



MANAGEMENT OF SUB-AXIAL CERVICAL SPINE INJURY

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Management of Sub-Axial Cervical Spine injury

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Introduction

Patients with spinal cord injuries comprise a major percentage of the total number of trauma patients who suffer with high mortality and long-term morbidity. The majority of these patients are of young age, resulting in a disproportionately high loss of productive years of life both individually and for society [1]. One third of these patients have injuries that involve the cervical spine [1]. The management of spinal cord injury remains controversial [2-5] as does the optimal timing of surgical intervention [3]. Recent laboratory and clinical trials indicate that early surgical intervention may be associated with improved results [5-11] though previous studies have reported an increased risk of neurological deterioration with early surgical intervention [2,12,13].

Review of Literature

There are several controversial issues regarding surgical intervention of management in the cervical spine injury. The most important issues are the role of decompression and the timing of the decompression. The goals of surgical treatment of cervical spine injuries include decompression of neural elements, realignment of the misaligned spine, and restoration of spinal stability. The role of surgical decompression in patients with complete spinal injury remains controversial.

Heiden & colleagues [14] retrospectively analyzed 199 patients with complete cervical spine injury and found that no patient of the surgical group (121) recovered ambulatory function. Of the 78 patients non-surgically treated patient two were ambulatory at 1 year follow up examination. Other investigators [15-18] have also reported spontaneous neurologic improvement with nonoperative treatment.

Some investigators [17,19,20] have indicated that neither spinal surgery nor anatomic realignment of the spinal column improved neurologic outcome in patients with acute SCI, with the possible exception of bilateral locked facets. The role of decompressive surgery in incomplete cervical cord injury is also unclear. The role of surgical decompression in incomplete cervical spinal cord injury is not yet determined. However, a number of retrospective studies have suggested that decompression, even when delayed can improve the neurological function in incomplete cervical spine injury [21-24]. The timing of spinal cord decompression in patients with cervical spinal cord injuries is unresolved issue.

Levi et al. [25] found no significant differences in outcome between the early and the late surgical group. Theoretically, early decompression and stabilization should decrease an ongoing secondary injury in the spinal cord and result in a better neurological outcome. Experimental studies on rats showed that early decompression of the spinal cord results in better neurological outcome.

In 1999, Dimar et al. [20] provided the evidence that spinal cord decompression after SCI is beneficial. The results in this study present strong evidence that the prognosis for neurologic recovery is adversely affected by both a higher percentage of canal narrowing and a longer duration of canal narrowing after a spinal cord injury. The tolerance for spinal canal narrowing with a contused cord appears diminished, indicating that an injured spinal cord may benefit from early decompression. Additionally, it appears that the longer the spinal cord compression exists after spinal cord injury, the worse the prognosis for neurologic recovery.

Carlson et al. [26] also reported similar results in dogs. Animals undergoing early decompression showed significantly better functional recovery and significantly smaller lesion volumes. Guha et al. [27] demonstrated a benefit from decompressing the spinal cord after 4 hours of injury in rats. Proponents of early surgery also cite the beneficial effects of early mobilization and a shorter duration of hospital stay.

Fehlings and Perrin et al. [24,28] reviewed the recent literature and concluded that animal studies consistently show that neurological recovery is enhanced by early decompression. Urgent decompression in acute cervical SCI remains a reasonable practice option and can be performed safely. There is emerging clinical evidence that surgery within 24 hours may reduce length of intensive care unit stay and reduce post-injury medical complications.

Several prospective series [15,29,30-32] suggest that early decompression done less than 12 hours post injury can be performed safely and may improve neurological outcomes. Currently, there are no standards regarding the role and timing of decompression in acute spinal cord injury [24,28]. La Rosa G et al. [33] after a Medline search covering the period 1966-2000, supplemented with manual search concluded that early decompression resulted in better outcome compared with both conservative and late management.

Papadopoulos et al. [30] concluded that immediate spinal cord decompression could improve neurological outcome. Chen et al. [34], showed that surgical decompression was associated with immediate neurologic improvement, faster recovery, early mobilization, better long term outcome. Mirza et al. [3] showed benefits from early decompression. Hakato et al. [35] also concluded that the early cervical spinal cord decompression creates optimal conditions for the neurological recovery. Schlegel et al. [36] concluded that surgical decompression, reduction, and fixation within the first 72 hours are indicated in patients with cervical injuries with a neurological deficit. Rosenfeld et al. [37] reviewed experimental and clinical research to clarify the benefits of early surgery for cervical spinal cord injury. Their conclusion was that the direct clinical benefit of early surgery is the theoretic improvement in neurologic recovery over that of delayed surgery. Additional benefits of early

surgery include the clinical advantages of a decreased length of hospitalization and its associated complications and a decreased time to rehabilitation and mobilization.

Albert et al. [37] concluded in 2005 that though clinical investigators have failed to provide convincing evidence that the timing of surgery significantly affects neurological outcome in most situations, early surgical stabilization of the injured spine has been shown to provide significant non neurological benefits such as more rapid patient mobilization, facilitation of treating associated injuries, reduction in rates of pulmonary and pressure sore complications, reduction in duration of intensive care unit and hospital stays, and a decrease in overall medical costs.

The findings of basic science studies have improved our understanding of the molecular and cellular events surrounding initial and secondary spinal cord injury (SCI), and analysis of these findings suggests that the early post injury period may present a unique opportunity for meaningful intervention. This possibility has been supported by results obtained in animal studies that demonstrate the potential for improving functional outcome when surgical intervention is performed within a few hours following experimental spinal cord injury.

Despite the absence of significant neurological recovery in most clinical studies, the results of most recent clinical studies strongly support the overall clinical benefits of early surgical intervention [6-11]. Whether early surgery results in functional improvement or not is still not known. However, given the knowledge from numerous studies, it appears that early surgical decompression of cord could benefit some patients [6-11].

Advocates of late surgery emphasize that most neurological damage occurs at the moment of injury and cite increased vulnerability of a recently traumatized cord, which could negate the benefits of an early operation. They further suggest that early surgery on a patient with a traumatized cord will increase the morbidity [12,15,38,39].

A widely cited multicenter analysis by Marshall and colleagues [12] showed that early surgery is hazardous in ways that later surgery (>6 days after injury) is not.

Kerwin et al. [38] concluded that Spinal fixation within 48 hours after vertebral fractures and dislocations appears to increase mortality despite similar anatomic and physiologic parameters in the later operative group. Incomplete resuscitation of patients before surgery may have contributed to this result. The shorter hospital stay may have been because of the higher number of early deaths. Prospective studies to identify the optimal timing of spinal fixation and the reason for these outcome differences are warranted.

Vaccaro & Daugherty [15] and colleagues revealed (prospective randomized study) no significant neurologic benefit when cervical spinal cord decompression after trauma is performed less than 72 hours after injury as opposed to waiting longer than 5 days.

Pollard & Apple [39] showed the most important prognostic variable relating to neurologic recovery in a patient with a spinal cord injury is the completeness of the lesion. No evidence was found to support high-dose steroid administration and early surgical decompression.

Tator & Fehlings [40] concluded that there is very little agreement on the optimum timing of surgical treatment. The results of this study confirm the need for a randomized controlled trial to assess the optimum timing of decompressive surgery.

Sapkas & Papadakis [41] concluded that timing of surgery does not affect neurological outcome. Wagner & Chehrizi [42] reported that surgical decompression within 48 hours of injury in 44 patients had no effect on neurologic recovery.

Miller [4] & Mahoney [43] reported increased complication rate with surgical intervention within 5 to 7 days. Kleyn [44] concluded that neurosurgical recovery did not correlate to the time interval from injury to reduction.

Epidemiology

Estimates of number of patients living with spinal cord injuries in the U.S.A range from 18500 to 400000 [45-47]. SCI tends to occur in the young individual in the 2nd or 3rd decade with a 2nd peak in elderly population [46-48]. Young males are affected 3 to 20 times more common than females [46,49,50]. Of the 14000 people who sustained SCI annually, 4200 die before reaching hospital and 1500 patients die during hospital stay [45,49,50] Motor vehicle accidents cause between 35% and 45% of all spinal injury. Athlete injuries result in between 5% and 10% of all spinal cord injuries [45-48].

Pediatric spinal trauma has an annual incidence of 1.8 per 100,00 population, with 80% of injuries occurring in patients older than 10 years [51]. 70%-87% injuries occur at C3 or higher in patients younger than 8 years [47]. Higher level of pediatric injury, likely to be fatal, mostly from atlanto-occipital dislocation 71% to 100% mortality [51].

Sixty percent pediatric cervical spine injury results in fracture without neurologic compromise [51]. Twenty percent pediatric injury is spinal cord injury without radiographic abnormality (SCIWORA) [51]. SCIWORA is usually complete injury, having poor prognosis [51]. The cost of caring for a SCI patient depends on the level involved. A high cervical injury C4 and above, costs an average of \$549000 in the first year \$98000 per year after that [52].

Cervical spine fracture constitutes 20 to 30 % of all spinal fractures while cervical cord injury constitutes upto 50% of all spinal cord injury [46,47]. Cervical spine injury results in severe disability and profound socio-economic impact. In 1990, the economic cost of SCI was estimated to be \$4 billion in direct cost and \$3.4 billion in lost wages [52].

In India, the incidence of cervical spine injury is 18% per year. Males are more affected than females (M: F=3.6:1). Fall from height and road traffic accidents constitute 58.9% and 21.3% respectively of the total number of cervical spine injuries. 25.6% were managed surgically and 74.4% were managed non-surgically [53].

The incidence of head and facial injuries in patients with cervical cord lesions can be as high as 50%, while thoracolumbar spine injury may be complicated by chest contusion, rib fractures, pelvis fracture

and other long bone fractures [55].

Anatomy of subaxial cervical spine

The normal cervical spine consists of 7 cervical vertebrae separated by intervertebral disks and joined by a complex network of ligaments. These ligaments keep individual bony elements behaving as a single unit.

Classifications of Cervical Spine Injuries

Cervical spine injuries can be classified as many ways.

Harris et al. [55] proposed a classification of osseomusculo ligamentous injuries.

Flexion injuries

Anterior dislocation (hyperflexion sprain): Anterior subluxation in the cervical spine occurs when posterior ligamentous complexes (nuchal ligament, capsular ligaments, ligamentum flavum, posterior longitudinal ligament) rupture. The anterior longitudinal ligament remains intact. No associated bony injury is seen. Radiographically, the lateral view shows widening of interspinous space, and anterior and posterior contour lines are disrupted in flexion views.

Bilateral facet dislocation (locked facet): Bilateral facet dislocation is an extreme form of anterior subluxation that occurs when a significant degree of flexion and anterior subluxation causes ligamentous disruption to extend anteriorly, which causes significant anterior displacement of the spine at the level of injury. This injury involves the annulus fibrosus, anterior longitudinal ligament and posterior ligamentous complex. At the level of injury, i.e., the upper vertebrae, inferior articulating facets pass superior and anterior to the superior articulating facets of the lower involved vertebrae because of extreme flexion of the spine. Radiographically, this is seen as a displacement of bilateral facet joints and of the vertebral body in the lateral view. This is an extremely unstable condition and is associated with a high prevalence of spinal cord injuries.

Simple wedge compression fracture: With a pure flexion injury, a longitudinal pull is exerted on the nuchal ligament complex that, because of its strength, usually remains intact. The anterior vertebral body bears most of the force, sustaining simple wedge compression anteriorly without any posterior disruption.

Radiographically the anterior border of the vertebral body has diminished height and increased concavity along with increased density due to bony impaction. The prevertebral soft tissues are swollen.

Flexion tear drop fracture: A flexion teardrop fracture occurs when flexion of the spine, along with vertical axial compression, causes a fracture of the antero-inferior aspect of the vertebral body. This fragment is displaced anteriorly and resembles a teardrop. For this fragment to be produced significant posterior ligamentous disruption must occur. Since the fragment displaces anteriorly, a significant degree of anterior ligamentous disruption exists.

Clay-shoveler fracture (spinal process avulsion): Abrupt flexion of the neck, combined with a heavy upper body and lower neck muscular contraction, results in an oblique fracture of the base of the spinous process, which is avulsed by the intact and powerful supraspinous ligament. Fracture also occurs with direct blows to the spinous process or with trauma to the occiput that causes forced flexion of the neck. Injury commonly is observed in a lateral view, since the avulsed fragment is readily evident. Injury commonly occurs in lower cervical vertebrae; therefore, visualization of the C7-T1 junction in the lateral view is imperative. Injury also may be seen in the antero-posterior view as a vertically split appearance of the spinous process in the lower vertebrae.

Flexion-rotation-unilateral facet dislocation: Unilateral facet dislocation occurs when flexion, along with rotation, forces one inferior articular facet of an upper vertebra to pass superior and anterior to the superior articular facet of a lower vertebra, coming to rest in the intervertebral foramen. Although the posterior ligament is disrupted, vertebrae are locked in place, making this injury stable. Radiographically, the lateral view shows an anterior displacement of the spine at the involved level of less than one half the diameter of the vertebral body. This is in contrast to the greater displacement seen with a bilateral facet dislocation, as discussed above. The antero-posterior view is useful in

diagnosis of unilateral dislocation because it shows a disruption in the line connecting the spinous processes at the level of the dislocation. The oblique view is also useful because it shows a disruption of the typical shingles appearance at the level of the involved vertebra. The dislocated superior articulating facet of the lower vertebra is seen projecting within the neural foramina.

Vertical compression (axial loading)

Burst fracture

When downward compressive force is transmitted to lower levels in the cervical spine, the body of the cervical vertebra can shatter outward, causing a burst fracture. This fracture involves disruption of the anterior and middle columns, with a variable degree of posterior protrusion of the latter.

Radiographically, a vertical fracture line in the frontal projection and evidences this fracture by comminution and protrusion of the vertebral body anteriorly and posteriorly with respect to the contiguous vertebrae in the lateral view. Posterior protrusion of the middle column may extend into the spinal canal and can be associated with anterior cord syndrome. Burst fractures always require an axial CT scan or MRI to document amount of middle column retropulsion.

Extension injury

Hyperextension dislocation (hyperextension sprain): The spinous process and lateral mass acts as a fulcrum, causing anterior longitudinal ligament and anterior disc to rupture. The radiographic examination may be normal. Radiographic evidence includes prevertebral soft tissue swelling, widened disc space, small anterior avulsion fracture.

Laminar fracture: Isolated laminar fracture may occur due to hyperextension. These injuries are stable.

Hyperextension fracture dislocation: the result of severe rotatory hyperextension causing compression of posterior elements including pedicle, laminae, spinous process, and articular masses. The vertebral body is frequently subluxed forward. The anterior longitudinal ligament and intervertebral disc may be disrupted causing anterior avulsion fracture.

Extension-Rotation-Lateral Mass (Pillar Fracture)

This is mild form of hyperextension fracture dislocation. It is stable fracture.

Lateral Flexion- Uncinate Process Fracture

It occurs by laterally directed force causing excessive lateral flexion. It is stable fracture.

Other Classification System

Allen et al. [56] - According to this system, there are six patterns of subaxial cervical spine fractures

- A. Compression-flexion
- B. Compression-extension
- C. Distraction-flexion
- D. Distraction-extension
- E. Lateral flexion

Cervical Spinal Stability [57,58]

Clinically cervical spine stability has been defined (56,57) using three parameters:-

1. Motion segment will not further displace or deform under physiological load.
2. There is no progressive displacement or deformity during healing process.
3. There is no progressive neural compression or injury.

Two and three column concept [59] - although these are designed to describe stability of thoraco-lumbar spine specifically, but they are also helpful in defining stability of cervical spine also. According to Holdsworth [60], anterior column is made up of the anterior longitudinal ligament, vertebral body, intervertebral disc, and posterior longitudinal ligament; posterior column consists of all bony and ligamentous component posterior to posterior longitudinal ligament. Instability was defined by disruption of posterior ligament complex.

Dennis [61] postulated a three-column concept of spinal stability. Anterior column is made up of anterior 2/3rd of vertebral body and disk and anterior longitudinal ligament, middle column consists of posterior 1/3rd of vertebral body and disk, and posterior column consists of

supra and infraspinous ligament, ligamentum flavum, joint capsules, laminae, spinous process and articular process. Instability is defined by injury to any two or more columns.

Pathophysiology [61-64]

The spinal cord is more vulnerable to injury than the brain. Immediately following blunt trauma or compression, hemorrhages are seen in the central gray matter. A zone of hemorrhage, edema and necrosis spreads from the central area to involve the entire diameter of the cord within 6 to 24 hours. Damage to the gray matter involves only two or three segments at the level of injury.

This will cause an interruption of nerve conduction in the fiber tracts, which isolates the region of the body below the level of injury from cerebral control. There is progressive loss of function after the initial impact for the first 24 hours related to associated secondary injury, edema, disc compression, hematoma and hypoperfusion to the spinal cord.

As edema subsides or circulation is reestablished, the function in some areas may improve slightly and in the absence of further injury, the pattern is usually stable after the first day. The rest of the patient's progress can be divided in an acute and chronic phase.

Acute phase of spinal cord injuries [61] (4-6 weeks)

The immediate response to spinal cord compression or injury is a massive sympathetic stimulation and reflex parasympathetic activity that usually lasts for 3 to 4 minutes and is mediated by alpha-adrenergic receptors. The hemodynamic effects are severe hypertension and reflex bradycardia or tachyarrhythmias.

After this initial response, loss of neurologic function below the injured cord will cause flaccid paralysis of voluntary muscles, areflexia, loss of sympathetic tone, which is called spinal shock. There is hypotension and bradycardia in high thoracic or cervical injuries, increased vascular capacitance, poikilothermia and flaccidity of the GI tract with generalized ileus and the paralysis bladder with urinary retention.

Respiratory system [65]

The extent of respiratory impairment depends on the level of the lesion. The diaphragm is innervated by C3-C5, and lesions above this level cause total diaphragmatic paralysis and an inability to generate adequate tidal volume. This accounts for a large number of the pre-hospital deaths. Patients, with lesions below C6, have an intact diaphragm with variable intercostal and abdominal muscle weakness depending on the level of the lesion.

There is reduction in functional residual capacity, forced vital capacity, maximum inspiratory pressure, maximum expiratory pressure as well as the presence of paradoxical respiration. Vital capacity is reduced to about 1500ml with acute cervical spinal cord injury, which usually improves with time to 50 percent of normal. The reduction in tidal volume can reach 60 percent in these patients. The overall effect is severe hypoventilation with a high incidence of hypoxia and hypercapnia. Quadriplegic patients have a higher vital capacity in the supine position than in the seated position, because of diaphragmatic mechanics. The inability to cough and clear secretions leads to a high incidence of atelectasis and pneumonia in the acute period. The initial surge of sympathetic activity with the injury can also cause neurogenic pulmonary oedema, the mechanism of which is unclear, but may be mediated by both haemodynamic and permeability factors.

Reflex vascular activity, sweating, and shivering are abolished in spinal shock; patients with high-level lesions are generally poikilothermic. Hyperglycemia, which commonly occurs in patients with severe systemic stress, has been associated with worse neurologic outcome in spinal cord injury.

Progression to the chronic phase [61]

Sympathetic tone returns to some extent in 4 to 7 days. Resting blood pressure returns to, or toward normal and there may be a mild hypertensive response (autonomic hyperreflexia) to various stimuli such as pain or bladder distention below the level of the lesion. Reflex activity returns after 4 to 6 weeks and the chronic phase begins. This is characterized by spastic motor paralysis with hyperactive tendon reflexes, occasionally severe autonomic hyperreflexia, and some return of involuntary bladder function.

A patient who sustains paralysis with no sign of distal sparing may have a complete and irreversible cord lesion. When the period of spinal shock is over, which is heralded by the return of the bulbocavernosus reflex (elicited by pulling on the glans penis, tapping the clitoris, or tugging on an indwelling urinary catheter and obtaining a rectal sphincter response), a definitive diagnosis can be made. If the reflex has returned and complete paralysis continues, there will be no neural recovery. Two additional considerations are particularly important in the chronic phase: supersensitivity of cholinergic receptors and autonomic hyperreflexia.

Supersensitivity of cholinergic receptors [61]

In response to denervation, cholinergic receptors proliferate beyond the end plates of voluntary muscle fibers, eventually to invest the entire cell membrane. The muscle becomes "supersensitive" and contracts maximally in response to a concentration of acetylcholine only 1/4th to 1/5th that needed to initiate contraction in normal muscle. Potassium ion is released suddenly along the entire length of the fiber rather than gradually as the action potential propagates.

Autonomic hyperreflexia [61]

The chronic phase in which spinal reflexes reappear is characterized by autonomic hyperreflexia in a high proportion of patients. Cutaneous, proprioceptive, and visceral stimuli, such as urinary bladder distention, may cause violent muscle spasm and autonomic disturbances. The symptoms of autonomic hyperreflexia are facial tingling, nasal obstruction, severe headache, shortness of breath, nausea and blurred vision. The signs are hypertension, bradycardia, dysrhythmias, sweating, cutaneous vasodilation above and palor below the level of the spinal injury, and occasionally loss of consciousness and seizures. The precipitous blood pressure increase may lead to retinal, cerebral, or subarachnoid hemorrhage, increased myocardial work and pulmonary edema.

Patients with chronic spinal cord lesions above T-6 are particularly at risk for this response: 85 % will display autonomic hyperreflexia at some time during the course of daily living. Of course, surgery is a potent stimulus to autonomic response even in patients who give no history of the problem.

The neuroanatomic pathway of this syndrome has been known for a long time. Afferent impulses enter the isolated spinal cord and elicit reflex autonomic output over the entire sympathetic outflow below the level of injury, which is not modulated by higher centers as in the neurologically intact subject. This causes vasoconstriction below the level of injury and resultant hypertension, which stimulates baroreceptors and may cause bradycardia via intact vagal pathways to the heart and vasodilation via intact pathways above the injury.

Therapeutic methods to reduce the hypertension of autonomic hyperreflexia must act below the level of injury. Ganglionic blockers, alpha-adrenergic blockers, catecholamine depleters, direct vasodilators, and general or regional anesthesia have been recommended for prevention or treatment of autonomic hyperreflexia.

Clinical Profile [66-68]

Common presentations include the following:

- A. Posterior neck pain on palpation of spinous processes
- B. Limited range of motion associated with pain
- C. Weakness, numbness, or paresthesias along affected nerve roots
- D. Quadriplegia with sensory loss with sphincter involvement
- E. Spinal shock
 - a. Flaccidity
 - b. Areflexia
 - c. Loss of anal sphincter tone
 - d. Fecal incontinence
 - e. Priapism
 - f. Loss of bulbocavernosus reflex
- F. Neurogenic shock
 - a. Hypotension
 - b. Paradoxical bradycardia
 - c. Flushed, dry, and warm peripheral skin
 - d. Autonomic dysfunction --Ileus
 - 1. Urinary retention
 - 2. Poikilothermia

Clinical Syndromes [65-69]

Spinal shock

- A. Severe spinal cord injury may cause a concussive injury of the spinal cord termed spinal shock syndrome.
- B. Spinal shock manifests as distal areflexia of a transient nature that may last from a few hours to weeks. Initially, the patient experiences

a flaccid quadriplegia along with areflexia. Segmental reflexes start to return usually within 24 hours as spinal shock starts to resolve. At that point, flaccid quadriplegia changes to spastic paralysis.

- C. Eventually, total resolution can be expected.

Neurogenic shock

- A. Neurogenic shock is spinal shock that causes vasomotor instability because of loss of sympathetic tone.
- B. Patients with neurogenic shock are hypotensive but have paradoxical bradycardia.
- C. Flushed, dry, and warm peripheral skin, (in contrast to findings with hypovolemic or cardiogenic shock) may be present. Other signs of autonomic dysfunction include ileus, urinary retention, and poikilothermia.
- D. Loss of anal sphincter tone with fecal incontinence and priapism suggest spinal shock. Return of bulbocavernosus reflex heralds resolution of spinal shock.

Complete and incomplete cord syndromes

- A. Besides spinal shock, complete and incomplete spinal cord syndromes may occur.
- B. Spinal shock mimics a complete spinal cord lesion. Emergency physicians should wait until spinal shock resolves to make an accurate estimate of the patient's prognosis.
- C. Incomplete cord syndromes include
 - a. Anterior spinal cord syndrome
 - b. Central spinal cord syndrome
 - c. Brown-Séquard syndrome.

Anterior spinal cord syndrome: Anterior spinal cord syndrome involves complete motor paralysis and loss of temperature and pain perception distal to the lesion. Since posterior columns are spared, light touch, vibration, and proprioceptive input are preserved. This syndrome is caused by compression of the anterior spinal artery, which results in anterior cord ischemia or direct compression of the anterior cord. It is associated with burst fractures of the spinal column with fragment retropulsion caused by axial compression.

Central spinal cord syndrome: This syndrome is caused by damage to the corticospinal tract. It is characterized by weakness, greater in the upper extremities than the lower extremities and more pronounced in the distal aspect of extremity. The syndrome usually is associated with a hyperextension injury in patients with spondylosis or congenital stenosis of the cervical canal. Extension of the cervical spine, causing buckling of the ligamentum flavum into the spinal cord, is believed to cause central spinal cord syndrome.

Brown-Séquard syndrome: This syndrome involves injury to only 1 side of spinal cord. It causes paralysis, loss of vibration sensation, and loss of proprioceptive input ipsilaterally, with contralateral loss of pain and temperature perception because of involvement of posterior columns and spinothalamic tracts on the same side. It is associated with hemisection of the spinal cord from penetrating trauma; however, it also can be caused by a lateral mass fracture of a cervical vertebra.

Causes [45,46,50,57]

Motor vehicle accidents and falls account for 50% and 20% of these injuries, respectively. Sports-related activities account for 15%. The remaining injuries are attributed to interpersonal violence.

Radiological workup [70-73]: Recent literature has examined the need for C-spine imaging in patients who are low risk for unstable fracture or ligamentous injury. Two clinical decision-making criteria, the Canadian C-Spine Rules (CCR) and the National Emergency X-Radiography Utilization Group (NEXUS) criteria allow clinicians to "clear" low-risk patients of C-spine injury, obviating the need for radiography. To be clinically cleared using the CCR, a patient must be alert (GCS 15), not intoxicated, and not have a distracting injury (eg, long bone fracture, large laceration). The patient can be clinically cleared providing the following:

- A. The patient is not high risk (age >65 y)
- B. A low risk factor that allows safe assessment of range of motion exists. This includes simple rear end motor vehicle collision, maintaining seated position, ambulation at any time post trauma, delayed onset of neck pain, and the absence of midline cervical spine tenderness.
- C. The patient is able to actively rotate their neck 45 degrees left and right.
- D. The NEXUS criteria state that a patient with suspected c-spine injury can be cleared providing the following:

- a. No posterior midline cervical spine tenderness is present.
- b. No evidence of intoxication is present.
- c. The patient has a normal level of alertness.
- d. No focal neurologic deficit is present.
- e. The patient does not have a painful distracting injury.

f. Both studies have been prospectively validated as being sufficiently sensitive to rule out clinically significant c-spine pathology. The CCR were shown to be more sensitive than the NEXUS criteria (99.4% sensitive vs 90.7%), and the rates of radiography were lower with the CCR (55.9% vs 66.6%). Debate still exists as to which criteria are more useful and easier to apply.

X-Rays [74,75]

A standard trauma series is composed of 5 views: trans-lateral, swimmer's, oblique, odontoid, and antero-posterior.

Trans-lateral view: Approximately 85-90% of cervical spine injuries are evident in lateral view, making it the most useful view from a clinical standpoint. A technically acceptable lateral view shows all 7 vertebral bodies and the cervico-thoracic junction. Approach analysis of this view methodically to avoid missing significant pathology. Check alignment of cervical spine by following 3 imaginary contour lines.

- A. The first line connects the anterior margins of all the vertebrae and is referred to as the anterior contour line
 - B. The second line should connect the posterior aspect of all vertebrae in a similar way and is referred to as the posterior contour line.
 - C. The third line should connect the bases of the spinous processes and is referred to as the spinolaminar contour line.
- a. Each of these lines should form a smooth lordotic curve. Suspect bony or ligamentous injury if disruption is seen in the contour lines.
 - b. An exception occurs in young children who, because of immature muscular development, may have a benign pseudosubluxation in the upper cervical spine. An imaginary straight line should connect the points bisecting the base of the spinous processes of C1, C2, and C3. In pseudosubluxation, these imaginary points should not be displaced more than 2 mm in front of or behind the straight line.
 - c. Check individual vertebrae thoroughly for obvious fracture or changes in bone density. Areas of decreased bone density are seen in patients with osteoporosis, osteomalacia, or osteolytic lesions and may represent weak areas predisposed to injury.
 - d. Areas of increased bony density may be seen with osteoblastic lesions or may represent compression fractures of an acute nature.
 - e. Look for soft tissue changes in predental and prevertebral spaces. The predental space, also known as the atlantodental interval, is the distance between the anterior aspect of the odontoid and the posterior aspect of the anterior arch of C1. This space should be no more than 3 mm in an adult and 5 mm in a child. Suspect transverse ligament disruption if these limits are exceeded.
 - f. Prevertebral space extends between the anterior border of the vertebra to the posterior wall of the pharynx in the upper vertebral level (C2-C4) or to the trachea in the lower vertebral level (C6).
 - g. At the level of C2, prevertebral space should not exceed 7 mm.
 - h. At the level of C3 and C4, it should not exceed 5 mm, or it should be less than half the width of the involved vertebrae.
 - i. At the level of C6, prevertebral space is widened by the presence of the esophagus and cricopharyngeal muscle. At this level, the space should be no more than 22 mm in adults or 14 mm in children younger than 15 years.
 - j. Children younger than 24 months may exhibit a physiologic widening of the prevertebral space during expiration; therefore, obtain images in small children during inspiration to assess prevertebral space adequately.
 - k. If the prevertebral space is widened at any level, a hematoma secondary to a fracture is the most likely diagnosis.
 - l. Check for fanning of the spinous processes. This is evident as an exaggerated widening of the space between 2 spinous process tips and suggests posterior ligamentous disruption.
 - m. Check for an abrupt change in angulation of greater than 11 degrees at a single interspace. This also suggests bony injury with possible ligamentous involvement.

Swimmer's view

Occasionally, it is impossible to fully visualize all 7 cervical vertebrae and, more importantly, the cervicothoracic junction in a true lateral image. Failure to fully visualize these areas has resulted in patient morbidity and successful malpractice litigation against emergency physicians. A swimmer's or transaxillary view adequately exposes these areas for scrutiny.

Oblique view

This view also is considered a laminar view because most pathologic conditions assessed on it manifest with some disruption in the normal overlapping appearance of the vertebral laminae. The normal structural appearance of the laminae is described as shingles on a roof, forming a regular elliptical curve with equal interlaminar spaces. If interlaminar space between 2 continuous laminae is increased, suspect subluxation of the involved vertebrae. Similarly, if the expected tiling of shingles is disrupted, suspect a unilateral facet dislocation. A posterior laminar fracture should be evident as disruption of the body of a single shingle.

Open mouth odontoid view

This view is used to evaluate an area that is difficult to visualize in the trans lateral view because of shadow superimposition. The most important structural relationship to evaluate in this view is alignment of the lateral masses of C1 with respect to the odontoid process. Masses should be bilaterally symmetric with the dens and odontoid process and must be checked for fractures or lateral displacement. Assess symmetry of the interspace between C1 and C2.

Anteroposterior view

- A. This is the least useful view from a clinical standpoint.
- B. A straight line should connect the spinous processes bisecting the cervical spine. If this is not seen, consider a rotation injury (ie, unilateral facet dislocation). Also consider a clay shoveler fracture if a spinous process appears vertically split. The cross-table lateral view if used alone will be missing 15%-25% of cervical spine injuries.

The combination of the cross-table lateral, anterior-posterior, and open-mouth views will be missing 8% of fractures. The missed injuries were often unstable in the above studies. As the sensitivity of plain films is only 75%-90%, negative plain radiographs cannot be used as sufficient criteria for ruling out a cervical spine fracture, especially if a patient is at high risk. High risk patients include front-end motor vehicle accidents (>35mph) without seatbelts, head-first falls and equivocal C-spine roentgenograms.

They are believed to have at least a 10% chance of having a C-spine injury. Given a 10% false-negative rate, a set of plain films negative for spine injuries reduces the probability of an injury to 1% (not 0%). Soft tissue injury (ie, unstable ligamentous injury) has traditionally been evaluated by flexion-extension views. If static views are normal, flexion-extension views are indicated under fluoroscopic guidance in presence of neurosurgeon to diagnose occult instability.

CT Scan [73]

- A. The advent of computed tomography has supplanted the use of plain radiography at many centers. If plain radiograph findings are negative but clinical suspicion for fracture is high, films should be followed by CT. CT is more sensitive to detect fracture. Three-dimensional view can give excellent anatomical orientation of the bony pathology.
- B. MRI [73] --- mainstay of diagnosing neural (cord and root) injury and/or compression and other soft tissue injury.

Treatment

Prehospital Care [67]: When a cervical spine injury is suspected, minimize neck movement during transport to the treating facility. Ideally, transport the patient on a backboard with a semirigid collar.

Emergency department care [76-79]

- A. Stabilization of vital and systemic parameters
- B. Temporary immobilization of spine
- C. Medical treatment of spinal cord injury

The goals of resuscitation should be stabilization of the cervical spine, prevention of secondary injury, reduction of the fracture as soon as possible and protection of the spinal cord.

Stabilization of vital and systemic parameters [76-79]

- A. Airway management [76,77,78]

Many airway management plans would be reasonable for patients with potential cervical spine injuries because there is no evidence for the superiority of any individual tracheal intubation technique. The urgency of airway intervention is the most important factor in planning airway management for patients with potential C-spine injuries. Other considerations include the assessment of the risk of cord injury with head and neck movement, the airway anatomy, the patient's degree of cooperation, and the anesthesiologist's expertise. For patients requiring immediate and/or urgent airway control, recommendation is rapid sequence induction followed by orotracheal intubation with

cricoid pressure and manual in-line immobilization of the head and neck. Precise cervical spine in-line immobilization should be maintained throughout the intubation maneuvers. This technique, also called manual in-line axial traction is an active process done by a second individual who is responsible for applying a varying amount of force to counteract the movements of the laryngoscopist, in an attempt to stabilize the cervical spine. The patient lies supine with the head in neutral position; an assistant applies manual in-line immobilization by grasping the mastoid processes, whereupon the front of a rigid collar can be removed safely; the collar can impede mouth opening, does not contribute significantly to neck stabilization during laryngoscopy, and will be an obstruction if surgical airway is required.

This technique of emergency airway management involves a minimum of three, but ideally four individuals:

- A. the first to pre- oxygenate and intubate,
- B. the second to apply cricoid pressure,
- C. The third to maintain manual in-line immobilization of the head and neck and
- D. The fourth to give intravenous drugs and assist.
- E. For non-urgent and elective airway control, awake, fiber optic intubation technique should be used. Although there is no proof that this method minimizes C-spine movement, it does not depend on atlanto-occipital extension and the head and the neck stabilizing devices can be left in place.
- F. Indications of intubation--
 - a. for ventilation
 - b. to prevent aspiration (impaired cough reflex)
 - c. airway obstruction in case of associated trauma

10.13.2. Indications of ventilation [66,80]:

PaO₂ <70mm of Hg (with O₂) (80---100), PH < 7.25 (7.35---7.45)

PaCO₂ >55mm of Hg (40---45), Vital capacity <15ml/kg (65---75)

FEV₁<10ml/kg (50-60), Tidal volume <5ml/kg (5---8)

Normal hydration and blood pressure should be maintained. Inotropes, CVP monitoring should be used whenever needed.

Other associated problems will be treated.

- G. Temporary immobilization of spine [79]

Cervical orthosis [74,75,79,80]:

- A. Soft collar –do little to prevent spinal movement, allowing for 96 percent of normal neck flexion and 73% of extension.
- B. Hard collar: Hard collars allow 72-73% of normal flexion and extension.
- C. Philadelphia collar [74] –The most effective collar allowing for almost no neck flexion, although 35% of extension is possible, ineffective in preventing axial rotation and lateral bending
- D. Poster-type orthoses [74]- Gilford orthoses and sterno-occipito-mandibular brace
- E. Cervico-thoracic device [74] -Minerva jacket and Yale orthoses

Skeletal traction [44,79]: Tongs used – Gardner-wells tongs [81], Crutchfield tongs [82]

Gardner- Wells tongs is most commonly used, because they are easy to apply and less likely to dislodge than Crutchfield tongs.

After shaving of small area, under local anaesthesia and aseptic preparation. These tongs consists of a rigid semicircle that follows the contour of calvaria. A threaded hole on each side accommodates a screw for advancement of skull fixation points through the skin into the calvaria. One of the cone shaped points has a self-contained, spring-loaded indicator of the compressive force exerted by the pin. Both pins tilt upward to avoid the danger of the tongs being pulled out with increasing traction weight. The insertion site is usually 2 cm cephalad to the pinna, in line with the tragus, to allow for straightway axial traction. However, initial placement of the Gardner-wells tongs can be altered to treat specific fracture-dislocations. Anterior placement of the tongs may be needed to produce hyper extension in case of hyperflexion injury and posterior placement may be needed in case of unilateral or bilateral facet joint dislocation injuries to get reduction. After skin preparation, pins are then tightened by the hand until the indicator on the spring-loaded pin protrudes 1mm. This indicates approximately 30 pounds of

compressive force against the skull. At 24 hours of application, the spring-loaded pin should be tightened, if needed. The amount of weight varies with the level of injury and the extent of ligamentous disruption. Debate exists in the literature regarding the amount of literature and the maximum weight that should be used. Minimal weight should be used initially to avoid over distraction and neurological deterioration. Crutchfield developed a rule of 5 lb per cervical level, starting with 10lb for head. After that weight may be increased every 30 or 60 minutes with 5 or 10lb. Complications of cervical traction includes pin site infection, tong dislocation with bleeding, over distraction with neurological worsening, skull penetration and meningitis or brain abscess or intracranial hemorrhage.

The main disadvantage of cervical traction as a definite therapy for cervical spine injury is confinement of the patient to the bed until bony fusion occurs which requires 8 to 12 weeks. For this reason, there has been a trend over the last two decades towards the initial realignment by tong traction followed by operative stabilization through anterior or posterior approach. Alternatively, long-term immobilization can be achieved with the halo orthosis, which allows earlier mobilization of patients.

Medical treatment of spinal cord injury [83-86]: Several trials of pharmacotherapy have been conducted, focusing on the effects of either steroids or gangliosides.

Steroids –

Mechanism of action includes

- 1) reducing the effects of lipid peroxidation
- 2) Improves spinal cord blood flow
- 3) Enhancing activity of Na⁺/K⁺ ATPase
- 4) Facilitating recovery of extracellular Ca⁺ ions

The first North American Spinal Cord Injury Study (NASCIS I) [83] –examined role of low and high dose methylprednisolone for 10 days in spinal cord injury. No difference was seen regarding outcome. This study lacked control group.

The second (NASCIS II) [84] trial was a prospective, randomized, double blind multicenter trial that demonstrated improved neurological outcome at 6 weeks, 6 months, and 12 months in patients who received a regimen of methylprednisolone in the dose of – 30 mg/kg of body weight IV bolus followed by 5.4mg/kg/hour for 23 hours. Neurological improvement was seen when drug was administered within 8 hours of injury as compared to Naloxone or placebo.

Third trial (NASCIS III) [85] demonstrated that the same dose should be given for 23 hours if patient comes within 3 hours and for 48 hours when patient comes within 3 to 8 hours. 21-aminosteroids was compared with high dose steroid.

Ganglioside [86]: A prospective, randomized, double blind, multicenter trial showed beneficial effect of ganglioside GM1 in spinal cord injury if given within 3 hours of injury.

Long-Term Immobilisation

- A. Halo immobilization of cervical spine injuries
- B. Minerva jacket
- C. Surgical stabilization

Halo immobilization of cervical spine injuries [74]

Perry and Nickel first introduced the halo as a traction apparatus in 1959 for treatment of cervical spine instability due to poliomyelitis. This early halo system has undergone significant modifications to expand their role in the management of spinal instability. Cervical stability can be achieved by attaching the halo to a body jacket by means of metal uprights. Halo-jacket system consists of three components – the halo ring with pins, the plastic jacket and adjustable uprights that connect the halo ring to the jacket. Once in place, it allows less than 4% of normal flexion-extension and less than 1% of normal rotation.

- A. Application 1: patient positioning – supine on bed with head beyond the edge. Traditionally, halo is held in position by the first assistant and the second assistant assists the surgeon with the materials. However, a specially designed positioning fixture is available to hold the halo and support the fixture.
- B. Fixation of the halo ring – halo ring are generally available in different sizes. Approximately 1 to 1.5cm halo-skull clearance should be present. Anterior pins of halo should be placed 1cm above the lateral third of the eyebrow. Posterior pins will be placed 1.5cm posterior to the ear and 1cm above the pinna. Aseptic skin preparation and local anaesthesia is required. One anterior and the diagonally opposite pin are simultaneously tightened with maximum torque of 6kg.

- B. Attachment of the jacket – anterior and posterior half of the jacket was connected with the strap and the jacket has been connected with halo ring by uprights. Adjustments among uprights are possible to get desired flexion, extension traction etc.
- C. X-ray - to ensure desired spinal alignment

Complications of halo –

- A. pin site infection, osteomyelitis
- B. superior migration
- C. skin, muscle necrosis
- D. brain abscess, hemorrhage
- E. overdistraction, malpositioning with neurological deterioration

After care – 1) pin site dressing 2) Soft tissue checking for sore 3) Retightening of pin after 24 hours.

- A. Minerva jacket [79]: it is hot, heavy and uncomfortable. Radiological evaluation is difficult with this plaster of Paris jacket. Adjustment of head and neck position is also impossible with this jacket.
- B. Surgical Management [79,87,88]: Decompression, fusion and stabilization-through anterior and/or posterior approach. Issues regarding the role of decompression and the timing of decompression have already been discussed.
- C. If the injury primarily involves the anterior column, a Smith-Robinson or standard anterior approach to the spine is used to allow anterior decompression and reconstruction with either allograft or autograft iliac crest or fibula followed by stabilization with anterior locking plates.
- D. Anterior surgical approach is recognized as being more effective in achieving decompression in cases involving anterior encroachment of neural elements (28,86).
- E. Blood flow studies indicated that initial (4) concerns of compromising local cord perfusion by the surgical approach are not accurate. Local blood flow is not altered or compromised by anterior surgical approach (87).
- F. The posterior approach is indicated when the pathoanatomy involves the posterior elements and is the basic midline approach with muscle retraction off the cervical spine to the lateral aspect of the facet joints bilaterally. Occasionally, a dual approach is necessary to remove an offending disk fragment anteriorly prior to reduction, followed by posterior stabilization with anterior reconstruction. These global injuries are usually quite unstable and benefit from both an anterior and posterior reconstruction. Neurological recovery is not dependant on approach (anterior or posterior). Any of one or both can be done according to pathoanatomy of injury [89].

Intraoperative details: When the patient is taken to the operating room, certain issues must be addressed. If the patient has been in traction or a halo for reduction of the spine, this must be maintained in the transfer to the operating table and while on the operating table. This sometimes requires fibre-optic intubation, with meticulous handling of the cervical spine during the entire process. Thus, the surgeon, anesthesiologist, and nursing staff must all be working in concert to ensure the patient's safety.

The operative approach obviously dictates whether the patient is prone or supine, and in some instances, both are needed for the combined approach. Whatever the position of the patient, the hips, knees, shoulders, hands, and feet should all be well padded to prevent pressure sores and peripheral nerve palsies.

- A. **Operative technique:** Anterior approach [88,90]
- B. **Positioning:** Supine, with or without traction depending on pathology, mild neck extension.
- C. **Mark incision:** Transverse incision, for long segment fixation incision along anterior border of sternocleidomastoid can be given.
- A. **Exposure:** After painting and draping, incision has to be given along above-mentioned incision down to platysma. Platysma is undermined. Investing layer of deep cervical fascia is excised. Plane developed in between tracheo-oesophageal bundle and carotid sheath towards infero-medially to reach anterior surface of vertebrae. Opening is enlarged after soft tissue release. Prevertebral fascia is excised. Longus coli muscle is elevated and retracted by Cloward self-retaining or hand-held retractor. Level is confirmed by C-arm on table with localiser. Discectomy or corpectomy is done according to need of that particular patient under microscope. For discectomy anterior longitudinal ligament and anterior annulus is incised with 11 no blade. Superficial discectomy is done and then vertebral spreader is placed in the disc space to distract the disc space and facilitate deep part of discectomy by curette and disc forceps. Posterior osteophytes are removed by Kerrison's punch to decompress foramina. Posterior longitudinal ligament is cut to see dura, epidural space and also to remove any subligamentous disc. For corpectomy, vertebral body is removed with nibbler, drill and Kerrison's punch after two discectomies. Bone

graft is taken from iliac crest and anterior column is reconstructed and checked by C-arm. Fixation is done by anterior cervical locking screw and plate under guidance of C-arm. Haemostasis is achieved, wound is washed with diluted betadine lotion and wound is closed in layers with a suction minivan drain in prevertebral space.

B. Posterior approach: Positioning: prone with head fixed in Mayfield head holder in neutral neck position with padded pressure points and semi-flexed limbs.

C. Incision: midline vertical incision from one level below and one level above the spinous process to be fixed.

D. Exposure: after painting and draping, incision is given along the above-mentioned incision through the avascular plane of ligamentum nuchae down to spinous process. Paraspinal muscles are elevated from laminae and retracted laterally to expose laminae and lateral mass. Laminectomy may be done depending on the pathology. Luque rod stabilization or lateral mass fixation is done followed by bone grafting. After haemostasis, wound is closed.

E. Spinal cord monitoring [43]: The indications for spinal cord monitoring are also somewhat controversial among surgeons. Somatosensory evoked potentials (SSEPs) offer a safe, noninvasive, and continuous technique for assessing the functional integrity of the spinal cord. Several reports suggest that changes in evoked potentials are predictive of neurologic change in the patient. When a signal deformity is detected, it can be checked with a wake-up test to ensure its accuracy.

However, the delayed nature of the change makes these tests of limited efficacy in preventing intraoperative injury. A major problem with the technique is that multiple false-positive and false-negative results that have been reported. In addition, the monitoring is only as good as the skill of the personnel performing the monitoring. In summary, SSEPs and motor-evoked potentials (MEPs) may have some benefit in the neurologically intact patient in whom significant intraoperative manipulation of the spinal column is anticipated.

F. Postoperative details [4,43]: Postoperatively, patients usually are maintained in some sort of cervical orthosis, depending on the quality of fixation obtained. Fixation techniques have improved dramatically over the past 10 years. Before, most patients would be in a halo postoperatively; now, patients are being placed in only a rigid or soft collar for comfort. The patient should be mobilized out of bed as soon as possible to prevent pulmonary and DVT complications. A return to independence should be approached aggressively, with the help of a comprehensive team including a physical therapist, social service personnel, and family. In patients with spinal cord injury and neurologic deficit, extensive physical and emotional rehabilitation should be instituted immediately following surgery to ensure the best physical and psychological outcome for the future. Postoperative management of bowel and bladder dysfunction should include instruction in intermittent catheterizations as well as the use of alternate day suppositories and a high-fiber diet to assist with bowel function and avoid impaction. Close attention to skin breakdown should include frequent turning of the patient and, as mentioned above, prompt mobilization.

G. Follow-up care [4,43,91]: Patients must be monitored closely for the first 4 weeks postoperatively to ensure maintenance of spinal alignment and no evidence of neurologic deterioration. Encouragement should be given to patients for independence, and attempts to regain preinjury activity levels should begin. Two to 3 months of healing usually is expected, and radiographic signs of osseous healing should be noted in this timeframe. Some patients may require up to 6 months to achieve preinjury activity levels, and others, based on concomitant injuries and age, may never reach this status. Aggressive therapy should continue during this entire period, and, once the injury is healed and the patient is functioning at maximal levels, return to full activity is begun. During the follow-up period, any deterioration in neurologic function should prompt further investigation to include MRI for posttraumatic syrinx or other intrinsic or extrinsic compressive lesions [92-107].

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