The Orthopedic Theory Examination

First Edition



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Paper 1

Applied Anatomy

Anatomy and physiology of the bone

Histology of Bone

A bone is made up of lamellae.Each lacuna contains one osteocyte. Spreading out from each lacuna are fine canals or canaliculi that communicate with those from other lacunae (Fig.1) The canaliculi are occupied by delicate cytoplasmic processes of osteocytes.

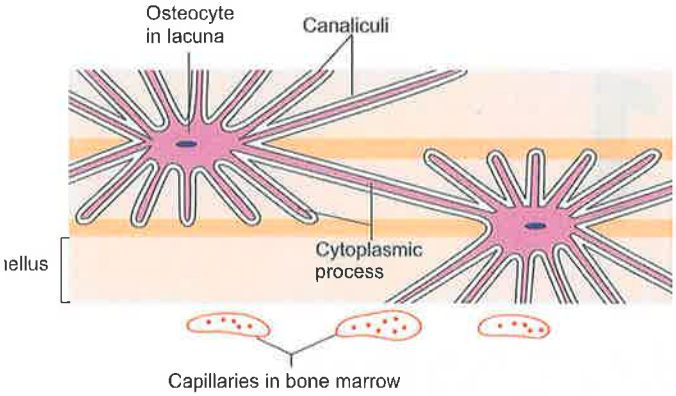


Fig 1: Relationship of osteocyte and bone lamellae

Histologically a bone can be categorized as compact and spongy.

Compact bone: Compact bone is made upof concentric lamellae, and ispervaded by lacunae (containingosteocytes), and by canaliculi(Fig 2). Most of the lamellaeare arranged in the form ofconcentric rings that surrounda narrow Haversian canalpresent at the centre of each ring.The Haversian canal is occupiedby blood vessels, nerve,fibers,and some cells. One Haversiancanal and the lamellae around, constitutes a Haversian systemor osteon.

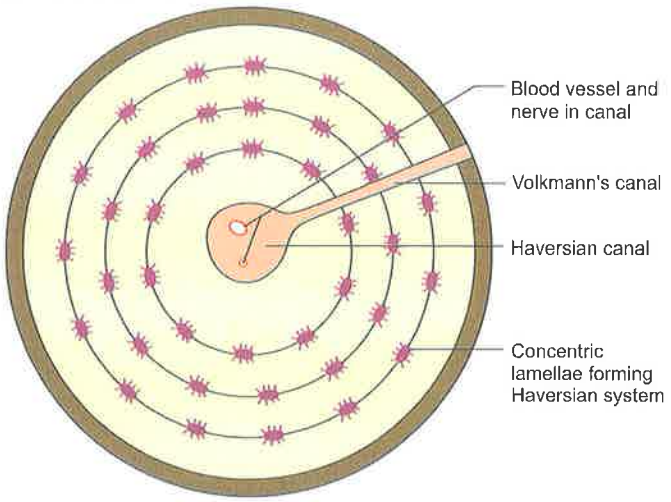


Fig 2: Haversian system in compact bone

Cancellous bone: Cancellous bone is made up of a meshwork of bony plates or rods called trabeculae. Each trabeculus is made up of a number of lamellae between which there are lacunae containing osteocytes. Canaliculi, containing the processes of osteocytes, radiate from the lacunae (Fig 3).The trabeculae enclose wide spaces that are filled in by bone marrow. They receive nutrition from blood vessels in the bone marrow. The trabeculae are covered externally by vascular endosteum containing osteoblasts, osteoclasts and osteoprogenitor cells.

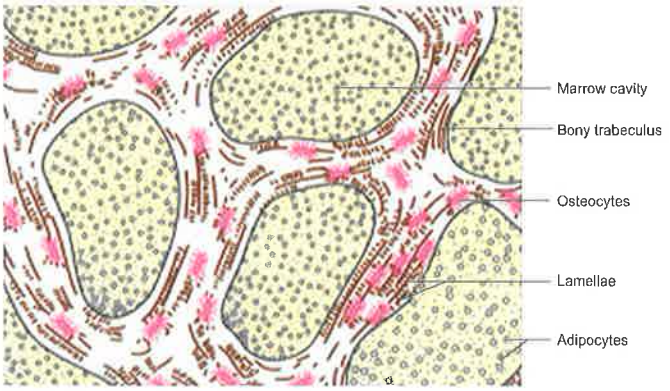


Fig 3: Structure of cancellous bone

Factors regulating bone metabolism are mentioned below:

- **Calcium:** Calcium is essential for normal cell function and physiological processes such as blood coagulation, nerve conduction and muscle contraction. An uncompensated fall in extracellular calcium concentration (hypocalcemia) may cause tetany; an excessive rise (hypercalcemia) can lead to depressed neuromuscular transmission. If the ionizedplasma calcium concentration falls, PTH is released and causes
  (a) Increased renal tubular reabsorption of calcium and reduced renal tubular reabsorption of phosphate and
  (b) A switch to increased 1,25-(OH)2 vitamin D production and enhanced intestinal calcium absorption.

  If the calcium concentration remains low, calcium is drawn from the skeleton by increased bone resorption through the influence of PTH.
- **Phosphorus:** Apart from its role (with calcium) in the composition of hydroxyapatite crystals in bone, phosphorus is needed for many important metabolic processes, including energy transport and intracellular cell signaling.The solubility product of calcium and phosphate is held at a fairly constant level; any increase in the one will cause the other to fall. Pi is regulated by the phosphaturic hormone FGF23. The other main regulators of plasma phosphate concentration are PTH and 1,25-(OH)2D. If Pi rises abnormally, a reciprocal fall in calcium concentration will stimulate PTH secretion which in turn will suppress urinary tubular reabsorption of Pi, resulting in increased Pi excretion and a fall in plasma Pi. High Pi levels also result in diminished 1,25-(OH)2D production, causing reduced intestinal absorption of phosphorus.
- **Magnesium:** Magnesium is necessary for the efficient secretion and peripheral action of parathyroid hormone. Thus, if hypocalcemia is accompanied by hypomagnesaemia, it cannot be fully corrected until normal magnesium concentration is restored.
- **Vitamin D & PTH** actions are summarized in table.

	Source	Secretion increased by	Secretion decreased by	Effects on intestine	Effects on kidney	Effects on bone	Effects on serum ca and Pi
PTH	Parathyroid gland	Fallin serum Ca	Rise in serum Ca Increase in 1,25-(OH)2D	No direct effect but Ca absorption increased through 1,25-(OH)2D	Increase in 1,25(OH)2D Increased resorption of Ca. Increased excretion of phosphate	No direct effect but resorption increased via action on 1,25-(OH)2D	Rise in serum Ca Fall in serum Pi
1,25(OH)2 vit D	Kidney tubule	Fall in serum Ca Fall in serum Pi Rise in serum PTH	Rise in serum Ca Rise in serum Pi Fall in PTH	Increased absorption of Ca Increased absorption of phosphate		Osteoclastogenesis and increased bone resorption	Rise in serum Ca and Pi

- **Estrogen:** acts to suppress longitudinal growth and periosteal expansion, and its rise around puberty contributes to growth plate closure.Estrogen deficiency in cases of primary amenorrhea results in reduction of bone mineral density, particularly at trabecular-rich sites such as the spine.Estrogen deficiency at the menopause leads to an increase in bone turnover, a decline in bone mineral density, a deterioration in bone architecture, and a consequential increase in fracture risk.
- **Androgen:**stimulate bone growth, and act tomaintain bone mass, through a combination of suppression of bone resorption and stimulation of bone formation.
- **Glucocorticoid:** increase in glucocorticoids, either in Cushing's or when used as a therapy has effects on bone metabolism characterized by suppressed bone formation, leading to adecrease in BMD and increased risk of osteoporosis.
- **Thyroxine:** thyrotoxicosis or increase in thyroxine is associated with increased bone turnover and is a recognized risk factor for low bone mineral density and osteoporosis.
- **Growth Hormone:** plays role in skeletal growth and also influences bone remodeling.
- **Calcitonin:** Calcitonin is produced by C cells of the thyroid, which reduces the osteoclast activity, and was used as an anti-resorptive drug treatment forosteoporosis.

Electric phenomenon and electrical changes in bone:

Living bones have small electrical potentials on their surfaces, the magnitudes of which change only slightly with the reaching of adulthood. When using a limb, stresses are put on the bones inside, and these create piezo-electric potentials (electric potentials generated in response to mechanical stress,Fig 4)which may themselves cause bone growth along the lines of stress, hence making the bone stronger. Voltages also appear at fracture sites and may be important in causing the bone to heal its wound. Fractures that have not healed properly can be stimulated into repair by the passage of small electric currents through them.

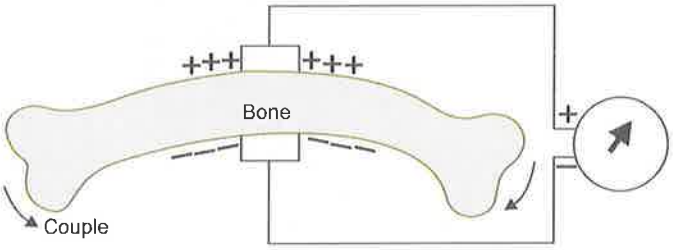


Fig 4: potentials developed are negative in the area of compression and positive in the area of tension.

Mechanical bone stress and osteoporosis - bone currents can affect bone collagen itself, especially the binding between collagen and hydroxyapatite. The currents generated during stress (during walking) promote stability of these bonds. Absence of these currents can probably cause the bindings (ionic: apatite - collagen) to weaken or be broken. The effect is loosening and weakening of the bone structure. As seen in astronauts.

Cartilage Anatomy

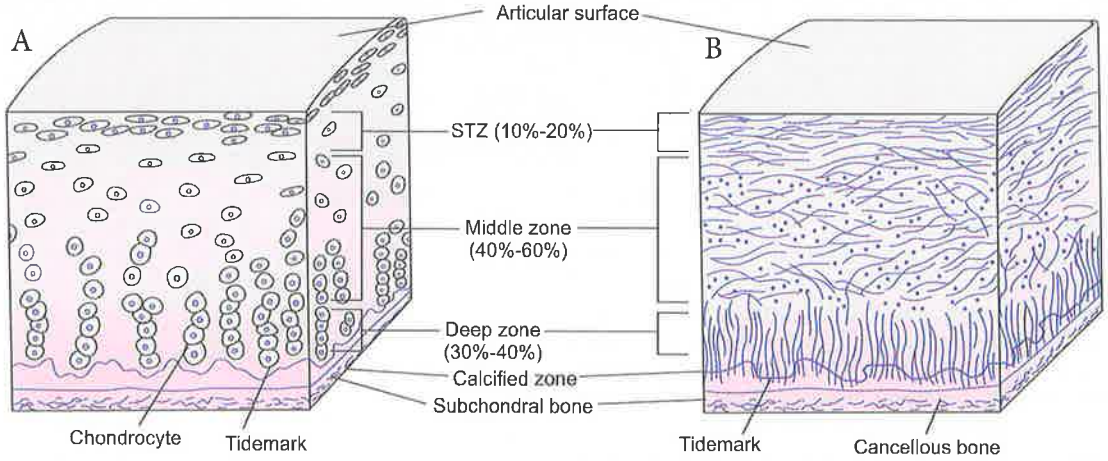
Normal structure

- Articular cartilage consists primarily of extracellularmatrix with a sparse population of cells
- Its made of hyaline cartilage of 2-4mm thick
- It does not have its own bloodvessels, lymphatic vessels and nerves
- It has a lowlevel of metabolic activity,
- There is little or no celldivision or cell death in normal adult articular cartilageexpect it has chondrocytes, making uponly about 1% of volume of adult human articular cartilage
- The articular cartilage needs to be elastic and have high tensilestrength to functional normally.
- The unique mechanical properties of articularcartilage depend on the extracellular matrix
- The extracellular matrix consists of two components(help

to retain water within the matrix)
  - Tissuefluid
  - The framework of structural macromolecules consisting of type II collagen fibers, proteoglycans andnon-collagenousproteins and glycoproteins,
- Tissue fluid and structural macromolecules are producedin the appropriateamounts and assembled and organizedinto a highly orderedmolecular framework by the chondrocytes
- The collagen matrix gives cartilage itsform and tensile strength.
- Proteoglycans and noncollagenousproteins bind to the collagenous network, helpstabilize the matrix macromolecular framework and helpchondrocytes bind to the macromolecules of the network

Histology of the cartilage

- Various zones of articular cartilage
  - The superficial zone, the middle zone, the deep zone, and the calcified zone
- Within each zone, 3 regions can be identified
  - The pericellular region, the territorial region, and the interterritorial region.
- **Zones of cartilage**



- Superficial zone
  - The thin superficial (tangential) zone protects deeper layers from shear stresses
  - 10% to 20% of articular cartilage thickness.
  - The collagen fibers of this zone (primarily, type II and IX collagen) are packed tightly and aligned parallel to the articular surface
  - It contains a relatively high number of flattened chondrocytes, and the integrity of this layer is imperative in the protection and maintenance of deeper layers.
  - This zone is in contact with synovial fluid and is responsible for most of the tensile properties of cartilage, which enable it to resist the sheer, tensile, and compressive forces imposed by articulation.
- Middle (transitional) zone
  - Immediately deep to the superficial zone
  - It provides an anatomic and functional bridge between the superficial and deep zones.
  - It represents 40% to 60% of the total cartilage volume,
  - It contains proteoglycans and thicker collagen fibrils.
  - In this layer, the collagen is organized obliquely, and the chondrocytes are spherical and at low density.
  - Functionally, the middle zone is the first line of resistance to compressive forces
- Deep zone
  - Is responsible for providing the greatest resistance to compressive forces, given that collagen fibrils are arranged perpendicular to the articular surface.
  - The deep zone represents approximately 30% of articular cartilage volume.
  - It contains the largest diameter collagen fibrils in a radial disposition, the highest proteoglycan content, and the lowest water concentration.
  - The chondrocytes are typically arranged in columnar orientation, parallel to the collagen fibers and perpendicular to the joint line.
- The tide mark
  - It distinguishes the deep zone from the calcified cartilage.
  - The collagen fibrils are arranged perpendicular to the articular cartilage.
  - The calcified layer plays an integral role in securing the cartilage to bone, by anchoring the collagen fibrils of the deep zone to subchondral bone.
  - In this zone, the cell population is scarce and chondrocytes are hypertrophic.
- Regions
- Pericellular matrix
  - This is a thin layer adjacent to the cell membrane, and it completely surrounds the chondrocyte.
  - It contains mainly proteoglycans, as well as glycoproteins and other noncollagenous proteins.
  - This matrix region may play a functional role to initiate signal transduction within cartilage with load bearing.
- Territorial matrix
  - Surrounds the pericellular matrix;

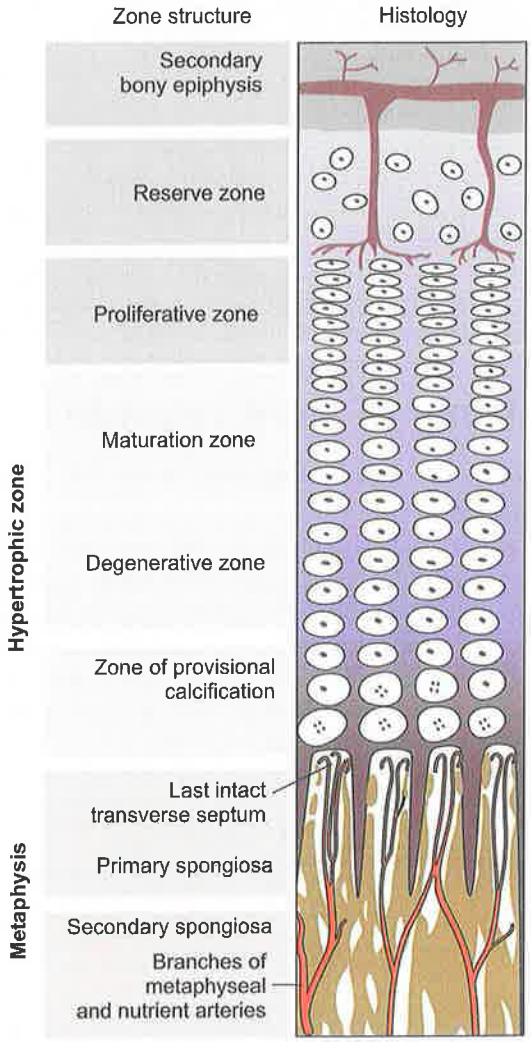
- It is composed mostly of fine collagen fibrils, forming a basketlike network around the cells.
- This region is thicker than the pericellular matrix, and it has been proposed that the territorial matrix may protect the cartilage cells against mechanical stresses and may contribute to the resiliency of the articular cartilage structure and its ability to withstand substantial loads

- Interterritorial region
  - It is the largest of the 3 matrix regions;
  - It contributes most to the biomechanical properties of articular cartilage.
  - This region is characterized by the randomly oriented bundles of large collagen fibrils, arranged parallel to the surface of the superficial zone, obliquely in the middle zone, and perpendicular to the joint surface in the deep zone.
  - Proteoglycans are abundant in the interterritorial zone

Physis anatomy

- Two types of growth plates exist in pediatric long bones
  - Horizontal growth plate (the true physis)
    - Well organized into layers
    - 3 zones
      - Reserve zone
      - Proliferative zone
      - Hypertrophy zone
        - Zone of maturation
        - Zone of degeneration
        - Zone of provisional calcification
  - Spherical growth plate
    - For growth of epiphysis/apophysis
    - Less organized
- Perichondrial artery → major source of nutrition to the growth plate

Physis zones	Functions	Disorders
Reserve zone	Stores glycogen and lipids for growth Production of organic matrix	Lysosomal storage disorders Diastrophic dwarfism
Proliferative zone	Responsible for longitudinal growth Production of organic matrix	Gigantism Achondroplasia
Hypertrophy zone (Zone of maturation and degeneration)	Chondrocytes increase in size Chondrocytes mature Preparation of matrix for calcification	SCFE Mucopolysaccharidosis
Hypertrophy zone (Zone of provisional calcification)	Calcification of matrix	Rickets



Management of physeal bar

- Imaging
  - Map bar to determine location, extent
    - Xrays
    - CT scans
    - MRI
  - To assess deformity
    - Scanogram
- Types
  I. Peripheral→angular deformity
  II. Central, tented physis→shortening
  III. Combined/complete → shortening
- Treatment guidelines
  - Assess
    - Remaining growth
    - Amount of physis involved
    - Degree of angular deformity
    - Expected LLD at maturity
  - Address
    - Angular deformity
    - Limb length discrepancy

- Physeal Bar Resection
  - Indications
    - >2 years remaining growth
    - <50% physeal involvement (cross- sectional)
    - Concomitant osteotomy for >15-20° deformity
    - Completion epiphysiodesis and contralateral epiphysiodesis may be more reliable in older child
  - Techniques
    - Direct visualization on the physis (± fluoroscopy) → curettage/burr → Interpositional fat graftto prevent reformation → Wire markers can be used to assess future growth

Anatomy of Joint capsule

Anatomy of Joint Capsule and Its Surgical Importance

The joint capsule resembles a sac-like envelope that forms a sleeve around the synovial joint and encloses its cavity. The joint capsule is a dense fibrous connective tissue that is attached to the bones via specialized attachment zones at the end of each involved bone. There are two connective tissue layers in the joint capsule. The outer fibrous layer is made up of collagen fibers. The capsule is thickened at places forming intrinsic ligaments while there are discrete ligaments outside the capsule(extrinsic ligaments). This layer surrounds the joint and attaches to the periosteum of the adjacent bones. The synovial membrane is its inner layer and is poorly innervated and highly vascularized. It seals the joint space, provides passive stability by limiting movements, provides active stability via its proprioceptive nerve endings, and may form articular surfaces for the joint. It varies in thickness according to the stresses to which it is subject, is locally thickened to form capsular ligaments, and may also incorporate tendons.

Surgical importance:

- Septic arthritis (capsulotomy)
- Deformity correction – eg. In a case of flexion deformity of knee, posterior capsules are released.
- Instability – eg. In a condition of shoulder instability, capsullorhaphy is performed.
- Iatrogenic – eg. During elective case of hip surgeries, capsule tear may lead to subluxation, hence capsules are commonly repaired during surgery.

Synovial Fluid Analysis In Various Disorders

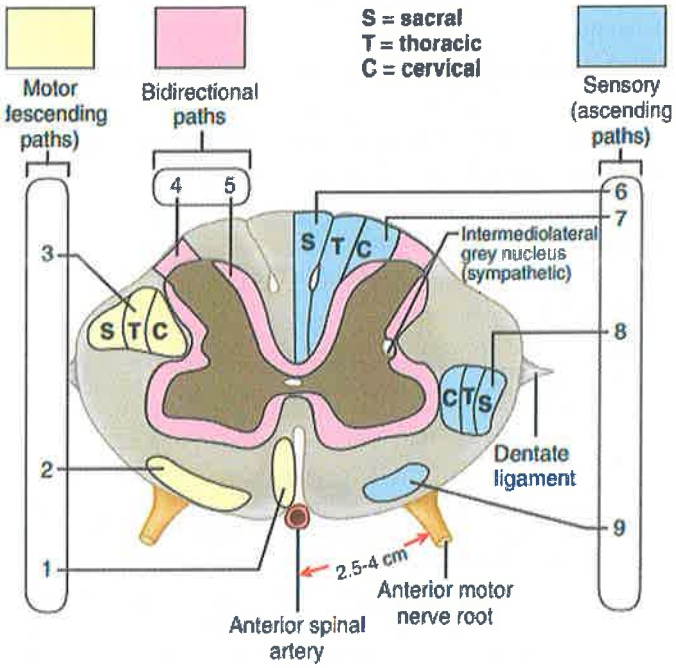
Synovial fluid analysis can differentiate between non-inflammatory, inflammatory and traumatic conditions. Systematic examination includes macroscopic and microscopic, and biochemical analysis.

Gross	Normal fluid composition	Noninflammatory (liketraumatic, reactive,degenerative)	Septic arthritis	Inflammatory arthropathies (like rheumatoid)	Crystal arthritis
Appearance	Clear, oil like viscous fluid	Reactive and degenerative effusion—clear,blood stained—posttraumatic	Turbid	Cloudy	Usually, cloudy
Viscosity	High	High	Inconsistent	Low	Inconsistent
Color	Straw	Yellow to red dependingon hemorrhage	Cloudy to yellow	Cloudy yellow	Cloudy yellow
Wbcs per mm3	<100, usually monocytes	Up to 2,000, usually monocytes	>80,000, usually poly	2,000–50,000 (poly and mono)	2,000–75,000 (poly and mono)
Neutrophil	<25%	<25%	>75%	≥50	≥50
Crystals	No	No	No	No	Yes
Culture	Sterile	Sterile	±	Sterile	Sterile
Gram's stain	-	-	±	-	-
Protein	<2 g/dl	N to (↓ dilutional) ↑with hemorrhage the proteins increase but seldom increase above 5 g/dl	↑↑↑(both albumin and characteristically globulins rise)	N to ↑↑↑(complement level reduced in rheumatoid arthritisbut n in ankylosing spondylitis). A:g ratio may bereversed inrheumatoid arthritis	↑↑(globulins increase in proportion to duration of inflammation but unlike rheumatoid arthritis a: g ratio reduces but does not reverse)
Mucin clot	Firm	Firm	Friable, whole aspirate cloudy	Friable	Friable
Glucose (difference from fasting blood sample)	≈ 0	≈ 0	>50 mg%	<50 mg%	>50 mg%

Anatomy of Intervertebral Foramen: (IVF)

- Bound by two mobile joints
  - Zygoapophyseal joints – posteriorly
  - Intervertebral disc – anteriorly
- Roof: Inferior notch of respective vertebral pedicle
- Floor: Superior notch of respective vertebral pedicle.
- Medial wall by thecal sac
- Lateral wall by a fascial sheath with overlying Psoas muscle.
- Content: Spinal nerves (combined dorsal & ventral roots in root sheaths with dorsal root ganglia). Dural sheath & its watershed area as it continues with epineurium of spinal nerve, lymphatics, spinal branch of segmental artery, communicating veins b/w internal & external vertebral venous plexus, sinus vertebral nerves & fat surrounding these structures.
- Basic principle is → Insertion of needle into the disc through safe triangle of Kambin's which lies b/w exiting & traversing nerve roots.

Cross section of spinal cord



Ascending and descending (Motor) Tracts			
Number	Path	Function	Side of body
1	Anterior corticospinal tract	Skilled movement	Opposite
2	Vestibulospinal tract	Facilitates extensor muscle tone	Same
3	Lateral corticospinal (pyramidal tract)	Skilled movement	Same
4	Dorsolateral fasciculus	Pain and temperature	Bidirectional
5	Fasciculus proprius	Short spinal connections	Bidirectional
6	Fasciculus gracilis	Position / fine touch	Same
7	Fasciculus cuneatus	Position/fine touch	Same
8	Lateral spinothalamic tract	Pain and temperature	Opposite
9	Anterior spinothalamic tract	Light touch	Opposite

- Spinal cord is enclosed in three protective membranes—the pia, arachnoid, and dura mater.
- Pia and arachnoid membranes are separated by the subarachnoid space, which contains the cerebrospinal fluid.
- The spinal cord has enlargements in the cervical and lumbar regions that correlate with the brachial plexus and lumbar plexus.
- Within the spinal cord are tracts of ascending (sensory) and descending (motor) nerve fibers.
- These pathways typically are arranged with cervical tracts located centrally and thoracic, lumbar, and sacral tracts located progressively peripheral.
- This accounts for the clinical findings of central cord syndrome and syrinx.
- Understanding the location of these tracts aids in understanding different spinal cord syndromes
- Spinal nerves exit the canal at each level. Spinal nerves C2-7 exit above the pedicle for which they are named (the C6 nerve root exits the foramen between the C5 and C6 pedicles).
- The C8 nerve root exits the foramen between the C7 and T1 pedicles.
- All spinal nerves caudal to C8 exit the foramen below the pedicle for which they are named (the L4 nerve root exits the foramen between the L4 and L5 pedicles).
- Because the spinal cord is shorter than the vertebral column, the spinal nerves course more vertically as one moves caudally. Each level gives of a dorsal (sensory) root and a ventral (mostly motor) root, which combine to form the mixed spinal nerve.
- The dorsal root of each spinal nerve has a ganglion located near the exit zone of each foramen.
- This dorsal root ganglion is the synapse point for the ascending sensory cell bodies. This structure is sensitive to pressure and heat and can cause a dysesthetic pain response if manipulated.

Pathoanatomy based differences in the presentation of brown sequard syndrome and central spinal cord syndrome

Clinical features depend upon the area of the cord that is injured

Central cord syndrome (common in hyperextension-hyper flexion injuries)

It consists of injury to the central area of the spinal cord, including gray and white matter

- Motor deficits (seen more in the upper limbs)
- Varied sensory loss
- Variable bowel and bladder involvement
- Centrally located upper extremity motor neurons in the corticospinal tracts are the most severely affected, and the lower extremity tracts are affected to a lesser extent.
- Generally, patients have a tetra-paresis involving the upper extremities to a greater degree than the lower extremities with greater dysfunction distally in the extremities than proximally.
- Sensory sparing varies, but usually sacral pinprick sensation is preserved. These patients frequently show early partial recovery and may have pre-existing cord compression and may not have spinal instability.
- Prognosis varies, but more than 50% of patients have return of bowel and bladder control, become ambulatory, and have improved hand function
- This syndrome usually results from a hyperextension injury in an older individual with preexisting osteoarthritis of the spine.
- The spinal cord is pinched between the vertebral body anteriorly and the buckling ligamentum flavum and lamina posteriorly
- It also may occur in younger patients with flexion injuries.

Brown Sequard syndrome (seen in hyperextension injuries and facet and compression fracture)

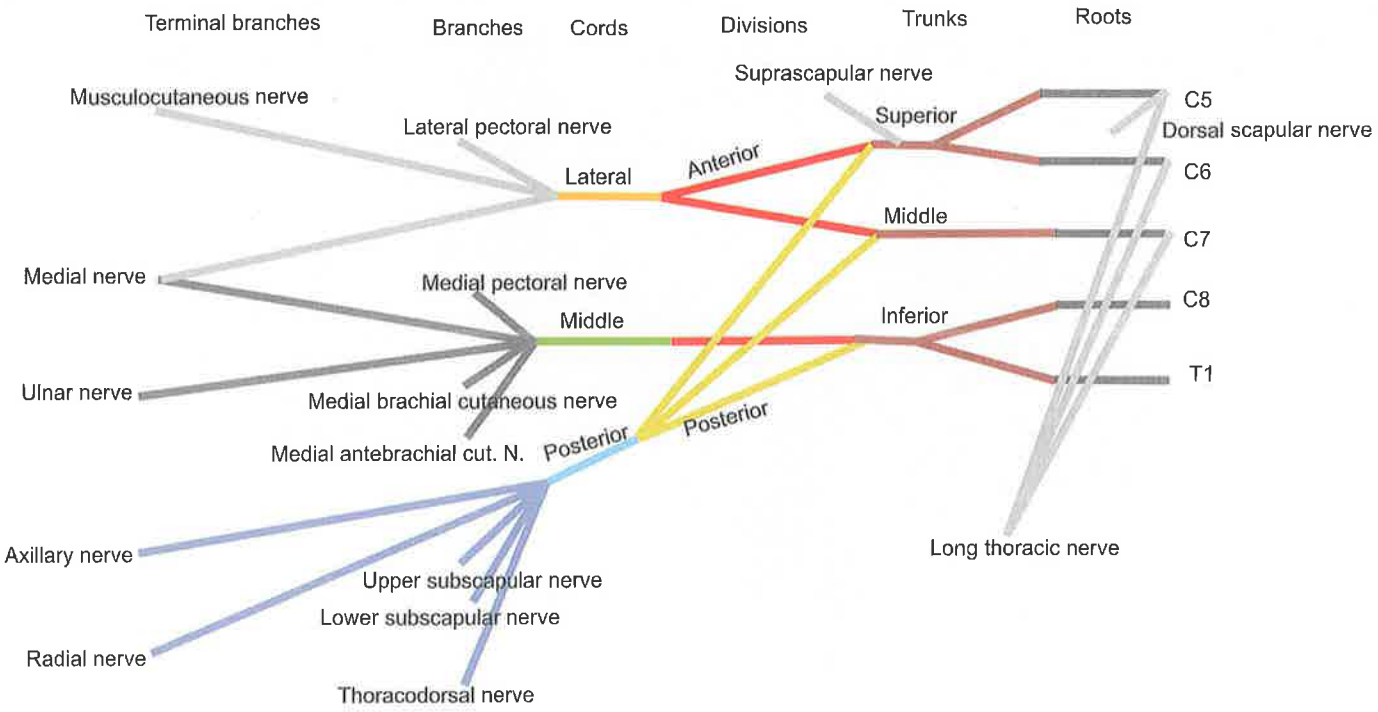
- Brown–Sequard syndrome refers to injury to the lateral half (hemisection) of the spinal cord.
- Injury to either side of the spinal cord and usually is the result of a unilateral laminar or pedicle fracture, penetrating injury, or rotational injury resulting in a subluxation.
- It is characterized by motor weakness on the side of the lesion and the contralateral loss of pain and temperature sensation.
- Ipsilateral loss of vibratory perception
- Segmental lower motor neuron signs at the level of the lesion
- Ipsilateral loss of proprioception (position sense)-below level of lesion
- Contralateral loss of pain and temperature sensibility - (one or two segments below level of lesions)
- Ipsilateral motor loss with spastic paresis
- Inability to walk
- Loss of normal bowel and bladder function
- Prognosis for recovery is good, with significant neurologic improvement often occurring. Pollard and Apple noted that only central cord and Brown-Séquard syndromes were statistically associated with improved recovery at 2 years after injury.

Anatomy of Brachial Plexus

The origin of the brachial plexus is from the fifth through eighth cervical (C5 through C8) and the first thoracic (T1) spinal nerves. Small contributions may originate from the fourth cervical (C4) and second thoracic (T2) nerves. The dorsal and ventral rootlets (six to eight rootlets per level) exit the spinal cord, merge to form the spinal nerves, which leave the intervertebral foramina, and quickly divide into dorsal and ventral rami. The small dorsal rami travel posterior to innervate the skin and muscles of the neck and upper back and are not part of the brachial plexus. The ventral rami emerge between the anterior and middle scalene muscles and are designated as the nerve roots of the brachial plexus. The upper roots (C5 through C8) descend toward the first rib, whereas the lower T1 root must ascend over the first rib to form the brachial plexus. Sympathetic fibers join the nerve roots as they traverse between the scalene muscles. C5 and C6 receive fibers from the middle sympathetic cervical ganglion, and C7, C8, and T1 acquire fibers from the cervicothoracic ganglion. These sympathetic fibers control blood-vessel smooth-muscle contraction (vasoconstriction) and sweat gland activity.

The motor cell bodies of the nerve roots for the brachial plexus are located within the ventral horn of the spinal cord gray matter. In contrast, the sensory cell bodies are positioned outside the spinal cord within the dorsal root ganglia. The dorsal root ganglia transfer afferent fibers to the spinal cord through the dorsal rootlets. The knowledge of the difference in anatomic location of cell bodies between motor and sensory fibers is important for the accurate diagnosis and treatment of proximal brachial plexus injuries.

The ventral rami of C5 and C6 combine to form the superior trunk, the C7 ramus continues alone as the middle trunk, and C8 and T1 unite to form the lower trunk. The trunks are located in the posterior triangle of the neck, enclosed by the posterior border of the sternocleidomastoid muscle, anterior border of the upper trapezius, and clavicle. The spinal accessory nerve (cranial nerve XI) crosses the posterior triangle to innervate the trapezius muscle. This nerve divides the posterior triangle into nearly equivalent upper and lower parts. The lower portion of the triangle contains the brachial plexus, with the upper and middle trunks superior to the omohyoid muscle and the lower trunk inferior. Each trunk divides into anterior and posterior divisions and proceeds behind the clavicle. The divisions then merge into three cords named in relation to the axillary artery. The anterior divisions of the upper and middle trunks combine to form the lateral cord. The three posterior divisions of the upper, middle, and inferior trunks converge to form the posterior cord. The anterior division of the lower trunk continues as the medial cord.



The cords proceed behind the pectoralis minor muscle into the axilla. Each cord divides into two terminal branches . The lateral cord terminates as the musculocutaneous nerve and a branch to the median nerve. The musculocutaneous nerve perforates and supplies the coracobrachialis muscle and then becomes the principal motor nerve of the flexor compartment of the arm. The posterior cord divides into the axillary and radial nerves. At the level of the glenohumeral joint, the axillary nerve, along with the posterior humeral circumflex vessels, travels inferior to the subscapularis muscle and across the upper border of the teres major to enter the quadrangular space for innervation of the deltoid and teres minor muscles. Compression of the nerve or artery can occur at the quadrangular space from hypertrophy or anomalies of the bordering muscles. The radial nerve is the largest branch of the brachial plexus and passes inferior to the teres major muscle to enter the posterior arm between the long head of the triceps and humerus. The medial cord continues as the ulnar nerve and a branch to the median nerve. The ulnar nerve travels down the arm medial to the brachial artery, pierces the medial intermuscular septum, and enters the cubital tunnel. The median nerve forms anterior to the axillary artery from the union of the medial and lateral cord branches and descends into the arm.

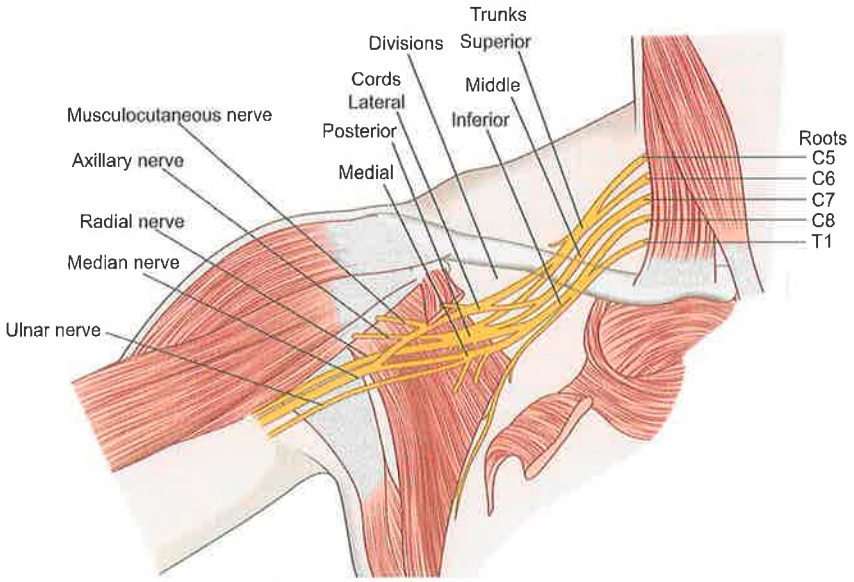


Fig: Brachial Plexus From Roots To Branches

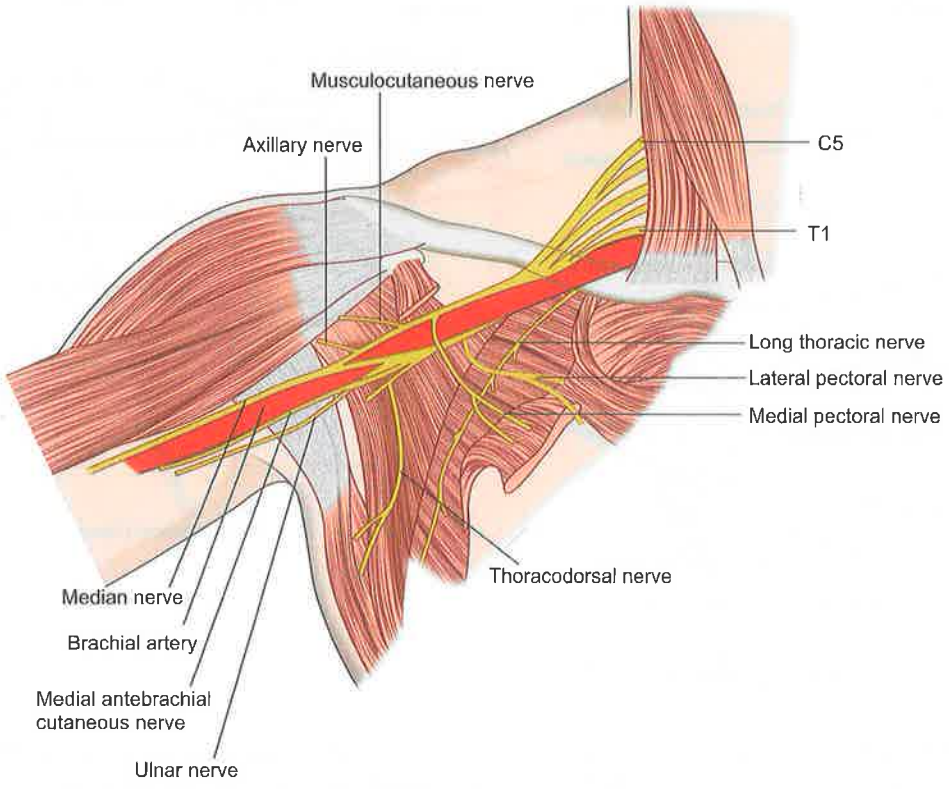


Fig: Brachial Plexus Behind The Clavicle Pectoralis Minor Muscle

Several branches arise from the roots, trunks, and cords of the brachial plexus . The status of these intermediate nerves provides valuable information about the location of nerve injury. At the root level, the dorsal scapular nerve arises from C5, pierces the middle scalene to enter the posterior triangle of the neck, and innervates the levator scapulae and rhomboid muscles. The long thoracic nerve arises from C5, C6, and C7 just distal to the intervertebral foramina and travels behind the brachial plexus along the chest wall to supply the serratus anterior muscle. The phrenic nerve originates at the root level from C3, C4, and C5 and crosses the anterior scalene muscle to enter the thorax. The phrenic nerve may be injured in nerve root injuries, which results in hemidiaphragm paralysis.

At the trunk level, the suprascapular nerve arises from the superior trunk to travel across the posterior cervical triangle to the suprascapular notch. The suprascapular nerve advances through this notch to innervate the supraspinatus and infraspinatus muscles.

There are no branches from the plexus at the division level. At the cord level, multiple branches are present. From the lateral cord, the lateral pectoral nerve arises to pass anterior to the axillary artery, to perforate the clavipectoral fascia, and to innervate the clavicular part of the pectoralis major. The posterior cord supplies three branches: the upper subscapular, thoracodorsal, and lower subscapular nerves. The upper subscapular nerve innervates the upper portion of the subscapularis muscle. The lower subscapular nerve innervates the lower subscapularis and the teres major muscle. The thoracodorsal nerve originates between the upper and lower subscapular nerves, passes behind the axillary artery, and supplies the latissimus dorsi muscle. The medial cord provides one motor and two sensory branches. The medial pectoral nerve traverses and innervates the pectoralis minor muscle and then continues to supply the sternocostal portion of the pectoralis major muscle. The medial brachial and medial antebrachial cutaneous nerves are the only sensory branches to arise directly from the plexus and supply the arm and forearm, respectively.

The vascular anatomy of the brachial plexus centers about the subclavian and axillary vessels. The subclavian artery originates from the arch of the aorta on the left side and from the brachiocephalic artery on the right. The subclavian artery ascends over the first rib to reside between the anterior and middle scalene muscles, with the roots and trunks of the brachial plexus. In contrast, the subclavian vein is located anterior to the anterior scalene muscle. The subclavian vessels cross the first rib and become the axillary vessels with the vein medial to the artery. The axillary vessels accompany the brachial plexus behind the pectoralis minor muscle to enter the axilla. The axillary vessels become the brachial vessels beyond the axilla.

There is variability and asymmetry in the neural and vascular anatomy of the brachial plexus. The plexus is termed prefixed when there is a relatively large contribution from C4 and a small allotment from T1. Similarly, a postfixed plexus has substantial contribution from T2 with little from C5. There are also variations in cord separation and peripheral branchings

of nerves that may or may not affect segmental innervation. Branches may occasionally arise from divisions that usually originate from trunks or cords. The vascular relation to the plexus can be altered, with the axillary artery or vein shifted in position or even piercing a nerve. The subclavian vein can travel with the artery and brachial plexus posterior to the anterior scalene muscle. Anomalies in nerve and/or vascular anatomy should be considered when clinical, diagnostic, and surgical findings do not correspond.

Nerve	Muscles supplied
Branches from Roots	
Dorsal scapular nerve	Levator scapulae and Rhomboid muscles
Long Thoracic nerve	Serratus Anterior
Phrenic nerve	Diaphragm
Branches from trunks	
Suprascapular nerve	Supraspinatus, Infraspinatus
Nerve to subclavius	Subclavius
Branches from cords	
Lateral cord	
Lateral pectoral nerve	Pectoralis Major(clavicular part)
Musculocutaneous nerve	Coracobrachialis, Brachialis, Biceps
Branch to median Nerve	*
Medial cord	
Branch to Median nerve	
Medial Ante brachial cutaneous nerve	Sensory nerve to medial half of forearm
Medial Brachial cutaneous nerve	Sensory nerve to medial half of arm
Medial Pectoral nerve	Pectoralis Major(sternocostal part)
Ulnar Nerve	#
Posterior cord	
Radial Nerve	##
Axillary nerve	Deltoid, Teres Minor
Upper Subscapular nerve	Upper Subscapularis
Lower Subscapular nerve	Lower Subscapularis, Teres Major
Thoracodorsal Nerve	Lattismus Dorsi

* Median nerve – Pronator teres, Flexor carpi radialis, flexor digitorum superficialis, flexor digitorum profundus (medial half), palmaris longus, flexor pollicis longus, pronator quadrates, flexor pllocis brevis, abductor pollicis brevis, Opponens Pollicis, first and second lumbricals

Ulnar nerve - flexor carpi ulnaris, the flexor profundus to the little and ring fingers, the lumbricals of the little and ring fingers, all of the interossei, the adductor of the thumb, and all of the short muscles of the little finger

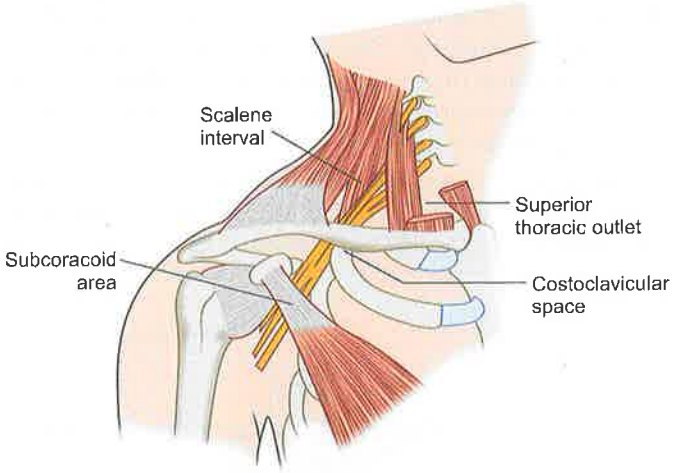
Radial nerve - triceps; the supinators of the forearm; and the extensors of the wrist, fingers, and thumb

Thoracic outlet

The thoracic outlet begins just distal to the intervertebral foramina and extends to the coracoid process. The outlet is surrounded by anatomic constraints that encompass the brachial plexus and associated vessels (subclavian and axillary). These structures include muscles (anterior and middle scalene muscles), skeleton (first rib, cervical ribs, clavicle, and coracoid), and fascia or fibrous bands.

The most common sites of compression in thoracic outlet syndrome are at the superior thoracic outlet, the scalene interval or triangle, the costoclavicular space, or the subcoracoid area. This compression can be static or dynamic (i.e., dependent on posture and activity).

The anterior border of the superior thoracic outlet is the sternum; lateral boundary in the first rib and the posterior border in the thoracic vertebrae. The inferior trunk must ascend from the intervertebral foramina to navigate over the first rib. A postfixed brachial plexus must climb even higher to exit from the thorax. The inferior trunk can be compressed or stretched over the first rib in this area.



The most important structure is the scalene, or inter-scalene, triangle formed by the anterior scalene muscle anteriorly and medially, the middle scalene posteriorly and laterally, and the superior border of the first rib inferiorly. Both scalene muscles insert at the first rib, the anterior scalene inserts at the scalene tubercle, and the middle scalene inserts 1 or 2 cm further laterally. The anterior scalene has its origin on the anterior tubercles of the lateral masses of the C3–C6 cervical vertebrae. The middle scalene originates from the posterior tubercles of the C2–C7 vertebrae. The scalene triangle has a narrow base (approximately 1 to 2 cm) and elongated sides. The upper roots (C5 through C7) descend, whereas the inferior roots (C8 and T1) and subclavian artery must ascend to pass through the scalene triangle.

The spinal nerves of the brachial plexus exit their neural foramina between the tubercles and thus, descend between the scalene in a lateral, caudal, slightly anterior direction. As the C5–C8 spinal nerves pass superior to the first rib, the T1

root must take a different course. Exiting the neural foramen between the T1 and T2 vertebrae in the thorax caudal to the first rib, the T1 spinal nerve passes superiorly, crossing the first rib anteriorly, and then turning superiorly around its neck to join the C8 nerve at the superior surface of the rib and form the lower trunk. Thus, the origins of the lower trunk embrace the neck of the first rib. This arrangement is important because T1 and the proximal portion of the lower trunk are tethered inferiorly by the origin of T1. Thus, the lower trunk and especially the T1 spinal nerve are vulnerable to any element compressing them from below, because they cannot be displaced superiorly and will therefore be deformed and compressed. This is the most important fact in the understanding of the neurogenic TOS.

The subclavian artery leaves the thorax and ascends between the scalenes anterior to the lower trunk of the plexus before entering the axilla above the first rib. Although the entire brachial plexus traverses the scalene triangle, only the lower trunk of the plexus and the subclavian artery come into contact with the first rib between the insertions of the scalenes.

It is in this area of the inferior scalene triangle that the lower trunk of the plexus and the subclavian artery can be compressed by anatomic anomalies such as an aberrant or fused scalene muscle insertion, cervical ribs, fibrous bands, or an anomalous first rib. Nearly all of the signs and symptoms of the various appearances of the TOS appear to involve the lower trunk of the brachial plexus.

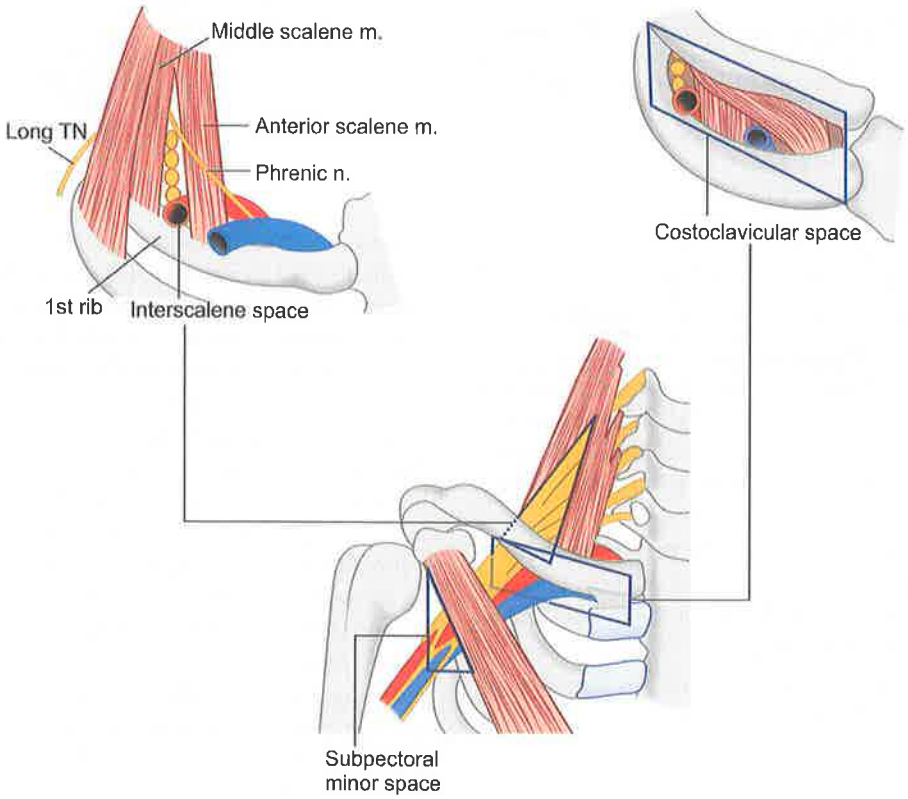
The presence of a cervical rib or a fibrous band extending from an incomplete cervical rib to the first rib can reduce the dimensions of the triangle by elevation of its base. This forces the inferior roots and subclavian artery to further ascend to enter the scalene triangle. Cervical ribs are present in 0.5% to 1% of individuals and occur bilaterally 50% to 80% of the time. The width of the scalene triangle can be narrowed by anterior or middle scalene muscle abnormalities, which can precipitate thoracic outlet compression.

Sites of compression in thoracic outlet syndrome

Site	Principal Cause
Superior thoracic outlet	First rib or cervical rib
Scalene interval or triangle	Scalene muscles or fibrous bands
Costoclavicular space	Narrow clavicle first rib distance
Subcoracoid area	Coracoid process

The costoclavicular interval is between the clavicle and first rib. Depression of the clavicle reduces this space and can compress the brachial plexus and subclavian vessels. A hypertrophied subclavius muscle or a clavicle with abundant callus formation can narrow the costoclavicular space. The subclavian vein is also susceptible to compression within the costoclavicular interval.

The subcoracoid area can compress the brachial plexus. The coracoid provides a fulcrum across the plexus during abduction and external rotation of the arm. The pectoralis minor and conjoined tendon (short head of the biceps and coracobrachialis) prevents slippage of the plexus from behind the coracoid. Excessive arm elevation can lead to a traction or compressive neuropathy along the coracoid process.



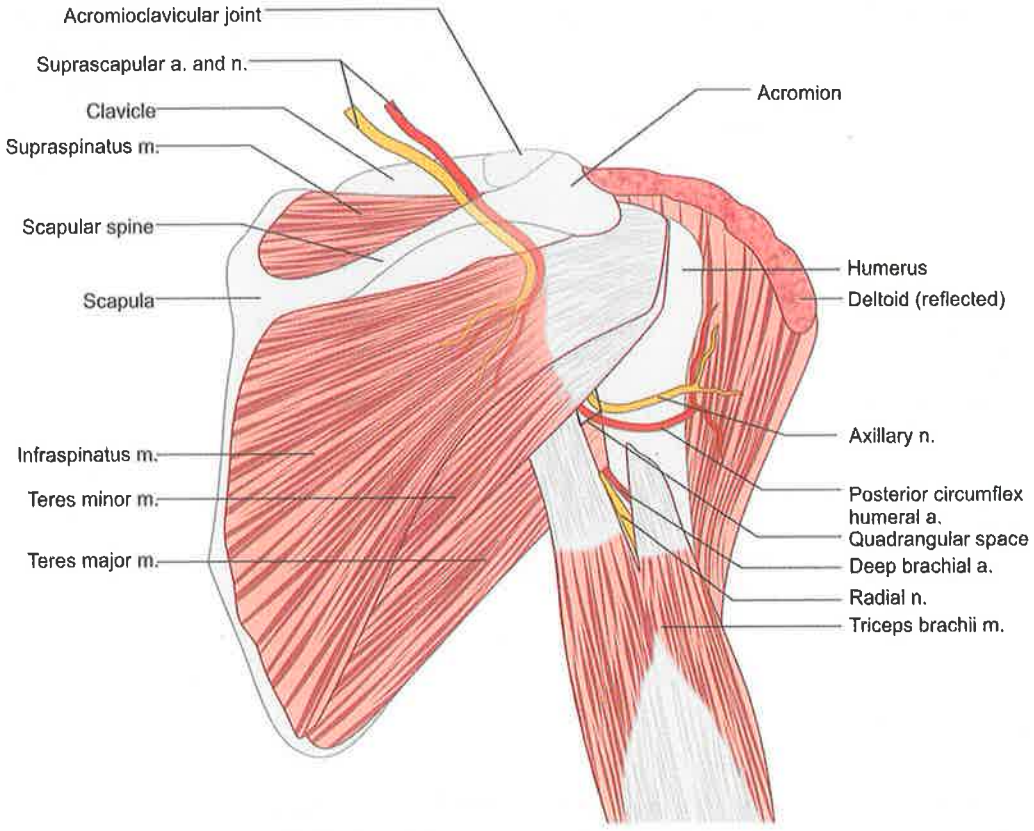
Boundaries of Quadrangular space. Describe quadrangular space syndrome, Features and management

The quadrilateral space is bounded superiorly by the

- Teres minor muscle,
- Inferiorly by the teres major muscle,
- Medially by the long head of the triceps, and
- Laterally by the humeral shaft.

The axillary nerve and PHCA reside in the quadrilateral space. The axillary nerve innervates the teres minor and deltoid muscles, which are primarily responsible for abduction and external rotation.

Quadrilateral space syndrome (QSS) is a rare disorder characterized by axillary nerve and posterior humeral circumflex artery (PHCA) compression within the quadrilateral space, first described by Cahill and Palmer.



Clinical features

- Younger patients, less than forty years of age,
- History of repeated overhead activities, for example, in athletes of volleyball, baseball, or swimming.
- Symptom presentation can be vague with involvement of neurogenic or vascular features.
- Neurogenic QSS is characterized by paresthesia, fasciculations, weakness, or neurogenic pain in a nonspecific pattern.
- Vascular QSS include signs of acute ischemia (pain, pallor, absent pulses), thrombosis, or embolism (coolness or cyanosis of the hand or digits).
- Muscular atrophy and accompanying weakness, thought to be a result of denervation.
- Tenderness over the quadrilateral space.
- In severe cases, thrombosis of the PHCA can block flow from the axillary artery, causing embolization and subsequent cyanosis, digital ischemia, and cold intolerance.
- Etio-pathology
- Repeated overhead activity (such as baseball or volleyball athletes),
- Other pathologies including lipomas, hematomas, and labral cysts may cause compression in the quadrilateral space.
- Characteristic fibrous bands are found within the quadrilateral space, which exacerbate symptoms, particularly pain, elicited by movements associated with the deltoid and teres minor muscles. The anatomical differences in innervation patterns in the glenohumeral joint between patients can make it difficult to distinguish whether pain is due to suprascapular nerve palsy or axillary nerve compression. Because the PHCA stretches around the neck of the humerus, repetitive tension and mechanical stress to the PHCA wall can lead to thrombosis and aneurysmal degeneration.

Management

Investigations- MRI to look for cause, arteriography to rule

out vascular blockade. Bilateral upper extremity arteriography is useful in establishing the patient's baseline healthy anatomy, as compared to the pathological shoulder. Decreased outflow from the PHCA would indicate compression of the adjacent axillary nerve leading to QSS.

One should have high level of suspicion of QSS when

The four distinct features of QSS are present:

1. Diffuse pain around the shoulder;
2. Paresthesia in a non-dermatomal distribution;
3. Point tenderness above the quadrilateral space; and
4. Positive angiogram finding in provocative positioning.

Treatment

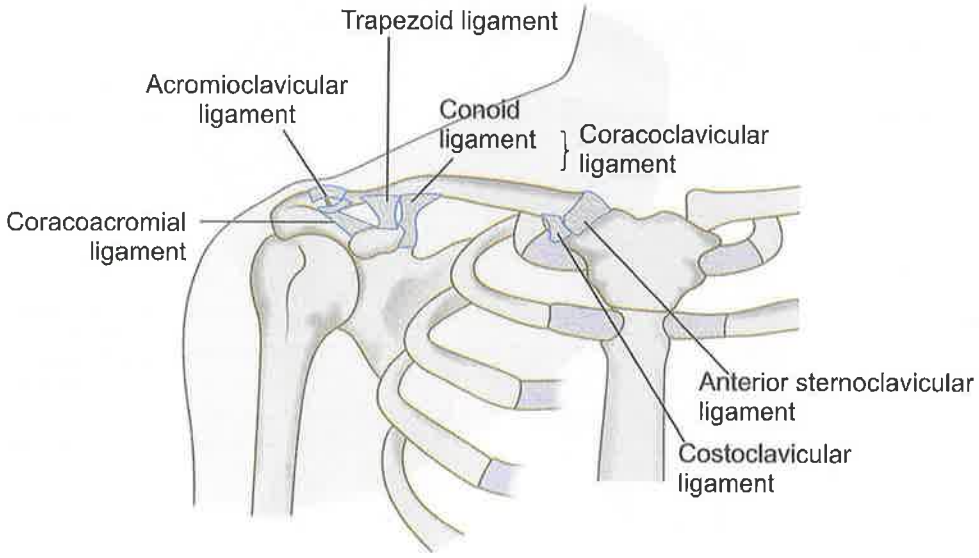
Mostly conservative. Life style modification. Physical therapy

Surgical decompression can be done in recalcitrant cases or in cases where evident compression is noted.

Pathoanatomy of AC Joint

Bone and Ligament Anatomy

An understanding of the anatomy and biomechanics of the acromioclavicular joint is essential to treating patients with acromioclavicular pathology. Knowledge of the exact locations of insertion of the acromioclavicular ligaments along with the conoid and trapezoid ligament is important prior to operative resection of the distal clavicle. Comprehensive knowledge of the bony and ligamentous anatomy of this joint will also give us a better understanding of newer and better operative techniques to recreate anatomy of the acromioclavicular joint.



The acromioclavicular joint is a diarthrodial joint involving the medial facet of the acromion and the distal clavicle. Its articular surfaces are covered with hyaline cartilage until age 17 when it acquires the characteristics of fibrocartilage on its acromial surface and at age 23 on its clavicular surface. Before the age of 3 to 5 years only a fibrocartilaginous bridge exists between the acromion and the clavicle and not a true joint cavity. Along with the sternoclavicular joint, the acromioclavicular joint provides a bony link of the shoulder to the axial skeleton. The inclination of the joint is variable in both the sagittal and coronal planes. The most common type of inclination is with the clavicle overriding the acromion. The least common inclination is with the clavicle underriding the acromion. Acromioclavicular joints with a more vertical orientation are more susceptible to osteolysis because higher forces were concentrated across the clavicle. A fibrocartilaginous disc of varying size and shape is present in the joint, which, unlike that of the sternoclavicular joint, usually has a large perforation in its center. Degenerative changes of the disc increase in frequency with age.

The presence of a coracoclavicular joint is uncommon (about 1%)but varies from the presence of a well-developed diarthrodial articulation to the presence of articular facets and synovial structures within the coracoclavicular ligaments. A bony bar may form in place of the coracoclavicular ligaments, the so-called coracoclavicular bar. This connection may be complete or incomplete. Coracoclavicular articulations have also been linked to compression of the subclavian artery and brachial plexus, along with early degenerative changes of the acromioclavicular and sternoclavicular joints.

The primary stabilizers of the acromioclavicular joints are the ligaments that surround the joint. A thin capsule surrounds the joint and is stabilized by anterior and posterior as well as superior and inferior ligaments. The superior acromioclavicular ligament is the most robust of the ligaments and is reinforced by fascial attachments of the deltoid and trapezial muscles. The inferior ligament in many cases is nonexistent. Anteroposterior stability of the acromioclavicular joint is controlled by the acromioclavicular ligaments.

Vertical stability (superoinferior) is provided by the coracoclavicular ligaments (conoid and trapezoid). These

ligaments pass from the inferior surface of the clavicle to the base of the coracoid process of the scapula. The trapezoid ligament is the most lateral of the two. It is a broad, thin, quadrilateral-shaped ligament running obliquely from the superior surface of the coracoid process to the oblique ridge on the inferior surface of the clavicle. The conoid ligament is more conical in form, with its base directed superiorly. Its apex attaches to a rough impression at the base of the coracoid process medial to the trapezoid ligament. It attaches by its base to the coracoid tuberosity on the inferior surface of the clavicle and a line proceeding medial for 1 cm on this bone. These ligaments are the primary stabilizers of the acromioclavicular joint and constitute the primary supports by which the scapula is suspended from the clavicle. Division of the acromioclavicular ligaments and joint capsule, along with detachment of the trapezius and deltoid, allows less than 50% subluxation superiorly. Posterior dislocation, though, is possible. Complete superior dislocation of the acromioclavicular joint will occur only after complete division of the coracoclavicular ligaments has also occurred.

The conoid ligament is the primary stabilizer with superior displacements. There is considerable variation in the length and width of the conoid and trapezoid ligaments. Recent literature also found variation in the insertional distances (from the acromioclavicular joint) of these ligaments. There was found to be a significant gender-related difference in the insertional distances of either the trapezoid or conoid ligaments. Resection of less than 11 mm should not violate the trapezoid ligament and resection less than 24 mm should not violate the conoid ligament in either gender.

In children, the clavicle is surrounded by a thick periosteal tube that extends all the way to the acromioclavicular joint. A secondary ossification center is present at the distal clavicle and fuses with the clavicle at about the age of 19. Because of the variation in anatomy compared with adults, children are more prone to fracture and pseudodislocations than true dislocation of the acromioclavicular joint.

An ossification center appears in the body of the coracoid at approximately 1 year of age. Another combined ossification center for the base of the coracoid and upper forth of the glenoid occurs close to age 10. These centers fuse to the scapula at about age 15. Near puberty, the acromion forms two to five ossification centers that fuse at about age 22. Failure to fuse the acromial ossification center leads to an os acromiale.

The blood supply to the acromioclavicular joint is derived mainly from the acromial artery, a branch of the deltoid artery of the thoracoacromial axis. There are rich anastomoses between this artery, the suprascapular artery, and the posterior humeral circumflex artery. The acromial artery comes off the thoracoacromial axis anterior to the clavipectoral fascia and perforates back through the clavipectoral fascia to supply the joint. It also sends branches anteriorly up onto the acromion. Innervation of the joint is supplied by the lateral pectoral, axillary, and suprascapular nerves.

Biomechanics

Acromioclavicular joint motion occurs as the arm abducts and forward flexes. While the clavicle rotates upward 50 degrees with overhead motion, only 5 to 8 degrees occurs at the acromioclavicular joint due to synchronous scapuloclavicular motion. Thus, while the clavicle rotates the scapula rotates with it, and therefore, minimal rotation (5 to 8 degrees) is required between the two. Hence, most scapulothoracic motion must occur at the sternoclavicular articulation. Clinically, fusion of the acromioclavicular joint will lead to minimal dysfunction.

It is seldom that AC ligaments are called on to resist trauma, and their usual function is to control joint motion. As noted earlier, this joint has relatively little motion, and muscles controlling scapulothoracic motion insert on the scapula. To a large extent, the ligaments function to guide motion of the clavicle. For example, the conoid ligament produces much of the superior rotation of the clavicle as the shoulder is elevated in flexion.

Vascular Anatomy of Proximal Humerus And Its Clinical Relevance In Fractures

The rich vascular anatomy of the proximal humerus has been delineated in the recent literature to help explain osteonecrosis of the humeral head. Studies have showed that the anterior lateral branch of the anterior humeral circumflex artery is the primary blood supply to the proximal humerus. The anterior humeral circumflex artery arises from the lateral side of the third division of the axillary artery approximately 1 cm distal to the inferior border of the pectoralis minor muscle (just above the teres major muscle) and courses laterally behind the coracobrachialis to reach the surgical neck of the humerus at the lower border of the subscapularis. The anterolateral branch sends twigs to the lesser tuberosity, crosses under the biceps tendon, and then arches superiorly adjacent to the lateral side of the intertubercular groove. The vessel then penetrates bone at the cephalad portion of the transition from greater tuberosity to intertubercular groove, staying distal to the position of the old epiphyseal plate. The intraosseous portion of this artery has been named the arcuate artery because of its posteromedial course after entering the humeral head. Multiple branches radiate to supply the subchondral bone of the humeral head.

The importance of both the extra- and intraosseous arterial anastomoses has recently been described. Abundant extraosseous anastomosis has been seen between the anterior humeral circumflex artery and posterior humeral circumflex arteries, as well as with the thoracoacromial, subscapular, suprascapular, and profunda brachii arteries. The anterior and posterior circumflex vessels connect with the profunda brachii artery through ascending deltoid vessels. Anastomosis of the profunda brachii artery occurs with the radial (anterior) and medial (posterior) collaterals. This rich collateral circulation about the shoulder can provide adequate circulation to maintain viability of the extremity despite axillary artery disruption after,

for example, a dislocation.

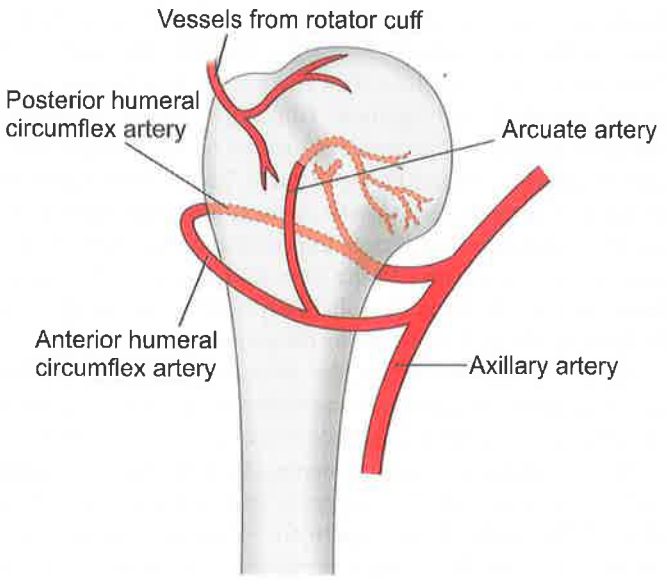
However, vascularization of the entire humeral head is possible only through the anterior lateral branch of the anterior humeral circumflex artery. There are inconsistent contributions to the humeral head from both the lesser and greater tuberosities. It has not been confirmed that vessels of the rotator cuff directly vascularized the underlying bone.

The posterior humeral circumflex artery arises closely juxtaposed to the anterior humeral circumflex artery and is much larger in diameter. The posteromedial vessels arising from the posterior humeral circumflex artery supply the posterior portion of the greater tuberosity and a small posterior inferior part of the head. The vessels then form an anastomosis to the arcuate artery on and within the greater tuberosity and on the joint capsule. These posteromedial vessels pass beneath the humeral capsular attachment (which at this site extends from 1 cm onto the surgical neck) and run toward the humeral head before entering the bone just below the articular margin.

Many authors have emphasized the importance of the intraosseous anastomoses and showed that the humeral head could be completely profused after ligation of the anterior humeral circumflex artery at its entry site into the humeral head. They found large metaphyseal arteries that passed through the fused growth plate to anastomose with the arcuate artery. In addition, significant intraosseous anastomoses occurred between the arcuate artery and the posterior humeral circumflex artery through the posteromedial vessels described in the foregoing and the vessels of the greater and lesser tuberosities.

Knowledge of the vascular anatomy may help predict osteonecrosis of the humeral head. Researchers have highlighted the clinical relevance of the intraosseous anastomosis in shoulder dislocations with both circumflex arteries interrupted and the extraosseous network compromised. Osteonecrosis did not develop in these patients, suggesting that the intraosseous anastomoses are important. A classic four-part fracture will lose the blood supply to the head from disruption of the anterior humeral circumflex artery, greater and lesser tuberosities, and any metaphyseal arterial anastomosis. Thus, a high rate of osteonecrosis is expected. Yet, authors have shown that perfusion of the humeral head by the arcuate artery may continue if the head fragment includes part of the medial aspect of the upper part of the neck where the posteromedial arteries enter. The so called four-part valgus-impacted fracture with limited lateral displacement of the head fragment may retain its vascularity from the posterior medial vessels, thus accounting for the lower incidence of osteonecrosis reported compared with classic four-part fractures. The vascular anatomy also supports the observation that minimally displaced fractures of the anatomic neck, in which the fracture line is at the junction between the articular surface and neck, may be complicated by osteonecrosis as a result of disruption of both the extra- and intraosseous blood supply.

Knowledge of the extensive collateral circulation around the shoulder helps explain the mechanism whereby the viability of the upper extremity can be maintained despite disruption of the axillary artery.



Anatomy of Rotator Cuff

Four of the scapulohumeral muscles "supraspinatus, infraspinatus, teres minor, and subscapularis"are called rotator cuff muscles because they form a musculotendinous cuff around the glenohumeral joint and all except the supraspinatus are rotators of the humerus.

The four muscles take their origin from the body of the scapula and envelope the humeral head as they insert along the tuberosities of the proximal humerus. The musculotendinous cuff is firmly adherent to the underlying glenohumeral capsule and provides circumferential reinforcement except at the rotator interval and axillary recess. The rotator interval is a triangular area that is made up of fibrous elements and bordered by the upper margin of the subscapularis tendon and anterior aspect of the supraspinatus. Within the interval lie the coracohumeral ligament, the biceps tendon, and the superior glenohumeral ligament.

The four rotator cuff muscles are often considered as distinct separate musculotendinous units that directly overlie the joint capsule and insert onto the proximal humerus. The fibres of the rotator cuff tendons interdigitate and fuse, forming a common insertion on the tuberosities of the humerus. Fibres from both the subscapularis and infraspinatus interdigitate with respective fibres of the supraspinatus. Microscopically, the rotator cuff complex is stratified into five distinct layers that also receive reinforcement from the coracohumeral ligament and is contiguous with the glenohumeral capsule

Layer 1 is composed of superficial fibres that overlie the cuff tendons and extend from the coracoid process to the greater tuberosity. These fibres form an extension of the coracohumeral ligament (CHL).Layers 2 and 3 contain the fibres of the supraspinatus (SP) and the infraspinatus (IS) tendons. The fibres in layer 2 are oriented parallel to the axes of the supraspinatus and infraspinatus tendons. The fibres of layer 3 are smaller and are obliquely oriented with