Degenerative Spine Joint Disease

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Key Points

- Facet joints, also known as zygapophysial joint, are formed from the articulation of the inferior articular process of one vertebra with the superior articular process of the adjacent caudal vertebra. Facet joints are crucial in stabilizing the spine and guiding flexion, extension, and rotation.
- Intervertebral motion can be isolated to the two vertebral bodies and the three-joint complex, consisting of the disc anteriorly and the two facet joints posteriorly. Degenerative change in disc can accelerate the degenerative changes in the facets.
- Spondylosis is defined as age- and stress-related degenerative changes that occur within the articular components of the spine.
- Because of the pars interarticularis' vulnerable position, stress forces make the area prone to fracture, and the vertebral body is then more inclined to forward slippage eventually leading to spondylolisthesis.
- Hyperkyphosis is a fixed and exaggerated convex anterior-posterior curvature of the thoracic spine that develops from age-related muscle weakening, degenerative disc disease, vertebral fractures, and genetic predisposition.
- While arthropathy of the sacroiliac joint can occur in isolation, sacroiliac joint dysfunction occurs more commonly in association with other degenerative syndromes, such as degenerative disc disease, spinal stenosis, and facet syndrome. Spinal fusion and laminectomy may be a significant predisposing factor.
- Hip osteoarthritis describes degenerative and degradative changes of the cartilaginous structures that occur in the hip joint. Primary osteoarthritis of the hip occurs due to normal wear and tear of the cartilaginous structures of the weight-bearing joint, typically becoming symptomatic in adults over the age of 40. Secondary hip osteoarthritis is caused by congenital or developmental etiologies.

Normal Anatomy and Function

Vertebral Column

The spine is composed of 7 cervical (levels C1–C7), 12 thoracic (levels T1–T12), 5 lumbar (levels L1–L5), 5 fused sacral (levels S1–S5), and 4 fused coccygeal segments [1]. The morphology of the first two cervical segments is the most distinctive and specialized to allow for movements of the skull, whereas the other segments are more consistent in appearance.

The C1 vertebra, also known as the atlas, is a ring-like structure, composed of the anterior arch, the posterior arch, and paired lateral masses, each articulating superiorly with the occipital condyles of the skull and inferiorly with the vertebral body of C2. C1 is unique in that it lacks a vertebral body. The C1–C2 junction is unique for the absence of an intervertebral disc. Instead, a bony isthmus, known as the dens or the odontoid process, originates at C2 projecting upward into the anterior arch of C1, thereby forming a bony articulation which confers substantial dynamic stability to the upper cervical spine (Fig. 13.1).

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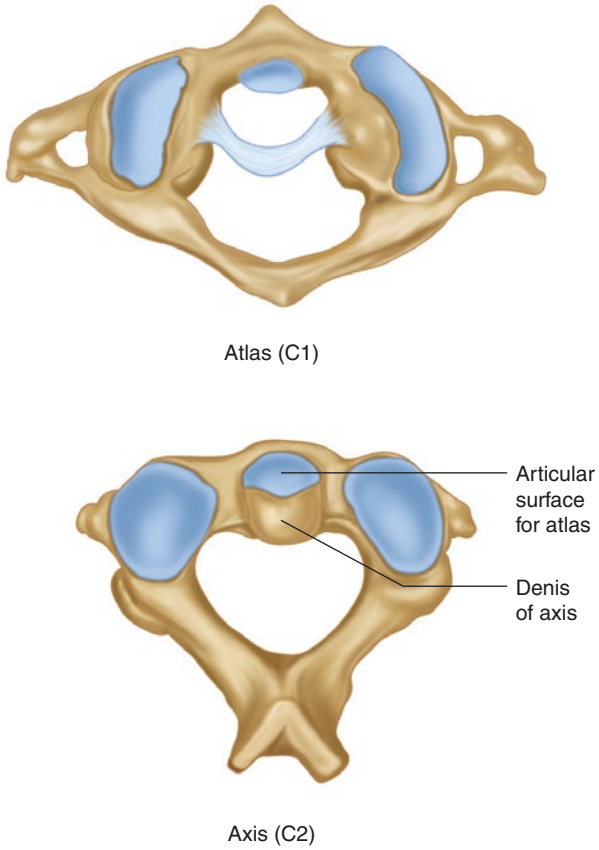

Fig. 13.1 Atlas (C1) and axis (C2): superior views. The denis of the axis projects upward into the atlas, forming a bony articulation

With the exception of C1 and C2, the remainder of the cervical, thoracic, and lumbar segments consist of vertebral bodies separated by intervertebral discs. Each vertebra consists of a vertebral body, the primary weight-bearing structure, and a bony arch, which projects dorsally from the vertebral body, forming the spinal canal where the spinal cord and spinal nerves travel [1]. Two short, thick, flanking processes known as pedicles originate from the vertebral body on either side, projecting dorsally and fusing with two broad bony plates known as lamina. The two laminae extend further posteromedially to the midline where they fuse and give off the spinous process (Fig. 13.2, top). The sliver of bone intervening between the ipsilateral pedicle and lamina is known as the pars interarticularis (Fig. 13.2, bottom). The pars interarticularis serves as the origin for the superior articular process, the inferior articular process, and the transverse process, thus producing four articular processes and two transverse processes for each segment. The three spinal processes, two transverse and one spinous, serve origins and insertions for muscles and ligaments, thereby anchoring and guiding spinal movement. The articular processes, which project superiorly and inferiorly, articulating with one another to form the zygapophysial or facet joints, confer sites to mechanically guide coordinated spinal movement, to bear roughly one-third of the axial load, and to resist anterior shear stress.

Joints of the Spine

Specific types of synovial joints exist from the skull base to the lumbo-pelvic junction, including atlanto-occipital, atlantoaxial, uncovertebral, and zygapophysial (facet) joints.

The atlanto-occipital joint is formed by the articulation between the occipital condyles of the cranium and the superior articular processes of the C1. The atlantoaxial joint is formed by the articulation between the posterior surface of the anterior arch of the atlas with the dens and the articulation of the lateral masses of C1 with the superior articular surface of C2 (Fig. 13.3). Together movement at these joints accounts for roughly 50% of total cervical rotation in addition to slight flexion, extension, and lateral flexion.

Uncovertebral joints, also known as joints of Luschka, are formed by the articulation between the uncinate processes, which arise from the posterior and lateral margins of the superior end plates of C3–C7, with the unci of the superior vertebrae. The joints of Luschka guide and permit flexion and extension of the cervical spine while simultaneously restricting lateral flexion.

The zygapophysial or facet joints are formed by the synovial articulation between the inferior articular and superior articular processes of the adjacent vertebrae [2] (see Fig. 13.2). Biomechanically, facet joints work in pairs to constrain the motion of the vertebrae while aiding the transmission of spinal loads [3]. In the cervical spine, the planes of the facet joints slope downward from an antero-superior to a postero-inferior position, thereby creating a plane of movement that facilitates rotation and extension while restricting lateral flexion and resisting shear stress in a coronal plane and bearing a minority of axial load [4]. Clinically this configuration guides one's ability to turn the neck and look up. In the thoracic spine, the facets are similarly oriented but at a more acute angle [5]. Taken together with the attachment of ribs along the lateral aspects of the vertebrae, this configuration confers significant rotational freedom of motion while limiting movement in all other planes. Finally, in the lumbar spine the planes of the facets are oriented to limit rotational range of motion but to allow greater range of motion in forward flexion, lateral flexion, and extension.

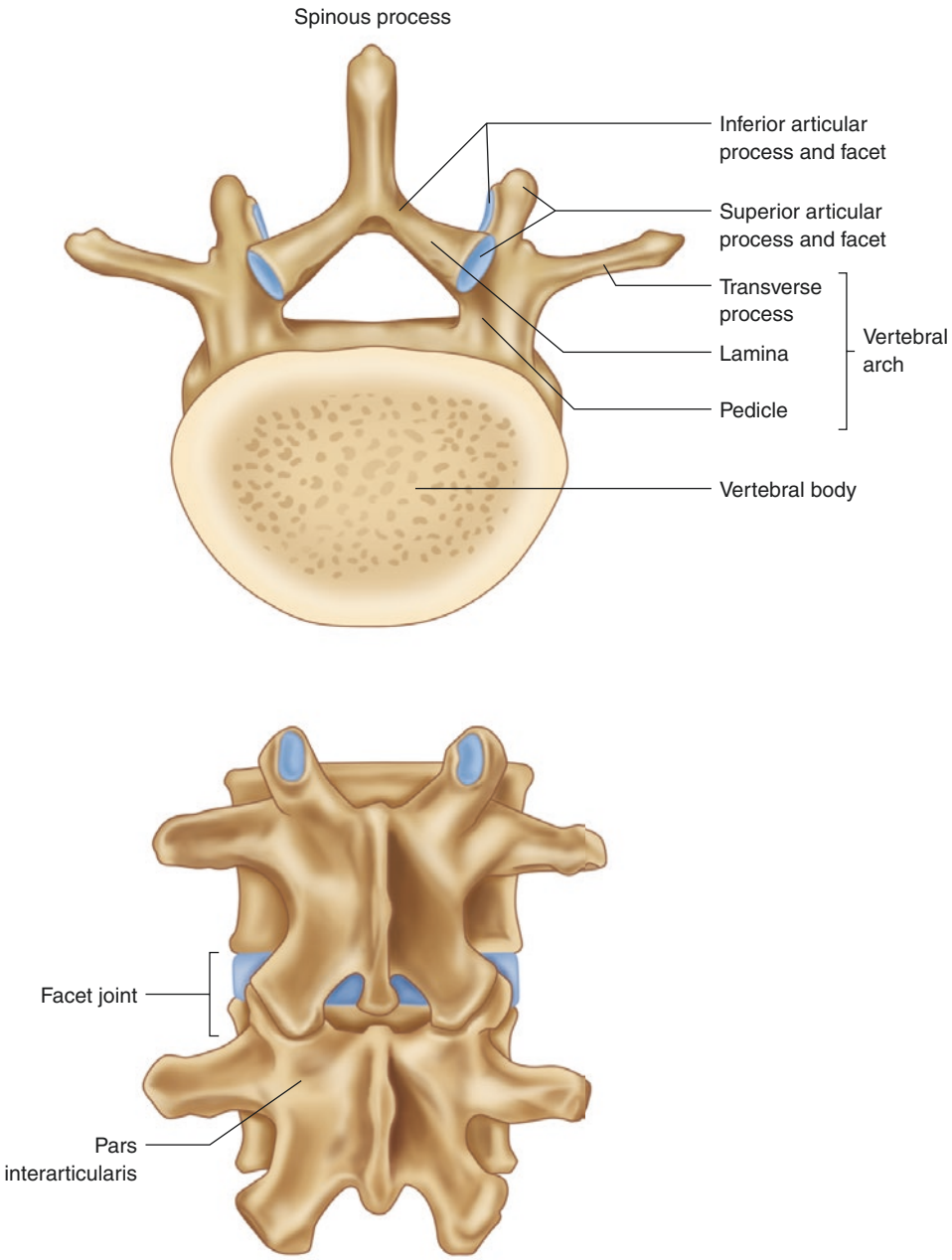

Fig. 13.2 Superior view of lumbar vertebral body (top). Labels include vertebral body, pedicle, lamina, transverse process, vertebral arch, superior articular process and facet, and inferior articular process and facet. Posterior view of two vertebrae (bottom) to better illustrate the pars interarticularis and the facet joint

Associated Joints: Sacroiliac Joints and Hip Joints

The bony pelvis is a large, relatively immobile ring-like basin designed primarily to bear weight of the body and transfer the load of the vertebral column laterally and inferiorly through the hip joints and into the lower limbs (Fig. 13.4). The pelvis is comprised of two innominate bones, joined anteriorly at the pubic symphysis and posteriorly to the sacrum at the sacroiliac joints [1]. Each innominate bone is comprised of three distinct bones which typically fuse by the end of puberty, the ilium, the ischium, and the pubis.

On the lateral aspect of the innominate, these three bones converge and fuse to form a cup-shaped concavity known as the acetabulum, which receives and articulates

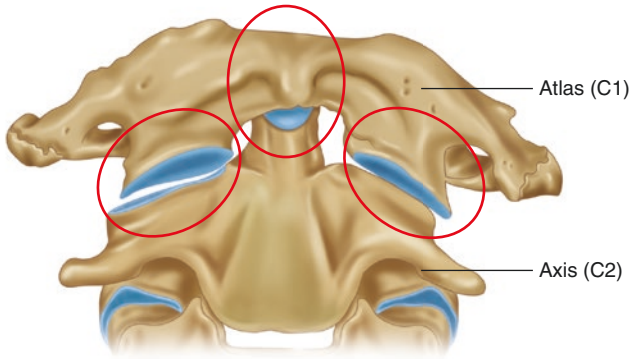

Fig. 13.3 Anterior view of atlas and axis. Circled components illustrate the atlantoaxial joint. The midline articulation is the dens of the axis projecting onto the atlas. The lateral articulations are that of the superior articular surface of the axis with the lateral masses of the atlas

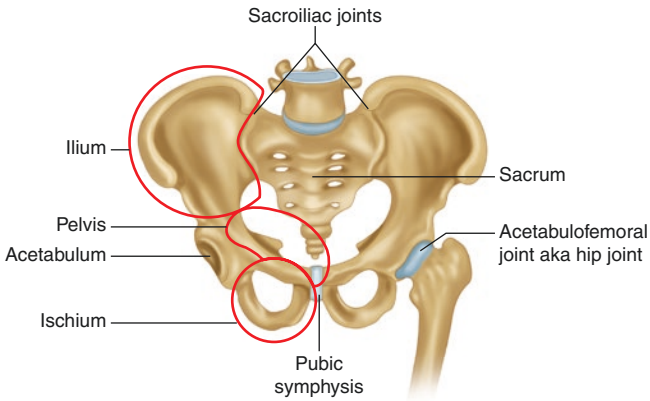

Fig. 13.4 Inferior view of a (male) bony pelvis to illustrate the sacroiliac joints, which is an articulation of the sacrum with the ilium, and the acetabulofemoral joint, which is an articulation of the acetabulum of the pelvis with the femur. Also illustrated are the three regions of the pelvis – ilium, pubis, and ischium – which are distinct regions that fuse by the end of puberty

with the head of the femur to form the acetabulofemoral joint, colloquially known as the hip joint. Each of the two sacroiliac joints (SI) is formed from the broad sinusoidal articulation occurring between the lateral border of the sacrum and the medial border of the ilium [6, 7]. Due to their role in bearing and dispersing axial spinal load, a sound understanding of both the sacroiliac and hip joints, biomechanically, in health, and in disease, is essential to a complete understanding of spinal degenerative disease and biomechanics.

Cascade of Degeneration and Osteophyte Formation

Intervertebral motion can be isolated to the two vertebral bodies and the three-joint complex, consisting of the disc anteriorly and the two facet joints posteriorly [8]. Degenerative change in any segment of the three-joint complex can influence degenerative changes in the other two [9–11]. Degeneration of the disc typically progresses to a subsequent loss of disc height. Lack of sufficient disc height can overwhelm the facet joints during axial loading and leads to inward bowing of the annulus and ligament's flavor, which will subsequently lead to neural foramina stenosis. The degenerated disc also allows for increased micro-axial rotation during axial loading and, consequently, exacerbating mechanical stress upon the longitudinal ligaments outermost annulus fibrosis [12–14]. Osteoblasts at the attachment sites on the margins of the vertebral bodies and annulus fibrosis are stimulated to form osteophytes. Disc-osteophyte formation further reduces spinal range of motion; posterior osteophytes can also contribute to central spinal stenosis.

Facet Arthropathy

Overview

Facet arthropathy refers to any acquired, degenerative, or traumatic process that affects the facet joints, often resulting in axial neck, mid-back, and low back pain as well as referred pain into the head as well as into the lower extremity [15–17]. Facet arthropathy can be a primary source of pain after whiplash injury or secondary to degenerative disease of the disc, vertebral compression fracture, or ligamentous injury. Facet arthropathy may result in pain due to the intrinsic nociception of the facet joints or its extrinsic compression of the lateral recess or neural foramen.

Normal Anatomy and Function

Facet joints, also known as zygapophysial joint, are formed from the articulation of the inferior articular process of one vertebra with the superior articular process of the adjacent caudal vertebra (see Fig. 13.2). The articular surface of the facet joints is covered by a layer of hyaline cartilage; the external joint is surrounded by a thin fibrous capsule and lined with a synovial membrane. Facet joints are crucial in stabilizing the spine and guiding flexion, extension, and rotation; when the spine is in extension, the facet joints bear a

significant portion of the weight of the spine [18, 19]. The facet joint ensures that the spinal column resists joint distraction, shear forces, and lateral or anterior-posterior translation and imparts sufficient torsional stiffness [8].

Nociceptive nerve endings are located in both the capsule and synovial membrane of the facet joints [20, 21]. Nociceptive signals from the facet joints are transmitted through the medial branch nerves, which also supply the motor innervation of the multifidus muscle as well as interspinous and supraspinous ligaments. Medial branch nerves originate from the posterior ramus which also divides into the lateral and intermediate branches, and in turn, the medial branch divides into two branches that supply the facet joint at the same level and the joint at the level below [21, 22].

The medial branch nerve courses over the medial posterior surface of the transverse process one level inferior to where it originates. Each lumbar facet joint is innervated by the medial branch of the nerve exiting at the same level as well as by the medial branch of the nerve one level above [23]. For example, at L4–L5 facet, which is the most common level for lumbar facet arthropathy, innervation of the inferior articular process of L4 is supplied by the medial branch nerve of L3, while the innervation of the superior articular process of L5 is supplied by the medial branch of L4. L5–S1 facet joint is unique in that it is innervated by the medial branch of L4 as well as the dorsal ramus of L5, which course along the junction of the S1 superior articular process and the sacral ala.

Pathology

Facet joints are weight-bearing structures and normally can carry up to one-third of the axial load. As described in the disc degeneration section, a cascade of degeneration can result when a degenerated disc can no longer contribute an adequate disc height. While it is challenging to determine the true sequence in the cascade, many researchers theorize that the disc degeneration usually precedes facet arthropathy since the degeneration in the disc is frequently accompanied by arthropathy of the associated facet joints, while arthropathy is minimal when the discs are relatively normal and disc degeneration also frequently occurs without facet arthropathy. With disc degeneration, studies suggest that it is the increased micro-axial rotation that results that places additional mechanical stress on facet joint [8]. The increased biomechanics load can lead to a molecular response, involving osteoarthrosis, osteophyte formation, and production of inflammatory cytokines within the cartilage and synovial membrane of the facet joints—the end result of which is hypertrophic and fibrocartilaginous changes of the facet joints [24, 25]. MRI findings may include hyperintensities of the bone marrow and periarticular soft tissue edema on T2 weighted imaging as well as widened facet joints with effusions [26]. The relationship between facet degeneration and back pain remains unclear. Establishing a clear relationship between joint degeneration and pain has been challenging, though in the past two decades, the trend is toward the conclusion that facet joints can be and often are a primary source for back pain [27]. Studies show that chemical or mechanical stimulation of the facet joints or the medial branches elicit concordant back pain [28–31], while local anesthetic blocks of the facet joints or the medial branches have been shown to relieve pain significantly in patients with chronic pain [32].

Spondylosis

Spondylosis is the term that describes age- and stress-related degenerative changes that occur within the articular components of the spine [24, 33, 34]. The degenerative changes affect all aspects and components of the spine, including the ligaments, discs, end plates, and bones. These degenerative changes result in spinal canal and nerve root narrowing. The general term for spondylosis is osteoarthritis of the spine. As with osteoarthritis that affects other areas of the body, it is a nonreversible, natural occurrence that is age and use related. Other degenerative changes that occur as a result of spondylosis are bony osteophytes and spurs [35]. These further contribute to degeneration and pathology by narrowing the spinal canal space, and the nerve roots exit. Thus, spondylosis is considered a mechanical hypertrophic response of adjacent vertebral bone to disc degeneration and includes facet joint osteoarthritis, degenerative disc disease from dehydrated discs, spinal canal stenosis, ligament and bony hypertrophy, or any other age-related degeneration of the spine [24].

Spondylolisthesis

Spondylolisthesis is the general term given to a slipped vertebra. Most often occurring in the lumbosacral region, it is broadly defined as anterior or posterior slipping of one vertebra over another [36]. Neugebauer was one of the first to describe the pathology of spondylolisthesis in 1888 as the separation of the posterior neural arch from the vertebral body. The bony defect, he noticed, was commonly encountered at the pars interarticularis which allowed for anterior displacement of the vertebral body, while the spinous process and inferior articulating surfaces remained aligned with

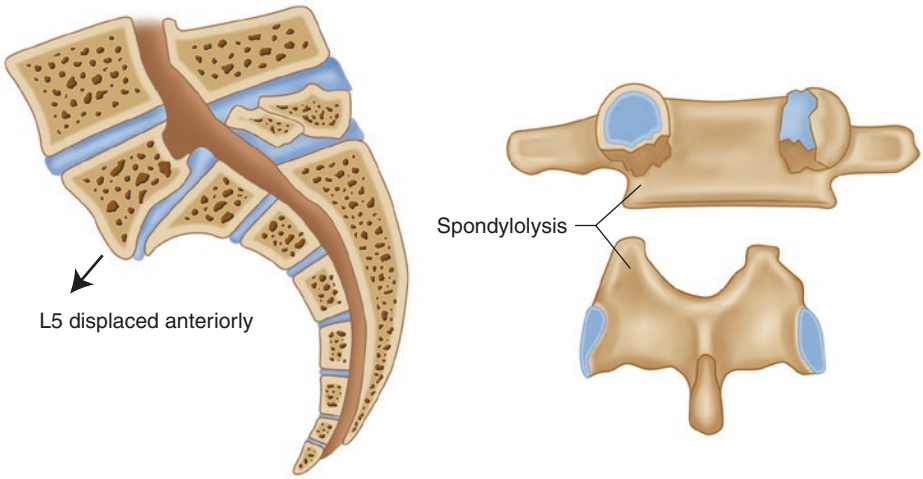

Fig. 13.5 Sagittal view illustrating spondylolisthesis, more specifically L5 anterolisthesis, in addition to posterior section illustrating spondylolysis, which is fracture of the pars interarticularis

the posterior sacrum [37]. The bony defect at the pars interarticularis would later be termed *spondylolysis* (Fig. 13.5). In the majority of cases, spondylolisthesis is an incidental finding on x-ray and is asymptomatic. The pars interarticularis appears to be a particularly vulnerable area in the spinal architecture that predisposes to the development of spondylolisthesis [38, 39].

Numerous studies have pointed to both a genetic predisposition as well as repetitive stress-related trauma to the pars as the leading cause of the development of spondylolisthesis [38, 39]. In 1957, Wiltse et al. described the presence of congenital bone abnormalities and defects of the pars interarticularis as a cause of spondylolysis which eventually progresses to spondylolisthesis [40].

Because of the pars interarticularis' vulnerable position, stress forces make the area prone to fracture. The structure thus weakened, and the vertebral body is then more inclined to forward slippage eventually leading to spondylolisthesis. The current classification of spondylolisthesis, made popular by Wiltse, accepts that the cause of the primary lesion is multifactorial [41, 42]. Type 1 spondylolisthesis is due to congenital dysplastic defects in the bony architecture of the vertebrae and occurs in approximately 20% of cases. A congenital deficiency of the superior sacral facet or the posterior neural arch of the fifth lumbar vertebra can allow for forward slippage of L5 over S1. Some argue that defects in the pars interarticularis are absent in this classification. Type 2 spondylolisthesis, also called isthmic spondylolisthesis, is the most common presentation of the defect occurring in approximately 50% of cases; traumatic or stress-related fracture in the pars interarticularis, both acute and chronic, results in the slippage. Type 3 spondylolisthesis occurs secondary to degenerative processes and is commonly found at L4/L5. The slippage occurs due to intersegmental instability and subsequent remodeling of the articular process. Type 4 often involves trauma in areas other than the pars resulting in slippage, and Type 5 involving pathologic causes like malignancy or other inherent bone abnormality is a very rare cause of spondylolisthesis.

Age-Related Hyperkyphosis

Overview

Hyperkyphosis is a fixed and exaggerated convex anterior-posterior curvature of the thoracic spine, also known as "postural roundback" or "dowager's hump." The presence of this deformity has been known to impair balance and increase risks of falls and injury. The condition develops from age-related muscle weakening, degenerative disc disease, vertebral fractures, genetic predisposition, or any combination of these. The prevalence of hyperkyphosis is 20–40% in both men and women over the age of 60 [43].

Normal Curvature

Developmentally, the lateral curvature of the spine is C-shaped and concave anteriorly. The thoracic and sacrococcygeal spine are referred to as primary curves as they remain kyphotic or concave anteriorly, whereas the cervical and lumbar curves are secondary curves as they change after birth [1]. Developmentally, lordosis of the cervical spine evolves from biomechanical changes that occur when a child begins to maintain an upright head posture. In contrast, lordosis of the lumbar spine evolves from the biomechanical changes elicited when a child begins to walk upright. The gold standard

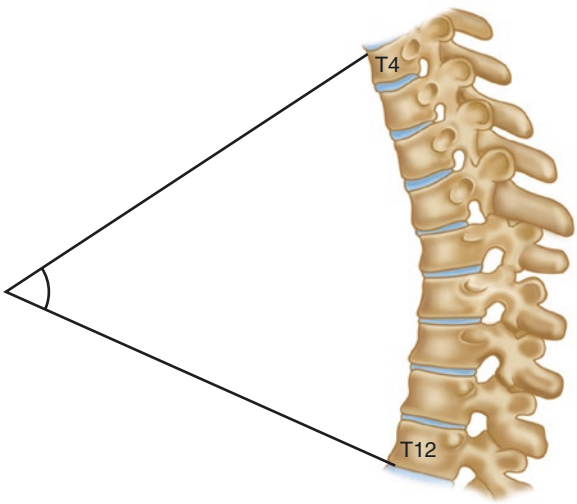

Fig. 13.6 Cobb angle can be calculated from the extension of the line from the superior end plate of T4 and the inferior end plate of T12. Cobb angle is one measure of the severity of the hyperkyphosis

for measuring kyphosis is with standing lateral radiographs from which the Cobb angle can be calculated from the interval of the thoracic curve (usually T4–T12) [44] (Fig. 13.6).

Pathology

A small amount of anterior curvature of the thoracic spine is normal. However, a kyphosis angle of greater than 40°, which is greater than the 95th percentile of normal, is considered hyperkyphosis. An increasing kyphotic angle is inversely correlated to quality of life and physical activity. Multiple musculoskeletal, neuromuscular, and sensory impairments are significant predictors of age-related hyperkyphosis [45, 46]. Stress loading on the aged, osteoporotic spine during daily activities can cause vertebral wedging and compression fractures. The severity of wedging and vertebral fractures correlates with decreased bone density and aging. A history of anterior wedge thoracic vertebral fractures is strongly correlated with hyperkyphosis [47].

Degenerative disc disease is another common finding seen on radiographs with patients with severe hyperkyphosis. There is significant correlation between anterior disc height and kyphotic angle. Some studies have shown hyperkyphosis in individuals without vertebral compression fractures, which supports the stronger correlation between degenerative disc disease [47]. With age-related extensor muscle weakness and the loss of inability to stand erect, the normal postural alignment is lost. Thus, others have postulated that hyperkyphosis is associated with spinal extensor muscle weakness [46, 48–50]. Additionally, the age-related calcification and ossification of anterior longitudinal ligament may also contribute to worsening hyperkyphosis. Finally, the aging population has loss of cerebellar function, vestibular, and proprioceptive feedback mechanisms. This may worsen already impaired erect vertebral alignment and serve to worsen hyperkyphosis [45, 50].

Sacroiliac Joint Dysfunction

Overview

The sacroiliac joint is the connection of the spine to the pelvis and therefore a significant region for transmission of weight from the trunk to the lower extremities. Sacroiliac joint dysfunction is estimated to be a generator of low back pain approximately 15–30% of the time [51, 52]. Predisposing factors for sacroiliac joint pain include leg length discrepancy, age, and previous spine surgery. With aging, the capsular surface of the ilium becomes coated with more fibrous plaques, and motion at the sacroiliac joint becomes noticeably restricted by the sixth decade. Erosions of the sacroiliac joint may be present by the eighth decade [53].

Normal Anatomy and Function

The sacroiliac joint is an ear-shaped articulation of the sacral segments S1–S3 with the ilium with only the inferior one-half to two-thirds of the joint to be considered truly synovial, while the superior aspect is more ligamentous (Fig. 13.7).

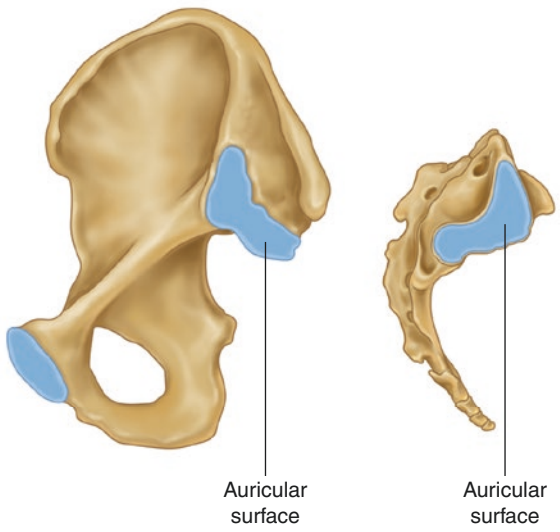

Fig. 13.7 The sacroiliac joint illustrated from the medial ilium view (on the left) and the lateral sacral view (on the right). The joint surface is often called the auricular surface as it is ear shaped

The sacral surface of the joint is made of hyaline cartilage, whereas the iliac surface of the joint is made of fibrocartilage—these surfaces are made of convoluted, interlocking grooves and ridges, which add to the stability of the joint against vertical and anterior shearing [54–56]. The intricate ligamentous system surrounding the sacroiliac joint further enhances the strength of the joint and functions to limit the amount of motion available to the sacrum and coccyx with respect to the ilium [57]. Due to the stability of the joint and surrounding ligaments, the sacroiliac joint is only capable of slight movement, such as flexion and extension of the sacrum, which are referred to as nutation and counternutation, respectively [58].

Innervation of the sacroiliac joint and adjacent ligaments is variable and may explain the different patterns of referred pain between individuals [59–61]. The anterior portion of the sacroiliac joint receives innervation from the lateral branches of dorsal rami of L2–S2, while the posterior portion of the joint receives innervation from L4 to S3. Effective reduction of sacroiliac joint pain has been achieved in multiple studies with lesioning of the L5 dorsal ramus in addition to the lateral branches of the dorsal sacral rami from S1 to S3 [62].

Pathology

Biomechanically, because there are intimate connections between the surrounding ligaments of sacroiliac joint with the biceps femoris, piriformis, and gluteus maximus, any imbalance of the muscle dynamics could lead to abnormal sacroiliac joint mobility, leading to mechanical stress and accelerated degeneration [63]. While arthropathy of the sacroiliac joint can occur in isolation, sacroiliac joint dysfunction occurs more commonly in association with other degenerative syndromes, such as degenerative disc disease, spinal stenosis, and facet syndrome [64]. Spinal fusion and laminectomy may be a significant predisposing factor as the altered spinal mechanics can subject the sacroiliac joints to increased mechanical load [65]. Pathologic changes, more commonly found in individuals over 50 years of age, include cartilage erosion, denudation of the joint surface, and osteophytic formation, in addition to para-articular and intra-articular fibrosis ankylosis—these can all lead to the gradual obliteration of the joint space until the sacrum and ilium are completely apposed [66].

Hip Joint Disorders

Overview

The hip is a ball and socket synovial joint. It consists of articulation between the head of the femur and acetabulum of the pelvis. It serves as a connection between the spine and the lower extremities, providing stability and dynamic support of the body by distributing axial load evenly to the lower extremities. The hip joint is subject to extreme forces, facilitating movement in three axes, all perpendicular to each other and centered around the femoral head [2].

Normal Anatomy and Function

The stability of the hip is maintained by the activity of the ligaments, muscles, and tendons working in tandem [67, 68] (Fig. 13.8). The "socket" of the hip joint consists of tight articulation of three bones: the pubis medially, the ischium inferiorly, and the ilium superiorly. At the junction of these three bones is the triradiate cartilage which fuses by the age of 16. The acetabular notch is the only part of the acetabulum that does not cover the femoral head. The transverse acetabular ligament runs inferiorly to the acetabular notch, creating a portion of the acetabular labrum, completing the ring of the joint, and helping to stabilize the hip joint by preventing inferior displacement of the femoral head [67, 68].

The femoral head is the most proximal part of the femur and forms the "ball" of the joint (see Fig. 13.8). Its articulating surface is lined with a durable layer of hyaline cartilage. A small depression in the head of the femur, called the fovea capitis femoris, houses the attachment of the round ligament. The femoral neck lies at an angle of approximately 130° to the axis of the femoral shaft. The greater and lesser trochanter of the femur serves as attachments for the hip joint's muscular stabilizers.

The acetabular labrum is a fibrocartilage inserted on the base of the acetabular edge. It blends in with the transverse acetabular ligament and helps increase the overall depth and stability of the hip joint. Additionally, the labrum helps to keep the synovial fluid within the joint capsule. As mentioned above, the femoral head, as well as the entire acetabular articulating surface, is covered by durable hyaline cartilage. Its main function is to serve as a shock absorber to help dissipate weight during weight-bearing activities, decrease friction, and allow free motion of the joint.

The articular capsule is a fibrous sheath which encases the hip joint from the acetabulum to the femoral neck. There are three peripheral thickenings of the capsule, named for their pelvic attachments, which form three important extracapsular ligaments. The *iliofemoral ligament* is the strongest ligament in the body and originates below the anterior inferior iliac spine and inserts on to the femur at the greater trochanter and onto the intertrochanteric line. It reinforces the anterior aspect of the joint capsule and serves to restrict overextension and external rotation of the hip joint. The *pubofemoral ligament* originates from the pubic portion of the acetabular edge and from the ilio-pectineal eminence and

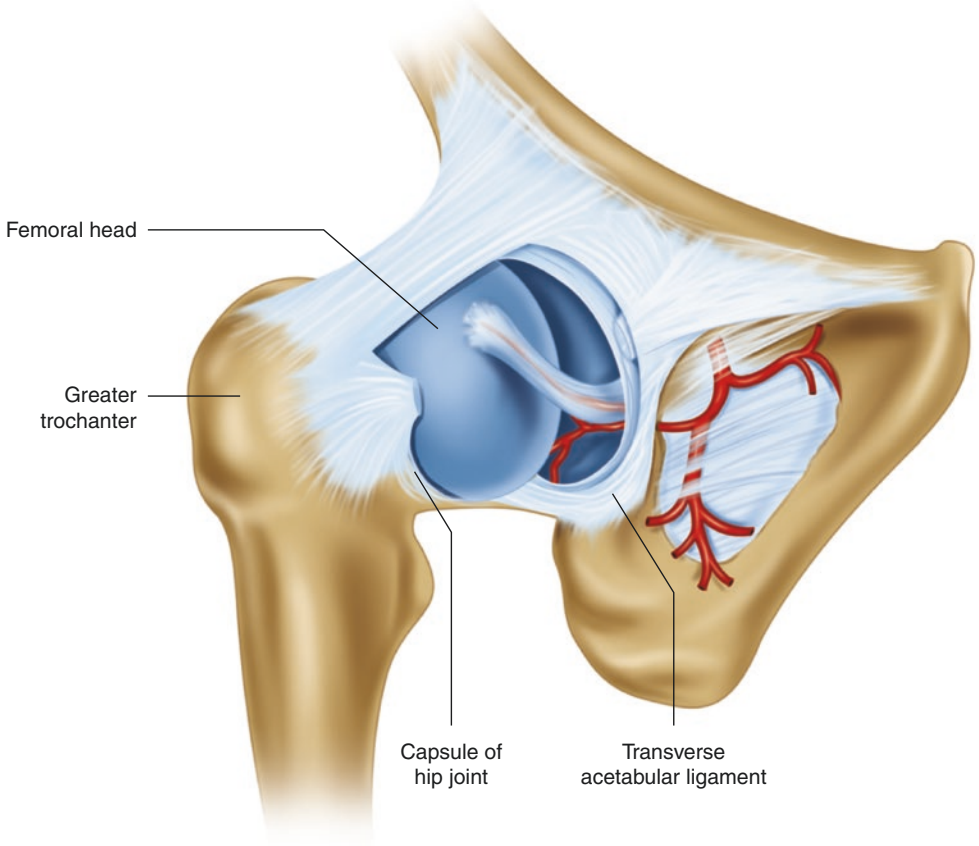

Fig. 13.8 Anterior view of the acetabular fossa and ligament of the femoral head

blends with the joint capsule. It serves to restrict movement in abduction. The *ischiofemoral* ligament spans from the ischium below and behind the acetabular edge and attach to the intertrochanteric line. It serves to restrict movement in internal rotation.

The *ligamentum teres femoris*, also known as ligament of the head of the femur, round ligament, or foveal ligament, is located intracapsular and attaches the apex of the notch to the fovea of the femoral head. The base of the ligament is attached by two bands that each attach to the acetabular notch and blend with the transverse ligament. The ligamentum teres was initially thought to be an embryological remnant. However, it is now accepted that the ligamentum teres is integral to the hip joint as it serves as a carrier for the foveal artery (posterior division of the obturator artery) which supplies the femoral head. Injuries to the ligamentum teres can occur in dislocations, which can cause injury to the foveal artery, resulting in osteonecrosis of the femoral head. Congenital absence of the ligamentum teres is a classic feature in patients with hip dysplasia.

The hip houses attachments of many muscles and muscle groups. They are arranged anteriorly, medially, laterally, and posteriorly around the hip joint. Anteriorly, the rectus femoris, sartorius, iliopsoas, and pectineus are the primary flexors of the hip. The adductors longus, brevis, and magnus form the medial group responsible for adduction of the hip. The gracilis muscle is often included in this group as well. Posteriorly are the semimembranosus, semitendinosus, and the biceps femoris. Laterally, the muscles are gluteus minimus, medius, and maximus, as well as the tensor fascia lata which serve to abduct the hip.

The bursae involved in hip articulation, the iliopsoas bursa and the peritrochanteric bursae, have a particularly important role from a clinical point of view. The iliopsoas bursa, the largest bursa in the human body, is bounded by the iliopsoas muscle and tendon anteriorly, the capsule of the hip posteriorly, and femoral vascular structures medially. The peritrochanteric bursae located in the subgluteus maximus region of the lateral thigh include multiple distinct bursal components. Inflammation of these bursae presents as lateral hip pain, often confused with lumbar radiculopathy.

Adhesive Hip Capsulitis

Primary or idiopathic adhesive hip capsulitis, "capsular constriction" or "frozen hip," is characterized by a painful limitation in active and passive hip motion, usually external

rotation and abduction, without known trauma or pathology. The disease usually affects middle-aged individuals without sexual predominance. Radiographic imaging usually discerns only mild degenerative changes. Adhesive hip capsulitis is thought to be the result of synovial inflammation that progresses to capsular fibrosis [69].

The incidence of the disease is rare and is usually diagnosed after careful consideration of other more common etiologies such as osteoarthritis and hip impingement syndromes. The most convincing physical evidence of adhesive hip capsulitis is capsular fibrosis on arthroscopy. Rodeo et al. biopsied surgical samples of 19 patients with adhesive capsulitis of the shoulder and concluded that while synovial hyperplasia and capsular fibrosis played a pivotal role in the pathology of adhesive capsulitis, cytokines were also involved in the inflammatory and fibrotic process of the disease as well. These cytokines provide a persistent stimulus that results in fibrosis of the capsule. Many believe that the same mechanism applies to capsulitis of the hip joint.

Hip Osteoarthritis

Osteoarthritis is the prevailing joint disorder in the United States, and over 200,000 hip arthroplasties are performed annually in an effort to combat this disease. Both primary and secondary causes of hip osteoarthritis have been described. Primary osteoarthritis of the hip occurs due to normal wear and tear of the cartilaginous structures of the weight-bearing joint, typically becoming symptomatic in adults over the age of 40. Secondary hip osteoarthritis is caused by congenital or developmental etiologies.

Hip osteoarthritis is the term used to describe degenerative and degradative changes of the cartilaginous structures that occur in the hip joint in a nonuniform manner [70–73]. As the lubricating surfaces of the joint capsule begin to deteriorate and the bony structures closely appose each other, the body compensates by forming bone spurs and osteophytes which further impedes motion and further causes painful degeneration. Mild to moderate synovial fluid inflammatory changes also occur at this time as well as thickening of the synovium and ligaments.

Risk factors of primary osteoarthritis of the hip include age, genetic disposition, high BMI, participation in weight-bearing activities, and occupations that require prolonged standing, lifting, or moving heavy objections. Risk factors for secondary osteoarthritis include hemochromatosis, hyperthyroidism, hypothyroidism, acromegaly, connective tissue disorders, Paget's disease, and gout, among others [70–73]. Some studies suggest that hip osteoarthritis is more prevalent in females because a reduction in hormones, particularly a decrease in estrogen levels, worsens the progression of the disease.

Spine-Hip Syndrome and Hip-Spine Syndrome

The spine and the hip joints are intimately related due to kinematics; pathologies in the spine can lead to compensatory changes in the hip joint, leading to hip pathology and vice versa. In spine-hip syndrome, the aging of the spine due to degenerative disc disease and osteoporotic vertebral collapse can lead to progressive loss of lumbar lordosis and increased pelvic retroversion; with time, the patient will become sagittally imbalanced with under-coverage of the femoral head anteriorly therefore developing increased risk for hip osteoarthritis. In the hip-spine syndrome, the osteoarthritic hip joint becomes stiffened and immobile due to osteophytosis; in reaction, the spine will compensate by increasing the lumbar lordosis so that the individual can stand upright – overtime, this compensation can lead to pathologic degeneration of the spine [74].

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