

BONE FRACTURE

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Fracture

- A bone fails when the loading forces exceed the load bearing capacity.
- Depending on both the force of the injury and the properties of the bone involved, load failure results in a fracture in several unique patterns.
- Three fundamental forces cause fractures: shear, compressive, and tensile.
- Bone is least able to withstand shear forces, followed by tension, then compression

Fracture

- Tensile bone failure causes fractures perpendicular to the direction of loading (transverse)
- Compression forces cause oblique stresses in a plane approximately 45 degrees to the bone's long axis.
- Bending forces result in a tensile stress to the convex side and a compressive stress to the concave side
- Results in transverse and oblique fractures on the tensile and compressive sides respectively.

Fracture

- The tensile-compressive pattern from bending can cause a resultant bone wedge referred to as a butterfly fragment or the characteristic greenstick fracture.
- Torsion (rotational) forces lead to more complex fractures by causing a small crack to extend into a spiral pattern.
- Many fractures, however, involve combinations of forces and therefore develop complex fracture patterns.

Fracture

- Buckle (torus) fractures follow compression failure, often at the junction between the porous metaphysis and the denser diaphysis.
- These injuries typically occur in the distal radius after longitudinal trauma (fall on an outstretched hand), but are also seen in the distal tibia, fibula, and femur.
- Buckle fractures are by definition stable and can often be managed with splinting.
- The greenstick fracture occurs in >84% of forearm re-fractures.

Fracture

- A plastic deformity (or bowing fracture) occurs when a longitudinal force exceeds the bone's ability to recoil to its normal position.
- If the tension side cannot propagate the fracture, microscopic fractures can occur to dissipate the impact energy, thus creating a plastic deformity.
- Most commonly seen in the ulna, the radius, and occasionally in the fibula.

Fracture

- If the deformation is less than 20 degrees or if the deformity occurs in a child <4 years of age, the angulation often corrects itself.
- Otherwise, closed reduction or operative intervention may be necessary to straighten the bone.

Growth plate

- The germinal zone of the growth plate borders the epiphysis.
- The epiphyseal cartilage cells grow toward the metaphysis and form columns of cells.
- These columns degenerate, undergo hypertrophy and then calcify at the metaphysis to form new bone.

Fracture

- The weakest part of the growth plate is the zone of hypertrophic cartilage and accordingly the most common site for physeal fractures .
- In infancy and early childhood, the physis is relatively thick and the epiphysis is mostly cartilaginous, serving as a shock absorber and transmitting forces to the metaphysis.
- During adolescence, when the epiphysis begins to ossify, these forces are less absorbed and consequently transmitted to the physis.

Fracture

- Growth plates are susceptible to fracture and represent a weak point in pediatric bone.
- Because the tensile strength of pediatric bone is less than that of the ligaments, the same injury mechanism causing a ligamentous injury in adults (sprain or strain) is more likely to cause a bone injury in children:
 - The physis will separate or fracture before disruption or "spraining" of an adjacent strong and flexible ligament.
- Occur in 21 to 30 percent of pediatric long bone fracture, more commonly involving the distal growth plates of the radius and ulna.

Fracture

- In girls, growth plate injuries occur between ages 9 and 12, while in boys they typically occur later, between ages 12 and 15.
- Although a majority of these fractures heal without incident, approximately 30 percent lead to premature closure and unilateral long bone shortening.
- Appropriate anatomic alignment is critical for optimal growth.

Epiphyseal fracture

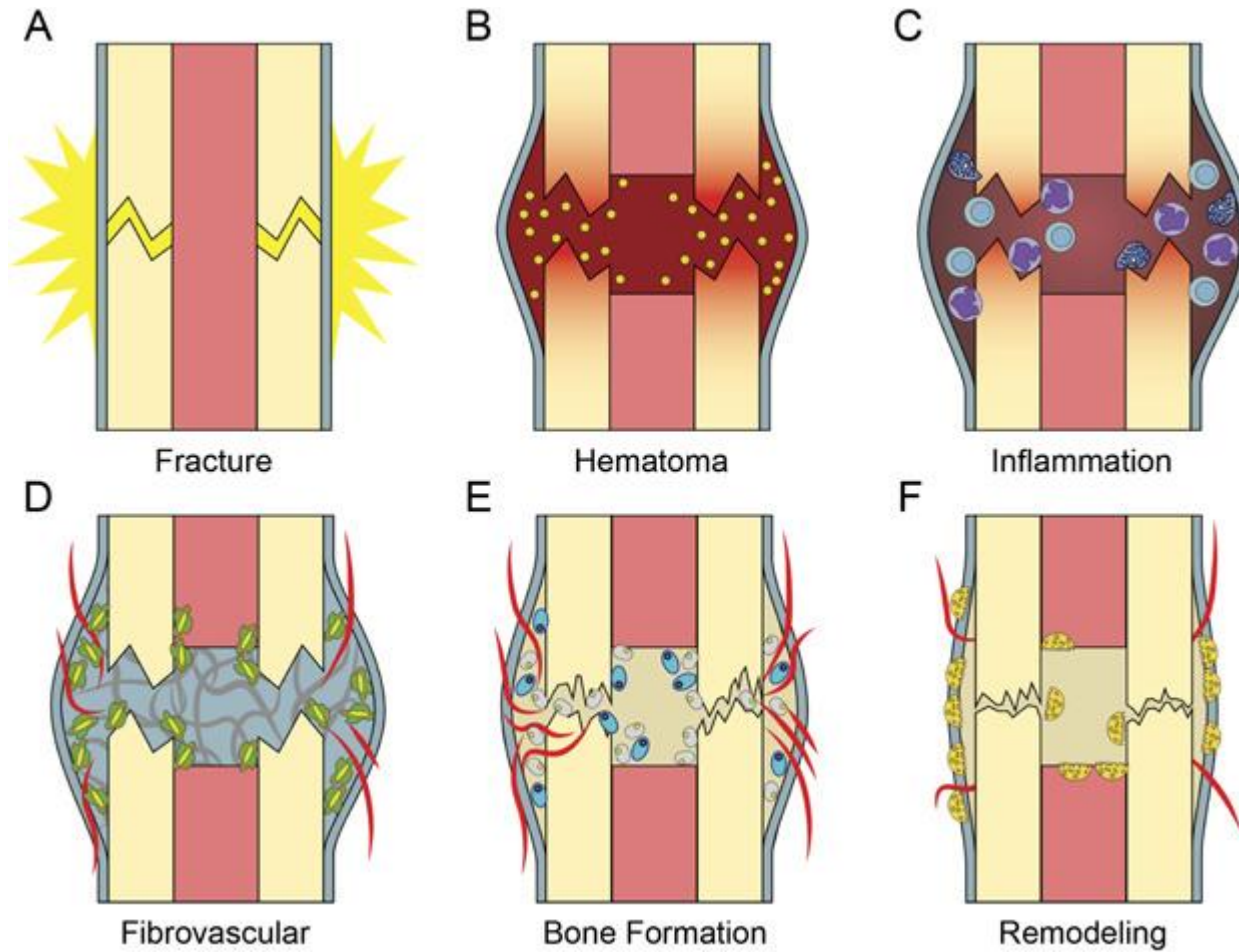
- Salter-Harris classification
- Metaphysis is above the growth plate
- Epiphysis is below the growth plate
- Type I Straight across the metaphysis
- Type II Above the metaphysis
- Type III Below the metaphysis
- Type IV Through the metaphysis
- Type V Erasure or crush of growth plate

Fracture

- Certain growth plates contain fibrocartilage instead of columnar cartilage (tibial tuberosity, inferior pole of the patella).
- These areas are tensile-responsive apophyseal centers and have unique failure patterns that result from the intersection of the ossification center and the fibrocartilage.
- These apophyseal centers are prone to overuse traction avulsions, termed apophysitis.
- Apophyseal injuries do not interfere with growth and are mostly self-limited injuries in adolescent athletes.

Fracture repair

- Initial inflammatory phase is dominated by vascular events.
- Following a fracture, a hematoma forms.
- Subsequently, reabsorption occurs of the 1 to 2 mm of bone at the fracture edges that have lost their blood supply.
- It is this bone reabsorption that makes fracture lines become radiographically distinct 5 to 10 days after injury.
- Next, multipotent cells are transformed into osteoprogenitor cells, which begin to form new bone.



Fracture healing is temporally-defined process.

Fracture repair

- In the reparative phase, new blood vessels develop from outside the bone that supply nutrients to the cartilage which begins to form across the fracture site.
- Nearly complete immobilization is desirable during both the inflammatory phase and the early reparative phase to allow for the growth of these new vessels.
- Once neovascularization is complete, progressive loading and motion of the fracture site are desirable to augment callus formation.

Fracture repair

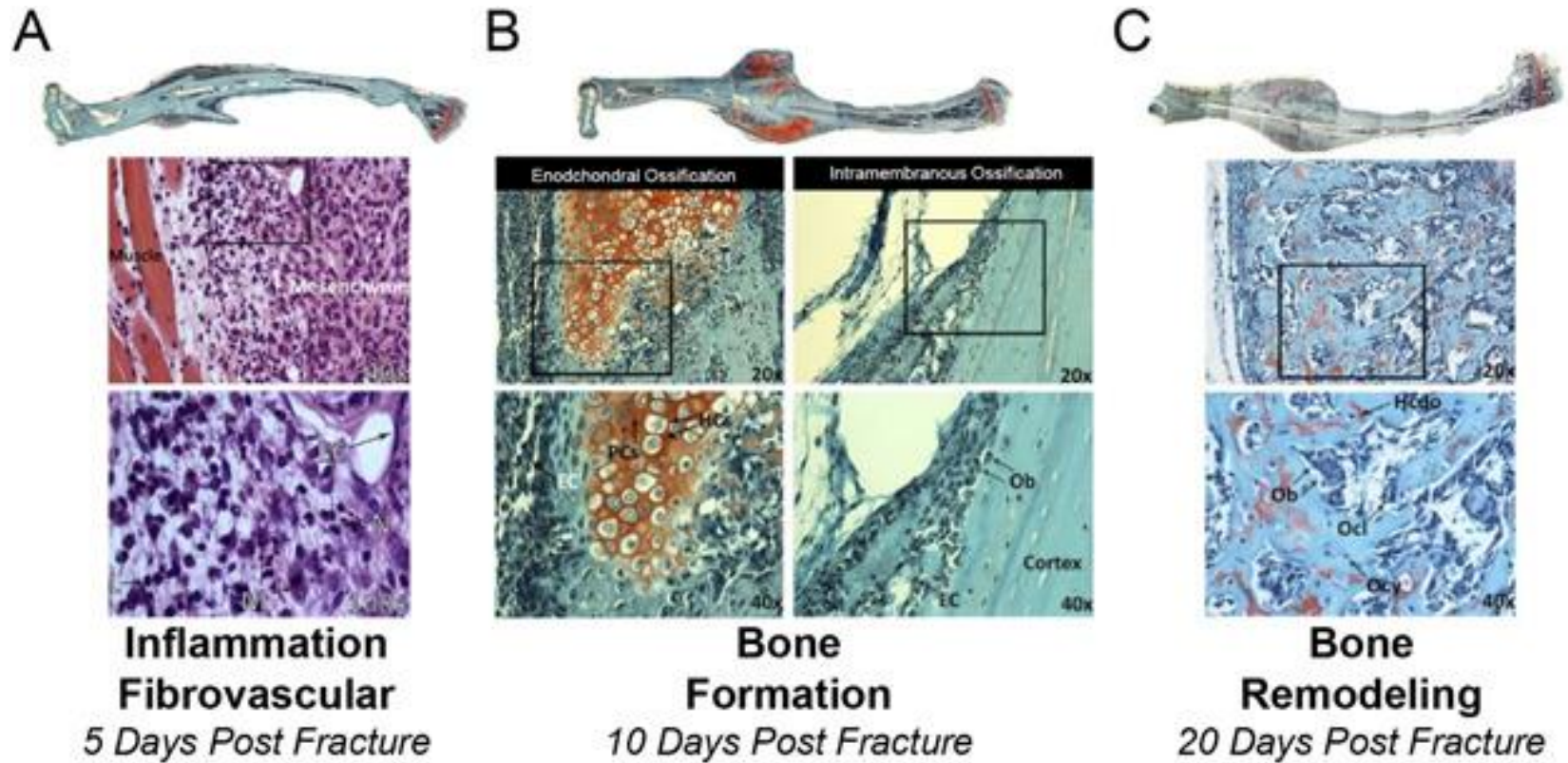
- Callus typically forms as a collar of new, endochondral bone around the fractured area.
- Callus is initially highly cartilaginous, but hardens as mineralization and endochondral calcification occur during the remodeling phase.
- Late in the reparative phase, clinical union of the fracture occurs.
- Clinical union occurs when the fractured bone does not shift on clinical examination, the fracture site is non-tender, and the patient can use the injured limb without significant pain.

Fracture repair

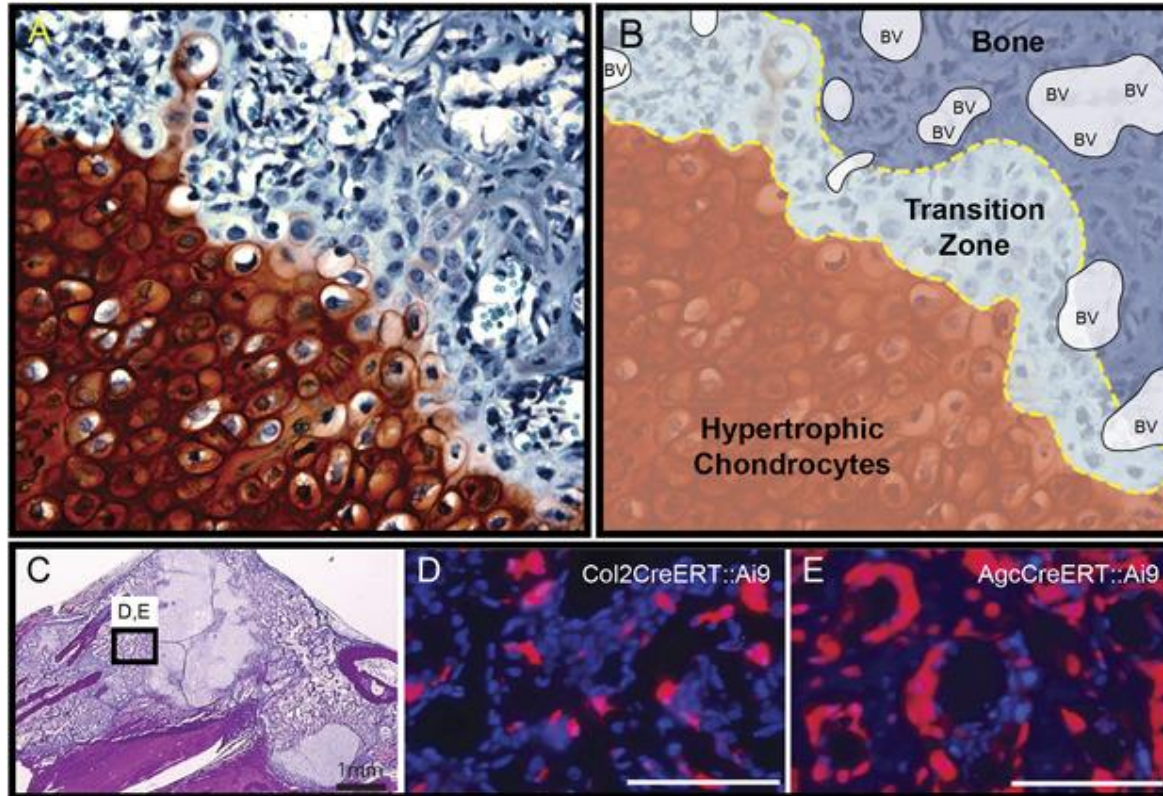
- Because the initial callus is cartilaginous, clinical union may occur before evidence of radiographic union is appreciable on x-rays.
- Clinical union classically marks the end of the reparative phase of fracture healing.
- In the remodeling phase, the endochondral callus becomes completely ossified and the bone undergoes structural remodeling.

Fracture repair

- The process of remodeling occurs quickly in young children, who remodel their entire skeleton every year.
- By late childhood, the rate of skeletal remodeling is approximately 10 percent per year and continues near this level throughout life.



(A) undifferentiated mesenchymal cells are present in the callus and areas of inflammation remain (B) 10 days post-fracture there is both endochondral ossification (red staining, safranin-o stains cartilage) and intramembranous bone formation occurring. (C). An extensive network of primary bone has formed and endochondral ossification is complete.



Hypertrophic chondrocytes develop into osteoblasts and osteocytes

Acute response

- TNF- α concentration has been shown to peak at 24h and to return to baseline within 72h post trauma.
- During this time-frame TNF- α is expressed by macrophages and other inflammatory cells, and it is believed to mediate an effect by inducing secondary inflammatory signals, and act as a chemotactic agent to recruit necessary cells.
- TNF- α also induces osteogenic differentiation of mesenchymal stem cells (MSC).

Acute response

- These effects are mediated by activation of the two receptors TNFR1 and TNFR2 which are expressed on both osteoblasts and osteoclasts.
- However, TNFR1 is always expressed in bone whereas TNFR2 is only expressed following injury

Acute response

- IL-1 and IL-6 are believed to be most important cytokines for fracture healing.
- IL-1 expression overlaps with that of TNF- α with a biphasic mode
- It is produced by macrophages in the acute phase of inflammation and induces production of IL-6 in osteoblasts
- Promotes the production of the primary cartilaginous callus, and also promotes angiogenesis at the injured site by activating either of its two receptors

Acute response

- IL-6 is only produced during the acute phase and stimulates angiogenesis, vascular endothelial growth factor (VEGF) production, and the differentiation of osteoblasts and osteoclasts.
- BMP-2 is essential for bone repair
- BMP-7 may play a more important role in the recruitment of progenitor cells.

Acute response

- Stromal cell-derived factor-1 (SDF-1) and its G-protein-coupled receptor CXCR-4 form an axis (SDF-1/CXCR-4) that is a key regulator of recruiting and homing specific MSCs to the site of trauma.
- SDF-1 expression is increased at the fracture site, especially in the periosteum at the edges of the fracture.
- SDF-1 has a specific role in recruiting CXCR-4 expressing MSCs to the injured site during endochondral fracture healing

Acute response

- Hypoxia inducible factor-1 α (HIF-1 α) induces the production of VEGF in the revascularization process
- Hypoxic gradients regulate MSC progenitor cell trafficking by HIF-1

Healing of bone fracture

- Following the initial trauma, bone heals by either direct intramembranous or indirect fracture healing, which consists of both intramembranous and endochondral bone formation.
- The most common pathway is indirect healing as direct bone healing requires an anatomical reduction and rigidly stable conditions.
- When such conditions are achieved, the direct healing cascade allows the bone structure to immediately regenerate anatomical lamellar bone and the Haversian systems without any remodeling steps necessary.

Contact healing

- Bone on one side of the cortex must unite with bone on the other side of the cortex to re-establish mechanical continuity.
- Under these conditions, osteoclasts which cross the fracture line, generate longitudinal cavities.
- These cavities are later filled by bone produced by osteoblasts residing at the rear of the juncture.
- This results in the simultaneous generation of a bony union and the restoration of Haversian systems formed in an axial direction.

Contact healing

- The re-established Haversian systems allow for penetration of blood vessels carrying osteoblastic precursors.
- The bridging osteons later mature by direct remodeling into lamellar bone resulting in fracture healing without the formation of periosteal callus.

Gap healing

- Bony union and Haversian remodeling do not occur simultaneously.
- It occurs if stable conditions and an anatomical reduction are achieved, although the gap must be less than 1 mm.
- In this process the fracture site is primarily filled by lamellar bone oriented perpendicular to the long axis, requiring a secondary osteonal reconstruction.

Gap healing

- The primary bone structure is then gradually replaced by longitudinal revascularized osteons carrying osteoprogenitor cells which differentiate into osteoblasts and produce lamellar bone on each surface of the gap.
- This lamellar bone, however, is laid down perpendicular to the long axis and is mechanically weak.

Gap healing

- This initial process takes approximately 3 and 8 weeks, after which a secondary remodeling resembling the contact healing cascade takes place.
- Although not as extensive as endochondral remodeling, this phase is necessary in order to fully restore the anatomical and biomechanical properties of the bone.

Healing of bone fracture

- Indirect healing
- Involves an acute inflammatory response and the recruitment of mesenchymal stem cells in order to generate a primary cartilaginous callus.
- This primary callus later undergoes revascularization and calcification, and is finally remodeled to fully restore a normal bone structure.

Healing phase

- Following the formation of the primary hematoma, a fibrin-rich granulation tissue forms.
- Within this tissue, endochondral formation occurs in between the fracture ends, and external to periosteal sites.
- These regions are mechanically less stable
- The cartilaginous tissue forms a soft callus which gives the fracture a stable structure.

Healing phase

- Peak of soft callus formation occurs 7–9 days post trauma with a peak in both type II procollagen and proteoglycan core protein extracellular markers.
- At the same time, an intramembranous ossification response occurs subperiostally directly adjacent to the distal and proximal ends of the fracture, generating a hard callus.
- It is the final bridging of this central hard callus that ultimately provides the fracture with a semi-rigid structure which allows weight bearing

Healing phase

- The WNT-family is thought to regulate the differentiation of pluripotent MSCs into the osteoblastic lineage, and, at later stages of development, to positively regulate osteoblastic bone formation.
- As fracture callus chondrocytes proliferate, they become hypertrophic and the extracellular matrix becomes calcified.
- Both osteoblasts and hypertrophic chondrocytes express high levels of VEGF
- Promote the invasion of blood vessels and transforming the avascular cartilaginous matrix into a vascularized osseous tissue.

Healing phase

- A cascade orchestrated primarily by macrophage colony-stimulating factor (M-CSF), RANKL, osteoprotegerin (OPG) and TNF- α initiates the resorption of this mineralized cartilage.
- During this process M-CSF, RANKL and OPG are also thought to help recruit bone cells and osteoclasts to form woven bone.
- TNF- α further promotes the recruitment MSC with osteogenic potential
- Initiates chondrocyte apoptosis.

Healing phase

- Mitochondria accumulate calcium-containing granules created in the hypoxic fracture environment.
- After elaboration into the cytoplasm of fracture callus chondrocytes, calcium granules are transported into the extracellular matrix where they precipitate with phosphate and form initial mineral deposits.
- These deposits of calcium and phosphate become the nidus for homogeneous nucleation and the formation of apatite crystals.
- The peak of the hard callus formation is usually reached by day 14

Remodeling

- The remodeling process is carried out by a balance of hard callus resorption by osteoclasts, and lamellar bone deposition by osteoblasts.
- Although the process is initiated as early as 3–4 weeks in the remodeling may take years to be completed to achieve a fully regenerated bone
- The process of remodeling occurs quickly in young children, who remodel their entire skeleton every year.
- By late childhood, the rate of skeletal remodeling is approximately 10 percent per year and continues near this level throughout life

Remodeling

- Bone remodeling has been shown to be a result of production of electrical polarity created when pressure is applied in a crystalline environment.
- This is achieved when axial loading of long bones occurs, creating one electropositive convex surface, and one electronegative concave surface, activating osteoclastic and osteoblastic activity respectively.
- By these actions the external callus is gradually replaced by a lamellar bone structure, whereas the internal callus remodeling re-establishes a medullar cavity characteristic of a diaphyseal bone.

Reparative phase

- Because the initial callus is cartilaginous, clinical union may occur before evidence of radiographic union is appreciable on x-rays.
- Clinical union classically marks the end of the reparative phase of fracture healing.
- Clinical union of the fracture occurs.
- Clinical union occurs when the fractured bone does not shift on clinical examination, the fracture site is non-tender, and the patient can use the injured limb without significant pain.

Maxillofacial fractures

- Maxillofacial injuries rarely cause immediate threat to life.
- Treatment of the fracture involves maintenance of function, particularly masticatory function.
- CT scanning required to evaluate injuries.
- The angle of the mandible is the most common area of mandibular fracture.
- The mandible is displaced as the pterygo-masseter sling causes the proximal fragment to ride superiorly.

Maxillofacial fractures

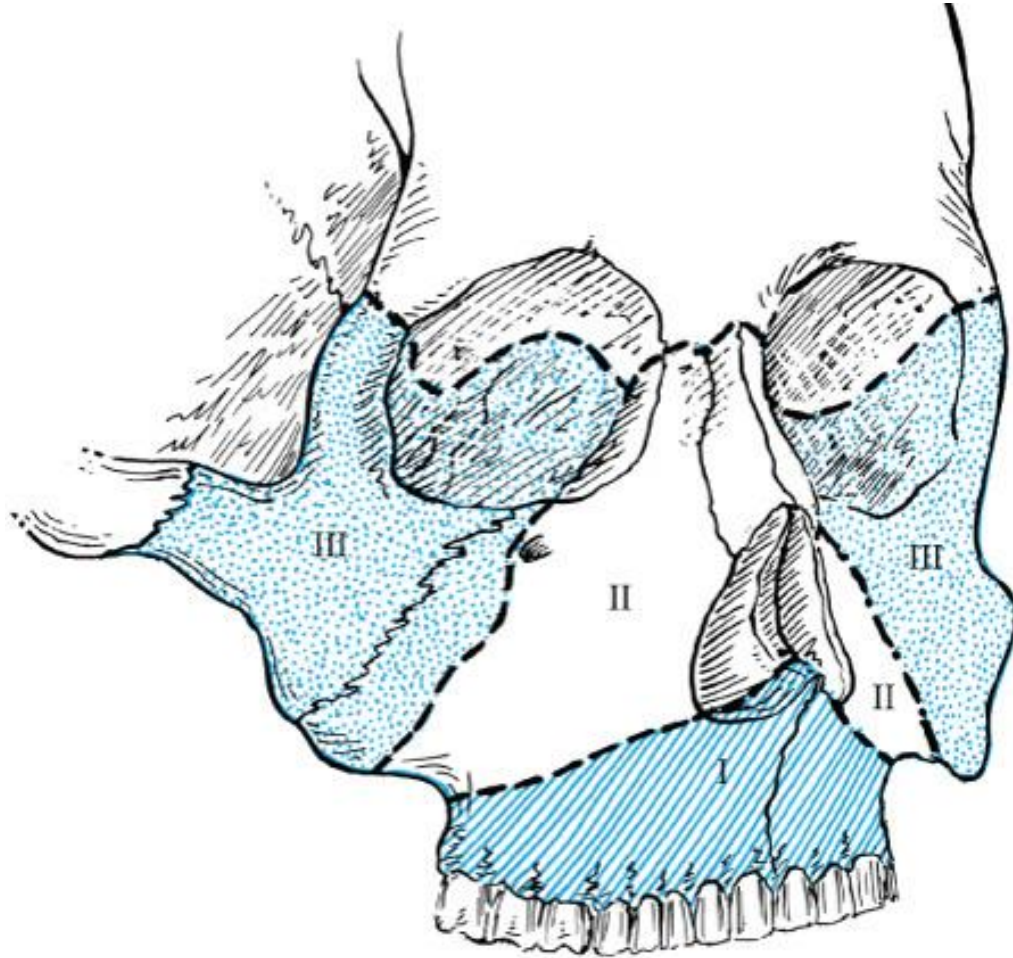
- Unilateral condylar fracture will cause the jaw to deviate toward the side of fracture on maximum opening.
- Bilateral subcondylar fracture presents with an anterior bite and occlusion on the posterior molars. There is no contact with incisor teeth.
- An alveolar ridge fracture is usually associated with a loss in the anterior or incisor region.
- (Mandibular fracture as well often involves the alveolar ridge.)

Midfacial fractures

- Isolated fractures of the zygomatic arch are uncommon.
- The zygoma may be depressed, impinge on the coronoid process of the mandible, and limit the jaw opening.
- A pure blowout fracture is caused by direct ocular trauma.
- The thin orbital floor is forced into the maxillary antrum.
- Entrapment of extra-ocular muscles may occur.

LaFort facial fracture

Illustration of the fracture lines of LeFort I (alveolar), LeFort II (zygomatic maxillary complex), and LeFort III (cranial facial dysostosis) fractures.



Source: Knoop KJ, Stack LB, Storrow AB, Thurman RJ: *The Atlas of Emergency Medicine, 3rd Edition*: <http://www.accessmedicine.com>
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LeFort facial fractures

- In all LeFort fractures, the mandible is free-floating.
- In type I, a horizontal fracture line runs from the lateral nasal apertures, along the lateral wall of the maxillary sinuses bilaterally, and across the pterygo-maxillary tissue to the lateral pterygoid plate.
- The body of the maxilla is separated from the base of the skull above the level of the palate and below the attachment of the zygomatic process.

LeFort facial fractures

- The dental arch is easily mobilized.
- Displacement is dependent upon muscular pull.
- The fracture may be associated with damage to CN V_2 as well as CN VII.
- Labial and maxillary arteries may be involved.

LeFort facial fracture

- A type II or pyramidal fracture consists of vertical fractures through the facial aspects of the maxilla extending upwards to the nasal and ethmoid bones.
- The fractures extend through the maxillary sinuses and the infraorbital rims bilaterally across the bridge of the nose.
- CSF rhinorrhea may be present.

LeFort facial fracture

- There is associated damage to CN V₂ as well as CN VII.
- The transverse facial artery may also be involved.
- Frequently the central portion of the orbital floor will also fracture as part of the zygomatic-maxillary complex.
- Entrapment of extra-ocular muscles may occur.

LeFort facial fracture

- A type III fracture extends through the frontozygomatic suture lines bilaterally, across the orbits, and through the base of the nose and the ethmoid region.
- The lateral rim of the orbit is separated and the infra-orbital rim may also be fractured.
- A pyramidal or a horizontal fracture may also be present.

LeFort facial fracture

- The face is depressed.
- The midface and zygoma move concurrently.
- CSF rhinorrhea is common.
- There may be significant damage to CN V₂ as well as CN VII.
- The transverse facial artery and the parotid duct may also be involved.

Abuse

- Any fracture in a child younger than one year of age or any lower extremity fracture in a non-ambulatory child should raise suspicion of abuse.
- Specific fracture patterns of concern include:
 - Posterior rib fractures
 - Metaphyseal lesions (bucket-handle or corner fractures)
 - Bilateral long bone fractures
 - Complex skull fractures
 - Spinous process fractures.

Abuse

- In addition, a repeat fracture occurring at an unusual location for repeat injury is suspicious for child abuse, and careful history and social assessment should be performed.
- Elder abuse should be considered in all elderly patients with falls and fractures.

Peripheral nerve injury

Nerve	Motor function	Sensory function	Scenario
Radial	Wrist drop (extension)	Back of forearm and hand (digits 1-3)	Humeral fracture
Ulnar	Claw hand (finger abduction)	Front and back of hand (digits 4-5)	Elbow dislocation or fracture
Median	Pronation, thumb opposition	Palmar surface of hand (digits 1-3)	Carpal tunnel syndrome; humeral fracture
Axillary	Abduction, lateral rotation of shoulder	Lateral shoulder	Humeral fracture or dislocation
Peroneal	Foot drop (dorsiflexion, eversion)	Dorsal foot and lateral leg	Knee dislocation; fibular fracture

Fat Embolism

