Spinal Injuries

Md Moshiur Rahman, Nazmin Ahmed and Forhad H. Chowdhury

Abstract: Spinal injury can be devastating for the patient and their family. It can be a big economic burden for families and society. Most spinal injury patients are young and injuries are usually caused by road traffic accidents, falls from heights, or by acts of violence. Injury can occur in any part of the spine. Spinal injury can be with or without neuro-deficit and with or without instability; for this reason, in these patients, clinical and radiological evaluations are very important. Management ranges from simple immobilization to complex decompression and stabilization. The early part of this chapter will discuss the history and epidemiology of spinal injury, the three-column theory of spinal stability, the clinical and radiological evaluation of spinal injury patients, and the classification of spinal injury. The later part of this chapter will briefly demonstrate the principles of management of specific types of spinal injury, as well as their complications and outcome prediction.

Abbreviations

AD	autonomic dysreflexia	AIS	ASIA Impairment Scale
CNS	central nervous system	CV	Cardiovascular
CVJ	craniovertebral junction dislocation	DBH	dopamine-S-hydroxylase
DVT	deep vein thrombosis	GW	Gardner–Wells
ICU	intensive care unit	MAO	monamine oxidase
MRI	magnetic resonance imaging	NLI	neurological level of injury
PE	pulmonary embolism	SCI	spinal cord injury

1. Introduction

Spinal injury can cause an overwhelming debilitation in the patient's life. Adjusting to the new condition is a test for all who are included, as it is particularly costly from the monetary perspective, both for the patient and their family and for healthcare administrations, as improving the patient's quality of life requires a wide range of assorted costs. Furthermore, most patients who experience the ill effects of spinal injury are of working age; therefore, they lose their source of revenue and become absolutely contingent upon their family, both monetarily and in terms of their basic needs, for example, eating, getting dressed, bathing, and so on, requiring in-home, personalized medical care. As indicated by different epidemiological examinations, spinal cord injury affects somewhere in the range of 236–1298 people per million worldwide (Guttman 1973). Spinal cord injury (SCI) can result from acute trauma, compression, and from hemisection. These three causes are reflected in the clinical scores for the study of SCI. Each of the three results in various levels of essential tissue damage. Despite noteworthy advancements in the recovery of SCI patients, the following areas have not been satisfactorily researched: (1) the overall fundamental pathophysiologic issues that happen in severely affected subjects, such as osteoporosis, periarticular bone alterations, hypertensive emergencies, etc.; (2) moderation of serious injury: anti-inflammatories or careful intervention to protect as much sensory and motor function as possible; and (3) the potential recovery of lost motor function. Acute injury to the spinal cord results in an immediate loss of motor function. The capacity to walk requires the support of a number of long tracts of the spinal cord. Moreover, pathophysiologic changes happen following SCI. If we are mindful of the specific pathophysiologic condition of the patient, it is conceivable to diminish the pace of progressive loss of function after acute spinal cord injury and to preserve some level of motor and sensory function by proper intervention. The morphologic changes that result from fall-related spinal cord injuries in exploratory animal models are like those that happen in most human spinal cord injuries. The cell layers of neurons, glia, and veins go through irreversible pathologic changes that lead to the degeneration of the spinal cord and incorporate mechanical and vascular disruption and the influx of free radicals. In spinal cord injury models, blood flow to the affected fragment is especially diminished, indicating that ischaemia might be a significant pathogenic component (DeVivo 2002). The reduced blood flow might lead to vasospasm or microvascular dysfunction, or both. The spinal cord and the remainder of the central nervous system (CNS) have high requirements to function correctly, more so than other vital organs. The blood–CNS barrier maintains a special climate for the CNS by barring certain substances and improving the transport of others, for example, ascorbic acid. Accordingly, lipid and protein membranes must be physicochemically flawless to maintain the functioning of the sensitive films engaged in their transport. Minor disruptions in the arrangement of key atoms in the membranes of the lipids that help keep up the dynamic state of key catalysts, for example, Na+, K+-ATPase, adenylate cyclase, and prostaglandin synthetase, can have adverse outcomes. Modifying lipid transmission will influence synaptosome arrangement and the coupling of transmitter receptors. Biogenic amines delivered inside the spinal cord after injury have been studied.

2. Epidemiology and History

SCI was first mentioned as "an illness not to be cured" by a mysterious physician from Egypt in the supposed Edwin Smith Papyrus over 5000 years ago (Guttman 1973). The prognosis for people with SCI has only recently improved. Before World War II, an individual with SCI had a future that was rarely longer than two years; the majority succumbed to renal failure, septic infections, and pressure ulcers. With the appearance of anti-microbials and improved restorative strategies, noteworthy advancement has been made toward viably alleviating and dealing with the various unexpected problems stemming from SCI. People with SCI have improved their capability for self-care and mobility, which typically enables them to reintegrate into their social networks. This has been made possible by improved intensive care strategies as well as early and comprehensive rehabilitation. The prevalence of SCI has slowly increased, and the relevance of this problem as a medical issue for society has increased, despite the fact that the annual frequency of SCI in the USA has remained stable and that the chances of death every year after injury have decreased. Even though the general incidence has stayed consistent in recent decades, the most prevalent causes of SCI have changed. Car accidents are the main cause of SCI, followed by falls and violence-related reasons (National Spinal Cord Injury Statistical Center 2005). Over time, the severity of injuries associated with sports has decreased while the severity of injuries from falls has increased. Before 1980, 13% of SCI cases were attributed to acts of violence. This percentage peaked at 25% between 1990 and 1999, then started to decline to 14% starting in 2000. Despite the fact that most injuries happen in people between the ages of 16 and 30, the mean age at acute SCI has increased to 37.6 years.The percentage of affected people over 60 years old increased from 5% prior to1980 to 11% after 2000. The origin of the injury differs between age groups, with sports-related and violence-related injuries commoner in the younger population and falling-related injuries commoner in the elderly population. There is a more prominent occurrence of SCI in the hotter months and on weekends. There has been an ongoing pattern toward a higher number of incomplete injuries, perhaps because of differing aetiology (e.g., falls cause an incomplete injury and violent trauma a complete injury), improved therapy at the site of injury by emergency services, and the availability of immediate medical attention. At the time of injury, over half of the individuals with SCI are secondary school graduates and of working age. Less than 33% of people are married at the time of spinal cord injury, with the majority (30%) being single (Go et al. 1995). Cervical injuries make up about 13% of all acute SCIs, whereas thoracic injuries make up the remaining 33%. The most common level of injury resulting in paraplegia is T12, and the most common injury resulting in neurological sequelae is C5, followed by C4 and C6, in the cervical region.

3. Three-Column Concept of the Spine

Francis Denis, in 1983, published a novel classification of spinal fractures as well as their management among stakeholder medical professionals (Zhang and Chauvin 2021; Denis 1983). In place of Sir Frank Holdsworth's earlier two-column theory, he devised a three-column theory (Holdsworth 1970). This newly developed three-column hypothesis later served as the cornerstone of a system for categorizing spinal injuries.

In the Denis classification system, the spinal architecture is divided into three columns including the following components (Figure 1) (Zhang and Chauvin 2021; Denis 1983; Holdsworth 1970):

Anterior column:

- ALL (anterior longitudinal ligament);
- Anterior two-thirds of the vertebral body and the annulus.

Middle column:

- Posterior one-third of the vertebral body and the annulus;
- Posterior vertebral wall;
- PLL (posterior longitudinal ligament).

Posterior column:

- Everything that is behind the PLL plus the posterior ligamentous complex as well as the posterior bony arch (supraspinous ligament, capsule of facet joint, interspinous ligament, and ligamentum flavum).

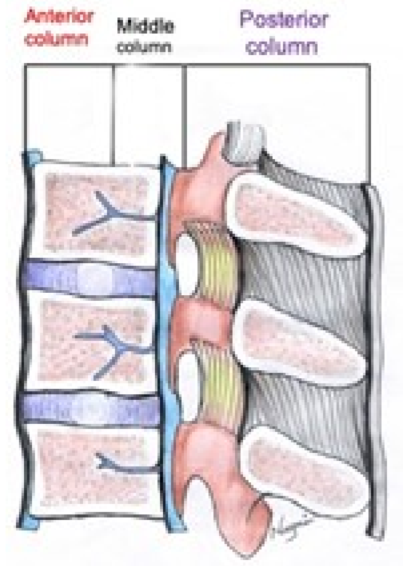

Figure 1. Three-column model of the spine. Source: Figure by authors.

To explain the innate instability of the middle column, the third column was created (Denis 1983; Denis 1984). When the posterior longitudinal ligament is injured in conjunction to the posterior annulus fibrosis, middle column fractures are deemed unstable, but an isolated total breakdown of the posterior ligamentous complex is not enough to cause complete instability (Zhang and Chauvin 2021).

4. Clinical Evaluation of SCI

The International Standards for Neurological Classification of Spinal Cord Injury, more commonly referred to as the American Spinal Injury Association (ASIA) guidelines, are the most precise method for evaluating SCI (Figure 2) (Sapru 2002; American Spinal Injury Association and International Spinal Cord Society 2006). This enables the clinician to determine the motor, physical, and neurological consequences of the injury, the level of the injury, and the ASIA Impairment Scale (AIS) score. The sensory and motor components, along with certain mandatory and optional components, make up the two main segments of the neurologic evaluation of a person with SCI. The necessary components include the assessment of neurological, motor, and sensory function levels, motor and sensory scores, and an assessment of the injury level. An anal examination that checks for voluntary anal contraction and deep anal pressure sensation is also necessary. A standardized neurological scale should be used to capture this information so that it may be retained in clinical records. The optional components are those portions of the neurological evaluation that do not contribute to the mathematical scoring but may reflect a more accurate scenario of the patient's clinical status. These include tests of additional muscles, proprioception, as well as reflexes. An instruction handbook and videotapes on the international regulations in this regard are available via the ASIA office in Atlanta, Georgia. These recommendations provide the definitions for the most often used terminology that doctors use to assess neurological function and evaluate SCI. The Model System Spinal Cord Injury information network uses the international recommendations since they are the most comprehensive and reliable system for evaluating SCI. This information network, which is maintained by designated SCI Model System centres in the USA, keeps track of information about spinal cord injury, provides rehabilitation services, and conduct research on SCI from the onset of injury to its progression over the long term.

For sensory functioning in incomplete spinal cord injury, a mathematical scale is utilized, which is as follows:

0—lack of sensation;
1—weakened sensation, characterized as fractional or modified sensation, encompassing hyperaesthesia;
2—typical sensation, with the face being the ordinary reference point.

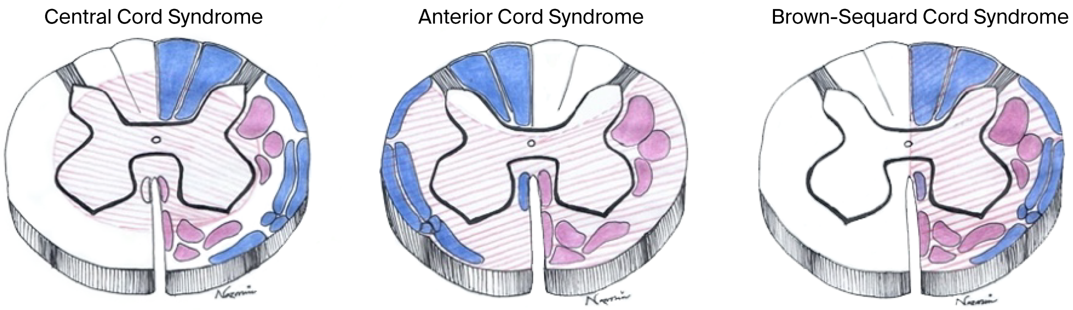

Figure 2. Illustrations of different types of incomplete spinal cord injury. Source: Figure by authors.

The patient must be capable of differentiating between a safety pin's sharp and dull edges in a pinprick test. The failure to distinguish between the two results in a score of 0. When a patient can differentiate between the sharp and dull end but the pin is not felt as sharply as it would be on the face, the patient receives a score of 1 for a weaker response to the pinprick test. If the pain the same as on the face, a score of 2 (normal) is assigned. A cotton swab is utilized to assess light touch, with a normal score of 2 representing a touch sensation equivalent to that on the face and a weakened score of 1 representing less sensation than on the face. The tactile level is described as having a characteristic sensation for both the pinprick and light touch on both sides of the body. This is also assessed on the caudal dermatome. For each dermatome, the scores for pinprick and light touch are added separately to produce a possible absolute score of 112 for tactile record scoring (56 on each side). A digital examination is conducted to evaluate deep anal sensation. The clinician approaches the patient and firmly presses a digit on the inside of the anus to feel for any tactile awareness, whether related to touch or pressure. One or the other must be present in order for deep anal sensation to be recorded. Proprioception, which refers to the ability to sense joint position and movement, temperature, and force, is an optional component of the sensory function assessment. In the unlikely event that precise tactile examination of any dermatome cannot be carried out, “not tried” should be noted, or another site within the dermatome can be examined, with proof that a different site was used. Ten important muscles on each side of the body—five in the upper appendages and five in the lower appendages—are tested as part of the motor assessment. Beginning with the elbow flexors (i.e., C5 innervated muscles) and ending with the plantar flexors, muscles should be tested in a cranial-to-caudal pattern. Although the majority of muscles are supplied by more than one nerve root, these muscles have been chosen for examination as they are consistently essentially innervated by the relevant segment and because they are straightforward to test while lying in a reclined position. A muscle is regarded to have complete innervation by at least one of its innervating segments in SCI if it receives a rating of 3/5. A muscle is regarded as important for useful activities if it has a grading of over 3 and if it can generate resistance against gravity (Welch et al. 1986). Different major muscles (such as the deltoids, abdominal muscles, and hip adductors) may be used; however, they are not utilized to detect the connection of specific regions of the spinal cord or to determine a motor level. In addition to the major muscles, the external anal sphincter should be tested by digital examination to identify voluntary contraction. To avoid confusing reflex contraction of the anal sphincter with voluntary contraction, care must be exercised. The most caudal key muscle group that is rated three or higher on the ASIA scale, with the portions cephalad to it retaining normal function, is known as the motor level (American Spinal Injury Association and International Spinal Cord Society 2006). The motor level is calculated when the motor scores for each muscle are recorded during a standard examination. The highest absolute motor score is 100, or 50 on each side. In many cases, the patient's clinical state may prevent the completion of an accurate assessment, like when a patient is unconscious owing to traumatic brain injury, has a lumbosacral or brachial plexus injury, or has an immobilized limb due to a broken bone. The clinician should enter NT, for “not tried”, rather than a numerical score, when the patient is not entirely tested in any way at all. The NLI (neurological level of injury) is the most caudal level above which the body's motor and sensory modalities on the two sides are both normal. For instance, the NLI is C7 if the motor level is C7 and the sensory level is C8. It is advised to note each side separately, as this may provide a clearer picture of the individual's status because the motor or sensory level may differ from side to side (i.e., right C6 motor, C7 sensory, left C7 motor, C6 sensory). Compared to the overall NLI, the motor level in the upper extremities is a good reflection of the level of function and of the severity of injury and disability after tetraplegia (total loss of motor function) (Marino et al. 1995).

5. Neurological Evaluation

Stabilizing spine protections, such as immobilization, should be continued until thorough clinical and radiological evaluations are completed. The underlying examination should include an itemized neurological examination that is completed as early as is practical and includes a time and date stamp. It is crucial that all motor and sensory function be assessed in an awake patient. The American Spine Injury Association (ASIA) framework should be used to assess muscle strength and pinprick and light touch sensation. In unconscious patients, muscular tone, deep tendon reflexes, long tract signs, and priapism in the male patient should all be noted at first. The evaluation should include an anal examination to check sphincter tone, types of contraction, the absence or presence of the bulbocavernosus response, the anal wink, and perineal sensation. The use of a feeding tube and of a Foley urinary catheter can occur as treatment progresses. This is because it is normal for these patients to develop paralytic ileus, which puts them at risk of malnutrition. Not only is the Foley urinary catheter useful for recording urinary production, but it can also prevent bladder overdistention, which frequently accompanies the urinary retention experienced by these individuals.

6. Radiographic Evaluation

A lateral cervical spine series is the first thing that the radiographs of patients with spinal injury are evaluated for. About 70–83% of the time, this view accurately detects significant deviations from the norm. This view has to be examined for arrangement, abnormalities in the bone and intervertebral disc spaces, and soft-tissue injuries. At the C3 level, prevertebral tissues often measure close to 4 mm in thickness. Prevertebral soft-tissue swelling should be taken into account just as much as vertebral facture. In 30–40% of patients, prevertebral soft-tissue swelling could be the primary radiological evidence of traumatic spinal cord injury (Harris 1986). Imaging should include each of the seven cervical vertebrae and the C7-D1 junction (Nichols et al. 1987). Consider using a "swimmer's perspective", offsetting the humeral heads, to visualize the cervical spine. Atlantoaxial, atlanto-occipital, or other instabilities identified in the initial examination are contraindications to using this view. An AP view, together with an odontoid view (also called open mouth), is typically all that is required to sufficiently view the cervical spine. Limitations of plain X-rays include the difficulty in identifying tendon injuries, over- and underexposure, and decreased perception of the cervicothoracic, occipitocervical, and thoracolumbar areas. CT scanning should also be used to investigate any areas or junctions that ordinary views are unable to adequately visualize. Bone dislocation is also better viewed with CT, and CT filters are more sensitive to vertebral fractures (Blackmore et al. 1999). The limitations of this method encompass missing fractures that correlate falsely to the imaging plane. This is no longer a major worry thanks to the use of transforms. However, CT is the imaging modality of choice for viewing neurological components because X-ray has limited sensitivity in diagnosing fractures. CT can be used in patients who have unexplained neurological damage, an additional neurological conditions, or who have inconsistent assessment results between the skeletal and neurological systems. This occurs in cases of traumatic spinal fracture. Spinal cord compression, intramedullary oedema and discharge, plate disruption, ligament injury, and vascular obstruction can all be identified with an X-ray. MRI also provides a better image of recurring injuries such myelomalacia and syrinx arrangement. The great majority of these cases can be assessed using sagittal T2W images. Cases with altered mental states and fractures that involve decussation always be suspected of this (Golueke et al. 1987). When MRI cannot be performed or when there is an MRI contraindication, like a pacemaker, emergency myelography is performed.

7. Types of Spinal Cord Injury

7.1. General Types of Spinal Cord Injury

To link the various types of SCI with their sequelae, clinicians have attempted to classify them depending on clinical symptoms. The Frankel classification is broadly considered to be useful and objective. Cases are evaluated as (A) complete neurological injury; (B) preserved sensation only; (C) preserved motor, nonfunctional; (D) preserved motor, functional; and (E) normal motor function. According to this classification, complete neurological injury has no preservation of motor or sensory function in at least three segments underneath the level of injury. As indicated by the guidelines published by the American Spinal Injury Association in 1992, for a spinal cord injury to be classified as incomplete, motor or sensory function or both should be preserved in the S4–S5 sacral segments (American Spinal Injury Association 1992). Otherwise, the patient is regarded to have a complete spinal injury. These definitions are significant in that, as far as prognosis goes, patients with incomplete

SCI have greater likelihood of achieving some functional recovery, while in those with complete SCI, avoiding spinal shock is the best-case scenario. In large cohort studies of patients with spinal cord injury, the majority are classified as incomplete SCI cases.

7.2. Specific Types of Spinal Cord Injuries

7.2.1. Injury to the Cervical Spine

Different pattern and classification of cervical spinal injuries are shown in Figures 3–11 and Table 1.

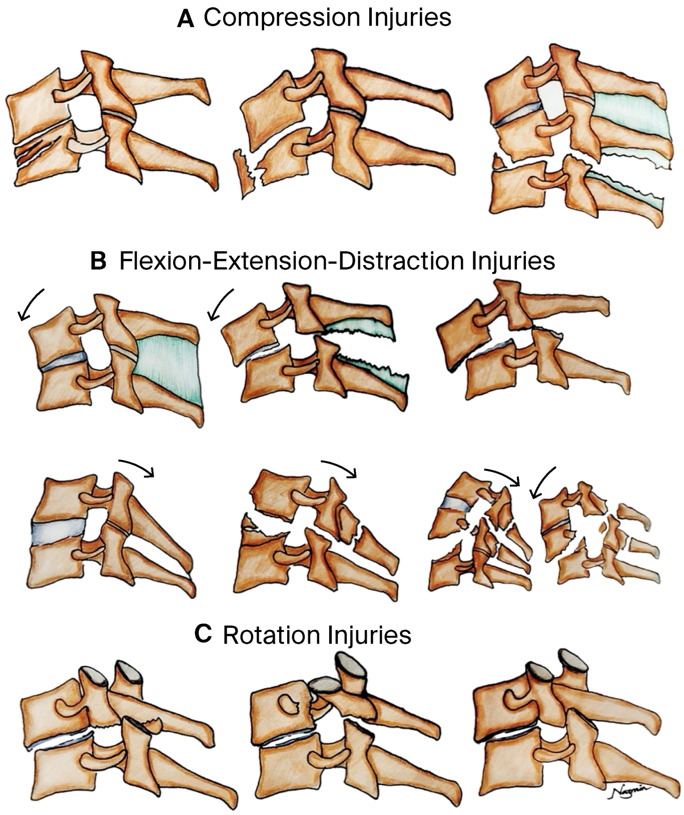

Figure 3. Illustration showing a schema for the classification of lower cervical spine injuries (Argenson et al. 1997). Source: Figure by authors.

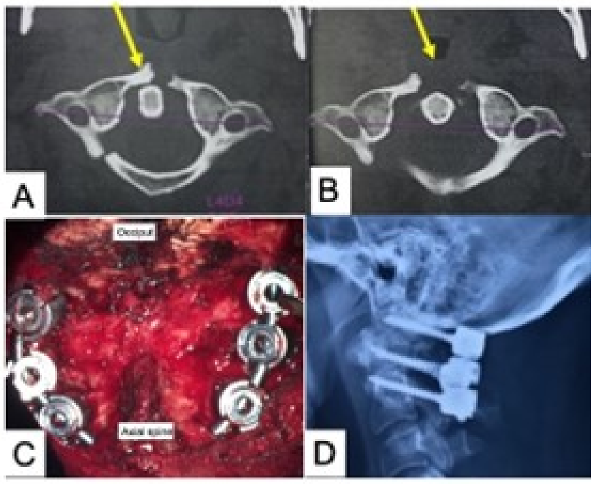

Figure 4. (**A**) CT scan of CVJ showing Jefferson fracture; (**B**) CT scan after 6 weeks showing unstable fracture; (**C**) perioperative picture of fixation of the occipital condyles (Co), C1 lateral mass, and C2 lateral mass with fusion for unstable Jefferson fracture; (**D**) postoperative X-ray showing fixation of Co-C1-C2. Source: Figure by authors.

Figure 5. (**A**,**B**) X-ray of cervical spine and MRI of CVJ showing traumatic AAD. (**C**,**D**) X-ray of CVJ showing fixation of C1 and C2 by lateral mass screws and rods, respectively. Source: Figure by authors.

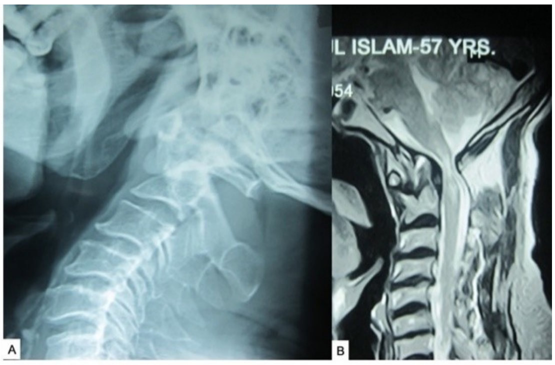

Figure 6. (**A**) X-ray of cervical spine showing AAD (traumatic); (**B**) MRI of CVJ showing AAD with spinal cord compression. Source: Figure by authors.

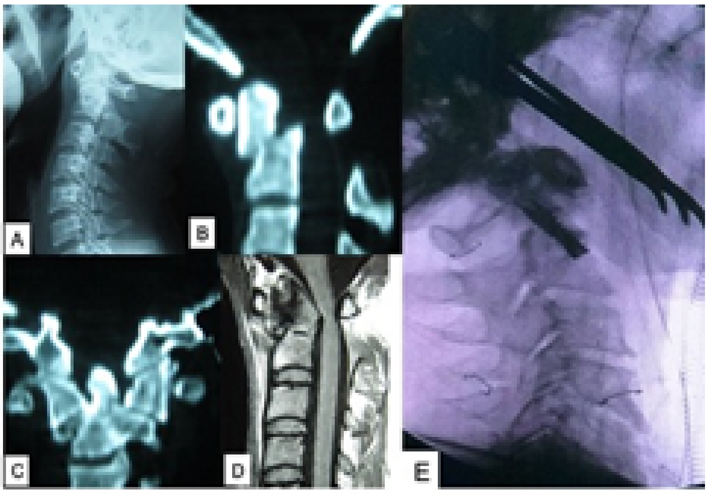

Figure 7. (**A**) X-ray of CVJ showing odontoid fracture with AAD; (**B**,**C**) CT scan of CVJ showing type 2 unstable odontoid fracture. (**D**) MRI of CVJ showing cord compression with instability. (**E**) Intraoperative X-ray showing reduction of odontoid fracture with fixation. Source: Figure by authors.

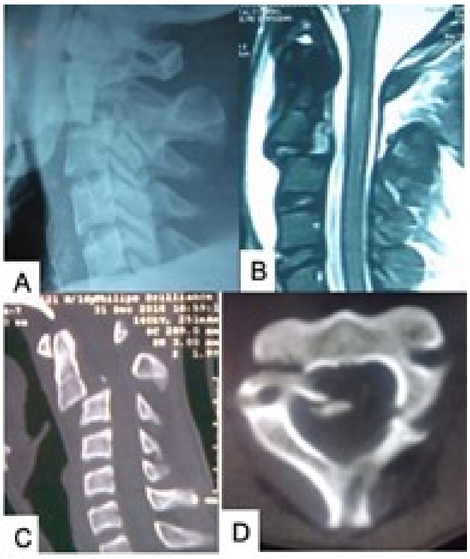

Figure 8. (**A**) X-ray, (**B**) MRI, and (**C**,**D**) CT scan of cervical spine showing hangman's fracture. Source: Figure by authors.

Figure 9. X-ray of cervical spine (lateral view) showing fracture and dislocation of C7 with locked facet. Source: Figure by authors.

Figure 10. MRI of cervical spine (sagittal view) showing traumatic fracture and subluxation at C5/6 with spinal cord injury. Source: Figure by authors.

Figure 11. (**A**) X-ray of cervical spine (lateral view) showing complete fracture and dislocation at C5/6. (**B**) MRI of cervical spine showing complete transection of cervical spinal cord. Source: Figure by authors.

Table 1. Classification of cervical spinal injury.

A. Cervicocranial Injuries—Anatomical Types		
Occiput	Atlanto-occipital dislocation (AOD)	Severe combined distractive force; often fatal
Atlas (C1)	Jefferson burst fractures	Axial compression
Odontoid process (Figure 7)	Type 1 Type 2 Type 3	Tip of dens (stable) Through base of dens (unstable) Through C2 vertebral body (stable)
Axis (C2)	Hangman's fracture	Pars fracture ("traumatic spondylolisthesis"')
B. Lower Cervical Spinal Injuries—Classified by Mechanism of Injuries (bones fail in compression and ligaments fail in distraction)		
Distractive flexion	Posterior ligament tear Hyperflexion sprain Bilateral "perched facets" Bilateral facet dislocation Unilateral facet dislocation (with rotational component)	
Compressive flexion	Anterior vertebral body fracture Wedge compression fracture Flexion teardrop	
Distractive extension	Anterior ligament tear Hyperextension sprain Hyperextension teardrop Hyperextension dislocation	
Compressive extension	Posterior element fractures Unilateral or bilateral laminar, lateral mass or spinous process fracture	
Axial compression	Vertebral body burst fracture	
Lateral bending	Uncinate process fracture Unilateral vertebral body Posterior element fracture	
Clay shoveler's fracture	Isolated spinous process fracture of C7 (C6 or T1)	

Source: Authors' compilation based on data from Schwartz (2008).

Most spine injuries happen at the cervical spine, which is the most flexible section of the vertebral column. Car accidents represent a large portion of these wounds. Tears and fractures are the most widely recognized types of injury, and subluxations and spinal cord injury without radiographic abnormality (SCIWORA), although generally rare, happen more habitually in younger patients (Hamilton and Mylks 1992).

According to estimates, 15% of people with spine injuries experience neurological and physical problems (Hagen 2015). The cervical spine is the most frequently involved segment, and it is calculated that 40–60% of all cervical spine injuries result in neurological morbidity and mortality (Hagen 2015). After clinical and radiographic evaluations are completed, unstable or severe injury should be treated immediately with cervical traction using Gardner–Wells (GW) pins (Hagen 2015).

Following careful cleansing of the skin in the area and administration of a local anaesthetic, pins are placed 1 cm cephalad to the pinna by the external acoustic meatus (Labronici et al. 2015). The pins are tightened until the spring-loaded indicator protrudes 1 mm above the surface (Labronici et al. 2015). Keeping the patient compliant and awake enough to comply with subsequent neurological examinations is crucial. Complications such pin dislodging, site contamination, and skull penetration are connected to cervical traction (Patel et al. 2009). Distracting injuries, skull fractures, and unstable upper cervical spine injuries are among the indications against using this technique (Iida et al. 1999).

Recommendations of the WFNS Spine Committee 2019 for Cervical Spine Trauma

Guidelines for preventing spine damage include the following (World Federation of Neurosurgical Societies Spine Committee 2019):

- The following are the most effective measures for preventing spinal cord injuries linked to auto accidents:
    - The implementation of measures such as enacting and upholding laws against drunk driving, which mandate a 0.05 g/dL blood alcohol limit for all drivers;
    - The use of head restraints;
    - The use of seatbelts and kid passenger restraints;
    - The setting and enforcement of speed limits.
- The best interventions for the prevention of spinal cord injury associated with road traffic motorcycle accidents comprise the following:
    - Motorcycle helmets;
    - Motorcycle daytime running lights;
    - Designs of roads that keep autos and larger vehicles apart from people and two-wheelers. comprehensive traffic-calming strategies;
    - Graduated driver licensing laws.
- The following interventions are included in the prevention of SCI caused by falls:
    - Clear floors free of debris and loose carpets; Adequate lighting; Handrails and furniture at the right height; Window guards in high-rise buildings; Roof barriers;
    - Safe harvesting equipment. When appropriate, wheelbarrows.

Guidelines for the transportation and immobilization of patients with cervical spine trauma include:

- In the prehospital context, immobilization of patients over 12 years old who are at high risk of spinal cord injury (SCI) should involve the use of a hard cervical collar and a spinal backboard with straps or tape to immobilize the patient completely.
- Alert individuals with mild blunt trauma without penetrating damage and any spinal discomfort can be carried without being immobilized in the event of a human resource shortage.
- As soon as possible, patients with acute traumatic spinal cord injuries should be sent to the primary hospital facility for SCI treatment.
- For alert, asymptomatic individuals, collar immobilization may be stopped after arrival at the hospital.
- Following a negative high-quality C-spine CT scan, an alert, symptomatic patient may no longer require in-hospital collar immobilization.

Guidelines for closed reduction of cervical spine fractures:

- Awake patients with partial injuries are better candidates for a closed reduction if one is attempted.
- There is no evidence that closed reduction of cervical locked facets is more beneficial than open reduction.
- Pre-reduction MRI and open reduction should be chosen when attempting a reduction in patients who have lost consciousness.
- Surgical reduction and prompt anterior decompression are preferable options in the event that a closed reduction attempt is unsuccessful.

- Although most publications recommend it should be done as soon as feasible, the ideal period for a closed reduction is not well established.
- Following closed reduction, all patients ought to undergo surgery to achieve stabilization and fusion. An anterior, posterior, or mixed anterior and posterior route may be used for this procedure.

Guidelines for radiologic evaluation of upper cervical trauma include the following:

- Cervical CT is the first study to be performed for cervical spine screening in patients whose history and physical examination results raise suspicions of cervical spinal trauma. It is crucial for diagnosis and surgical planning.
- Transverse atlantal ligament disruption and instability in C1–C2 may be indicated by an anterior atlanto-dental interval (AADI) > 3 mm or a posterior atlanto-dental interval (PADI) < 13 mm.
- In patients with cervical injuries, preoperative 3D CT scanning should be carried out to rule out anatomical bone abnormalities before to the implantation of screws at the upper cervical spine.

Suggesations for occipital condyle fractures:

- For the treatment of occipital condyle fractures (OCF), Mueller et al. (2012)'s classification scheme would be better.
- For OCF diagnosis and treatment, CT imaging is the best option.
- To evaluate the stability of OCFs and determine the integrity of the craniocervical ligaments, MRI is advised in addition to CT scan.
- For OCFs without atlanto-occipital dislocation (AOD), conservative care should be given priority over surgical care.

Guidelines for atlanto-occipital dislocation injuries:

- In patients suspected of having atlanto-occipital dislocation (AOD), CT may be sufficient to define the condylo-C1 interval (CCI).
- An atlanto-occipital dislocation may be suspected in cases of severe traumatic brain injury (TBI), lower cranial nerve impairments, and/or spinal cord injury.
- Patients with AOD should have occipitocervical fixation surgery if their overall status is stable.
- Cervical traction is not advised for AOD.

Guidelines for atlas fractures:

- In order to determine the type of fracture and the integrity of the transverse atlantal ligament (TAL), treatment for isolated atlas fractures should be based on CT and MRI criteria.
- Most atlas fractures are stable and respond well to conservative management.
- Any "unstable" atlas fracture as well as atlanto-occipital instability and an intraligamentous TAL rupture are indications for surgery for atlas fractures.

Guidelines for odontoid fractures:

- An anterior atlanto-dental interval (AADI) > 3 mm in adult patients with odontoid fractures suggests disruption of the transverse atlantal ligament (TAL) and instability in C1–C2, whereas an AADI $\geq$ 5 mm suggests rupture of the transverse ligament and accessory stabilizing ligaments.
- If type 2 odontoid fractures are fixed with a posterior C1 lateral screw in conjunction with a C2 pedicle/laminar screw, the following factors increase the risk of fracture nonunion: advanced age, prolonged duration, and preoperative separation of the odontoid fracture > 4 mm.
- The "gap" in the fracture and the time between injury and operation are important predictors of fusion failure in anterior odontoid screw fixation.

Guidelines for Hangman Fractures:

- In addition to a CT scan, an upright X-ray taken under physician supervision may be helpful for hangman's fractures.
- Surgery is advised for hangman's fractures of Levine type 2A.
- Due to its complexities, conservative therapy for hangman's fracture should be done with a rigid collar rather than with Halovest.
- Levine type 3 hangman's fractures may require both anterior and posterior surgery.

Treatment recommendations for combined atlas and axis fractures are as follows:

- No superior evidence exists for combination atlas–axis fractures.
- In the majority of C1–C2 combination fracture cases, external immobilization is employed.
- The following are suggestions for the classification of subaxial cervical spine injuries:

- The Subaxial Injury Classification (SLIC) system is a safe and useful tool for directing subaxial cervical spine injury therapy. The SLIC score (morphology, neurology, and disco-ligamentous complex—DLC) and the selected course of treatment have a high degree of agreement (>90%).
- We also recommend the use of MRI to obtain a more accurate categorization of subaxial fractures.

Strategies for managing injuries to the subaxial cervical spine include:

- Treating C1 hangman's fractures with C2–C3 angulation of $\geq$11 degrees and C1 type 2 odontoid combination fractures with an atlanto-dental interval of $\geq$5 mm with surgery.
- For injuries with a SLIC score of less than 3, nonsurgical treatment with a rigid collar for 6–12 weeks is advised.
- Early surgery is advised for injuries with a SLIC score greater than 4.
- Anterior operations are advised for major anterior column injuries.
- Surgery is indicated for stable incomplete impairments with significant spinal canal disruption or for growing neurological deficits.
- Patients with significant dislocation (complex) injuries and those in need of multilevel corpectomy should be evaluated for additional posterior procedures.
- There is disagreement over the recommendation to do posterior procedures on patients who have ankylosing spondylitis and osteoporosis.

Recommendations for traumatic locked facets:

- Preoperative MRI is advised in the management of locked facets if a posterior approach is being investigated.
- Traction aids in immobilizing the shaky section and could facilitate reduction.
- Anterior surgical procedures are sufficient for effective therapy of most acute ($\leq$3 days) locked aspects.
- When an anterior approach is impractical, a posterior technique is recommended for lower cervical locked facets with no or minimal disc prolapse, as well as chronic locked aspects lasting more than two weeks.

The following are recommendations for paediatric cervical spine injuries:

- MRI is required for children without abnormal X-ray or CT scan results who have neurological spinal cord symptoms.
- For irreducible rotatory atlanto-occipital dislocation, surgery is recommended.
- If a child under five years old has a cervical spine fracture or dislocation and there is no surgical rationale, a Minerva cast may be utilized in place of a halo.

Guidelines for cervical trauma-related vertebral artery damage include the following:

- Computed tomographic angiography (CTA) should be used as a screening method in certain individuals who have experienced blunt cervical trauma and have fractures close to the vertebral artery course.
- Conventional catheter angiography is advised if CTA is abnormal for vertebral artery injury (VAI) and endovascular therapy is a possible course of treatment.
- The choice of therapy—anticoagulation therapy versus antiplatelet therapy versus no treatment—for patients in whom endovascular treatment for VAI is not advised should be tailored to the patient's specific characteristics of the vertebral artery injury, the accompanying injuries, and the risk of bleeding.
- Since the function of endovascular therapy in VAI is yet unclear, no advice can be given regarding its application in treating VAI.

7.2.2. Craniocervical Fracture and Dislocation

Occipital Condylar Fractures

Classifications of occipital condylar fractures are shown in Table 2.

Jefferson Fracture

This fracture (Figure 4) was first described by Sir Geoffrey Jefferson (Jefferson 1920). Typically, it is a four-point (burst) fracture of the C1 ring, yet the term is presently regularly utilized to incorporate the more frequent three- or two-point fractures of the arches of C1 (the most delicate of the vertebra) (Papadopoulos 1993; Alker et al. 1975). The most common cause of Jefferson fractures are diving accidents. There is a 41% possibility of a related C2 break. In children, it is essential to separate a C1 fracture from the standard synchondroses. A fracture may additionally happen through the unfused synchondrosis.

Table 2. Classifications for evaluating occipital condylar fractures.

Classification	Type	Description	Stability	Treatment
Anderson and Montesano (1988)	I	Comminuted: minimal/no displacement	Stable	Collar
	II	Direct trauma with basilar skull fracture	Stable	
	III	Avulsion fracture involving the alar ligament	Unstable	Surgical or halo fixation
Tuli et al. (1997)	1	Nondisplaced	Stable	Collar
	2A	Displaced, ligaments intact	Stable	
	2B	Displaced plus craniocervical instability	Unstable	Surgical or halo fixation

Source: Authors' compilation based on data from Anderson and Montesano (1988); Tuli et al. (1997).

Hangman's Fractures

- Vertical or angled fractures (Figure 8) of the C2 pars interarticularis, disengaging the posterior arch from the vertebral body.
- Usually brought about by hyperextension (MVA or diving accident); the posterior C1 arch as well as C2–C3 disc should likewise be assessed for injury.
- Most isolated fractures can be managed with a collar; displaced, unstable fractures can be dealt with halo immobilization.
- Surgery might be indicated if there is C2–C3 facet dislocation or if the patient has another significant unstable spinal injury.

Classification of hangman fracture is shown in Table 3.

Table 3. Levine and Edwards classification of hangman fractures.

Type	Description	Notes
I	<3 mm anterolisthesis, no angulation	Axial load and hyperextension
II	>3 mm anterolisthesis, angulation and disruption of posterior longitudinal ligament	Hyperextension and axial loading force linked to severe flexion
IIa	Horizontal fracture line and angulation without anterolithesis	Flexion, distraction; no or mild displacement but very severe angulation
III	Type I plus bilateral facet joint dislocation	Flexion, compression

Source: Authors' compilation based on data from Levine and Edwards (1985).

The commonest subluxation of the axis on C3 occurs regularly. Schneider et al. (1965) coined the term "Hangman's fracture" (HF), despite the fact that the most common causes of HFs nowadays are hyperextension and secondary flexion from MVAs or diving accidents. Nonetheless, modern-day HFs share some similarities with those seen in judicial hangings (where the submental position of the noose brings about hyperextension as well as distraction) (Wood-Jones 1913).

Craniovertebral Junction Dislocations

CVJ dislocations are uncommon and are categorized as atlantoaxial subluxation without fracture, vertical atlanto-axial dislocation (vertical AAD), and joint vertical dislocations (vertical AOD and vertical AAD). Typically occurring in children and adolescents, traumatic posterior atlantoaxial subluxation can be accompanied by odontoid fracture (Fielding et al. 1978). It is a rare occurrence in elderly patients and adults and has only been described in eight case reports (Fox and Jerez 1977; Haralson and Boyd 1969; Jamshidi et al. 1983; Sassard et al. 1974; Sud et al. 2002; Wong et al. 1991; Yoon et al. 2003). The C2 odontoid process/dens is physically joined with the C1's anterior arch and the TAL's synovial joint, and thanks to its special anatomical configuration, it can act as a pivot for the rotation of C1 above C2. As a result, it is extremely difficult to achieve the dislocation of the

C2 odontoid process from the C1 arch, meaning that this traumatic lesion is associated with extensive ligament damage. This unusual injury's nature can be confirmed by radiological analysis using an X-ray, CT scan, and 3D MRI. In the acute stage, neuroimaging is helpful to accurately define the ligament damage and to prevent the presence of a traumatic haematoma in the spinal canal. Spinal cord injury was not severe in any of the cases discussed in the text. Three patients had no neurological deficits (Yoon et al. 2003; Haralson and Boyd 1969), and the remaining five displayed mild or temporary motor weakness (Sud et al. 2002; Wong et al. 1991; Yoon et al. 2003). This illustrates that before spinal cord compression occurs at the C1-2 junction, there is a significant amount of free space. The vast extent of the spinal canal suggests that there can be a lot of instability in this area. Amir Jamshidi et al. (Jamshidi et al. 1983), in 2009, hypothesized that severe rotating hyperextension of the neck is the basis of these uncommon and traumatic events. The pathophysiology of the injury is still under debate. Rotating subluxation is caused by general damage and injury to the C1-2 lateral facet joints. Currently, clinical treatment is debatable and is being discussed. It is clear that the reduction of dislocation is difficult when the patient is alert (Jamshidi et al. 1983). This is because of extent of ligament damage.

Atlanto-Occipital Dislocation (AOD)

Until recently, treating this kind of injury was unusual because most patients would either die at the site of the accident or right away after reaching at the hospital due to traumatic brain stem injury (Consortium for Spinal Cord Medicine 1997). Strong resuscitative measures taken on the scene have transformed AOD into a physical problem that may be treatable (Consortium for Spinal Cord Medicine 1997). Untreated AOD is associated with extreme morbidity and mortality (Hagen 2015). Clinically, individuals may exhibit complete tetraplegia, respiratory distress, or neurological normalcy (Hagen 2015). When a patient is conscious, it is occasionally possible to hear them complain of occipital pain, and lower cranial nerve paralysis can be seen (Schneider et al. 1965). Brown–Sequard syndrome, focal neurological deficits, or Bell's palsy have been described with this injury (Schneider et al. 1965). Increased signal intensity in and around the tendons on MRI may indicate tendon injury. Numerous symptoms, ranging from mild neurological issues to bulbar–cervical separation leading to respiratory arrest and death, may be present.

Atlantoaxial Dislocation (AAD)

This term indicates a loss of continuity between atlas and axis (C1 and C2) (Figures 5 and 6). The expected distance between the anterior arch of C1 and the dens is <2–3 mm in adults and <5 mm in children. The causes of AAD can be traumatic or secondary to specific infections. Following a comminuted C1 fracture, the cross over tendon is currently attached to a bone segment. The spine is weak, and the C1 lateral mass has been dislodged. Rheumatoid arthritis, Down syndrome, and Morquio syndrome are related disorders (Consortium for Spinal Cord Medicine 1997). Patients with the aforementioned diseases should be carefully assessed, and these conditions should be excluded in individuals with deficits limited to the upper cervical spinal cord. A traumatic atlanto-axial dislocation alone, without further displacement, is uncommon (Consortium for Spinal Cord Medicine 1997). Treatments include posterior fixation and fusion.

7.2.3. Subaxial Cervical Spine Injuries

These will, in general, happen in young patients because of a car accident or some sports injury (Figures 3 and 9–11). In this age group, if cervical immobilization is used, one-level arthrodesis be employed to avoid further injury and preserve the range of motion. Similarly, a posterior approach may be used depending on the number of levels involved.

- The most regularly affected levels are C5 and C6.
- The three categories of injury are compression fractures, burst/distraction fractures, or translational injuries.
- These injuries may affect either bony tissue or soft tissue.

A schematic drawing from Argenson et al.'s classification of lower cervical spine injuries is shown in Figure 3 (Argenson et al. 1997).

7.2.4. Dorsolumbar Fracture and Sacral Fracture

Injuries to the Dorsal, Dorsal–Lumbar, and Lumbar Spine

Illustrations in Figure 12 show the AO/Magerl classification of dorsolumbar spinal injuries.

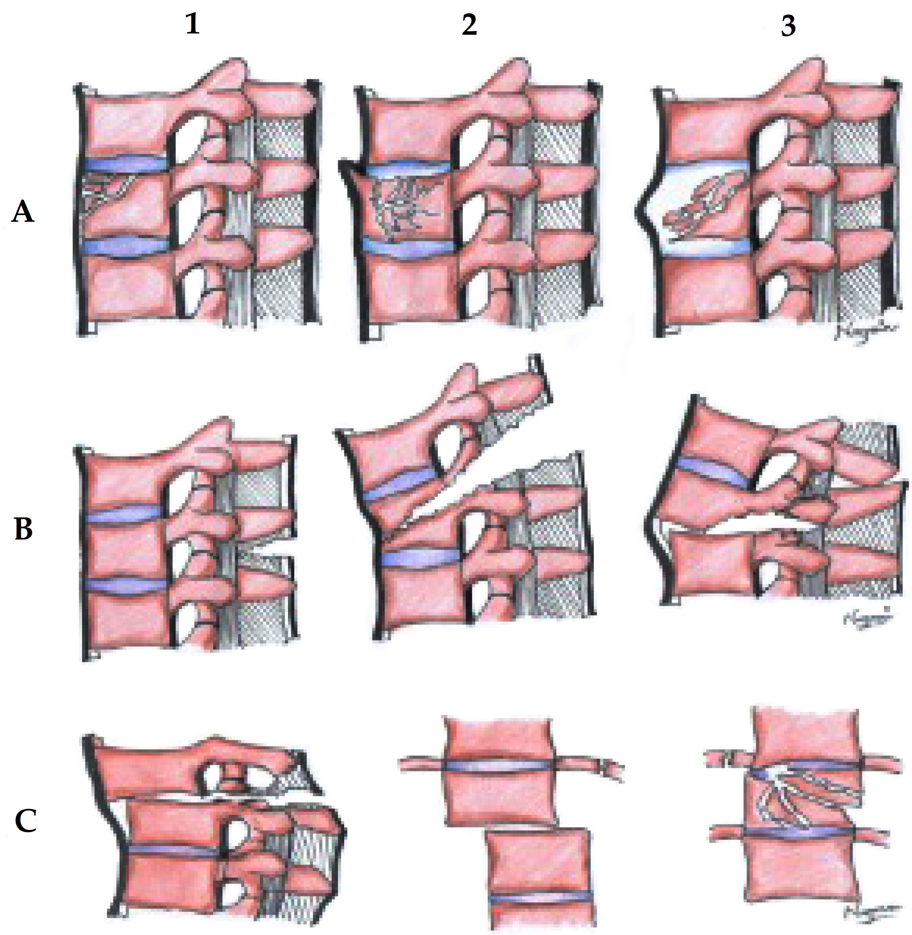

Figure 12. Illustrations showing AO/Magerl classification of dorsolumbar spinal injuries showing three main types ((**A**) compression, (**B**) distraction, (**C**) torsional injury) and their subtypes (**1**, **2** and **3**). Source: Figure by authors.

Injuries to the dorsal (D2 to D10), dorsolumbar (D11 to L2), and lumbar (L3 to L5) spine (Figures 12–18) are managed separately, as the bone arrangement, biomechanics, and neurological functions of each of these segments are unique. Even though the fracture mechanisms that happen in these segments are similar, the clinical interpretation and, thus, the treatment and prognosis shifts as indicated by the degree of the injury.

Figure 13. (**A**,**B**) X-ray and (**C**,**D**) MRI of dorsal spine showing unstable fracture and posterior dislocation of D11 with cord compression. Source: Figure by authors.

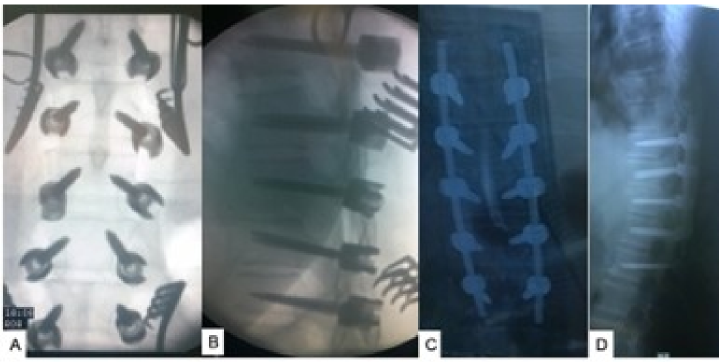

Figure 14. (**A**,**B**) Intraoperative X-ray of fracture reduction, stabilization (with pedicular screws and rods), and fusion of D11 fracture and dislocation in patient in Figure 13. (**C**,**D**) Postoperative X-ray of patient in Figure 13. Source: Figure by authors.

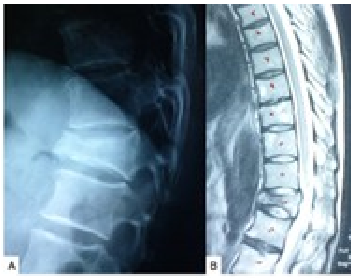

Figure 15. (**A**) X-ray and (**B**) MRI of dorsal spine showing relatively stable D12 compression fracture which was managed conservatively. Source: Figure by authors.

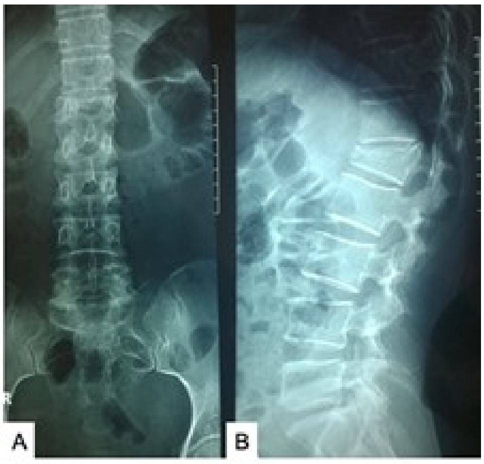

Figure 16. (**A**,**B**) X-ray of dorsolumbar spine showing L1 wedge fracture. Source: Figure by authors.

Figure 17. (**A**,**B**) X-ray and (**C**) MRI of dorsolumbar spine showing unstable L1 wedge fracture with compression of conus medullaris. Source: Figure by authors.

Figure 18. (**A**) X-ray lumbar spine showing L3 compression fracture and (**B**) MRI of lumbar spine showing L3 compression fracture with cauda equina compression. (**C,D**) Postoperative X-ray after decompression, fusion, and stabilization of L3 fracture. Source: Figure by authors.

Thoracic Spine Injuries

Thoracic spinal injuries are shown in Figures 12–15. Fifteen percent of all spinal cord injuries are to the thoracic spine (Tator 1994). Even though the the thoracic spine is difficult to damage, the spinal canal is extremely vulnerable to injury and has the worst chance of recovering in a way that will be useful. Solely 10% of dorsal vertebral body injuries are caused by spinal cord injuries. In contrast, the cervical spine comprises 39% of this total. The degree of the neurological deficits commonly associated with injuries to this area is represented by a weak spinal-canal-to-spinal-cord ratio and weak CSF flow. The sternum, the back, the costovertebral tendons, the ribs, the chest enclosure and divider muscles, and this region's musculature all serve to stabilize the spinal column here. In addition to strengthening the thoracic region, they also limit the amount of physiological growth that is permitted. For instance, the ribcage restricts expansion movement by around 70% (White and Panjabi 1979). The fact that a healthy ribcage is associated with a fourfold rise in the compression resistance of the adjacent vertebral segment confirms that the ribcage also gives stiffness to the spine (White and Panjabi 1979). The upper thoracic region's flexion and expansion are also constrained by the ribs, and the back structures mostly prevent expansion (White and Panjabi 1979).

Injury to the Lower Spinal Cord and the Cauda Equina: The Role of Surgical Decompression

Individuals with lesions to the lower spinal cord and the cauda equina may sustain total or partial injury (Germon et al. 2015). Decompressive surgery typically does not produce positive results in patients who have complete injuries for longer than 48 h (Germon et al. 2015). Both early and late decompression have been shown to improve function, which suggests that the role of time is yet unknown.

Injury to the T11–L1 Segment

Anterior and posterohorizontal methods of decompression are used for this part of the spine (Figures 16 and 17). The main ways to deal with T11 lesions include a thoracotomy cut, while the involvement of T12 and L1 requires a thoracoabdominal surgical approach (Germon et al. 2015). This methodology might be used in incomplete SCI or compressive cauda equina lesions (Germon et al. 2015). Where there is evidence of posterior neural compression, this system is contraindicated. Postero-lateral surgeries incorporate the extracavitary and costotransversectomy technique (Gokaslan et al. 1998). These procedures also necessitate the excision of at least one rib and the use of cross-over cycles to gain access to the lateral vertebral body. Posterior instrumentation can be refined with the two procedures, even though the extracavitary approach considers the perception of the level inverse to the pedicle base (Gokaslan et al. 1998). With this form, it is difficult to excise of large midline bone pieces. These methodologies are related to the danger of inadequate decompression because of the presentation, and there is a high risk of deformation without anterior bone joining (Germon et al. 2015).

Injury to the L2–L4 Segment

In injuries to L2-L4 segment (Figure 18) functional recovery is varied because these lesions cause damage to the cauda equina. If there is neurological compression, a laminectomy with transpedicular decompression might offer sufficient exposure (Gokaslan et al. 1998). With an S-shaped incision from the tip of the 12th rib into the lower abdomen, a retroperitoneal surgery enables the viewing of the peritoneum and the contents of the abdominal cavity (Zindrick et al. 1986). The lumbar segmental vessels are then linked, and the psoas muscle is subsequently mobilized to give caudal access from L2 to the anterolateral region of the lumbar spine (Zindrick et al. 1986). After instrumentation, a vertebrectomy plus grafting is accomplished (Gokaslan et al. 1998). Maintaining physiological lordosis in this area is important since losing it can result in postural abnormalities and a flat back.

Injury to the L5 Segment

At L5, neural decompression with a transpedicular approach can be successfully accomplished following a laminectomy. In cases of extreme neural compression, a paramedian abdominal incision can be used to conduct anterior decompression. Through a retroperitoneal or transperitoneal approach, the lower lumbar spine may then be revealed. More extensive exposure is provided by the transperitoneal method, but it also involves mobilizing the large vessels and the plexus of the hypogastric nerve. An increased probability of impotence is correlated with the mobilization of the above structure. For L5 fractures, sacral fixation is required. Excellent fixation is provided by the utilization of posterior instrumentation with the positioning of pedicle screws laterally at 45° in the sacrum ala or medially in the first pedicle (Zindrick et al. 1986). To improve the build, sacral sublaminar wiring is often used. Patients are put in a lumbosacral orthotic system postoperatively.

Thoracolumbar Spine Trauma: WFNS Spine Committee Recommendations

Epidemiology and incidence (World Federation of Neurosurgical Societies Spine Committee 2020):

- The most frequent causes of thoracolumbar fractures are falls and traffic accidents.
- Including osteoporotic fractures, the annual incidence of TL fractures is around 30 per 100,000 people.
- Little is known about the true prevalence and epidemiology in poor nations.
- Low-velocity falls are becoming more common, particularly among the elderly.
- In industrialized nations, the mortality rate following a spinal injury is declining. This is particularly true for spinal injuries sustained in auto accidents because of advancements in traffic laws and vehicle safety.
- Many vertebral fractures occur in youngsters;
- The death rate from thoracolumbar trauma is relatively high in male older patients.

Radiological diagnosis and classification:

- In clinical practice, the revised AO classification and TLICS should be utilized as trustworthy classifications of traumatic thoracolumbar fractures.
- Recent research indicates that the new AO classification may be more beneficial in the treatment of thoracolumbar fractures even if it is more complicated.
- In the event that an MRI or CT scan are not accessible, AP and lateral standard radiographs may be taken.
- Although MRI should be taken into consideration, CT still plays a significant role in the assessment of trauma because it is unable to accurately show the disco-ligamentous complex.
- The most popular advanced imaging technique is magnetic resonance imaging (MR imaging), which is the preferred tool for disco-ligamentous abnormalities, spinal cord abnormalities, and other disorders related to spinal trauma.

Indications for nonsurgical and surgical treatment:

- It is preferable not to treat AO type B and C fractures conservatively.
- There is no clinical evidence that bracing for the conservative treatment of TL fractures will improve the outcome.
- AO type A2, A3, and A4 fractures can be treated conservatively if there is no significant kyphotic angulation, significant vertebral body collapse, or canal compromise with neurological impairment.
- It is preferable to operate on fracture dislocations and instances with substantial instability (TLISS classification score > 5).
- Although there is insufficient data to support it, surgical decompression and stabilization may be considered for burst fractures resulting in neurological impairments.
- Conservative or surgical methods can be used to treat burst fractures in the absence of neurological impairments.

Surgical approaches for thoracolumbar fractures:

- Short-segment posterolateral pedicle screw fixation is usually enough for burst fractures.
- In order to strengthen the construct in cases of burst fractures of the thoracolumbar junction, a fracture-level screw should be used. If it is not possible to incorporate a fracture-level screw, long-segment fixation ought to be used.

- There is no proof that fusion is necessary when utilizing long-segment screws because the results are the same with or without fusion.
- The clinical results for TL burst fractures are insensitive to the choice of anterior or posterior approach.
- There is insufficient data to conclude that nonoperative treatment for burst fractures of the lumbar and thoracic spine is superior in terms of clinical results.
- Since the data points to comparable clinical results, minimally invasive methods of treating thoracolumbar burst fractures may be taken into consideration.
- Non-fusion surgery for thoracolumbar burst fractures has the following benefits over fusion surgery: less donor site problems, shorter recovery times, and less bleeding.
- There is no statistical evidence that suggests regional kyphosis will worsen following non-fusion surgery.

Factors influencing surgical results:

- After thoracolumbar burst fracture surgery, obesity may exacerbate segmental kyphosis.
- Bad things seem to happen to those who are older.
- Poor results are predicted by smoking, comorbidities, and long-term high-dose steroid use.
- It is not recommended to rule out early surgery due to polytrauma or high injury severity scores.
- Kyphotic deformity may worsen if there is a greater than 50% loss of anterior vertebral body height.
- Since it greatly affects the result, it is crucial to identify injuries to the posterior longitudinal ligament complex.
- Burst fractures with sagittal–transverse canal diameter ratio <0.40 are substantially related with brain damage and outcomes.
- After surgery, Cobb's angle greater than 10.5° may indicate unfavorable results.

Post-traumatic kyphosis following thoracolumbar fractures:

- Untreated, unstable burst fractures are the most frequent cause of post-traumatic kyphosis.
- There is no specific kyphosis angle that would require surgery in the treatment of post-traumatic kyphosis. Instead, one must evaluate the global sagittal equilibrium.
- Remarkable kyphosis correction can be obtained with posterior surgery, with minimal blood loss and complications.

Sacral Fractures

These are uncommon and are regularly brought about by shear forces (Jefferson 1920). They can harm sacral roots and plexus and influence pelvic and spinopelvic stability. Injuries beneath S2 ought not to influence ambulation; however, they might be unstable and lead to trauma that improves after careful intervention (Bellabarba and Bransford 2015).

8. Medical Complications of Spinal Injuries

8.1. Cardiovascular Complications

Direct cardiovascular (CV) issues after SCI are caused by the disruption of the autonomic nervous system and the loss of interaction between receptor organs and brainstem regions (Hagen 2015). SCI-related CV problems encompass pulmonary embolism, deep vein thrombosis (DVT), autonomic dysreflexia (AD), orthostasis, cardiac arrhythmia, abnormalities of the thermoregulatory system, and orthostasis (PE) (Hagen 2015). The most common CV abnormalities in acute SCI are bradycardia and hypotension, both of which are brought on by a lack of sympathetic tone (Tator 1994).

8.2. Orthostatic Hypotension

Orthostatic hypotension is a reduction in circulatory effort caused by an alteration in body posture toward the upright. There are several warning signs, including drowsiness, unsteadiness, syncope, and pallor. People with a complete injury above the T6 level are prone to orthostasis. The orthostatic hypotension component includes preganglionic sympathetics in the spinal cord and interferes with the sensory CV contribution to the brainstem. Aortic and carotid baroreceptors sense a decrease in blood pressure when the patient stands up. This can often result in an increase in sympathetic tone, causing vasoconstriction and tachycardia. But the efferent route is blocked in SCI, preventing an increase in sympathetic outflow (i.e., norepinephrine and epinephrine). Due to these factors, the pulse only slightly increases as a result, which is insufficient to counteract the decline in blood pressure. Additionally, venous pooling occurs, which reduces venous return to the right atrium and, as a result, decreases cardiovascular output. Orthostasis's adverse effects depend more on cerebral blood flow than on high blood pressure (Gonzalez et al. 1991). As spinal postural reflexes that generate vasoconstriction develop and cerebrovascular autoregulation of dissemination improves in response to low perfusion pressures, this condition gradually worsens (Corbett et al. 1971).

8.3. Thermoregulation

Body temperature is usually under hypothalamic control (i.e., thermoregulation). When the core temperature needs to be raised, the hypothalamus may utilize shivering and vasoconstriction, which increase heat production and decrease heat loss. Similar to sweating, vasodilation reduces temperature by boosting heat loss. SCI reduces the peripheral nervous system's capacity to direct the hypothalamus. Individuals with lesions above T5 frequently exhibit poikilothermy, or difficulties in responding to local temperature. For instance, a person with a high level of SCI may have a high core temperature while in a hot environment or outdoors, which could be misconstrued for an infectious source-induced fever. Therefore, it is critical to teach patients self-protection strategies against both hypothermia and hyperthermia.

8.4. Autonomic Dysreflexia

Autonomic dysreflexia, also called autonomic hyper-reflexia, is a syndrome that occurs in cases with SCI above the T6 level and leads to an uncoordinated sympathetic response. It is is a medical emergency that needs prompt detection and care. The rate ranges from 48% to 85% in SCI patients, and it is caused by an exaggerated sympathetic response below the level of injury that the patient is unaware of as they lack feeling (Lindan et al. 1980; Erickson 1980). Overdistention, frequently of the bladder, is the primary cause of these dangerous hypertensive episodes. They may also result from inappropriate catheterization or a clogged indwelling Foley catheter. Any injury, regardless of the cause, such as constipation, a pressure ulcer, tight clothing, an ingrown toenail, or a fracture, may worsen the disease, as these causes and trigger an episode of dysreflexia. A lack of compensatory descending parasympathetic stimulation and intrinsic post-traumatic hypersensitivity cause the sympathetic response to be exaggerated. The outcome is local vasoconstriction despite maximum parasympathetic vasodilatory efforts and a steep rise in blood pressure. Reflex bradycardia might be caused by this increased blood pressure; however, it is not sufficient to decrease it altogether. When it comes to older adult patients, who may naturally be hypertensive, monitoring hypertension is especially important. AD can cause headaches, excessive sweating, and piloerections due to intentional stimulation of hair follicles. Flushing may occur above the level of injury. The patient typically complains of nasal congestion and discomfort. Retinal discharge, myocardial localized necrosis, subarachnoid or intracerebral drain, seizures, and potentially death are all expected complications of AD. AD may occur soon after injury and exists in many patients within six months of injury. Within one year of injury, 92% of patients are affected by AD (Lindan et al. 1980).

8.5. Deep Vein Thrombosis

The most well-recognized CV-related risk after SCI is deep vein thrombosis (DVT). Its occurrence fluctuates somewhere in the range of 8% and 100% depending on the analytic tests used (Green et al. 1992, 2005). Recent information shows that the rate of DVT might be decreasing, maybe because of prophylaxis (Green et al. 2005; Ragnarsson et al. 1995). Virchow's triad, namely venous stasis, vascular injury, and hypercoagulability, comprises the risk factors for the development of DVT in SCI. A lower appendage fracture, obesity, older age, diabetes, a history of past thrombosis, and vascular disease are additional risk factors. Individuals with tetraplegia and those who have neurologically recovered from SCI have DVT more frequently. The onset often occurs within fourteen days, and after two months, the frequency starts to decline. One of the leading aetiologies of death in patients during the intensive phase of SCI is PE, which occurs in 1–7% of those with the condition (Green et al. 2005). Most incidences occur in the lower appendages' deep veins. The degree or severity of SCI has no bearing on PE. After SCI, DVT prevention is crucial. Clinical testing is important but less effective in SCI patients due to the absence of some of the common signs and symptoms, like sensitivity to palpation. The degree of apoplexy is inversely correlated with the perimeter of the legs; in any event, a 1 cm difference between perimeter of the two lower limbs at the calf or thigh should arouse suspicion. Clinical recommendations for the prevention and management of DVT in SCI have been made available by the Consortium for Spinal Cord Injury (Consortium for Spinal Cord Medicine 1997). A plan for anticoagulant prophylaxis and mechanical prophylaxis—i.e., pneumatic pressure devices—is suggested. Usually, it is advised to employ mechanical prophylaxis when the patient is sleeping, 24 h a day. If there is no evidence of brain injury, or coagulopathy, anticoagulant prophylaxis with either unfractionated or low-molecular-weight heparin should begin within 72 h of the initial injury. The duration of DVT prevention depends on the kind and severity of SCI as well as any concurrent clinical conditions that are present (Consortium for Spinal Cord Medicine 1997). Patients in whom anticoagulant prophylaxis has failed or who are contraindicated to anticoagulation may be eligible for the placement of retrievable vena cava filters; nonetheless, this is by no means a replacement for pharmacological thromboprophylaxis. When using mechanical coagulation, lower appendage preparation and exercise are typically continued for 48–72 h following the implementation of the appropriate therapeutic treatment.

8.6. Respiratory Complications

Following SCI, respiratory complications such as atelectasis, pneumonia, and aspiration are very common (Jackson and Groomes 1994). All things considered, the older the patient, the higher the neurological level, and the more severe the neurological injury, the more likely these conditions are to arise. Patients with SCI are more vulnerable to the development of respiratory complications due to prior respiratory problems (e.g., having a history of smoking, ongoing bronchial asthma, chronic obstructive pulmonary disease, and excess weight). Several factors may contribute to inadequate respiratory function during severe SCI. Severe SCI causes the respiratory muscles to lose all or some of their mobility. Patients who have an injury at or above the C3 level are typically unable to breathe on their own at first and need artificial ventilation. Mechanical ventilation may be necessary for a while for injuries below the C3 vertebra. These individuals may not be able to empty their lungs of secretions because their abdominal muscles are losing mobility to varying degrees, which affects their ability to cough. Thoracic SCI is typically associated with chest injuries, such as rib fractures, hemopneumothorax, and lung injuries, which can result in acute respiratory failure. To prevent any potential respiratory complications following severe SCI, careful respiratory management is essential. Following SCI, a preventative respiratory treatment program should start as soon as possible. For those with severe SCI, breathing medications with saline solutions or bronchodilators should be available. Significant components of respiratory management include dynamic chest stretches and exercises, incorporating percussion, clearing lung secretions as needed, and assisted coughing techniques. Regularly placing the patient in the Trendelenburg and reverse Trendelenburg positions, and side-lying positions, if possible, should help with perfusion. Due to the weakness of the cough in people with high-level SCI, assisted coughing is provided by different manual techniques (such as the quad cough) or by using mechanical insufflation–exsufflation equipment. When treating a patient with a freshly implanted inferior vena cava filter, caution should be taken when using the quad coughing method. In contrast to suctioning, people with SCI prefer mechanical insufflation–exsufflation for the treatment of their secretions. Strengthening the abdominals as well as other innervated accessory muscles of respiration should begin as soon as possible after injury (Jackson and Groomes 1994; Garstang et al. 2000). During mechanical ventilation, the abdominal muscles may weaken quickly, and if mechanical ventilation stops, they will not be able to provide enough aspiratory work.

9. Prognosis for Recovery and Prediction of Outcome

The primary factor in determining how well the injured brain will recover neurologically is the actual assessment. Once the patient's physical condition has been identified, it will be possible to predict when the injury will be resolved. The use of radiographic and electrodiagnostic testing, as well as neurological assessment, are just a few clinical techniques that can be used to predict neurological recovery after SCI (Kirshblum and O'Connor 2000). The neurological assessment's determination of whether a person has a neurologically complete or incomplete injury is the primary predictor of recovery (Kirshblum and O'Connor 2000). The degree of the underlying injury, the underlying muscular strength, and the subject's age are other significant assessment factors (Theisen et al. 2014). Recovery from spinal injuries below the cervical spine that cause paraplegia is been the same as recovery from injuries causing quadriplegia, although some predictions regarding future recovery are comparable (Theisen et al. 2014). Numerous studies have linked MRI observations to neurological status and recovery following SCI and discovered that the kind and degree of MRI change correlate with the severity and prognosis of the injury (Ditunno et al. 2002). In the immediate aftermath of an SCI, numerous electrophysiological tests are used to assess the severity and level of the SCI and predict neurological and functional outcomes (Ditunno et al. 2002). Techniques for improving clinical and neuroradiological tests include nerve conduction studies, late responses (H-reflex and F-wave), motor evoked potentials, somatosensory evoked potentials, and sympathetic skin responses (Theisen et al. 2014).

10. Pharmacological and Surgical Procedures to Increase Recovery and Regeneration

After the underlying injury, various changes happen inside the spinal cord that prevent the return of function. In recent decades, various pharmacological techniques for the treatment of SCI to improve recuperation have emerged. No significant differences in neurological or functional results were found between the two regimens at 6 months or 1 year; notwithstanding this, the maximum tolerated dose was below the therapeutic threshold. Patients with cauda equina injuries and injuries to the cervical spine were not included in the study. A month and a half, half a year, and a year later, the study revealed that methyl prednisolone (MP) given within 8 h of injury increased neurological recovery, even though practical recovery was not explicitly taken into account. Patients treated after 8 h exhibited no beneficial effects. Enhancing blood flow to the spinal cord, preventing lipid peroxidation, being a free radical scavenger, and having a moderating capacity are all aspects of MP activity. Tirilizad is a potent steroid that has no glucocorticoid effect, providing the positive effects of MP (such as lipid peroxidation and the movement of cancer prevention agents) without the negative effects. According to the study, patients who are treated within three hours of injury should receive steroids for 24 h, and those who are treated between three and eight hours after the accident should have them for 48 h. There are several recent reports that call into doubt the current normal use of steroids, and this more recent practice using 48 h of therapy has not been widely accepted. In the central nervous system (CNS), GM-1 ganglioside (Sygen) is present in high concentration and is involved in the structures of a large portion of cell membranes. It is hypothesized that GM-1 ganglioside can stimulate and protect protein kinases, inhibit glutamate-induced neuronal excitotoxicity, increase neurite outgrowth, and reduce CNS tissue damage. An unpublished small study that treated individuals within 48 h of injury for an average of 26 days found greater mean recovery after 1 year, with somewhat better recovery for muscles that were weak at the time of the study (Geisler et al. 1991). At the primary endpoint of 26 weeks, no significant effects were seen in the entire group of patients being evaluated (Bracken 2001). In a pharmacological trial, fampridine-SR, a long-acting version of 4-aminopyridine (4-AP), was used on people with chronic incomplete spinal cord injury. The potassium (K+) channel blocker 4-AP binds to internodal axonal K+, improving nerve conduction. Phase 2 research on SCI showed trends toward improvement in pain and stiffness (Gokaslan et al. 1998; Davis et al. 1990; Segal and Brunnemann 1997). A phase 1 clinical trial of electrical stimulation was carried out on 10 participants with neurologically complete SCI. Their injuries ranged between C5 and T10 in severity, and MRI did not reveal any evidence of other lesions. Pain scores assessed using a visual analogue scale indicated a reduction in their level of pain at one year, and improvements in light touch and pinprick sensitivity as well as in the strength of some muscles were found (Shapiro et al. 2005). The Food and Drug Administration approved the enrolment of 10 more severe SCI patients after it was determined that the use of this therapy in patients with severe SCI was safe and could be useful. Additional results and preliminary clinical reports have shown similar outcomes.

11. Conclusions

The progressive degradation of both vascular and brain tissue, which undermines the anatomical substrate required for neurological recovery, is linked to all cellular and molecular alterations and events. These neurodegenerative mechanisms increase the need for various therapeutic approaches to lessen the harm brought on by secondary injury. These approaches are frequently based on knowledge of the pathophysiology of spinal cord injury, which aids in the development of systematic and multivariable therapies that facilitate functional recovery, prevent secondary injury, and promote regeneration. Spine injuries can be neurologically damaging. To avoid neurological worsening, prompt identification in the setting of multiple injury is important. Initial evaluation includes a thorough history and physical examination, which includes a thorough neurological assessment, in addition to airway safety and haemodynamic stability. The radiographic evaluation of life-threatening injuries starts in the emergency room. A high index of scepticism should always be maintained in order to recognize and treat such injuries, like atlanto-occipital dislocation. The most frequently injured area of the spinal cord is the cervical spine. The identification and treatment of injuries in this region are crucial due to the high morbidity and mortality rates associated with them. For certain types of injuries that, if effective, can be repaired without surgery, closed reduction can be explored.

Author Contributions: Conceptualization, methodology, validation, formal analysis, investigation, resources, data curation, M.M.R. and F.H.C.; writing—original draft preparation, M.M.R. and N.A.; writing—review and editing, visualization, supervision, F.H.C. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Conflicts of Interest: The authors declare no conflicts of interest.

References

Alker, George, Jr., Young S. Oh, Eugene V. Leslie, Judith Lehotay, Victor A. Panaro, and Edward G. Eschner. 1975. Postmortem radiology of head neck injuries in fatal traffic accidents. *Radiology* 114: 611–17. [CrossRef] [PubMed]

American Spinal Injury Association. 1992. *Standards for Neurological and Functional Classification of Spinal Cord Injury*. revised. Chicago: American Spinal Injury Association.

American Spinal Injury Association and International Spinal Cord Society. 2006. *International Standards for Neurological Classification of Spinal Cord Injury*, 6th ed. Chicago: ASIA and ISCS.

Anderson, Paul A., and Pasquale X. Montesano. 1988. Morphology and treatment of occipital condyle fractures. *Spine (Phila Pa 1976)* 13: 731–36. [CrossRef] [PubMed]

Argenson, Claude, F. de Peretti, A. Ghabris, P. Eude, J. Lovet, and I. Hovorka. 1997. Classification of lower cervical spine injuries. *European Journal of Orthopaedic Surgery & Traumatology* 7: 215–29. [CrossRef]

Bellabarba, Carlo, and Richard J. Bransford. 2015. Spinopelvic fixation. In *AOSpine Masters Series, Volume 6: Thoracolumbar Spine Trauma*. New York: Thieme.

Blackmore, C. Craig, Scott S. Emerson, Frederick A. Mann, and Thomas D. Koepsell. 1999. Cervical spine imaging in patients with trauma: Determination of fracture risk to optimize use. *Radiology* 211: 759–65. [CrossRef] [PubMed]

Bracken, Michael B. 2001. Summary statement: The Sygen (GM-1 ganglioside) clinical trial in acute spinal cord injury. *Spine* 26: S99–S100. [CrossRef]

Consortium for Spinal Cord Medicine. 1997. *Prevention of Thromboembolism in Spinal Cord Injury: Clinical Practice Guidelines*. Washington: Paralyzed Veterans of America.

Corbett, J. L., H. L. Frankel, and P. J. Harris. 1971. Cardiovascular reflex responses to cutaneous and visceral stimuli in spinal man. *The Journal of Physiology* 215: 395–409. [CrossRef]

Davis, Floyd A., Dusan Stefoski, and Jean Rush. 1990. Orally administered 4-aminopyridine improves clinical signs in multiple sclerosis. *Annals of Neurology* 27: 186–92. [CrossRef]

Denis, Francis. 1983. The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. *Spine (Phila Pa 1976)* 8: 817–31. [CrossRef]

Denis, Francis. 1984. Spinal instability as defined by the three-column spine concept in acute spinal trauma. *Clinical Orthopaedics and Related Research* 189: 65–76. [CrossRef]

DeVivo, Michael J. 2002. Epidemiology of traumatic spinal cord injury. In *Spinal Cord Medicine*. Edited by Steven Kirshblum, Denise I. Campagnolo and Joel A. DeLisa. Philadelphia: Lippincott Williams & Wilkins, pp. 69–81.

Ditunno, J. F., A. E. Flanders, S. Kirshblum, V. Graziani, and A. Tessler. 2002. Predicting outcomes in traumatic spinal cord injury. In *Spinal Cord Medicine*. Edited by Steven Kirshblum, Denise I. Campagnolo and Joel A. DeLisa. Philadelphia: Lippincott Williams & Wilkins, pp. 108–22.

Erickson, R. P. 1980. Autonomic hyperreflexia: Pathophysiology and medical management. *Archives of Physical Medicine and Rehabilitation* 61: 431–40.

Fielding, J. William, Richard J. Hawkins, Robert N. Hensinger, and William R. Francis. 1978. Atlantoaxial rotary deformities. *Orthopedic Clinics of North America* 9: 955–67. [CrossRef] [PubMed]

Fox, John L., and Alvaro Jerez. 1977. An unsual atlantoaxial dislocation: Case report. *Journal of Neurosurgery* 47: 115–18. [CrossRef]

Garstang, Susan V., Steven C. Kirshblum, and Kenneth E. Wood. 2000. Patient preference for inexsufflation for secretion management in spinal cord injury. *The Journal of Spinal Cord Medicine* 23: 80–85. [CrossRef] [PubMed]

Geisler, Fred H., Frank C. Dorsey, and William P. Coleman. 1991. Recovery of motor function after spinal-cord injury: A randomized, placebo-controlled trial with GM-1 ganglioside. *The New England Journal of Medicine* 324: 1829–38. [CrossRef] [PubMed]

Germon, Timothy, Sashin Ahuja, Adrian TH Casey, Nicholas V. Todd, and Am Rai. 2015. British Association of Spine Surgeons standards of care for cauda equina syndrome. *The Spine Journal* 15 S3: S2–S4. [CrossRef] [PubMed]

Go, B. K., M. J. DeVivo, and J. S. Richards. 1995. The epidemiology of spinal cord injury. In *Spinal Cord Injury: Clinical Outcomes from the Model Systems*. Edited by Samuel L. Stover, Joel A. DeLisa and Gale G. Whiteneck. Gaithersburg: Aspen Publishers.

Gokaslan, Ziya L., Julie E. York, Garrett L. Walsh, Ian E. McCutcheon, Frederick F. Lang, Joe B. Putnam, David M. Wildrick, Stephen G. Swisher, Dima Abi-Said, and Raymond Sawaya. 1998. Transthoracic vertebrectomy for metastatic spinal tumors. *Journal of Neurosurgery* 89: 599–609. [CrossRef]

Golueke, P., S. Sclafani, T. Phillips, A. Goldstein, T. Scalea, and A. Duncan. 1987. Vertebral artery injury: Diagnosis and management. *Journal of Trauma and Acute Care Surgery* 27: 856–65. [CrossRef]

Gonzalez, F., J. Y. Chang, K. Banovac, D. Messina, A. Martinez-Arizala, and R. E. Kelley. 1991. Autoregulation of cerebral blood flow in patients with orthostatic hypotension after spinal cord injury. *Paraplegia* 29: 1–7. [CrossRef]

Green, David, Russell D. Hull, Eberhard F. Mammen, Geno J. Merli, Saul I. Weingarden, and James S. T. Yao. 1992. Deep vein thrombosis in spinal cord injury summary and recommendations. *Chest* 102: 633S–635S. [CrossRef]

Green, David, Susan Sullivan, Janet Simpson, Robert C. Soltysik, and Paul R. Yarnold. 2005. Evolving risk for thromboembolism in spinal cord injury (SPIRATE Study). *American Journal of Physical Medicine & Rehabilitation* 84: 420–22. [CrossRef]

Guttman, Ludwig. 1973. *Spinal Cord Injuries: Comprehensive Management and Research*. Oxford: Blackwell Scientific Publications.

Hagen, Ellen Merete. 2015. Acute complications of spinal cord injuries. *World Journal of Orthopedics* 6: 17–23. [CrossRef] [PubMed]

Hamilton, Mark G., and S. Terence Mylks. 1992. Pediatric spinal injury: Review of 174 hospital admissions. *Journal of Neurosurgery* 77: 700–4. [CrossRef] [PubMed]

Haralson, Robert H., III, and Harold B. Boyd. 1969. Posterior dislocation of the atlas without fracture. Report of a case. *The Journal of Bone and Joint Surgery* 51: 561–66. [PubMed]

Harris, John H., Jr. 1986. Radiographic evaluation of spinal trauma. *Orthopedic Clinics of North America* 17: 75–86. [CrossRef] [PubMed]

Holdsworth, Frank. 1970. Fractures, dislocations, and fracture-dislocations of the spine. *The Journal of Bone & Joint Surgery* 52: 1534–51.

Iida, Hideo, Shigekuni Tachibana, Takao Kitahara, Shigeharu Horiike, Takashi Ohwada, and Kiyotaka Fujii. 1999. Association of head trauma with cervical spine injury, spinal cord injury, or both. *The Journal of Trauma and Acute Care Surgery* 46: 450–52. [CrossRef]

Jackson, Amie B., and Thomas E. Groomes. 1994. Incidence of respiratory complications following spinal cord injury. *Archives of Physical Medicine and Rehabilitation* 75: 270–75. [CrossRef]

Jamshidi, Saied, Michael W. Dennis, Charles Azzam, and Najmaldin Karim. 1983. Traumatic posterior atlantoaxial dislocation without neurological deficit. Case report. *Neurosurgery* 12: 211–13. [CrossRef]

Jefferson, Geoffrey. 1920. Fractures of the atlas vertebra: Report of four cases, and a review of those previously recorded. *British Journal of Surgery* 7: 407–22. [CrossRef]

Kirshblum, Steven C., and Kevin C. O'Connor. 2000. Levels of spinal cord injury and predictors of neurologic recovery. *Physical Medicine and Rehabilitation Clinics of North America* 11: 1–27. [CrossRef]

Labronici, Pedro José, Diogo do Nascimento Pereira, Pedro Henrique Vargas Moreira Pilar, José Sergio Franco, Marcos Donato Serra, José Carlos Cohen, and Rogério Carneiro Bitar. 2015. Safe localization for placement of percutaneous pins in the calcaneus. *Revista Brasileira de Ortopedia* 47: 455–59. [CrossRef] [PubMed]

Levine, Alan M., and C. C. Edwards. 1985. The management of traumatic spondylolisthesis of the axis. *The Journal of Bone and Joint Surgery* 67: 217–26. [CrossRef] [PubMed]

Lindan, R., E. Joiner, A. A. Freehafer, and C. Hazel. 1980. Incidence and clinical features of autonomic dysreflexia in patients with spinal cord injury. *Paraplegia* 18: 285–92. [CrossRef] [PubMed]

Marino, R. J., D. Rider-Foster, G. Maissel, and J. F. Ditunno. 1995. Superiority of motor level over single neurological level in categorizing tetraplegia. *Paraplegia* 33: 510–13. [CrossRef] [PubMed]

Mueller, Franz Josef, Bernd Fuechtmeier, Bernd Kinner, Michael Rosskopf, Carsten Neumann, Michael Nerlich, and Carsten Englert. 2012. Occipital condyle fractures. Prospective follow-up of 31 cases within 5 years at a level 1 trauma centre. *European Spine Journal* 21: 289–94. [CrossRef]

National Spinal Cord Injury Statistical Center. 2005. Spinal cord injury: Facts and figures at a glance. *The Journal of Spinal Cord Medicine* 28: 379–80.

Nichols, Constance G., David H. Young, and William R. Schiller. 1987. Evaluation of cervicothoracic junction injury. *Annals of Emergency Medicine* 16: 640–42. [CrossRef]

Papadopoulos, Stephen M. 1993. Biomechanics of Occipito-Atlanto-Axial Trauma. In *Spinal Trauma: Current Evaluation and Management*. Edited by AANS Publications Committee, Gary L. Rea and Carole A. Miller. Hoboken: WileyBlackwell, pp. 17–23.

Patel, Shanon, Shalini Kanagasingam, and Thomas Pitt Ford. 2009. External cervical resorption: A review. *Journal of Endodontics* 35: 616–25. [CrossRef]

Ragnarsson, Kristjan T., Karyl M. Hall, Conal B. Wilmot, and R. Edward Carter. 1995. Management of pulmonary, cardiovascular and metabolic conditions after spinal cord injury. In *Spinal Cord Injury: Clinical Outcomes from the Model Systems*. Edited by Samuel L. Stover, Joel A. DeLisa and Gale G. Whiteneck. Gaithersburg: Aspen Publishers.

Sapru, Hreday N. 2002. Spinal cord: Anatomy, physiology and pathophysiology. In *Spinal Cord Medicine*. Edited by Steven Kirshblum, Denise I. Campagnolo and Joel A. DeLisa. Philadelphia: Lippincott Williams & Wilkins.

Sassard, Walter R., Charles F. Heining, and William R. Pitts. 1974. Posterior atlantoaxial dislocation without fracture. Case report with successful conservative treatment. *The Journal of Bone & Joint Surgery* 56: 625–28.

Schneider, Richard C., Kenneth E. Livingston, A. J. E. Cave, and Gilbert Hamilton. 1965. 'Hangman's Fracture' of the Cervical Spine. *Journal of Neurosurgery* 22: 141–54. [CrossRef]

Schwartz, David T. 2008. Section 5. Cervical Spine. In *Emergency Radiology: Case Studies*. Edited by David T. Schwartz. New York: McGraw-Hill. Available online: https://accessemergencymedicine.mhmedical.com/ (accessed on 31 May 2023).

Segal, Jack L., and Sherry R. Brunnemann. 1997. 4-Aminopyridine improves pulmonary function in quadriplegic humans with longstanding spinal cord injury. *Pharmacotherapy* 17: 415–23. [CrossRef] [PubMed]

Segal, Jack L., and Sherry R. Brunnemann. 1998. 4-Aminopyridine alters gait characteristics and enhances locomotion in spinal cord injured humans. *The Journal of Spinal Cord Medicine* 21: 200–4. [CrossRef] [PubMed]

Shapiro, Scott, Richard Borgens, Robert Pascuzzi, Karen Roos, Michael Groff, Scott Purvines, Richard Ben Rodgers, Shannon Hagy, and Paul Nelson. 2005. Oscillating field stimulation for complete spinal cord injury in humans: A phase 1 trial. *Journal of Neurosurgery: Spine* 2: 3–10. [CrossRef] [PubMed]

Sud, S., S. Chaturvedi, T. B. S. Buxi, and S. Singh. 2002. Posterior atlantoaxial dislocation without associated fracture. *Skeletal Radiology* 31: 529–31. [CrossRef] [PubMed]

Tator, Charles H. 1994. Epidemiology and general characteristics of the spinal cord injured patient. In *Contemporary Management of Spinal Cord Injury*. Edited by Edward C. Benzel and Charles H. Tator. Park Ridge: American Association of Neurological Surgeons.

Theisen, D., L. Malisoux, R. Seil, and A. Urhausen. 2014. Injuries in youth sports: Epidemiology, risk factors and prevention. *Deutsche Zeitschrift für Sportmedizin* 65: 248–52. [CrossRef]

Tuli, Sagun, Charles H. Tator, Michael G. Fehlings, and Margot Mackay. 1997. Occipital condyle fractures. *Neurosurgery* 41: 368–76. [CrossRef]

Welch, Robert D., Susanne J. Lobley, Susan B. O'Sullivan, and Murray M. Freed. 1986. Functional independence in quadraplegia: Critical levels. *Archives of Physical Medicine and Rehabilitation* 67: 235–40.

White, Augustus A., and Manohar M. Panjabi. 1979. The basic kinematics of the human spine: A review of past and current knowledge. *Spine* 3: 12. [CrossRef]

White, Augustus A., and Manohar M. Panjabi. 1990. *Clinical Biomechanics of the Spine*. Philadelphia: JB Lippincott.

Wong, David A., Robert P. Mack, and Thomas K. Craigmile. 1991. Traumatic atlantoaxial dislocation without fracture of the odontoid. *Spine* 16: 587–89. [CrossRef]

Wood-Jones, Frederic. 1913. The Ideal Lesion Produced by Judicial Hanging. *Lancet* 181: 53. [CrossRef]

World Federation of Neurosurgical Societies Spine Committee. 2019. Cervical Spine Trauma Recommendations. Available online: https://wfns-spine.org/cervical-spine-trauma-recommendations (accessed on 5 March 2024).

World Federation of Neurosurgical Societies Spine Committee. 2020. Thoracolumbar Spine Trauma: WFNS Spine Committee Recommendations. Available online: https://wfns-spine.org/pdf/Thoracolumbar%20Spine%20Trauma%20Recommendations.pdf (accessed on 5 March 2024).

Yoon, Do Heum, Kook Hee Yang, Keung Nyun Kim, and Sung Han Oh. 2003. Posterior atlantoaxial dislocation without fracture: Case report. *Journal of Neurosurgery: Spine* 98: 73–76. [CrossRef] [PubMed]

Zhang, Andrew, and Brad J. Chauvin. 2021. Denis Classification. In *StatPearls [Internet]*; Treasure Island: StatPearls Publishing. Available online: https://www.ncbi.nlm.nih.gov/books/NBK544310/ (accessed on 23 July 2022).

Zindrick, Michael R., Leon L. Wiltse, Eric H. Widell, James C. Thomas, W. Russell Holland, B. Ted Field, and Curtis W. Spencer. 1986. A biomechanical study of intrapeduncular screw fixation in the lumbosacral spine. *Clinical Orthopaedics and Related Research* 203: 99–111. [CrossRef]