Spinal Anatomy, Mobility, Balance, and Deformity

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Abstract: The spinal column is formed of vertebrae and intervertebral discs. The vertebral column contains the spinal cord, nerve roots, dura, and blood vessels within the vertebral canal. In brief, the spine allows for flexion, extension, lateral flexion, and rotatory movement. Human mobility and posture importantly depend on the structural integrity and homeostasis of spinal balances (particularly sagittal and coronal balances). The derangement of structural integrity and the alteration of spinal balance homeostasis lead to spinal deformities like kyphosis, scoliosis, and kyphoscoliosis. This chapter will shortly discuss spinal anatomy, mobility, spinal balance, and spinal deformity. The principles of the management of common spinal deformities such as kyphosis and scoliosis will also be discussed.

Abbreviations

ALV	apical lumbar vertebrae	ASD	adult spinal deformity
CSVL	central sacral vertical line	CT	computed tomography
DDD	degenerative disc disease	FDA	Food and Drug Administration
MRI	magnetic resonance imaging	MT	main thoracic
PT	proximal thoracic	TL/L	thoracolumbar/lumbar
SVA	sagittal vertical axis	TDR	total disc replacement

1. Anatomy of the Spine

1.1. History

Before proceeding to understand the concept of modern spine surgery, knowledge on the anatomy and physiology of the spine needs to be acquired. Herophilus of Chalcedon (300 B.C.), known as the father of anatomy, as well as Galen of Pergamon (130–200 A.D.) were early pioneers who made observations on the nervous system and the spine. Following his 1543 publication of then most up-to-date anatomical textbook *De Humani Corporis Fabrica Libri Septi*, Andreas Vesalius (1514–1564) became regarded as the founder of modern spinal anatomy (Acar et al. 2005).

1.2. Basic Concepts

Vertebrates are an animal subphylum in the phylum Chordata and are defined by their possessing a vertebral column, or spine, which contains vertebrae. In humans, the spine is made up of thirty-three vertebrae, which are divided as follows: seven cervical, twelve thoracic, five lumbar, five sacral, and four coccygeal. The spinal column runs from the occiput to the coccyx. The neurological system and physical structure supported by the vertebral column also make possible correct movement and sensation. Spinal pathology can have a crippling effect on one's quality of life. The axial skeletal system is composed of the vertebrae, the skull, the ribs, and the sternum.

The vertebrae vary in terms of shape and size. This is more applicable to different regions of the spinal column, though they share a similar basic structure.

The vertebral body is the weight-bearing component positioned anteriorly of each vertebra. The superior and the inferior aspects of the vertebral body, lined with hyaline cartilage, are known as superior and inferior endplates. The posterior part is the vertebral arch, with several bony prominences which act as attachment sites for muscles and ligaments. Each vertebra has a single centrally placed spinous process, posteriorly at the point of the arch. On both sides, the transverse processes, which articulate with the ribs, extend laterally and posteriorly. Pedicles are bony structures that connect the vertebral body to the transverse processes. The lamina connects the transverse processes to the spinous processes (Figure 1a,b).

Intervertebral discs are short, cylindrical fibrocartilaginous structures between the vertebrae. They are wedge-shaped in the thoracic and lumbar regions to support the spinal curvature and absorb jolts. Each disc has two parts—a tough fibrous annulus fibrosus and a jelly-like nucleus pulposus, which it surrounds. Disc herniation occurs when the nucleus pulposus herniates in the posterolateral direction, breaking through the annulus fibrosus.

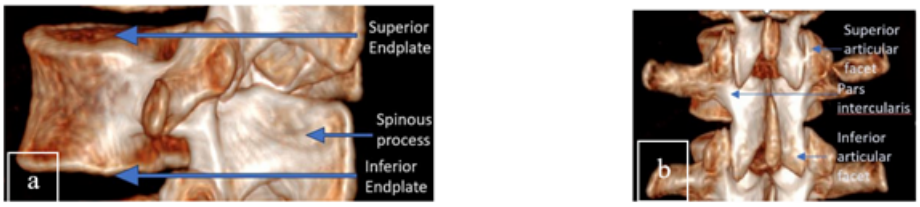

Figure 1. Lateral (**a**) and posterior (**b**) view of lumbar vertebra showing different parts. Source: Figure by authors.

1.2.1. Classification of the Vertebrae

Cervical Vertebrae

There are seven cervical vertebrae. They have three features that distinguish them from other vertebrae (Figure 2a,b):

1. The spinous process is bifid at its distal end, except in the atlas (C1), which has no spinous process, and C7, which has the longest spinous process among the cervical vertebra and may not be bifid.
2. In the first six cervical vertebrae, the vertebral artery and vein run in the transverse foramen. In the seventh, only the vertebral vein occupies the transverse foramen (also known as the foramen transversarium). The vertebral artery is especially significant due to it supplying oxygenated blood to the brain and spinal cord.
3. Cervical vertebrae have a triangular vertebral foramen, except for the atlas (C1) and axis (C2), as those are specialized to allow for the movement of the head.

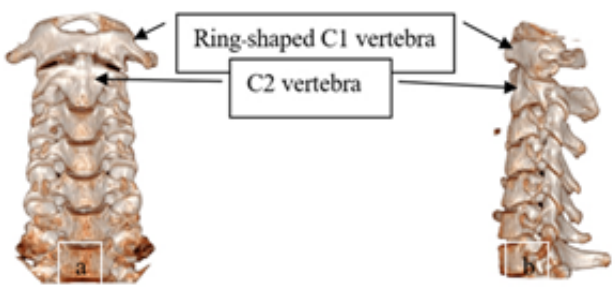

Figure 2. Anterior (**a**) and lateral (**b**) view of cervical spine (3D view). Source: Figure by authors.

Thoracic Vertebrae

The twelve thoracic vertebrae are medium-sized and increase in size as they go down. To produce the bony thorax, there are demifacets in the upper and lower half of each side of the vertebral body. Those demifacets articulate with the heads of two different ribs (Figure 3a,b).

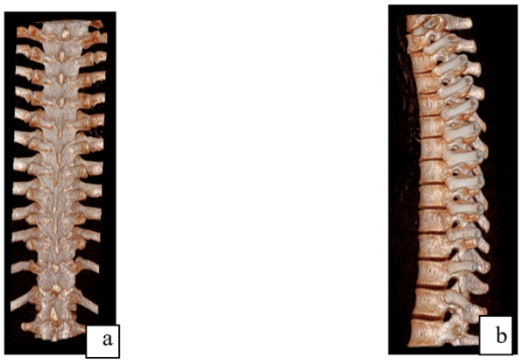

Figure 3. Posterior (**a**) and lateral (**b**) view of the thoracic spine along with the L1 vertebra, showing the position and attachment of the ribs and the anatomy of the facets, joints, and spinous processes of the thoracic vertebra (3D view). Source: Figure by authors.

The spinous processes of the thoracic vertebrae are positioned obliquely inferiorly and posteriorly. The vertebral foramen of the thoracic vertebrae is circular, in contrast to that of the cervical vertebrae.

Lumbar Vertebrae

The largest vertebrae in the spinal column are the lumbar vertebrae, and there are five of them in most individuals (Figure 4a,b). They have a specialized structural design to bear the weight of the torso.

The kidney-shaped vertebral bodies of the lumbar vertebrae are very big. They share a triangular-shaped vertebral foramen with the cervical vertebrae. Compared to the thoracic vertebrae, their spinous processes are shorter and do not reach inferiorly beyond the level of the vertebral body.

Because of their shape and size, needles can enter the spinal cord and spinal canal in this region of the spine, which is not feasible between the thoracic vertebrae. A lumbar puncture and the injection of epidural anaesthesia are two examples of this.

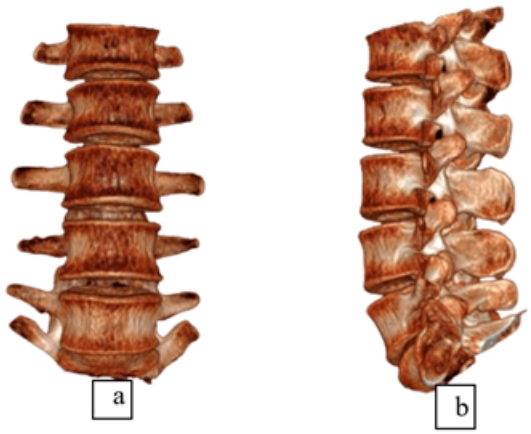

Figure 4. Anterior (**a**) and lateral (**b**) view of lumbar spine (3D view). Source: Figure by authors.

Sacrum and Coccyx

There are five fused vertebrae that make up the sacrum. With the apex pointing downwards, it resembles an inverted triangle. There are facets for articulation with the pelvis at the sacroiliac joints on the lateral sides of the sacrum.

A little bone called the coccyx articulates with the sacrum's apex. It can be identified by the absence of vertebral arches. There is no spinal canal because there are no vertebral arches.

"Sacralization" refers to the fusion of L5 with the sacrum, whereas "lumbarization" describes the separation of S1 from the sacrum. These are developmental in nature.

Ligaments and Joints

The articular facets and joints connecting the bodies of the mobile vertebrae allow them to articulate with one another.

Left and right superior articular facets articulate with the upper vertebrae, while left and right inferior articular facets articulate with the lower vertebrae. Through intervertebral discs, vertebral bodies indirectly articulate with one another. The cartilaginous joints that make up the vertebral body are designed for weight bearing. Intervertebral discs connect the articular surfaces, which are covered by hyaline cartilage.

The anterior and posterior longitudinal ligaments, which extend the whole length of the vertebral column, are two ligaments that support the vertebral body joints. Because of its thickness, the anterior longitudinal ligament keeps the spinal column from overextending. Even though the posterior longitudinal ligament is weaker, it inhibits hyperflexion.

The joints between the articular facets are called facet joints. They allow for some gliding motions between the vertebrae. They are strengthened by several ligaments:

- Ligamentum flavum—extends between the lamina of adjacent vertebrae;
- Interspinous and supraspinous ligaments—join the spinous processes of adjacent vertebrae. Interspinous ligaments attach between processes, and supraspinous ligaments attach to the tips;
- Intertransverse ligaments—extend between transverse processes.

The spinal cord is approximately 45cm long and originates from the medulla. It descends down through the foramen magnum and ends as the conus medullaris, which terminates at S2 in foetuses, L3 in newborns,

and at the lower border of L1 in adults. There are two enlargements: cervical C5–T1 for the brachial plexus and lumbosacral L2–S3 for the lumbar and sacral plexuses. The meningeal coverings of the cord are continuous with the brain, and the cord is closely ensheathed by pia mater. The pia continues inferiorly as filum terminale, piercing the distal extremity of the dural sac and attaching to the coccyx. Pia mater forms the denticulate ligaments which secure the cord within the dural sac (Bican et al. 2013). The dura forms a tough sheath and ends distally at S2, anchored to the coccyx by the filum terminale. The space between the arachnoid and pia mater contains CSF. All layers continue along spinal nerve roots. The anterior spinal artery is made up of two branches, one from each vertebral artery (VA). Radicular arteries arise from different levels—C3 (VA), C6 (from the deep cervical artery, with branches from the costocervical trunk and from the left subclavian), C8 (from the costocervical trunk), and D4/D5 (from the intercostal artery). The artery of Adamkiewicz (arteria radicularis anterior magna), which needs special mention, arises between T9 and L2 in 85% and between T5 and T8 in 15% of the population. It is the principal source of blood for the spinal cord from the T8 segment to the conus and comes from the left side in 80% of cases (Frostell et al. 2016). Two paired posterior spinal arteries arise from the posterior inferior cerebellar artery (PICA), which is less prominent than the anterior, and receive additional supply from 10–23 radicular branches. The midthoracic part has poor blood circulation, with a radicular artery from T4/5. This is a "watershed zone" and is more susceptible to vascular insult. There is anatomical variation, as at the conus medullaris, the anterior spinal artery joins the paired posterior spinal arteries. Ascending and descending tracts of spinal cord are shown in Tables 1 and 2, respectively.

Table 1. Ascending tracts.

Tract	Function	Point of Decussation
Dorsal columns Fasciculus gracilis Fasciculus cuneatus	Joint position, vibration, light touch	Brainstem: lower medulla and medial lemniscus
Posterior spinocerebellar	Unconscious proprioception (stretch receptors)	Uncrossed
Anterior spinocerebellar	Unconscious proprioception (whole limb position)	Uncrossed
Lateral spinothalamic	Pain and temperature	Spinal cord: at level of entry across anterior white commissure
Anterior spinothalamic	Light touch	Spinal cord: 2–3 segments above point of entry

Source: Authors' compilation based on data from Diaz and Morales (2016).

Table 2. Anatomy: descending tracts (motor).

Tract	Function	Point of Decussation
Lateral corticospinal	Skilled movement	Medullary pyramids
Anterior corticospinal	Skilled movement	Uncrossed
Rubrospinal	Facilitates flexor muscle tone	Midbrain
Vestibulospinal	Facilitates extensor muscle tone	Uncrossed

Source: Authors' compilation based on data from Diaz and Morales (2016).

2. Concept of Balances

2.1. Spine Sagittal Balance

2.1.1. Introduction

Humans stand and move in an environment subject to gravity. Constraints are placed on the spine as a result. Due to the bipedal stance, the pelvis and spine have a close association. The spine and body function inside a cone of equilibrium to maintain sagittal as well as coronal alignment with little energy consumption. This involves lumbar lordosis, dorsal kyphosis, cervical lordosis, and the pelvic anatomy. The primary goal is to retain mechanical equilibrium in both the sagittal and coronal planes, with the centre of cranial load, heads of the femur, and inferior extremities as the focal points. Scoliosis and spinal deformity surgery in the past emphasized coronal plane alignment (Glassman et al. 2005; Schwab et al. 2010).

According to White and Panjabi (White and Panjabi 1990), spinal instability, both acute and chronic, points to substantial spinal displacement that would lead to neuro-deficit, pain. or deformity. Spinal stability is the capability to restrict patterns of deformation under physiologic stresses so as not to harm the spinal cord and nerve roots and, furthermore, to avoid pain or incapacitating deformity resulting from structural alteration.

2.1.2. Aetiology

The aetiologies of sagittal imbalance include congenital, degenerative, traumatic, and iatrogenic. The majority of individuals with spinal sagittal imbalance have a kyphotic or hypolordotic fusion mass, with degenerated segments below and above the fusion (Booth et al. 1999).

Spinal biomechanics can be altered in case of congenital scoliosis, which can involve underdeveloped vertebrae or fixed kyphotic segments (Figure 5). Surgical intervention by fusion of long segments for adolescent idiopathic scoliosis may result in spinal degeneration distally that will affect sagittal balance later on (Wang et al. 2008).

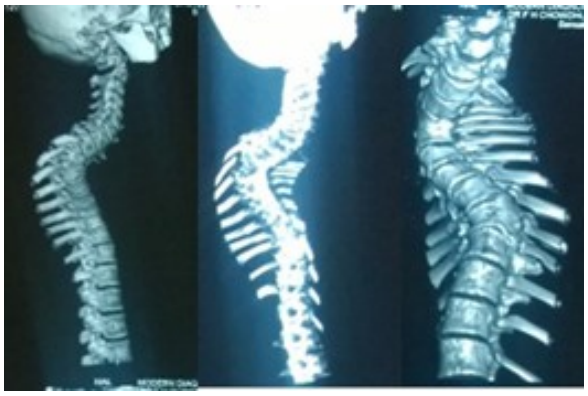

Figure 5. Three-dimensional CT scan of spine showing upper dorsal kyphoscoliosis due to D4 hemivertebra. Source: Figure by authors.

Spinal trauma, regardless of operative or nonoperative treatment, can result in chronic alterations in spinal balance, particularly when the thoracolumbar junction is involved. This phenomenon can be seen in disease conditions which cause degenerative or fixed alterations of the spine, like ankylosis spondylitis or rheumatoid arthritis (Bradford et al. 1987).

Because of the proximal lumbar hyperlordosis caused by thoracolumbar fusion, increased lordosis or kyphosis compensate for the rest of the lumbar and thoracic segments, respectively. The resultant compensating effect wears off over time, resulting in flat back syndrome and thoracic hyperkyphosis (Rose et al. 2009).

2.1.3. Epidemiology

Sagittal imbalance is a complicated problem that can arise from a variety of causes of spinal deformity. Table 3 shows the prevalence of sagittal imbalance based on a large cohort study (Glassman et al. 2005).

Table 3. Distribution of causes of sagittal imbalance (Figures 6–9).

Causes	Percentage (%)
Adolescent idiopathic scoliosis (Figures 6–9)	30.4
Kyphotic angulation	20.7
Iatrogenic imbalance and combined junctional degeneration	14
Congenital, neuromuscular, or scoliosis	25.5

Source: Authors' compilation based on data from Glassman et al. (2005).

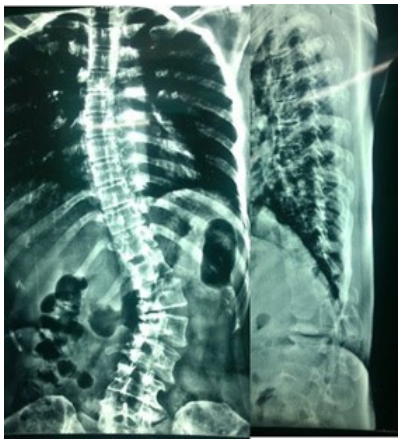

Figure 6. X-ray of dorsolumbar spine, with anterior–posterior and lateral views showing lumbar scoliosis. Source: Figure by authors.

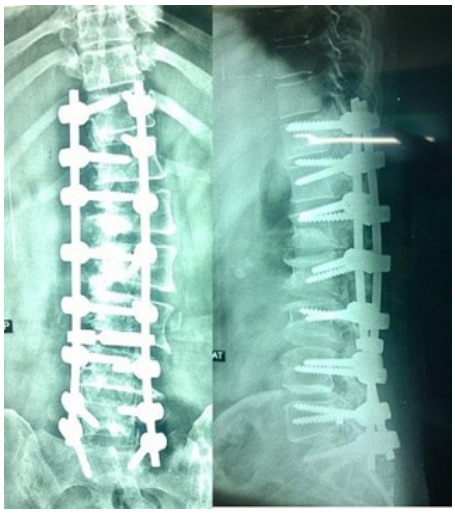

Figure 7. Postoperative X-rays of patient in Figure 6 showing correction of lumbar kyphosis with pedicular screws and rods. Source: Figure by authors.

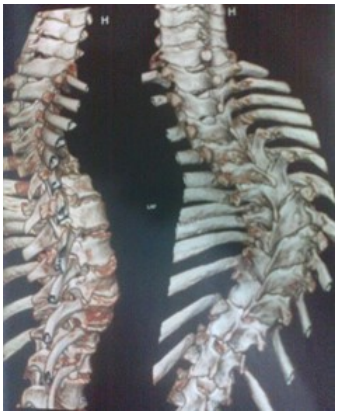

Figure 8. Three-dimensional CT scan of spine showing dorsal kyphosis. Source: Figure by authors.

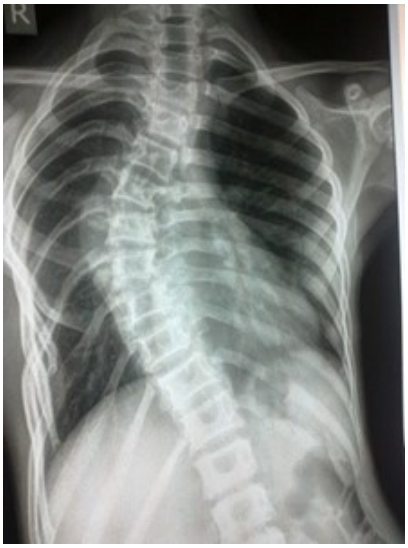

Figure 9. X-ray showing dorsal scoliosis with altered cardiac shadow in a patient with compromised cardiopulmonary function. Source: Figure by authors.

Angular measurement of spinal curves are shown in Table 4.

Table 4. Angular measurement of spinal curves.

Spinal Zone	Normal Curvature
Cervical lordosis	20°–40°
Dorsal kyphosis	20°–50° (can usually range 30° more than lumbar lordosis)
Thoracolumbar junction	D12/L1 junctional area should be neutral, with less than 10° or regional kyphosis
Lumbar lordosis	Mean 60°
Pelvic tilt	<20°
Pelvic incidence	55° ± 10°
Pelvic incidence/lumbar lordosis mismatch	<10°
Sagittal vertical axis	<5 mm

Source: Table by authors.

Sagittal Vertical Axis (SVA) and C7 Plumb Line

The C7 plumb line is the commonest method of determining spinal sagittal balance. An imaginary vertical line is drawn in a caudal direction from the centre of the vertebral C7 body, and it should intersect with or remain within 05 mm of the S1 posterior–superior endplate. For health-related quality of life outcomes, this is regarded to be within the acceptable range. Patients lean forwards and acquire slightly more positive sagittal alignment as they get older.

Preserving the harmonious anatomy of sagittal plane considerations is crucial. To preserve an approximately neutral posture, the cervical spine might compensate with a hyperlordotic presentation. Knee flexion as well as an increased pelvic tilt can help the patient compensate even more. However, this is a physically demanding and tiring position that is not anatomically typical (Schwab et al. 2010).

Pelvic Incidence

Pelvic incidence (PI) (Figure 10) is the angle created by a line drawn 90° to the surface of the sacrum's superior endplate as well as a line from the midpoint of the sacrum's superior endplate to the femoral head's centre. This measurement's usual range is 55° ± 10° (Rose et al. 2009).

PI is a roentgenographic comparison of the biomechanic interaction between the lumbar spine as well as the pelvis in individuals with different pelvic morphologies. An individual's lumbar lordosis (LL) and PI may change. However, to maintain sagittal balance in the lumbopelvic junction as well as SVA in the whole spine, their relationship must be maintained. Changes in pelvic and hip posture often compensate for fixed sagittal abnormalities in the lumbar spine, preserving SVA.

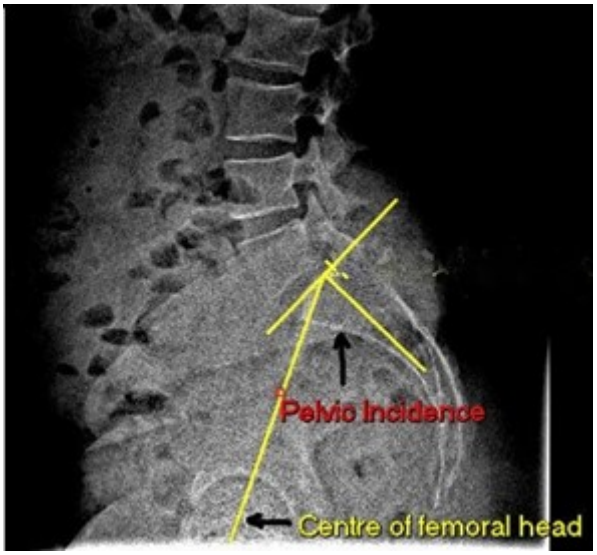

Figure 10. X-ray of lumbosacral spine (lateral view) demonstrating calculation of "pelvic incidence". Source: Figure by authors.

Pelvic Tilt

Pelvic tilt (Figure 11) is the angle between the following two roentgenographic lines:

- A line drawn from the centre of the S1 endplate to the centre of the femoral head;
- A vertical line intersecting the femoral head's centre.

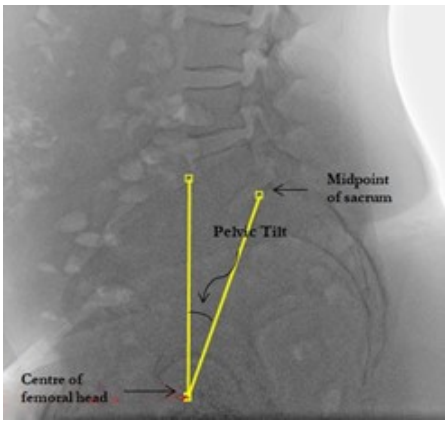

Figure 11. X-ray of lumbosacral spine (lateral view) demonstrating calculation of "pelvic tilt". Source: Figure by authors.

The normal measurement is <20°, but it can vary with differences in position and contracture (Roussouly et al. 2005).

2.1.4. History

A detailed medical history, as well as a history of spinal pathology, must be collected, with special attention paid to any previous spine procedures. Discogenic, mechanical, and neuropathic causes of pain must be distinguished.

A comprehensive musculoskeletal and neurologic examination should be carried out. The patient must be examined in both the standing and supine positions. Gait analysis should be carried out to look for secondary compensatory mechanisms.

The range of motion of the patient's hip and their pain or discomfort should be examined for any probable contractures or dysfunction in addition to the spinal assessment. The cranium must also be considered in terms of the cervical spine's range of motion. Vertical gaze functional constraints, which are fairly frequent consequences of sagittal imbalance, should similarly be evaluated (Kim et al. 2021).

2.1.5. Neurological Examination Related to Spinal Pathologies

Neurological examination (Boyraz et al. 2015; Fuller 2019; Shimizu et al. 1993; Walker 1990; Watson et al. 1997) is a very important tool in diagnosis, especially in the case of spinal pathologies. In this chapter, important examinations relevant to diagnosing the level of spinal pathology will be described.

MOTOR System

Grading of muscle power: Muscle power is conventionally graded using the Medical Research Council (MRC) scale. This is usually amended to divide grade 4 into 4+, 4, and 4−. The scale is as follows:

5 = normal power;
4+ = submaximal movement against resistance;
4 = moderate movement against resistance;
4− = slight movement against resistance;
3 = moves against gravity but not resistance;
2 = moves with gravity eliminated;
1 = flicker;
0 = no movement.

Power should be graded according to the maximum power attained, even it is maintained for a brief period.

Myotomes: Myotomes can be remembered by counting numerically:

"1, 2"—S1 and S2 supply the back of the legs (hip extension, knee flexion, and plantar flexion). This is also the root value of the ankle reflex.

"1, 2"—hip flexion is supplied by L1 and L2.

"3, 4"—L3 and L4 supply the quadriceps and the knee reflex (below the hip).

"5"—foot dorsiflexion is below L3 and L4, so it is supplied by L5.

"5"—the biceps are mainly supplied by C5 and this is the root value of the biceps reflex.

"6"—the brachioradialis is mainly supplied by C6 and this is the root value of the supinator reflex.

"7"—C7 supplies elbow extensors, i.e., the triceps, and extensors of the wrist and fingers; this is the root value of the triceps reflex.

"8"—finger flexion is below C7 and so it is supplied by C8.

"1"—small muscles of the hands are innervated by T1.

Tone: Testing muscle tone is a very important indicator of the presence and site of pathology. It can be surprisingly difficult to evaluate. Patients need to be fully relaxed. Commands to relax usually do not help. Irrelevant conversation or asking the patient to count down may help.

Normal tone is slight resistance through the whole range of motion. When the knee is lifted, the heel will lift minimally off the bed.

Decreased tone means loss of resistance throughout the range of motion. The heel does not lift off the bed when the knee is lifted quickly. Flaccid means marked loss of tone. This happens in lower motor neuron lesions or cerebellar lesions. Rare causes are myopathies, "spinal shock" (e.g., early after a stroke), and chorea. Increased tone means resistance increases suddenly ("a catch"). When the knee is lifted quickly, the heel easily leaves the bed. This is spasticity, which is a consequence of upper motor neuron lesions. Increased tone through the whole range of motion, as if bending a lead pipe, is named lead pipe rigidity. Regular intermittent breaks in tone through the whole range of motion are known as cogwheel rigidity. If the patient seems to oppose your attempts to move their limb, this is called gegenhalten or paratonia, and can happen in bilateral frontal lobe damage. Common causes are cerebrovascular disease and dementia.

Special situations:

- Myotonia: slow relaxation following action. This is demonstrated by asking the patient to make a fist and then release it suddenly. In myotonia, the hand will only unfold slowly.
- Dystonia: patient maintains posture at the extreme end of motion with contractions of agonists and antagonists.
- Percussion myotonia: may be demonstrated when a muscle dimples following percussion with a patella hammer. Most commonly seen in the abductor pollicis brevis and the tongue.

Basic Neurological Screening Examination

Upper limbs: A simple screening procedure can be performed as outlined below. It is to be tested on one side and then compared to the other side.

Shoulder abduction—this can be tested by asking the patient to lift both elbows out to the side. This tests the deltoid muscle, which is innervated by the axillary nerve, C5 root. Elbow flexion—patient's elbow and wrist are held and the hand is pulled towards their face. This tests the biceps brachii, which is innervated by the musculocutaneous nerve, C5 and C6 roots. Elbow extension—the patient is asked to extend the elbow against resistance. This tests the triceps muscle, which is innervated by the radial nerve, (C6), C7, and (C8) roots. Wrist extension—the patient's forearm is supported and the wrist is bent backwards. This tests the flexor carpi ulnaris and radialis muscles, which are innervated by the radial nerve, (C6), C7, and (C8) roots. Finger extension—the patient's hand is supported and extended fingers are to be pressed downward for the test. This tests the extensor digitorum muscle, which is innervated by the posterior interosseous nerve (a branch of the radial nerve), C7 and (C8) roots. Finger flexion—the examiner's fingers are placed on the patient's fingers, palm to palm, so that both sets of fingertips are on the other's metacarpophalangeal joints. The patient is then asked to "flex the fingers" or to "grip your fingers"; the examiner will attempt to "resist the finger flexion" or will try to "open the patient's grip". The flexor digitorum superficialis and profundus, which are innervated by the median and ulnar nerves, C8 root, are tested here. Finger abduction—the patient is asked to spread their fingers out against resistance. This tests the first dorsal interosseous muscle, which is innervated by the ulnar nerve, T1 root. Finger adduction—this can be tested using the card test, i.e., putting a card between the index and middle, middle and ring, or ring and little fingers. This tests the second palmar interosseous muscle, which is innervated by the ulnar nerve, T1 root.

Serratus anterior—the patient is examined from the back while standing in front of a wall. The patient is asked to push against the wall with their arms straight and their hands at shoulder level. If the muscle is weak, the scapula lifts off the chest wall, forming a "winged scapula". The long thoracic nerve, formed from C5, C6, and C7 roots, is tested here.

Brachioradialis—the examiner should hold the patient's semipronated forearm and wrist. The patient is asked to pull their hand towards their face. This tests the brachioradialis muscle, which is innervated by the radial nerve, C6 root.

Flexor digitorum profundus (FDP)— the patient is asked to grip the examiner's fingers while the examiner attempts to extend the distal interphalangeal joint of the little and ring fingers. The medial part of the muscle, which inserts to the fourth and fifth digits, is innervated by the ulnar nerve (C8-T1). The lateral part, which inserts to the second and third digits, is innervated by the median nerve via the anterior interosseous branch (C8-T1).

Lower limbs: The femoral and sciatic nerves are the two major nerves that supply the lower limbs; the former for knee extension and the later for knee flexion. The posterior tibial branch of the sciatic nerve supplies foot plantar flexion and inversion and the small muscles of the foot. The common peroneal branch of the sciatic nerve supplies dorsiflexion and eversion of the ankle.

The examination starts, ideally, by an inspection of the legs for wasting and fasciculation. Wasting is to be checked especially on the quadriceps, the anterior compartment of the shin, the extensor digitorum and brevis, and the peroneal muscles.

A simplified root distribution in the legs can be demonstrated as follows: L1 and L2 for hip flexion, L3 and L4 for knee extension and knee reflex, L5 for dorsiflexion of the foot, inversion and eversion of the ankle, and extension of the big toe, and S1 for hip extension, knee flexion, plantar flexion, and ankle reflex.

Screening Examination for the Lower Limbs

One side is to be compared with the other side. Hip flexion—the patient can be asked to lift their knee towards the chest. When the knee is at a right angle, the patient should be asked to pull it up as hard as they can against resistance applied by the examiner. This tests the iliopsoas muscle, which receives nerve supply from the lumbosacral plexus, L1 and L2 roots. Hip extension—this can be tested while the patient is lying flat with their legs straight. The patient is asked to push down on the examiner's hand, placed under the patient's heel. The gluteus maximus muscle is responsible for the movement and is innervated by the inferior gluteal nerve, (L5) and S1 root. Knee extension—the patient is asked to bend their knee. When it is flexed at 90 degrees, they are asked to straighten the leg against resistance applied at the ankle. This tests the quadriceps femoris muscle, which is innervated by femoral nerve, L3 and L4 roots. Knee flexion—the patient is asked to bend the knee and

to bring the heel towards their bottom against resistance applied at the ankle. This tests the hamstring muscles (semitendinosus, semimembranosus, and biceps femoris), which are innervated by the sciatic nerve, (L5) and S1 root. Foot dorsiflexion—the patient is asked to move their ankle back and bring their toes towards their head. When the ankle is past 90 degrees, resistance is applied in the other direction. This tests the tibialis anterior muscle, which is innervated by the deep peroneal nerve, L4 and L5 roots. Plantar flexion of the foot—the patient is asked to point their foot and toes downwards with the leg straight against resistance applied under the foot. This tests the gastrocnemius muscle, which is innervated by the posterior tibial nerve, S1 root.

Big toe extension—the patient is asked to pull their big toe up towards their face against resistance applied by the examiner. This tests the extensor hallucis longus muscle, which is innervated by the deep peroneal nerve, L5 root. Extension of the toes—the patient is asked to bring all toes towards their head against resistance. This tests the extensor digitorum brevis muscle, which is innervated by the deep peroneal nerve, L5 and S1 roots. Foot inversion—the patient is asked to turn their foot inwards while the ankle is at 90 degrees. This tests the tibialis posterior muscle, L4 and L5 roots. Foot eversion—the patient is asked to turn their foot out to the side. This tests the peroneus longus and brevis muscles, which are innervated by the superficial peroneal nerve, L5 and S1 nerve roots.

Tendon Reflexes

Tendon reflexes are increased in upper motor neuron lesions and decreased in lower motor neuron lesions and muscle abnormalities.

Reflexes can be graded as follows (Walker et al.):

0 = absent;
± = present only with reinforcement;
1+ = present but depressed;
2+ = normal;
3+ = increased;
4+ = clonus.

Biceps—the biceps muscle tendon is stretched with a tendon hammer above the elbow. The involved nerve is the musculocutaneous nerve and the root value is C5 (C6). Supinator—the brachioradialis muscle tendon is stretched with a tendon hammer above the wrist with the hand on a semipronated position. The involved nerve is the radial nerve and the root value is C6 (C5). Triceps—the triceps muscle tendon is stretched by stroking the back of the elbow with a tendon hammer. The involved nerve is the radial nerve and the root value is C7. Finger reflex—the examiner holds the patient's hand in a neutral position, places their hand opposite the fingers, and strikes the back of their fingers. The involved muscles are the flexor digitorum profundus and superficialis, the involved nerves are the median and ulnar nerves, and the root value is C8. Scapulohumeral reflex (SHR)—the SHR of Shimizu is elicited by tapping the tip of the spine of the scapula and acromion in a caudal direction. The SHR is classified as hyperactive only when an elevation of the scapula or an abduction of the humerus have been clearly defined after tapping at these points. The major muscles involved are the upper portion of the trapezius, the levator scapulae, and the deltoid. The reflex centre of the SHR is clinically presumed to be located between the posterior arch of C1 and the caudal edge of the C3 body. The implication of a hyperactive SHR provides useful information about dysfunctions of upper motor neuron lesions cranial to the C3 vertebral body level (Shimizu et al. 1993).

Pectoralis reflex—the examiner lightly places the index finger of their left hand on the tendon of the pectoralis major at the deltopectoral groove and strikes the finger with a reflex hammer. The pectoralis major muscle is to be observed. A brisk pectoralis jerk is seen only in patients with spinal cord compression at the C2–3 and/or C3–4 levels. The presence of a hyperactive pectoralis reflex is specific to lesions of the upper cervical spinal cord.

If the pectoralis reflex is brisk, the SHR is be tested to determine the level, as the SHR centre is higher on the spinal cord. If the SHR and jaw jerk are both positive, then the lesion is at the level of the pons or above. If the jaw jerk is negative, the lesion is below the level of the pons.

Lower Limb Reflexes

Knee reflex—the patient's knee on one side is supported by the examiner at 90 degrees. The examiner strikes the knee below the patella and watches the quadriceps. The involved nerve is the femoral nerve and the root value is L3 and L4. Ankle reflex—the examiner holds the patient's foot at 90 degrees, with the medial malleolus

facing the ceiling if the patient is lying on a bed. In the sitting position, one leg can be crossed over the other. The Achilles tendon is to be stroked directly. The muscles of the calf are to be watched. The involved nerve is the tibial nerve and the root value is S1 and S2.

Ankle Reflex Alternatives

1. The ankle reflex can also be tested by keeping the patient's legs straight and placing the examiner's hand on the ball of the patient's foot, with the ankles at 90 degrees. The examiner's hand is to be stroked with a tendon hammer and the muscles of the calf are to be watched. If the reflex is absent, reinforcement is needed. 2. Another way to check the ankle reflex is to ask the patient to kneel on a chair so that their ankles are hanging loose over the edge of the chair. The Achilles tendon is to be stroked directly.

Reinforcement of Reflexes

Jendrassik Ernő, a Hungarian physician, described the Jendrassik manoeuvre, where the patient clenches the teeth, flexes both sets of fingers into a hook-like form, and interlocks those sets of fingers together and at the same time, the reflexes are elicited with a tendon hammer. This can be used to reinforce the reflexes of the lower limbs when they cannot be normally elicited.

Clonus

Clonus is a rhythmic oscillating stretch reflex that occurs in upper motor neuron lesions and is generally accompanied by hyper-reflexia. Testing for clonus is performed as part of the neurological exam.

The ankle or Achilles reflex (S1/S2 nerve roots) is the most common site to test for clonus. This is tested while keeping the patient in the supine position. The hip and knee are flexed at a right angle; the knee is supported, the ankle is stretched to a dorsiflex position, then free movement is allowed. A persisted flapping movement of the ankle (plantar flexion) is positive for ankle clonus.

Some other commonly tested clonus reflexes are as follows (Boyraz et al. 2015):

- In the lower limbs, the patella/quadriceps/knee, L2 to L4 (mostly L4), are tested just inferior to the patella (or by pushing the patella distally);
- In the upper limbs:
  - Biceps: C5 to C6, just anterior to the elbow;
  - Triceps: C7 to C8 (mostly C7), just posterior to the elbow;
- For lesions in the brainstem, jaw jerk/masseter (trigeminal nerve) is tested at the chin/mental protuberance.

Interpretation:

- Increased reflex or clonus indicates an upper motor neuron lesion above the root at that level.
- Absent reflexes indicate peripheral neuropathy.
- Reduced reflexes (more difficult to judge) occur in peripheral neuropathy, muscle disease, and cerebellar syndrome. Reflexes can be absent in the early stages of severe upper motor neuron lesion, like "spinal shock".
- Reflex spread indicates an upper motor neuron lesion occurring above the level of innervation of the muscle to which the reflex has spread. The reflex being tested is present but this response goes beyond the muscle normally seen to contract. For example, the fingers are seen to flex when the supinator reflex is tested, or the hip adductors are seen to contract when testing the knee reflex.
- An inverted reflex is a combination of loss of the reflex tested with reflex spread to a muscle at a lower level. The level of the absent reflex indicates the level of the lesion. For example, a biceps reflex is absent but produces a triceps response. This indicates a lower motor neuron lesion at the level of the absent reflex (in this case, C5) with an upper motor neuron lesion below indicating spinal cord involvement at the level of the absent reflex.

Superficial Reflexes

Abdominal reflexes—using the sharp end of a reflex hammer, the examiner lightly scratches the abdominal wall from the abdominal margins toward the umbilicus, observes a quivering motion of the abdominal muscles, and watches the abdominal wall. It should contract on the same side. The afferents are the segmental sensory

nerves and the efferents are the segmental motor nerves. The root value above the umbilicus is T8–T9; below the umbilicus, it is T10–T11.

An absent abdominal reflex may occur due to obesity, previous abdominal operations, frequent pregnancy or age, as well as due to pyramidal tract involvement above that level or a peripheral nerve abnormality. Brisk abdominal reflexes are not clinically significant, though they are said to be brisk in cerebral palsy and motor neuron disorders.

Plantar response—the examiner draws the sharp end of the reflex hammer up a lateral border of the foot and across the foot pad. The big toe and the remainder of the foot are observed.

If all toes flex, this is a flexor plantar response. This will be interpreted as negative Babinski's sign, i.e., normal. If the big toe extends (goes up) and the other toes flex or spread, this is extensor plantar response, or positive Babinski's sign. This indicates upper motor neuron lesion. If the big toe extends (goes up), the other toes extend, and the ankle dorsiflexes, this is a withdrawal response. The test needs to be repeated gently or alternative stimuli need to be tried. If there is no movement of the hallux (even if the other toes flex), this indicates no response. A positive test at clinical examination should be reproducible.

Alternative stimuli for plantar response—these alternative stimuli are only useful when the plantar response is present. A stimulus on the lateral aspect of the foot can be tried to elicit a plantar response known as Chaddock's reflex. The same can be elicited by running the thumb and index finger down the medial aspect of the tibia; this is known as Oppenheim's reflex.

An upper motor neuron lesion pattern includes the signs and symptoms of increased tone, brisk reflexes, pyramidal pattern of weakness, and extensor plantar responses. A lower motor neuron lesion pattern includes wasting, fasciculation, decreased tone, decreased or absent reflexes, and flexor plantar responses.

If the reflexes are absent, the diagnosis will go towards polyradiculopathy, peripheral neuropathy, or myopathy. Sensory testing should be normal in myopathy.

In the state of "spinal shock", which occurs after a recent acute and severe upper motor neuron lesion, tone will be reduced and reflexes may be absent, even though this is an upper motor neuron lesion.

Mixed upper motor neuron (in the legs) and lower motor neuron (in the arms) weakness suggests motor neuron disease, which can either be associated with no sensory loss or with mixed cervical myelopathy and radiculopathy, which feature sensory loss.

Weakness in both legs with increased reflexes and extensor plantar responses suggests a lesion in the spinal cord. The lesion must be above the root level of the highest motor abnormality. The level may be ascertained with sensory signs.

Weakness in both legs, along with absent reflexes in the legs, indicates polyradiculopathy, cauda equina lesion, or peripheral neuropathy. Unilateral arm and leg weakness indicates upper motor neuron lesion in the high cervical cord, brainstem, or above.

Laterality of the Lesion

The corticospinal tracts (the pyramidal tracts) cross over in the pyramids in the medulla. Thus, lesions in the brain and brainstem above the medulla result in weakness on the opposite side of the body, and lesions in the spinal cord result in weakness on the same side of the body.

Upper motor neuron signs limited to a single limb can be caused by lesions in the spinal cord, brainstem, or cerebral hemisphere. Motor signs need to be assessed along with cranial nerve or sensory abnormality investigation to reach a diagnosis. If lower motor neuron lesion occurs, the following symptoms are seen.

Upper Limb

Lesion of the C5 root causes weakness of shoulder abduction, external rotation, elbow flexion, impaired sensation in the outer aspect of the upper arm, and loss of biceps reflex. Lesion of the C6 root causes weakness of elbow flexion, pronation, impaired sensation of the lateral aspect of the forearm and thumb, and loss of supinator reflex. Lesion of the C7 root causes weakness of elbow and wrist extension, impaired sensation of the middle finger, and loss of triceps reflex. Lesion of the C8 root causes weakness of finger flexion, impaired sensation of the medial aspect of the forearm, and loss of finger reflex. Lesion of the T1 root causes wasting of all small muscles of the hand and impaired sensation of the medial forearm.

Lesion of the median nerve causes weakness and wasting of the thenar eminence in the abductor pollicis brevis and impaired sensation of the thumb, index, and middle fingers. Lesion of the ulnar nerve causes weakness with or without wasting of all muscles in the hand excepting the LOAF muscles (lateral two lumbricals, opponens pollicis, abductor pollicis brevis, flexor pollicis brevis, and impaired sensation of the little and half ring fingers). Lesion of the radial nerve causes weakness of finger, wrist, and probably triceps and brachioradialis extension, impaired sensory changes at the anatomical snuffbox, and loss of reflex of the supinator and triceps, if the lesion is above the spiral groove.

Bilateral wasting of small muscles can be the presentation of peripheral neuropathy (with distal sensory loss) or motor neuron disease (without sensory loss). Lesion of the axillary nerve causes weakness of shoulder abduction by paralyzing the deltoid and impaired sensation of a small patch on the lateral part of the shoulder.

Lesion of the L4 root will result in weakness of knee extension and foot dorsiflexion; sensation in the medial shin will be impaired and the knee reflex will be affected. Lesion of the L5 root will result in weakness of foot dorsiflexion, inversion, and eversion, extension of the big toe, and hip abduction, and sensation will be impaired in the lateral shin and the dorsum of the foot. Common peroneal palsy results in weakness of foot dorsiflexion and eversion with preserved inversion, and sensation will be impaired in the lateral shin and the dorsum of the foot. Lesion of the S1 root causes weakness of plantar flexion and foot eversion, impaired sensation in the lateral border of the foot and sole of the foot, and ankle reflex loss. If in doubt, re-examine them with these factors in mind.

Sacral Sensation

This is not usually screened. However, it is important to test sacral sensation in any patient with urinary or bowel symptoms, bilateral leg weakness, sensory loss in both legs, and a possible cord conus medullaris or cauda equina lesion.

Patterns of Sensory Loss

Sensory deficits can be classified into eight levels of the nervous system:

1. Single nerve: sensory loss within the distribution of a single nerve may occur. This happens most commonly in the median, ulnar, peroneal, and lateral cutaneous nerves of the thigh.
2. Root or roots: sensory deficit may be confined to a single root or several roots in close proximity. Common roots in the arm are C5, C6, and C7, and in the leg, these are L4, L5, and S1. An important example is cauda equina syndrome, which involves multiple nerve roots in the lumbosacral spine (usually the S1–S5 roots bilaterally). This causes sensory loss in the perianal region and buttocks (saddle anaesthesia) and the back of both thighs.
3. Peripheral nerves can be affected by neuropathy, e.g., in distal glove and stocking deficit.
4. Spinal cord: five patterns of loss can be recognized:
    (a) Complete transverse lesion: hyperaesthesia (increased appreciation of touch/pinprick) at the upper level, with loss of all modalities a few segments below the lesion.
    (b) Hemisection of the cord (Brown-Séquard syndrome): loss of joint position and vibration sensation on the same side as the lesion and pain and of temperature sensation on the opposite side a few levels below the lesion.
    (c) Central cord: loss of pain and temperature sensation at the level of the lesion, where the spinothalamic fibres cross in the cord, with other modalities preserved. This is also known as dissociated sensory loss and is seen in syringomyelia.
    (d) Posterior column loss: loss of joint position and vibration sensation, but pain and temperature sensation are preserved.
    (e) Anterior spinal syndrome: loss of pain and temperature sensation below the level of the lesion, but joint position and vibration sensation are preserved.
5. Brainstem: loss of pain and temperature sensation on the face and on the opposite side of the body. This is found in lateral medullary syndrome.
6. Thalamic sensory loss: hemisensory loss of all modalities.
7. Cortical loss: in parietal lobe damage, the patient is able to recognize all sensations but localizes them poorly and the damage results in the loss of two-point discrimination, astereognosis, and sensory inattention.
8. Functional loss: this diagnosis is suggested by a non-anatomical distribution of sensory deficit, frequently with inconstant findings.

Tinel's Test

Tinel's test is the percussion of a nerve at the putative site of compression (usually using a tendon hammer). It is positive when paraesthesiae are produced in the distribution of the nerve concerned. It is commonly performed to test for median nerve compression at the wrist. Lhermitte's phenomenon is when forward flexion of the neck produces a feeling of electric shock, usually running down the back. The patient may complain of this spontaneously or you can test for it by flexing the neck. Occasionally, patients have the same feeling on extension (reverse Lhermitte's). This indicates cervical pathology, usually demyelination. It occasionally occurs with cervical spondylitic myelopathy, or, rarely, with B12 deficiency or cervical tumours.

Straight leg raising (SLR): this is a test for lumbosacral radicular entrapment. The examiner lifts the leg straight by the heel while patient is lying flat on the bed. The angle and any differences between the two sides are noted. It is regarded as normal when it is >90°, less in older patients. SLR is considered positive when it evokes radiating pain along the course of the sciatic nerve and below the knee between 30° and 70° of hip flexion. Hoover's sign: Hoover's sign demonstrates functional weakness by showing a discrepancy between voluntary hip extension and automatic hip extension. A patient lying on a bed flexing the hip to lift their left leg off the bed will inevitably automatically extend their right hip.

Bladder and Bowel Function

Spinal bladder: initially, the patient presents with urinary retention with or without overflow incontinence. Later, the bladder contracts and automatically voids small volumes of urine and dribbles. This is associated with constipation, but with normal anal tone. The patient may develop reflex penile erections, called priapism (after the Greek god Priapus). This is commonly caused by trauma and multiple sclerosis, rarely by spinal tumours.

Peripheral neurogenic bladder: this is a painless distension of the flaccid bladder with overflow incontinence and large residual volumes. This is associated with faecal incontinence and impotence. Anal tone is reduced. There may be saddle anaesthesia. This occurs in cauda equina lesions. A common cause is central lumbar disc protrusion; rarer causes are spina bifida, ependymomas, cordomas, and metastases. This also occurs in peripheral nerve lesions, where the most common cause is diabetes mellitus and rarer causes are pelvic surgery or malignancy.

2.1.6. Evaluation

Thirty-six-inch erect posture films are the gold standard for imaging spinal alignment. To demonstrate appropriate alignment, symmetry, and compensation, the pelvis, femoral heads, and spinal structure must be evaluated. Certain measures are quite important.

The most recent innovation in X-ray technology enables 2D-to-3D reconstructions using biplanar X-rays, which expose the patient to 800–1000 times lower radiation than a CT scan to produce the same image. MRI and CT can be used as part of a neurosurgical preoperative study or to assess the individual's clinical condition. Plain films, on the other hand, remain the mainstay of diagnosis.

2.1.7. Treatment

The main aim is to arrange the body in a physiological position that allows it to preserve its cone of stability with the lowest possible effort. The effects of sagittal and coronal plane spinal misalignment on pain and impairment in adults are now well recognized. Nonoperative treatment options include bracing for anatomical structural support and physiotherapy for strengthening. When surgical treatment is considered, spinal osteotomies are becoming more common in cases where nonoperative treatment has failed (Menger et al. 2020).

Adult scoliosis, iatrogenic fixed sagittal imbalance, flat back syndrome, kyphotic decompensation syndrome, as well as flat buttocks are all common disorders that necessitate surgical treatment. Smith-Petersen osteotomy, pedicle subtraction osteotomy, Ponte osteotomy, total/partial corpectomies, as well as spinal column resections are some of the main realignment procedures mentioned in the literature for these pathologies (Boachie-Adjei et al. 2006; Bridwell et al. 2004; Smith-Petersen et al. 1969; Thomasen 1985).

Schwab et al. described a complete classification of spinal osteotomies based on anatomy and established the inter- and intra-rater reliability of this classification system (Schwab et al. 2014) (Table 5).

Prevalence of Adult Spinal Deformity (ASD) and Role of Surgery Depending on the Magnitude of the Disease

Schwab and colleagues (Schwab et al. 2003) utilized the Short Form (SF)-36 questionnaire in the US to assess the illness burden in cases with adult scoliosis, comparing the findings to data from the general US population and those with other medical comorbidities. In all eight SF-36 domains, adults with scoliosis performed worse than the general population. For the US population with symptomatic spinal deformity, Bess and colleagues (Bess et al. 2016) gathered summary values of the SF-36 physical and mental components and observed comparable findings with a faster generational decline than the average (Roussouly et al. 2005).

ASD is a matter of discussion in healthcare due to its frequency among people over the age of 65, a population that is growing due to a variety of circumstances. Nonoperative care of ASD has been shown to be ineffective, with individuals with smaller abnormalities and people who are already happy with their spine-related health benefiting the most. Surgical treatment is favoured by patients hoping to improve their quality of life, according to publications, but complications are common (Diebo et al. 2019).

Table 5. Spinal osteotomy classification.

Grade	Description of Osteotomy
1	Partial facet joint excision—resection of the joint capsule and inferior facet at a particular spinal level.
2	Total facet joint resection—both inferior and superior facets at a specific spinal segment are excised with whole ligamentum flavum excision; other posterior parts of the vertebra incorporating the spinous processes and the lamina may also be excised.
3	Partial body/pedicle resection—partial wedge excision of a segment of the posterior vertebral body and of a part of the posterior vertebral elements, including the pedicle.
4	Partial body/pedicle/disc—wider wedge excision through the vertebral body, including a substantial part of the posterior vertebral body, posterior elements with pedicles, as well as excision of at least a portion of 1 endplate with the nearby intervertebral disc.
5	Discs and total vertebra—total excision of a vertebra and of both nearby discs (rib excision in the dorsal region).
6	Discs and multiple vertebrae—excision of more than one total vertebra and of nearby discs. Grade 5 excision as well as additional nearby vertebral excision.

Source: Authors' compilation based on data from Schwab et al. (2014).

Degenerative Spine Disease Without Instability

Without overt instability, whether a degenerative spine needs fusion or not is a matter of debate. There are no class I data to prove the efficacy of fusion. With the fact that the fusion reduces the range of movement, novel surgical plans are required to substitute surgical fusion.

Dynamic Stabilization

Two techniques are being studied: disc arthroplasty and posterior dynamic stabilization devices. The US Food and Drug Administration (FDA) has approved some artificial disc brands to manage symptomatic degenerative lumbar disc disease. Disc arthroplasty and lumbar fusion yielded similar results in short-term studies (Lin and Wang 2006).

A prospective, randomized, controlled multicentre trial aimed at demonstrating the "noninferiority" of cervical total disc replacement (TDR) in terms of outcome at 24 months found that this technique was at least similar in terms of outcome to anterior cervical discectomy and fusion (Murrey et al. 2009). Though most key outcome criteria (like pain scores and neurologic success) were comparable across both arms at 24 months, the disc replacement arm required fewer analgesics and had a lesser number of reoperations than the fusion arm.

Despite the fact that these findings are encouraging for total disc replacement, they should not be applied to the group of cases with multilevel disc herniations, spondylolisthesis, spondylosis, or degenerative disc disease (DDD), as this research was applicable to patients with radiculopathy and single-level disc disease. Long-term follow-up studies are essential to see whether these advantages are long-lasting, whether motion preservation using artificial discs is maintained over time, and whether the frequency of adjacent segment disease is reduced.

There are various types of posterior dynamic stabilization devices. Pedicle screw-based systems, in which the screws are joined by flexible elements rather than rigid rods, are the most promising. Theoretically, their purpose is to restrict movement to an area where spine loading is near-neutral or neutral or to avoid movement into a zone where excessive loading happens. Again, the studies undertaken thus far have generated clinical outcomes that are comparable to fusion (Schwarzenbach et al. 2005).

Contrary to conventional spinal instruments, which are sheltered from biomechanical stress as soon as the bony fusion is established, artificial discs and posterior dynamic stabilization devices should have better outcomes than fusion surgery and must function for the lifetime of the patient. Biologic management strategies aimed at repairing and preserving deteriorated spine elements, rather than mechanical treatment strategies, are more likely to give an acceptable remedy to degenerative spine disease in the future. Until then, lumbar TDR may be a preferable way of treatment over lumbar fusion for young patients with degenerative disc disease (DDD) who do not have severe facet joint degeneration, deformity, instability, or osteopenia/osteoporosis (Salzmann et al. 2017).

3. Kyphosis

When the dorsal spine curve is outside of the usual limit on the sagittal plane, it is called kyphosis. The Cobb angle is taken to determine the angle of the thoracic curve. Angle calculations between the superior endplate of D5 and the inferior endplate of D12 were reported by the Scoliosis Research Society (SRS) to range from 10 to 40 degrees (O'Brien et al. 2004). Thoracic kyphosis develops more commonly in men than in women (9.6%) (Yaman and Dalbayrak 2014).

3.1. Aetiology of Kyphosis

- Congenital kyphosis (Figure 12);
- Scheuermann kyphosis;
- Degenerative disc disease;
- Tumour-related kyphosis;
- Post-traumatic kyphosis;
- Postlaminectomy iatrogenic kyphosis;
- Infection-related kyphosis (Pott);
- Kyphosis developing due to neuromuscular diseases;
- Muscular dystrophy;
- Myelomeningocele;
- Spinal muscular atrophy;
- Paget's disease;
- Neurofibromatosis;
- Skeletal dysplasia (Yaman and Dalbayrak 2014).

The most important aetiologies of kyphosis will be discussed here.

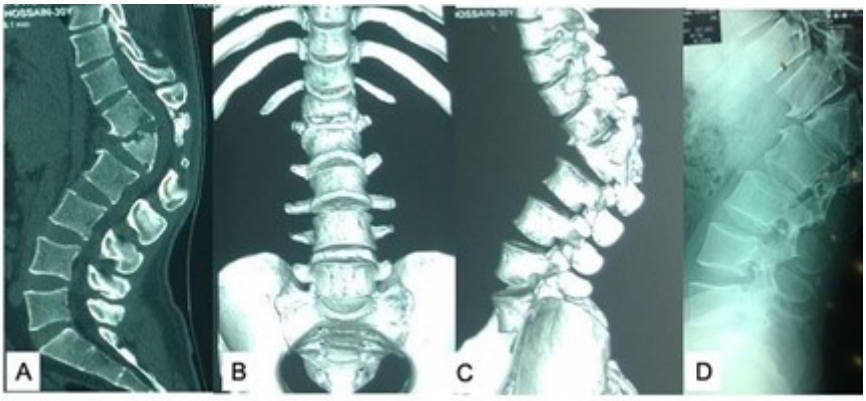

Figure 12. (**A**) Sagittal view; (**B**,**C**) three-dimensional reconstruction of CT scan of lumbodorsal spine and (**D**) X-ray lumbodorsal spine lateral view showing congenital kyphosis due to anomalous L1 vertebra. Source: Figure by authors.

3.2. Scheuermann Kyphosis

The Danish radiologist Holger Werfel Scheuermann was the first to define Scheuermann disease, also called "osteochondritis deformans juvenilis dorsi". The osteochondritis of secondary ossification centres causes rigid kyphosis which is commonest in young adults (Scheuermann 1920). It most frequently affects the lower dorsal and upper lumbar regions. It mostly affects adolescents between the ages of 13 and 16. The majority of patients are taller than their peers (Fotiadis et al. 2008).

Sorenson proposed the first diagnostic criteria for Scheuermann disease (Sörensen 1964):

1. Angulation of wedging of at least three adjacent vertebrae greater than 5°.
2. Kyphosis in the sagittal plane of more than 40°.
3. Irregularities of vertebral endplate.
4. Though Scheuermann's disease generally affects the dorsal spine (classic type), Edgren et al. also published an atypical type affecting the lumbar spine (Edgren and Vainio 1957).

3.2.1. Clinical Findings

1. Pain: after being seated for a long time, pain develops in the apical area. This pain diminishes once growth stops. Type 2 Scheuermann kyphosis causes greater pain than type 1 kyphosis.
2. Deformity: it is most commonly found during school age. To counteract kyphosis, lordosis of the lumbar and cervical spine may increase (Yaman and Dalbayrak 2014).

3.2.2. Treatment

When kyphosis can be minimized, rehabilitation is indicated to relieve discomfort and enhance sagittal balance. Postural control, trunk stretching and strengthening, and musculotendinous stretching, notably of tense pectoral and hamstring muscles, are all examples of rehabilitation treatments. In the event of restrictive pulmonary disease, respiratory rehabilitation can be beneficial (Zaina et al. 2009). For uncomfortable Scheuermann's disease with mild kyphosis or for mild kyphosis itself, bracing is recommended (Bradford et al. 1974).

When kyphosis between 55° and 80° is diagnosed before skeletal maturity, brace treatment is almost always successful, according to Lowe (Lowe 2007; Lowe and Line 2007). Until the patient achieves skeletal maturity, the brace should be put on for 21 h every day (Zaina et al. 2009; Gutowski and Renshaw 1988). Due to both the psychological and aesthetic influence of a neck ring, compliance may be lower with the Milwaukee brace than with other braces.

Scheuermann's disease rarely needs surgery. Surgery is suggested for stiff and symptomatic kyphosis (neurological impairment) with considerable and increasing curvature (>70°) when conservative measures fail (Lowe and Line 2007; Papagelopoulos et al. 2008; Palazzo et al. 2014). In skeletally mature patients, it must be performed by qualified surgeons. The first surgical procedure for treating Scheuermann's kyphosis was the posterior operative technique (Papagelopoulos et al. 2008). Various approaches have been developed. They all have the following steps: spinal structures are released, kyphosis is corrected (at least 50% of the curve is corrected), and instrumentation with arthrodesis is performed.

A number of authors paired an anterior release with a posterior correction to make the curve easier to adjust. The benefits of the additional anterior technique, on the other hand, are uncertain, and side effects may be more common (Lee et al. 2006; Lonner et al. 2007). Electrophysiologic monitoring is increasingly used to control neurological problems that may develop after surgery, especially during kyphosis reduction. Neurological (paraplegia), infectious, and respiratory problems are the most common (Lowe and Line 2007). The degeneration of the segment above or below the arthrodesis is known as junctional syndrome (Kim et al. 2012).

3.3. Postlaminectomy Kyphosis

Following extensive laminectomy, the likelihood of developing kyphosis increases. The facets of the posterior column in the neck area carry 65% of the load, while the rest is transmitted to the forearm. It is important to remember that when the posterior tension band (the ligamentum flavum, interspinous ligaments, and the ligamentum nuchae) is destroyed, stability is compromised (Yaman and Dalbayrak 2014).

The removal of more than a third of the cervical facets has been linked to instabilities (Epstein 1988). In certain studies, the rate of postlaminectomy kyphosis development in the paediatric age group was reported to be 100% (Dickson et al. 1978). To preclude the development of postlaminectomy kyphosis, cases should be carefully

opted for laminectomy. The likelihood of developing postlaminectomy kyphosis is reduced if there is preoperative lordosis (10 degrees or more), no instability findings on extension and flexion radiographs, and the facets are preserved perioperatively. In patients without cervical lordosis, the likelihood of postlaminectomy kyphosis is twice as high. In patients in whom the facets could not be protected, posterior fusion after decompression is indicated (Rao et al. 2011; Scioscia et al. 2011). When 30%–50% of the facets are eliminated, McAllister et al. recommend fusion (McAllister et al. 2012). In one study, anterior corpectomy with instrumentation, posterior fusion plus instrumentation and combined anterior corpectomy and posterior fusion with instrumentation were offered as surgical options for postlaminectomy kyphosis. During follow-up, none of the patients managed with cervical laminectomy and fused with lateral mass screws developed kyphosis, according to Kumar et al. (Kumar et al. 1999).

Post-traumatic kyphosis is a type of kyphosis that develops at the thoracolumbar junction after a trauma or surgery (Vaccaro and Jacoby 2002). The loads posed on the thoracic spinal segment during compression and flexion may create segmental kyphosis by causing a height decrease in the anterior column. Progressive kyphosis can occur as a result of pseudoarthrosis that occurs following surgery to cure the patient's spinal fracture and lack of fusion. Progressive neurological deficit and discomfort are the specific surgical signs of post-traumatic kyphosis (Yaman and Dalbayrak 2014; Vaccaro and Jacoby 2002).

4. Scoliosis

4.1. Introduction and Classification

Scoliosis is a broad term that describes a range of disorders characterized by alterations in the shape and the position of the spine, trunk, and thorax. Hippocrates used the term "spina luxate" to refer to all spinal abnormalities. Galen is credited with coining the term "scoliosis" (from the Greek skolios, which means "crooked or curved") (Vasiliadis et al. 2009) to describe an abnormal lateral spinal curvature.

"Structural scoliosis," or simply scoliosis, must be distinguished from "functional scoliosis," which is a curvature of the spine caused by extraspinal factors (e.g., paraspinal muscle tone asymmetry or shortening of a lower limb). When the underlying cause is removed, it usually reduces or disappears completely (e.g., in a recumbent position) (Negrini et al. 2018).

Kleinberg (Kleinberg 1922) coined the phrase "idiopathic scoliosis," which refers to all cases in which a specific disease is unable to be identified as the reason of the deformity; in reality, it can arise in otherwise healthy children and it progresses in response to a variety of stimuli throughout the fast interval of growth. Idiopathic scoliosis, by definition, has no known aetiology and is most likely caused by a combination of factors.

Idiopathic scoliosis is an aetiopathogenetically characterized spinal deformity that is an indication of a syndrome with a complex aetiology (Xiong et al. 1994; Burwell et al. 1983; Brooks et al. 1975). Scoliosis almost often appears as a single deformity, but careful examination may uncover several significant subclinical signs (Grivas et al. 2002; Weinstein 1999).

An "upper-end vertebra" and a "lower-end vertebra," both used as a control level to determine the Cobb angle, limit the curvature in the frontal plane (AP X-ray in upright posture) (Figure 13). The diagnosis is verified, according to the Scoliosis Research Society (SRS), when the Cobb angle is 10 degrees or above and axial rotation is visible. At the apical vertebra, the maximum axial rotation is measured. A Cobb angle of less than 10° (Xiong et al. 1994), however, can indicate structural scoliosis with the possibility of progression.

Progression is more prevalent in female children during the puberty growth spurt, and it is known as progressive idiopathic scoliosis then. If left untreated, it can progress to significant trunk abnormalities, limiting chest capacity as well as functional biomechanics, exercise capability, work abilities, and overall fitness, all of which are linked to a decrease in the quality of life (Negrini et al. 2018).

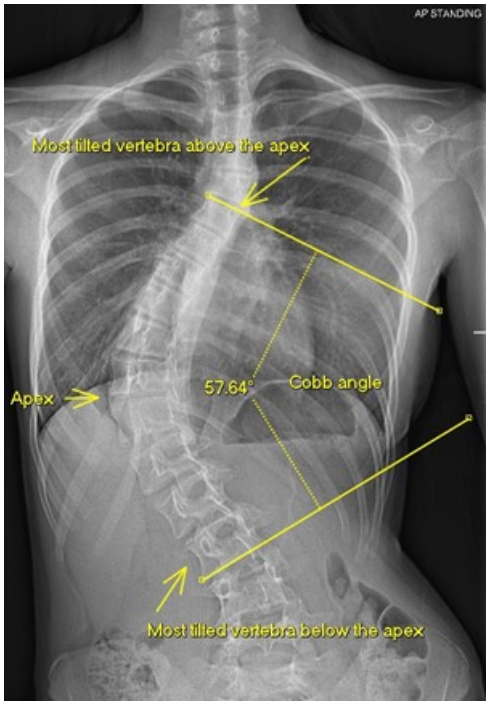

Figure 13. X-ray showing dorsolumbar scoliosis and demonstration of calculation of Cobb angle. Source: Figure by authors.

The three-step Lenke (Lenke et al. 2001) classification for adolescent idiopathic scoliosis has gained acceptance.

1. Label primary curve as type 1 to 6.

- Calculate regional curves:
  - Proximal thoracic (PT);
  - Main thoracic (MT);
  - Thoracolumbar/lumbar (TL/L).
- Define major curve (biggest curve): always either MT (type 1–4) or MT/L (type 4*, 5, 6).
- Detect whether minor curve is structural or not:

  Definition of structural: >25 degrees in the coronal plane on standing AP and not bending out to <25 degrees on bending films OR >20 degrees in the sagittal plane.

Assign type 1 to 6 based on the chart from Table 6.

Table 6. Assignment of types 1 to 6 based on chart below.

Curve Type	Curve Name	Comment on PT **, MT, TL/L
1	Main thoracic	Structural (major*) for MT, none for PT, TL/L
2	Double thoracic (DT)	Structural for PT and structural (major*) for MT, none for TL/L
3	Double major (DM)	None for PT, structural (major*) for MT, structural for TL/L
4	Triple major (TM)	Structural for PT, structural (major*) for MT and TL/L
5	TL/L	None for PT and MT, structural (major*) for TL/L
6	TL/L-MT	None for PT, structural for MT, structural (major*) for TL/L

Structural (major*)—has the largest Cobb angle and is always structural. In type 4, it can be either MT or FL/L based on which Cobb angle is greater. ** If PT is the largest curve, then by default assign major curve to MT. Source: Authors' compilation based on data from Lenke et al. (2001).

2. Assign lumbar modifier (A, B, C).

- Identify apical lumbar vertebrae (ALV): is it the lower lumbar body which falls outside of the curve?
- Draw the central sacral vertical line (CSVL) and determine its relationship to the pedicles of the ALV.
- Modifier:

- A if CSVL runs between pedicles of apical lumbar vertebrae (ALV);
  CSVL falls between pedicles of the lumbar spine up to stable vertebra.
- B modifier if CSVL touches pedicle of apical lumbar vertebrae (ALV);
- C modifier if CSVL does not touch apical lumbar vertebrae (ALV);
  Apex of lumbar curve falls completely off the midline, showing a curve with complete apical translation off the CSVL.

3. Assign sagittal modifier (-,N,+).

- Measure the sagittal Cobb angle from D5 to D12.
- Modifier:

  (a) hypokyphotic (−) if <10°;
  (b) normal if 10°–40°;
  (c) hyperkyphotic (+) if >40°.

4.2. Management

Scoliosis is a difficult condition to treat medically. Observation, bracing, and surgery are the traditional alternatives, in order. Spinal deformities are surgically corrected or improved to maintain sagittal balance, improve or preserve lung function, limit pain or morbidity, maximise postoperative function, and elevate or at least not affect lumbar spine function (Canale and Beaty 2008).

Developed curvature in a growing paediatric patient, severe deformity C > 50 with asymmetry of the trunk in teens, discomfort that is not contained by nonsurgical management, dorsal lordosis, and considerable physical deformity are all indications for the surgical treatment of AIS. Fusion surgery approaching from the anterior, posterior, or a combination of both are among the surgical options. Anterior procedures for idiopathic scoliosis include anterior instrumentation and fusion, which is now considered a standard procedure for certain dorsolumbar and lumbar curves (Canale and Beaty 2008; Muschik et al. 2006). Without using the anterior method, posterior instrumented fusion with pedicle screws or hooks is used. There are two stages in the combined technique:

(1) Anterior release plus fusion.
(2) Posterior fusion plus instrumentation with multi-hook segments (Canale and Beaty 2008; Muschik et al. 2006).

In more serious cases, a combination of these two steps is used.

In skeletally immature patients, the anterior technique has long been recommended because it can effectively prevent the crankshaft phenomenon (Dwyer 1973; Giehl et al. 1992) and it has historically been recorded to achieve better curve and rib hump correction as well as save fusion levels caudally. Another benefit derived from anterior spinal instrumentation is that it can treat thoracic hypokyphosis, which is common in AIS patients (Betz et al. 1999).

The results of thoracoscopic anterior instrumented spinal fusion are likewise excellent. Many surgeons, on the other hand, are unfamiliar with thoracoscopic surgery (Geck et al. 2009). When compared to anterior open or posterior methods, the learning curve remains steep, and operating times appear to be much longer (Lonner et al. 2006; Newton et al. 2009). All of these factors have limited its widespread adoption among orthopaedic surgeons. For the same duration of time, posterior pedicle screw instrumentation with posterior Ponte osteotomies (Geck et al. 2009) for both dorsal and dorsolumbar idiopathic scoliosis have gained popularity due to less morbidity, better respiratory function outcomes, and similar roentgenographic outcomes compared to anterior surgery (Lonner et al. 2006; Newton et al. 2009).

Although anterior thoracoscopic instrumentation can produce significant scoliosis correction (average 55–65%) with favourable cosmetic results, the danger of instrumentation problems (proximal screw pull-out) and of pseudoarthrosis is considerable (Reddi et al. 2008).

In the literature, there are several studies that compare anterior releasing and posterior fusing procedures for the treatment of scoliosis (Muschik et al. 2006). SA-phased treatment for extreme inflexible scoliosis with a Cobb angle > 80° at the coronal plane was described in a retrospective study by Yamin et al. (Yamin et al. 2008). The first stage involved anterior release and halopelvic tension, while the second stage involved posterior instrumentation plus spinal fusion. They came to the conclusion that staged surgery is a reliable method for managing serious stiff scoliosis (Yamin et al. 2008). Min et al. (Min et al. 2007) investigated the radiological and clinical results of patients

who had selective short anterior fusion of the main thoracolumbar/lumbar (TL/L) curve for the management of idiopathic AIS. They came to the conclusion that a balanced and satisfactorily repaired spine is produced by selected short anterior fusion of the TL/L curve scoliosis with a dorsal curve of less than 25 degrees. Short fusions allow for global spinal equilibrium by leaving enough movable lumbar segments (Min et al. 2007).

An anterior approach is, therefore, unnecessary with thoracic pedicle screw instrumentation. When compared to segmental hook instrumentation, posterior pedicle screw instrumentation results in much superior major and minor curvature correction frequencies sans any neurological problems and enhanced lung function (Sanders et al. 2003). According to Betz et al., both the anterior and posterior groups had equal coronal correction and balancing (Betz et al. 1999). In both the axial and coronal planes, thoracic pedicle screws enhanced correction. When compared to posterior segmental hook instrumentation, lumbar lordosis can be adequately managed to allow for more dorsal hypokyphosis (Pourfeizi et al. 2014).

Due to an increased risk of wound- or anaesthesia-related issues, two-stage surgery to repair scoliosis may give rise to some difficulties. Other potential hazards include thoracotomy complications (haemothorax, pneumothorax, etc.). Because of the unique challenges of ICU admissions, statistically significant differences are a critical aspect to consider when making an ICU admission decision. Another element to consider is the total length of stay in the hospital. Long-term hospitalization can raise hospitalization risks (medical errors, nosocomial infections, psychological impacts, and so on) (Weiss and Goodall 2008).

Scoliosis can cause soft-tissue inflammation or deep inflammatory processes as well as respiratory difficulties, haemorrhage, and nerve injury. Approximately 5% of individuals require reoperation as little as five years after surgery (Hawes and O'Brien 2008). The physical consequences of surgery are not always predictable (Kouwenhoven and Castelein 2008). In severe scoliosis, posterior segmental pedicle screw fixation without anterior release resulted in plausible deformity correction with minimal loss of curvature correction (Pourfeizi et al. 2014).

Growth modulation procedures, like the Shilla operation, growing rods, stapling of vertebral body, and, recently, vertebral body tethering (VBT), all rely on the Hueter–Volkmann principle to control growth and to rectify the curvature in adolescent idiopathic scoliosis (Aronsson and Stokes 2011; McCarthy et al. 2014; Betz et al. 2010; Hueter 1863). These approaches are expected to help maintain the underlying structure of the spine, which can result in a variety of advantages (Hoernschemeyer et al. 2020).

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