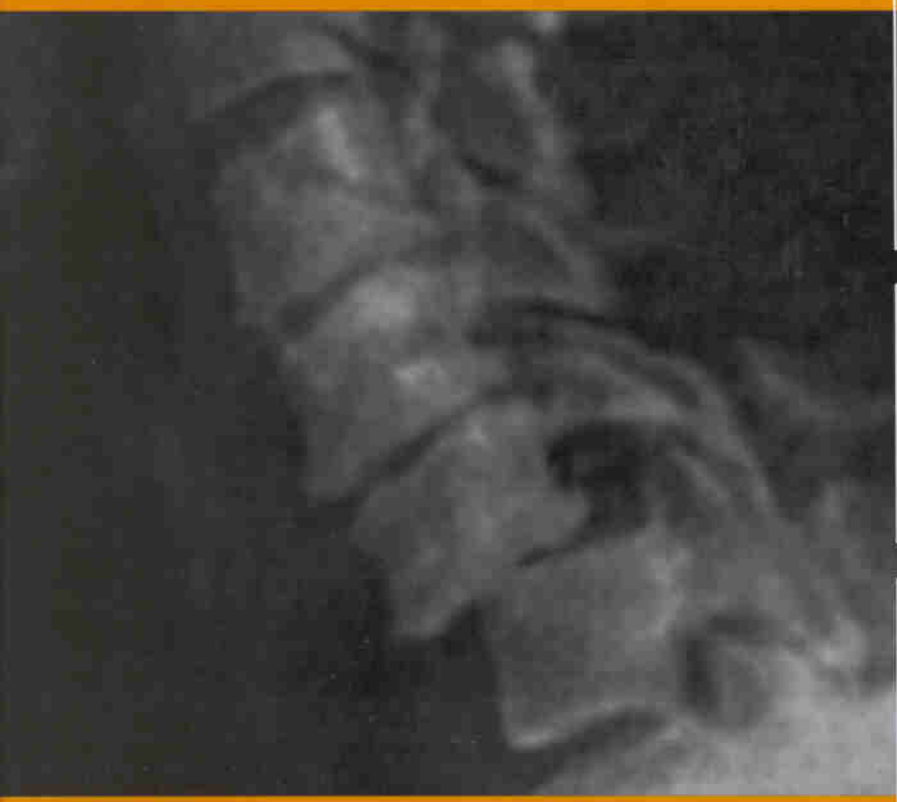
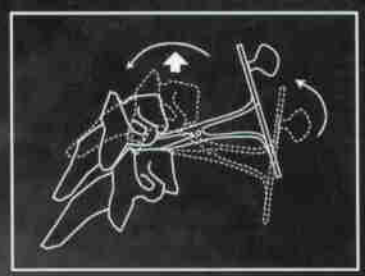
FRACTURES OF THE CERVICAL, THORACIC, AND LUMBAR SPINE





ALEXANDER R. VACCARO

FRACTURES OF THE CERVICAL, THORACIC, AND LUMBAR SPINE

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MARCEL DEKKER, INC. NEW YORK • BASEL

CRC Press
Taylor & Francis Group
6000 Broken Sound Parkway NW, Suite 300
Boca Raton, FL 33487-2742

Version Date: 20130723

International Standard Book Number-13: 978-0-8247-4397-0 (eBook - PDF)

Preface

Spinal trauma is a potentially devastating occurrence associated with significant morbidity and mortality. Fortunately, with improved ''in-field'' management protocols, ''in-hospital'' resuscitation measures, and further insight into the pathophysiology of spinal cord injury, progress is being made toward improving neurological function and quality of life for this patient population. In order to optimize postinjury intervention efforts by spinal care providers, a thorough understanding of all facets of spinal injury and spinal cord injury pathomechanics must be attained.

The medical literature is replete with generalized assessment and treatment guidelines for broad groups of spinal injuries; it lacks, however, a comprehensive and precise investigation into the particulars of various subgroups of traumatic pathology. Each fracture type has individualized assessment concerns, fracture subclassifications, immobilization techniques, nonoperative and operative indications, operative fixation strategies, and prognostic outlook. It is imperative that such vital information be included in any volume exploring contemporary spinal injury management. A comprehensive textbook that allows quick and easy reference to fracture subtypes, spinal injury management protocols, and aftercare is indispensable to the spinal care provider regardless of the level of academic training.

In an effort to develop a broad, in-depth but readable text on the comprehensive management of spinal injury, world-renowned experts were asked to focus on particular issues of spinal pathophysiology and fracture subtypes rather than on regional spinal pathology, as is often the case in other contemporary textbooks. This approach is invaluable when precise and current information is necessary to manage common and uncommon spinal traumatic pathology.

This book serves as an up-to-date review and a comprehensive resource. With a thorough understanding of the contemporary issues surrounding spinal trauma management, the spinal care provider can supply the best care and optimize the potential for gainful rehabilitation.

Alexander R. Vaccaro
Justin P. Kubeck

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1

Spinal Injury: Etiology, Demographics, and Outcomes

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I INCIDENCE AND PREVALENCE

The incidence of spinal cord injury (SCI) has been estimated to be between 30 and 40 cases per million per year, or about 10,000 new cases annually (1). A recent report from a state-based registry found an incidence rate of 59 cases per million for patients in hospitals, and 77 per million including prehospital fatalities (2). There is not sufficient reporting of SCI in the United States to determine whether the incidence has changed in recent years. Prevalence of SCI has been estimated to be between 721 and 906 persons per million population, or about 183,000 to 230,000 persons in the United States (1).

II DEMOGRAPHICS

The information regarding the demographics and case descriptions of spinal cord injury patients in this chapter comes from the Model Spinal Cord Injury Systems of care funded by the National Institute on Disability and Rehabilitation Research, Office of Special Education and Rehabilitative Services, Department of Education. The Model SCI Systems program has been funded since 1970, and data collected in a national SCI database (the Database) since 1973. Over the years, the number and geographical spread of centers have varied. The Database is not population-based, so it cannot be used to determine incidence and prevalence. Demographic data represent an incidence series of cases admitted to the Model SCI Systems, which may differ from prevalence data. The Model SCI System, the Database, and limitations of the Database have been described elsewhere (3,4). Where relevant, information

from the Database is supplemented by data from state registries of SCI and other sources.

A Age at Injury

Data from the Database indicate that the mean age at injury is 32.3 years [standard deviation (sd) = 15.8 years], and the median age at injury is 27 years (4). The highest incidence occurs in the 16- to 30-year-old range, with 54% of all injuries (Fig. 1) (4). The percentage of individuals over age 60 sustaining a SCI has been climbing steadily in the Database, from 4.5% during the 1973 to 1977 period to 11.5% during the 1994 to 1998 period (4). This trend reflects the aging in the general population over the same time.

B Sex

Males sustain SCI more frequently than females by a 4:1 ratio. In the Database, 81.5% of the sample is male (4). This is a slightly higher proportion than that reported by most state registries. The proportion of males in state registries ranges from a low of 69% in Louisiana (5) to a high of 80.4% in Arkansas (6).

C Race/Ethnicity

Of those entered in the Database since 1990, 58.1% are White, 28% are African-American, 8.4% are Hispanic, 0.4% are Native American, and 2.1% are Asian (7). These proportions are significantly different from the proportions in the general population, where 80.3% are White (8). State registries also demonstrate this disproportionate inclusion of minorities. The incidence of SCI among African-Americans is nearly twice that for Whites in Louisiana (5) and Virginia (9), while the rates were found to be similar in Mississippi (2). Where rates for minorities are increased, much of the increase has been due to greater rates of violence as a cause of SCI (6,10).

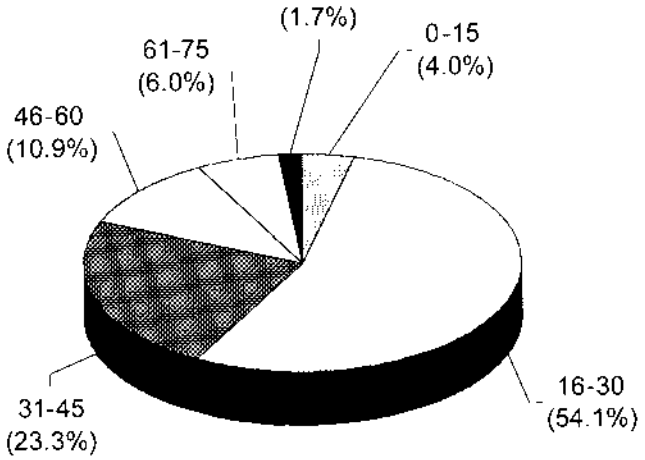

Figure 1 Age at injury. (From Ref. 4.)

III ETIOLOGY

Since 1990, the most frequent cause of SCI in the Database has been motor vehicle crash (37.4%), followed by violence (25.9%) and falls (21.5%) (Fig. 2) (7). Over time, there has been an increase in the proportion of injuries due to violence and falls and a decrease in the proportion due to motor vehicle crashes (7). Due to limitations of the Database, it is not clear whether these changing proportions are due to changes in incidence rates or other factors, such as a change in the centers that contribute to the Database or changing referral patterns. However, others have noted an increasing proportion of admissions due to violence-related SCI (11).

Etiology of SCI is influenced by age, gender, and race/ethnicity. Vehicular crash is the leading cause of SCI up to age 45, after which falls become the most common etiology (Fig. 3) (1). The proportion of injuries due to violence and sports decreases with age, while that due to falls increases. According to the Database, the leading causes of SCI for both males and females are auto accidents, falls, and gunshot wounds. However, auto accidents account for a higher proportion of injuries in females than males (51.5 vs. 31.4%) and gunshot wounds for a higher proportion in males than females (18.3 vs. 11.3%) (4). Males are more likely than females to sustain SCI as a result of diving (8.0 vs. 3.2%) and motorcycle accidents (6.1 vs. 1.7%) (4). Etiology of SCI by race is shown in Fig. 4 (4). Vehicular crash is the leading cause of injury for Whites, Native Americans, and Asians, while violence is the leading cause of injury for African-Americans and Hispanics (4).

IV SEVERITY OF INJURY

There are approximately equal proportions of people with complete and incomplete injuries in the Database, with a slightly higher percentage of persons with tetraplegia than paraplegia. Based on neurological status at discharge, SCI is classified as complete tetraplegia in 23.3%, incomplete tetraplegia in 30.2%, complete paraplegia in 26.1%, and incomplete paraplegia in 19.7% (4). The severity of injury is dependent upon etiology (Fig. 5) (4). Vehicular crash results in incomplete tetraplegia in about

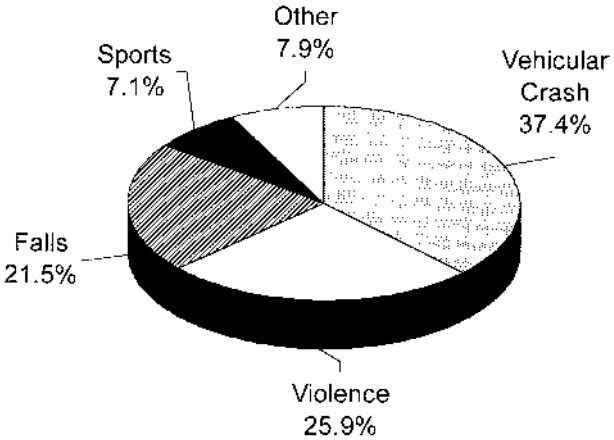

Figure 2 Etiology of SCI since 1990. (From Ref. 7.)

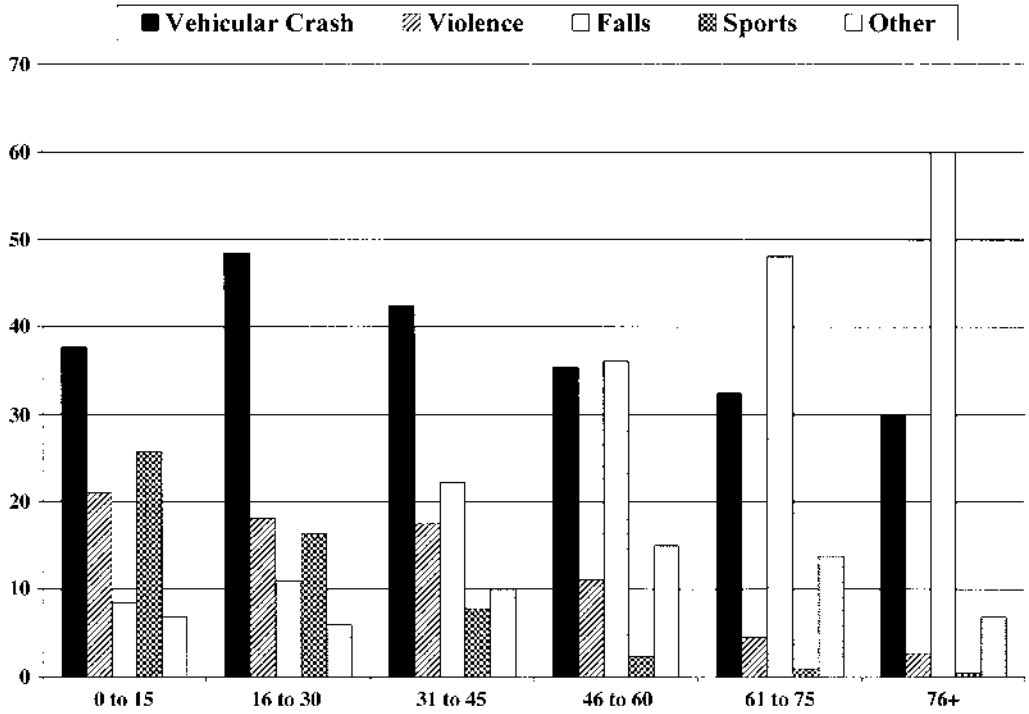

Figure 3 Etiology of SCI by age group. (Adapted from Ref. 1.)

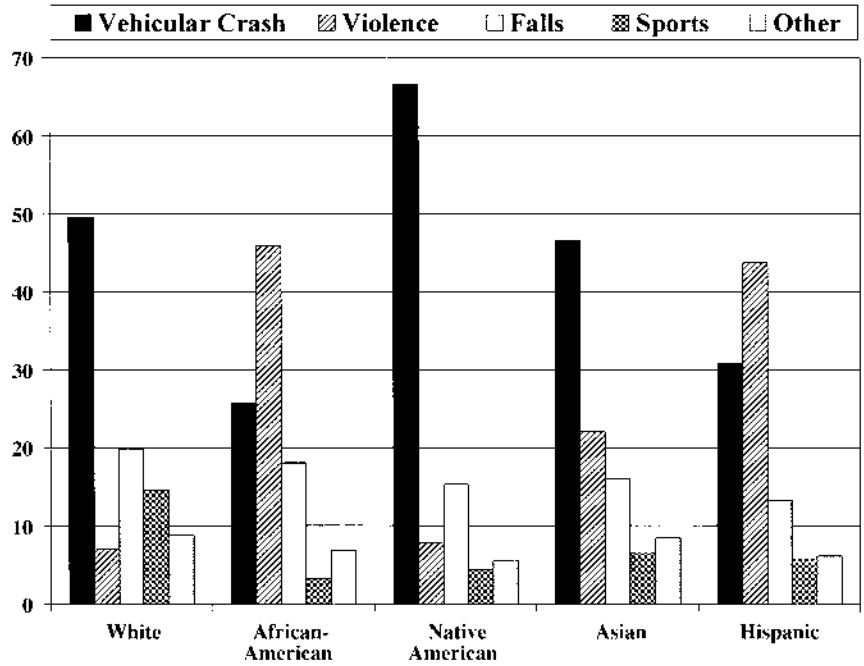

Figure 4 Etiology of SCI by racial/ethnic group. (Adapted from Ref. 4.)

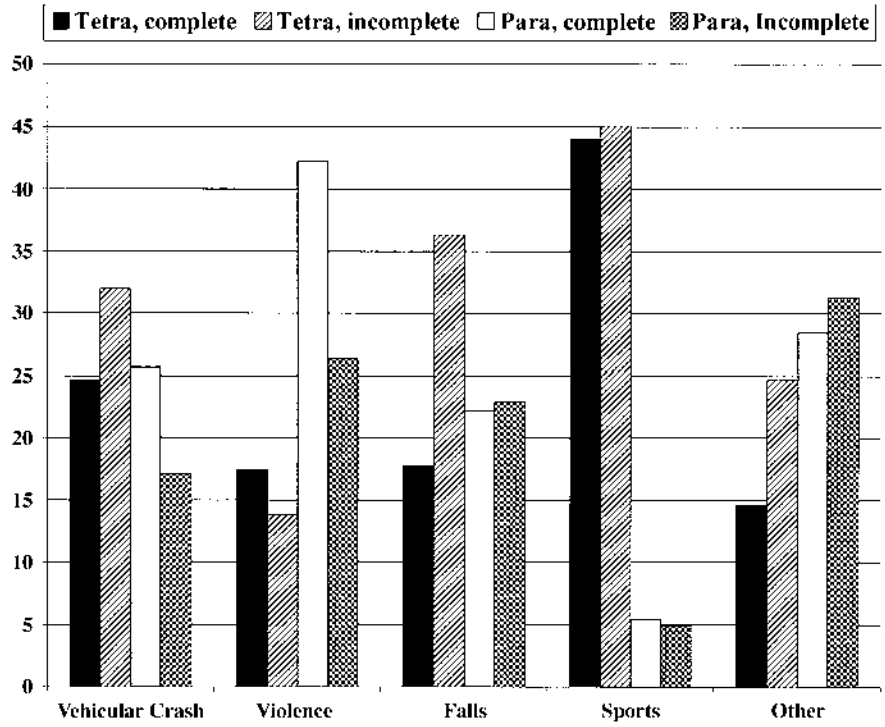

Figure 5 Severity of SCI by etiology. (Adapted from Ref. 4.)

one-third of cases, and in complete tetraplegia or complete paraplegia in 25% of cases each. Acts of violence are more likely to result in paraplegia (68% paraplegia: 42% complete, 26% incomplete), while sports injuries largely result in tetraplegia (89% tetraplegia: 44% complete, 45% incomplete) (4). Compared to nonviolent injuries, SCI caused by violence is more likely to be complete at admission and to remain complete at discharge (12).

V COSTS

Based on 10,000 new cases of SCI yearly, the total direct cost for all causes of SCI in the United States has been estimated at $7.736 billion (13). This estimate does not include indirect costs such as lost wages, fringe benefits, and productivity, which can account for up to 65% of total aggregate costs (14). Lifetime costs vary by severity of injury and age at injury (Table 1). Higher costs are associated with more severe injuries and with younger age at injury. These data, derived from the Model Systems, may overestimate costs for incomplete SCI because they include only those individuals who required inpatient rehabilitation. Johnson et al. (15) used a state-based registry to examine costs of all SCI in Colorado (15). They found that 30% of individuals with SCI received no inpatient rehabilitation services. This is reflected in the lower average first-year cost for those with motor functional injuries, only $60,267 in 1992 dollars (15).

Table 1 Direct Costs of SCI by Severity of Injury (in 1999 dollars)

Severity of injury	Average cost first year	Average cost each subsequent year	Lifetime cost if age 25 at injury	Lifetime cost if age 50 at injury
High tetraplegia (C1-C4)	$549,800	$98,483	$2,100,185	$1,236,390
Low tetraplegia (C5-C8)	$355,037	$40,341	$1,187,507	$752,019
Paraplegia (all levels)	$200,897	$20,442	$701,716	$478,614
Motor functional (any level)	$162,032	$11,355	$468,097	$339,239

Source: National Spinal Cord Injury Statistical Center. Spinal Cord Injury: Facts and Figures at a Glance. Birmingham, AL: University of Alabama at Birmingham, 2000.

VI MORTALITY

Although life expectancy has been increasing for individuals with SCI, it still lags that of the general population, even for the least severe injuries (motor functional). Life expectancy is shorter as severity of injury and age increase, and is shortest for ventilator-dependent individuals (Table 2) (7). Individuals aged 60 and older who are ventilator-dependent have a life expectancy of only 1.2 years. Mortality is greater during the first year postinjury than in subsequent years, so that life expectancy is greater in those who survive 1 year after SCI (Table 3) (7). One-year mortality has steadily decreased since the 1970s, according to the Database. Compared to the 1973 to 1977 period, the odds of dying by 1 year postinjury for persons injured during 1993 to 1998 dropped by 67% (16).

During the first year postinjury, respiratory and cardiac-related causes account for over half of all deaths. After the first year, no single cause accounts for more than 20% of deaths. The top three causes of death after 1 year are heart disease (18.8%), external causes (18.3%), and respiratory complications (18.0%) (16). The only significant trend over time has been a decrease in deaths from urinary causes (16).

Table 2 Life Expectancy for Persons Who Survive the First 24 Hours After SCI

Age at injury (years)	No SCI	Motor functional (any level)	Para	Low-tetra (C5–C8)	High-tetra (C1–C4)	Ventilator-dependent (any level)
20	57.2	51.6	45.2	39.4	33.8	16.2
40	38.4	33.5	27.8	23.0	18.7	7.2
60	21.2	17.5	13.0	9.6	6.8	1.2

Source: National Spinal Cord Injury Statistical Center. Spinal Cord Injury: Facts and Figures at a Glance. Birmingham, AL: University of Alabama at Birmingham, 2000.

Table 3 Life Expectancy for Persons Who Survive at Least 1 Year Postinjury

Age at injury (years)	No SCI	Motor functional (any level)	Para	Low-tetra (C5–C8)	High-tetra (C1–C4)	Ventilator-dependent (any level)
20	57.2	52.5	46.2	41.2	37.1	26.8
40	38.4	34.3	28.7	24.5	21.2	13.7
60	21.2	18.1	13.7	10.6	8.4	4.0

Source: National Spinal Cord Injury Statistical Center. Spinal Cord Injury: Facts and Figures at a Glance. Birmingham, AL: University of Alabama at Birmingham, 2000.

VII SUMMARY

There are between 183,000 and 230,000 people in the United States with traumatic SCI, with about 10,000 new cases annually. While relatively infrequent, costs of SCI are high, with lifetime costs for new injuries estimated at $7.736 billion. SCI occurs predominantly in males and in the young (<30 years of age). Compared to younger individuals, older individuals are more likely to sustain SCI as a result of falls, and less likely as a result of violence or sports. African-American and Hispanic minority groups have a higher incidence of violence-related SCI than Whites. Severity of injury is influenced by etiology. Mortality is decreasing, but life expectancy continues to lag that of the general population, particularly in more severe injuries.

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2

Anatomy and Pathophysiology of Traumatic Spinal Cord Injury

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I GROSS ANATOMY

The spinal cord extends from the foramen magnum to approximately the L1–L2 disc space. It is continuous with the medulla oblongata and terminates in the conus medullaris. Below this level the nerve roots running inferiorly are collectively called the cauda equina (Fig. 1A–D). The cauda equina runs within the spinal canal, which is bordered anteriorly by the vertebral bodies and posteriorly by the dorsal bony arch. The spinal canal measures approximately 45 cm in length in males and 42 cm in females (1). There are two enlargements in the spinal cord that run from C4 to T1 and from L2 to S3. These enlargements correspond to areas of upper and lower extremity innervation. In all there are 31 nerve roots that branch from the spinal cord (8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 1 coccygeal). In cross-section the cord is slightly flatter in the AP plane. It reaches its maximum transverse diameter between C3 and C6, which is approximately 13 to 14 mm (2).

Membranous layers covering the spinal cord are referred to as the meninges (3). The meninges consist of three layers—the dura, arachnoid, and pia mater. The dura is attached anteriorly to the posterior longitudinal ligament. The pia mater is composed of a superficial layer (epi-pia) and a deep layer (pia-glia). This pia mater

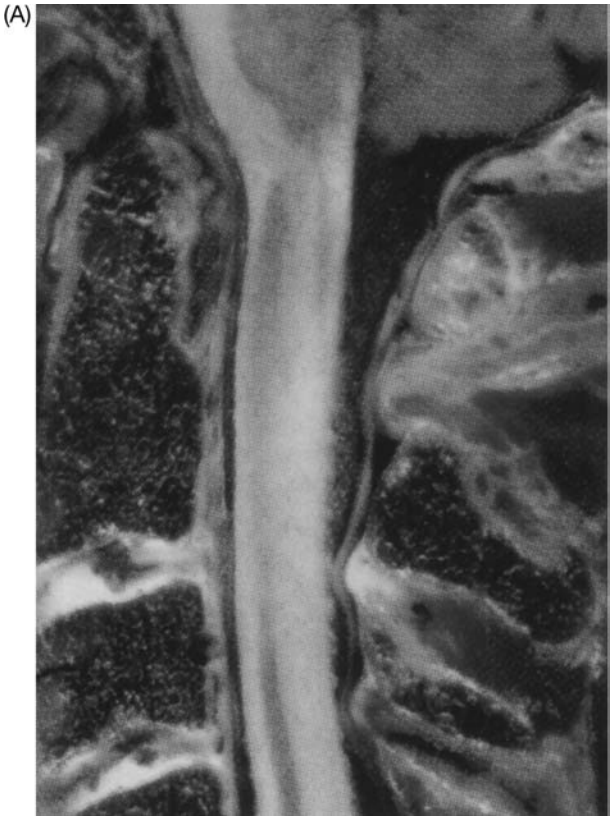

Figure 1 (A) Midsagittal section through the upper cervical spine of a 34-year-old man. The odontoid process is the most prominent structure. The synovial joint between the anterior arch of the atlas and the dens shows signs of slight degeneration. Note also the transverse portion of the cruciate ligament which holds the odontoid process posteriorly. The transverse ligament is covered by the tectorial membrane which constitutes a reinforcement of a parietal blade of the dura mater and which is continuous with the dura mater of the skull. In addition, the thin apical ligament of the dens directly anchors the tip of the dens to the clivus portion of the foramen magnum. Posteriorly, the thin atlanto-occipital membrane connects the posterior arch of the atlas with the rim of the foramen magnum.

invests the spinal cord. A small thread of the pia mater extends from the distal end of the spinal cord as the filum terminale, which connects the conus medullaris to the periosteum of the first coccygeal vertebrae.

Topographically there are six distinct grooves in the spinal cord (4) (Fig. 2). Posteriorly there is a posterior median sulcus and two posterior lateral sulci. These two posterior lateral sulci correspond to the regions of entrance for the posterior rootlets. They are referred to as the dorsal root entry zones (DREZ). Anteriorly in the midline there is an anterior median fissure in which the anterior spinal artery runs. There are two ventral lateral sulci that correspond to the exit zones for the anterior rootlets. They are referred to as the anterior root exit zones (AREZ) (5).

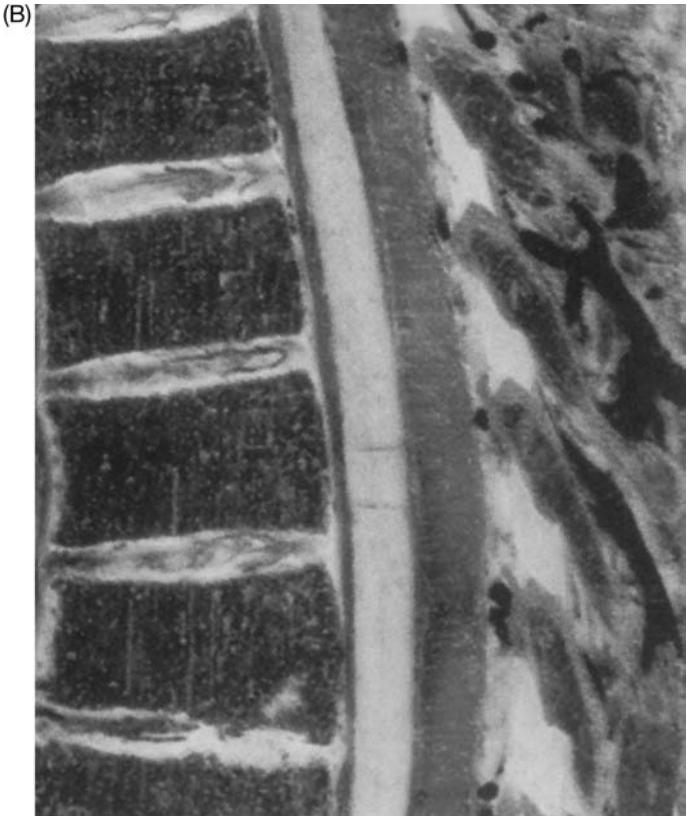

Figure 1 (B) Midsagittal section through the midthoracic spine of a 64-year old female. Due to the supine position of the cadaver, there is engorgement of the deep posterior veins. All disks show degenerative changes. The disk between T9 and T10 is completely resorbed and the cartilaginous end plates have fused. On the most spondylitic segments, the anterior longitudinal ligament is thicker than in the less degenerated segments. Normally thoracic disks have a perfectly straight posterior margin. Of note is the relationship of the laminae to the intervening ligamentum flavum. The long slender spinous processes as well as the flat wide laminae overlap like obliquely sloping shingles, completely hiding the ligamentum flavum. The latter attaches to the adjacent laminae in a consistent fashion: in the anterior surface of the lamina above and in the upper rim of the lamina below. Viewed from the spinal canal (anteriorly), only a narrow band of bone is visible; the posterior wall of the spinal canal thus is predominantly elastic-ligamentous, yet shielded by the "hidden" lamina portion. Note that the veins behind the dura (belonging to the posterior internal venous plexus) are invariably located at the level of the bony lamina, not the ligamentum flavum.

II MICROSCOPIC ANATOMY

Internally the spinal cord is divided into gray and white matter (Fig. 2). The gray matter is roughly in the shape of the letter "H" (6). It is composed of nerve cell bodies of efferent and interneural neurons arranged into distinct vertical columns, connective tissue, and vascular components. This gray matter extends the entire

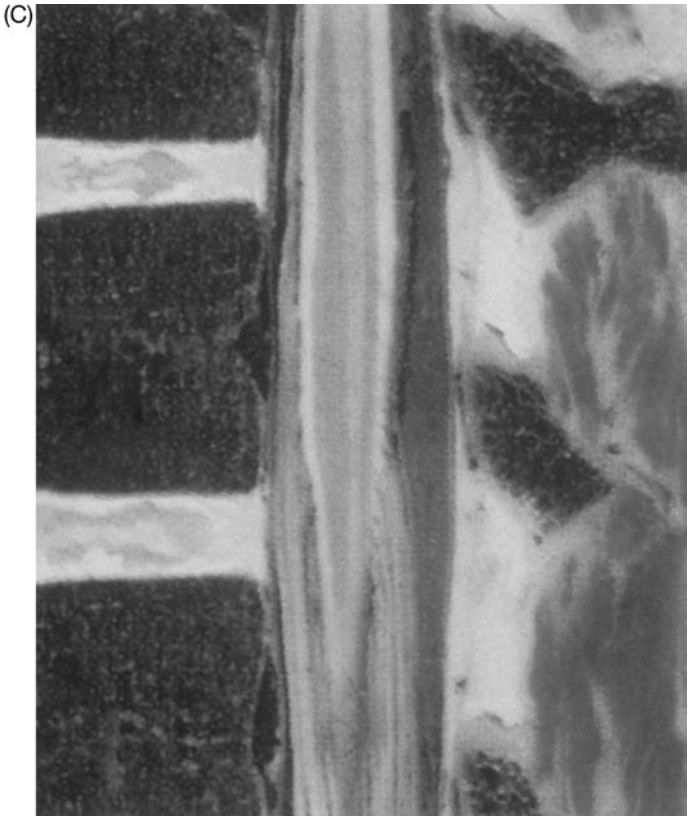

Figure 1 (C) Sagittal section in the midline through the thoracolumbar spine of a young female adult. The conus medullaris typically terminates at the L1-L2 disk level. This specimen also shows the caudal extension of the conus, the filum terminale, and also outlines the central canal of the spinal cord. The upper two vertebrae show vascular venous outlet foramina (Batson) through which the veins in the vertebral body communicate with the anterior epidural veins. Note the marked increase in disk height from the lower thoracic to the upper lumbar spine. The posterior annulus of all disks is straight, rendering the anterior wall of the spinal canal straight. The conus medullaris is surrounded by bundles of thick cauda equina roots, motor anterolaterally and sensor dorsolaterally. The intrathecal reserve space is much smaller than in the thoracic and cervical spine.

length of the spinal cord. Surrounding the gray matter is the white matter, which is composed of ascending and descending fibers in distinct tracts. The gray matter is divided into three horns, posterior, intermediate, and anterior (7). The posterior horn is composed of somatosensory neurons, interneurons, and tract cells. This posterior horn is further subdivided into three main segments. The substantia gelatinosa relays information regarding pain, temperature, and touch (7). The nucleus proprius relays information concerning proprioception, two-point discrimination, and body movement. Both of these run the entire length of the spinal cord. The nucleus dorsalis relays proprioceptive information and runs from C8 to L3.

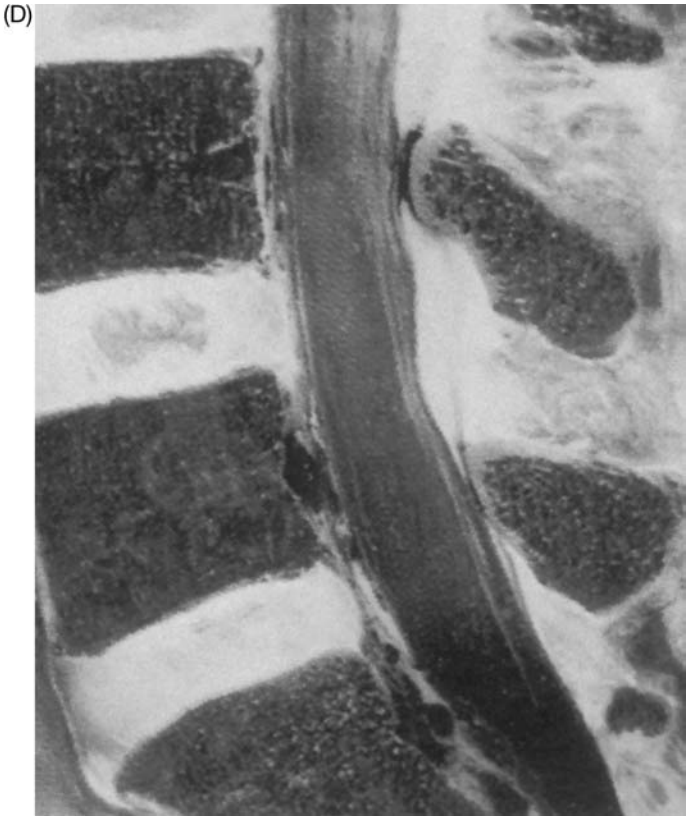

Figure 1 (D) Sagittal section through a normal lower lumbar spine and upper sacrum of a young female through the lateral portion of the thecal sac. The segmental root bundles converge toward each intervertebral foramen. The L3, L4, and L5 disks all display a slight posterior convexity, and the outermost layers of the annulus fibrosus attach beyond the apophyseal ring. The vertebral bodies typically have a concave posterior contour. In the midportion of the posterior vertebral wall at L5 a large venous vascular foramen (outlet foramen of the Batson plexus) exists through which veins traversing the vertebral bodies communicate with the ventral internal venous plexus. The laminae have a characteristic shape: toward the spinal canal only a narrow vertical band of cortical bone is exposed, superiorly the laminae have a sharp ridge, and from behind the laminae slope postero-inferiorly. The ligamentum flavum attaches superiorly to the laminae's inferior posteriorly receding surface. At the infra-adjacent lamina the ligamentum flavum attaches to the sharp upper ridge and a small area behind it. Note that the ligamentum flavum at the lumbosacral level is much thinner than at levels above.

The intermediate gray matter is composed of cells of the preganglionic sympathetic neurons from T1 to L2 (6). It also contains preganglionic parasympathetic fibers from S2 through S4.

The anterior horn gray matter contains somatomotor neurons. It is subdivided into medial, lateral, and central columns (7). All of them function to innervate muscle units (1). The medial column innervates axial, abdominal, spinal, and intercostal

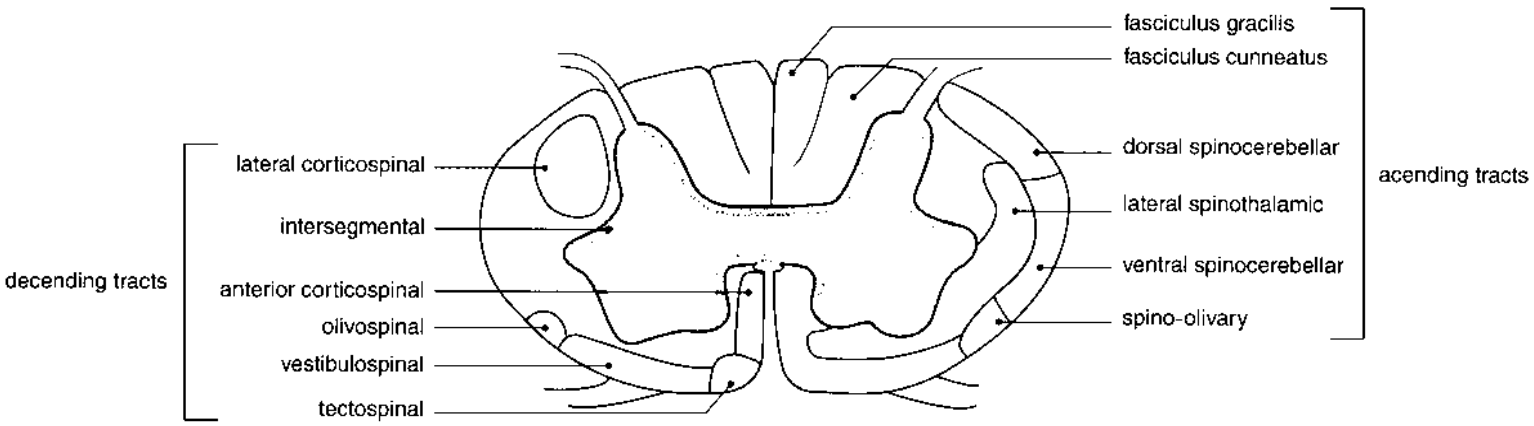

Figure 2 Cross-sectional depiction of the spinal cord. Sensory nerve roots enter posteriorly at the dorsal root entry zones, and motor nerve roots exit anteriorly at the ventral root exit zones. An anterior median fissure communicates the anterior spinal artery while a posterior median sulcus provides for a dorsal vascular channel. The H-shaped gray matter is composed mainly of neuronal cell bodies. The white matter is divided into dorsal, lateral, and ventral columns transmitting ascending and descending neuronal axons that are grouped into tracts: (1) fasciculus gracilis, (2) fasciculus cunneatus, (3) dorsal spinocerebellar tract, (4) ventral spinocerebellar tract, (5) lateral spinothalamic tract, (6) spino-olivary tract, (7) anterior corticospinal tract, (8) tectospinal tract, (9) vestibulospinal tract, (10) olivospinal tract, (11) intersegmental or propriospinal tract, (12) lateral corticospinal tract.

muscles. The central portion innervates the diaphragm. The lateral portion innervates the appendicular skeleton and is present only in the cervical and lumbosacral regions.

The white matter of the spinal cord is composed of motor and sensory nerve tracts, connective tissue, and vascular tissue (Fig. 2). These ascending and descending fibers are organized into distinct tracts. The white matter is divided into posterior, lateral, and anterior columns (1).

The posterior column lies between the posterior horns of the gray matter. It is divided centrally by the posterior median septum. Below T6 it is composed of the fasciculus gracilis and above this level it is joined by the fasciculus cuneatus (6). These two tracts are separated by the posterior intermediate sulcus on each side. They carry proprioception, vibratory, and tactile information from the lower and upper extremities, respectively (8).

The lateral column is located between the anterior and posterior root entry zones (4). It contains both ascending and descending tracts. The lateral corticospinal tract is a descending pathway relaying voluntary motor function. This is a crossed tract. The fibers are arranged so that more caudad motor fibers are located laterally. The posterior spinocerebellar tract is an ascending uncrossed tract that relays pain as well as proprioception, touch, and pressure. The anterior spinocerebellar tract is similar, only its fibers are crossed. The lateral spinothalamic tract is an ascending crossed tract relaying pain and temperature sensations. The spinotectal tract is an ascending crossed pathway relaying information of pain, temperature, and tactile sense to visual reflexes. The spinoreticular tract carries sensory input from the skin, joints, and muscles. The spino-olivary tract relays cutaneous and proprioceptive input. This area of the spinal cord also carries autonomic fibers of a descending nature to regulate visceral functions.

The anterior column lies between the two anterior root entry zones. The anterior corticospinal tract carries 10 to 15% (6) of the uncrossed pyramidal tract fibers that did not become part of the lateral corticospinal tract. The anterior spinothalamic tract carries ascending fibers relaying information regarding light touch. The tectospinal tract is a crossed tract relaying information to control reflexes in response to visual stimuli. It terminates in the upper thoracic cord. The medial and lateral vestibulospinal tracts are descending fibers relaying information regarding muscle control and equilibrium from inner ear and cerebellum.

III PATHOPHYSIOLOGY

Neurological dysfunction following traumatic spinal cord injury is a result of both an initial mechanical insult and ongoing processes that disrupt normal cord anatomy and function. The primary traumatic insult may cause cord transection, compression, or distraction that disrupt neuronal and glial architecture. The amount of energy applied to the cord, the specific mechanism of injury, the level of injury to the spinal cord, and patient factors including the preinjury space available for the cord and medical comorbidities all help determine the pattern and degree of neurological deficit following this primary insult (9). The secondary cascade of events then exacerbates injury to the cord and may propagate the spinal cord injury caudally or rostrally, which explains why some patients with acute spinal cord injury may experience neurological deterioration in the acute postinjury period. While the primary traumatic insult can be addressed by clinicians only through developing and implementing

preventative measures, understanding the secondary processes of spinal cord injury may afford an opportunity for therapeutic intervention.

The secondary cascade was first suggested almost a century ago by Allen (10), who highlighted the deleterious effects of ongoing hemorrhage, edema, and ischemia. Secondary mechanisms have since been found to involve vascular dysfunction leading to ischemia and hemorrhage, inflammation with cellular and molecular mechanisms of continued neuronal and glial destruction, and other modes of injury including excitotoxicity and apoptosis (Fig. 3). In paralleling the discovery of these processes, it is useful to examine the pathophysiology of spinal cord injury first on a histopathological level and then on a molecular level. Agents that may intervene in some of these secondary processes give further insight into the pathophysiology of spinal cord injury.

IV HISTOPATHOLOGICAL CHANGES FOLLOWING SPINAL CORD INJURY

The first changes evident in spinal cord anatomy following traumatic injury are punctate hemorrhages in the gray and white matter within 15 min of injury. Disruption of thin-walled capillary or postcapillary venules causes bleeding, and red-cell diapedesis into the substance of the cord may result from inflammatory second messenger effects on blood vessel endothelium (11). The area involved in bleeding appears to progress centrifugally, with hemorrhagic lesions found predominantly in the gray matter but also in the white matter 3 to 5 days postinjury (12).

An edematous reaction also begins within this central area of hemorrhage and progresses outward. Presumably from inflammatory changes in endothelial leakiness, the interstitial swelling develops at first as a nonproteinacious edema. With further cell breakdown and intracellular/extracellular osmotic imbalance, the interstitial space becomes engorged with protein-rich fluid. The swelling obliterates the subarachnoid and subdural spaces. The edema and hemorrhage progress outwardly both at the level of injury as well as rostrally and caudally, which may be evident as soon as 4 h after the trauma (13).

Occlusion of small intramedullary and pial arteries and veins has been documented within 20 min of injury. Larger vessels, including the anterior and posterior spinal arteries and sulcal arteries, appear to be spared (12). The smaller vessels that do get occluded undergo a process of thrombosis and fibrinoid necrosis. The resultant ischemia helps to propagate spinal cord injury, especially in remote areas of the posterior columns where infarction may be evidenced months after the injury (12). Release of cord compression early after spinal cord injury results in a reperfusion hyperemia response. In an experimental study, longer compression times led to less hyperemia (14).

Inflammatory cells become evident in the zone of injury within several hours. Leukocytes infiltrate the site of trauma, including systemically derived inflammatory cells and local microglial-derived macrophages (15). In one study, depletion of peripheral macrophages after spinal cord injury in rats improved functional outcome, suggesting the central role of hematogenously derived inflammatory cells (16).

Cell death and dysfunction become apparent with evidence of axonal disruption, cell fragmentation, and myelin breakdown occurring within several hours of injury. Cell ultrastructural integrity and intracellular transport are disrupted, so a

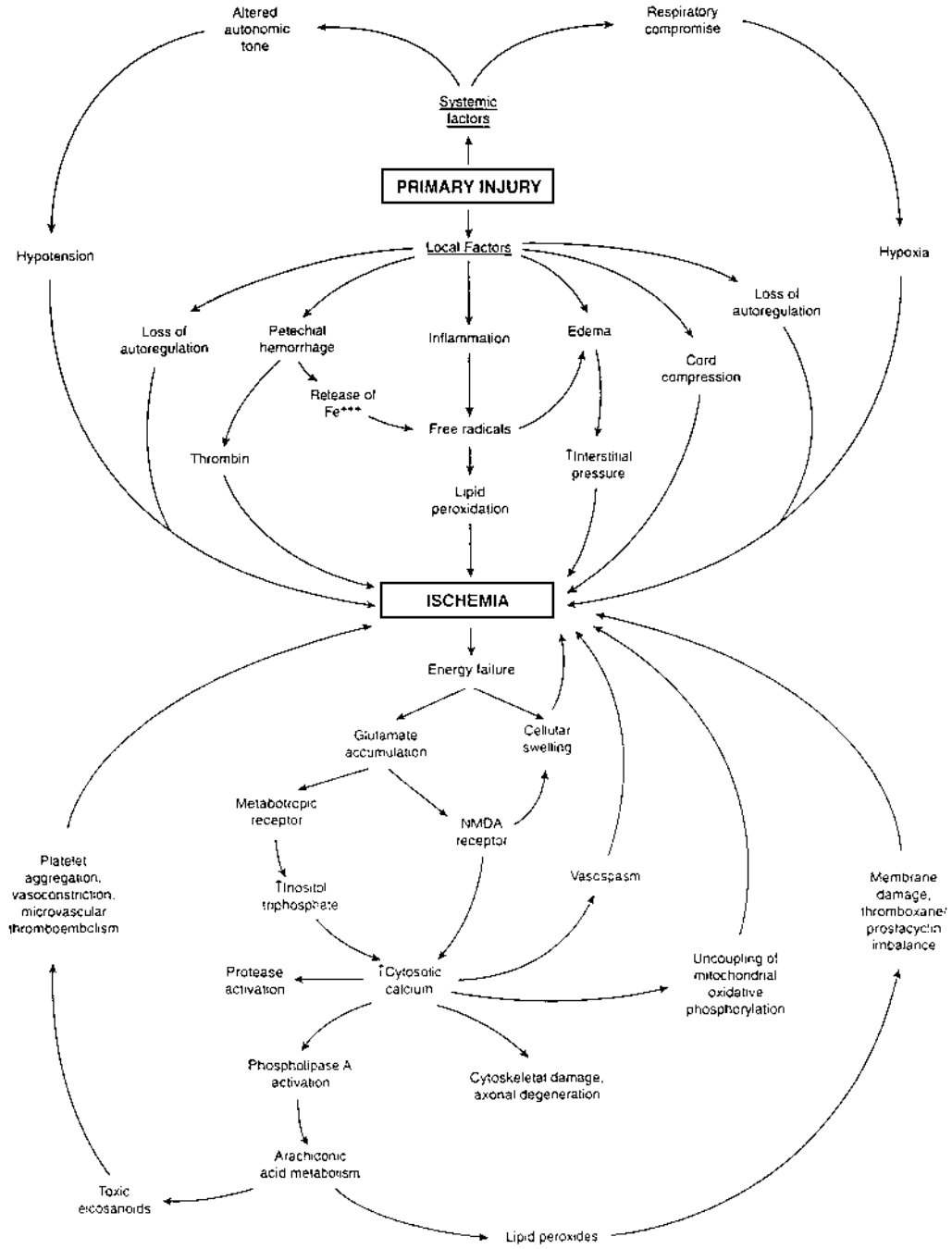

Figure 3 Schematic representation of key mechanisms, molecular species, and interrelations underlying the pathogenesis of acute SCI. Principal pathways of secondary injury that converge upon ischemia are emphasized, and others have been omitted for simplicity. These pathogenetic determinants represent the logical targets for therapeutic modulation. (Reproduced with permission from Ref. 9.)

buildup of multiple organelles is seen in axonal processes (17). Demyelination and Wallerian degeneration of long fiber tracts distant from the primary zone of injury may be observed.

These acute changes in spinal cord histopathology last from approximately 48 h to 1 week after the injury. Afterward, a decrease in interstitial edema and a resorption of cellular debris and hemorrhage occurs. As degenerated tissue is absorbed, cavitation of the cord begins centrally. In the months to years following the traumatic injury, cystic areas may coalesce to form macroscopic syrinxes filled with cerebrospinal fluid, which occur more frequently in patients with greater degrees of neurological deficit (18). Vessels occluded by clot gradually are replaced first by intimal hyperplasia and then, to a variable degree, by recanalization (19). Also, the degenerated gray and white matter are replaced with scar tissue, either from surviving astrocytes surrounding the zone of injury or from systemic fibroblast cells. The fibrous scar tissue includes the damaged spinal cord, the surrounding leptomeninges, and the contiguous vertebral periosteum. This scar tissue formation yields a sclerotic and adherent cord with obliterated subarachnoid and subdural spaces.

V MOLECULAR EVENTS FOLLOWING SPINAL CORD INJURY

Traumatic spinal cord injury induces metabolic dysregulation in neuronal, axonal, and glial tissue. Ischemia leads to energy store depletion, and ATP-dependent ion pumps fail (9). Within 5 min of severe spinal cord injury, intracellular sodium begins to increase while potassium and magnesium concentrations decrease, correlating with a decrease in axonal compound action potential transmission (a measure of axonal function). Calcium concentration and water content seem to follow the increase in intracellular sodium within 15 min of injury, leading to axonal swelling. These ionic shifts can be attenuated or arrested by sodium channel blockade with tetrodotoxin in vitro, suggesting the sodium channel as a major effector of spinal cord injury (20). Certain potassium channels also exhibit altered behavior shortly after spinal cord injury, with ''fast'' potassium channel blockade by 4-aminopyridine improving axonal compound axon potentials in vitro (21). The elevated extracellular potassium concentration may impede axonal signal conduction, leading to early neurological dysfunction and possibly being the etiology of spinal shock. The increased intracellular calcium may have myriad deleterious effects on calcium-dependent enzymatic pathways, including the uncoupling of mitochondrial oxidative phosphorylation thereby depleting energy stores, disrupting intracellular transport, degrading cell membranes, and generating free radicals (22). Furthermore, intracellular calcium concentration has been found to be directly proportional to the degree of tension experienced by the injured axon (23).

Expression of the inflammatory cytokine tumor necrosis factor-alpha (TNF-α) has been found to be upregulated in the spinal cord following acute spinal cord injury (24). Permeability to TNF-α across the blood–brain and blood–cord barrier increases after spinal cord injury. This increase in TNF-α transport occurs diffusely —even in the brain following a lumbar spinal cord injury—and remains saturable in experimental studies (25). Therefore, there is a diffuse upregulation of TNF-α transport rather than a local mechanical disruption of the blood–brain/cord barrier. A major effect of TNF-α is to activate the proinflammatory transcription factor NF-

κB, which upregulates various genes coding for cytokines, adhesins, and other inflammatory agents (26).

Inhibition of TNF-α may improve functional recovery, providing evidence that TNF-α plays a central role in the inflammatory response to spinal cord injuy. Systemic administration of the anti-inflammatory cytokine IL-10 30 min after spinal cord injury can decrease TNF-α expression and improve functional recovery of injured axons in a murine model (24). Another study showed a decrease in lesion volume following systemic IL-10 administration 3 days following simulated spinal cord injury (27). Infusion of basic fibroblast growth factor, another anti-inflammatory cytokine, diminished the zone of injury following spinal cord injury; while IL-4, ciliary neurotrophic factor, and nerve growth factor showed nonstatistical trends toward reducing the zone of injury (28).

The success of anti-inflammatory steroids in improving functional recovery following spinal cord injury yields further insight into the molecular mechanisms of neurological damage. To date, only methylprednisolone treatment has been shown to be clinically effective in large multicenter, double-blinded, randomized trials (29). Progesterone administration was also neuroprotective in an experimental study (30). The proposed mechanisms by which steroids prevent neurological deterioration include decreasing the area of ischemia in the cord (31); reducing TNF-α expression and NF-κB binding activity (32); decreasing free radical oxidation and thereby stabilizing cell and lysosomal membranes; checking calcium influx into damaged cells; and reducing cord edema (9).

Stimulation of receptor-mediated enzyme pathways such as the phospholipase A2 and phospholipase C/diacylglycerol lipase systems can lead to membrane phospholipid degradation (33). Excitatory amino acid receptors may be responsible for initiating lipase and phospholipase activity as well as facilitating calcium ion disequilibrium (34); and levels of norepinephrine, epinephrine, and dopamine are increased in the zone of injury following spinal cord injury (31). Certain glutamate receptors are also involved in mediating spinal cord injury, with evidence implicating both N-methyl D-aspartate (NMDA)-sensitive (35) and non-NMDA, kainate-sensitive glutamate receptors (36).

Free radical formation within the traumatized spinal cord may further intracellular damage. Free radicals are extremely reactive and cause lipid peroxidation, structural protein oxidation, and membrane destabilization (33). Hypoxia from disrupted microcirculation may promote free radical formation, as can calcium-mediated enzyme reactions, iron ion release from red blood cell degradation, and leukocyte-mediated cytotoxicity(22).

Another recently discovered mechanism of cell death following spinal cord injury is apoptosis in the white matter. Apoptotic glial cells may be noted over a wide distribution of vertebral levels following spinal cord injury (37). Biochemical signals from injured axons, inflammatory cells, or the surrounding vasogenic edema may lead to glial autodestruction. The loss of oligodendrocytes through apoptosis is a further mechanism for axonal demyelination, thereby contributing to neurological deterioration.

Spinal cord injury is therefore an ongoing process following acute injury. Research into intervention in this secondary cascade is yielding new potential treatments for patients with spinal cord injuries.

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3

Biomechanics of the Injured Cervical Spine

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I INTRODUCTION

Cervical spine injuries account for numerous disabilities and deaths in the United States annually (1). Most of the catastrophic cervical spine injuries result from head impacts when the head stops and the moving neck is forced to stop the moving torso. The forces generated are complex and can lead to a variety of cervical spine injury patterns.

To understand the mechanisms of injury and the methods of treatment for the various cervical spine injury patterns, knowledge of the functional anatomy and the physiological motion of the cervical spine is necessary.

II FUNCTIONAL SPINAL ANATOMY

The cervical spine consists of seven vertebrae with unique morphometry aligned normally in lordosis. The cervical vertebrae can function as a unit through complex interactions to provide support for the cranium, to protect vital neural structures, and to provide a wide range of motions (2).

The cervical spine may be divided into an upper cervical region, the occipitoatlantoaxial region (occiput-C1–C2), and a lower cervical region, the subaxial region (C3–C7).

The occipitoatlantoaxial complex serves as a transitional zone between the cranium and the spine. It is one of the most complex articulations in the body. The unique anatomy of the first and second vertebrae contribute to the versatile flexibility of the occipitoatlantoaxial complex.

The first cervical vertebra, atlas or C1, is a modified ring of bone. By virtue of embryological development, C1 has no central body, and has a thin posterior neural arch just posterior to the facet joints (3). This thin area of the ring is a frequent area for fractures, and is a result of a depression in the superior aspect of the ring that accommodates the vertebral artery as this artery passes between the ring of C1 and the occiput. The large lateral masses of C1 contain the concave elliptical superior articulating facets that accommodate the convex occipital condyles, and provide the only weight-bearing articulations between the skull and the spinal column. The stability of the occipitoatlantal motion segment is significantly reduced after 50% or more resection of a unilateral occipital condyle. At the occipital-cervical junction, significant hypermobility occurs during flexion/extension after a 25% unilateral condlectomy, during axial rotation after a 75% unilateral condlectomy, and during lateral bending after a 100% unilateral condlectomy (4). The inferior facets of the atlas and the superior facets of the axis are slightly convex; this allows significant flexibility at the expense of stability (5).

The second cervical vertebra, the axis or C2, more typically resembles the other cervical vertebrae. Posteriorly, the axis contains a bifid spinous process and anteriorly, the odontoid process or dens. The dens projects vertically from the superior aspect of the body of the axis and articulates with the posterior aspect of the anterior arch of the atlas.

The atlantoaxial articulation is heavily dependent on ligamentous interconnections, which collectively are referred to as the cruciate complex (6–10). As no intervertebral disk is present between the occiput and C1, nor between C1 and C2, in normal circumstances, stability is maintained in part by the ligaments of the cruciate complex. Anteriorly, the atlanto-occipital ligament or membrane, appearing as a continuation of the anterior longitudinal ligament, attaches to the anterior aspect of the body of C2. The anterior longitudinal ligament is stronger at C1-C2 than at any other level in the spine (11). Posteriorly, the posterior atlanto-occipital ligament connects the posterior ring of C1 to the posterior aspect of the foramen magnum.

The apical and alar ligaments contribute to the stability of the atlantoaxial and the atlanto-occipital joints (12,13). The apical ligament extends from the tip of the odontoid to the anterior lip of the foramen magnum. The alar ligaments consist of atlanto and occipital fibers that connect the dens to the occipital condyles and to the lateral masses of the atlas. The alar ligaments are primary restraints to axial rotation and to lateral bending in the occipitoatlantoaxial complex (6,7).

The transverse atlanto ligament is the primary restraint to anterior atlantoaxial subluxation. The tensile strength of the transverse ligament is greater than that of the alar ligaments (8).

In the lower cervical spine (C3–C7), the subaxial vertebrae have a similar appearance. Dorsal lateral superior projections of the vertebral body, called the uncinate processes, help prevent lateral displacement (14).

The facet joints in the lower cervical spine arise from the superior and inferior aspects of the pedicle. The facets tilt upward approximately 45 degrees from the horizontal. This characteristic inclination allows for flexion/extension and for lateral bending. Lateral bending is coupled with rotation (15). Any rotation of the lower cervical spine is accompanied by lateral bending in the same direction (16–18).

The facets can act as important stabilizing structures to absorb compression forces and to limit flexion. In normal motion, the facets may absorb approximately

20 to 30% of compression loads (19). In addition to resisting hyperflexion, the intact facets may resist shear forces. In experimental studies, a cervical facetectomy of more than 50% significantly compromised the shear strength of the cervical motion segment (20–22).

As in the occipitoatlantoaxial complex, stability in the lower cervical spine is enhanced by the circumferential attachments of the anterior longitudinal ligament, the posterior longitudinal ligament, and the interspinous ligaments.

A role of the cervical musculature is the production of physiological motion of the cervical spine. Several groups of muscles may be activated simultaneously to create coupled motion patterns. The musculature may resist excessive forces and may provide a neutralizing and stabilizing effect; however, the precise role of the musculature in maintaining spinal stability is not exactly clear (23,24).

III SPINAL STABILITY

The three-column theory of spinal anatomy, as in the thoracolumbar spine (25), helps to define factors associated with spinal stability.

The anatomical structures of the anterior column include the anterior longitudinal ligament, the anterior half of the annulus fibrosis, and the anterior half of the vertebral body. The middle column contains the posterior half of the annulus fibrosis and vertebral body, and the posterior longitudinal ligament. The posterior column consists of all structures posterior to the posterior longitudinal ligament.

The anatomical structures that contribute the most resistance to compressive forces are the vertebral centrum and the intervertebral disk. Biomechanical studies of the vertebral body have demonstrated compression failure occurring more consistently at the end plates (26).

The cervical disk may resist compressive and other pathological loads more effectively than do the vertebrae (19). In laboratory testing with forces evenly distributed, cervical vertebral endplate failure preceded disk failure (27,28). The cervical intervertebral disc is relatively resistant to direct shear loading, and it might provide the greatest resistance during horizontal translation. However, during all but direct compression, a portion of the disk is subjected to tensile forces (29).

In the middle column, compressive forces are transmitted through the posterior vertebral body wall to the uncovertebral joints. The uncovertebral joints can reduce primary and coupling motions, especially in response to axial rotation and lateral bending loads (30).

In a study to quantify the extent of injury to the distinct anatomical structures and spinal columns, it was observed that flexion instability correlated best with injury to the interspinous/supraspinous ligaments and the ligamentum flavum; extension instability correlated best with anterior longitudinal ligament and pedicle injury; axial rotation instability correlated best with anterior disk/endplate and capsular ligament injuries; lateral bending instability correlated best with posterior disk/endplate injuries; anterior column injuries correlated best with extension, axial rotation, and lateral bending instabilities; and posterior column injuries correlated best with flexion instability (31,32).

The complexity of the cervical vertebral interactions makes the formation of an unequivocally acceptable and standardized definition and classification of spinal stability difficult. The integrity of the anatomical and neural elements and the char-

acteristics of spinal motion must be carefully considered. A classification system proposed by Panjabi (33) appears most comprehensive and contains parameters that may be useful for other systems of classification. Parameters and guidelines vary from region to region within the cervical spine and at each level.

In the upper cervical spine (occipitoatlantal complex) clinical instability is suggested by axial rotation to one side greater than 8 degrees, and translation of greater than 1 mm between the basion of the occiput and the top of the dens with flexion/extension. At C1–C2, instability is suggested by a lateral overhang of C1 on C2 of greater than 7 mm total, unilateral C1–C2 axial rotation of greater than 45 degrees, C1–C2 translation of greater than 4 mm between the anterior border of the dens and the posterior border of the anterior ring of C1, and less than 13 mm of space between the posterior body of C2 and the posterior ring of C1.

In the lower cervical spine, criteria for instability include anterior and/or posterior column element disruption, abnormal disk narrowing, spinal cord damage and/or nerve root damage, a developmentally narrow spinal canal (sagittal diameter less than 13 mm or Pavlov's ratio less than 0.8), sagittal plane translation greater than 3.5 mm or 20%, and/or sagittal plane rotation greater than 20 degrees on dynamic flexion/extension lateral cervical radiographs, or sagittal plane translation greater than 3.5 mm or 20%, and sagittal plane angulation greater than 11 degrees on resting cervical lateral radiographs.

Establishing the integrity of the osseous structures is simpler, since bony fractures can be demonstrated on plain x-rays or CT scans. Anterior column failure and instability may occur when there is greater than 25% loss of vertebral body height. This degree of compression can be associated with ligamentous rupture (34). Instability may also occur with bony injury to the anterior column when at least 20% of the vertebral body is sheared off in compression, as in a teardrop-type fracture (35).

Ligamentous injury cannot always be reliably detected. The extent of injury can be inferred from radiographs by the presence of abnormal angulation, translation, and separation. The annulus fibrosis and the posterior longitudinal ligament play a crucial role in providing stability. Experimentally, it can be demonstrated that the cervical spine can resist flexion forces despite sectioning of the interspinous, supraspinous, and facet capsule attachments. Not until sectioning of the posterior longitudinal ligament did sudden angulation of at least 11 degrees or translation of at least 3.5 mm occur in the cervical motion segment. This also occurred when the ligamentous complexes were sectioned from anterior to posterior under extension forces (35).

IV DESIGNATION AND ANALYSIS OF INJURY USING FORCE VECTORS

It is difficult to clinically determine the precise force vectors that create a specific cervical injury. The primary force vector of the acute cervical spinal injury can only be inferred, since the condition of the injury mechanism is not controlled and is usually not directly observed. Soft tissue trauma to the head may be misleading, and may occur from secondary impact after spinal cord injury. Furthermore, cervical spinal cord injury has been demonstrated without craniocervical impact (36,37), and in the absence of radiographic evidence (38). For these reasons, a universally accepted classification for cervical spine fractures and dislocations does not exist.

Controlled laboratory experiments have demonstrated that isolated force vectors such as flexion, extension, vertical compression, lateral flexion, rotation, and/or a combination of these can produce isolated injuries specific to the force vector (39–42). Most likely, the injury results from multiple simultaneous forces that are resolved into a predominant vector, rather than by a single isolated injury force. Classifications of injury based on a predominant injury force vector have been established (43) and can in part be explained using a three-dimensional coordinate system with sagittal, horizontal, and frontal axes (44,45).

The column concept of the spine is valuable in understanding the pathophysiology and mechanistic action of the force vectors producing the cervical spine injuries. A predominant flexion force vector causes compression of the vertebral body and disc (anterior and middle columns) and simultaneous distraction of the posterior elements (posterior column). Conversely, a hyperextension force vector causes simultaneous distraction of the anterior column and simultaneous compression of the posterior column. This indicates a dynamic and reciprocal action that involves all the columns of the spine. Despite this, however, a single force vector, such as flexion, may cause different types of injuries that may be grouped together. It should also be reasonably noted that a direct relationship between the magnitude of a causative force and the type of injury may be present (i.e., the greater the force, the more severe the injury) (46).

V CLINICAL BIOMECHANICS OF SPECIFIC CERVICAL SPINE INJURIES

A Flexion Injuries

Flexion injuries are caused by a predominant force vector exerted axially primarily in the region of the anterior elements. Forward translation and/or rotation of the cervical vertebrae may occur in the sagittal plane. Simultaneous compression of the anterior column and distraction of the posterior column of the spine occur (47).

B Simple Wedge Compression Fractures

A hyperflexion force sufficient to cause impaction of one vertebra against an adjacent vertebra may cause this type of fracture. Minimal deformations suggest a midline, axially directed force of low magnitude (48). A compression fracture with central depression probably results from a greater force in which the intervertebral disk, as a wedge, is driven through the end plate into the vertebral body (49). During axial compression testing, 88% of the applied load passed through the disk, and maximum intradiskal pressure occurred in flexion with axial compression (50).

C Clay-Shoveler's Fractures

A clay-shoveler's fracture is an avulsion fracture of the spinous process. It occurs most commonly at C7 (51). This fracture develops when the head and upper cervical spine are forced into flexion and overcome the opposing action of the interspinous ligaments.

D Anterior Subluxation (Ligamentous Hyperflexion Strain)

Hyperflexion and simultaneous distractive forces during rapid head acceleration or deceleration can cause tensile failure of the posterior osseous-ligamentous structures. Progressive ligamentous injury can occur from posterior to anterior and can contribute up to a 30 to 50% incidence of delayed instability (52). Minor sprains may be painful but have minimal long-term consequence. Major ligamentous injuries are highly unstable; their diagnosis may be initially missed or frequently delayed, because initial radiographs are interpreted as negative. Plain radiographs may show only subtle signs of instability: local kyphosis, angulation of adjacent endplates at a single level, or interspinous widening. A supine cross-table lateral x-ray may not reveal injury, because the supine position places the neck in extension which may reduce the deformity. Dynamic flexion/extension radiographs should be avoided, as they may cause dislocation and spinal cord injury. CT with sagittal reconstruction and MRI with fat suppression techniques may be useful in identifying posterior ligamentous injuries (53).

E Teardrop Fractures

The major injury forces in the teardrop fracture appear to be acute flexion and compression with the spine in an attitude of flexion (54–58). In contradistinction, strong vertical forces applied to the neck in a straightened position create burst-type fractures. Shearing forces across the intervertebral disk and retrolisthesis of the vertebral body into the spinal canal can occur (59). Tensile failure of the posterior osseous-ligamentous structures can cause interspinous separation and fracturing of the lamina and spinous processes. The posterior longitudinal ligament usually is preserved and it may guide realignment during fracture reduction.

Stages of this type of compression-flexion injury have been proposed (54). Stage I injuries consist of blunting of the anterior superior vertebral margin of the vertebral body with no evidence of failure of the posterior ligaments. Stage II injuries show changes as seen in Stage I and, in addition, wedging of the anterior vertebral body and loss of height of the anterior centrum. A ''beak'' appearance of the anterior inferior vertebral body results. In Stage III lesions, the beak is fractured, and a fracture line passes obliquely from the anterior surface of the vertebral body through the inferior subchondral endplate. With further force, a Stage IV lesion develops having less than 3 mm of displacement of the posteroinferior margin into the spinal canal. In Stage V injuries, there is evidence of posterior ligament disruption and more significant retropulsion into the spinal canal. The higher stages represent unstable injuries and are frequently associated with spinal cord injuries. Complete spinal cord injury has been observed in 25% of Stage III injuries, 38% of Stage IV injuries, and 91% of Stage V injuries (54).

Compression-flexion teardrop injuries should be differentiated from an avulsion fracture of the anterior inferior corner of the vertebral body caused by hyperextension. The avulsion-type teardrop fracture is usually stable and should not be confused with the highly unstable flexion teardrop fracture.

F Flexion-Distraction Injuries

Flexion-distraction injuries represent a continuum of ligamentous injuries. They range from sprains or minor tears of the interspinous ligaments and the facet capsules,

to more severe, unstable injuries that involve complete disruption of the ligamentous structures and disk and result in subluxations, dislocations, and fracture/dislocations of the facets and posterior elements.

Bilateral facet subluxation results from a flexion distraction force, usually without a rotational component; it results in a sprain of the posterior cervical ligaments. There may be a partial disruption of the interspinous ligaments as well as a partial disruption of the facet capsules. Widening between the spinous processes locally may be detected on a flexion radiograph. The facet joints may be subluxated superiorly and anteriorly, and may result in slight kyphosis of less than 10 degrees on a lateral radiograph (60).

The next stage of injury is the bilateral ''perched'' facet. This injury represents a progression of the previous flexion-distraction mechanism. The inferior articular process translates superiorly and anteriorly until the inferior tip of the facet comes to rest on the top of the superior facet below. A local segmental kyphosis results, a complete rupture of the facet capsules and the interspinous ligaments occurs, and there may be partial disruption of the ligamentum flavum and posterior annulus. The force between the tips of the perched facets is usually considerable, and little motion is detected on dynamic flexion extension radiographs.

Bilateral facet dislocation injuries represent the terminal progression of the predominant hyperflexion force vector. Considerable tensile loading of the posterior elements causes significant ligamentous disruption all through the functional spinal unit. This causes the facets to ride up and be displaced superiorly and anteriorly. In approximately 30 to 50% of these cases, an associated traumatic disk herniation develops (61,62). The disk disruption creates an anterior column flexion instability that must be considered when reconstruction of the posterior tension band is performed to resist flexion. Any significant asymmetrical forces can contribute to lateral bending and to axial rotation; this creates a more dominant unilateral injury rather than a bilateral dislocation. Fractures of the posterior elements, such as laminar fractures, spinous process fractures, and facet fractures may be observed in more than 50% of cases. Vertebral artery injury and occlusion have been detected by angiography in approximately 50 to 60% of patients with facet dislocation (63). When enough force is present to create translation of greater than 50%, patients are at increased risk of having spinal cord injury and/or progression of their neurological deficits.

G Flexion-Rotation

A flexion-rotation mechanism more commonly leads to unilateral ligamentous injuries that are less common than bilateral ligamentous hyperflexion distraction-type injuries. They differ from their bilateral counterparts since a rotational force vector is present. The degree of rotational deformity increases proportionately with the amount of disruption and translation of the facet. That is, with less rotational force, subluxation results rather than a unilateral dislocation.

H Unilateral Facet Dislocations

A unilateral facet dislocation results from an exaggeration of the physiological coupling motion of the cervical spine. In normal circumstances, lateral bending is coupled with axial rotation. During injury, an exaggeration of flexion, lateral bending,

and axial rotation results in a unilateral subluxation or dislocation. The combination of simultaneous flexion-distraction and rotational forces, when most severe, can cause complete disruption of the facet capsule, attenuation of the interspinous ligaments, and partial disruption of the posterolateral corner of the disc and uncinate process. Translation is approximately 25% and, in general, is less than that observed in bilateral facet dislocations.

When significant shear and/or vertical compression force components are added to the predominant flexion-rotation injury vector, unilateral facet fractures, bilateral facet fractures, unilateral fracture dislocations, and fracture-separation of the lateral mass may occur (64).

I Bilateral Facet Fractures

Unlike the ligamentous injuries involving the cervical facets and lateral masses, facet fractures do not represent a continuum of the injury mechanisms. Fractures may occur bilaterally involving the superior articular processes, the inferior articular processes, or a combination of the two (65). These fractures develop from a mechanism of injury involving slight flexion and translation. If the shearing action is severe, disruption of the posterior longitudinal ligament and disk may occur. Bilateral inferior facet fractures occur more commonly with lesser shear forces as compared with bilateral superior facet fractures. In both types of fractures, the interspinous ligaments are stretched but not completely disrupted. Tensile loading of the posterior elements during the flexion force may lead to disruption of the ligamentum flavum, to spinous process fractures, or to laminar fractures (66). Unlike the facet dislocations resulting from ligamentous injury, the facet capsule in general is not disrupted, and the instability occurs through the bony fracture margins of the facet complex. The resulting instability patterns are in bidirectional rotation and anterior translation.

J Unilateral Facet Fractures

Unilateral facet fractures are distinct types of injuries, with slightly different mechanisms of injury. Inferior, superior, and/or lateral mass separation-type fractures may occur.

1 Superior Facet Fracture

A superior facet fracture is the most common type of facet fracture (64). The mechanism of injury is predominantly rotation in slight flexion. The capsule of the injured facet is usually intact and, as a result, may carry a displaced fragment into the neural foramen. Disruption of the posterior superior corner of the intervertebral disk may also occur. The capsule of the contralateral noninjured facet remains intact, and there may be stretching of the interspinous ligaments.

2 Inferior Facet Fracture

An inferior facet fracture is also a flexion-rotation injury. The involved facet capsule is usually disrupted, and a posterior greenstick fracture of the facet develops. The resulting instability is predominantly rotational with a minimal flexion component (64).

3 Fracture Separation of the Lateral Mass

The mechanism of injury in this type of fracture, unlike the other two types of facet fractures, is usually extension-rotation. The predominant injury force is rotational. The rotational movement can produce instability at more than one level (65). Most of the instability occurs at the level of the fracture or at the level below the fracture separation (60). A pedicle fracture, and a vertical laminar fracture with associated injury to the facet capsules develop. A true dislocation at one level or at two levels may result, and a free-floating lateral mass can be observed. Various combinations of injury patterns may develop: two-level fracture separations of the lateral mass, two-level unilateral facet fractures, or alternate-level injuries.

K Flexion-Lateral Bending

Rarely does a cervical spine injury result from a predominant lateral flexion or lateral bending injury force vector. Because of the physiological coupling motion in the cervical spine, a lateral flexion force vector is usually accompanied by a rotational force vector, and acts as a modifying injury force rather than as a primary injury force. An uncinate process fracture may be the only discrete fracture resulting from lateral flexion of the cervical spine (57). In cases where lateral displacement occurs, as in a Jefferson fracture, the lateral displacement may be attributable to a simultaneous vertical compression force.

L Hyperextension Injuries

Hyperextension injuries of the cervical spine are not uncommon (66,67). Forced hyperextension is the common mechanism of injury. The predominant injury force vector is usually a direct frontal blow to the head, or through an acceleration/deceleration mechanism.

During hyperextension, the posterior spinal elements experience compressive forces. Distractive tensile forces coupled with shearing forces cause disruption of the anterior longitudinal ligament, separation of the intervertebral disc at the vertebral end-plate junction, disruption of the posterior longitudinal ligament, fracture or dislocation of the facets, and posterior displacement of the vertebral centrum (54). When the injury develops primarily through the soft tissue structures, the overall residual deformity may have a normal or near-normal appearance (68). This may increase the chance of not detecting a serious and unstable spinal injury.

Neurological injury is not uncommon in hyperextension injuries (69). In patients with congenital spinal stenosis and/or cervical spondylosis, spinal cord compression can occur during hyperextension. During hyperextension, the area of the spinal canal is decreased, the spinal cord is shortened, and direct pressure is placed on the spinal cord by encroaching degenerative osteophytes, a posterior bulging disc, and an invaginating ligamentum flavum. Spinal cord necrosis may occur and may lead to a central cord syndrome (70).

M Atlas Fractures

When the predominant injury force vector is hyperextension, fractures of the posterior arch of C1 may occur (71). During hyperextension at the C1–C2 articulation, the anterior arch of C1 is compressed against the dens. The posterior arch of C1 is

compressed between the impinging occiput and the posterior elements of C2. The bending moment applied to the atlas increases the tensile forces through the ring, and fractures develop at the areas of least resistance—the thin bone of the bilateral groove for the vertebral artery. This mechanism of injury may also result in associated injuries such as a traumatic spondylolisthesis of the axis and an anterior teardrop fracture of C2. A hyperextension avulsion fracture of the anterior arch of C1 may occur as tensile forces are increased at the insertion site on C1 of the longus colli and the anterior longitudinal ligament (71).

When lateral bending and axial rotation force vectors simultaneously overpower the hyperextension force, asymmetrical injuries of the atlas, such as a lateral mass fracture, may develop. These combined forces may cause displacement of the lateral mass without displacement of the entire ring of C1. Other types of C1 fractures that may develop from these combined injury forces include an ipsilateral anterior and posterior arch fracture, a unilateral anterior arch fracture, a simple and/or comminuted lateral mass fracture, and a transverse process fracture (72–74) (Fig. 1).

When predominantly vertical axial compression forces are applied to the atlas, a burst fracture, the Jefferson fracture, occurs.

N Hyperextension Teardrop Fractures

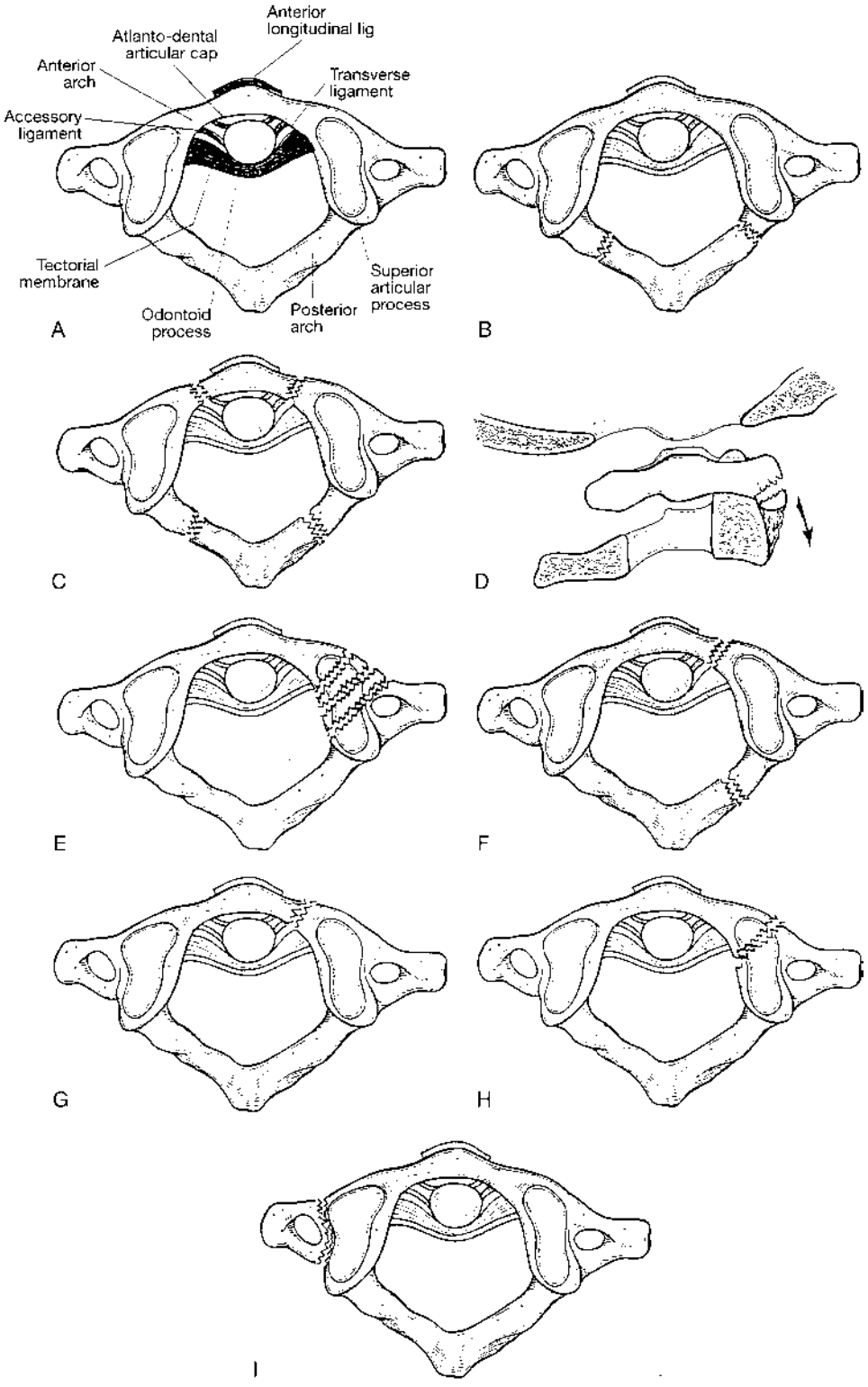
Hyperextension teardrop fractures can be confused with the more common flexion teardrop fracture. Radiographically, a small bony fragment appears displaced from the anterior vertebral body endplate. The avulsion is thought to occur through the fibrous attachments of the annulus to the end plate. Associated posterior element fractures may occur, but the midsagittal body fracture of the flexion teardrop fracture is absent (75).

O Axis Fracture (Hangman's Fracture)

The pathogenesis of traumatic spondylolisthesis of the axis, a fracture through the C2 pars interarticularis, has been studied extensively.

Experimental and clinical studies have demonstrated that the initial injurious force is predominantly hyperextension with axial loading (76). During injury, tensile forces through the anterior longitudinal ligament, the intervertebral disk, and the posterior longitudinal ligament are balanced by compression forces through the posterior elements. When the hyperextension and axial loading forces overcome this balance, shearing forces produce fractures through the C2 pars interarticularis, the weakest and most susceptible region to fatigue failure in this instance. If a significant

Figure 1 An illustration of various types of fractures of the atlas. (A) An axial projection of the atlantoaxial complex illustrating certain ligamentous and bony structures. (B) A fracture through the posterior arch, bilaterally. (C) A four-part burst fracture. (D) An avulsion fracture of the anterior inferior arch resulting from hyperextension. (E) A comminuted lateral mass fracture. (F) An ipsilateral fracture of the anterior and posterior arches. (G) An isolated unilateral anterior arch fracture. (H) A simple unilateral fracture of the lateral mass. (I) An ipsilateral transverse process fracture. (From Ref. 74a.)


Anterior longitudinal lig
Atlanto-dental articular cap
Anterior arch
Transverse ligament
Accessory ligament
Tectorial membrane
Superior articular process
Odontoid process
Posterior arch
A
B
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D
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lateral bending force component is present, an asymmetrical unilateral neural-arch fracture may develop. In the case of bilateral fractures, an uncommon oblique fracture may propagate through the vertebral body of C2 sparing injury to the ipsilateral neural arch. Angulation and translation result from flexion or distraction that follows the hyperextension force that caused the fracture. This propagating flexion-distraction mechanism, from a posterior to anterior direction, can cause disruption of the annulus fibrosis (77). The anterior longitudinal ligament may rupture from tensile forces propagated by the hyperextension force.

Various combinations of associated fracture patterns involving the facets and pedicles can occur. The most common variation is a bipedicular fracture with bilateral facet dislocation. Most likely, a flexion-distraction force produces the bilateral facet dislocation, and the hyperextension force causes the pars interarticularis fractures. A useful classification (72,73) describes the injured anatomical structures, the mechanism of injury, the sequence of injury, and provides guidance for management options (Fig. 2).

Traumatic spondylolisthesis of C2 on C3 may be caused not only by a Hangman's fracture but by other fractures, such as bilateral facet or laminar fractures. These fractures usually result from flexion-distraction or shear forces, and may be associated with ligamentous injuries as well as other fractures.

P Whiplash

Whiplash injuries, or acceleration-deceleration injuries of the cervical spine, represent a syndrome of various clinical circumstances and abnormalities that may not be obvious at initial or follow-up examinations. Classically, the clinical history is of a rear-end vehicular accident.

The exact mechanism of whiplash injury is not clear and numerous factors influence the extent of injury (78). At impact, it appears that the head moves first into flexion and then, within 0.2 s, into extension (79). However, this initial pattern of head motion during injury is debated (80). During impact, the cervical spine and the remainder of the body are accelerated forward while the head remains behind, held in its resting position by its own inertia (81). The sudden forward pull by the trunk creates shear forces through the neck and the lower cranium ''whipping'' the head forcefully backward and causing extension of the cervical spine. Likewise, with sudden deceleration of the body, the head moves into a forward or anterior position and then recoils into extension (82).

Motion analysis from simulated testing of the cervical vertebrae during whiplash reveals distinct patterns of vertebral motion during impact. C6 rotates into extension and the upper cervical spine moves into initial flexion. Maximum rotation of C6 induces C5 to extend. With the upper cervical motion segments in flexion and the lower segments in extension, the cervical spine assumes an S-shaped posture, and C5–C6 exhibits an open-book motion with an upward-shifted instantaneous axis of rotation (83).

Injuries to capsular and spinal ligaments may be part of the mechanism contributing to the whiplash symptom complex. During whiplash simulation testing, peak strains of the capsular ligaments occurred at the C6–C7 level (84).

Motion of the head and the loads causing whiplash can be affected by preventive safety factors. An appropriately constructed and positioned headrest at the levels

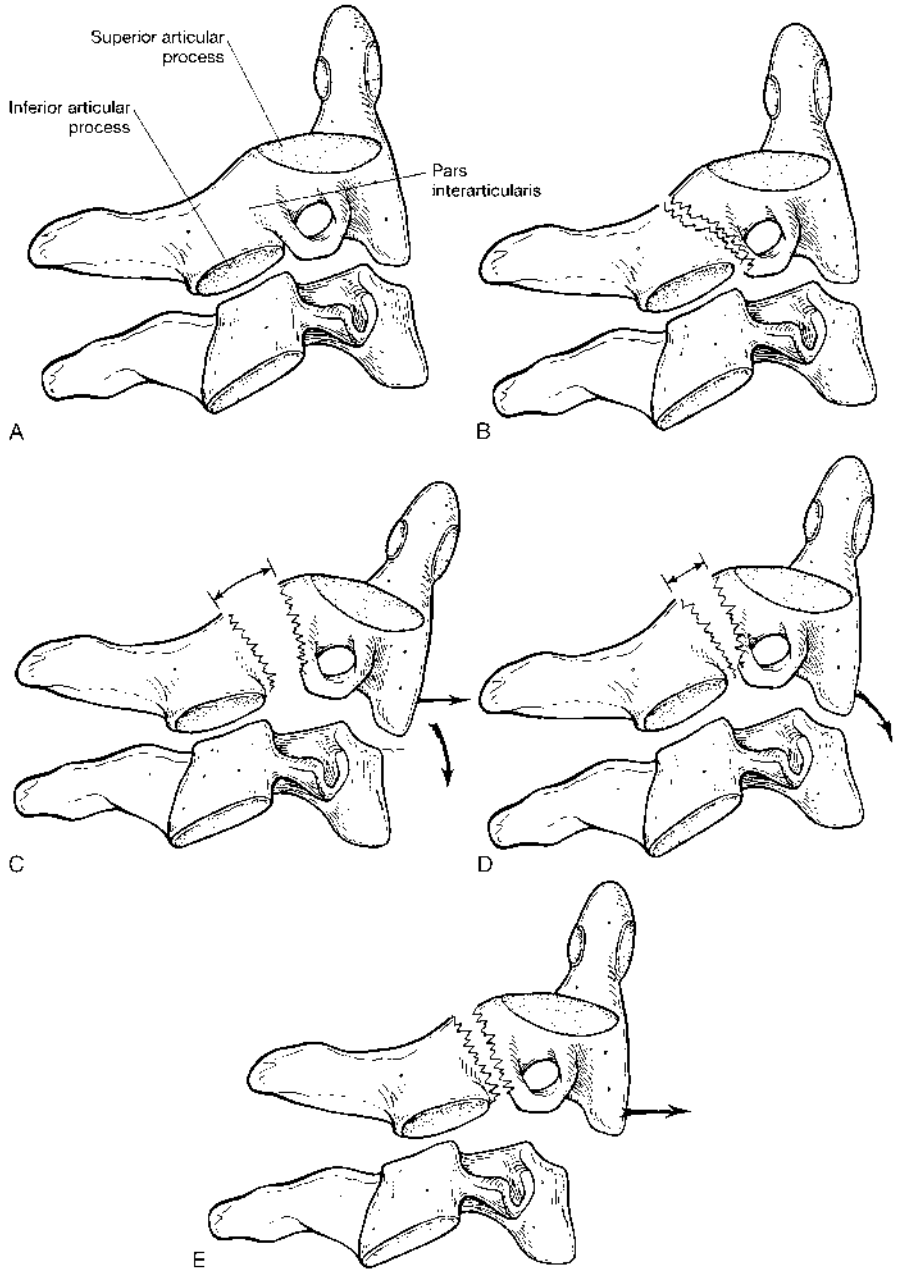

Figure 2 An illustration of different types of atlas fractures that may lead to traumatic spondylolisthesis. (A) Normal anatomical relationships of the atlas to C3. (B) A fracture through the pars interarticularis, bilaterally, without angulation and/or anterior translation. (C) A pars interarticularis fracture with anterior displacement and significant angulation. (D) A pars interarticularis fracture with significant angulation without anterior translation. (E) A pars interarticularis fracture with anterior translation leading to a unilateral or bilateral facet dislocation of C2 on C3. (From Ref. 72.)

of the ears can limit extension. A seat-belt shoulder strap can restrain and decrease the acceleration forces of the torso, thereby decreasing the inertial forces on the cervical spine. A stiffer car seat can also decrease the acceleration of the torso; this minimizes the shear forces and the bending stresses in the neck (82).

An airbag restraint system may reduce fatalities in frontal vehicular crashes. Few data exist to document specific fracture patterns after airbag deployment. In a small study (85), it was found that non-seat-belted drivers demonstrated flexion injuries of the cervical and thoracic spine and direct impaction fractures to the face and sternum. One lap-shoulder-belted driver demonstrated an extension injury of the upper cervical spine.

Q Odontoid Fractures

High-energy trauma and motor vehicle accidents are the most common causes of adult odontoid fractures (86,87). The odontoid fracture might not be diagnosed during the initial emergency room evaluation because of diverting factors such as facial trauma, altered mental status, head injury, and other associated injuries of high-energy trauma. Low-velocity falls may more commonly cause odontoid fractures in children and the elderly. In the elderly, myelopathic symptoms may be caused by an occult odontoid fracture (88).

Concomitant cervical spine injuries, such as a Jefferson fracture, may occur in approximately 18% of patients with odontoid fractures (89).

Various mechanisms have been suggested for odontoid fractures, such as hyperflexion, hyperextension, axial compression, rotation, shear, and lateral bending (90–92). From experimental studies, it appears that the predominant mechanism is a combination of axial compression and horizontal shear (93).

Anterior displacement of the odontoid occurs more frequently than posterior displacement, except in the elderly. When a hyperflexion force is present, an intact transverse ligament may be involved in translating the odontoid anteriorly; when hyperextension is present, the anterior ring of the atlas may displace the odontoid posteriorly (8).

When rotational forces are applied, an avulsion fracture of the odontoid tip may occur. This type of odontoid fracture represents an avulsion injury to the alar ligaments and cranial cervical ligamentous complex rather than a direct injury to the tip of the odontoid (94). An oblique odontoid tip avulsion fracture is a relatively uncommon odontoid injury, and it most likely results from injury to the alar ligament rather than to the apical ligament. This fracture can be confused with the rare ossiculum terminale, the secondary center of ossification.

The most common odontoid fracture develops at the waist of the odontoid just superior to the body of the axis. This fracture has the highest rate of nonunion for odontoid fractures. The high rate of nonunion and poor prognosis may be related to the age of the patient, to a delay in diagnosis (greater than 7 days), to the direction of displacement (posterior greater than anterior), to the degree of translation (greater than 2 mm), and to the degree of comminution injuring the extraosseous and intraosseous anastomotic vasculature (95,96).

Another type of odontoid fracture is one that propagates through the body of C2. Contrasted with the waist-type dens fracture, a larger fracture surface area of bleeding cancellous bone with this type of fracture accounts for the relatively higher rates of union (97).

When an odontoid fracture develops, strong simultaneous additional forces to vertical compression and horizontal shear may lead to associated injuries such as fractures of the ring of C1, traumatic spondylolisthesis of C2, and traumatic atlantoaxial (C1–C2) subluxations and dislocations.

R Atlantoaxial Subluxations and Dislocations

The mechanism of these types of injuries is related to any pathological process that compromises the C1–C2 ligamentous structures and causes an abnormal relationship between the ring of C1 and the ring of C2. The main causes are trauma or inflammation.

In traumatic cases, either anterior or posterior abnormal displacement of C1 in relation to C2 may occur, with or without an associated rotary subluxation/dislocation of C1 on C2. The type of injury is related in part to the predominant injury force vector.

For an anterior dislocation of C1 from C2, the predominant injury force vector is hyperflexion. In this case, the force pattern is similar to that of an odontoid fracture. If the force is severe enough, continuation of the injury leads to the displacement of the ring of C1. With hyperextension, a posterior displacement of C1 in relation to C2 occurs. In this case as well, a posteriorly displaced dens fracture may occur, as the progressive horizontal compression of the anterior ring of C1 shears and breaks the dens. In two cases, a posterior dislocation of C1 on C2 occurred in which the dens was not fractured and the ring of C1 was lifted over the dens (98,99). In these instances, a strong vertical distractive force combined with hyperextension contributed to the injury.

Rotary subluxation, which may lead to a dislocation or to a fixed rotational deformity, occurs when the predominant injury force vector is at an angle to the sagittal plane and is sufficiently off-center enough to develop a torque on C1 (100).

The transverse ligament acts as the primary restraint to anterior atlantoaxial translation. The alar ligaments act as the primary restraint to excessive rotation of the atlas in relation to the axis, and they act as secondary restraints to anterior displacement of the atlas. When the transverse ligament is incompetent, the alar ligaments cannot fully prevent further displacement of the atlas when the injury force is sustained (8).

Continued rotatory subluxation may occur when the secondary restraints to rotation (the tectorial membrane, the accessory atlantoaxial ligaments, and the facet capsules) become compromised (101). A rotary dislocation of C1 from C2 may develop as rotation progresses to approximately 63 to 65 degrees. If the transverse ligament is disrupted and anterior translation of C1 exceeds 5 mm, rotatory dislocation of C1 on C2 may develop as rotation progresses to approximately 45 degrees (102).

Lateral tilt of the head may occur when a lateral flexion force combined with rotation exceeds the physiological coupling movement of C1 on C2. During lateral flexion, the opposite alar ligament tightens. On the side of flexion, the concave inferior facet shifts inferiorly and posteriorly as the contralateral superior facet shifts anteriorly and superiorly. With further lateral bending, rotation of the dens tightens the opposite alar ligament, and motion is inhibited. In this rotated, flexed position, the alar ligaments are most taut and are at greatest risk for rupture (103). Radio-

graphically, this may be demonstrated on open-mouth views that indicate an overlap of the lateral mass of C1 in relation to the superior articular facet of C2. Abnormal anterior displacement of the posterior wall of the anterior ring of C1 from the anterior surface of the dens may be observed on the lateral x-ray view (104).

In nontraumatic cases, chronic subluxation may lead to a fixed atlantoaxial rotational deformity. The pathophysiology for this condition is not well defined. Various hypotheses have been proposed concerning the dynamic effects on the soft tissue structures of the neck that are created from pathological inflammatory changes (105,106). Spasm and inflammation of the sternocleidomastoid muscle may lead to contracture of that muscle and to a gradual restrained posture of the neck. Effusion and synovitis of the C1–C2 facet joints and capsules may lead to attenuation and excessive laxity of the capsules. As rotation and stretching of the capsules and ligamentous structures progresses, invaginated inflamed synovial folds may prevent the reduction of the displaced facet joints. If reduction is not obtained and deformity persists, secondary contractures may develop that lead to fixation of the atlantoaxial rotational deformity.

S Compression Burst Fracture

A burst fracture of the ring of C1 results from forceful axial compression, usually a direct blow to the head (107). The caudally driven occipital condyles act like a wedge to compress the ring of C1 between the condyles and the ring of C2. Although classically described as a four-part Jefferson fracture, a two-part or three-part fracture occurs more frequently (91,108) (Fig. 1). On open-mouth radiographs, splaying and bilateral lateral displacement of the lateral masses of the ring of C1 in relation to C2 may be observed. A total of $\geq$7 mm of displacement indicates instability.

When axially loaded, the straightened cervical spine acts as a segmented column (109,110). When the neck is partially flexed, the cervical spine is in fact straightened. In the straightened cervical spine, the axial load is transmitted with greater force from the head to the thoracic spine compared to when the cervical spine is in the normal lordotic posture (111,112). When the cervical spine is not straight, the load-carrying capacity is reduced and the segmental spinal column may buckle (113,114). Compression of the vertebral body may cause varying degrees of retropulsion of the posterior vertebral wall into the spinal canal (115). Positioning a cervical burst fracture into either extension or compression can significantly increase canal occlusion as compared with canal occlusion in the neutral position (116).

T Occipital Condyle Fractures

Fractures of the occipital condyle are rare. They may be associated with other fractures of the spine (117).

Occipital condyle fractures may be classified as impacted fractures, fractures associated with a basilar skull fracture, or an avulsion fracture (82). Impacted fractures and fractures associated with a basilar skull fracture are usually the result of a forceful axial compression force. This force coupled with a rotation or shear can lead to an avulsion fracture of the occipital condyle (118). In the avulsion-type fracture, the restraint and stabilizing effect of the alar ligament is compromised, and significant instability may occur.

U Occipitoatlantal Dislocations

The exact mechanism of this often fatal injury is not definitive. The injury might not be recognized unless specifically looked for. From postmortem examinations, it has been suggested that the primary injury force vector is hyperflexion (119); others suggest hyperextension (120,121). In either case, there is displacement and translation of the occipital condyles away from the C1 articular surfaces, as the stout stabilizing craniocervical ligaments and articular capsule are ruptured.

V Spinal Cord Injury Without Radiographic Abnormality (SCIWORA)

In a child with a spinal cord injury and the absence of any abnormal findings on plain radiographs, a hyperextension injury to the cervical spine should be suspected. Several large studies have provided insight into this interesting spinal injury (38,122). It appears that the unique anatomical and biomechanical characteristics of the skeletally immature spine account for the normal radiographic appearance of the cervical spine despite a high-energy injury with neurological impairment. Failure was found to occur through the weak physeal or the cartilaginous endplate (123). Hyperextension appeared to be the predominant mechanism of injury. A high index of suspicion is recommended, since lack of recognition most often leads to an error in diagnosis of this injury (124).

VI CONCLUSION

The response of the cervical spine to impact loading during injury is complex. Various cervical injury patterns can be produced from specific force vectors. An understanding of the mechanisms of injury and the pathological forces acting on the cervical spine can guide the treating physician to an accurate diagnosis and effective treatment of these injuries.

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4

Emergency Management of Spine Trauma

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Spinal trauma and its cost in health-care dollars continue to increase, especially in developed countries where high-speed transportation and high-speed recreation are on the increase. In first-, second-, and third-world countries, violence also contributes heavily to the spine trauma population. Trauma remains a disease that represents a leading cause of death in the first four decades of life. Trauma also permanently disables many members of society at an estimated cost of 100 billion dollars annually. Emergency treatment of spine and multiple traumas represents the first opportunity to successfully impact ultimate outcomes of the injury.

In a large percentage of severe high-energy trauma cases, death occurs instantly. The emergency medical transport system in the United States and similarly developed countries allows an increased rate of survivability. However, in the best of circumstances, 30% succumb in the earlier, initial phases of care and an additional 20% succumb to later problems in the initial weeks and months following the trauma. Most often, these causes of death relate to infection or multiple organ failure, including the chronic manifestations of head trauma, hemorrhagic shock and spinal cord injury (1).

Isolated spine trauma most frequently results from sports-related injuries, falls, and some low-velocity gunshot wounds (2). This represents approximately half of the cases seen in the major spinal cord injury centers. Multisystem involvement must be addressed early in the treatment of these low-energy injuries, but early attention to the spine is usually possible without risk of hemorrhage, head trauma, or other injury that often delays spine surgery in high-energy injury (3–5).

The trauma associated with a spine injury in the remaining group is most frequently associated with high-energy vehicular trauma, falls from heights, and some

violence. The breakdown of associated trauma with spinal cord injury includes fractures of the trunk (18%); long bone fractures (14%); head and facial trauma (14%); and chest and abdominal injury (18%) (6,7).

I INITIAL MANAGEMENT

Initial management of the multiple trauma or isolated spinal trauma patient is done by emergency medical technicians, doctors in the field, paraprofessionals in the field, and even ''good samaritan'' citizens trained in CPR. This initial care is often instrumental in allowing the patient to survive with a minimum of injury and with some reduction in the likelihood of complete paralysis. Enhancement of recovery begins with this care (1,8). The actual delivery of field care from emergency medical technicians and paramedics can often be instrumental in survivability. The Emergency Medical Services Act of 1973 established guidelines for this care. Since the advent of that system, steady improvement in emergency services has been noted in the United States and other developed countries.

Toscano (1) cited an incidence of severe complications associated with transfer, but in the past 10 years significant improvement from the sited 26% neurological complication rate has been noted. During patient transport, the emphasis has been placed on the ABCs of life support together with splinting of all fractures, establishment of intravenous lines, and administration of initial fluid done by the field technicians, sometimes under the guidance of emergency room physicians. Universal teaching on splinting the spine with bed board, orthotic devices, and some traction apparatus have permitted early immobilization or reduction before the patient reaches the hospital. Attending sports physicians are well versed in contact sports injuries, including support of the spine, logrolling, and immediate bracing of the spine (9). Some traction devices are available to some emergency technicians. Inboard traction devices are also available to immobilize cervical spine fractures during transportation. All uncontrolled motion of the spine must be avoided to preserve as much neurological function as possible. It has been emphasized that any unconscious or semiconscious patient must be assumed to have a spinal cord injury until proven otherwise.

Assessment of the airway and cardiovascular status is also done in the field. Field emergency personnel initialize protection of vital functions, maintaining airway, supporting and assisting respiratory efforts, and assessing most phases of bodily function. In rural areas, fix-winged airplane and helicopter transportation are also available so that transportation to a major trauma center can take place.

Nasal tracheal, oral tracheal, and even sometimes surgical tracheal intubation are required, especially in maxillofacial trauma. Patients are often in severe distress and clearing the airway to establish gas exchange is critical. Attention to the hemodynamic system is also essential. Establishing of intravenous lines and fluid flow in the field is often instrumental in preventing or providing early treatment of shock, as well as protecting the spinal cord itself from hypovolemia.

A physician or health paraprofessional often supervises athletic events in the United States. Contact sports, in particular, have a relatively high incidence of potential for spinal trauma. Athletic competition becomes secondary when there is potential for spinal cord injury. Temporary loss of consciousness, temporary neurological extremity injury, or any suggestion that the central nervous system or spinal

cord has been damaged necessitates immediate cessation of the activity, transportation for evaluation, and communication between the health-care professional and the coach, parents, and injured athlete.

II EMERGENCY TREATMENT

Once the patient is transported to the emergency room, the emergency physician and trauma team are called upon to triage as well as evaluate and begin treatment of all injuries (10–12). Life-threatening injuries are treated first. Intoxication can complicate the initial evaluation. Establishing of basic life support methods includes evaluation of the patient for the presence of coma. The Glasgow Coma Scale (Table 1) is frequently utilized to assess neurological status. In the conscious patient, full cooperation for a neurological exam is facilitated. In the presence of severe maxillofacial or other trauma (as noted on the Glasgow Coma Scale) spinal cord evaluation is made much more difficult. The oral tracheal and endotracheal system can be injured in high-energy trauma with maxillofacial fractures, blood in the nares, and, occasionally, even tracheal injury. Establishing an airway, even if it requires surgical tracheotomy, is essential to survival.

Large lines may need to be established to support the cardiovascular system. Hemodynamic stability is essential to avoid shock and multiorgan failure, and to preserve spinal cord function when the cord is injured.

Basic trauma treatment frequently involves the attempt to triage secondary injuries but provide some emergency treatment (13). Obviously, spinal deformity and spinal support can be established while life-threatening injuries are treated. The early phases of management require obvious efforts to attempt to diagnose any spinal cord injury.

Table 1 Glasgow Coma Scale

Eye opening
Spontaneous
To voice
To pain
None
Verbal response
Oriented
Confused
Inapposite
Incomprehensible
None
Motor response
Obeys commands
Localizes pain
Withdrawal (pain)
Flexion (pain)
Extension (pain)
None

Early treatment of spinal cord injury has been enhanced over the years by administration of methylprednisolone. Investigators for the Third National Acute Spinal Cord Injury Study concluded that methylprednisolone improves neurological recovery after the first 24 to 48 h. Tirilazad mesylate can be used for 48 h after acute spinal cord injury. Bracken and coworkers have clearly defined the benefits in this national research, randomized, double-blind group (14–16). The National Acute Spinal Cord Injury Study Group documented with 16 acute spinal cord injury centers in North America that the effect of the 48-h methylprednisolone regimen was significant at 6 weeks and 6 months. Patients who received the 48-h regimen and who started treatment at 3 to 8 h were more likely to improve one full neurological grade. The 48-h group was also reported to experience greater sepsis and severe pneumonia than patients in the 24-h methylprednisolone and tirilazad groups, but other complications were similar.

Tirilazad is a lazeroid in the drug class of lipid peroxidase inhibitors. In addition, it exhibits neuroprotective effects by a variety of other mechanisms, such as improving spinal cord blood flow and membrane stabilization. Because lazeroids have none of the glucocorticoid properties of methylprednisolone, tirilazad may have fewer side effects (12). Except for gunshot trauma to the spine, the corticosteroid protocol, when implemented immediately, is very effective at reducing cord swelling and establishing the optimum environment for recovery. The dosage profile described in the study involves a loading intravenous dose of 30 mg/kg of methylprednisolone for 15 to 30 min, followed by 5.4 mg/kg/h for 23 h. The incidence of stress ulceration and other systemic complications exists, but there is definitive, positive, documented effect on recovery. This treatment must be administered and is considered standard of care. It cannot be used in children under the age of 13, during pregnancy, or in patients with diabetes or infection.

Table 2 describes several common multiple trauma problems that require triage through the interaction of the general surgeon and the emergency physician. Intra-

Table 2 Life-Threatening Complications of Common Injuries

Injury complication contributing factor
Pneumothorax
Tension pneumothorax
Positive pressure ventilation
Blunt chest trauma
Arrhythmia
Pericardial tamponade
Myocardial contusion
Atrial rupture
Tracheal
Airway obstruction
Hemorrhage, edema
Subcapsular hematoma (spleen, liver)
Massive hemorrhage
Delay in diagnosis

abdominal and intrathoracic hemorrhage, as well as extremity hemorrhage, must be immediately controlled. Large volumes of fluid plus blood replacement and use of plasma expanders are all modalities to reduce the period of time the patient has severe hypoperfusion. The general surgeon must be able to recognize, diagnose, and treat severe liver lacerations, the effects of blunt chest trauma causing pericardial tamponade, and spleen rupture or avulsion of intestines, all of which represent severe, potentially life-threatening disorders. Emergency abdominal surgery may be required while temporizing measures are instituted for the spine trauma. Once life-threatening chest and abdominal injuries are treated or ruled out, attention can be given to the spine.

In the polytrauma patient with a spinal cord injury, there is an additional diagnostic dilemma. Hypotension can be a direct result of hypovolemia. However, hypotension can also be due to neurogenic problems associated with loss of vascular tone and increased vascular capacitance if bradycardia is present in these patients with neurogenic shock (17).

Soderstrom documented systemic blood pressure less than 100 in 69% of patients reviewed with cervical spine injury (3). Obvious recognition of this problem, as well as ruling out potential intra-abdominal trauma, is essential. The treatment for neurogenic shock includes limited volume replacement with use of vasopressors; overinfusion can result in fatal pulmonary edema.

Vasopressors in the face of hypovolemic hypotension or cardiogenic causes can present the physician with life-threatening complications. If intra-abdominal injuries and long bone fractures are ruled out, treatment of neurogenic shock should be instituted if cord injury is present. Table 3 delineates a reasonable protocol for the essentials of initial treatment of a multitrauma patient, whether it is from gun violence or high-speed, high-energy trauma.

III SPINAL EVALUATION

Once life-threatening injuries have been treated, hemodynamic status is established, and after clear documentation of cerebral function, assessment of the spine and spinal cord must be made. Initially, evaluation of the spine must include radiographs of the entire spine. Numerous studies have clearly documented that missile injuries do not destabilize the spine, except in extremely rare occurrences (2). However, fractures can be missed in high-energy trauma because reduction may have taken place spontaneously or can have been achieved by the emergency technicians. Any pretracheal edema on cervical spine films is an obvious clue. Slight angulations or slight facet incongruity, as well as small anterior vertebral fractures or spinous process fractures can also be clues.

In the thoracic spine, it is critical not to accept AP and lateral chest x-rays as being diagnostic of spine stability. Thoracic spine instability can be suspected in multiple rib fractures, first rib fractures, or any trauma where a direct blow is evidenced in the posterior upper thoracic spine. Otherwise, the thoracic spine is usually stable. In the thoracolumbar spine and lumbar spine, fractures must be ruled out in the presence of pelvic instability or in the presence of significant transverse process fractures. Pedicle widening on the AP film is also considered a hallmark of spinal fractures. The literature is replete with information on late diagnoses of peripheral fractures. It is also evident that multiple traumas have an association with multiple

Table 3 Basic Trauma Treatment

1. Contact and consult with field team.
2. Prepare emergency area for victim.
3. Establish airway and begin respiratory support, if needed.
4. Establish fluid replacement to restore circulation; treat shock.
5. Evaluate and reevaluate physical status:
   - neurological
   - chest
   - abdomen
   - extremities
6. Foley catheter for drainage and monitoring.
7. Chest tube (prn):
   - abdominal tap (prn)
   - nasogastric tube
8. Evaluate:
   - CNS
   - spine
   - long bones
9. Spinal protection:
   - skeletal traction
   - neck bracing
10. Methylprednisolone for spinal cord injury.
11. In coma, everything is injured or fractured until proved otherwise.
12. Plan appropriate treatment for spine injury.

noncontiguous spinal fractures (18–20). Finding one spinal fracture does not assure the physician that no other fractures exist. Up to 15% of patients have noncontiguous spinal fractures. In the event of some plain radiographic suggestion of spinal instability, CT and MRI scanning may be employed early on to further delineate the problem. Emergency information from diagnostic MRI and CT scanning has proven valuable, especially when ligamentous or disk injury is present and accompanies instability. Emery eloquently described a black stripe sign indicative of posterior torn ligaments (20). Others have also described the value of early CT and MRI imaging. Reference 21 provides useful information to assist the physician in interpretation of subtle signs of instability.

Table 4 Acute Management of Spinal Cord Injury

1. Maintenance of perfusion systolic blood pressure >90 mmHg.
2. 100% O_2 saturation via nasal cannula.
3. Early diagnosis by plain radiography.
4. Methylprednisolone therapy (loading dose 30 mg/kg followed by infusion at rate of 5.4 mg/kg/h for 23 to 48 h).
5. Immediate traction reduction for cervical fracture and dislocation.
6. Spinal imaging (MR imaging and/or computed tomography).
7. Surgery, if indicated, for residual cord compression or fracture instability.

Source: Ref. 12.

One of the other absolute essentials in dealing with emergency management of the spinal cord and spinal trauma patient is to thoroughly evaluate and reevaluate neurological status. Unfortunately, the presence of head trauma and intoxication can initially delay or distort neurological evaluation and makes the evaluation extremely difficult.

IV LEVEL OF CONSCIOUSNESS

Making certain the patient has an acceptable level of consciousness and documenting neurological function are essential. Serial evaluations of functional physical activity and the patient's neuromotor skills are assessed. The ASIA motor index form is a detailed evaluation of motor function. The Frankel scale is a generic scale addressing function, but is nonspecific with respect to the gradation and exact level of function.

Focal tenderness that is elicited from the patient creates an index of suspicion of a ligamentous injury. Transitory sensory deficit that is not present at the time of the examination (Lhermitte's sign) also creates a high index of suspicion with regard to the possibility of spinal instability. The diagnoses of any motor or sensory loss are also heavily relied upon to determine the presence of spinal instability. If a totally normal neurological evaluation is present, there is much more justification for motion studies to determine the nature and extent of any potential instability. The presence of a neurological deficit mandates CT or MRI scanning to fully assess the spine. In larger individuals, when C7–T1 is not visualized on the lateral view, a CT scan of that region can be important.

Radiographic hints regarding the presence of instability include (1) abnormal relationship between the anterior margins of the posterior bony structures in the foramen magnum and the anterior margin of the spinous process of C1 and C2; (2) prevertebral edema in the cervical spine; (3) widening of spinous process relationships when compared to other segments; and (4) pedicular widening on the AP in the lumbar spine. All of these signs and others are described in Reference 21. The extent of evaluation accuracy when a patient has a depressed level of consciousness (Glasgow Coma score of less than 10) is difficult for the practitioner. Many major skeletal injuries are overlooked because of decreased pain response and because the brain injury takes immediate precedence. In a large series, Mackersie et al. (4) described a 14% incidence of axial spine fractures in the setting of head trauma. Obviously, in any obtunded or unconscious patient, complete study of the spine is imperative.

Accurate assessment of the extent of spinal trauma and instability is imperative. There can be a determination of the need for treatment based upon the extent of the spine trauma. The American Spine Injury Assessment (ASIA) impairment scale (Table 5) can serve as a guide for appropriate treatment and for expected outcomes. Initial evaluation must include an assessment of sensorimotor function and bowel and bladder innervation. Performance of a rectal exam is essential if any question exists regarding neurological loss. Obvious sensory deficits or subtle loss of sharp–dull discrimination, temperature, and light touch are key. Manual muscle testing is graded by a standard system: Grade 0—no function; Grade 1—trace or palpable contraction without the ability to move the joint; Grade 2—poor joint range of motion with gravity eliminated; Grade 3—fair joint motion against gravity; Grade 4—some ability to resist gravity and some manual resistance; Grade 5—normal range of motion against significant resistance. This represents one of the key factors

Table 5 ASIA Impairment Scale

A = Complete: No motor or sensory function is preserved in the sacral segments S4–S5.
B = Incomplete: Sensory but not motor function is preserved below the neurological level and extends through the sacral segments S4–S5.
C = Incomplete: Motor function is preserved below the neurological level, and the majority of key muscles below the neurological level have a muscle grade less than 3.
D = Incomplete: Motor function is preserved below the neurological level, and the majority of key muscles below the neurological level have a muscle grade greater than or equal to 3.
E = Normal: Motor and sensory function is normal.

in the ASIA motor index rating system, but also complies with grading systems in guidelines described for physical examination.

If a neurological disorder exists, establishing the lowest level, where complete function, both motor and sensory, exists is important. Damage to specific areas of the spinal cord creates specific syndromes. The anterior cord involves damage to the anterior two-thirds of the cord and is heralded by complete loss of motor and sharp–dull sensation. The posterior columns are spared, which allows crude sensation. This syndrome offers a poor prognosis. Central cord syndromes are heralded by damage and dysfunction primarily in the hand, with little loss in the lower extremities, although a spastic gait often is retained. Fine motor use of the upper extremities is often the last to recover. Brown Sequard–type syndromes, which are rare in high trauma but common in missile or stab injuries, are characterized by the loss of ipsilateral motor function and contralateral pain. Posterior cord syndromes are rare and associated with direct trauma; they involve loss of proprioception and position sense. Isolated root injuries can also occur from unilateral facet translation, dislocation, or traumatic instability with disk injury. Once a neurological evaluation has been established, maintenance and reevaluation to diagnose either improvement or decline is essential. Multiple serial neurological examinations by a standardized approach are essential to provide feedback for the patient. Loss of neurological function occurs in unrecognized instability, cord edema, and ascending hematoma. These conditions must be treated immediately or they can preclude recovery. No clear evidence exists to establish that emergency surgery is beneficial beyond 8 h from the time of trauma. In most university settings, early decompression is accepted, but has not been documented to provide superior functional outcome.

In emergency treatment of spinal trauma, if any suspicion of spinal cord trauma exists it is imperative to implement the methylprednisolone regimen. When it is clear that spine stability has been established, secondary care to long bone fractures and conservative care to non-destabilizing spine fractures can be given. In any circumstance, it cannot be overemphasized that serial neurological evaluations must be performed in the first 24 to 48 h in any polytrauma patient. If discharge from the emergency setting is contemplated, several factors must be considered. First and foremost, absolutely no neurological symptomatology has existed at any time during the trauma. Second, the entire spine has been radiographed and evaluated completely and successfully. Third, other than musculoskeletal pain, no significant or severe symptomatology exists. Fourth, no other system injury can be present. Fifth and

final, the patient must be able to interact completely on his own, without having had any periodic or transitory functional loss.

V ANESTHESIA IN MULTISYSTEM TRAUMA

If spinal trauma is present together with life-threatening cardiovascular, abdominal, or cranial injuries, the spinal surgeon must be involved in the surgical team. This must be considered a part of the emergency care in spine trauma, as nonspinal surgery is often required prior to complete spine evaluation.

Spinal personnel must assure safe transport to and from the operating room, along with maintenance of spinal alignment during the procedure. Most trauma centers deal with multiple trauma and spine trauma, and multiple personnel are often available. However, in hospitals where the problem is less frequently seen, the orthopedic or spinal surgeon must be prepared for hands-on management.

Reestablishing reasonable blood pressure, blood flow, and oxygenization for cord benefit, realignment of the spine, and maintenance of alignment are all imperative to assure good outcomes and are the responsibility of the spinal surgeon. The anesthiologist must also be aware that cervical, thoracic, and lumbar trauma may accompany maxillofacial trauma, head trauma, and multisystem injury. The spinal surgeon must also provide information during the resuscitation period regarding avoidance of overdose of glucose in trauma as described in Refence 22.

VI REDUCTION AND ALIGNMENT OF THE SPINE IN THE EMERGENCY SETTING

Many cases of multiple traumas are present with spinal fractures. Realignment or establishment of external stabilization can be performed in the emergency room or in the first 12 to 24 h. The procedures can be accomplished with minimal sedation and analgesia so that continuous neurological evaluation can take place.

Failure to maintain patient contact can lead to serious complications. Tongs and traction or halo traction can result in disaster if continuous monitoring is not performed. Increasing traction and leaving the patient unattended can be fraught with risk due to the presence of partial ligamentous injury. Once the traction exceeds the ligamentous tolerance, over distraction can occur, resulting in severe neurological abnormality. In the presence of C-arm fluoroscopic visualization or with continuous monitoring, distraction can be avoided. A gentle amount of sedation that is reversible together with muscle relaxation can assist in the procedure. General anesthesia to permit reduction is fraught with complications, although Vaccaro has described safe emergent cervical reductions with awake intravenous sedation. With closed reduction, as with surgery, there is a window of safe surgical and nonsurgical intervention. Increased risk of neurological complication due to cord edema does occur after the first 24 to 48 h. After that, some physicians wait up to 7 days before they manipulate the spinal elements, especially in the presence of spinal instability.

Accurate assessment and early supervision of the spine in multiple trauma helps to reduce complications, achieve the desired results, and maximize outcomes. Rapid reestablishment of spinal alignment also creates the environment for maximum cord recovery. Once the spine has been realigned in the face of deteriorating neurological function, surgery on an urgent basis can be contemplated.

VII SUMMARY

Initial contact with trauma victims is the initial step to recovery. Life-threatening injury may delay spinal surgery but must not delay urgent measures to reduce or stabilize the spine and to optimize recovery of the cord by medical treatment.

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5

Timing of Surgery Following Spinal Cord Injury

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I TIMING OF SURGERY IN SPINAL CORD INJURY

As many as 10,000 people a year sustain and survive spinal cord injury (SCI) and approximately 200,000 people live with some degree of SCI in the United States today. Many of the recent medical developments in the treatment of SCI have been related to injury prevention and rehabilitation. Advances in pharmacological therapy have been shown to have a positive effect on preventing progression and even improving neurological function following SCI.

Evidence to support surgical management of SCI due to fractures or dislocations of the spine is substantial. What remains controversial, however, is the optimal timing of surgical intervention. Timing of surgery should be analyzed in terms of a risk/benefit ratio in regard to the potential for neural recovery, associated systemic medical complications, as well as cost to society.