Immediate Care of the Wounded

Clifford C. Cloonan, MD, FACEP
COL (ret) U.S. Army

Associate Professor
Deptartment of Military & Emergency Medicine
Uniformed Services University of the Health Sciences

Disability

If thou examinest a man having a dislocation in a vertebra of his neck, shouldst thou find him unconscious of his two arms and his two legs on account of it, while his phallus is erected on account of it, and his urine drops from his member without his knowing it; His flesh has received wind; his two eyes are bloodshot; It is a dislocation of a vertebra of his neck.[1(p 5)]

The Edwin Surgical Papyrus

The British Admiral Lord Horatio Nelson, at the time of his greatest victory, the defeat of Napoleon's fleet at Trafalgar in 1805, was mortally wounded by a gunshot to his chest that struck his thoracic spine and caused paraplegia below the breast. Mr. Beatty, the ship's surgeon, was called and upon his arrival Nelson is reported to have said, "*Ah, Mr. Beatty! I have sent for you to say what I forgot to tell you before, that all power of motion and feeling below my chest are gone and you very well know I can live but a short time…You know I am gone.*" Mr. Beatty's reply was: "*My Lord, unhappily for our Country, nothing can be done for you.*" Nelson died a few hours later.[2]

The Death of Lord Nelson by William Beatty

Overview and Epidemiology of Head and Spine Injury

As reported by Picket et al. in their population-based study of brain injuries,[3] blunt head injury is a leading cause of death[4] and disability in the United States.[5] In the United States roughly 1.5 million people sustain a head injury each year and head injury causes about 50,000 deaths annually.[6] Of those with traumatic brain injury [TBI] at least 80,000 are left with some degree of long-term disability.[7] Head trauma in the U.S. is very costly generating roughly $346 million in emergency care costs[8] and $54 billion in associated hospital care costs each year [in 1990 dollars].[9]

Despite its rarity, spinal cord injury is also a significant contributor to health care costs in the United States. Acute spinal cord injury occurred in only 2.6% of the 114,510 patients entered into the Major Trauma Outcome Study between 1982 – 1989.[10] Within the U.S. only around 10,000 individuals sustain a spinal cord injury (SCI) each year.[11(p.329)] Despite a low annual rate because of the permanent nature of cord injury almost 200,000 people in the United States have some degree of paralysis caused by SCI.[12] Annual aggregate costs, in 1988 dollars, for spinal cord injury were estimated by Berkowitz to be $5.6 billion.[13]

Combat-associated wounds of the head and neck are also costly in terms of human life, disability, and long-term cost. Such injuries occur at a frequency greater than would be predicted by body surface area and cause a disproportionate number of combat deaths. Although the head and neck make up only about 9% of adult body surface area, in the past 75 years, roughly 17% of all combat wounds have been to the head and neck. Head wounds have accounted for around 14% of all isolated or major combat caused wounds[14] and spine injuries make up about 2% to 3%.[15]

The relative frequency of combat-related head and neck wounds varies considerably depending upon the character of ongoing military operations. During the Korean War, for all types of combat operations, injuries to the head and neck accounted for approximately 20% of all wounds, whereas during withdrawal operations, only 13% of all wounds were to the head and neck.[16(p.44)] These differences in frequency reflect differences in anatomic exposure and variations in the type of weaponry employed in different operations.[16 (p.46)] During World War II, of 14,000 battle casualties who survived long enough to be treated in Fifth U.S. Army hospitals, 6.17% had wounds involving the head (excluding maxillofacial wounds). One-third of these wounds were classified as intracranial and two-thirds involved only the scalp; this reflecting the high lethality of wounds that involve penetration of the cranium.[17(p.99)]

One reason the head and neck are disproportionately injured is because combatants frequently expose this part of their body to engage the enemy. Some of this disproportionality may also be due to there being a higher percentage of combatants with minor head injuries who seek medical care than occurs in those sustaining similar wounds elsewhere on the body. Improvements in, and increased use of, body armor will certainly affect the total number of casualties with head and neck wounds occurring in a given combat engagement; but there is currently no definitive evidence that shows this is occurring. Given the vital structures contained within the head and neck, it is not surprising that combat-associated wounds to the head and

neck have a high incidence of morbidity and mortality. Head wounds have accounted for almost half of all ground combat deaths since World War II.[18] During the Korean War, bullet wounds to the head resulted in death 59.9% of the time and fragment wounds to the head from explosive devices caused death 37.7% of the time.[15 (p.44)]

Head Injury

Despite significant advances in neurosurgery and critical care, penetrating head wounds remain the second most common cause of combat-related deaths (following hemorrhage). Spinal cord injuries, particularly those in the cervical spine, although rare, are also highly lethal. The case fatality rate for bullet-caused head wounds during the Korean War was 14.7%; this only being exceeded by bullet-caused abdominal wounds (14.9%) as a single cause of death following hospitalization (the category "body generally," which covered multiple causes and locations of wounds that were otherwise not identified is listed as the cause of death in 33% of all DOW during the Korean War).[16(p.44)]

During the Korean War, the relative proportion of DOW from all causes was 25.4% for wounds of the head (for wounds of the face it was 5.4%).[16 (p.44)] Overall, major head wounds constitute about 14% of all combat casualties. Of these, almost 50% die either immediately or shortly after wounding. Of the 50% who do not die immediately, about 20% require advanced medical care or they will die within 6 hours; without neurosurgical care another 30% will die within 24 hours. Most of the remaining casualties with a major combat-caused head wound in which the dura has been penetrated will die within a week of wounding from infection unless properly treated.

Thus, without timely and proper treatment, nearly 90% of all combat casualties with serious head wounds involving penetration of the dura will die; even if such treatment is immediately available, a significant number will still die.[14] In Vietnam 20% of those with penetrating head wounds who survived beyond the first few minutes had very severe wounds and died without surgery soon after admission. The other 80% who survived long enough to reach a hospital had surgery, with a mortality of about 10%. Fortunately, most of these eventually returned to productive lives.[19] It is fortunate that a sizeable number of head wounds do not involve penetration of the cranium, because when penetration occurs, death usually results.

Although head and neck wounds are still associated with a high morbidity and mortality, the prognosis of casualties with these injuries has improved considerably in the past century. The mortality associated with all head wounds was 73.9% in the Crimean War and 71.7% in the American Civil War.[20, 21] In World War I, under the guidance of the famous neurosurgeon, Harvey Cushing, the DOW rate of penetrating head wounds, fell from 78% to 28.8% (keep in mind that this was in the pre-antibiotic and pre-diathermy era).[22, 23]

When antibiotics were introduced during World War II, the DOW rate for penetrating head wounds fell to 14%[24] and during the Korean War, to below 10%.[16 (p.44),20, 25] The relative improvement in survival of casualties with head and/or neck wound seen during the Korean and Vietnam Wars, as compared to earlier conflicts, was most certainly even better than the DOW rate would suggest because in both of these conflicts, rapid aeromedical evacuation of such casualties was routine;[26] rapid transport of such casualties to a hospital would be expected to cause a

paradoxical increase in the DOW rate because more casualties with unsurvivable wounds live long enough to reach the hospital, only to die shortly thereafter.

In a study of 690 missile head wound casualties of the Iran-Iraq war (1980-1988) who had dural penetration (which occurred in 74% of all casualties with head wounds),[21, 27] Aarabi found that 72.1% of these wounds were caused by shell fragments (mainly from shrapnel, artillery, bomb, mine, hand grenade and surface-to-surface missile) and 11.8% were from gunshot wounds (mostly 7.62 bullets fired from AK-47 assault rifles). In 16.1% of cases, the wounding agent could not be determined.[28]

Small fragments, not bullets, produce most wounds in modern combat[29] and bullets, being much more lethal than fragments, are much less likely to produce a living casualty. This probably accounts, in part, for the overwhelming predominance of fragment, as opposed to bullet, wounds of the head seen in combat medical treatment facilities. The Kevlar helmet effectively prevents penetration of most fragments but not most bullets (see following discussion), so when it is used, the relative percentage of bullet, as compared to fragment-caused head wounds (lethal and non-lethal) would be expected to increase.

Neck Injury

Penetrating wounds of the neck also have a high morbidity and mortality. In a recent study of 54 Israeli soldiers sustaining penetrating neck wounds due to combat-type mechanisms, 26% (14) died before reaching the hospital and another 15% (8) died after reaching the hospital, for an overall mortality rate of 41%. Most of these casualties sustained projectile wounds (38) and gunshot wounds (13).[30]

Even in a civilian setting, overall mortality due to penetrating neck trauma is as high as 11%,[31] and if there is injury to major vascular structures such as the carotid or subclavian vessels, mortality may exceed 60%.[32]

Of 4,555 cases in the WDMET database of Vietnam casualties, 614 records were coded as belonging to combat casualties with some form of penetrating neck wound (open neck wounds; superficial neck wounds; open laryngeal injury; vascular injury of the head and neck; and spinal column injury, with or without cord injury) for an approximate incidence of 13% of all combat casualties having some type of penetrating neck injury.[33] Because of their relatively high lethality casualties with penetrating neck wounds make up a considerably smaller percent of the surviving wounded.

Injuries Involving the Spinal Cord

A survey conducted 1 month after D-Day in World War II showed that about 10% of all casualties who reached general hospitals were neurosurgical. Of all injuries in casualties surviving to present for care, head injuries accounted for about 4%, injuries to the spinal cord about 1.5%, and peripheral nerve injuries 5–6%.[34 (p82)]

The history of Soviet medical care of spine injuries during World War II is illustrative of the influence that the tactical situation can have on the relative frequency of the various causes of penetrating spine injury in combat casualties and on wounding patterns. In the Soviet experience in World War II bullet wounds caused on average 42.5% of all combat-related damage to the spine, fragments caused 57.3%, and blunt trauma caused only 0.2%. The frequency with which each of these mechanisms caused spine injury varied,

however, depending upon the tactical situation. In defensive operations, fragment wounds made up a much higher percentage, whereas in offensive operations, bullet wounds of the spine increased in frequency.[35(p45)]

Flexion Teardrop, Unstable Fracture of C5, from Operational Medicine 2001

Different types of penetrating injury were also associated with different rates of associated spinal cord injury. Significant injury was caused to the spinal cord more frequently when the wounding agent was a large fragment from an artillery shell or bomb (unimproved munitions), whereas there was less cord damage from smaller fragments that produced "multi-fragment wounds" of the spine.[35 (p.46)] Bullets would also be expected to more commonly cause spinal cord injury as compared to fragments from improved conventional munitions.

The tactical situation also caused differ-

ences in the relative frequency of cord injury and in the anatomic distribution of spine injuries in casualties presenting to hospitals. The preponderance of wounds of the thoracic spine in surviving casualties was, at least in part, due to the fact that those with penetrating neck wounds were more likely to die prior to reaching a hospital (selection bias).[35 (p52)]

Actually, despite noting a relative infrequency of cervical spine injuries, the published frequency in this text of such injuries was still 19.4%, with 38.7% occurring in the thoracic spine, 35.8% in the lumbar spine, and 5.7% in the sacral spine (it is unclear from the Soviet text what injuries accounted for the remaining 19.8%). What was significantly different, however, was that, as compared to those with other injuries of the spine, those with cervical spine injuries rarely had wounds that penetrated into the spinal canal. This was explained in the Soviet text as being due to the fact that those with cervical cord injuries "…more frequently perished on the field of battle."[35 (p.55)]

Despite their infrequency among surviving casualties, patients with spinal cord injuries often require significant medical resources, both initially and throughout their lives. As noted by Frohna,[36] in 1992 dollars, the average direct cost of caring for a ventilator-dependent patient with a high cervical spine injury was $417,067 for the first year and $74,707 for each subsequent year.[37(p.1-5)]

These are very debilitating injuries, so efforts to minimize cord damage wherever and whenever possible are certainly reasonable and appropriate. The great majority of combat-caused head and neck wounds continue to be penetrating, but as the total number of U.S. casualties caused by direct enemy action has declined the

relative frequency of blunt trauma from motor vehicle and aircraft crashes and from falls has likely increased. As of June 2003, accidents of all causes had accounted for 37% of all Operation Iraqi Freedom fatalities, and almost half of those were due to vehicular accidents.[38] For this and other reasons, it is important to review how such injuries are managed at or near the point of injury in civilian and battlefield settings.
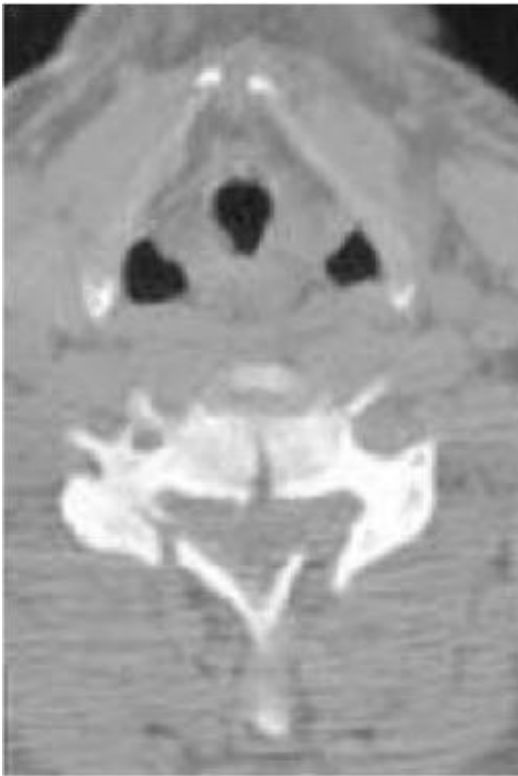
Flexion Teardrop, Unstable Fracture of C4, from Operational Medicine 2001

Much of how we care for patients with head and spine injuries in combat today comes from civilian practice, so it is important to understand that medical practices appropriate in a civilian setting may not always be appropriate in a combat environment. There are significant differences in combat mechanisms of injury as compared to injury in civilian settings.

Even blunt spinal trauma in combat may be different from civilian blunt trauma. Combat casualties may suffer spine injury due to mechanisms such as ejection from high-performance aircraft and aircraft accidents, which either do not occur at all in civilian settings or occur much more rarely. The environment in which these injuries are cared for is also significantly different.

The exact impact of these differences on the frequency of unstable fractures is unclear, but it is likely that the frequency of unstable fractures among combatants with cervical spine injuries is different than that in a random mix of civilian patients with cervical spine injuries. These differences alter the relative risk-benefit ratio of any care provided to combat casualties such that a medical practice that might produce more good than harm in a civilian setting might well have the opposite effect in a combat environment.

To understand how modern prehospital care for head and spine injuries has come to be what it is, it is important to examine the epidemiology of civilian head and spine injuries and to consider the modern civilian prehospital practice environment. Within the civilian population approximately 11% of all trauma admissions have a head injury[39] and 1-3% of all blunt trauma victims with cervical fractures[40, 41] have an injury to the spine [spinal cord]. According to the American College of Surgeon's ATLS course, approximately 55% of spine injuries occur in the cervical region, 15% in the thoracic region, 15% at the thoracolumbar junction, and 15% in the lumbosacral area.[42]

Blunt trauma, primarily from motor vehicle accidents, accounts for approximately 82% of all serious non-fatal civilian head injuries (Traumatic Brain Injury in the

United States: A Report to Congress CDC Estimates of Traumatic Brain Injury-Related Disability Current Data @ http://www.cdc.gov/doc.do/id/0900f3ec800101e6/) and a similar percentage of all civilian spine injuries.[36] In civilian trauma, only around 15% of all spine injuries[43-45] and approximately 10% of all head injuries (Traumatic Brain Injury in the United States: A Report to Congress CDC Estimates of Traumatic Brain Injury-Related Disability Current Data @ http://www.cdc.gov/doc.do/id/0900f3ec800101e6/) are caused by a penetrating mechanism, most of these being the result of a low-velocity gunshot wound from handguns.

A number of articles have placed the frequency of spinal column injury from gunshot wounds to the neck (primarily low velocity) from a low of 2.7%[46] to a high of 22%[47] with neurological deficits being present in 1.9%.[46, 47]

Civilian vs. Military Spine Injuries

The above discussion about epidemiology of wounding is relevant to a discussion about point-of-wounding care for casualties with spine injuries. When making risk-benefit decisions about the care of such patients, it is necessary to know, at least approximately, the portion of surviving casualties who will have spinal cord and unstable cervical spine injury. The benefit of reduced risk of spinal cord injury must be balanced against the logistical challenges and the risks associated with spine immobilization in a combat zone; the outcome of this analysis is substantially influenced by the incidence of unstable spine injuries in combatants. The more common unstable spine injury is in a given population the greater the relative benefit of spine immobilization.

The estimate by Arishita et al.[33] of an in-

cidence of 1.4% unstable penetrating cervical spine injuries in combat casualties with penetrating neck wounds is significantly influenced by how many casualties with potentially unstable cervical spine injuries died prior to treatment. In their analysis 20 of 365 [5.5%] combat casualties who had potentially survivable penetrating neck wounds, died. If these 20 casualties had been included in Arishita et al.'s estimate of unstable spinal cord injury, the frequency of unstable cervical spine injuries among those who do not die immediately could be more than twice as high (3.7%).[48]

However, unless it becomes significantly easier in the future to reach and treat combat casualties with penetrating neck wounds, there will always be a group of patients who will die before they receive care; this fact must be considered in any risk-benefit analysis of medical care.

In the article by Arishita et al.,[33] the case of a soldier shot through the neck and found lying face down in the water is illustrative. This casualty was dragged out of the water by his comrades without any regard for his cervical spine, and was later found, on autopsy, to have an unstable cervical spine injury with cord damage. This individual was described by the authors as someone who might potentially have benefited from cervical spine immobilization, when in fact it seems probable that if the casualty were not already paralyzed, he would have removed his own face from the water. Had care not been almost immediately available, it is highly likely that this casualty would have died at the site of wounding from exsanguination or drowning, rather than later. Thus, the fact that his cervical spine was not immobilized in the middle of a firefight seems not to be especially relevant.

Injury to the head and neck from frag-

ments is quite rare in a civilian setting but is relatively common on the battlefield. The great majority of civilian bullet wounds are caused by low-velocity handguns, whereas most bullet wounds to the head and neck sustained during combat are caused by high-velocity weaponry, with low-velocity bullet wounds being rare in combat. These differences in mechanism of penetrating trauma make it difficult to compare penetrating neck trauma in civilian and military settings. The frequency of cord injury and spine instability is certainly different between wounds caused by high-velocity, full-metal-jacket rounds and those caused by soft-lead, low-velocity bullets shot from a handgun. An extensive discussion of these differences is provided later in this chapter.

Another difference between civilian and military head and neck trauma is that the great majority of combatants are healthy males between the ages of 18 and 35, whereas the civilian population includes both the very young and the very old: two groups of patients with a much higher risk of head and neck injury and of spine instability.

Different age groups even have different patterns of injury. In infants and small children, the head is relatively much larger than the neck and body as compared to adults, and the supporting musculature is much weaker, making certain injuries considerably more likely. On the other end of the age spectrum, degenerative changes predispose the elderly to spine and spinal cord injuries. In both the very young and the elderly, high cervical spine injuries are relatively more common compared to those in the age range of most combatants (18-35), in whom injuries to the lower cervical spine predominate.

In the multicenter National Emergency X-

radiography Utilization Study[49] (NEXUS) participants between 20 and 30 years of age sustained their injury between the levels of C5 and C7 around 50% of the time and the vertebral body was the structure fractured in roughly 30% of these lower cervical vertebrae – making it the single most commonly injured structure.[50] Importantly isolated vertebral body fractures are rarely associated with spinal cord injury.[50] In the 20- to 30-year old age group, spinal cord injury occurred in only 4.1% of patients with a radiologically significant cervical spine injury.[51, 52] In the 60- to 80+ age range, however, fractures of C1 and C2 accounted for 57% all fractures.[51] A high percentage of C1-C2 fractures are unstable. In NEXUS, the relative risk for cervical spine injury in elderly blunt trauma victims was 2.09, compared to 0.87 for other adult blunt trauma victims.[52] All this suggests that the risk of spinal cord injury and spine instability is probably considerably higher in a civilian population that includes an elderly population than in combatants, nearly all of whom are young, previously fit, adults.

Definition of "Instability"

Before discussing the frequency of spine instability in blunt and penetrating injuries, it is important to define exactly what constitutes "instability." The term, "unstable fracture" is commonly used in the literature without there being general agreement as to exactly what this term means. Guttmann, in his 1976 text, *Spinal Cord Injuries: Comprehensive Management and Research*, [53] [(p179)] noted that, *"There is still disagreement as to the definition of stable and unstable fractures, and the criteria used differ considerably."* Because fear of causing spinal cord injury in a neurologically intact patient who has an "unstable" spine injury has led to the

current, nearly universal, practice of spine immobilization, it is important to define exactly what constitutes an "unstable" injury. Unfortunately, most investigators do not provide a definition when they use the term "unstable." Without an agreed-upon definition, it is not possible to do an accurate meta-analysis of studies to determine the true frequency of this condition. Given the rarity of unstable cervical spine injury, it is difficult to do a large enough study to determine its true incidence.

Instability is generally defined by the anatomic structures that are injured.[54, 55] White et al. performed an analysis of the clinical stability of cadaveric spines following sequential transection of the anterior and posterior structures (ligaments, annulus fibrosus, and articular facets), in flexion and extension, to determine which of these structures contributes most to spinal stability.[55] They found that the spine tends to remain stable even when most of the ligaments are transected.

They also found that when instability occurred in this model, it occurred suddenly and completely, without any warning of intermediate instability. With regard to the influence of the paracervical muscles on spinal stability, White et al. stated that, *"Although muscles exert some forces, we do not believe that they play a significant role in clinical stability."* They based this conclusion upon the clinical observation that in "severe motor paralysis of the paracervical muscles, significant displacement of the vertebral bodies and facets does not occur provided the bone and ligamentous structures remain intact." Obviously because their study used cadaveric spines, they could not test this hypothesis.

In any case, it should be noted that the converse situation is the issue in most patients; i.e., when the bone and ligamentous

structures are <u>NOT</u> intact, how much do the paracervical muscles contribute to stability? No study to date has assessed this important question, although this author is aware of unreported anecdotes that suggest that patients with unstable cervical spine injuries are, at least occasionally, able to stabilize their cervical spines through the use of paracervical and other muscles (reports of patients presenting to emergency departments stabilizing their own highly unstable cervical spine fractures with their hands "holding their head on").

Spine instability is occasionally defined functionally as well as anatomically. Hockberger et al., in Rosen's *Emergency Medicine: Concepts and Clinical Practice* text, state that if neurological injury has occurred, particularly delayed neurological injury, then, by definition, the original injury was *"unstable."*[56(p372)] They note that the concept of stability is *"...complex and somewhat confusing"* and that mechanically stable injuries may be associated with spinal cord injuries (this is certainly the case in penetrating trauma) whereas many patients with mechanically unstable injuries may have no neurological deficit.

Generally anterior column injuries, i.e. those involving only the vertebral body and/or intervertebral disks, are considered stable. Injuries of the posterior column (pedicles, transverse processes, articulating facets, laminae and spinous processes, together with their associated ligaments) are more likely to be unstable, especially if there is some degree of dislocation with associated ligamentous injury. Isolated fractures of posterior elements without dislocation have a relatively low incidence of associated neurological injury.

The most unstable of all injuries are those

involving elements of both the anterior and posterior columns.[50] In an extensive epidemiologic study by Riggins & Kraus, patients with isolated vertebral body fractures had a 3% incidence of neurologic deficit, whereas those sustaining fractures of the posterior elements and body with some degree of associated malalignment of the spine had an incidence of 61%.[51] Guttmann, in his *Spinal Cord Injuries* text, notes that data on the frequency of unstable spine injury vary considerably from as low as 3.5% to as high as 12%.[53]

Influence of Mechanism of Injury on Spine instability

It is unclear whether a fracture caused by blunt force is the same as a similar fracture caused by a penetrating mechanism in terms of spinal stability. It is likely that blunt trauma and penetrating trauma are quite different in this regard. The kinetic energy transferred to the spine and its associated structures during a motor vehicle or aircraft accident or during a fall from a height is considerably different than that transferred during penetrating injury. In penetrating injury, the kinetic energy of a bullet or fragment is expended within a relatively small space, whereas in blunt trauma, the kinetic energy is spread over a much larger area.

The rate of energy release is also different. In high-velocity penetrating injury, all of the kinetic energy is released into the tissue within microseconds. In blunt force injury, the transfer of energy is relatively much slower. These differences in energy magnitude, distribution, and transfer time are probably important in terms of likelihood of spine instability.

Many authors have stated that bullets cause spinal cord injury from "direct trauma" rather than from movement of an unstable spinal column after the initial injury.[57-60]

Many factors may contribute to the observed differences but it appears that spine instability is considerably less common when the mechanism of injury is penetrating. It is therefore probably erroneous to assume that the degree of spine instability present in a patient with a fracture caused by blunt force would be the same in a patient with an identical fracture caused by a penetrating mechanism.

Barkana et al. note that all of the literature concerning definition, description, and experiments of spine instability is based on blunt trauma.[30, 61-67] They go on to comment that when penetrating injuries are evaluated, it is very rare to find unstable injury, and they state that it is *"…conceptually impossible for a penetrating injury to cause such substantial spinal damage leading to instability without completely destroying the cord."*[30] It should be noted that this Israeli study by Barkana et al., and their collective experience, is probably influenced by a predominance of penetrating injuries caused by high-velocity military bullets.

Apfelbaum et al. describe a case of "unstable" cervical spine injury without cord damage caused by a 22-caliber long rifle bullet fired from a handgun.[44] Although this patient's spine may have met an anatomic definition of "instability," the functional "instability" of even this injury is disputable. This patient did not sustain a spinal cord injury despite periods of time, both before and after receiving medical attention, in which her cervical spine was not immobilized. During the time her spine was not immobilized there was quite probably movement that would have

caused cord injury had there been significant instability; it is certainly unlikely that a patient who had been shot in the neck would lie perfectly still while waiting for the ambulance to arrive. Low-velocity bullets and fragments certainly can cause a spine injury that fits the commonly used anatomical definition of "unstable" *without* causing concomitant cord injury even when movement occurs.

Although current literature suggests that the risk of an unstable cervical spine following penetrating trauma is minimal,[33, 56] there is little data to support this statement. In their retrospective review of cases in the WDMET report with penetrating neck wounds, Arishita et al. conclude that the risk of an unstable cervical spine injury is very low in combat casualties.[33] Only one study has looked specifically at the frequency of cervical spine instability in penetrating trauma, and it concluded *that "spinal stability following a gunshot wound is not guaranteed, especially in the cervical spine, and each case should be assessed individually for the presence of instability"*(see earlier discussion).[58] No study in the current literature, however, contains a side-by-side comparison of overall morbidity and mortality associated with management of penetrating neck with immobilization vs. no immobilization. For many reasons it is quite unlikely that such a study will ever be done. Only recently have multicenter trials begun to compare different approaches to the management of patients with blunt cervical trauma.[67]

Isiklar & Lindsey[58] retrospectively evaluated patients with low-velocity gunshot wounds to the spine who presented to a civilian facility. Of 12 cases of gunshot wounds involving the cervical spine, 3 (25%) were described as "unstable." In this study, clinical stability of the subaxial cervical spine was defined "according to a

scoring system developed by White & Panjabi," and described as

> *...a quantitative analysis of the behavior of the spine as a function of the systematic destruction of various anatomic elements. Under controlled conditions designed to maintain the biological integrity of the specimens, 17 motion segments from 8 cervical spines were analyzed. The spines were studied with either flexion or extension simulated using physiologic loads."*[69]

In the Isiklar and Lindsey study, 11 (92%) patients had neurological deficits, and 8 (67%) had related vascular injuries. Only 1 patient (8%) had an unstable cervical spine without a neurological deficit.[58] In order to assess cervical spine instability in the manner described by White et al.,[68] it would have been necessary for Isiklar and Lindsey to perform flexion and extension of the cervical spine to determine whether there was > 3.5 mm of linear intervertebral displacement and/or > 11° of angular displacement. There is no evidence in their retrospective record review that this is how cervical instability was determined; in fact, the contribution of bony injury to instability was not considered.[58] This brings into question how cervical spine stability was assessed in this study and suggests that this study can't be used to estimate the frequency of spine instability in penetrating neck trauma.

High-velocity bullets tend to cause "all-or-none" injuries in the neck. If no vital structures are hit, they may pass through the neck causing little damage, but if the spine is struck (excluding the tip of a spinous or transverse process), the damage tends to be catastrophic, with immediate quadraparesis and, often, death.
This "all-or-none" phenomenon tends also

to be true for high velocity gunshot wounds to the head. Where low-velocity handgun bullets may penetrate the skull to cause neurological damage, but not death, such an event rarely occurs in casualties sustaining high-velocity gunshot wounds to the head.

Apfelbaum et al.[44] make the observation that lower velocity, smaller caliber handguns are associated with a different injury pattern[58, 63, 70] than previously seen in military studies,[33] including an increased frequency of fracture without neurological impairment and increased associated vascular injury. They postulate that one reason for the increased incidence of cervical spine instability with low-velocity gunshot wounds may be, "…the decreased amount of surrounding soft tissue [in the neck], compared to the thoracic or lumbar spine, such that an increased amount of the bullet's kinetic energy is conveyed to the spinal column with increased skeletal injuries."[58]

Although this mechanism may partially account for the higher incidence of cervical, as compared to thoracic or lumbar, spine instability, another explanation is more likely. Most studies assessing spine instability have been done on admitted patients and do not assess spine instability in nonsurvivors. Such studies therefore fail to identify the most likely explanation for the low incidence of spine instability among survivors of such wounds, i.e., that most patients with gunshot wounds who have a spine injury severe enough to produce instability also sustained injury to critical structures that lead to rapid death (Since a high velocity bullet is more likely than a low velocity bullet to kill it's victim, a patient with a low velocity bullet wound is more likely to survive with an unstable spine injury; thus since only survivors tend to be assessed for the presence

of spine instability those with low velocity GSW appear to have a higher incidence of unstable fractures).

It is clear, therefore, that the mechanisms causing civilian head and neck trauma, even when comparing blunt with blunt and penetrating with penetrating injury, are different in ways that should impact on management decisions because each has a different likelihood of causing an unstable spine injury in a surviving casualty. It is also clear that, in a combat setting, where there is often a persisting risk of death and injury both to the casualty and to anyone attempting to rescue and treat the casualty, the risk-benefit ratio of any procedure(s) that might be done is also affected. In the WDMET database one of every ten casualties was wounded or killed while attempting to render aid to another casualty.[33]

Selection Bias

The true incidence of instability in blunt and penetrating spinal trauma is difficult to ascertain because of selection bias. The great majority of studies that address the question of spine stability have been performed by neurosurgeons. Patients treated by neurosurgeons, however, have a higher incidence of instability because, in many cases, concerns about instability led to their being referred to a neurosurgeon in the first place.

Frequency of Spinal Cord Injury and Instability in Penetrating Trauma

Barkana et al.[30] note that in a study done by Hammoud et al.[71] of spinal cord injuries during the Lebanese civil war, none of the 24 injuries reported had spine instability. They go on to say that spine instability

occurs very rarely in spine injuries from fragments and bullets because the bone architecture is only a little disturbed. Despite a statement by Yoshida et al. in their article on gunshot wounds to the spine that, *"With few exceptions [gunshot wounds to the spine] result in a spinal cord injury,"*[72] in fact, this is *not* the case. More importantly, among those who survive the initial injury (which is the only population of medical concern) the percentage of patients with gunshot wounds to the neck who also have an injury to the spine is in the 5 to 20% range.[30, 33, 47, 73-75] Although there may be a higher incidence of associated spinal cord injury in penetrating spinal trauma as compared to blunt, the fact remains that overall spinal cord injury is uncommon in this group, especially if only those who survive the initial injury are considered.

The writings of Sir Zachary Cope, based on his and others' experiences during World War II, seem to agree with Barkana et al.[30] that damage to the spinal cord by high-velocity missiles is generally caused by direct trauma, not by spine instability that later produces spinal cord damage. Cope noted that:

> *...the stability of the spine was not much endangered by a shell fragment...small pieces of metal moving at high velocity were the usual wounding agents [during World War II]. These did damage by penetration or by traversing the body but they did not very often shatter the spine...[not] all of these wounds appeared to result in total and lasting paraplegia. Recovery seemed possible in about 25-30 percent of cases.*[76(p381)]

Although concern for patients is the primary factor that has shaped current civilian EMS, guidelines regarding the prehos-

pital management of patients with head and neck injuries, fear of litigation, and dogmatic adherence to practices that lack scientific evidence of efficacy have also played a role. These issues will be discussed in detail below in the section on spine immobilization.

Diagnosis

Although medical personnel at or near the point of wounding have, for many reasons, a limited ability to make specific diagnosis in patients with head and neck trauma, a degree of diagnostic certainty can often be achieved. Despite the challenges, it is important to be as diagnostically precise as possible in the forward areas for the following reasons:

1. Diagnostic accuracy can considerably improve patient management at or near the point of wounding. If it is reasonably clear what the patient's diagnosis is, or perhaps more importantly, what it is not, then treatment can be focused on those who will benefit most from it. In forward areas, a focus on diagnostic accuracy also allows the most efficient use of limited staffing and equipment resources.

2. An accurate diagnosis is essential to making proper evacuation decisions. Combat casualties with unmistakable neurological injury or an unstable spine should be evacuated to the care of a neurosurgeon once they are clinically stable and as soon as logistically possible. Early access to a neurosurgeon has been associated with improved outcomes for patients with serious head and spine injuries.[77(p.177)]

Clues to a patient's diagnosis can be ob-

tained by ascertaining an accurate mechanism of injury. This can often be accomplished by simply asking the patient what happened. Patients who are conscious and able to speak can often describe the exact mechanism of injury and can frequently describe their symptoms in terms that allow for an accurate diagnosis. The challenge is to be able to accurately interpret what the patient has said and to use that information to make the proper diagnosis.

A rapid physical examination can also provide critical clues. Most importantly, care providers at or near the point of wounding must maintain a high index of suspicion for serious head and neck trauma, because the effect of a missed diagnosis can be catastrophic. The focus of the following discussion will be on the diagnosis of spine injury and unstable spine injury. Head injury diagnosis and management is discussed later. Although it is not possible, at or near the point of wounding, to diagnose spine instability with certainty (indeed this is difficult to do even in a fully equipped hospital) it is possible, primarily by knowing the mechanism of injury, to roughly determine the probability of spine instability.

Clearing the Cervical Spine at or near the Point of Wounding

A major current area of discussion and controversy in civilian emergency medical services is the "clearing" of patients with potential cervical spine injuries prior to transport, allowing prehospital personnel to exercise judgment as to which patients with possible spine injury actually need to be immobilized. This civilian controversy is primarily fueled by

  (1) The costs associated with placing a high percentage of trauma victims into cervical spine immobilization

(estimated at $75 million annually within the United States)[36] and

  (2) The risks associated with cervical spine immobilization. This issue of cervical spine immobilization will be discussed later when the treatment of patients with known and potential spine injuries is discussed.

How capable are prehospital care providers at accurately sorting determining which patients do and do not have spine injury, particularly unstable spine injury? As Hoffman et al. point out, an assessment for spine injury could be almost 100% sensitive if every casualty with even a remote possibility of spine injury were presumed to have such an injury.[78] Unfortunately, such an approach in a military setting would cause substantial over-evacuation, would create a significant burden on care providers, and would expose a large population of casualties without spine injury to the risks of spine immobilization (discussed below).

For these reasons, it is essential that a reasonable degree of specificity be obtained. So, what evidence is there that prehospital care providers can accurately identify those with spine injuries? More importantly, how able are they to identify those who may suffer adverse consequences if there were an error in diagnosis, i.e. patients whose spines are unstable and those who must be treated by a neurosurgeon? There are certainly some spine injuries (some spinous and transverse process fractures, and some compression fractures for example) that, if missed, will result in no adverse consequences to the patient.

As part of the large multicenter National Emergency X-Radiography Utilization Study (NEXUS), prospective data were collected on cervical spine injuries.[78] One part of this study involved prospective

assessment of the efficacy and safety of selecting patients with a very low probability of cervical spine injury for treatment not involving spine immobilization or radiography. The instrument used to make this determination is the NEXUS cervical spine criteria; i.e., patients with none of the following criteria were deemed to be safely *not* immobilized or radiographed:

- A focal neurological deficit

- Any evidence of intoxication

- Any tenderness at the posterior midline of the cervical spine

- Any painful injury that might distract the patient from the pain of a cervical spine injury

- Any alteration of consciousness from any cause

(The first four criteria demonstrated high inter-rater reliability in a study of blunt trauma patients assessed for cervical spine injury that was published in the same year.)[80] The Canadian C-Spine Rule, developed for the same purpose, uses the following criteria to decide which trauma victims need cervical spine radiography.[81, 82] By these guidelines, no cervical spine radiographs are indicated in alert and stable trauma victims if:

(1) There is no high-risk factor, including
   a. Age >64
   b. Dangerous mechanism (fall > 3 feet, axial load to head [e.g., diving], motor-vehicle crash at > 100 km/hour and/or involving rollover and/or ejection from vehicle, motorized recreational vehicle crash, or

   bicycle crash), or
   c. Paresthesias in extremities

(2) <u>And</u> if there are indications of low risk such as
   a. Simple rear-end motor-vehicle crash
   b. Patient in sitting position in emergency department
   c. Patient ambulatory at any time
   d. Delayed onset of neck pain, and
   e. Absence of midline cervical tenderness

(3) Patient is able to actively rotate neck 45 degrees to right and left.

Both the NEXUS and Canadian C-Spine Rule studies only assessed the efficacy and safety of their decision instrument when applied by physicians. Some authors have studied whether emergency medical services providers could apply an identical or similar instrument to make decisions about spine immobilization at or near the point of injury.

Brown et al. conducted a study comparing the application of the NEXUS instrument by EMS providers and emergency department physicians. The emergency physicians and the EMS providers were blinded to each others' assessments. There was 78.7% agreement. In only 7.7% of cases, the emergency physician indicated that the patient should be immobilized when the EMS assessment did not. In general the EMS provider's assessments were more conservative than those of the emergency physician's.[83] Unfortunately, this study does not provide any evidence as to the safety and efficacy of having EMS providers use the NEXUS guidelines to avoid immobilization.

Stroh et al. conducted a retrospective assessment of a prehospital protocol for out-of-hospital spine clearance that was used on 42,000 patients in Fresno County, California.[84] The charts of all patients (N=861) discharged from five Fresno County trauma centers with the diagnosis of "significant" cervical injury were examined. EMS personnel brought in 504 patients, of whom 495 (98.2%) had cervical spine immobilization in place. Of the remaining 9 patients, 2 refused immobilization and 2 could not be immobilized; 3 cervical spine injuries were missed by the protocol criteria and 2 were missed because of protocol violations. Of the 5 patients with injuries who were not immobilized, 1 had an adverse outcome and two had injuries that were considered unstable. The three patients missed by the protocol were at extremes of age: 9 months, and 68 and 83 years. Further, the two missed patients who were considered protocol violations were elderly (73 and 76 years of age), which suggests that cervical spine injury in infants and the elderly may be more difficult to ascertain in the prehospital setting (note that this is not an issue in military combatants).

Hoffman et al. in their review of the Stroh study,[78] offer the following words of caution about this study:

(1) For a variety of reasons, the actual sensitivity of the EMS practice in Fresno for immobilizing patients with cervical spine injury may well be much lower than reported and

(2) Chart reviews are subject to substantial biases and errors, even when done rigorously.[85]

One of the most important questions raised by Hoffman et al. is, "Did the use of the prehospital spine injury clearance protocol really lead to meaningful selective immobilization?" As noted above, immobilizing everyone would produce a sensitivity of 100%. Unfortunately, there is no way to calculate the specificity of the Fresno approach or to know whether, or to what degree, EMS personnel in the Fresno study were able to reduce *unnecessary* immobilization among patients with blunt trauma.[78] This is certainly the important issue because one of the primary objectives of any prehospital cervical spine injury clearance protocol must be to safely minimize the number of unnecessary immobilization procedures.

Finally, although the complexities of the issues raised and the resources that would be necessary to prospectively determine which patients require immobilization make it unlikely that a definitive answer will ever be found, the following points (elucidated by Hoffman et al.) should be kept in mind:[78]

(1) Any out-of-hospital protocol should emphasize safety (sensitivity) over efficiency (specificity). The cumulative small benefits associated with avoiding spine immobilization in many patients without injury must be balanced against the rare but extremely important harm associated with failing to immobilize injured patients. [*It should be noted however that the "cumulative small benefits" have never been well quantified (see following discussion about the risks of cervical spine immobilization) so it is not possible to know the true extent of benefit that might be derived from a selective immobilization protocol.*]

(2) Decision instruments proven to be effective in the hands of emergency physicians should not be assumed to

work equally well when applied by others (eg, paramedics or nurses), especially in a very different (out-of-hospital) environment [*certainly a true statement that applies to all procedures*].

(3) Any out-of-hospital cervical spine clearance protocol that is created should incorporate those elements with the best face validity (Is there neck pain? Did the mechanism involve forces that could possibly hurt the spine?), as well as elements from any protocols that have been proven to be useful in the ED. The number of patients immobilized by EMS should probably end up somewhat higher than the number radiographed in the ED.[78]

Probability of Spine Injury in Patients with other Injuries above the Clavicle

The American College of Surgeon's Advanced Trauma Life Support (ATLS) course states that, *"Any injury above the clavicle should prompt a search for a cervical spine injury."* According to ATLS approximately 15% of patients sustaining such an injury will have an actual c-spine injury and approximately 5% of head-injured patients have an associated spine injury. [42 (p217)]

Others also consider the presence of head or facial injuries to be an indication for cervical spine radiography.[86, 87] The rate of cervical spine injury in facial trauma series varies from 0% to 4%.[88] Bayless et al. reviewed 1382 cases of mandibular fractures and found cervical spine injuries to be rare.[89] They concluded that history and physical examination, without radio-

graphic studies, are sufficient to evaluate the alert, cooperative patient with blunt, low-velocity mandibular trauma and no other complicating features.

Other reports[36, 90, 91] have confirmed the low incidence (1.04% and 1.8%) of cervical spine injuries in patients with facial trauma. Williams et al. reviewed the records of 5,021 trauma patients and found that there was no higher incidence of cervical spine injury in head- injured patients, in patients with facial trauma, or in patients with clavicular fracture than in those without.[92] If the Glasgow Coma Scale (GCS) was used to stratify head-injured patients, a higher incidence of cervical spine injury was noted in patients with a GCS < 14 than in those with a GCS > 14.

Hills and Deane reviewed a series of 8,285 blunt trauma victims and found that facial injuries were not associated with cervical spine injuries;[93] however, they found a much greater risk of cervical spine injury in victims with "clinically significant" head injury. In another study by Bayless et al., of 228 cases of blunt head trauma, only 3 were found to have cervical spine injuries for an incidence of 1.7(3)% [Note that 3/228 = 1.3% while 4/228 = 1.7% but original article states incidence is 1.7%].[89] However, when only those patients with serious head injury (more than a mild concussion) were considered, the frequency of cervical spine injury in this study rose to 5%. It is not surprising that in patients with evidence of serious head trauma, there is a significantly higher incidence of cervical spine injury.

A recent study by Patton et al. has some relevance to the care of combat casualties. In this study 102 individuals sustaining a blunt assault to the head and neck region were evaluated, but no clinically significant cervical spine injuries were detected

(there was one patient with a spinous process fracture who had been hit in the back of the neck with a pipe – not clinically significant).[94]

Also of military relevance are three retrospective studies that concluded that patients with gunshot wounds limited to the head do not have cervical spine injuries and do not require immobilization.[95-97] Despite there being little evidence to support cervical spine immobilization for victims of blunt trauma solely on the basis of injury above the clavicle, the majority of studies still conclude that, "Immobilization of the patient with an injury above the clavicle is prudent until a physician is able to evaluate the patient fully for possible cervical spine injury and determine the need for radiographs."[36]

This recommendation is, no doubt, based upon an assumption that the overall risk of spine immobilization is so low that there is no reason not to immobilize every patient in whom there is any question about whether a cervical spine injury is present. This may or may not be a reasonable assumption in a civilian setting, but as noted later in the discussion about cervical spine immobilization, it is probably *not* a reasonable assumption in a combat setting.

Clinical Findings in Cervical Spine Injury

Fortunately subtle, occult, or delayed neurological injury is quite rare. In most patients with spinal cord injury, neurological impairment is clinically apparent early in the course of evaluation.[98, 99] This certainly seems to be true in the case of penetrating neck injury, although delayed neurological damage has been described.[44, 52, 99]

Prehospital evaluation of penetrating neck

trauma should include consideration of the mechanism of injury, wound location, and the presence of suspected entry and exit wounds. Although it has been suggested by some that the reliability of the prehospital clinical evaluation for the potential of spine injury is not affected by the mechanism of injury (Domeier et al.),[101] there is no question that, in fact, the *probability* of spine injury is affected by the mechanism of injury and the amount of potential energy transfer. There are differences in probability of spine injury between penetrating and blunt trauma. It is also certain that a victim of a high-speed motor vehicle accident or a fall from a significant height is much more likely to have a spine injury than someone who has tripped and fallen.

Undoubtedly the results of the civilian Domeier study were influenced by the inclusion of elderly patients who tend to have degenerative bone disease and a higher probability of malignant lesions that would predispose them to injury from even minimal forces. This is not an issue for active duty soldiers, for whom a substantial amount of force is required to cause an injury to the spine.

In terms of causing spine injury, penetrating wounds caused by knives and bayonets are different from those caused by projectiles, and wounds caused by high-velocity bullets are different from those caused by handgun bullets and fragments.[100] Although the true path of a penetrating wound, even when there is an entrance and an exit, cannot be determined by examination of the external wound(s) none of these wounds should ever be probed.

Knowledge of the size and type of blade and the angle of entry (if it can be determined) is useful in estimating what struc-

tures might have been damaged by a stab wound. The position of the casualty and the trajectory of the wounding projectile can also be used for the same purpose.[99] In most instances, stab wounds to the neck cause fewer severe injuries than do projectiles. In a review of 218 patients with penetrating neck injuries undergoing mandatory surgical exploration, stab wounds had a 10% higher rate of negative exploration than injuries from projectiles.[102] The bottom line, however, is that a physical examination of the patient and a good description of symptoms from the patient are most important in making a diagnosis of spine, and particularly neurological, injury.

Priaprism, or penile erection due to retention of blood, is a diagnostic finding in spinal cord injury, especially cervical spine injury. The following guidelines are given for patient assessment in the field in the 2004 edition of *Intermediate Emergency Care: Principles & Practice*:

> *Examine the male organ for priaprism, a painful, prolonged erection usually caused by spinal cord injury or blood disturbances. Suspect a major spinal cord injury in any patient with a priaprism.[103(p.524)]*

Despite considerable effort no studies where found that addressed the incidence of priapism in spinal cord injured patients or that associated specific types of spine injury with the development of priapism. Although there is a paucity of data numerous texts and training manuals make reference to the need to check for the presence of priapism as a marker for spinal cord injury so it seems reasonable to make the same recommendation here as well. The

sensitivity and specificity of priapism as a marker for spinal cord injury is unknown.

Patients may have either complete or partial spinal cord injury. A complete spinal cord injury is defined as total loss of sensory or motor function below a certain level. If any motor or sensory function remains (e.g., sacral sparing), it is considered an incomplete injury. The prognosis for recovery from an incomplete injury is significantly better than from a complete spinal cord injury. It is particularly important that patients with incomplete injury be handled with care to prevent worsening of their condition and that they be quickly transferred to the care of neurosurgeon.

Signs of sacral sparing include the presence of perianal sensation, rectal sphincter tone, and any ability to move the toes[104] The sensory level of the cord injury is defined by the most caudal segment of the spinal cord with normal sensory function on both sides of the body. The motor level is defined as the lowest key muscle innervation that maintains a 3/5 (able to move against gravity) muscle grade.

Assessing deep tendon reflexes is also helpful in assessing for the presence of a spinal cord injury and this can certainly be done near the point of wounding. In the acute setting, muscle paralysis with intact deep tendon reflexes typically indicates a spinal cord (upper motor neuron) lesion, whereas paralysis with absent deep tendon reflexes suggests a nerve root or cauda equina (lower motor neuron) lesion. Because lower motor neuron lesions are often surgically correctable, this distinction is important. The deep tendon reflexes that are important to assess are as follows:

Location of Loss of Deep Tendon Reflexes	Indicated Location of Lesion
Biceps	At or above C6
Triceps with intact reflexes at biceps	C7
Patellar and Achilles tendon with intact upper extremity reflexes	T1 – L4
Achilles tendon only	L3 – S1

Penetrating injury can cause isolated lower motor neuron injury either at the nerve root or more distal. Such an injury produces a loss of motor function and sensation over a specific dermatomal area that corresponds with the level of the involved nerve root. Although nerve root damage from penetrating injury can involve more than one level, it can be differentiated from spinal cord injury by the fact that motor function and sensation below the level of injury are preserved. This condition may be confused with Brown-Séquard's syndrome of spinal cord injury (see below), which also involves unilateral sensory and motor function loss, but it only involves one or two levels of dermatomes and does not have a contralateral loss of position and vibratory sensation.

Spinal shock (not to be confused with neurogenic shock) is characterized by flaccidity and loss of reflexes after a spinal cord injury. Because spinal shock involves complete loss of neurological function, it can cause an incomplete spinal cord injury to mimic a complete cord injury. Spinal shock is a concussive injury to the spinal cord that usually lasts less than 24 hours. Return of the bulbocavernosus reflex may signal the end of spinal cord shock.[104] Neurogenic shock, which has already been discussed at length in the chapter on Circulation, refers to the shock state caused by loss of vasomotor tone and sympathetic innervation of the heart. The loss of vasomotor tone leads to vasodilatation with pooling of blood that produces hypoten-

sion. Loss of sympathetic innervation of the heart, if it is present, results in a lack of the normal tachycardia seen in other shock states.

Incomplete spinal cord injuries are associated with specific patterns of neurological involvement. Approximately 90% of incomplete spinal cord injuries are classified as one of three distinct clinical syndromes:

(1) Central cord syndrome,

(2) Anterior cord syndrome, and

(3) Brown-Séquard's syndrome.[104]

The most common of these syndromes in the general population is the central cord syndrome.[104] Because the central cord syndrome usually occurs in older patients with degenerative arthritis who have sustained a hyperextension injury, it is not the most common injury pattern found in military combatants (although it has occurred in older military personnel). In the central cord syndrome, the ligamentum flavum is thought to buckle into the spinal cord, injuring the central gray matter and the most central portions of the pyramidal and spinothalamic tracts.[104] This damage produces weakness, possibly accompanied by a variable degree of sensory loss, that is disproportionately greater in the upper than in the lower extremities. Central cord syndrome can occur with or without cervical spine fracture or dislocation.

The anterior cord syndrome usually results from a flexion mechanism that produces a spinal cord contusion or is the result of a protrusion of bony fragments or a herniated disk into the spinal canal. Injury, thrombosis, or laceration of the anterior spinal artery can also cause the anterior cord syndrome. On physical examination, this syndrome is characterized by bilateral paralysis and hypalgesia below the level of injury, with preservation of the posterior column functions of position and vibration sense. Anterior cord syndrome has the poorest prognosis of the incomplete injuries.

Brown-Séquard's syndrome, or hemisection of the spinal cord, is a rare injury in civilian settings. This syndrome usually results from penetrating injuries, especially stab injuries, but it can also occur following lateral mass fractures of the cervical spine.[104] Brown-Séquard's syndrome consists of ipsilateral loss of motor function and vibratory and position sense (posterior column) associated with contralateral sensory loss beginning one to two levels below the level of injury. As noted in the experience of the Russians during WWII, *"Neurologic violations with the puncture and cut wounds were frequently expressed in the Brown-Sequard Syndrome."*[35 (p.21)]

Spine Injury Treatment

Initial Management

The goal in the management of casualties with potential spinal cord injuries at or near the point of wounding is to prevent new primary cord injury and to minimize the effects of delayed secondary injury. Delayed secondary cord injury results from a cascade of autodestructive forces[105]

and is particularly common following blunt spine injury. Secondary cord injury can occur hours or even days after the initial injury and is responsible for much of the spinal cord damage that follows non-penetrating injury.

Although prevention and treatment of spinal cord damage is very important, it does the casualty no good if he or she dies with an intact cord; so the initial management of all such casualties must be on securing the airway and stopping all controllable blood loss. Management of the multiply injured trauma patient with potential cervical spine injury should proceed in an organized manner, following the ABCDE approach to trauma care as recommended by the American College of Surgeons' Committee on Trauma in the Advanced Trauma Life Support (ATLS) course [American College of Surgeons, Committee On Trauma. ATLS - Advanced Trauma Life Support Program for Doctors. 7th ed. Chicago, IL: American College of Surgeons; 2004]. Airway maintenance with cervical spine protection is the first step in the ABCDEs of the primary survey.

Concern about cervical spine injury should not delay performance of the primary survey or resuscitation. Such resuscitation should be initiated as soon as the need is identified, with due consideration for the possibility of spine injury. This is especially important when the mechanism of injury is a fall from a height or a motor vehicle crash.[36]

Airway Management

The prehospital care provider must identify the casualty whose airway is at risk. Combat casualties with potential cervical spine injury can have many reasons for airway compromise. Maxillofacial inju-

ries, foreign bodies (e.g., teeth); blood and secretions; cervical cord lesions; and associated head, neck, or chest injuries can all place the combat casualty's airway at risk. Initial airway management, which is often all that is required, should include basic maneuvers such as the chin-lift, jaw thrust, placement of a nasal or oral airway, and suctioning.[36]

As Chiles and Cooper note in their *New England Journal of Medicine* review of acute spinal cord injury, *"The most immediate threat to patients with injury of the cervical spinal cord is hypoxemia from hypoventilation or aspiration of gastric contents."*[106] Suderman et al. note that, *"...neurologic complications of intubation are rare provided that the unstable cervical spine is immobilized during establishment of the airway."*[107] And finally Apfelbaum et al. state that, *"The primary concern in managing [patients with penetrating neck wounds] has been control of bleeding and airway management."*[44]

Choosing the optimal airway management technique is often perceived as a clinical dilemma due to the belief that orotracheal intubation is hazardous in the presence of a cervical spine injury.[108] According to Rhee et al.[109] and Einav,[110] the most important considerations in deciding how to provide a definitive airway in a potentially cervical spine injured patient are operator skill and comfort with the procedure. These authors and others[107], [111], [112] have demonstrated that orotracheal intubation with in-line immobilization is a safe and effective method for definitive airway management. Gerling et al., using a cadaver model, showed no significant vertebral body movement during orotracheal intubation with manual in-line stabilization, although they did find that a significant amount of distraction occurred during orotracheal intubation with cervical collar

immobilization.[113] In addition, the authors report no significant difference in vertebral body movement when using different laryngoscope blades.

ATLS guidelines recommend orotracheal intubation with in-line manual cervical spine immobilization as the initial definitive airway procedure in the apneic patient. In the breathing patient who requires a definitive airway, the following sequence is recommended:

1. *Perform nasotracheal or orotracheal intubation*

2. *Perform orotracheal intubation with pharmacologic adjuncts if intubation is both required and impossible without such adjuncts*

3. *Avoid paralytic agents if at all possible, because patients who are paralyzed must be ventilated or they will die*

In trauma patients with potential cervical spine injuries who require intubation (a very rare circumstance in combat casualties), a surgical airway may have to be established when intubation cannot be accomplished by other means.[36]

Individuals providing care at or near the point of wounding should not delay transport to perform detailed clinical examinations or extensive stabilization. Patient evaluation and management should follow, or at least be concurrent with, resuscitation.

Airway management of patients with penetrating neck injuries is risky, even in the best of circumstances, and the point of wounding is *not* the best of circumstances. Numerous potentially life-threatening complications (e.g., severe hemorrhage

and inability to intubate secondary to distorted anatomy) can occur when advanced airway management is attempted on casualties with penetrating neck wounds. For this reason, it is recommended that intubation, or other advanced airway techniques, be attempted only in casualties who are

(1) Anticipated to have a long transport time and have some sign of airway obstruction such as stridor suggesting severe respiratory compromise and

(2) Apneic (note: an apneic and pulseless combatant casualty with a penetrating neck wound is dead and no procedures are indicated).

If evacuation can be done expeditiously, advanced airway management should be delayed until a more skilled provider with better equipment, support, and lighting is available. In such circumstances advanced airway procedures should be attempted only in the case of impending or full respiratory or cardiac arrest.

If indicated, and if the care provider is qualified, orotracheal intubation is the preferred route[99] because the airway can be visualized directly and there are fewer associated complications. It is also the technique that most care providers are trained and experienced in performing. If a casualty with a penetrating neck wound must be intubated, it should be done without neuromuscular paralytic agents if at all possible.

Cricothyrotomy may need to be performed when orotracheal intubation is unsuccessful, or is impossible[114-116] but it must be considered the final airway option in patients with penetrating neck trauma because of the risk of life-threatening hemorrhage.

Controversy exists about the performance of cricothyrotomy by prehospital personnel.[115, 117, 118] Mortality rates are high when prehospital cricothyotomy is performed on patients with penetrating neck trauma, but it is unclear whether this is a function of the experience (or lack thereof) of the provider or the degree of injury sustained by these patients If a surgical airway must be established, perhaps a safer technique would be percutaneous needle cricothyotomy, which requires little to no incision, and thus may reduce the risk of life-threatening hemorrhage.[119]

No studies address the safety and efficacy of this procedure when performed on casualties with penetrating neck wounds. Cricothyrotomy, with or without a needle, is risky and difficult, particularly when an anterior neck hematoma is present (presenting a high risk of catastrophic hemorrhage). Even bag-valve-mask ventilation (BVM) may be hazardous when used on patients with penetrating neck trauma. If there is injury to the airway, the positive pressure generated by BVM ventilation may cause dissection of air into the surrounding tissues, resulting in death from airway or vascular complications. BVM ventilation should be regarded as a temporizing measure until a more definitive airway is achieved.[99]

Immobilization

In civilian settings, ambulances generally carry the needed immobilization materials to the patient, but in combat, immobilization materials are carried to the patient on the back of the care provider. In the combat setting, therefore, a backboard is out of the question; no combat medic would, or should, carry a backboard into a combat environment, although a field-expedient backboard, such as a door, is a reasonable

option if available and clinically indicated. Even a stretcher may be unavailable at or near the point of wounding. Usually, the only question for the combat medic is whether or not to carry a cervical collar. An evaluation of the efficacy of using a cervical collar alone to stabilize cervical spine injuries has yet to be done. If a patient cannot be secured to a backboard (or at least a litter), it is unclear if there is any added value from applying a cervical collar.
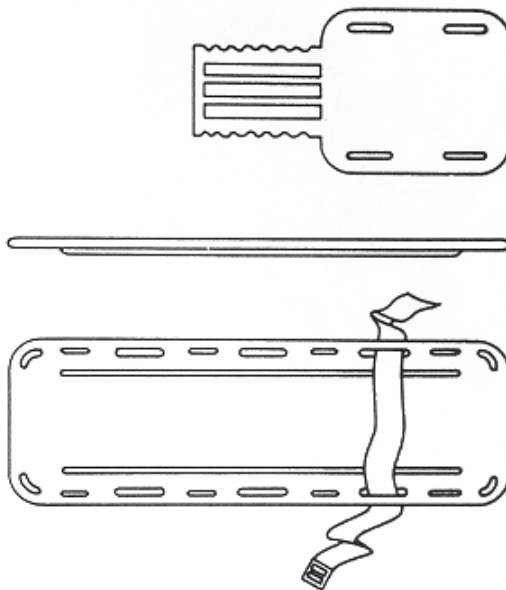
Spine Boards From United States Naval Hospital
Corpsman 3 & 2 Training Manual
NAVEDTRA 10669-C June 1989

The issue of spine immobilization at or near the point of wounding is somewhat contentious. That a small subset of casualties with blunt or penetrating neck trauma could benefit from spine immobilization at or near the point of wounding is indisputable. The issue is whether or not the civilian EMS model of immobilization for all patients with a *possible* spine injury applies in combat. At or near the point of wounding, immobilization is often impractical, if not impossible, and the poten-

tial benefits do not necessarily outweigh the risks/costs.

The Literature Supporting Immobilization

As noted by Hoffman et al.,[78] no attempt will ever be made to prove the efficacy and safety of prehospital spine immobilization with a randomized controlled trial because it is, *"unimaginable that emergency physicians would allow patients with known cervical spine injury to remain unrestrained."* Ethics certainly preclude *"allowing patients with [cervical spine injury] to bump around unprotected on an ambulance just to prove that it is or is not really dangerous,"[78]* but a study that would help determine whether the benefit of immobilizing *all* patients with potential spine injury outweigh the risks would be extremely useful. The underlying assumption is that the risk to patients from spine immobilization is so small that it would be unethical to conduct such a study. When applied to a combat scenario, however this assumption may not be correct, and only a study that assesses not just neurological outcomes but also overall morbidity and mortality from <u>all</u> causes will be able to answer this question.

The studies and other articles currently available on this topic are more anecdotal than rigorous, and when scrutinized carefully do not make the case with any degree of certainty. Routine immobilization of all patients with possible cervical spine injury is based on literature that warns of spinal cord injuries being sustained during the prehospital phase of care. Rogers, in his sentinel 1957 retrospective review of 77 patients with blunt cervical spine injuries, stated that, *"It is a sad commentary that in one in every ten patients symptoms of cord compression or an increase of cord symp-*

toms developed subsequent to the time of original injury - during emergency care, during the time when the diagnosis was being established, during definitive treatment, or following reduction."[120]

This comment is often quoted as the reason for the prehospital cervical spine immobilization practice that has become the standard of care. In fact, this article has served as a major reason for the universal practice of spine immobilization with rigid cervical collar, sandbags or taped block, and a long spine board.[120] It is interesting to note, however, that in Rogers' review, in all cases in which spinal cord damage occurred during treatment, the cord injury occurred either during surgical stabilization or following it, <u>not</u> as the result of prehospital care. Rogers describes not a single case in which spinal cord injury occurred during transport from an accident site or during treatment in an emergency department, yet his article is often cited as evidence of the risk of causing cervical spine cord injury during prehospital movement.[120]

Other articles, including those by Podolsky et al.,[121] Cloward & Netter,[122] and Geisler et al.[123] attribute delayed spinal cord injuries to improper prehospital handling, but fail to provide supporting data in their published reports. A 1977 review and epidemiologic study by Riggins & Kraus,[50] reported a 39% incidence of neurologic deficit for all cervical spine injuries, but they did not attribute these injuries to "improper handling during transport." None of this should be interpreted to suggest that spinal cord injury cannot occur, or even has not occurred, as the result of improper handling; it most certainly can and has. It is clear, however, that no evidence supports the assertion that this is, or ever was, a common occurrence.

Further complicating the debate is the role patients themselves play in minimizing their injury. In the same way that trauma patients instinctively hug their ribcage and breathe more shallowly to minimize internal injuries, or hold an injured arm close to the body for splinting, patients with spine injury may often be capable of protecting themselves from spinal cord damage.[124]

In a retrospective study by Hauswald et al., patients cared for in New Mexico, where prehospital cervical spine immobilization is standard practice, were compared with patients cared for in Kuala Lumpur, where prehospital cervical spine immobilization is not the standard. Types of injuries and patient ages were similar in both groups. Despite near universal spine immobilization in the United States, the odds ratio for disability was actually *higher*, after all independent variables were corrected for, for U.S. patients than for Malaysian patients (OR 2.03), corresponding *"to a <2% chance that immobilization has any beneficial effect."[124]* Although it is unlikely that immobilization actually causes spinal cord injury, this study at least serves to raise questions about our current practice.

The current practice of near universal spine immobilization for all trauma victims with suspected spine injury has evolved despite a lack of systematic analysis of the risks associated with immobilization or lack thereof. In civilian practice, the potentially disastrous consequences associated with failing to properly immobilize patients who have unstable spine injuries are viewed as far outweighing any other consideration. At the point of wounding in combat, however, immobilization has inherent risks that are not typically seen in civilian EMS.

Risks Associated with Immobilization at, or near, the Point of Wounding

Figure 3-13. Casualty lying on side.

From FM 21-10

As mentioned above, the realities of combat often preclude immobilization of casualties. Even when immobilization is possible under these circumstances, it may not be recommended. Immobilized combat casualties are often left unattended during evacuation or when attendants must turn their attention to protecting themselves and their patients from enemy fire. This places such casualties at risk for aspiration or impaired ventilation. Cervical spine immobilization may also conceal life-threatening injuries such as an expanding hematoma or blood loss.[30]

Aspiration is a risk in immobilized patients, particularly in those with some degree of altered mental status who are restrained in a supine position. Relatively recent studies reiterate the risks of aspiration,[125] and the need to have suctioning equipment on hand.[55] (p.364) If vomiting does occur, patients should immediately be placed on their sides, maintaining spine alignment, while suctioning is performed. In the case of immobilized patients who are intoxicated, Kirk & Pace[126] suggest that the backboard not be attached to the gurney so the patient can be turned on the board to aid in airway clearance. In short, immobilized patients must be constantly observed by a provider with the necessary skills and equipment needed to keep the airway clear. Unfortunately, in a combat setting, this is rarely possible so aspiration is a very real risk. Even in civilian settings, aspiration occasionally occurs as the result of immobilization.

Immobilization on a backboard has also been associated with impaired ventilation.[127] It has been clearly demonstrated that standard and appropriately applied spine immobilization devices can significantly reduce pulmonary function and respiratory capacity, even in healthy individuals.[128, 129] The supine position itself has been noted to have a detrimental effect on pulmonary function.[130, 131] Bauer & Kowalski note that, "…closer observation of patient ventilatory function while affixed to these devices is indicated. The common practice of leaving patients strapped to these [spine] boards while in the emergency center could hamper respiratory function."[128]

Unfortunately, at or near the point of wounding and throughout evacuation to definitive care, it is often not possible to keep immobilized casualties under close enough surveillance to ascertain whether their respiratory function is impaired. Furthermore, combat casualties often have multiple injuries, so it is not uncommon for an individual to have both a potential cervical spine injury and a lung injury with compromised ventilation. In cases of isolated cervical spine injury ventilation will be impaired if there is cord injury above C5.

Another way in which immobilization on a long backboard can cause harm is by creating pressure sores. This is not an insignificant problem, particularly for patients who have sensory loss as a result of spinal cord injury. Pressure sores not only add to the burden of injury, but are complex wounds that heal slowly.[132] Cordell et al.[133] note that several studies have associated use of the spine backboard with both

patient discomfort and the development of pressure ulcers.[134-137] Casualties immobilized and evacuated in combat could spend a considerable amount of time on a backboard, thus increasing their risk of pressure sores if they have spinal cord injury. Even as few as 2 hours spent on a spine board has been reported to cause pressure ulcers.[136]

Finally, lying on a backboard can be quite painful even if the patient has no injuries at all[133, 134, 137] It is quite possible that the pain associated with immobilization may become so severe that a patient with a spine injury may move around in an effort to get comfortable and might thus cause neurological damage that might not otherwise have occurred. Cordell et al. suggest that through this mechanism, spine boards could actually contribute to "antiimmobilization."[133]

In sum, the following guidelines should be followed at or near the point of wounding:

- Do not immobilize patients who don't need to be immobilized

- Safely remove immobilized patients from the backboard as soon as possible, <u>especially</u> if they have a known spinal cord injury

- If possible, lay an air mattress on top of the spine board to reduce pain and pressure that can lead to pressure sores[133] (this may not be feasible in a combat situation)

- Remove all hard objects, such as knives and other weapons, from the pockets of immobilized casualties, particularly in those who are paralyzed. Failure to do so may cause severe pressure sores.[120]

Steroids for Known Spinal Cord Injury

Steroids have long been used in hopes of limiting the extent of secondary spinal cord injury, despite the lack of supporting scientific data. The use of steroids in the treatment of neurotrauma was based upon their theoretical ability to inhibit lipid peroxidation, stabilize lysosomal membranes, and modify edema production.[138] Based upon the results of the Second National Acute Spinal Cord Injury Study (NASCIS 2), the current civilian recommendation is to treat patients with *nonpenetrating* spinal cord injury with high-dose methylprednisolone within the first 8 hours of injury.[139]

This protocol was derived from a study done by Bracken et al., who found that patients treated within 8 hours of injury with methylprednisolone, 30 mg/kg intravenous bolus given over 15 minutes, followed by a 45-minute pause then a 5.4 mg/kg/hr infusion for 23 hours, showed significant neurological improvement at 6 weeks, 6 months, and 1 year when compared with patients treated with naloxone or placebo.[139, 140]

But even this government recommendation has been recently challenged.[141-143] Although it may be reasonable to apply this recommendation to combat casualties with nonpenetrating spinal cord injuries, it is unclear whether, in total, such a recommendation would produce the best patient outcomes in this patient population, in a combat setting; and there is no evidence to support the use of high dose steroids in casualties, civilian or military, who have cord damage from penetrating injury.

A high-dose, short course of corticosteroid appears to have no documented serious side effects but, as noted in a recent re-

view of the effects of single-dose gluco-corticoid administration, data are only available from small-scale heterogenic studies.[144] No large-scale studies of standardized surgical procedures have been done that could establish the safety of glucocorticoid administration in situations that might have a higher risk of wound and infectious complications (e.g. combat casualties).

Evacuation

Few patients with penetrating injuries of the spine need to be evacuated directly to a facility with neurosurgical capabilities because experience has borne out that most such patients, particularly if the injury involves the cervical spine, have other injuries that are more immediately life threatening. Neurosurgical care is rarely available within a 1-hour evacuation time, so patients needing urgent surgical intervention should be taken to the closest facility with resuscitative surgical capability. It is only appropriate to evacuate directly to a neurosurgeon when

(1) No associated life- or limb-threatening injury is present (rarely possible to establish at or near the point of wounding given the absence of advanced diagnostic capability or prolonged observation) or

(2) There is a neurosurgeon at the nearest resuscitative surgery facility.

During evacuation, immobilized casualties must be attended by a care provider who is capable of and properly equipped to maintain the airway. If the evacuation is anticipated to take longer than 1 hour, especially if the casualty is paralyzed, the risk of pressure ulcers can be reduced by pad-

ding the backboard and by shifting the patient's weight periodically by tilting the board from side to side.

Care of the Casualty with Head Trauma

Initial Management

As in the management of patients with spine injuries, initial management of patients with both blunt and penetrating head trauma should focus on immediate life threats, e.g. airway, breathing, and circulation. Absent any obvious exsanguinating hemorrhage, the first concern should be the airway.

Often casualties with penetrating head injuries, and some with blunt head trauma, have maxillofacial injuries that involve the airway. Even when there is no direct damage to the upper airway, the airway of head-injured casualties is at risk. Such patients quite frequently have altered mental status and, particularly when in the supine position, they are at risk of airway obstruction. Obstruction can occur from prolapse of the tongue and/or aspiration of vomit, blood and tissue. Wounds to the head and face are common causes of airway obstruction in combat casualties. In both civilian and military combat settings head injury is the leading indication for intubation in a trauma setting (see Airway chapter, above).[145, 146]

The airway management recommendations for head and face injured casualties such as those provided by Jolly during the Spanish Civil War[147(p.137)] and by Beecher and others during WWII[148 (p.982)] remain valid today. Specifically, casualties with serious head injuries should be transported in the lateral recumbent or prone position with the head turned to the side. If it is

necessary for any reason, such as cervical spine immobilization, to transport such casualties in a supine position, an attendant with ready access to suction and skilled in airway management must be present throughout the transport.

Early and adequate ventilation and oxygenation are particularly critical for head-injured casualties. With gunshot injuries to the head, fatal apnea often occurs immediately after injury as energy is transferred from the bullet to the brain. Available evidence suggests that intracranial shock waves caused by high-velocity bullets can cause brain-stem compression and thus interfere with the cardiorespiratory function of the medulla oblongata.[21, 28, 29, 149-151] It is therefore likely that, even under ideal circumstances in which a casualty with penetrating brain injury is immediately attended to and rapidly evacuated, survival is unlikely, even if the initial injury was not otherwise lethal.[152, 153] The evidence from Korean War casualties certainly suggests that this is the case.[16 (p.44)]

It is essential that <u>all</u> controllable hemorrhage be controlled. The critical influence of cerebral perfusion pressure on outcome in brain-injured patients is discussed in detail below. A difficult balancing act must be carried out when treating multiply injured combat casualties with head injury and internal bleeding. Although uncontrolled internal hemorrhage is increased when intravenous fluids are administered, which is why aggressive fluid resuscitation is not recommended in most such circumstances, more aggressive fluid resuscitation may be necessary when serious head injury is present. As discussed later in detail, it is important to maintain cerebral perfusion pressure even at the risk of increased internal bleeding. Even bleeding from scalp wounds should be aggressively controlled because blood loss from these

injuries can be significant over time. Scalp injury is present in a significant percentage of head-injured casualties.[17]

Blunt Head Trauma

Although the preceding discussion of the epidemiology of head injuries in combat casualties focused almost entirely on those with injuries involving scalp lacerations or intracranial penetration, an increasing percentage of combat casualties are sustaining blunt closed-head trauma. It was noted during World War II that

> *"Emphasis, rather naturally was upon penetrating head wounds as compared with closed head injuries. It soon became evident, however, that in a mechanized army, closed injuries and the factor of blast could not be casually dismissed as potential causes of cerebral trauma."[154(p.91)]*

This statement is even more relevant today. As the number of penetrating injuries has decreased in American troops during combat, the frequency of blunt injuries has increased. Increased insertion of troops by parachute, fast roping, and helicopter or other aircraft, and more urban combat all contribute to an increased risk of closed head injury.

Closed-head injury ranges from minor concussion, with transient or no loss of consciousness (LOC), to severe intracranial injury, and death. The challenge today is for military prehospital care providers to rapidly and effectively triage and treat all patients within this spectrum. Any blunt head injury that produces LOC, no matter how minor or brief, produces some degree of cerebral pathology (as imaging technology improves, more of this pathology is being identified). The great majority of such patients suffer either minor, or no

demonstrable, long-term consequences. Those with persistent symptoms probably have sustained a cerebral contusion from a contracoup mechanism.

In patients at both ends of the spectrum of severity of injury, evacuation for the purpose of rapid treatment is typically not indicated. Evacuation of patients with minor head injury generally has no effect on their outcome, which is almost always good.

At the far end of the spectrum of blunt brain injury with no associated intracranial bleeding is diffuse axonal injury (DAI), which may range from mild to severe. DAI is present to some degree in many patients with severe head trauma, and is almost always the result of an injury in which rotational acceleration has created high shear forces on the brain parenchyma. Patients with DAI are generally those who were rendered unconscious at the moment of sudden injury and in whom the pathological changes induced by these shear forces are not identifiable as a mass lesion on head CT.[155]

The duration of DAI-associated LOC may range from 6 to 24 hours in its mildest form (15% associated mortality) to a permanent comatose/vegetative state (or death) in severe DAI (>25% mortality with a poor prognosis in virtually all cases).[156] Despite, or perhaps because of, the poor outcomes associated with DAI, patients with DAI typically benefit little from early evacuation to a neurosurgeon.

Patients who benefit most from accurate, far-forward diagnosis and appropriate treatment are those with focal intracranial bleeding, especially those with epidural bleeding, Epidural bleeding usually occurs in association with a temporal bone fracture and is caused by rupture of the middle

meningeal artery. This can occur even when there has been relatively little energy transfer to the brain. Because epidural hematomas are caused by arterial bleeding, they expand rapidly and cause death if not treated quickly. Conversely, if quickly diagnosed and treated, such patients typically do well because the brain itself usually sustains no serious damage.

Other forms of intracranial bleeding are cerebral, subdural, subarachnoid, intracerebral, and intraventricular bleeding. Unlike epidural hematoma, these other forms of bleeding usually indicate that the brain has been subjected to a high energy force. Even with appropriate initial treatment and rapid evacuation to skilled neurosurgical care, patients with such injuries often do poorly. In the general civilian population, patients with epidural hematomas make up only about 0.5% of all patients with closed head injury and about 1% of those presenting in coma.[156(p.308)] It can be calculated that approximately 10% of this same group sustain subdural hematomas.[156, 157]

The much higher percentage of elderly, alcoholic, and chronically ill patients in the civilian population, as compared to the military combatant population, increases the relative percentage of patients with subdural and other types of intracranial bleeding. Thus the probability that a combatant with blunt head trauma and intracranial hemorrhage will develop an epidural hematoma is higher as compared to the general civilian population. For this reason, in a military population, the percentage of patients with epidural hematoma would be expected to be several times higher than that seen in civilian series; but still quite rare nonetheless.

It should be clear from the preceding that the primary focus of military prehospital

personnel caring for combat casualties with blunt head trauma must be on

(1) Immediate resuscitation, and then

(2) Early identification and evacuation of patients who have neurosurgically correctable lesions, especially those with epidural hematoma.

Prevention

Despite considerable advances in the diagnosis and treatment of patients with neurological injury, after such injury has occurred, there is often little beyond palliative care that can be provided. The central nervous system, once damaged, heals very slowly, if at all. Therefore, the focus of medical efforts in the realm of central nervous system injuries must be on prevention.

Kevlar Helmet

During World War II, a significant number of deaths resulted from motorcycle accidents. A sizeable percentage of these deaths were either solely, or in part, caused by head trauma. As the result of a recommendation to the British Army Medical Research Council in 1941 by Sir Hugh Cairns, the use of crash helmets by British Army motorcyclists was made compulsory. A study published in 1943 by Cairns & Holbourn documented a dramatic decrease in motorcycle fatalities following the implementation of this recommendation.[158] A similar aggressive head-injury prevention policy needs to be followed today in all areas where head trauma is reasonably likely.

Considerable advances have been made since World War II in head-injury prevention, but there is still far to go. The Kevlar helmet represents a significant improvement over the steel helmet in preventing penetrating injury from fragments and other low-velocity projectiles. The former can even defeat some high-velocity projectiles in some circumstances. In general, however, high-velocity assault rifle bullets will penetrate the standard Kevlar helmet and, contrary to popular belief, when penetration does occur, the velocity of the bullet is only minimally reduced as it moves through the helmet. When a bullet penetrates a Kevlar helmet, the resultant head injury may be as bad, or even worse, than if a helmet had not been worn at all because the bullet retains most of its energy as it penetrates but the full metal jacket may be deformed. Despite this fact, because the primary risk in most battlefield situations remains fragments helmets should always be used on the battlefield.

Overall Kevlar helmets are quite efficient at reducing the risk of penetrating injury, particularly lower velocity injury, but they

are much less effective against blunt injury. Unlike a motorcycle helmet that reduces intracranial injury by deforming when force is applied, thus dissipating energy, the Kevlar helmet is quite rigid (which allows it to defeat penetrating trauma) so it does a poor job at reducing the transfer of blunt energy.

In the past some special operations troops used a crushable, bicycle-type, helmet, rather than the standard Kevlar helmet, for some close combat situations because the greatest risk for head trauma was felt to be from blunt trauma and high velocity bullets, which Kevlar does not usually defeat. The combat deaths of some of these elite SOF troops from penetrating head wounds while wearing these bicycle-type helmets led to a change to a ballistic helmet. What is clearly needed, especially for troops with an increased risk of closed-head injury, e.g. airborne and special operations troops, is a helmet that combines both ballistic protection and protection from blunt force injury. Such helmets are already used by aviators who also face the dual threat of penetrating and blunt head trauma. Airborne troops use soft foam inserts inside the Kevlar helmet to reduce the risk of closed head injury during airborne operations; although these certainly help, a better helmet would further reduce the risk.

No improvements in helmet technology, however, will address the main problem, which is compliance. Troops frequently cite a long litany of reasons why they don't wear the helmet they are provided. These include heat load, weight, perceptions of decreased situational awareness due to decreased ability to hear, and a desire to blend in with the local population and to appear less "threatening." Measures taken to address these concerns would also help

reduce the problem of CHI in combat troops.

Diagnosis

Maj. Douglas Jolly's 1938 notes from the Spanish Civil War regarding the difficulty of accurate and far-forward diagnosis in head trauma are still true today. He stated that

> *The subdivision of head wounds into two clear-cut groups - those in which operation can, and those in which it cannot, be recommended - is unattainable even by those with an extensive experience in war surgery...All that the discriminating surgeon should allow himself to say of the most severe head injuries is that they do not merit priority treatment; but they should unfailingly be reviewed when there is some respite from the rush of casualties.*[147(p. 84)]

Those who provide care at or near the point of wounding must determine which head-injured patients must be rapidly evacuated and which do not need to be evacuated. As a general rule, all patients with a head injury, except those with unequivocally minor scalp injuries, should be regarded as having a potentially life-threatening injury, and none, except those having an injury that is obviously incompatible with life, should be treated as if they were unsalvageable.

Head injury in itself is not always an indication for evacuation, but today it is impossible to definitively rule out serious head injury at or near the point of wounding. While this might be interpreted to mean that every patient with any head injury should be evacuated the contingencies of the battlefield would certainly not allow this. What must happen is that easy

to use, durable, and light weight diagnostic tools must be developed and fielded for far forward use as soon as possible. Absent this the forward care provider must use common sense, knowledge of the epidemiology of serious head injury, and the basic assessment tools of history and physical examination to make these difficult triage decisions.

Most of Jolly's observations about the management of casualties with head wounds during the Spanish Civil War remain relevant today. He noted that, *"Attempts at prognosis based on the course of the projectile through the cranium are liable to serious error....*[147(p. 85)]*"* and that, *"...no scalp wound is so trivial that it should not be regarded as potentially serious."*[147(p. 84)] His admonishment that, *"Blind groping for foreign bodies with forceps can hardly be condemned too strongly"*[147 (p. 97)] should be strictly observed in all wound cases, but especially in the case of penetrating head wounds.

When confronted with a casualty with blunt head trauma at or near the point of wounding, the key question is, "Must the casualty be evacuated, and, if so, how urgently?" In the patient with a serious head injury, particularly one with blunt head injury, making the correct decision is essential. It has been shown that patients with blunt head trauma who require and receive surgery within 4 hours of their injury have a mortality rate that is three times lower than those who need surgery but do not receive it for more than 4 hours after their injury.[159]

For the patient with penetrating head injury, the evacuation decision is generally both less difficult to make and less critical. It is usually easier to determine that a casualty has sustained a penetrating head injury than it is to diagnose an early sub-

dural or epidural hematoma caused by blunt trauma. For this reason, the primary focus of the following discussion will be on the assessment and disposition of the casualty with the closed head injury. When it is unclear whether a penetrating head wound is present, experienced military neurosurgeons agree that obtaining an immediate definitive diagnosis is unnecessary because such patients are not likely to deteriorate en route.

During combat operations in Mogadishu Somalia on 3 October 1993 a combatant with a small, lightly bleeding head wound of unclear cause presented to a casualty collection point and was quickly neurologically assessed by a physician. He was found to be neurologically intact, asked to be returned to combat, and was allowed to do so. A few days after the battle this individual returned again for medical evaluation complaining of a headache and clear fluid draining from the wound. A radiograph of the head was obtained that revealed a small fragment approximately 6 cm inside the brain. The casualty was started on antibiotics and was evacuated. During evacuation the patient seized and developed a brain abscess, but eventually recovered with good neurological function (personal communication with Dr. Rob Marsh, Special Operations Surgeon). This case demonstrates that although early evacuation is optimal, and certainly all patients with a known or reasonably suspected penetrating head injury should be evacuated to competent neurosurgical care, such patients will often do well even if there is a delay.

Because minor blunt head trauma, with and without transient LOC, is fairly common in military populations in both peacetime training and in war, it would be extremely useful to be able to quickly sort out those who need the prompt services of

a neurosurgeon from those who do not. As the U.S. military becomes increasingly smaller and more specialized, over-evacuation (evacuating casualties who do not need to be evacuated) must be limited. There is no piece of equipment and no exam that far forward medical personnel can use to quickly and accurately make good triage and evacuation decisions for head-injured casualties. There are, however, some things that can improve decision making in such circumstances.

Demographically most combatants are quite similar to high school and college athletes, so it is reasonable and appropriate to examine how athletes with closed-head injuries are evaluated on the field of play to guide a similar evaluation of casualties with closed head injury at or near the point of injury.[160] Although serious head injury is rare in organized athletics, it does occur, and minor head injury is quite common, particularly in contact sports such as football. In the United States, there are, on average, eight deaths each year as a result of football-related head injury, most which are caused by an acute subdural hematoma.[161 (p913),162]

More than 200,000 minor head injuries occur during organized football each year,[163, 164] and 70% of American football players who are "knocked out" return to play the same day.[161 (p913)] Returning to combat is also what most likely happens to the average combat casualty who suffers a brief loss of consciousness from a closed head injury. Unfortunately, as in athletics, a combatant who is rendered unconscious may have only a mild concussion and can safely return to the field, or he may have a developing intracranial hematoma or other intracranial process that may result in death or permanent neurological injury.[165] The question of whether it is possible to effectively and

efficiently differentiate between these two types of injury at or near the point of wounding is the focus of the following discussion.

Although there appears to be no difference in severity of head injuries between groups based on sex or race, it does appear that age may be a factor. Jennett noted that victims over the age of 60 are four times more likely to have an intracranial abnormality on CT than their [166] Mostly this information is useful because it indicates that the risk of serious head injury for a given degree of trauma is lower in population of military combatants than would be the case in a civilian population that includes elderly patients. Most intracranial injuries result from unprotected trauma involving large forces. In the civilian setting, pedestrians and bicyclists struck by automobiles have the highest potential for serious intracranial injury.[167] This only suggests the obvious, i.e. that those sustaining higher energy blunt head injury have the highest probability of serious intracranial injury.

Information about mechanism of injury is useful at or near the point of wounding to risk-stratify head-injured combat casualties. It is possible to ascertain that patients with high energy mechanisms of injury will be at higher risk of serious intracranial injury, but it is not possible to achieve a high degree of sensitivity or specificity with this information. Some casualties sustaining high-force impacts to the head, for example, will have minor or no injury and some who have sustained apparently low-force impacts will later be found to have serious intracranial injury.[166, 167]

Knowing whether a casualty with blunt head trauma has had loss of consciousness (LOC) is one of the most valuable pieces of information in helping to make triage,

management, and evacuation decisions for head-injured patients. It is commonly thought that casualties with blunt head injury but no LOC have a very low probability of serious intracranial injury and probably do not need to be evacuated. As pointed out by Cheung & Kharasch, however, this has not been proven; no study has specifically analyzed the negative predictive value of LOC.[167] In an evaluation of patients with a GCS of 15 and LOC, the incidence of identifiable intracranial pathology ranged from 6.1% to 9.4%.[167] Most studies of the incidence of intracranial injury in patients with LOC do not address the duration of LOC as a variable. When this variable is considered, increasing duration of LOC is associated with increasing incidence of intracranial lesions.[169] Miller et al. noted an increase in intracranial injury on CT from 5.9% in patients with LOC < 5 minutes to 8.5% in those with LOC >5 minutes,[170] confirming the earlier findings of Rivara et al. in a pediatric population.[171]

Many if not most intracranial lesions identified on a CT scan following blunt injury do not require surgery and cannot be significantly improved with any form of medical intervention. The number of casualties who would benefit from rapid evacuation to a neurosurgeon following a closed head injury, then, is quite low, and it is identification of this small group of casualties that presents the challenge for point-of-wounding care providers.

The available data appear to support the widely held belief that the longer the LOC, the higher the likelihood of serious intracranial pathology and the greater the need for rapid evacuation of the casualty to the care of a neurosurgeon. It is important to note that the above discussion only relates to the evaluation of casualties with blunt head injury (a significant *minority* of combat-related head injuries) and should *not* be applied to casualties with penetrating head wounds. Unless intracranial penetration can be ruled out all patients who may have sustained a penetrating head wound need to be evacuated to a facility that can rule out intracranial pathology.

It would be useful if the presence or absence of such symptoms as headache and nausea and vomiting could be effectively used to raise or lower the clinical suspicion of an intracranial abnormality. Although the presence of these symptoms often influences emergency department decisions such as whether to obtain a CT scan, such findings are neither sensitive nor specific enough to predict intracranial abnormality. The presence of headache, and nausea and vomiting has not been shown to be associated with an increased risk of

(1) A surgically correctable intracranial lesion,
(2) New-onset, post-traumatic seizures, or
(3) CT-demonstrable intracranial pathology.[166]

In terms of physical examination, the Glascow Coma Scale (GCS) is the most widely used system for grading the neurological status of trauma victims. The GCS is relatively easy to calculate and has been shown, in civilian trauma settings, to be reproducible with low inter-observer variability. When calculated by prehospital care providers in the heat of combat, however, the inter-observer variability of the GCS, however, is likely to increase dramatically, making the test much less reliable in this setting. Even with low inter-observer variability, the GCS is not much help in answering the most important

question, "Does this patient need to be evacuated?"

Another problem with the GCS is that most studies that have evaluated its predictive value have not addressed the important question of timing. A GCS obtained in an unconscious patient immediately after injury certainly has a different predictive value than a GCS obtained in the same patient an hour later.[172, 173] It would be very helpful if it were possible to simply say that any casualty with a closed head injury who has a GCS of 15 does not need to be evacuated and can be safely returned to duty – unfortunately it is not.

Unfortunately, even a perfect GCS of 15 does not exclude significant intracranial abnormalities. The incidence, in the published literature, of CT-demonstrable intracranial injury in patients with a GCS of 15 varies between 2.5% and 22.5%.[166] The high of 22.5% originates from the neurosurgical literature and thus suffers considerably from selection bias, i.e., only those with a significant problem are referred to a neurosurgeon so patients with a GCS of 15 and no intracranial pathology never get into the neurosurgery database. If only emergency medicine studies are considered, a much lower incidence of 2.5% to 9.8% is found.[166] The relevance of this information to combat casualty management is hard to ascertain because surgically correctable lesions were not differentiated from other CT abnormalities: reported lesions varied from surgical epidural hematomas (urgent neurosurgery required) to simple linear skull fractures (no surgery needed).

In the largest study assessing the frequency of CT abnormality in head trauma patients with a GCS of 15, Shackford found that 14.8% of 2,166 such patients

had an abnormal CT but only 3.2% required a craniotomy.[159] Miller et al., in the largest emergency medicine series of head-trauma patients with a GCS of 15, reported that 6.1% of 1382 patients had an abnormal CT but only 3 (0.2%) required surgical intervention; all three having obvious depressed skull fractures.[170] In another study of emergency department head trauma patients with a GCS of 15, Jeret et al. found a 9.4% incidence of CT abnormality but only 0.3% (2 of 712) required surgery, one of whom died.[174] It would therefore seem that the percentage of head trauma patients presenting to an ED or battalion aid station with a GCS of 15 but still requiring neurosurgical intervention is very low -- but not zero. Although a GCS of 15 does not exclude a CT-demonstrable lesion (overall negative predictive value of 90.7%), there is clearly an increased risk of serious brain injury with lower GCS scores.

In every study to date there has been an inverse relationship between GCS score and frequency of CT-demonstrable intracranial abnormalities. This information is of little use in making evacuation decisions for head-injured patients because all patients with a low GCS score are generally evacuated as soon as possible in any case and the GCS is not useful in deciding what to do with the much larger number of surviving head injury casualties who have a GCS of 15.[166]

Physical Findings

Attempts have been made to establish a relationship between anisocoria, external signs of trauma, and neurologic findings and the presence of significant intracranial abnormalities.[175] But, as Cheung & Kharasch point out, few studies have looked at these types of exam findings as independent variables, so it is difficult to

arrive at a definitive conclusion regarding such a relationship.[167]

Between 30% and 80% percent of head injury patients with anisocoria have been found to have demonstrable intracranial injury, [167, 171, 175] and the incidence of intracranial abnormalities has been found to increase with the degree of anisocoria.[175] In a study by Borczuk of 210 head trauma patients with anisocoria greater than 1 mm, only about one-third had an intracranial abnormality,[169, 176] so although there appears to be a reasonably high probability that a head trauma patient with anisocoria will have an intracranial abnormality, a sizeable number of head trauma victims with this finding will not. Signs suggesting basilar skull fracture such as "raccoon" eyes and hemotympanum have been associated with a 53%-90% incidence of intracranial pathology.[171, 176]

Even if a casualty with blunt head injury and history of LOC has regained full consciousness and does not have a neurosurgically correctable lesion, a cerebral concussion has nonetheless been sustained that is likely to produce some degree of confusion and amnesia. Casualties with such injuries may exhibit easy distractibility, poor vigilance, inability to maintain a coherent stream of thought, inability to carry out a sequence of goal-directed actions, delayed verbal responses or slowed actions, disorientation, slurred or incomprehensible speech, problems with motor coordination, emotional lability, and/or short-term memory deficits. This confusion and memory disturbance can be immediate or delayed, so returning such casualties to important duty positions is problematic.[177]

The classic clinical picture of a patient with an epidural hematoma is transient LOC at the time of the injury, followed by a normal level of consciousness (lucid interval) over a variable period. This is followed by a decline in mental status, with a unilaterally (usually ipsilateral to clot) dilated pupil, decerebrate posturing, contralateral weakness, and often, unless treated quickly, death. Although this sequence is considered characteristic of epidural hematoma, only about a third of patients with an epidural hematoma present in this manner .[178]

Acute subdural hematomas occur approximately three times more frequently in athletes than epidural hematomas.[177] A similar relative frequency of these conditions would be expected in combat troops because they are demographically a similar population although exposure of combat troops to potentially much higher forces may affect the relative frequency of these conditions. In the general population that has sustained head trauma, subdural hematoma occurs almost twenty times more commonly than epidural hematoma.[156(p.308)]

The reason for this difference between athletes (and presumably combatants) and the general population is because the general population includes young children and the elderly both of whom have a relatively lower rate of occurrence of epidural hematoma. This has been attributed to there being a closer adherence of the dura to the skull in these two groups and due to skull elasticity in young children.[156(p.308)] Furthermore, because of brain atrophy, subdural hematomas are also much more common in the elderly than in young adult combatants and athletes. Because there is less subdural space within which blood can collect in young adults, in them when subdural hematoma does occur there is generally less pooling of blood in the subdural space and there is a higher degree of underlying brain injury. For this reason

the symptoms associated with acute sub-dural hematoma in younger adults are more often related to the underlying brain injury rather than to the mass effect of a clot so in this group an operation is less likely to be beneficial.[178]

The combatant with an acute subdural hematoma usually has prolonged uncon-sciousness and a focal neurological find-ing, such as pupillary asymmetry or de-cerebrate posturing so the evacuation de-cision is not difficult. Such casualties should be evacuated to a neurosurgeon as quickly as possible.

The above information is important be-cause it makes it clear that in combatants who have sustained serious head injury epidural hematoma is relatively much more common than would be the case in the general population; this has disposition and management implications. Compared to a civilian population, combat casualties with serious head injury are much more likely to benefit from rapid evacuation to a neurosurgeon.

One condition that has been well de-scribed in athletes engaged in contact sports is a syndrome of massive cerebral edema and death that follows relatively minor head trauma occurring shortly after a previously similar "minor" head in-jury.[179, 180] This rare condition typically presents in an athlete who, while still symptomatic from a previous "minor" head injury, returns to play before com-pletely clearing his sensorium and then suffers a second head injury. This syn-drome, believed to be caused by persisting cerebral vascular sensitivity, is called the "*second impact syndrome.*" A second injury then leads to cerebral autoregula-tory dysfunction, vascular congestion, and subsequent intracranial hypertension. This condition, which can follow minor head

injury without loss of consciousness, has resulted in sudden death.[181]

Unfortunately there is no evidence to sup-port the notion that a normal neurological examination can accurately identify those head injured patients who have minimal risk of serious intracranial injury.[166, 181, 182] Despite there being no good evidence to support a point-of-wounding policy of returning to duty most soldiers with blunt head trauma, a GCS of 15, and a normal neurological examination, such a policy is probably still reasonable in the absence of better far-forward diagnostic capability.

It is important to note, however, that, as Cheung and Kharasch have pointed out,[167] most studies simply do not address the question of whether such emergency de-partment patients can safely be sent home. They focus instead on predictive factors such as GCS < 15, focal neurologic defi-cits, and skull fractures. This is of little use to the care provider at or near the point of wounding, who will feel com-pelled to evacuate any head-injured casu-alty to someone who can rule out intracra-nial injury. In such a circumstance, the evacuation decision will be made primar-ily upon the tactical situation and the availability of transport. If both preclude rapid evacuation and if, after some period of observation, the patient appears to re-main well, the care provider will likely return him or her to duty. On the other hand, if evacuation is available and the tactical situation permits, the care provider will likely evacuate the casualty.

Treatment

In the initial resuscitation of a combat casualty with a potentially serious head injury, the emphases should be on

  (1) Ensuring an adequate airway and adequate ventilation, and
  (2) Controling hemorrhage.

Although the scalp can bleed significantly, and can contribute to the development of shock, scalp wounds in adults are rarely the sole cause of hemorrhagic shock. It is also true that serious brain injury can produce significant hypotension without concomitant loss of intravascular volume. Despite these facts, any patient with a serious head injury who is also in shock should be assumed to have another source of bleeding (usually internal) until proven otherwise. It should also be assumed that altered mental status in a combat casualty is due to head injury and is <u>not</u> due to blood loss until proven otherwise.

Except in the case of severe shock, altered mental status (aside from anxiety) is rarely present in previously healthy young combatants without head injury or hypoxia. In discussing the management of head injured combat casualties, during the Spanish Civil War, Jolly makes the still very relevant observation that, *"Time spent in the systematic prevention and control of bleeding is not lost."*[147(p89)] It is absolutely critical to the casualty with a serious head injury to maintain adequate blood pressure, hematocrit, and oxygenation and ventilation.[183] All controllable bleeding should be stopped; the airway kept patent; and, if available, supplemental oxygen should be provided (although achieving a supranormal oxygen level provides no benefit). The mean arterial pressure must be kept greater than 70 mm Hg to ensure adequate cerebral perfusion pressure.

In managing the head-injured casualty at or near the point of wounding, the objectives must be to determine who needs urgent evacuation to the care of a neurosurgeon and who can be safely returned to duty. Until there is an effective way, in far-forward situations, to sort out which casualties have sustained serious intracranial injury, there should be a low threshold for rapidly evacuating such casualties when circumstances permit.

Recently developed guidelines have been published[185, 186] to minimize the use of head CT for patients with minor head trauma. These guidelines could be used to help make appropriate forward area triage and evacuation decisions at or near the point of wounding, but they have not been evaluated for their effectiveness at reducing CT use even in a civilian population. Several of the criteria are not generally relevant to military combatants, such as "age over 60" and drug or alcohol intoxication. Most of the others are so common following head injury that they are not likely to be very effective at reducing head CT use and would result in substantial over-evacuation if used solely to make evacuation decisions in head injured combatants. These include

  (1) Headache,
  (2) Vomiting,
  (3) Deficits in short-term memory and,
  (4) Physical evidence of trauma above the clavicles.

The remaining criteria of seizure following head trauma would likely result in evacuation even without implementation of a published guideline. This despite the fact that there is no proven correlation between the presence or absence of new-

onset, post-traumatic, seizures and CT-demonstrable intracranial pathology.[167]

The Canadian CT Head rule has more stringent criteria than those described above and may be more useful in making evacuation decisions and reducing over-evacuation.[187]

Canadian Head CT Rule: Selected Criteria
GCS score < 15 at 2 hours after injury
Suspected open or depressed skull fracture
Any sign of basal skull fracture (hemotympanum, 'raccoon' eyes, cerebrospinal fluid otorrhea, Battle's sign)
More than two episodes of vomiting
Retrograde amnesia > 30 minutes
Significant mechanism of injury (pedestrian struck by motor vehicle, occupant ejected from motor vehicle, fall from height > 3 feet or five stairs)
Age > 65 years[186] [Stiell, I. G., G. A. Wells, et al. (2001). "The Canadian CT Head Rule for patients with minor head injury." Lancet 357(9266): 1391-6]

In combat, dealing with the issue of the second-impact syndrome is problematic because there is simply no way to minimize the risk of recurrent head injury in most combatants who are returned to duty. When circumstances permit, casualties sustaining minor head injury should be observed for a period of time and only returned to duty when they are absolutely symptom-free, or when their duties make recurrent head injury very unlikely.

Although the circumstances of combat will always be a major consideration in the evaluation, management, and disposition of head-injured casualties the following approach, derived from recommendations for on-the-field management of head-injured athletes should be followed whenever possible:

- Any combatant who has received a blow to the head or any significant acceleration-deceleration-type force to the head should be considered to have sustained potentially serious head injury and should be thoroughly evaluated for level of consciousness, steadiness of gait, orientation, post-traumatic amnesia, and retrograde amnesia.

- Casualties who have sustained only a grade 1 concussion (no LOC or post-traumatic amnesia of < 1 hour duration) should be observed for 20–30 minutes. If there is complete clearing of the sensorium and no residual symptoms, such casualties can reasonably be returned to duty.

- Circumstances permitting, casualties with persisting symptoms, such as headache, dizziness, or confusion, should be evacuated for evaluation by a physician.[188]

- All casualties with a grade 2 concussion (< 5 minutes loss of consciousness, amnesia < 24 hours) should be evacuated for evaluation by a physician.

- Casualties sustaining a grade 3 concussion, where there is prolonged or severe alteration in level of consciousness or orientation, or a focal neurological deficit should be evacuated, when possible, directly to a facility capable of performing a CT-directed craniotomy.

Because of the "second-impact syndrome," casualties with minor head injury present a dilemma because most in this category do fine if returned to duty but a very small number who sustain a second minor head injury shortly following the first may die. For this reason, when possible, no combatant who has sustained a mild concussion should be returned to duties that have a relatively high risk of recurrent head injury for at least 1 to 2 weeks.[181] Symptoms such as persistent headache, irritability, fatigue, dizziness, double vision, impairments in memory and concentration, and/or problems with behavior should lead to neurological or neurosurgical evaluation.[177] Repeated "minor" head injuries have an additive effect on cognitive abilities,[164, 189-193] so any combatant with recurrent head injury should be neurologically evaluated.

Steroids do not appear to offer any benefit in the management of acute head injury.[194] Because free radicals are thought to play a major role in producing secondary injury, free radical scavengers have been evaluated for possible use in the treatment of head injured patients. Although there have been some promising results from an initial human trial in which patients with se-

vere head injury (GCS ≤ 8) were treated with the oxygen radical scavenger superoxide dismutase combined with polyethylene glycol,[195] free radical scavengers are not ready today for use at, or near, the point-of-wounding.

Antibiotic prophylaxis for central nervous system wounds

The prevention of infection of wounds of the central nervous system is very important because such infections can rapidly result in meningitis, cerebritis, abscess formation and even death. Unfortunately, the efficacy of prophylactic antibiotic administration at, or near, the point-of-wounding, is unproven. Israeli data from the Yom Kippur war even suggests that the prophylactic use of antibiotics might not only select for gram negative and mixed microbial infections but may actually increase the overall risk of wound infection.[196]

Although antibiotic prophylaxis in neurosurgical practice is not only very common but is considered the standard of care by many, the strength of evidence from civilian studies that proves that prophylactic antibiotics are effective at reducing the risk of infection from operations and wounding is limited.

Savitz et al., advocates for antibiotic prophylaxis in neurosurgery admit, in their 2003 meta-analysis, that "To date, individual studies have not demonstrated a significant benefit for prophylactic antibiotic therapy in spinal operations."[197(pp.243-5)] But they go on to point out that, in their meta-analysis, pooled infection rates were 2.2% (10 of 451 patients) with antibiotics and 5.9% (23 of 392 patients) without antibiotics. The pooled odds ratio in their meta-analysis was 0.37 (95% confidence interval, 0.17-0.78), favoring antibiotic

treatment (P < 0.01). They concluded that prophylactic antibiotic therapy is beneficial for spinal surgery, even when expected infection rates without antibiotic treatment are low; and the rate of infection in combat casualties with CNS injuries is certainly much higher than that occurring in planned surgeries.

During the Vietnam conflict sepsis, particularly in the setting of extensive burns or penetrating trauma to central nervous system, was the major cause of mortality in rear echelon hospitals.[198] The higher the risk of infection the greater the likely benefit of prophylactic antibiotic administration.

A number of double and single blinded studies[199-204] have been done that suggest that antimicrobial prophylaxis reduces the rate of wound infection following craniotomy. The recommendation of the 1988 U.S. edition of the NATO War Surgery Manual[205] regarding antibiotic prophylaxis for combat casualties with CNS injuries, is that, although the efficacy of prophylactic antibiotics has not been proven, "intravenous antibiotics should be administered at meningeal doses for one week." Based upon the currently available evidence such a policy seems reasonable, especially in the case of penetrating central nervous system injury, and should probably be implemented without long delay. The only question is which antibiotic should be used for prophylaxis of CNS combat injury?

For combat wounds in general, cefoxitin, was initially recommended by Butler and Hagmann[206] but prior to Operation Enduring Freedom Butler and O'Connor examined the concept of prophylactic antibiotic administration by Special Operations Forces medics and concluded that prophylactic administration of cefotetan, par-

enterally,or gatifloxacin orally, were better choices.[207] Their recommendation lead to a U.S. Army Special Operations Command policy of pre-hospital prophylactic antibiotic administration by SOF medics and these antibiotics were issued to SOF medics for this purpose during Operations Enduring Freedom and Iraqi Freedom. Unfortunately cefotetan has poor penetration into the CSF and it is generally not recommended for prophylaxis of CNS injury. A better choice for antimicrobial prophylaxis of combat caused CNS injuries is ceftriaxone.

Opponents of antibiotic prophylaxis argue that great emphasis should be placed on aseptic technique and wound care because the administration of antibiotics alone will not compensate for inattention to detail;[207] they certainly have a point. The emphasis in the training of pre-hospital personnel should be on minimizing wound contamination and speedy evacuation to surgical care. At best antibiotics are an adjunct to good wound care not a substitute. It would be ideal if a study were conducted to assess the efficacy of prophylactic antibiotic administration prior to there being a Department of Defense-wide implementation of a policy of prophylactic pre-hospital antibiotic administration but this seems unlikely.

Maintaining Adequate Oxygen Delivery to the Brain-Injured Casualty

The brain lies in a rigid container that has a fixed volume of approximately 1500 cm^3. Within this fixed space lie the parenchyma of the brain (approx. 80% of the total volume), cerebral spinal fluid, and blood (remaining 20%). Because the cranium is a rigid container, any increase in one component must come at the expense of one or more of the other components. If

trauma produces swelling of the brain or an intracerebral hematoma, some cerebral spinal fluid (CSF) and blood are excluded from the space.

When the swelling and/or hematoma are sufficiently large and no more CSF can be excluded from the space, intracranial pressure rises and the flow of blood into the brain slows and eventually stops. As cerebral blood flow drops, oxygen delivery to brain cells drops, and when the threshold is crossed where insufficient oxygen is being delivered to meet cerebral metabolic needs, ischemia, and eventually infarction, occur.[209]

Through the mechanism of autoregulation, primarily achieved by altering cerebral vascular resistance, the body is able to maintain a constant delivery of oxygen to the brain despite a significant drop in blood pressure or a significant rise in intracranial pressure (ICP). As intracranial pressure rises, if the body is able to raise the blood pressure, it does so in order to maintain a cerebral blood flow great enough to prevent irreversible neuronal damage (> 18-20 mL/100 g brain tissue/min is the critical amount of blood flow needed to deliver enough oxygen to meet the minimal metabolic needs of the brain). This is the Cushing response, i.e., increased ICP leading to hypertension and a reflexive bradycardia. Cerebral blood flow is equal to the cerebral perfusion pressure, or mean arterial pressure (MAP) minus ICP, divided by the cerebral vascular resistance.

(Cerebral blood flow) = (Cerebral perfusion pressure)

= (MAP-ICP) ÷ (Cerebral vascular resistance)

Under normal conditions, ICP is minimal (0-10 mm Hg) so CPP is essentially equal to MAP. In serious head trauma, the ICP may increase enough to reduce the CPP to zero. Thus, a decrease in MAP and/or an increase in ICP can result in decreased cerebral blood flow.[210] The critical issue in the management of patients with serious head injury is not intracranial pressure but rather the delivery of sufficient oxygen to meet cerebral metabolic needs. When efforts to reduce ICP reduce oxygen delivery, seriously brain injured patients may have worse outcomes even if ICP is reduced. Measures that improve oxygen delivery to an injured brain include the following:

1. Maintaining MAP > 70 mm Hg

2. Ensuring an adequate amount of functional hemoglobin

3. Optimizing oxygenation and ventilation

4. Ensuring that oxygen can be offloaded at the cellular level

5. Preventing and/or reducing cerebral edema

Measures that focus on treating elevated ICP but also reduce cerebral blood flow may be necessary if herniation is imminent, but depriving an ischemic brain of oxygen to prevent herniation may result in brain death.

Another way to ensure that sufficient oxygen is supplied to meet demand is to reduce demand. Preventing increases in the cerebral metabolic requirement for oxygen by preventing elevations in temperature (or by inducing mild hypothermia), preventing and/or rapidly treating seizures, and avoiding hyperglycemia are the most commonly agreed-upon methods. Lowering the cerebral metabolic rate by prophylactically inducing a barbituate coma, however, is more controversial. Despite

some theoretical advantages to the use of prophylactic barbituate coma in treating severely head-injured patients, it does not appear to be beneficial in improving outcome[211] and should not be attempted at or near the point of wounding.

Elevation of the head has long been advocated in the prehospital treatment of head-injured patients. Despite this practice having been recently challenged on the grounds that elevation of the head leads to decreased cerebral perfusion pressure studies by Feldman et al.[212] and Hickey & Sloan.[139] have shown that head elevation from 0° to 30° reduces intracranial pressure without producing any significant change in cerebral perfusion pressure, cerebral blood flow, cerebral metabolic demand for oxygen, or cerebral vascular resistance. Based upon these findings patients being treated for serious head injury at or near the point of wounding should have the head of the litter elevated by 15° to 30° unless the patient is in shock that is unresponsive to hemorrhage control and fluid resuscitation.

Appropriate prehospital measures aimed at minimizing the cerebral metabolic rate that can, and should, be undertaken for the seriously head-injured casualty include controlling fever and other causes of elevated temperature (even mild hypothermia has been shown to be protective in ischemic brain injury),[213] and control of agitation and seizure activity.[214]

Mannitol and hypertonic saline can be used individually to control cerebral edema at or near the point of wounding. Mannitol has long been used to control intracerebral pressure and brain swelling in patients with serious head injury. Recently two relatively large blinded, randomized, controlled, multi-center trials conducted by Cruz et al. have looked at the efficacy of high dose mannitol in the treatment of patients with blunt traumatic subdural hematomas[215] and traumatic intraparenchymal temporal lobe hemorrhages.[216]

Both of these studies demonstrated significant decreases mortality and morbidity when a standard early initial bolus of 0.6 – 0.7 grams mannitol/kilogram body weight mannitol was followed by an additional bolus of 0.6 – 0.7 gm/kg if no pupillary widening and up to 1.2 to 1.4 gm/kg if pupillary widening was present. These studies differ from earlier findings of no benefit or even possible harm associated with either repeated dosing with the use of doses higher than .25 g/kg.[217] If the Cruz study findings of dramatic benefit from high dose mannitol are confirmed by additional studies and especially if benefit is shown from pre-hospital administration of high dose mannitol then it would seem reasonable to recommend the use of high dose mannitol (1.4 gm/kg) to treat serious blunt head injured combatants at or near the point of wounding.

There is increasing interest in the use of hypertonic saline both to treat hemorrhagic shock and cerebral edema. The use of hypertonic saline alone, and combined with dextran, has been studied extensively for these indications.[218-229]

Although hypertonic saline is effective at reducing cerebral edema and intracranial pressure, and improving cerebral blood flow, there is no definitive evidence it is responsible for improved outcomes in head injured patients.[229] Most studies comparing the efficacy of hypertonic saline with mannitol suggest a similar efficacy in terms of reduced intracranial pressure but indicate that mannitol may last longer. Qureshi & Suarez,[229] in their recent extensive review of the use of hyper-

tonic saline to treat cerebral edema and intracranial hypertension, note that the following adverse effects have been associated with the use of hypertonic saline:

- Electrolyte abnormalities
- Bleeding diatheses
- Phlebitis
- Cardiac failure
- Central pontine myelinolysis and rebound intracranial hypertension (unproven but possible)[229]

With a low frequency of significant side effects and with the majority of studies showing a definite reduction in intracranial pressure following the administration of hypertonic saline, it seems reasonable to recommend the use of hypertonic saline for the treatment of seriously head injured patients at or near the point of wounding when (and if) it is approved for use. The use of hypertonic saline as a single resuscitative fluid for prehospital use in the treatment of hemorrhagic and burn shock and head injury patients is discussed at length in the Circulation section.

High glucose levels should be avoided. Specifically, patients with head injury should not be resuscitated with glucose-containing solutions and patients with altered mental status of unknown etiology should not be reflexively given 50% glucose in water unless hypoglycemia has been confirmed. High glucose levels may aggravate ischemic injury by increasing lactic acidosis and contributing to cerebral edema.[230-233] The goal in head-injured patients is to maintain blood sugar levels between 100 and 150 mg/dL. In practical terms, at or near the point of wounding, this simply means that previously healthy combat troops who may have serious head injury should not be given glucose-containing solutions.

Hyperventilation

Although falling into disfavor, hyperventilation continues to be used by some to treat patients with traumatic brain injury. Ghajar et al. found in their survey of 277 medical centers specializing in neurotrauma that hyperventilation was used in the treatment of intracranial hypertension for most patients in 83% of the center surveyed.[233] Despite this continued use the evidence supporting the efficacy of hyperventilation is limited.

The important question is not whether hyperventilation reduces intracranial pressure, because it most certainly does, at least acutely (but even this benefit is lost after a few hours); it does so by reducing cerebral arteriolar diameter (CO_2 reactivity of cerebral arterioles) and by secondarily reducing cerebral blood flow.[234] The important question is, "Does hyperventilation reduce mortality and improve neurologic outcomes in head-injured patients?" There is little evidence to suggest that it does and some evidence that, in some circumstances, it actually increases mortality and worsens neurologic outcomes (i.e. prolonged hyperventilation).[235-237]

Most important is not the intracranial pressure but rather the

(1) Amount of oxygen the brain needs (cerebral metabolic rate),

(2) Amount that is actually delivered, and

(3) Ability of the brain to use the oxygen that is delivered.

Cerebral oxygen content is dependent upon cerebral blood flow, oxygen content in the cerebral blood (primarily deter-

mined by hemoglobin content), and ability of the brain to extract the available oxygen (oxygen-hemoglobin dissociation curve). By increasing cerebrovascular resistance through hypocarbic vasoconstriction hyperventilation reduces cerebral blood flow and secondarily reduces ICP. Unfortunately this reduced ICP comes at the cost of decreased oxygen delivery to already ischemic gray matter. Hyperventilation reduces cerebral oxygen delivery both by reducing cerebral blood flow and by inducing an alkalemia that shifts the oxygen-hemoglobin dissociation curve to the left and reduces the ability of hemoglobin to off-load oxygen at the cellular level.

The 1995 recommendation of the Joint Section on Neurotrauma and Critical Care, approved by the Boards of the American Association of Neurological Surgeons and Congress of Neurological Surgeons, states that hyperventilation should *not* be used prophylactically "…during the first 24 hours after severe traumatic brain injury…because it can compromise cerebral perfusion during a time when cerebral blood flow is reduced."[179, 238] A somewhat contrary position taken by Stocchetti & Maas et al., in a recent review of hyperventilation for head injured patients, states, "Our opinion is that the careful use of hypocapnia for the short-term control of raised ICP remains useful…"[239] In sum it is certainly reasonable to limit the aggressiveness and duration of any hyperventilation that is done to treat intracranial hypertension.

Evacuation of Head-Injured Patients

It has been known for many years that expert neurosurgical care is critical to ensure optimal outcomes for head-injured patients. As long ago as 1939 Jefferson noted that, "…the mortality of head wounds is distinctly lower in the hands of those who understand them…It is only too easy to increase damage to the nervous system by injudicious operating, and so to leave the community's charge a permanently crippled individual."[147(p.82)]

Balancing the need to get head-injured patients to the care of a neurosurgeon with the small number of such specialists has long been a challenge. Placing neurosurgeons far forward in the evacuation chain creates critical shortages elsewhere and leads to inefficient use of their specialized talents. Placing them in centralized facilities far in the rear, however, excessively prolongs the time it takes a head-injured patient to reach this specialized level of care. A variety of solutions to this problem have been tried, but the best seems to be a combination of placing neurosurgeons mid-way in the evacuation chain and then moving them and their team around to areas of active or anticipated combat.[17(pp.40-43), 76(pp. 378-379,387-389,392),147(p.52)]

In the modern military neurosurgical record there appears to be an inconsistency as to the impact of travel time on the morbidity and mortality of the head trauma victim. Some sources indicate that such patients "travel well,"[17 (pp.101-102),147 (p.384)] while elsewhere the need for rapid transport and minimizing the time from wounding to neurosurgical care is emphasized.[147 (p.52)] This is probably explained by the differences between types of head injury. Patients sustaining serious blunt head trauma are at much greater risk of developing an expanding intracranial hematoma with increasing intracranial pressure than are those sustaining penetrating head trauma (at least those that survive to receive medical care).[17 (p.102)]

There is no question that when there is an expanding hematoma and increasing in-

tracranial pressure, increased length of time to surgery increases the likelihood of death or permanent disability. In patients with an acute subdural hematoma, Seelig et al. found a fourfold increase in the mortality rate if surgery to evacuate the hematoma was delayed 4 hours or more after injury compared with those patients who had surgery within 2 hours.[240, 241] Conversely, those with penetrating head injuries who have survived the immediate post-injury period appear to tolerate long transport reasonably well.

Any trained general surgeon can perform surgical decompression of an expanding intracranial hemorrhage when the location of the hematoma is known. Unfortunately, the only currently available means for accurately localizing an intracranial hematoma is computerized axial tomography. Although weight is not an issue in civilian health care, this piece of equipment is so heavy that it is found no further forward than a combat support hospital. If a new portable, lightweight, durable, and easily used device were introduced that could localize an intracranial hematoma, it would then be possible, far forward, to sort head-injured patients into those who can tolerate a long transport from those who need early decompression by a general surgeon.

Making the appropriate evacuation decision is of critical importance in head-injured patients. Over-evacuation of patients sustaining minor closed-head injuries leads to a drain on forward combat power that, as our army shrinks in size, must be prevented at all costs.

During World War II it was noted that after such a patient was evacuated out of the forward areas *"an early return to duty was unlikely."*[17(p.102)] Wounds of the scalp present a particular dilemma in that the general recommendation is that, *"Every wound of the scalp must be regarded as a possible penetrating wound until the presence of foreign bodies within the cerebrum are ruled out with roentgenograms"*[17(p.102)] and *"...no scalp wound is so trivial that it should not be regarded as potentially serious,"*[147(p.84)] yet minor scalp wounds can and should be cared for in the forward, prehospital environment, followed by a quick return to duty. Mechanism of injury can often be used to sort out those patients with simple, uncomplicated, scalp injuries from those with a penetrating scalp injury or serious underlying injury. An effective, lightweight, durable, and easy to use diagnostic tool to help differentiate between these groups of patients would also be very helpful.

It certainly continues to be the case that patients who need the services of a neurosurgeon do best when evacuated directly to a facility with neurosurgical capability unless another more pressing injury takes precedence. As well stated by Jolly in 1938, *"Wounds of the head do not mix well with other casualties in the rota for operation...if they take their turn on the same table with injuries of other regions they come either to be operated upon by those without the necessary knowledge and ability, or they hold back other serious but more expeditiously dealt with injuries to their disadvantage."*[147(p.52)]

Summary

Penetrating and blunt head and neck wounds injuries, while relatively uncommon in combat casualties, are disproportionately common relative to body surface area and have a disproportionately high morbidity and mortality. Although penetrating injuries predominate as the mechanism of injury in combat-caused head and neck wounds, blunt trauma is becoming relatively more common. Despite considerable advances in trauma care the morbidity and mortality of head and neck wounds remains high.

In the forward combat areas little can be done for casualties with such injuries and sorting out minor head and neck injuries from those requiring more advanced care is problematic even for physicians, much less combat medics. New, lightweight, durable, and easy-to-use tools capable of effectively sorting out these categories of patients should be a high priority for the military medical research and development community.

Combat-caused neck injuries, both penetrating and blunt, appear to be different from their civilian counterparts in terms of structures injured and likelihood of instability. Although any discussion about cervical spine instability is problematic because a consistent definition of "instability" is lacking in the literature, it appears that combat casualties who survive a penetrating neck wound are considerably less likely to have an "unstable" cervical spine than civilian patients with anatomically similar blunt trauma injuries.

There is even some suggestion in the literature that casualties who survive a low-velocity penetrating wounding of the neck are more likely to have spinal cord and "unstable" injuries than casualties who survive high-velocity bullet wounds of the neck (a significant portion of this difference no doubt being due to a higher rate of survival in low-velocity wounding). Although casualties with blunt neck trauma and cervical spine instability can certainly sustain spinal cord injury if they are handled injudiciously, it would seem that the risk of this, in a prehospital combat setting, may be overstated especially if the casualty is awake, responsive, and able to protect his or her own cervical spine.

How to best manage neck-injured casualties at or near the point of wounding is problematic. There is significant controversy, even in civilian EMS literature, regarding the appropriate management of patients with these injuries. Even if there were a general consensus regarding the appropriate prehospital management of civilian patients with neck injuries, the differences between civilian and combat circumstances would render the decision mostly irrelevant to the management of neck-injured combat casualties. Not only are there significant differences between typical civilian and combat-related neck injuries in terms of likelihood of spinal cord injury and instability, virtually all aspects of prehospital combat casualty care are different in ways that alter the risk-benefit ratio of the typical civilian EMS approach to the management of such casualties.

In civilian settings, EMS supplies are transported to the scene of the injury by vehicle while in most combat situations medical supplies are carried on the backs of combat medics. Backboards are out of the question and even cervical collars might be left behind in favor of more field dressings.

The care environment is different as well. In civilian settings, it is rare that patients remain at continued risk of serious injury following the initial wounding and even rarer that prehospital personnel are seriously at risk while attempting to render initial care. During initial evacuation, however, combat casualties must often be left unattended (or without skilled attendance) during transport. Evacuation times are usually significantly longer in combat than in civilian settings.

All of these differences increase the risk associated with applying the standard treatment of civilian neck-injured casualties to combat casualties, i.e., complete immobilization on a long backboard. Combat casualties thus treated would be exposed to serious risk of aspiration, impaired ventilation, and pressure sores and would be unable to protect themselves or assist in any way should there be an attack on their evacuation vehicle.

Although it is common practice for combat medics to carry a rigid cervical collar to treat combat casualties with a presumed high risk of cervical spine injury, no data supports this practice. It is possible that, in sum, there may be more risk than benefit from applying just a rigid cervical collar when compared to not applying one. However, rather than abandon this apparently reasonable practice because of the lack of supportive evidence, it would seem sensible to continue to apply a cervical collar to those combat casualties at relatively high risk of cervical spine injury. A study to determine exactly what are the risks and benefits of isolated cervical collar application to casualties with potential cervical spine injury needs to be done.

When the mechanism of injury is blunt and significant (fall from a height, motor vehicle or aircraft crash, etc…) and especially when the casualty has a complaint of neck pain and/or there are neurologic findings consistent with spine injury, spine immobilization should be carried out to the extent possible with available supplies and appropriate for the tactical and evacuation circumstances.

It is essential that every forward area medical provider recognize that any casualty restrained in a supine position is at risk of aspiration, impaired ventilation, and pressure sores. Further, they are entirely at the mercy of others and the circumstances of the moment. During evacuation, an attendant skilled in basic airway management must be continually in attendance of any casualty strapped down or otherwise unable to protect his or her own airway.

To prevent pressure sores, all hard objects must be removed from the pockets and from behind the casualty and, especially if the transport is long, the weight of the casualty should be periodically shifted. These are all reasonable guidelines that should be provided to prehospital combat medical personnel. Even in this selected population, the likelihood of prehospital care converting a spine injured, cord-intact, patient into a cord-damaged patient is very small (but certainly not zero). The risk of this occurrence has probably been seriously overstated in the literature and is probably even lower in a population of young, previously healthy, unintoxicated military combatants.

When the mechanism of injury is penetrating, the primary management focus must be on ensuring that the airway remains patent, that the patient is adequately ventilating, and that hemorrhage is controlled. Speed of evacuation to resuscitative sur-

gery is probably the key factor in survival of such patients. The application of advanced airway management techniques by prehospital personnel to casualties with neck injuries, especially penetrating neck injuries, carries significant risk and should be avoided unless unequivocally indicated *and* the care provider is trained and experienced in the technique(s). Specific efforts to secure the cervical spine of casualties with penetrating neck wounds are likely to result in greater harm than benefit. This is not to suggest that the cervical spine should be entirely disregarded, just that concerns for the cervical spine should not dictate management. Certainly, if a casualty complains of increased pain or develops new neurologic symptoms with movement, reasonable and appropriate measures should be taken to stabilize the spine.

The civilian controversy regarding clinical "clearing" of the cervical spine, either in the field, or in an emergency department, is probably not directly relevant to prehospital combat casualty care. In most circumstances at or near the point of wounding, casualties will "clear" their own cervical spine if able to do so. Absent any specific guidelines, most combat medics will use common sense to "clear" most casualties with neck injuries who come to their attention. For this reason, it would probably be useful to provide to combat medics simple guidelines for making a decision regarding which casualties to return to duty, which to evacuate, and which would likely benefit from cervical spine immobilization.

If it is clear that a casualty with a blunt trauma mechanism has sustained spinal cord injury, the current guidelines are to administer high-dose methylprednisolone as soon as possible. In most situations, this should be done at the level of the Battalion Aid Station rather than in the field.

For head injured casualties a patent airway, adequate ventilation, and control of all controllable hemorrhage are the main priorities. Casualties with serious head injuries should be transported in the lateral recumbent position or in the prone position with the head turned to the side. If it is necessary, for any reason (such as cervical spine immobilization), to transport such casualties in a supine position an attendant with ready access to suction and skilled in airway management must be present throughout the transport.

Early and adequate ventilation, oxygenation and cerebral perfusion are critical.[183] All controllable hemorrhage should be controlled. Contrary to the approach for most patients with uncontrolled internal bleeding, head injured patients should be fluid resuscitated to maintain a systolic blood pressure above 90 mm of mercury. In casualties with significant hemorrhage and with altered mental status, head injury, not hypovolemia, should be considered the cause until proven otherwise. As noted in the circulation chapter, most combat casualties in hemorrhagic shock have clear mental status until cerebral perfusion pressure drops below 70 mm of mercury.

In managing the potentially head injured casualty at, or near, the point-of-wounding, the job of the pre-hospital combat care provider is to identify which casualties may have sustained serious head injury, and decide which need urgent evacuation to the care of a neurosurgeon and which can be safely returned to duty. As circumstances permit there should be a low threshold for rapid evacuation of head injured casualties. Guidelines developed

to minimize head CT use in trauma victims with minor head injuries may be useful in developing head injury evacuation guidelines and in making return-to-duty decisions.[184, 186]

The critical issue in the management of patients with serious head injury is delivery of sufficient oxygen to meet cerebral metabolic needs. The forward health care provider achieves this by maintaining an adequate blood pressure, preventing all preventable blood loss, optimizing oxygenation and ventilation, insuring that oxygen can be off-loaded at the cellular level by avoiding alkalosis from excessive hyperventilation, and preventing and/or reducing cerebral edema by avoiding overhydration (especially with hypotonic solutions). The current recommendation is that hyperventilation should <u>not</u> be used prophylactically.[179, 238]

Outcomes of head injured patients can also be improved by reducing cerebral metabolic demand. Temperature elevation should be avoided, seizures prevented and/or rapidly treated, and hyperglycemia avoided. The head of the litter of head injured casualties should elevated by 15 to 30 degrees unless the patient is in shock unresponsive to hemorrhage control and fluid resuscitation.[138, 212, 214] Mannitol and hypertonic saline are both capable of reducing cerebral edema [216-219, 222-224, 226-229, 242] but it remains unclear how much either of these agents contributes of overall survival of head injured patients.[229] It is still seems reasonable to recommend the use of Mannitol at the level of a battalion aid station to treat seriously head injured patients.

Combat casualties with penetrating head wounds who survive the initial wounding apparently "travel well" and, with appro-

priate enroute care, can usually tolerate relatively long evacuation.[17(pp101-102),76 (p384)] Patients with serious blunt head trauma appear to do less well if evacuation is lengthy.[17 (p102),147 (p52)] When there is an expanding hematoma and increasing intracranial pressure the head injured casualty should be evacuated to the closest surgeon who can localize the lesion and perform surgical decompression. Patients who need the services of a neurosurgeon do best when evacuated directly to a facility with neurosurgical capability unless another more pressing injury takes precedence.[147 (p52)]

Although clear scientific proof is lacking to support the efficacy of prophylactic antibiotic administration by pre-hospital personnel to prevent central nervous system wound infections, the weight of the available evidence supports such a policy. The specific antibiotic selected for this purpose should have low risks, a long half-life, should readily penetrate into the CNS, should be effective against common CNS pathogens, and should not have any special handling considerations, such as a requirement for refrigeration.

References

1. Gibbs M, Jones A. Cervical spine injury: A state-of-the-art approach to assessment and management. Emerg Med Pract. 2001;10:1-24.

2. Beatty W. The death of Lord Nelson: The authentic narrative. 4th ed. London, UK: Stobart & Son; 1985.

3. Pickett W, Ardern C, Brison RJ. A population-based study of potential brain injuries requiring emergency care. Cmaj. Aug 7 2001;165(3):288-292.

4. Kraus JF, Black MA, Hessol N, et al. The incidence of acute brain injury and serious impairment in a defined population. Am J Epidemiol. Feb 1984;119(2):186-201.

5. Thurman DJ, Alverson C, Dunn KA, Guerrero J, Sniezek JE. Traumatic brain injury in the United States: A public health perspective. J Head Trauma Rehabil. Dec 1999;14(6):602-615.

6. Sosin DM, Sniezek JE, Waxweiler RJ. Trends in death associated with traumatic brain injury, 1979 through 1992. Success and failure. Jama. Jun 14 1995;273(22):1778-1780.

7. Foundation NHI. Facts about Traumatic Brain Injury. Washington D.C.; 1995.

8. Jager TE, Weiss HB, Coben JH, Pepe PE. Traumatic brain injuries evaluated in U.S. emergency departments, 1992-1994. Acad Emerg Med. Feb 2000;7(2):134-140.

9. Max W, MacKenzie EJ, Rice DP. Head injuries: costs and consequences. Journal of Head Trauma Rehabilitation. 1991;6:76-91.

10. Burney RE, Maio RF, Maynard F, Karunas R. Incidence, characteristics, and outcome of spinal cord injury at trauma centers in North America. Arch Surg. May 1993;128(5):596-599.

11. Hockberger RS , Kirsehnbaum KJ. Chapter 36 Spine. In: Marx J, ed. Rosen's Emergency Medicine Concepts and Clinical Practice. 5th ed. St. Louis, MO: Mosby; 2002:329-370.

12. Berkowitz M, O'Leary P, Kruse D, Harvey C. Spinal cord injury: An analysis of medical and social costs. New York: Demos Medical Publishing Inc.; 1998.

13. Berkowitz M. Assessing the socioeconomic impact of improved treatment of head and spinal cord injuries. J Emerg Med. 1993;11 Suppl 1:63-67.

14. Bellamy RF. The causes of death in conventional land warfare: implications for combat casualty care research. Mil Med. Feb 1984;149(2):55-62.

15. Spurling R. Chapter III "The European Theater of Operations". In: Coates JJ, Spurling R, Woodhall B, eds. Medical Department of the US Army in World War II - Surgery in World War II: Neurosurgery. Vol 1. Washington, DC: Office of the Surgeon General; 1958:82.

16. Reister F, ed. Battle casualties and medical statistics - U.S. Army experience in the Korean War. Washington, D.C.: Office of the Surgeon General; 1986.

17. Campbell EH. Chapter VI The Mediterranean theater of operations. In: Coates JB, Spurling RG, B W, eds. Surgery in World War II: Neurosurgery, Vol. 1. Medical Department of the US Army in World War II. Vol 1. Washington, DC]: Office of the Surgeon General; 1958.

18. Carey ME. Learning from traditional combat mortality and morbidity data used in the evaluation of combat medical care. Mil Med. Jan 1987;152(1):6-13.

19. Salazar AM, Schwab K, Grafman JH. Penetrating injuries in the Vietnam war. Traumatic unconsciousness, epilepsy, and psychosocial outcome. Neurosurg Clin N Am. Oct 1995;6(4):715-726.

20. Gurdjian ES. The treatment of penetrating wounds of the brain sustained in warfare. A historical review. J Neurosurg. Feb 1974;40(2):157-167.

21. Rosenfeld JV. Gunshot injury to the head and spine. J Clin Neurosci. Jan 2002;9(1):9-16.

22. Lepore FE. Harvey Cushing, Gordon Holmes, and the neurological lessons of World War I. Arch Neurol. Jul 1994;51(7):711-722.

23. Rish BL, Dillon JD, Caveness WF, Mohr JP, Kistler JP, Weiss GH. Evolution of craniotomy as a debridement technique for penetrating craniocerebral injuries. J Neurosurg. Dec 1980;53(6):772-775.

24. Matson DD. The Treatment of Acute Craniocerebral Injuries Due to Missiles. Vol 1. Springfield IL: Charles C. Thomas; 1948.

25. Meirowsky AM. Penetrating Craniocerebral Trauma. Springfield IL: Charles C. Thomas; 1984.

26. George ED, Dagi TF. Military penetrating craniocerebral injuries. Applications to civilian triage and management. Neurosurg Clin N Am. Oct 1995;6(4):753-759.

27. Aarabi B. Management of traumatic aneurysms caused by high velocity missile head wounds. Neurosurg Clin N Am. 1995;6:775-797.

28. Arabi B. Surgical outcome in 435 patients who sustained missile head wounds during the Iran-Iraq War. Neurosurgery. Nov 1990;27(5):692-695; discussion 695.

29. Carey ME. Experimental missile wounding of the brain. Neurosurg Clin N Am. 1995;6:629-642.

30. Barkana Y, Stein M, Scope A, et al. Prehospital stabilization of the cervical spine for penetrating injuries of the neck - is it necessary? Injury. Jun 2000;31(5):305-309.

31. Asensio JA, Valenziano CP, Falcone RE, Grosh JD. Management of penetrating neck injuries. The controversy surrounding zone II injuries. Surg Clin North Am. Apr 1991;71(2):267-296.

32. Demetriades D, Skalkides J, Sofianos C, Melissas J, Franklin J. Carotid artery injuries: experience with 124 cases. J Trauma. Jan 1989;29(1):91-94.

33. Arishita GI, Vayer JS, Bellamy RF. Cervical spine immobilization of penetrating neck wounds in a hostile environment. J Trauma. Mar 1989;29(3):332-337.

34. Spurling RG. Part I - Administrative Considerations in Neurosurgery Chapter III "The European Theater of Operations". In: Coates JB, Spurling RG, Woodhall B, eds. Surgery

in World War II: Neurosurgery. Vol I. Washington D.C.: Office of the Surgeon General; 1958:82.

35. *Goldberg DG. Chapter I, "Short Historical Survey/Coverage of the Surgical Treatment of bullet wounds and damages of spine and spinal cord. Wright Patterson Air Force Base, OH: Transated from Russian and Published by Foreign Technology Division; 1952.*

36. *Frohna WJ. Emergency department evaluation and treatment of the neck and cervical spine injuries. Emerg Med Clin North Am. Nov 1999;17(4):739-791, v.*

37. *Garland DE, Lankenau JE. Epidemiology and costs of spine trauma. In: Capen DA, Haye W, eds. Comprehensive Management of Spine Trauma. St. Louis: Mosby-Year Book; 1998:1-5].*

38. *Bailey L. Motor-vehicle accidents take a grisly toll in Iraq. Army Times. June 16, 2003, 2003: 12.*

39. *Tator CH. Strategies for recovery and regeneration after brain and spinal cord injury. Inj Prev. Dec 2002;8 Suppl 4:IV33-36.*

40. *Orledge JD, Pepe PE. Out-of-hospital spinal immobilization: is it really necessary? Acad Emerg Med. Mar 1998;5(3):203-204.*

41. *Hauswald M, Braude D. Spinal immobilization in trauma patients: is it really necessary? Curr Opin Crit Care. Dec 2002;8(6):566-570.*

42. *American College of Surgeons Committee On Trauma. Advanced Trauma Life Support Program for Physicians. Chicago, IL: American College of Surgeons; 1997.*

43. *Zipnick RI, Scalea TM, Trooskin SZ, et al. Hemodynamic responses to penetrating spinal cord injuries. J Trauma. Oct 1993;35(4):578-582; discussion 582-573.*

44. *Apfelbaum JD, Cantrill SV, Waldman N. Unstable cervical spine without spinal cord injury in penetrating neck trauma. Am J Emerg Med. Jan 2000;18(1):55-57.*

45. *Walters RL, Adkins RH. The effects of removal of bullet fragments retained in the spinal canal: A collaborative study by the National Spinal Cord Injury Model System. Spine. 1991;16:934-939.*

46. *Carducci B, Lowe RA, Dalsey W. Penetrating neck trauma: consensus and controversies. Ann Emerg Med. Feb 1986;15(2):208-215.*

47. *Ordog GJ, Albin D, Wasserberger J, Schlater TL, Balasubramaniam S. 110 bullet wounds to the neck. J Trauma. Mar 1985;25(3):238-246.*

48. *Sumchai AP. Cervical spine immobilization of penetrating neck wounds in a hostile environment [Letter to the editor]. J Trauma. 1989;29:1453-1454.*

49. *Hoffman JR, Mower WR, Wolfson AB, Todd KH, Zucker MI. Validity of a set of clinical criteria to rule out injury to the cervical spine in patients with blunt trauma. National Emergency X-Radiography Utilization Study Group. N Engl J Med. Jul 13 2000;343(2):94-99.*

50. *Goldberg W, Mueller C, Panacek E, Tigges S, Hoffman JR, Mower WR. Distribution and patterns of blunt traumatic cervical spine injury. Ann Emerg Med. Jul 2001;38(1):17-21.*

51. *Riggins RS, Kraus JF. The risk of neurologic damage with fractures of the vertebrae. J Trauma. Feb 1977;17(2):126-133.*

52. *Lowery DW, Wald MM, Browne BJ, Tigges S, Hoffman JR, Mower WR. Epidemiology of cervical spine injury victims. Ann Emerg Med. Jul 2001;38(1):12-16.*

53. *Guttmann L. Spinal Cord Injuries: Comprehensive Management and Research. 2nd ed. Oxford: Blackwell Scientific Publications; 1976.*

54. *Louis R. Spinal stability as defined by the three-column spine concept. Anat Clin. 1985;7(1):33-42.*

55. *White AA, 3rd, Johnson RM, Panjabi MM, Southwick WO. Biomechanical analysis of clinical stability in the cervical spine. Clin Orthop Relat Res. 1975(109):85-96.*

56. *Rosen P, Barkin RM, eds. Emergency Medicine, Concepts and Clinical Practice. 3rd ed. St. Louis, MO: Mosby; 1992.*

57. *Jorden RC. Neck Trauma. In: Rosen P, Barkin R, eds. Emergency Medicine: Concepts and Clinical Practice. 4th ed. St. Louis, MO: Mosby Year Book; 1998:505-514.*

58. *Isiklar ZU, Lindsey RW. Low-velocity civilian gunshot wounds of the spine. Orthopedics. Oct 1997;20(10):967-972.*

59. *Waters RL, Hu SS. Penetrating injuries of the spinal canal. Stab and gunshot injuries. In: Frymoyer JW, ed. The Adult Spine. New York, NY: Raven Press; 1991:815-826.*

60. *Gordon K. Head and neck trauma. In: Hamilton GC, Hamilton M, Jehle D, Trott M, eds. Emergency Medicine: An Approach to Clinical Problem-Solving. Philadelphia, PA: W B Saunders; 1991:894-923.*

61. *Benzel ED. Biomechanics of Spine Stabilization - Principles and Clinical Practice. New York, NY: : McGraw-Hill; 1996.*

62. *White AA, Panjabi MM. Clinical Biomechanics of the Spine. Philadelphia, PA: Lippincott Williams & Wilkins; 1990.*

63. *Kupcha PC, An HS, Cotler JM. Gunshot wounds to the cervical spine. Spine. Oct 1990;15(10):1058-1063.*

64. *Bailey H, ed. Surgery of Modern Warfare. 2nd ed. Edinburg: E & S Livingston; 1942; No. 1.*

65. *Holdsworth F. Fractures, dislocations, and fracture-dislocations of the spine. J Bone Joint Surg Am. Dec 1970;52(8):1534-1551.*

66. *Kelly RP, Whitesides TE, Jr. Treatment of lumbodorsal fracture-dislocations. Ann Surg. May 1968;167(5):705-717.*

67. *Denis F. The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. Spine. Nov-Dec 1983;8(8):817-831.*

68. *White AA III, Panjabi MM. The problem of clinical instability in the human spine: A systematic approach. In: White AA, Panjabi MM, eds. Clinical Biomechanics of the Spine. 2 ed. Philadelphia, PA: Lippincott Williams & Wilkins; 1990.*

70. *Heiden JS, Weiss MH, Rosenberg AW, Kurze T, Apuzzo ML. Penetrating gunshot wounds of the cervical spine in civilians. Review of 38 cases. J Neurosurg. May 1975;42(5):575-579.*

71. Hammoud MA, Haddad FS, Moufarrij NA. Spinal cord missile injuries during the Lebanese civil war. Surg Neurol. May 1995;43(5):432-437; discussion 437-442.

72. Yoshida GM, Garland D, Waters RL. Gunshot wounds to the spine. Orthop Clin North Am. Jan 1995;26(1):109-116.

73. Saletta JD, Lowe RJ, Lim LT, Thornton J, Delk S, Moss GS. Penetrating trauma of the neck. J Trauma. Jul 1976;16(7):579-587.

74. Roon AJ, Christensen N. Evaluation and treatment of penetrating cervical injuries. J Trauma. Jun 1979;19(6):391-397.

75. Demetriades D, Theodorou D, Cornwell E, et al. Transcervical gunshot injuries: mandatory operation is not necessary. J Trauma. May 1996;40(5):758-760.

76. Cope Z. Chapter 10 Neurosurgery - The skull and brain. In: Copes Z, ed. Surgery. London: Her Majesty's Stationery Office; 1953.

77. Seelig JM, Becker DP, Miller JD, al e. Traumatic acute subdural hematoma: Major mortality reduction in comatose patients treated within four hours. N Engl J Med. 1981;304:1511.

78. Hoffman JR, Mower WR. Out-of-hospital cervical spine immobilization: making policy in the absence of definitive information. Ann Emerg Med. Jun 2001;37(6):632-634.

79. Hoffman JR, Wolfson AB, Todd K, Mower WR. Selective cervical spine radiography in blunt trauma: methodology of the National Emergency X-Radiography Utilization Study (NEXUS). Ann Emerg Med. Oct 1998;32(4):461-469.

80. Mahadevan S, Mower WR, Hoffman JR, Peeples N, Goldberg W, Sonner R. Interrater reliability of cervical spine injury criteria in patients with blunt trauma. Ann Emerg Med. Feb 1998;31(2):197-201.

81. Stiell IG, Wells GA, Vandemheen KL, et al. The Canadian C-spine rule for radiography in alert and stable trauma patients. Jama. Oct 17 2001;286(15):1841-1848.

82   .Bandiera G, Stiell IG, Wells GA, et al. The Canadian C-spine rule performs better than unstructured physician judgment. Ann Emerg Med. Sep 2003;42(3):395-402.

83. Brown LH, Gough JE, Simonds WB. Can EMS providers adequately assess trauma patients for cervical spinal injury? Prehosp Emerg Care. Jan-Mar 1998;2(1):33-36.

84. Stroh G, Braude D. Can an out-of-hospital cervical spine clearance protocol identify all patients with injuries? An argument for selective immobilization. Ann Emerg Med. Jun 2001;37(6):609-615.

85. Gilbert EH, Lowenstein SR, Koziol-McLain J, Barta DC, Steiner J. Chart reviews in emergency medicine research: Where are the methods? Ann Emerg Med. Mar 1996;27(3):305-308.

86. Bertolami CN, Kaban LB. Chin trauma: a clue to associated mandibular and cervical spine injury. Oral Surg Oral Med Oral Pathol. Feb 1982;53(2):122-126.

87. Scher AT. A plea for routine radiographic examination of the cervical spine after head injury. S Afr Med J. Jun 11 1977;51(24):885-887.

88. *Kellman R. The cervical spine in maxillofacial trauma. Assessment and airway management. Otolaryngol Clin North Am. Feb 1991;24(1):1-13.*

89. *Bayless P, Ray VG. Incidence of cervical spine injuries in association with blunt head trauma. Am J Emerg Med. Mar 1989;7(2):139-142.*

90. *Beirne JC, Butler PE, Brady FA. Cervical spine injuries in patients with facial fractures: a 1-year prospective study. Int J Oral Maxillofac Surg. Feb 1995;24(1 Pt 1):26-29.*

91. *Merritt RM, Williams MF. Cervical spine injury complicating facial trauma: incidence and management. Am J Otolaryngol. Jul-Aug 1997;18(4):235-238.*

92. *Williams J, Jehle D, Cottington E, Shufflebarger C. Head, facial, and clavicular trauma as a predictor of cervical-spine injury. Ann Emerg Med. Jun 1992;21(6):719-722.*

93. *Hills MW, Deane SA. Head injury and facial injury: is there an increased risk of cervical spine injury? J Trauma. Apr 1993;34(4):549-553; discussion 553-544.*

94. *Patton JH, Kralovich KA, Cuschieri J, Gasparri M. Clearing the cervical spine in victims of blunt assault to the head and neck: what is necessary? Am Surg. Apr 2000;66(4):326-330; discussion 330-321.*

95. *Kaups KL, Davis JW. Patients with gunshot wounds to the head do not require cervical spine immobilization and evaluation. J Trauma. May 1998;44(5):865-867.*

96. *Chong CL, Ware DN, Harris JH, Jr. Is cervical spine imaging indicated in gunshot wounds to the cranium? J Trauma. Mar 1998;44(3):501-502.*

97. *Kennedy FR, Gonzalez P, Beitler A, Sterling-Scott R, Fleming AW. Incidence of cervical spine injury in patients with gunshot wounds to the head. South Med J. Jun 1994;87(6):621-623.*

98. *Colterjohn NR, Bednar DA. Identifiable risk factors for secondary neurologic deterioration in the cervical spine-injured patient. Spine. Nov 1 1995;20(21):2293-2297.*

99. *Esce PG, Haines SJ. Acute Treatment of Spinal Cord Injury. Curr Treat Options Neurol. Nov 2000;2(6):517-524.*

100. *Kendall JL, Anglin D, Demetriades D. Penetrating neck trauma. Emerg Med Clin North Am. Feb 1998;16(1):85-105.*

101. *Domeier RM, Frederiksen SM, Welch K. Prospective performance assessment of an out-of-hospital protocol for selective spine immobilization using clinical spine clearance criteria. Ann Emerg Med. Aug 2005;46(2):123-131.*

102. *Markey JC, Jr., Hines JL, Nance FC. Penetrating neck wounds: a review of 218 cases. Am Surg. Feb 1975;41(2):77-83.*

103. *Bledsoe BE, Porter RS, Cherry RA. Intermediate Emergency Care: Principles & Practice. Upper Saddle River, NJ: Pearson/Prentice Hall; 2004.*

104. *Hockberger RS, Kirshenbaum KJ, Doris PE. Spinal injuries. In: Rosen P, Barkin R, Ling LJ, eds. Emergency Medicine: Concepts and Clinical Practice. Vol 1. 4th ed. St. Louis, MO: Mosby Year Book; 1998:462-505.*

105. *Anderson DK, Hall ED. Pathophysiology of spinal cord trauma. Ann Emerg Med. Jun 1993;22(6):987-992.*

106. Chiles BW, 3rd, Cooper PR. Acute spinal injury. N Engl J Med. Feb 22 1996;334(8):514-520.

107. Suderman VS, Crosby ET, Lui A. Elective oral tracheal intubation in cervical spine-injured adults. Can J Anaesth. Sep 1991;38(6):785-789.

108. Walls RM. Airway management in the blunt trauma patient: how important is the cervical spine? Can J Surg. Feb 1992;35(1):27-30.

109. Rhee KJ, Green W, Holcroft JW, Mangili JA. Oral intubation in the multiply injured patient: the risk of exacerbating spinal cord damage. Ann Emerg Med. May 1990;19(5):511-514.

110. Einav S. Intubation of the trauma patient with a fractured cervical spine: controversies and consensus. Isr J Med Sci. Nov 1997;33(11):754-756.

111. Criswell JC, Parr MJ, Nolan JP. Emergency airway management in patients with cervical spine injuries. Anaesthesia. Oct 1994;49(10):900-903.

112. Shatney CH, Brunner RD, Nguyen TQ. The safety of orotracheal intubation in patients with unstable cervical spine fracture or high spinal cord injury. Am J Surg. Dec 1995;170(6):676-679; discussion 679-680.

113. Gerling MC, Davis DP, Hamilton RS, et al. Effects of cervical spine immobilization technique and laryngoscope blade selection on an unstable cervical spine in a cadaver model of intubation. Ann Emerg Med. Oct 2000;36(4):293-300.

114. Murphy-Macabobby M, Marshall WJ, Schneider C, Dries D. Neuromuscular blockade in aeromedical airway management. Ann Emerg Med. Jun 1992;21(6):664-668.

115. Spaite DW, Joseph M. Prehospital cricothyrotomy: an investigation of indications, technique, complications, and patient outcome. Ann Emerg Med. Mar 1990;19(3):279-285.

116. Xeropotamos NS, Coats TJ, Wilson AW. Prehospital surgical airway management: 1 year's experience from the Helicopter Emergency Medical Service. Injury. Apr 1993;24(4):222-224.

117. Johnson DR, Dunlap A, McFeeley P, Gaffney J, Busick B. Cricothyrotomy performed by prehospital personnel: a comparison of two techniques in a human cadaver model. Am J Emerg Med. May 1993;11(3):207-209.

118. Nugent WL, Rhee KJ, Wisner DH. Can nurses perform surgical cricothyrotomy with acceptable success and complication rates? Ann Emerg Med. Apr 1991;20(4):367-370.

119. Johnson JL, Cheatham ML, Sagraves SG, Block EF, Nelson LD. Percutaneous dilational tracheostomy: a comparison of single- versus multiple-dilator techniques. Crit Care Med. Jun 2001;29(6):1251-1254.

120. Rogers WA. Fractures and dislocations of the cervical spine; an end-result study. J Bone Joint Surg Am. Apr 1957;39-A(2):341-376.

121. Podolsky S, Baraff LJ, Simon RR, Hoffman JR, Larmon B, Ablon W. Efficacy of cervical spine immobilization methods. J Trauma. Jun 1983;23(6):461-465.

122. Cloward RB, Netter FH. Acute cervical spine injuries. Clinical Symposia. 1980;321:1 - 32.

123. Geisler WO, Wynne-Jones M, Jousse AT. Early management of the patient with trauma to the spinal cord. Med Serv J Can. Jul-Aug 1966;22(7):512-523.

124. Hauswald M, Ong G, Tandberg D, Omar Z. Out-of-hospital spinal immobilization: its effect on neurologic injury. Acad Emerg Med. Mar 1998;5(3):214-219.

125. Gibbs MA, Jones AE. Cervical spine injury: A state-of-the-art approach to assessment and management. Emerg Med Pract. 2001;10:1-24.

126. Kirk M, Pace S. Pearls, pitfalls, and updates in toxicology. Emerg Med Clin North Am. May 1997;15(2):427-449.

127. Walsh M, Grant T, Mickey S. Lung function compromised by spinal immobilization. Ann Emerg Med. May 1990;19(5):615-616.

128. Bauer D, Kowalski R. Effect of spinal immobilization devices on pulmonary function in the healthy, nonsmoking man. Ann Emerg Med. Sep 1988;17(9):915-918.

129. Schafermeyer RW, Ribbeck BM, Gaskins J, Thomason S, Harlan M, Attkisson A. Respiratory effects of spinal immobilization in children. Ann Emerg Med. Sep 1991;20(9):1017-1019.

130. Abraham E, Gong H, Jr., Tashkin DP, Baraff LJ, Geehr E. Effect of pneumatic trousers on pulmonary function. Crit Care Med. Nov 1982;10(11):754-757.

131. Briscoe WA. Lung volumes. In: Fenn WO, Rahn H, eds. Handbook of Physiology: Respiration, Section 3. Vol 2. Washington D.C.: American Physiological Society; 1964:1363.

132. Spurling RG, Woodhall B, eds. Neurosurgery. Washington D.C.: Office of the Surgeon General; 1958. JB C, ed. Surgery in World War II, Medical Department, US Army; No. 1.

133. Cordell WH, Hollingsworth JC, Olinger ML, Stroman SJ, Nelson DR. Pain and tissue-interface pressures during spine-board immobilization. Ann Emerg Med. Jul 1995;26(1):31-36.

134. Mawson AR, Biundo JJ, Jr., Neville P, Linares HA, Winchester Y, Lopez A. Risk factors for early occurring pressure ulcers following spinal cord injury. Am J Phys Med Rehabil. Jun 1988;67(3):123-127.

135. Chan D, Goldberg R, Tascone A, Harmon S, Chan L. The effect of spinal immobilization on healthy volunteers. Ann Emerg Med. Jan 1994;23(1):48-51.

136. Barney RN, Cordell WH, E M. Pain associated with immobilization on rigid spine boards [abstract]. Ann Emerg Med. 1989;18:918.

137. Linares HA, Mawson AR, Suarez E, Biundo JJ. Association between pressure sores and immobilization in the immediate post-injury period. Orthopedics. Apr 1987;10(4):571-573.

138. Hickey R, Sloan T. Protecting the injured brain and spinal cord. Anesth Clin N Amer. 1996;14:39-58.

139. Bracken MB, Shepard MJ, Collins WF, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the Second National Acute Spinal Cord Injury Study. N Engl J Med. May 17 1990;322(20):1405-1411.

140. Bracken MB, Shepard MJ, Collins WF, Jr., et al. Methylprednisolone or naloxone treatment after acute spinal cord injury: 1-year follow-up data. Results of the second National Acute Spinal Cord Injury Study. J Neurosurg. Jan 1992;76(1):23-31.

141. Short D. Use of steroids for acute spinal cord injury must be reassessed. Bmj. Nov 11 2000;321(7270):1224.

142. Short D. Is the role of steroids in acute spinal cord injury now resolved? Curr Opin Neurol. Dec 2001;14(6):759-763.

143. Short DJ, El Masry WS, Jones PW. High dose methylprednisolone in the management of acute spinal cord injury - a systematic review from a clinical perspective. Spinal Cord. May 2000;38(5):273-286.

144. Holte K, Kehlet H. Perioperative single-dose glucocorticoid administration: pathophysiologic effects and clinical implications. J Am Coll Surg. Nov 2002;195(5):694-712.

145. Zajtchuk R, Grande CM, eds. Chapter 3 Airway management by Hecker RB, Kingsley CP in Anesthesia and Perioperative Care of the Combat Casualty. Washington, DC: Department of the Army, Office of the Surgeon General, and Borden Institute; 1995. Zajtchuk R, Bellamy RF, eds. Textbook of Military Medicine.

146. Adnet F, Jouriles NJ, Le Toumelin P, et al. Survey of out-of-hospital emergency intubations in the French prehospital medical system: a multicenter study. Ann Emerg Med. Oct 1998;32(4):454-460.

147. Jolly DW. Field Surgery in Total War. New York: Paul B Hoeber, Inc., Medical Book Department of Harbor & Brothers; 1939.

148. Office of the Chief Surgeon, Headquarters European Theater of Operations, United States Army. Circular Letter No. 23 Care of Battle Casualties Supplemental to Manual of Therapy - ETO - 5 May 1944. In: Coates JB, Carter BN, eds. Appendix D in Medical Department United States Army In World War II - Surgery in World War II - Activities of Surgical Consultants. Vol II. Washington D.C.: Office of the Army Surgeon General; 1945:982.

149. Butler EG, Puckett WO, Harvey EN, McMillen JH. Experiments on head wounding by high velocity missiles. J Neurosurg. 1945;2:358-367.

150. Carey ME, Young HF, Mathis JL. The neurosurgical treatment of craniocerebral missile wounds in Vietnam. Surg Gynecol Obstet. Sep 1972;135(3):386-389.

151. Crockard HA. Early intracranial pressure studies in gunshot wounds of the brain. J Trauma. Apr 1975;15(4):339-347.

152. Brandvold B, Levi L, Feinsod M, George ED. Penetrating craniocerebral injuries in the Israeli involvement in the Lebanese conflict, 1982-1985. Analysis of a less aggressive surgical approach. J Neurosurg. Jan 1990;72(1):15-21.

153. Byrnes DP, Crockard HA, Gordon DS, Gleadhill CA. Penetrating craniocerebral missile injuries in the civil disturbances in Northern Ireland. Br J Surg. Mar 1974;61(3):169-176.

154. Woodhall B. Chapter V - Head injuries in the zone of the interior. In: Coates JB, Spurling RG, Woodhall B, eds. Medical Department of the US Army in World War II - Surgery in World War II: Neurosurgery. Vol I. Washington, DC: Office of the Army Surgeon General; 1958.

155. Sahuquillo J, Vilalta J, Lamarca J, Rubio E, Rodriguez-Pazos M, Salva JA. Diffuse axonal injury after severe head trauma. A clinico-pathological study. Acta Neurochir (Wien). 1989;101(3-4):149-158.

156. Biros WH, W H. Section II System Injuries Chapter 34 Head. In: Marx J, ed. Rosen's Emergency Medicine Concepts and Clinical Practice. 5th ed. St. Louis, MO: Mosby; 2002:299-311.

157. Narayan RK. Closed head injury. In: Rengachary SS, Wilkens RH, eds. Principles of Neurosurgery. London: Wolfe; 1994.

158. Cope Z. Surgery. In: Cope Z, ed. Shock and Resuscitation. London: Her Majesty's Stationary Office; 1953:78-88.

159. Shackford SR, Wald SL, Ross SE, et al. The clinical utility of computed tomographic scanning and neurologic examination in the management of patients with minor head injuries. J Trauma. Sep 1992;33(3):385-394.

160. Warren WL, Jr., Bailes JE. On the field evaluation of athletic neck injury. Clin Sports Med. Jan 1998;17(1):99-110.

161. Alves WM, Polin RS. Sports-related head injury. In: Narayan RK, Wilberger JE Jr, Povlishock JT, eds. Neurotrauma. New York, NY: McGraw-Hill; 1996.

162. Mueller FO, Blyth CS. Fatalities from head and cervical spine injuries occurring in tackle football: 40 years' experience. Clin Sports Med. Jan 1987;6(1):185-196.

163. Buckley WE. Concussions in college football. A multivariate analysis. Am J Sports Med. Jan-Feb 1988;16(1):51-56.

164. Wilberger JE. Minor head injuries in American football. Prevention of long term sequelae. Sports Med. May 1993;15(5):338-343.

165. McLatchie G, Jennett B. ABC of sports medicine. Head injury in sport. Bmj. Jun 18 1994;308(6944):1620-1624.

166. Jennett B. Skull X-rays after recent head injury. Clin Radiol. Jul 1980;31(4):463-469.

167. Cheung DS, Kharasch M. Evaluation of the patient with closed head trauma: an evidence based approach. Emerg Med Clin North Am. Feb 1999;17(1):9-23, vii.

168. Schutzman SA, Barnes PD, Mantello M, Scott RM. Epidural hematomas in children. Ann Emerg Med. Mar 1993;22(3):535-541.

169. Davis RL, Mullen N, Makela M, Taylor JA, Cohen W, Rivara FP. Cranial computed tomography scans in children after minimal head injury with loss of consciousness. Ann Emerg Med. Oct 1994;24(4):640-645.

170. Miller EC, Derlet RW, Kinser D. Minor head trauma: Is computed tomography always necessary? Ann Emerg Med. Mar 1996;27(3):290-294.

171. Rivara F, Tanaguchi D, Parish RA, Stimac GK, Mueller B. Poor prediction of positive computed tomographic scans by clinical criteria in symptomatic pediatric head trauma. Pediatrics. Oct 1987;80(4):579-584.

172. Zink BJ. Traumatic brain injury outcome: concepts for emergency care. Ann Emerg Med. Mar 2001;37(3):318-332.

173. Marion DW. *Outcome from severe head injury. In: Narayan RK, Wilberger JE J, Povlishock JT, eds. Neurotrauma. New York, NY: McGraw-Hill; 1996:767-778.*

174. Jeret JS, Mandell M, Anziska B, et al. *Clinical predictors of abnormality disclosed by computed tomography after mild head trauma. Neurosurgery. Jan 1993;32(1):9-15; discussion 15-16.*

175. Chesnut RM, Gautille T, Blunt BA, Klauber MR, Marshall LE. *The localizing value of asymmetry in pupillary size in severe head injury: relation to lesion type and location. Neurosurgery. May 1994;34(5):840-845; discussion 845-846.*

176. Borczuk P. *Predictors of intracranial injury in patients with mild head trauma. Ann Emerg Med. Jun 1995;25(6):731-736.*

177. Warren WL, Jr., Bailes JE. *On the field evaluation of athletic head injuries. Clin Sports Med. Jan 1998;17(1):13-26.*

178. Bruno LA, Gennarelli TA, Torg JS. *Management guidelines for head injuries in athletics. Clin Sports Med. Jan 1987;6(1):17-29.*

179. Bullock R, Chesnut RM, Clifton G, et al. *Guidelines for the management of severe head injury. Brain Trauma Foundation. Eur J Emerg Med. Jun 1996;3(2):109-127.*

180. Patterson D. *Legal aspects of athletic injuries to the head and cervical spine. Clin Sports Med. Jan 1987;6(1):197-210.*

181. Wekesa M, Asembo JM, Njororai WW. *Injury surveillance in a rugby tournament. Br J Sports Med. Mar 1996;30(1):61-63.*

182. Dietrich AM, Bowman MJ, Ginn-Pease ME, Kosnik E, King DR. *Pediatric head injuries: can clinical factors reliably predict an abnormality on computed tomography? Ann Emerg Med. Oct 1993;22(10):1535-1540.*

183. Fulton RL, Everman D, Mancino M, Raque G. *Ritual head computed tomography may unnecessarily delay lifesaving trauma care. Surg Gynecol Obstet. Apr 1993;176(4):327-332.*

184. Gruen P, Liu C. *Current trends in the management of head injury. Emerg Med Clin North Am. Feb 1998;16(1):63-83.*

185. Haydel MJ, Preston CA, Mills TJ, Luber S, Blaudeau E, DeBlieux PM. *Indications for computed tomography in patients with minor head injury. N Engl J Med. Jul 13 2000;343(2):100-105.*

186. Jagoda AS, Cantrill SV, Wears RL, et al. *Clinical policy: neuroimaging and decisionmaking in adult mild traumatic brain injury in the acute setting. Ann Emerg Med. Aug 2002;40(2):231-249.*

187. Stiell IG, Wells GA, Vandemheen K, et al. *The Canadian CT Head Rule for patients with minor head injury. Lancet. May 5 2001;357(9266):1391-1396.*

188. Vegso JJ, Lehman RC. *Field evaluation and management of head and neck injuries. Clin Sports Med. Jan 1987;6(1):1-15.*

189. Cantu RV, Cantu RC. *Guidelines for return to contact sports after transient quadriplegia. J Neurosurg. Mar 1994;80(3):592-594.*

190. Carlsson CA, von Essen C, Lofgren J. Factors affecting the clinical course of patients with severe head injuries. 1. Influence of biological factors. 2. Significance of posttraumatic coma. J Neurosurg. Sep 1968;29(3):242-251.

191. Gerberich SG, Priest JD, Boen JR, Straub CP, Maxwell RE. Concussion incidences and severity in secondary school varsity football players. Am J Public Health. Dec 1983;73(12):1370-1375.

192. Tysvaer AT. Head and neck injuries in soccer. Impact of minor trauma. Sports Med. Sep 1992;14(3):200-213.

193. Tysvaer AT, Storli OV. Soccer injuries to the brain. A neurologic and electroencephalographic study of active football players. Am J Sports Med. Jul-Aug 1989;17(4):573-578.

194. Shapira Y, Artru AA, Yadid G, Shohami E. Methylprednisolone does not decrease eicosanoid concentrations or edema in brain tissue or improve neurologic outcome after head trauma in rats. Anesth Analg. Aug 1992;75(2):238-244.

195. Muizelaar JP. Cerebral ischemia-reperfusion injury after severe head injury and its possible treatment with polyethyleneglycol-superoxide dismutase. Ann Emerg Med. Jun 1993;22(6):1014-1021.

196. Klein RS, Berger SA, Yekutiel P. Wound infection during the Yom Kippur war: observations concerning antibiotic prophylaxis and therapy. Ann Surg. Jul 1975;182(1):15-21.

197. Savitz MH, Malis LI, Savitz SI. Efficacy of prophylactic antibiotic therapy in spinal surgery: a meta-analysis. Neurosurgery. Jul 2003;53(1):243-244; author reply 244-245.

198. Feltis JJ. Surgical experience in a combat zone. Am J Surg. Mar 1970;119(3):275-278.

199. Djindjian M, Lepresle E, Homs JB. Antibiotic prophylaxis during prolonged clean neurosurgery. Results of a randomized double-blind study using oxacillin. J Neurosurg. Sep 1990;73(3):383-386.

200. Geraghty J, Feely M. Antibiotic prophylaxis in neurosurgery. A randomized controlled trial. J Neurosurg. Apr 1984;60(4):724-726.

201. van Ek B, Dijkmans BA, van Dulken H, van Furth R. Antibiotic prophylaxis in craniotomy: a prospective double-blind placebo-controlled study. Scand J Infect Dis. 1988;20(6):633-639.

202. Young RF, Lawner PM. Perioperative antibiotic prophylaxis for prevention of postoperative neurosurgical infections. A randomized clinical trial. J Neurosurg. May 1987;66(5):701-705.

203. Blomstedt GC, Kytta J. Results of a randomized trial of vancomycin prophylaxis in craniotomy. J Neurosurg. Aug 1988;69(2):216-220.

204. Gaillard T, Gilsbach JM. Intra-operative antibiotic prophylaxis in neurosurgery. A prospective, randomized, controlled study on cefotiam. Acta Neurochir (Wien). 1991;113(3-4):103-109.

205. Bowen TE, Bellamy RF. Emergency War Surgery Second United States Revision of The Emergency War Surgery NATO Handbook. Washington, D.C.: United States Government Printing Office; 1988.

206. Butler FK, Jr., Hagmann J, Butler EG. Tactical combat casualty care in special operations. Mil Med. Aug 1996;161 Suppl:3-16.

207. Butler F, O'Connor K. Antibiotics in tactical combat casualty care 2002. Mil Med. Nov 2003;168(11):911-914.

208. Haines SJ. Prophylactic antibiotics. In: Wilkins RH, Rengachary SS, eds. Neurosurgery. New York: McGraw Hill; 1985:448-452.

209. Allen CH, Ward JD. An evidence-based approach to management of increased intracranial pressure. Crit Care Clin. Jul 1998;14(3):485-495.

210. Marik PE, Varon J, Trask T. Management of head trauma. Chest. Aug 2002;122(2):699-711.

211. Ward JD, Becker DP, Miller JD, et al. Failure of prophylactic barbiturate coma in the treatment of severe head injury. J Neurosurg. Mar 1985;62(3):383-388.

212. Feldman Z, Kanter MJ, Robertson CS, et al. Effect of head elevation on intracranial pressure, cerebral perfusion pressure, and cerebral blood flow in head-injured patients. J Neurosurg. Feb 1992;76(2):207-211.

213. Sano T, Drummond JC, Patel PM, Grafe MR, Watson JC, Cole DJ. A comparison of the cerebral protective effects of isoflurane and mild hypothermia in a model of incomplete forebrain ischemia in the rat. Anesthesiology. Feb 1992;76(2):221-228.

214. Fessler RD, Diaz FG. The management of cerebral perfusion pressure and intracranial pressure after severe head injury. Ann Emerg Med. Jun 1993;22(6):998-1003.

215. Cruz J, Minoja G, Okuchi K. Improving clinical outcomes from acute subdural hematomas with the emergency preoperative administration of high doses of mannitol: a randomized trial. Neurosurgery. Oct 2001;49(4):864-871.

216. Cruz J, Minoja G, Okuchi K. Major clinical and physiological benefits of early high doses of mannitol for intraparenchymal temporal lobe hemorrhages with abnormal pupillary widening: a randomized trial. Neurosurgery. Sep 2002;51(3):628-637; discussion 637-628.

217. Tommasino C. Fluids and the neurosurgical patient. Anesthesiol Clin North America. Jun 2002;20(2):329-346, vi.

218. Mirski AM, Denchev ID, Schnitzer SM, Hanley FD. Comparison between hypertonic saline and mannitol in the reduction of elevated intracranial pressure in a rodent model of acute cerebral injury. J Neurosurg Anesthesiol. Oct 2000;12(4):334-344.

219. Fisher B, Thomas D, Peterson B. Hypertonic saline lowers raised intracranial pressure in children after head trauma. J Neurosurg Anesthesiol. Jan 1992;4(1):4-10.

220. Gemma M, Cozzi S, Tommasino C, et al. 7.5% hypertonic saline versus 20% mannitol during elective neurosurgical supratentorial procedures. J Neurosurg Anesthesiol. Oct 1997;9(4):329-334.

221. Gunnar W, Jonasson O, Merlotti G, Stone J, Barrett J. Head injury and hemorrhagic shock: studies of the blood brain barrier and intracranial pressure after resuscitation with normal saline solution, 3% saline solution, and dextran-40. Surgery. Apr 1988;103(4):398-407.

222. Hartl R, Ghajar J, Hochleuthner H, Mauritz W. Hypertonic/hyperoncotic saline reliably reduces ICP in severely head-injured patients with intracranial hypertension. Acta Neurochir Suppl. 1997;70:126-129.

223. Qureshi AI, Suarez JI, Bhardwaj A, et al. Use of hypertonic (3%) saline/acetate infusion in the treatment of cerebral edema: Effect on intracranial pressure and lateral displacement of the brain. Crit Care Med. Mar 1998;26(3):440-446.

224. Schwarz S, Schwab S, Bertram M, Aschoff A, Hacke W. Effects of hypertonic saline hydroxyethyl starch solution and mannitol in patients with increased intracranial pressure after stroke. Stroke. Aug 1998;29(8):1550-1555.

225. Schwarz S, Georgiadis D, Aschoff A, Schwab S. Effects of hypertonic (10%) saline in patients with raised intracranial pressure after stroke. Stroke. Jan 2002;33(1):136-140.

226. Shackford SR, Zhuang J, Schmoker J. Intravenous fluid tonicity: effect on intracranial pressure, cerebral blood flow, and cerebral oxygen delivery in focal brain injury. J Neurosurg. Jan 1992;76(1):91-98.

227. Suarez JI, Qureshi AI, Bhardwaj A, et al. Treatment of refractory intracranial hypertension with 23.4% saline. Crit Care Med. Jun 1998;26(6):1118-1122.

228. Worthley LI, Cooper DJ, Jones N. Treatment of resistant intracranial hypertension with hypertonic saline. Report of two cases. J Neurosurg. Mar 1988;68(3):478-481.

229. Qureshi AI, Suarez JI. Use of hypertonic saline solutions in treatment of cerebral edema and intracranial hypertension. Crit Care Med. Sep 2000;28(9):3301-3313.

230. Lanier WL. Glucose management during cardiopulmonary bypass: cardiovascular and neurologic implications. Anesth Analg. Apr 1991;72(4):423-427.

231. Lam AM, Winn HR, Cullen BF, Sundling N. Hyperglycemia and neurological outcome in patients with head injury. J Neurosurg. Oct 1991;75(4):545-551.

232. Rovlias A, Kotsou S. The influence of hyperglycemia on neurological outcome in patients with severe head injury. Neurosurgery. Feb 2000;46(2):335-342; discussion 342-333.

233. Ghajar J, Hariri RJ, Narayan RK, Iacono LA, Firlik K, Patterson RH. Survey of critical care management of comatose, head-injured patients in the United States. Crit Care Med. Mar 1995;23(3):560-567.

234. Ropper AH, Rockoff AM. Treatment of intracranial hypertension. In: Ropper AH, Kennedy SF, eds. Neurological and Neurosurgical Intensive Care. Rockville, MD: Aspen Publications; 1988:9-22.

235. Bullock R. Hyperventilation. J Neurosurg. Jan 2002;96(1):157-159.

236. Chesnut RM. Hyperventilation versus cerebral perfusion pressure management: time to change the question. Crit Care Med. Feb 1998;26(2):210-212.

237. Chesnut RM. Hyperventilation in traumatic brain injury: friend or foe? Crit Care Med. Aug 1997;25(8):1275-1278.

238. Yundt KD, Diringer MN. The use of hyperventilation and its impact on cerebral ischemia in the treatment of traumatic brain injury. Crit Care Clin. Jan 1997;13(1):163-184.

239. Stocchetti N, Maas AI, Chieregato A, van der Plas AA. *Hyperventilation in head injury: a review. Chest. May 2005;127(5):1812-1827.*

240. Seelig JM, Becker DP, Miller JD, Greenberg RP, Ward JD, Choi SC. *Traumatic acute subdural hematoma: major mortality reduction in comatose patients treated within four hours. N Engl J Med. Jun 18 1981;304(25):1511-1518.*

241. Marion DW. *Neurologic Emergencies - Head and Spinal Cord Injury. Neurologic Clinics. 1998;16(2):485-502.*

242. Gunnar W, Kane J, Barrett J. *Cerebral blood flow following hypertonic saline resuscitation in an experimental model of hemorrhagic shock and head injury. Braz J Med Biol Res. 1989;22(2):287-289.*