) at 2–3 times normal.

Pulmonary embolism

warfarin therapy is recommended in all patients who have sustained a pulmonary embolus, and therapy is normally continued for 6 months. If the patient cannot be anticoagulated, or sustains further PE despite anticoagulation then consideration can be given to placing an inferior vena caval (IVC) filter.

G)Wound complications

Infection

Subcutaneous haematoma is a common prelude to a wound infection, and large haematomas may require evacuation. The onset is usually within 7 days of operation. **Symptoms include** malaise, anorexia, and pain or discomfort at the operation site. **Signs include** local erythema, tenderness, swelling, cellulitis, wound discharge or frank abscess formation, as well as an elevated temperature and pulse rate. Antibiotics are only required if there is evidence of associated cellulitis or septicaemia. If the wound infection is chronic, the presence of a suture sinus or an enterocutaneous fistula must be excluded.

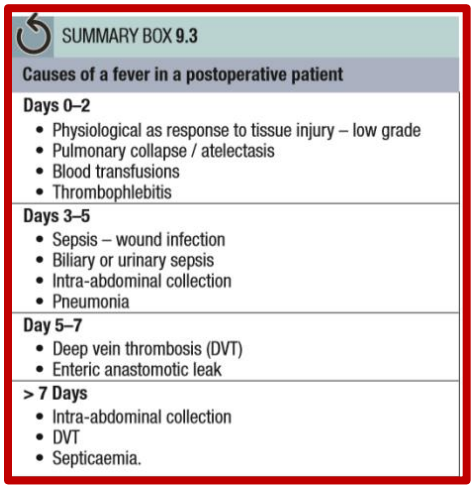
Dehiscence

may be **partial** (deep layers only) or **complete** (all layers, including skin). A **serosanguinous discharge** is characteristic of partial wound dehiscence.

The extrusion of abdominal viscera through a complete abdominal wound dehiscence is known as evisceration. This rare complication **usually occurs within the first 2 weeks after operation**.

Risk factors include obesity, smoking, respiratory disease, obstructive jaundice, nutritional deficiencies, renal failure, malignancy, diabetes and steroid therapy; however, the most important causes are poor surgical technique, persistently increased intra-abdominal pressure, and local tissue necrosis due to infection. The wound should be resutured under general anaesthesia. Incisional herniation complicates approximately 25% of cases.

H) Postoperative fever



SUMMARY BOX 9.3

Causes of a fever in a postoperative patient

Days 0–2
- Physiological as response to tissue injury – low grade
- Pulmonary collapse / atelectasis
- Blood transfusions
- Thrombophlebitis

Days 3–5
- Sepsis – wound infection
- Biliary or urinary sepsis
- Intra-abdominal collection
- Pneumonia

Day 5–7
- Deep vein thrombosis (DVT)
- Enteric anastomotic leak

> 7 Days
- Intra-abdominal collection
- DVT
- Septicaemia.

Common Congenital Neurological Diseases

Spinal Dysraphism

- This is a congenital abnormality of the spinal axis, with or without abnormalities of the spinal cord, meninges and nerves, owing to failure of the neural tube to close.
- Most affect the lumbar/lumbosacral region.
  - **Maternal folate supplementation** and **prenatal screening for raised serum α-fetoprotein** at 16 weeks' gestation reduced the incidence of myelomeningocoele.

Open spinal dysraphism

- This also known as classic spina bifida aperta or myelomeningocoele
- The child has an obvious **open spinal defect** and lower motor neuron signs below the level of the lesion, with numbness, weakness and a neuropathic bladder.

Management: surgical closure of the spinal lesion and require ventriculo-peritoneal shunting as they commonly develop hydrocephalus.

Complication: Chiari II malformation, which may cause respiratory or feeding difficulties and they may develop scoliosis as they grow.

Closed spinal dysraphism

- This is also known as spina bifida occulta and encompasses lesions such as lipomyelomeningocoele, meningocoele, tight filum terminale syndrome, sinus tracts and intradural dermoids, split cord malformations and caudal agenesis.
- The condition may be apparent at birth owing to the characteristic overlying skin lesions, which include midline lumbar lipomas, hairy patches, dimples and sinuses.

Management: surgical untethering and affected individuals do not develop hydrocephalus and there is no association with the Chiari malformation.

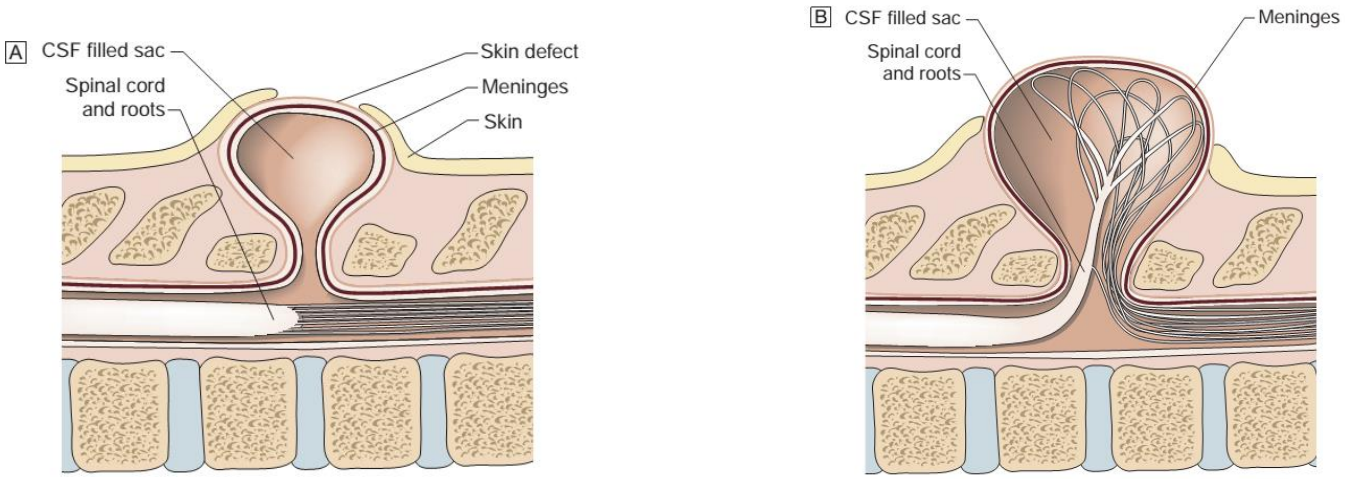


Fig. 24.16 Spinal dysraphism. A Simple meningocoele. B Myelomeningocoele.

Hydrocephalus

Etiology and clinical features

Hydrocephalus is the accumulation of CSF within the ventricles or over the surface of the brain due to

1- Overproduction of CSF (because of a choroid plexus papilloma)
2- Reduced drainage secondary to obstruction of normal CSF flow:
   - congenital, such as in aqueduct stenosis
   - Acquired, as a result of tumour or arachnoidal adhesions and fibrosis secondary to intraventricular or subarachnoid haemorrhage.

- 'Internal', 'non-communicating' or 'obstructive' hydrocephalus is due to obstruction of flow within the ventricular system, leading to dilatation of the ventricles.
- In 'external' or 'communicating' hydrocephalus, the ventricular system is patent but there is reduced flow through the basal cisterns or absorption of CSF by the arachnoid granulations. In this type, the ventricles and the CSF spaces around the surface of the brain will be enlarged.
- In adults, chronic hydrocephalus may cause the 'normal pressure hydrocephalus' syndrome of gait ataxia, incontinence and cognitive decline.

Symptoms & signs:
Congenital hydrocephalus presents at birth or in early infancy:

1. The cranial sutures may start to open and the fontanelle will be tense and bulging.
2. The veins of the scalp and the bridge of the nose will be dilated.
3. As the hydrocephalus worsens, the eyes may become downcast (sunsetting).
4. The child may be floppy and develop apnoeic spells and episodes of bradycardia.

In older children and children with closed fontanelles:

1. headache, vomiting and drowsiness
2. The eyes may develop a squint owing to VIth cranial nerve palsy.
3. Papilloedema may be present
4. if severe or chronic, may lead to blindness.

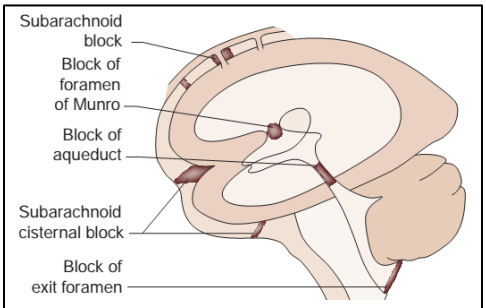


Fig. 24.17 Hydrocephalus: sites of cerebrospinal fluid (CSF) blockage.

Management and prognosis
Treatment consists of relieving the pressure by bypassing the block to CSF drainage such as in the following cases:

Endoscopic third ventriculostomy: in case of aqueduct stenosis (endoscope a small hole is formed in the floor of the third ventricle, allowing CSF to flow into the basal cisterns)

ventriculo-peritoneal (VP) shunt: in most cases insert catheter in the **lateral ventricle**, which drains CSF through a valve (that sits on the skull under the scalp) into the **peritoneal cavity**.

Complication: bleeding into the ventricular system - early infection, usually with skin commensal organisms such as *Staphylococcus epidermidis* - over-drain the ventricles, leading to premature closure of the cranial sutures and microcephaly.

The long-term prognosis depends very much on the underlying cause of the hydrocephalus. In cases of simple aqueduct stenosis treated early, the prognosis for normal IQ and normal neurological function is good. Repeated episodes of raised ICP or ventriculitis can lead to loss of IQ and neurological deficit.

Malformations Of The Skull

Subgaleal haematomas:

- often related to underlying skull fractures
- Can cause the haemoglobin to drop significantly.

Growing skull fractures:

- Peculiar to infancy
- Caused when a fracture is associated with an underlying dural tear.
- The treatment is to repair the dura. The defect can be repaired with bone, but this is not always required.

Craniosynostosis

- Premature closure or absence of a cranial suture.
- Several intramembranous ossification centres occur in the skull vault and form plates of bone. Sutures form where these plates of bone meet each other. This is where further bone growth occurs. Overall bone growth is driven by the expanding brain. The brain has reached 85% of its adult size by the age of 2 years but continues to grow slowly after this time. The midline frontal metopic suture fuses at the age of 2 years.
- **The commonest head-shape are**
  1. Scaphocephaly (sagittal suture)
  2. Plagiocephaly (coronal or lambdoid suture): Many cases of plagiocephaly are due to head moulding, when the baby lies on its back to sleep.
- Sometimes, more than one suture can be affected. This can be syndromal (e.g. Crouzon's or Apert's syndrome). These syndromes are associated with characteristic craniofacial deformities.
- **Signs and symptoms:** reduction in cranial volume, causing raised ICP.
- **Management:** Surgery can be undertaken to remodel the skull into a more acceptable shape or to increase the cranial volume.

Cranial dermal sinuses and angular dermoids

- Dermal sinuses are **midline tracts lined with squamous epithelium** that may communicate with the intracranial cavity and may predispose to **meningitis.**
- **In the head,** most are found in the **occipital region**; 70–80% are associated with inclusion dermoids and 80% extend subdurally.
- **In the face,** because of the complex embryology, dermoids can be found at **the tip of the nose and the lateral aspect of the eye.**

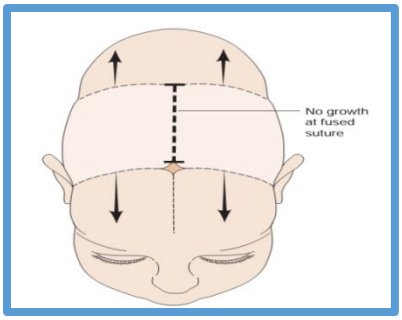


Fig. 24.20 Sagittal suture craniosynostosis. The sagittal suture is prematurely fused (broken line). Growth normally occurs perpendicular to the suture line. In this case, the skull cannot widen, as there is no growth at the fused suture, which may be palpable as a ridge in the midline. There is compensatory growth at the coronal and lambdoid sutures, leading to an elongated head shape (scaphocephaly), with bulging of the forehead (frontal bossing) in severe cases.

Intracranial pressure:

❖ Introduction:

Intracranial pressure (ICP) depends on volumes of:

1- intracranial blood
2- CSF
3- Brain parenchyma.

Note: ICP also fluctuates in response to changes in **intrathoracic pressure** (e.g. increased by coughing, defecation) and **cardiac pulsation.**

- ICP is the same as the CSF pressure obtained at **lumbar puncture** (**5–15 cm H2O, 4–10 mmHg**).

- ICP is often severely elevated following neurotrauma because of oedema, haematoma, contusions, engorgement of the brain vasculature, hydrocephalus or even infection.
  What happened if there is increase in ICP?
  In patients with intracranial mass lesions (tumour, haemorrhage), oedema or CSF obstruction, the extra volume is
  at first compensated for by a **reduction in cerebral blood volume and CSF volume**.

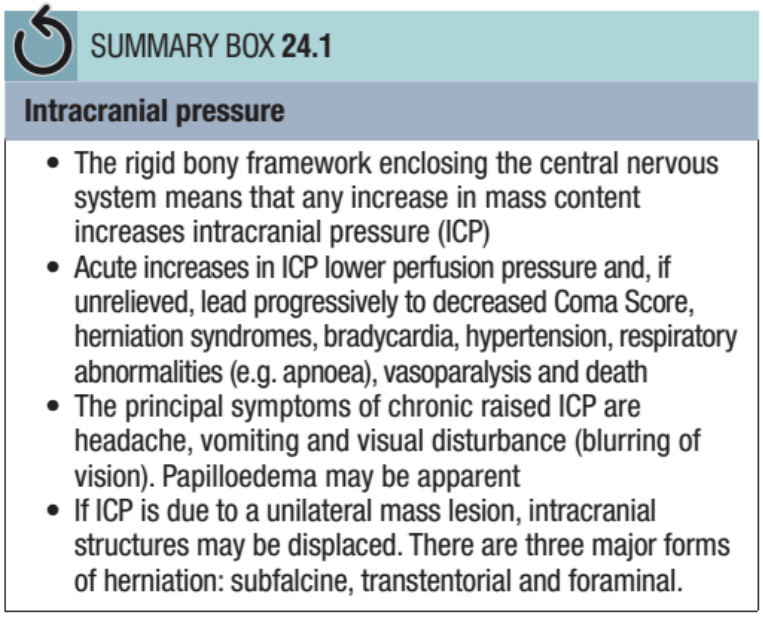
If compensated mechanism failed Generalized or localized increases in ICP may lead to:

1- marked displacement of intracranial structures (**brain herniation syndromes**)

2- Compromise brain perfusion leading to **cerebral ischaemia and infarction**

- The cerebral perfusion pressure (CPP) equals mean arterial pressure (MAP) less the ICP **(CPP = MAP – ICP).**
- Progressive rises in ICP lead to **increases in MAP and reflex bradycardia.**
- A **CPP of > 60 mmHg** is generally required to sustain adequate cerebral perfusion.

The rate of increase in the volume of intracranial mass is crucial to the shape of the ICP pressure–volume curve With more chronic, slow-growing lesions such as brain tumours, abscesses or congenital abnormalities, extraordinary degrees of compensation can occur.



SUMMARY BOX **24.1**

Intracranial pressure

- The rigid bony framework enclosing the central nervous system means that any increase in mass content increases intracranial pressure (ICP)
- Acute increases in ICP lower perfusion pressure and, if unrelieved, lead progressively to decreased Coma Score, herniation syndromes, bradycardia, hypertension, respiratory abnormalities (e.g. apnoea), vasoparalysis and death
- The principal symptoms of chronic raised ICP are headache, vomiting and visual disturbance (blurring of vision). Papilloedema may be apparent
- If ICP is due to a unilateral mass lesion, intracranial structures may be displaced. There are three major forms of herniation: subfalcine, transtentorial and foraminal.

❖ Brain herniation syndromes:

1- Subfalcine (cingulate gyral) herniation:

With a **parasagittal mass**, the **ipsilateral cingulate gyrus** may herniate **beneath the free edge of the falx.**

Leading to:

Anterior cerebral artery compression and cause **medial hemispheric infarction**, but otherwise there are no obvious clinical signs except **deteriorating conscious level.**

2- Transtentorial (uncal) herniation:

With **large ipsilateral brain lesions**, the **medial part of the temporal lobe** is pushed down **through the tentorial notch**
to become wedged between the tentorial edge and the midbrain.

Leading to:

1. The opposite cerebral peduncle is pushed against the sharp tentorial edge, and the midbrain and uncus become wedged at the tentorium.
2. The aqueduct is compressed, obstructing CSF flow, and venous obstructionleads to midbrain haemorrhage.

- **The clinical features of an uncal herniation, most often due to a traumatic intracranial haematoma, are:**
  - The Glasgow Coma Score (GCS) falls.
  - The motor component of the GCS becomes asymmetrical
  - The ipsilateral pupil dilates and becomes non-reactive to light
  - The blood pressure rises
  - The pulse slows
  - The respiratory rate falls and the patient become apnoeic.

3- Foraminal (tonsillar) herniation:

With **mass lesions of the posterior cranial fossa,** the **cerebellar tonsils and medulla** are displaced downwards **through**
the foramen magnum.

Leading to:

Cerebellar impaction leads to medullary compression can lead to:

- dramatic decrease in the GCS,
- acute hypertension
- bilateral extensor responses
- bilateral fixed dilated pupils
- sudden respiratory arrest

A similar syndrome may occur following **the removal of CSF at lumbar puncture in patients with raised ICP** due to a **posterior fossa tumour**, and is also known as **'coning'.**

- There is a rapid deterioration in conscious level, with decerebration.
- Lumbar puncture **must NOT be performed** in patients suspected of having raised ICP due to a mass lesion.

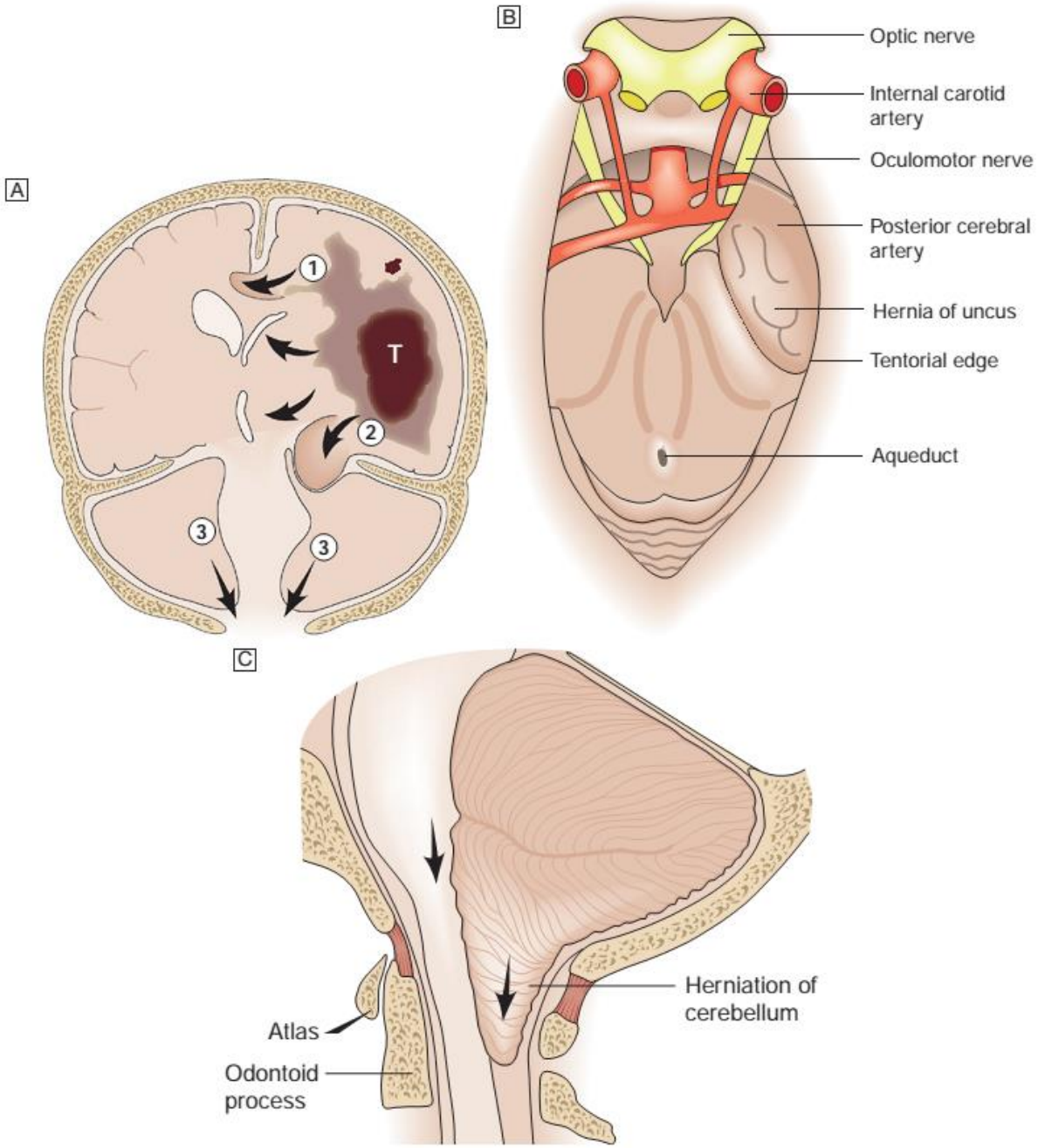


Fig. 24.3 Herniation syndromes. A Coronal diagram of the dynamics of an intracranial mass lesion. Tumour (T) causes mass effect that compresses the midline and the ventricles (unnumbered arrows), provoking subfalcine or cingulated herniation (arrow 1), and transtentorial herniation (arrow 2). If the mass effect is uncontrolled, tonsillar herniation may also occur (arrows 3). B Transtentorial herniation in the axial plane (plane of dotted line in Fig. 24.3A). C Foraminal herniation.

Glasgow Coma Score

- The GCS is a **measure of conscious level especially after head injury**.
- It records the best verbal response, best motor response and eye opening.
- The **maximum score is 15** and the **minimum is 3.**
- Mild injury: 15 to 13; moderate: 12 to 9; and severe less than 8.
- Coma is also defined as a GCS of 8 or less.

Table 24.1 Glasgow Coma Scale

Eyes open	
• Spontaneously	4
• To verbal command	3
• To pain	2
• No response	1
Best motor response	
To verbal command	
• Obeys verbal command	6
To painful stimulus	
• Localizes pain	5
• Flexion withdrawal	4
• Abnormal flexion (decorticate rigidity)	3
• Extension (decerebrate rigidity)	2
• No responses	1
Best verbal response	
• Orientated and converses	5
• Disorientated and converses	4
• Inappropriate words	3
• Incomprehensible sounds	2
• No response	1
Total number of points (minimum 3, maximum 15)	

Management

- The principal aim of management is to limit **secondary damage due to ischaemia and brain herniation caused by raised ICP, hypoxia and hypotension.**
- As with all injured patients, management commences with **airway, breathing and circulation.**
- **The neck should be immobilized** until a **cervical spine injury has been excluded.**
- **The GCS should be documented on arrival and following resuscitation because it is the key aspect in management:**

  - **Patients with a GCS of 8 or less** are intubated and ventilated; to prevent hypoxia and aspiration pneumonitis, and to allow hyperventilation, which reduces the PaCO2 and so lowers ICP through cerebral vasoconstriction.
  - **a head CT** is performed to visualize intracranial haematoma, brain contusions (bruises), depressed bone fragments, intracranial air and associated maxillofacial fractures.
  - Mass lesions such as extradural haematoma, subdural haematoma and haemorrhagic contusions may cause brain swelling and shift, and are often **surgically evacuated.**
  - Indications for clot evacuation are **> 5 mm midline shift, significant impairment of GCS, or protracted headache or vomiting.**
  - Compound cranial wounds need to be surgically explored, dead tissue and foreign bodies removed, depressed bone fragments elevated, haemostasis secured and the dura closed in a watertight fashion.
  - A sustained ICP that exceeds 25 mmHg is associated with a **poorer outcome.**
  - Severely brain-injured patients are therefore kept sedated and ventilated and their ICP is monitored.
  - **Hyperventilation, mannitol and barbiturates** are used to reduce ICP, and the systemic blood pressure may be raised using fluids and inotropes.

Introduction to Mechanisms of Trauma + Trauma Care

The most common cause of death from birth to the fourth decade, and the fourth most common cause overall, is trauma.

☒ Accident features associated with major trauma

- Death of another individual in the same accident
- High-velocity impact, e.g. pedestrian or cyclist (motor or pedal) struck at > 30 kph, or vehicle occupants in collisions with closing speeds > 60 kph
- Entrapment in, or intrusion into, the passenger compartment of the vehicle
- Ejection from a vehicle
- Falls from heights > 3 m
- Penetrating injury of the chest, abdomen or neck.

☒ Injury severity assessment

Glasgow coma scale is used to assess the neurological state of injured patients objectively, and which also has prognostic value.

Table 7.1 Glasgow Coma Scale

Eyes open	
• Spontaneously	4
• To verbal command	3
• To pain	2
• No response	1
Best motor response	
To verbal command	
• Obeys verbal command	6
To painful stimulus	
• Localizes pain	5
• Flexion withdrawal	4
• Abnormal flexion (decorticate rigidity)	3
• Extension (decerebrate rigidity)	2
• No responses	1
Best verbal response	
• Orientated and converses	5
• Disorientated and converses	4
• Inappropriate words	3
• Incomprehensible sounds	2
• No response	1
Total number of points (minimum 3, maximum 15)	

☒ primary survey

- Airway with cervical spine control
- Breathing with ventilation
- Circulation with haemorrhage control
- Neurological disability and pupils
- Exposure and environment.

Airway

- The patency of the airway is first assessed by direct inspection, identifying and removing obstructions.
- The most common cause of airway obstruction is a reduced conscious level, with the tongue falling back blocking the oropharyx.
- Airway clearance, together with the ‘chin-lift’ or ‘jaw-thrust’ manoeuvres,
- A patient speaking in complete sentences does not have an immediate airway problem
- if the GCS score is < 8/15, usually mandates early definitive airway intervention,
- Orotracheal intubation is the advanced airway technique of choice .the patient is pre-oxygenated and must be carefully monitored throughout the process.

Breathing

- Optimal ventilation requires a patent upper and lower airway and effective function of the thoracic wall, lungs and diaphragm
- Respiratory compromise is characterized by tachypnea or bradypnoea, the use of accessory muscles of respiration, and paradoxical (see-saw) movement of the chest and abdomen, Hypoxia may be manifest by restlessness, tachycardia, confusion, agitation, pallor or sweating
- Clinical inspection, palpation and auscultation of the neck and chest (including the back) should detect immediately life-threatening injuries such as flail segment, penetrating wounds, tension or open pneumothoraces, major haemothorax and cardiac tamponade. These conditions need immediate treatment, e.g. needle thoracocentesis for tension pneumothorax, or the insertion of an intercostal drain for haemothorax.

Circulation

- The clinical detection of blood loss and the resulting haemodynamic effects is crude and non-specific. Pulse rate, cuff blood pressure and peripheral perfusion (assessed by capillary refill time) are routinely noted every 5–10 minutes in the initial stages.
- **Situations where blood loss may be misappreciated**
  - The elderly
  - Patients on concurrent drug therapy (e.g. β-blockers, antihypertensives, anti-anginals)
  - Patients with a pacemaker
  - Athletes
  - Pregnancy
  - Hypothermia.
- The next priority is to insert and secure two largebore (12–14 G) intravenous cannulae. The forearms or Antecubital fossae are the most accessible peripheral sites.
- **Initial blood samples should be taken from the trauma patient for :-**
  - Blood grouping and cross-matching
  - Full blood count and haematocrit
  - Urea and electrolytes
  - Plasma glucose
  - Arterial blood gases
- **To ensure effective resuscitation** → 1- Measurement of urine output (should be >1 ml/kg body weight/hr normally implies adequate renal perfusion)
  2- continuous intra-arterial blood pressure monitoring
  3- serial lactate levels assist in monitoring the response to infusion.
- **Monitoring trauma patient :-**
  - Heart rate

- Blood pressure (cuff and intra-arterial) (in both arms if
- suspected aortic injury)
- Capillary refill
- Respiratory rate
- Glasgow Coma Scale
- Urine output
- ECG
- Pulse oximetry
- Core temperature.

☒ Analgesia

Opioid drugs such as morphine or diamorphine, given in small intravenous (intramuscular absorption may be poor and inconsistent) doses titrated to effect are unsurpassed for analgesia.

☒ Secondary survey

a more detailed history and examination is undertaken, with appropriate laboratory and imaging investigations to determine the full extent of the patient's injuries and the requirement for surgery or other care. This review should enable a definitive management plan to be formulated.

Hand Injuries

Necrotizing fasciitis

This is an uncommon but severe, life-threatening infection of **skin and subcutaneous tissues characterized by necrosis of deep fascia**

There are two main types depending on causative organisms

- **Type I: Polymicrobial aetiology** which is also known as synergistic bacterial gangrene; Fournier's gangrene is a special type affecting the perineal area
- **Type II: Single organism infection**, usually by β-haemolytic Group A streptococci (Streptococcus pyogenes).

The infection usually starts at a site of (often minor) trauma and can spread very quickly as bacterial exotoxins and enzymes lead to necrosis of fat and fascia and eventually overlying skin.

The patient is usually febrile, toxic and in severe pain. Initially, the overlying skin may appear deceptively normal but as the infection progresses there is oedema, discoloration and crepitus (due to gas production).

Urgent surgical debridement of all necrotic tissue is essential and several visits to theatre may be required. Initial antibiotic choice is usually empirical with a combination of broad-spectrum agents against likely pathogens e.g. carbapenems, clindamycin and metronidazole. Antibiotic therapy can later be tailored according to the results of pus and tissue cultures.

Wrist disease

The cause of pain around the wrist can be a challenging diagnosis to make. Common causes include those **secondary to trauma** such as a distal radius fracture (Colles fracture) or a scaphoid fracture

degenerative causes such as rheumatoid arthritis or osteoarthritis and those secondary to inflammation affecting the tendon sheaths such as De Quervain's disease which affects the abductor pollicis longus and extensor pollicis brevis tendons.

Trigger finger

Thickening of the flexor tendon causes it to jam under the pulley system that would normally allow the tendon to slide backwards and forwards. Injection around the tendon or release of the first pulley results in a cure of the condition.

Dupuytren's disease

Thickening of the palmar fascia draws the fingers (predominantly the fourth followed by the fifth) into a flexed and deformed position, resulting in disability and loss offunction. It is more common in diabetics, epileptics and can often be associated with previous trauma to the area affected.

The condition mainly affects men over the age of 40 years. Surgery is considered if there is significant disability affecting the fingers.

This involves either dividing the thickened tissue (fasciotomy), dissecting and removing the thickened fascial bands alone (fasciectomy) or with the overlying skin (dermo-fasciectomy), taking great care to preserve the associated nerves and blood vessels. In severe cases, amputation of the little finger may be the best line of treatment

Pediatric Hand Fractures

Fractures often behave differently in children. Because their bones are more pliable, children may suffer from greenstick fractures, in which the cortex of the bone does not break but bends instead. Fractures may also affect the growth plate (epiphysis) of the bone, leading to problems with slowing of the growth of the bone (growth arrest) or deformity of the growing bone .Generally, fractures heal much more quickly in children.

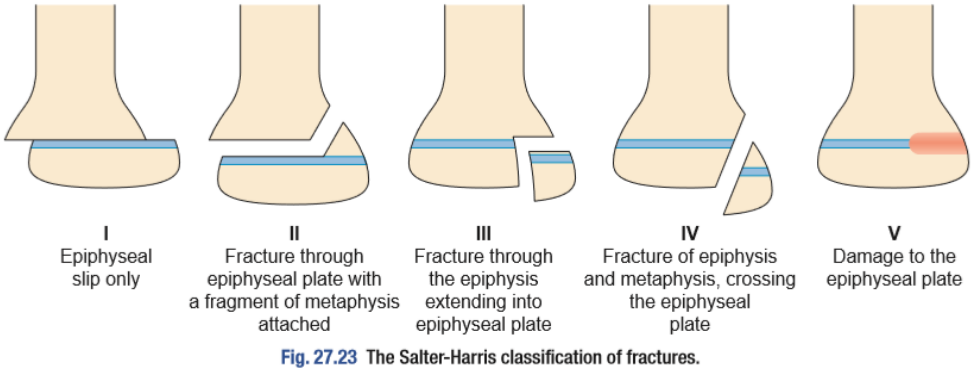


Fig. 27.23 The Salter-Harris classification of fractures.

There are many differing ways of treating fractures. However, they all have the same fundamental objectives: namely, the close approximation of uncontaminated, well-vascularized bone ends in a stable configuration that will maximize bone and soft tissue healing without deformity or loss of function. **In general, the fracture healing process takes approximately 8–10 weeks in adults** but can take longer depending on the severity of the injury and whether it is an open fracture

Breast Diseases

Anatomy

- Composed of glandular tissue, fibrous and fat.
- Lies between the skin and pectoral fascia.
- Extends from the clavicle down to the abdominal wall muscles.
- Axillary tail runs between pectoralis & latissimus dorsi to blend with the axillary fat.
- **Functional unit** -> Terminal Duct Lobular Unit. (where usually tumors arise from)
- Secretion from the terminal duct -> 12-15 subareolar ducts -> nipple.
- In non-pregnant, the nipple is plugged by keratin. (Dislodge of keratin? -> watery **physiological** secretions could be multiple colors)

Blood Supply and Lymphatic Drainage:

- Lateral thoracic branch of the axillary artery superiolaterally.
- Perforating branches of the internal mammary artery superiomedially.
- The main route of lymphatic spread of breast cancer is to the axillary node behind the axillary vein. (20 Nodes)
- These nodes separated in 3 levels.
  Level 1 -> Lateral to pectoralis minor. (nearest to the breast, **1st affected by breast cancer**)
  Level 2 -> Below the pectoralis minor.
  Level 3 -> Medial to pectoralis minor.
- Occasionally, the main route of lymph drainage of cancer is to the interpectoral (**Rotter's**) nodes. Lies between P. major and minor.

Histology:

- The functional unit is lined with single layer of cuboidal epithelial cells surrounded by myoepithelial cells.
- The subareolar is lined with stratified squamous epithelium.

Congenital Anomalies

- **Supernumerary Nipple** → (between normal breast and umbilicus)
- **Supernumerary breast** → (Lower the axillary line)
- **Absence or hypotrophic breast** → pectoral muscle defect.
- **Poland's Syndrome** -> characteristic deformity of the upper limb + hypotrophic / absence breast.

Hormonal Control of Breast & Development & Function

- **Witch's Milk:** Because of maternal circulating estrogen in the newborn, the breast bud enlarges in the 1st two weeks & secret colostrum-like fluid the it'll regress until puberty.
- Three phases of development of breast:
  A) **Development:** At puberty, proliferation of ducts & ductules.
  B) **Mature Reproductive Life:** During pregnancy. The breasts double in weight, further proliferation for milk production. Milk production is inhibited during pregnancy by the steroids of ovaries & placenta which will decrease after delivery.
  C) **Involution:** From the age of 30 (persistent). Glandular and fibrous tissues atrophy. The breasts become droopy. Microscopically; Fibrosis, microcysts formation, adenosis. **(Physiological)**

Clinical Features

- **Presenting Complains:**

1) Breast Lump,
2) Nipple Discharge, scaling, retraction or eczema.
3) Breast distortion,
4) Swelling or inflammation.

- **The most important pointer to the diagnosis is age.**

Duration; cyst develops overnight, malignancy is slow

Evaluation of The Patient with Breast Disease:

Examination And Assessment of regional lymph nodes	Investigations
- Inspection; patient's hands first by the patient's side, above the head, then pressing on the hip. - If the patient complain was discharge, **try to produce it.** - 4 Groups of lymph nodes; 1) Ant. group, behind pectoralis major, Ant. Axillary fold. 2) Post. group, Ant. to subscapularis muscle, Post. axillary fold. 3) Medial group, Against the ribs and chest wall. 4) Lateral group, against the medial side of the neck of Humerus. - Supraclavicular lymph nodes are assessed from behind. - **Ultrasound is better in assessing the axillary nodes than clinical examination.**	**1) Mammography:** **The best screening tool.** Primary signs: mass lesion (e.g. microcalcification) Secondary signs: parenchymal architectural distortion (e.g. nipple/areola changes) Because the breasts are relatively radiodense in women under 35 years of age, mammography is rarely of value in this group. **2) Ultrasonography:** **Guide for biopsy** Differentiate between solid and cysts. Cysts → transparent lesions. Benign lesions → well-circumscribed. Malignant lesions → irregular & hypoechoic. **3) MRI:** Accurate, **high sensitivity** for breast cancer. **Differentiate between** invasive and non-invasive tumors. MRI is the optimum method of imaging breast **implants** and detecting implant leakage or rupture. **4) Ductoscopy, ductography, scintigraphy.** **Ductoscopy & ductography** → to locate abnormal ducts. **Scintigraphy** -> used in case of vague other imaging results using isotope to detect metabolic activity of a lesion to indicate neoplasm. **5) Nipple Discharge** → Assess volume, color, consistency & number of affected ducts. **Discharge from multiple ducts is managed by total excision of the major subareolar using a periareolar incision.** **6) FNAC and Biopsy:** **Core biopsy** → Image-guided core biopsy is **the most accurate** tool for diagnosis. FNAC -> Could be used to assess metastasis in the axillary nodes. Open biopsy → If the **core biopsy** didn't exclude malignancy or if the patient requested an excision for a benign mass. Sentinal node → the first lymph node that drains the tumor and is most commonly level 1
Clinical Presentation: • Most common complain → painless lump, firm and irregular. • Malignant lesion produces asymmetrical breasts such as dimpling of the skin, or retraction of the nipple. • 50% of breast cancers are located in the upper outer quadrant. • Some patients present with manifestations of advanced breast cancer, such as skin ulceration or edema. • **Breast pain is rare.** • Nipple appearance: Dry scaling, red weeping → **Paget's Disease**, and it signifies an underlying invasive or non-invasive cancer. • **Paget's disease** → **Always** affect the **nipple**, areola is secondary. • **Eczema** → **Always** affect the **areolar**, the nipple is secondary.	

Notes:

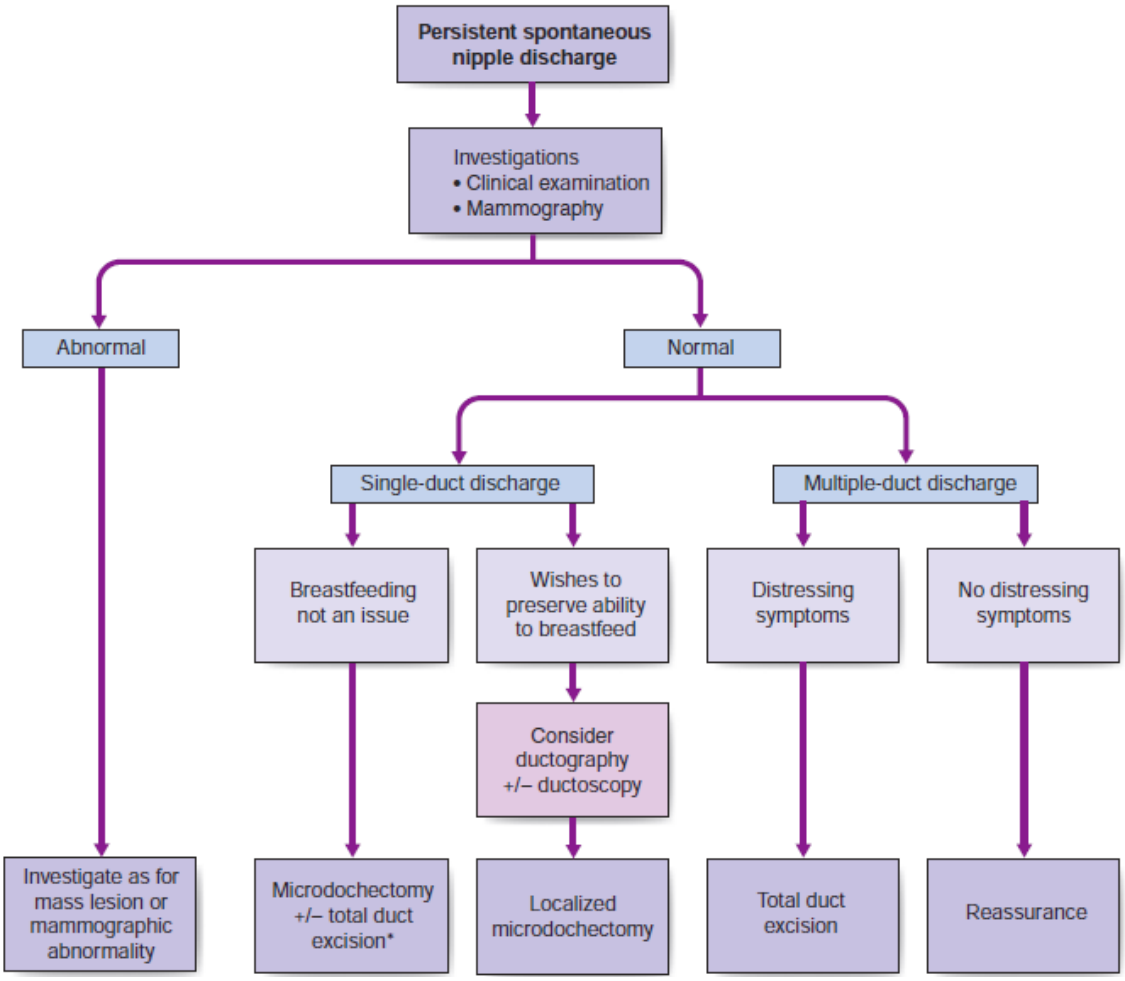
- MRI is more effective than mammography at screening women < 50 years who are at very high risk of breast cancer either because they carry a BRCA1 or BRCA2 mutation or because of their family history.
- Patients presenting with breast lump or suspicious lesion on image should undergo triple assessment:

A) Mammography +/- ultrasound for women > 35.
B) Ultrasound for women < 35.
C) Core biopsy +/- cytology.

<table>
<tr><th colspan="3">Disorder of Development</th></tr>
<tr><td colspan="2">Juvenile Hypertophy: Uncontrolled overgrowth of breast tissue after puberty.
- Usually bilateral.
- Due to increase in stromal tissue, not lobules of ducts.
- Presenting symptoms: pain in shoulders & back & large breasts.
- Treated with reduction mammoplasty.</td><td>Fibroadenoma: Aberration of development
- Develop from a whole lobule rather than a single cell (that's why it's considered as a developmental aberration rather than a benign tumor)
- Hormonal dependence & increase in size.
- Common in (15-25) age group.
- Well-circumscribed, firm, smooth, mobile lumps.
- Could be multiple or bilateral.
- Visualized margins in Ultrasound.
- Diagnosis is confirmed by core biopsy.
- Management if not disappeared alone;
A) <4 cm → Reassurance, no excision nor follow-up is needed.
B) >4 cm → excised to check for phyllodes tumors.</td></tr>
<tr><th colspan="3">Disorders of cyclic changes: premenstrual nodularity and breast discomfort are common and considered normal.</th></tr>
<tr><td>Cyclical Mastalgia:
- Severe premenstrual pain interferes with daily activities.
- Managed with Danazole and Tamoxifen.</td><td>Nodularity:
- Could be diffused or focal, diffuse nodularity is normal premenstrual. Focal nodule must be investigated by core biopsy to exclude malignancy.</td><td>Non-cyclical Mastalgia:
- It's actually a chest wall pain instead of breast pain as the patient may think.
- NSAIDs are effective, if not, use local anesthesia and steroids.</td></tr>
</table>

<table>
<tr><td rowspan="4">Disorders Related to Aberrations of Normal Development and Involution (ANDI): NORMAL AGING PROCESS</td><td>Palpable breast cysts: Smooth discrete lumps that can be painful and visible.
- Management:
A) Symptomatic cysts → aspiration.
Cysts that contain blood-stained fluid are excised to exclude intracystic malignancy.
B) Asymptomatic cyst → no aspiration.
C) All patients with cyst should have mammography.
- Galatocoele: Subareolar cyst lesion which usually occurs in lactating women.</td></tr>
<tr><td>Sclerosis: Occurs in stromal involution. They include;
A) Radial scars.
B) Complex sclerosing lesion.
C) Sclerosing Adenosis.
- All can produce stellate lesions or localized <u>calcification</u> that mimic breast cancer in mammography.
- Radial scars are difficult to differentiate on imaging from small cancers.
- Most scars are removed.</td></tr>
<tr><td>Duct Ectasia: Symptomatic major subareolar ductal dilation and shorten due to aging.
- The dilation result in cheesy discharge.
- Retraction of the nipple is slit-like in this condition.
- Surgery is indicated if the discharge is bothersome, or the patients wants the nipple to be everted.</td></tr>
<tr><td>Epithelial Hyperplasia: Increase in cells lining the terminal ducts lobular unit.
- Graded into 3; mild, moderate, or florid (papillomatosis).
- Atypical hyperplasia → hyperplastic cells show atypia. (Women with atypical hyperplasia have significant increase risk for development of breast cancer)</td></tr>
</table>

Benign Neoplasia		
Duct Papillomas: - Single or multiple. - Can cause persistent & troublesome nipple discharge which can be frank blood or serum. - **Management:** Removal of discharging duct (**microdochectomy**) which removes the papilloma and exclude an underlying neoplasm.	**Lipomas:** - Soft, lobulated, radiolucent lesions. - Could be misinterpreted with **pseudolipoma**, which is a mass that can be felt around a tumor. - They may be present as a lump in the axilla where it has to be differentiated from accessory breast. - **Management:** Excision for **cosmetic** reasons.	**Phyllodes Tumors:** - Localized, discrete fibro-epithelial masses. Clinically mimic fibroadenoma but larger > 4 cm. - **Management: Wide excision** (whether malignant or benign) or mastectomy if the lesion was large. **No axillary clearance required.**
Other benign tumors that occur in the breast include granular cell tumor, neurofibromas, and leiomyomas.		



Breast infections:

- Most commonly affect women between 13-50.
- Divided into **lactating** and **non-lactating**.
- An **infection of the skin** overlying the breast is either **primary** of **secondary**.
- **Principle of treatment:**

A) Antibiotics.
B) Surgical Drainage of abscess. (presence of abscess is confirmed by ultrasound or aspiration)
C) Excluding breast cancer using imaging and core biopsy.

- **Antibioma:** localized abscess or inflammation mimicking a tumor due to **prolonged** antibiotics use.

Lactating infection	Non-lactating infection
- Usually develops within the **1st six weeks** of breast feeding. - Reduce drainage of milk from the affected segment. - The stagnant milk will be infected, so women are encouraged to breastfeed before this happen. - Pain, swelling, tenderness, with cracked nipple +/- skin abrasion. - **S. aureus** is the most common cause. - **Management:** 1) Flucloxacillin (**allergy?**) use co-amoxicalv instead. 2) **Present abscess?** → recurrent aspiration or incision and drainage.	**A) Central (periareolar) infection:** - Most commonly in 32-year-old women. - Etiology → **periductal mastitis.** - Smoking is an important risk factor. - Periareolar inflammation +/- abscess. - Nipple retraction could occur. - **Management:** 1) Co-amoxiclav. 2) **Recurrent infection?** → Total Ductal Excision. - After abscess drainage, 1/3 develop fistula. **B) Mammary Ductal Fistula:** Communication between the skin & major subareolar duct at the areolar margin. - **Management:** Excision of the fistula & the diseased duct, under antibiotics cover. **C) Peripheral non-lactating abscess:** - Associated with DM, RA, Steroids treatment or trauma. - **Managed as all abscess.** **D) Tubercular Mastitis:** - Affect females in the reproductive age group. - Present with a lump painless or painful, and with multiple inflammatory nodules which could involve the skin & sinuses. - **Hx of TB.** - FNAC shows **Caseous necrosis** and +ve acid-fast bacilli. - **Not clear diagnosis?** → Open biopsy. - **PCR is diagnostic with histopathology.** - **Treatment: Anti-TB**.

Skin Infections:

- **Primary:** affects the lower half of the breast, recurrent in overweight patients/large breast. More common after surgery or radiotherapy.
  Managed with antibiotics and abscess drainage, with weight reduction in those with overweight and hygiene.
- **Secondary:** Infected subaceous cysts.
- Some recurrent infections in the skin of the lower part of the breast are due to **Hidradenitis Suppurativa**, more common in smokers. This is **managed** with excision of the affected skin.

Table 19.4 Established and probable risk factors for breast cancer

Factor	Relative risk	High-risk group
Age	> 10	Elderly
Geographical location	5	Developed country
Age at first full pregnancy	3	First child in early 40s
Previous benign disease	4–5	Atypical hyperplasia
Cancer in other breast	> 4	Women treated for breast cancer
Socioeconomic group	2	Social classes I and II
Diet	1.5	High intake of saturated fat
Exposure to ionizing radiation	3	Abnormal exposure in young females after age 10
Taking exogenous hormones		
Oral contraceptives	1.24	Current use
Combined hormone replacement therapy	2.3	Use for ≥ 10 years
Family history	≥ 2	Breast cancer in first-degree relative

Breast Cancer:

- Also, one of the risk factors is early menstrual cycle and late menopause.
- Breastfeeding is protective.
- Only 10% of breast cancers are due to genetic predisposition, also not all gene carries develop breast cancer.
- **Human breast cancer genes that have been identified and that affect different families include:**

1) **BRCA1** on chromosome **17**,
2) **BRCA2** on chromosome **13**,
3) **p53** on chromosome **17** and,
4) **PTEN** on chromosome **10**.

- **Options for high-risk women include:**

A) Regular **screening**,
B) **Prevention** using hormonal agents such as Tamoxifen, Raloxifene or Aromatase Inhibitors (e.g. Letrozole)
C) **Prophylactic** bilateral mastectomy.

Types of Breast Cancer: Derived from the epithelial cells that line the terminal duct lobular unit.

Non-Invasive (In-situ)	Invasive
- Remain **within the basement membrane** of the tubule and draining ducts. - **Two types:** **A) Ductal Carcinoma In Situ (DCIS):** **The most common**. In mammogram there's microcalcification, which could be either localized or widespread. **B) Lobular Intraepithelial Neoplasia.** Incidental finding. **Managed** with regular follow-ups.	- **Move out from the basement membrane** of the ducts and lobules into the surrounding tissues. - Two types: A) **Ductal,** which has five subtypes: Well differentiated. Good prognosis I) Tubular. II) Cribriform. III) Mucinous. (produce mucin) IV) Papillary. V) Medullary. (High-grade, pleomorphic cells surrounded by lymphoid cells) B) **Lobular**. (Large at the time of diagnosis)

Grading:

- Presence or absence of glands.
- The extent of nuclear pleomorphism.
- Mitotic rate of the tumor.
  Grade I → Best differentiated, best prognosis.
  Grade II → Intermediate prognosis.
  Grade III → (high-grade) Poor prognosis.
- The presence of tumor cells in lymphatics or blood vessels is a marker of **aggressive disease** & is associated with increase local and systemic recurrence.

Hormones and Growth Factors

Hormones

- Estrogen Receptors **(ER)** expressed in large amounts in cancer cells compared to a normal tissue.
- Depriving cancer cells from estrogen inhibits them for growing.
- If the cancer cells are –ve for ER or PgR (progesterone receptor) → **ineffective hormonal therapy.**

Growth Factors:

- **HER2** is the most important receptor in Human Epidermal Growth Factor Receptors.
- 15 – 20% of cancers have this receptor activation.
- Associated with poor prognosis.
- Drugs that block this receptor:
  1) **Trastuzumab** (Hereceptin)
  2) **Laptanib** (blocks HER1 & HER2)
  3) **Pertuzumab** (blocks HER1, HER2 & HER3)
- **Triple negative (ER, PgR, HER2) seen in women with BRCA1 gene abnormalities → Worse outcome.**

Screening

- **Core Biopsy is the gold standard of diagnosis.**
- **Mammography is the best screening tool.**
- Cancer most commonly appears to as a dense opacity and an irregular outline.
- Microcalcification. (Alone?) → **DCIS.**
- Skin tethering or thickening.
- Distortion of the shape of the breast.
- Tenting or direct involvement of underlying muscle.

Notes for staging:

- **If < 4 cm mass**, with no symptoms, no Investigations for metastases are required. **BUT** check the axillary lymph nodes, by FNAC, ultrasound, or core biopsy.
- **If > 4 cm mass**, do bone scan & ultrasound for the liver.
- **Simple staging;**

A) Operable.
B) Locally advanced.
C) Metastasis.

The Curability of Breast Cancer:

- Prognostic Factors:

A) Stage.
B) Genetic factors.
C) Grading.

- **The most important prognostic factor is the the number of axillary node involved.**
- The Nottingham Prognostic Index is the most widely used. (Next page)

Table 19.6 TNM Staging for breast cancer

T (Primary tumour)

- T_X Primary tumour cannot be assessed
- T_0 No evidence of primary tumour
- T_{is} Carcinoma in situ: intraductal carcinoma, lobular carcinoma in situ, or Paget's disease of the nipple with no associated tumour mass[1]
- T_1 Tumour 2.0 cm or less in greatest dimension[2]
- T_{1a} 0.5 cm or less in greatest dimension
- T_{1b} More than 0.5 cm but not more than 1.0 cm in greatest dimension
- T_{1c} More than 1.0 cm but not more than 2.0 cm in greatest dimension
- T_2 Tumour more than 2.0 cm but not more than 5.0 cm in greatest dimension[2]
- T_3 Tumour more than 5.0 cm in greatest dimension[2]
- T_4 Tumour of any size with direct extension to chest wall or skin
- T_{4a} Extension to chest wall
- T_{4b} Oedema (including peau d'orange), ulceration of the skin of the breast or satellite nodules confined to the same breast
- T_{4c} Both of the above (T_{4a} and T_{4b})
- T_{4d} Inflammatory carcinoma

N (Regional lymph nodes)

- N_X Cannot be assessed (e.g. previously removed)
- N_0 No regional lymph node metastasis
- N_1 Movable ipsilateral axillary lymph node(s)
- N_2 Ipsilateral lymph node(s) fixed to one another or to other structures
- N_3 Ipsilateral internal mammary lymph node(s)

M (Distant metastases)

- M_X Cannot be assessed
- M_0 No distant metastasis
- M_1 Distant metastasis present (includes metastasis to ipsilateral supraclavicular lymph nodes)

Note: Chest wall includes ribs, intercostal muscles and serratus anterior muscle, but not pectoral muscle.

[1]Paget's disease associated with tumour mass is classified according to the size of the tumour.

[2]Dimpling of the skin, nipple retraction or other skin changes may occur in T_1, T_2 or T_3 without changing the classification.

Management of Operable Breast Cancer

In Situ Breast Cancer & Operable Breast Tumors:	Local Therapy	Systemic Therapy
- Localized DCIS **< 4cm** → wide complete excision + radiotherapy post-operatively. - **Low grade** DCIS → Excision without radiotherapy. - Tamoxifen decrease recurrence & contralateral malignancy in those with +ve ER. - Widespread DCIS **> 4cm** → Mastectomy. - **Operable Breast Disease → T1, T2, T3, N0, N1, M0**	**A) Breast Conserving Therapy:** - Wide local excision + radiotherapy for 4-5 weeks, of 45-50 Gy dose followed by 10-15 Gy. - **Excision of the tumor with additional 1cm of the surrounding normal tissue.** - **Only suitable for < 4cm (both invasive and non-invasive)** **B) Modified Radical Mastectomy:** - Consists of total removal of the breast together with sentinel node biopsy, or axillary clearance. - Considered in the following; 1) Large tumor. 2) Unavailable radiotherapy. 3) Patient's wishes. 4) Who has localized invasive cancer with large, surrounding non-invasive cancer. 5) When breast conservation gives an unacceptable cosmetic outcome. - **Patey's Surgery** → pectoralis minor is excised. - **Auchincloss' surgery** → pectoralis minor is retracted. - **Scanlon's surgery** → the pectoralis minor is divided.	- Could be given before or after surgery or radiotherapy. - Adjuvant therapy: Chemotherapy, or hormonal therapy. ER +ve → Hormonal therapy. ER -ve → Chemotherapy. It decreases the risk of relapse. **A) Adjuvant chemotherapy:** - Four cycles post-operatively. - Chemotherapy will be offered to all except for those with poor performance, or tumor size < 1 cm, or grade I or absent vascular or lymphatic invasion. - Regimens that contain Anthrcyclines (Adriamycin/Doxorubicin) are more effective than others. **B) Adjuvant Hormonal Therapy:** - Consists of oophorectomy, tamoxifen. - Aromatase inhibitors (Letrozole) are more effective than tamoxifen. **C) Primary Systemic Therapy:** - The usage of chemotherapy (3-4 cycles) preoperatively to allow Breast Conservative instead of radical modified mastectomy.

Complications of Therapy

Axillary Surgery	Radiotherapy	Chemotherapy	Hormonal Therapy
Intercostobrachial nerve damage → numbness and paresthesia down the upper inner aspect of the arm. Long thoracic n. damage → winging of the scapula. Thoracodorsal n. damage → atrophy of latissimus dorsi and prominence of scapula. Frozen shoulder → short term reduction in shoulder movement. Lymphoedema → Bandaging and supportive elastic arm stalking.	- Erythematous reaction. (avoid sunlight) - Fibrosis around the shoulder → restriction of movement.	- Hair loss (mostly) - Fatigue and lethargy. - Alopecia → reduced by scalp cooling. - Nausea and Vomiting. - Usage of Trastuzumab → Cardiac Failure.	- Vaginal dryness or discharge. - Loss of libido. - Muscular aches. - Fractures. **Vaginal discharge and loss of libido are rare in Letrozole.**

Options of Reconstructions:

- Placement of an implant behind the chest wall muscles at the time of mastectomy.
- Expander behind the pectoralis muscle.
- Myocutaneous flap (most commonly latissimus dorsi)

Follow-up: The aim is → to detect local recurrence at a stage when it is treatable and patients should have annual mammography for one or both breasts, for life.

Management of Locally Advanced Breast Cancer (LABC):

- LABC characterized by infiltration of skin or chest wall (inoperable) which will may lead to:

A) Inflammatory cancer present with erythema

B) +/- widespread orange peel appearance affecting the skin.
Skin edema from obstruction of dermal lymphatics by tumor cells gives rise to orange peel skin.
The pits are because skin is tethered by hair follicles and sweat gland ducts.

- Inflammatory carcinomas have the worst prognosis of all LABCs.
- Management → Systemic Therapy → Surgery → Radiotherapy.
- If inoperable → Systemic Therapy → Radiotherapy.
- Systemic Therapy is either chemotherapy (3-4 months) or Hormonal Therapy (5 years).

Breast Cancer in Pregnancy.

- **Management: 1st two trimesters** → Mastectomy, avoid radiotherapy, and you can give chemotherapy but it could side effects on the fetus.
  3rd Trimester → Surgery or monitoring & deliver the fetus after the 32 week.

Management of metastatic or advance breast cancer:

The primary aim is to improve symptoms as well as improving the quantity and quality of life.

A) Chemotherapy:
The most common used drugs in metastasis breast cancer are the Anthracyclins and Epirubicin.

B) Hormonal Therapy:
Premenopausal → oophorectomy + radiation + Tamoxifen.
Postmenopausal → Letrozole + progestogens.

C) Anti-HER2 Therapy:
Trastuzumab → HER2.
Laptanib → HER1 & HER2.

Surgery in Metastatic Breast Cancer:

1) **Bone Disease** → External beam radiotherapy, NSAIDs, opiate, and bisphosphonate. Internal fixation and radiotherapy should be when fractures are likely.
2) **Hypercalcemia** → Hydration + saline and Bisphosphonate.
3) **Marrow Infiltration** → Chemotherapy.
4) **Spinal Cord Compression** → Surgery, radiotherapy and systemic therapy. In those who are not fit use steroids and fractionated radiotherapy.
5) **Pleural Effusion** → Tube drainage.
6) **Liver Metastases** → Chemotherapy (usually). +ve ER → Letrozole. Where jaundice is due to nodal disease at the porta hepatis, a stent is inserted in the common bile duct using ERCP.

7) **Brain Metastases** → Diagnosed with MRI/CT. High-dose of corticosteroids followed by radiotherapy. If isolated brain metastasis → Local excision + radiotherapy + appropriate systemic therapy.

Miscellaneous Tumors of The Breast:

1) **Lymphoma**: Characterized by discrete smooth rubber mass.
2) **Sarcomas**: Induced by radiotherapy. Treated by excision.
3) **Malignant Phyllodes Tumors:** Initial treatment is wide excision or mastectomy.
4) **Secondary tumors:** Produce a well-defined mass both clinically and mammography.

Gynecomastia	**Male Breast Cancer**
- The Growth of breast tissue in males to any extent in all ages. - Benign and reversible. - Persistent enlargement is indication for surgery. - Senescent Gynecomastia: affect men between 50 and 80. - Causes include; alcoholism, drugs including cannabis, cirrhosis, hypogonadism, testicular tumor. - **Management** → Excision of glandular tissue with liposuction.	- Klinefelter's Syndrome and a strong family history are the risk factors. - Associated with BRCA2 mutation. - Present with eccentric breast mass or retraction of the overlying skin. - Mammography and core biopsy to confirm. - **Management** → Breast conserving surgery or total mastectomy, and the removal of the sentinel or all the axillary nodes. Tamoxifen to reduce recurrence. Chemotherapy for fit patients with –ve ER.

Venous Diseases

- Normal ambulatory venous pressure (AVP) shouldn't exceed **25mmHg** at the level of the ankle.
- **Long standing hypertension can then result in:** 1. Varicose veins. 2.Chronic venous insufficiency
- AVP is the minimal pressure in foot veins on walking (Normally ≤ 25mmHg), on motionless standing, venous pressure can reach 90mmHg, this pressure fall by 60-80% in few seconds after walking.
- Causes of increases AVP: Primary superficial valve defects, AV fistula, DVT and Tumor beside deep veins.

Varicose veins

- **Most common vascular disorder**, can be saphenous or non-saphenous.
- F > M.

Risk factors	Accelerators (when the risk is there they accelerate their occurrence)
- **Female gender** - **Advance Age** - **Caucasian race** - **Family history.**	- Pregnancy - Obesity - Professions need long standing - Oral contraceptives

Types of varicose veins:

Telangiectasia	Reticular veins	Trunk varices(varicose vein)
Dilated intradermal venues less than 1mm **in diameter**	Dilate bluish tortuous sub dermal veins **1-3mm** in diameter	Subcutaneous dilated, elongated, tortuous veins **grater than 3mm**

Chronic venous insufficiency

- CVI collectively describes the manifestations of impaired venous return due to abnormal venous system function.
- Primary causes (superficial veins)**: related to structural weakness of valves (Floppy valves) or venous wall as in primary varicose veins.** Tx: Surgery or conservative.
- Secondary causes (deep veins) **include those due to previous DVT as in post-phlebitis syndrome, a tumor blocking deep veins , AV fistula or Pregnancy .** Tx: Treat the underlying cause.

Clinical picture

Asymptomatic → Pain → Swelling and leg heaviness → Itching → Skin pigments

- Severity of symptoms and signs depend on the degree and duration of venous hypertension.
- Patients can also manifest with complications such as Bleeding, Leg ulcers, Lipodermatosclerosis and Superficial thrombophlebitis.
- Leg ulcers are painful and primarily located near the ankle; their development can be preceded by brownish pigmentations. (Venous ulcer located around the ankle on the medial aspect).

Investigations

1) **Doppler ultrasound**

- It gives a qualitative assessment of venous reflux as well as an evaluation of reflux in Sapheno-femoral & popliteal junctions.

Duplex Scan: best initial test, save in pregnancy

- Direct detection of valvular efflux, Visualization of valve leaflet motions, Quantify degree of incompetence.

Physiologic reflux < 0.5sec.
Pathologic reflux > 0.5sec.

2) **Plethysmography:** Detects volume change of limb secondary to changes in venous blood flow
3) **Pressure measurements:** 1. Transmural pressure 2. Ambulatory venous pressure (AVP).

AVP abnormal results if:

- **Lack of sufficient drop: p**ressure doesn't decrease enough on walking and the **difference between the standing and walking pressure is <50%**
- **Short venous refill time: It takes less than 20 seconds**. (This means the blood is filling veins quickly and the valves aren't working efficiently to stop the blood from

4) **Invasive Procedures**

- Ascending venography, Descending venography, CT Venography and MRV.
- **Venography: the gold standard:** Contrast injected to visualize veins, not used much nowadays, due to its complications. But still has specific indication.

In order to standardize the reporting and treatment of the diverse manifestations of chronic venous disorders, a comprehensive classification system (CEAP) has been developed to allow uniform diagnosis.

Treatment Options

Varicose veins	Chronic venous insufficiency
• Conservative treatment Many patients with uncomplicated VV can be managed conservatively in primary care. Elastic support hose, weight reduction, regular exercise and the avoidance of constricting garments and prolonged standing all help to relieve tiredness and reduce swelling. • Surgery **(gold standard)** - VV surgery aims to remove varices and intercept incompetent connections between deep and superficial veins so that further varices do not form. - In patients with GSV disease, the SFJ is ligated flush with the femoral vein. - Recurrence is very much less likely if the LSV is stripped out from knee to groin. - Care must be taken not to damage the saphenous nerve, which joins and runs with the GSV below the knee. • Endovenous treatment - Surgery is being increasingly replaced by a range of minimally invasive endovenous treatments that can be performed under local anaesthesia as a day case or even as an outpatient procedure. The techniques include: • Radiofrequency ablation (RFA) • Endovenous LASER ablation (EVLA) • Ultrasound guided foam sclerotherapy (UGFS)	• Medical therapy Patients with leg ulcers often have multiple medical co-morbidities, the treatment of which must be optimal if the chances of ulcer healing are to be maximized (If b-haemolytic streptococcus or *Staphylococcus aureus* is cultured, oral antibiotics guided by sensitivities are indicated.Topical antibiotics and antiseptics are contraindicated.). • Dressings • Compression therapy (mainstay of treatment) - To be maximally effective, compression should be: ▪ elastic, ▪ multilayer, ▪ graduated, with the pressure greatest at the ankle (c. 30–40 mmHg) and least at the knee (c. 15–20 mmHg). - It is vitally important to exclude arterial disease before compression is applied • Surgical and endovenous therapy Eradication of superficial venous reflux by means of surgery or endovenous treatment in addition to compression therapy definitely reduces CVU recurrence and probably increases CVU healing rates when compared to compression alone.

Deep vein thrombosis (DVT)

- DVT originates in lower extremity venous system Starts at calf veins &progress proximally into popliteal, femoral, iliac veins & IVC 80-90% of PE originate here.
- **Virchow's Triad: > 100 yrs ago Virchow described triad of factors for the development of venous thrombosis**
  - Alteration in normal blood flow
  - Injury to vascular endothelium

- Alteration in constituents of blood

DVT Symptoms:

- **Sudden Swelling**
- **Limb pain** → pain on dorsiflexion of foot with knee flexed 90 degrees (Homan's sign)→ not sensitive or specific.
- **Dilated veins, cyanosis,pallor.**

- Extensive thrombosis in thigh and pelvis may lead to **Phlegmasia Alba Dolens (inflammation white pain),** Associated with arterial spasm and Painful pale with poor or absent pedal pulses.

Diagnosis:

D-dimer Assay

- Fibrin fragment present in fresh fibrin clot & FDP of cross linked fibrin
- Marker for action of plasmin on fibrin
- Sensitivity 98.4% (low specificity)
- Also elevated in trauma, recent surgery, haemorrhage, sepsis, cancer, pregnancy, liver disease.

Duplex

- **96% sensitive & 100% specific**
- In DVT vein is dilated , lumen shows thrombus and non compressible veins with poor or no venous flow.

Venography

- DVT is visualized as filling defect
- Invasive
- Contrast complications

Prevention:

- 5000 units of heparin 2 hours preoperatively than every 12 hours post operatively (5 days or until discharged).

Treatment:

Uncomplicated DVT:

1. Bed rest is unnecessary and the patient can be mobilized immediately, wearing an appropriately fitted compression stocking.
2. LMWH given by intermittent subcutaneous injection.

Complicated DVT: because of one or more of the following:

1- The DVT is more extensive (iliofemoral, vena cava, phlegmasia)
2- The DVT is recurrent
3- The patient has had a PE
4- The patient has one or more major irreversible congenital and/or acquired thrombophilia
5- Heparinization is contraindicated.

Insert a caval filter or consider thrombectomy.

Lymphedema

- **Primary lymphedema:** Developmental error in lymphatic vessels, depending on the severity it appears.
    - At birth < year – lymphedema congenital – familial - Milroy;s Disease
    - Between 1-35yr – lymphedema precox
    - Later >35yr - lymphedema tarda

- **Secondary lymphedema:**
  • Trauma, infections, filariasis*, post plebitic limb, irradiation, malignancy, allergy.

Clinical features: Lymphedema is like other forms of edema, in that it is present only upon dependency; that is, **worse at the end of the day and absent in the morning**.

Diagnosis:

- Lymphoedema is essentially a clinical diagnosis and most patients require no further investigation.
- Lymphangiography (invasive, inject contrast)
- Isotope lymphosintigraphy (best initial test)

Management:

- The patient should elevate the foot above the level of the hip when sitting, elevate the foot of the bed when sleeping, and avoid prolonged standing. Various forms of massage are effective at reducing oedema.
- **Surgical treatment**
    - Reducing Surgery
    - Bypass surgeries **(best procedure)**
        - lymph venous shunts.

Vascular investigations

Ankle to brachial pressure index (ABPI):

- The severity of ischemia in the leg can be simply estimated by determining the ratio between the ankle and brachial blood pressures. The latter is recorded in the normal way, the former using an ankle cuff and a hand-held Doppler device.
- In health, the ankle to brachial pressure index (ABPI) should be at least 1; that is, the pressure at the ankle should be at least as high as that in the arm.
- Patients with Intermittent Claudication usually have an ABPI of 0.5–0.9, in CLI usually the ABPI is less than 0.5.

Color flow duplex ultrasound:

- Color duplex ultrasound imaging has largely replaced conventional venography in the diagnosis of DVT
- It is non- invasive, avoids ionizing radiation and contrast, and is as accurate as venography in most cases
- At times of doubt, MR or CT venography may be useful.

Specific organ trauma

Spleen

Anatomy:

- The spleen is a vascular organ lying in the left upper quadrant of the abdomen alongside the 9th, 10th and 11th ribs, and is usually impalpable.
- The convex outer surface and superior pole lies against the diaphragm, the concave inner surface is related to the fundus of the stomach, the tail of the pancreas and the upper pole of the right kidney, and its lower pole rests on the splenic flexure of the colon below.
- Arterial inflow is primarily through the tortuous splenic artery arising from the coeliac axis, which carries 40% of the splanchnic blood flow into the spleen
- Venous blood drains into the portal venous system via the splenic vein
- The spleen has a secondary vascular inflow and outflow via the short gastric vessels that run within the lienogastric ligament to the upper part of the greater curvature of the stomach, which assume importance when the main splenic vessels are occluded through surgical division, radiological embolization or spontaneous thrombosis.

Trauma:

- The potential for injury is relatively obvious through a history of blunt or penetrating trauma

- Cardinal features in splenic injury: blood loss and signs of peritoneal irritation (peritonitis – left shoulder tip pain)
- Management: 80% of splenic injuries may be managed conservatively, and of these the requirement for interven- tion is apparent within 72 hours in 95%.

1. In the responding patient, cross-sectional imaging is advised
2. If the patient doesn't respond do not respond to initial resuscitation require an emergency laparotomy and usually a splenectomy along with a careful exploratory laparotomy to exclude injury to other structures.
3. Unstable patient, control of hemorrhage and restoration of circulating volume are paramount and consideration regarding organ preservation is of secondary importance
4. Unlike an elective splenectomy a midline laparotomy is usually performed with packing of the left upper quadrant which will normally control the splenic haemorrhage to allow the remainder of the abdomen to be examined
5. if examination of the spleen reveals bleeding from the splenic hilum, preservation is not appropriate and a splenectomy should be performed.

- Indications of splenectomy: Blunt – Penetrating – Iatrogenic intraoperative – Endoscopic trauma.

Liver

Anatomy:

- The liver is divisible into right and left hemilivers (each having four segments) using a line running from the gallbladder fossa to the inferior vena cava.
- Each hemiliver receives a branch of the hepatic artery and portal vein; 75% of the liver blood flow and 50% of its oxygen supply are provided by the portal vein.
- The hepatocytes are arranged in lobules, each of which has a central branch of the hepatic vein and peripheral portal tracts (containing a branch of the hepatic artery, portal vein and bile duct)
- It extends from the fifth intercostal space to the right costal margin.
- It is triangular in shape, its apex reaching the left midclavicular line in the fifth intercostal space.
- In the recumbent position, the liver is impalpable under cover of the ribs.
- The liver is attached to the undersurface of the diaphragm by suspensory ligaments that enclose a 'bare area', the only part of its surface without a peritoneal covering
- Its inferior or visceral surface lies on the right kidney, duodenum, colon and stomach
- The liver is divided by the attachment of the falciform ligament into right and left lobes; fissures on its visceral surface demarcate two further lobes, the quadrate and caudate
-

Trauma:

- After the spleen, the liver is the solid organ most commonly damaged in abdominal trauma, particularly following road traffic accidents.
- Stab injuries and gunshot wounds of the liver are also increasing in incidence.

Common Neck Swelling

1-Thyroid gland

Surgical anatomy and development

The lobes of the thyroid lie on the front and sides of the trachea and larynx at the **level of the 5–7th cervical vertebrae**

It is invested by the pre-tracheal fascia, which binds it to the larynx, cricoid cartilage and trachea. The strap muscles (sternohyoid and sternothyroid) lie in front of the pretracheal fascia and must be separated to gain access to the gland.

The superior thyroid artery runs down to the upper pole of the gland as a branch of the external carotid artery, whereas **the inferior thyroid artery** runs across to the lower pole from the thyrocervical trunk (a branch of the subclavian artery).

Blood drains through superior, middle and inferior thyroid veins into the internal jugular and innominate veins.

The superior laryngeal nerve (also a branch of the vagus) runs with the superior thyroid vessels.

Enlargement of the thyroid gland (goitre):

It is a visible or palpable enlargement of the thyroid. The swelling characteristically moves upwards on swallowing because of the gland's attachment to the trachea. All forms of thyroid cancer can produce goitre. Lymphoma and anaplastic tumours may cause diffuse thyroid swellings. Medullary and follicular tumours are often solitary swellings

Assessment of thyroid disease

1-Measurement of T3, T4 and TSH

2-ultrasonography

3-radioisotope scanning (99mTc-sodium pertechnetate. "Hot,warm or cold nodule")

	Non-toxic nodular goitre	Thyrotoxic goitre			
		Autoimmune thyroiditis (Hashimoto's disease)	Solitary thyroid nodules	Primary thyrotoxicosis (Graves' disease)	Toxic multinodular goitre and toxic adenoma
Aetiology and pathophysiology	Endemic (like iodine deficiency), sporadic or a reaction to drugs	Destruction of thyroid follicles by lymphocytes. Antibodies are detected in the serum against thyroglobulin, thyroid cell cytosol and microsomes.	Half are benign adenomas and the rest are cysts or differentiated cancers.	An autoimmune disease in which TSH receptors in the thyroid are stimulated by circulating thyroid receptor antibodies (TRAbs). TRAbs can cross the placental barrier, so that neonatal thyrotoxicosis can occur.	One or more nodules become hyperactive and begin to hyperfunction independently of TSH levels.
Clinical features	Asymptomatic, tracheal compression and dyspnea, dysphagia, pain or respiratory distress due to retrosternal goiter.	-Start with thyrotoxicosis, then euthyroid and finally with hypothyroidism. -Female to male ratio: 10:1 - Diffusely enlarged and firm thyroid. - Lymphoma could occur as a complication	- single thyroid nodule	-(male:female ratio 1:8) -Diffusely enlarged and soft thyroid -Bruit may be audible because of high vascularity - Causes a lot of symptoms.	-Eye signs are rare -more common in older women -Frequent cardiac complications such as arrhythmias
Investigations	T3, T4 and TSH are usually normal. Chest X ray may reveal tracheal deviation and CT scan may show tracheal compression.	1-antithyroid antibodies 2-Biopsy for cytology	The diagnostic test is fine-needle aspiration cytology, complemented by ultrasonography, isotope scans and thyroid function tests.	Raised T3 and T4 levels, coupled with low TSH levels, are confirmatory.	In a toxic multinodular goitre, the isotope scan demonstrates one or more areas of increased uptake. In toxic adenoma, the nodule is 'hot' and the remainder of the gland is 'cold'.
Management by surgery	Large goitres and those causing symptoms of compression require total or subtotal thyroidectomy. Some patients request surgery for cosmetic reasons.	Thyroidectomy is rarely needed.	Depends on the nature of the nodule	-Antithyroid drugs (Carbimazole) -Radioactive iodine : Many consider this to be the treatment of choice. Not used in pregnant ladies. Might lead to hypothyroidism. -Surgery: Thyroidectomy is a highly successful form of treatment. Total thyroidectomy being the operation of choice. -Before surgery, patients must be rendered euthyroid with antithyroid drugs. β-Adrenergic blocking drugs can be used to block the sympathetic activity .	By total thyroidectomy (multinodular goitre) or lobectomy (toxic adenoma), or if surgery is contraindicated, radioiodine.

Other rare causes of goiter:

Subacute thyroiditis (de Quervain's disease)

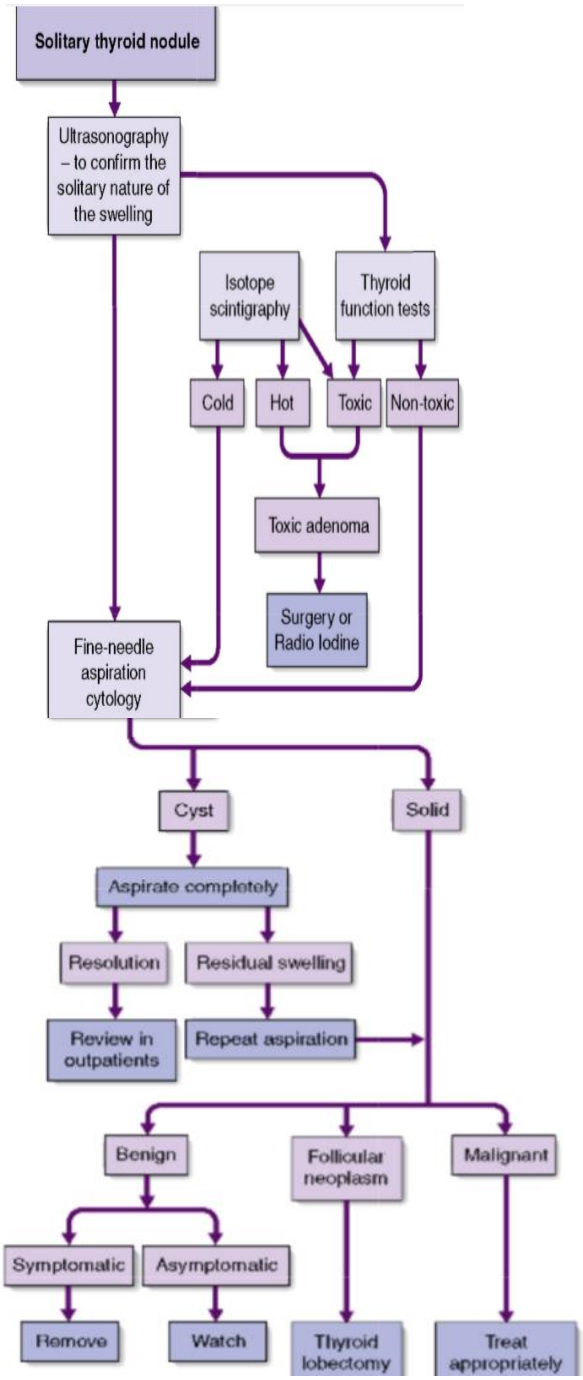
Rare condition. it is associated with an influenza-like illness. Diffuse painful swelling. The disease may be due to a viral infection and usually resolves spontaneously.

Riedel's thyroiditis

Very rare condition and the cause is unknown. Firm painless swelling and tracheal compression. Decompression of the trachea may be required

Symptoms of thyrotoxicosis:

Heat intolerance, skin is moist and warm, weight loss, Tachycardia, arrhythmia, tremor, exophthalmos, anxiety, ophthalmoplegia, pretibial myxoedema, proximal muscle myopathy, menstrual irregularity and infertility can occur.



Malignant tumours of the thyroid

The two main types of thyroid carcinoma are papillary (50%) and follicular (30%), with the remainder comprising medullary carcinoma, anaplastic carcinoma and lymphoma.

There might be a history of ionizing radiation exposure.

Enlarged lymph nodes may be the only finding in some patients with a microscopic primary thyroid cancer. (the so-called 'lateral aberrant thyroid')

Notes:

-Cardiac failure, obstructive airways disease and diabetes (where they may mask hypoglycaemic symptoms) are contraindications to the use of β-blockers.

- A cytopathologist cannot distinguish between a follicular adenoma and follicular carcinoma; this can only be achieved on definitive histopathology by looking for capsular or vascular invasion.

Thyroid cancer	Papillary carcinoma	Follicular carcinoma	Anaplastic carcinoma	Medullary carcinoma	Lymphoma
Clinical features	-before the age of 40 -solitary thyroid swelling -Enlarged lymph nodes -Could be discovered as an incidental finding	-solitary thyroid nodule -Patients aged 30–50 years. -haematogenous spread -Vascular and capsular invasion distinguish it from a benign follicular adenoma.	-rapidly growing, highly malignant tumors -older patients. -Hoarseness of voice, dyspnea and dysphagia due to invasion of adjacent strucures. -Horner's syndrome	-arises from the parafollicular C cells -50% of patients the cervical lymph nodes are involved -could be part of MEN syndrome type II (Sipple's syndrome). Calcitonin levels are elevated - Mutation of Ret proto-oncogene -Prophylactic thyroidectomy at different ages depending on the specific mutation and level of risk associated with that mutation.	It is a rare complication of autoimmune thyroiditis.
Management and prognosis	-Bilateral total lobectomy if it is multifocal. -Microscopic disease (< 1cm and unifocal) treated by single lobectomy alone. -lymph nodes usually identified by preoperative ultrasound scanning. -Isotope scan should be performed postoperatively to identify any iodine uptake in the neck or elsewhere. -The disease has an excellent prognosis, with 10-year survival rates approaching 90%.	-Treatment consists of total thyroidectomy with preservation of the parathyroids. -If a postoperative radioisotope scan (challenge scan) reveals increased uptake in the skeleton or neck, therapeutic doses of radioiodine are given. -Plasma thyroglobulin levels should be undetectable after surgery and radioiodine therapy, if high level=recurrence. -The disease is more aggressive than papillary carcinoma, with 10-year survival rates approaching 75%.	Resection is rarely appropriate but surgery can relieve tracheal compression. External beam radiotherapy may be of value.	Treatment consists of total thyroidectomy and, if the calcitonin level is raised, dissection of the lymph nodes in the central compartment of the neck. Preoperative CT of the neck and mediastinum is advised and the exclusion of a phaeochromocytoma before neck surgery is mandatory.	It is treated by radiotherapy and chemotherapy. Patients often require core biopsy of the gland to characterize the type of lymphoma. -CT is used to stage the disease fully.

Note: The age for prophylactic thyroidectomy in MEN syndrome varies from age 1 for the 918 mutation through to age 5 for the commonest 634 mutation and in some good prognosis families may be delayed to age 13.

Thyroidectomy

Technique

The recurrent laryngeal nerves should be identified, so that they can be protected from injury. Generally nothing less than a total lobectomy should be performed, to avoid the need for reoperation on that side.

Complications

Haemorrhage

Nerve damage

-The external branch of the superior laryngeal nerve injury: weak voice

-Recurrent laryngeal nerve injury: hoarseness of voice and stridor.

Hypothyroidism

Hypoparathyroidism

Scar complications: The scar can become hypertrophic or keloid.

2-Parathyroid glands

Surgical anatomy

The parathyroid glands receive a rich blood supply from the inferior thyroid artery

Primary hyperparathyroidism

Pathology

90% due to adenoma, 10% hyperplasia and less than 1% carcinoma.

Clinical features

-Female to male ratio is 2:1

-asymptomatic

-digestive symptoms, kidney stones, psychiatric abnormalities, and bone disease.

Diagnosis

High serum calcium and PTH (PTH may be normal) levels, low serum phosphate and raised 24-hour urinary calcium excretion. In familial hypercalcaemic hypocalciuria there will be low urinary calcium.

Management

Preoperative imaging with ultrasound and MIBI scans should be done. CT, MRI could be used. Adenoma is removed. In hyperplastic parathyroid glands we should remove three and half.

Secondary and tertiary hyperparathyroidism

There is over-secretion of PTH in response to low plasma levels of ionized calcium, usually because of renal disease or malabsorption. It is managed initially by giving 1-α-hydroxyvitamin D_3 (alfacalcidol) to increase calcium absorption. If it does not work Total or subtotal parathyroidectomy may be needed.

Hypoparathyroidism

Hypoparathyroidism may occur temporarily after parathyroidectomy until the suppressed residual glands assume normal function. This leads to Symptoms and signs of hypocalcemia. It is treated with intravenous calcium gluconate or oral calcium and vitamin D.

3-Other neck swellings:

Thyroglossal cyst

-The thyroid remains attached to the tongue by the thyroglossal duct. This duct should completely atrophy, otherwise a thyroglossal cyst may develop.

-It moves on swallowing or tongue protrusion and can become infected.

-Confirmation by an ultrasound scan

-Treatment is by surgical excision.

Skin and subcutaneous swellings

-Sebaceous cysts occur commonly in the head and neck and have a characteristic punctum.

-Treated by drainage or excision.

Branchial cyst and fistula

-Swellings lying laterally in the upper neck may be branchial cysts.

-Treated by excision

Carotid body tumors

- These are rare tumors arising from chemoreceptor tissue in the carotid body

- pulsatile swellings

-Diagnosed by angiography

-Treated by excision

Lymph node swellings

-Caused by: infection, primary tumor (lymphoma) , metastases or systemic diseases

- Careful examination of the upper aerodigestive tract is therefore mandatory in assessing an undiagnosed neck node.

- PET CT scanning of the neck, fine needle aspiration cytology and rigid endoscopy.

4-Summary boxes from the book

SUMMARY BOX 20.1

Goitres

- Physiological thyroid enlargement may occur during puberty or pregnancy
- Non-toxic nodular goitre can be associated with iodine deficiency and drug reactions; it is usually asymptomatic but can cause compression symptoms
- Thyrotoxic goitre results from stimulation of the gland by TSH or TSH-like proteins, resulting in excessive production of T_3 and T_4. About 25% of cases of thyrotoxicosis are due to a toxic multinodular goitre (a long-standing non-toxic goitre develops hyperactive nodule(s) that function independently of TSH levels)
- Thyroiditis can produce diffuse painful swelling that may be subacute (de Quervain's disease) or autoimmune (Hashimoto's disease). Riedel's thyroiditis is a very rare cause of painless thyroid swelling and tracheal compression
- A solitary thyroid nodule is often a conspicuous palpable nodule in a multinodular goitre. True solitary nodules may be adenomas, cysts or cancers, conditions that are distinguished by fine-needle aspiration cytology, ultrasonography, isotope scans and function tests
- Thyroid cancers can produce a goitre, particularly in the case of medullary carcinoma of the thyroid and lymphoma.

SUMMARY BOX 20.3

Hyperparathyroidism

- Serum calcium levels are normally controlled by parathormone (mobilizes calcium from bone, and increases renal calcium absorption and phosphate excretion) and vitamin D (promotes absorption from the intestine and augments effect of parathyroid hormone (PTH) on osteoclasts), with an uncertain contribution from calcitonin
- Hyperparathyroidism may be primary (90% adenoma, 10% hyperplasia, 1% carcinoma), secondary to renal disease/malabsorption (low serum Ca^{2+} triggers PTH secretion) or tertiary (development of autonomous secretion in secondary hyperparathyroidism)
- Hyperparathyroidism is now normally diagnosed while asymptomatic, but can produce renal effects (nephrocalcinosis, calculi and failure), skeletal effects (demineralization), gastrointestinal upsets (peptic ulcer, pancreatitis) and psychotic symptoms, i.e. 'stones, bones and groans'
- The diagnosis of primary hyperparathyroidism is supported by detection of circulating PTH in the presence of hypercalcaemia
- Primary hyperparathyroidism is treated surgically by removing an adenoma. If all four glands are involved by hyperplasia, all but a portion of one gland is removed.

SUMMARY BOX 20.2

Thyroid cancer

- Thyroid cancers may arise from the epithelium (papillary 50%, follicular 30%). Remainder comprise anaplastic, parafollicular C cells (medullary carcinoma) or lymphoreticular tissue (lymphoma)
- Papillary cancers are rare after the age of 40 years, are often multifocal and spread to lymph nodes, but rarely disseminate widely. Total or near-total thyroidectomy with the removal of involved nodes may be followed by radioiodine, and thyroid replacement therapy to suppress TSH. Ten-year survival rates approach 90%
- Follicular carcinoma occurs in the 30–50-year age group, spreads preferentially via the bloodstream, and is treated by total thyroidectomy. Residual neck or skeletal radioisotope uptake signals the need for radioiodine therapy. T_4 is used routinely to suppress TSH production. The 10-year survival rate is 75%
- Anaplastic carcinoma occurs in older patients, spreads locally and frequently gives rise to pulmonary metastases. Curative resection is rarely possible, radiotherapy/chemotherapy is of little value, and most patients die within 1 year
- Medullary carcinomas secrete calcitonin, may involve both lobes, and involve neck nodes. They may be sporadic or part of MEN II. Treatment consists of total thyroidectomy and node dissection.

SUMMARY BOX 26.6

Salivary gland swellings

- Swellings in the submandibular gland are more often due to calculi, but those in the parotid gland are commonly benign neoplasms
- The most common salivary gland tumours are pleomorphic adenomas
- Parotid swellings generally require removal with a cuff of normal salivary tissue (superficial parotidectomy)
- The facial nerve runs through the parotid gland as a series of branches and is at risk during parotid surgery.

الحمد لله الذي بنعمته تتم الصالحات