INTRODUCTION

Femoral shaft fractures represent approximately 1.6 % of all bony injuries in children. It has a bimodal distribution. The first peak is in early childhood (age range: 1-4 years) and the second is in mid-adolescence (age range: 14-17 years).[1]

In adolescence (age range : 6-14 years) femoral fractures are most likely to be caused by high-energy injuries such as motor vehicles accidents which account for over 90% of femoral fractures in this age group.[2]

Treatment of femoral fractures in adolescence between 6-14 years of age is controversial and age dependent. So, there are several methods by which this can be achieved, each of which has advantages and disadvantages.[3]

Femoral fractures in children are frequently treated conservatively by skin or skeletal traction aiming to overcome the effect of the strong pull of the quadriceps and hamstring muscles. This method is effective, however it has the disadvantage of prolonged recumbancy, the need for proper nursing, continuous monitoring, and frequent readjustment, as well as psychological problems and increased cost.[4]

Early plaster spica allows early hospital discharge,[5] but problems in transportation, cast intolerance, displacement, shortening and angulations of the fracture are frequent.[1]

The management of paediatric femoral shaft fractures gradually has evolved towards a more operative approach in the past decade. The aim of surgical treatment is to provide rapid recovery and avoid negative effects of prolonged immobilization.[6]

The use of AO compression plates for treatment of femoral shaft fractures in older children is an effective surgical alternative. It offers the advantages of anatomical reduction, ease of insertion, simplified nursing care, and rapid mobilization without casting. However, its disadvantages includes: the long incision, risk of infection, risk of plate breakage, stress fracture after plate removal and the need of another operation to remove the implant.[3,7]

External fixation of femoral shaft fractures offers a fast convenient method to align and hold the fractured femur .It is the method of choice when severe soft tissue injury is present and therefore, recommended for open or severely comminuted fractures. [8] The most common complication of external fixation is pin track infection, which has been reported to occur in up to 72% of patients.[9]

Reamed locked intramedullary nails are most effective in the isolated fracture or in the hemodynamically stable, multiply injured patient. However, it is not recommended for patients younger than 12 years of age (proximal femoral physis is still open) because proximal femoral growth abnormalities and osteonecrosis of the femoral head due to disruption of the vascular supply are possible complications.[10]

Flexible intramedullary nailing has become an increasingly popular method of fixation of femoral fractures in children.

This technique has been also called Elastic Stable Intramedullary Nailing (ESIN).ESIN relies on the flexibility of the nail to provide a stable reduction and dynamization of femur fractures in children. ESIN uses two small diameter flexible nails which are pre-bent and inserted in either retrograde or antegrade fashion into the fractured femur .[11]

ANATOMY OF THE FEMUR

The femur is the largest and strongest bone in the human body. The femur can be divided into regions consisting of the head, neck, intertrochanteric, subtrochanteric (extending 5 cm distal to the lesser trochanter), shaft, supracondylar, and condylar regions. The femoral shaft is gently curved with forward convexity. It is divided into upper, middle and lower thirds.[13]

The middle third:

The middle third is the narrowest part of the shaft; it possesses three surfaces and three borders. The anterior surface is smooth and extends between the lateral and medial borders, both of which are rounded and ill-defined. The lateral surface is directed more backwards than laterally and is bound in front by the lateral border and behind by the posterior border (linea aspera) which usually forms a crest like projection with distinct lateral and medial lips. The medial surface is directed medially and slightly backwards, smooth like the other two surfaces, it is bound in front by the medial border and behind by the linea aspera. (Figure 1,2)[13]

The upper third:

The shaft possesses a fourth surface that is directed backwards. On the medial side, the posterior surface is bounded by the spiral line (which continues below with the medial lip of the linea aspera and above with the lower end of the intertrochanteric line). On the lateral side, the posterior surface is bounded by a broad roughened ridge termed the gluteal tuberosity (which continues below with the lateral lip of the linea aspera and above with the root of the greater trochanter) (Figure 1,2).[13]

The lower third:

The shaft also possesses a fourth posterior surface which is placed between the lateral and medial supracondylar ridges (which continue above with the corresponding edges of the linea aspera and extend downwards to the epicondyles). This flattened triangular area is termed popliteal surface of the femur (Figure 1,2).[13]

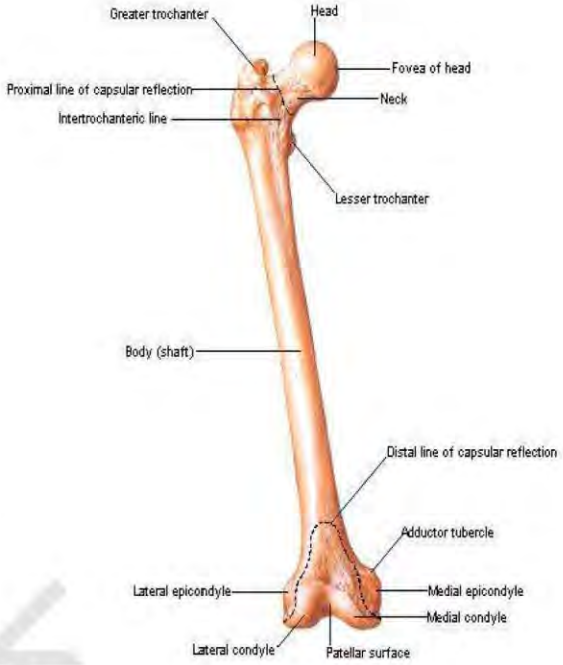


Figure (1): Anatomy of femur - Anterior view) [13]

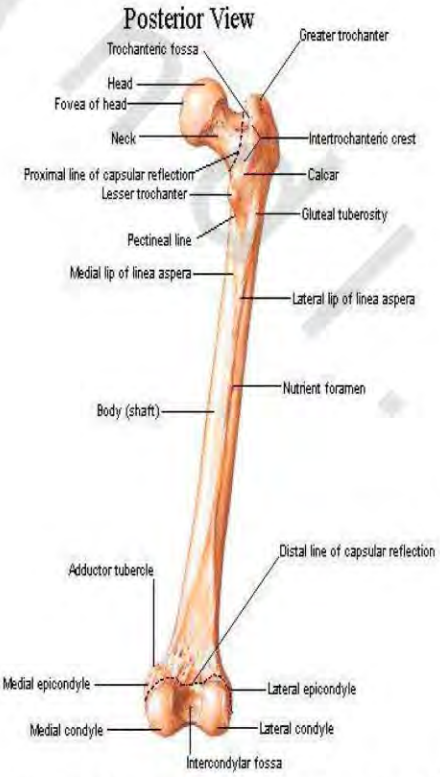


Figure (2): Anatomy of femur –posterior view) [13]

Ossification of the femur (Figure 3)

The femur is ossified from *five* centres: one for the body, one for the head, one for each trochanter, and one for the lower end. Of all the long bones, except the clavicle, it is the first to show traces of ossification; this commences in the middle of the body, at about the seventh week of foetal life, and rapidly extends upward and downward. The centres in the epiphyses appear in the following order: in the lower end of the bone, at the ninth month of foetal life (from this centre the condyles and epicondyles are formed); in the head, at the end of the first year after birth; in the greater trochanter, during the fourth year; and in the lesser trochanter, between the thirteenth and fourteenth years. The order in which the epiphyses are joined to the body is the reverse of that of their appearance; they are not united until after puberty, the lesser trochanter being first joined, then the greater, then the head, and, lastly, the inferior end, which is not united until the twentieth year. (13)

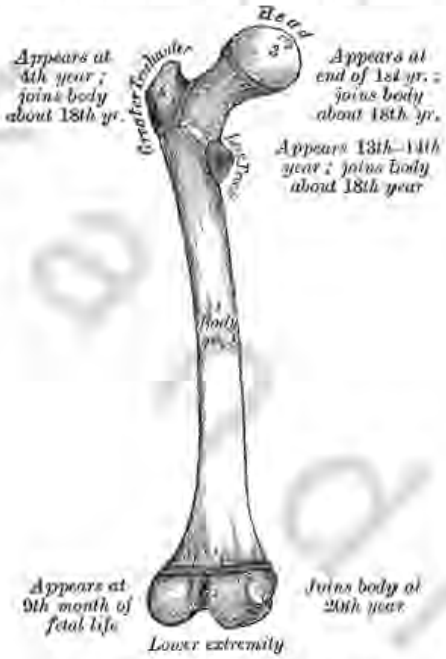


Figure (3): Ossification of the femur. (13)

The muscles of the thigh (Figures 4,5,6,7)

The muscles of the thigh can be divided into three groups:

1. The anterior femoral muscles:

Included in this group are the tensor fascia lata, sartorius and rectus femoris, which can act on both the hip and knee joints. The group also includes three vasti; medialis, lateralis and intermedius, which act on the knee joint only. The articularis genu, a derivative of vastus intermedius, acting as a retractor of the synovial capsule of the knee joint completes this group. The rectus femoris and the vasti extend the knee joint through a common tendon, and are hence considered as one muscle, the quadriceps femoris.(13)

2. The medial femoral muscles:

Included in this group are the adductor longus, gracilis, adductor magnus, adductor brevis and obturator externus. The contents of this compartment are separated from the anterior muscle group by the medial intermuscular septum. There is no septum separating this group from the posterior flexor compartment. All these muscles cross the hip joint, but only the gracilis is inserted distal to the knee joint. Collectively, they are known as the adductors of the thigh, but their actions are more complex.[13]

3. The posterior femoral muscles:

These are the biceps femoris, semitendinosus and semimembranosus muscles, which are often called the hamstrings. They span the hip and knee joints.[13] This compartment is separated from the anterior compartment by the lateral intermuscular septum, but there is no septum separating it from the medial femoral compartment.[16] The lateral and medial heads of the gastrocnemius muscle and the popliteus muscle arise from the lower end of the back of the shaft.[17]

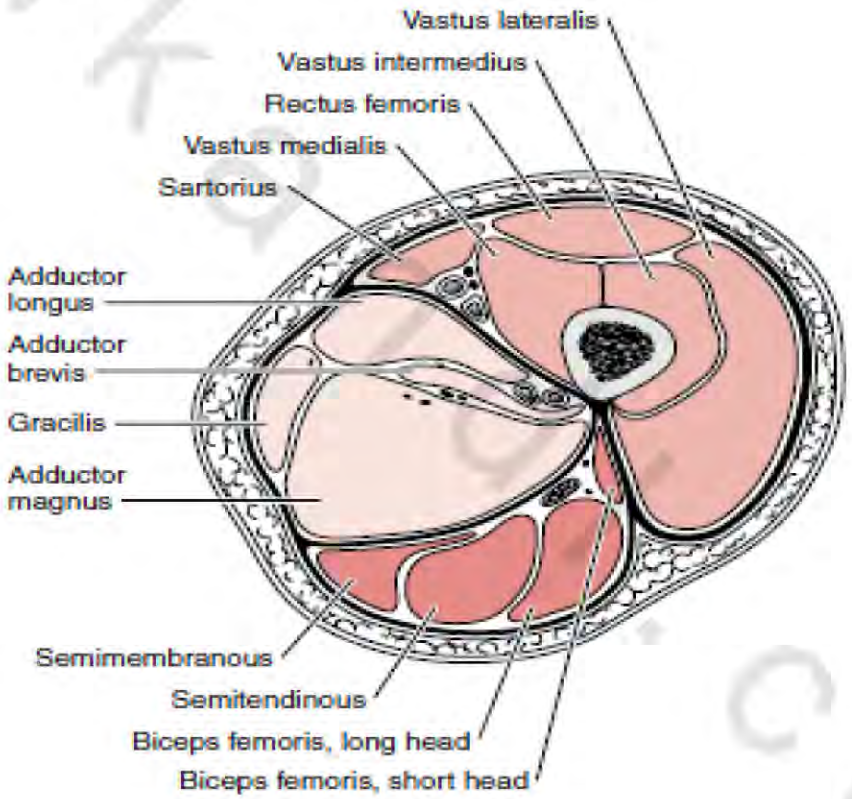


Figure (4): Cross section of the middle third of the thigh showing the relative positions of the neurovascular structures, bones, and muscles. Different shading indicates the different compartments in the thigh. .[13]

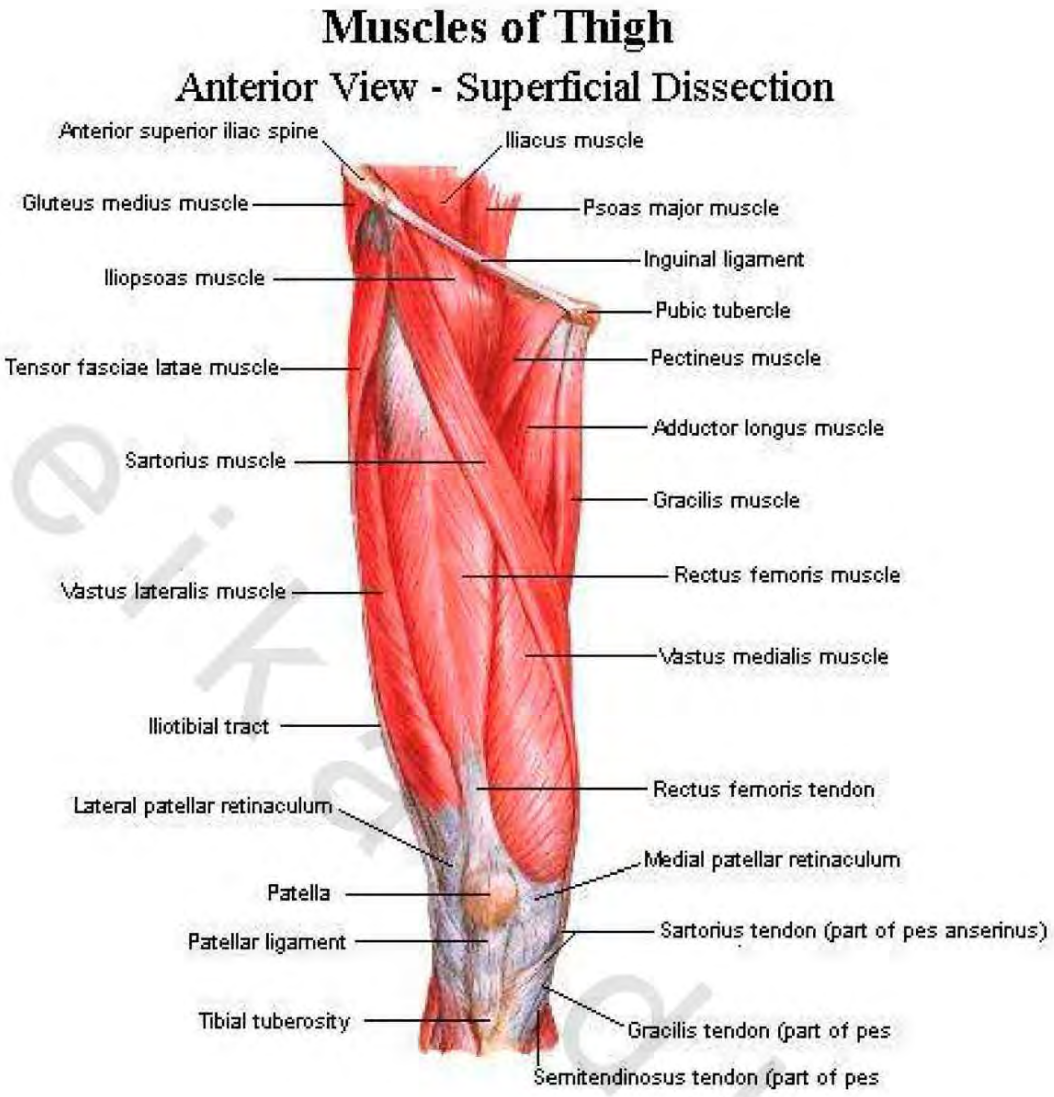


Figure (5): muscles of the thigh anterior view.[16]

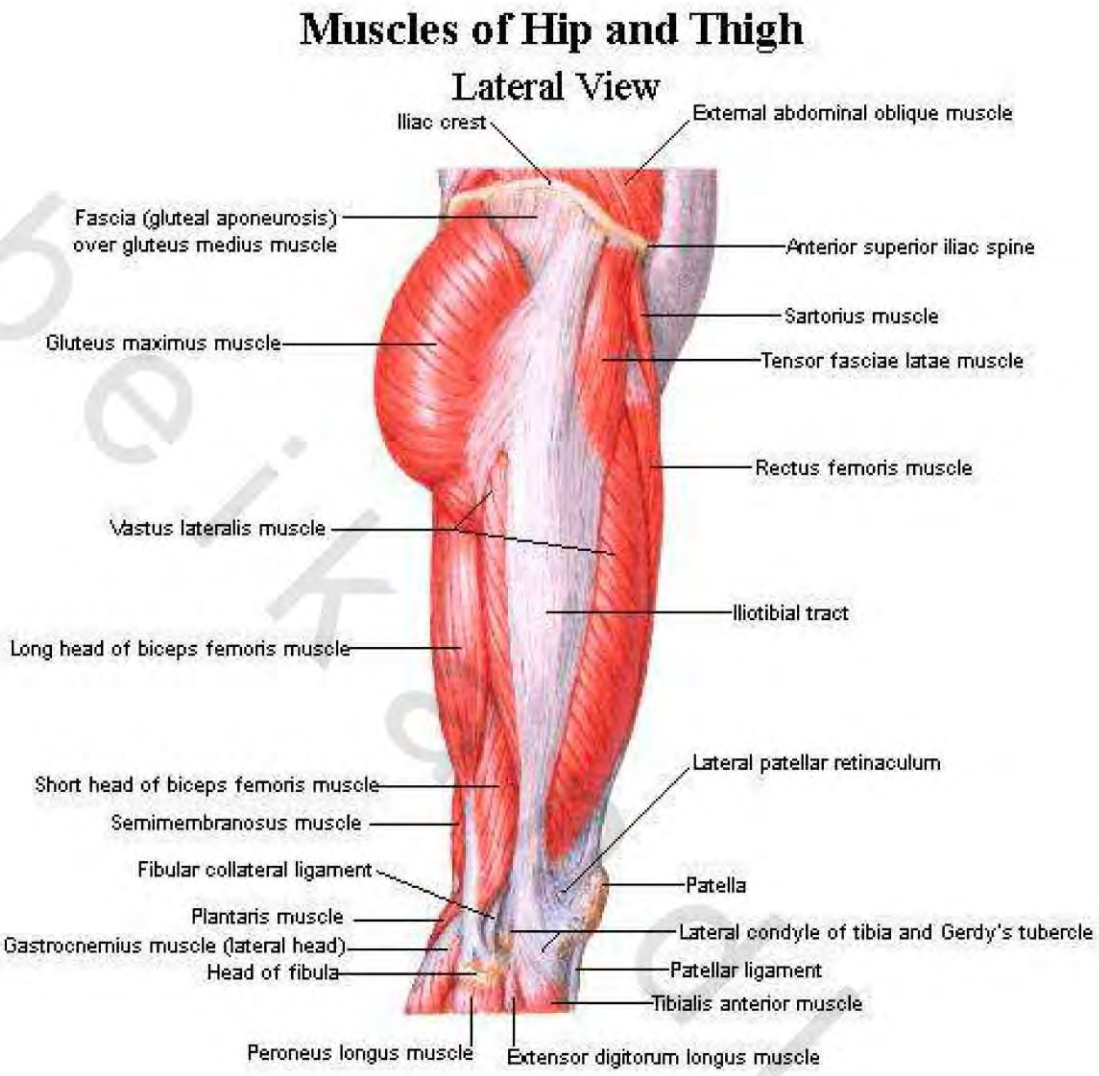


Figure (6): muscles of the thigh lateral view.[16]

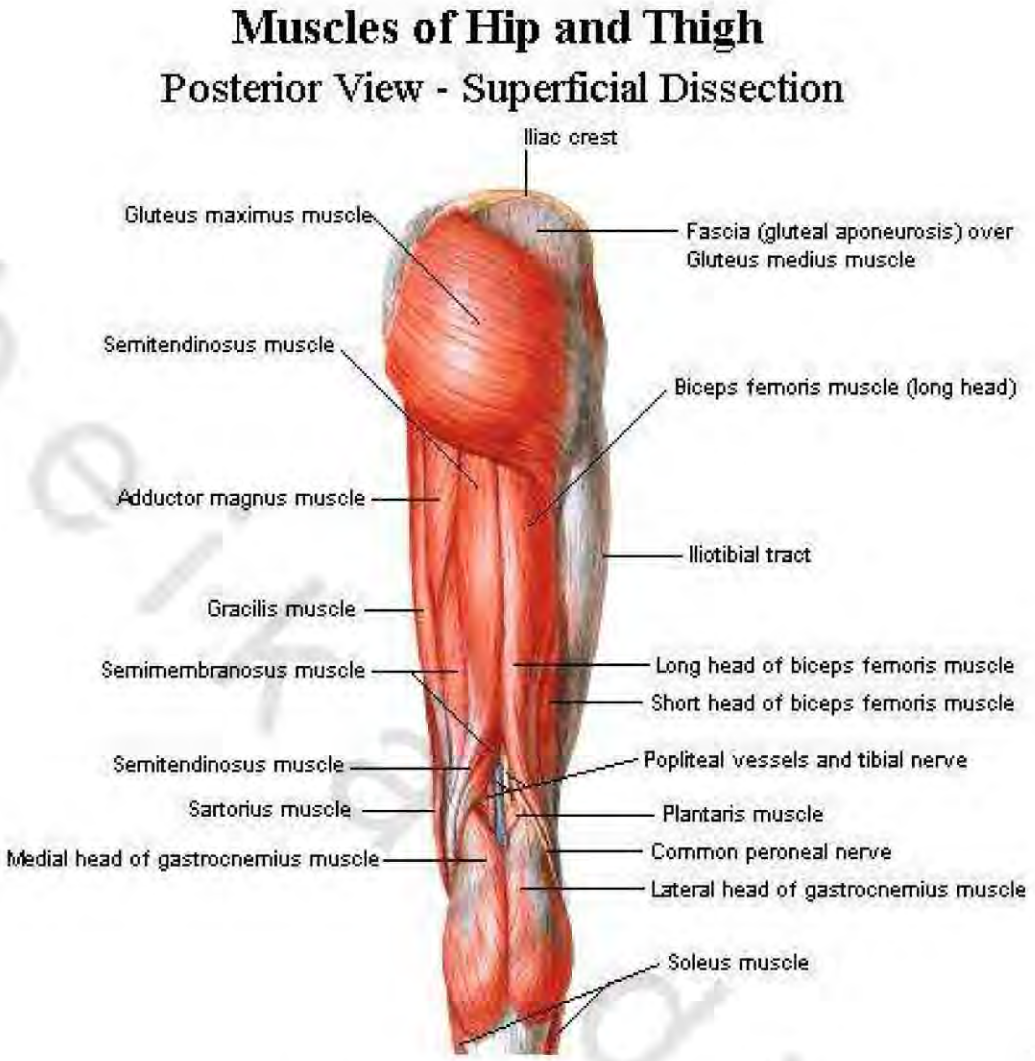


Figure (7): muscles of the thigh posterior view.[16]

Blood supply of the femur

The blood supply to the femur, like that of all tubular bones, is through periosteal and endosteal vessels. The periosteal blood supply is related to the multiple muscle origins from the femoral shaft and thus is seldom in danger in fractures of the femoral shaft except in case of extensive stripping.[18] The nutrient artery provides the endosteal blood supply. Usually the femur has only one nutrient artery supplying the shaft. It arises from the first perforating branch of the profunda femoris artery. This artery enters the shaft along the linea aspera.[18] In some cases, there is a second nutrient artery which arises from one of the remaining perforating vessels in an inconstant manner. It also enters the femoral shaft in the region of the linea aspera at a more distal level. The arteries branch as soon as they reach the medullary canal and these branches pass up and down the medullary cavity.[19]

The relative contributions of the nutrient vascular system and the periosteal system remain controversial. Most evidences indicate that the primary arterioles of medullary origin arborise within the inner two-thirds to three-quarters of the cortex and then anastomose with the scattered blood vessels of the periosteal circulation. [18, 20] By means of haemodynamic experiments and microangiographic analysis, it became evident that the blood flow in the cortical bone passes centrifugally from the medullary arterial system into the cortical capillary mesh then out into the capillaries of the periosteal membrane and the interfascicular capillaries and veins of the attached muscles. [21] Recently, observation of blood flow has disclosed that blood flow in the cortex is not completely centrifugal, but blood returns to the venous sinusoids of the intramedullary canal. [18] This is a fact because the cortical capillary mesh communicates, not only with the capillaries outside the bone, but also with the sinusoids of the marrow which are connected to the longitudinally disposed central venous sinus.[21]

Vascular response in 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 especially along the linea aspera. The early external callus receives its blood supply from the periosteal circulation through vessels which are oriented perpendicular to the cortical surface.[20]

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.[20]

BIOMECHANICS

Axes of the femur (Figure 8)

THE MECHANICAL AXIS

The mechanical axis is the line connecting the centre of the hip joint to the centre of the knee joint. It makes 3° from the vertical line. The shaft of the femur is convex anteriorly, so that an imaginary line drawn from the midpoint of the femoral head (i.e. the centre of the hip joint) downward through the midpoint of the knee joint does not pass through the centre of the femoral shaft but is instead posterior to it, especially in the lower two thirds of the thigh. This arrangement is important in understanding the action of some muscles inserting on the femur: although they insert on the posterior surface of the femur, they work as medial rotators of the thigh because the axis of rotation of the thigh passes posterior to the femur along some of its length. [14]

THE ANATOMICAL AXIS

The anatomical axis is the line drawn through the centre of the trochanteric area to the centre of the knee joint line. It makes 9° from the vertical line. The anatomical axis does not coincide with the mechanical axis; it forms an angle of about 6° with it. The shaft inclines downwards and medially so that the medial sides of both knees almost touch.[14]

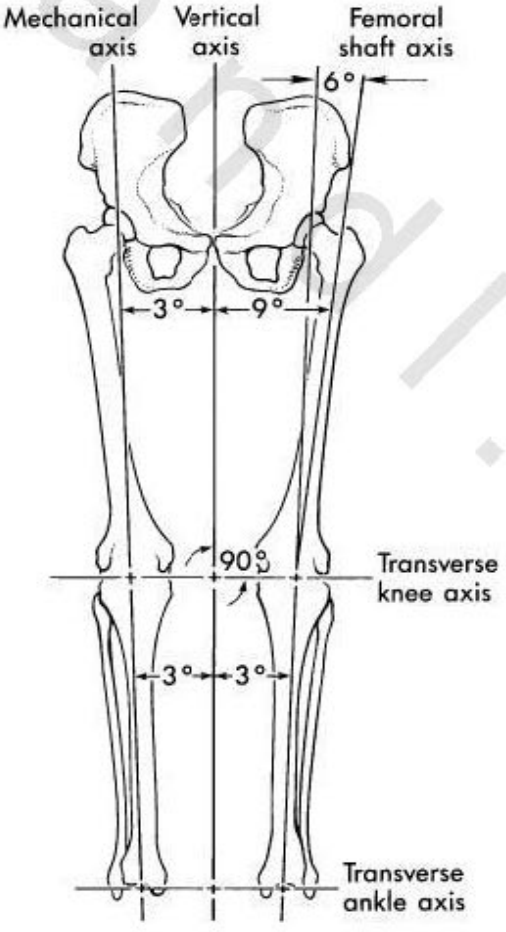


Figure (8): Axes of the femur. [14]

Significance of the Inner Architecture of the Shaft of the femur

1. Resisting shear:

The shearing stresses are at a minimum in the shaft. As shearing stresses are most efficiently resisted by material placed near the neutral plane, in the shaft a minimum amount of material will be needed near the neutral axis. In this region there is very little if any material in the central space, practically the only material near the neutral plane being in the compact bone, but lying at a distance from the neutral axis.[13]

2. Resisting bending moment:

"The bending moment increases from a minimum at section 4 to a maximum between sections 16 and 18, then gradually decreases almost uniformly to 0 near section 75." Figure (9) "To resist bending moment stresses most effectively the material should be as far from the neutral axis as possible." It is evident that the hollow shaft of the femur is an efficient structure for resisting bending moment stresses, all of the material in the shaft being relatively at a considerable distance from the neutral axis.[11]

3. Resisting axial stress.

The inner architecture of the shaft is adapted to resist axial stresses.
The structure of the shaft is such as to secure great strength with a relatively small amount of material.[13]

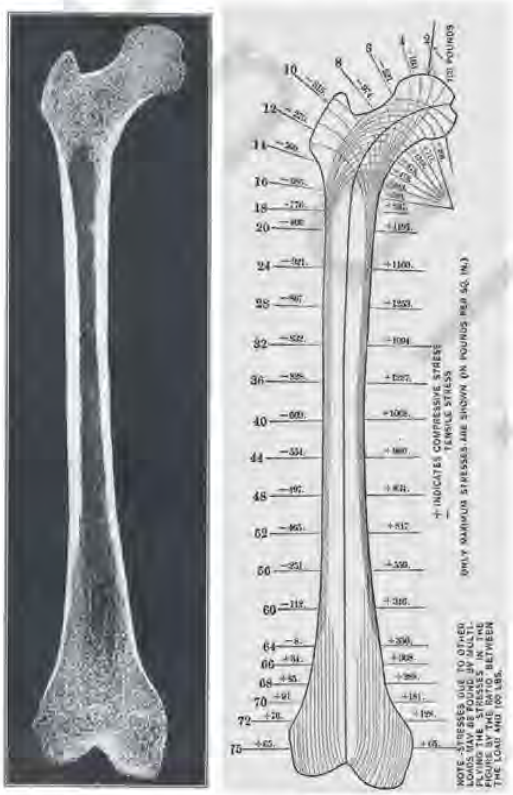
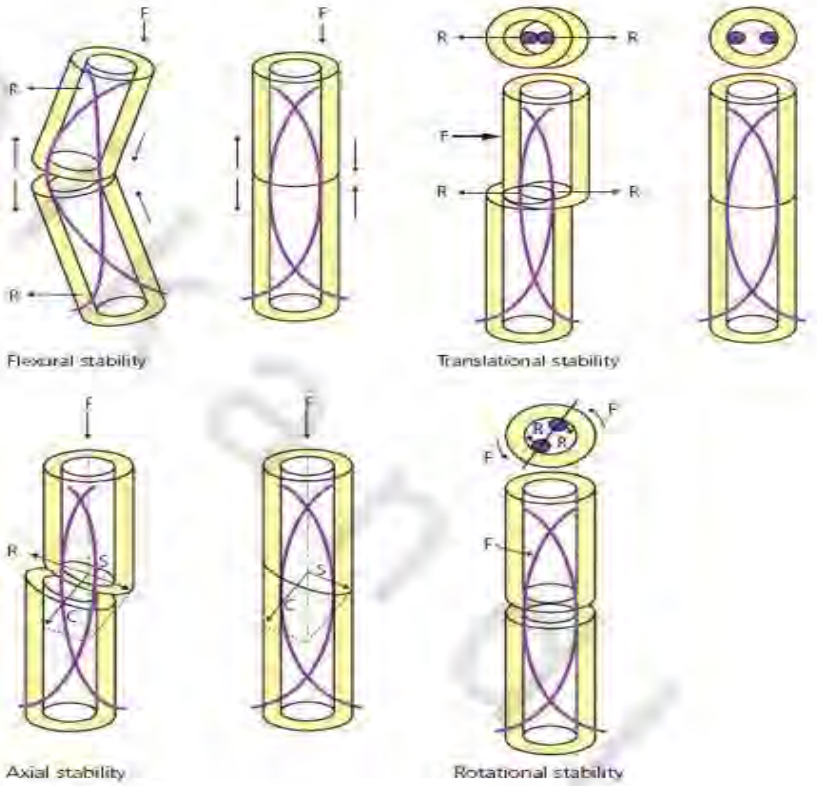


Figure (9): Inner architecture of the shaft of the femur.
Left: Frontal longitudinal midsection of right femur.
Right: Diagram of the computed lines of maximum stress in the right femur. [13]

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 that is appropriate to the age of the child. 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 (Figure 10).[22,75]



F = force acting on the bone
R = restoring force of the nail
S = shear force
C = compressive force

Figure (10): Biomechanical principle of the titanium elastic nail. [22]

Biomechanical principle of the Dynamic Compression Plate (DCP):

Plates are a fundamental element of internal fixation. Compression plates can be used to reduce and stabilize transverse or short oblique fractures when lag screw fixation alone is inadequate. The plate can produce static compression in the direction of the long axis of bone in three ways: by over bending of the plate, by application of a tension device, and by a special plate design that generates axial compression by combining screw hole geometry with screw insertion. The special geometry of the DC plate hole allows for two basic functions: independent axial compression and the ability to place screws at different angles of inclination. In the compressive mode, the screw is inserted 1.0 mm eccentrically to its final position in the hole on the side away from the fracture site. When the screw is tightened, its head slides down along the inclined

plane, merging the eccentric circles and causing horizontal movement of the plate (1.0 mm). This results in fracture compression, assuming that a plate screw has previously been inserted to affix the plate into the other fracture fragment. This procedure can produce a maximum of 600 N of axial compression if anatomic reduction of the fragments is accomplished (Figure 11).[73,74]

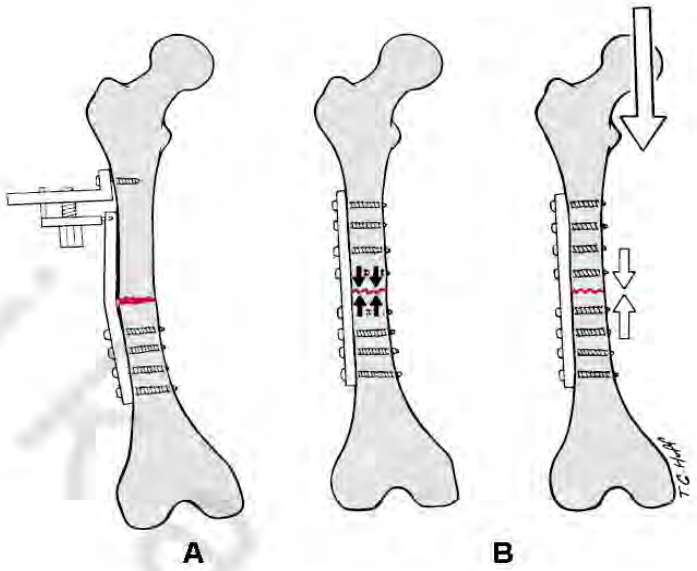


Figure (11): Application of a compression plate to the lateral aspect of The femur demonstrates the combination of static and dynamic compression.

A, Immediately after the plate is applied, static compression is achieved at the fracture site. *B,* After functional loading of the curved bone, additional dynamic compression is obtained at the fracture site because of the effect of the laterally placed plate, which acts as a tension band.[73]

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 the fractures. Fracture healing can be divided into the following phases:[23]

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.[24]

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.[23]
B. Necrosis of the bone adjacent to the fracture line.[25]
C. Necrosis of the periosteum, marrow and other surrounding soft tissues around the fracture site.[25]

The injury initiates a set of inflammatory events that has two beneficial effects:

- Inflammation causes substantial pain and swelling, resulting in hydraulic splinting of the limb and voluntary immobilization. A stiffened, immobile limb optimizes the local mechanical environment for fracture healing by minimizing interfragmentary motion.
- 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, 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. [26]

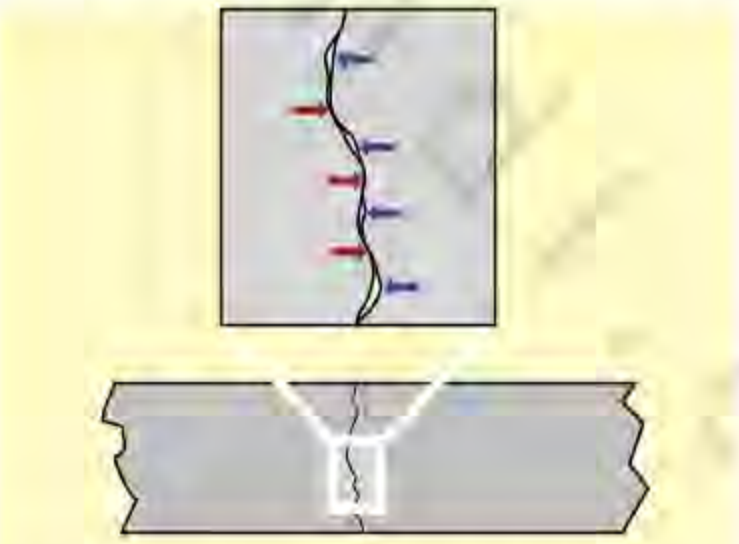
2. Reparative phase:

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.[25, 27]

Later, formation of the bridging callus in the fracture gap occurs between the first and third week. [26] 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.[23] 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. [25,27] If the fracture is held less rigidly, e.g. conservative treatment using a cast, 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.[26]

Direct bone healing (Figure 12)

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.[26]



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.

Figure (12): Direct bone healing. [26]

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.

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. After a lag phase of several weeks, the basic remodelling unit forms a cutting cone, with osteoclasts on the leading surface of the cone (Figure 7). 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.[26]

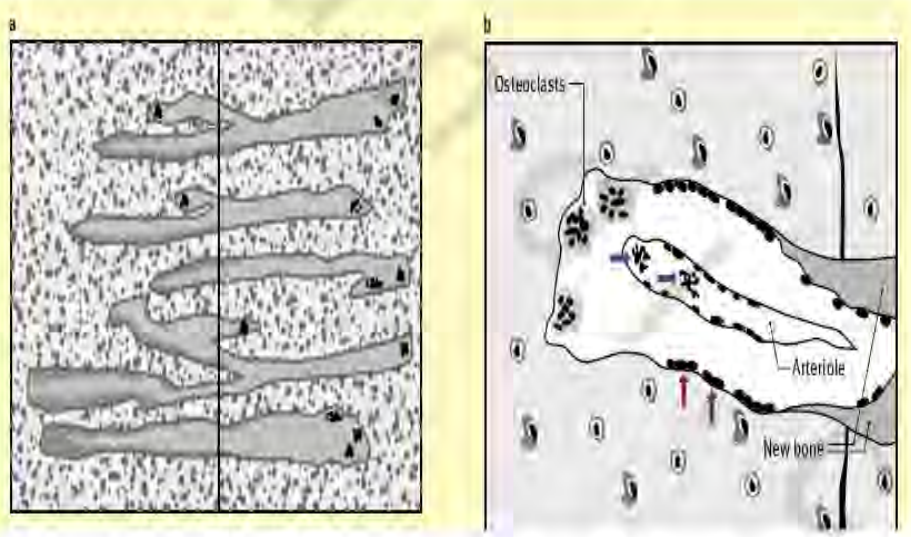


Figure (13): Contact healing: cutting cone. [26]

Secondary bone healing (Figure 14)

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. 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.[26]

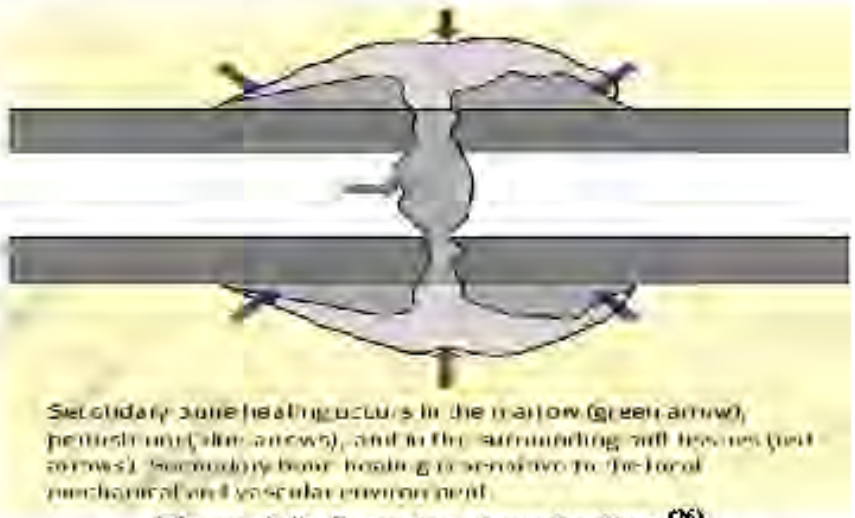


Figure (14): Secondary bone healing. [26]

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.

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.

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.[26]

Healing response in the surrounding soft tissues – like periosteal healing, this healing is sensitive to its mechanical environment, being substantially dampened 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.[26]

Secondary bone healing following flexible intramedullary nailing

Flexible intramedullary nails have enjoyed dramatic success in the treatment of diaphyseal femur fractures. This biological fixation is not rigid but sufficiently stable against angular, translational and torsional deforming forces (Elastic Stability). Therefore, beneficial interfragmentary micromotion between bone ends cause these fractures to unite by secondary bone healing with early formation of exuberant callus. Typically, no external immobilization is required. 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 children's fractures.[12]

3. Remodelling phase:

Remodelling occurs from the sixth week to one year or longer.[27] It involves a process of simultaneous bone removal and replacement through the respective action of osteoclasts and osteoblasts together with the accompanying blood vessels. 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.

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 thin where loads are diminished.[24,25]

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.[25]

CLASSIFICATION OF FEMORAL SHAFT FRACTURES IN CHILDREN

Femoral shaft fractures can be classified by location, as follows: proximal third, middle third and distal third fractures. The geometry of the fracture, comminution and open versus closed status are also used. A universal classification system does not exist. Two of the most common classification systems are the Winquist-Hansen and Arbeitsgemeinschaft für Osteosynthesefragen/Association for the Study of Internal Fixation (AO/ASIF) systems. The most common femoral fracture in children is a middle third, transverse, closed injury.[1]

Winquist-Hansen classification (Figure 15)

Type I: Minimal or no comminution
Type II: Cortices of both fragments at least 50% intact
Type III: 50% to 100% cortical comminution
Type IV: Circumferential comminution with no cortical contact

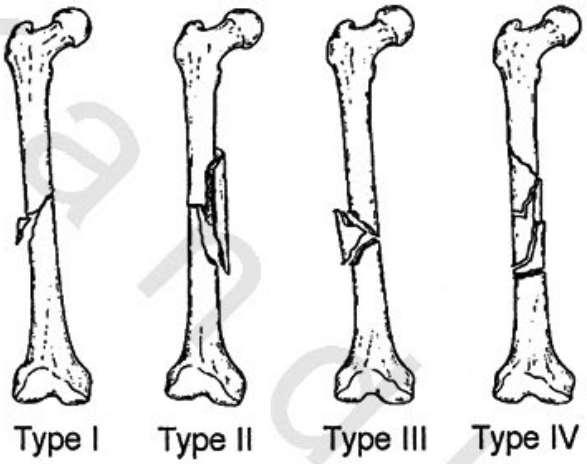


Figure (15): Winquist and Hansen classification of femoral shaft fractures.[1]

AO classification

A1 simple fracture, spiral
- Subtrochanteric zone
- Middle zone
- Distal zone

A2 simple fracture, oblique (30° or more)
- Subtrochanteric zone
- Middle zone
- Distal zone

A3 simple fracture, transverse (<30°)
- Subtrochanteric zone
- Middle zone
- Distal zone

B1 wedge fracture, spiral wedge
- Subtrochanteric zone
- Middle zone
- Distal zone

B2 wedge fracture, bending wedge
- Subtrochanteric zone
- Middle zone
- Distal zone

B3 wedge fracture, fragmented wedge
- Subtrochanteric zone
- Middle zone
- Marked displacement

C1 complex fracture, spiral
- With two intermediate fragments
- With three intermediate fragments
- With more than three intermediate fragments

C2 complex fracture, segmental
- With one intermediate segmental fragment
- With one intermediate segmental and additional wedge fragment(s)
- With two intermediate segmental fragments

C3 complex fracture, irregular
- With two or three intermediate fragments
- With limited shattering (<5 cm)
- With extensive shattering (5 cm or more)

The level of the fracture (Figure 16) leads to characteristic displacement of the fragments based on the attached muscles.

a. Proximal third fracture: the displacement is often constant; the short proximal fragment is flexed, abducted and externally rotated. Flexion is caused by the iliopsoas muscle which is inserted into the lesser trochanter. Abduction is caused by the gluteus medius and to a lesser extent the gluteus minimus. While external rotation is due to the unopposed action of the short rotators, chiefly the obturator internus and externus, the two gemelli and the pyriformis. All are attached to the greater trochanter. The long distal fragment is pulled behind the proximal fragment by the muscles that bridge the fracture site.

b. Middle third fracture: the displacement is less extreme because there is compensation by the abductors and extensor attachment on the proximal fragment.

c. Distal third fracture: results in little alteration in the proximal fragment position because most muscles are attached to the same fragment providing balance. The distal fragment, however, is rotated to a greater degree by the gastrocnemius muscle and may exert pressure on the popliteal artery, and thus may interfere with the blood flow through the leg and foot. [1]

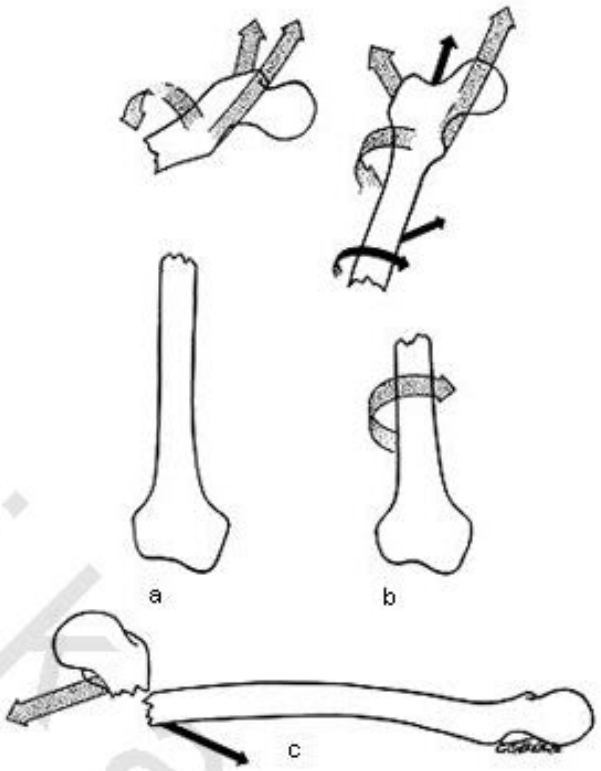


Figure (16): The relationship of fracture level and displacement of the fragments.[1]

a. proximal third fracture
b. middle third fracture
c. distal third fracture

DIAGNOSIS OF FEMORAL SHAFT FRACTURES IN CHILDREN

Most patients with femoral shaft fractures are unable to walk and are in extreme pain with an obvious deformity. The diagnosis is more difficult in patients with multiple trauma or head injury, and in non-ambulatory, severely disabled children. A physical examination usually is sufficient to document the presence of a femoral fracture. Swelling, instability, crepitance, and tenderness are usually present.[1]

The entire child must be carefully examined. Hypotension rarely results from an isolated femoral fracture. "Waddell's triad" of femoral fracture, intra-abdominal or intrathoracic injury, and head injury are associated with high-velocity automobile injuries.[1]

Radiographic evaluation should include the entire femur, hip and the knee, because injury of the adjacent joints is common. Quality plain x-rays, in the anteroposterior and lateral planes, are usually sufficient for making the diagnosis.[1]

MANAGEMENT OF FEMORAL SHAFT FRACTURES IN CHILDREN

Treatment of femoral shaft fractures in children between six and fourteen years of age is controversial. There are several methods of treatment, each one has advantages and disadvantages. There are many factors that must be considered in treatment of femoral fractures in children, such as the age, the size of the bone, the cause of injury, whether the femoral fracture is an isolated injury or part of poly-trauma, economic concerns, and the psychological implication of the treatment.[1]

The long-term goals of femoral shaft fracture management include a united fracture, acceptable limb-length equality at maturity and lack of significant angulations and malrotation.[28]

Conservative methods:

Hip spica casting:

Hip spica casting after reduction under anaesthesia is the usual method of treatment, especially in children younger than six years of age, unless (1) shortening of more than 2cms is present, (2) massive swelling of the thigh is noted, or (3) associated injuries are present. Its primary advantages are simplicity, low cost and generally good results based on leg-length equality, healing time, and motion after removal of the cast. The greatest problems encountered by the family in caring for a child in spica cast are the difficult transportation, cast intolerance by the child and keeping the child clean. Counselling and planning with the family before cast application must be done, if possible. Shortening and angulation deformities are problems faced in immediate spica casting. These can be detected and consequently corrected with weekly radiological evaluation during the first two weeks after injury.[1]

The position of the hips and knees in the spica cast is controversial. Varying the amounts of hip and knee flexion in the spica cast based on the position of the fracture has also been recommended: the more proximal the fracture, the more flexed the hip should be. Generally, the spica cast is worn for four to eight weeks, depending on the age of the child and the severity of the soft-tissue damage accompanying the fracture. After the cast has been removed, management should include skin care in young children and crutch-assisted or walker-assisted ambulation in older children. Ambulation is accomplished with weight bearing as tolerated. Stiff knees and weak hamstrings and quadriceps should be identified after the child begins to walk, and a physical therapy program should be started. Long-term follow-up for leg-length evaluation, range of motion, and quadriceps weakness should be maintained.[1,29,30]

Traction and casting:

Traction followed by spica casting, is another method used to treat femoral shaft fractures in children, especially those without multiple fractures, head trauma, severe soft-tissue or vascular injuries. The patient must be able to comply with the period of bed rest and spica cast immobilization. The indications for skin or skeletal traction include (1) unstable femoral fracture in a child younger than six years of age with more than 3cms shortening, (2) femoral fracture that fails to maintain proper length and alignment in a spica cast in a child younger than six years of age, (3) femoral fracture in

a child six to eleven years of age, without multiple fractures, head trauma, or severe soft-tissue or vascular injuries, who is able to cooperate with a period of bed rest and spica cast immobilization. In general, however, skeletal traction is not recommended for children twelve years of age or older because of a significant incidence of shortening and angular malformation. Comparative studies and retrospective reviews have demonstrated unsatisfactory results in a small, yet significant, percentage of patients treated in this manner.[31]

The limit of skin traction is the interface between skin and tape. Skin complications, such as skin slough and blistering, usually occur when more than five pounds of traction is applied. When more than five pounds of traction is required, or simply for ease in patient management, skeletal traction may be used to maintain alignment. In patients with femoral fractures, skeletal traction is best accomplished using a threaded or smooth skeletal traction pin. Threaded pins, although more difficult to remove, are preferable to smooth pins because of their secure fixation within the bone without side-to-side movement; however, they have a slightly higher incidence of skin interface complications. The distal femur is the location of choice for a traction pin, although proximal tibial traction pins have been recommended by some. The time in traction depends on the age and size of the child and the time required for the fracture to become "sticky". It may range from seven to twenty one days.[1,31]

Operative methods:

External fixation:

External fixation provides a relatively safe method of femoral fracture treatment in children from ages 5 to 11 years, especially in those with extensive soft tissue damage, multiple trauma, head injury, and open fractures. 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 (Figure 17) ORTHOFIX consist of an external single rod to which stout Shanz screws are attached to provide bone fixation. Length adjustment is possible in these devices. [32]

The proximal pin should be placed at least 2 cm below the greater trochanteric apophysis, a point often corresponding to the lesser trochanter. The distal pin should be a similar distance above the distal femoral physis to prevent physeal damage.

The fracture is reduced by manual traction or on a fracture table. The fixator and pins are usually removed with the use of general anaesthesia in an outpatient surgery setting after union or spica should be applied at the same setting. [32]

Despite reports of good results with these devices, concerns about adverse psychological consequences, delayed fracture healing, infection, loosening , refracture, and overgrowth have reduced broad acceptance of external fixation devices. These devices do not provide the benefits of anatomical alignment and compression obtained by plate fixation or rod systems, and the rigid nature of the frames prevents the normal dynamics of fracture healing in children by inhibiting callus formation. Delayed fracture healing may cause refracture and present additional problems. Pin-track infections may also complicate the use of external fixation devices. Superficial infections should be treated aggressively with pin-track releases and antibiotics. Deep

infections are rare, but if they are present, drainage and antibiotic therapy are mandatory. Any skin tenting over the pins should be released at the time of application or at follow-up. [33]

Figure (17): Unilateral external fixator. [32]

Open reduction and internal fixation with plate and screws:

The use of AO compression plates for treatment of femoral shaft fractures in children between six and fourteen years of age, especially those with multi-system injuries, is another surgical alternative. Plate fixation offers the advantages of anatomical reduction, ease of insertion, simplified nursing care and rapid mobilization without casting. Its disadvantages include long incision, extensive dissection and periosteal stripping, infection, risk of implant failure, refractures and the need of another operation to remove the plate. [34] Overgrowth ranges from 0.5cm to 2.5cms especially in children younger than 12 years of age. Refracture may occur after removal of the implant due to bone atrophy under the plate.[35]

Intramedullary fixations: there are two types of intramedullary nailing, the first one is rigid intramedullary rod fixation and the second one is flexible intramedullary nailing.

Rigid intramedullary rod fixation:

Problems with angular malalignment and maintenance of length can be avoided with the use of rigid intramedullary fixation. Interlocking proximally and distally to maintain length and rotational alignment in unstable fractures, as in adults, appears to be of benefit to adolescents. Fractures treated by this method usually unite with no angular or rotational deformities and the nail can be removed after an average of 14 months.[35] However, rigid nailing is not routinely recommended unless the child is near skeletal maturity because of proximal femoral growth abnormalities, avascular necrosis of the femoral head, size of the proximal femur, and the relative success of other treatment methods.[37]

Flexible intramedullary nailing (Elastic stable intramedullary nailing):

The technique of elastic stable intramedullary nailing, adapted from existing flexible rod systems, was first described by surgeons from *Nancy*, France. The titanium nails (Figure 18) 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 children's fractures. Sometimes three or more flexible rods are inserted in order to better fill the medullary canal to enhance cortical contact, and provide more stable fixation. This constitutes a form of rigid intramedullary fixation, quite different from the *Nancy* nailing concept.[38]

Elastic stable intramedullary nailing is an accepted method of managing most, but not all, paediatric femoral fractures that need operative stabilization. It is indicated for fixation of diaphyseal femoral fractures in children between six and fourteen years of age. It is ideally suited for mid-diaphyseal transverse, short oblique or short spiral fractures with minimal comminution, more than 10° of varus-valgus angulation, more than 15° of anteroposterior angulation and more than 2 cm of shortening, and in children with multiple system injury, head injury, multiple long bone fractures, a floating knee injury and pre-existing pulmonary dysfunction. [1]

The flexible rod is initially bent or curved (plastically deformed). During intramedullary insertion, which is typically retrograde in the femur (Figure 18), 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. However, any significant imbalance in the magnitude or the direction of the moment created by the two nails will result in angulation of the fracture in the direction of the stronger nail. [12]

The most common complications reported are pain and skin irritation at the entry site associated with the prominence of the ends of the nails. Nail ends should not be bent, as was originally recommended, but advanced so that they lie against the supracondylar flare of the femur in order to avoid symptoms at the insertion site. Use of

nails of two different diameters is associated with a high rate of loss of reduction in the direction of the stronger rod. Multifragmentary fractures might be better stabilized by alternative methods of fixation. If used in comminuted fractures, these should be monitored weekly for early loss of reduction, and they might benefit from some additional external immobilization. Although the originators of this technique recommended routine removal of the nails, there is no evidence that this is necessary in the absence of nail-related symptoms.[12]

Figure (18): Titanium nails.

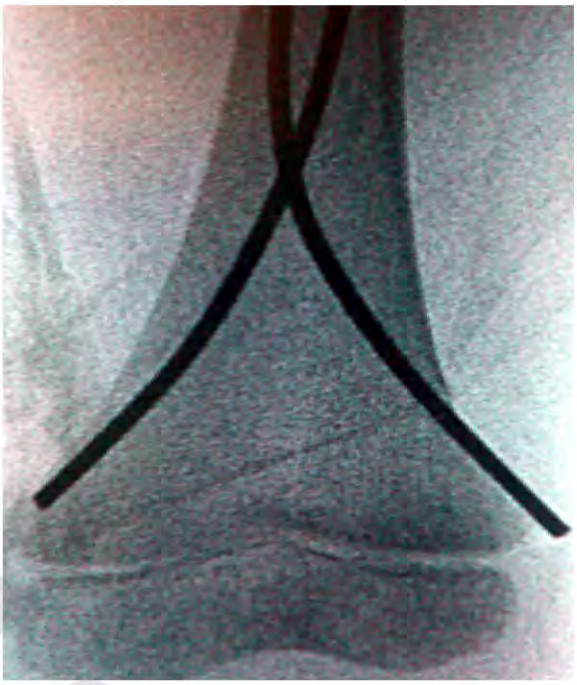


Figure (19): Retrograde flexible intramedullary nailing.

COMPLICATIONS OF FEMORAL SHAFT FRACTURES IN CHILDREN

I-Early Complications:

1. Shock:

One to two litres of blood in children can be lost even with a closed fracture, and shock may be severe. In most cases transfusion is required. [39]

2. Fat embolism:

This is an uncommon condition. It may be due to the sudden release of fat from the fracture site or due to coalescence of chylomicrons in response to injury. This can usually be accommodated without serious consequences, but in some cases (and especially in those with multiple injuries and severe shock) it results in progressive respiratory distress syndrome and multi-organ failure. Treatment is supportive, with the emphasis on preventing hypoxia and maintaining blood volume.[40]

3. Neurovascular injuries:

Nerve and vascular injuries are uncommon with femoral shaft fractures in children. Nerve abnormalities reported with femoral fractures in older children include those caused by direct trauma to the sciatic or femoral nerve at the time of fracture or injuries to peroneal nerve during treatment. If peroneal, femoral, or sciatic nerve deficit is present at initial evaluation of a closed fracture, no exploration is indicated. If a nerve deficit occurs during reduction or treatment, exploration of the nerve should be done. Persistent nerve loss without recovery over a 4- to 6-month period is an indication for exploration. [41]

4. Compartment syndrome:

Compartment syndromes of the thigh musculature are rare, because of large volume of the thigh compartments, which blend with those of the hip. Compartment syndrome occurs because of an increase in volume or pressure or because a force decreases the capacity of the compartments. It has been reported in patients with massive thigh swelling after femoral fracture and in patients treated with intramedullary rod fixation. If massive swelling of thigh musculature occurs and pain is out of proportion to that expected from a femoral fracture, compartment pressure measurements should be obtained and decompression by fasciotomy should be considered.[42]

II-Late Complications:

1. Angular deformity:

Some degree of angular deformity is frequent after femoral shaft fractures in children. Guidelines for acceptable alignment vary widely. The range of acceptable anterior and posterior angulation is up to 10° in older children and adolescents.[1] The range of acceptable varus and valgus angulation varies with age. Varus angulation in infants and children should be between 10° and 15°, although greater degrees of angulation may have a satisfactory outcome. Acceptable valgus angulation is 20° to 30° in infants, 15° to 20° in children up to age 5 years, and 10° in older children and adolescents. The muscle mass of the femur and remodelling with age generally hides femoral deformity from direct observation.[1]

However, the significant angular malunion is not common with long term follow up, because of the remodelling power that occurs with growth.[43] The basic mechanism which corrects angular deformities in growing bones is not well understood.

According to **Wolff's law**: Remodelling occurs at the fracture site with appositional new bone formation in the concavity of the long bone. Differential physeal growth also occurs in response to diaphyseal angular deformity. It is clear that angular remodelling occurs best in the direction of motion at the adjacent joint that is, anterior and posterior remodelling in the femur occurs rapidly and with little residual deformity. In contrast, remodelling of a varus or valgus deformity occurs more slowly.[44]

F0actors that may influence remodelling include age of the patient, direction of angulation21, fracture location within the femoral shaft and fracture configuration.[44]

2. **Rotational deformity:**

Rotational deformities of 10° to more than 30° occur in one third of children after conservative treatment of femoral shaft fractures.[45] Up to 25° of rotational malalignment at the time of healing of femoral fractures appears to be well tolerated in children.[46]

In older children, no significant remodelling will occur, but in infants and juveniles some rotational deformity can be accepted because either true rotational remodelling occurs or functional adaptation allows resumption of normal gait.

The goal, however, should be to reduce a rotational deformity to 10°, based on alignment of the proximal and distal femur radiologically, interpretation of skin and soft-tissue envelope alignment and the muscle pull on the proximal fragment. The distal fragment should be lined up with the position of the proximal fragment determined by the muscles inserted upon it.[1]

3. **Delayed union:**

Delayed union of femoral shaft fractures is uncommon in children. The time to fracture union in most children is rapid and age dependent. In children younger than 5 years of age, healing usually occurs in 4 to 6 weeks. In children between the ages of 5 and 10 years, fracture healing is somewhat slower, requiring 8 to 10 weeks. Throughout adolescence, the time to healing continues to lengthen. By the age of 15 years, the mean time to healing is about 13 weeks with range from 10 to 15 weeks. The rate of healing is also related to soft-tissue injury and type of treatment.[1]

4. **Non-union:**

Nonunions of paediatric femoral fractures are not very rare. They tend to occur in adolescents, in infected fractures, or in fractures with segmental bone loss or severe soft-tissue loss. Femoral fractures account for only 15% of nonunions in children. Even in segmental fractures with bone loss, young children may have sufficient osteogenic potential to fill in a significant fracture gap. For the rare femoral shaft nonunion in a child aged from five to ten years, bone grafting and plate-and-screw fixation have been traditional treatment methods, but more recently insertion of an interlocking intramedullary nail and bone grafting have been preferred, especially in children older than ten to twelve years of age.[47]

5. **Leg-length discrepancy:**

The most common sequel after femoral shaft fractures in children is leg-length discrepancy.

Shortening: The fractured femur may be initially short from overriding of the fragments at union; growth acceleration occurs to "catch up" the difference.

Lengthening: The potential for growth stimulation from femoral fractures has long been recognized, but the exact cause of this phenomenon is still unknown. Growth acceleration has been attributed to age, sex, fracture type, fracture level, and the amount of distraction of the fracture fragments. Fractures in the proximal third of the femur and oblique comminuted fractures have been associated with relatively greater growth acceleration. In children between 2 and 10 years of age, overgrowth is more likely, especially if traction has been used.[48]

Surgical intervention for correction of limb length discrepancy should be avoided for at least one year after injury or better until skeletal maturity compensation and remodelling to allow femoral growth to stabilize and an accurate determination of ultimate limb-length discrepancy to be ascertained.[43]

6. **Infection:**

Infection may rarely complicate a closed femoral shaft fracture, with haematogenous seeding of the haematoma and subsequent osteomyelitis. Fever is commonly associated with femoral fractures during the first week after injury, but persistent fever or fever that spikes exceedingly high may be an indication of infection.[49]

Pin-track infections occasionally occur with the use of skeletal traction or external fixator, but most are superficial infections that resolve with local wound care, antibiotic therapy and removal of pins. Occasionally, however, the infections may lead to osteomyelitis of the femoral metaphysis or a ring sequestrum that requires surgical debridement.[1]

7. **Muscle weakness:**

Weakness after femoral fracture has been described in the hip abductor musculature, the quadriceps, and the hamstrings, but persistent weakness in some or all of these muscle groups seldom causes a clinical problem. These deficits are related to the degree of initial displacement of the fracture. This weakness is present whether patients are treated operatively or nonoperatively. Injury to the quadriceps muscle probably occurs at the time of femoral fracture, and long-term muscle deficits may persist in some patients regardless of treatment. Severe scarring and contracture of the quadriceps occasionally require quadricepsplasty.[1]

8. **Proximal femoral physis injury:**

Antegrade rodding may result in growth disturbance in the proximal femur leading to coxa valga and mild hip subluxation from trochanteric physeal arrest. Also rod insertion may result in femoral neck fracture and avascular necrosis of the femoral head (as the space between the trochanter and the femoral head is extremely narrow in children which leads to vascular disruption).[1]