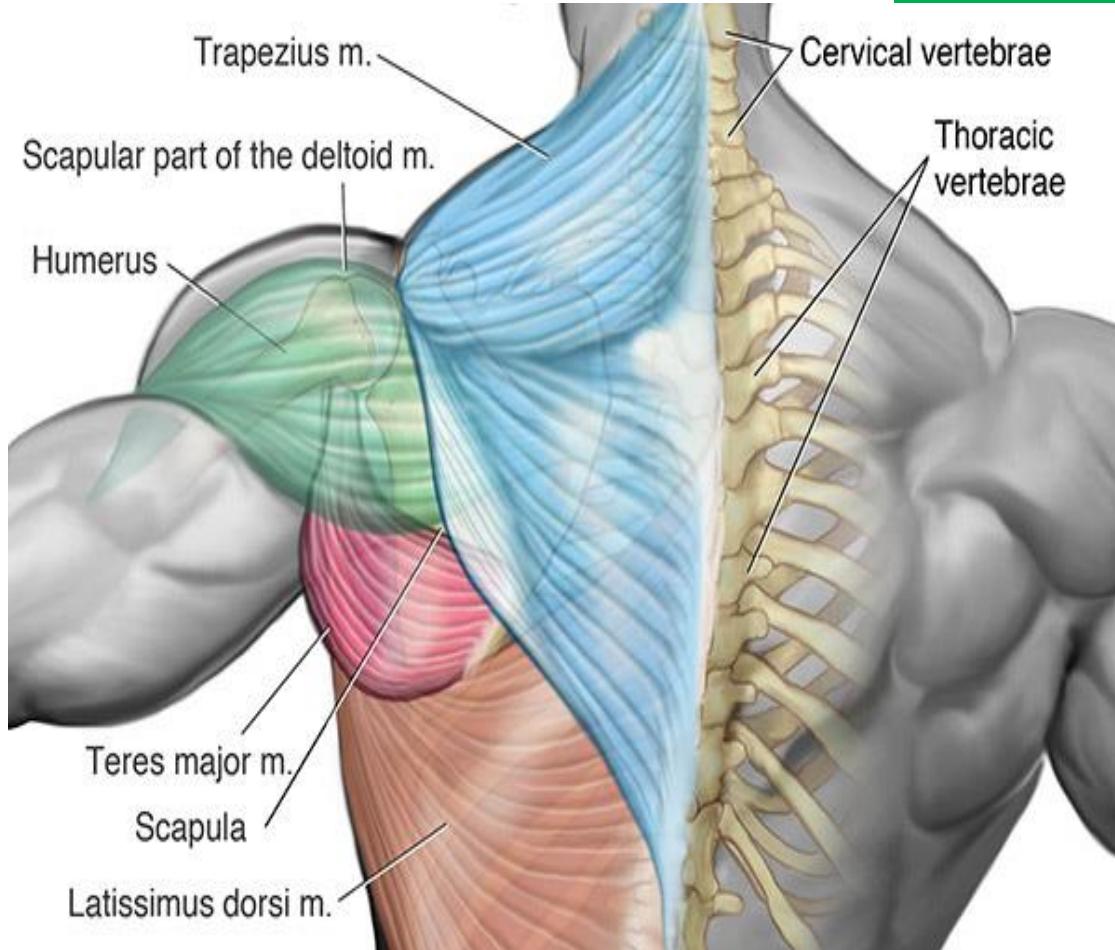


2020

For MRCS part A

Upper Limb

MRCS in Capsule

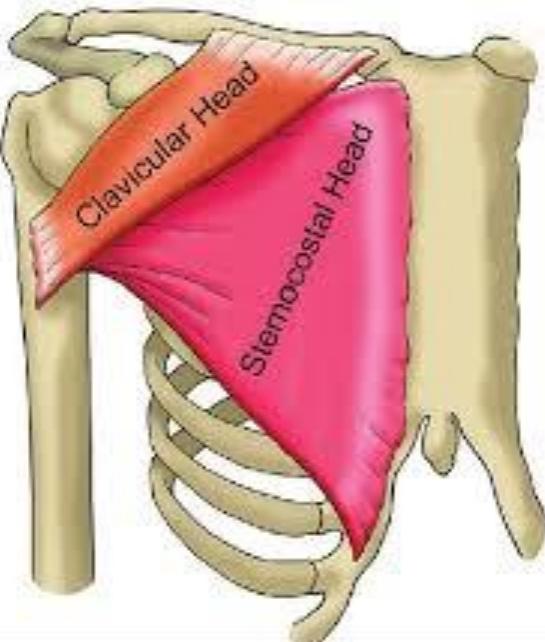


Dr. Ahmed Elsabbagh

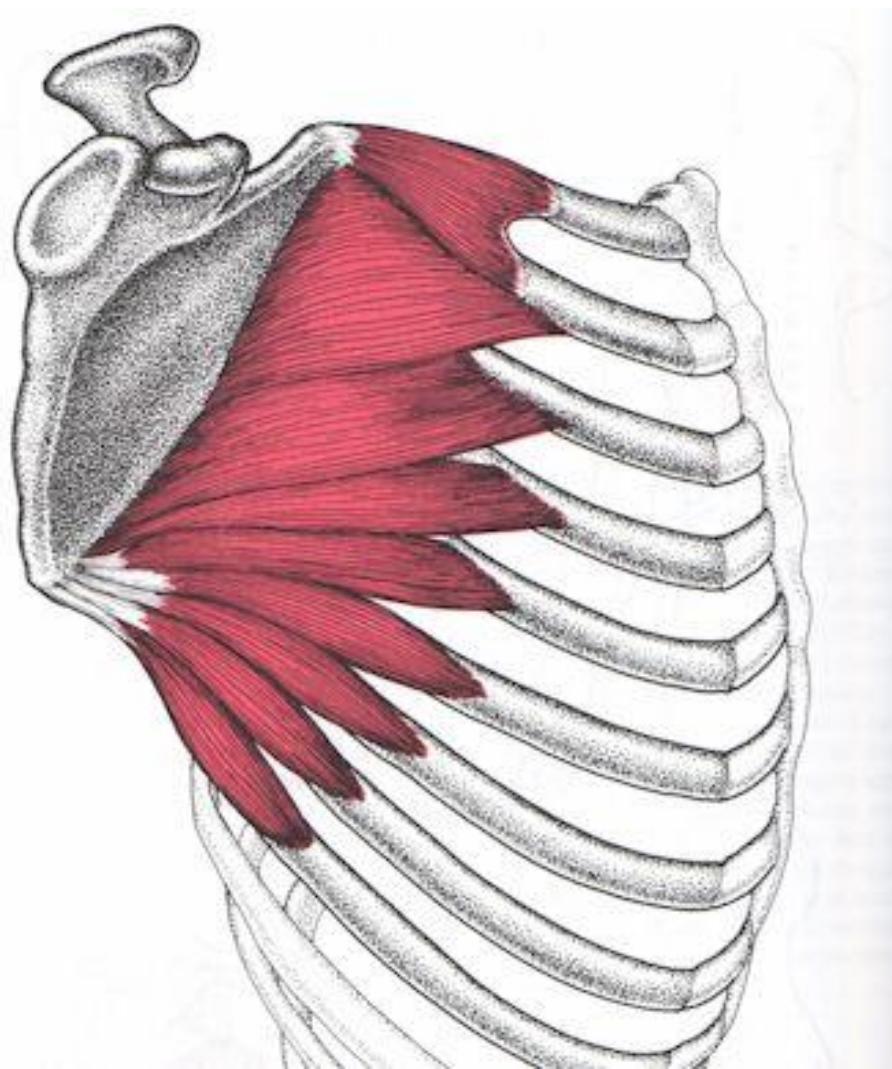
A. Muscles of pectoral region

	Pectoralis major	Pectoralis minor	Serratus anterior
Origin	<u>Clavicular head:</u> Front of medial $\frac{1}{2}$ of the clavicle. <u>Sternocostal head:</u> From: 1- Front of sternum. 2- Upper 6 costal cartilages.	Outer surface of 3 rd , 4 th & 5 th ribs near costal cartilages.	- By 8 digitations from outer surface of upper 8 ribs.
Insertion	In lateral lip of the bicipital groove.	Coracoid process of the scapula.	The muscle is inserted into ventral surface of the medial border of scapula,
Nerve supply	Medial and lateral pectoral nerves.	Medial pectoral nerve	Long thoracic nerve (of Belly)
Action	1. <u>Clavicular head:</u> Flexion of arm. 2. <u>Whole muscle:</u> adduction & medial rotation of arm		Fixing scapula to posterior thoracic wall so its paralysis leads to winging of scapula .

PECTORALIS MAJOR



pectoralis minor



Serratus anterior

Clavi-pectoral fascia

Def: strong fibrous membrane of deep fascia filling the gap between pectoralis minor & the clavicle

Site: Deep to pectoralis major

Attachments:

- **Above:** it splits to enclose subclavius muscle then it attaches to lower surface of the clavicle
- **Below:** it splits to enclose pectoralis minor muscle then continue downward to be attached to axillary fascia forming (suspensory ligament of axilla).
- **Medially:** 1st costal cartilage
- **Laterally:** coracoid process & clavicle

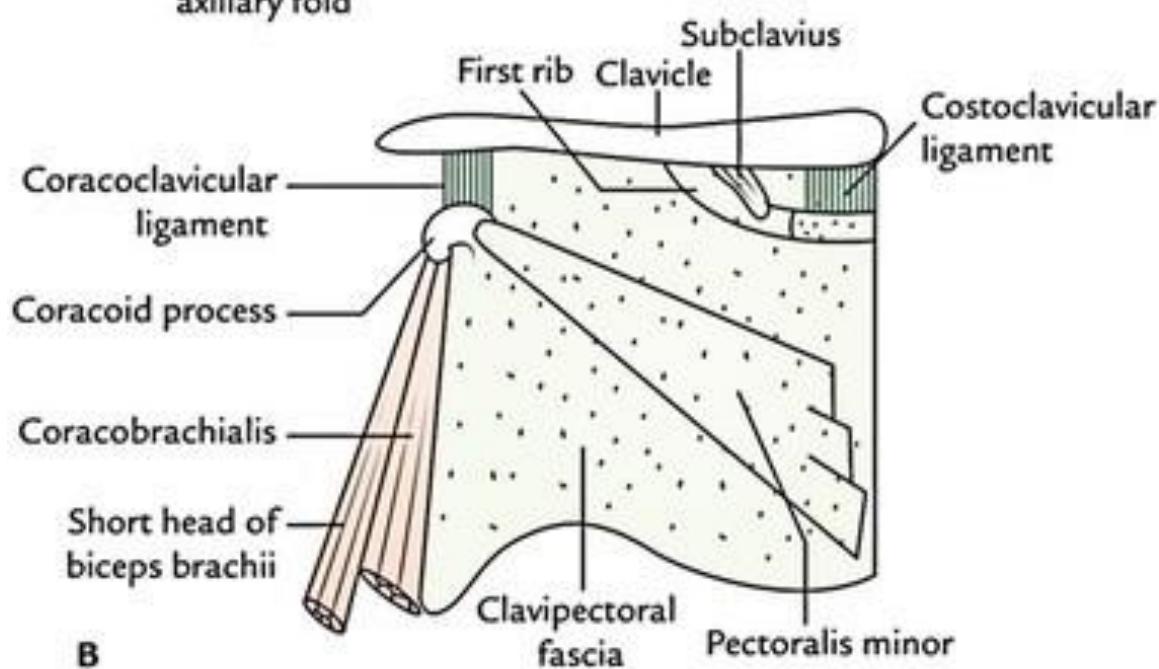
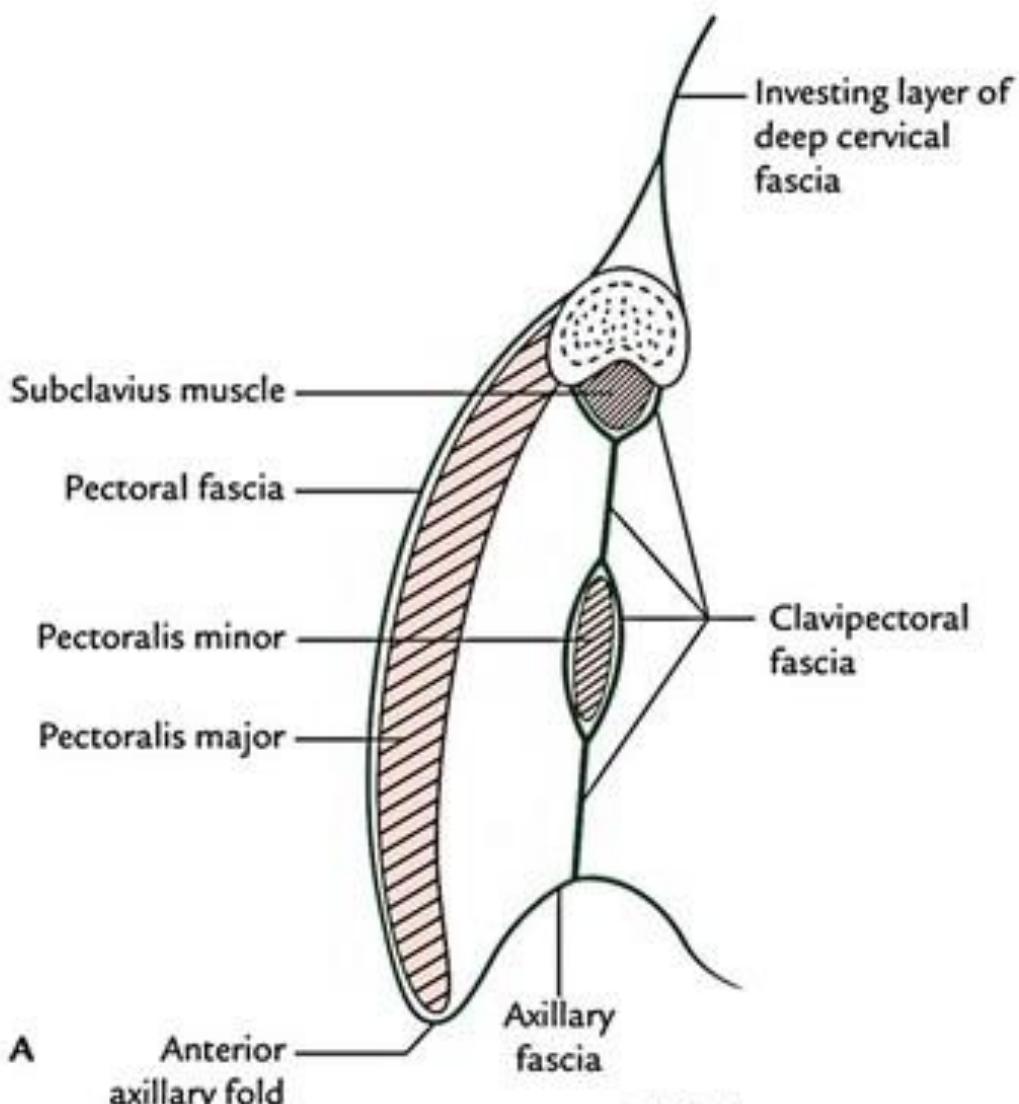
Structures piercing it:

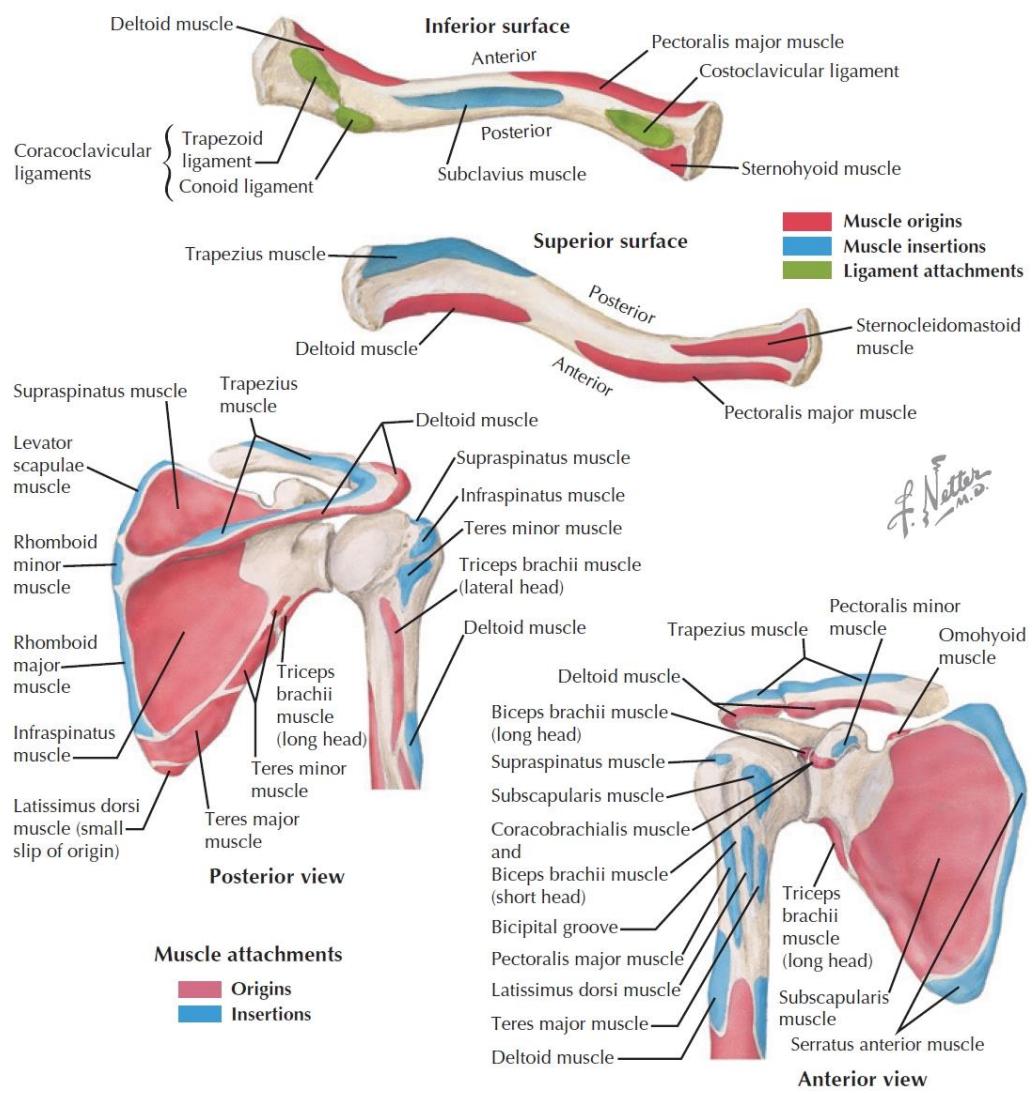
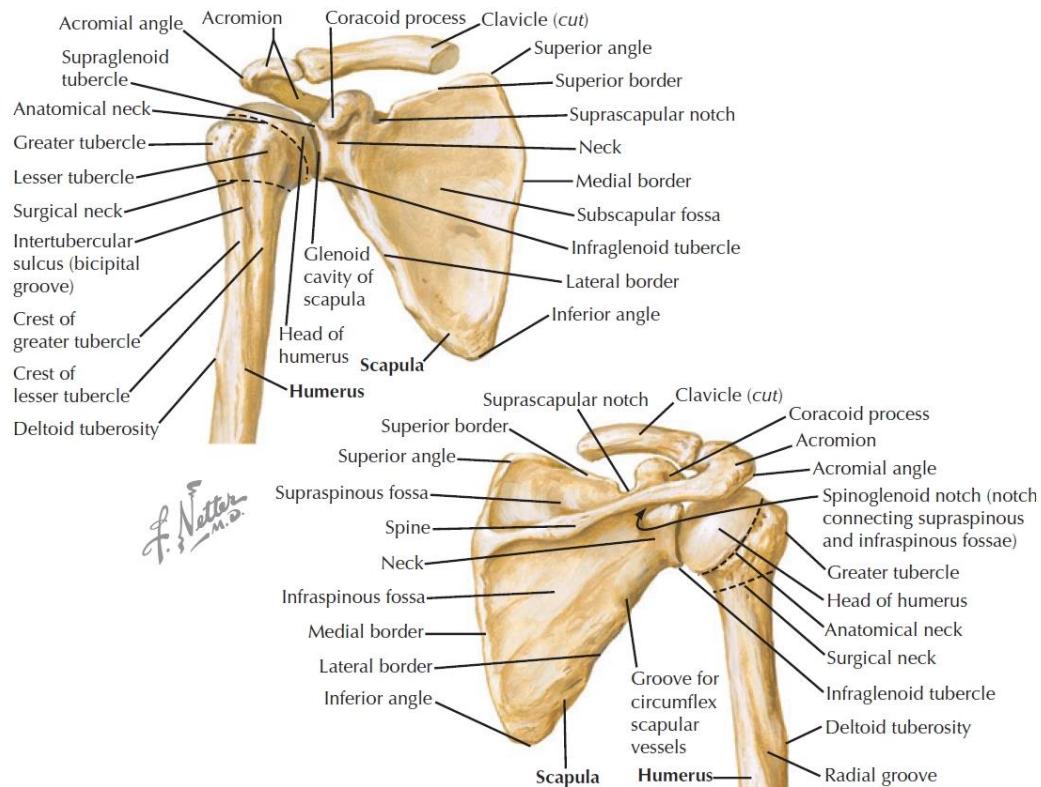
- Cephalic vein
- Lateral pectoral nerve
- Acromiothoracic artery
- Fat, LNS



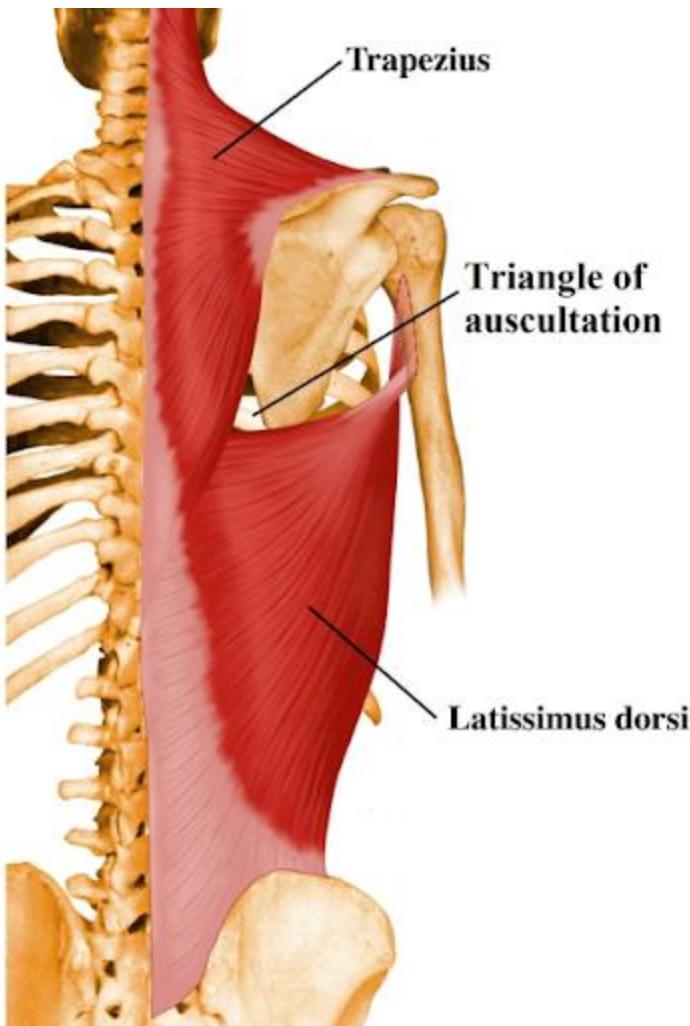
Structures piercing clavipectoral fascia

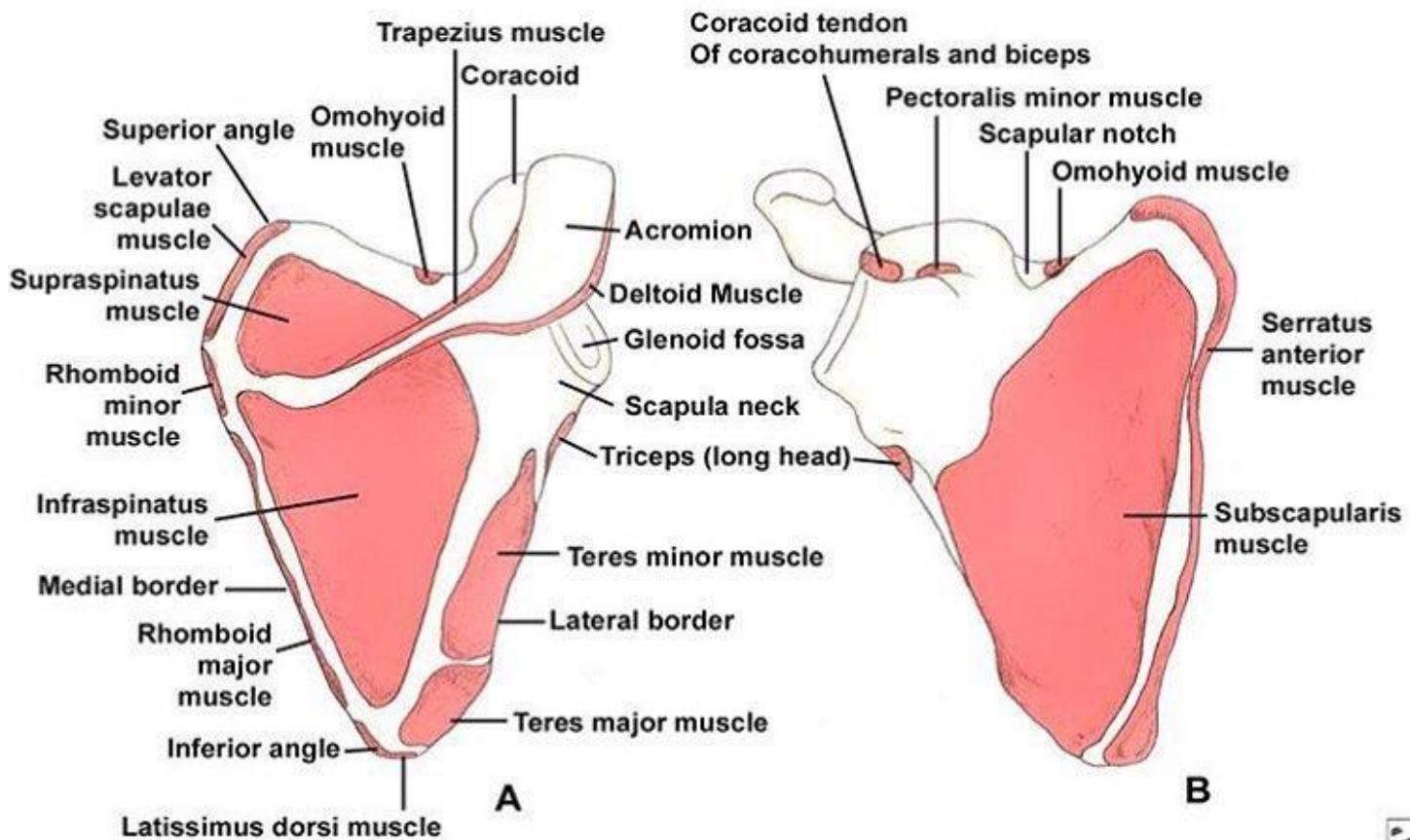
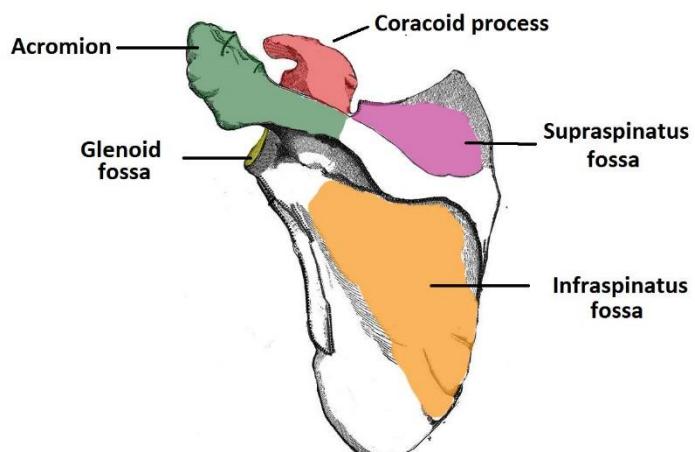
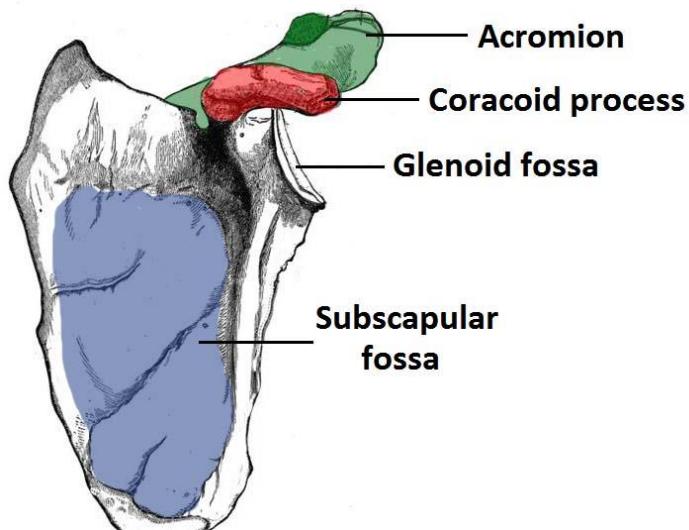
1. Thoraco-acromial artery
 2. Cephalic vein
 3. Lateral pectoral nerve
 4. Lymphatics
-
- The diagram illustrates the structures that pierce the clavipectoral fascia. It shows a cross-section of the fascia covering the pectoralis minor muscle. Several structures are labeled: the Thoraco-acromial artery (red), the Cephalic vein (pink), the Lateral pectoral nerve (yellow), and Lymphatics (represented by wavy lines). To the right, the Axillary vein (pink), Axillary artery (blue), and the Lateral cord of the brachial plexus (yellow) are shown passing through or adjacent to the fascia. A large red shaded area represents the pectoralis minor muscle.





	Trapezius	Latissimus dorsi
Origin	<ul style="list-style-type: none"> - Spine of c7 - All thoracic spines & supra spinous ligaments 	<ul style="list-style-type: none"> - Lower 6 thoracic spines & all lumbar vertebrae with their supraspinous ligaments
Insertion	Clavicle & scapula	Floor of the bicipital groove
NS	spinal accessory nerve	N. to latissimus dorsi (thoraco-dorsal nerve)
Action	It rotates the scapula upward 90 – 180	Adduction, extension and medial rotation of shoulder joint





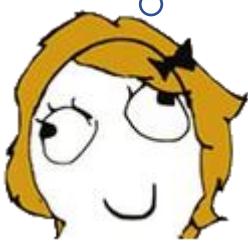
Flexor Muscles of forearm

NB: common flexor origin (CFO) presents in front of medial epicondyle of humerus.

Superficial flexors	Pronator teres	Flexor carpi radialis	Palmaris longus	Flexor carpi ulnaris	Flexor digitorum superficialis
Origin	<ul style="list-style-type: none"> + <u>Humeral head:</u> CFO + <u>Ulnar head:</u> from medial border of coronoid process of ulna <p>NB: median nerve pass between its 2 heads.</p>	<ul style="list-style-type: none"> + <u>Humeral head:</u> CFO 	<ul style="list-style-type: none"> + <u>Humeral head:</u> CFO 	<ul style="list-style-type: none"> + <u>Humeral head:</u> CFO + <u>Ulnar head:</u> <ul style="list-style-type: none"> - medial surface of olecranon process of ulna - Upper 2/3 of post. Border of ulna <p>NB: ulnar nerve pass between its 2 heads.</p>	<ul style="list-style-type: none"> + <u>Humero-ulnar head:</u> <ul style="list-style-type: none"> - CFO - medial border of coronoid process of ulna + <u>Radial head:</u> <ul style="list-style-type: none"> - From ant. Oblique line of radius
Insertion	Into rough area on the middle of lateral surface of radius (at its maximum convexity)	Base of 2 nd , 3 rd metacarpal bones	Apex of palmer aponeurosis	<ul style="list-style-type: none"> - Base of 5th metacarpal bone - Pisiform - Hook of hamate 	The muscle is divided into 4 tendons & inserted into the middle phalanges of the medial 4 fingers
NS	Median nerve		Ulnar nerve		Median nerve
Action	1- Flexion of elbow joint 2- pronation		2. Flexion in wrist joint 3. Adduction of hand		
					3) Flexion of M/P & proximal I/P joints of medial 4 fingers

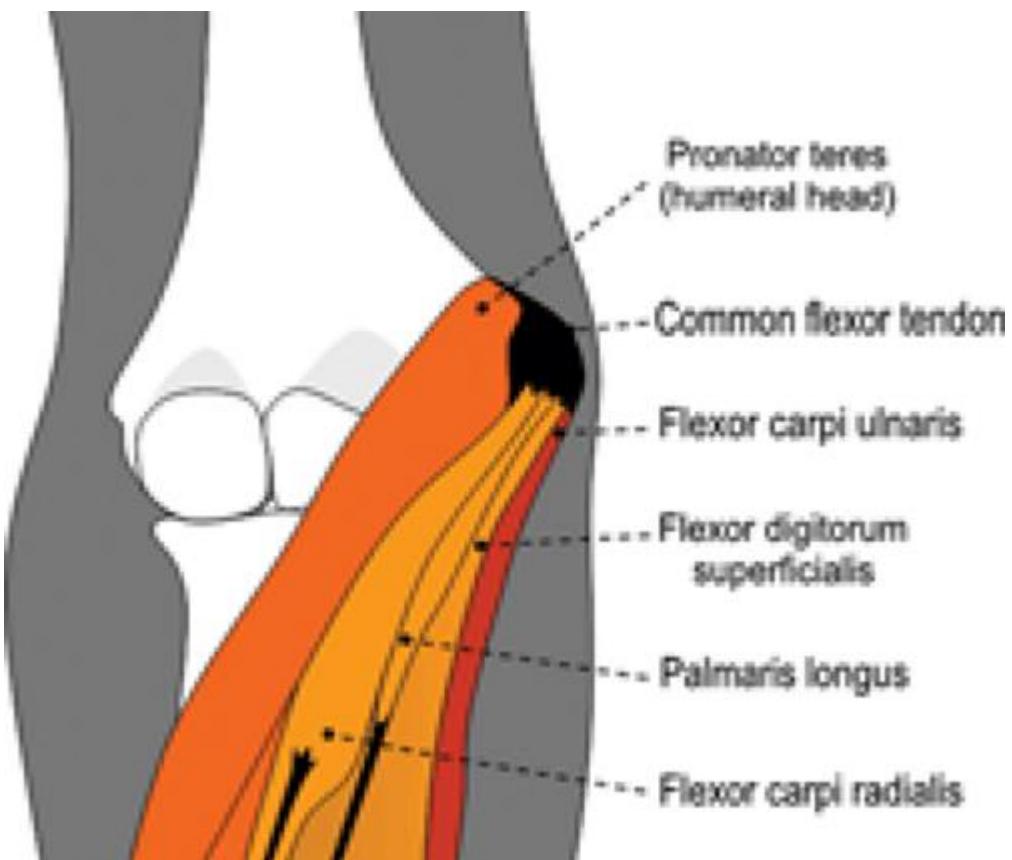
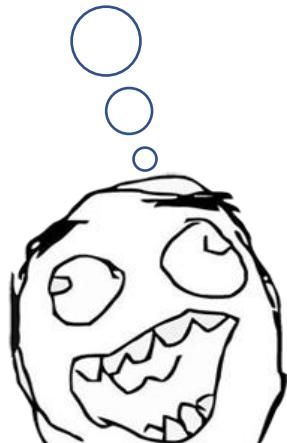
Pronator teres

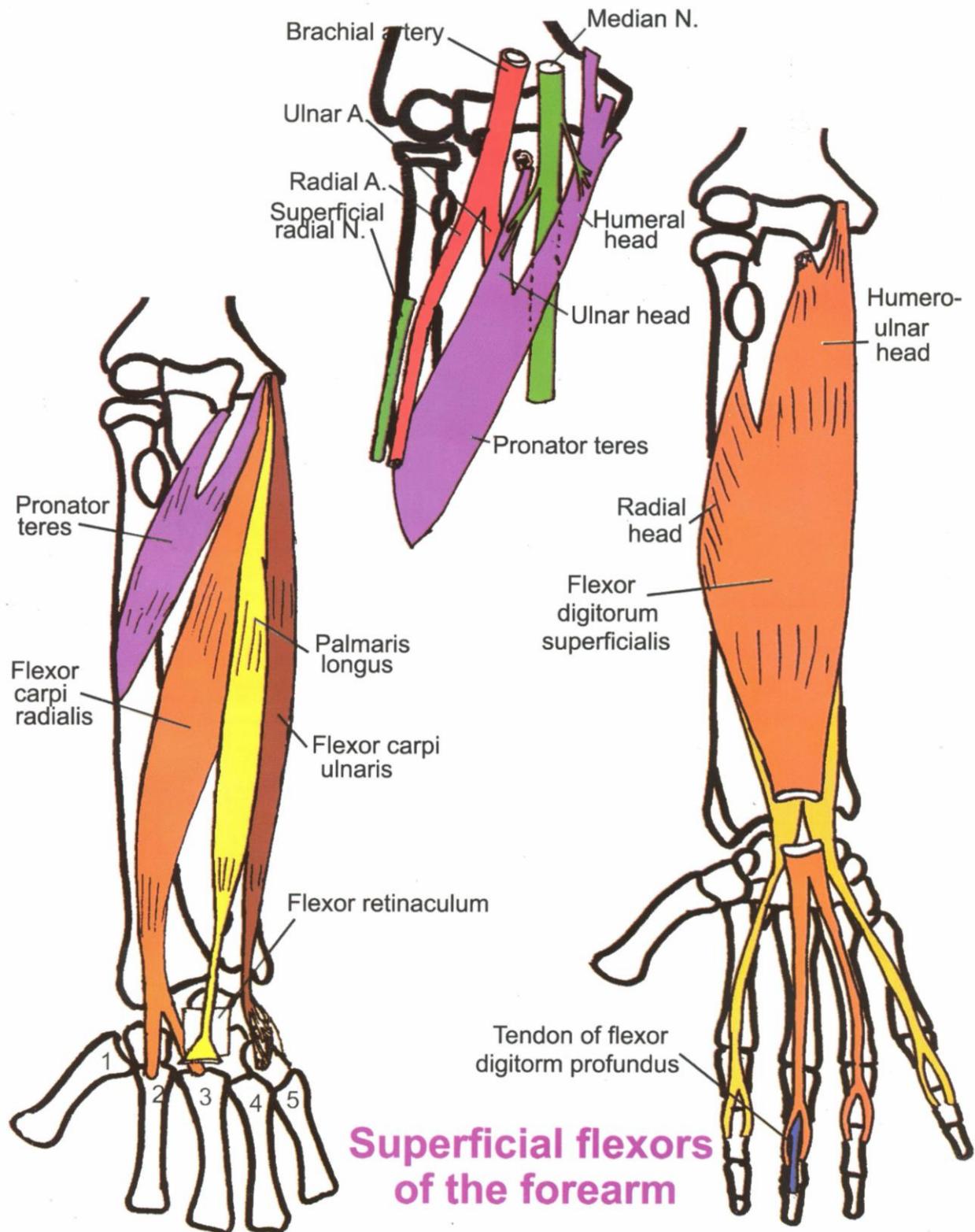
- CFO
- Coronoid
- MSCR



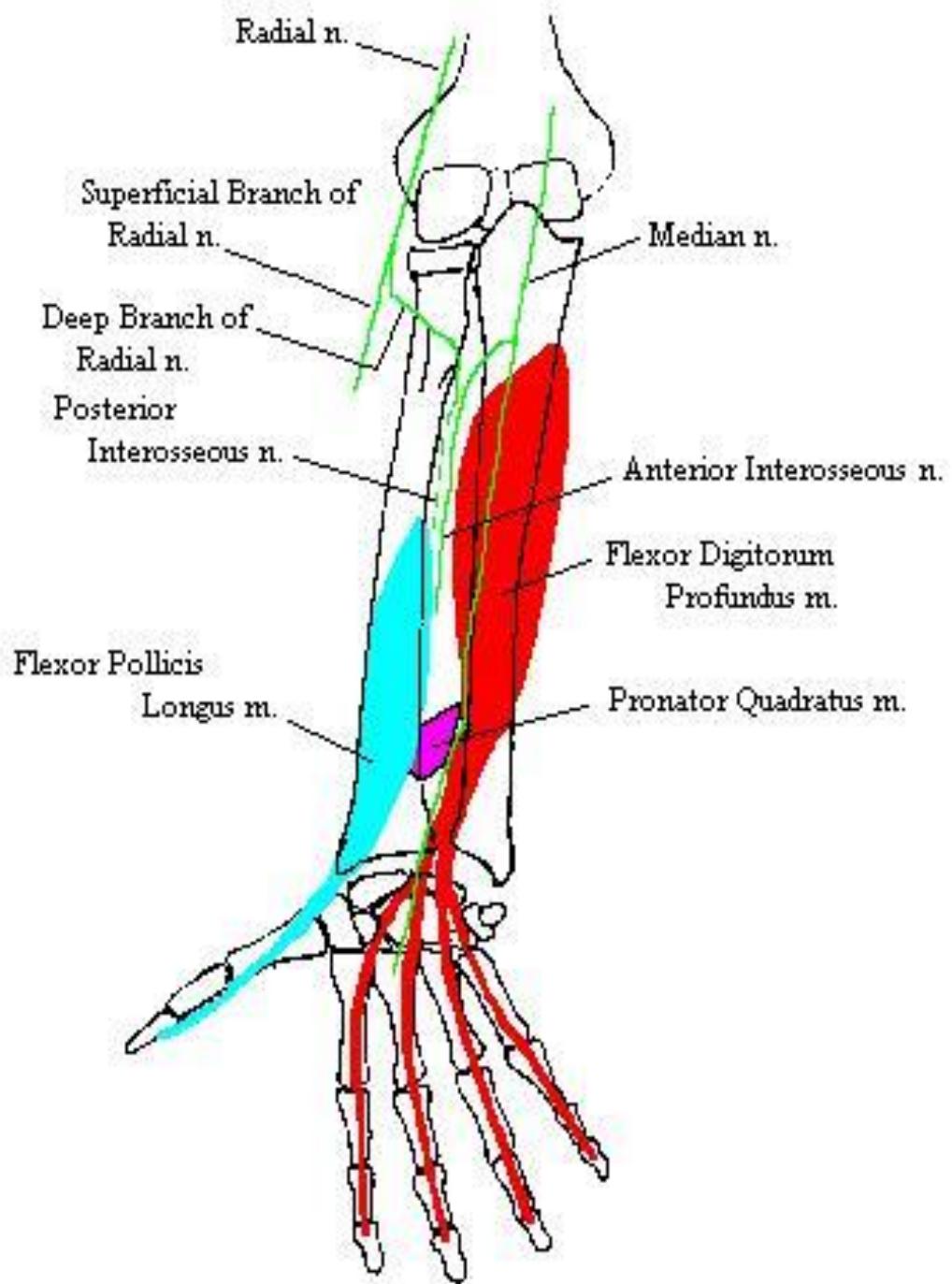
Flexor digitorum superficialis

- CFO
- Coronoid
- AOL





Superficial flexors of the forearm



Deep Forearm Flexors

Deep flexors	Flexor pollicis longus	Pronator quadratus	Flexor digitorum profundus
Origin	- Upper 2/3 of ant. Surface of radius	Lower 1/3 of ant. Surface of ulna	- Upper 2/3 of ant. & medial surfaces of ulna
Insertion	Base of distal phalanx of thumb	Lower 1/3 of ant. Surface of radius	Base of distal phalanges of medial 4 fingers.
NS	Anterior interosseous nerve		
Action	<ul style="list-style-type: none"> - Flexion in wrist - Flexion in all joints of thumb 	<ul style="list-style-type: none"> - Main pronator of forearm - fixes radius to ulna 	<ul style="list-style-type: none"> - Flexion in wrist - Flexion of all joints of medial 4 fingers

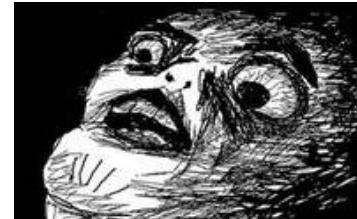
Flexor pollicis longus



Flexor digitorum profundus



Pronator quadratus



The Scalp

Layers of scalp

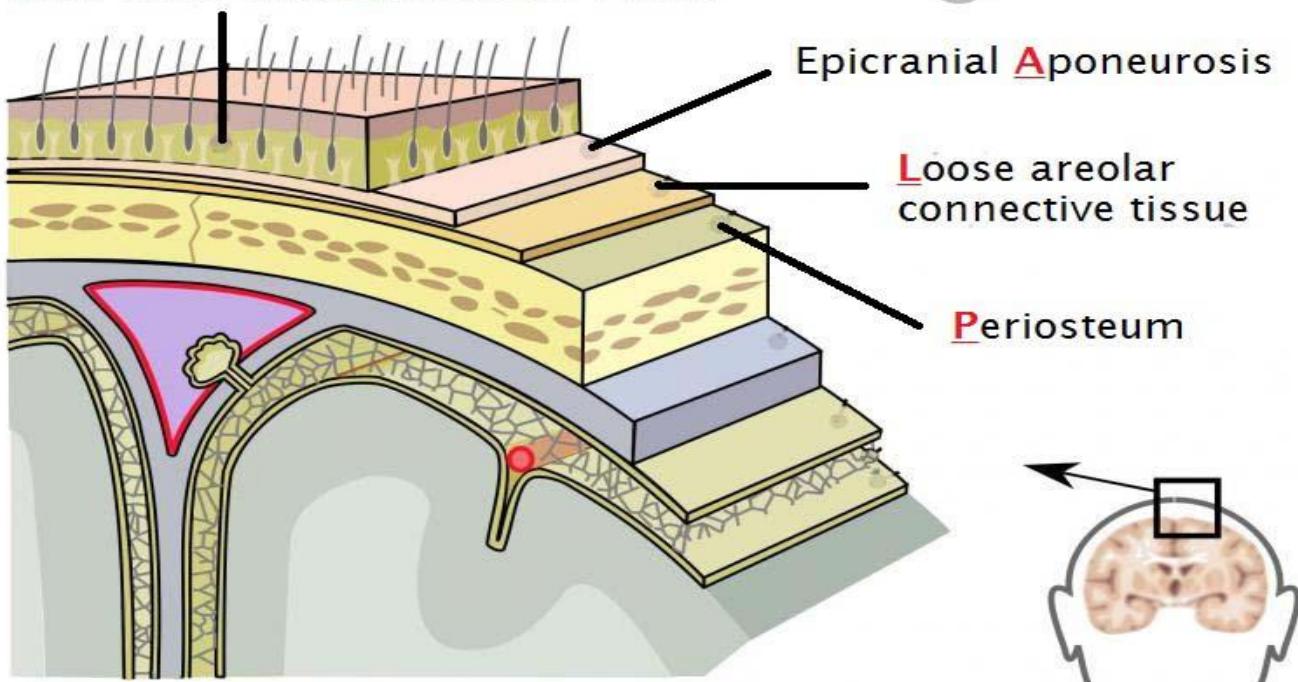
- 1) Skin (S)
- 2) Connective tissue layer (superficial fascia) (C)
- 3) Aponeurotic layer (A) (epicranial aponeurosis, galea aponeurotica)
- 4) Loose areolar tissue (L): This area is most dangerous as infections can spread easily.
The emissary veins that drain this area may allow sepsis to spread to the cranial cavity.
- 5) Pericranium (P): It is the outer periosteum of the skull.

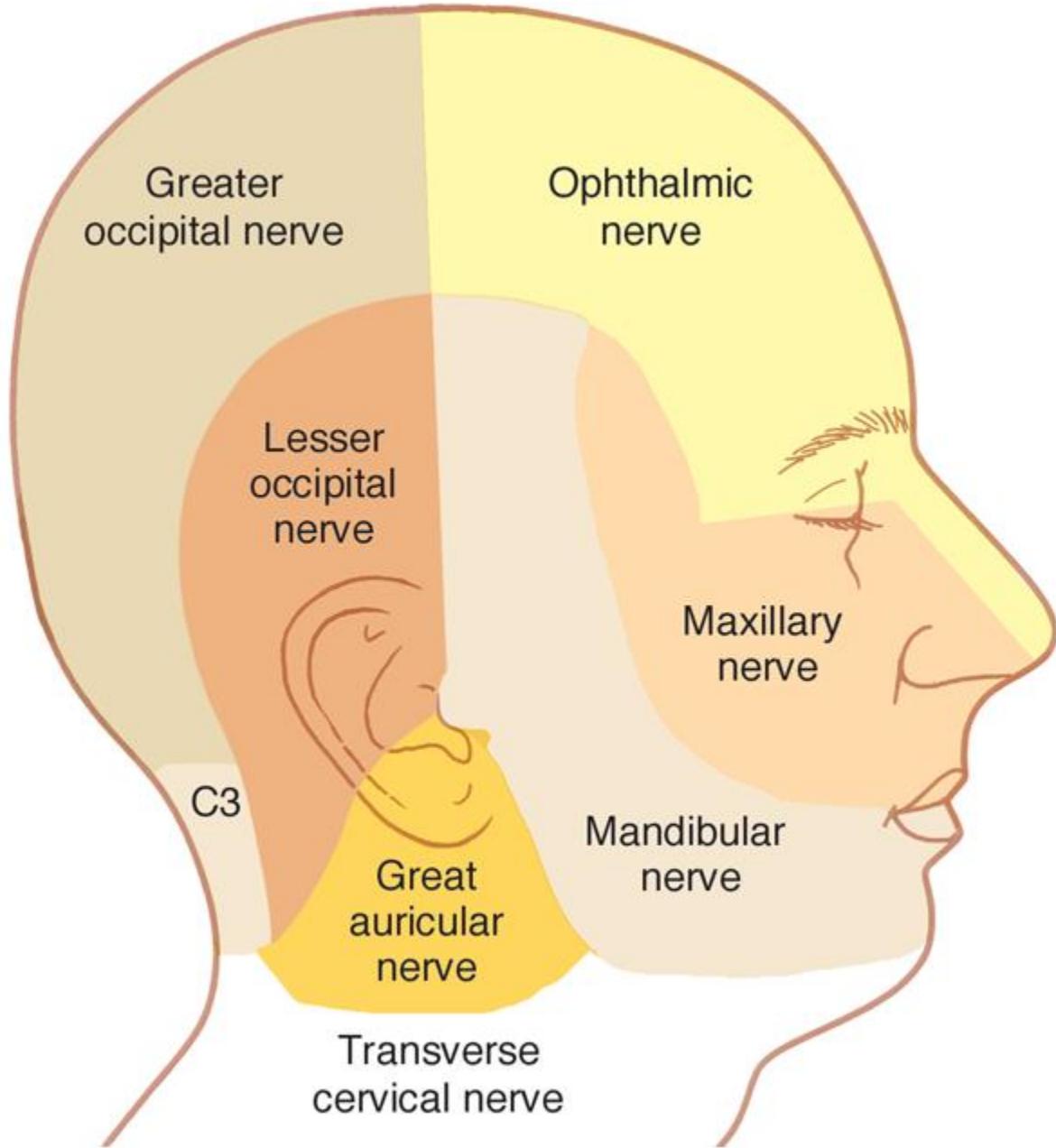
Nerve supply:

1. Anterior $\frac{1}{2}$ → by branches of trigeminal nerve
2. Posterior $\frac{1}{2}$ → by C2 & C3

Skin and Dense Connective Tissue

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Parotid Gland

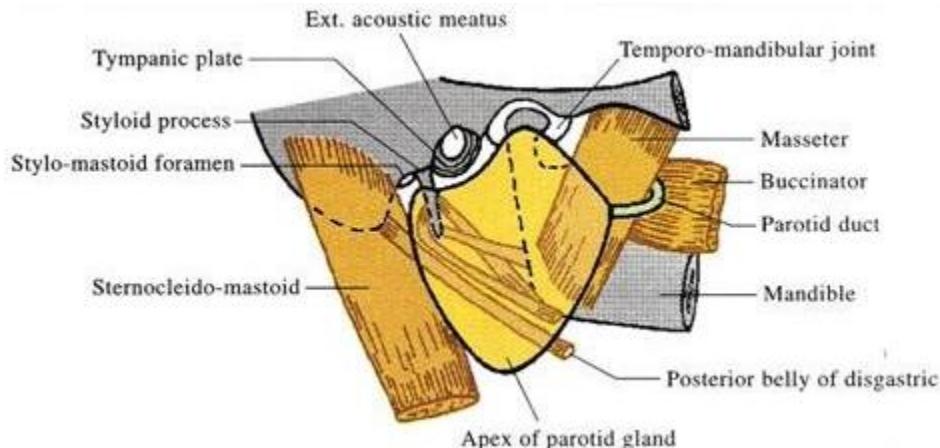
It is the largest salivary gland in the body. It is a large pyramidal or wedge shaped glands, weight about 15 gm. It is purely serous gland on each side at the upper part of the side of the neck.

A small detached or accessory lobe may be found between the zygomatic arch and parotid duct on the masseter muscle.

Position and extension:

It is enclosed in the gap between:

- External auditory meatus (above),
- Ramus of mandible (anteriorly),
- Mastoid process and sternomastoid muscle (posteriorly).



Capsules of the gland:

The gland is surrounded by 2 capsules:

- 1- **True capsule:** Formed by the condensed connective tissue of the gland.
- 2- **False capsule:** Formed by the deep fascia surrounds the gland.

Structures embedded within parotid gland:

1- Facial nerve:

The most superficial structure

It divides within the gland into 5 terminal branches:

- Temporal branch
- Zygomatic branch
- Buccal branch
- Mandibular branch
- Cervical branch

2- Retromandibular vein:

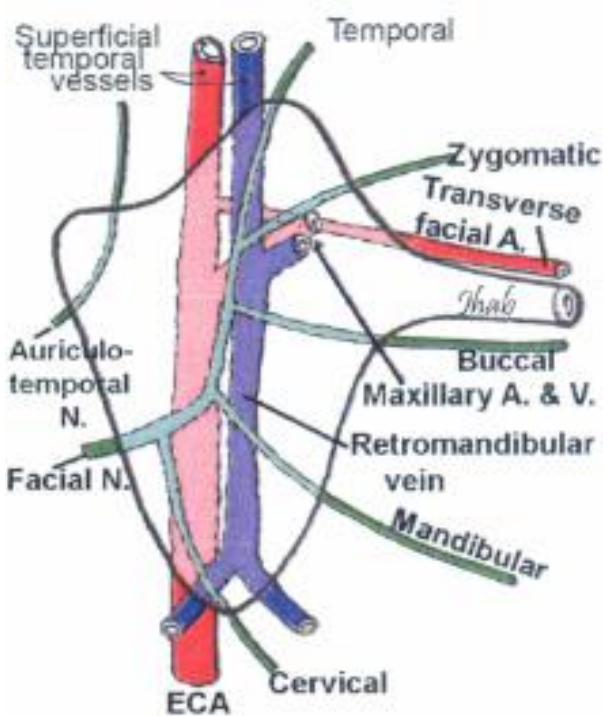
It is formed inside the gland by fusion of: maxillary & superficial temporal veins

3- External carotid artery:

It enters the gland at its apex and divides inside it into:

- **Maxillary artery.**
- **Superficial temporal artery.**

4- Auriculo-temporal nerve

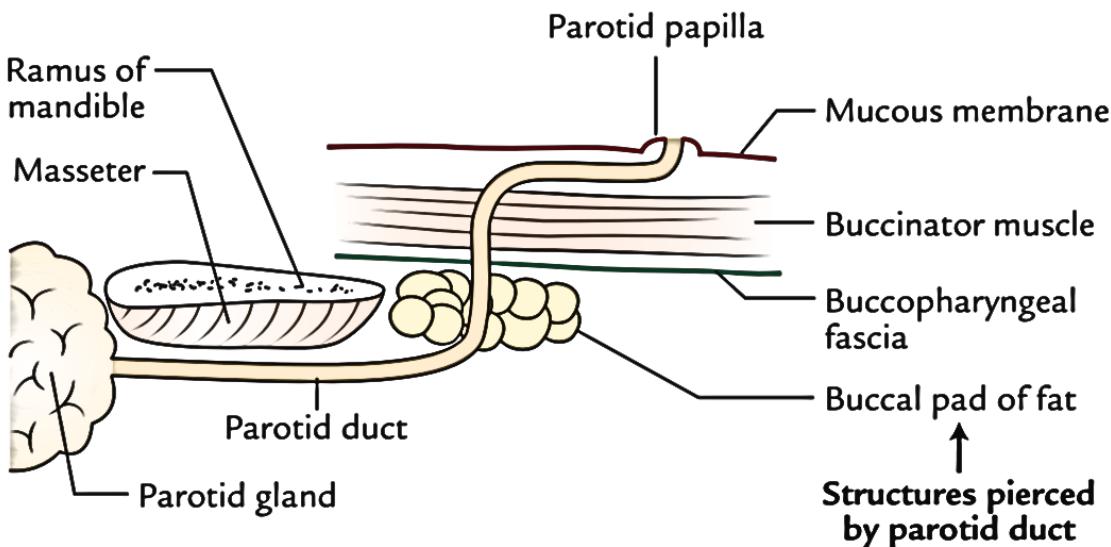


NB: greater auricular nerve is routinely injured during superficial parotidectomy

Parotid duct (Stensen's duct):

- It is a cord like duct arises from the anteromedial surface of the gland at its anterior border.
- It passes forward on the masseter muscle till its anterior border, then curves medially piercing:
 - Buccal pad of fat,
 - Buccopharyngeal fascia,
 - Buccinator muscle
 - Mucous membrane of the cheek.
- It opens into the vestibule of the mouth opposite the **upper 2nd molar teeth**.

Surface anatomy: It is represented by the middle 1/3 of a line extends from the tragus of the auricle till a point midway between the ala of the nose and the red margin of the upper lip



Blood supply of the gland:

- 1- **Arterial:** from External carotid artery& its branches.
- 2- **Venous:** it drains into retromandibular vein.

Nerve supply:

- 1- **Sensory supply:** By auriculo-temporal nerve.
- 2- **Sympathetic supply:** It arises from the superior cervical sympathetic ganglion around external carotid artery.
- 3- **Parasympathetic:** Otic ganglion

Lymph drainage: The parotid gland drains into Superficial and Deep parotid lymph nodes

The Otic Ganglion

Site: It is a small parasympathetic ganglion that lies **below** the foramen ovale & suspended from **mandibular nerve**.

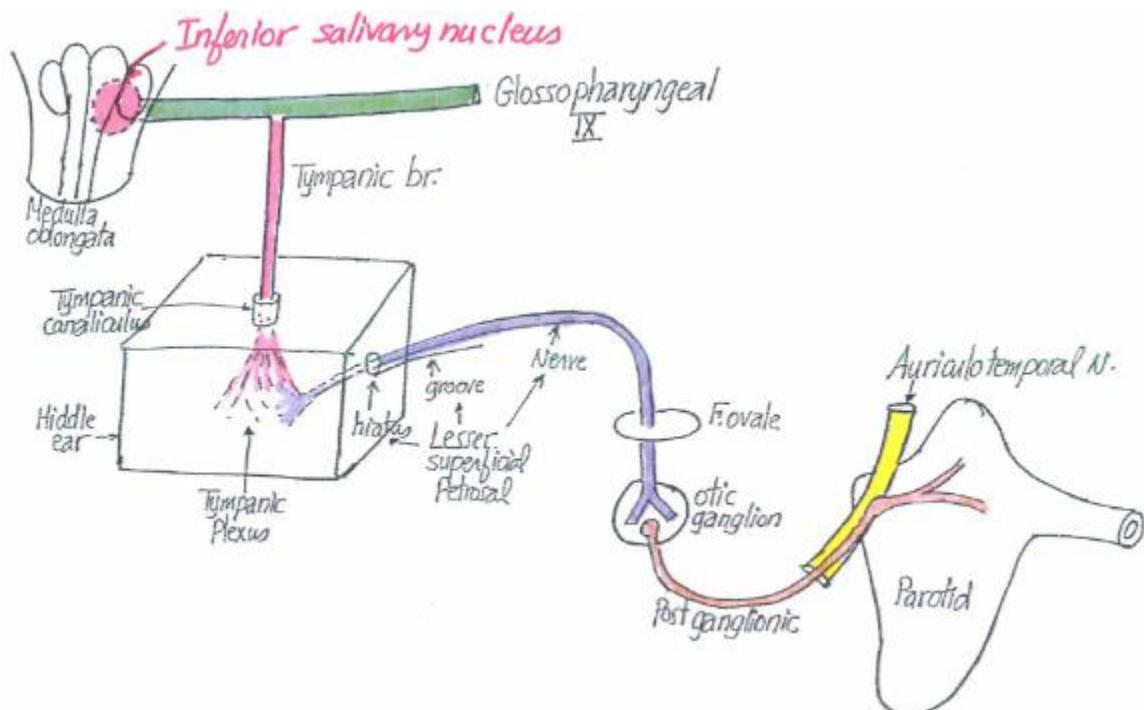
Roots of the otic ganglion:

1- Parasympathetic root:

- arise from the **inferior salivary nucleus** in the medulla oblongata. Then to **glossopharyngeal nerve** then with its **tympanic branch** then to middle ear inside petrous bone.
- The nerve leaves petrous bone & now is called **lesser superficial petrosal nerve** that relay in the **otic ganglion**.
- Postganglionic fibers pass with the **auriculotemporal nerve** to supply the parotid gland

2- Sympathetic root:

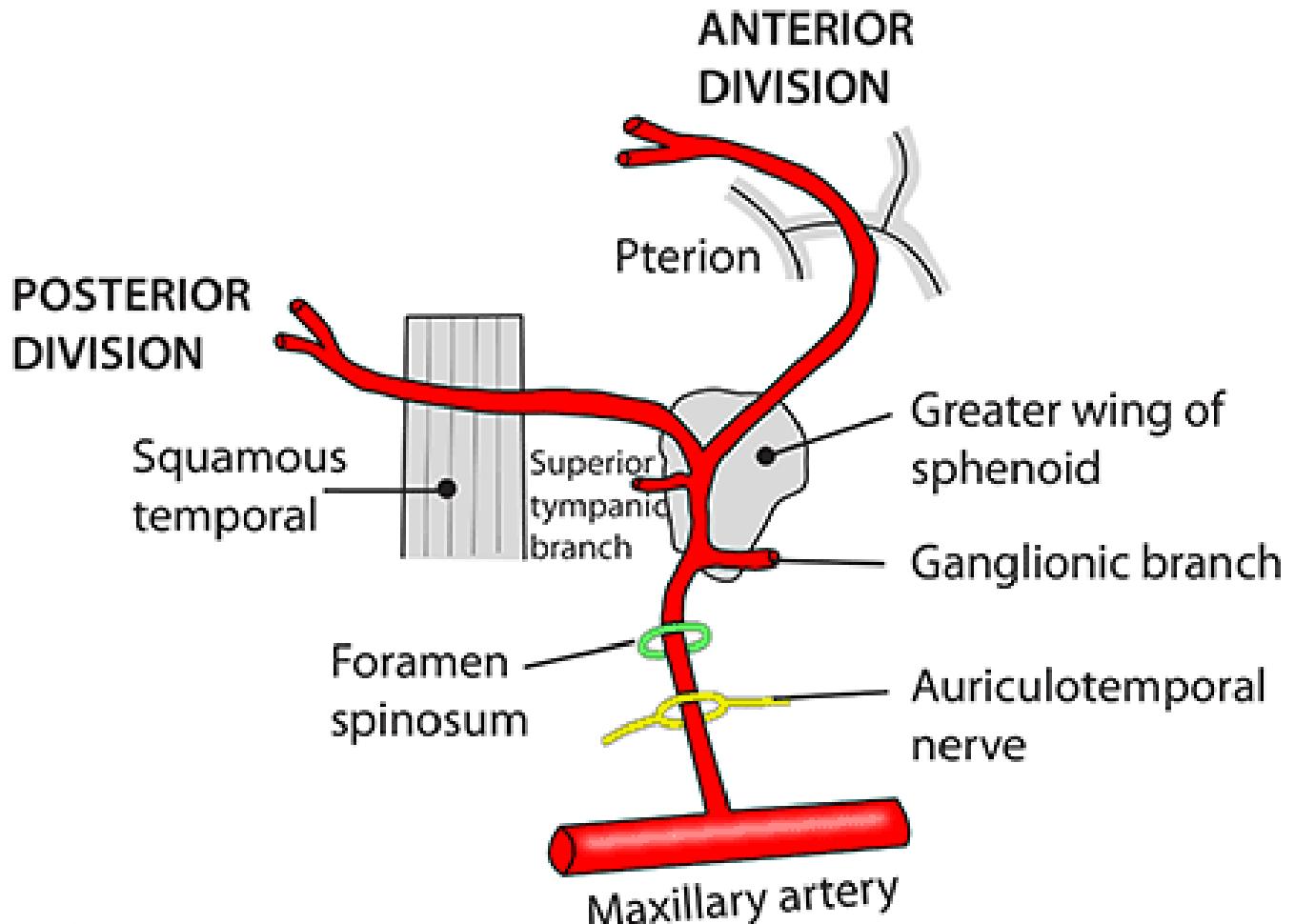
- arise from the **superior cervical sympathetic ganglion** around the external carotid artery.



The middle meningeal artery

- **Origin:** 1st part of the maxillary artery, it enters skull through the **foramen spinosum** to supply the dura mater (the outermost meninges).
- The middle meningeal artery is the largest of the three (paired) arteries which supply the meninges, the others being the anterior meningeal artery and the posterior meningeal artery.
- The middle meningeal artery runs **beneath the pterion**. It is vulnerable to injury at this point, where the skull is thin. Rupture of the artery may give rise to an **extra dural hematoma**.
- In the dry cranium, the middle meningeal, which runs within the dura mater surrounding the brain, makes a deep indentation in the calvarium.
- The middle meningeal artery is intimately **associated with the auriculotemporal nerve** which wraps around the artery making the two easily identifiable in the dissection of human cadavers and also easily damaged in surgery.

RIGHT MIDDLE MENINGEAL ARTERY



Submandibular salivary gland

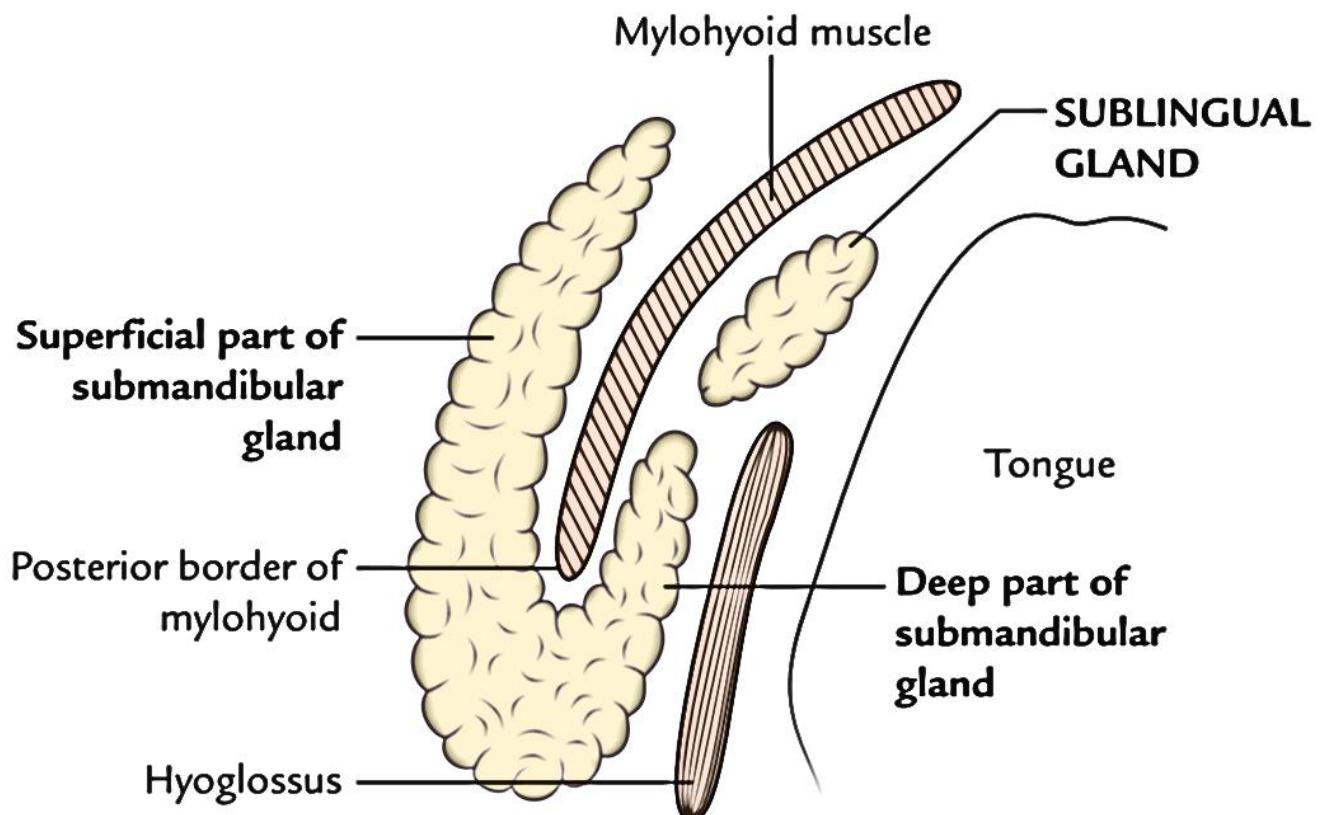
It is a mixed exocrine gland formed of mucous and serous parts.

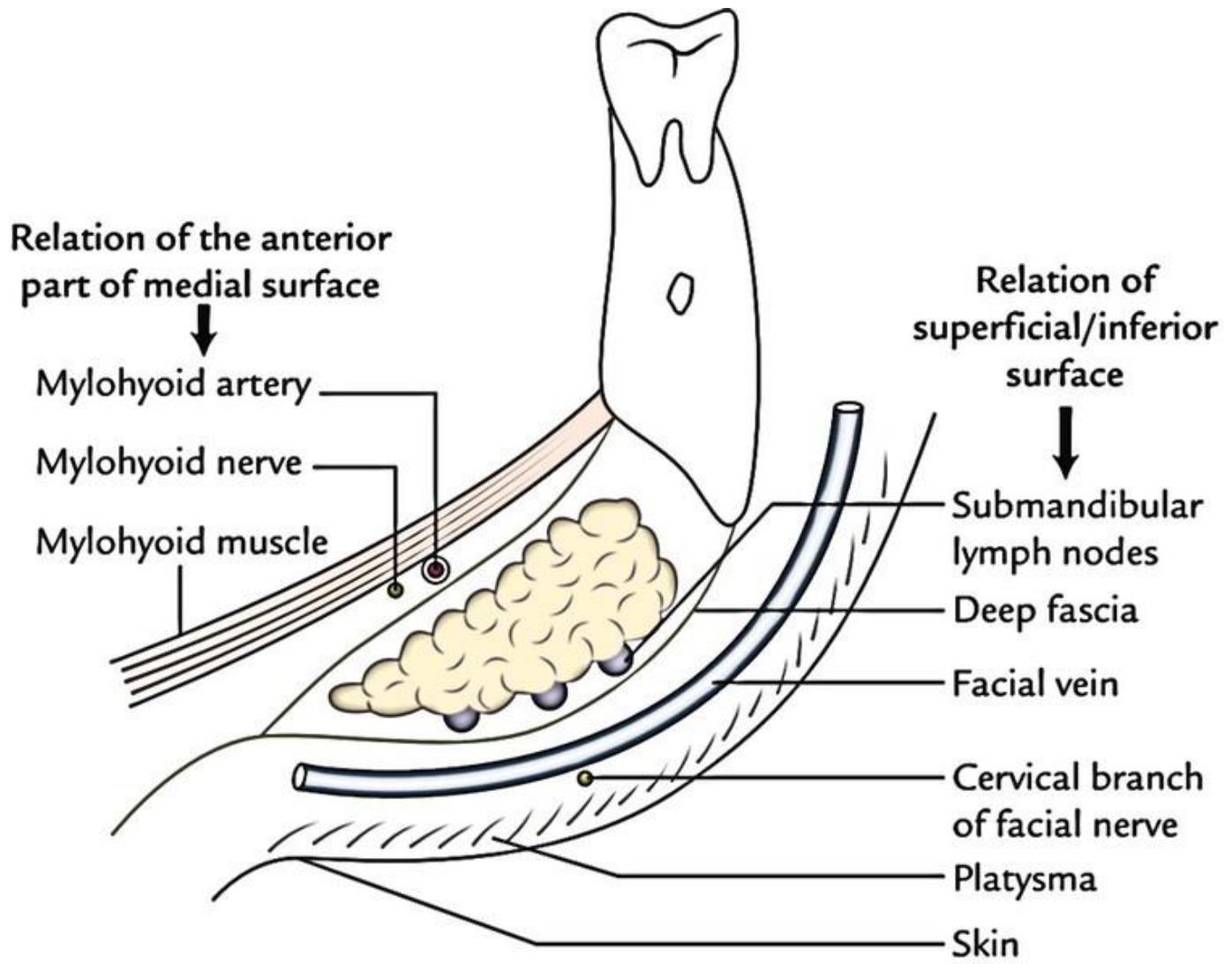
Aspects and position

- It is a C-shaped gland consisting of a large superficial and a small deep parts.
- The 2 parts continues with each other around the posterior border of mylohyoid muscle by a connecting part of the gland.

Relations:

Superficial part	Deep part
Platysma, deep fascia and mandible Submandibular lymph nodes Facial vein (facial artery near mandible) Marginal mandibular nerve Cervical branch of the facial nerve	Facial artery (inferior to the mandible) Mylohyoid muscle Sub mandibular duct Hyoglossus muscle Lingual nerve Submandibular ganglion Hypoglossal nerve





Submandibular (Wharton's) duct:

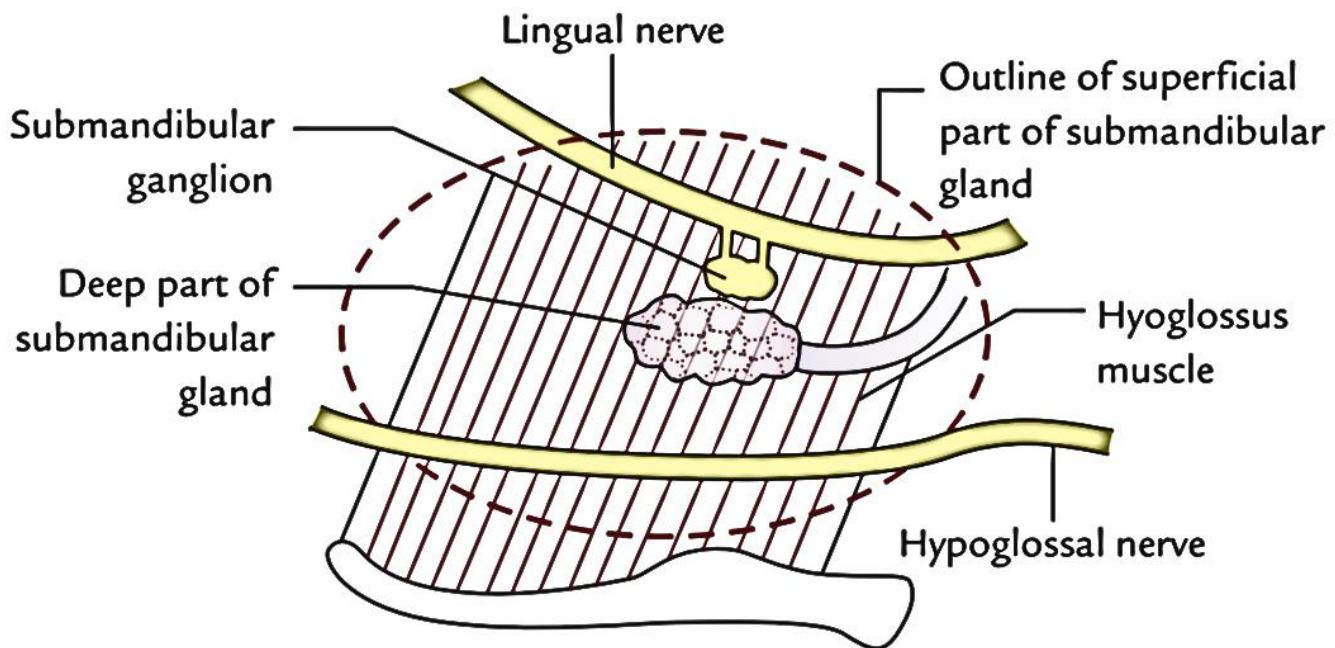
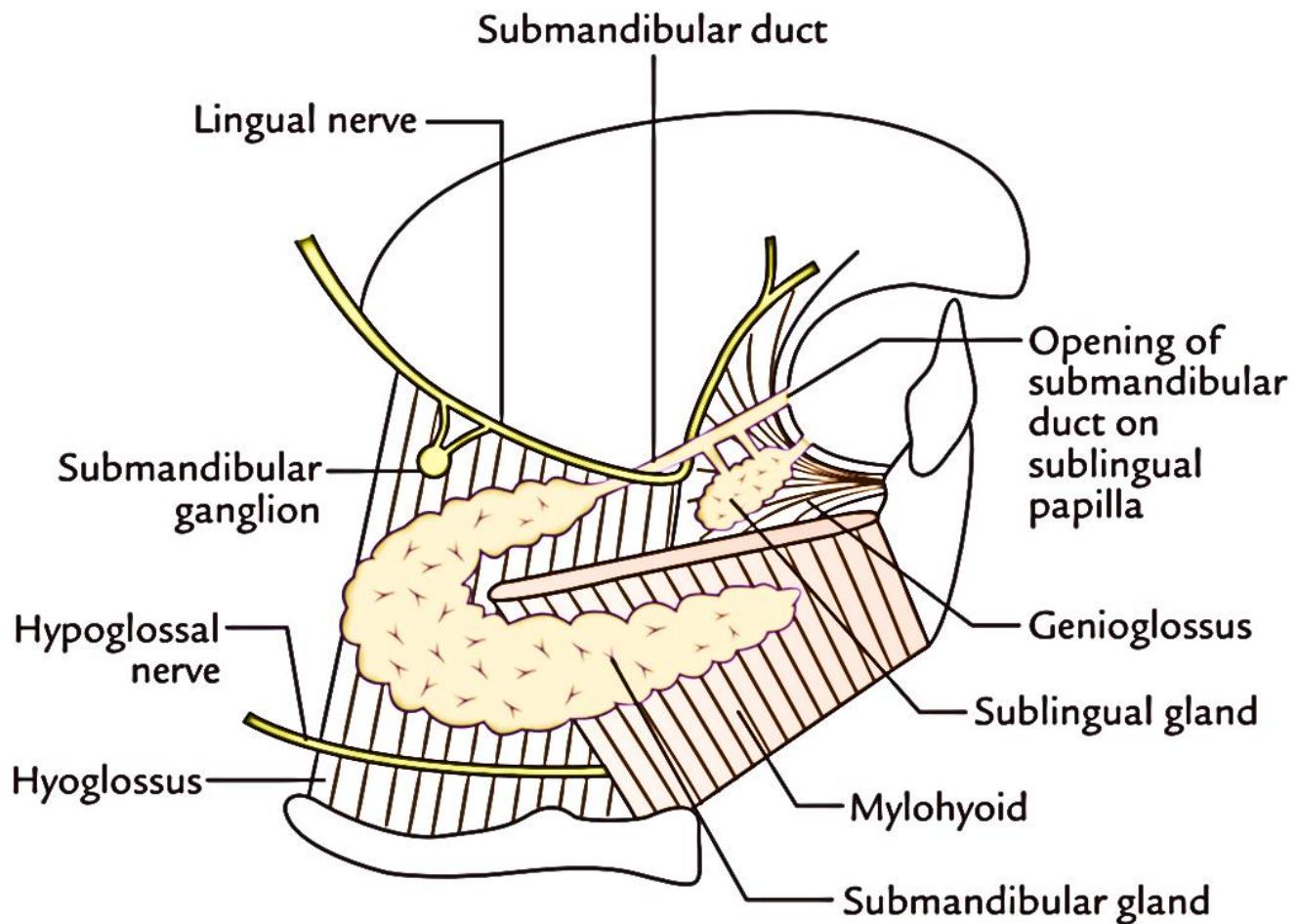
- It arises from the anterior part of the deep part of the gland.
- It opens in the floor of the mouth **on the sublingual papilla** at the side of the frenulum of the tongue.

Blood supply:

- **Arterial:** from the facial and lingual arteries
- **Venous:** into the common facial vein.

Lymph drainage: drain into submandibular lymph nodes.

Nerve supply: *submandibular ganglion.*



Submandibular ganglion

Roots of the ganglion:

1- Parasympathetic fibers:

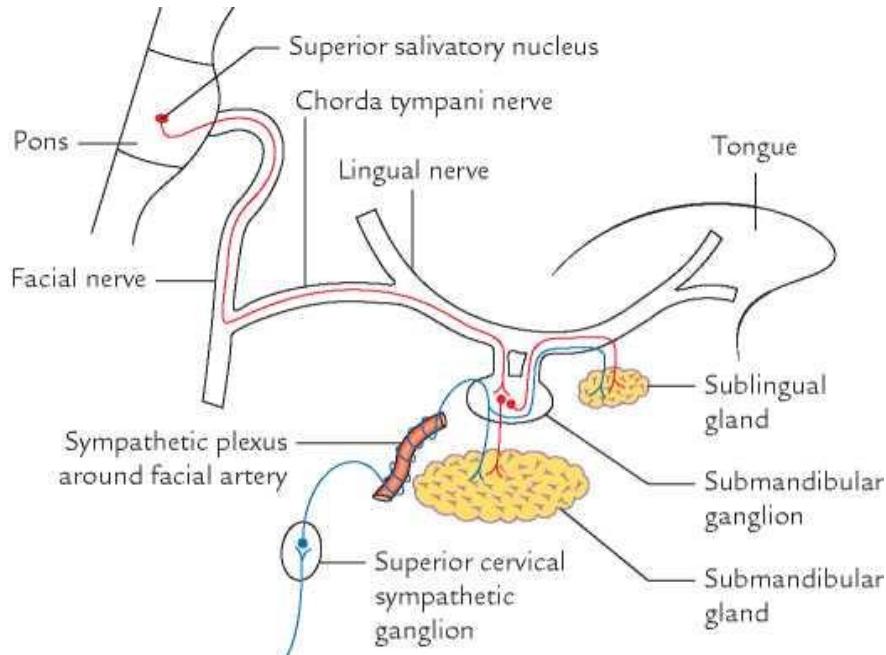
- It arises from superior salivary nucleus in the pons accompanying the facial nerve, chorda tympani branch and joins the lingual nerve to relay in submandibular ganglion.

2- Sympathetic fiber:

- It arises from the superior cervical sympathetic ganglion and reaches the gland through the plexus around the facial artery.

3- Sensory fibers:

- They are branches from the lingual nerve.



Inguinal canal

Definition: It is an oblique passage through lower part of anterior abdominal wall muscles

Direction: It extends down and medially.

Length: 4 cm.

Contents:

1. Ilioinguinal nerve (**in both sexes**).
2. Spermatic cord (& its contents) in **male**.
3. Round ligament of the uterus in **female**.

Beginning → At the Deep (internal) inguinal ring:

- It is an oval opening *in fascia transversalis*
- It is $\frac{1}{2}$ inch above mid point of inguinal ligament.

End → Superficial (external) inguinal ring: It is triangular opening *in the aponeurosis of external oblique* just above & lateral to the pubic tubercle.

Boundaries:

1) Anterior wall:

- Ext. oblique aponeurosis.
- Internal oblique aponeurosis

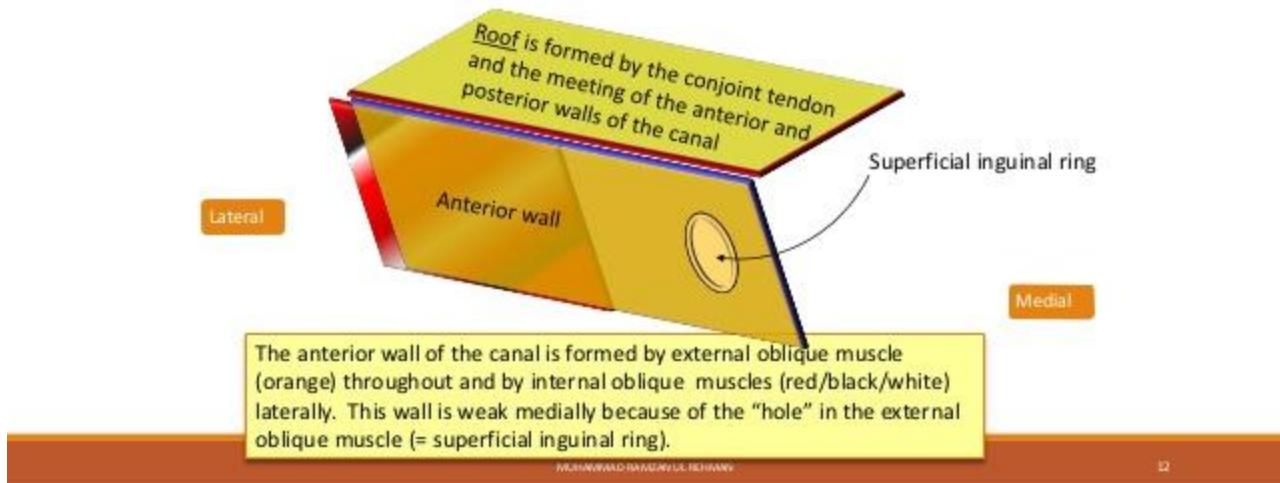
2) Posterior wall:

- Fascia **t**ransversalis.
- Conjoint **t**

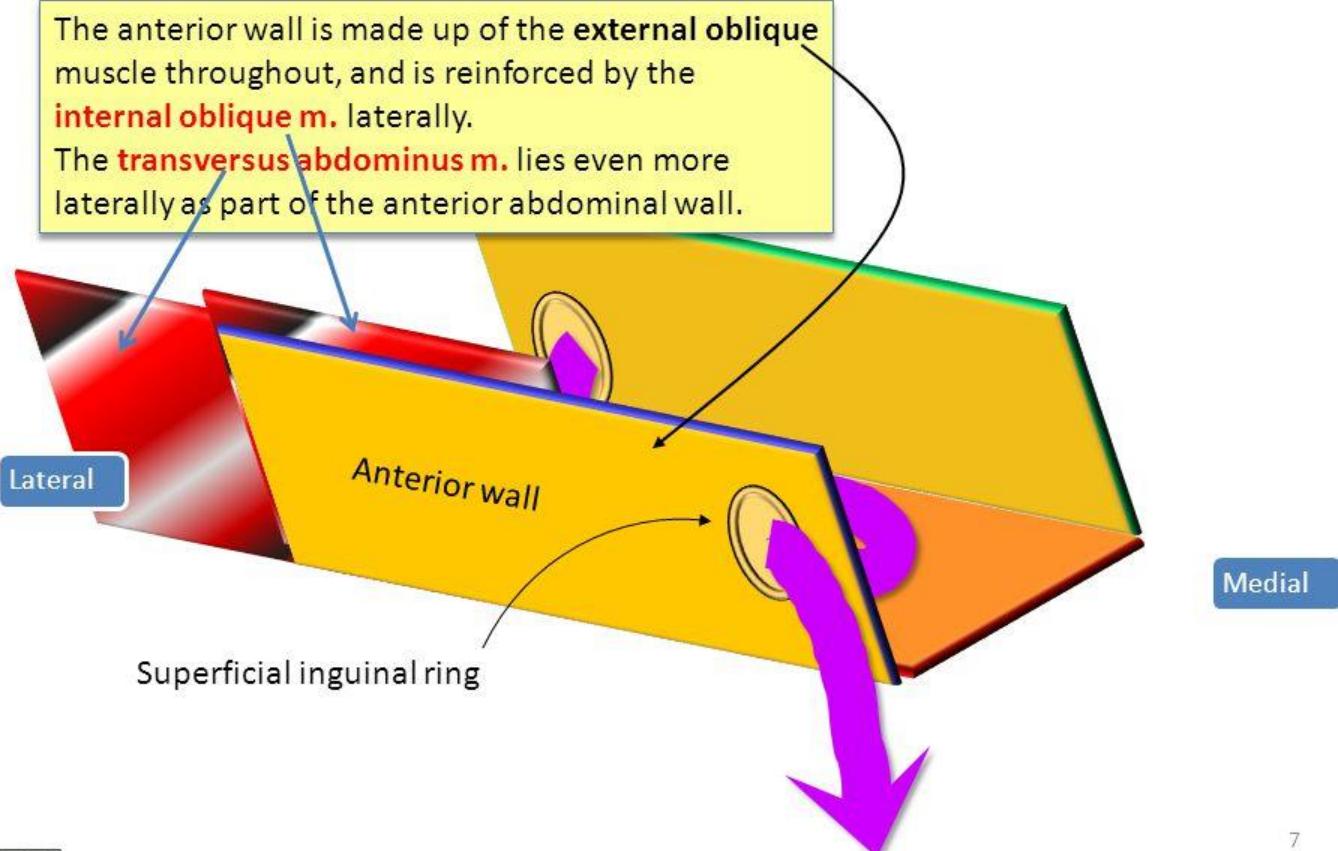
3) Roof: conjoint tendon.

4) Floor: inguinal ligament.

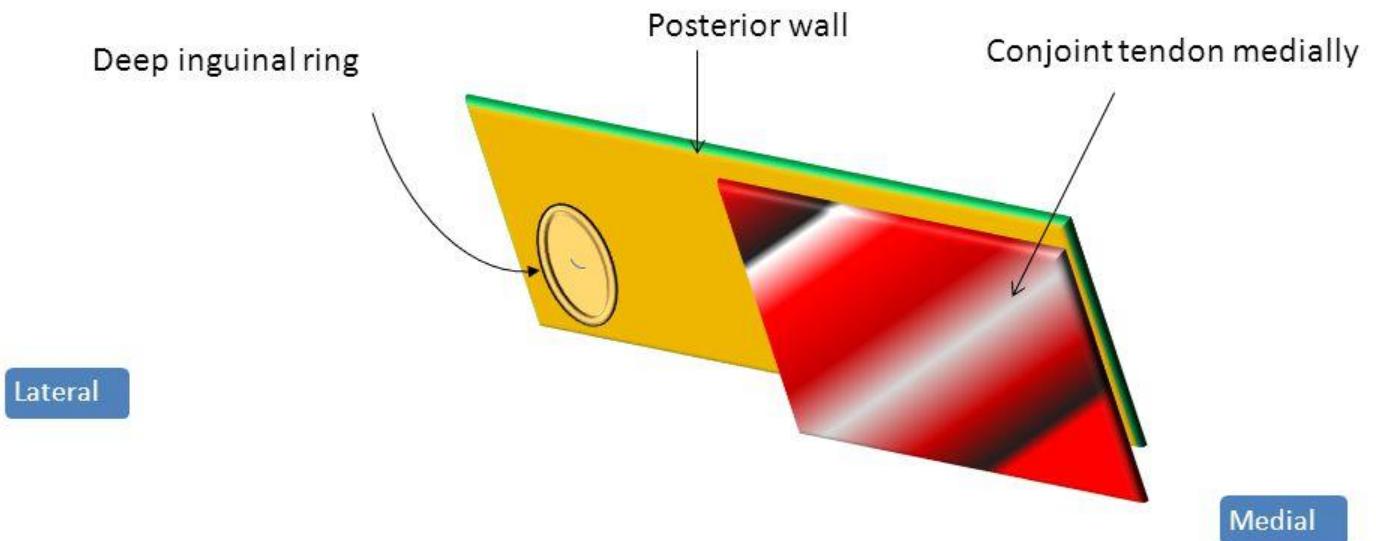
Roof and anterior wall of the inguinal canal



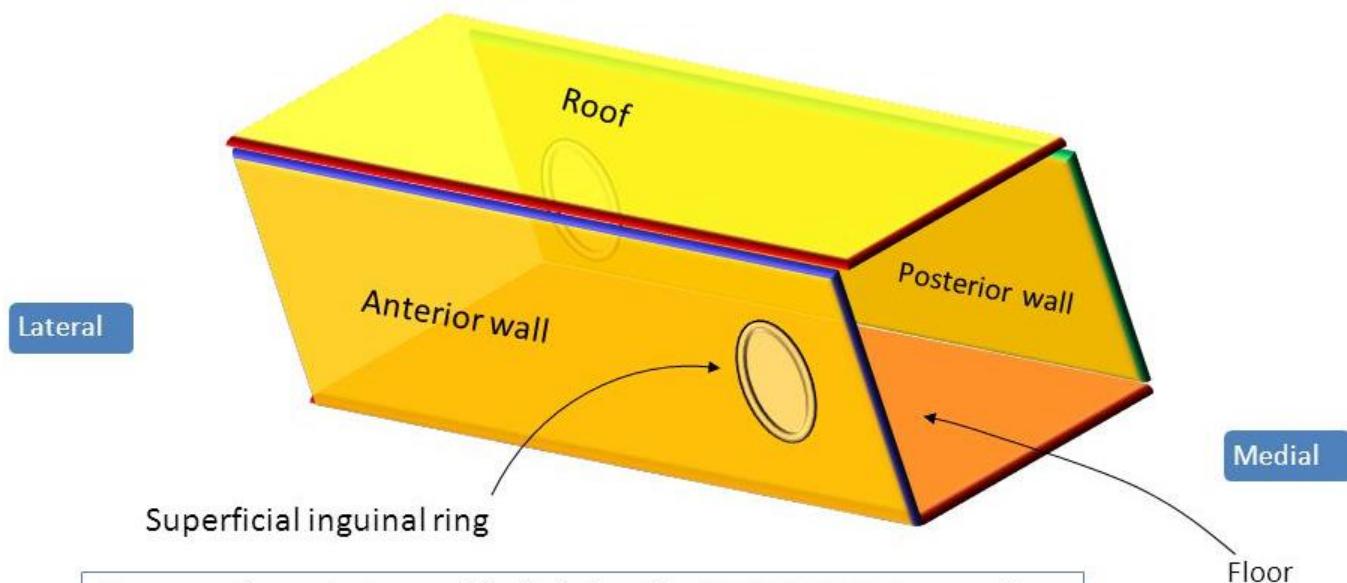
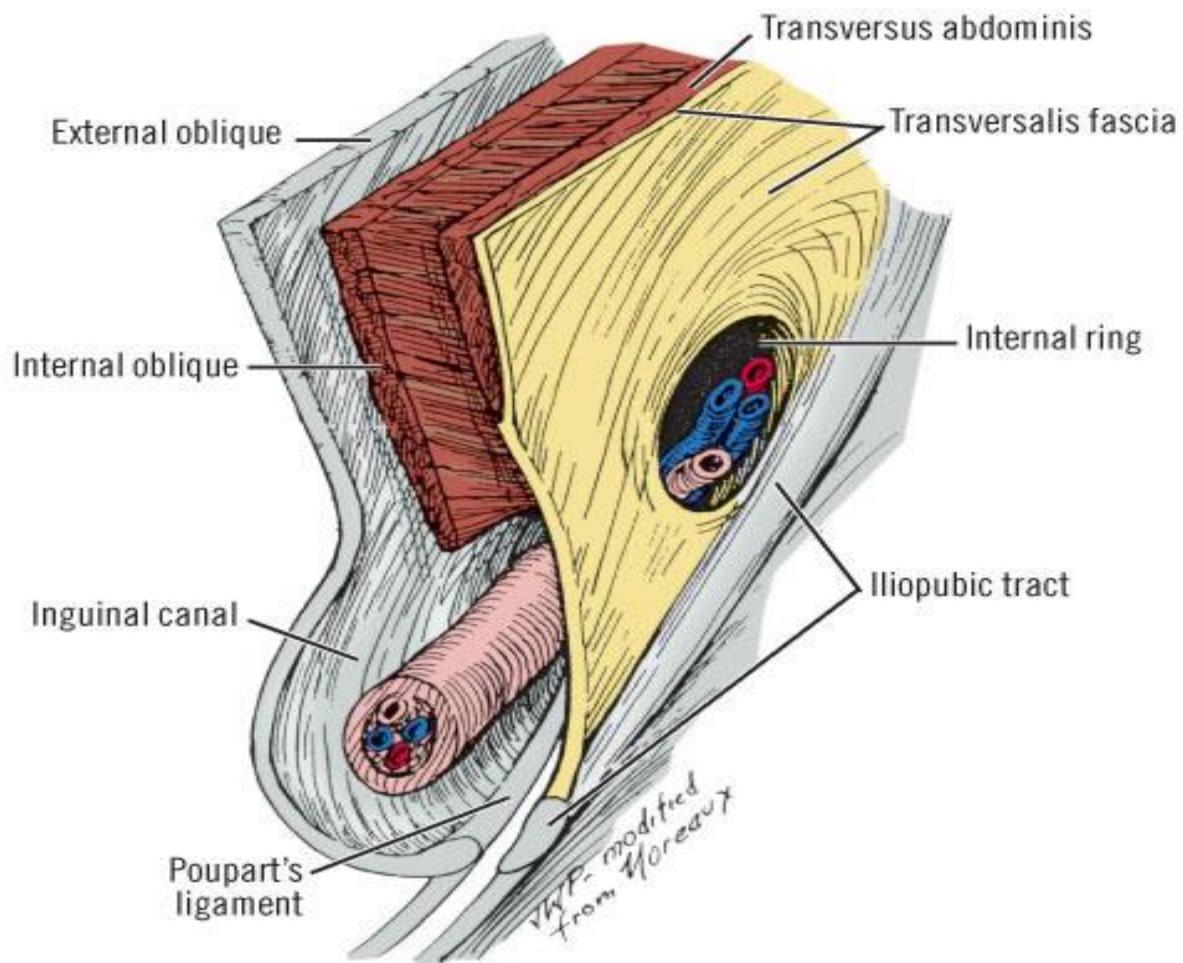
Inguinal canal



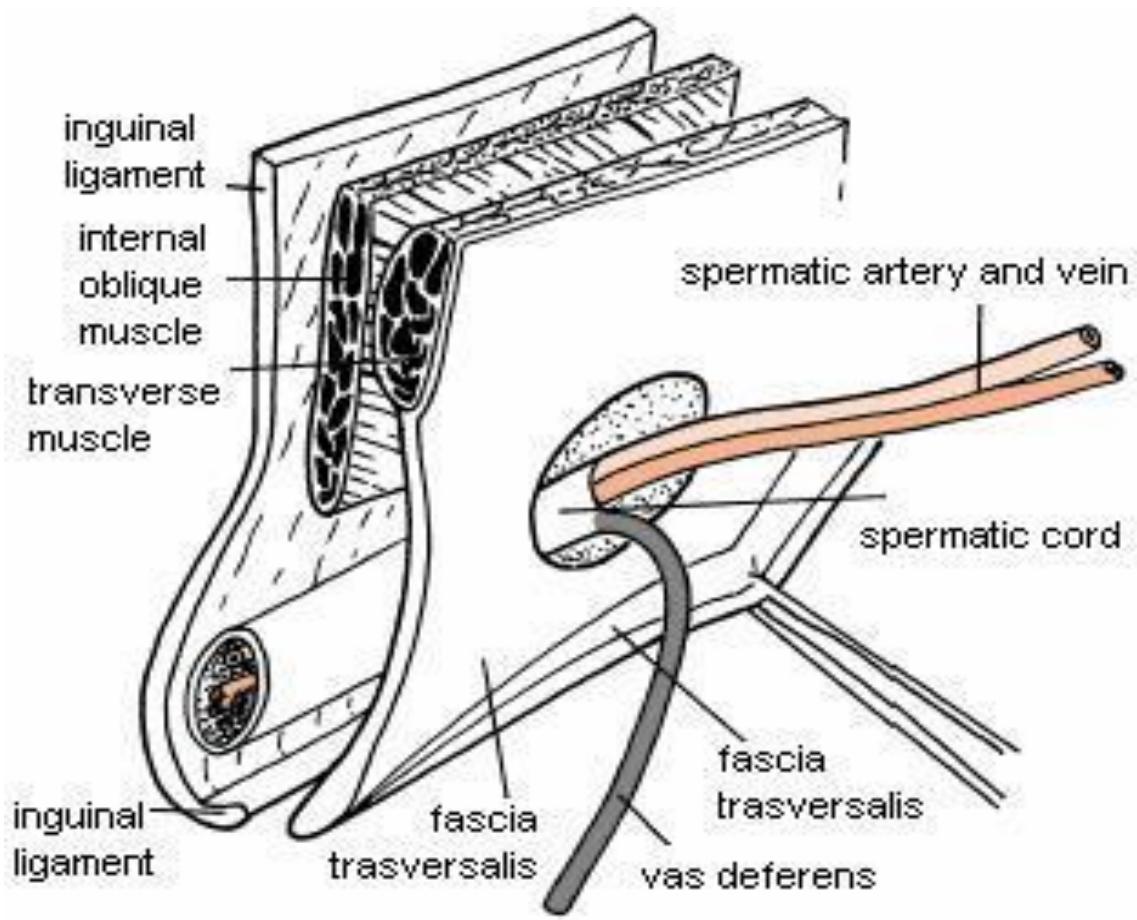
Posterior wall of the inguinal canal

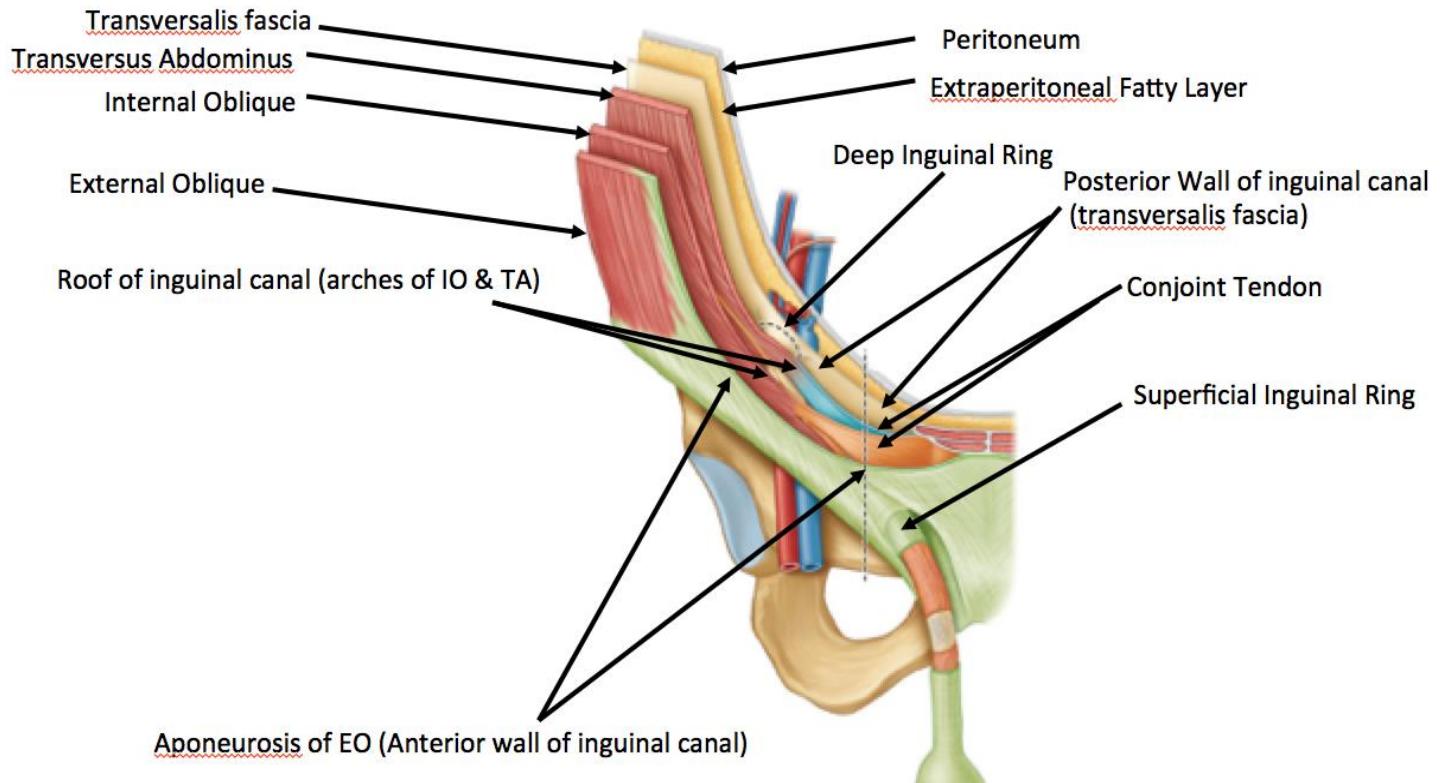


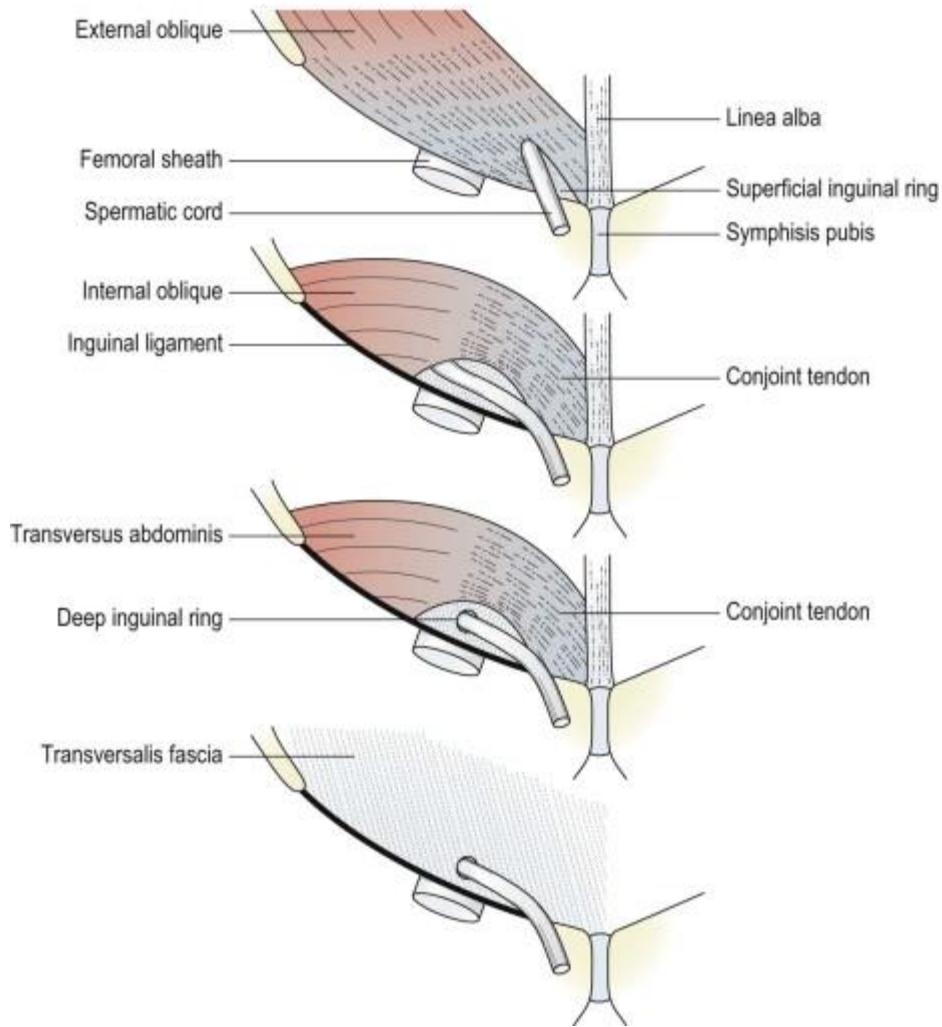
The posterior wall is formed by transversalis fascia (orange) throughout and the conjoint tendon (red) medially. The wall is particularly weak over the deep inguinal ring



Here are the anterior wall (which has the SUPERFICIAL inguinal ring situated medially), and the roof.



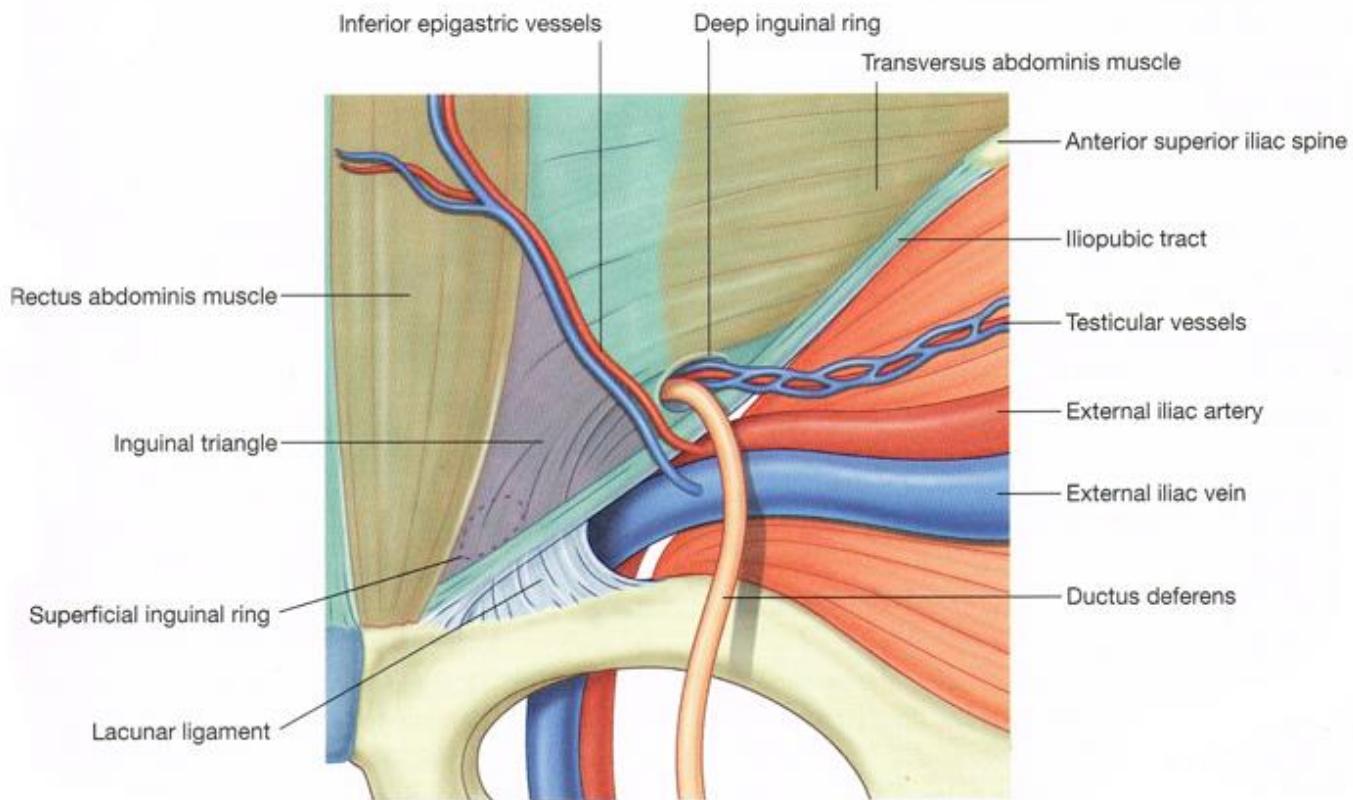




Inguinal triangle (Hasselbach triangle)

Boundaries:

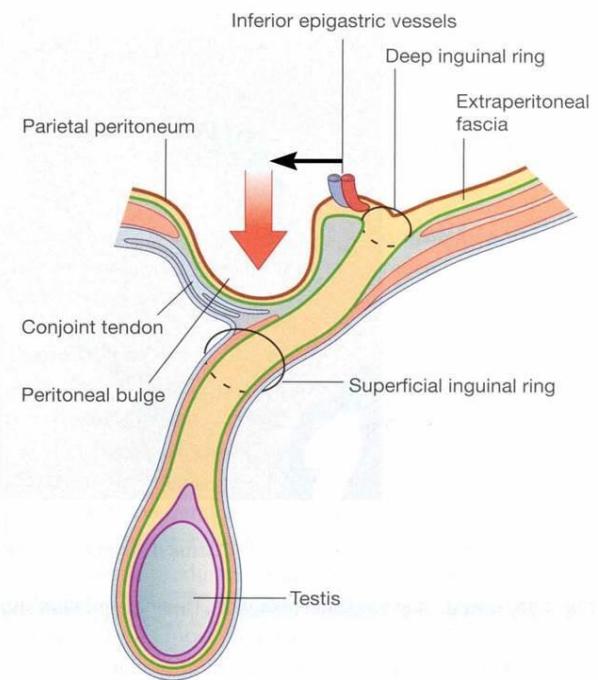
- **Medially:** Lateral border of rectus abdominis muscle.
- **Laterally:** the inferior epigastric vessels.
- **Inferiorly:** the inguinal ligament



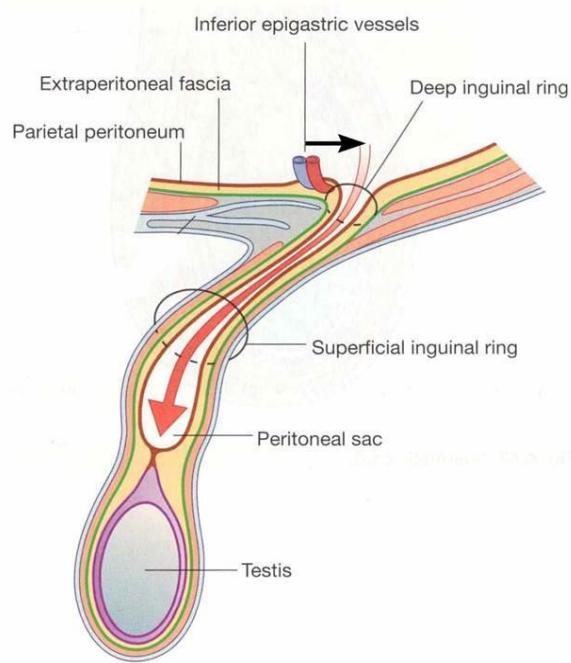
Inguinal hernia

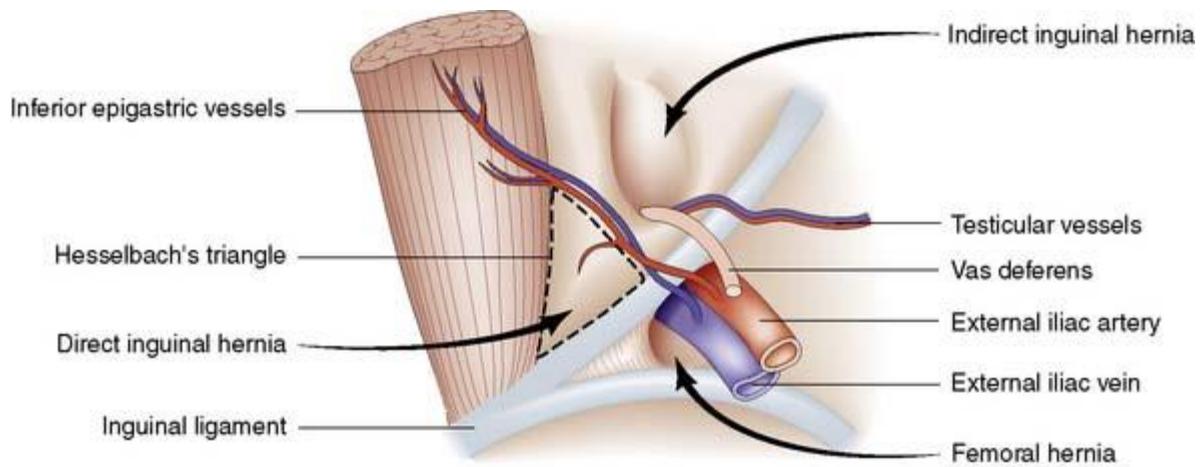
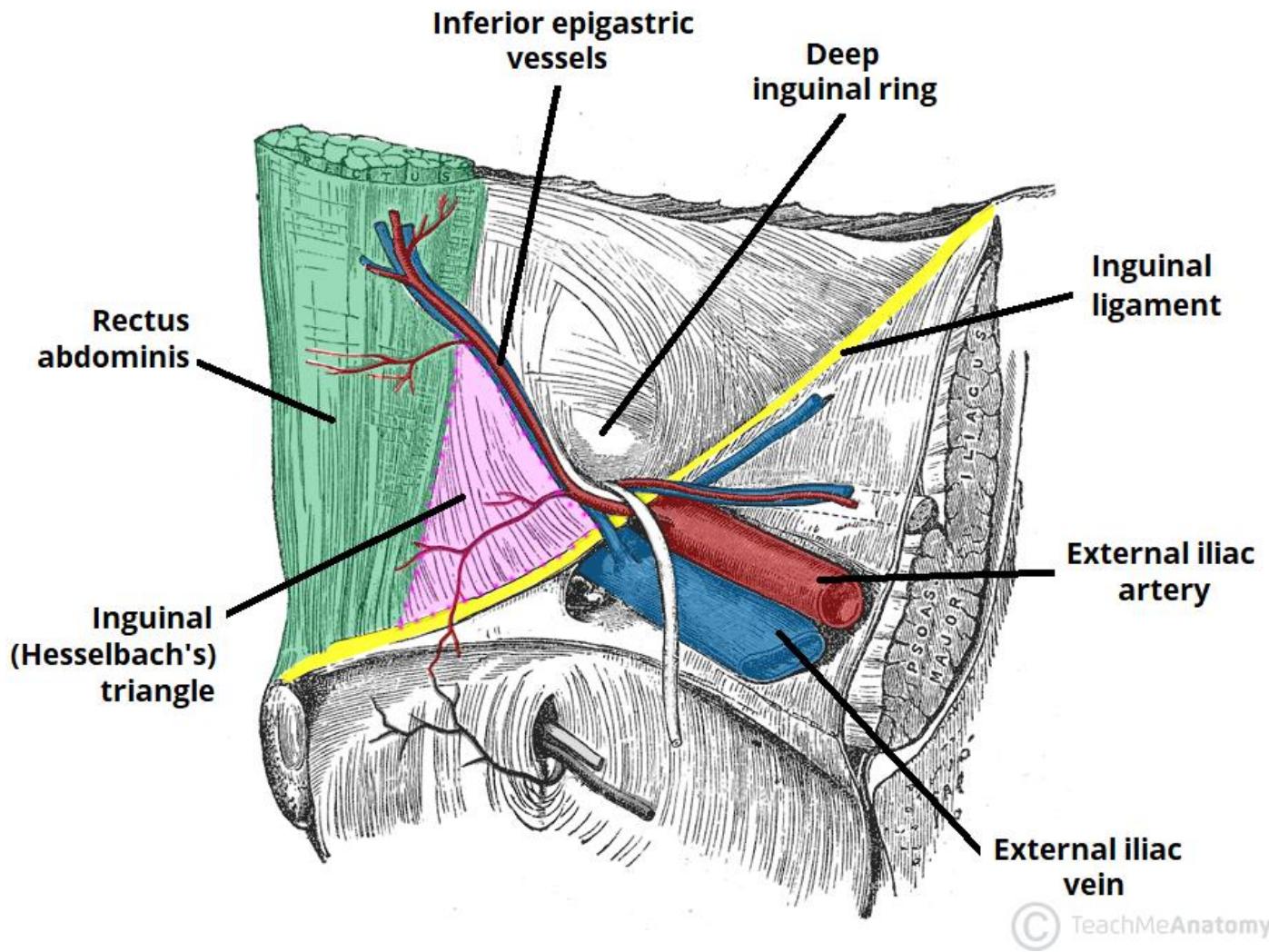
	Direct inguinal hernia	Indirect inguinal hernia
Defect	In inguinal triangle	Deep inguinal ring
	Medial to inferior epigastric artery	Lateral to inferior epigastric artery
	Never enters the scrotum	May enter the scrotum

Direct



Indirect





Rectus sheath

Definition: It is an aponeurotic sheath enveloping the rectus abdominis and pyramidalis muscles.

It is formed by the aponeuroses of the three muscles of the lateral abdominal wall.

Formation:

A) Upper part (above xiphoid process):

- **Anterior wall:** external oblique aponeurosis.
- **Posterior wall:** 5th, 6th and 7th costal cartilages.

B) Middle part (Between xiphoid process & arcuate line):

- **The anterior wall:** external oblique & anterior lamina of internal oblique aponeurosis.
- **The posterior wall:** posterior lamina of internal oblique, transversus abdominis aponeurosis & fascia transversalis.

C) Lower part (below arcuate line):

- **Anterior wall:** the 3 aponeuroses.
- **The posterior wall:** fascia transversalis.

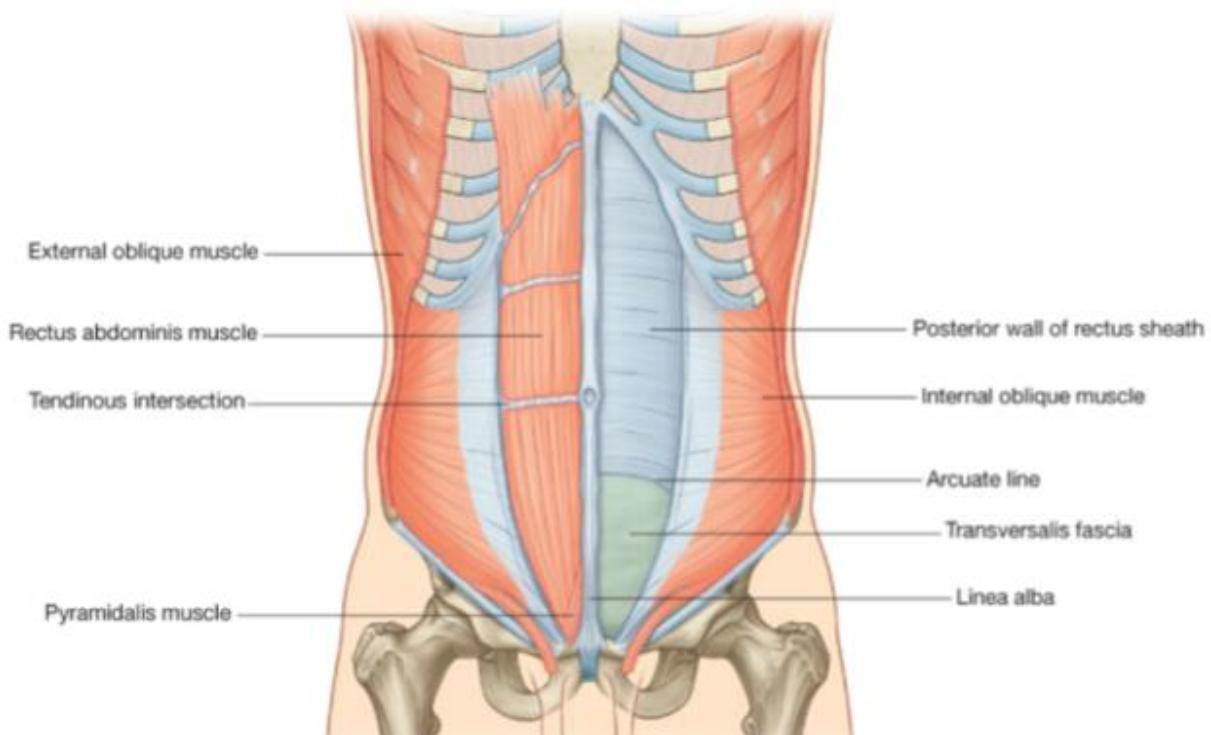
Contents of the rectus sheath:

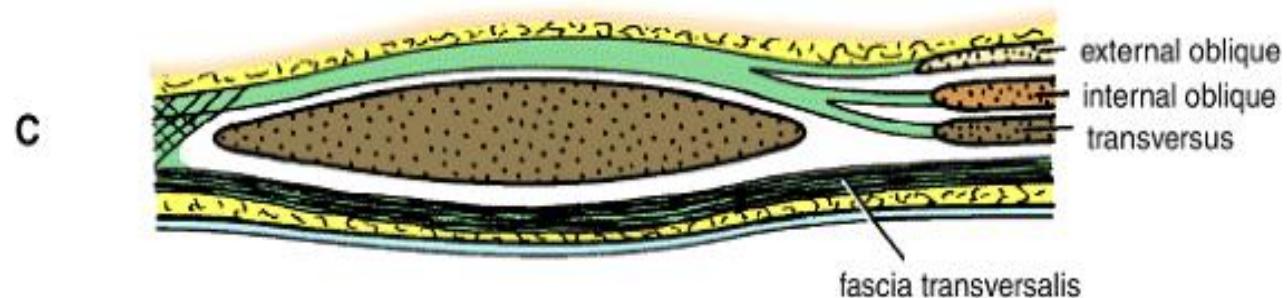
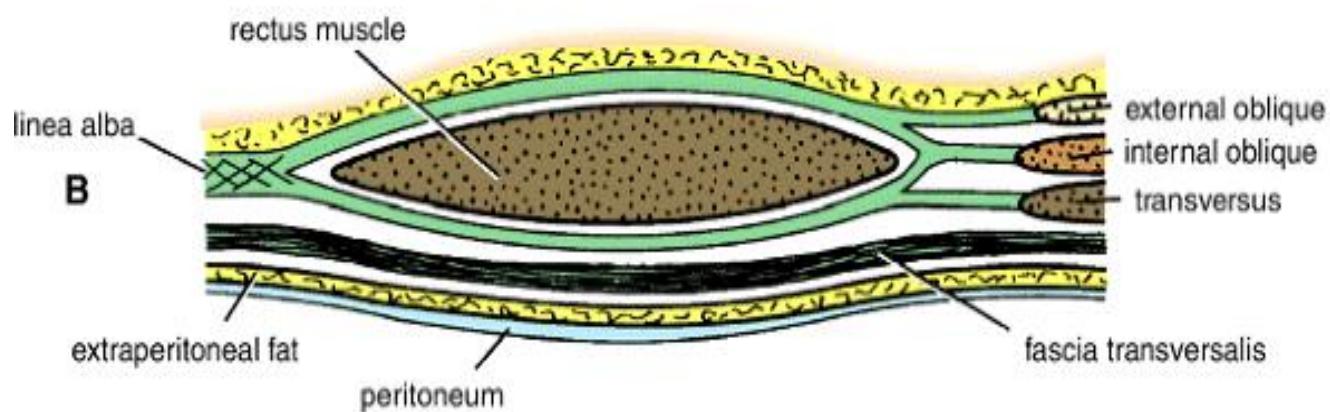
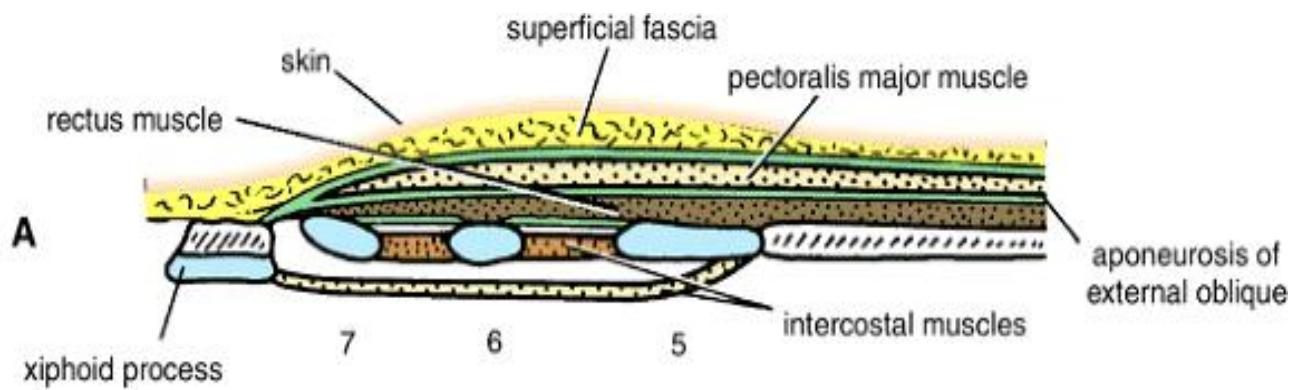
a) **Two muscles:** Rectus abdominis & Pyramidalis.

b) **2 vessels:** sup. & inferior epigastric vessels.

c) **Lower 5 intercostal, subcostal Ns & vessels.**

d) **Connective tissue, lymphatics.**





Spermatic cord

Def: group of structures passing to or from the testis surrounded by tubular covering

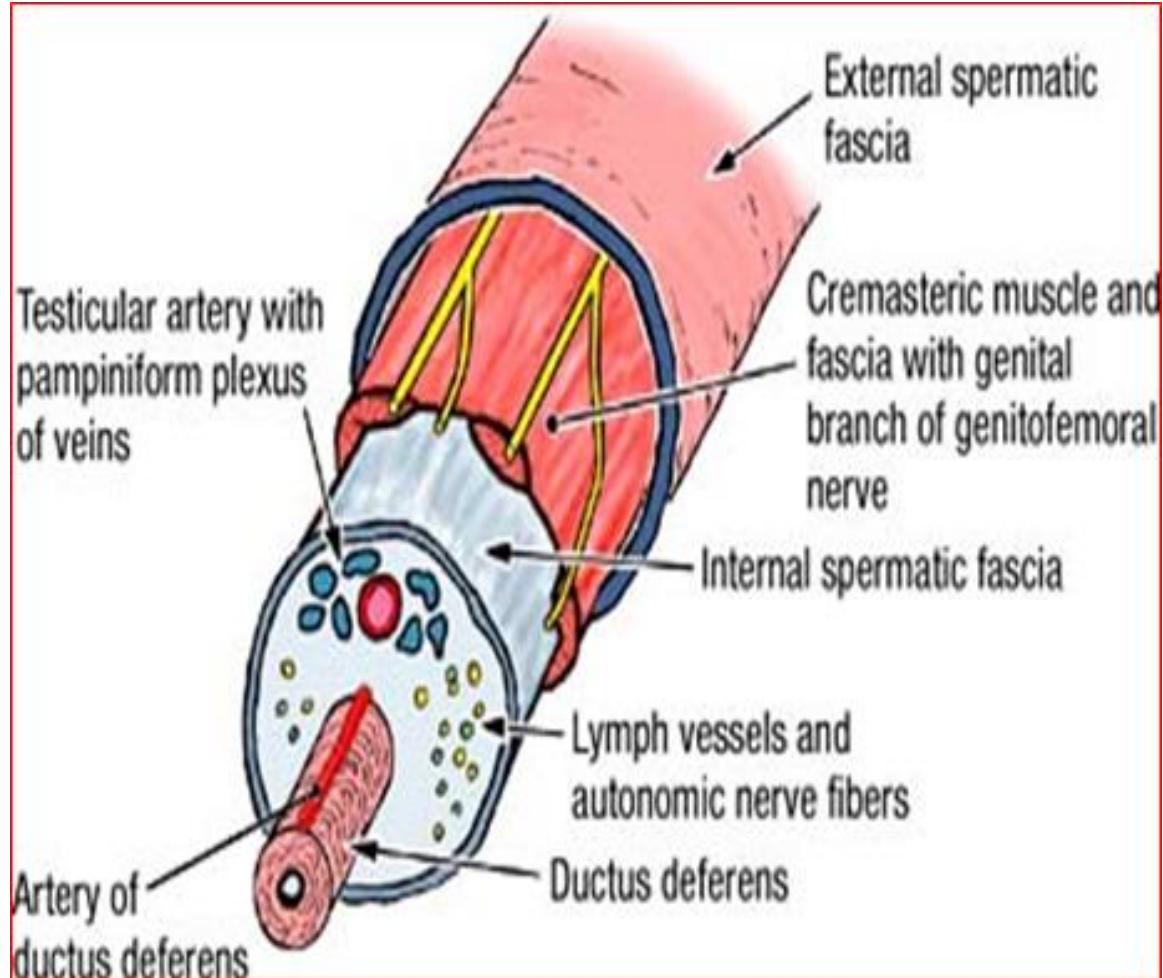
Site: in scrotum & in inguinal canal

Covering: Formed by the vas deferens and is covered by the following structures:

Layer	Origin
Internal spermatic fascia	Transversalis fascia
Cremasteric fascia	From the fascial coverings of internal oblique
External spermatic fascia	External oblique aponeurosis

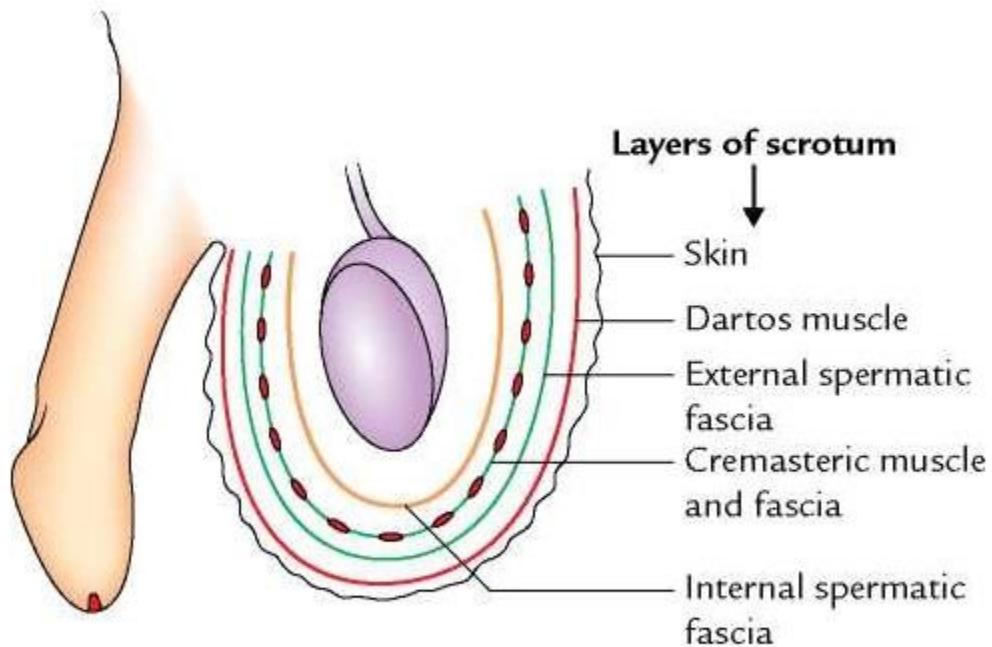
Contents of the cord:

Vas deferens	Transmits sperm and accessory gland secretions
Testicular artery	Branch of abdominal aorta supplies testis and epididymis
Artery of vas deferens	Arises from inferior vesical artery
Cremasteric artery	Arises from inferior epigastric artery
Pampiniform plexus	Venous plexus, drains into right or left testicular vein
Sympathetic nerve fibres	Lie on arteries, the parasympathetic fibres lie on the vas
Genital branch of the genitofemoral nerve	Supplies cremaster
Lymphatic vessels	Drain to lumbar and para-aortic nodes



Scrotum

- **Arterial supply:** anterior and posterior scrotal arteries
- **Lymphatic drainage:** to the superficial inguinal lymph nodes
- **Nerve supply:**
 - Anterior → ilioinguinal nerve
 - Posterior → pudendal nerve
- **Layers of the scrotum** (Some Damn Englishman Called It The Testis)
 - Skin
 - Dartos fascia and muscle → **derived from scarpa's fascia**
 - External spermatic fascia
 - Cremasteric fascia
 - Internal spermatic fascia
 - Tunica vaginalis
 - Testes



Testes

The testes are surrounded by the tunica vaginalis (closed peritoneal sac). The parietal layer of the tunica vaginalis adjacent to the internal spermatic fascia.

Blood supply: testicular arteries arise from the aorta immediately inferiorly to the renal arteries.

Venous drainage: pampiniform plexus drains into the testicular veins, the left drains into the left renal vein and the right into the inferior vena cava.

Lymphatic drainage: is to the para-aortic nodes.

Testicular embryology

During foetal life the testicles are located within the abdominal cavity. They are initially located on the posterior abdominal wall on a level with the upper lumbar vertebrae (L2).

Testicular descent is guided by the gubernaculum which extends caudally to the inguinal region, through the canal and down to the superficial skin.

As the foetus grows the gubernaculum becomes progressively shorter. It carries the peritoneum of the anterior abdominal wall (the processus vaginalis). As the processus vaginalis descends the testis is guided by the gubernaculum down the posterior abdominal wall and the back of the processus vaginalis into the scrotum.

By the third month of foetal life the testes are located in the iliac fossae, by the seventh they lie at the level of the deep inguinal ring.

The processus vaginalis usually closes after birth, but may persist and be the site of indirect hernias. Part closure may result in development of cysts on the cord.

Wound healing

Surgical wounds are either incisional or excisional and either clean, clean contaminated or dirty. Although the stages of wound healing are broadly similar their contributions will vary according to the wound type.

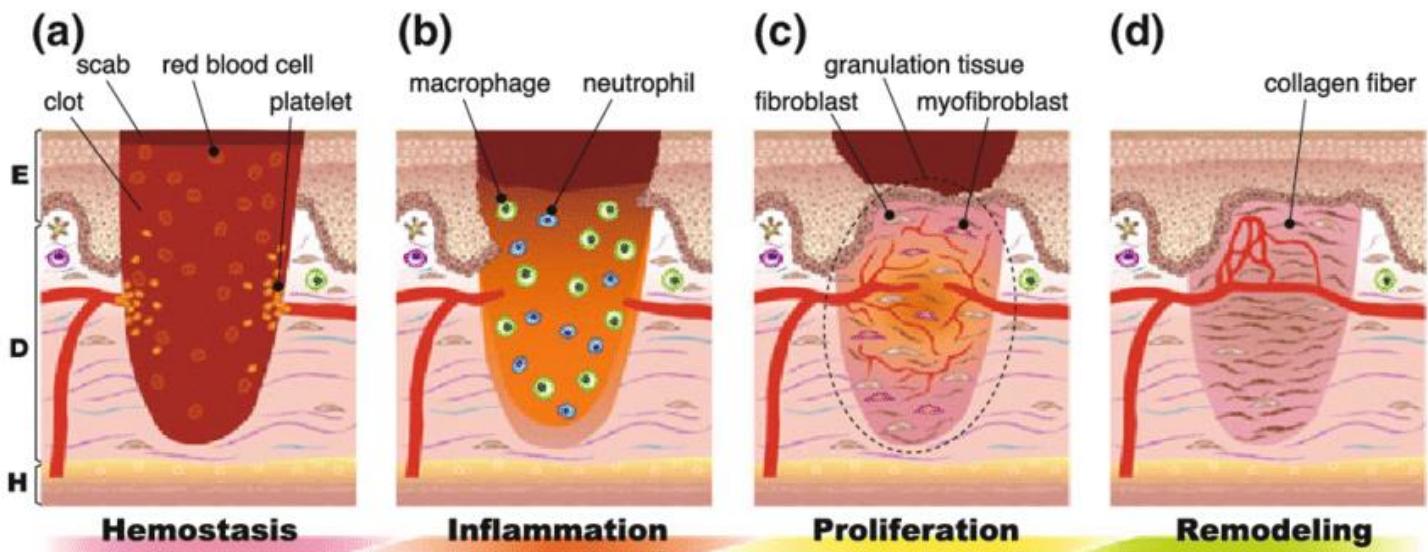
The main stages of wound healing include:

Phase	Key features	Cells	Time
Haemostasis	<ul style="list-style-type: none"> ✓ Vasospasm in adjacent vessels ✓ Platelet plug formation and generation of fibrin rich clot 	Erythrocytes & platelets	Seconds/ Minutes
Inflammation	<ul style="list-style-type: none"> ✓ Neutrophils migrate into wound (function impaired in diabetes). ✓ Growth factors released, including basic fibroblast growth factor and vascular endothelial growth factor. ✓ Fibroblasts migrate into wound. ✓ Macrophages and fibroblasts couple matrix regeneration and clot substitution. 	Neutrophils, fibroblasts and macrophages	Days (1 – 5)
Regeneration	<ul style="list-style-type: none"> ✓ Platelet derived growth factor and transformation growth factors stimulate fibroblasts and epithelial cells. ✓ Fibroblasts produce a collagen network. ✓ Angiogenesis occurs and wound resembles granulation tissue. 	Fibroblasts, endothelial cells, macrophages	Weeks
Remodeling	<ul style="list-style-type: none"> ✓ Longest phase of the healing process and may last up to one year (or longer). ✓ During this phase fibroblasts become differentiated (myofibroblasts) and these facilitate wound contraction. ✓ Collagen fibres are remodelled. ✓ Microvessels regress leaving a pale scar. 	Myofibroblasts	6 weeks to 1 year (Months)

Factors impairing wound healing:

- Vascular disease, shock and sepsis can all compromise microvascular flow and impair healing.
- Jaundice will impair fibroblast synthetic function and immunity with a detrimental effect in most parts of the healing process.
- Drugs which impair wound healing:

- Non steroidal anti inflammatory drugs
- Steroids
- Immunosuppressive agents
- Anti neoplastic drugs



Cushing's syndrome

The symptoms and signs of Cushing's syndrome are associated with high cortisone level

Clinical picture

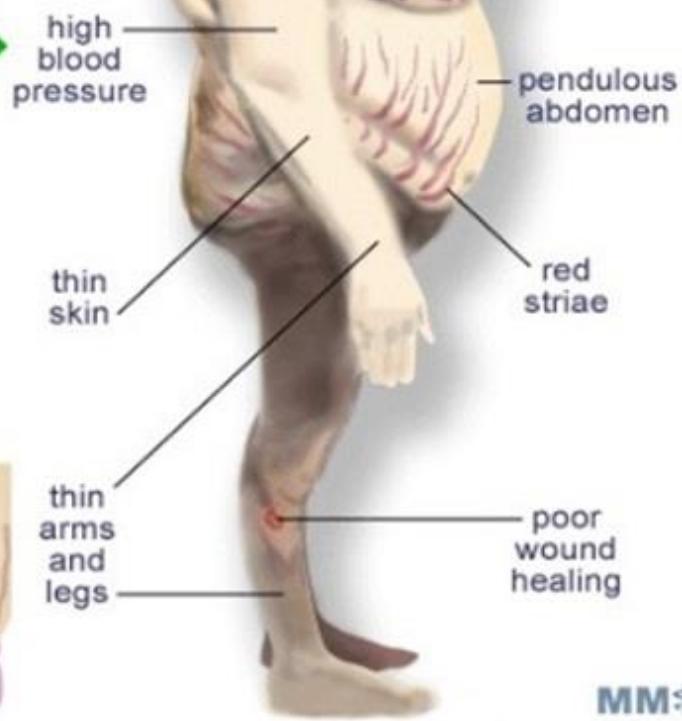
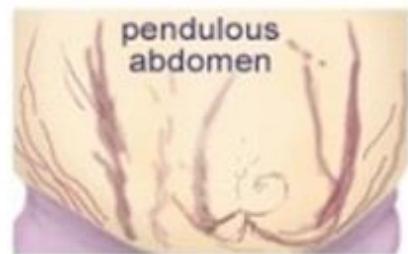
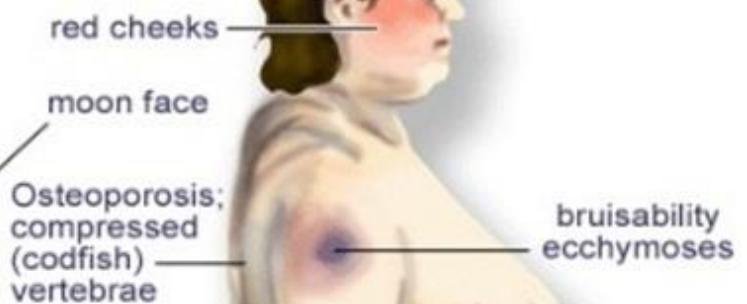
- central obesity and moon face
- plethora and acne
- menstrual irregularity
- hirsutism and hair thinning
- hypertension
- diabetes
- osteoporosis—may cause collapse of vertebrae, rib fractures
- muscle wasting and weakness
- atrophy of skin and dermis—paper thin skin with bruising tendency, purple striae.

Cushing's disease	Cushing's syndrome
Pituitary hypersecretion of ACTH → bilateral adrenal hyperplasia	Iatrogenic steroid therapy

Investigations:

- Plasma cortisol (raised)
- 24hrs urine cortisol (raised)
- **Dexamethasone suppression test** is diagnostic: 2mg oral dexta given at midnight and shows no change in morning cortisol level
- Plasma ACTH: Reduced, following CRH administration in CD but not, if source is ectopic
- Pituitary & Adrenal CT or MRI

Cushing's Syndrome



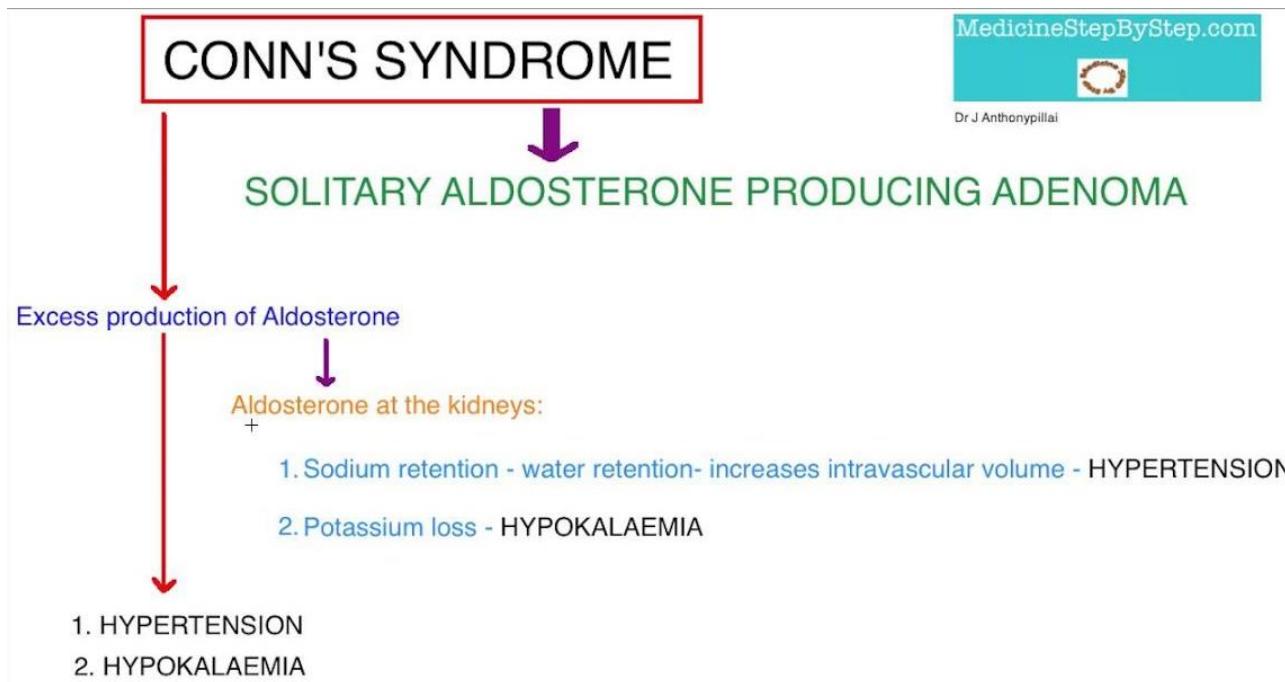
MM‡

Conn's syndrome (primary hyperaldosteronism)

Cause: excess aldosterone caused by an adenoma of the zona glomerulosa.

High aldosterone level causes:

- **hypernatremia** → hypertension.
- **Hypokalaemia** → muscular weakness, cardiac arrhythmias & metabolic alkalosis causing tetany and paraesthesia.



Actinomycosis

- Chronic, progressive granulomatous disease caused by filamentous gram positive anaerobic bacteria from the Actinomycetaceae family.
- Actinomyces are commensal bacteria that become pathogenic when a mucosal barrier is breached.
- The disease most commonly occurs in the head and neck, although it may also occur in the abdominal cavity and in the thorax.
- The mass will often enlarge across tissue planes with the **formation of multiple sinus tracts**.
- Abdominopelvic actinomycosis occurs most frequently in individuals that have had appendicitis (65%) cases.

Pathology :

- On histological examination gram positive organisms and **evidence of sulphur granules**.
- Sulphur granules are colonies of organisms that appear as round or oval basophilic masses.
- They are also seen in other conditions such as nocardiosis.

Treatment

- Long term antibiotic therapy usually with penicillin.
- Surgical resection is indicated for extensive necrotic tissue, non healing sinus tracts, abscesses or where biopsy is needed to exclude malignancy.



Bacterial Gastroenteritis

Campylobacter jejuni	<ul style="list-style-type: none"> - Most common cause of acute infective diarrhoea - Spiral, gram negative rods - Usually infects terminal ileum but spreads to involve colon and rectum. Local lymphadenopathy is common - May mimic appendicitis as it has marked right iliac fossa pain - Reactive arthritis is seen in 1-2% of cases
Shigella spp.	<ul style="list-style-type: none"> - Members of the enterobacteriaceae - Gram negative bacilli - Clinically causes dysentery - Shigella soneii is the commonest infective organism (mild illness) - Usually self limiting, ciprofloxacin may be required if individual is in a high risk group
Salmonella spp	<ul style="list-style-type: none"> - Facultatively anaerobic, gram negative, enterobacteriaceae - Infective dose varies according to subtype - Salmonellosis: usually transmitted by infected meat (especially poultry) and eggs
E. coli	<ul style="list-style-type: none"> - Enteropathogenic - Enteroinvasive: dysentery, large bowel necrosis/ulcers - Enterotoxigenic: small intestine, travelers diarrhoea - Enterohaemorrhagic : cause a haemorrhagic colitis, haemolytic uraemic syndrome in children and thrombotic thrombocytopenic purpura
Yersinia enterocolitica	<ul style="list-style-type: none"> - Gram negative, coccobacilli - Enterocolitis, acute mesenteric lymphadenitis or terminal ileitis - Differential diagnosis acute appendicitis - May progress to septicaemia in susceptible individuals - Usually sensitive to quinolone or tetracyclines
Vibrio cholera	<ul style="list-style-type: none"> - Short, gram negative rods - Transmitted by contaminated water, seafood - Symptoms include sudden onset of effortless vomiting and profuse watery diarrhoea - Correction of fluid and electrolyte losses are the mainstay of treatment - Most cases will resolve, antibiotics are not generally indicated

Clostridium Difficile

Clostridium difficile is a Gram positive rod often encountered in hospital practice. It produces an exotoxin which causes intestinal damage leading to a syndrome called **pseudomembranous colitis**.

Clostridium difficile develops when the normal gut flora are suppressed by broad-spectrum antibiotics. Clindamycin is historically associated with causing *Clostridium difficile* but the aetiology has evolved significantly over the past 10 years. Second and third generation cephalosporins are now the leading cause of *Clostridium difficile*.

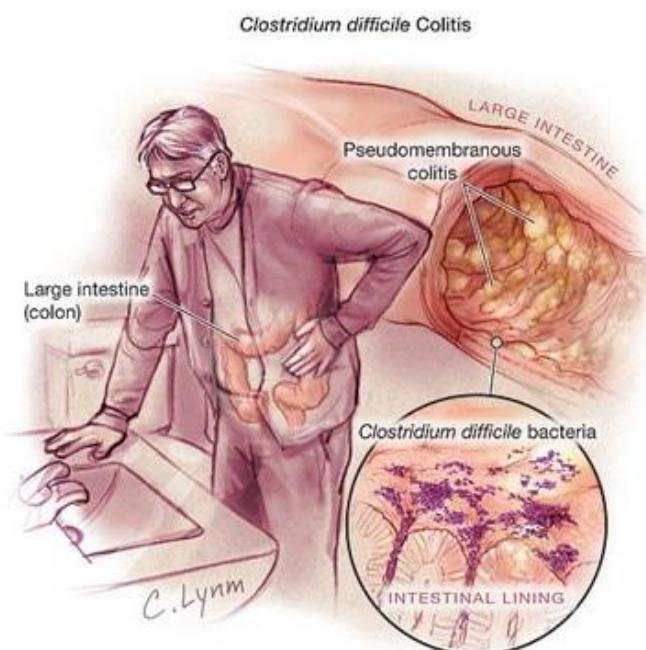
Features

- Diarrhoea
- Abdominal pain
- A raised white blood cell count is characteristic
- If severe, toxic megacolon may develop

Diagnosis is made by detecting *Clostridium difficile* toxin (CDT) in the stool

Management

- First-line therapy is oral metronidazole for 10-14 days
- If severe, or not responding to metronidazole, then oral vancomycin may be used
- For life-threatening infections a combination of oral vancomycin and intravenous metronidazole should be used



Epiphyseal fractures

Fractures involving the growth plate in children are classified using the Salter - Harris system. There are 5 main types.

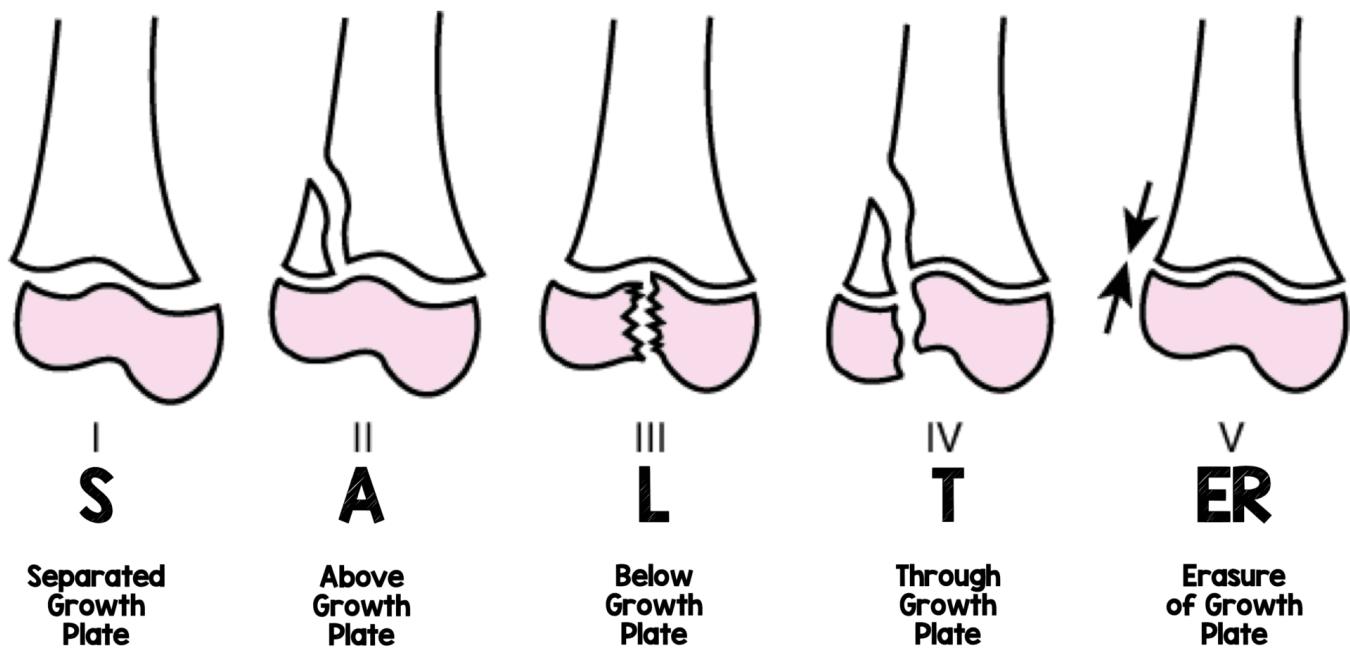
Salter Harris Classification

Type	Description
Type 1	Transverse fracture through the growth plate
Type 2	Fracture through the growth plate to the metaphysis (commonest type)
Type 3	Fracture through the growth plate and the epiphysis with metaphysis spared
Type 4	Fracture involving the growth plate, metaphysis and epiphysis
Type 5	Compression fracture of the growth plate (worst outcome)

Management

- Non displaced type 1 injuries can generally be managed conservatively.
- Unstable or more extensive injuries will usually require surgical reduction and/ or fixation, as proper alignment is crucial.

Salter-Harris Classification



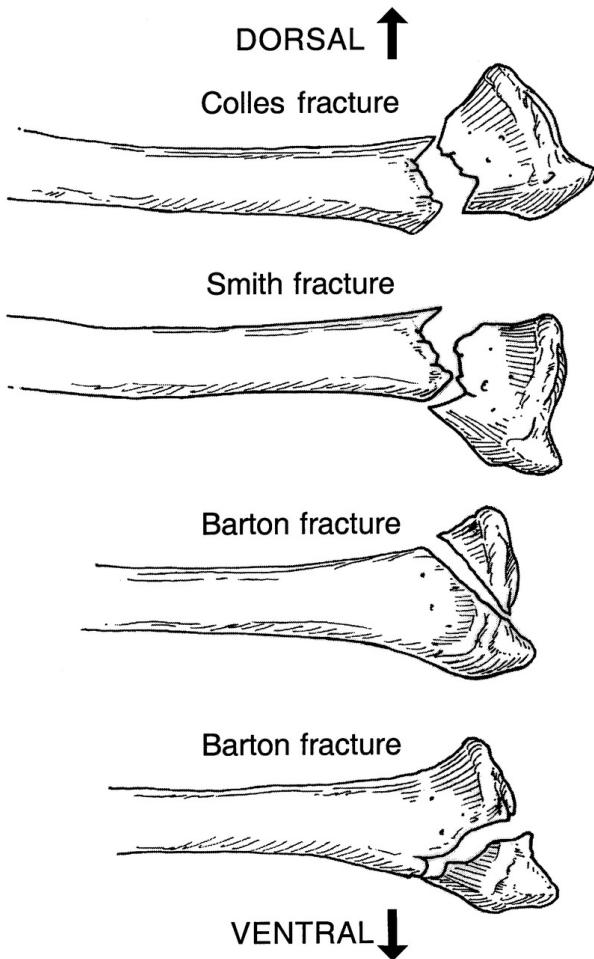
Eponymous fractures

Colles' fracture (dinner fork deformity)

- Fall onto extended outstretched hand
- Classical Colles' fractures have the following 3 features:
 1. Transverse fracture of the radius
 2. 1 inch proximal to the radio-carpal joint
 3. Dorsal displacement and angulation

Smith's fracture (reverse Colles' fracture)

- Volar angulation of distal radius fragment (Garden spade deformity)
- Caused by falling backwards onto the palm of an outstretched hand or falling with wrists flexed



Paediatric fractures

Paediatric fracture types

Type	Injury pattern
Complete fracture	Both sides of cortex are breached
Toddlers fracture	Oblique tibial fracture in infants
Greenstick fracture	Unilateral cortical breach only
Buckle fracture	Incomplete cortical disruption resulting in periosteal haematoma only

Growth plate fractures

In paediatric practice fractures may also involve the growth plate and these injuries are classified according to the Salter- Harris system (given below):

Type	Injury pattern
I	Fracture through the physis only (x-ray often normal)
II	Fracture through the physis and metaphysis
III	Fracture through the physis and epiphysis to include the joint
IV	Fracture involving the physis, metaphysis and epiphysis
V	Crush injury involving the physis (x-ray may resemble type I, and appear normal)

As a general rule it is safer to assume that growth plate tenderness is indicative of an underlying fracture even if the x-ray appears normal. Injuries of Types III, IV and V will usually require surgery. Type V injuries are often associated with disruption to growth.

Non accidental injury

- Delayed presentation
- Delay in attaining milestones
- Lack of concordance between proposed and actual mechanism of injury
- Multiple injuries
- Injuries at sites not commonly exposed to trauma
- Children on the at risk register

Pathological fractures

Genetic conditions, such as osteogenesis imperfecta, may cause pathological fractures.

Osteogenesis imperfecta

- Defective osteoid formation due to congenital inability to produce adequate intercellular substances like osteoid, collagen and dentine.
- Failure of maturation of collagen in all the connective tissues.
- Radiology may show translucent bones, multiple fractures, particularly of the long bones, wormian bones (irregular patches of ossification) and a trefoil pelvis.

Subtypes

- Type I The collagen is normal quality but insufficient quantity.
- Type II- Poor collagen quantity and quality.
- Type III- Collagen poorly formed. Normal quantity.
- Type IV- Sufficient collagen quantity but poor quality.

Osteopetrosis

- Osteoclastic problem
- Bones become harder and more dense.
- Autosomal recessive condition.
- It is commonest in young adults.
- Radiology reveals a lack of differentiation between the cortex and the medulla described as marble bone.

Paediatric orthopaedics

Diagnosis	Mode of presentation	Treatment	Radiology
Developmental dysplasia of the hip	Usually diagnosed in infancy by screening tests. May be bilateral, when disease is unilateral there may be leg length inequality. As disease progresses child may limp and then early onset arthritis. More common in extended breech babies. Neonate with abnormal skin crease in groin	Splints and harnesses or traction. In later years osteotomy and hip realignment procedures may be needed. In arthritis a joint replacement may be needed. However, this is best deferred if possible as it will almost certainly require revision	Initially no obvious change on plain films and USS gives best resolution until 3 months of age. On plain films Shentons line should form a smooth arc
Perthes Disease	Hip pain (may be referred to the knee) usually occurring between 5 and 12 years of age. Bilateral disease in 20%.	Remove pressure from joint to allow normal development. Physiotherapy. Usually self-limiting if diagnosed and treated promptly.	X-rays will show flattened femoral head. Eventually in untreated cases the femoral head will fragment.
Slipped upper femoral epiphysis	Typically seen in obese male adolescents. Pain is often referred to the knee. Limitation to internal rotation is usually seen. Knee pain is usually present 2 months prior to hip slipping. Bilateral in 20%.	Bed rest and non-weight bearing. Aim to avoid avascular necrosis. If severe slippage or risk of it occurring then percutaneous pinning of the hip may be required.	X-rays will show the femoral head displaced and falling inferolaterally (like a melting ice cream cone) The Southwick angle gives indication of disease severity
Osgood schlatter disease	presents with knee pain. It is worst during activity and settles with rest. On examination, there is tenderness overlying the tibial tuberosity and an associated swelling at this site	Cold compresses and conservative management	

Biliary atresia

- 1 in 17000 affected
- Biliary tree lumen is obliterated by an inflammatory cholangiopathy causing progressive liver damage

Clinical features

- Infant well in 1st few weeks of life
- No family history of liver disease
- **Jaundice in infants > 14 days in term infants (>21 days in pre term infants)**
- Pale stool, yellow urine (colourless in babies)
- Associated with cardiac malformations, polysplenia, situs inversus

Investigation

- Conjugated bilirubin (prolonged physiological jaundice or breast milk jaundice will cause a rise in unconjugated bilirubin, whereas those with obstructive liver disease will have a rise in conjugated bilirubin)
- Ultrasound of the liver (excludes extrahepatic causes, in biliary atresia infant may have tiny or invisible gallbladder)
- Hepato-iminodiacetic acid radionuclide scan (good uptake but no excretion usually seen)

Management

- Early recognition is important to prevent liver transplantation.
- Nutritional support.
- Roux-en-Y portojejunostomy (**Kasai procedure**).
- If Kasai procedure fails or late recognition, a liver transplant becomes the only option.

Bilious vomiting in neonates

Causes of intestinal obstruction with bilious vomiting in neonates

Disorder	Incidence and causation	Age at presentation	Diagnosis	Treatment
Duodenal atresia	1 in 5000 (<u>higher in Downs syndrome</u>)	Few hours after birth	AXR shows "double bubble sign, contrast study may confirm	Duodenoduodenostomy
Malrotation with volvulus	Usually cause by incomplete rotation during embryogenesis	Usually 3-7 days after birth, volvulus with compromised circulation may result in peritoneal signs and haemodynamic instability	<u>Upper GI contrast study</u> may show DJ flexure is more medially placed, USS may show abnormal orientation of SMA and SMV	<u>Ladd's procedure</u>
Jejunal/ ileal atresia	Usually caused by vascular insufficiency in utero, usually 1 in 3000	Usually within 24 hours of birth	AXR will show air-fluid levels	Laparotomy with primary resection and anastomosis
Meconium ileus	Occurs in between 15 and 20% of those babies with cystic fibrosis, otherwise 1 in 5000	Typically in first 24-48 hours of life with abdominal distension and bilious vomiting	sweat test to confirm cystic fibrosis	Surgical decompression, serosal damage may require segmental resection (enema with N acetyl cysteine)
Necrotising enterocolitis	<ul style="list-style-type: none"> ✓ Prematurity is the main risk factor ✓ Early features include abdominal distension and passage of bloody stools ✓ Increased risk when empirical antibiotics are given to infants beyond 5 days 	Usually second week of life	Dilated bowel loops on AXR, pneumatisis and portal venous air	Conservative and supportive for non perforated cases, laparotomy and resection in cases of perforation of ongoing clinical deterioration

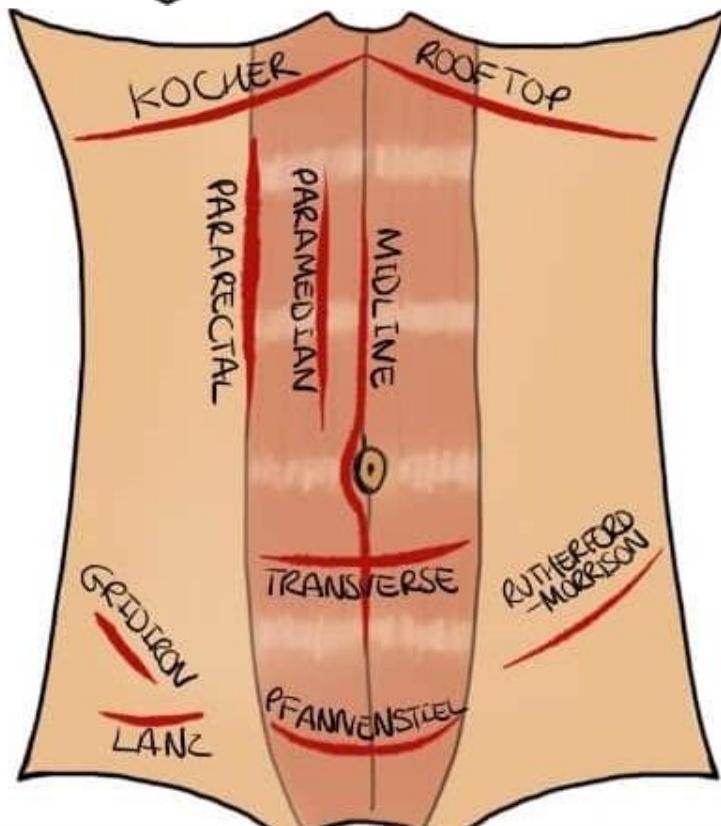
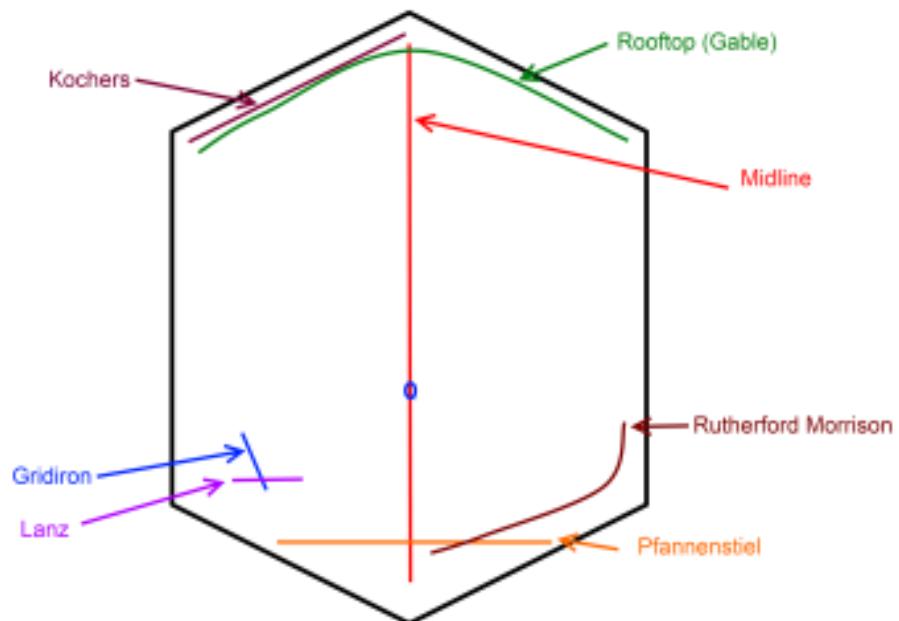
Paediatric Gastrointestinal disorders

Pyloric stenosis	M>F 5-10% Family history in parents Projectile non bile stained vomiting at 4-6 weeks of life Diagnosis is made by test feed or USS Treatment: Ramstedt pyloromyotomy (open or laparoscopic)
Acute appendicitis	Uncommon under 3 years When occurs may present atypically
Mesenteric adenitis	Central abdominal pain and URTI Conservative management
Intussusception	Telescoping bowel Proximal to or at the level of, ileocaecal valve 6-9 months age Colicky pain, diarrhoea and vomiting, sausage shaped mass, red jelly stool. Treatment: reduction with air insufflation
Malrotation	High caecum at the midline Feature in exomphalos, congenital diaphragmatic hernia, intrinsic duodenal atresia May be complicated by development of volvulus, infant with volvulus may have bile stained vomiting Diagnosis is made by upper GI contrast study and USS Treatment is by laparotomy, if volvulus is present (or at high risk of occurring then a ladds procedure is performed
Hirschsprung's disease	Absence of ganglion cells from myenteric and submucosal plexuses Occurs in 1/5000 births Full thickness rectal biopsy for diagnosis Delayed passage of meconium and abdominal distension Treatment is with rectal washouts initially, thereafter an anorectal pull through procedure
Oesophageal atresia	Associated with tracheo-oesophageal fistula and polyhydramnios May present with choking and cyanotic spells following aspiration VACTERL associations
Meconium ileus	Usually delayed passage of meconium and abdominal distension Majority have cystic fibrosis X-Rays may not show a fluid level as the meconium is viscid (depends upon feeding), PR contrast studies may dislodge meconium plugs and be therapeutic Infants who do not respond to PR contrast and NG N-acetyl cysteine will require surgery to remove the plugs
Biliary atresia	Jaundice > 14 days Increased conjugated bilirubin Urgent Kasai procedure

Abdominal incisions

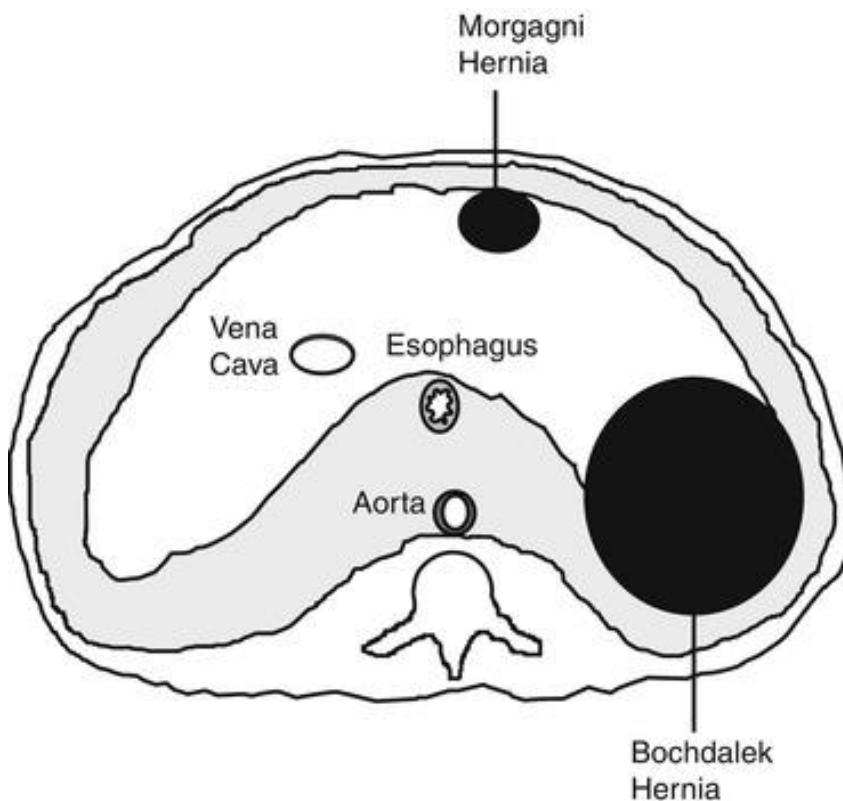
Midline incision	<ul style="list-style-type: none">• Commonest approach to the abdomen• Structures divided: linea alba, transversalis fascia, extraperitoneal fat, peritoneum (avoid falciform ligament above the umbilicus)• Bladder can be accessed via an extraperitoneal approach through the space of Retzius
Paramedian incision	<ul style="list-style-type: none">• Parallel to the midline (about 3-4cm)• Structures divided/retracted: anterior rectus sheath, rectus (retracted), posterior rectus sheath, transversalis fascia, extraperitoneal fat, peritoneum• Incision is closed in layers
Battle	<ul style="list-style-type: none">• Similar location to paramedian but rectus displaced medially (and thus denervated)• Now seldom used
Kocher's	Incision under right subcostal margin e.g. Cholecystectomy (open)
Lanz	Incision in right iliac fossa e.g. Appendicectomy
Gridiron	Oblique incision centered over McBurneys point- usually appendicectomy (less cosmetically acceptable than Lanz)
Gable	Rooftop incision
Pfannenstiel's	Transverse supra pubic, primarily used to access pelvic organs
McEvedy's	Groin incision e.g. Emergency repair strangulated femoral hernia
Rutherford Morrison	Extraperitoneal approach to left or right lower quadrants. Gives excellent access to iliac vessels and is the approach of choice for first time renal transplantation.

Abdominal incisions



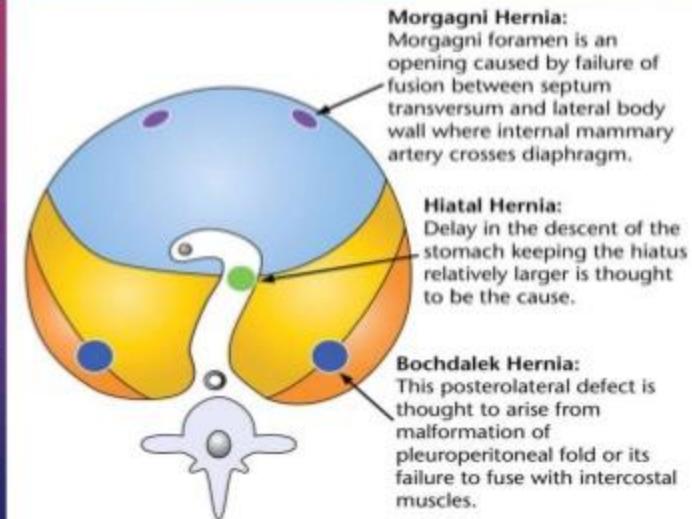
Hernia

Inguinal Hernia	<ul style="list-style-type: none">➤ Indirect: Leaves Abd. Cavity lat. to Inf. Epigastric Vessel → Travels entire canal → Enters scrotum.➤ Direct: Leaves Abd. Cavity med. to Inf. Epigastric Vessel (inside hasselbach triangle) → Don't traverse entire canal & rarely enters scrotum.
Epigastric or Paraumbilical	<ul style="list-style-type: none">➤ Linea Alba➤ Multibarity & obesity are risk factors
Obturator Hernia	<ul style="list-style-type: none">➤ Through the Obturator canal➤ Hernia passes between Pectineus & Adductor Longus➤ Causes pain along medial side of thigh due to affection of obturator nerve
Litter's Hernia	<ul style="list-style-type: none">➤ Meckel's Diverticulum as sac content➤ Strangulation of Meckel's
Richter's Hernia	<ul style="list-style-type: none">➤ Portion of circumference of Intestine as sac content➤ Part of bowel @ antemesenteric margin strangulated
Maydel's Hernia	<ul style="list-style-type: none">➤ "W" loops of small bowel➤ 2 separate loops of bowel as sac content
Morgagni Hernia	<ul style="list-style-type: none">➤ Transverse colon enters Thoracic cavity – diaphragmatic hernia
Bochdalek Hernia	<ul style="list-style-type: none">➤ If Morgagni hernia causes displaced apex beat; respiratory problem; scaphoid abdomen➤ Associated with complication
Amyand's hernia	<ul style="list-style-type: none">➤ Hernia containing appendix



CONGENITAL DIAPHRAGMATIC HERNIA

- Diaphragmatic hernias include Bochdalek (posterolateral), Morgagni (retrosternal), and hiatal hernias
- Antenatal USG scan can diagnose all types earlier
- USG can aid in determining the survivability of the foetus.
- Congenital diaphragmatic hernia (CDH) is a major surgical emergency in newborns. The key to survival lies in prompt diagnosis and treatment
- Pulmonary hypertension and Pulmonary hypoplasia are complications



Inguinal hernia surgery

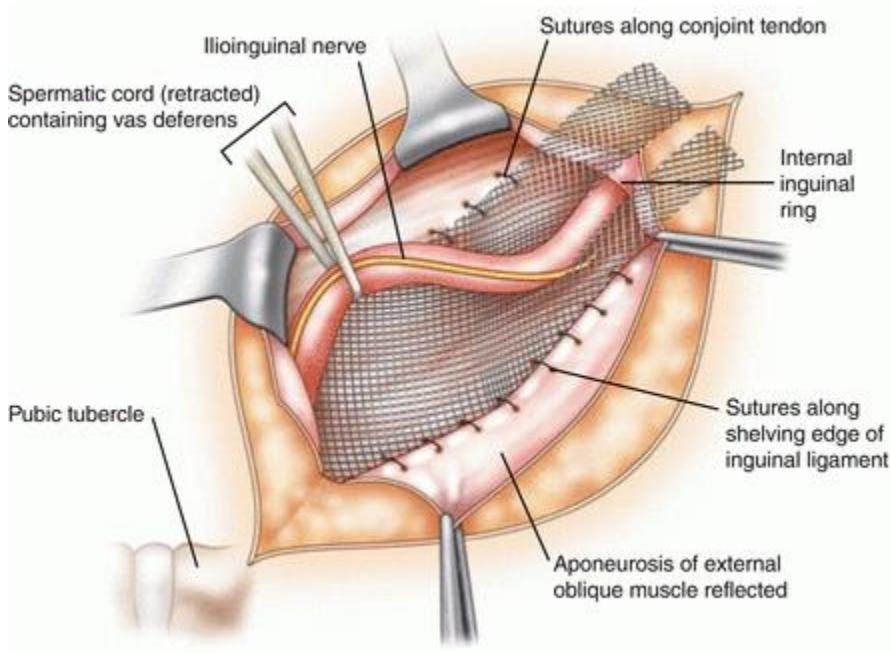
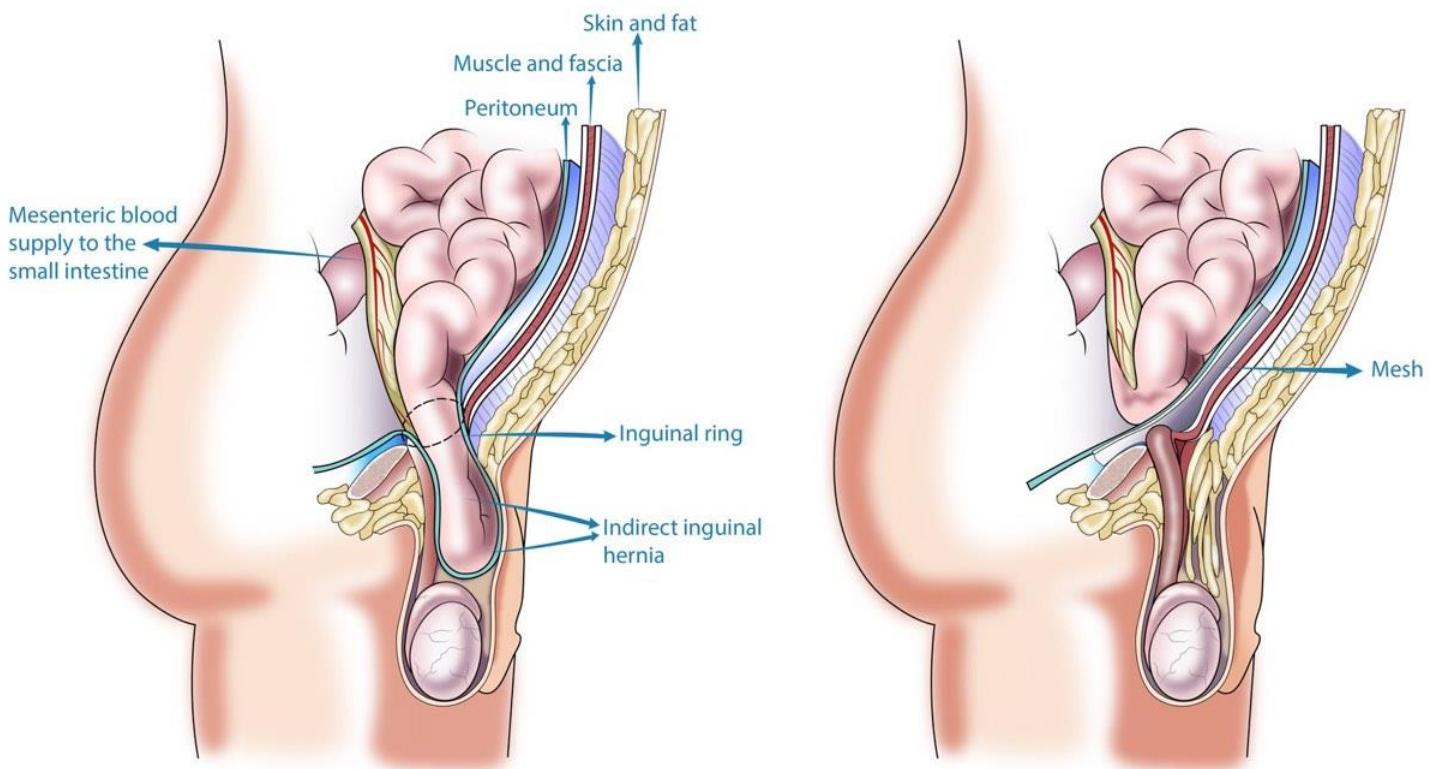
Diagnosis

- Most cases are diagnosed clinically, a reducible swelling may be located at the level of the inguinal canal. Large hernia's may extend down into the male scrotum, these will not trans-illuminate and it is not possible to "get above" the swelling.
- Cases that are unclear on examination, but suspected from the history, may be further investigated using ultrasound or by performing a herniogram.

Treatment

Hernias associated with few symptoms may be managed conservatively. Symptomatic hernias or those which are at risk of developing complications are usually treated surgically.

- **First time hernias** may be treated by performing an open inguinal hernia repair; the inguinal canal is opened, the hernia reduced and the defect repaired. A prosthetic mesh may be placed posterior to the cord structures to re-enforce the repair and reduce the risk of recurrence.
- **Recurrent hernias** and **bilateral hernias** are generally managed with a laparoscopic approach. This may be via an intra or extra peritoneal route. As in open surgery a mesh is deployed. However, it will typically lie posterior to the deep ring.
- **Inguinal hernia in children**
Inguinal hernias in children are **almost always of an indirect type** and therefore are usually dealt with by herniotomy, rather than herniorraphy. Neonatal hernias especially in those children born prematurely are at highest risk of strangulation and should be repaired urgently. Other hernias may be repaired on an elective basis.



Abdominal compartment syndrome

Background

Intra-abdominal pressure is the steady state pressure concealed within the abdominal cavity.

- In critically ill adults the normal intra abdominal pressure = 5-7 mmHg
- Intra abdominal hypertension has pressures of 12-25mmHg
- Changes >15mmHg are associated with microvascular hypoperfusion
- Abdominal compartment syndrome is defined as **sustained intra abdominal pressure >20mmHg coupled with new organ dysfunction / failure**
- It may occur either primarily without previous surgical intervention e.g. Following intestinal ischaemia or secondarily following a surgical procedure
- Diagnosis is typically made by transvesical pressure measurements coupled with an index of clinical suspicion.

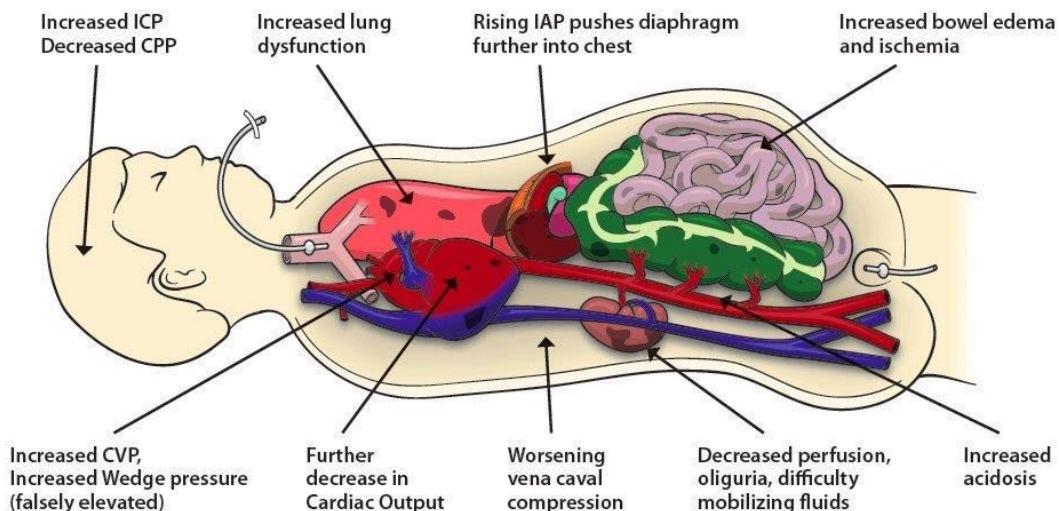
Management

Once the diagnosis is made non operative measures should be instituted including:

- Gastric decompression
- Improve abdominal wall compliance e.g. muscle relaxants/ sedation
- Drain abdominal fluid collections.
- Consider fluid restriction/ diuretics if clinically indicated

In those whom non operative treatment is failing; the correct treatment is laparotomy and laparostomy. Options for laparostomy are many although the Bogota bag or VAC techniques are the most widely practised. Re-look laparotomy and attempts at delayed closure will follow in due course.

Occult Organ Ischemia IAP 16 – 20 mmHg



Abdominal radiology

Plain abdominal x-rays are often used as a first line investigation in patients with acute abdominal pain. A plain abdominal film may demonstrate free air, evidence of bowel obstruction and possibly other causes of pain (e.g. renal or gallbladder stones).

Investigation of potential visceral perforation is usually best performed by obtaining an erect chest x-ray, as this is a more sensitive investigation for suspected visceral perforation than recumbent films.

Features which are usually abnormal

- Large amounts of free air (colonic perforation), smaller volumes seen with more proximal perforations.
- A positive Riglers sign (gas on both sides of the bowel wall).
- **Caecal diameter of >8cm**
- **Fluid levels in the colon**
- Ground glass appearance to film (usually due to large amounts of free fluid).
- Sentinel loop in patients with inflammation of other organs (e.g. pancreatitis).

Features which should be expected/ or occur without pathology

- Following ERCP (and sphincterotomy) air may be identified in the biliary tree.
- Free intra abdominal air following laparoscopy / laparotomy, although usually dissipates after 48-72 hours.

Abdominal signs

A number of eponymous abdominal signs are noted. These include:

- **Rovsing's sign** → appendicitis
- **Boas sign (hyperesthesia below right scapula)** → cholecystitis
- **Murphys sign (fingers in Rt. Hypochondrium & ask patient to take deep breath)** → cholecystitis
- **Cullens sign** → pancreatitis (other intraabdominal haemorrhage)
- **Grey-Turners sign** → pancreatitis (or other retroperitoneal haemorrhage)

In clinical practice haemorrhagic pancreatitis is thankfully rare. The signs are important and thus shown below:

Cullen's sign

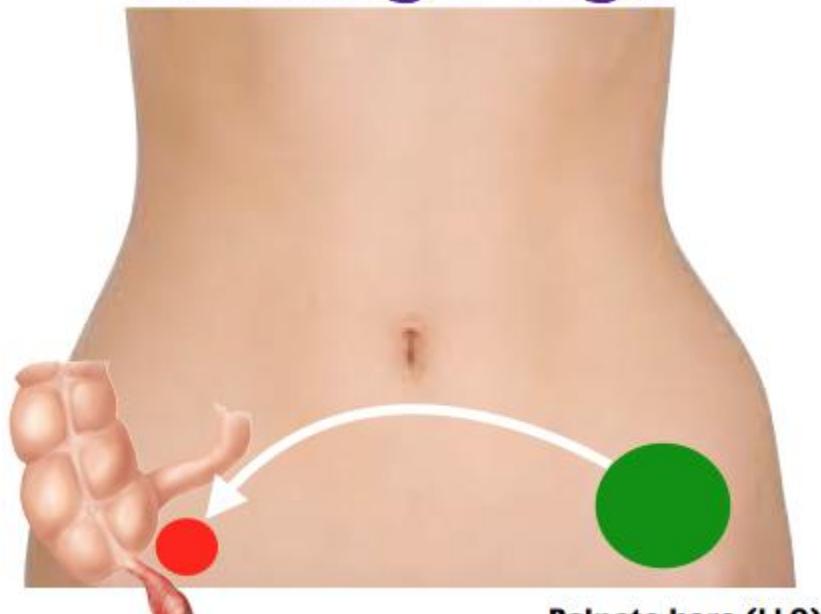


Grey Turner's sign



Rovsing's sign

Rovsing's Sign



Palpate here (LLQ)

Pain elicited in RLQ

Suggestive of acute appendicitis

Acute abdominal pain

Acute abdominal pain is a common cause of admission to hospital. The relative proportions of conditions presenting with abdominal pain is given below:

- **Non specific abdominal pain (35%)**
- Appendicitis (17%)
- Intestinal obstruction (15%)
- Urological disease (6%)
- Gallstone disease (5%)
- Colonic diverticular disease (4%)
- Abdominal trauma (3%)
- Perforated peptic ulcer (3%)
- Pancreatitis (2%)

Non specific abdominal pain should really be a diagnosis of exclusion and if care is taken in excluding organic disease the proportion of cases labeled such should decline. It should also be appreciated that a proportion of patients may have an underlying medical cause for their symptoms such as pneumonia or diabetic ketoacidosis.

Key points in management

- Early administration of adequate analgesia (including opiates).
- Abdominal ultrasound is safe, non invasive and cheap and yields significantly more information than plain radiology. However, plain radiology is still the main test for suspected perforated viscus, especially out of hours.
- In up to 50% cases with perforated peptic ulcer, the plain x-rays may show no evidence of free air. If clinical signs suggest otherwise, then a CT scan may be a more accurate investigation, if plain films are normal.
- Plain film radiology usually cannot detect <1mm free air, and is 33% sensitive for detection of 1-13mm pockets of free air.
- Think of strangulated intestine when there is fever, raised white cell count, tachycardia and peritonism.
- In suspected large bowel obstruction a key investigation is either a water soluble contrast enema or CT scan.
- Where need for surgery is difficult to define and imaging is inconclusive the use of laparoscopy as a definitive diagnostic test is both safe and sensible.

Gynaecological causes of abdominal pain

Differential diagnoses of abdominal pain in females

Diagnosis	Features	Investigation	Treatment
Mittelschmerz	Usually <u>mid cycle pain</u> . Often sharp onset. Little systemic disturbance. May have recurrent episodes. Usually settles over 24-48 hours.	Full blood count- usually normal Ultrasound- may show small quantity of free fluid	Conservative
Endometriosis	25% asymptomatic, in a further 25% associated with other pelvic organ pathology. Remaining 50% may have menstrual irregularity, infertility, pain and deep dyspareunia. Complex disease may result in pelvic adhesional formation with episodes of intermittent small bowel obstruction. Intra-abdominal bleeding may produce localised peritoneal inflammation. Recurrent episodes are common.	Ultrasound- may show free fluid Laparoscopy will usually show lesions	Usually managed medically, complex disease will often require surgery and some patients will even require formal colonic and rectal resections if these areas are involved
Ovarian torsion	Usually sudden onset of deep seated colicky abdominal pain. Associated with vomiting and distress. Vaginal examination may reveal adnexial tenderness.	US may show free fluid Laparoscopy is usually both diagnostic and therapeutic	Laparoscopy
Ectopic gestation	Symptoms of pregnancy without evidence of intra uterine gestation. Present as an emergency with evidence of rupture or impending rupture. Open tubular ruptures may have sudden onset of abdominal pain and circulatory collapse, in other the symptoms may be more prolonged and less marked. Small amount of vaginal discharge is common. There is usually adnexial tenderness.	Ultrasound showing no intra uterine pregnancy and beta HCG that is elevated May show intra abdominal free fluid	Laparoscopy or laparotomy is haemodynamically unstable. A salphingectomy is usually performed.
Pelvic inflammatory disease	<u>Bilateral lower abdominal pain associated with vaginal discharge</u> . Dysuria may also be present. Peri-hepatic inflammation secondary to Chlamydia (<u>Fitz Hugh Curtis Syndrome</u>) may produce right upper quadrant discomfort. Fever >38°	Full blood count- Leucocytosis Pregnancy test negative (Although infection and pregnancy may co-exist) Amylase - usually normal or slightly raised High vaginal and urethral swabs	Usually medical management