Fifth Edition For Students and Teachers Fifth Edition Manipal Manual of the Book for Students and Teachers Fifth Edition

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Chapter

Wound, Keloid, Hypertrophic Scar and Metabolic Response to Injury

- □ Types of wounds: Classification
- □ General principles of management of open wounds
- Components of wound healing
- □ Factors affecting wound healing
- Compartment syndrome
- □ Hyperbaric oxygen
- Hypertrophic scar and keloid

- □ Classification of surgical wounds
- Healing of specialised tissues
- □ Metabolic response to injury
- Recent advances
- Description Negative pressure assisted wound closure
- □ Tissue engineering and regeneration
- □ What is new in this Chapter?/Recent advances

INTRODUCTION

Wound is a discontinuity or break in the surface epithelium. A wound is simple when only skin is involved. It is complex when it involves underlying nerves, vessels and tendons.

TYPES OF WOUNDS

A few classifications have been given below.

I A. Closed Wounds

- Contusion
- Abrasion
- Haematoma

Contusion: Can be minor soft tissue injury without break in the skin, or major such as when being run over by a vehicle. Generally, it produces discolouration of the skin due to collection of blood underneath.

Abrasion: In this wound, epidermis of the skin is scraped away exposing the dermis. They are painful as dermal nerve endings are exposed. These wounds need cleaning, antibiotics and proper dressings.

Haematoma: This refers to collection of blood usually following injury. It can occur spontaneously in patients who have bleeding tendencies such as haemophilia. Depending upon the site, it can be subcutaneous, intramuscular, intra-articular, even subperiosteal. A knee joint haematoma may need to be aspirated followed by application of compression bandage. Small haematomas get absorbed. If not, they can get infected.

B. Open Wounds

- Incised
- Lacerated
- Penetrating
- Crushed

Incised wounds: They are caused by sharp objects, such as knife, blade, glass, etc. This type of wound has a sharp edge and is less contaminated. Primary suturing is ideal for such wounds, as it gives a neat and clean scar.

Lacerated wounds: They are caused by blunt injury, such as fall on a stone or due to road traffic accidents (RTA). Edges are jagged. The injury may involve only skin and subcutaneous tissue or sometimes deeper structures also. Due to the blunt nature of the object, there is crushing of the tissue which may result in haematoma, bruising or even necrosis of the tissue. These wounds are treated by wound excision and primary suturing provided they are treated within six hours of injury.

Penetrating wounds: They are not uncommon. Stab injuries of abdomen are very notorious. It may look like an innocent injury with a small, 1 or 2 cm long cut but

internal organs such as intestines, liver, spleen or mesenteric blood vessels may have been damaged. All penetrating wounds of the abdomen should be admitted and observed for at least 24 hours. Layer by layer exploration and repair, though recommended, may not be possible at times due to oblique track of the wound.

Crushed or contused wounds: They are caused by blunt trauma due to run over by vehicle, wall collapse, earthquakes or industrial accidents. These wounds are dangerous as they may cause severe haemorrhage, death of the tissues and crushing of blood vessels. These patients are more prone to gas gangrene, tetanus, etc. Adequate treatment involves good debridement and removal of all dead and necrotic tissues.

II. Tidy and Untidy Wounds

- a. **Tidy wounds:** Incised, clean, healthy tissue and seldom associated with tissue loss (Key Box 1.1).
- b. **Untidy wounds:** Crushed or avulsed, contaminated, devitalised tissues and often with tissue loss.

Key Box 1.1

Repair in Tidy Wound

- Nerves: Fascicular repair under magnification (loupe or microscope) using 8–0 or 10–0 monofilament nylon
- Artery: To be repaired by using 6-0 prolene
- Tendon repaired by monofilament nonabsorbable suture (polypropylene 3–0 or 4–0)
- o Skin loss: Skin flap/skin graft

III. Acute Wound and Chronic Wound

- a. **Acute wound:** Stab wounds, following RTA and blast injuries.
- b. Chronic wound: Leg ulcers, pressure sores.

General Principles of Management of Open Wounds (Fig. 1.1)

- Admission or observation in the hospital.
- Monitoring of temperature, pulse and respiration.
- Systemic antibiotics depending upon the contamination of wound.
- Injection tetanus toxoid for prophylaxis against tetanus.
- Treatment of the wound in the form of cleaning, dressing or suturing.

Healing of the Wound

Healing by primary intention occurs in a clean incised wound such as a surgical incision wherein there is only

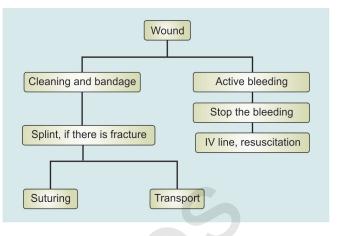


Fig. 1.1: Wound management

a potential space between the edges. It produces a clean, neat, thin scar.

Healing by secondary intention refers to a wound which is infected, discharging pus or wound with skin loss. Such wounds heal with an ugly scar.

COMPONENTS OF WOUND HEALING (Table 1.1)

I. Inflammatory Phase (Lag Phase)

- Injury results in the release of mediators of inflammation, mainly histamine from platelets, mast cells and granulocytes. This results in increased capillary permeability.
- Later, kinins and prostaglandins act and they play a chemotactic role for white cells and fibroblasts.
- In the first 48 hours, polymorphonuclear (PMN) leucocytes dominate. They play the role of scavengers by removing the dead and necrotic tissue (Figs 1.2 to 1.6).

II. Proliferative Phase (Collagen Phase)

- Between 3rd and 5th days, polymorphonuclear leukocytes diminish in number but monocytes increase. They are the specialised scavengers.
- By 5th or 6th day, **fibroblasts** appear, proliferate and eventually give rise to a protocollagen which is converted into collagen in the presence of an enzyme, protocollagen hydroxylase. O₂, ferrous ions and ascorbic acid are necessary for this step.
- Fibroplasia along with capillary budding gives rise to granulation tissue.

OTHER SPECIAL TYPES OF PYOGENIC INFECTIONS

BOIL (Key Boxes 2.5 and 2.6)

- This is also called furuncle. It is a hair follicle infection caused by *Staphylococcus aureus* or secondary infection of a sebaceous cyst.
- It starts with a painful indurated swelling with surrounding oedema. After about 1–2 days, softening occurs in the centre and a pustule develops which bursts spontaneously discharging pus. Necrosis of subcutaneous tissues produces a greenish slough. Skin overlying the boil also undergoes necrosis. *Hence, boil is included under acute infective gangrene.*
- Furuncle of the external auditory meatus is a very painful condition because of the rich nerve supply of the skin. Pain is also due to dense adherence of skin to the perichondrium (there is no subcutaneous tissue).

Treatment of Boil

Incision and drainage with excision of slough. Antibiotic cloxacillin is given. Diabetes, if present, is treated.

Complications of Boil

- Necrosis of the skin
- Pyaemic abscess and septicaemia.
- **Cavernous sinus thrombosis** due to boil on the face or stye on the eyelid.

CARBUNCLE (Figs 2.17A and B)

• This is an **infective gangrene** of the subcutaneous tissue caused by *Staphylococcus aureus* (Key Box 2.7). It commonly occurs in **diabetic patients**. Patients with poor immunity, or undergoing radiotherapy can also develop carbuncle.

Key Box 2.5

Precipitating Factors	Common Locations
o Scratching	Face and back of the neck
o Diabetes	Axilla
• Poor immunity	Gluteal region

Key Box 2.6

Facts about a Boil

o Dangerous boil	: On the skin of face
o Tender boil	: External auditory
o Sweet boil	: Diabetic patients
o Boil likes	: Oily skin
o Blind boil or dull boil	: Subsides without suppuration

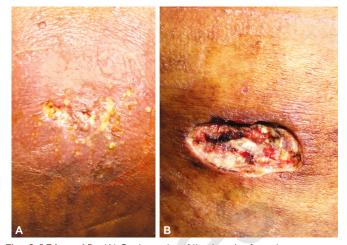


Fig. 2.17A and B: (A) Carbuncle of the back of neck—common site, sieve-like appearance, (B) after excision of the carbuncle wound heals within 2 to 3 weeks. Some cases require split skin grafting

Key Box 2.7

Staphylococcal Infections of Surgical Importance

- Boil • Carbuncle
- Parotitis Osteomyelitis
- Breast abscess
- Sites: Nape of the neck is the commonest site followed by back and shoulder region. Skin of these sites is coarse and has poor vascularity.

Pathology

The initial lesion is similar to a boil in the form of hair follicle infection with perifolliculitis. Since majority of patients are diabetics, infection takes a virulent course and results in necrosis of subcutaneous fat which gives rise to multiple abscesses. These abscesses are **intercommunicating** and they open to the exterior by multiple openings which are called **sieve-like openings**. This appearance is described as **cribriform** appearance which is pathognomonic of carbuncle.

Clinical Features

- Typically, the patient is a diabetic.
- Severe pain and swelling in the nape of the neck.
- Constitutional symptoms such as fever with chills and rigors are severe.
- Surface is red, angry looking like red hot coal.
- Surrounding area is indurated.
- Later, skin on the centre of carbuncle softens and peripheral satellite vesicles appear, which rupture discharging pus and give rise to a **cribriform** appearance (Fig. 2.17A).
- The end result is development of a large crateriform ulcer with central slough.

Complications

- 1. Worsening of the diabetic status resulting in diabetic ketoacidosis.
- 2. Extensive necrosis of skin overlying carbuncle. Hence, it is included under acute infective gangrene.
- 3. Septicaemia, toxaemia.

Treatment (Key Box 2.8)

Key Box 2.8

Summary of Carbuncle

- Caused by Cocci
- Abscesses **Communicating**
- o Red hot like Coal
- Appearance **Cribriform**, **Crateriform ulcer**
- o Gangrene **Cutaneous** (subcutaneous)
- Drug of choice Cloxacillin
- o Diabetes Control
- o Incision Cruciate

Pearls of Wisdom

Treatment of carbuncle is excision.

- Diabetes control, preferably with injectable insulin.
- Appropriate **parenteral antibiotics** are given till complete resolution occurs. Most strains of *Staphylococcus aureus* are sensitive to cloxacillin, flucloxacillin, erythromycin and some of the cephalosporins. However, *methicillin-resistant Staphylococcus aureus (MRSA)* bacteria are resistant to the drugs mentioned above. They are sensitive only to expensive drug **vancomycin** which has to be given intravenously.
- Improve general health of the patient.
- If carbuncle does not show any softening or if it shows evidence of healing, it is not incised. It can be left open to the exterior or saline dressings may be applied to reduce oedema. Complete resolution may take place within 10–15 days.
- Surgery is required when there is pus. *Cruciate incision* is preferred because of multiple abscesses and extensive subcutaneous necrosis. Edges of the skin flap are excised, pus is drained, loculi are broken down, slough is excised, and *cavity is irrigated with antiseptic agents*. Like pyogenic abscess, wound heals with granulation tissue from the depth (Fig. 2.18)—excision of carbuncle.



Fig. 2.18: Carbuncle with central slough. A cruciate incision should be given followed by excision.

ERYSIPELAS

Observe 8 Cs

- It is an acute inflammation of the skin and subcutaneous tissue associated with severe lymphangitis. Causative organism is *Streptococcus pyogenes*. Precipitating factors are malnourishment, chronic diseases, etc. Thus, children and old people are commonly affected.
- Infection sets in after a small scratch or abrasion and spreads very rapidly resulting in toxaemia. Sites: Face, eyelids, scrotum and in infants, the umbilicus.

Clinical Features

- **Rose-pink** rash with raised edge, appreciated on palpation and has a consistency of button hole.
- Vesicles appear later, rarely become pustular.
- Oedema of the eyelids or scrotum depending upon the site.
- Features of *toxaemia*
- When it occurs in the face, it involves pinna because erysipelas is basically a *cuticular lymphangitis*. This is described as *Milian's ear sign positive*. This sign is used to differentiate cellulitis of face from facial erysipelas. In cellulitis of face, pinna does not get involved because of close adherence of skin to the cartilage.

Complications

- 1. Toxaemia and septicaemia
- 2. Gangrene of skin and subcutaneous tissue
- 3. Lymphoedema of face and eyelids due to lymphatic obstruction causing fibrosis of lymphatics.

Treatment

Injection crystalline penicillin 10 lakh units 6th hourly IM/IV for 5–10 days.

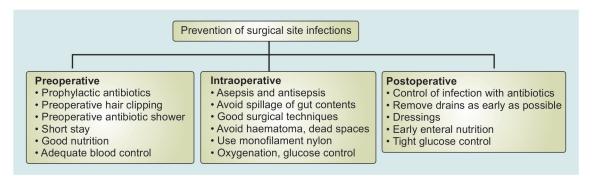


Fig. 2.24: Prevention of surgical site infections

- Autoclaving and sterilisation should be optimally done
- Proper ventilation of the wards
- Proper scrubbing before any procedure
- Proper disposal of urine, faeces, sputum
- Use of disinfectants.
- Antibiotic prophylaxis (Key Boxes 2.14 and 2.15).

ASEPSIS AND ANTISEPSIS

Strictly speaking, they are equivalent and there is not much of a difference between these.

Key Box 2.14

Antimicrobial Prophylaxis

Operation	Likely pathogens			
o Breast	S. aureus, coagulase-negative,			
	staphylococci			
• Appendicectomy	Gram-negative bacilli, anaerobes			
o Biliary tract	Gram-negative bacilli, anaerobes			
o Upper Gl	Gram-negative bacilli, strepto-			
cocci, oropharyngeal anaerobes				
(peptostreptococci)				
Cefazolin is generally accepted as the antimicrohial				

agent of choice for clean-contaminated operations.

- Dose: 1–2 g/adult dose
- **Timing:** No more than 30 minutes before skin is incised.

Prophylactic regimens

- **Vascular:** 3 doses of flucloxacillin with or without gentamicin, vancomycin
- **Oesophagogastric:** 1 dose of 2nd generation cephalosporin and metronidazole
- o Biliary: One dose of 2nd generation of cephalosporin
- **Small bowel:** 1 dose of 2nd generation of cephalosporin with metronidazole
- **Appendix/colorectal:** 1 dose of 2nd generation of cephalosporin with metronidazole

Key Box 2.15

Key Points in SSI

- SSI within 24 hours is caused by clostridia and streptococci
- SSI after 48 hours (5 days) is caused by gram-negative and other bacteria.
- Nasal carriers of *Staph. aureus* have ↑ risk of SSI.
- Prevention of SSI is by aseptic and antiseptic technique in OT as introduced by Lister, use of prophylactic antibiotics and patient's own ability to prevent infection.
- Skin to be prepared by germicidal antibiotics such as tincture of iodine, povidone iodine or chlorhexidine.
- The first dose of prophylactic antibiotics is given intravenously at the induction of anaesthesia.
- o Monofilament sutures are better to decrease the SSI.

Asepsis means precautions taken before any surgical procedure against development of infection. Some examples are: Wearing gloves before any procedure, cleaning the patient's abdomen with iodine and spirit, sterilisation of instruments and autoclaving.

Antisepsis: All surgical procedures today are done after taking aseptic precautions.

- Dressing of an already contaminated wound using carbolic acid, iodine.
- Broad-spectrum antibiotics are used in presence of infection.
- Wearing mask and cap in the operation theatre.

SINUS AND FISTULA

Sinus

- It is a blind track leading from the surface down into the tissues (Fig. 2.25). It is lined with granulation tissue. Following are a few examples:
 - 1. *Congenital sinus:* Pre-auricular sinus, post-auricular sinus (Fig. 2.27).

- 2. Acquired sinus:
 - *Median mental sinus (see* page 295): Occurs as a result of tooth abscess.
 - *Pilonidal sinus:* Occurs in the midline in the anal region (Fig. 2.27).
 - *Osteomyelitis:* Gives rise to sinus discharging pus with or without bony spicules (Figs 2.28 and 2.29)
- Most common sinus in the neck is due to tubercular lymphadenitis. It discharges cheesy material. Skin surrounding the sinus shows bluish discolouration.

Fistula

It is an abnormal communication between the lumen of one viscus and the lumen of another (internal fistula) or communication of one hollow viscus with the exterior, i.e. body surface (external fistula) (Fig. 2.26).

Examples of Internal Fistula

- Tracheo-oesophageal fistula
- Colovesical fistula

Examples of External Fistula

• Orocutaneous fistula due to carcinoma of the oral cavity infiltrating the skin

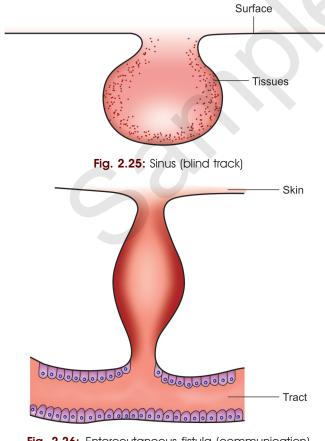


Fig. 2.26: Enterocutaneous fistula (communication)



Fig. 2.27: Postauricular sinus

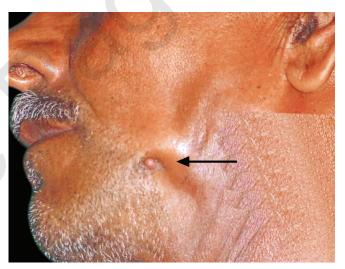


Fig. 2.28: Mandibular sinus due to badly infected caries teeth, osteomyelitis of mandible



Fig. 2.29: Tuberculous sinuses in the chest wall. Observe that the edges of the sinuses are in flush with the skin

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Multiple Choice Questions

1. Which of the following statement is false in tetanus?

- A. Exotoxins are produced by Clostridium tetani
- B. The toxin gets fixed to motor cells of anterior horn cells
- C. It stimulates the release of cholinesterase
- D. Period of onset is more important than incubation period

2. Favourable conditions for development of tetanus include following *except*:

- A. Injury
- B. Foreign body
- C. Devitalised tissues
- D. Aerobic conditions

3. Hyperbaric oxygen can be used in the following conditions *except*:

- A. Gas gangrene
- B. Decompression sickness
- C. Carbon monoxide poisoning
- D. Necrotising fasciitis

4. Following facts are true in gas gangrene except:

- A. It is caused by *Clostridium welchii*
- B. Severe myonecrosis is a feature
- C. Brownish fluid has foul odour
- D. Alpha toxin causes cell membrane damage

5. Positive Nagler reaction in gas gangrene is caused by:

- A. Lecithinase
- B. Beta toxin
- C. Proteinase
- D. Hyaluronidase

6. Sponge-like consistency of the part is typically seen in which condition:

- A. Necrotising fasciitis
- B. Diabetic ulcer leg
- C. Gas gangrene
- D. Pyomyositis

7. Following are true for Clostridium welchii except:

- A. Anaerobic B. Gram-positive
- C. Nonspore-bearing D. Produces a toxins
- 8. Following are clinical features of gas gangrene, except:
 - A. Crepitus
 - B. Khaki coloured skin
 - C. Low grade fever
 - D. Hypertension
- 9. Following are features of necrotising fasciitis type II *except:*
 - A. It is monomicrobial— β -haemolytic streptococci
 - B. It can occur in young healthy individuals also
 - C. Organism is Clostridium perfringens
 - D. It can give rise to toxic shock syndrome
- 10. Which of the following is false to prevent gas gangrene?
 - A. Antigas gangrene in risk groups
 - B. Prophylactic antibiotics
 - C. Blood sugar level should be maintained within 200 mg/dl
 - D. Avoid tourniquets while operating on crushed wounds in the legs

				Ansv	vers				
1. C	2. D	3. D	4. C	5. A	6. C	7. C	8. D	9. C	10. C

Section 1 • General Surgery

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C

Table 5.1	le 5.1 Comparison of tuberculoid leprosy with lepromatous leprosy			
		Tuberculoid leprosy	Lepromatous leprosy	
1. Cell-media	ated immunity	Strong	Low	
2. Histology		Giant cells, epithelioid cells, histiocytes, lymphocytes are present. Bacilli are few.	Bacilli distending the macrophages—'GLOBI'. Plenty of bacilli invading nerves, adnexa, sweat glands, etc.	
3. Clinical		Localised. Anaesthetic, hypopigmented, raised skin patch. Early nerve damage, nerve thickening is a characteristic feature. Involvement of face and nose is not seen.	Generalised. Erythematous multiple macular rashes. Nerve involvement is usually not seen. Leonine facies, collapse of the bridge of the nose are characteristic.	
4. Prognosis	3	Good	Not good	

Treatment

- 1. Lepromatous and borderline lepromatous leprosy (multibacillary disease)
 - 3-drug regimen is the most ideal treatment.
 - Dapsone 100 mg/day Clofazimine 50 mg/day Rifampicin 600 mg once monthly, supervised. Clofazimine 300 mg once monthly, supervised.

For a minimum period of 2 years. Skin smear should be negative.

- 2. Tuberculoid and borderline tuberculoid leprosy (paucibacillary disease)
 - Dapsone 100 mg daily
 - Rifampicin 600 mg once a month, supervised.
- For a period of 6 months.

DEFORMITIES IN LEPROSY

I. Primary Deformity (Figs 5.1 to 5.3)

- It occurs directly due to the disease.
- Face: It is involved in lepromatous leprosy and is described as leonine facies with multiple nodules over the face, pigmentation, *loss of lateral portion* of

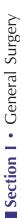




Fig. 5.1: Observe small muscle wasting in both the hands



Fig. 5.2: Autoamputation of the toes in a case of leprosy



Leonine facies, collapse of the bridge of the nose are a few other features of lepromatous leprosy

Fig. 5.3: Observe deformed ear

the eyebrows (madarosis), collapse of bridge of the nose due to destruction of nasal cartilages (warm and moist area) and paralysis of facial nerve.

- Hands: Involvement of ulnar nerve at the elbow and median nerve at wrist gives rise to *'claw hand'*.
- **Foot:** Posterior tibial nerve is involved at the ankle leading to clawing of the toes. Foot drop occurs when lateral popliteal nerve below the knee joint is involved.

----- Clinical Notes

MS exam case, KMC, Manipal (2004)

A 28-year-old lady complained of swelling in the posterior triangle of 8 months duration. It was a bit irregular with restricted mobility and it was tender. 3 candidates offered nonspecific lymphadenitis as a diagnosis. Only one candidate thought of nerve abscess. The clue was thickened nerve above. It was a case of Hansen's disease (Fig. 5.4).

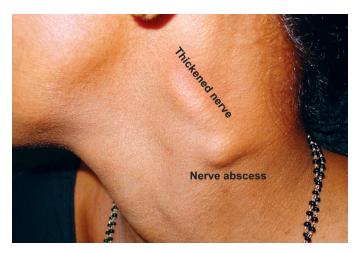


Fig. 5.4: Nerve abscess and thickened postauricular nerve

Ulnar Nerve Abscess and Small Muscles Atrophy in the Hand (Figs 5.5 and 5.6 and Key Box 5.2)







Fig. 5.6: Ulnar nerve abscess

A young man, 33 years of age, presented to the outpatient department with inability to use his right hand for the last 6 months. He said he could not work, or hold objects. Examination revealed atrophy of small muscles, specially hypothenar muscles and interossei muscles. A 2 cm 'skin ulcer' was also noticed. On careful questioning, he said he also had loss of sensation. Further examination revealed 4 cm x 3 cm oval swelling on the medial side of right arm. It was firm. Some fluctuation could be elicited.

What is the diagnosis?

Ulnar nerve abscess (cold) secondary to leprosy. This is a case of pure neuritic leprosy with nerve abscess usually caused by *Mycobacterium leprae*. It is an uncommon manifestation. No other peripheral nerve was involved.

Key Box 5.2

Ulnar Nerve Abscess

- May be a part of pure neuritic leprosy
- Presents as oval fluctuant swelling on the medial side of arm
- Granuloma is common. Progression to abscess occurs in tuberculoid leprosy.
- Schwann cell is affected. Slowly whole endoneurial zone is occupied by endothelial cells.
- High resolution ultrasound can demonstrate echotexture of masses.
- MRI—with the post-gadolinium T₁W sequence peripheral rim enhancement with central necrosis.
- Treatment: Incision, drainage, excision of granulomatous mass followed by treatment for leprosy.

Correction of Deformity of the Face by Plastic Reconstruction

- 1. A prosthesis to correct the nose
- 2. Lateral tarsorrhaphy, to prevent exposure keratitis.
- 3. Temporalis muscle flap to the upper eyelid to prevent exposure keratitis.

Correction of Deformity of the Hand and Foot

- Claw hand can be corrected using extensor carpi radialis brevis muscle (Paul Brand's procedure).
- Otherwise, flexor digitorum superficialis can be used (Bunnell's procedure).
- Foot drop can be corrected by using tibialis posterior muscle tendon transfer (**Ober's and Barr's procedure**).

LYMPH NODES SECONDARIES (METASTASIS) IN THE HEAD AND NECK

Introduction

Very often, the patients present to the surgeon with lymph node swelling in the neck with or without any complaints. If there is an obvious lesion in the oral cavity, the diagnosis is easy. On the other hand, difficulty arises in locating the primary malignancy, which is hidden or occult. It is important to know the anatomical location of the lymph nodes in the neck and drainage area, so that drainage areas can be investigated.

Before understanding the various lymph nodes, drainage area and block dissection, it is advisable to know surgical anatomy of the neck and lymphatic drainage.

SURGICAL ANATOMY OF NECK

Neck is that region of the body that lies between the lower border of the mandible and the suprasternal notch and the upper border of the clavicle. Its boundaries include mandible, zygomatic process of temporal bone, external auditory canal, mastoid process, superior nuchal line, external occipital protuberance, manubrium sterni, clavicle, acromioclavicular joint and spinous process of 7th cervical vertebra.

Fascial Layers of Neck

- Two fascial layers in the neck: Superficial cervical fascia and deep cervical fascia.
- Superficial cervical fascia corresponds to subcutaneous tissue.
- Deep cervical fascia (fascia colli) is the important layer for functional and selective neck dissection. It is this layer that divides neck into various compartments.

Deep Cervical Fascia

It has superficial and deep components.

A. Superficial layer (investing or anterior fascia): It is attached to following structures—occipital protuberance, mastoid process, capsule of parotid gland, angle of jaw, and body of the mandible to the symphysis. And here, it proceeds around contralateral side in a similar manner. It then goes posteriorly across the spinal process of the cervical vertebrae. Anteriorly, it passes from the mandible to hyoid bone. Inferiorly, it attaches to sternum, upper edge of clavicle, acromion, and spine of scapula. At the inferior border, in the midline, superficial layer splits in two different layers just superior to the manubrium of sternum. The space between these two layers is known as the **suprasternal space of Burns**.

From posterior to anterior, the superficial layer splits to enclose the trapezius, the portion of the omohyoid muscle that crosses the posterior triangle of the neck, and the sternocleidomastoid muscle. In a similar way, it envelops the strap muscles, before ending in the midline. The superficial veins of the neck lie on or within this superficial layer of the deep cervical fascia.

B. Deep layer (prevertebral fascia): The deep or prevertebral layer, like the superficial layer, attaches posteriorly to the spinous process of the cervical vertebrae. Above, it reaches the skull base at the jugular foramen and carotid canal, then passes across the basilar process to the opposite side. It covers the muscles of the back that enter into the neck immediately deep to the trapezius muscle. At the upper limit of the posterior triangle, the spinal accessory nerve crosses the posterior triangle at this level, along with some lymph nodes. At the lower end, both fascial layers further separate, the deep layer covers the scalene muscles, whereas superficial layer remains attached to the trapezius muscle and the clavicle.

Lymphatic Drainage of Neck

- The cervical lymphatics are divided into superficial and deep. The superficial lymphatics perforate the cervical fascia and drain into the deep lymphatics. The deep vessels and nodes are most commonly found along blood vessels, nerves and muscles. For example: Along the internal jugular vein classically described as jugular chain of lymph nodes. Look for this finding in ultrasound examination of neck.
- Lymph nodes have been given numbers according to their levels and drainage sites which are given below (Fig. 16.87).

Memorial Sloan-Kettering Cancer Centre: Lateral Lymph Node Classification

- Level I : Lymph nodes in the **submental triangle and submandibular triangle.**
- Level II : Upper jugular nodes. Deep to the sternocleidomastoid muscle, anterior to posterior border of sternocleidomastoid, posterior to posterior aspect of the posterior belly of diagastric, superior to level of hyoid. Level II is further divided into (IIa) which is inferior and in front of the line of spinal accessory nerve, (IIb) is above and posterior to line of spinal accessory nerve.

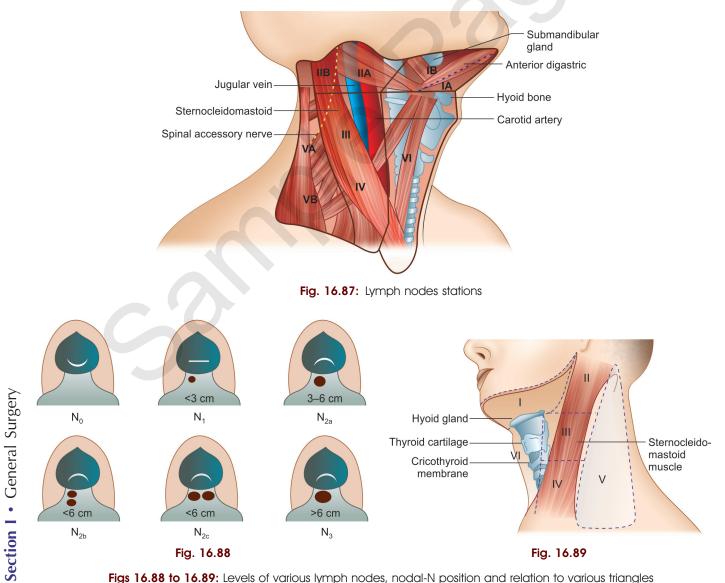
- Level III : Middle jugular nodes from hyoid superiorly to cricothyroid membrane inferiorly.
- Level IV : Lower jugular nodes from cricothyroid membrane superiorly to clavicle inferiorly.
- Level V : **Posterior cervical region** from anterior border of trapezius posteriorly to posterior border of sternocleidomastoid anteriorly and clavicle inferiorly.

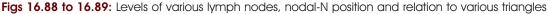
a. Above the line of spinal accessory nerve. b. Below the level of spinal accessory nerve.

- Level VI : Anterior compartment nodes from hyoid bone superiorly to suprasternal notch inferiorly and laterally by medial border of carotid sheath.
- Level VII: Upper mediastinal nodes inferior to suprasternal notch (no longer used).

Drainage Area

- Level Ia lymph nodes: Floor of mouth, anterior oral tongue, anterior mandibular alveolar ridge, lower lip.
- Level Ib lymph nodes: Oral cavity, anterior nasal cavity, soft tissue of midface, submandibular gland.
- Level IIa and IIb nodes: Oral cavity, nasal cavity, nasopharynx, oropharynx, hypopharynx, larynx, parotid gland.
- Level III lymph nodes: Oral cavity, nasopharynx, oropharynx, hypopharynx, larynx.
- Level IV lymph nodes: Hypopharynx, thyroid, cervical oesophagus, larynx.
- Level V lymph nodes: Scalp, parotid gland, nasopharynx, thyroid gland, etc.
- Level VI lymph nodes: Thyroid gland, glottic and subglottic larynx, apex of pyriform sinus, cervical oesophagus (Figs 16.88 and 16.89).





Key Box 22.17

Laparoscopic Ultrasound (LUS)

- LUS is also a staging modality and may provide improved accuracy in T and N staging.
- LUS may also be more accurate for staging coeliac nodes because EUS probe is more distant and does not provide direct inspection.

Depth of invasion

- For T1 lesions, conservative oesophageal resection such as vagal-sparing, transhiatal or minimally invasive oesophagectomy can be opted.
- For localised intramucosal tumours of limited extent, EMR or ESD are an acceptable alternative.
- There is almost no role for chemoradiotherapy for treatment of T1 lesions.
- Surgical or endoscopic resection alone carries a good long-term survival, as high as 88% in some series.

Depth of tumour vs lymph node involvement

1. T1 lesion (intramucosal)	18% LNI
2. T1 lesion (submucosal)	55% LNI
3. T2 lesion	60% LNI
4. T3 lesion	80% LNI

General Assessment for Staging

- General health of the patient, co-morbid conditions, fitness for anaesthesia—all these factors decide the choice of therapy.
- TNM staging
- In the absence of systemic spread, surgical cure may not be possible as depicted in Key Box 22.14.

TREATMENT

A few considerations before treatment of carcinoma since they often present at an advanced stage, 5-year survival is very low in the majority of cases. The following factors are considered for *palliative treatment*:

1. Comorbid conditions:	Cardiac diseases, bad chest
2. Blood spread metastasis:	Para-aortic lymph nodes
3. Contiguous organ	Trachea, aorta, pericardium
	invasion (CT scan)
4. Diagnostic laparoscopy:	Peritoneal spread
	Omental nodules, ascites
5. CT scan or laparo-	Lymph nodes metastasis
scopic ultrasound	Para-aortic lymph nodes,
	supraclavicular lymph
	nodes

TREATMENT WITH CURATIVE INTENT

• Once diagnosis and accurate staging is done, the histological diagnosis is also considered for treating carcinoma oesophagus.

TNM STAGING AJCC, 8th edition

Tumour

- **T0** No primary tumour
- Tis Carcinoma in situ—high-grade dysplasia
- **T1a** Tumour invades lamina propria, muscularis mucosa
- T1b Tumour involving submucosa
- T2 Tumour involving muscularis propria
- T3 Tumour with pararioesophageal spread
- T4 Involvement of recurrent laryngeal nerve, phrenic nerve, sympathetic chain, azygos vein; malignant effusion (adjacent structures)
 T₄a Resectable structure
- T_4 Tumor invades unresectable structure

Nodal Status

- Nx Lymph nodes cannot be assessed
- **N0** No regional lymph node metastasis
- N1 Regional lymph node metastasis—1 to 2 nodes
- N2 Metastasis in 3–6 regional nodes
- N3 Metastasis in 7 or more regional nodes

Metastasis

- M0 No distal spread
- M1 Upper thoracic oesophageal carcinoma with spread to other nonregional nodes or distant spread. Middle thoracic oesophageal carcinoma with spread to neck nodes/coeliac nodes or other nonregional nodes. Lower thoracic oesophageal carcinoma with spread to other nonregional nodes or distant spread.
- Mx Distant metastasis cannot be assessed.

TNM Stage Grouping

0	Tis N0 M0 T1 N0 M0	GX	Grade cannot be ass- essed—stage grouping
Stage IIA : Stage IIB :	T2 N0 M0, T3 N0H0 T1N1N0, T2N1, M0	G1 G2 G3 G4	
0	T3N1M0, T4 and N1N0		stage grouping as G3
Stage IV : A—Any T and N, M1a B—Any T1 and N, M1b			

• Tumor location is also important because of close proximity of oesophagus to pleura, pericardium azygos vein, aorta, etc.

While treating cancers in the oesophagus can be broadly classified as follows:

• **Cervical oesophagus:** It is from hypopharynx to thoracic inlet (at sternal notch), upper, thoracic oesophagus is from thoracic inlet to azygos vein.

Chapter

28

Small Intestine

- □ Embryology and development
- □ Anatomy
- □ Physiological functions
- Abdominal tuberculosis
- Tuberculous peritonitis
- **D** Tuberculous mesenteric lymphadenitis
- Glandular tuberculosis
- □ Intestinal tuberculosis
- □ Inflammatory bowel diseases
- □ Ileostomy
- Crohn's disease

- Surgical complications of enteric fever
- Intestinal amoebiasis
- Radiation enteropathy
- Peutz-Jeghers syndrome
- Adenocarcinoma
- □ GIST
- Neuroendocrine tumours
- □ Short gut syndrome
- Intestinal fistulae
- □ Small intestinal diverticula
- What is new in this Chapter?/Recent advances

INTRODUCTION

Truly speaking small intestines extends from pylorus to ileocaecal junction. However, for all practical purposes, it is discussed as starting from duodenojejunal flexure till caecum. Small intestines play an important role not only in the transfer of food contents distally but in the digestion, absorption and secretion of the contents. Being the central portion of the GI tract with long length, many diseases affect the intestine. Surgically important topics have been covered in this chapter.

EMBRYOLOGY AND DEVELOPMENT

- Small intestine develops from midgut.
- This midgut loop has cranial and caudal limbs.
- As the elongation starts from 5th week of foetal life, cranial limb develops into distal duodenum, jejunum and proximal ileum.
- Distal ileum and proximal two-thirds of transverse colon are developed from caudal limb.
- Midgut also rotates 270°. Thus, proximal jejunum will go to left side and ileum will go to right side.
- Anomalies can occur during rotation resulting in malrotation.

- Vitellointestinal duct (VI) joins the junction of cranial and caudal intestines which is about 2 feet away from ileocaecal junction.
- When VI duct is not obliterated, various anomalies occur. One of them is Meckel's diverticulum.
- Caecum which is present in the right hypochondrium descends into the right iliac fossa region.

ANATOMY

- Small intestine consists of proximal 2/5 jejunum and distal 3/5 ileum. It is about 6 metres in length.
- Small intestine starts at duodenojejunal flexure just to the **left of the inferior mesenteric vein**. **Surgical importance:** To identify the first (short) loop of jejunum for gastrojejunostomy.
- Small intestine ends at ileocaecal junction. In cases of intestinal obstruction, trace up to ileocaecal junction. If caecum is distended, it is a case of large bowel obstruction. If caecum is collapsed, it is a case of small bowel obstruction.
- Jejunum resides in the left side of the peritoneal cavity and ileum on the right side.
- Differences between jejunum and ileum have been given in Key Box 28.1.

C) Key Box 28.	1				
	Differences between Jejunum and Ileum					
		Jejunum	lleum			
0	Length	2/5	3/5			
0	Diameter	Wider (2–4 cm)	Less (2–3 cm)			
0	Wall	Thick and double	Thin			
		(mucous membrane				
		can be felt)				
0	Colour	Deep red	Pale pink			
0	Peyer's	Very, very less	More			
	patch					
0	Blood	Long and a few	Short and			
	supply	vasa recta (1 or 2)	numerous (5 or 6			
0	Mesentery	Transparent, less fat	More fat			

Blood Supply (Fig. 28.1)

- Superior mesenteric artery is the **artery of the midgut** which supplies the entire midgut (entire small intestine). Jejunal arteries are end-arteries.
- Mesenteric border of the intestine gets more blood supply when compared to anti-mesenteric border. Hence, in cases of diminished blood supply, antimesenteric border becomes ischaemic first.
- Venous drainage is through superior mesenteric vein.

Mesentery

- It is a fan-shaped fold of peritoneum which attaches jejunum and ileum to posterior abdominal wall.
- Blood vessels and lymphatics course in between the folds of peritoneum.
- It extends from the left of duodenojejunal flexure (left of L2) vertebra to the right sacroiliac joint, thus fixing the ileocaecal junction there.

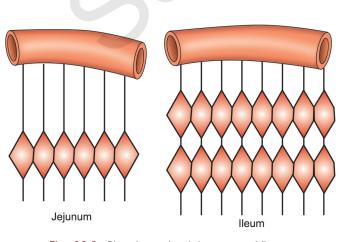


Fig. 28.1: Blood supply—jejunum and ileum

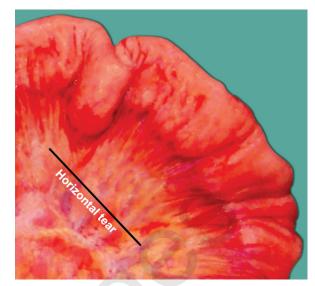


Fig. 28.2: Observe blood supply of the mesentery and it is but natural that a horizontal tear causes more gangrene

- The importance of direction of the mesentery is appreciated in the following examples:
 - **A. Mesenteric cyst** moves at right angles to the direction of the mesentery (*see* page 735).
 - **B. Mesenteric lymph nodes** can be clinically palpable as a nodular or a smooth mass. (Page 751)
 - **C. Horizontal tear** in the mesentery causes more gangrene of the bowel than vertical tear (Fig. 28.2).
- Structures crossed by mesentery: Duodenum, aorta, inferior vena cava, right ureter, right psoas major and right gonadal vein.

Innervation

- **Parasympathetic:** These are derived from vagus. It is secretomotor, thus helping in secretion and motility of small intestines. Nerve fibres traverse through coeliac ganglion. Afferent fibres do not carry pain impulses.
- **Sympathetic:** These fibres arise from three sets of splanchnic nerves. Their ganglion cells are located in a plexus around base of superior mesenteric artery. Pain is mediated through sympathetic system.

Lymphatics

- From mucosa, lymphatics pass through the wall of the bowel to regional lymph nodes, then into lymph nodes at base of superior mesenteric artery.
- Then it flows into cisterna chyli and then into thoracic duct and empty into venous system at confluence of left internal jugular and subclavian veins.
- Peyer's patches are major deposits of lymphatic tissue in the distal bowel. Tuberculosis and typhoid fever affect the Peyer's patches.

ANORECTAL ANOMALIES

Developmental Anatomy

- To start with, there is a common chamber called cloaca, which is later divided into 2 chambers, anteriorly allantois gives rise to urinary bladder and posteriorly, postallantoic gut gives rise to rectum and upper 2 cm of anal canal.
- Postallantoic gut fuses with proctodeum, thus giving rise to anal canal. If there is a defective fusion of this, it results in imperforate anus.

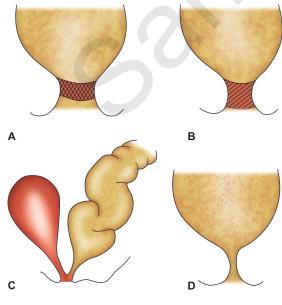
IMPERFORATE ANUS

Incidence

1:4500 live births. Common in female children.

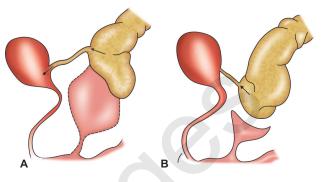
Types of Imperforate Anus

- **I.** Low anomalies: It refers to termination of the bowel below the anorectal bundle.
 - **1. Covered anus** (Fig. 30.106A): The anal orifice is covered by a tag of skin.
 - **2. Membranous anus** (Fig. 30.106B): Covered with a thin membrane
 - **3. Anterior ectopic anus** (Fig. 30.106C): Anus is situated anteriorly
 - **4. Stenosed anus** (Fig. 30.106D): Anal orifice is microscopic
- **II. High anomalies:** They are supralevator—the bowel terminates above the anorectal bundle.
 - 1. Anorectal and agenesis, with fistula: Rectovesical, rectovaginal, rectourethral fistulae (Fig. 30.107A).



Figs 30.106A to D: Low anorectal anomalies

- 2. Rectal atresia: Colon ends as a blind pouch below (Fig. 30.107B).
- 3. Cloaca: In this variety, bowel, urinary and genital tracts open into a common chamber. This occurs in females only.



Figs 30.107A and B: High anorectal anomalies

Diagnosis—Wangensteen's Invertogram

12 hours after birth, the child is held upside down (12 hours is the time for the gas shadow to reach the distal portion of the gut). A metal coin is strapped to the site of anus and X-ray is taken. If the gas shadow is above the pubococcygeal line, it is a high anomaly. If the distance between the coin and gas shadow is more than 2.5 cm, it is a high anomaly. If the gas shadow is below the pubococcygeal line, it is a low anomaly. Pubococcygeal line is called Stephen's line.

Treatment

- **I.** Low anomaly: Easy to treat, division of membrane or skin followed by dilatation is all that is required with some amount of plastic reconstruction (anoplasty is necessary).
- II. High anomaly: Repaired by 3-stage procedure:
 - 1st stage: Preliminary transverse colostomy to relieve intestinal obstruction.
 - 2nd stage: When the child is 8–10 kg of weight, a "pull-through" operation is done with division of fistula.
 - 3rd stage: After 2 months, colostomy is closed.

CAUSES OF INTESTINAL OBSTRUCTION AS PER AGE

NEONATES: 0 TO 7 DAYS (FIGS 30.108 TO 30.115)

- 1. Atresia and stenosis
- 2. Hirschsprung's disease
- 3. Arrested rotation with bands
- 4. Volvulus neonatorum
- 5. Meconium ileus
- 6. Imperforate anus

Key Box 46.1

- Mechanical obstruction
- o GIT bleeding
- Tumours of small intestine
- o Unexplained abdominal pain
- Diarrhoea

Key Box 46.2

Contraindications

- Complete obstruction
- o Suspected perforation
- o Massive dilatation of small bowel
- Duodenal obstruction
- o Gastrojejunostomy

Procedure

- Bilbao Dotter tube is inserted with the guide wire through one of the nostrils and advanced caudally with the swallowing action till the tip reaches the stomach. The tube is then advanced through the antrum of the stomach to the pyloric canal. Then it is advanced under fluoroscopic guidance to about 4–5 cm distal to the Trietz ligament (duodenojejunal junction).
- 200 ml barium suspension is injected at a rate of 75 ml/min followed by 5% of methylcellulose at a rate of 100 ml/min. The head of the barium column is followed with intermittent fluoroscopy and films exposed wherever necessary.
- Ileocaecal spot films are taken when the junction is opacified and distended.

Interpretation of Study

- 1. Normal small bowel shows a decrease in calibre from jejunum to ileum and the change of prominent valvulae conniventes to featureless ileum is evident.
- 2. Malignancies and lymphomas show evidence of strictures, proximal dilatations and mucosal abnormality. Large mesenteric nodal masses displace the bowel loops.
- 3. Strictures and ulceration of terminal ileum: Dilatation of the segment proximal to the narrowed segment and conical shrunken caecum are seen in ileocaecal tuberculosis. In later stages, ileal strictures, fistulae, etc. may be seen.

Complications

- Perforation
- Inspissation of barium
- Transient bacteraemia

ANGIOGRAPHY (Figs 46.4 and 46.5)

Definition

This is the study of blood vessels by injection of a contrast medium containing iodine into the vessel.

Indications

- 1. Primary vascular diseases such as vaso-occlusive disease, aneurysm, arteriovenous malformation (AVM).
- 2. Vascularity assessment of a tumour.
- 3. Congenital vascular conditions such as coarctation.
- 4. Percutaneous interventional vascular procedures.



Fig. 46.4: Arch aortogram: Contrast in the arch of aorta demonstrating the major vessels arising from it

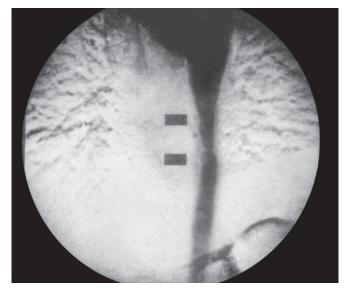


Fig. 46.5: Aortogram: Contrast in the thoracic aorta showing narrowing

VIRTUAL COLONOSCOPY

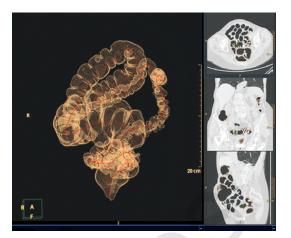
- It is a recently developed technique that uses a CT scanner and computer virtual reality software to look inside the body without having to insert a long tube (conventional colonoscopy) into the colon or without having to fill the colon with liquid barium (barium enema).
- More formally known as three-dimensional CT colonography, the virtual procedure allows radiologists to obtain 3D images from different angles, providing a sort of movie of the colon's interior without having to insert an endoscope into the bowel.

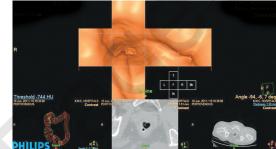
Advantages

- Noninvasive procedure, well-tolerated by patient
- Requires no sedation, less time-consuming
- Useful in elderly who are frail and infirm
- Useful when a tumour is large enough to block passage of scope (Figs 46.24 and 46.25).

Disadvantages

- Exposure to radiation, less detail of inner lining of colon
- Small polyps are located more reliably by colonoscopy
- Strictly a diagnostic procedure (unlike colonoscopy).





Figs 46.24 and 46.25: Virtual colonoscopy shows opened-up view to look for small mass or polyp

What is new in this Chapter? | Recent Advances

- □ All topics have been updated.
- PET scan, virtual colonoscopy have been upgraded
- A few photographs have been added

Multiple Choice Questions

1. Niche and a notch is diagnostic of:

- A. Chronic duodenal ulcer
- B. Chronic gastric ulcer
- C. Carcinoma stomach
- D. Stromal tumour

2. Trifoliate/Clover deformity is diagnostic of:

- A. Chronic duodenal ulcer
- B. Chronic gastric ulcer
- C. Carcinoma stomach
- D. Stromal tumour

3. The substance used in barium studies is:

- A. Barium chloride
- B. Barium sulphate

- C. Barium carbonate
- D. Barium sulphide

4. Barium follow through extends up to:

- A. Proximal duodenum
- B. Fundus of the stomach
- C. Ileocaecal junction
- D. Proximal jejunum
- 5. Which of the following is a contraindication for barium study?
 - A. Dysphagia and odynophagia
 - B. Motility disorders of the GIT
 - C. Perforation of gastric mucosa
 - D. Assessing mediastinal masses

XX. MALIGNANT MELANOMA

- 1. What is this specimen?
 - Specimen of foot showing a large ulcerated growth in the sole of the foot.
- 2. What is the diagnosis and why do you say so?
 - Malignant melanoma because the lesion is pigmented.
- 3. What is the commonest type of malignant melanoma?
 - Superficial spreading is the first followed by nodular variety.
- 4. What are the staging systems available for this condition?
 - Clark's level of invasion and Breslow's thickness are important staging systems in addition to TNM staging.

5. What are the ABCDE of melanoma?

- Asymmetry
- Border irregular
- Colour variegation
- Diameter >6 mm
- Elevation



Fig. 52.20: Malignant melanoma

XXI. THYROIDECTOMY SPECIMEN

- 1. What is this specimen?
 - Specimen of thyroid gland showing both lobes and isthmus.
- 2. What is the diagnosis and why do you say so?
 - Probably it is a subtotal thyroidectomy specimen surgery is done for multinodular goitre.
- 3. What is the commonest type of malignancy of the thyroid gland?
 - Papillary carcinoma—63%. 2nd common type is follicular carcinoma thyroid.
- 4. What is the surgical treatment for welldifferentiated carcinoma thyroid gland?
 - Most centres follow total thyroidectomy. If the patient is in low-risk category, lobectomy can be done.

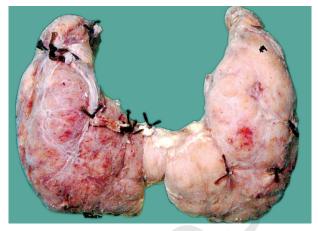


Fig. 52.21: Thyroidectomy specimen

- 5. What blood investigation is useful in the followup period of papillary carcinoma thyroid gland?
 - Thyroglobulin

XXII. WIDE EXCISION SPECIMEN OF SKIN

- 1. What is this specimen?
 - Specimen of skin which has been excised with normal skin. Hence, wide excision specimen.
- 2. What is the diagnosis and why do you say so?
 - Probably it is a squamous cell carcinoma because edges are everted.
- 3. What are the common sites of squamous cell carcinoma skin?
 - Areas with chronic irritation, e.g. Kangri cancer in the abdominal wall, chimney sweepers cancer, etc.
- 4. What are the common precancerous lesions for squamous cell carcinoma?
 - Leukoplakia, Bowen's disease, chronic irritation and scar tissues, etc.
- 5. What do you call squamous cell carcinoma arising in a scar tissue?
 - Marjolin's ulcer



Fig. 52.22: Wide excision skin

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	6	
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