CHAPTER ONE

1-Introduction

Deep venous thrombosis (DVT) is a common disease with potentially serious consequences such as pulmonary embolism (PE). It should be noted that venous thromboembolism (VTE) includes both DVT and PE. (Oger, 2000) (Murphy et al., 2015)

Venous thromboembolic disease in hospitalized patients results in substantial mortality, morbidity, and healthcare resource use. While the true incidence of venous thromboembolism (VTE) is difficult to determine, autopsy studies have shown that 5–10 % of hospital deaths are attributable to pulmonary embolism. Major orthopedic surgery is associated with a very high risk of VTE. (Kakkar et al., 1969) (Kakkar.,1972).

The incidence of DVT in the general population is between 1.6 and 1.8 per 1.000 per year. (Oger, 2000) .Three factors, namely, blood flow, hypercoagulability, and endothelial injury play an important role in the process of thrombus formation and they are referred to as Virchow's triad. Notably, major trauma often precipitates one or all of these risk factors. (Kitagawa and Sakoda, 2009). Trauma patients often have all three of these factors, which causes a high risk of thromboembolism. Direct injury to blood vessels can cause intimal damage, leading to thrombosis, prolonged bed rest, immobilization, hypo perfusion, and paralysis, all promoting venous stasis. (Hak, 2001)

The association between injury and VTE is well recognized, and the reported incidence of VTE after trauma varies from 7% to 58% depending on patient demographics, kind of injury, method of detection, and type of VTE prophylaxis used. (Knudson et al., 2004)

Vascular injuries constitute less than 3% of all traumatic findings, yet they are associated with potentially serious events. Injuries of the greater blood vessels may have devastating complications resulting in amputations or even death. An understanding of the mechanism of trauma is of utmost importance for accurate diagnosis. The majority of vessel injuries affect the upper and lower limbs. Vessel injury is a serious problem for physicians, as some vascular lesions may not be initially

recognized based on clinical evaluation and vital signs. Thus, imaging and follow-up should be the basis for diagnosis and care. (Rozycki et al., 2003)

Angiography is the gold standard method for the examination of trauma-related vascular complications; however, noninvasive diagnostic modalities such as computed tomography angiography (CTA), magnetic resonance angiography (MRA), and venous duplex ultrasound (VDUS) have recently emerged as adequate alternatives. (Gaitini et al., 2008) VDUS uses two components to assess for DVT, namely, the brightness mode (B-mode) imaging with probe compression and Doppler evaluation including color flow Doppler imaging and spectral Doppler waveform analysis. In VDUS, the B-mode imaging is applied while the lower limb veins are compressed along their extent with a US transducer for presentation of an intraluminal thrombus. A patent vein will exhibit whole vein wall coaptation on compression with the US probe .Absence of compressibility is the most reliable indicator of an existing of thrombus within the studied vein. (Gornik and Sharma, 2014) (Kassai et al., 2004)

Color flow Doppler is useful for examining residual flow within a thrombosed venous section and for verifying patency of venous segments that are not approachable for compression. The pulsed Doppler spectral waveform in a normal lower limb vein exhibits spontaneous and respiro-phasic flow pattern. An alteration of this flow pattern might include obstruction proximally to the level of probe location. Also, respirophasicity could be assessed by distal augmentation maneuvers during spectral Doppler evaluation to further demonstrate patency of the veins. While the distal augmentation maneuver is applied, there should be a sharp “spike” of augmented anterograde venous flow. Blunted or absence of flow augmentation indicates venous obstruction distally. (Gornik and Sharma, 2014) (Kassai et al., 2004)

1.1-The Statement of the problem

Lower limb DVT becomes a significant challenge in traumatic patients which may cause pulmonary embolism or at least may delay orthopedic surgery. It can cause pain and swelling which were the same symptoms of fracture symptoms this condition increase difficulty of the clinical diagnosis thus the duplex ultrasound plays a great rule to exclude the DVT because it's simple, fast and non invasive procedure.

1.2-The objectives of the study:-

1-The aim of the current study was to evaluate the role of duplex ultrasound (DUS) in the evaluation of deep venous blood flow to diagnose the DVT in fractured lower extremities

2-To know the prevalence of DVT in the target group

3-To correlate between the deep vein thrombosis and LL fracture location

4-To identify the link between DVT and other risk factor in patients with LL fracture

5-To identify the link between LL fracture types and DVT

6-To find out the relationship between the DVT and type of fixation

7-To know the relationship between the DVT and types of blood groups

1.3-Thesis outline

The thesis is outlined into five chapters as follows: Chapter one general introduction of DVT and PE its criteria scores, and objectives of the study. Chapter two is devoted to the literature review to the previous local and international studies, anatomy physiology and pathology. Chapter three Materials and Methods features of the machine used in the study and patients sample and characteristics, inclusion and exclusion criteria. Chapter four represents the results, and data collected from the investigation. Chapter five discusses the findings of the study, gives some conclusions. And brief recommendations for future research are also specified

1.4-Thesis outcome

The following papers were published and prepared during this study:

1. Awadalla Adam, Mohamed Yousef1, Babiker A.Wahab1, Ahmed Abukonna, Mustafa Z. Mahmoud Duplex Ultrasound for Evaluation of Deep Venous Blood Flow in Fractured Lower Extremities © Polish Journal of Radiology, 2017; 82: DOI: 10.12659/PJR.904445 .(Appendix D)

2. Awadalla Adam M. Yousef, Babiker A.Wahab1, Ahmed Abukonna, Mohammed Elfadil Incidence of Deep Venous Thrombosis Following Fixation of Fractured Lower Extremity

CHAPTER TWO
Literature Review

2. Literature Review

2.1-Anatomy

2.1.1-Lower limb bones

The bones of the lower limb are the three fused components of the pelvic girdle; the femur and patella (thigh); the tibia and fibula (leg); the tarsus, metatarsus and phalanges (foot) The hip bones (especially the ilium and ischium), femur, tibia and bones of the hind foot are strong and their external (cortical) and internal (trabecular) structure is adapted for weight bearing. The free lower limb is connected to the pelvic girdle by the hip joint. It consists of the thigh bone (femur), the (lower) leg (crus) including tibia and fibula, and the foot (pes), which includes the ankle (tarsus), metatarsals, and toes (digits). The movements between femur and lower leg occur at the knee joint (genu), which is a principal factor in the movement sequence of walking. The tibia and fibula are connected to the foot by the ankle joint. Together with the mid-tarsal (talo calcaneo navicular) joint and the other joints of the foot, this freely mobile joint enables the foot to roll during walking. (Susan Standring2008) (A Faller et al 2004)

The pelvic girdle connects the lower limb to the axial skeleton via the sacroiliac joint, a synovial joint in which mobility has been sacrificed for stability and strength, to allow for effective weight transmission from the trunk to the lower limb. Anteriorly, the pelvic girdle articulates with the contralateral girdle at the pubic symphysis, a secondary cartilaginous joint that may display a slight degree of mobility during hip and sacroiliac movement and during childbirth. The hip joint, a synovial ball-and-socket joint, exhibits a very effective compromise between mobility and stability that allows movement in all three orthogonal planes. The more distal

joints have gained mobility at the expense of stability the knee joint includes the patella-femoral articulation, which allows the patella to glide over the distal femur. However, the main component of the knee joint is a bi-compartmental synovial articulation between the femur and the tibia that allows flexion, extension and some medial and lateral rotation of the leg. It is not a true hinge joint because its axes of flexion and extension are variable and there is coupled rotation. The tibia and fibula articulate with each other at the superior and inferior tibiofibular joints. The superior joint, a plane synovial joint, allows slight gliding movement only. The inferior joint, a fibrous joint, lies just above the ankle and allows a degree of fibular rotation linked to ankle motion. The ankle (talo-crural) joint is formed by the distal ends of the tibia and fibula ‘gripping' the talus, and allows dorsiflexion and plantar flexion. There are multiple joints in the foot that may be classified topographically on the basis of whether they are in the hind foot, mid foot or forefoot. Collectively, these joints allow the complex movements required as the foot fulfils its functional roles as a platform for standing and for shock absorption and propulsion in gait. Both knee and ankle are commonly subject to closed injuries, and the relatively superficial location of the knee renders it susceptible to open injury and infection. Although the ankle is frequently injured and is a major loadbearing joint, the incidence of clinically significant degenerative arthritis is surprisingly low when compared with that found in the hip and knee (Susan Standring2008)

2.1.1.1 Long Bones

Bones are classified according to their shape. Long bones are longer than they are wide. Short bones are cube shaped—that is, their lengths and widths are about equal. Flat bones, such as those of the skull, are plate-like with broad surfaces. Irregular bones have varied shapes that permit connections with other bones. Round bones are circular in shape. A long bone, can be used to illustrate certain principles of bone anatomy. The bone is enclosed in a tough, fibrous, connective tissue covering called the **periosteum,** which is continuous with the ligaments and tendons that anchor bones. The periosteum contains blood vessels that enter the bone and service its cells. At both ends of a long bone is an expanded portion called an **epiphysis;** the portion between the epiphyses is called the **diaphysis.** The diaphysis is not solid but has a **medullary cavity** containing yellow marrow. Yellow marrow contains large amounts of fat.

The medullary cavity is bounded at the sides by compact bone. The epiphyses contain spongy bone. Beyond the spongy bone is a thin shell of compact bone and, finally, a layer of hyaline cartilage called the **articular cartilage.** Articular cartilage is so named because it occurs where bones articulate (join). **Articulation** is the joining together of bones at a joint. The medullary cavity and the spaces of spongy bone are lined with endosteum, a thin, fibrous membrane. (Mader 2004)

2.1.1.2 Compact bone, or dense bone, contains many cylinder shaped units called osteons. The osteocytes (bone cells) are in tiny chambers called *lacunae* that occur between concentric layers of matrix called lamellae. The matrix contains collagenous protein fibers and mineral deposits, primarily of calcium and phosphorus salts. In each osteon, the lamellae and lacunae surround a single central canal. Blood vessels and nerves from the periosteum enter the central canal. The osteocytes have extensions that extend into passageways called canaliculi, and thereby the osteocytes are connected to each other and to the central canal. (Mader 2004)

2.1.1.3 Spongy bone, or cancellous bone, contains numerous bony bars and plates, called trabeculae. Although lighter than compact bone, spongy bone is still designed for strength. Like braces used for support in buildings, the trabeculae of spongy bone follow lines of stress. In infants, **red bone marrow,** a specialized tissue that produces blood cells, is found in the cavities of most bones. In adults, red blood cell formation, called **hematopoiesis,** occurs in the spongy bone of the skull, ribs, sternum (breastbone), and vertebrae, and in the ends of the long bones. (Mader 2004)

2.1.1.4 Femur

The femur is the longest and strongest bone in the human body. Its length is associated with a striding gait, its strength with the weight and muscular forces it is required to withstand. Its shaft, almost cylindrical along most of its length, is bowed forward. It has a proximal rounded, articular head projecting medially from its short neck, which, in turn, is a medial extension of the proximal shaft. The distal extremity is wider and more substantial, and presents a double condyle that articulates with the tibia. In standing, the femoral shafts show an inclination upwards and outwards from their tibial articulations, with the femoral heads being

separated by the pelvic width. Since the tibia and fibula descend vertically from the knees, the ankles are also in the line of body weight in standing or walking. The degree of femoral obliquity varies between individuals, but is generally greater in women, reflecting the relatively greater pelvic breadth and shorter femora. Proximally the femur consists of a head, neck, and greater and lesser trochanters.(Susan Standring2008)
The thighbone (femur) is the longest and strongest bone of the human skeleton. Proximally, the shaft of the femur (diaphysis) continues into the femoral neck (collum femoris), which is directed obliquely upward and which ends in the head of the femur (caput femoris). In the adult the angle of the neck to the shaft (collo diaphyseal angle) is about125−126°. In the new born the angle is markedly greater (ca.150°), while in the elderly it may be smaller than126°. At the junction between the shaft and neck there are two processes, the robust externally directed greater trochanter, and the somewhat smaller internally directed lesser trochanter. Both trochanters are the site of muscle insertions. Anteriorly and laterally, the femoral shaft is smooth, while along its posterior aspect runs a rough line (linea aspera) with an inner and an outer lip for muscle insertions. Distally, the femur expands to form the two femoral condoyles, extensively covered by hyaline cartilage. Anteriorly, they are extended into a cartilaginous groove, the patellar surface, in which the kneecap (patella) glides downward during flexion of the knee. Posteriorly, the two condyles are separated by abroad groove, the intercondylar notch (inter condylar fossa). (A Faller et al 2004)

2.1.1.4.1 Femoral head

The femoral head faces anterosuperomedially to articulate with the acetabulum. The head, often described as rather more than half a ‘sphere', is not part of a true sphere but is spheroidal. Its smoothness is interrupted posteroinferior to its centre by a small, rough fovea. The head is intracapsular and is encircled, distal to its equator, by the acetabular labrum. Its articular margin is distinct, except anteriorly, where the articular surface extends on to the neck. The ligamentum teres is attached to the fovea. The anterior surface of the head is separated inferomedially from the femoral artery by the tendon of psoas major, the psoas bursa, and the articular capsule. (Susan Standring2008)

As the femur is the longest bone in the human body the long bones develop centers of ossification in their epiphyses. At birth, ossification is not yet complete and continues throughout childhood. In long bones, growth occurs in the epiphyseal discs at the junction of the diaphysis with each epiphysis. An epiphyseal disc is still cartilage, and the bone grows in length as more cartilage is produced on the epiphysis side. On the diaphysis side, osteoblasts produce bone matrix to replace the cartilage. Between the ages of 16 and 25 years (influenced by estrogen or testosterone), all of the cartilage of the epiphyseal discs is replaced by bone. This is called closure of the epiphyseal discs (or we say the discs are closed), and the bone lengthening process stops. Also in bones are specialized cells called osteoclasts (a *clast* cell is a "destroying" cell), which are able to dissolve and reabsorb the minerals of bone matrix, a process called resorption. Osteoclasts are very active in embryonic long bones, and they reabsorb bone matrix in the center of the diaphysis to form the marrow canal. Blood vessels grow into the marrow canals of embryonic long bones, and red bone marrow is established. After birth, the red bone marrow is replaced by yellow bone marrow. Red bone marrow remains in the spongy bone of short, flat, and irregular bones. (Scanlon & Sanders 2008)

2.1.1.4.2 Femoral neck

The femoral neck is approximately 5 cm long, narrowest in its mid part and widest laterally, and connects the head to the shaft at an average angle of 135° (angle of inclination; neck–shaft angle): this facilitates movement at the hip joint, enabling the limb to swing clear of the pelvis. The neck also provides a lever for the action of the muscles acting about the hip joint, which are attached to the proximal femur. The neck–shaft angle is widest at birth and diminishes gradually until adolescence; it is smaller in females. The neck is laterally rotated with respect to the shaft (angle of anteversion) some 10–15°, although values of this angle vary between individuals and between populations (Eckhoff et al 1994). The contours of the neck are rounded: the upper surface is almost horizontal and slightly concave, the lower is straighter but oblique, directed inferolaterally and backwards to the shaft near the lesser trochanter. On all aspects the neck expands as it approaches the articular surface of the head. The anterior surface of the neck is flat and marked at the junction with the shaft by a rough intertrochanteric line. The posterior surface,

facing posteriorly and superiorly, is transversely convex, and concave in its long axis; its junction with the shaft is marked by a rounded intertrochanteric crest. There are numerous vascular foramina, especially anteriorly and posterosuperiorly. The anterior surface is intracapsular, the capsule attaching laterally to the intertrochanteric line. Facets, often covered by extensions of articular cartilage, and various imprints frequently occur here. These facets may sometimes be associated with squatting. One such feature, the cervical fossa, may be a racial characteristic. On the posterior surface the capsule does not reach the intertrochanteric crest; little more than the medial half of the neck is intracapsular. The anterior surface adjoining the head and covered by cartilage is related to the iliofemoral ligament. A groove, produced by the tendon of obturator externus as it approaches the trochanteric fossa, spirals across the posterior surface of the neck in a proximolateral direction. (Susan Standring2008)

2.1.1.4.3 Greater trochanter

At the proximal end of the femur are the greater and lesser trochanters, large projections that are anchors for muscles.The greater trochanter is large and quadrangular, projecting up from the junction of the neck and shaft. Its posterosuperior region projects superomedially to overhang the adjacent posterior surface of the neck and here its medial surface presents the rough trochanteric fossa. The proximal border of the trochanter lies approximately a hand's breadth below the iliac tubercle, level with the centre of the femoral head. It has an anterior rough impression. Its lateral surface, continuous distally with the lateral surface of the femoral shaft, is crossed anteroinferiorly by an oblique, flat strip, which is wider above. This surface is palpable, especially when the muscles are relaxed. The trochanteric fossa occasionally presents a tubercle or exostosis. (Susan Standring2008) (Scanlon & Sanders 2008)

2.1.1.4.4 Lesser trochanter

The lesser trochanter is a conical posteromedial projection of the shaft at the posteroinferior aspect of its junction with the neck. Its summit and anterior surface are rough, but its posterior surface, at the distal end of the intertrochanteric crest, is smooth. It is not palpable. (Susan Standring2008)

2.1.1.4.5 Intertrochanteric line

The intertrochanteric line, a prominent ridge at the junction of the anterior surfaces of the neck and shaft, descends medially from a tubercle on the upper part of the anterior aspect of the greater trochanter to a point on the lower border of the neck, anterior to the lesser trochanter, where there may also be a tubercle. This line is the lateral limit of the hip joint capsule anteriorly. The upper and lower bands of the iliofemoral ligament are attached to its proximal and distal ends and the associated tubercles. Distally it is continuous with the spiral line. (Susan Standring2008)

2.1.1.4.6 Intertrochanteric crest

The intertrochanteric crest, a smooth and prominent ridge at the junction of the posterior surface of the neck with the shaft, descends medially from the posterosuperior angle of the greater trochanter to the lesser trochanter. A little above its centre is a low, rounded quadrate tubercle. It is covered by gluteus maximus, from which it is separated, medial to the tubercle, by quadratus femoris and the upper border of adductor magnus. (Susan Standring2008)

2.1.1.4.7 Gluteal tuberosity

The gluteal tuberosity may be an elongated depression or a ridge. It may at times be prominent enough to merit the unofficial title of ‘third trochanter'. (Susan Standring2008)

2.1.1.4.8 Shaft

The shaft is surrounded by muscles and is impalpable. The distal anterior surface, for 5–6 cm above the patellar articular surface, is covered by a suprapatellar bursa, between bone and muscle. The distal lateral surface is covered by vastus intermedius. The medial surface, devoid of attachments, is covered by vastus medialis. The shaft is narrowest centrally, expanding a little at its proximal end, and substantially more at its distal end. Its long axis makes an angle of approximately 10° with the vertical, and diverges 5–7° from the long axis of the tibia. Its middle third

has three surfaces and borders. The extensive anterior surface, smooth and gently convex, is between the lateral and medial borders, which are both round and indistinct. The posterolateral surface is bounded posteriorly by the broad, rough linea aspera, usually a crest with lateral and medial edges. Its subjacent compact bone is augmented to withstand compressive forces, which are concentrated here by the anterior curvature of the shaft. The linea aspera gives attachment to adductor longus intermuscular septa and the short head of biceps femoris, all inseparably blended at their attachments. Perforating arteries cross the linea laterally under tendinous arches in adductor magnus and biceps femoris. Nutrient foramina, directed proximally, appear in the linea aspera, varying in number and site, one usually near its proximal end, a second usually near its distal end. The posteromedial surface, smooth like the others, is bounded in front by the indistinct medial border and behind by the linea aspera. In its proximal third the shaft has a fourth, posterior surface, bounded medially by a narrow, rough spiral line that is continuous proximally with the intertrochanteric line and distally with the medial edge of linea aspera. Laterally this surface is limited by the broad, rough, gluteal tuberosity, ascending a little laterally to the greater trochanter and descending to the lateral edge of the linea aspera. In its distal third the posterior surface of the shaft presents a further surface, the popliteal surface between the medial and lateral supracondylar lines. These lines are continuous above with the corresponding edges of the linea aspera. The lateral line is most distinct in its proximal two-thirds, where the short head of biceps femoris and lateral intermuscular septum are attached. Its distal third has a small rough area for the attachment of plantaris, often encroaching on the popliteal surface. The medial line is indistinct in its proximal two-thirds, where vastus medialis is attached. Distally, the medial line is crossed obliquely by the femoral vessels entering the popliteal fossa from the adductor canal. Further distally, the line is often sharp for 3 or 4 cm proximal to the adductor tubercle. (Susan Standring2008)

The popliteal surface, triangular in outline, lies between the medial and lateral supracondylar lines. In its distal medial part it is rough and slightly elevated. Forming the proximal part of the floor of the popliteal fossa, the popliteal surface is covered by a variable amount of fat that separates the popliteal artery from bone. The superior medial genicular artery, a branch

of the popliteal artery, arches medially above the medial condyle. It is separated from bone by the medial head of gastrocnemius. The latter is attached a little above the condyle; further distally there may be a smooth facet underlying a bursa for the medial head of gastrocnemius. More medially, there is often an imprint proximal to the articular surface: in flexion this is close to a rough tubercle on the medial tibial condyle for the attachment of semimembranosus. The superior lateral genicular artery arches up laterally proximal to the lateral condyle but is separated from bone by the attachment of plantaris to the distal part of the lateral supracondylar line. (Susan Standring2008)

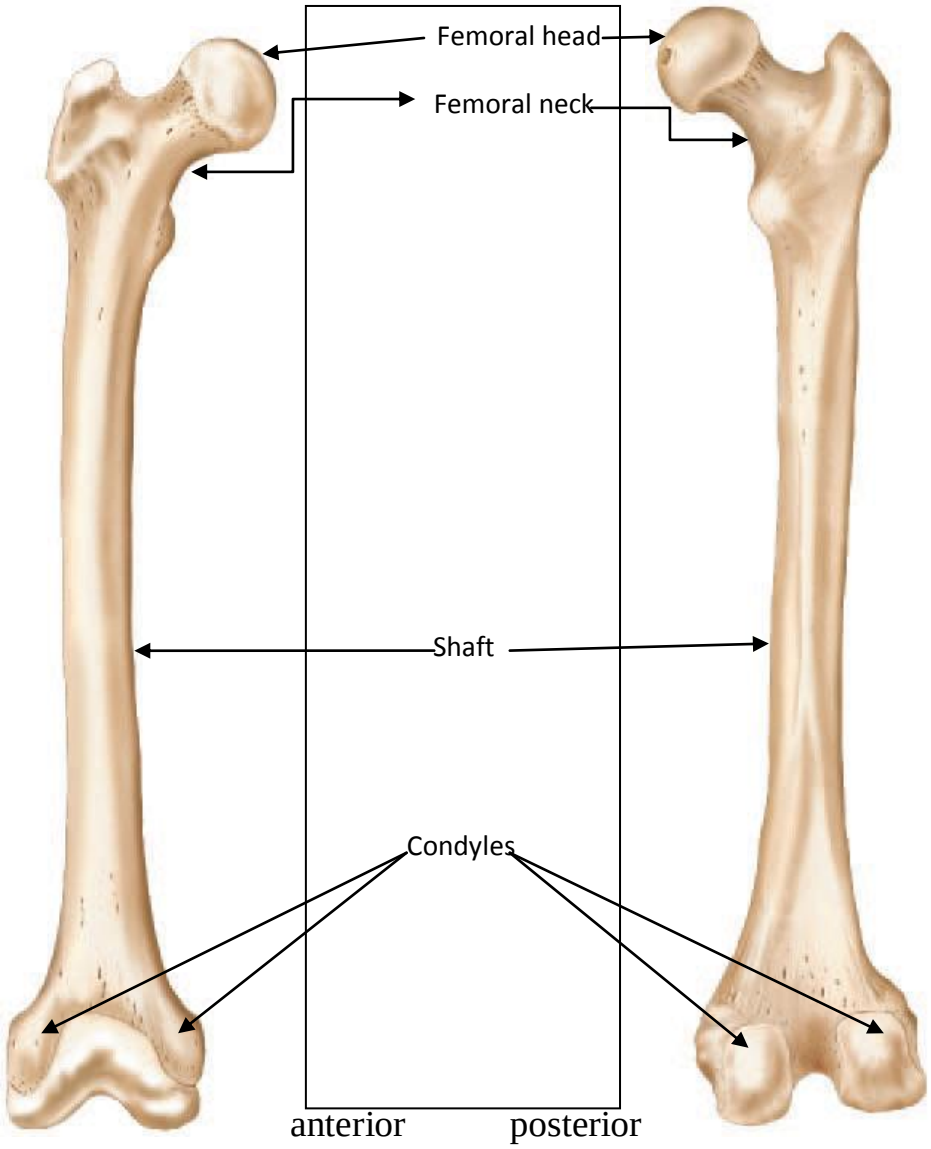


Figure (2-1) **Right femur. a. Anterior view. b. Posterior view Understanding** Human Anatomy & Physiology, Mader 2004 Page No 102)

2.1.1.4.9 Distal end

The distal end of the femur is widely expanded as a bearing surface for transmission of weight to the tibia. It bears two massive condyles, which are partly articular. Anteriorly the condyles are confluent and continue into the shaft; posteriorly they are separated by a deep intercondylar fossa and project beyond the plane of the popliteal surface. The articular surface is a broad area, like an inverted U, for the patella and the tibia. The patellar surface extends anteriorly on both condyles, especially the lateral. It is transversely concave, vertically convex and grooved for the posterior patellar surface. The tibial surface is divided by the intercondylar fossa but is anteriorly continuous with the patellar surface. Its medial part is a broad strip on the convex inferoposterior surface of the medial condyle, and is gently curved with a medial convexity. Its lateral part covers similar aspects of the lateral condyle but is broader and passes straight back. The tibial surfaces are convex in all directions. The medial and lateral tibial surfaces have dissimilar anteroposterior curvatures. However views differ as to the exact representation of these differences. One view holds that in both tibial portions of the femoral condyles the sagittal radius of curvature is ever decreasing (a ‘closing helix'). More recently it has been suggested that the medial articular surface describes arcs of two circles. The more posterior has a smaller radius. Laterally there may only be one arc of fixed curvature with a radius similar to that of the posterior arc of the medial femoral articular surface. These differences are believed to be important determinants of knee joint motion. (Susan Standring2008)

2.1.1.4.10 Patellar surface (trochlear groove)

The patellar surface extends more proximally on the lateral side. Its proximal border is therefore oblique and runs distally and medially, separated from the tibial surfaces by two faint grooves that cross the condyles obliquely. The lateral groove is the more distinct. It runs laterally and slightly forwards from the front of the intercondylar fossa and expands to form a faint triangular depression, resting on the anterior

edge of the lateral meniscus when the knee is fully extended. The medial groove is restricted to the medial part of the medial condyle and rests on the anterior edge of the medial meniscus in full extension. Where it ceases, the patellar surface continues back to the lateral part of the medial condyle as a semilunar area adjoining the anterior region of the intercondylar fossa. This area articulates with the medial vertical facet of the patella in full flexion; its outline is indistinct in most femora. In habitual squatters articular cartilage may extend to the lateral aspect of the lateral condyle under vastus lateralis. The trochlear groove helps to stabilize the patella. An abnormally shallow groove predisposes to instability. (Susan Standring2008)

2.1.1.4.11 Intercondylar fossa

The intercondylar fossa separates the two condyles distally and behind. In front it is limited by the distal border of the patellar surface, and behind by an intercondylar line, separating it from the popliteal surface. It is intracapsular but largely extrasynovial. Its lateral wall, the medial surface of the lateral condyle, bears a flat posterosuperior impression which spreads to the floor of the fossa near the intercondylar line for the proximal attachment of the anterior cruciate ligament. The medial wall of the fossa, i.e. the lateral surface of the medial condyle, bears a similar larger area, but far more anteriorly, for the proximal attachment of the posterior cruciate ligament. Both impressions are smooth and largely devoid of vascular foramina, whereas the rest of the fossa is rough and pitted by vascular foramina. A bursal recess between the ligaments may ascend to the fossa. The capsular ligament and, laterally, the oblique popliteal ligament, are attached to the intercondylar line. The ligamentum mucosum (infrapatellar synovial fold or plica) is attached to the anterior border of the fossa. (Susan Standring2008)

2.1.1.4.12 Lateral condyle

The lateral condyle is larger anteroposteriorly than the medial. Its most prominent point is the lateral epicondyle to which the lateral collateral ligament is attached. A short groove, deeper in front, separates the lateral epicondyle inferiorly from the articular margin. This groove allows the tendon of popliteus to run deep to the lateral collateral ligament and insert

inferior and anterior to the ligament insertion. Adjoining the joint margin is a strip of condyle, 1 cm broad. It is intracapsular and covered by synovial membrane except for the attachment of popliteus. The medial surface is the lateral wall of the intercondylar fossa. Its lateral surface projects beyond the shaft. Part of the lateral head of gastrocnemius is attached to an impression posterosuperior to the lateral epicondyle. (Susan Standring2008)

2.1.1.4.13 Medial condyle

The medial condyle has a bulging, convex medial aspect which is easily palpable. Proximally its adductor tubercle, which may only be a facet rather than a projection, receives the tendon of adductor magnus. The medial prominence of the condyle, the medial epicondyle, is anteroinferior to the tubercle. The lateral surface of the condyle is the medial wall of the intercondylar fossa. The condyle projects distally so that, despite the obliquity of the shaft, the profile of the distal end is almost horizontal. A curved strip, 1 cm wide, adjoining the medial articular margin, is covered by synovial membrane and is inside the joint capsule. Proximal to this, the medial epicondyle receives the medial collateral ligament (Susan Standring2008)

2.1.1.4.14 Structure

The femoral shaft is a cylinder of compact bone with a large medullary cavity. The wall is thick in its middle third, where the femur is narrowest and the medullary cavity most capacious. Proximally and distally the compact wall becomes progressively thinner, and the cavity gradually fills with trabecular bone. The extremities, especially where articular, consist of trabecular bone within a thin shell of compact bone, their trabeculae being disposed along lines of greatest stress. At the proximal end the main trabeculae form a series of plates orthogonal to the articular surface, converging to a central dense wedge, which is supported by strong trabeculae passing to the sides of the neck, especially along its upper and lower profiles. Force applied to the femoral head is therefore transmitted to the wedge and thence to the junction of the neck and shaft. This junction is strengthened by dense trabeculae extending laterally from the lesser trochanter to the end of the superior aspect of the neck, thus

resisting tensile or shearing forces applied to the neck through the head. Tensile and compressive tests indicate that axial trabeculae of the femoral head withstand much greater stresses than peripheral trabeculae. A smaller bar across the junction of the greater trochanter with the neck and shaft resists shearing produced by muscles attached to it. These two bars are proximal layers of arches between the sides of the shaft and transmit to it forces applied to the proximal end. A thin vertical plate, the calcar femorale, ascends from the compact wall near the linea aspera into the trabeculae of the neck. Medially it joins the posterior wall of the neck; laterally it continues into the greater trochanter, where it disperses into general trabecular bone. It is thus in a plane anterior to the trochanteric crest and base of the lesser trochanter. At the distal end of the femur, trabeculae spring from the entire internal surface of compact bone, descending perpendicular to the articular surface. Proximal to the condyles these are strongest and most accurately perpendicular. Horizontal planes of trabecular bone, arranged like crossed girders, form a series of cubical compartments (Susan Standring2008)

2.1.1.5-Patella

The patella is the largest sesamoid bone and is embedded in the tendon of quadriceps femoris, anterior to the distal femur (femoral condyles). It is flat, distally tapered, proximally curved, and has anterior and posterior surfaces, three borders and an apex which is the distal end of the bone. With the knee in extension, the apex is just proximal to the line of the knee joint. The subcutaneous, convex anterior surface is perforated by nutrient vessels. It is longitudinally ridged, separated from the skin by a prepatellar bursa, and covered by an expansion from the tendon of quadriceps femoris, which blends distally with superficial fibres of the patellar tendon (patellar ligament), the continuation of the tendon of quadriceps. The posterior surface has a proximal smooth, oval articular area, crossed by a smooth vertical ridge, which fits the intercondylar groove on the femoral patellar surface and divides the patellar articular area into medial and lateral facets; the lateral is usually larger. Each facet is divided by faint horizontal lines into equal thirds. A seventh ‘odd' facet is present as a narrow strip along the medial border of the patella; it contacts the medial femoral condyle in extreme flexion. Distal to the articular surface, the apex is roughened by the attachment of the patellar

tendon. Proximal to this, the area between the roughened apex and the articular margin is covered by an infrapatellar pad of fat. The articular cartilage is the thickest in the body, reflecting the magnitude of the stresses to which it is subjected. The thick superior border (surface) slopes anteroinferiorly. The medial and lateral borders are thinner and converge distally; the expansions of the tendons of vastus medialis and lateralis (medial and lateral patellar retinacula respectively) are attached to them. The lateral retinaculum receives contributions from the iliotibial tract. Ossification occasionally extends from the lateral margin of the patella into the tendon of vastus lateralis. The shape of the patella can vary, and certain configurations are associated with patellar instability. Not infrequently a bipartite patella is seen on radiographs. The bone seems to be in two parts, usually with a smaller superolateral fragment: this has long been attributed to the presence of a separate ossification centre, but in some structure. The patella consists of more or less uniformly dense trabecular bone, covered by a thin compact lamina. Trabeculae beneath the anterior surface are parallel to the surface; elsewhere they radiate from the articular surface into the substance of the bone. Ases could represent failed union following a stress fracture. (Susan Standring2008)

2.1.1.6-The Leg

The skeleton of the lower leg is formed by the tibia and the fibula. The tibia is the stronger bone and the true supporting pillar and connection between the femur and the foot. Proximally, the two bones are connected by the strong ligamentous tibiofibular joint. Distally, the two bones are connected by a fibrous joint(syn- desmosis). The mortise formed by the medial and lateral malleoli of these bones, together with the trochlea of the talus, makes up the ankle joint. Proximally, the tibia expands and features on its anterior surface the tibial tuberosity, into which the quadriceps femoris is inserted. The superior articular surface formed by the medial and lateral condyles, forms the knee joint with the femur. Like the radius and ulna, the tibia and fibula are connected by an interosseus membrane, which gives origin to some muscles in the leg.
(A Faller et al 2004)

2.1.1.6.1-The Tibia & Proximal End

The tibia lies medial to the fibula and is exceeded in length only by the femur. Its shaft is triangular in section and has expanded ends; a strong medial malleolus projects distally from the smaller distal end. The anterior border of the shaft is sharp and curves medially towards the medial malleolus. Together with the medial and lateral borders it defines the three surfaces of the bone. The exact shape and orientation of these surfaces show individual and racial variations. The expanded proximal end is a bearing surface for body weight, which is transmitted through the femur. It consists of medial and lateral condyles, an intercondylar area and the tibial tuberosity. (Susan Standring2008)

2.1.1.6.2-Condyles

The tibial condyles overhang the proximal part of the posterior surface of the shaft. Both condyles have articular facets on their superior surfaces that are separated by an irregular, non-articular intercondylar area. The condyles are visible and palpable at the sides of the patellar tendon, the lateral being more prominent. In the passively flexed knee the anterior margins of the condyles are palpable in fossae that flank the patellar tendon. The fibular facet on the posteroinferior aspect of the lateral condyle faces distally and posterolaterally. The angle of inclination of the superior tibiofibular joint varies between individuals, and may be horizontal or oblique. Superomedial to it the condyle is grooved on its posterolateral aspect by the tendon of popliteus; a synovial recess intervenes between the tendon and bone. The anterolateral aspect of the condyle is separated from the lateral surface of the shaft by a sharp margin for the attachment of deep fascia. The distal attachment of the iliotibial tract makes a flat but definite marking, Gerdy's tubercle, on its anterior aspect. This tubercle, which is triangular and facet-like, is usually palpable. The anterior condylar surfaces are continuous with a large triangular area whose apex is distal and formed by the tibial tuberosity. The lateral edge is a sharp ridge between the lateral condyle and lateral surface of the shaft. (Susan Standring2008)

2.1.1.6.3-Tibial Tuberosity

The tibial tuberosity is the truncated apex of a triangular area where the anterior condylar surfaces merge. It projects only a little, and is divided into a distal rough and a proximal smooth region. The distal region is palpable and is separated from skin by the subcutaneous infrapatellar bursa. A line across the tibial tuberosity marks the distal limit of the proximal tibial growth plate. The patellar tendon is attached to the smooth bone proximal to this, its superficial fibres reaching a rough area distal to the line. The deep infrapatellar bursa and fibroadipose tissue intervene between the bone and tendon proximal to its site of attachment. The latter may be marked distally by a somewhat oblique ridge, onto which the lateral fibres of the patellar tendon are inserted more distally than the medial fibres. In habitual squatters a vertical groove on the anterior surface of the lateral condyle is occupied by the lateral edge of the patellar tendon in full flexion of the knee. (Susan Standring2008)

2.1.1.6.4-Shaft

The shaft is triangular in section and has (antero) medial, lateral and posterior surfaces separated by anterior, lateral (interosseous) and medial borders. It is narrowest at the junction of the middle and distal thirds, and expands gradually towards both ends. The anterior border descends from the tuberosity to the anterior margin of the medial malleolus and is subcutaneous throughout. Except in its distal fourth, where it is indistinct, it is a sharp crest. It is slightly sinuous, and turns medially in the distal fourth. The interosseous border begins distal and anterior to the fibular facet and descends to the anterior border of the fibular notch; it is indistinct proximally. The interosseous membrane is attached to most of its length, connecting tibia to fibula. The medial border descends from the anterior end of the groove on the medial condyle to the posterior margin of the medial malleolus. Its proximal and distal fourths are ill defined but its central region is sharp and distinct. The anteromedial surface, between the anterior and medial borders, is broad, smooth and almost entirely subcutaneous. The lateral surface, between the anterior and interosseous borders, is also broad and smooth. It faces laterally in its proximal three-fourths and is transversely concave. Its distal fourth swerves to face anterolaterally, on account of the medial deviation of the anterior and

distal interosseous borders. This part of the surface is somewhat convex. The posterior surface, between the interosseous and medial borders, is widest above, where it is crossed distally and medially by an oblique, rough soleal line. A faint vertical line descends from the centre of the soleal line for a short distance before becoming indistinct. A large vascular groove adjoins the end of the line and descends distally into a nutrient foramen. Deep fascia and, proximal to the medial malleolus, the medial end of the superior extensor retinaculum, are attached to the anterior border. Posterior fibres of the medial collateral ligament and slips of semimembranosus and the popliteal fascia are attached to the medial border proximal to the soleal line, and some fibres of soleus and the fascia covering the deep calf muscles are attached distal to the line. The distal medial border runs into the medial lip of a groove for the tendon of tibialis posterior. The interosseous membrane is attached to the lateral border, except at either end of this border. It is indistinct proximally where a large gap in the membrane transmits the anterior tibial vessels. Distally the border is continuous with the anterior margin of the fibular notch, to which the anterior tibiofibular ligament is attached. The anterior part of the medial collateral ligament is attached to an area approximately 5 cm long and 1 cm wide near the medial border of the proximal medial surface. The remaining medial surface is subcutaneous and crossed obliquely by the long saphenous vein. Tibialis anterior is attached to the proximal two-thirds of the lateral surface. The distal third, devoid of attachments, is crossed in mediolateral order by the tendons of tibialis anterior (lying just lateral to the anterior border), extensor hallucis longus, the anterior tibial vessels and deep fibular nerve, extensor digitorum longus and fibularis tertius. On the posterior surface, popliteus is attached to a triangular area proximal to the soleal line, except near the fibular facet. The popliteal aponeurosis, soleus and its fascia, and the deep transverse fascia are all attached to the soleal line: the proximal end of the line does not reach the interosseous border, and is marked by a tubercle for the medial end of the tendinous arch of soleus. Lateral to the tubercle, the posterior tibial vessels and tibial nerve descend on tibialis posterior. Distal to the soleal line, a vertical line separates the attachments of flexor digitorum longus and tibialis posterior. Nothing is attached to the distal quarter of this surface, but the area is crossed medially by the tendon of tibialis posterior travelling to a groove on the posterior aspect of the

medial malleolus. Flexor digitorum longus crosses obliquely behind tibialis posterior; the posterior tibial vessels and nerve and flexor hallucis longus contact only the lateral part of the distal posterior surface. (Susan Standring2008)

2.1.1.6.5-Distal End

The slightly expanded distal end of the tibia has anterior, medial, posterior, lateral and distal surfaces. It projects inferomedially as the medial malleolus. The distal end of the tibia, when compared to the proximal end, is laterally rotated (tibial torsion). The torsion begins to develop in utero and progresses throughout childhood and adolescence till skeletal maturity is attained. Some of the femoral neck anteversion seen in the newborn may persist in adult females: this causes the femoral shaft and knee to be internally rotated, and the tibia may develop a compensatory external torsion to counteract the tendency of the feet to turn inwards. The smooth anterior surface projects beyond the distal surface, from which it is separated by a narrow groove. The capsule of the ankle joint is attached to an anterior groove near the articular surface. The medial surface is smooth and continuous above and below with the medial surfaces of the shaft and medial malleolus respectively: it is subcutaneous and visible. The posterior surface is smooth except where it is crossed near its medial end by a nearly vertical but slightly oblique groove, which is usually conspicuous and extends to the posterior surface of the malleolus. The groove is adapted to the tendon of tibialis posterior, which usually separates the tendon of flexor digitorum longus from the bone. More laterally, the posterior tibial vessels and nerve and flexor hallucis longus contact this surface. The lateral surface is the triangular fibular notch; its anterior and posterior edges project and converge proximally to the interosseous border. The floor of the notch is roughened proximally by a substantial interosseous ligament but is smooth distally and sometimes covered by articular cartilage. The anterior and posterior tibiofibular ligaments are attached to the corresponding edges of the notch. The distal surface articulates with the talus and is wider in front, concave sagittally and slightly convex transversely, i.e. it is saddle-shaped. Medially it continues into the malleolar articular surface which

may extend into the groove that separates it from the anterior surface of the shaft. Such extensions, medial or lateral or both, are squatting facets, and they articulate with reciprocal talar facets in extreme dorsiflexion. These features have been used in the field of forensic medicine to identify the racial origins of skeletal material. (Susan Standring2008)

2.1.1.6.6-Medial Malleolus

The short thick medial malleolus has a smooth lateral surface with a crescentic facet that articulates with the medial surface of the talus. Its anterior aspect is rough and its posterior aspect features the continuation of the groove from the posterior surface of the tibial shaft for the tendon of tibialis posterior. The distal border is pointed anteriorly, posteriorly depressed, and gives attachment to the deltoid ligament. The tip of the medial malleolus does not project as far distally as the tip of the lateral malleolus; the latter also being the more posteriorly located of the two malleoli. The capsule of the ankle joint is attached to the anterior surface of the medial malleolus, and the flexor retinaculum to its prominent posterior border. (Susan Standring2008)

2.1.1.7-Fibula

The fibula is much more slender than the tibia and is not directly involved in transmission of weight. It has a proximal head, a narrow neck, a long shaft and a distal lateral malleolus. The shaft varies in form, being variably moulded by attached muscles: these variations may be confusing. (A Faller et al 2004)

2.1.1.7.1-Fibuler head

The head of the fibula is irregular in shape and projects anteriorly, posteriorly, and laterally. A round facet on its proximomedial aspect articulates with a corresponding facet on the inferolateral surface of the lateral tibial condyle. It faces proximally and anteromedially and has an inclination which may vary among individuals from almost horizontal to an angle of up to 45°. A blunt apex (styloid process) projects proximally from the posterolateral aspect of the head and is often palpable approximately 2 cm distal to the knee joint. The fibular collateral

ligament is attached in front of the apex, embraced by the main attachment of biceps femoris. The tibiofibular capsular ligament is attached to the margins of the articular facet. The common fibular nerve crosses posterolateral to the neck and can be rolled against bone at this location. (A Faller et al 2004)

2.1.1.7.2-Fibuler shaft

The shaft has three borders and surfaces, each associated with a particular group of muscles. The anterior border ascends proximally from the apex of an elongated triangular area that is continuous with the lateral malleolar surface, to the anterior aspect of the fibular head. The posterior border, continuous with the medial margin of the posterior groove on the lateral malleolus, is usually distinct distally but often rounded in its proximal half. The interosseous border is medial to the anterior border and somewhat posterior. Over the proximal two-thirds of the fibular shaft the two borders approach each other, with the surface between the two being narrowed to 1 mm or less. The lateral surface, between the anterior and posterior borders and associated with the fibular muscles, faces laterally in its proximal three-fourths. The distal quarter spirals posterolaterally to become continuous with the posterior groove of the lateral malleolus. The anteromedial (sometimes simply termed anterior, or medial) surface, between the anterior and interosseous borders, usually faces anteromedially but often directly anteriorly. It is associated with the extensor muscles. Though wide distally, it narrows in its proximal half and may become a mere ridge. The posterior surface, between the interosseous and posterior borders, is the largest and is associated with the flexor muscles. Its proximal two-thirds is divided by a longitudinal medial crest, separated from the interosseous border by a grooved surface that is directed medially. The remaining surface faces posteriorly in its proximal half; its distal half curves onto the medial aspect. Distally this area occupies the fibular notch of the tibia, which is roughened by the attachment of the principal interosseous tibiofibular ligament. The triangular area proximal to the lateral surface of the lateral malleolus is subcutaneous; muscles cover the rest of the shaft. The anterior border is divided distally into two ridges that enclose a triangular subcutaneous surface. The anterior intermuscular septum is attached to its proximal

three-fourths. The lateral end of the superior extensor retinaculum is attached distally on the anterior border of the triangular area and the lateral end of the superior fibular retinaculum is attached distally on the posterior margin of the triangular area. The interosseous border ends at the proximal limit of the rough area for the interosseous ligament. The interosseous membrane attached to this border does not reach the fibular head, which leaves a gap through which the anterior tibial vessels pass. The posterior border is proximally indistinct, and the posterior intermuscular septum is attached to all but its distal end. The medial crest is related to the fibular artery. A layer of deep fascia separating the tendon of tibialis posterior from flexor hallucis longus and flexor digitorum longus is attached to the medial crest. (A Faller et al 2004)

2.1.1.7.3-Lateral Malleolus

The distal end forms the lateral malleolus which projects distally and posteriorly. Its lateral aspect is subcutaneous while its posterior aspect has a broad groove with a prominent lateral border. Its anterior aspect is rough, round and continuous with the tibial inferior border. The medial surface has a triangular articular facet, vertically convex, its apex distal, which articulates with the lateral talar surface. Behind this facet is a rough malleolar fossa pitted by vascular foramina. The posterior tibiofibular ligament and, more distally, the posterior talofibular ligament, are attached in the fossa. The anterior talofibular ligament is attached to the anterior surface of the lateral malleolus; the calcaneofibular ligament is attached to the notch anterior to its apex. The tendons of fibularis brevis and longus groove its posterior aspect: the latter is superficial and covered by the superior fibular retinaculum. (A Faller et al 2004)

2.1.1.8-Foot (Pes)

The foot, like the hand, is divided into three groups arranged end to end: the ankle (tarsus), the intermediate group (metatarsus), and the toes (digits). There are seven tarsal bones, of which the talus, the navicular, and the three cuneiform bones lie on the medial side, while the calcaneum (calcaneus, oscalcis), and the cuboid bone are lateral. The calcaneum is the largest bone of the foot, its posteriorly directed calcaneal tubercle

(tuberculumcalcanei) forming the bony base of the heel. The five metatarsal bones are long bones, like the metacarpal bones of the hand. Each is divided into a base, a shaft, and a head. The first (medial) metatarsal bone is the thickest and strongest metatarsal bone. The 2nd to 5th toes consist of proximal, middle and distal phalanges, while the 1st toe (big toe, hallux), only has a proximal and a distal phalanx. Inspection of the skeleton of the foot reveals that, while the talus lies above the calcaneum behind, the two bones lie side by side in the middle and infront. This arrangement creates medially a marked longitudinal arch, and at the level of the cuneiform bones and the metatarsal bones a transverse arch running from medial to lateral. Muscles and tendons serve to keep the searches intact. The calcaneal tubercle and the heads of the 1st to 5th metatarsals may be regarded as the bases on which all of the body weight rests on level ground. (A Faller et al 2004)

2.1.1.8.1-Distal Tibia

The distal end of the tibia has anterior, medial, posterior, lateral and distal surfaces, and projects inferomedially as the medial malleolus. The distal surface articulates with the talus and is wider anteriorly than posteriorly. It is concave sagittally and slightly convex transversely and continues medially into the malleolar articular surface. The medial malleolus is short and thick and has a smooth lateral surface with a crescentic facet that articulates with the medial surface of the talar body. (Susan Standring2008)

2.1.1.8.2-Distal fibula

The distal end of the fibula or lateral malleolus projects distally and posteriorly relative to the medial malleolus. Its lateral aspect is subcutaneous, the posterior surface has a broad groove with a prominent lateral border, and the anterior surface is rough and somewhat rounded and articulates with the anteroinferior aspect of the tibia. The medial surface has a triangular articular facet, vertically convex with its apex directed distally. It articulates with the lateral talar surface. Behind the facet is a rough malleolar fossa. (Susan Standring2008)

2.1.1.8.3-Tarsus

The seven tarsal bones occupy the proximal half of the foot. The tarsus and carpus are homologous, but the tarsal elements are larger, reflecting their role in supporting and distributing body weight. As in the carpus, tarsal bones are arranged in proximal and distal rows, but medially there is an additional single intermediate tarsal element, the navicular. The proximal row is made up of the talus and calcaneus; the long axis of the talus is inclined anteromedially and inferiorly, its distally directed head is medial to the calcaneus and at a higher level. The distal row contains, from medial to lateral, the medial, intermediate and lateral cuneiforms and the cuboid. Collectively these bones display an arched transverse alignment that is dorsally convex. Medially, the navicular is interposed between the head of the talus and the cuneiforms. Laterally, the calcaneus articulates with the cuboid. The tarsus and metatarsus are arranged to form intersecting longitudinal and transverse arches. Hence thrust and weight are not transmitted from the tibia to the ground (or vice versa) directly through the tarsus, but are distributed through the tarsal and metatarsal bones to the ends of the longitudinal arches. For the purposes of description, each tarsal bone is arbitrarily considered to be cuboidal in form, with six surfaces. (Susan Standring2008)

2.1.1.8.4-Talus

The talus is the link between the foot and leg, through the ankle joint

2.1.1.8.4.1-The head of the talus

Directed distally and somewhat inferomedially, the head has a distal surface which is ovoid and convex; its long axis is also inclined inferomedially to articulate with the proximal navicular surface. The plantar surface of the head has three articular areas, separated by smooth ridges. The most posterior and largest is oval, slightly convex and rests on a shelf-like medial calcaneal projection, the sustentaculumtali anterolateral to this and usually continuous with it, a flat articular facet rests on the anteromedial part of the dorsal (proximal) calcaneal surface; distally it continues into the navicular surface. Between the two calcaneal facets, a part of the talar head, covered with articular cartilage, is in contact with the plantar calcaneonavicular ligament, which is covered

here, superiorly, by fibro cartilage. When the foot is inverted passively, the dorsolateral aspect of the head is visible and palpable approximately 3 cm distal to the tibia; it is hidden by extensor tendons when the toes are dorsiflexed. (Susan Standring2008)

2.1.1.8.4.2-Neck

The neck is the narrow, medially inclined region between the head and body. Its rough surfaces are for ligaments. The medial plantar surface has a deep sulcus tali which, when the talus and calcaneus are articulated, forms a roof to the sinus tarsi, which is occupied by inter-osseous talocalcaneal and cervical ligaments. The long axis of the neck, inclined downwards, distally and medially, makes an angle of approximately 150° with that of the body; it is smaller (130–140°) at birth, accounting in part for the inverted foot in young children. The dorsal talonavicular ligament and ankle articular capsule are attached distally to its dorsal surface. Thus the proximal part of this surface lies within the capsule of the ankle joint. The medial articular facet of the talar body and part of the trochlear surface may extend onto the neck. The anterior talofibular ligament is attached on the lateral aspect of the neck, spreading along the adjacent anterior border of the lateral surface. The interosseous talocalcaneal and cervical ligaments are attached to the inferior surface of the neck. A dorsolateral, so-called ‘squatting facet' is commonly present on the talar neck in those individuals who habitually adopt the squatting position: it articulates with the anterior tibial margin in extreme dorsiflexion and may be double. (Susan Standring2008)

2.1.1.8.4.3-Body

The body is cuboidal, covered dorsally by a trochlear surface articulating with the distal end of the tibia. It is anteroposteriorly convex, gently concave transversely, widest anteriorly and, therefore, sellar. The triangular lateral surface is smooth and vertically concave for articulation with the lateral malleolus. Superiorly, it is continuous with the trochlear surface; inferiorly its apex is a lateral process. Proximally, the medial surface is (posterosuperiorly) covered by a comma-shaped facet, which is deeper in front and articulates with the medial malleolus. Distally, this surface is rough and contains numerous vascular foramina. The small

posterior surface features a rough projection termed the posterior process. The process is marked by an oblique groove between two tubercles which lodges the tendon of flexor hallucis longus. The lateral tubercle is usually larger; the medial is less prominent and immediately behind the sustentaculum tali. The plantar surface articulates with the middle one-third of the dorsal calcaneal surface by an oval concave facet, its long axis directed distolaterally at an angle of approximately 45° with the median plane. The medial edge of the trochlear surface is straight, but its lateral edge inclines medially in its posterior part and is often broadened into a small elongated triangular area which is in contact with the posterior tibiofibular ligament in dorsiflexion. The posterior talofibular ligament is attached to the lateral tubercle of the posterior process. Its attachment extends up to the groove, or depression, between the process and posterior trochlear border. The posterior talocalcaneal ligament is attached to the plantar border of the posterior process. The groove between the tubercles of the process contains the tendon of flexor hallucis longus and continues distally into the groove on the plantar aspect of the sustentaculum tali. The medial talocalcaneal ligament is attached below to the medial tubercle, whereas the most posterior superficial fibres of the deltoid ligament are attached above the tubercle. The deep fibres of the deltoid ligament are attached still higher to the rough area immediately below the comma-shaped articular facet on the medial surface. (Susan Standring2008)

2.1.1.8.5-Calcaneus

The calcaneus is the largest tarsal bone and projects posterior to the tibia and fibula as a short lever for muscles of the calf attached to its posterior surface. It is irregularly cuboidal, its long axis being inclined distally upwards and laterally. The superior or proximal surface is divisible into three areas. The posterior one-third is rough and concavo-convex; the convexity is transverse and supports fibroadipose tissue (Kager's fat pad) between the calcaneal tendon and ankle joint. The middle one-third carries the posterior talar facet, which is oval and convex anteroposteriorly. The anterior one-third is partly articular; distal (anterior) to the posterior articular facet, a rough depression, the sulcus calcanei, narrows into a groove on the medial side and completes the sinus tarsi with the talus. Distal and medial to this groove, an elongated

articular area covers the sustentaculum tali and extends distolaterally on the body of the bone. This facet is often divided into middle and anterior talar facets by a non-articular interval at the anterior limit of the sustentaculum tali. Rarely, all three facets on the upper surface of the calcaneus are fused into one irregular area. The anterior surface is the smallest, and is an obliquely set concavo-convex articular facet for the cuboid. The posterior surface is divided into three regions: a smooth proximal (superior) area separated from the calcaneal tendon by a bursa and adipose tissue; a middle area, which is the largest, limited above by a groove and below by a rough ridge for the calcaneal tendon; a distal (inferior) area inclined downwards and forwards, vertically striated, which is the subcutaneous weight-bearing surface. The plantar surface is rough, especially proximally as the calcaneal tuberosity, the lateral and medial processes of which extend distally, separated by a notch. The medial process is longer and broader. Further distally, an anterior tubercle marks the distal limit of the attachment of the long plantar ligament. The lateral surface is almost flat. It is proximally deeper and palpable on the lateral aspect of the heel distal to the lateral malleolus. Distally, it presents the fibular trochlea, which is exceedingly variable in size and palpable 2 cm distal to the lateral malleolus when well developed. It bears an oblique groove for the tendon of fibularis longus and a shallower proximal groove for the tendon of fibularis brevis. About 1 cm or more behind and above the fibular trochlea, a second elevation may exist for attachment of the calcaneofibular part of the lateral ligament. The medial surface is vertically concave, and its concavity is accentuated by the sustentaculum tali, which projects medially from the distal part of its upper border. Superiorly, the process bears the middle talar facets and inferiorly a groove which is continuous with that on the talar posterior surface for the tendon of flexor hallucis longus. The medial aspect of the sustentaculum tali can be felt immediately distal to the tip of the medial malleolus; occasionally it is also grooved by the tendon of flexor digitorum longus. (Susan Standring2008)

2.1.1.8.6-Cuboid

The cuboid, the most lateral bone in the distal tarsal row, lies between the calcaneus proximally and the fourth and fifth metatarsals distally. Its dorsolateral surface is rough for the attachment of ligaments. The plantar

surface is crossed distally by an oblique groove for the tendon of fibularis longus and bounded proximally by a ridge that ends laterally in the tuberosity of the cuboid, the lateral aspect of which is faceted for a sesamoid bone or cartilage that is frequently found in the tendon of fibularis longus. Proximal to its ridge, the rough plantar surface extends proximally and medially because of the obliquity of the calcaneocuboid joint, making its medial border much longer than the lateral. The lateral surface is rough; the groove for fibularis longus extends from a deep notch on its plantar edge. The medial surface, which is much more extensive and partly non-articular, bears an oval facet for articulation with the lateral cuneiform, and proximal to this facet (sometimes absent) for articulation with the navicular: the two form a continuous surface separated by a smooth vertical ridge. The distal surface is divided vertically into a medial quadrilateral articular area for the fourth metatarsal base and a lateral triangular area, its apex lateral, for the fifth metatarsal base. The proximal surface, triangular and concavo-convex, articulates with the distal calcaneal surface; its medial plantar angle projects proximally and inferior to the distal end of the calcaneus. (Susan Standring2008)

2.1.1.8.7-Cuneiforms

The wedge-like cuneiform bones articulate with the navicular proximally and with the bases of the first to third metatarsals distally; the medial cuneiform is the largest, the intermediate the smallest. The dorsal surfaces of the intermediate and lateral cuneiforms form the base of the wedge. The wedge is reversed in the medial cuneiform, which is a prime factor in shaping the transverse arch. The proximal surfaces of all three form a concavity for the distal surface of the navicular. The medial and lateral cuneiforms project distally beyond the intermediate cuneiform and so form a recess for the second metatarsal base. (Susan Standring2008)

2.1.1.8.7.1-Medial cuneiform

The medial cuneiform articulates with the navicular and first metatarsal base. It has a rough, narrow dorsal surface. The distal surface is a reniform facet for the first metatarsal base, its ‘hilum' being lateral. The proximal surface bears a piriform facet for the navicular, which is

concave vertically and dorsally narrowed. The medial surface, rough and subcutaneous, is vertically convex; its distal plantar angle carries a large impression which receives the principal attachment of the tendon of tibialis anterior. The lateral surface is partly non-articular; there is a smooth right-angled strip along its proximal and dorsal margins for the intermediate cuneiform. Its distal dorsal area is separated by a vertical ridge from a small, almost square, facet for articulation with the dorsal part of the medial surface of the second metatarsal base. Plantar to this, the medial cuneiform is attached to the medial side of the second metatarsal base by a strong ligament. Proximally, an interosseous inter cuneiform ligament connects this surface to the intermediate cuneiform. The distal and plantar area of the surface is roughened by attachment of part of the tendon of fibularis longus. (Susan Standring2008)

2.1.1.8.7.2-Intermediate cuneiform

The intermediate cuneiform articulates proximally with the navicular and distally with the second metatarsal base. It has a narrow, plantar surface that receives a slip from the tendon of tibialis posterior. The distal and proximal surfaces are both triangular articular facets and articulate with the second metatarsal base and the navicular, respectively. The medial surface is partly articular: it articulates via a smooth, angled strip that is occasionally double with the medial cuneiform along its proximal and dorsal margins. The lateral surface is also partly articular: along its proximal margin a vertical strip, usually indented, abuts the lateral cuneiform. Strong interosseous ligaments connect non-articular parts of both surfaces to the adjacent cuneiforms. (Susan Standring2008)

2.1.1.8.7.3-Lateral cuneiform

The lateral cuneiform is between the intermediate cuneiform and cuboid, and also articulates with the navicular and, distally, with the third metatarsal base. Like the intermediate cuneiform, its dorsal surface, which is rough and almost rectangular, is the base of a wedge. The plantar surface is narrow and receives a slip from tibialis posterior and sometimes part of flexor hallucis brevis. The distal surface is a triangular articular facet for the third metatarsal base. The proximal surface is rough on its plantar aspect, but its dorsal two-thirds articulate with the navicular

by a triangular facet. The medial surface is partly non-articular and has a vertical strip, indented by the intermediate cuneiform, on its proximal margin; on its distal margin, a narrower strip (often two small facets) articulates with the lateral side of the second metatarsal base. The lateral surface, also partly non-articular, bears a triangular or oval proximal facet for the cuboid; a semilunar facet on its dorsal and distal margin articulates with the dorsal part of the medial side of the fourth metatarsal base. Non-articular areas of the medial and lateral surfaces receive intercuneiform and cuneocuboid ligaments, respectively, which are important in the maintenance of the transverse arch. (Susan Standring2008)

2.1.2-Musculs

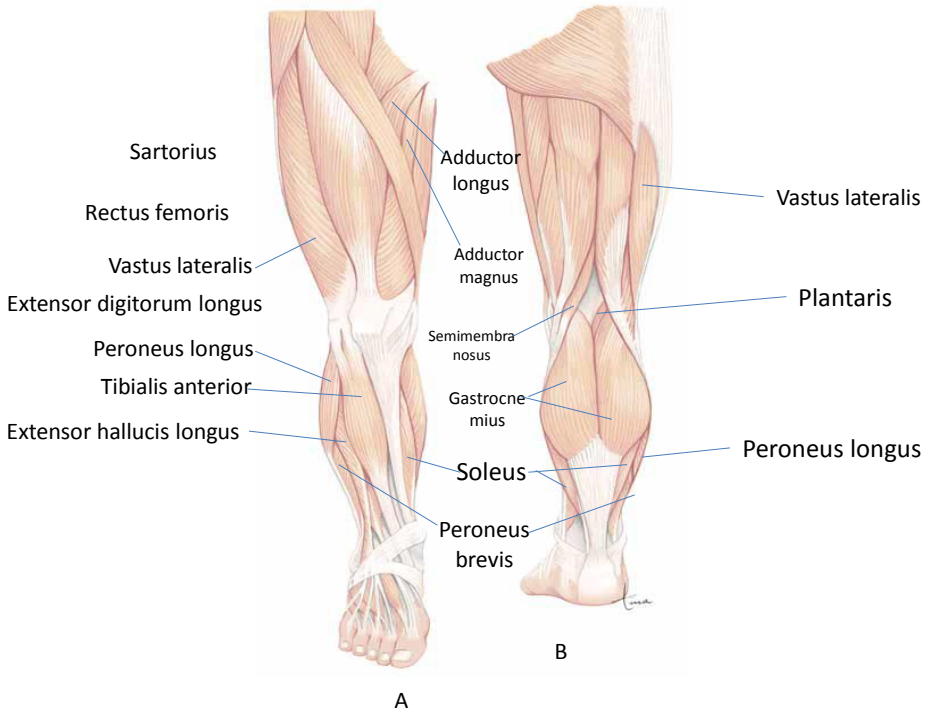
Muscle tissue is specialized for contraction. When muscle cells contract, they shorten and bring about some type of movement. There are three types of muscle tissue: skeletal, smooth, and cardiac. The movements each can produce have very different purposes. (Valerie C. Scanlon & Tina Sanders 2008)

2.1.2.1-Skeletal muscles

Skeletal muscle may also be called striated muscle or voluntary muscle. Each name describes a particular aspect of this tissue, as you will see. The skeletal muscle cells are cylindrical, have several nuclei each, and appear striated, or striped. The striations are the result of the precise arrangement of the contracting proteins within the cells. Skeletal muscle tissue makes up the muscles that are attached to bones. These muscles are supplied with motor nerves, and thus move the skeleton. They also produce a significant amount of heat, which is important to help maintain the body's constant temperature. Each muscle cell has its own motor nerve ending. The nerve impulses that can then travel to the muscles are essential to cause contraction. Although we do not have to consciously plan all our movements, the nerve impulses for them originate in the cerebrum, the "thinking" part of the brain. Let us return to the three names for this tissue: "skeletal" describes its location, "striated" describes its appearance, and "voluntary" describes how it functions. (Valerie C. Scanlon & Tina Sanders 2008)

2.1.2.2 Smooth muscles

Smooth muscle may also be called involuntary muscle or visceral muscle. The cells of smooth muscle have tapered ends, a single nucleus, and no striations. Although nerve impulses do bring about contractions, this is not something most of us can control, hence the name involuntary. The term visceral refers to internal organs, many of which contain smooth muscle. The functions of smooth muscle are actually functions of the organs in which the muscle is found. In the stomach and intestines, smooth muscle contracts in waves called peristalsis to propel food through the digestive tract. In the walls of arteries and veins, smooth muscle constricts or dilates the vessels to maintain normal blood pressure. The iris of the eye has two sets of smooth muscle fibers to constrict or dilate the pupil, which regulates the amount of light that strikes the retina. Other functions of smooth muscle are mentioned in later chapters. This is an important tissue that you will come across again and again in our study of the human body. (Valerie C. Scanlon & Tina Sanders 2008)



Figure(2-2). Muscles of the leg. (**A**) Anterior view. (**B**) Posterior view (Valerie C. Scanlon & Tina Sanders 2008 Page No 158)

2.1.2.3 Muscles of the hip and leg

The hip muscles that move the thigh are anchored to the pelvic bone and cross the hip joint to the femur. Among these are the gluteus maximus (extension), gluteus medius (abduction), and iliopsoas (flexion). The muscles that form the thigh include the quadriceps group anteriorly and the hamstring group posteriorly. For most people, the quadriceps is stronger than the hamstrings, which is why athletes more often have a "pulled hamstring" rather than a "pulled quadriceps." Movement of the knee joint depends on thigh muscles and lower leg muscles. Movement of the foot depends on lower leg muscles such as the gastrocnemius (dorsiflexion or flexion) and the tibialis anterior (plantar flexion or extension). (Valerie C. Scanlon & Tina Sanders 2008)

The effects of extension and medial rotation of the limb that occur during fetal development are manifest in the relative positions of the muscle groups in the thigh and the leg, and in the adult pattern of segmental innervation (dermatomes). In broad outline, the anterior aspect of the adult limb is the extensor aspect, while the flexors lie posteriorly; the reverse is true at the hip. The role of the muscles of the lower limb in the maintenance of equilibrium during locomotion and in stance is rarely emphasized sufficiently. Many of the muscles act frequently or predominantly from their distal attachments. During both stance and locomotion, the distal attachment is often fixed and the proximal attachment is mobile, e.g. the predominant action of gluteus medius is as a pelvic stabilizer rather than as a hip abductor. (In contrast, in the upper limb the proximal muscle attachments are usually fixed and the distal attachments are mobile, an arrangement that is consistent with the prehensile function of the hand.) The lower limb contains many muscles that act upon more than one joint, and it is unusual for any joint of the lower limb to move in isolation. Muscles of the lower limb may be subdivided into muscles of the iliac and gluteal regions, and of the thigh, leg and foot. The main muscles of the iliac region are psoas major and iliacus, the major flexors of the hip that run from the lumbar spine and inner surface of the ilium respectively to the lesser trochanter of the femur. The much less important psoas minor (when present) runs from the lumbar spine to the pubis. The muscles of the gluteal region include the three named gluteal muscles and the deeper short lateral rotators of the hip joint. Gluteus maximus lies most superficially, running from the

posterior pelvis to the proximal femur and fascia lata. It is a powerful extensor of the hip joint, acting more often to extend the trunk on the femur than to extend the limb on the trunk. Gluteus medius and minimus, attaching proximally to the outer iliac surface and distally to the greater trochanter of the femur, are abductors of the hip; their most important action is to stabilize the pelvis on the femur during locomotion, and they are helped in this function by tensor fasciae latae, a more anteriorly placed muscle that arises from the anterolateral ilium and inserts into the fascia lata. Two of the short lateral rotators of the hip, piriformis and obturator internus, arise from within the pelvic walls, while the others, obturator externus, the gemelli and quadratus femoris, are attached externally: all of these muscles are attached distally to the proximal femur. The muscles of the thigh lie in three functional compartments. The anterior or extensor compartment includes sartorius and the quadriceps group. Sartorius and rectus femoris are attached proximally to the pelvis and can thus act on the hip joint as well as on the knee, whereas the vasti are attached proximally to the femoral shaft, and, acting as a unit, are powerful knee extensors. The medial or adductor compartment contains the named adductor muscles and gracilis; pectineus may also be included. These muscles are attached proximally to the anterior aspect of the pelvis, and distally to the femur; gracilis has no femoral attachment, being attached distally to the proximal tibia, while a part of adductor magnus has a proximal attachment to the ischial tuberosity. The posterior (‘hamstring') compartment includes semitendinosus, semimembranosus and biceps femoris. These muscles are attached proximally to the ischial tuberosity and act both to extend the trunk on the femur and to flex and rotate the knee. Adductor magnus, as may be inferred from the extent of its proximal attachment and its dual innervation, shares the first of these functions with the hamstrings. Biceps femoris is the only muscle of the thigh that is attached distally to the fibula, and has no tibial attachment. In the leg the anterior or extensor compartment includes the extensors (dorsiflexors) of the ankle and the long extensors of the toes. Tibialis anterior, the main ankle dorsiflexor, also inverts the foot at the subtalar joint, while the smallest muscle of the compartment, fibularis (peroneus) tertius, is a dorsiflexor that everts the foot. The posterior or flexor (plantarflexor) compartment has superficial and deep components. The superficial component contains gastrocnemius and soleus, powerful

plantar flexors of the ankle, and the slender plantaris. Gastrocnemius and soleus are attached distally via the calcaneal (Achilles) tendon. The deep component of the flexor compartment contains popliteus, a rotator of the knee, the long flexors of the toes and tibialis posterior, the main invertor of the foot. The lateral compartment contains the main evertors of the foot, fibularis (peroneus) longus and brevis; both muscles are also plantar flexors at the ankle. Gastrocnemius, plantaris and popliteus are the only muscles of the leg that are attached proximally to the femur and they can therefore act on the knee as well as at the ankle. The remaining leg muscles are attached proximally to the tibia, fibula or both, and to the interosseous membrane. The intrinsic muscles of the sole of the foot are arranged in layers. They facilitate the actions of the long flexors of the toes, and by effecting subtle changes in the shape of the foot they help control foot posture in stance and locomotion. (Susana Standring 2008)

2.1.3 The Vessels

The arterial and venous systems are often thought of as a series of tubes that transport blood to and from organs and tissues. In reality, blood vessels are highly complex structures that respond to nervous stimulation and interact with chemicals in the blood stream to regulate the flow of blood throughout the body. Changes in cardiac output and the tone of the smooth muscle cells in the arterial walls are crucial factors that affect blood flow. The structure of a blood vessel wall varies considerably depending on its position within the vascular system. Arteries and veins are composed of three layers of tissue, with veins having thinner walls than arteries. The outer layer is called the adventitia and is predominantly composed of connective tissue with collagen and elastin. The middle layer, the media, is the thickest layer and is composed of smooth muscle fibers and elastic tissue. The intima is the inner layer and consists of a thin layer of epithelium overlying an elastic membrane. The capillaries, by contrast, consist of a single layer of endothelium, which allows for the exchange of molecules through the capillary wall. It is possible to image the structure of larger vessel walls using ultrasound and to identify the early stages of arterial disease, such as intimal thickening. The arterial tree consists of elastic arteries, muscular arteries and arterioles. The aorta

and subclavian arteries are examples of elastic or conducting arteries and contain elastic fibers and a large amount of collagen fibers to limit the degree of stretch. Elastic arteries function as a pressure reservoir, as the elastic tissue in the vessel wall is able to absorb a proportion of the large amount of energy generated by the heart during systole. This maintains the end diastolic pressure and decreases the load on the left side of the heart. Muscular or distributing arteries, such as the radial artery, contain a large proportion of smooth muscle cells in the media. These arteries are innervated by nerves and can dilate or constrict. The muscular arteries are responsible for regional distribution of blood flow. Arterioles are the smallest arteries, and their media is composed almost entirely of smooth muscle cells. Arterioles have an important role in controlling blood pressure and flow, and they can constrict or dilate after sympathetic nerve or chemical stimulation. The arterioles distribute blood to specific capillary beds and can dilate or constrict selectively around the body depending on the requirements of organs or tissues. (Abigail Thrush & Timothy Hartshorne 2005)

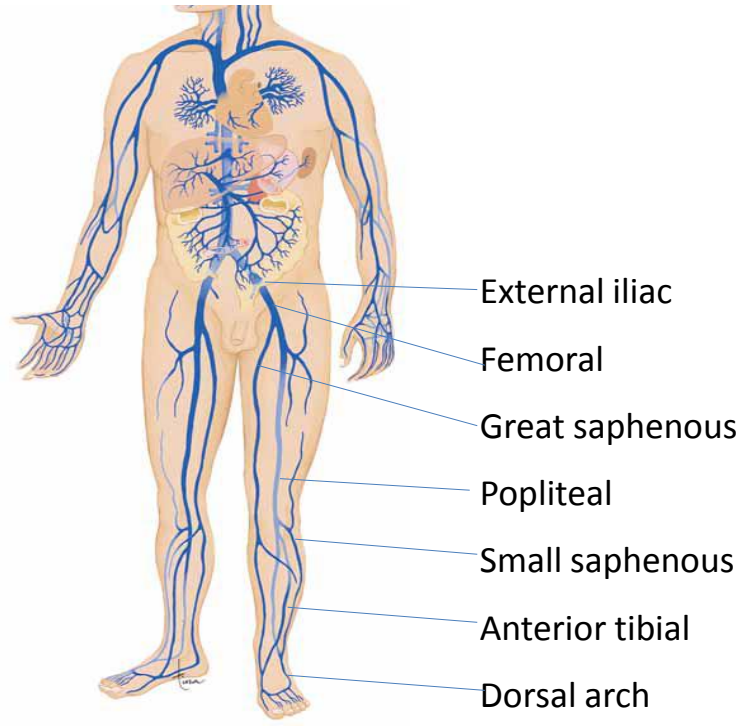


Figure (2–3.) Systemic veins shown in anterior view, Essential anatomy and physiology Scanlon & Sanders 2008Page298

The circulatory system connects the capillary bed, which serves the ex - change of substances, with the heart through the arteries and veins. The function of the arteries and veins is strictly to channel the blood, not to exchange substances. Arteries and veins may be distinguished by the fact that pressure in the arteries is high and that in the veins is low**.** (Myron Pozniak & Paul Allan 2014)

2.1.3.1-The Structure of Arteries and Veins

Arteries and veins resemble each other in that their walls contain three coats. However, the vessels adapt to their different circulatory tasks by differing in the structure of these coats. The inner coat (vascular endothelium, tunica intima) consists of a single layer of endothelial cells applied to a thin connective tissue layer, the basement membrane. The middle coat (tunica media) contains primarily smooth muscle and elastic tissue fibers. The outer coat (tunica adventitia, adventitious coat) embeds the vessel in its surroundings and consists mainly of connective tissue. In addition, the arteries have an elastic, fenestrated membrane (membrane elastic interna) between the inner and middle coat. Usually another, thinner elastic membrane (membrane elastic externa) lies between the middle and the outer coat. Arteries are distinguished by an especially well developed muscle coat, which contains a varying amount of elastic fiber according to its site (predominantly elastic and predominantly muscular arteries).This layer is the driving force of the blood vessels. By dilating (vaso dilatation) and constricting (vasoconstriction) the diameter of the blood vessels, it regulates blood flow and blood pressure. The arteries near the heart contain a high proportion of elastic fibers and this creates elastic recoil. The blood ejected during systole is partly stored by expansion of the arterial wall, and is then moved forward during diastole by elastic recoil, thus achieving a continuous blood flow Veins in general have wider lumina and thinner walls than arteries. The three coats are less well defined and the muscular coat is less well developed. Most veins, with the exception of those close to the heart, contain venous valves. These endothelial folds, projecting like pockets into the lumen of the vessel, act as one-way valves that guide the blood toward the heart and prevent backward flow (A Faller et al 2004)

2.1.3.2- Structure of the Capillaries

In the smallest blood vessels, the capillaries, the coats are reduced to one, the tunica intima. This facilitates the exchange of fluids and gases. The exchange of substances basically occurs in both directions: from the blood through the endothelium and basement membrane into the surrounding tissue, and in the reverse direction. (A Faller et al 2004)

2.1.3.3-The arteries of the lower limb arise at the bifurcation of the abdominal aorta, the common iliac arteries run down the posterior wall of the pelvis and divide into the internal and external iliac arteries in front of the sacroiliac joint. The internal iliac artery continues down into the pelvis and is difficult to demonstrate with transabdominal ultrasound, although transvaginal or transrectal scanning will show some of its branches. The external iliac artery continues around the side of the pelvis to the level of the inguinal ligament, it lies anteromedial to the psoas muscle and is normally superficial to the external iliac vein. The common femoral artery runs from the inguinal ligament to its division into superficial and deep femoral arteries in the upper thigh; this division is usually 3–6 cm distal to the inguinal ligament. The deep femoral artery, or profunda femoris artery, passes posterolaterally to supply the major thigh muscles. The importance of the profunda femoris lies in its role as a major collateral pathway in patients with significant superficial femoral artery disease. Several other branches arise from the external iliac, common femoral and profunda femoris arteries and occasionally one of these may be mistaken for the profunda femoris artery, especially if it is enlarged as a collateral supply. The superficial femoral artery (also referred to as the femoral artery) passes downwards along the anteromedial aspect of the thigh lying anterior to the vein; in the lower third of the thigh it passes into the adductor canal, deep to sartorius and the medial component of quadriceps femoris. Passing posteriorly behind the lower femur it enters the popliteal fossa and becomes the popliteal artery, which lies anterior to the popliteal vein and gives off several branches, the largest of which are the superior and inferior geniculate arteries. Below the knee joint the popliteal artery divides into the anterior tibial artery and the tibioperoneal trunk, although the exact level of the division may vary; after 2–4 cm the latter divides into the posterior tibial artery and the peroneal artery. The anterior tibial artery passes forwards through the interosseous membrane between the fibula and tibia. It then

descends on the anterior margin of the membrane, deep to the extensor muscles on the Anterolateral aspect of the calf. At the ankle it passes across the front of the joint to become the dorsalis pedis artery of the foot which runs from the front of the ankle joint to the proximal end of the first intertarsal space where it gives off metatarsal branches and passes through the first intertarsal space to unite with the lateral plantar artery and form the plantar arterial arch. The posterior tibial artery passes down the deep medial aspect of the calf to pass behind the medial malleolus, after which it divides into the medial and lateral plantar arteries of the foot; the lateral plantar artery joins with the dorsalis pedis artery in the plantar arch. The peroneal artery passes down the calf behind the tibia and interosseous membrane and divides into several periarticular branches behind the ankle joint. The size of the calf arteries can be quite variable, the posterior tibial artery is the least variable in caliber but the anterior tibial and peroneal arteries may vary considerably in caliber and overall length in the calf. The arterial supply to the foot is not normally examined but if a bypass procedure to the pedal arteries is being considered then the dorsalis pedis and posterior tibial artery and its plantar branches should be assessed. (Myron Pozniak & Paul Allan 2014)

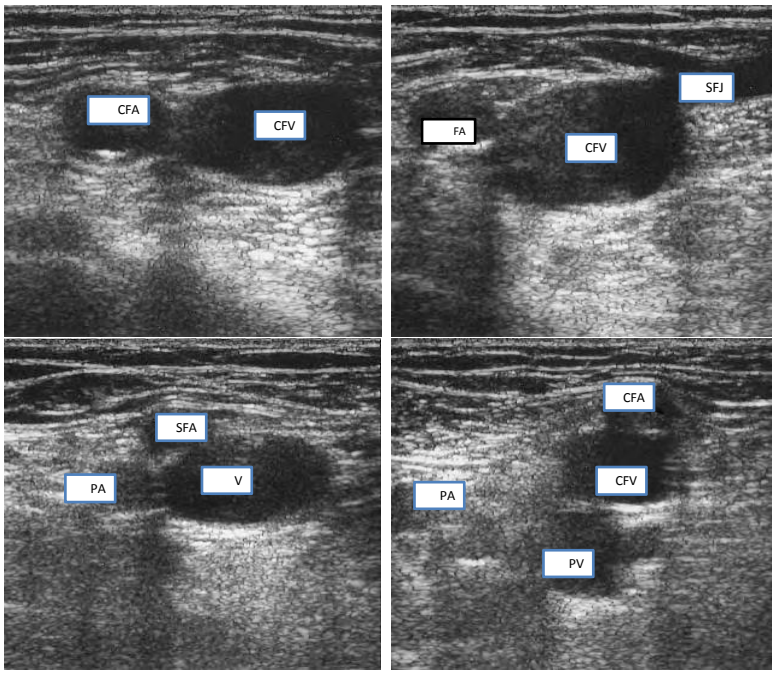


Figure (2-4) the anatomy of the right femoral artery and vein at the groin, with corresponding transverse B-mode images at four different levels. Vessels shown on the diagram are: 1 common femoral artery, 2 common femoral vein,3 saphenofemoral junction, 4 superficial femoral artery, 5 profunda femoris artery, 6 superficial femoral vein, 7 profunda vein. Vessels demonstrated on the images are the common femoral vein (V), common femoral artery (CFA),saphenofemoral junction (SFJ), superficial femoral artery (SFA), profunda femoris artery (PA), superficial

femoral vein(SFV) and profunda vein (PV). Note that the femoral artery bifurcation is sometimes found above the level of thesaphenofemoral junction. In addition, the superficial femoral artery tends to roll on top of the superficial femoral vein, asshown in the B-mode image. . (How where and when and where Thrush & Hartshorne 2005 page No118)

2.1.3.4-The veins of the lower limb are divided into deep and superficial systems. These are linked by a variable number of perforator veins which carry blood from the superficial to the deep systems. The Deep Veins The anatomy of the lower limb veins is rather variable. Generally the veins accompany the arteries but their number may vary and the communications with other veins along the way can show a variety of patterns; however, a general arrangement is usually apparent. In the calf there are veins running with the main arteries: the posterior tibial, peroneal and anterior tibial veins; there are usually two, occasionally three veins with each artery. In addition there are veins draining the major muscle groups in the posterior calf. These are seen in the upper calf as they pass upwards to join the other deep veins in the lower popliteal region; the gastrocnemius and soleal veins are the largest of these. The gastrocnemius vein is the more superficial and may be mistaken for the small saphenous vein; clues to its true identity are that it is usually accompanied by the artery to the muscle and it can be followed distally down into the muscle rather than outwards to lie subcutaneously on the fascia around the calf, which is the position of the small saphenous vein. The calf veins join to form the popliteal vein, or veins – there may be two, or sometimes three channels, especially if there is a dual superficial femoral vein. The popliteal vein runs up through the popliteal fossa, lying more posterior and usually medial to the artery. As well as the veins from the calf and calf muscles, it is joined by the small saphenous vein at the sapheno popliteal junction. The popliteal vein becomes the femoral vein at the upper border of the popliteal fossa; rarely, the popliteal vein runs more deeply to join with the profunda femoris vein. The femoral vein passes through the femoral canal and runs up the medial aspect of the thigh, posterior to the femoral artery to join with the profunda femoris vein (which can alternatively be called the deep femoral vein) in the femoral triangle below the groin; the profunda femoris vein drains the thigh muscles. The confluence of the femoral and profunda femoris veins to form the common femoral vein is normally a little more caudal than the bifurcation of the common femoral artery into the femoral and

profunda femoris arteries. The femoral vein may have significant segments of duplication along its length in up to 25–30% of subjects these dual segments may have a variable relation to the artery, so that they may be overlooked unless care is taken in the examination of the thigh veins with both transverse and longitudinal views being obtained. In the pelvis and groin, the anatomy is generally consistent. The femoral vein and profunda femoris vein join to form the common femoral vein, which lies medial to the common femoral artery. The common femoral vein is joined by the great saphenous vein at the sapheno femoral junction; the appearance of the common femoral vein, great saphenous vein and the common femoral artery in transverse section is sometimes referred to as the 'Mickey Mouse' view. The common femoral vein is also joined by veins from the muscles around the hip. These veins are variable in size and number, occasionally one of these is large veins then join at the level of the aortic bifurcation to form the inferior vena cava, which normally passes cranially on the right side of the aorta. The left common iliac vein passes behind the right common iliac artery just distal to this confluence. In a small number of individuals this confluence does not occur and the two common iliac veins continue cranially as dual inferior venae cavae; this reflects the arrangement of paired cardinal veins in the embryo. The deep veins have a series of valves along their course. These are somewhat variable in their number and location. They are most numerous in the veins below the knee; in the thigh, the femoral vein usually has one just below the confluence with the profunda femoris vein and at several levels below this. The iliac veins, in contrast, have relatively few valves rarely a valve may be seen in the inferior vena cava The Superficial Veins The two main superficial venous channels in the lower limb are the great and small saphenous veins. The great saphenous vein arises from the medial aspect of the dorsal venous arch of the foot and passes in front of the medial malleolus to run up the medial aspect of the calf and knee into the thigh. In the upper thigh, the great saphenous vein curves laterally and deeply to join the common femoral vein just below the inguinal ligament. The great saphenous vein has two components in the calf: the posterior division passes up from the medial malleolus and communicates with the perforator veins; the anterior division usually joins the posterior division just below the level of the knee joint. Duplication of the great saphenous vein can be seen in the

thigh in up to 50% of people, this usually takes the form of parallel channels. The great saphenous vein receives many superficial tributaries and is connected to the deep veins by perforating veins; some of these tributaries in the thigh can be quite prominent and may be mistaken for the main vein if their true nature is not recognized. In the region of the saphenofemoral junction the great saphenous vein receives several tributaries draining the groin, lower abdominal wall and perineum. These veins are of significance in the recurrence of varicose veins following high ligation, as they provide a network of collateral channels which may bypass the resected segment. The small saphenous vein arises from the lateral aspect of the dorsal venous arch of the foot, passing below and behind the lateral malleolus to run up the posterolateral aspect of the calf to the popliteal fossa, where it passes through the deep fascia to join the popliteal vein. Classically, it enters the lateral aspect of the popliteal vein at the level of the popliteal skin crease, or within a few centimeters above this but the level of the confluence can be quite variable. It can be distinguished from the posterior muscle venous sinuses as it does not have an accompanying artery and it is seen to lie within the fascial triangle in the posterior thigh defined by the deep muscular fascia and the superficial fascia. Occasionally there is a thigh extension of the small saphenous vein, passing upwards to join the profunda femoris vein in the lower thigh – a giacomini vein. Burihan and Baptista- Silva dissected 200 adult cadaver legs and reported 20 different patterns of termination of the small saphenous vein. In 27.5% of legs the small saphenous vein terminated in the principal deep vein of the leg (popliteal or lower femoral vein), in 25% of legs the small saphenous vein, or a branch arising from it, communicated with the great saphenous vein. In the remaining legs, there was a wide variety and combination of communications with other veins, including the deep femoral vein, the mid-thigh perforator vein, muscular veins and even the inferior gluteal vein in three legs. Other studies have shown that Giacomini veins can be affected by varicose disease with reflux either upwards or downwards in the thigh to the greater and lesser saphenous veins respectively. The Perforating Veins The perforating veins connect the superficial veins to the deep veins. They are numerous and very variable in both size and location. In the past, they were often known by eponymous designations but with the revised nomenclature they are now identified by their

anatomical location – for example: medial, lateral, or anterior ankle perforator – full details are given in the consensus statement on venous nomenclature. They are normally less than 5 mm in diameter and blood flows inwards from the superficial to the deep systems. (Myron Pozniak & Paul Allan 2014)

2.2-Physiology

2.2.1- Bones and Joints

The function of the skeleton:- 1-Provides a framework that supports the body; the muscles that are attached to bones move the skeleton 2-Protects some internal organs from mechanical injury; the rib cage protects the heart and lungs, for example 3-Contains and protects the red bone marrow, the primary hemopoietic (blood-forming) tissue. Provides a storage site for excess calcium4-Calcium may be removed from bone to maintain a normal blood calcium level, which is essential for blood clotting and proper functioning of muscles and nerves. (Valerie C. Scanlon & Tina Sanders 2007)

The bones of the lower limbs support the entire body when we are standing, and the pelvic girdle supports the abdominal. All bones in the fetus have red bone marrow that produces blood cells. In the adult, only certain bones produce blood cells. The skeleton stores minerals and fat. All bones have a matrix that contains calcium phosphate, a source of calcium ions and phosphate ions in the blood. Fat is stored in yellow bone marrow. The skeleton, along with the muscles, permits flexible body movement. (Mader 2004)

2.2.1.1-The Hip Joint (Articulatio Coxae)

The hip joint is a ball-and-socket joint in which the head of the femur moves in the acetabulum. The acetabulum is formed by all three components of the hipbone, (ilium, ischium and pubic bone). It grasps almost half the ball of the femur, giving the hip joint a solid bony channel. The joint contact is augmented by a fibro cartilaginous lip, (acetabular labrum), which extends the joint beyond its equator by forming a ring around the acetabulum. In this way the joint maintains solidity and stability in every position. The capsule of the joint is rein forced by three strong ligaments running from the three parts of the hip

bone: the iliofemoral, the pubofemoral, and the ischiofemoral ligaments. The iliofemoral ligament is the strongest ligament of the body; with at ensile strength of 350kg. The ligaments run a characteristic spiral course around the neck of the femur. In the erect posture they are tense, while they relax during flexion of the hip joint, allowing considerable mobility during flexion. (A Faller et al 2004)

Because a ball-and-socket joint has three main axes of movement, the leg can be moved around a transverse axis anteriorly and posteriorly (flexion and extension), a sagittal axis away from and toward the trunk (abduction and adduction), and a vertical axis around which it is rotated (internal and external rotation). The most important hip flexors muscles include the deep iliopsoas, the sartorius, and the rectusfemoris muscles. The extensor sat the hip are the gluteusmaximus muscle and the muscles running from the ischial tuberosity to the lower leg (ischiocrural muscles) the biceps femoris, the semitendinosus, and semimembranosus muscles). The gluteusmedius and the gluteusmaximus muscles function both as abdoctors and as internal and external rotators. On the medial side of the thigh, between the extensors and the flexors runs a group of five muscles that adduct the thigh toward the trunk (adductors, e.g., m. adductormagnus). They take their origin from the pelvis and are inserted into the medial lip of the lineaaspera of the femur. The external rotators arise from the posterior aspect of the pelvis and run to the femur, e.g., the gluteusmaximus. Internal rotators are the anterior parts of the gluteusmedius and gluteusminimus. During movement of the hip, the leg may be moved freely on the hip or the leg may be fixed (standing) and the hip moved against it. In walking these functions alternate. (A Faller et al 2004)

2.2.1.2-The Knee Joint (Articulatio Genus)

The knee joint is the largest joint of the human body. It is a compound joint, formed by the combined action of the femur, tibia, patella and two fibro cartilaginous disks, (menisci). The joint consists of the joint between the condyles of the femur and the tibia and the joint between the femur and the patella. The two menisci adapt the joint surfaces of the femur and the tibia to each other and increase the surface for the transmission of force. When the knee joint is flexed, the femur executes a combined gliding and rolling motion over the tibial articular surface, during which the menisci are shifted more posteriorly the further the knee

is flexed. The movements of the knee joint are guided by two sets of ligaments, the medial and lateral collateral ligaments and the anterior and posterior cruciate ligaments. While the collateral ligaments primarily stabilize the extended leg, the cruciate ligaments take over that function in the flexed knee joint. Because of the uneven curvature of the femoral condyles, the collateral ligaments are fully tensed only during extension of the knee joint; they are relaxed during flexion. With the knee flexed, the cruciate ligaments Limit internal and external rotation of the lower leg, internal rotation being more severely limited than external rotation by the coiling of the cruciate ligaments. (A Faller et al 2004)

The hip muscles that move the thigh are anchored to the pelvic bone and cross the hip joint to the femur. Among these are the gluteus maximus (extension), gluteusmedius (abduction), and iliopsoas (flexion). The muscles that form the thigh include the quadriceps group anteriorly and the hamstring group posteriorly. For most people, the quadriceps is stronger than the hamstrings, which is why athletes more often have a "pulled hamstring" rather than a "pulled quadriceps." Movement of the knee joint depends on thigh muscles and lower leg muscles. Movement of the foot depends on lower leg muscles such as the gastrocnemius (dorsiflexion or flexion) and the tibialis anterior (plantar flexion or extension. (Valerie C. Scanlon & Tina Sanders 2007).

Muscles inserted into the medial surface of the tibia. semitendinosus and semimembranosus) are internal rotators. The biceps femoris is inserted into the head of the fibula, and is the only external rotator of the lower leg. All three muscles are flexors of the knee joint, as is the sartorius. The most important extensor of the knee joint is the quadriceps femoris, inserted by the patellar ligament into the tibial tuberosity. The patella, the largest sesamoid bone in the human body, is embed in the patellar ligament. It is triangular in shape and articulates with the anterior surface of the distal femur (patellar surface). As the knee flexes, the patella moves downward because there is considerable transfer of force in the joint between femur and patella, especially during flexion, this is the most highly stressed joint in the body, and is the earliest to show degenerative changes in its cartilage. The Ankle Joint (Articulatio Talocruralis) and the Intertarsal Joint (Articulationes Subtalaris and Talocalcaneonavicularis) The movements of the foot against the lower leg occur in two joints: the ankle joint and the talocalcaneonavicular joint. (A Faller et al 2004)

2.2.1.3-Ankle Joint

The ankle joint is formed by the tibia, fibula, and talus. The distal ends of the tibia and fibula forma mortise made up of the medial and lateral malleoli, which grasp the trochlea of the talus. The talus and connects both malleoli. The movements at this joint are called dorsiflexion (lifting of the point of the foot) and plantar flexion (lowering of the point of the foot toward the sole=plantar side of the foot). When the foot is fixed, the lower leg moves backward and forward. A strongest of ligaments stabilizes the ankle joint. It consists of three lateral ligaments and the medial deltoid ligament. (A Faller et al 2004)

2.2.1.4-Intertarsal Joint

In the intertarsal joint, the talus articulates with the calcaneus and the navicular bone. These joints, in fact, composed of two joints that are anatomically entirely separate: posteriorly the subtalar joint, and anteriorly the talocalcaneonavicular joint. In the subtalar joint, the talus articulates with the calcaneus; in the talocalcaneonavicular joint, the ball-shaped head of the talusarticulates with the calcaneus and the navicular bone. The axis of movement in the talocalcaneonavicular joint runs from the center of the navicular obliquely downward, laterally, and backward through the calcaneum. The movements in this joint are lateral movements called supination (lifting of the inner =medial border of the foot) and pronation (lifting of the outer lateral border of the foot) (A Faller et al 2004)

2.2.2-Muscles

There are more than 600 muscles in the human body. The primary function of the muscular system is to move the skeleton. The muscle contractions required for movement also produce heat, which contributes to the maintenance of a constant body temperature. The other body systems directly involved in movement are the nervous, respiratory, and circulatory systems. The nervous system transmits the electrochemical impulses that cause muscle cells to contract. The respiratory system exchanges oxygen and carbon dioxide between the air and blood. The circulatory system brings oxygen to the muscles and takes carbon dioxide away. (Scanlon & Sanders 2008)

The muscles running from the lower leg to the foot acton the ankle joint and the talocalcaneonavicular joint. They are divided onto three groups according to their position on the leg: the posterior calf muscles. triceps

surae, tibialis posterior, flexordigitorumlongus, and flexorhallucislongus) act as supinators, plantar flexors (point of the foot turned down),and flexors of the toes(mm. flexordigitorumlongus and flexorhallucislongus only).The muscles of the anterior compartment of the lower leg(mm. tibialis anterior, extensordigitorumlongus, and extensorhallucislongus) act as dorsiflexors (lifting of the point of the foot) and supinators of the foot (m. tibialisanterior only). The muscles of the lateral compartment of the calf (mm. peroneilongus and brevis) are primarily pronators and assist plantar flexion. The strongest group are the superficial muscles of the calf (m. triceps surae), usually considered to be composed of two muscles, the soleus and the gastrocnemius muscles. They join to form the achilles tendon, which is inserted into the calcanealtubercle. If the Achilles tendon is torn, the triceps surae cannot act and it becomes impossible to stand on tip toe; supination is also severely impaired. (A Faller et al 2004)

Skeletal muscle contains long organized units called muscle fibers. The characteristic long fibers are under voluntary control, allowing us to contract a **muscle** and move a joint. The blood vessels, lymphatics, and nerves follow the fibrous partitions between the bundles of muscle. Several different types of muscles are present in the human body. Muscles have fibers that run parallel to the bone, have a fan shape, or form a **pennate** pattern. These feather-like muscle patterns run oblique to the long axis of the muscle and are unipennate, bipennate, multipennate, or circumpennate. Think of a feather and how thefibers grow from a central section. Half of this feather is unipennate, whereas the whole feather is bipennate. A multipennate muscle is a division of several feather-like sections in one muscle, whereas the circumpennate is the convergence of fibers to a central tendon.

(Sandra 2012)

All muscle cells are specialized for contraction. When these cells contract, they shorten and pull a bone to produce movement. Each skeletal muscle is made of thousands of individual muscle cells, which also may be called muscle fibers. Depending on the work a muscle is required to do, variable numbers of muscle fibers contract. When picking up a pencil, for example, only a small portion of the muscle fibers in each finger muscle will contract. If the muscle has more work to do, such as picking up a book, more muscle fibers will contract to accomplish the task. Muscles are anchored firmly to bones by tendons. Most tendons are

rope-like, but some are flat; a flat tendon is called an aponeurosis. Tendons are made of fibrous connective tissue, which, you may remember, is very strong and merges with the fascia that covers the muscle and with the periosteum, the fibrous connective tissue membrane that covers bones. A muscle usually has at least two tendons, each attached to a different bone. The more immobile or stationary attachment of the muscle is its origin; the more movable attachment is called the insertion. The muscle itself crosses the joint of the two bones to which it is attached, and when the muscle contracts it pulls on its insertion and moves the bone in a specific direction. (Scanlon & Sanders 2008)

2.2.2.1-Muscles of the Hip and Lower Limb

The muscles of the hip and thigh are tending to be large and heavy because they are used to move the entire weight of the body and to resist the force of gravity. Therefore, they are important for movement and balance. (Mader 2004)

2.2.2.2-Muscles That Move the Thigh

The muscles that move the thigh have at least one origin on the pelvic girdle and insert on the femur. Notice that the iliopsoas is an anterior muscle that moves the thigh, while the gluteal muscles ("gluts") are posterior muscles that move the thigh. The adductor muscles are medial muscles. Before studying the action of these muscles, review the movement of the hip joint when the thigh flexes, extends, abducts, and adducts Iliopsoas (includes psoas major and iliacus) originates at the ilium and the bodies of the lumbar vertebrae, and inserts on the femur anteriorly. This muscle is the prime mover for flexing the thigh and also the trunk, as when we bow. As the major flexor of the thigh, the iliopsoas is important to the process of walking. It also helps prevent the trunk from falling backward when a person is standing erect. The gluteal muscles form the buttocks. We will consider only the gluteus maximus and the gluteus medius. Gluteus maximus is the largest muscle in the body and covers a large part of the buttock (gluteus means buttocks in Greek). It originates at the ilium and sacrum, and inserts on the femur. The gluteus maximus is a prime mover of thigh extension, as when a person is walking, climbing stairs, or jumping from a crouched position. Notice that the iliopsoas and the gluteus maximus are antagonistic muscles. Gluteus medius lies partly behind the gluteus maximus. It runs

between the ilium and the femur, and functions to abduct the thigh. The gluteus maximus assists the gluteus medius in this function. Therefore, they are synergistic muscles. Adductor group muscles (pectineus, adductor longus, adductor magnus, gracilis) are located on the medial thigh. All of these muscles originate from the pubis and ischium, and insert on the femur. Adductor muscles adduct the thigh—that is, they lower the thigh sideways from a horizontal position. Because they press the thighs inward, these are the muscles that keep a rider on a horse. Notice that the gluts and the adductor group are antagonistic muscles. (Mader 2004)

2.2.2.3-Muscles That Move the Leg The muscles that move the leg originate from the pelvic girdle or femur and insert on the tibia. Before studying these muscles, review the movement of the knee when the leg extends and when it flexes. Quadriceps femoris group (rectus femoris, vastus lateralis, vastus medialis, vastus intermedius), also known as the "quads," is found on the anterior and medial thigh. The rectus femoris, which originates from the ilium, is external to the vastus intermedius. These muscles are the primary extensors of the leg, as when you kick a ball by straightening your knee. Sartorius is a long, strap-like muscle that has its origin on the iliac spine and then goes across the anterior thigh to insert on the medial side of the knee. Because this muscle crosses both the hip and knee joint, it acts on the thigh in addition to the leg. The insertion of the sartorius is such that it flexes both the leg and the thigh. It also rotates the thigh laterally, enabling us to sit cross-legged, as tailors were accustomed to do in another era. Therefore, it is sometimes called the "tailor's muscle," and in fact, sartor means tailor in Latin. Hamstring group (biceps femoris, semimembranosus, and semitendinosus) is located on the posterior thigh. Notice that these muscles also cross the hip and knee joint because they have origins on the ischium and insert on the tibia. They flex and rotate the leg medially, but they also extend the thigh. Their strong tendons can be felt behind the knee. These same tendons are present in hogs and were used by butchers as strings to hang up hams for smoking—hence, the name. Notice that the quadriceps femoris group and the hamstring group are antagonistic muscles in that the quads extend the leg and the hamstrings flex the leg. (Mader 2004)

2.2.2.4-Muscles That Move the Ankle and Foot Gastrocnemius is a muscle of the posterior leg, where it forms a large part of the calf. It arises from the femur; distally, the muscle joins the strong calcaneal tendon, which attaches to the calcaneus bone (heel). The gastrocnemius is a powerful plantar flexor of the foot that aids in pushing the body forward during walking or running. It is sometimes called the "toe dancer's muscle" because it allows a person to stand on tiptoe. Tibialis anterior is a long, spindle-shaped muscle of the anterior leg. It arises from the surface of the tibia and attaches to the bones of the ankle and foot. Contraction of this muscle causes dorsiflexion and inversion of the foot. Peroneus muscles (peroneus longus, peroneus brevis) are found on the lateral side of the leg, connecting the fibula to the metatarsal bones of the foot. These muscles avert the foot and also help bring about plantar flexion Flexor and extensor digitorum longus muscles are found on the lateral and posterior portion of the leg. They arise mostly from the tibia and insert on the toes. They flex and extend the toes, respectively, and assist in other movements of the feet. (Mader 2004)

2.2.3-The Blood

One of the simplest and most familiar life-saving medical procedures is a blood transfusion. As you know, however, the blood of one individual is not always compatible with that of another person. The ABO blood types were discovered in the early 1900s by Karl Landsteiner, an Austrian-American. He also contributed to the discovery of the Rh factor in 1940. In the early 1940s, Charles Drew, an African-American, developed techniques for processing and storing blood plasma, which could then be used in transfusions for people with any blood type. When we donate blood today, our blood may be given to a recipient as whole blood, or it may be separated into its component parts, and recipients will then receive only those parts they need, such as red cells, plasma, factor 8, or platelets. Each of these parts has a specific function, and all of the functions of blood are essential to our survival. The general functions of blood are transportation, regulation, and protection. Materials transported by the blood include nutrients, waste products, gases, and hormones. The blood helps regulate fluid–electrolyte balance, acid–base balance, and the body temperature. Protection against pathogens is provided by white

blood cells, and the blood clotting mechanism prevents excessive loss of blood after injuries. (Valerie C. Scanlon & Tina Sanders 2008)

2.2.3.1-Characteristic of Blood

Blood has distinctive physical characteristics: **Amount**—a person has 4 to 6 liters of blood, depending on his or her size. Of the total blood volume in the human body, 38% to 48% is composed of the various blood cells, also called formed elements. The remaining 52% to 62% of the blood volume is plasma, the liquid portion of blood. **Color**—you're probably saying to yourself, "Of course, it's red!" mention is made of this obvious fact, however, because the color does vary. Arterial blood is bright red because it contains high levels of oxygen. Venous blood has given up much of its oxygen in tissues, and has a darker, dull red color. This may be important in the assessment of the source of bleeding. If blood is bright red, it is probably from a severed artery, and dark red blood is probably venous blood. **PH—**the normal pH range of blood is 7.35 to 7.45, which is slightly alkaline. Venous blood normally has a lower pH than does arterial blood because of the presence of more carbon dioxide. **Viscosity**—this means thickness or resistance to flow. Blood is about three to five times thicker than water. Viscosity is increased by the presence of blood cells and the plasma proteins, and this thickness contributes to normal blood pressure. (Valerie C. Scanlon & Tina Sanders 2008)

2.2.3.2- Plasma

Plasma is the liquid part of blood and is approximately 91% water. The solvent ability of water enables the plasma to transport many types of substances. Nutrients absorbed in the digestive tract, such as glucose, amino acids, and minerals, are circulated to all body tissues. Waste products of the tissues, such as urea and creatinine, circulate through the kidneys and are excreted in urine. Hormones produced by endocrine glands are carried in the plasma to their target organs, and antibodies are also transported in plasma. Most of the carbon dioxide produced by cells is carried in the plasma in the form of bicarbonate ions (HCO_3^-). When the blood reaches the lungs, the CO_2 is re-formed, diffuses into the alveoli, and is exhaled. Also in the plasma are the **plasma proteins**. The clotting factors **prothrombin**, **fibrinogen**, and others are synthesized by the liver and circulate until activated to form a clot in a ruptured or damaged blood vessel. **Albumin** is the most abundant plasma protein. It

too is synthesized by the liver. Albumin contributes to the colloid osmotic pressure of blood, which pulls tissue fluid into capillaries. This is important to maintain normal blood volume and blood pressure. Other plasma proteins are called **globulins**. Alpha and beta globulins are synthesized by the liver and act as carriers for molecules such as fats. The gamma globulins are antibodies produced by lymphocytes. Antibodies initiate the destruction of pathogens and provide us with immunity. Plasma also carries body heat. Heat is one of the by-products of cell respiration (the production of ATP in cells). Blood is warmed by flowing through active organs such as the liver and muscles. This heat is distributed to cooler parts of the body as blood continues to circulate. (Valerie C. Scanlon & Tina Sanders 2008)

2.2.3.3-Blood cells

There are three kinds of blood cells: red blood cells, white blood cells, and platelets. Blood cells are produced from stem cells in **hemopoietic tissue**. After birth this is primarily the **red bone marrow**, found in flat and irregular bones such as the sternum, hip bone, and vertebrae. Lymphocytes mature and divide in **lymphatic tissue**, found in the spleen, lymph nodes, and thymus gland. The thymus contains stem cells that produce T lymphocytes, and the stem cells in other lymphatic tissue also produce lymphocytes. (Valerie C. Scanlon & Tina Sanders 2008)

2.2.3.3.1-Red Blood Cells

Also called **erythrocytes**, red blood cells (RBCs) are biconcave discs, which means their centers are thinner than their edges. You may recall from Chapter 3 that red blood cells are the only human cells without nuclei. Their nuclei disintegrate as the red blood cells mature and are not needed for normal functioning. A normal RBC count ranges from 4.5 to 6.0 million cells per microliter (_L) of blood (1 microliter _ 1 mm3 _ one millionth of a liter, a very small volume). RBC counts for men are often toward the high end of this range; those for women are often toward the low end. Another way to measure the amount of RBCs is the **hematocrit**. This test involves drawing blood into a thin glass tube called a capillary tube, and centrifuging the tube to force all the cells to one end. The percentages of cells and plasma can then be determined. Because RBCs are by far the most abundant of the blood cells, a normal hematocrit range is just like that of the total blood cells: 38% to 48%. Both RBC count and

hematocrit (Hct) are part of a complete blood count (CBC). (Valerie C. Scanlon & Tina Sanders 2008)

2.2.3.3.1.1-Blood types

Our blood types are genetic; that is, we inherit genes from our parents that determine our own types. There are many red blood cell factors or types; we will discuss the two most important ones: the **ABO group** and the **Rh factor**. The **ABO group** contains four blood types: A, B, AB, and O. The letters A and B represent antigens (protein-oligosaccharides) on the red blood cell membrane. A person with type A blood has the A antigen on the RBCs, and someone with type B blood has the B antigen. Type AB means that both A and B antigens are present, and type O means that neither the A nor the B antigen is present. Circulating in the plasma of each person are natural antibodies for those antigens not present on the RBCs. Therefore, a type A person has anti-B antibodies in the plasma; a type B person has anti-A antibodies; a type AB person has neither anti-A nor anti-B antibodies; and a type O person has both anti-A and anti-B antibodies. These natural antibodies are of great importance for transfusions. If possible, a person should receive blood of his or her own type; only if this type is not available should another type be given. For example, let us say that a type A person needs a transfusion to replace blood lost in hemorrhage. If this person were to receive type B blood, what would happen? The type A recipient has anti-B antibodies that would bind to the type B antigens of the RBCs of the donated blood. The type B RBCs would first clump (**agglutination**) then rupture (**hemolysis**), thus defeating the purpose of the transfusion. An even more serious consequence is that the hemoglobin of the ruptured RBCs, now called free hemoglobin, may clog the capillaries of the kidneys and lead to renal damage or renal failure. You can see why **typing** and **cross-matching** of donor and recipient blood in the hospital laboratory is so important before any transfusion is given. This procedure helps ensure that donated blood will not bring about a hemolytic transfusion reaction in the recipient. You may have heard of the concept that a person with type O blood is a “universal donor.” Usually, a unit of type O negative blood may be given to people with any other blood type. This is so because type O RBCs have neither the A nor the B antigens and will not react with whatever antibodies the recipient may have. If only one unit (1 pint) of blood is given, the anti-A and anti-B antibodies in the type O

blood plasma will be diluted in the recipient's blood plasma and will not have a harmful effect on the recipient's RBCs. The term negative, in O negative, the universal donor, refers to the Rh factor, which we will now consider. The **Rh factor** is another antigen (often called D) that may be present on RBCs. People whose RBCs have the Rh antigen are Rh positive; those without the antigen are Rh negative. Rh-negative people do not have natural antibodies to the Rh antigen, and for them this antigen is foreign. If an Rh-negative person receives Rh-positive blood by mistake, antibodies will be formed just as they would be to bacteria or viruses. A first mistaken transfusion often does not cause problems, because antibody production is slow upon the first exposure to Rh-positive RBCs. A second transfusion, however, when anti-Rh antibodies are already present, will bring about a transfusion reaction, with hemolysis and possible kidney damage. (Valerie C. Scanlon & Tina Sanders 2008)

2.2.3.3.2-White Blood Cells

White blood cells (WBCs) are also called **leukocytes**. There are five kinds of WBCs; all are larger than RBCs and have nuclei when mature. The nucleus may be in one piece or appear as several lobes or segments. Special staining for microscopic examination gives each kind of WBC a distinctive appearance. A normal WBC count (part of a CBC) is 5,000 to 10,000 per _L. Notice that this number is quite small compared to a normal RBC count. Many of our WBCs are not circulating within blood vessels but are carrying out their functions in tissue fluid or in lymphatic tissue. (Valerie C. Scanlon & Tina Sanders 2008)

2.2.3.3.2.1-Classification

The five kinds of white blood cells, all produced in the red bone marrow (and some lymphocytes in lymphatic tissue), may be classified in two groups: granular and agranular. The granular leukocytes are the **neutrophils**, **eosinophils**, and **basophils**, which usually have nuclei in two or more lobes or segments, and have distinctly colored granules when stained. Neutrophils have light blue granules, eosinophils have red granules, and basophils have dark blue granules. The agranular leukocytes are **lymphocytes** and **monocytes**, which have nuclei in one piece. Monocytes are usually quite a bit larger than lymphocytes. A **differential WBC count** (part of a CBC) is the percentage of each kind of leukocyte.. (Valerie C. Scanlon & Tina Sanders 2008)

2.2.3.3.2.2-Functions

White blood cells all contribute to the same general function, which is to protect the body from infectious disease and to provide **immunity** to certain diseases. Each kind of leukocyte makes a contribution to this very important aspect of homeostasis. Neutrophils and monocytes are capable of the **phagocytosis** of pathogens. Neutrophils are the more abundant phagocytes, but the monocytes are the more efficient phagocytes, because they differentiate into **macrophages**, which also phagocytize dead or damaged tissue at the site of any injury, helping to make tissue repair possible. During an infection, neutrophils are produced more rapidly, and the immature forms, called **band cells** may appear in greater numbers in peripheral circulation (band cells are usually less than 10% of the total neutrophils). The term "band" refers to the nucleus that has not yet become segmented, and may look somewhat like a dumbbell. Eosinophils are believed to detoxify foreign proteins and will phagocytize anything labeled with antibodies. This is especially important in allergic reactions and parasitic infections such as trichinosis (a worm parasite). Basophils contain granules of heparin and histamine. **Heparin** is an anticoagulant that helps prevent abnormal clotting within blood vessels. **Histamine**, you may recall, is released as part of the inflammation process, and it makes capillaries more permeable, allowing tissue fluid, proteins, and white blood cells to accumulate in the damaged area. There are two major kinds of lymphocytes, T cells and B cells, and a less numerous third kind called natural killer cells. For now we will say that **T cells** (or T lymphocytes) help recognize foreign antigens and may directly destroy some foreign antigens. **B cells** (or B lymphocytes) become plasma cells that produce antibodies to foreign antigens. Both T cells and B cells provide memory for immunity. **Natural killer cells** (NK cells) destroy foreign cells by chemically rupturing their membranes. As mentioned earlier, leukocytes function in tissue fluid as well as in the blood. Many WBCs are capable of self-locomotion (ameboid movement) and are able to squeeze between the cells of capillary walls and out into tissue spaces. Macrophages provide a good example of the dual locations of leukocytes. Some macrophages are "fixed," that is, stationary in organs such as the liver, spleen, and red bone marrow (part of the tissue macrophage or RE system—the samemacrophages that phagocytize old RBCs) and in the lymph nodes. They phagocytize pathogens that circulate in blood or

lymph through these organs. Other "wandering" macrophages move about in tissue fluid, especially in the areolar connective tissue of mucous membranes and below the skin. Pathogens that gain entry into the body through natural openings or through breaks in the skin are usually destroyed by the macrophages and other leukocytes in connective tissue before they can cause serious disease. The alveoli of the lungs, for example, have macrophages that very efficiently destroy pathogens that enter with inhaled air. A high WBC count, called **leukocytosis**, is often an indication of infection. **Leukopenia** is a low WBC count, which may be present in the early stages of diseases such as tuberculosis. Exposure to radiation or to chemicals such as benzene may destroy WBCs and lower the total count. Such a person is then very susceptible to infection. **Leukemia**, or malignancy of leukocyte-forming tissues. The white blood cell types (analogous to RBC types such as the ABO group) are called human leukocyte antigens (HLA): White Blood Cell Types: HLA. (Valerie C. Scanlon & Tina Sanders 2008)

2.2.3.3.3-Platelates

The more formal name for platelets is thrombocytes, which are not whole cells but rather fragments or pieces of cells. Some of the stem cells in the red bone marrow differentiate into large cells called megakaryocytes ,which break up into small pieces that enter circulation. These small, oval, circulating pieces are platelets, which may last for 5 to 9 days, if not utilized before that. Thrombopoietin is a hormone produced by the liver that increases the rate of platelet production. A normal platelet count (part of a CBC) is 150,000 to 300,000/_L (the high end of the range may be extended to 500,000). Thrombocytopenia is the term for a low platelet count. (Valerie C. Scanlon & Tina Sanders 2008)

2.2.3.3.3.1-Function

Platelets are necessary for **hemostasis**, which means prevention of blood loss. There are three mechanisms, and platelets are involved in each.

2.2.3.3.3.1.1-Vascular spasm—when a large vessel such as an artery or vein is severed, the smooth muscle in its wall contracts in response to the damage (called the myogenic response). Platelets in the area of the rupture release serotonin, which also brings about vasoconstriction. The diameter of the vessel is thereby made smaller, and the smaller opening may then be blocked by a blood clot. If the vessel did not constrict first,

the clot that forms would quickly be washed out by the force of the blood pressure. (Scanlon & Sanders 2008)

2.2.3.3.3.1.2-Platelet plugs—when capillaries rupture, the damage is too slight to initiate the formation of a blood clot. The rough surface, however, causes platelets to change shape (become spiky) and become sticky. These activated platelets stick to the edges of the break and to each other. The platelets form a mechanical barrier or wall to close off the break in the capillary. Capillary ruptures are quite frequent, and platelet plugs, although small, are all that is needed to seal them. Would platelet plugs be effective for breaks in larger vessels? No, they are too small, and though they do form, they are washed away (until a clot begins to form that can contain them). Would vascular spasm be effective for capillaries? Again, the answer is no, because capillaries have no smooth muscle and cannot constrict at all. (Scanlon & Sanders 2008)

2.2.3.3.3.1.3-Chemical clotting—the stimulus for clotting is a rough surface within a vessel, or a break in the vessel, which also creates a rough surface. The more damage there is, the faster clotting begins, usually within 15 to 120 seconds. The clotting mechanism is a series of reactions involving chemicals that normally circulate in the blood and others that are released when a vessel is damaged. The chemicals involved in clotting include platelet factors, chemicals released by damaged tissues, calcium ions, and the plasma proteins prothrombin, fibrinogen, Factor 8, and others synthesized by the liver. (These clotting factors are also designated by Roman numerals; Factor 8 would be Factor VIII.) Vitamin K is necessary for the liver to synthesize prothrombin and several other clotting factors (Factors 7, 9, and 10). Most of our vitamin K is produced by the bacteria that live in the colon; the vitamin is absorbed as the colon absorbs water and may be stored in the liver. Chemical clotting is usually described in three stages, Stage 1 begins when a vessel is cut or damaged internally, and includes all of the factors shown. As you follow the pathway, notice that the product of stage 1 is prothrombin activator, which may also be called prothrombinase. Each name tells us something. The first name suggests that this chemical activates prothrombin, and that is true. The second name ends in “ase,” which indicates that this is an enzyme. The traditional names for enzymes use the substrate of the enzyme as the first part of the name, and add

“ase.” So this chemical must be an enzyme whose substrate is prothrombin, and that is also true. The stages of clotting may be called a cascade, where one leads to the next, as inevitable as water flowing downhill. Prothrombin activator, the product of stage 1, brings about the stage 2 reaction: converting prothrombin to thrombin. The product of stage 2, thrombin, brings about the stage 3 reaction: converting fibrinogen to fibrin (see Box 11–6: Hemophilia). The clot itself is made of **fibrin**, the product of stage 3. Fibrin is a thread-like protein. Many strands of fibrin form a mesh that traps RBCs and platelets, and creates a wall across the break in the vessel. Once the clot has formed and bleeding has stopped, clot retraction and fibrinolysis occur. Clot retraction requires platelets, ATP, and Factor 13 and involves folding of the fibrin threads to pull the edges of the rupture in the vessel wall closer together. This will make the area to be repaired smaller. The platelets contribute in yet another way, because as they disintegrate they release platelet-derived growth factor (PDGF), which stimulates the repair of blood vessels (growth of their tissues) . As repair begins, the clot is dissolved, a process called fibrinolysis. It is important that the clot be dissolved, because it is a rough surface, and if it were inside a vessel it would stimulate more and unnecessary clotting, which might eventually obstruct blood flow. (Valerie C. Scanlon & Tina Sanders 2008)

2.2.3.3.3.2-Prevention of abnormal clotting

Clotting should take place to stop bleeding, but too much clotting would obstruct vessels and interfere with normal circulation of blood. Clots do not usually form in intact vessels because the **endothelium** (simple squamous epithelial lining) is very smooth and repels the platelets and clotting factors. If the lining becomes roughened, as happens with the lipid deposits of atherosclerosis, a clot will form. Heparin, produced by basophils, is a natural anticoagulant that inhibits the clotting process (although heparin is called a “blood thinner,” it does not “thin” or dilute the blood in any way; rather it prevents a chemical reaction from taking place). The liver produces a globulin called **antithrombin**, which combines with and inactivates excess thrombin. Excess thrombin would exert a positive feedback effect on the clotting cascade, and result in the splitting of more prothrombin to thrombin, more clotting, more thrombin formed, and so on. Antithrombin helps to prevent this, as does the fibrin

of the clot, which adsorbs excess thrombin and renders it inactive. All of these factors are the external brake for this positive feedback mechanism. Together they usually limit the fibrin formed to what is needed to create a useful clot but not an obstructive one. Thrombosis refers to clotting in an intact vessel; the clot itself is called a **thrombus**. Coronary thrombosis, for example, is abnormal clotting in a coronary artery, which will decrease the blood (oxygen) supply to part of the heart muscle. An **embolism** is a clot or other tissue transported from elsewhere that lodges in and obstructs a vessel. (Scanlon & Sanders 2008)

2.2.3.4- Dissolving clot

Abnormal clots may cause serious problems in coronary arteries, pulmonary arteries, cerebral vessels, and even veins in the legs. However, if clots can be dissolved before they cause death of tissue, normal circulation and tissue functioning may be restored. One of the first substances used to dissolve clots in coronary arteries was **streptokinase**, which is actually a bacterial toxin produced by some members of the genus Streptococcus. Streptokinase did indeed dissolve clots, but its use created the possibility of clot destruction throughout the body, with serious hemorrhage a potential consequence. Safer chemicals called third-generation thrombolytics are now used (thrombo _ "clot" and lytic _ "to lyse" or "split"). In a case of coronary thrombosis, if a thrombolytic can be administered within a few hours, the clot may be dissolved and permanent heart damage prevented. The same procedure is also used to prevent permanent brain damage after strokes (CVAs) caused by blood clots. (Valerie C. Scanlon & Tina Sanders 2008)

2.2.3.5-Summary

All of the functions of blood— transport, regulation, and protection— contribute to the homeostasis of the body as a whole. However, these functions could not be carried out if the blood did not circulate properly. The circulation of blood throughout the blood vessels depends upon the proper functioning of the heart, the pump of the circulatory system, which is the subject of our next chapter. (Scanlon & Sanders 2008)

The circulatory system acts as the transport system for the body. It brings oxygen to tissues and carries carbon dioxide away. Nutrients are picked

up from the digestive system and delivered throughout the body, while waste products are carried away so that certain organs may remove them. The circulatory system consists of the heart and blood vessels and includes arteries, veins, and capillaries. Blood is the transport medium that is pumped throughout the body. It is a liquid tissue that consists of plasma and formed elements (red blood cells, white blood cells, and platelets). It is important for the medical assistant to have an understanding of this system in order to effectively perform electrocardiograms, phlebotomy, and blood tests. (Kathryn A. Booth & Terri D. Wyman 2008**)**

2.3 PATHOLOGY

2.3.1-Aneurysm

Aneurysms most commonly affect the aorta and the poplitealartery but may also occur in the femoral and iliac vessels, in particular in patients with dilated angiopathy (Schuler et al. 1993). Aneurysms are identified on gray-scale scans as saccular or spindle-shaped dilatations of the vessel lumen. Mural thrombi in the aneurysm are often identified by their slightly higher echogenicity relative to flowing blood in the patent lumen. Confirmation is obtained by color duplex ultrasound, which indicates thrombosis by the absence of color coding. The thromboticdeposits can cause stenosis, inparticular when they occur at the distal end of an aneurysm. No flow signals will be depicted in a completely thrombosed aneurysm. Angiography is not the method of reference for assessing a partially thrombosed aneurysm while computed tomography (CT) depicts the morphology and extent of an aneurysm but provides no hemodynamic information. Patients with an isolated occlusion in the popliteal territory should be examined ultrasonographically for exclusion of a thrombosed aneurysm or compression syndrome prior to a radiologic intervention. Aneurysms of the popliteal artery often occur bilaterally and a concomitant aneurysm of the abdominal aorta is present in 25–30%of cases. Aneurysms can occlude or rupture or cause embolic occlusion of peripheral vessels with the necessity of amputation. Surgery is indicated for all aneurysms with a diameter greater than 2 cm and smaller ones when they are saccular or contain thrombotic deposits. Thrombotic aneurysms in the knee area carry a higher risk of arterial embolism even

when they are small because they are exposed to the shearing forces of the bending knee. Duplex scanning is the method of first choice, yielding reliable information on the diameter of the aneurysm, its shape, and the presence of thrombosis and thus providing the foundation for planning the surgical procedure. Scanning of the superficial aneurysm is performed with a high-resolution transducer. The diameter is measured and thromboticmaterial identified in the lumen (absence of color coding) in transverse orientation while the shape is assessed in the longitudinal plane. False aneurysms (pseudoaneurysms) are a typical complication of arterial puncture performed for diagnostic angiography or interventional procedures. Their incidence is up to 4% after percutaneous transluminal angioplasty (PTA) and cardiac catheterization (Hust and Schuler 1992; Moll et al. 1991). A subgroup of false aneurysms are suture aneurysms developing after vascular surgery, in particular after bypass operations. A false aneurysmmust be differentiated froma perivascular hematoma with transmitted pulsation but this is difficult on clinical grounds (Thomas et al. 1989). Using duplex ultrasound, an aneurysm can be differentiated fromhypoechoic, perivascular structures such as hematoma, seroma, or lymphocele by the depiction of to-and-fro flow, which is diagnostic of a false aneurysm and requires no angiographic confirmation. To-and-fro flow occurs in the neck of an aneurysm due to changing pressures: At the high intraluminal pressure during systole, blood flows through the narrow neck into the aneurysm at a rather high velocity. Under the reversed pressure conditions during diastole, the blood flows back into the vessel at a slightly lower flow rate. Reflux is typically turbulent. Before ultrasound enabled the precise localization of the aneurysmal neck relative to the skin surface, the therapy of choice was surgical revision with closure. With the ultrasound scanners now available, it is possible to occlude over 90%of the aneurysms by ultrasound-guided compression of the neck (Fellmeth et al. 1991; Hust et al. 1993). Thrombosis occurs after 10 to 30 minutes of compression of the neck. Alternatively, thrombosis of a false aneurysm can be induced by injecting thrombin into the aneurysm under ultrasound guidance. The hyperechoic signal reflected by the needle ensures reliable placement of the needle tip inside the hypoechoic aneurysm for instillation of 5,000 IU thrombin dissolved in 3–5 ml saline solution. Injection produces immediate thrombosis and there are no publications reporting thrombus dislocation into a peripheral vessel

induced by this procedure. Nevertheless, some investigators recommend to first inject thrombin near the wall before proceeding to the neck. Thrombin injection has the advantage of rapidly inducing thrombosis while compression therapy is less expensive and has the added advantage of reducing the aneurysm volume, leaving a smaller hematoma that produces less swelling and pressure. (W. Schaberle 2005).

2.3.1.1-Causes Most causes are unknown. One identified risk to developing an aneurysm is **atherosclerosis,** which is a hardening of the arteries usually associated with a diet high in cholesterol. Smoking and obesity also increase the risk of atherosclerosis. Congenital conditions may cause an aneurysm—some individuals are born with weak aortic walls. A traumatic injury to the chest may also be a risk factor. The risk of developing an aneurysm can be reduced by not smoking, by losing excess weight, and by having a diet low in cholesterol. Periodic screening is an option for patients with a family history of aortic aneurysms. (Kathryn A. Booth & Terri D. Wyman 2008**)**

2.3.1.2-Signs and symptoms there are usually no signs and symptoms of an aneurysm, although hypertension can be a sign. When symptoms do exist, a pulsation in the abdomen and back pain are the most commonly seen. A sudden pain in the abdomen or back, dizziness, a fast pulse, or a loss of consciousness are signs that an aneurysm has burst.

2.3.1.3-Treatment The primary treatment is surgery to repair the aneurysm. (Kathryn A. Booth & Terri D. Wyman 2008**)**

2.3.2-Thrombophlebitis

Is a condition in which a blood clot and inflammation develop in a vein. It most commonly occurs in the veins of the legs. The danger of this disorder is that the blood clot may break loose. Once it reaches the heart, it is pumped to the lungs and is likely to block a blood vessel, causing a pulmonary embolism (an obstruction in the lungs). If the blood clot reaches the aorta and is pumped into arterial circulation, it can block either a coronary artery, causing a heart attack, or an artery in the brain, causing a stroke. (Booth &Wyman 2008**)**

2.3.2.1-Causes The causes and risk factors include prolonged inactivity, oral contraceptives, hormone replacement therapy for estrogen, certain types of cancer, paralysis in the arms or legs, the presence of a catheter in

a vein, a family history of this condition, varicose veins, and trauma to veins. (Booth &Wyman 2008**)**

2.3.2.2-Signs and symptoms the most common symptoms are tenderness and pain in the affected area, redness, swelling, and fever.

2.3.2.3-Treatment This disorder is most often treated by the application of heat to the affected area, elevation of the legs, anti-inflammatory drugs, blood-thinning medications, the wearing of support stockings, and the removal of varicose veins. Surgery to remove the clot may be needed in some cases. (Booth &Wyman 2008**)**

2.3.3-Varicose veins

Are dilated veins that are usually seen in the legs. They affect women more often than men.

2.3.3.1-Causes Varicose veins may be caused by prolonged sitting or standing, damage to valves in the veins, a loss of elasticity in the veins, obesity, pregnancy, oral contraceptives, or hormone replacement therapy. Varicose veins may be prevented through exercise and elevation of the legs. (Booth &Wyman 2008**)**

2.3.3.2-Signs and symptoms Signs and symptoms include discomfort in the legs, discolorations around the ankles, clusters of veins, and enlarged, dark veins that are seen through skin. (Booth &Wyman 2008**)**

2.3.3.3-Treatment The treatment of varicose veins includes the following:

• Sclerotherapy, which is a procedure that prevents blood from flowing through varicose veins. Laser surgery to prevent blood from flowing through affected veins.

• Vein stripping, which involves removing affected veins.

• Insertion of a catheter in the affected veins in order to destroy them.

• Endoscopic vein surgery to close off affected veins.

(Kathryn A. Booth & Terri D. Wyman 2008**)**

2.3.4-Fractures

Fractures are the most common skeletal abnormality seen in a general radiology practice. A fracture is defined as a disruption of bone caused by mechanical forces applied either directly to the bone or transmitted along the shaft of a bone. Although often obvious, some fractures are subtle and

difficult to detect. A fracture typically appears as a radiolucent line crossing the bone and disrupting the cortical margins. However, the fracture line may be thin and easily overlooked, whereas overlap of fragments may produce a radiopaque line. Secondary signs of an underlying fracture include joint effusion, soft tissue swelling, and interruption of the normal pattern of bony trabeculae. (Ronald L &Nancy 2012)

2.3.4.1-Types of Fractures

Fractures are described and classified by their extent, direction, and position; the number of fracture lines; and the integrity of the overlying skin. A fracture that results in discontinuity between two or more fragments is a **complete fracture**; an **incomplete fracture** causes only partial discontinuity, with a portion of the cortex remaining intact. In **closed fractures** the overlying skin is intact; if the overlying skin is disrupted, the fracture is open, or **compound**. Although it is a clinical distinction, the radiographic demonstration of bone clearly protruding through the skin and the presence of air in soft tissues about the fracture site on radiographs obtained immediately after the injury are highly suggestive of an **open fracture.** The direction of a fracture is determined by its relationship to the long axis of long and short bones and to the longest axis of irregular bones (e.g., the talus or carpal navicular). A **transverse fracture** runs at a right angle to the long axis of a bone and most commonly results from a direct blow or is a fracture within pathologic bone. An **oblique fracture** runs a course of approximately 45 degrees to the long axis of the bone and is caused by angulation or by both angulation and compression forces. A **spiral fracture** encircles the shaft, is generally longer than an oblique fracture, and is caused by torsional forces. **Avulsion fractures** are generally small fragments torn from bony prominences; they are usually the result of indirectly applied tension forces within attached ligaments and tendons rather than direct blows. A **comminuted fracture** is composed of more than two fragments. A **butterfly fragment** is an elongated triangularfragment of cortical bone generally detached from two other larger fragments of bone. A **segmental fracture** consists of a segment of the shaft isolated by proximal and distal lines of fracture. A **compression fracture** results from a compression force that causes compaction of bone trabeculae and results in decreased length or width of a portion of a bone. Compression

fractures most commonly occur in the vertebral body as a result of flexion of the spine; they may also be seen as impacted fractures of the humeral or femoral heads. A **depressed fracture** occurs in the skull or tibial plateau. In the skull, a small object with great force can produce a comminuted fracture, with portions of the fracture fragments driven inward. In the knee, the relatively hard lateral femoral condyle may impact on the relatively soft lateral tibial plateau with sufficient force to push the cortical surface of the tibia into the underlying cancellous bone. A **stress, or fatigue, fracture** is the response of bone to repeated stresses, none of which is sufficient to cause a fracture. The earliest pathologic process in a stress fracture is osteoclastic resorption, followed by the development of periosteal callus in an attempt to repair and strengthen the bone. A **pathologic fracture** occurs in bone at an area of weakness caused by a process such as tumor, infection, or metabolic bone disease. A **greenstick fracture** is an incomplete fracture with the opposite cortex intact. Greenstick fractures are found almost exclusively in infants and children because of the softness of their cancellous bone. A **torus (buckle) fracture** is one in which one cortex is intact with buckling or compaction of the opposite cortex. A **bowing fracture** is a plastic deformation caused by a stress that is too great to permit a complete recovery of normal shape but is less than the stress required to produce a fracture. An **undisplaced fracture** occurs when a plane of cleavage exists in the bone without angulation or separation. **Displacement** refers to separation of bone fragments; the direction of displacement describes the relationship of the distal fragment with respect to the proximal fragment and is usually measured in terms of the thickness of the shaft. **Angulation** indicates an angular deformity between the axes of the major fragments and also describes the position of the distal fragment with respect to the proximal one. **Dislocation** refers to the displacement of a bone that is no longer in contact with its normal articulation. If there is only partial loss of continuity of the joint surfaces, the displacement is called a **subluxation**. (Ronald L &Nancy 2012)

2.3.4.2-Treatment The overall goal of fracture treatment is to restore function and stability with an acceptable cosmetic result and a minimum of residual deformity. In **external, or closed, reduction,** the fracture is treated by manipulation of the affected body part without surgical incision. (Ronald L &Nancy 2012)

Open reduction is a surgical procedure using direct or indirect manipulation of the fracture fragments and usually involving the application or insertion of some type of appliance or device to achieve and maintain the reduction. **External fixation** is accomplished with the use of splints, external reduction devices, or casts; **internal fixation** uses metal plates and screws, wires, rods, and nails, either alone or in combination, to maintain the reduction. Most reduced fractures are immobilized or protected by an overlying cast. Fiberglass casting material causes less attenuation than plaster and thus produces less artifact. The radiopaque cast causes some obscuration of fine bony detail and, in severely osteoporotic bone, may make it difficult to visualize the fracture site. Therefore, if there is a question of healing that requires the demonstration of early callus formation or if there is a possibility of osteomyelitis, it is essential that the cast be removed by the physician before radiographs are obtained so that there is sufficient visibility of bone detail to resolve the questions. (Ronald L &Nancy 2012)

2.3.4.3-Fracture Healing

The radiographic evidence of fracture healing is a continuous external bridge of callus (calcium deposition) that extends across the line of fracture and unites the fracture fragments. The callus uniformly ossifies and approaches the density of normal bone. It is essential that at least two views be taken (preferably 90 degrees to each other) to ensure that there is callus about the fracture line in all directions. Proper exposure of the radiograph is required because underexposed images may produce the illusion of obliteration of the fracture line by bony trabeculae, whereas a properly exposed image would demonstrate the continued presence of the fracture line and a lack of healing. If the findings are equivocal, either CT or conventional tomography may be required to determine the degree of union. “Stress” images, a series of radiographs obtained with the injured part in the neutral position and during the application of stress by a physician or designated assistant on the distal fragment or part in the plane of suspected motion, may demonstrate a change in the alignment of the fragment, which indicates a lack of union. Malunion is the healing of fracture fragments in a faulty position. It leads to impairment of normal function or a cosmetic appearance that may require surgical correction. *Delayed union* is an ill-defined term arbitrarily applied to any fracture that takes longer to heal than the average fracture at that anatomic

location. Delayed union may result from infection, from inadequate immobilization, from limited blood supply, or from loss of bone at the fracture site. *Nonunion* refers to a condition in which the fracture healing process has completely stopped and the fragments remain ununited even with prolonged immobilization. Radiographically, nonunion characteristically appears as smooth, well-defined sclerosis about the fracture margins with occlusion of the medullary canal by sclerotic bone. A persistent defect, consisting of fibrous tissue and cartilage, appears between the fragments (Ronald L &Nancy 2012)

2.3.5-DVT

Over a century ago, Rudolf Virchow described three factors that are critically important in the development of venous thrombosis: (1) venous stasis, (2) activation of blood coagulation, and (3) vein damage. These factors have come to be known as the Virchow triad.

Venous stasis can occur as a result of anything that slows or obstructs the flow of venous blood. This results in an increase in viscosity and the formation of micro-thrombi, which are not washed away by fluid movement; the thrombus that forms may then grow and propagate. Endothelial (intimal) damage in the blood vessel may be intrinsic or secondary to external trauma. It may result from accidental injury or surgical insult. A hypercoagulable state can occur due to a biochemical imbalance between circulating factors. This may result from an increase in circulating tissue activation factor, combined with a decrease in

circulating plasma anti-thrombin and fibrinolysins.

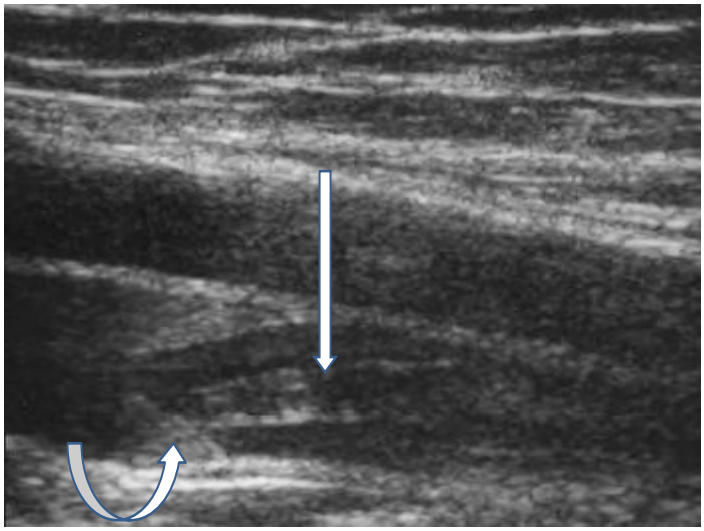


Figure (2-5) The proximal end of a free-floating thrombus (arrow) is seen in the superficial femoral vein. The thrombus is relatively anechoic and the thrombus tip is touching a valve cusp (curved arrow). (How where and when and where Thrush & Hartshorne 2005 page No196)

Over time, refinements have been made in the description of these factors and their relative importance to the development of venous thrombosis. The origin of venous thrombosis is frequently multi-factorial, with components of the Virchow triad assuming variable importance in individual patients, but the end result is early thrombus interaction with the endothelium. This interaction stimulates local cytokine production and facilitates leukocyte adhesion to the endothelium, both of which promote venous thrombosis. Depending on the relative balance between activated coagulation and thrombolysis, thrombus propagation occurs.

Decreased vein wall contractility and vein valve dysfunction contribute to the development of chronic venous insufficiency. The rise in ambulatory venous pressure causes a variety of clinical symptoms of varicose veins, lower extremity edema, and venous ulceration. (Bryan A Mitton 2015)

2.3.5.1-Development of thrombosis

Thrombosis is the homeostatic mechanism whereby blood coagulates or clots, a process crucial to the establishment of hemostasis after a wound.

It may be initiated via several pathways, usually consisting of cascading activation of enzymes that magnify the effect of an initial trigger event. A similar complex of events results in fibrinolysis, or the dissolution of thrombi. The balance of trigger factors and enzymes is complex. Microscopic thrombus formation and thrombolysis (dissolution) are continuous events, but with increased stasis, procoagulant factors, or endothelial injury, the coagulation-fibrinolysis balance may favor the pathologic formation of an obstructive thrombus. Clinically relevant deep venous thrombosis is the persistent formation of macroscopic thrombus in the deep proximal veins. For the most part, the coagulation mechanism consists of a series of self-regulating steps that result in the production of a fibrin clot. These steps are controlled by a number of relatively inactive cofactors or zymogens, which, when activated, promote or accelerate the clotting process. These reactions usually occur at the phospholipids surface of platelets, endothelial cells, or macrophages. Generally, the initiation of the coagulation process can be divided into two distinct pathways, an intrinsic system and an extrinsic system. The extrinsic system operates as the result of activation by tissue lipoprotein, usually released as the result of some mechanical injury or trauma. The intrinsic system usually involves circulating plasma factors. Both of these pathways come together at the level of factor X, which is activated to form factor Xa. This in turn promotes the conversion of prothrombin to thrombin (factor II). This is the key step in clot formation, for active thrombin is necessary for the transformation of fibrinogen to a fibrin clot.

Once a fibrin clot is formed and has performed its function of hemostasis, mechanisms exist in the body to restore the normal blood flow by lysing the fibrin deposit. Circulating fibrinolysins perform this function. Plasmin digests fibrin and also inactivates clotting factors V and VIII and fibrinogen. Three naturally occurring anticoagulant mechanisms exist to prevent inadvertent activation of the clotting process. These include the heparin-antithrombin III (ATIII), protein C and thrombomodulin protein S, and the tissue factor inhibition pathways. When trauma occurs, or when surgery is performed, circulating ATIII is decreased. This has the effect of potentiating the coagulation process. Studies have demonstrated that levels of circulating ATIII is decreased more, and stay reduced longer, after total hip replacement (THR) than after general surgical cases. Furthermore, patients who have positive venograms

postoperatively tend to be those in whom circulating levels of ATIII are diminished. Under normal circumstances, a physiologic balance is present between factors that promote and retard coagulation. A disturbance in this equilibrium may result in the coagulation process occurring at an inopportune time or location or in an excessive manor. Alternatively, failure of the normal coagulation mechanisms may lead to hemorrhage. Thrombus usually forms behind valve cusps or at venous branch points, most of which begin in the calf. Venodilation may disrupt the endothelial cell barrier and expose the subendothelium. Platelets adhere to the subendothelial surface by means of von Willebrand factor or fibrinogen in the vessel wall. Neutrophils and platelets are activated, releasing procoagulant and inflammatory mediators. Neutrophils also adhere to the basement membrane and migrate into the subendothelium. Complexes form of the surface of platelets and increase the rate of thrombin generation and fibrin formation. Stimulated leukocytes irreversibly bind to endothelial receptors and extravasate into the vein wall by means of mural chemotaxis. Because mature thrombus composed of platelets, leukocytes and fibrin develops, and an active thrombotic and inflammatory process occurs at the inner surface of the vein, and an active inflammatory response occurs in the wall of the vein. Studies have shown that low flow sites, such as the soleal sinuses, behind venous valve pockets, and at venous confluences, are at most risk for the development of venous thrombiHowever, stasis alone is not enough to facilitate the development of venous thrombosis. Experimental ligation of rabbit jugular veins for periods of up to 60 minutes have failed to consistently cause venous thrombosis. Although, patients that are immobilized for long periods of time seem to be at high risk for the development of venous thrombosis, an additional stimulus is required to develop DVT. (Bryan A Mitton 2015)

2.3.5.2-Evolution of venous insufficiency

Over time, thrombus organization begins with the infiltration of inflammatory cells into the clot. This results in a fibroelastic intimal thickening at the site of thrombus attachment in most patients and a fibrous synechiae in up to 11%. In many patients, this interaction between vessel wall and thrombus leads to valvular dysfunction and overall vein wall fibrosis. Histological examination of vein wall remodeling after venous thrombosis has demonstrated an imbalance in connective tissue

matrix regulation and a loss of regulatory venous contractility that contributes to the development of chronic venous insufficiency. Some form of chronic venous insufficiency develops in 29-79% of patients with an acute DVT, while ulceration is noted in 4-6%.The risk has been reported to be 6 times greater in those patients with recurrent thrombosis. Over a few months, most acute DVTs evolve to complete or partial recanalization, and collaterals develop. Although blood flow may be restored, residual evidence of thrombus or stenosis is observed in half the patients after 1 year. Furthermore, the damage to the underlying valves and those compromised by peripheral dilation and insufficiency usually persists and may progress. Venous stasis, venous reflux, and chronic edema are common in patients who have had a large DVT. The acute effect of an occluded outflow vein may be minimal if adequate collateral pathways exist. As an alternative, it may produce marked pain and swelling if flow is forced retrograde. In the presence of deep vein outflow obstruction, contraction of the calf muscle produces dilation of the feeding perforating veins, it renders the valves nonfunctional (because the leaflets no longer coapt), and it forces the blood retrograde through the perforator branches and into the superficial system. This high-pressure flow may cause dilation of the superficial (usually low-pressure) system and produce superficial venous incompetence. In clinical terms, the increased incidence of reflux in the ipsilateral greater saphenous vein increases 8.7-fold on follow-up of DVT. This chain of events (ie, obstruction to antegrade flow producing dilation, stasis, further valve dysfunction, with upstream increased pressure, dilation, and other processes) may produce hemodynamic findings of venous insufficiency. Another mechanism that contributes to venous incompetence is the natural healing process of the thrombotic vein. The thrombotic mass is broken down over weeks to months by inflammatory reaction and fibrinolysis, and the valves and venous wall are altered by organization and ingrowth of smooth muscle cells and production of neointima. This process leaves damaged, incompetent, underlying valves, predisposing them to venous reflux. The mural inflammatory reaction breaks down collagen and elastin, leaving a noncompliant venous wall. Persistent obstructive thrombus, coupled with valvular damage, ensures continuation of this cycle. Over time, the venous damage may become irreversible. Hemodynamic venous insufficiency is the underlying

pathology of postthrombotic syndrome (PTS), also referred to as postphlebitic syndrome. If numerous valves are affected, flow does not occur centrally unless the leg is elevated. Inadequate expulsion of venous blood results in stasis and a persistently elevated venous pressure or venous hypertension. As fibrin extravasates and inflammation occurs, the superficial tissues become edematous and hyperpigmented. With progression, fibrosis compromises tissue oxygenation, and ulceration may result. After venous insufficiency occurs, no treatment is ideal; elevation and use of compression stockings may compensate, or surgical thrombectomy or venous bypass may be attempted. With anticoagulation alone, as many as 75% of patients with symptomatic DVT present with PTS at 5-10 years. However, the incidence of venous ulceration is far less, at 5%. Of the half million patients with venous ulcers in the United States, 17-45% report having a history of DVT. (Bryan A Mitton 2015)

2.3.5.3- Thrombi in Lower-Extremity

Most small thrombi in the lower extremities tend to resolve spontaneously after surgery. In about 15% of cases, however, these thrombi may extend into the proximal femoral venous system of the leg. Untreated proximal thrombi represent a significant source of clinically significant pulmonary emboli. In the absence of rhythmic contraction of the leg muscles, as in walking or moving, blood flow in the veins slows and even stops in some areas, predisposing patients to thrombosis. In the postoperative patient, as many as one half of all isolated calf vein thrombi resolve spontaneously within a few hours, whereas approximately 15% extend to involve the femoral vein. A many as one third of untreated symptomatic calf vein DVT extend to the proximal veins. At 1-month follow-up of untreated proximal DVT, 20% regress and 25% propagate. Although calf vein thrombi are rare sources of clinically significant PE, the incidence of PE with untreated proximal thrombi is 29-50%.Most PEs are first diagnosed at autopsy. (Bryan A Mitton 2015)

2.3.5.4-Pulmonary Embolism

PE develops as venous thrombi break off from their location of origin and travel through the right heart and into the pulmonary artery, causing a ventilation perfusion defect and cardiac strain. PE occurs in approximately 10% of patients with acute DVT and can cause up to 10% of in hospital deaths. However, most patients (up to 75%) are

asymptomatic. Traditionally, proximal venous thrombosis are thought to be at highest risk for causing pulmonary emboli; however, the single largest autopsy series ever performed to specifically to look for the source of fatal PE was performed by Havig in 1977, who found that one third of the fatal emboli arose directly from the calf veins. (Bryan A Mitton 2015)

2.3.5.5-Superior vena cava syndrome

Superior vena cava syndrome is caused by gradual compression of the superior vena cava (SVC). Patients can present with dyspnea, cough, dysphagia, and swelling of the neck and upper extremities. SVC syndrome is most commonly caused by extrinsic compression from a malignant process, such as lung or breast cancer. However, thrombotic causes of SVC syndrome are increasing due to the more widespread use of central venous catheters and pacemakers. SVC syndrome is a clinical diagnosis, but it can be confirmed with plain radiography, computed tomography (CT) scanning, and venography.[1] For cancer-related SVC syndrome, the treatment consists of chemotherapy and radiation directed at the obstructing tumor. For thrombotic causes, thrombolysis and anticoagulation may be used. Increasingly, endovascular treatment with balloon dilation and stenting are being used with rapid resolution of symptoms. (Bryan A Mitton 2015)

2.3.5.6-Problems and Pitfalls

Problems and Pitfalls in the Diagnosis of Deep Vein Thrombosis the value of ultrasound as a technique for the diagnosis of DVT depends on the operator performing a careful, complete examination, being aware of potential pitfalls and recognising when a less than adequate examination has been performed. The main problem areas which should be remembered are shown in table (1-2). Inadequate Visualisation. **(**Myron A. Pozniak & Paul L. Allan2014**)**

Table (1-2) problems and pitfalls in the diagnostic of DVT

• Swollen/oedematous/fat legs
• Dual thigh and popliteal veins
• Non-occlusive thrombus
• Segmental calf vein thrombus
• Segmental iliac vein thrombus
• Pregnant patients

The essential requirement for a satisfactory examination is good ultrasound access to the veins of the limb. Many patients with a possible diagnosis of DVT have swollen or oedematous legs; this situation is aggravated if the patients are also obese. If visualisation is poor then it is possible to miss significant thrombus unless the situation is recognized and appropriate care is taken with the examination and machine settings, as well as with the selection of an appropriate transducer Duplicated Venous Segments. Dual segments of femoral vein may be overlooked unless they are actively sought with transverse scanning. If they are not recognised, then one component may be patent and seen on colour Doppler, whereas the other component may contain thrombus and be overlooked. Non-occlusive Thrombus. Similarly, non-occlusive thrombus may be missed if the vein is not seen adequately. If there is only a small amount of thrombus in the vein then good flow signals will be obtained on spectral and colour Doppler and the presence of the thrombus may not be recognized. This is particularly important in obese or oedematous legs. Isolated Calf Vein Thrombus. The calf veins are multitudinous in number and variable in their anatomy. Even with a careful, patient, time-consuming examination it is difficult to exclude completely the presence of a small segmental thrombus in a calf vein or muscular sinus In a mobile patient with a little calf tenderness or swelling this is not a problem, as the body's normal thrombolytic mechanisms will probably clear this. However, in a patient who is immobile following surgery or a stroke, a small segmental calf thrombus indicates that the clotting cascade has been activated and there is a possibility that this small thrombus may increase in size, resulting in a significant, occlusive thrombus. Therefore a follow-up scan should be considered in these patients in order to identify any progression of thrombus from the calf. A study by Labropoulos et al. reviewed 5250 patients; isolated calf vein thrombus was found in 4.8% (282 limbs in 251 patients). In these patients, variable patterns of involvement of the calf veins were demonstrated with the soleal veins involved in 20% of cases, gastrocnemius veins in 17%, peroneal veins in 15% and the posterior tibial veins in 12%; in 64% of these positive cases, only a single vein group was involved. A review by Scarvelis et al, discussing the management of patients with deep vein thrombosis, comments that only 1–2% of patients who have a negative initial ultrasound will be confirmed to have a proximal DVTupon serial

testing, so serial examinations are not cost-effective. However, whilst re-scanning should not be a routine expectation, it should be considered in cases with a high clinical probability or clinical concern and an initial negative scan. Asymptomatic Thrombus. The accuracy of Doppler in the detection of asymptomatic thrombus is less impressive than that for symptomatic thrombus, and the technique is therefore generally inadequate as a screening tool for the detection of asymptomatic thrombus. This is probably because asymptomatic thrombi are more likely to be small and non-occlusive; in addition, there is a higher incidence of distal thrombi in the calf veins, which may be more difficult to demonstrate with ultrasound. **(**Myron A. Pozniak & Paul L. Allan2014**)**

2.3.5.7-Other causes of leg swelling, pain or tenderness

Unlike venography, ultrasound allows examination of other structures in the pelvis and leg. Other pathologies may be seen which account for the patient's symptoms of a swollen, or painful, tender leg; these are given in table (2-2). The deep veins must still be examined carefully, as a coexistent DVT may otherwise be overlooked. Labropoulos et al. demonstrated popliteal cysts in 3% of asymptomatic individuals, rising to 10% of patients with symptoms of possible DVT and 20% of patients with painful knees. Langsfeld et al. found popliteal cysts in 3% of patients being examined for possible DVT, 7% of those with cysts had a coexisting DVT. **(**Myron A. Pozniak & Paul L. Allan2014**)**

Table(2-2) Other causes of leg swelling, pain or tenderness

Popliteal (Baker's) cysts
• Haematoma/muscle injury
• Superficial thrombophlebitis
• Iliac nodes/pelvic masses
• Arteriovenous fistula
• Lymphoedema

Accuracy in Relation to Other Techniques Despite these potential problems, ultrasound is a good non-invasive method for the diagnosis of symptomatic DVT, especially between the lower popliteal region and the groin. The key to its value in any given department is that the sonographers must not only be well trained in the technique, but must also be able to recognise an inadequate examination so that appropriate

further measures, such as venography or a repeat scan, can be arranged. Should venography be required to clarify areas of doubt, this can be focused on the area of concern identified at the ultrasound examination and only a limited examination may be required **(**Myron A. Pozniak & Paul L. Allan2014**)**

2.3.5.8-DVT Duplex Examination

During deep venous thrombosis (DVT) examination, the following should be checked**:**

1. Whether thrombosis present?
2. If yes, the extent and age of thrombosis
3. Whether the thrombosis is adherent to the vessel vessel wall and
4. The cause of thrombosis

2.3.5.8.1-Specific Problems and Solutions

Poor visualization of the femoral vein in the adductor canal

Support the thigh from behind with the left hand during the examination. Otherwise, try a posterior apporach for imaging the distal portions of the adductor canal.

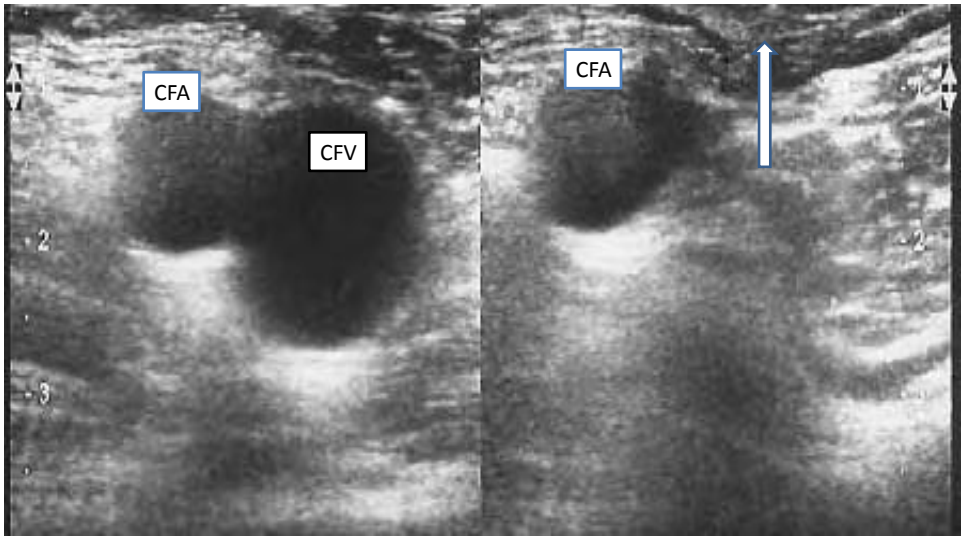


Figure (2-7) A: A transverse image of the right common femoral vein (CFV) and femoral artery (CFA). B: Patency of the CFV is demonstrated by complete collapse of the vein (arrow) during transducer pressure (How where and when and where Thrush & Hartshorne 2005 page No193)

The leg appears too swollen for ultrasound examination.

First consider alternative modalities. If this is not possible, locate the femoral vein in the groin and also locate the popliteal vein. Both sites can always be evaluated with ultrasound. The findings, though minimal, are useful for selecting therapeutic options when thrombosis is present.

Thrombosis is present, but the pelvic vessels are difficult to evaluate.

The external iliac vein can almost always be evaluated in its distal portion, but often the proximal end of the thrombus cannot be seen. There is usually no difficulty in compressing the vena cava. However, this finding is usually adequate if conservative treatment is desired, since ultrasound has demonstrated involvement at the pelvic level while excluding vena cava thrombosis.

Figure (2-8) The Doppler waveform in the femoral vein distal to an iliac vein occlusion often demonstrates continuous low-velocity flow with a loss of phasicity. (How where and when and where Thrush & Hartshorne 2005 page No197)

Thrombosis is absent, but the pelvic vessels are difficult to evaluate

If there is no reason to suspect that isolated pelvic vein thrombosis is present (e.g., pelvic mass, malignant lymphoma), thrombosis at that level can be indirectly excluded by a normal response of the common femoral vein to a Valsalva maneuver.

Significant atherosclerosis of the accompanying arteries creates acoustic shadows that obscure the veins

Try changing the transducer position to scan past the artery and interrogate the vein directly.

The veins in the lower leg cannot be positively identified

In a patient with a very thick calf, adjust the transducer position to minimize the distance from the transducer face to the veins of interest. If they still cannot be adequately visualized, try flexing the leg over the edge of the table.

2.3.5.8.2- Findings and pitfalls

Echogenic lumen (suspected thrombosis)

Intravascular echoes can be caused by overamplification of the Bmode image (B-mode gain too high) or unfavorable acoustic conditions.

Echo-free lumen (no sign of thrombosis)

Fresh thrombi may appear sonolucent

No detectable flow signal in the vessel lumen (suspected thrombosis)

Very slow flow may be below the threshold of detection even with optimum instrument settings. Often, a color signal cannot be obtained just

proximal or distal to thrombosis, in the lower leg, or in a standing examination. Shadowing from calcified plaque in the accompanying artery can prevent color flow imaging.

Detectable flow signal in the vessel lumen (no sign of thrombosis)

Thrombosis that is incomplete or partially recanalized may produce a detectable flow signal, so make certain that color fills the lumen before excluding thrombosis. Occasionally, this is difficult to achieve even in healthy subjects, which is why distal compression is often used. This technique may cause a partial thrombosis to become swamped with echoes. (Jaypee 2009)

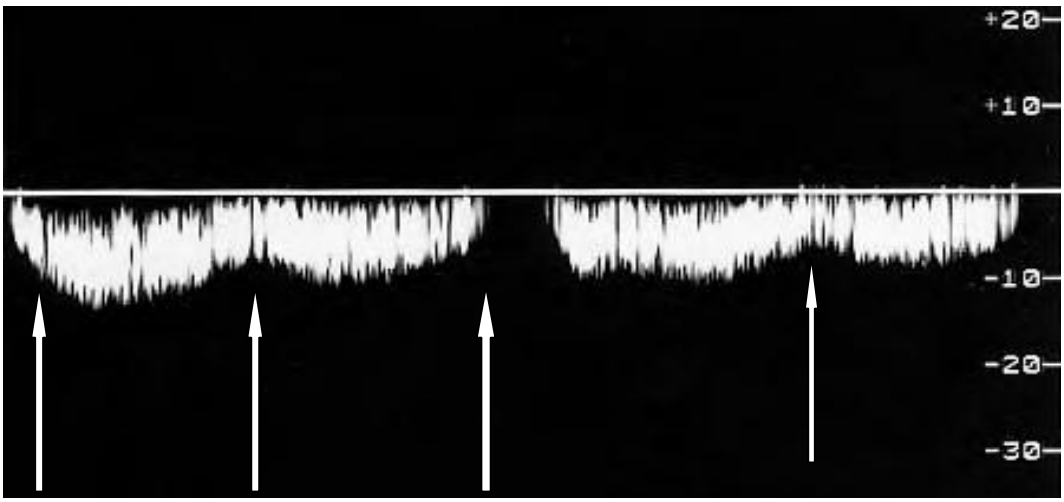


Figure (2-9) Doppler waveform demonstrating the effect of respiration on the blood flow in the common femoral vein. The large arrow indicates the cessation of flow during inspiration and the small arrows show small changes in flow due to the cardiac cycle, which may not always be seen in the common femoral vein. (How where and when and where Thrush & Hartshorne 2005 page No61

2.4- The Previous Studies

Seung Yeol Lee and others evaluated the nationwide incidence and risk factors for symptomatic deep vein thrombosis (DVT) after major lower limb orthopedic surgeries, they investigated the incidence of DVT from 2007 to 2011 using a nationwide database maintained by the Korean Health Insurance Review and Assessment Service (HIRA). The HIRA is a non-profit agency that is responsible for reviewing medical fees. They found that the age- and gender-adjusted annual incidence of DVT was 70.67 per 100000 persons/year. Compared to patients aged <49 years, the relative risk of DVT was five times higher in patients aged 50–69 and 10 times higher in patients aged >70 years (p<0.001). Females showed a greater relative risk for DVT than males (1.08; p<0.001). The incidence

of postoperative DVT, according to the type of surgery, was significantly greater for knee replacement arthroplasty than for other forms of surgery ($p<0.002$). The relative risk of postoperative DVT was higher in females in knee replacement arthroplasty (1.47) and hip fracture surgery (2.25) groups, although relatively lower in those who underwent hip replacement arthroplasty (0.97). Unfortunately, their data did not include information regarding the diagnostic method. The study conclude that among major lower limb surgeries, advanced age, female gender, and undergoing a knee replacement arthroplasty were found to be risk factors for developing postoperative DVT. These findings further emphasize the need for orthopedic surgeons to consider the development of DVT after surgery in high-risk patients.

Smith Shaha et al were using Doppler ultrasonography to determine the prevalence of deep vein thrombosis with lower limb trauma patients, their study was conducted on 125 patients having lower limb fractures (excluding foot fractures and grade III compound fractures) in a tertiary care centre in a rural area (Dhiraj General Hospital Piparia India) from September 2011 to June 2013. The patients were thoroughly evaluated based on history, fracture pattern, associated injuries and co morbid conditions along with colour doppler pre operatively. In their series of 125 patients, 107 were male and 18 female (M: F=5.9:1). Out of them 6 patients were DVT positive (4.8%). Amongst them 3(2.4%) had proximal DVT and 3(2.4%) had distal DVT. Only 1 had pulmonary embolism. Out of 47 patients with periacetabular fractures, 4 (8.51%) developed DVT and Out of 8 patients with floating knee injury, 2 (25%) developed DVT. in their study they found that the combination of risk factors rather than a single risk factor had played important role for development of DVT. They concluded that the prevalence of DVT is low in Indian population thereby avoiding the need for chemoprophylaxis in all patients. However based on fracture geometry and co morbid conditions chemoprophylaxis may be justified in few individuals

Several Studies done by Lasse Lapidus 2007 its aims was to assess the incidence of symptomatic DVT and PE during first six week after orthopedic surgery (paper I), to assess the extent of DVT after orthopedic surgery (paper I) and to evaluate the sensitivity and specificity of CDS with those of phlebography in patient surgically treated for an ankle

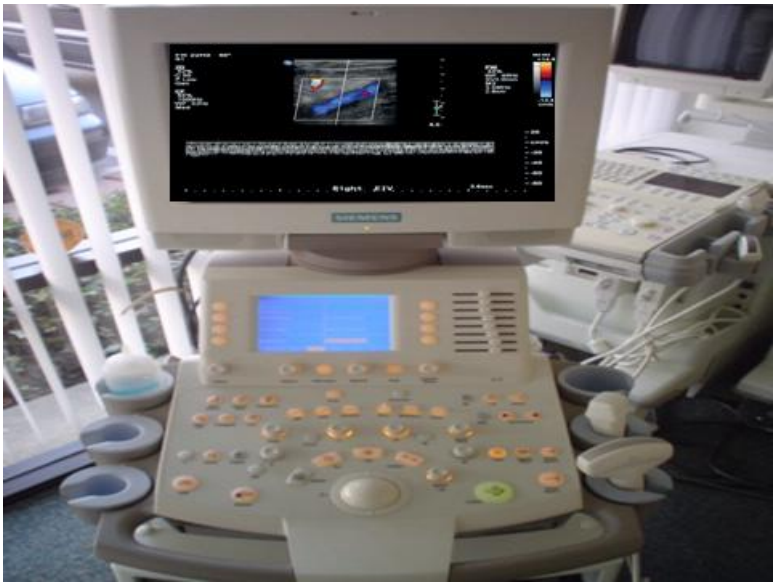
fracture (paper II). In an observational study of 30816 consecutive patients 13397(43.5%) men and 17419 women (56.5%) (*Paper I*) undergoing orthopedic surgery between 1 March 1996 and 31 December 2003 the mortality and incidence of DVT and PE was recorded prospectively during a six week follow up. After major joint surgery of lower extremity, after spinal surgery and after lower limb fracture surgery thromboprophylaxis (LMWHs) was administered during 7 to 10 days. The overall DVT and PE incidence was 1.0% and 0.3% respectively. The highest incidence of VTE with the LMWH prophylaxis was seen after pelvic fracture surgery (13.0%). The sensitivity and specificity color duplex sonography with that of phlebography in a prospective trial (paper II) with 180(83 male&97 female) consecutive patients surgically treated for ankle fracture. With a sensitivity of 96% and a negative predictive value of 99%, the result showed that CDS is highly reliable for ruling out DVT for screening purposes.

CHAPTER THREE

3-Material and Methods

3.1-Study population

This study was carried out at Rabat Universal Hospital in Khartoum Sudan from January 2016 and May 2018, a total of 176 patients (129 males and 47females), aged ranged between 18 and 86 years (mean age of 51.5±19.5 years) who presented with fractured lower extremities were enrolled in this prospective study. Patients were thoroughly evaluated before surgery and some undergone orthopedic surgery with respect to medical history, fracture pattern, associated injuries, comorbid conditions, and VDUS findings. Informed consent was obtained from all patients, and the study was reviewed and approved by an institutional review board.



Figure(3-1) Sonoline G 60S US imaging system; Siemens

3.2-Ultrasound examination protocol

Immediately upon patient admission to the ultrasound (US) department, the affected limb was assessed for DVT by VDUS using a Sonoline G 60S US imaging system; Siemens – Germany, equipped with a high frequency (7–10 MHz) linear probe. Gray-scale images and color and

spectral Doppler waveforms were recorded without and with compression and from the long axis, respectively using UP-D898MD digital, black and-white A6 printer; Sony and UPP-110S high density standard US thermal paper roll (110 mm×20 m). Distal augmentation maneuvers were performed with manual calf compression while investigating the common femoral vein (CFV), superficial femoral vein (SFV), and popliteal vein (POPV) segments. Foot compression was used to evaluate the posterior tibial veins (PTVs). US examination was considered to be negative if there was a normal blood flow in CFV, SFV, POPV, and PTVs, with the vessel lumen fully compressible and completely filled with color. DVT was diagnosed if the vessel wall was not compressed. Because color Doppler ultrasound (CDUS) is operator dependent, all scans were performed by an experienced musculoskeletal US sonologist US scanning was conducted with the patient in the supine position with the head raised from 15 to 30 degrees and the examination table tilted by 5 to 10 degrees (reverse Trendelenburg tilt). The investigated leg was outwardly rotated at the hip with the knee slightly. In the short axis, starting at CFV and advancing into the distal external iliac vein (EIV), the transducer was moved moderately inferiorly to completely scan CFV and SFV throughout the thigh and to scan POPV from a posterior approach throughout the popliteal fossa. From a posteromedial access in the lower extremity, PTVs were evaluated. Probe compression was applied at 1 to 2 cm intervals for all vein sections, with each section evaluated for complete compressibility and for the presence of any intraluminal echoes suggestive of thrombus. Longitudinal inspections were applied to assure the presence of intraluminal echoes seen on short axis imaging and to obtain Doppler spectral waveforms of venous hemodynamics in CFV, SFV, POPV, and PTVs. These Doppler waveforms were studied for the presence of spontaneous flow, respiratory phasicity, and augmented flow in response to manual distal limb compression [12]. The patients who were diagnosed with DVT of a CFV, SVF, and/-or POPV received warfarin and dalteparin for three months. The international normalized ratio (INR) was used to monitor warfarin treatment. Once the INR was greater than 2, dalteparin was stopped and warfarin continued.

Chapter Four

4.1-Results

This chapter represents the results in tables and graphs as the following:

Table(4-1) Gender

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Male	129	73.3	73.3	73.3
	Female	47	26.7	26.7	100.0
	Total	176	100.0	100.0	

Table (4-2) Operation status

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Preoperation	126	71.6	71.6	71.6
	Postoperation	50	28.4	28.4	100.0
	Total	176	100.0	100.0	

Table (4-3) Anticoagulant drug

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Used	147	83.5	83.5	83.5
	Not Used	29	16.5	16.5	100.0
	Total	176	100.0	100.0	

Table (4-4) DM & HTN

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	None	132	75.0	75.0	75.0
	DM	17	9.7	9.7	84.7
	HTN	14	8.0	8.0	92.6
	Both	13	7.4	7.4	100.0
	Total	176	100.0	100.0	

Table (4-5) Patient habits

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	None	104	59.1	59.1	59.1
	snuff	26	14.8	14.8	73.9
	cigarate	24	13.6	13.6	87.5
	tabbacco	5	2.8	2.8	90.3
	complex	17	9.7	9.7	100.0
	Total	176	100.0	100.0	

Table (4-6) Coffee Drinker

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	yes	74	42.0	42.0	42.0
	No	102	58.0	58.0	100.0
	Total	176	100.0	100.0	

Table (4-7) Others Sonographic Findings

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	None	108	61.4	61.7	61.7
	Lymph Nodes	62	35.2	35.4	97.1
	Hematoma	3	1.7	1.7	98.9
	Baker's cyst	1	.6	.6	99.4
	Others	1	.6	.6	100.0
	Total	175	99.4	100.0	
Missing	System	1	.6		
Total		176	100.0		

Table (4-8) Regionalist

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Not available	32	18.2	18.2	18.2
	Khartoum	63	35.8	35.8	54.0
	Middle States	27	15.3	15.3	69.3
	East States	11	6.3	6.3	75.6
	West States	32	18.2	18.2	93.8
	North States	11	6.3	6.3	100.0
	Total	176	100.0	100.0	

Table (4-9) Gender * DVT Crosstabulation

			DVT		
			No DVT	DVT	Total
Gender	Male	Count	112	17	129
		% within Gender	86.8%	13.2%	100.0%
	Female	Count	42	5	47
		% within Gender	89.4%	10.6%	100.0%
Total		Count	154	22	176
		% within Gender	87.5%	12.5%	100.0%

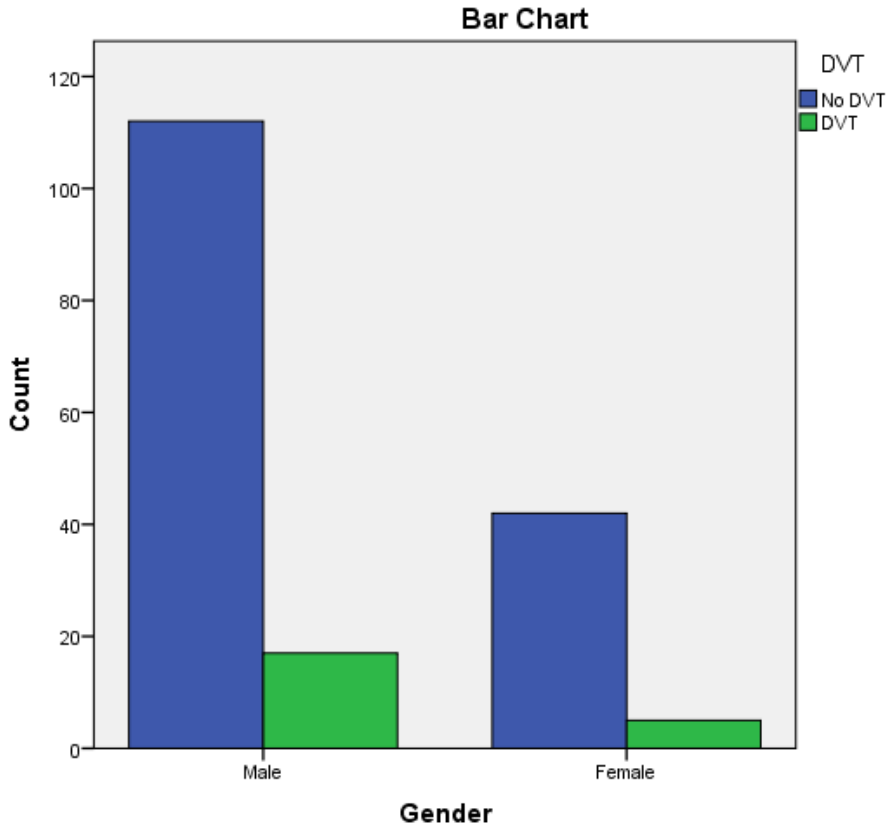


Figure (4-1) Gender * DVT Crosstabulation

Table (4-10) Age group * DVT Crosstabulation

			DVT		
			No DVT	DVT	Total
Age group	(18-37)year	Count	61	6	67
		% within Age group	91.0%	9.0%	100.0%
	(38-57)year	Count	30	4	34
		% within Age group	88.2%	11.8%	100.0%
	(58-77)year	Count	51	10	61
		% within Age group	83.6%	16.4%	100.0%
	(78-97)	Count	12	2	14
		% within Age group	85.7%	14.3%	100.0%
Total		Count	154	22	176
		% within Age group	87.5%	12.5%	100.0%

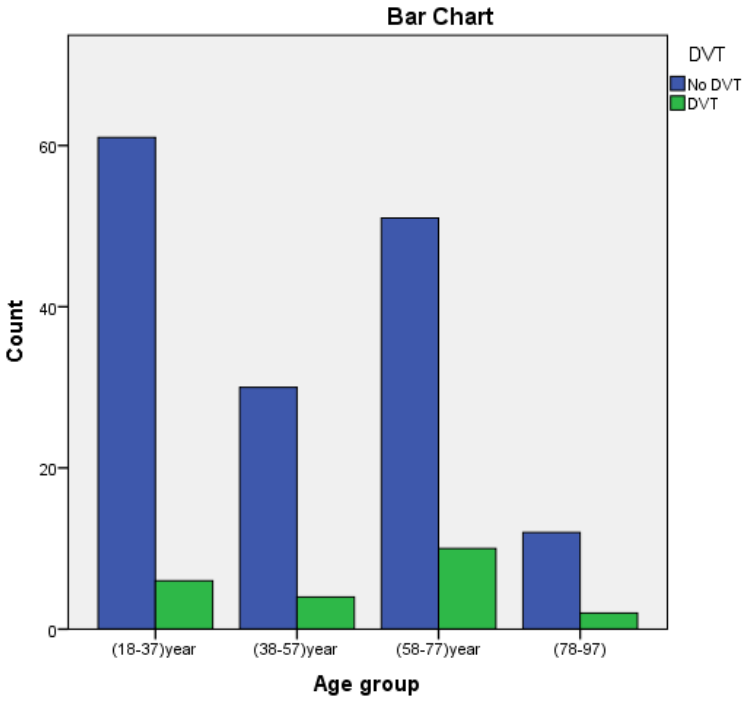


Figure (4-2) Age group * DVT Crosstabulation

Table (4-11) Blood group * DVT Crosstabulation

			DVT		
			No DVT	DVT	Total
Blood group	not avilable	Count	59	14	73
		% within Blood group	80.8%	19.2%	100.0%
	A+ve	Count	27	2	29
		% within Blood group	93.1%	6.9%	100.0%
	A-ve	Count	2	0	2
		% within Blood group	100.0%	0.0%	100.0%
	B+ve	Count	19	1	20
		% within Blood group	95.0%	5.0%	100.0%
	O+ve	Count	38	4	42
		% within Blood group	90.5%	9.5%	100.0%
	O-ve	Count	6	0	6
		% within Blood group	100.0%	0.0%	100.0%
	AB	Count	3	1	4
		% within Blood group	75.0%	25.0%	100.0%
Total		Count	154	22	176
		% within Blood group	87.5%	12.5%	100.0%

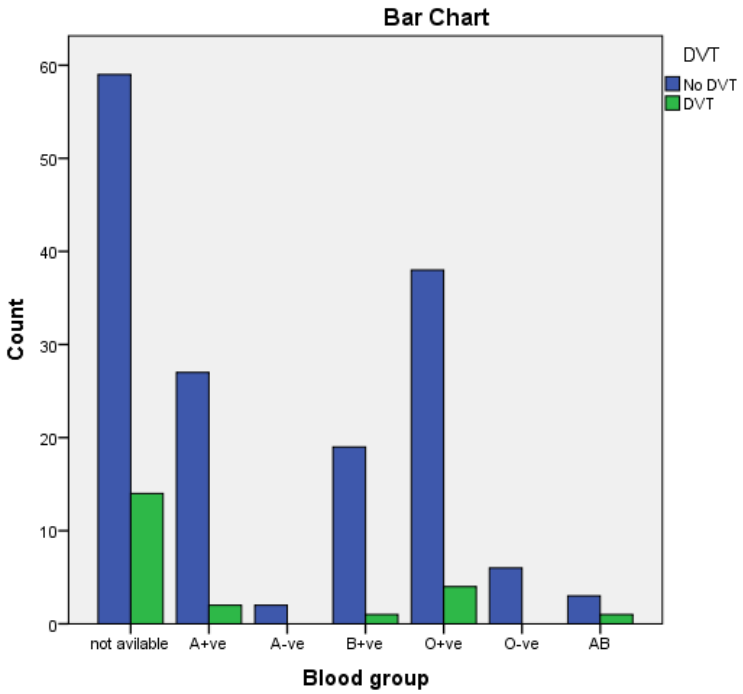


Figure (4-3) Blood group * DVT Crosstabulation

Table (4-12) b**ody mass index * DVT Crosstabulation**

			DVT		
			No DVT	DVT	Total
Body mass index	BMI<25	Count	95	12	107
		% within Body mass index	88.8%	11.2%	100.0%
	BMI>25	Count	58	10	68
		% within Body mass index	85.3%	14.7%	100.0%
Total		Count	153	22	175
		% within Body mass index	87.4%	12.6%	100.0%

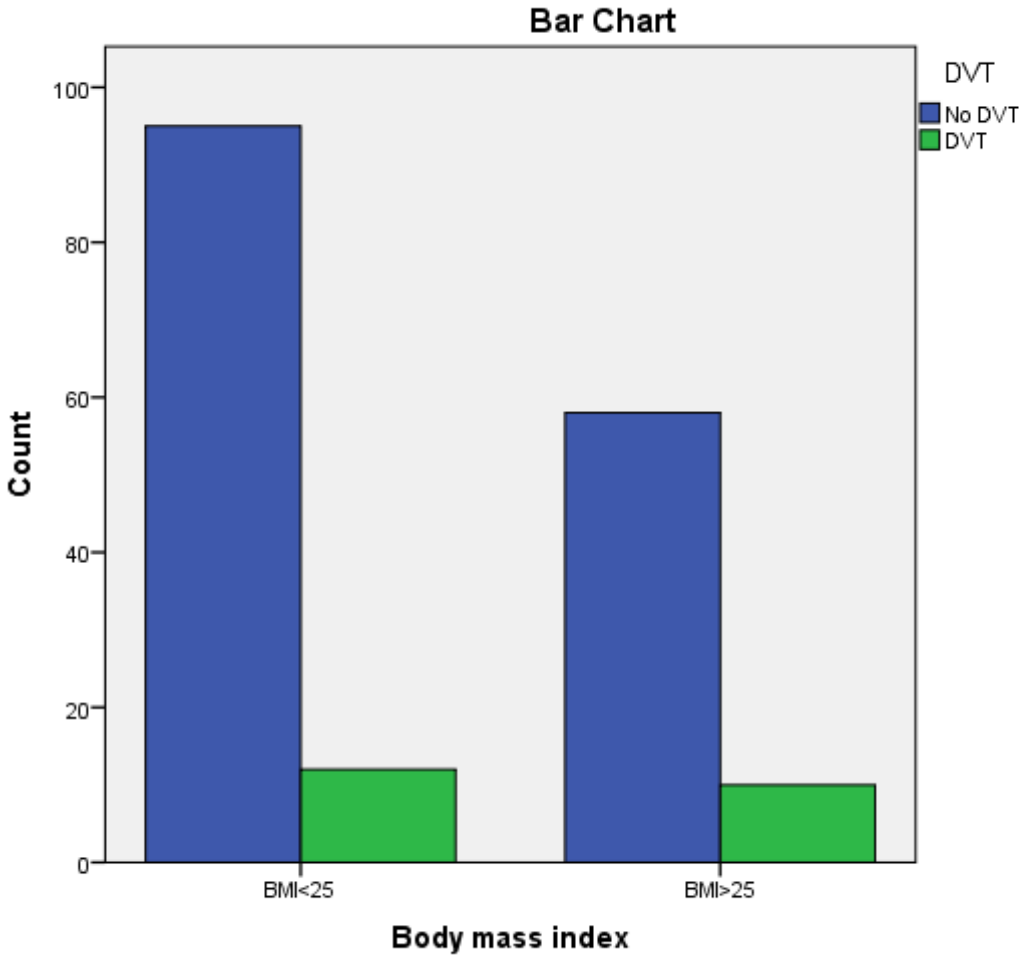


Figure (4-4) b**ody mass index * DVT Crosstabulation**

Table (4-13) Fracture site * DVT Crosstabulation

			DVT		Total
			No DVT	DVT	
Fracture site	periacetabuler	Count	63	10	73
		% within Fracture site	86.3%	13.7%	100.0%
	femur	Count	20	5	25
		% within Fracture site	80.0%	20.0%	100.0%
	knee	Count	14	0	14
		% within Fracture site	100.0%	0.0%	100.0%
	leg&foot	Count	47	4	51
		% within Fracture site	92.2%	7.8%	100.0%
	multiple fractures	Count	10	3	13
		% within Fracture site	76.9%	23.1%	100.0%
Total		Count	154	22	176
		% within Fracture site	87.5%	12.5%	100.0%

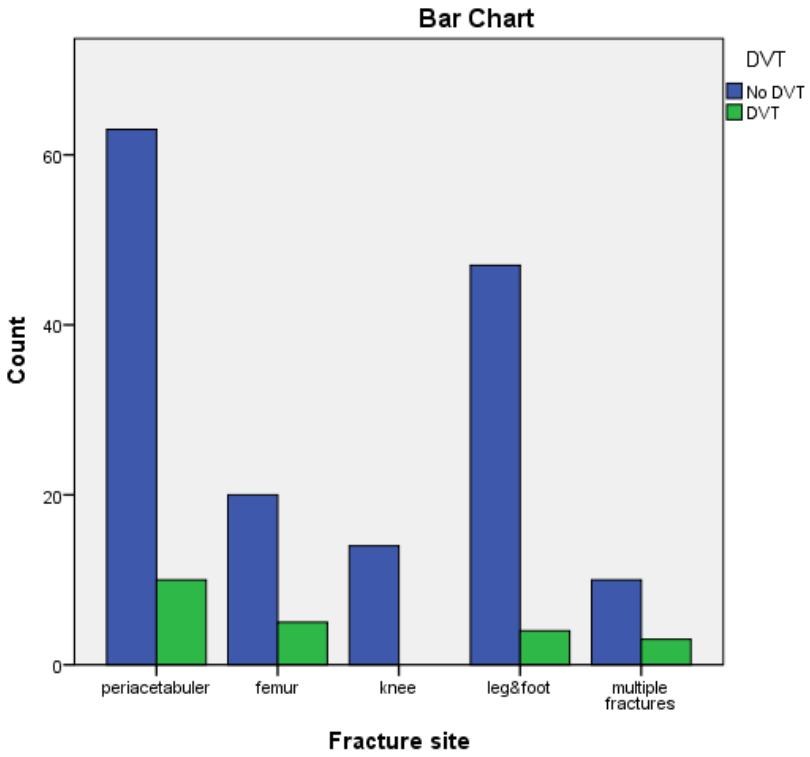


Figure (4-5) Fracture site * DVT Crosstabulation

Table (4-14) Time before restoration * DVT Crosstabulation

			DVT		Total
			No DVT	DVT	
Time before restoration	within first hour	Count	46	8	54
		% within Time before restoration	85.2%	14.8%	100.0%
	(2-4)hours	Count	33	2	35
		% within Time before restoration	94.3%	5.7%	100.0%
	(5-7)hours	Count	12	1	13
		% within Time before restoration	92.3%	7.7%	100.0%
	(8-10)hours	Count	8	1	9
		% within Time before restoration	88.9%	11.1%	100.0%
	more than10 hours	Count	24	6	30
		% within Time before restoration	80.0%	20.0%	100.0%
	no restoration	Count	31	4	35
		% within Time before restoration	88.6%	11.4%	100.0%
Total		Count	154	22	176
		% within Time before restoration	87.5%	12.5%	100.0%

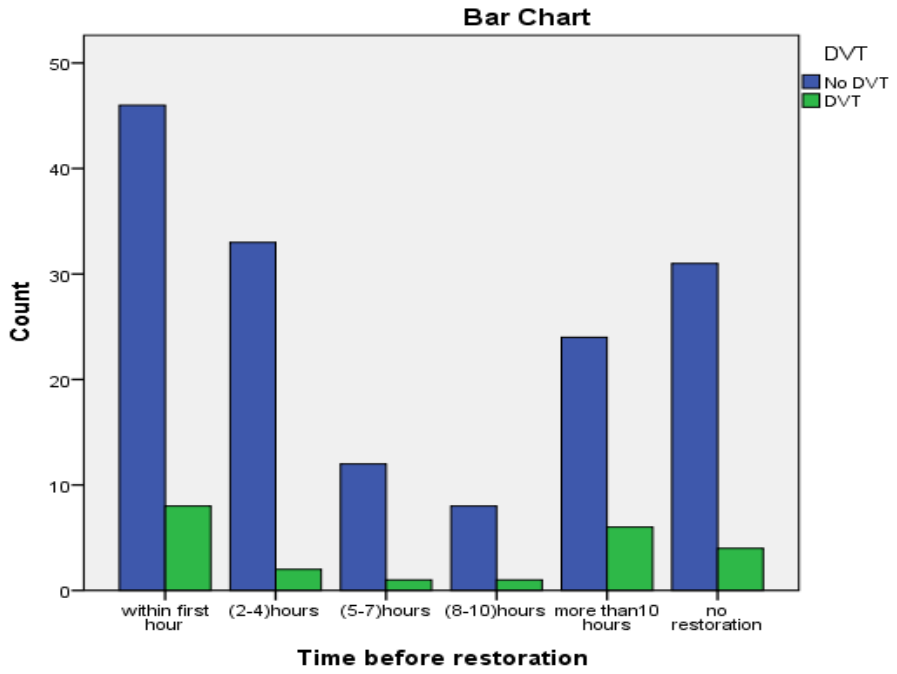


Figure (4-6) Time before restoration * DVT Crosstabulation

Table (4-15) Type of Restoration * DVT Crosstabulation

			DVT		
			No DVT	DVT	Total
Type of Restoration	plaster	Count	56	4	60
		% within Type of Restoration	93.3%	6.7%	100.0%
	external fixation	Count	6	0	6
		% within Type of Restoration	100.0%	0.0%	100.0%
	traction	Count	56	11	67
		% within Type of Restoration	83.6%	16.4%	100.0%
	TBS	Count	4	3	7
		% within Type of Restoration	57.1%	42.9%	100.0%
	None	Count	32	4	36
		% within Type of Restoration	88.9%	11.1%	100.0%
Total		Count	154	22	176
		% within Type of Restoration	87.5%	12.5%	100.0%

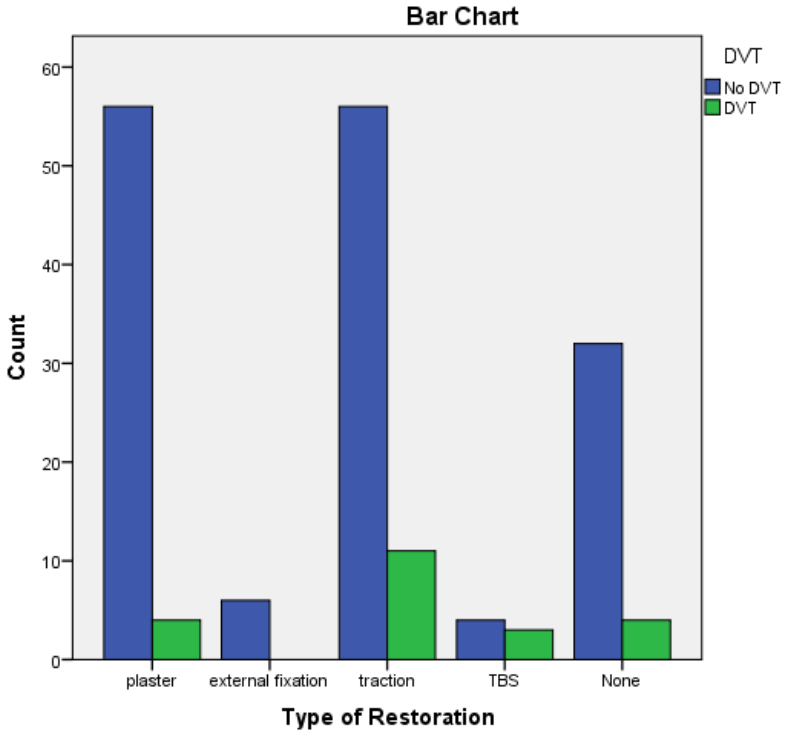


Figure (4-7) Type of Restoration * DVT Crosstabulation

Table (4-16) Fracture Type * DVT Crosstabulation

			DVT		
			No DVT	DVT	Total
Fracture Type	Single Closed	Count	102	16	118
		% within Fracture Type	86.4%	13.6%	100.0%
	Single Open	Count	22	2	24
		% within Fracture Type	91.7%	8.3%	100.0%
	Multiple Closed	Count	23	3	26
		% within Fracture Type	88.5%	11.5%	100.0%
	Multiple Open	Count	7	1	8
		% within Fracture Type	87.5%	12.5%	100.0%
Total		Count	154	22	176
		% within Fracture Type	87.5%	12.5%	100.0%

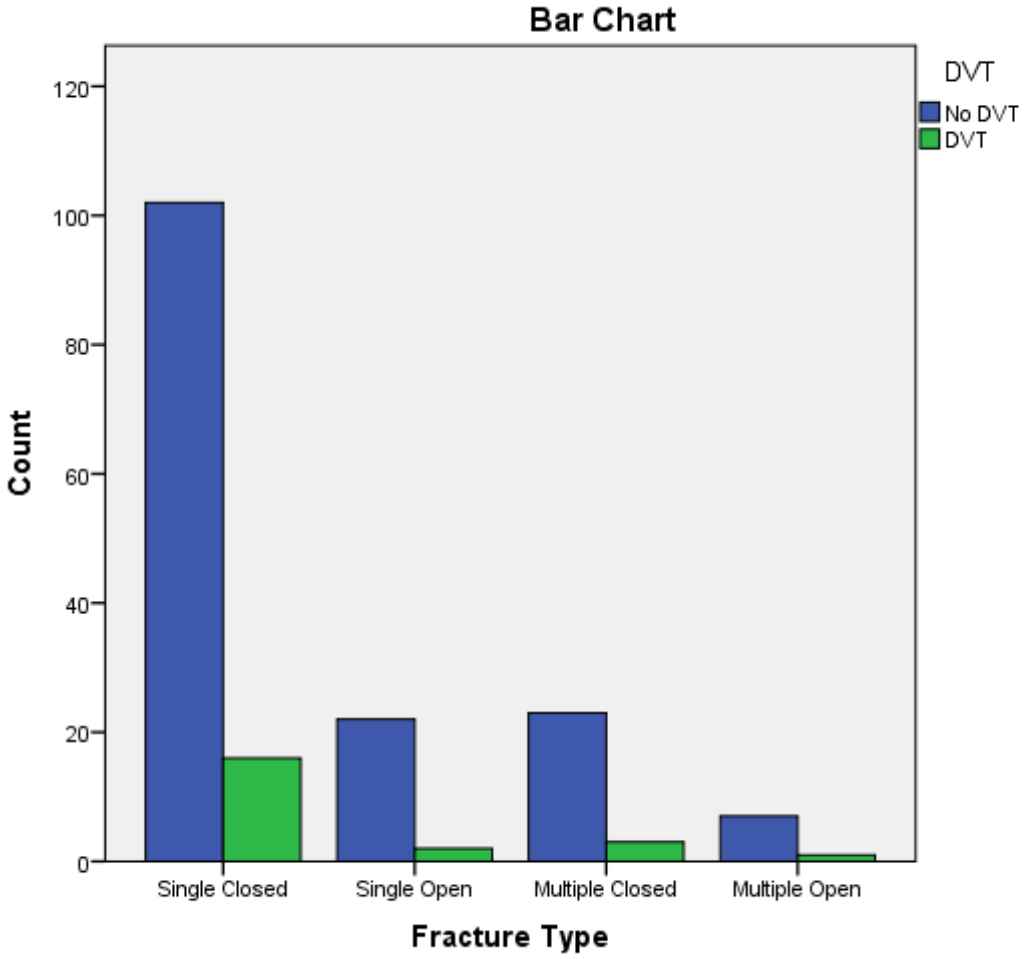


Figure (4-8) Fracture Type * DVT Crosstabulation

Table (4-17) Anticoagulant drug * DVT Crosstabulation

			DVT		
			No DVT	DVT	Total
Anticoagulant drug	Used	Count	129	18	147
		% within Anticoagulant drug	87.8%	12.2%	100.0%
	Not Used	Count	25	4	29
		% within Anticoagulant drug	86.2%	13.8%	100.0%
Total		Count	154	22	176
		% within Anticoagulant drug	87.5%	12.5%	100.0%

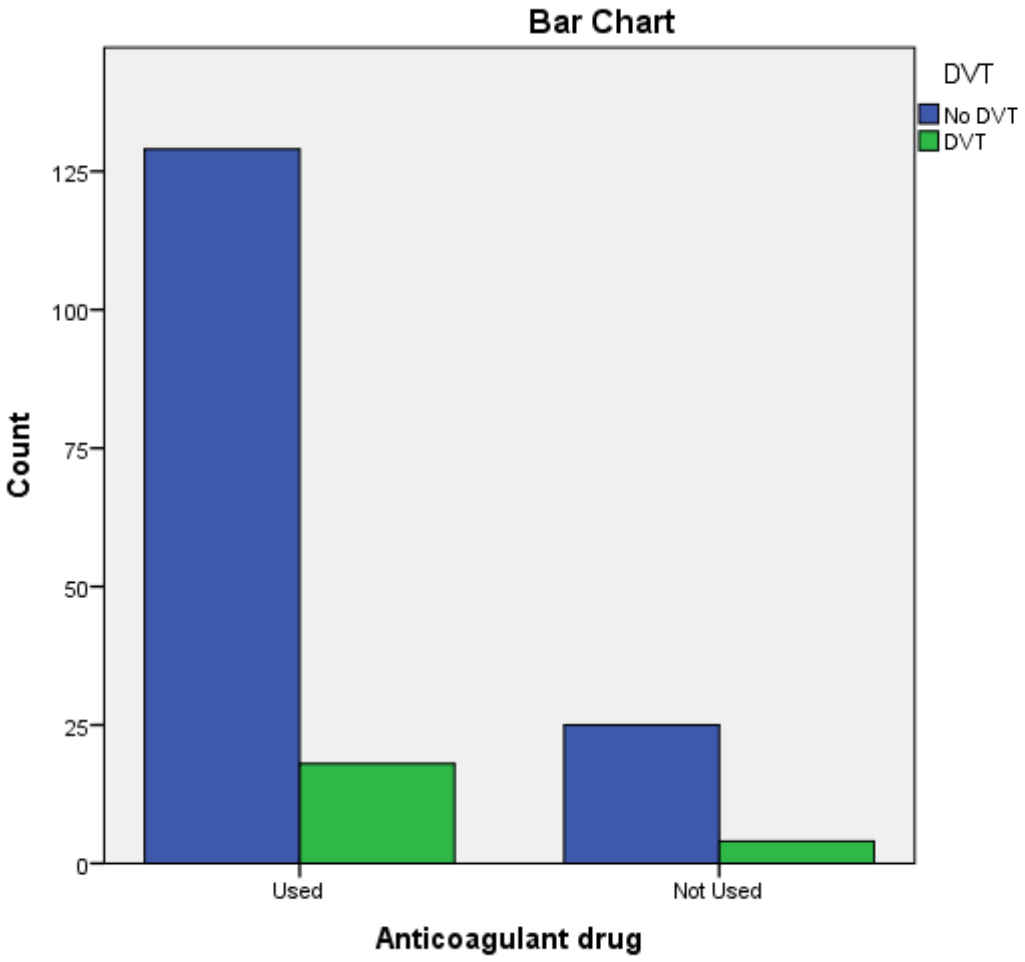


Figure (4-9) Anticoagulant drug * DVT Crosstabulation

Table (4-18) DM & HTN * DVT Crosstabulation

			DVT		
			No DVT	DVT	Total
DM & HTN	None	Count	120	12	132
		% within DM & HTN	90.9%	9.1%	100.0%
	DM	Count	12	5	17
		% within DM & HTN	70.6%	29.4%	100.0%
	HTN	Count	10	4	14
		% within DM & HTN	71.4%	28.6%	100.0%
	Both	Count	12	1	13
		% within DM & HTN	92.3%	7.7%	100.0%
Total		Count	154	22	176
		% within DM & HTN	87.5%	12.5%	100.0%

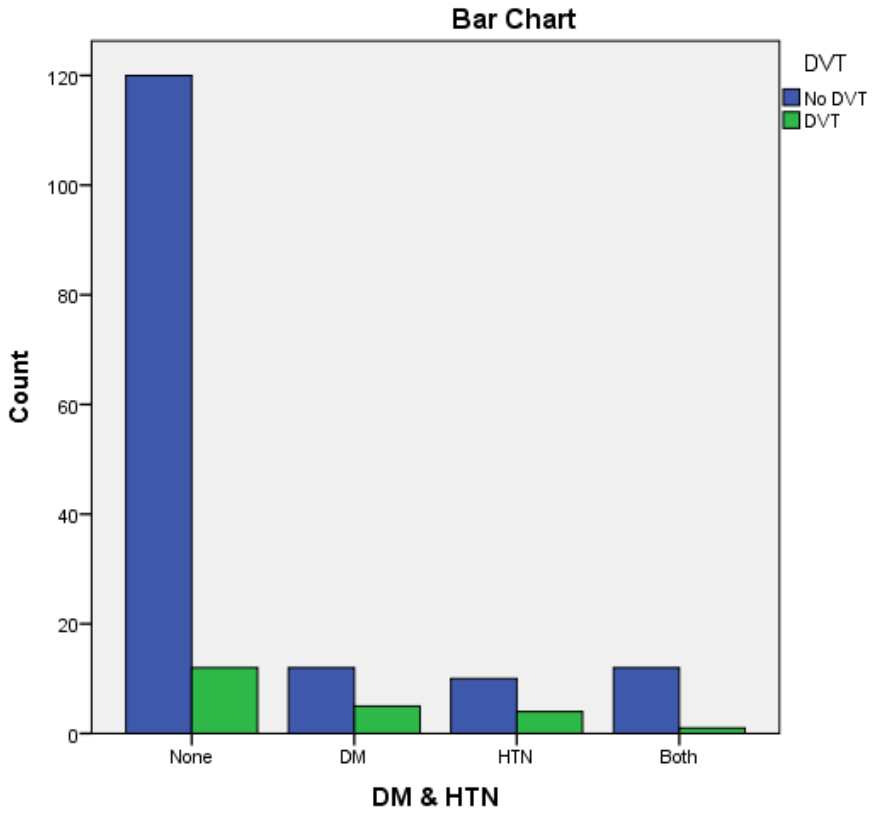


Figure (4-10) DM & HTN * DVT Crosstabulation

Table (4-19) Respiratory phasicity * DVT Crosstabulation

			DVT		
			No DVT	DVT	Total
Respiratory phasicity	Yes	Count	146	3	149
		% within Respiratory phasicity	98.0%	2.0%	100.0%
	No	Count	8	19	27
		% within Respiratory phasicity	29.6%	70.4%	100.0%
Total		Count	154	22	176
		% within Respiratory phasicity	87.5%	12.5%	100.0%

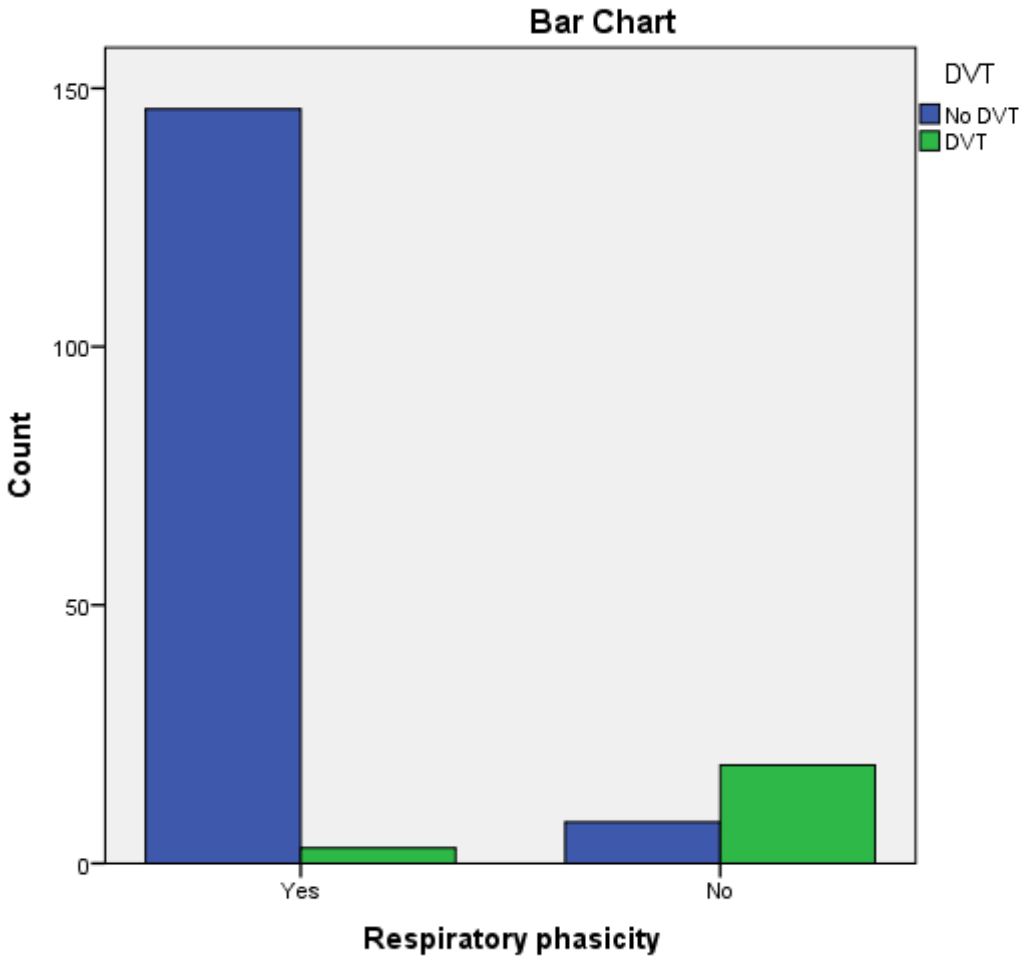


Figure (4-11) Respiratory phasicity * DVT Crosstabulation

Table (4-20) compressibility * DVT Crosstabulation

			DVT		
			No DVT	DVT	Total
compressibility	full	Count	154	0	154
		% within compressibility	100.0%	0.0%	100.0%
	semi	Count	0	11	11
		% within compressibility	0.0%	100.0%	100.0%
	Noncompressible	Count	0	11	11
		% within compressibility	0.0%	100.0%	100.0%
Total		Count	154	22	176
		% within compressibility	87.5%	12.5%	100.0%

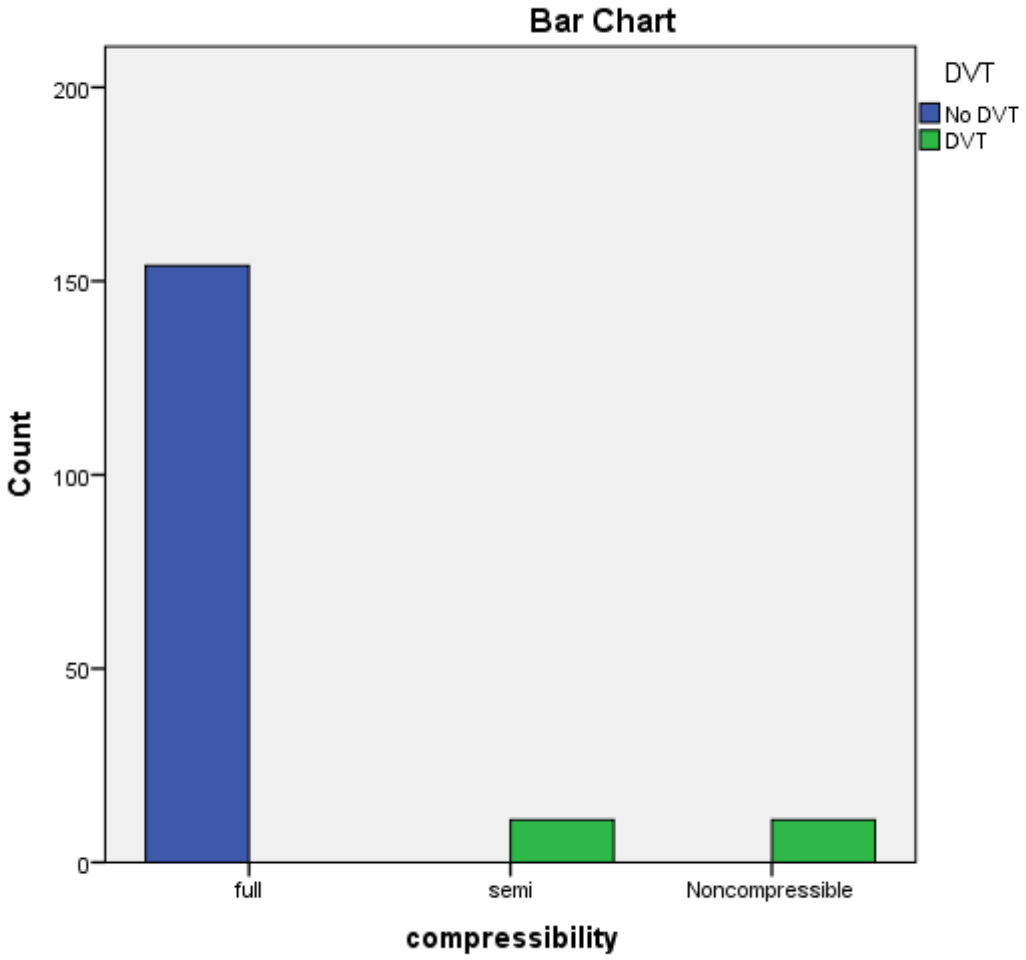


Figure (4-12) compressibility * DVT Crosstabulation

Table (4-21) Regonality * DVT Crosstabulation

			DVT		
			No DVT	DVT	Total
Regonality	Not available	Count	26	6	32
		% within Regonality	81.3%	18.8%	100.0%
	Khartoum	Count	56	7	63
		% within Regonality	88.9%	11.1%	100.0%
	Middle States	Count	23	4	27
		% within Regonality	85.2%	14.8%	100.0%
	East States	Count	10	1	11
		% within Regonality	90.9%	9.1%	100.0%
	West States	Count	29	3	32
		% within Regonality	90.6%	9.4%	100.0%
	North States	Count	10	1	11
		% within Regonality	90.9%	9.1%	100.0%
Total		Count	154	22	176
		% within Regonality	87.5%	12.5%	100.0%

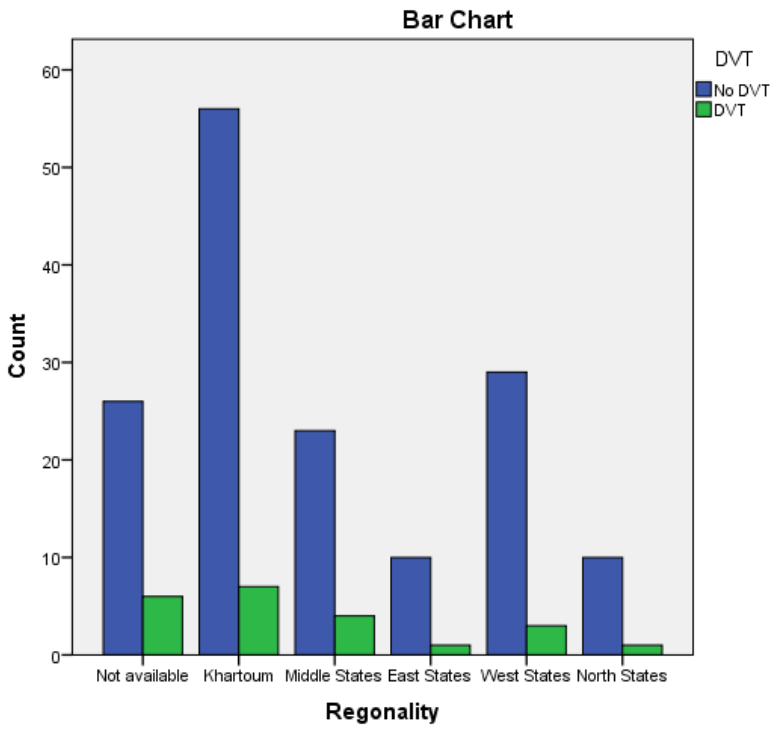


Figure (4-13) Regionalist * DVT Crosstabulation

Table (4-22) Performance of absence of compressibility and phasicity in the detection of deep venous thrombosis in fractured lower extremities

Absence of compressibility in detection of DVT in fractured lower extremities	Number of cases (*n*)	Absence of phasicity in detection of DVT in fractured lower extremities		Number of cases (*n*)	
True positive	13	True positive		14	
True negative	45	True negative		44	
False positive	0	False positive		0	
False negative	3	False negative		2	
Performance of absence of compressibility in detection of DVT in fractured lower extremities	Value	95% CI	Performance of absence of phasicity in detection of DVT in fractured lowerextremities	Value	95% CI
Sensitivity (%)	81.25%		Sensitivity (%)	87.50%	61.65-98.45%
Specificity (%)	100.00%		Specificity (%)	100.00%	91.96-100.00
Negative likelihood ratio	0.19		Negative likelihood ratio	0.12	0.03-0.46
DVT prevalence in fractured lower extremities (%)	26.23%		DVT prevalence in fractured lowerextremities (%)	26.67%	16.07-39.66%
PPV (%)	100.00%		PPV (%)	100%	–
NPV (%)	93.75%		NPV (%)	95.65%	85.75-98.77%

DVT – deep venous thrombosis, PPV – positive predictive value, NPV – negative predictive value

CHAPTER FIVE

5.1-Discussion

The study takes place between January 2016 and March 2018, 129men and 47 women (mean age, 49.8 years).According to the results of this study, fractures of the lower extremities were classified according to the complexity, and wound pattern either in single closed, single open, multiply closed, or multiple open. The DVT was found in 16 (13.6%) patients with single closed fractures, 2 (8.3%) patients with single opened fractures, 3 (11.5%) patients with multiple closed fractures, one (12.2%) patient with multiple closed fractures, and one (12.5%) patients with multiple opened fracture Table (4-16). Such as that by Takahiro Niikura et al. [16], in which the authors evaluated the rate of venous thromboembolism (VTE) after complex lower limb fracture surgery without pharmacological prophylaxis. A total of 154 (87.5%) patients had no DVT, while 22 (12.5%) patients had lower limb DVT. Table (4-9)

It should be noted that the time before restoration divided into six groups. the fracture fixation of the first group was done within the first hour of trauma events which contained 54 patients (30.7%), the DVT events was(14.8%), the fracture fixation of the 2nd group was done within two to four hours contained 35 patients (19.9%), the DVT events was(5.7%), the 3rd group within five to seven hours which contained 13 patients (7.4%), the DVT events was(7.7%), the 4th group within eight to ten hours contained 9 patients (5.1%), the DVT events was(11.1%), the 5th group the fixation done after ten hours of fracture occurring which this group involved 30 patients (17.0%), the DVT events was(20.0%), the last group contained 35patients (19.9%) without fixation. the DVT events

was(11.4%), Table (4-14). Immobilization more than 4 days was the most common risk factor present in patients with DVT. A study by Abelseth.1 G et al suggested a higher DVT incidence in more proximal fractures, but little risk of embolization. Thrombus formation proximal to the popliteal fossa is rare. Older age, longer operating times, and longer times before fracture fixation all correlate with an increased incidence of DVT.

In this study, fractures of the lower extremities were classified according to the type of fixation either by plaster, traction, external fixation or TBS none fixated fractured patient considered the fifth type of fixation. Six patients (3.4%) fixated by external fixation no one of them have DVT, 67 patients (38.1%) fixated by traction, eleven (16.4%) of them were developed DVT, 7 patients (4.0%) used TBS, three out of them (42.9%) afflicted by DVT. And 36 patients (20.5%) procedure code listed as (None) with free lower extremities no fixation done, four (11.1%) of them were developed DVT. Table (4-15). Sharma et al conclude that the post-TBS complications present a heavy burden to the hospital and consume a large amount of government exchequer. These complications stretch even further the already bursting workload and Bed-capacity of the hospital. Orthopaedic Surgeons , Physicians and other staff of the hospital are forced to spent a lot of their busy time looking after these patients. What is ridiculous is that in spite of the true scenario, common people still believe that TBS is the best for their bone and soft tissue problems. People only present to the hospital if and when serious complications arise. The available literature suggests that this trend is present in many developing nations throughout Africa, Asia and South America.[16] Concerning the above results we believe that the TBS

procedure is the most high risk facture of DVT may be due to unsuitable local tight materials that damaged or obstructed the lower limb veins.

In this study, the overall prevalence of DVT in fractured lower extremities was (12.5%) Table (4), immobilization alone is not a strong enough predisposing factor to affect the incidence of DVT in patients with fracture lower extremity. However, the risk of DVT increases significantly in patients with 3 or more predisposing factors.

Traumatized veins may undergo post-traumatic thrombosis, and this may change the normal characteristics of these vessels. The presence of hematoma at the site of major veins without visualization of the vein is a robust indicator of whole vein rupture. In trauma patients, vein thrombosis develops minutes or hours after trauma. For the diagnosis of DVT, one can apply compression techniques and CDUS. In normal veins, blood flow is uninterrupted and spontaneous at rest, and the velocity changes with respiration, rising on inhalation and falling on exhalation. Active compression of the distal parts of the legs causes an increase or augmentation of the velocity. In the case of partial thrombosis, blood flow may be normal or abnormal, without response to respiration. In complete obstructive thrombosis, there will be an entire absence of blood flow on DUS investigation.

In the current of study, VDUS showed that some patients with fractures lower extremities had DVT (22, 12.5%). The absence of compressibility (4, 18.2%) and phasicity (4, 14.8%) was detected in DVT involving CFV (4, 18.2%), Absence of compressibility (8, 36.4%) was noted in DVT involving SFV (8, 36.4%), but the absence of phasicity was noted only in (5, 18.5%) of the affected patients. The incidence of absence of compressibility and phasicity was found in DVT that affected solely POPV (1, 4.5%), SFV (8, 36.4%), and both POPV and SFV (2, 9.1%).

Was (4.5% & 3.7%) , (36.4% , 18.5%) and (9.1% 7.4%) respectively. As regards the cases in which DVT involved CFV, SFV, and POPV (7, 31.8%), absence of compressibility and phasicity was found in 7 (31.8%) and 7 (29.9%) patients, respectively

Color flow Doppler (CFD) is currently considered as the first-line examination for evaluation of vascular injuries, with sensitivity and specificity of 89-100% relative to venography. Moreover, CFD has an accuracy of 95% to 98% in detecting lower extremity DVT. (Gaitini D, Razi NB, Ghersin E et al 2008.)(Hammers LW, Cohn SM, Brown JM et al 1996) In the current study, a diagnostic testing applied to 58 patients was revealed that the sensitivity of 81.25% and specificity of 100% of absence of compressibility in the detection of DVT in fractured lower extremities. Furthermore, positive predictive value (PPV) of 100% and negative predictive value (NPV) of 93.75% were found (Table 4-22). In addition, absence of phasicity in the diagnosis of DVT in fractured lower extremity has sensitivity of 87.50% and specificity of 100%. PPV and NPV in these cases were 100% and 95.65%, respectively (Table 4-2). Obesity, large hematomas, emphysema, and large uncovered wounds of the skin can hinder US examination performed in order to rule out trauma-related vascular injuries. Further limitations are due to operator dependence and duration of examination, which may be inappropriate in an acute setting. Despite these limitations, venous US can help diagnose other pathologies such as Baker’s cysts, superficial or intramuscular hematomas, lymphadenopathy, femoral aneurysm, superficial thrombophlebitis, and absceses. Moreover, there is no exposure to radiation. In the current study, we diagnosed one Baker’s cysts (1, 0.6%), lymphadenopathy (62, 35.2%), and intramuscular hematomas (3, 1.7%).

5.2-Conclusions

In conclusion, we evaluated the role of DUS in the detection of deep venous blood flow in fractured lower extremities to rule out DVT. The incidence of DVT after orthopedic trauma was high (16, 27.6%) and appears to be associated with fractures of the peri-acetabular area& femur, age and fixation type. VDUS with gray-scale imaging with compression maneuvers should be the first-line imaging modality for suspected lower extremity DVT. Loss of phasicity and compressibility in a venous segment, often with accompanying Doppler abnormalities, indicates DVT with a high degree of accuracy, and no additional examination is required to initiate management.

5.3-Recommandation

Finally according to the current study we recommended that:-

1. As the LL Doppler US scan is a noninvasive, high specificity procedure should be the first investigation to exclude the DVT
2. the LL Doppler US scan should be a routine exam pre and post major orthopedic surgery
3. patient on prophylaxis should be followed up by LL Doppler ultrasound scan
4. LL Doppler exam should be as part of the treating surgeon's protocol

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Appendix (A)

DATA COLLECTION SHEET					
1-No					
2-Age					Year
3-Sex	Male		Female		
4-Obesity	Kg		cm	BMI	
5-Residency					
6-blood group					
Fracture					
7-Fracture site	Periacetabular	knee	Femur	Tibia Fibula ankle foot	
8-Date of fracture					
9-Time before restoration					
10-Type of restoration	plaster	Ext fix.	Traction	TBS جبيره	None
11-Fracture type	Single/closed	Single/open	Multiple/close	Multiple/open	
12-Operation	Pre operation	Pos operation			
13-operation duration					
14-Walking after operation					
15-Operation date					
16-blood translation	No	Yes	the amount=		
17- patient transfer	Bed redden		Walking with support		
18-Anticoagulant	Used		Not		
Patient history					
19-Family history	Yes		No		
20-DM&HTN	DM	HTN	Both	None	
21-Patient habits	Snuff	Cigarette	coffee	Tobacco	Other
22-previus history of DVT					
23- vascular interventional					
24-Oral contraceptive	Yes		No		
25-other risk factors					
Sonographic Finding					
26-Blood velocity(cm/s)	CFV=	SFV=	POPV=		
27-Respiratory phasisity	Yes		No		
28-Compressibility	Full	Semi	None		
29-DVT	Yes		No		
30-Site of DVT	CFV	SFV	POPV	Extensive	

31-Other sonographic findings

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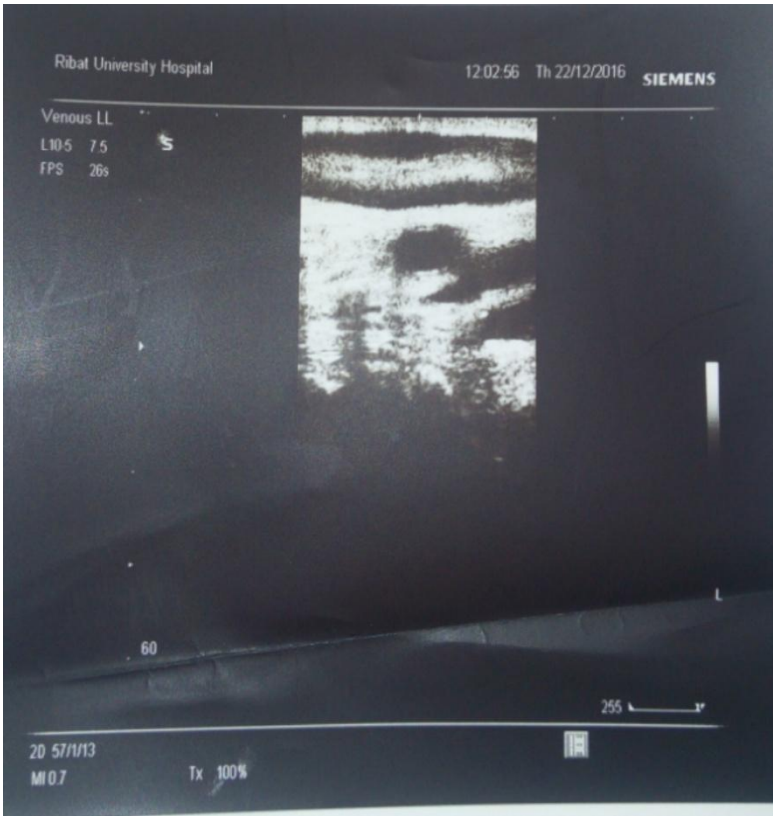
……..…

32-comment

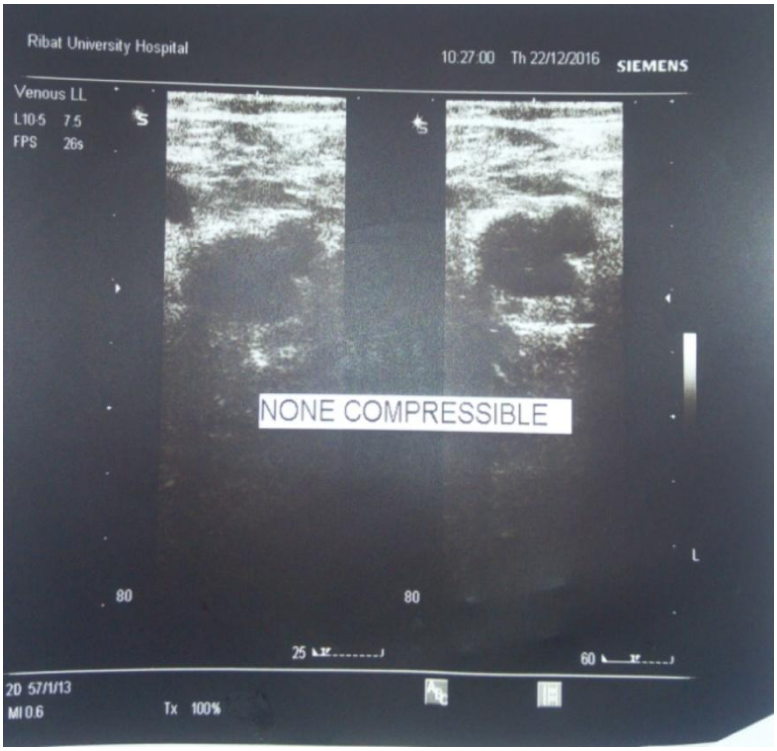
……..……

Appendix (B) Images



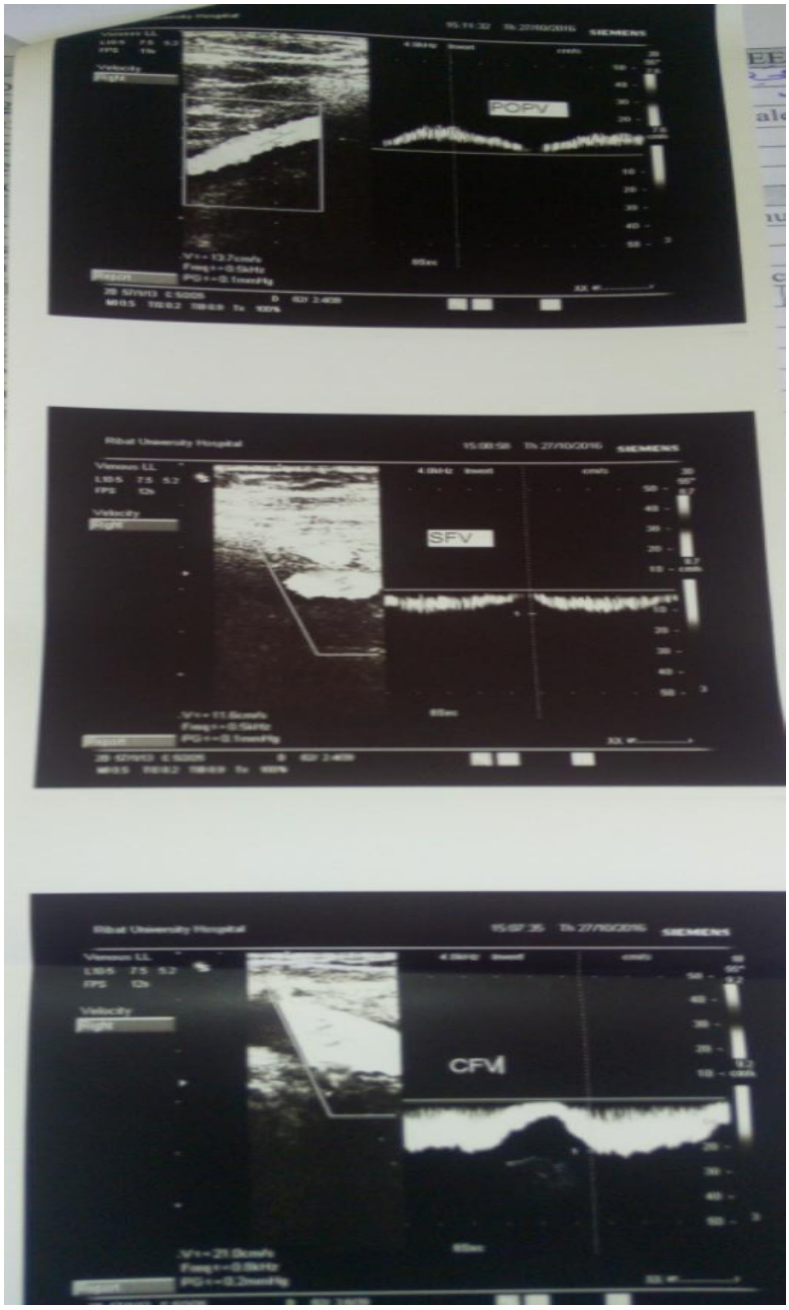
Male 45 y

Gray scale image for CFV DVT+ Enlarged Lymph node



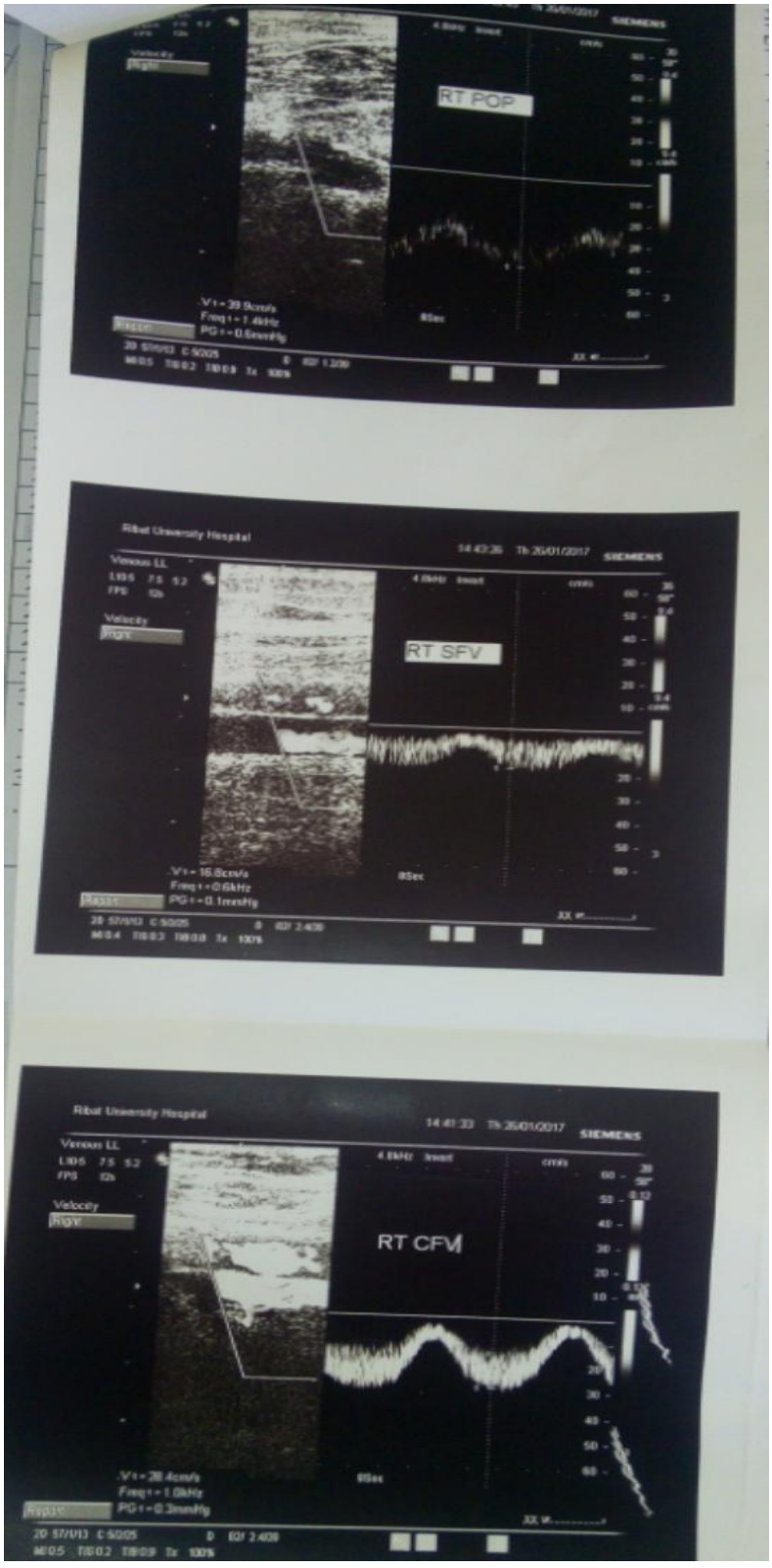
Male 46years old

Grayscale U/S image for CFV DVT. Note the non compressibility



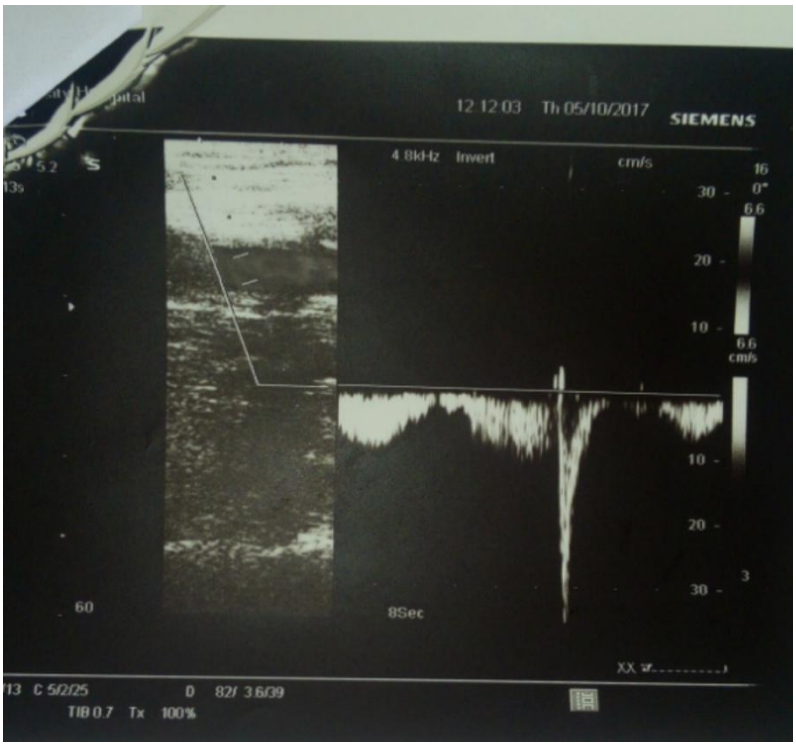
Male 22years old.

Spectral Doppler image for normal deep Venus flow

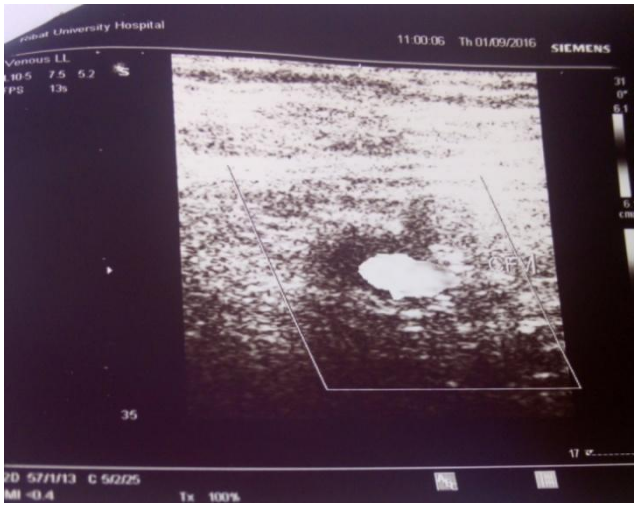


Male 38 years old

Spectral Doppler image for normal flow Note the Respiratory phasicity

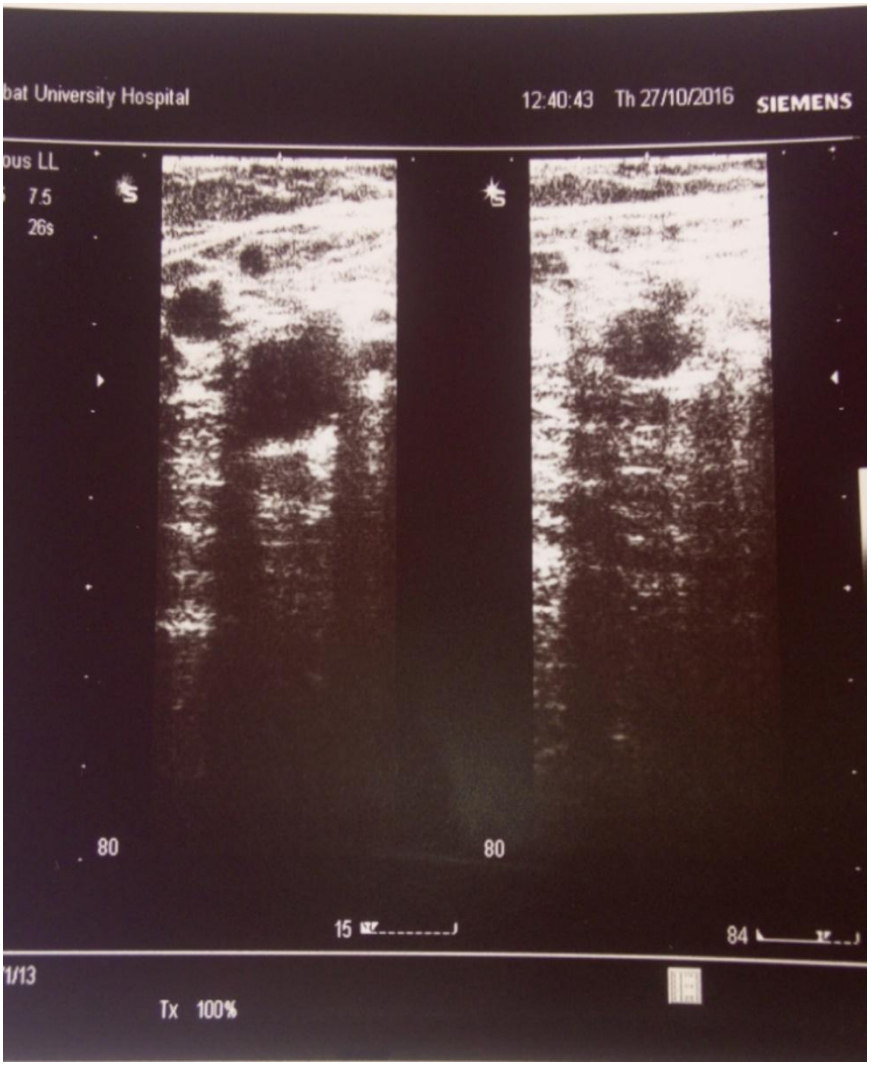


Male 35 years old normal Spectral Doppler imaging for SFV venus flow. Note the spike sign of augmentation test



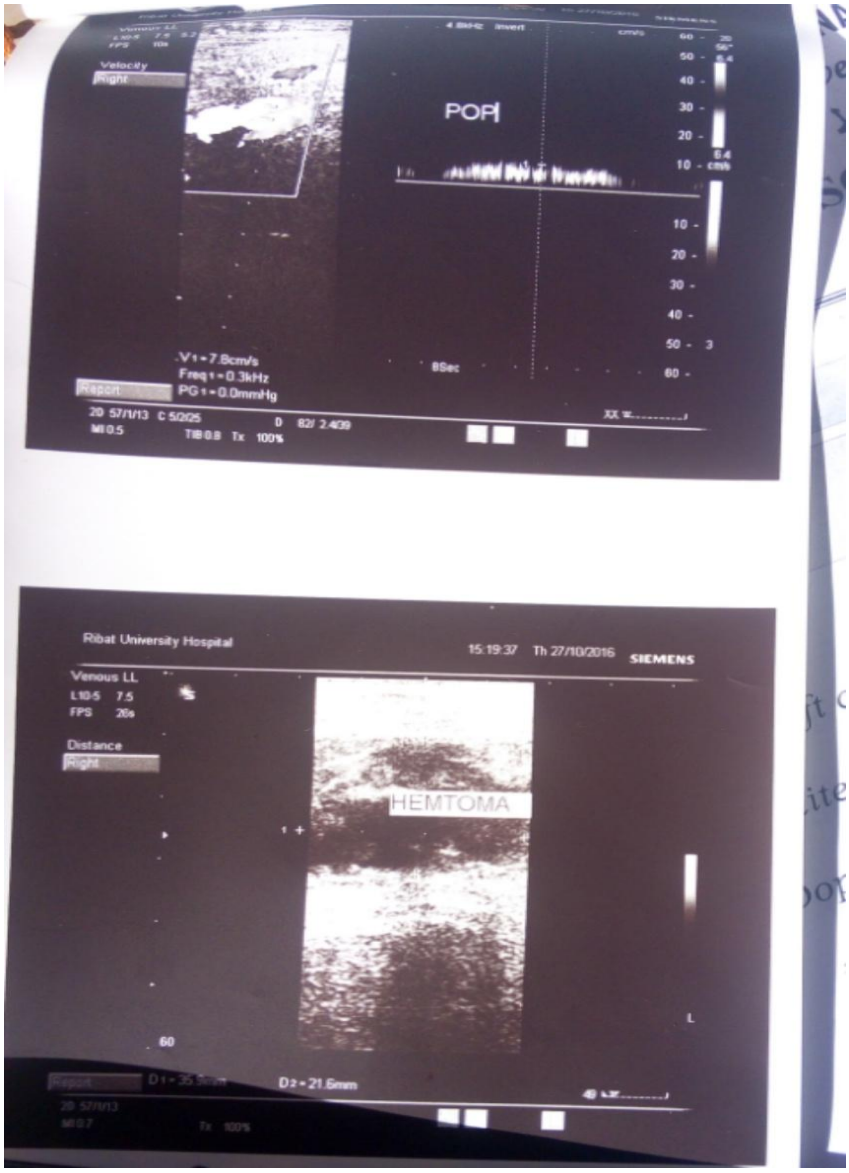
Female 75 years old

Color Doppler imaging demonstrated CFV DVT



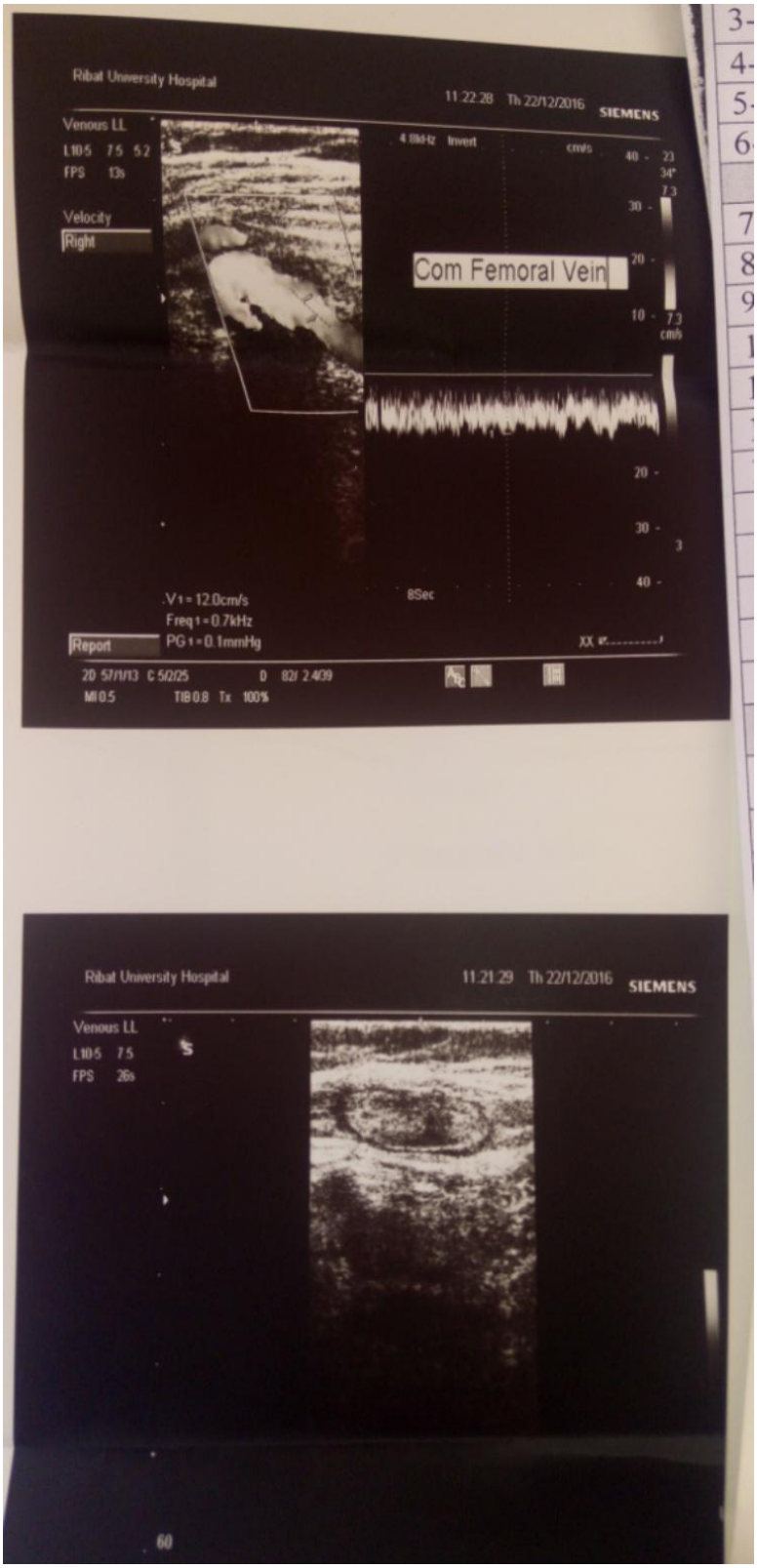
Female 37 years

2D imaging showed extensive DVT note the non compressible CFV



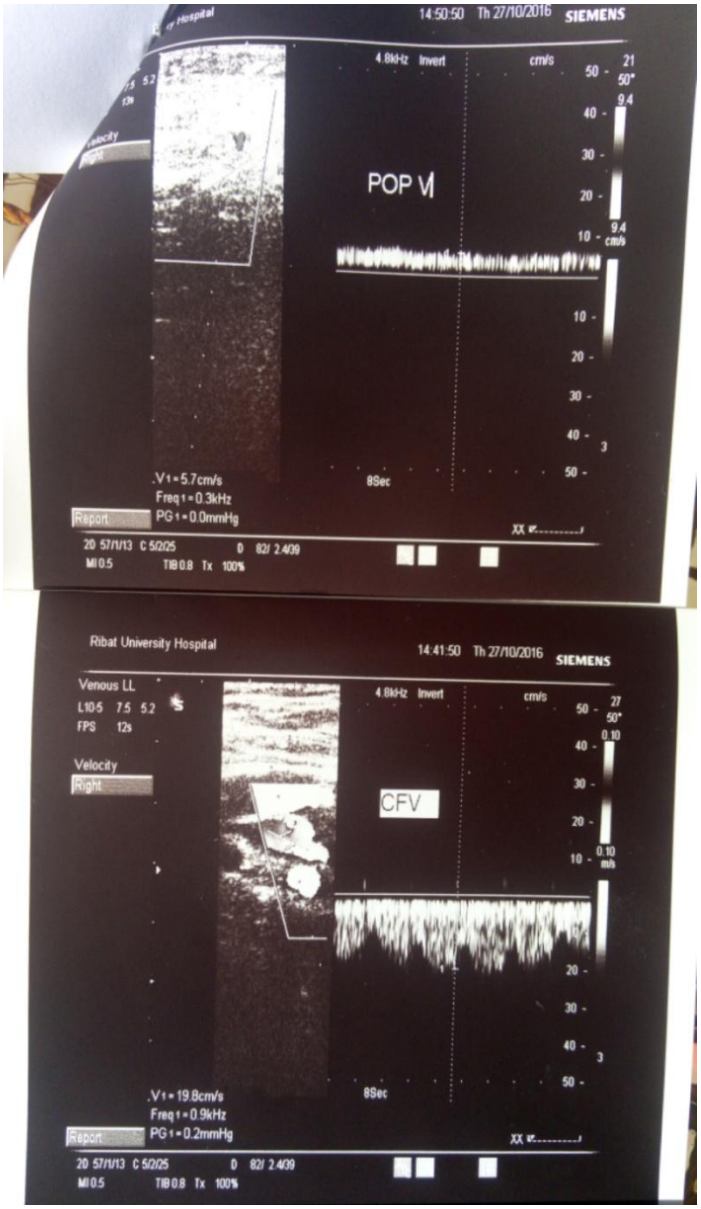
Male 27 years

2D & Spectral Doppler image for old hematoma + POP DVT



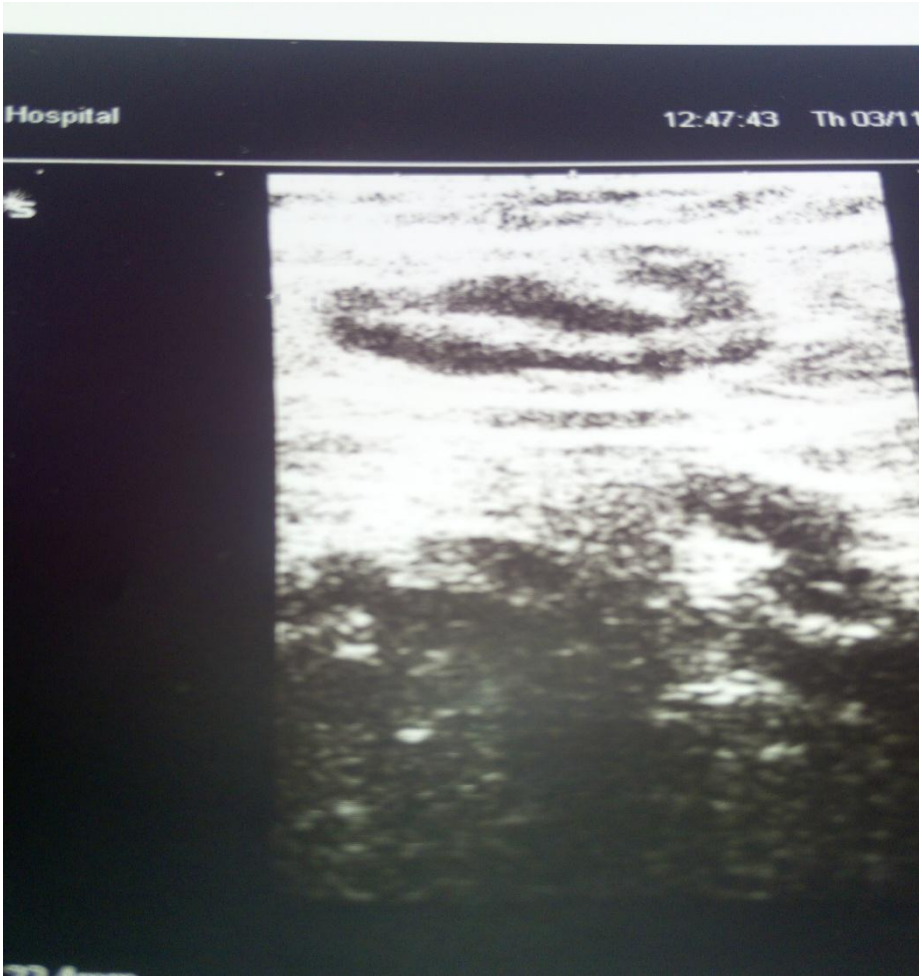
Female 70 years old

Spectral Doppler image for extensive DVT +Lymph node



Male 27 years old

Spectral Doppler image demonstrated partial DVT in the CFV . Note the absence of respiratory phasicity at the POP vein



Male 70 years old 2D image showed enlarged inguinal lymph node

No DVF

Published papers

Awadalla Adam, Mohamed Yousef1, Babiker A. Wahab, Ahmed Abukonna, Mustafa Z. Mahmoud, Duplex ultrasound for evaluation of deep venous blood flow in fractured lower extremities, Pol J Radiol 2018; 83: e47-e53

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