INTRODUCTION

Tibial fractures are the third most common pediatric long bone fractures, after fractures of the forearm and femur. [1,2] Approximately 50% of pediatric tibial fractures occur in the distal third of the tibia. This is followed by fractures of the tibia midshaft (39%), and least commonly, the proximal third of the tibia is involved. [1,3] Seventy percent of pediatric tibial fractures occur as an isolated injury. More than half of the combined tibia/fibula fractures are secondary to motor vehicle accidents. [1,4]

Patients are presented with pain, altered weight bearing status, and swelling or deformity, physical examination will usually demonstrate point of tenderness at the fracture site, there will frequently be ecchymosis, edema and deformity. During physical examination, careful neurologic, vascular, and soft-tissue examination should be performed to evaluate for compartment syndrome which requires emergent fasciotomies. [5]

Surgical stabilization of pediatric tibial fractures may be indicated for (a) children in whom acceptable positioning is not maintained after closed reduction; (b) children who are ten years of age and older, (c) children who have selected open fractures, spasticity due to head injury or cerebral palsy; (f) Children who have multiple long-bone fractures or multiple-system injuries; and (g) children who have concomitant severe soft − tissue injuries. [1, 6-8]

Tibial fractures requiring surgical stabilization are treated differently in children than in adults. Locked intramedullary nails are not used in children because of the risk of physeal injury. Treatment options include external fixation, elastic stable intramedullary nailing, plate fixation and transfixation pins and casts. [8,9-15]

The use of external fixation has been well described. The result have been satisfactory, but have been associated with complications including infection, delayed union, refracture, malalignment and joint stiffness. [16]

Treatment of pediatric fractures dramatically changed in 1982, when Métaizeau and the team from Nancy, France, developed the technique of flexible intramedullary pinning using titanium pins. [17]

Intramedullary nails function as an internal splint and provide three-points fixation to maintain bony alignment. Smaller incisions, shorter surgical times, and minimal dissection at the fracture site have been discribed as advantages of nailing over plating in children. In addition intramedullary fixation provides early motion, easy hardware removal and excellent cosmesis. [18,19]

The advantages of Elastic Stable Intramedullary Nailing (ESIN) also include low infection rates and shorter hospital stay. Possible complications such as malunion and refracture remain as they would with conservative treatment. The nails can be removed once the fracture has united, depending on the patient and surgeon preference. [20]

REVIEW OF LITERATURE

Anatomy of the tibia

Bony structure (Fig. 1,2)

The tibia is the main weight-bearing bone in the leg. Its shaft is triangular in cross section presenting three borders and three surfaces. Between the anterior and the medial borders is the medial surface which is subcutaneous. The anterior border is prominent forming the chin. At the junction of the upper end of the tibia with the anterior border is the tibial tuberosity to which is attached the ligamentum patellae. The anterior border becomes rounded as it extends down where it becomes continuous with the medial malleolus. Its lateral or interosseous border gives attachment to the interosseous membrane. The posterior surface of the shaft shows an oblique line, the soleal line, below the soleal line is a vertical ridge which passes downwards dividing the posterior surface into medial and lateral areas. [21-26] (Fig. 1,2)

In addition, the shaft of the tibia has an expanded upper end and a smaller lower end. At the upper end are the medial and the lateral condyles which articulate with the medial and the lateral condyles of the femur. The lateral condyle possesses on its lateral aspect a facet for the head of the fibula. The medial condyle shows a groove on its medial aspect for the insertion of the semimembranosus muscle. [21-26]

The lower end of the tibia is slightly expanded and on its inferior aspect shows a plafond shaped articular surface for the talus. The lower end is prolonged medially to form the medial malleolus. The lateral surface of the medial malleolus articulates with the talus. The lower end of the tibia shows a rough depression on its lateral surface for the articulation with the fibula. [21-26]

The fibula takes no part in articulation of the knee joint but below it forms the ankle joint. It has an upper end, shaft and a lower end. [21-25]

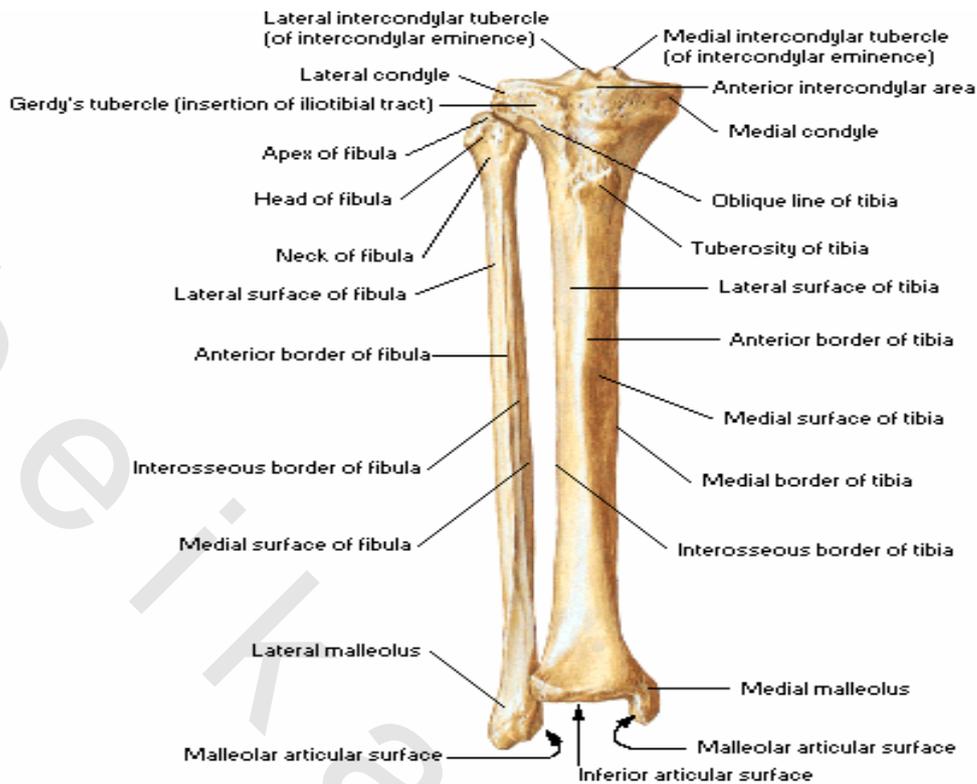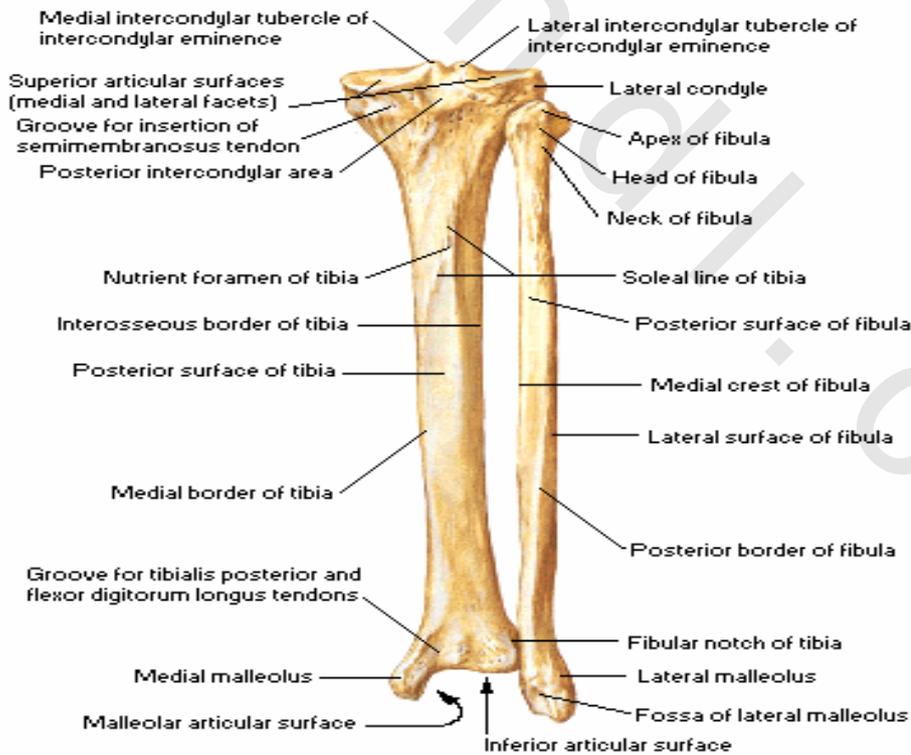**Fig. [1]:** Anterior view of the tibia and fibula.[25]

Fig. [2]: Posterior view of the tibia and fibula.[25]

Ossification of the tibia (Fig. 3)

The tibia ossifies from three centres, one in the shaft and one in each epiphysis. Ossification begins in midshaft at about the seventh intrauterine week. The proximal epiphyseal centre is usually present at birth. At 10 years a thin anterior process from the centre descends to form the smooth part of the tibial tuberosity. [27]

A separate centre for the tuberosity may appear at about the twelveth year and soon fuses with the epiphysis. Distal strata of the epiphyseal plate are composed of dense collagenous tissue in which the fibres are aligned with the patellar tendon. [27]

The proximal epiphysis fuses in the sixteenth year in females and the eighteenth in males. [27]

The distal epiphyseal centre appears early in the first year and joins the shaft in about the fifteenth year in females and the seventeenth in males. The medial malleolus is an extension from the distal epiphysis and starts to ossify in the seventh year: it may have its own separate centre.[27]
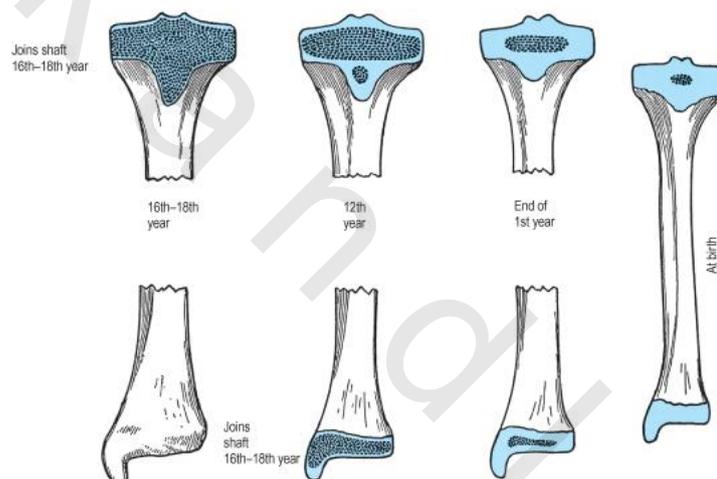

Fig. [3]: Stages in the ossification of the tibia (not to scale). Blue =unossified (cartilaginous) regions [27]

The muscles of the leg [28] (Fig. 4)

Muscle Origins and Insertions on the Tibia

Semimembranosus	Inserts on inner tuberosity of the proximal tibia
Tibialis anterior, extensor digitorum longus, biceps femoris	Attach to the lateral condyle of the tibia
Sartorius, gracilis, semitendinosus	Insert on the proximal medial surface of the tibial metaphysis
Tibialis anterior	Arises on the lateral surface of the tibial diaphysis
Popliteus, soleus, flexor digitorum longus, tibialis posterior	Attach to the posterior diaphysis of the tibia
Patellar tendon	Inserts into the tibial tubercle
Tensor fascia lata	Attaches to Gerdy's tubercle, the lateral aspect of the proximal tibial metaphysis
Secondary slip of fascia lata	Occasionally inserts into the tibial tubercle

Muscle Origins and Insertions on the Fibula

|	
Soleus, flexor hallucis longus	Arise from the posterior aspect of the diaphysis
peroneus longus, peroneus brevis	Arise from the lateral aspect of the fibular diaphysis
Biceps femoris, soleus, peroneus longus	Attach to the head of the fibula
Extensor digitorum longus, fibularis tertius, extensor hallucis longus	Attach to the anterior surface of the fibular shaft
Tibialis posterior	Arises from the medial aspect of the fibular diaphysis
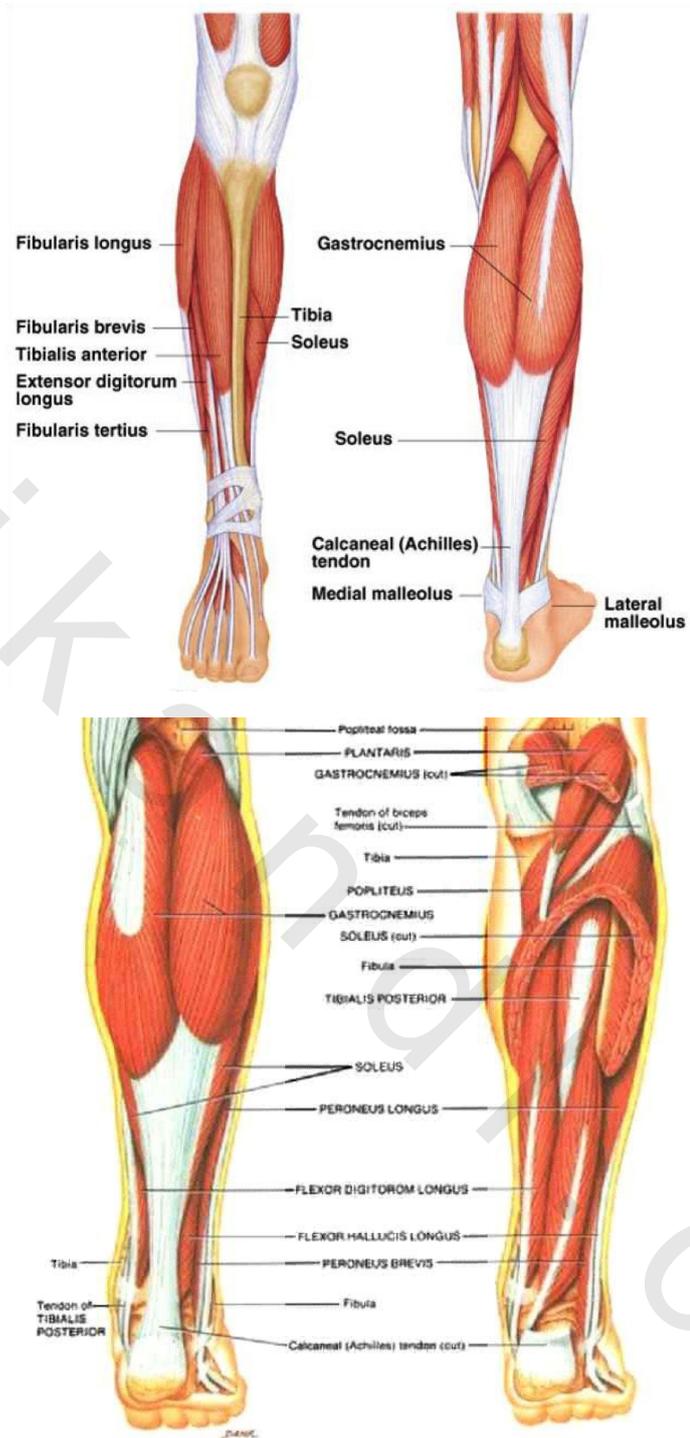
Fig. [4]: Muscles of the leg.[28]

Blood supply of the leg. (Fig. 5)

The popliteal artery descends vertically between the condyles of the femur and passes between the two heads of the gastrocnemius muscle. It ends at the distal border of the popliteus muscle, where it divides into the anterior and posterior tibial arteries. The anterior tibial artery passes between the tibia and the fibula over the proximal aspect of the intraosseous membrane. The posterior tibial artery divides several centimeters distal to this point, giving rise to the peroneal artery. [28]

Blood supply to the tibia itself derived from nutrient artery and periosteal vessels:

Posterior tibial artery (nutrient artery):

It enters into the posterior tibial cortex, distal to the soleal line at the middle 1/3, enters posterolateral cortex of the tibia at the origin of the soleus muscle, the artery may traverse distance of 5.5 cm before entering its oblique nutrient canal, it divides into three ascending branches and a single descending branch, which gives off smaller brances to the endosteal surface, so it provides the endosteal blood supply to the inner tibial cortex.

Anterior tibial artery (periosteal vessels):

It passes through hiatus in upper interosseous membrane, periosteum has abundant blood supply from anterior tibial artery branches as it courses down the interosseous membrane.
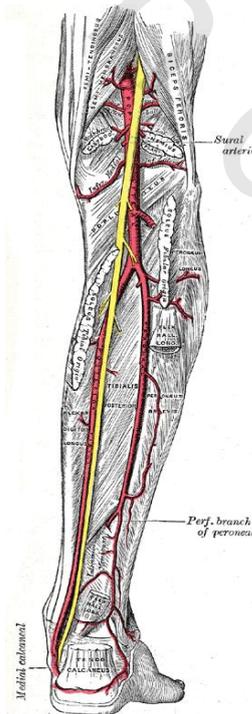

Fig. [5]: Blood supply to the tibia [28]

Nerve supply to the leg. (Fig. 6)

The posterior tibial nerve runs adjacent and posterior to the popliteal artery in the popliteal fossa. The common peroneal nerve passes around the proximal neck of the fibula. It divides into the deep and superficial branches, passing into the anterior and the lateral compartments of the lower leg, respectively. Each branch innervates the muscles within its compartment. The deep peroneal nerve provides sensation to the first web space. The superficial branch is responsible for sensation across the dorsal surface of the foot. [28]
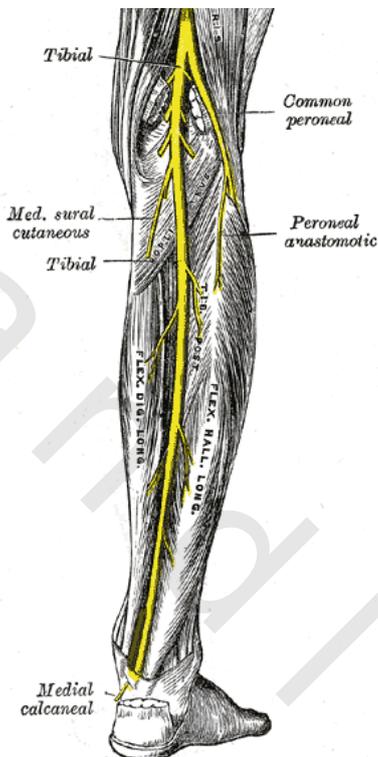
Fig. [6]: Nerve supply tothe tibia. [28]

Fascial Compartments (Fig. 7)

The lower leg has four fascial compartments. The anterior compartment contains the extensor digitorum longus, the extensor hallucis longus, and the tibialis anterior muscles; the anterior tibial artery and deep peroneal nerve run in this compartment. The lateral compartment contains the peroneus longus and brevis muscles. The superficial peroneal nerve runs through this compartment. The superficial posterior compartment contains the soleus and gastrocnemius muscles. The deep posterior compartment contains the flexor digitorum longus, the flexor hallucis longus, and the tibialis posterior muscles. The posterior tibial artery, peroneal artery, and posterior tibial nerve run in this compartment[28].
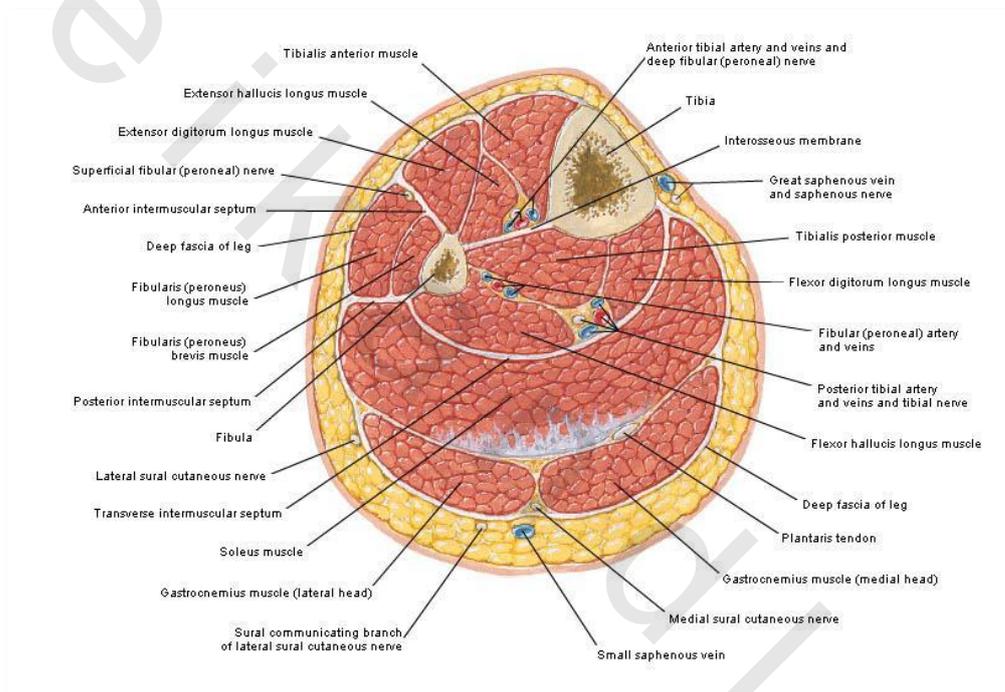

Fig. [7]: Fascial compartments of the leg. [28]

Vascular changes during fracture healing

When a fracture is displaced, the medullary vessels are disrupted and the periosteal vessels predominate as the vascular supply to the fracture site during early healing. In response to fracture, the periosteal vessels proliferate, while the endosteal circulation is restored much later. The significance of periosteal blood flow in healing also emphasizes the importance of avoiding periosteal stripping. The early external callus receives its blood supply from the periosteal circulation through vessels which are oriented perpendicular to the cortical surface.[29]

As healing progresses, the medullary circulation is re-established across the fracture site. This circulation continues to hypertrophy while the temporarily increased periosteal circulation recedes and soon, the medullary system provides osseous bridging. Finally, as bone remodelling advances, the normal vascular pattern is re-established with the medullary vessels meeting the periosteal vessels in the outer cortical layers. [29]

Biology of fracture healing in long bones

The primary goal of fracture healing is to re-establish the structural integrity of the injured bone and restore the function of the affected limb. Interruption of the continuity of long bones is followed by definite sequences aiming at bridging the defect. A basic understanding of this process is necessary for handling of fractures. Fracture healing can be divided into the following phases:[30]

1. Acute phase:

When a long bone is fractured, the soft tissue envelope around the fracture site including the periosteum and surrounding muscles is torn in varying degrees. Numerous blood vessels that cross the fracture site are torn leading to haematoma formation at the fracture site.[31]

The following events occur for a variable distance on each side of the fracture line:

A. Blood flow ceases in the Haversian systems back to the site where they anastomose with functioning blood vessels of other Haversian systems at the fracture ends.[30]

B. Necrosis of the bone adjacent to the fracture line.[32]

C. Necrosis of the periosteum, marrow and other surrounding soft tissues around the fracture site.[32]

Infiltrating inflammatory cells resorb necrotic ends of fractured bone back to viable tissue capable of healing. Inflammatory cells are a source of potent molecular signals that recruit cells to initiate fracture repair. Interleukins 1 and 6 (released by polynucleated inflammatory cells) are important molecules in the inflammatory phase. Transforming growth factor-beta and platelet-derived growth factor (released by degranulating platelets) cause local mesenchymal cells to proliferate and differentiate into osteoblastic cell types. The inflammatory response is a potent, non-specific response that is capable of initiating healing. However, if left unchecked, the inflammatory response is also capable of producing local injury. Therefore, it is self-limiting and subsides after several days. The mechanisms that diminish the inflammatory response are not well understood.[33]

2. Reparative phase:

The diaphyseal fracture repair process starts with primary callus response which leads to formation of the periosteal callus during the first week. It is a very fundamental reaction of bone to injury. A well-developed collar of callus is formed in each fragment. The activity of these collars is of limited duration. If contact between the bone fragments is not made, soon the callus will disappear (it is a once only phenomenon) and this reaction will never become reactivated. It seems that the cells responsible for this activity arise from the bony tissues themselves.[32,34]

Later, formation of the bridging callus in the fracture gap occurs between the first and third week.[32] A new tissue; callus, develops around and between the fragments forming a bridge by which the fragments are initially united. Callus that develops around the outer aspect of the opposing ends of the bone fragments is named external callus, and that which forms between the bone ends is named medullary callus.[30] If the fracture is treated by extreme mechanical rigidity healing will occur by a process of primary bone union (direct bone healing) which is dependent on the activity of medullary cells and direct osteonal penetration with no external callus formation and no intermittent cartilaginous stage.[32,34] If the fracture is held less rigidly, e.g. conservative treatment using a brace, healing will occur by a process of secondary bone healing. It involves healing processes within the bone marrow, periosteum and the soft tissues surrounding the bone. An intermittent cartilaginous or fibrocartilaginous stage precedes bone formation.[33]

Direct bone healing: (Fig. 8)

Direct bone healing occurs primarily after the fractured ends of cortical bone are directly reduced and rigidly opposed under compression. Rigid compression fixation of the opposed cortical ends creates a mechanical environment with minimal interfragmentary motion. Once this mechanical environment is established, an elaborate biological process occurs, eventually uniting the fracture. Perfect apposition of the fractured cortices is not achieved on a microscopic basis, and the cortical ends are opposed with a series of contact points and gaps. This establishes a sequential healing process, with gap healing occurring initially, followed by contact healing.[33]

Rigid compression plating results in regions of cortical bone apposition (red arrows) interspersed with regions of small gaps (blue arrows). Direct appositional bone healing must occur across the gaps before contact healing can proceed.
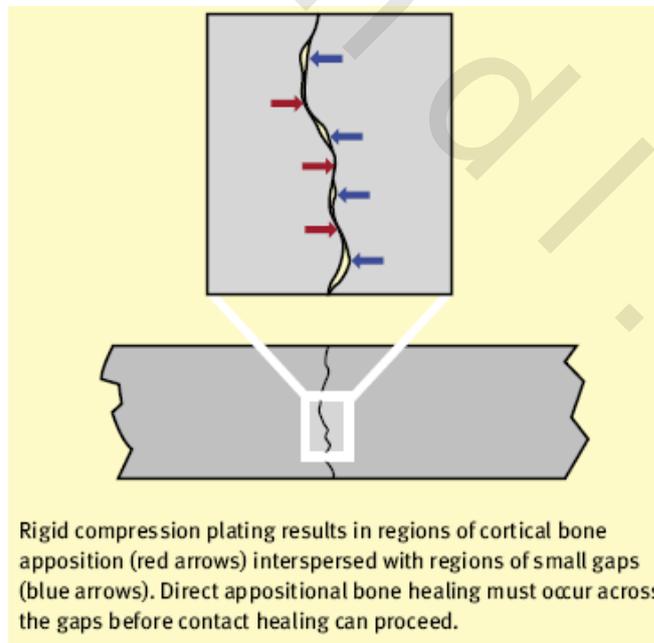
Fig. [8]: Direct bone healing.[33]

Gap healing is primarily characterized by direct bone formation between the ends of the bone bracketing the gap. This is mainly appositional bone growth, not relying on preceding osteoclastic resorption. Smaller gaps, approximately 200 micrometers or less (about the width of an osteon, previously known as a 'Haversian system'), fill rapidly with mature lamellar bone. Larger gaps fill more slowly, filling with primitive woven bone.[33]

Contact healing: gaps that have filled with primitive woven bone during the initial phase of gap healing require remodelling to achieve pre-fracture strength. This is achieved by contact healing, which occurs in a series of events controlled by the basic remodelling units. Basic remodelling units are activated by the fracture. They facilitate bone resorption, and then direct bone formation in the tunnels spanning the fracture. Basic remodelling units are constantly active at a low level in uninjured bone, regulating ongoing bony remodelling. After a fracture, activation of basic remodelling units increases dramatically.[33]

After a lag phase of several weeks, the basic remodelling unit forms a cutting cone, with osteoclasts on the leading surface of the cone (Fig. 9). The cutting cones burrow through the fracture cortices and across the fracture plane, creating a void. The leading edge of the cutting cone advances approximately 50 μm/day. Osteoblasts line the trailing edges of the cutting cone and begin bone formation on a delayed basis. The osteoblasts produce bone matrix at approximately 1 μm/day, filling the void carved out by the osteoclasts. This healing process takes approximately 3 to 6 months.[33]

Cutting cones are depicted spanning a fracture. The cutting cones appear after gap healing is complete.

Expanded view of a cutting cone. The leading edge of the cutting cone burrows through bone by osteoclastic resorption. Nutrients are brought in by small blood vessels that are also a source of mesenchymal cells (red arrows) that differentiate into osteoblasts (blue arrows).
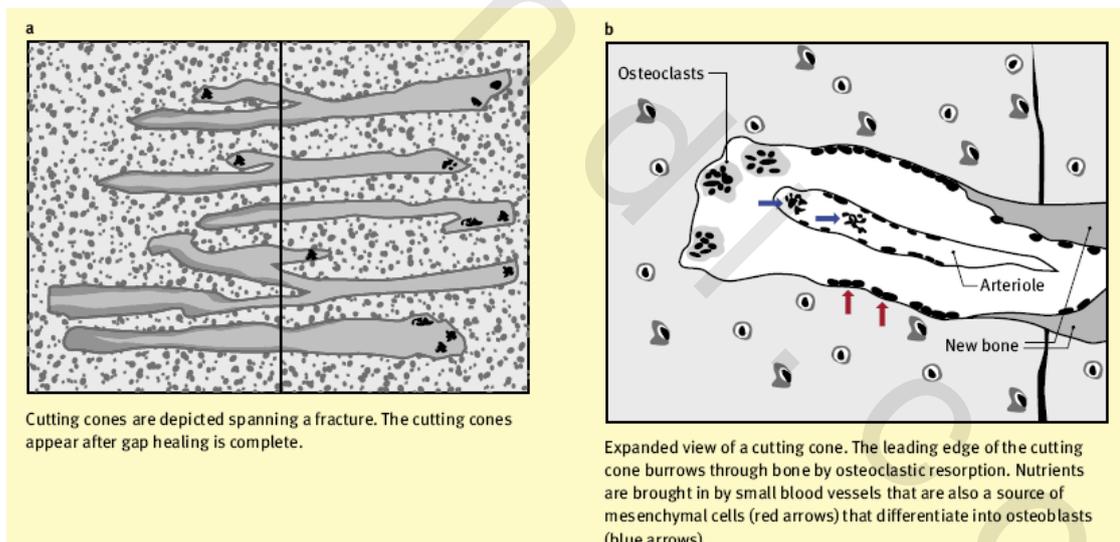
Fig. [9]: Contact healing: cutting cone.[33]

Secondary bone healing: (Fig. 10)

Fractures treated by closed methods, intramedullary fixation, external fixation, and less than rigid plate fixation unite by secondary bone healing. The fracture causes localized bleeding with the formation of a haematoma. Following the initial period of inflammation, secondary healing processes begin within the marrow, cortical bone, periosteum and surrounding soft tissues.[33]

Secondary bone healing employs a combination of direct intramembranous bone formation and enchondral ossification, using processes virtually identical to bone formation processes seen in skeletal growth. Secondary bone healing initially produces an excessive volume of primary woven bone. Following the initial repair, remodelling transforms the primitive woven bone into a more efficient secondary structure, eventually restoring the pre-injury bony architecture. Secondary bone healing is one of very few healing processes in the human that restores injured tissue to its normal state.[33]
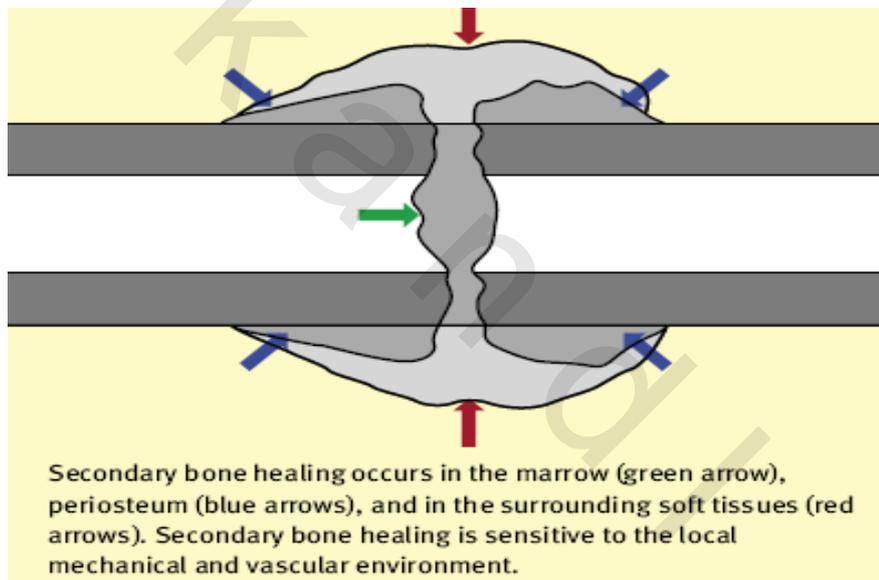
Secondary bone healing occurs in the marrow (green arrow), periosteum (blue arrows), and in the surrounding soft tissues (red arrows). Secondary bone healing is sensitive to the local mechanical and vascular environment.

Fig. [10]: Secondary bone healing.[33]

Secondary bone healing occurs by various pathways in the marrow cavity, in the periosteum, and in the surrounding soft tissues.

Endosteal healing originates in the marrow cavity. The densely packed marrow cells transform into osteoblastic cell types and are responsible for the production of endosteal callus. Unlike healing responses in the surrounding periosteum, soft tissue, and between the cortical ends, marrow healing is independent of mechanical influences.[33]

Periosteal healing begins within days of the injury. Periosteal healing employs both direct intramembranous bone formation and enchondral ossification. Intramembranous bone formation occurs very early after the fracture in the periosteum. This response is observed proximal and distal to the fracture, but not directly adjacent to the fracture plane. This is an extremely rapid response. Early hard callus seen on radiographs results from

intramembranous bone formation in the periosteum. This response provides most of the early fracture stability. Direct periosteal bone formation requires adequate blood supply and low local mechanical strain. These conditions exist in the periosteal region at a distance from the fracture. The periosteal healing response is sensitive to the mechanical environment surrounding the fracture. It is dampened by rigid fixation and enhanced by motion. However, excessive motion from fracture instability can retard and arrest healing.[33]

Healing between the cortical ends – hypoxia and high tissue strain immediately adjacent to the fracture prevent direct intra-membranous bone formation. Therefore, in this hypoxic, unstable region, the body initially forms cartilage and fibrocartilage. Cartilage is more compliant and can withstand the elevated tissue strains encountered near the fracture plane. Chondrocytes are capable of withstanding the hypoxic environment near the fracture gap. Once adequate stability is achieved, chondrogenic tissue calcifies, establishing a 'scaffold' for vascular invasion that promotes classic enchondral ossification.[33]

Healing response in the surrounding soft tissues – like periosteal healing, this healing is sensitive to its mechanical environment, being substantially diminished by rigid immobilization and enhanced by some fracture motion. However, this healing response can be arrested by excessive fracture motion. Most bony healing in the external soft tissues occurs by enchondral ossification. The relative hypoxic conditions (combined with increased motion) drive the enchondral process.[33]

Secondary bone healing following flexible intramedullary nailing:

Flexible intramedullary nails have a considirable success in the treatment of diaphyseal tibial fractures. This biological fixation is not rigid but sufficiently stable against angular, translational and torsional deforming forces (Elastic Stability). Therefore, beneficial interfragmentary micromotions between bone ends stimulate these fractures to unite by secondary bone healing with early formation of exuberant callus. The titanium nails have been distinguished from other flexible nail systems such as Ender nails, made of stainless steel. The latter are believed to be insufficiently elastic for the fractures.[35]

3. Remodelling phase:

Remodelling occurs from the sixth week to one year or longer. It involves a process of simultaneous bone removal and replacement through the respective action of osteoclasts and osteoblasts together. At first, osteoclastic resorption of poorly placed trabeculae occurs and then osteoblasts lay down new struts of bone that correspond to the lines of force. [34]

Wolff's law is a theory developed by the German Anatomist/Surgeon Julius Wolff (1835-1902) in the 19th century that states that "Bone will adapt to the loads it is placed under". If loading on a particular bone increases, the bone will remodel itself over time to become stronger to resist that sort of loading. Bone will be laid down where stresses are the greatest, and thined where loads are diminished.[30,32]

The control mechanism that modulates this cell behaviour is believed to be electrical. When a bone is subjected to stress, electropositivity occurs on the convex side and electronegativity occurs on the concave side. Experimental evidence indicates that regions of electropositivity are associated with osteoclastic activity and regions of electronegativity

with osteoblastic activity. The end result of the remodelling process is a bone that, if it has not returned to its original form, it has been altered in such a way that it may best perform the function demanded of it.[32]

Classification of tibial fractures in children

Classification of tibial shaft fractures

Any classification of tibial fractures is useless unless it helps the surgeon to choose a method of treatment or helps to predict the prognosis.[36]

Basis for classification

Many factors influence the treatment, prognosis and outcome of tibial shaft fractures, while many of these factors are controversial, four of them are generally considered important. [36]

1- The accident mechanism that produced the fracture

Fractures produced by indirect trauma have a better prognosis than those produced by direct trauma. [37]

2- The degree of resulting comminution

The risk of delayed union and non-union in closed and open treatment is increased by comminution. [38]

3- The soft tissue injury

Open fractures have a higher infection rate than closed fractures which increase with increasing severity of the soft tissue injury. [39]

4- The initial and final displacement

Slightly or non-displaced fractures allow more simple treatment than displaced fractures. [40]

Numerous classification systems have been proposed for tibial fractures ranging from the simple stable versus unstable to more detailed alphanumeric systems as proposed by Muller. [41]

Elli's classification

Elli's classified fractures into three basic groups: minor severity, moderate severity and major severity.

- A minor severity fracture is a fracture that is undisplaced, not angulated and has only a minor degree of comminution or a minor open wound.

- Moderate severity is total displacement or angulation with a small degree of comminution or a minor open wound.

- Major severity has complete displacement of the fracture fragments with major comminution or a major open wound. [36]

Weissman's classification

It is based largely on the initial displacement of the fragments. He felt that this was an accurate indication of the severity of the initial trauma and that the degree of initial angulation was of less importance. Although he agreed that both comminution and open wounds were important, he did not use them in his classification.

- Minimal displacement was defined as being one-fifth the width of the tibial shaft in the horizontal plane which is usually associated with slight angulation (10 degrees or less).

- Mild displacement was one-fifth to two-fifth the width of the tibia with angulation of approximately 10 to 30 degrees.

- Marked displacement occurred when there was more than 50% horizontal shift of the shaft of the tibia.

- Severe cases had total displacement with a total loss of contact between the fragments.[42]

The AO group classification (Fig. 11)

The Orthopaedic Trauma Association Committee for Coding and Classification OTA classification includes types 42A (simple patterns—ie, spiral, transverse, oblique), 42B (wedge), and 42C (complex, comminuted) [43]:

The Orthopedic Trauma Association classification [41] is as follows: (Fig. 7)

- Type A – simple

  o   A1 – Spiral

  o   A2 – Oblique greater than 30°

  o   A3 – Transverse

- Type B – Wedge (butterfly fragment)

  o   B1 – Spiral

  o   B2 – Bending

  o   B3 – Fragmented

- Type C – Complex (comminuted)

  o   C1 – Spiral
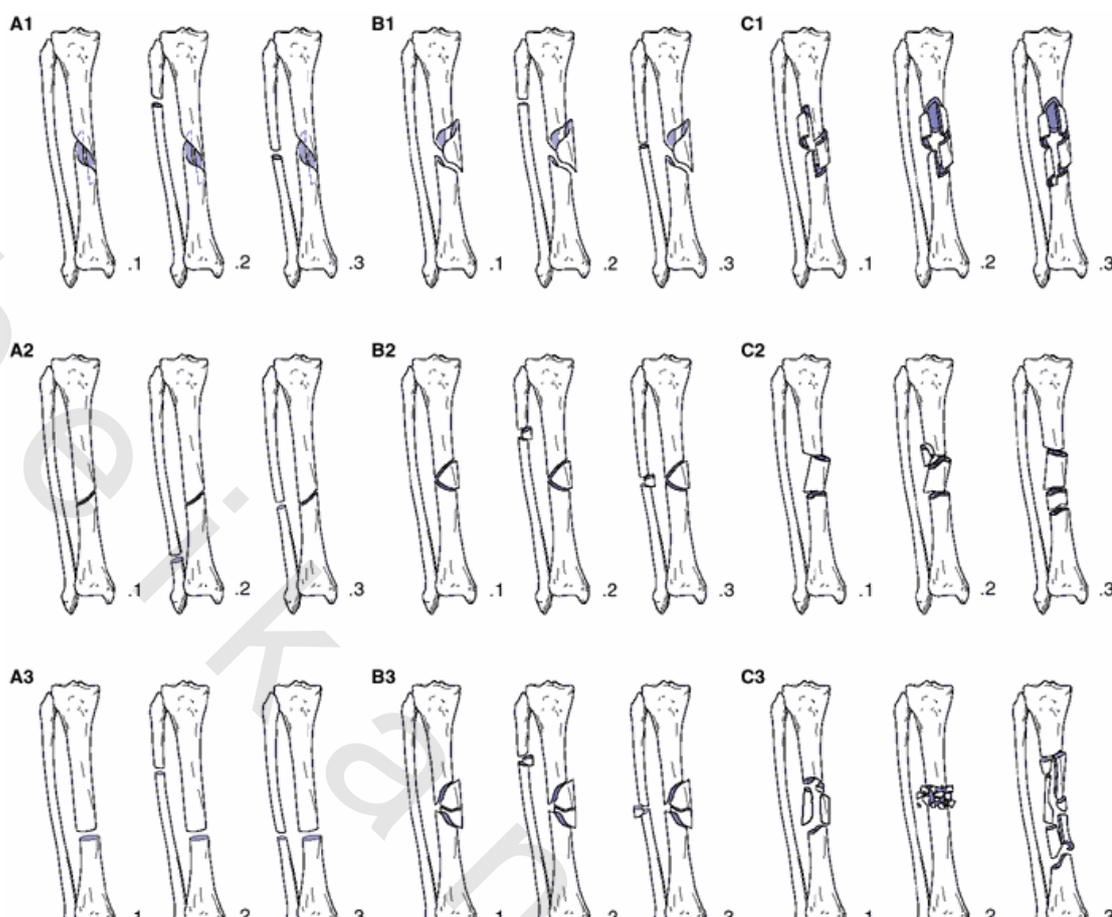
  o   C2 – Segmented

  o   C3 – Irregular

Fig. [11]: OTA classification of tibia/fibula diaphyseal fractures.[43]

Diagnosis of tibial fractures in children

Children present with pain, altered weight bearing status, swelling or deformity. Physical examination usually demonstrates point tenderness at the fracture site, frequently with ecchymosis, edema and deformity. During physical examination, careful neurologic, vascular and soft tissue examination should be performed to detect any compartment syndrome which requires emergent fasciotomies.[5]

Radiographic evaluation should include the entir tibia, ankle and the knee, because injury of the adjacent joints is common. Plain x-rays, in the anteroposterior and lateral planes, are usually sufficient for making the diagnosis. [2]

Management of tibial fractures in children

There is a little disagreement concerning the treatment of long bone fractures in children less than 6 years (plaster of paris "POP cast") and adolescents older than 14 years (locked intramedullary nailing). Controversy persists regarding the age between 6 to 14 years with several options: external fixator, flexible intramedullary nails and plate fixation.[44-48]

Regardless of the method of treatment, the goals are to stabilize the fracture, control length and alignment, promote bone healing, and to minimize the morbidity and complications.[49,50]

Long leg cast management avoids the potential complication of surgical intervention but it carries the risk of delayed union, non-union and mal-union.[8]

Conservative methods: (Reduction and above knee cast)

Uncomplicated tibial and fibular fractures in children can be treated by manipulation and cast application in most of patients. Fractures of the tibial shaft without concomitant fibular fracture may develop varus malalignment. Valgus angulation and shortening can present a significant problem in children who have complete fractures of both the tibia and the fibula. A recurvatum deformity also may develop. [51]

Displaced fractures should be managed by reduction under appropriate sedation. The child's ankle initially may be left in some plantar flexion (20 degrees for fractures of the middle and distal thirds, 10 degrees for fractures of the proximal third) to prevent posterior angulation at the fracture. There is little risk of developing an equinus contracture. [1]

The alignment of the fracture must be checked weekly during the first 3 weeks after the cast has been applied. Muscle atrophy and a reduction in tissue edema may allow the fracture to drift into unacceptable alignment. Some children require a second cast application with remanipulation of the fracture under general anaesthesia several weeks after injury. Acceptable position is somewhat controversial. Remodeling of angular deformity is limited. No absolute number can be given, but the following general principles may be beneficial in decision making: [1]

- Varus and valgus deformity in the upper and mid-shaft tibia remodel slowly if at all. Up to 5 degrees of deformity can be accepted, but not more than 10 degrees.

- Moderate translation of the shaft of the tibia in a young child is satisfactory, whereas in an adolescent, at least 50% apposition should be obtained.

- Up to 10 degrees of anterior angulation may be tolerated, although remodeling is slow.

- Little posterior angulation can be accepted because this forces the knee into extension at heel strike during walking and can produce pain.

- No more than 1 cm of shortening is acceptable, because overgrowth is minimal.

Operative methods:

Surgical stabilization may be indicated for:

(a) children in whom, the accepted position could not be maintained, (b) children who are ten years of age and older, (c) children who have open fractures, (d) fractures associated with compartment syndrome, (e) children who have spasticity due to head injury or cerebral palsy, (f) children who have multiple long-bone fractures or multiple-system injuries and (g) children who have concomitant severe soft-tissue injuries.[1, 6-8]

There are 3 main methods of tibial fracture fixation which are:

External fixation:

External fixation traditionally has been the method of choice for the stabilization of open tibial fractures in children when cast immobilization is not sufficient. It provides excellent stability and allows for multiple debridements of soft-tissue injuries. . [7,9,10,52]

External fixation provides a relatively safe method of tibial fracture treatment in children from ages 6 to 14 years, especially in those with extensive soft tissue damage, multiple trauma, head injury, and open fractures. There are many designs of external fixators. In tibial diaphyseal fractures, the easiest frames to apply are uniplanar frames, which are usually applied to the anteromedial subcutaneous border of the tibia. Uniplanar frames are held in position by four or six half pins, and most modern unilateral fixators have a facility to distract or compress the fracture. External skeletal fixation maintains skeletal alignment by using a combination of pins secured by an external frame. The characteristics of both the pins and the frames vary, but the concept and inherent problems do not. Unilateral frames consist of an external single cylinder to which shanz screws are attached to provide bone fixation. Length adjustment is possible in these devices. [7,9,10,52,53] (Fig. 12)

A good initial reduction is important no matter what type of fixator is applied, as it is often surprisingly difficult to achieve a secondary reduction if the primary reduction is unsuccessful. The incidence of malunion varies between 0% and 50%. Malunion should be avoidable if a multiplanar fixator is used, as adjustment of the device is usually straight forward. Uniplanar devices, with a rigid side bar, are usually more difficult to adjust, and the surgeon must take care to ensure a satisfactory reduction before the external fixator is applied. [53]

The use of external fixation is well established, the results have been satisfactory, but have been associated with complications including infection, delayed union, re-fracture, mal-alignment and joint stiffness. [16]

Pin tract sepsis is common, more than 20% of the patients show pin tract infection. Pin tract sepsis may be common, but it is rarely a problem, and pin will have to be changed only occasionally. The main drawback of pin tract sepsis is that either the patient must be taught to care for his or her pin tracts or regular nursing has to be arranged. [53]

Fig. [12]: Unilateral external fixator.[9]

Open reduction and internal fixation with plate and screws:

The use of AO compression plates for treatment of tibial shaft fractures in children between six and fourteen years of age, especially those with multi-system injuries, is another surgical alternative. The use of compression plates offers the advantages of anatomical reduction, stability and rapid mobilization. However, its disadvantages include: a long incision, risk of infection, risk of plate breakage, stress fracture after plate removal and the need of another operation to remove the implant.[19]

Flexible intramedullary nailing (Elastic stable intramedullary nailing): (Fig. 13,14)

Treatment of pediatric fractures has dramatically changed since 1982, when Metaizeau and the team of Nancy, France, developed the technique of flexible intramedullary pinning using titanium pins. Flexible Intramedullary nails function as an internal splint and provide three-point fixation to maintain bony alignment. [18,19,54]

Intramedullary fixation with elastic nails that are placed percutaneously through the proximal tibial metaphysis without violating the physis has become a popular technique for the treatment of pediatric tibial fractures.[14,55,56] Two flexible intramedullary nails, introduced in an antegrade, cross the fracture site and act as internal splints to maintain length and alignment while allowing sufficient fracture motion to generate callus formation. This technique is commonly referred to as ESIN or flexible intramedullary nailing.[14] It has been used successfully for the treatment of pediatric fractures of the tibia, femur, humerus, and forearm. [11,13,14,56,57]

Smaller incisions, shorter surgical times, and minimal dissection at the fracture site have been described as advantages of nailing over plating in children. In addition, intramedullary fixation provides early motion, easy hardware removal and excellent cosmesis.[11,29,57]

The advantages of ESIN also include early mobilization, low infection rates and shorter hospital stay. The nails can be removed once the fracture has united, depending on the patient and surgeon preference.[20,35]

Fig. [13]: Titanium nails.[14]

Biomechanical principle of the Titanium Elastic Nail:

The aim of this biological, minimally invasive fracture treatment is to achieve a level of reduction and stabilization. The biomechanical principle of the Titanium Elastic Nail is based on the symmetrical bracing action of two elastic nails inserted into the metaphysis, each of which bears against the inner bone at three points. This produces the following four properties: flexural stability, axial stability, translational stability and rotational stability. All four are essential for achieving optimal results (Fig.14).[56]

Elastic stable intramedullary nailing is ideally suited for mid-diaphyseal transverse, short oblique or short spiral fractures with minimal comminution. [55]

The flexible rod is initially bent or curved (plastically deformed). During intramedullary insertion, which is typically antegrade in the tibia, the relatively straight medullary canal (compared with the contoured nail) forces the curved rod to straighten within the bone. This elastic deformation creates a bending moment within the long bone which will tend to angulate the fracture in the direction and the plane of the concavity of the curved rod, as the rod wants to return to its initial curved state. This moment is counteracted by a second rod of matched diameter and curve, which balances the first rod with an equal but opposite moment. The two intramedullary nails act complimentarily to stabilize the fracture. This biologic fixation is not rigid but sufficiently stable against angular, translational and torsional deforming forces and is associated with early formation of exuberant callus. Typically, no additional external immobilization is required.[56,58]
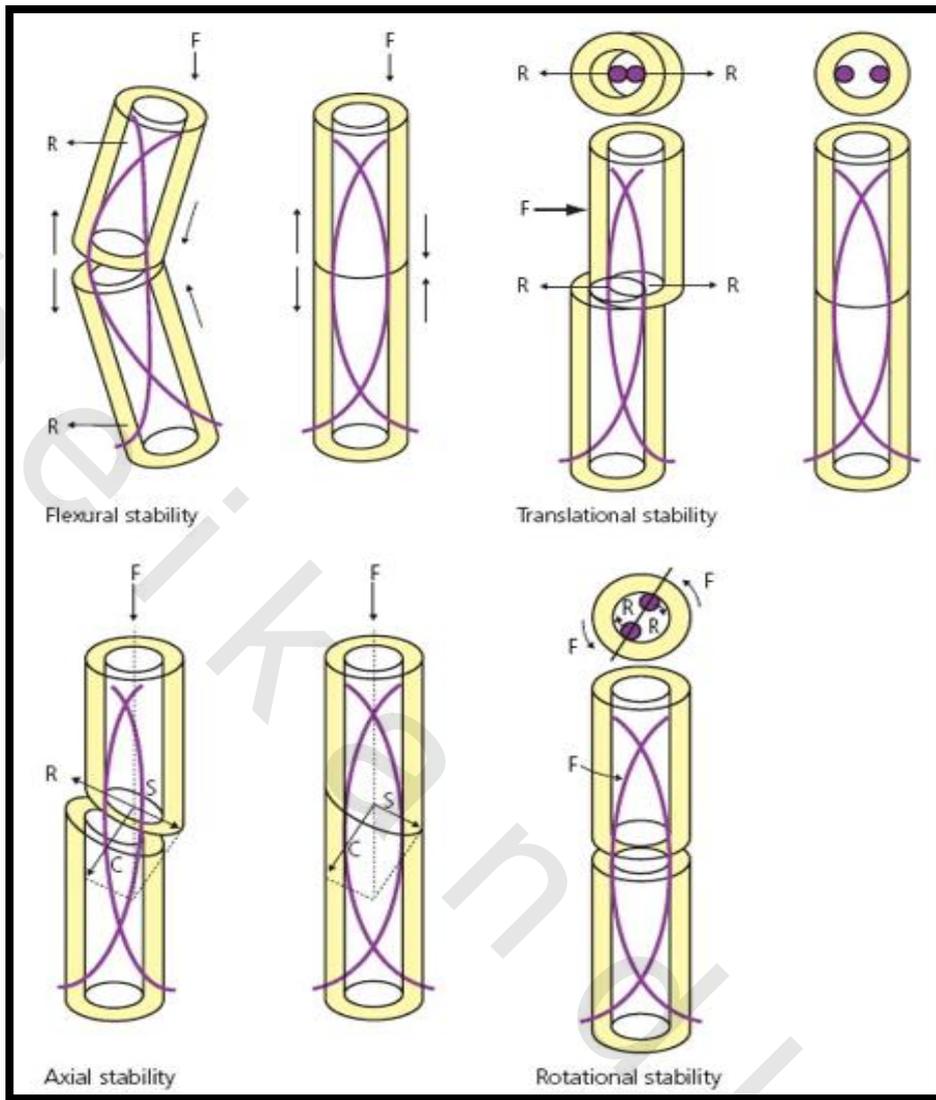
Fig. [14]: Biomechanical principle of the titanium elastic nail.[56] F = force acting on the bone, R = restoring force of the nail, S = shear force, C = compressive force

Fig. [15]: Flexible intramedullary nailing.[59]

Complications of tibial fractures in children

1- Compartment Syndrome

A compartment syndrome may occur after either a minor closed fracture or a severe injury in which the interosseus membrane is disrupted. Compartment syndromes may occur in any or all of the four compartments of the lower leg after trauma. Haemorrhage and soft tissue edema produce an elevation in the pressure within the myofascial compartment that impairs venous outflow. The small arterioles leading into the compartment become less efficient in delivering blood as venous outflow becomes occluded. The arterioles and capillaries close when the pressure in the compartment exceeds the pressure in the vessels. Ischemia soon follows.[60]

Patients with a compartment syndrome may complain of pain out of proportion to the severity of the injury. The compartment is firm to palpation. The patient may have a sensory defect in the distribution of the nerves that run through the compartment. Weakness of the muscles within the involved compartment and pain on passive motion of the toes also are common. Paralysis of the muscles in the involved compartment is a late finding. Pain with passive range of motion appears to be an early and strong clinical finding. Patients with a compartment syndrome involving the deep posterior compartment have severe pain that increases with passive extension of the toes, plantar hyperesthesias, and weakness of toe flexion.[61] Late complications of untreated compartment syndrome include clawed toes and limited subtalar motion secondary to a fibrous contracture of the muscles in the deep posterior compartment.[62]

2- Vascular Injuries

Vascular injuries associated with tibial fractures are uncommon in children; however, when they do occur, the sequelae can be devastating. One factor leading to a poor outcome was a delay in diagnosis. Evaluation for vascular compromise is imperative (during the primary and secondary trauma surveys) in all children with tibial fractures. [63]

The tibial fracture most frequently associated with vascular injury is that of the proximal metaphysis. The anterior tibial artery is in close proximity to the proximal tibia as it passes over the interosseous membrane into the anterior compartment. [64,65]

Distal tibial fractures also are associated with injuries to the anterior tibial artery. The vessels are injured when the distal fragment is translated posteriorly. Posterior tibial artery injuries are rare, except in fractures caused by heavy machinery or gunshots.[64,65]

3- Angular Deformity

Spontaneous correction of axial malalignment after a diaphyseal fracture of a child's forearm or femur is common. Remodeling of a malaligned tibial fracture, however, often is incomplete. As such, the goal of treatment should be to obtain as close to an anatomic alignment as possible. [66]

Spontaneous remodeling of malunited tibial fractures in children has been reported to end at approximately 18 months after fracture. [67]

4- Malrotation

Because rotational malalignment of the tibia does not spontaneously correct with remodelling, any malrotation should be avoided. A computerized tomographic evaluation of tibial rotation can be performed if there is any question about the rotational alignment of the fracture. [67]

Rotational malalignment of more than 10 degrees may produce significant functional impairment and necessitate a late derotational osteotomy of the tibia. The derotational osteotomy should be performed in the supramalleolar aspect of the distal tibia. The tibia is osteotomized and internally fixed. The fibula may be left intact, particularly for planned derotation of less than 20 degrees. [67]

5- Leg Length Discrepancy

Hyperemia associated with fracture repair may stimulate the physes in the involved leg, producing growth acceleration. Tibial growth acceleration after fracture is less than that seen after femoral fractures in children of comparable ages. The average growth acceleration of a child's tibia after fracture is approximately 4.5 mm. Comminuted fractures have the greatest risk of accelerated growth and overgrowth. [2]

The young children have a greater chance for overgrowth than older children. [68] Accelerated growth after tibial fracture occurs in children under 10 years of age, whereas older children may have a mild growth inhibition associated with the fracture. [67] The amount of fracture shortening also has an effect on growth stimulation. Fractures with significant shortening have more physeal growth after fracture union than injuries without shortening at union. [69]

6- Delayed Union and Nonunion

Delayed union and nonunion are uncommon after tibial fractures in children. The use of an external fixation device may lengthen the time to union in some patients, particularly those with open fractures resulting from high energy injury. Inadequate immobilization that allows patterned motion also can slow the rate of healing in patients with delayed union or nonunion; a fibulectomy approximately 4 cm from the tibial fracture allows compression at the delayed union or nonunion site and induces healing. A posterolateral bone graft also is an excellent technique to produce union in younger children. [1]

7- Anterior Tibial Physeal Closure

At present, no universally acceptable explanation can be given for this phenomenon. Some patients have demonstrated apparently iatrogenic closure after placement of a proximal tibial traction pin, the application of pins and plaster, or the application of an external fixation device. Other children have an undiagnosed injury of the tibial physis at the time of the ipsilateral tibial diaphyseal fracture. Regardless of aetiology, premature closure of the physis produces a progressive recurvatum deformity and loss of the normal anterior to posterior slope of the proximal tibia as the child grows, also there is risk for developing another fracture. [1,70]