Hip Anatomy

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Introduction

The osseous layer (Layer 1) is composed of the femur, acetabulum, and a pair of innominate bones that form the pelvis. This provides joint congruence while allowing four planes of motions: abduction, adduction, flexion, and extension in a normal hip [1].

The femur is the heaviest, strongest, and the longest bone in the human body. The femoral head is encased within the acetabulum cavity and is convex in shape [2]. The femoral head stabilizes the hip joint during gait and allows weight bearing for the upper body [3]. It is the reliable shape of the femur that allows it to play a role in both the movement of the hip and knee joint and serves as the site of origin and insertion for many muscles and ligaments that play a role in the continuum of the lower extremity kinetic

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chain [3]. The femur can be divided into three separate sections: proximal, shaft, and the distal bone. In this chapter, the proximal part of the femur and its role in the hip joint will be the focus of discussion. The femoral head has an anterior, medial, and superior surface that serves as insertion sites for multiple tendons [3]. To accommodate the full range of motion of the hip joint, the femoral head is overlayed with an articular cartilage [3]. Hyaline cartilage covers approximately 60–70% of the femoral head [3]. The rest of the area not covered by hyaline cartilage is called the fovea capitis and is located at the center area of the femoral head [3] which serves as the insertion site of the ligamentum teres [3].

The femoral neck supports the femoral head by playing a vital role in the connection between the femoral head and shaft [4]. The neck shaft angle (NSA), measures to 125 ± 5, in a normal adult [3]. This angle is also termed the angle of inclination, which is the angle measured from the medial side of the femoral shaft to the femoral neck [3]. If the NSA were to exceed 130°, this condition is termed coxa valga [3], if less than 120°, this condition is termed coxa vara [3], and outside the range of 120–130°, it would decrease the range of motion of the hip joint [3]. The NSA is measured at 150° at birth, but because of the loaded stresses at the femoral neck (due to increased weight bearing and walking during adolescents and childhood), the NSA will decline to 125° by adulthood [3].

Is the measured angle located between the mediolateral line through the anterior knee and a line through the femoral shaft and head [3]. Femoral anteversion creates an anterior rotation oriented in the coronal plane and is measured to be 15–20° [3]. Pelvic parameters are necessary to measure in order to characterize the position of the lumbar spine and the effects of the pelvic tilt in the sagittal plane [5]. The pelvic parameters consist of pelvic incidence (PI), pelvic tilt (PT), and the sacral slope (SS). PI is defined as the relative position of the sacral plate in relation to the femoral heads [5]. Patients with a low PI have a small anterior posterior (AP) diameter [5]. This is also termed a vertical pelvis, which is narrow horizontally and strong vertically [5]. A pelvis with a high PI has a large AP diameter and is large in the horizontal plane [5]. The pelvic incidence is the sum of

the sacral slope and the pelvic tilt [5]. The pelvic tilt is the angle between a vertical line of the two sacral endplates and indicates the spatial orientation of the pelvis [5]. The sacral slope is the angle of the sacral plateau to the horizontal plane [5]. For a given pelvic incidence, a specific pelvic tilt will be associated with sacral slope [5].

There are important radiographic parameters a physiatrist should be familiar with to accurately assess and manage various hip morphologies. The alpha angle is the angle measured that connects the femoral head to the distance from the center of the head to the point where the head exceeds the radius of the femoral head [6]. The alpha angle is used to measure the femoral head sphericity and is the measurement used by clinicians to diagnose CAM lesion [6]. The Tonnis angle is the measurement that quantifies the weight-bearing portion of the acetabular roof. It is seen on AP pelvis radiographs and is the line formed between the horizontal line and the tangential line that connects the medial edge to the lateral edge of the sourcil [6]. Clinically, the Tonnis angle has been correlated with subluxation of the hip, femoroacetabular impingement (FAI), and developmental dysplasia of the hip (DDH) [6]. The lateral center edge angle (LCEA) is the X-ray assessment of the lateral acetabular coverage in the frontal plane [6]. This measurement is used to classify hip dysplasia or characterize acetabular under coverage. A LCEA value of 20–25° is considered normal in an adult. Hips with a value of LCEA of less than 15° (in children) and less than 20° (in adults) are considered to have developmental hip dysplasia. If the LCEA shows over coverage of the femur head on radiographic imaging, FAI can be diagnosed [6]. The anterior center edge angle (ACEA) is the angle formed by the line passing through the center of the femoral head to the most anterior part of the acetabular sourcil [6]. An ACEA as low as 20° can indicate hip pathology such as acetabular dysplasia or FAI [7]. Low ACEA is correlated with anterolateral acetabular deficiency [6].

The greater and less trochanter, which projects superolateral and posteromedial, respectively, sit at the base of the femoral neck, where the periarticular muscles are inserted [4]. The trochanters' bony prominences significantly impact the muscle

groups that are attached to them [3]. Three main groups supply the arterial blood to the proximal femur which are the extracapsular arterial ring, ascending cervical branches of the extracapsular arterial ring on the surface of the femoral neck, and the arteries that supply the round ligament [3]. Injury to the retinacular arteries can be seen in any fracture of the femoral neck [3].

The acetabulum is a cup-shaped cavity, created by the innominate bones (ilium, ischium, and the pubis bones) [3]. Before the age of 14, these bones are skeletally immature and are divided by the triradiate cartilage [3]. Between the ages 14 and 16, these bones start to fuse together and are completed by the age of 23 [3]. The acetabulum is a Y-shaped triradiate cartilage that covers 70% of the femoral head [8]. The acetabulum is 40% made from the ilium, 40% from the ischium, and 20% from the pubic bone [3]. Acetabulum is a cavity located outside the pelvis where the ball shaped femoral head sits in and is encompassed by the acetabular edge which is a circular bone flap [4]. The lunate surface is the most peripheral portion of the acetabulum, and the acetabular surface is the most central portion [4]. The lunate contains a central inferior acetabular fossa that engulfs fat tissue, and the origin of the ligamentum teres [3]. These two surfaces are used during the articulation of the femoral head and acetabulum. The acetabular pit contains the round ligament which is surrounded by vascular anatomy and adipose tissue [4].

Within the osseous layer, structural pathologies may exist which are classified as dynamic or developmental [9]. There are three groups of dysfunctions, which are identified as static overload, dynamic impingement, or dynamic instability [1]. Dynamic pathologies related to the hip joint include trochanteric impingement, sub-spine impingement, delamination, and femoroacetabular impingement [9]. Pathologies that fall within the developmental category include femoral inclination, acetabular protrusion/profunda, dysplasia, acetabular version, femoral version [9]. Dynamic pathology also includes loss of the femoral head sphericity due to increase in age leading to impingements, labral tears or delamination [9].

The inert layer (Layer 2) is composed of the labrum, capsule, ligamentous complex, ligamentum teres, capsule-ligamentous

structures, and the synovial joint. The purpose of the inert layer is to distribute stability to the hip joint [9].

The labrum has many functions of the hip which includes shock absorption, joint lubrication, and pressure distribution [8]. It forms a fibrocartilaginous extension of the acetabulum which increases the containment of the femoral head [10]. The fibrocartilage characteristic of the labrum creates a negative intra-articular pressure within the hip joint [11]. The labrum works to hinder the fluid flow in and out of the hip joint, creating a "seal effect" that creates distraction of the femoral head from the acetabular pocket [10]. The seal limits lateral motion of the femoral head creating joint stability and preserves the quality of the hip joint [12]. Studies suggest that the labrum helps to center the femoral head in the cavity of the acetabulum, maintaining the thickness of the fluid layer between the bony surfaces [10]. The seal effect also functions to increase joint stability, and distribute compressive loads to the articular surfaces, which in return decreases the stress on the hip during weight-bearing action [10]. Ferguson et al. studies suggest that the labrum maintains joint stability in the most extreme range of motion, especially in flexion, due to the inconsistent nature of the femoroacetabular joint [10]. The labrum circumferentially encircles the acetabulum's perimeter from the base of the fovea capitis to the transverse acetabular ligament anteriorly and posteriorly [2]. During loading in the gait cycle, the labrum acts as a tension band that limits the expansion during the motion between the anterior and posterior columns [13]. If this tension is lost, due to labral tears, the joint will be more susceptible to increased load forces and repetitive trauma [2]. Increasing the concavity of the acetabular fossa, as well as increasing the contact area with the femoral head; the labrum plays a significant role in the stability of the hip joint [4]. In the absence of stability, the hip joint is vulnerable to many pathologies. The surface of the labrum has limited vascularity and minimum synovial coverage, making the labrum more susceptible to tears and low healing probability [2]. Blood vessels that supply the labrum are found in the outmost layer in the capsular surface, thus explaining why the labrum remains avascular [2]. Labral tears clinically manifest as hip instability more than pain, due to

free nerve endings located in the superficial layer of the labrum tissue [2]. The labrum is innervated by the branches of the nerve to the quadratus femoris and the obturator nerve [3]. The anterior labrum is more prone to tears due to the anterior section of the labrum being thinner while the posterior portion is thicker [2]. Labral tears can arise due to repetitive strain to the anterior section during abduction or external rotation in flexion or extension [2].

The capsule is a strong fibrous tissue that enforces hip stability established by the labrum [3]. The capsule fibers that encircle the femoral neck (zona orbicularis) help counteract the stresses placed on the acetabular labrum [3]. The capsule, along with the labrum, plays a significant role in bearing weight load in the flexion range of motion, thus explaining instability of the hip joint if capsular laxity is present [13]. The capsule is part of capsular ligamentous complex system that is made of the deep and superficial layers of the longitudinal ligaments [2]. The function of the ligamentous complex is to reinforce stability to the hip joint [2]. Repetitive tears of the capsule tissue can create a rotational instability of the hip joint, which is also associated with deficiency of labral tissue [2]. The capsule is the primary location that receives multiple innervations at the hip joint [3]. The posterior and inferior regions of the capsule are innervated by the posterior articular nerve, which is a branch of the nerve to the quadratus femoris [3]. The superior gluteal nerve innervates the superior regions of the capsule, while the branch of the femoral nerve innervates the anterior region of the capsule [3]. The anteroinferior and anteromedial regions of the capsule are innervated by the medial articular nerve which is a branch of the obturator nerve [3].

The function of the ligamentum teres remains to be unclear. Due to the existence of arteries around the ligamentum, this suggests that it plays a role in the blood supply to a developing hip [14]. The ligamentum teres tightens in hip flexion, adduction and external rotation [2]. It has been hypothesized that the ligamentum teres aids in hip stabilization and can resist dislocation forces placed on the femoral head [15]. The innervation of the ligamentum teres is by the posterior branch of the obturator nerve [3].

Ligaments involved in the attachment of the pelvis and femur to reinforce the capsule of the hips are the iliofemoral, pubofemoral, and ischiofemoral ligaments [3]. In the motion of hip extension, these ligaments are noted to be tight [3]. They are most slack in the motion of flexion, abduction, and external rotation [3]. Of the three ligaments, the iliofemoral ligament is the strongest, as it extends from the anterior inferior iliac spine to the anterior intertrochanteric line in an inverted Y shape [3]. The primary function of the iliofemoral ligament, also known as the ligament of Bigelow, is to help prevent hyperextension of the hip joint [3]. The pubofemoral ligament attaches to the superior pubic ramus proximally and the inferior femoral neck distally [3]. The key role of the pubofemoral ligament is to prevent hyperabduction of the hip. The ischiofemoral ligament, the thinnest extracapsular ligament, will attach the ischial rim of the acetabulum to the femoral neck crossing the posterior aspect of the hip joint [3]. The primary function of this ligament is to stabilize the joint in the extension motion. The innervation to the ischiofemoral ligament is the posterior articular nerve [3].

The synovial fluid in the hip joint is also part of the inert layer. The synovial joint lines the inner surface of the joint capsule and will form a sheath that covers the joint of the round ligament [4].

The osseous layer (Layer 1) and the inert layer (Layer 2) have a consequential relationship where if there is damage to either layer, it will affect the other [1]. For example, mechanical stress that influences the hip joint due to underlying abnormalities in the osseous layer lead to certain pathologies of the inert layer [1]. These pathologies can include labral tears and ligamentous tears, capsular irritation, hip instability or adhesive capsulitis [1].

Layer III, as known as the contractile layer, consists of all the muscular tissue that initiates, controls, and supports specific movement about the hip joint [9]. The contractile layer includes trunk stabilizers and the pelvic floor musculature. The main purpose of this layer is to provide dynamic stability to the hip, pelvis, and trunk [9]. Abnormal mechanics between the osseous (Layer 1) and inert layer (Layer 2) may lead to instability and increased tension to the lower spine, pubic symphysis, and SI joint. This tension therefore creates secondary stress to the muscles attached

to the same pelvic structures. When examining the muscles and tendons of the hip joint, it is great practice to examine the muscles by anatomical position: the anterior, lateral, medial, and posterior muscle groups.

The Iliopsoas group consists of the iliacus, psoas major, and minor muscle. These muscles originate from the lateral surface of the spine between the 12th thoracic vertebra and the upper region of the fifth lumbar vertebrae, the iliac crest, and the sacrum [3]. The muscles then insert on to the lesser trochanter of the femur. As the muscle begins to enter the thigh region and passes under the inguinal ligament, it becomes thinner leaving the muscle more susceptible to injury [4]. The psoas muscle joins the inguinal ligament to form the iliopsoas muscle [4]. Innervated by the femoral nerve, the iliopsoas muscle is the major hip flexor of the hip joint [3]. The medial femoral circumflex and iliolumbar arteries act as the main blood supply of the muscle [3]. The iliacus muscle originates from the upper portion of the iliac fossa and the anterior inferior iliac spine (AIIS) [3]. The rectus femoris muscle originates from the AIIS and the iliac crest while inserting on the superior patella [3]. This muscle also plays a significant role in flexion of the hip joint and is innervated by the femoral nerve, specifically the L2–L4 nerve root [3]. The blood supply of the rectus femoris muscle is the lateral femoral circumflex artery of the descending branch [3].

Structural pathology (Layer 1) of the hip joint is related to muscular tissue pathology (Layer 3). Mechanism of such pathology can be acute, traumatic, developmental avulsions, or overuse tendinosis [1]. Hip internal snapping of the psoas muscle can be seen when the muscle is displaced laterally with flexion and medially with hip extension [9]. When making a diagnosis of the anterior muscles of the hip, certain enthesopathy pathologies should be considered when making a differential diagnosis. Hip flexor strains, psoas impingement, and sub-spine impingement syndrome can be seen on physical examination of patients with hip flexor pathology [1]. Psoas tendonitis and rectus femoris avulsion pathologies may be associated with patients complaining of groin pain [16]. When the psoas and iliacus muscle merge to become a common tendon, it is called iliopsoas musculotendinous junction

[17]. It is at this junction that the iliopsoas tendon is susceptible to pathologic conditions such as iliopsoas bursitis, distal iliopsoas strain, partial or complete tears, and snapping of the iliopsoas tendon [17]. Snapping iliopsoas syndrome, defined as pain or discomfort occurring in the groin due to the snapping motion of the psoas tendon [17], was thought to be caused by the impingement of the psoas tendon against the iliopubic bone [17]. Recent studies under sonography have shown that it is due to the distal iliopsoas tendon that causes iliopsoas snapping syndrome, as the iliopubic eminence remained medial to the psoas tendon during dynamic and static evaluations [17]. It is during the motion of flexion, abduction, and external rotation that the psoas major tendon glides around the medial part of the iliacus muscle, which then can become entrapped between the tendon and the superior pubic ramus, therefore causing the snapping sensation [17]. Internal snapping of the psoas muscle could occur over either the femoral head or the iliopectineal eminence [9]. During the physical exam, the psoas muscle will be noted to be displaced laterally with flexion and medially with hip extension [9]. Pain will be localized over the anterior part of the groin when diagnosing internal snapping of the hip [18].

The gluteus maximus, medius, minimus, and the tensor fascia lata are considered to be the lateral muscles of the hip joint and the main function is abduction of the hip joint. The gluteus maximus originates from the posterior part of the ilium, dorsal sacral surface, and coccyx [3]. The muscle then inserts onto the posterior iliotibial tract and the gluteal tuberosity [3]. The gluteus maximus, innervated by the inferior gluteal nerve (L5, S1, S2), is considered the main hip extensor, but also plays a role in external rotation, abduction, and adduction [3]. The muscle receives its blood supply from both the superior and inferior gluteal artery [3]. The gluteus medius originates from the anterior gluteal line and inserts onto the lateral surface of the greater trochanter [3]. The gluteus minimus also inserts onto the lateral surface of the greater trochanter but originates from the outer cortex of the ilium [3]. Both muscles are involved in internal rotation and abduction of the hip joint. The muscles are also innervated by the superior gluteal nerve and receive their blood supply from the superior gluteal

artery [3]. The tensor fascia lata takes its origin from the anterior superior iliac spine and is inserted onto the iliotibial tract [3]. This muscle plays a role in hip abduction and internal rotation, but also part takes in hip flexion and external rotation and receives its blood supply from the ascending branch of the lateral circumflex femoral artery [3]. The piriformis muscle also originates from the inner surface of the sacrum and inserts onto the greater trochanter [3]. The piriformis muscle will aid in hip extension and external rotation. The muscle receives its blood supply from a branch of the inferior gluteal artery [3].

As a physiatrist, it is important to recognize lateral enthesopathies when examining the lateral muscle groups of the hip. These pathological conditions may involve the peri-trochanteric space and injuries to the gluteus minimus and medius tendons [1]. Tears of the gluteus medius and minimus muscle can clinically result in a positive Trendelenburg sign and weakness when patients are ascending stairs [9]. Tears of the muscle were also found in approximately 20% of hip fractures [9]. The tensor fascia lata can show decreased activation during hip flexion in patients diagnosed with femoroacetabular impingement syndrome (FAI) [1]. One must also consider deep gluteal syndrome, which is a syndrome that involves posterior soft tissue injury and irritation or compression of the sciatic nerve [1]. This can cause a constellation of pain patterns that involve the hamstrings but also the gluteal muscles as well [1]. Tears of the hip abductors may present as a musculoskeletal condition called the greater trochanter pain syndrome (GTPS), in which the gluteus medius and minimus become irritated at their insertion (greater trochanter) [19]. Patients may complain of lateral sided hip pain that is dull and aching and is exacerbated by weight bearing and resisted hip abduction motion [19]. Gluteus medius tears are seen more than gluteus minimus tears in GTPS due to the medius muscle having three anatomical attachments (central, posterior, and anterior) to the greater trochanter compared to the gluteus minimus which only has two insertions. Therefore, the gluteus medius is more prone to tears and degeneration during the gait cycle [19]. Hip snapping, specifically external type, can be caused by snapping of the posterior border of the iliotibial band or the anterior border of

the gluteus maximus over the greater trochanter when the hip is seen in flexion from an extended position [18].

Besides the gluteus maximus, the extensor muscles of the hip joint are the semitendinosus, semimembranosus, and bicep femoris muscles [3]. All three muscles originate from the ischial tuberosity, while having different insertion sites [3]. The semitendinosus inserts onto the anteromedial tibial plateau, while the semimembranosus inserts onto the posterolateral tibial plateau [3]. The bicep femoris inserts onto the fibular head of the fibula and the posterolateral tibial plateau as well [3]. All three muscles are innervated by the tibial branch of the sciatic nerve (L5, S1, and S2) [3]. The inferior gluteal and the perforating arteries are the blood supply to the semitendinosus muscle. The blood supply of the semimembranosus muscle is the profunda femoris and gluteal arteries [3]. Bicep femoris (long and short head) are supplied by the inferior gluteal, popliteal arteries, and the perforating branches from the inferior gluteal and profunda femoris arteries [3].

The semitendinosus, semimembranosus, and bicep femoris muscles are considered the posterior muscle group of the hip joint. Pathologies that can be seen are posterior enthesopathy, and proximal hamstring strains [9]. Proximal hamstring tendonitis is a differential diagnosis with patients that present with groin pain [16].

The external rotators of the hip include the piriformis, obturator internus and externus, quadratus femoris, superior and inferior gemellus muscles. These muscles run in the horizontal plan of the hip below the piriformis muscle [3]. All muscles play a role in hip adduction and external rotation; these muscles are also innervated by the sacral plexus branches L5–S1 [3]. The obturator internus muscle originates from the inner surface of the obturator membrane and inserts onto the medial greater trochanter, receiving innervation from the nerve to the obturator internus (L5–S1) [3]. The blood supply of the obturator internus muscle is the superior gluteal artery [3]. The obturator externus muscle originates from the outer surface of the obturator membrane (including the pubic ramus and the ischium bone) and inserts onto the trochanteric fossa. The innervation to the obturator externus is the obturator nerve (L3, L4), while receiving blood from the anterior branch of

the obturator artery and the medial circumflex femoral artery [3]. The superior and inferior gemellus muscle originate from the ischial spine and the ischial tuberosity, respectively [3]. Both muscles insert onto the posterior greater trochanter and are supplied by the branches of the inferior gluteal artery [3]. The superior gemellus is innervated by the nerve to the obturator internus (L5, S1), while the inferior gemellus is innervated by the nerve to quadratus femoris [3]. The piriformis muscle originates from both the anterior surface of the sacrum and sacrotuberous ligament, while inserting into the posterosuperior trochanter [3]. The ventral rami of S1 and S2 acts as the nerve supply to the piriformis muscle [3]. The last muscle of the external rotator group is the quadratus femoris muscle. This muscle originates from the lateral border of the ischial tuberosity while inserting into the quadratus tubercle [3]. The innervation to the quadratus femoris muscle is the nerve to the quadratus femoris [3].

The medial muscles of the hip include the adductor longus and brevis, adductor magnus, adductor minimus, gracilis, and the pectineus muscles [4]. All the medial muscles of the hip function to adduct the hip joint [3]. The adductor longus originates from the superior pubic ramus and inserts into the middle third of the linea aspera [3]. Not only does this muscle help with hip adduction but can also flex the hip up to 70° [3]. The adductor brevis originates from the inferior ramus and body of the pubis and inserts onto the proximal linea aspera and the pectineal line [3]. The adductor magnus muscle originates from the inferior pubic ramus, ischial ramus, and the ischial tuberosity. While inserting into the gluteal tuberosity, and the adductor tubercle of the medial femur [3]. This muscle not only functions in hip adduction but also contributes to hip extension and external rotation [3]. The adductor minimus originate from the inferior pubic ramus and insert into the medial portion of the linea aspera. This muscle will contribute with hip extension along with adduction [3]. The gracilis muscle originates from the inferior pubic ramus and inserts onto the distal knee [3]. The obturator nerve (L2–L4) is the innervation to the adductors of the hip [3]. The adductor magnus also receives innervation from

the sciatic nerve, while the pectineus muscle receives innervation from the femoral nerve [3].

Medial enthesopathies are the pathological patterns seen with medial muscles of the hip. These can include adductor rectus tendinopathies which are also called athletic pubalgia or sports hernias [1]. This can be caused due to overactivity of the adductor muscles. For example, the adductor muscles are seen to be overactive especially when the abductor muscles are weak [9]. This forces the adductor longus to act as a hip stabilizer in the frontal plane, causing repetitive motion and overactivity, leading to a tendinosis of the adductor muscles [9]. The adductor muscles work synergistically with the abdominal muscles to stabilize the pelvis during motion [16]. When there is injury to the adductors, patients will most commonly complain of groin pain and would not be able to take part in athletic activity [16]. During the physical exam, if there is tenderness to palpation at the adductor enthesis, with pain on passive adductor stretching and resisted adduction, adductor pathologic injury should be highly considered [16].

Lumbosacral muscles can be further distinguished by their function related to the hip. The rectus abdominus, external and internal abdominal oblique serve as the core muscles. The transversus abdominus and the multifidus muscle function as the stabilizers/deep core muscles. The erectors (ILS) include the interspinales, iliocostalis lumborum, and longissimus lumborum muscles.

Due to the attachments of the lumbosacral muscles to the pelvis, this can often affect the abdominal wall architecture correlating to the prevalence of sports hernias, and femoroacetabular impingement syndrome (FAI) seen in clinic [9]. When examining layer III, the physiatrist must consider postural adaptive changes that can lead to the Lower Crossed Syndrome. The Lower Crossed Syndrome is defined as an imbalance of muscle strength between the abdominal and gluteal muscles, iliopsoas, and thoracolumbar extensor muscles thus creating an anterior tilt of the pelvis [9]. Rectus abdominis tendinopathies are often seen with overuse of the rectus abdominal tendon and can lead to athletic pubalgia aka sports hernia. Sports hernias can be illustrated as occult hernias

that are caused by a tear or weakness of the posterior inguinal wall without a visible hernia on physical exam [16]. Other etiologies of sports hernias can be due to reduced force of conjoined tendons, injury at the insertion of the rectus abdominis muscle, tearing of the internal oblique muscle at the pubic tubercle, tearing within the internal oblique musculature, or abnormality seen in the external oblique aponeurosis [16]. Gilmore's groin pathology is seen when there is a tear seen in the external oblique aponeurosis [16]. Often athletic pubalgia can be co-diagnosed with FAI during a physical exam. This can be explained due to restriction in flexion and internal rotation at the hip joint resulting in abnormal motion in the pelvis [16]. The abnormal motion is responsible for the injury to the ligaments and musculature associated with sports hernia and FAI [16]. When athletes have weak abdominal muscles but relatively strong adductor muscles, this can result in trunk hyperextension and thigh hyperabduction therefore increasing the risk of sports hernias [16]. On physical exam, pain can be reproducible with resisted hip adduction or Valsalva maneuver [16].

The neuromechanical layer, also known as layer IV, is the theoretical layer that is composed of physiological processes, anatomical formation, and kinematic changes that occur throughout the pelvic area that drive proprioception and pain generators through the hip joint [9]. The neuro-kinetic layer serves as the neuromuscular link between the spine and the lower extremity as it functions within its environment [1]. Layer IV can be viewed either locally or globally. Locally, this layer contains the neuro-vascular structures, mechanoreceptors, and nociceptors at the site of the hip [9]. From the global view, layer IV refers to the posture and position of the pelvis over the femur bone [9]. In this section, we will discuss the neuro-vascular anatomy of the hip, regional mechanical receptors, the thoracolumbar mechanic and its relation to the hip, and the lower extremity mechanics and the compensation response from the hip. This section will also aim to illustrate how the neuromechanical layer entails examining the functional movement patterns and how it effects the dynamic movement of the pelvis over the femur, or femur under the pelvis [9]. The compensatory injuries that are seen within this layer are pain syndromes, nerve compressions, neuromuscular dysfunction, and spinal referral patterns [1].

The hip joint is innervated by multiple nerves specifically pertaining to the capsule of the hip. The inferior region of the hip and the ischiofemoral ligament is innervated by the posterior auricular nerve which is a branch of the nerve to the quadratus femoris [3]. The hip capsule is also innervated by the superior gluteal nerve which supplies the superior aspect of the capsule [3]. The branch of the femoral nerve supplies the innervation to the anterior capsule [3]. Peripheral nerve disorders are commonly seen in hip pathology. This includes femoral neuropathy, lateral femoral cutaneous neuropathy (Meralgia taresthetica), sciatic neuropathy (aka piriformis syndrome), obturator neuropathy, pudendal neuropathy, ilioinguinal, ilio-hypogastric, genitofemoral, superior and inferior gluteal neuropathies [1].

The hip receives its blood supply from several different arteries [3]. The medial and lateral circumflex femoral arteries are the main blood supply to the hip joint [9]. These arteries originate from the profunda femoral artery, which is the deep artery of the thigh, but there are variations where the arteries arise from the femoral artery [9]. The femoral head is also supplied by a small branch of the posterior division of the obturator artery which passes through the ligamentum teres [9]. The gluteal arteries and the femoral or the deep artery of the thigh are the main components of the gluteal and trochanteric anastomosis of the hip [9]. The acetabulum receives its blood supply from the obturator, superior, and inferior gluteal arteries [3]. The foveal artery, which is a branch of the posterior division of the obturator artery, runs along the ligamentum teres to supply the fovea centralis of the femur bone [3]. The proximal end of the femur bone is supplied by the extracapsular arterial ring (located at the femoral neck composed of the medial and lateral femoral circumflex, and the superior and inferior gluteal arteries), the ascending cervical branches of the arterial ring, and the arteries of the round ligament [3]. Overall, the labrum is not well supplied with blood, reasons why there is a slow healing process after the labrum is torn [9]. Even though it has scarce blood supply, the labrum does receive blood from a combination of arteries which include the inferior and superior gluteal arteries, and the obturator artery [9].

Nerve endings can be further categorized by mechanoreceptors and free nerve endings. The four types of mechanoreceptors are Ruffini endings, Pacinian corpuscles, and Golgi tendon organs [9]. Free nerve endings are also known as intra-articular nociceptive receptors [9]. Joint mechanoreceptors can be further subcategorized in type I, type II, type III, and type IV receptors. Type I receptors are found in tendons, ligaments, periosteum, and the joint capsule [9]. These receptors are responsible for proprioception and are also known as the Ruffini endings [9]. Type II receptors are located in the fat pads and the deep joint capsule [9] These receptors are responsible for kinesthesia and are also called Pacinian corpuscles [9]. Type III receptors are found in the extrinsic and intrinsic ligaments [9]. They are also known as the Golgi tendons and are responsible for proprioception [9]. Type IV receptors, free nerve endings, are responsible for pain sensation and are found in the blood vessels, fat pads, joint capsule, ligaments, and tendons [9]. Type II receptors are receptors that have a low threshold and are rapidly adapting [9]. These receptors are inactive at rest but are active during point position and movement [9]. Type I receptors, when compared to type II, have a higher threshold and are slow adapting [9]. These receptors are located in the inferior joint capsule of the hip and are less notable in the peripheral joints [9]. Type I play a significant role in the hip joint by being able to respond to stress and stretch in the hip joint capsule [9]. Past studies have shown that there is a minimum number of mechanoreceptors found in the hip joint capsule, further explaining the lack proprioception response when injury occurs [9]. This explains the lack of local reflexive response during injury, the increased vulnerability to neuromuscular inhibition, and dysfunction in the hip joint compared to peripheral joints [9].

The lumbopelvic rhythm explains how the lumbar spine and surrounding muscles move with the pelvis. It is the motion relationship between the hip joints and the lumbar spine in the sagittal plane. The first part of the lumbosacral rhythm involves the lumbar and sacral spine in flexion while the pelvis has an anterior tilt orientation at the hip joint. When a subject is returning to the erect posture, the rhythm is then reversed. This motion is commenced by the posterior tilting of the pelvis at the hip joint, which is fol-

lowed by extension of the lumbar spine. Existing hip or spinal pathology can affect the muscles of the hip which may change the mechanics of the lumbar pelvic rhythm [9]. Low back pain, especially amongst athletes, is usually associated with injury at L5–S1 vertebrae. There is a local response to the lumbosacral muscle due to this injury, which is inhibition to the segmental stabilizers, multifidus, and transversus abdominus [9]. This injury will also affect the myotomal distribution of the hip abductors, extensors, external rotators, knee flexors, peroneal, dorsiflexor, and plantar flexors [9]. Thus, understanding the spine and hip neuromuscular relationship is essential for thorough examination and successful treatment of hip pathology.

The neuromechanical layer can also explain lower extremity mechanics and the effect it has on the hip. There is an existent kinetic chain of adaptive reactions that can cause changing movement patterns from the ankle and foot to the hip [9]. These adaptive reactions can either occur from the foot to the pelvis or from the pelvis to the foot [9]. Past studies have shown evidence that neuromuscular patterns and reflexive relationships exist. For example, patients with chronic ankle sprains will demonstrate delayed activation of the gluteus maximus muscle during hip extension in a prone position [9]. The muscles of the gluteus maximus will have a change in the firing pattern at the hip (in hip extension) due to the sprained ankle [9]. Lower extremity pathology can significantly affect the arthrokinematics, muscle timing, and performance around the hip [9]. Other examples can be seen with external or internal foot rotations. Patients with a progressing external foot walking gait can indicate signs of trauma, effusion to the hip, femoral retroversion or FAI [1]. Patients with internal rotation of the foot can indicate increased femoral anteversion or acetabular retroversion pathology [1]. Treatment of the hip starts with addressing the kinetic chain in layer IV. It is important for physiatrists to examine the foot and how it hits the ground while patients are walking. The walking mechanics can affect the muscles (myotome patterns) of the hip and pelvis.

Furthermore, it is important and essential for physiatrists to communicate the correct pelvic girdle physical therapy prescriptions to the therapist while a patient is completing rehabilitation.

A "one for all" type of treatment approach regarding pelvic rehabilitation will likely fail [20]. Depending on the type of injury, different treatment approaches should be taken into consideration. When dealing with static overload type of injuries, rehabilitation should be focused on strengthening the hip abductors and extensors [20]. Exercises that should be included are squatting, lunging, and hip hinging with body weight. Optimization of the pelvic tilt should be completed in order for the acetabulum to cover the femoral head [20]. Patients should not participate in aggressive soft tissue exercises and avoid worsening pain in the soft tissue surrounding the stabilizers of the hip [20]. To avoid instability, exercises that focus on flexibility, such as barre and yoga, should be avoided as well [20]. As it pertains to patients that suffer from dynamic overload pathology, exercises that strengthen hip abductors, extensors, and flexors should be the focus of pelvic rehabilitation. The exercises should also include squats, lunges, and hip hinging but without aggressive end range stretching to avoid impingement syndrome. Physical therapists should allow slight variations of the foot and knee angles during these exercises to allow muscle strengthening optimization [11]. Pelvic tilt should be optimized to avoid hip impingement.

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