Immediate Care of the Wounded

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Introductory Thoughts

"It will be tragic if medical historians can look back on the World War II period and write of it as a time when so much was learned and so little remembered."

Beecher H. Early Care of the Seriously Wounded Man.[1],[2]

"It is highly desirable that anyone engaged in war surgery should keep his ideas fluid and so be ready to abandon methods which prove unsatisfactory in favour of others which, at first, may appear revolutionary and even not free from inherent danger."[3],[4]

Bailey H, ed. Surgery of Modern Warfare. 2nd ed.

"Fight on, my men," Sir Andrew says,
"A little I'm hurt, but not yet slain;
"I'll but lie down and bleed awhile,
"And then I'll rise and fight again."

Ballad of Sir Andrew Barton, author unknown, c. 1550

Introduction

This text represents a thirty year culmination of my thoughts about pre-hospital combat casualty care.

My earliest introduction to this topic came in 1973 when I attended the 91A Combat Medic course in San Antonio Texas, a necessary pre-requisite before I could attend the 300 F-1 Special Forces Medic course. It was mostly poorly taught and improperly focused and fortunately I never had to take care of anyone in, or out of, a combat situation using only the knowledge gained in that course. It was run by nurses and the academic focus of the course was on nursing care skills that were mostly irrelevant to pre-hospital combat casualty care, presumably the realm of the combat medic. Some of the combat skills instruction provided by veteran combat medics with experience in the Vietnam War was a notable exception.

The 300 F-1 Special Forces Medic Course that followed was exactly the opposite; it remains to this date the best and most intense medical instruction I have ever received. I learned more relevant medical information in the short span of that course than I would ever again learn in a similar time span. Upon completion of that course I was left wondering why it took physicians four years of college, four years of medical school and several more years of internship and residency training to learn what I had learned in less than a year; I was blessedly unencumbered with the knowledge of what I didn't know and youthfully confident in my skills and knowledge of combat casualty care. I was to never have the opportunity to learn my shortcomings as a combat medic since the Vietnam War wound down faster than I completed my training. It was this training and experience as a Special Forces medic that sent me on the path to become a military physician.

What I learned in the ensuing years is that most often:

- The simple answer is the right answer,
- Well-performed basic techniques are usually better for the patient than more complicated and "sophisticated" techniques,
- Conscious inaction is better than mindless action,
- Training is more important than equipment, and
- The day I graduated from the Special Forces Medic Course I was "smarter" than I would ever be again because mostly what I learned later was all the things that I didn't, and would never, know.

What I also learned in my nearly thirty years in military medicine is that most people believe that all relevant history began the day they were born and therefore nothing much of use for the present or the future can be learned from the past. **Nothing could be further from**

the truth -- there is actually very little that is completely new in the realm of military medicine. If you are looking for the solution to a problem the first place to look is in the past because there is a good chance that someone else either already solved the same or a similar problem or at least was able to find out what didn't work.

Throughout my career in military medicine I spent a considerable amount of time as a trainer/educator; I observed how people learn, what motivates them to learn, what they are likely to remember, and what they are likely to forget. I learned that it is much better to create systems (educational or otherwise) that take into account and accommodate probable human behavior than it is to try to shape or modify human behavior.

I also learned that much, if not most, of what is believed to be true has not been proven true and further it is likely that within my lifetime much of what I have been taught will be proven to be false – this as certainly been the case over the past thirty years. I discovered that almost everything that is believed to be true about pre-hospital combat casualty care is completely unproven; this certainly does not mean it is false, just that it has not been, and will likely never be, proven true. The reason for this is because, for obvious reasons, there are no randomized, double-blind, prospective studies of pre-hospital combat casualty care; and there are remarkably few such studies of civilian pre-hospital care.

What then is the basis for modern day pre-hospital combat casualty care? It is a composite of battle proven, albeit anecdotally supported, procedures and techniques combined with civilian EMS standards of care (whether appropriate to combat casualty care or not), some of which are based upon well done studies, many of which are not.

This text represents my best efforts to glean from the pages of military medical history and from such civilian pre-hospital care and other relevant studies as have been done the evidence, weak though it may be, that supports or refutes the performance of various procedures in a pre-hospital combat casualty care environment.

In the interest of full disclosure I admit to the reader certain biases that I have acquired as a result of the observations, experiences, and education I have described above. One of these biases is against teaching pre-hospital combat casualty care providers complex and potentially hazardous medical procedures even when there is no alternative (e.g. cricothyrotomy); another is a bias in favor of better training over better equipment. I also have a bias that sometimes the most important thing is to know what not to do and when not to do it.

I independently observed that there is strong psychological predisposition toward action over inaction among pre-hospital personnel, a predisposition that is certainly characteristic of Special Operations medics/corpsmen. I had also

observed that when a procedure is taught, particularly one that seems "heroic" (e.g. cardiac massage), that the procedure will be performed much more often than indicated. This observation being epitomized by the often used, tongue-in-cheek, comment by emergency medicine residents that the indication for a particular procedure they had performed was that the RANDO criteria had been fulfilled (Resident Ain't Never Done One).

I later found that these concepts had been previously well-described by one of the fathers of modern emergency medicine, Dr. Peter Rosen, in a rather obscure July 1981 publication, Topics in Emergency Medicine, titled, "The Technical Imperative: its definition and application to pre-hospital care." The technical imperative and its implications are defined and described in the early portion of the "Airway" section in this text and I would encourage readers to obtain and read the original article for greater detail (unfortunately it is hard to find in most libraries – I eventually found a copy in the library of the National Fire Academy in Emmitsburg Maryland).

One of the reviewers of this material suggested that it be re-written to appeal to a larger audience, noting that it is presented in the manner of a textbook. I was pleased by this comment because that is exactly what I intended. The primary intended audience for my work has been, from the beginning, military medicine trainers/educators and medical commanders who must make decisions as to what should be taught and to whom, and what material should be provided to those who provide pre-hospital combat casualty care.

The material in this book was originally written for inclusion in an unpublished book, Combat Surgery, to be part of the multi-volume "Textbook of Military Medicine" series published by the U.S. Army's Office of the Surgeon General. Unfortunately this book was never completed despite its obvious importance as a key volume in any series on the subject of military medicine, and the whole Textbook of Military Medicine project has, I am told, been discontinued. Thus as I finished my military career I was left having done a considerable amount of work for a book that, it seemed, was never to be published.

So the situation remained until I was contacted about a year ago by CAPT (fmr) Mike Hughey MD, a fellow military medicine educator and friend. Mike has a medical education website,

http://www.brooksidepress.org

that provides military medicine oriented educational material. He inquired as to whether I had any unpublished material on the subject of military medicine that I would like to get published? I replied that I certainly did and provided him with several megabytes of material that I used throughout my career, some of which I am pleased to see have already been incorporated into his Website. I also told him about this unpublished work which he encouraged me to com-

plete and offered to provide some editing services as well as a place to publish the material. Finally after more than four years in the writing and thirty years in the conceptualization here it is.

The commonly used ABCD approach is utilized to frame the discussion of the management of potentially life threatening problems in the pre-hospital combat environment. Historical information and current studies are incorporated to provide the reader an evidentiary basis upon which to make decisions as to what is likely to provide casualties benefit in a pre-hospital combat setting and what is either likely to be ineffectual or possibly harmful. Where it seems reasonable, based upon the available evidence, to offer what I believe is a best practice I have done so, but mostly the reader is given the available evidence and is left to decide for him or herself what materials, techniques, and procedures are most appropriate for pre-hospital combat casualty care providers.

Acknowledgements

I would like to take the opportunity here to thank those who made this work possible; most importantly Mike Hughey who has provided a venue for the material and who has also provided considerable editorial support. I would also like to thank Dr. Robert Joy, Professor Emeritus at the Uniformed Services University of the Health Sciences, COL(ret) Ron Bellamy MD, Lt. Col. John Wightman MD, USAF and COL Candice Castro MD, for their detailed reviews of my work and their helpful feedback. Others too numerous to cite individually have also reviewed this work and have offered helpful suggestions and for their comments I am also appreciative. Finally I would like to thank my wife Ok Cha for her patience and support while I labored on this work to the exclusion of, at times, all else.

About the Author

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Dr. Cloonan is currently a staff emergency medicine physician at Carlisle Regional Medical Center in Carlisle Pennsylvania. He is also an Associate Professor at the Uniformed Services University of the Health Sciences in Bethesda Maryland.

Prior to his retirement from the United States Army in January 2004 Dr. Cloonan was the Interim Chairman of the Department of Military and Emergency Medicine at the Uniformed Services University. From 1999 to 2002 he was the Consultant to the Surgeon General for Emergency Medicine and he served as the Department of Defense representative to the National Registry for Emergency Medicine Technicians from 2001 to 2004.

COL Cloonan was born January 16, 1954 in Boulder Colorado. He was an enlisted soldier assigned to the 10th Special Forces at Fort Devens Massachusetts between 1972 and 1975. Initially trained as an infantryman with specialized training in light and heavy weapons, he was later trained, and spent most of his enlistment, as a Special Forces medic.

After his discharge as a Sergeant in August 1975 he joined the reserves and began his undergraduate schooling at California Polytechnic State University at San Luis Obispo. He majored in biochemistry and was in the university's ROTC program, serving as the ROTC cadet battalion commander during his last year and receiving the George C. Marshall Award. While in college he worked as a registered nurse in a local hospital emergency department having used his previous Special Forces medic training to challenge the state registered nurse exam.

In the fall of 1979 he began medical school at the Uniformed Services University of the Health Sciences (USUHS), graduating with honors in 1983. Following a transitional internship at Brooke Army Medical Center in San Antonio Texas and after a brief stint as an instructor at the SF Aidman course, he attended the Army's Flight Surgeon course and spent the next 15 months in Korea as a flight surgeon with the 43rd MASH.

Upon returning to the U.S. he attended the Medical Officer Advanced Course and in July 1986 he began his emergency medicine residency at Madigan Army Medical Center, Ft Lewis Washington, serving as one of the chief residents.

After residency he was assigned to Tripler Army Medical Center in Hawaii where he served as a staff physician. In addition to his regular duties he served in a volunteer capacity in support of Joint Special Opera-

tions out of Ft Bragg North Carolina. In this role he was in combat with the Forward Surgical Team of the 44th Medical Brigade at Howard Air Force Base, Panama during Operation Just Cause.

Also while at Tripler he served as the head of the hospital's Emergency Medical Response Team (E.M.R.T.) which was responsible for providing emergency medical backup for Johnston Island, a major chemical weapons storage site in the Pacific. This team was also responsible for providing regional military medical disaster assistance as required. As chief of the E.M.R.T., COL Cloonan was sent to Western Samoa in February, 1990 as part of a team to assess the damage caused to that country by Cyclone Ofa.

He was next assigned to the Uniformed Services University of the Health Sciences (USUHS) medical school in August 1990 as an assistant professor in the Department of Military and Emergency Medicine. While at USUHS from 1990-1993 COL Cloonan was the course director for both the Combat Medical Skills course and for the Introduction to Combat Casualty Care course and he was Director of the Department's Basic Science Division.

In addition to his teaching duties at USUHS Dr. Cloonan also provided medical support for several federal law enforcement agencies involved in high risk law enforcement activities. In this capacity he provided medical support for the FBI Hostage Rescue Team during the Ruby Ridge standoff and during the Branch Davidian Waco Texas incident as well as medical support for numerous less high profile local area SWAT missions.

From July 1993 through June 1994, COL Cloonan attended the US Marine Corps Command and Staff College at Quantico, Virginia, obtaining a Master of Military Studies and graduating as the top army student. He then served as Division Surgeon for the 2D Infantry Division in Uijongbu South Korea following which he was Chief of Emergency Medical Services at the 121 General Hospital in Seoul.

From August 1996 to January 1997 COL Cloonan served as the Chief of the Emergency Department at Womack Army Medical Center, Fort Bragg, before becoming Dean of the Joint Special Operations Medical Training Center. Prior to his second assignment to the Department of Military and Emergency Medicine at the Uniformed Services University, from January 1977 to July 2000, COL Cloonan served as the Dean of the newly established Joint Special Operations Medical Training Center within the JFK Special Warfare Center and School at Fort Bragg, North Carolina where all Special Operations enlisted medical personnel within the Department of Defense receive their training.

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Airway

And when Elisha was come into the house, behold, the child was dead, and laid upon his bed. And he went up, and lay upon the child, and put his mouth upon his mouth…and the flesh of the child waxed warm…and the child opened his eyes

II Kings Chapter 4 verses 32-35, King James Version

"Of all the surgical operations which are performed in man for the preservation of life by the physician, I have always judged to be the foremost that by which man is recalled from a quick death to a sudden repossession of life, a feat which raises to the level of Aesculapius; the operation is the opening of the asperia arteria [trachea], by which patients, from a condition of almost suffocating obstruction to respiration, suddenly regain consciousness, and draw again into their heart and lungs that vital ether the air, so necessary to life, and again resume an existence which had been all but annihilated." [1]

Hieronymus Fabricius 1537–1619
Italian Anatomist and Surgeon

"Wounds of the face, when they do not implicate the brain, are not usually of a serious character."

Dr. Julian John Chisolm, Confederate Surgeon
Manual of Military Surgery, Chapter 8 *Wounds of the Face* p. 268

The mental image suggested by the term "gunshot wound of the face and jaws" invariably induces an emotion of sympathy, pity, or out and out revulsion. While numbering few in the total of war casualties, they demand and deserve every conceivable effort in the matter of medical care[2]

"Maxillo-Facial Injuries" by Major Henry B. Clark in
Forward Surgery of the Severely Wounded
(A History of the Activities of the 2nd Auxiliary Surgical Group)
World War II

Introduction

Having a patent airway is an essential prerequisite to life. Of this, there is no dispute. All textbooks on pre-hospital care identify airway management as the preeminent skill in resuscitation because humans can only briefly tolerate anoxia; if there is no airway, there is no life. Although there is no argument about these fundamental facts, almost every other aspect of pre-hospital airway management has become disputable, particularly in a combat environment. The fundamental questions in these debates are not whether airway management is important, but rather (1) how likely is airway obstruction, which is amenable to and requires medical correction, to be a problem in a pre-hospital military environment and, (2) after establishing that airway management must be done,

- When should it be done;
- Who should do it;
- How should it be done; and
- What equipment or medications, if any, should be used?

How likely is airway obstruction to be a medically correctable problem in a population of military patients; more specifically how likely is it to be a problem in a population of combat casualties? Although basic airway management should be part of every pre-hospital care provider's array of skills, if airway obstruction is an extremely rare medical problem in the population of interest (living combat casualties), teaching intubation may result in more problems then benefits -- especially if done incorrectly or when not indicated.

History of Airway Management

For most of the history of man conditions that cause complete airway obstruction have resulted in death, there being little that those in the medical professions could do to change this result. This does not mean that nothing has tried, occasionally with some success. Most of the efforts have been surgical but, particularly in more recent history, not all. Stock, in his "A Short History of the Development of the Tracheostomy",[4] noted that the Babylonian Talmud (AD 352-427) makes reference to a case of swelling of the throat (possibly referring to a peritonsilar abscess) saying that while a transverse division of the trachea will be fatal, a longitudinal section will not; suggesting that surgery had been tried for this condition, perhaps with some success. For some cases of cynanche [infection/inflammation of the upper airway with associated difficulty breathing] surgical intervention has been suggested and even tried since not long after the death of Christ. Around 117 AD Antyllus wrote that,

> *"In cases of cynanche we entirely disapprove of the operation [tracheostomy] because the incision is wholly unavailing when all the arteries [the whole of the trachea and bronchi] and the lungs are affected; but inflammation about the mouth and palate, and in cases of indurated tonsils, which obstruct the mouth of the windpipe and the trachea is unaffected, it will be proper to have recourse to pharyngotomy, in order to avoid the risk of suffocation."*

Thus, it is clear that tracheostomy was being performed by western surgeons some 2,000 years ago.[4] In 1546 Antonio Musa Brasavalo published the first account of a tracheostomy actually being done,[5] stating that, "When there is no other possibility, in angina, of admitting air to the heart, we must incise the larynx below the abscess." Fabricius (1537-1619) described tracheostomy in detail, in glowing terms (see opening quote), but he never performed the operation himself.[1]
By the early 1600's, according to Stock, tracheostomy appears to have been considered an acceptable procedure for the treatment of upper airway obstruction. Sanctorious (1561-1636) performed tracheostomy with a trocar and recorded leaving the cannula in place for three days. Nicolas Habicot performed four successful tracheostomies in 1620; one on a 14 year old boy who had swallowed a bag of gold coins to prevent their theft whereupon the bag became lodged in the esophagus causing partial airway obstruction.[6]

Marco Aurelio Severino (1580 – 1656), a well-known anatomist and famous surgeon, performed tracheotomy several times during the Naples diphtheria epidemic, this procedure having been previously described and recommended by Guidi, Fabricius, and Sanctorius.[7] Luckhaupt, in the German Journal, *Laryngologie, Rhinologie, Otologiel,*[8] noted that while the Arabian doctor Avicenna (980-1037) described the first orotracheal intubation for the treatment of dsypnea, the real beginning of endotracheal intubation begins with this procedure being performed by obstetricians and lifesavers in the 18th century.

In 1884, after four years of experimentation, intubation was first successfully performed by Joseph O'Dwyer (1841-98).[7] Intubation of the larynx for croup was first performed by Eugene Bouchut (1856), who inserted a small thimble-like tube in the larynx, but results were so poor that it failed to replace tracheotomy. In 1880 McEwen is documented to have preoperatively intubated a patient to prevent the aspiration of blood. Later, in 1900, Franz Kuhn first performed routine intubation to keep the airway clear during narcosis.[8]

In 1950, in the first volume of the Armed Forces Medical Journal, Tarrow and Knight describe the manufacture and use of the modern, long-cuff, endotracheal tube.[9]

During the Civil War, as a new way of treating neck injuries that obstructed the airway, surgeons performed tracheostomies, which were then referred to as bronchostomies. As reported in Bollet's book, "Civil War Medicine, Challenges and Triumphs", they even left the tube in for long periods if necessary.[10] Bollet records that twenty of these procedures were performed with seven survivors. Six of the twenty were done to treat patients with gunshot wounds and the other fourteen to treat patients with diseases such as diphtheria and abscesses which threatened to occlude the airway. In at least one instance a silver tube was inserted as a tracheostomy tube that was later replaced by a "double fenestrated cannula" which the wounded soldier continued to wear six months after his initial injury.

Frequency of Airway Obstruction in Civilian Population

In a 6-month long French survey of out-of-hospital emergency intubations in the suburbs of Paris,[11] there were 691 out-of-hospital endotracheal intubations; the most common indication for this procedure being cardiac arrest (333/691 [48.2%]). Among patients without cardiac arrest, the

most common indication was unconsciousness (198/358 [55.3%]). Only 9.3% (64/691) of intubated patients were trauma patients; of these, 22 (3.2%) had multiple injuries, 31 (4.5%) had head injuries and 11 (1.6%) had burns.

Although it is unclear from the report whether any of the patients with multiple trauma also had head trauma (a common indication for intubation in trauma patients), it is clear that at least half of all pre-hospital intubated trauma patients were intubated due to head trauma (and it is almost certain that nearly all of these had sustained blunt, not penetrating trauma).

Frequency of Airway Obstruction in Combat

What is the incidence of potentially survivable airway obstruction in combat trauma patients? Estimates of the number of combat casualties needing airway management (generally defined as those needing either intubation or a surgical airway) are based upon data obtained from medical treatment facilities, not pre-hospital data, which is entirely unavailable. This likely represents only a small portion of all casualties experiencing acute posttraumatic airway obstruction, however, because such casualties have, in the great majority of instances, already survived for more than an hour prior to reaching medical care.

As stated by Bellamy in "Combat Casualty Care Guidelines -- Operation Desert Storm"[12] in the Vietnam Wounding Data Munitions Effectiveness Team (WDMET) data[13], only 1.3% of combat casualties who arrived at medical facilities required emergency airway management; and only about half of these casualties (0.7% of the total) had sustained traumatic airway injury. Another 0.6% required airway control for ventilation due to severe neurological injury.

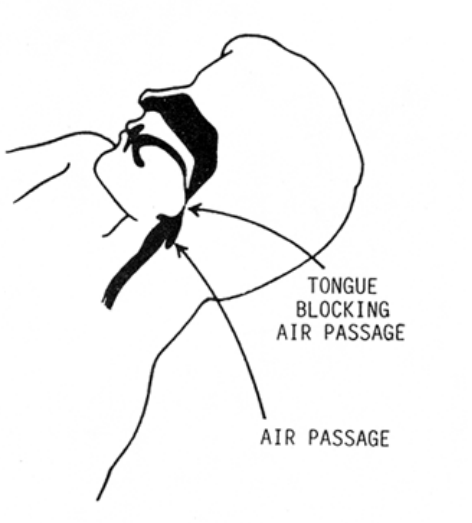
The incidence of casualties needing airway management upon arrival at a medical treatment facility in Vietnam (0.6%) is quite similar to that occurring during Operation Desert Storm in 1990. Burkle et al.[14] reported that 2 (1.2%) of 164 combat casualties admitted to the Khanjar Navy-Marine Trauma Center had an airway obstruction that required tracheotomy. It should be noted that these patients arrived alive (probably after a lengthy evacuation) without any pre-hospital surgical airway having been established, so the degree of urgency of the airway problem is unclear.

Bailey, in his World War II era text, "Surgery of Modern Warfare,"[15] noted that, "Wounds of the larynx are less commonly seen than are those of the jaws and pharynx, because of their greater mortality" and he notes that injuries to the cervical spine and great vessels of the neck are common and rapid causes of death in casualties with penetrating neck injuries.[15(p.808)][Chapter LXXI Wounds of the Air Passages and Air Sinuses – Larynx, Penetrating Wounds].

As is the case with other types of injuries [see discussion about the Soviet incidence of cervical spine injuries during WII in the section on "Disability"] the incidence of airway injuries in combat appears to be affected by the type of combat. In World War II it was noted that, "During the hedgerow fighting in Normandy, there was an unusually high incidence of maxillofacial injuries resulting from the close type of combat and the necessity for the men to expose their heads to see the enemy. These wounds were predominantly caused by small arms fire and were characterized by being exceedingly severe and

not being associated with wounds elsewhere in the body."[16]

It is also likely that the incidence of airway injuries in combat is affected by both the type of armaments used and the frequency of body armor use. Weapons that cause burn injury, for example, might create more casualties with airway injury that is not immediately fatal (caused by progressive swelling) and thus might be amenable to medical intervention. Likewise body armor, by reducing the number of casualties killed outright by fatal wounds to the thorax and abdomen, might result in an increase in the number of airway injured casualties who survive initial injury.



Airway Obstruction by Tongue, from United States Naval Hospital Corpsman 3 & 2 Training Manual NAVEDTRA 10669-C June 1989

A large civilian series of patients with facial trauma[17] found the causes of airway obstruction in trauma patients to be the following:

(1) obstruction by foreign body (e.g. vomitus)
(2) obstruction by soft tissue (e.g. tongue), hematoma, or avulsed parts
(3) disruption of the trachea

Most of these patients had mid-face fractures caused by blunt trauma and the obstruction was caused by posterior displacement of the midface. Only 17 of 1,025 facial fracture patients (1.7%) developed airway compromise that required emergency airway procedures. The authors of this study found that the rate of endotracheal intubation in patients with maxillofacial injury ranged from 2% to 6%. Because roughly 17% of seriously traumatized patients have maxillofacial trauma[17] this means that somewhere between 0.4% and 1% of all civilian trauma patients, most having blunt injury, require intubation for airway problems resulting from maxillofacial trauma.

It is worth noting that in this study the number of trauma patients requiring airway intervention for maxillofacial trauma is quite similar to that described earlier in military series despite the differences in patient populations. This is somewhat surprising because it would seem that patients sustaining blunt trauma to the head and neck would be more likely to present alive and needing urgent airway management than would those having sustained penetrating trauma.

The other major indication for intubation of civilian trauma patients is inability to protect the airway, which occurs in severely head-injured or intoxicated patients. Unfortunately, these statistics from a civilian population cannot be extrapolated to a population of combat trauma patients because, as noted by Smith and Bellamy, "Combat casualties do not have the same propensity for blunt trauma, cervical spine injury, upper airway problems, etc., as do civilian trauma victims."[18]

The specific nature of combat-related airway injury was elucidated by Rehwald[19]

(in the Textbook of Military Medicine Part IV, Surgical Combat Casualty Care: Anesthesia and Perioperative Care of the Combat Casualty, Chapter 3 Airway Management by Hecker and Kingsley[20(p.57)]). Using German data from World War II, Rehwald reported the major causes of asphyxiation associated with injury to the face and jaw as obstruction by the tongue (40%), oropharyngeal tissue (29%), or soft tissue (5%), tracheal compression (23%), and aspiration of blood and vomitus (3%).[19]

Fortunately, among those surviving long enough to reach hospital level care the mortality rate in maxillofacial wounds tends to be low. In Fifth U.S. Army hospitals during World War II, there were only 6 deaths from this cause described in 1,450 battle casualty deaths.[21]

Indications for Airway Management
Management at Point of Wounding

If airway management is necessary it usually needs to be accomplished within a few minutes of wounding if the casualty is to survive. This fact has significant implications in the pre-hospital combat casualty care airway management debate. Combat casualties who develop an airway obstruction either immediately after or within a few minutes of wounding will only survive if they themselves, or someone nearby who can reach them immediately, can clear their airway.

In general, airway clearing need not be part of self-care training provided to all combatants because individuals whose condition permits them to do so will reflexively position themselves to allow the most unobstructed breathing and to remove blood and secretions from the airway. However, at a conference conducted at the end of the Korean War, it was suggested that instruction be provided to maxillofacial-wounded casualties as to "how to hold [their] head or how to lie on the litter" because this may be lifesaving during the period of evacuation. It was also suggested that "With bleeding about the nose and mouth [such patients] should be instructed to lie in a manner that will allow the blood to drain to the exterior and not pass into the throat and cause aspiration and suffocation."[22]

If maxillofacial wounded casualties are unable to clear their own airway, then someone else must perform the necessary airway management procedure(s) or they will die quickly. The number of casualties who die within minutes of airway obstruction is not known because it is difficult to determine the precise cause of death at autopsy, particularly in those who have sustained multiple injuries. Although it is impossible to determine from the WDMET data the exact number of soldiers who died on the battlefield in Vietnam due to acute airway obstruction prior to medical intervention, autopsy results suggest that this occurrence was uncommon.[(Bellamy RF, Colonel, Medical Corps, US Army. Personal Communication, March 1995)][20(p. 56)] Unfortunately, autopsy data fail to reveal whether these casualties died from airway obstruction or with airway obstruction (exsanguination and fatal neurologic injury being common in patients with severe wounds to the head and/or neck, as was noted earlier).

Prophylactic Airway Management

Of course, not all airway obstruction occurs at or near the time of injury. There are instances, such as with inhalation injury or an expanding hematoma in the neck, where the airway should be managed well before there is any significant obstruction. There are also instances, such

as when a casualty becomes progressively less able to maintain his or her own airway (e.g. as with decreasing level of consciousness) where it is necessary for a care provider to manage the casualty's airway at a time considerably past the time of initial injury.

This type of patient is not uncommon in civilian pre-hospital care where closed head injury (expanding subdural or epidural hematoma) and drug and/or alcohol intoxication is usually the cause. Neither of these conditions, however, is common in battlefield casualties. Another category of patients needing airway management in the civilian pre-hospital setting includes those with disease processes such as severe asthma, cardiac problems, or severe pneumonia, which are rarely found in the pre-hospital battlefield patient population. It is worth noting, however, that individuals with a prior history of childhood asthma are no longer precluded from enlistment so the frequency of respiratory emergencies from this cause is likely to rise in the future.

Responsibility for Airway Management at Point of Wounding

As described in the Anesthesia volume of this series,

> *Many of the battle casualties who require immediate airway intervention will not survive to be evacuated from the site of injury to the field hospital level without first receiving airway control. For these casualties, airway intervention must take place in the first or second echelons of care…. Therefore, medics, physician assistants, and physicians providing care at the first and second echelons must be able to provide early airway management.* [20(p.57)]

Because airway obstruction must be alleviated within minutes or the patient will not survive, moving an airway obstructed patient to someone who can clear his or her airway is not an effective option in most instances due to the associated inherent time delay. From a training perspective, this means that better training in basic airway management for every soldier, during basic training and within the combat lifesaver program, is more likely to save lives than will advanced airway management training for any higher, and less immediately available, levels of medical care (See surgical airway section, below, for a discussion of training considerations for advanced airway procedures).

Decision Making in Airway Management

It is reasonable to question the efficacy of the standard civilian approach to pre-hospital airway management especially if it is to be applied to a population of combat casualties. Every intervention has consequences. Actions taken that might provide benefit in one situation might cause more harm than benefit in another. Airway management of the potentially cervical-spine injured patient is an example where the civilian approach may be inappropriate in a combat setting.

Standard Advanced Trauma Life Support (ATLS)[23] and Emergency Medical Technician (EMT) training emphasizes that because patients with head and neck injuries (i.e., injuries above the clavicle) have a higher probability of unstable cervical spine injury, the cervical spine must be stabilized in all such patients. This means that they are to be placed on a backboard, a rigid cervical collar applied, and their heads and bodies strapped and taped to the backboard. The airway of such "pack-

aged" patients is put at risk in the following ways:

- Being forcibly held in a supine position makes the patient extremely dependent upon someone else to clear his or her airway in the event that vomitus, blood, tissue, or other debris cause obstruction;
- Obstruction of the airway by the tongue is much more likely in the supine position
- The cervical collar, even if properly applied, and it often is not (and improperly applied may actually physically obstruct the airway), forces the mouth closed.

Despite these concerns, because blunt trauma is much more likely than penetrating trauma to produce an unstable cervical spine[24], and because there are usually adequate medical personnel with appropriate equipment to keep the airway clear, this is probably, nonetheless, appropriate for the majority of civilian trauma patients. Because none of these circumstances are likely to be present in forward combat areas, however, such management carries substantial risk with little hope of benefit. When the possibility of an unstable cervical spine is present in a combat casualty (e.g. a motor vehicle accident, fall from a height) and when medical vigilance can be provided and sustained to ensure that the airway remains clear, Advanced Trauma Life Support guidelines for civilian management of possible cervical spine injury remain reasonable and appropriate in the combat setting. (A detailed discussion of patient packaging for suspected spinal injuries appears in a following section on "Disability")

Unfortunately simple and basic lifesaving airway management techniques are often not well emphasized or taught in pre-hospital medical education. The very fact that they are basic and relatively simple to perform tends to cause healthcare workers to undervalue their importance. For this reason, and others, pre-hospital care providers who are taught more advanced airway skills often go directly to the use of advanced procedures even when more basic techniques would have sufficed if properly performed; occasionally with tragic consequences. The following case provided from civilian EMS literature, is illustrative:[25]

> *A 17 year old girl was an unrestrained passenger of a vehicle that struck a stationary truck. She was eventually found to have sustained a liver laceration, right pulmonary contusion, Le-Forte II/III and mandible fractures, and an acetabular fracture -- a CT of the head revealed no intracranial hemorrhage.*
>
> *Citing instability of "unstable" facial fractures as their justification, the on-scene paramedics elected to intubate the patient [perhaps the patient developed respiratory difficulty when she was placed in a supine position and secured on a backboard for cervical-spine control].*
>
> *Several unsuccessful attempts were made to orally intubate the patient without administration of a paralytic agent. When the aeromedical team arrived they described the patient as "awake, alert, and oriented X 3 with full recall of the accident." Citing again "deformed" and "unstable facial bones" and "respiratory difficulty" the aeromedical team elected to perform rapid sequence intubation of the patient using etomidate and succinylcholine. This despite that fact that there was no documented drop in*

oxygen saturation and vital signs remained unchanged. Furthermore their notes reflect that the patient had, "A normal mental status, and "clear speech."

Endotracheal tube placement was confirmed by appropriate breath sounds [notoriously unreliable under the best of circumstances-- which this was not]. No end-tidal CO_2 was recorded. The record then reflects the endotracheal tube became "dislodged" while it was being secured with ground crew assistance. At this point, "multiple" unsuccessful oral intubation attempts were made (no number recorded by the crew).

The patient's oxygen saturation fell from 96% to 88% and then to 74% over a period of 8 minutes. Bag-valve-mask ventilation was reportedly difficult so a cricothyrotomy (unsuccessful) was attempted. Cardiac arrest occurred 5 minutes prior to ED arrival.

A final pre-hospital attempt at oral intubation was successful. In the emergency department the patient was noted to be in a rhythm of pulseless electrical activity; this was successfully treated with return of spontaneous circulation. Unfortunately in the intensive care unit, multi-system organ failure ensued, brain death was declared; 55 hours after admission life support was withdrawn. Tragically, a trauma review panel concluded that <u>all of the patient's initial injuries were survivable and death resulted from hypoxia</u> due to failed airway interventions.[25]

The Technical Imperative

In July 1981, Rosen et al. introduced the concept of the "technical imperative," and applied it to pre-hospital care (although this concept actually applies to all levels of care).[26] First, the authors noted the psychological obstacle that exists in emergency medical personnel to inaction;

> *…there is a pervasive theme [in emergency care]…one of action orientation, that is, of specific technical intervention…. [B]efore addressing the individual procedures, the psychological basis underlying the performance of technical acts must [should] be considered.…[W]e select for, and demand, an aggressive, action-oriented psychological profile in our personnel. The consequence of this is that the hardest task for any good paramedic, nurse, or emergency physician to perform is inaction.*

Next, they described the technical imperative as follows:

> *If a procedure is taught, it will be used with a frequency greater than its indications. [Further], every procedure…has a cost that must be assessed as a complex therapeutic ratio, the deficit to the patient produced by not performing the procedure versus the deficit produced by performing the procedure "improperly" or performing it properly but unnecessarily.*

Even when medical procedures are properly performed and done only for valid, indicated, reasons, there can be life-threatening complications. Airway injury during airway management even in the best of circumstances, such as endotracheal intubation performed by anesthesiologists/anesthetists during general anes-

thesia, is a significant source of morbidity for patients.[27] Although most of these injuries are not life-threatening, some, such as pharyngeal and esophageal injury, can be. Esophageal injuries are more severe than all other types of airway injury combined and represent a significant portion of intubation-induced mortality.[28-31]

Airway Management Procedures

The ideal approach to emergent airway management of a combat casualty in a given set of circumstances depends upon the following:

- Degree of patient acuity
- Level of training and experience of the pre-hospital care provider(s),
- Tactical and environmental situation,
- Mode and distance of evacuation,
- Number of other casualties present
- Availability of medical personnel to manage the patient after the definitive airway management procedure has been performed.

When managing airway problems in combat environments pre-hospital personnel should consider using the least invasive techniques first, and refrain from using advanced procedures until they have balanced the inherent risks against the anticipated benefit. The least invasive procedures such as proper positioning, maneuvers such as the head-tilt or jaw-thrust, and clearing of the airway of secretions or debris, carry the least risk to the patient and can be most easily taught and sustained. For the majority of patients needing airway management in the pre-hospital combat environment, these procedures are usually adequate.

Basic Airway Management

As described by Bailey in World War II, in managing patients with maxillofacial wounds, "The immediate aim is to save the life of the patient." This means maintaining the airway, controlling hemorrhage, and preventing blood and other things from entering the trachea and causing airway obstruction.[15(p.811)] As noted above, these tasks can usually be effectively achieved by timely and effective application of a few simple, basic, procedures.

Body Position

During World War II the following guidance regarding proper litter evacuation of casualties was provided by the Surgical Consultant in the European Theater of Operations:

> *In the litter evacuation of fresh maxillofacial casualties, adequate attention occasionally has not been paid to the proper position for such cases. Improper position may result in fatality during evacuation. Instructions on this matter should be provided all personnel handling fresh casualties. Severe cases should be arranged in a prone position on the litter with the head supported by blankets. Such position gives the greatest assurance that the airway will be maintained and provides against the danger of aspiration of blood and oral secretions.*[32]

Also in World War II, Henry K. Beecher noted that among "common but grave errors" was the "transporting of patients in a face-up position instead of face-down, when they have pharyngeal wounds."[33] And the WWII history of the 2nd Auxiliary Surgical Group notes that "Maintenance of a clear respiratory tract may be accom-

plished by…extension of the tongue, and by postural drainage."[34] Earlier, in the Spanish Civil War (1939), Douglas Jolly, a British military surgeon, noted that following emergent treatment, patients who have sustained buccal wounds should be:

1) Placed in the prone position with a folded coat or haversack under the chest and their heads turned to the side
2) Under continuous surveillance by an orderly during evacuation and in the event that hemorrhage occurs from the face or pharynx
3) Kept by the orderly in the prone position and the orderly must swab the pharynx to prevent aspiration of blood into the glottis.[35]

Supporting these earlier observations is a recent study[36, 37] of poisoned comatose patients that demonstrated that the prone and semi-recumbent (head-down) positions are associated with a reduced incidence of aspiration pneumonia.

In the Anesthesia volume of "The Textbook of Military Medicine", Hecker & Kingsley note that,

> *Casualties with facial lacerations (common in penetrating head trauma) often present with copious bleeding that may contribute to airway obstruction. Relief of obstruction can frequently be obtained with gentle suctioning and if possible, by allowing the casualty to sit forward rather than supine. Gentle forward traction on an unstable, fractured mandible may also relieve simple obstruction."* [20(p. 74)]

As noted above, World War II data indicate that asphyxiation associated with facial and jaw wounds was due to prolapse of the tongue into the pharynx in 40% of cases and to aspiration of blood and vomitus in 3% of cases.[20(p. 57)] Given that some of the other causes of asphyxiation described in these data may have been correctable with simple airway maneuvers and/or patient positioning, it may be surmised that at least half of all cases of death from asphyxiation after maxillofacial trauma may be preventable with basic airway maneuvers and proper patient positioning.

At, and forward of, the Battalion Aid Station or similarly equipped and staffed treatment post, it is difficult to do more than the most basic of procedures so the maxillo-facial wounded casualty should be transported as soon as possible to a better equipped and staffed facility. According to Bailey, "Obvious bleeding points should be controlled…however, if the compression has the effect of increasing the amount of inhaled blood, it must then be decided to allow free external escape, for fear of asphyxiating the patient…If a suction pump is available, its use will be of great benefit in dealing with these complications."[15]

Interviews of paramedics by Karch et al. found that although failure to intubate trauma patients in the field was most common in gagging or combative patients, in nearly a quarter of survivors, inability to intubate was due to blockage of the airway with blood or vomitus.[38]

Even the ATLS course[23], which strongly advocates rapidly obtaining a definitive airway, makes it clear that not all patients with airway trauma require immediate advanced airway management. Even patients who have sustained massive trauma to the upper airway can often be managed, at least initially, using the basic airway management techniques described above. The patient shown below in Fig 1 is such a

patient. Note that this patient is not intubated and does not have a surgical airway. Only suctioning and positioning were used to keep this patient's airway clear in the emergency department.

Frequently, at the point-of-wounding, patients with such injuries have already positioned themselves to optimize their airway and if able to do so will continue to protect their airway unless interfered with by later arriving medical personnel. Sometimes well-intentioned pre-hospital personnel, while attempting to minimize the risk of injury to a potentially unstable cervical spine, will interfere with the patient's efforts to maintain a patent airway, thereby doing more harm than good. Often patients with massive facial trauma do best when allowed to sit up and lean forward, allowing blood, secretions, teeth, and soft tissue to be kept out of the upper airway.

This author personally recalls the case of a civilian patient with blunt facial trauma who developed airway obstruction as a direct result of such well-intentioned cervical spine management. The patient had been struck in the face with a baseball bat fracturing both sides of his mandible and a number of teeth. When EMS arrived the patient was on his hands and knees with blood dripping from his mouth, spitting out teeth and in pain but not in any respiratory distress. EMS personnel, following their guidelines, placed the patient in a supine position, with his cervical spine immobilized, on a backboard. When the patient arrived in the emergency department he was having marked difficulty breathing due to prolapse of his unsupported tongue into the posterior pharynx; this was alleviated with suction and outward digital traction of the jaw. In this instance well-intentioned EMS intervention almost killed the patient.

Figure 1. This man was the victim of an attempted assassination by shotgun blast to the head (turned head at moment shot was fired). The patient was responsive on ED admission. Only suctioning and positioning were used to keep this patient's airway clear in the ED. A surgical airway was placed in an operating room. Patient lost one eye and had his face reconstructed. Photo provided courtesy of Dr. Peter Rhee, military trauma surgeon

Administration of morphine to combatants with facial wounds - It was Maj. Jolly's opinion, during the Spanish Civil War, that morphine must be given early in all such cases, preferably intravenously so as to produce an immediate effect.[35(p.137)] But just a few years later, in World War II, Bailey recommended that morphine be avoided in maxillofacial wounded casualties "owing to its depressing effect on the cough reflex and respiratory centre" because, "the patient's life may depend on his ability to protect his airway by coughing."[15] On this same issue the WWII history of the activities of the 2nd Auxiliary Surgical Group noted that, "Due to mechanical respiratory difficulties in many of these cases it is wise to give morphine sparingly and in no case is it advisable to give more than ¼ grain (15 mg) every four hours."[34] Interestingly today even this smaller amount of morphine is higher than most pre-hospital personnel are comfortable with administering or, in some instances, are allowed to give.

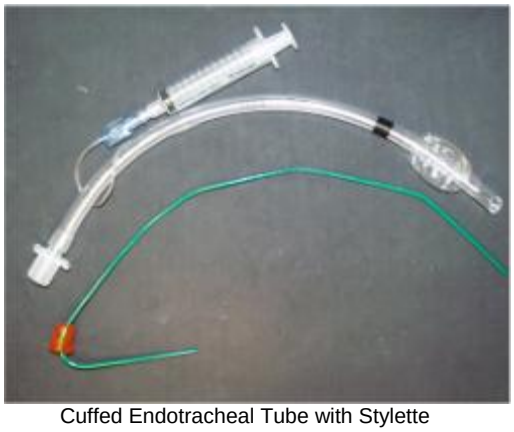
Advanced Airway Management

There is little dispute that when a patient has either significant or impending airway obstruction, establishing a definitive airway with a cuffed tube is ideal. Depending upon the circumstances, this is appropriately accomplished either by endotracheal intubation or by establishment of a surgical airway. The pre-hospital environment is, of course, a less-than-ideal surgical venue, being characterized by varying degrees of chaos; imminent danger; environmental extremes; inadequate lighting; inadequate equipment; and healthcare providers who are either untrained in, or at least relatively inexperienced in, the performance of advanced airway procedures. All of these factors decrease the likelihood of successful performance of definitive airway procedures. Under these circumstances, even if definitive airway management can be successfully accomplished without serious complications, most intubated patients not only are completely dependent upon others for every element of their survival but in the majority of situations they must be continually ventilated. In the pre-hospital combat environment, this creates a difficult, if not impossible, logistical challenge during evacuation to higher levels of care.

Indications

Emergency airway management requiring advanced airway procedures in the setting of trauma is most often indicated in cases of severe head injuries. WDMET data indicate that more than half of the 1.3% of combat casualties requiring emergency airway management had severe head injuries[20(p. 56)] [It should be noted here that Bellamy's "The Nature of Combat Injuries and the Role of ATLS in their Management" and the Anesthesia volume of the Textbook of Military Medicine[20(p.56)] have the incidence of head injuries and traumatic airway injury as an indication of intubation reversed (although both are so close – 0.6% and 0.7% -- that it is probably not a significant error)]. In a 1997study of pre-hospital cricothyrotomies done by physicians in Israel, cricothyrotomies were done on only 3.3% of all patients requiring pre-hospital airway procedures and, of these, roughly two-thirds had sustained penetrating trauma [39] (Israel may be somewhat unique among developed countries because of its disproportionately high percentage of patients sustaining penetrating trauma).

Cricothyrotomies were successfully completed in 26 of the 29 study patients (89.6%), but overall mortality in the study was 55.1%.



Cuffed Endotracheal Tube with Stylette

Efficacy

Unfortunately in many, if not most, cases, if endotracheal intubation can be performed on a severely head injured casualty without the use of paralytic and sedative drugs (i.e., when the patient is unresponsive, flaccid, or has no gag reflex) the likelihood of survival is very low. This means that, in most cases, if intubation is to help save patients with survivable injuries, rapid sequence intubation must be done. Some civilian emergency medical systems authorize paramedics to perform rapid-sequence intubation but a recent study by Dunford et al. [40] raises serious doubts about the safety of this procedure when performed by pre-hospital personnel in an out-of-hospital setting.

In this study the incidence of desaturation and pulse rate reactivity during paramedic rapid sequence intubation in 54 patients with severe head injury (Glasgow Coma Scale ≤8) was determined.[40] Thirty-one (57%) patients experienced desaturation during rapid sequence intubation, with 26 (84%) of the desaturation episodes occurring in patients whose initial SpO_2 value was greater than or equal to 90%. Therefore, patients given rapid-sequence intubation experienced more hypoxia, the very condition intubation is intended to prevent, than those managed with basic airway techniques and assisted bag-valve-mask ventilation. The extent and duration of desaturation were significant in several of these cases and six (19%) of the patients experienced marked bradycardia (defined here as a pulse rate of < 50 beats per minute so presumably a heart rate between 50 and 60 was not considered to be "marked").

Perhaps the most unsettling finding of this study is that the paramedics performing the rapid sequence intubation described the procedure as "easy" in 26 (84%) of 31 patients who experienced desaturation. Spaite and Criss[41] note that these findings make the near absence of reported significant complications in other studies of pre-hospital rapid sequence intubation seem implausible; they point out the following:

- The Dunford study, despite being carried out in a busy, metropolitan EMS system with substantial medical oversight and involving experienced paramedics, had a staggering rate of significant desaturation during rapid-sequence intubation, suggesting that an even higher rate could be expected under less optimal circumstances
- The fact that the paramedics assessed the rapid-sequence intubation procedure as "easy" despite the 84% associated morbidity suggests that other EMS studies that rely on self-reporting of com-

plications very likely suffer from significant under-reporting.

In a study of field-intubated trauma patients by Karch et al.,[38] in which roughly one-third had sustained gunshot wound(s), the following findings were noted:

- Intubated gunshot wounded patients were the least likely of all intubated patients to survive to discharge
- All surviving intubated trauma patients had head and/or facial injuries
- No patients with truncal injuries who required intubation survived
- Only slightly more than half of these patients were successfully intubated in the field
- Field intubation was three times more likely to be associated with nosocomial pneumonia than was hospital intubation (almost certainly due to a high rate of associated aspiration)[38]

Early studies show that field intubation is infrequently used in civilian trauma, perhaps because of its high failure rate. In a 1983 study of pre-hospital endotracheal intubation in Boston, only 16.2% of 178 field-intubated patients were trauma victims (e.g., blunt and penetrating injury, drowning and asphyxiation) [42] In a 1984 study of field intubation in Pittsburgh only 4% of attempted intubations were on trauma patients and of these almost one-third were unsuccessful.[43]

This failure rate in trauma patients was dramatically higher than the less than 10% failure rate cited in later studies.[44] Abraham et al. reported that based on unpublished data of field intubations performed on Israeli military trauma victims the failure rate of the first intubation attempt could exceed 40%.[45] Based on this preliminary finding the authors conducted a study to determine which factors might raise the risk of problematic intubation in soldiers. They concluded that difficult field intubations in soldiers is most probably unrelated to anatomical causes (e.g., high Mallampati score), and is more likely due to "complicated scenarios and deficient skills…."[44]

Thus much of the literature does not support intubation of combat casualties specifically showing that

- Pre-hospital intubation is rarely required for trauma patients (civilian or combat); the great majority of combat casualties who require intubation do so because they are to undergo general anesthesia and subsequent surgery;[14]
- There are virtually no survivors in intubated trauma patients who sustain other than head and/or facial trauma
- Blunt trauma to the head, neck, or face, which is proportionately much more common in civilian settings than in combat, is more likely than penetrating trauma to produce a patient who will benefit from advanced pre-hospital airway intervention
- A sizable portion of trauma patients with airway obstruction have a problem that is correctable with suctioning, basic airway maneuvers and/or proper positioning of the patient
- There is a high incidence of failure when intubating trauma patients, even in systems where the

overall intubation success rate is high

Finally, it is important to recognize, as Karch et al.[38] point out, that "Neither the success nor the complication rate for field intubation of trauma patients is known with any certainty."

Complications

Despite numerous case reports in the literature, including reports during routine anesthesia care, pharyngoesophageal perforation is an under-appreciated complication of tracheal intubation[27] that may cause life-threatening injury. Overall mortality after esophageal perforation is high (25%), even with rapid diagnosis and treatment.[45] Difficult intubation, emergency intubation, and intubation by inexperienced personnel are risk factors for pharyngoesophageal perforation.[45]

Even if military pre-hospital medical personnel are trained in advanced airway procedures, it is likely that a substantial number of intubation attempts will be unsuccessful. Despite the fact that civilian paramedics are well trained in airway management and frequently respond to patients in respiratory distress, in some systems as many as 25% of endotracheal tubes inserted by pre-hospital personnel in urban emergency medical systems are misplaced.[46] Although a number of studies showing high intubation success rates for civilian pre-hospital personnel have been published, it is unlikely that such rates could be obtained by military pre-hospital personnel even if there were an indication for the procedure. Reasons for this are as follows:

- Paramedics in emergency medical systems that report 90+% success rates for intubation generally have relatively frequent opportunities to perform indicated intubations
- These paramedics are also, in most instances, part of an emergency medical system that has an intubation skills sustainment requirement of performance of a set number of intubations under the supervision of an anesthesiologist on an annual basis
- There is also reason to suspect, in a number of instances, that the actual intubation success rates in civilian emergency medical systems studies are not actually as high as reported (Mizelle et al. note that in many reported series, intubation success is defined by the intubator and is unconfirmed by an independent observer utilizing an objective measure such as capnographic waveform analysis[25])
- Trauma, not disease, is the predominate cause of airway difficulties in a combat setting and, as was previously noted[38], the intubation success rate in trauma victims, even by experienced paramedics, is significantly lower than the average rate.

According to a 1987 study, anesthesia residents require a mean of 58 intubations before they become successful more than 90% of the time, and require more than 90 intubations to be successful more than 95% of the time.[47] If this learning curve applies to highly trained physicians working in a generally well-controlled, well-lit, well-equipped hospital environment, how can pre-hospital personnel with less training, less experience, and operating in an uncontrolled and suboptimal environment have equal

or better success rates?[25] It is also likely that serious complications are relatively common following pre-hospital intubation, particularly when the intubator is inexperienced, as most military pre-hospital providers are; and especially when the procedure is done in a chaotic combat environment. Dr. Ron Walls, a recognized expert in difficult airway management, notes that many factors increase the difficulty of intubation, including the following, many of which are present in trauma victims in a pre-hospital setting:

- Inability to achieve the necessary alignment of the geometric axes,
- Limitations to oral cavity access,
- Interference with the passage of an endotracheal tube, and
- Distortion of normal anatomy.[48]

Further compounding these difficulties is the fact that trauma patients are often uncooperative or even combative due to pain, hypoxia, and/or brain injury. All of these factors, combined with others common in combat situations such as extreme cold or heat, poor visibility (due to darkness, sand, etc.), influx of large numbers of patients at once, incoming enemy fire, and need for rapid extraction to name a few, may make intubation difficult, if not impossible, even for individuals who are experienced in airway management.

In 2004 Wang, Davis, Wayne, and Delbridge[49] did a thorough review of the evidence supporting pre-hospital rapid sequence intubation. They came to the conclusion that,

> *A growing body of evidence suggests that invasive airway management comes with a price that may outweigh any benefits with regard to oxygenation and airway protection. Thus, it may no longer be acceptable to simply arrive at the hospital with an endotracheal tube in place; instead, the means by which the ETI [endotracheal intubation] was achieved and the adverse physiologic conditions imposed upon the patient during the procedure may be more important. In fact, these results force us to ask whether early ETI itself is beneficial at all in any patient subsets.*

Establishment of a Surgical Airway

Although intubation is indicated in some cases, the majority of patients with airway obstruction resulting from combat-associated trauma can be managed successfully with simple positioning and/or suctioning; some, however, require surgical intervention. In many of those who do not respond to basic airway management techniques, endotracheal intubation is not possible for the reasons previously described.

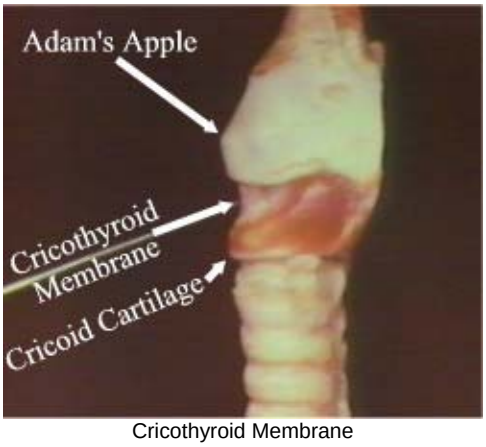
For this reason the only advanced airway technique taught by medical educators for the British Special Air Services to their highly specialized medics is a surgical airway; they do not teach endotracheal intubation. This is a logical decision given the epidemiology of combat-associated airway obstruction. It should, however, be noted that there is no evidence indicating that this approach saves more airway-obstructed casualties than intubation or even just basic airway maneuvers, or that total mortality (including those associated with inappropriately performed or unindicated surgical airway procedures) is reduced.

Cricothyrotomy Training

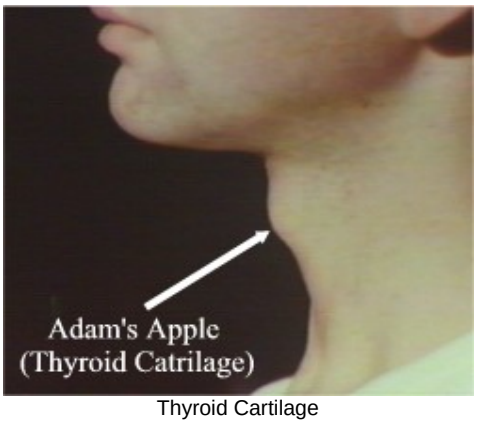
Because the "can't intubate, can't ventilate" circumstance, although quite rare, does occur, should pre-hospital personnel be taught to establish surgical airways as a last-ditch method? A recent panel discussion involving emergency medicine physicians, trauma surgeons, and combat medics conducted at the Special Operations Medical Association annual conference in Tampa Florida (December 9, 2003, moderated by this author)[50] addressed this question but did not reach a consensus.

A number of the panelists stated that all military pre-hospital personnel who are taught and authorized to perform endotracheal intubation should be taught and authorized to perform cricothyrotomies as well. There was consensus, however, that those authorized to perform cricothyrotomies must be properly trained, their competency effectively evaluated (a good simulator is needed), and their competency sustained over time.

It is important to note that both the initial and sustainment training needed to achieve and maintain competency in this procedure are relatively expensive in time, human resources, and equipment. Given the rarity of treatable life-threatening airway problems in combat casualties that require a surgical airway, it is worth asking whether these resources might be better allocated to more pervasive problems, such as ensuring highly effective hemorrhage control.



Cricothyroid Membrane



Thyroid Cartilage

Training to establish a surgical airway is problematic because it is very difficult to replicate the circumstances, anatomy, and pathophysiology of a combat casualty with a traumatically obstructed airway. In reality, patients with complete, or near-complete, obstruction of the airway are either struggling for air (not lying still) or they are very near death from hypoxia (extreme urgency); both of these situations can make the procedure difficult even for experienced physicians. In a combat setting, many patients with airway obstruction will have penetrating trauma of the neck and/or face with hemorrhage that extends into the anterior neck, distorting the anatomy (often the source of obstruction) and presenting a risk of exsanguination if a

surgical airway is attempted. Proper lighting, asepsis, and other environmental considerations are also frequently far less than ideal in a combat setting.

Current surgical airway management training is limited by the use of plastic models and cadavers which do not bleed or struggle and die. New sophisticated simulators can replicate physiology [http://www.meti.com] and thus are a significant improvement over simple plastic models and some of the new plastics are very lifelike and can even simulate bleeding when cut. Animal models such as large goats and pigs are much better for training purposes but they are expensive and the use of animals is becoming increasingly problematic. Interestingly, it is documented that as long ago as 1000 AD, Ibn Zuhr is reported to have successfully performed a tracheotomy on a goat, so this animal model for a surgical airway has a long history [http://www.entlink.net/museum/exhibits/Early-History.cfm?renderforprint=1].

Large pigs are the best animal model for surgical airway training because, of the animals used for this type of training, their anatomy is the closest to that of humans. Goats, by comparison, have a very long and superficial trachea that is easily accessible with low risk of serious hemorrhage. For this reason, it is deceptively easy to perform cricothyrotomy on a goat; successful students gain a false perception that the procedure is easy to perform in a combat casualty. The other problem with animal models is that the animals are anesthetized when the airway procedure is performed (not struggling), their anatomy is not distorted, and they generally have no airway obstruction so there is no risk of immediate death. Army combat medics in the 91W training program currently (as of 22 June 2004) receive two hours of lecture/discussion and twelve and one-half hours of practical exercise on all aspects of airway management; 30 minutes of this being dedicated to didactic teaching of a surgical airway and 4 hours spent on a practical exercise teaching a standard cricothyroidotomy. Previously this instruction had included training on the use of the Cook Melker percutaneous cricothyrotomy device[51] but, because of the lack of a inexpensive training device for the Melker airway this was discontinued.

Top: Rick Rescorla, left, and Myron Diduryk. Clinton Poley, center, in the battalion aid station. Bottom: Doc Carrara works on Arthur Viera.

Arthur Viera receiving a surgical airway by Doc Carrara at LZ X-Ray in Vietnam, in ***We Were Soldiers Once - And Young***, by Joe Galloway and Hal Moore. Viera is obviously awake, alert, and has survived for some period of time with his penetrating neck wound.

Combat medics (91W) are now taught to perform a standard emergency crichothyroidotomy with insertion of a small endotracheal tube. [91W Lesson Plan Airway Management C191W002 / Version 1 22 June 2004 - 300-91W10 1 Health Care Specialist Task 081-833-4528 Perform a cricothyroidotomy]

Sustainment skill training in cricothyrotomy skill training is a unit responsibility and units, for the most part, do not have models or devices for providing this training. The reality is as follows for most medics who would perform a surgical airway procedure on a combat casualty:

- He or she will have never before done the procedure on live tissue (animal or human)
- The last time he or she did the procedure was on a plastic manikin, more than a year before, during a mandated (not unit) training requirement
- The procedure will be unsupervised by someone experienced in the successful performance of the procedure
- The surgical environment will be far from ideal.

All of these factors suggest that when a combat medic performs a cricothyoidotomy on a casualty with an apparent airway obstruction there is a reasonably high probability that the procedure will be done when not indicated and/or it will be unsuccessful. In fact the procedure is most likely to be successfully performed on casualties in whom it is not indicated because they are less likely to have grossly distorted anatomy, they are probably not struggling, and there is likely less urgency, so the procedure can be performed in a more controlled manner. While it is certainly true that there are some situations in which a surgical airway would be indicated in a patient without an immediately critical airway obstruction, the ability of a combat medic to discern this patient from one who does not need this procedure is probably not very good.

Risks

Unfortunately, it is quite possible that although some patients could be saved by establishment of a surgical airway, almost as many may be killed or permanently harmed by the procedure itself. Inexperienced medics in the field are not only less likely to effectively manage an airway using non-surgical methods but they have a higher likelihood of complications and a lower likelihood of success when attempting to open an airway surgically. If the adverse outcomes only occurred in patients who truly had airway obstruction that couldn't be relieved with less-invasive methods, there would be no harm in attempting to establish a surgical airway (no matter how unskilled the medic) because otherwise these patients would surely die. Unfortunately, the less trained and less experienced the provider, the more likely it is that a surgical airway will be attempted when it is not indicated; which subjects patients who do not stand to benefit from the procedure to increased risk of complications and death.

It is worth noting that the recipient of the first recorded "successful" cricothyrotomy, performed in 1852, later died from airway stenosis.[52] Cricothyrotomy is associated with a historically high rate of difficult-to-manage subglottic stenosis and other complications that include life-threatening hemorrhage, airway obstruction, bronchospasm, cuff leak, esophageal perforation, paratracheal insertion, aspiration, infection, pneumomediastinum, pneumothorax, subcutaneous emphysema, or tracheoinnominate fistula.[53] Given the rarity of combat trauma patients sustaining airway obstruction that requires surgical intervention, it is

probable that the number of times this procedure would be done when not indicated is likely to greatly exceed the number of times it would be done when indicated; and some percentage of these casualties will suffer procedure-associated complications.

Although similar concerns exist about the establishment of a surgical airway when performed by a physician's assistant or medical officer, these individuals should have had more training and more experience than the typical combat medic. The Israeli experience, as reported in a 1997 study of physician-performed pre-hospital cricothyrotomies, is that cricothyrotomy performed in the field by physicians, even ones inexperienced in the performance of cricothyrotomy is successful in most (89.6%) cases.[54] In this study cricothyrotomies were done on only 3.3% of all patients requiring pre-hospital airway procedures; nearly two-thirds having sustained penetrating trauma (Israel may be somewhat unique among developed countries because of its disproportionately high percentage of patients sustaining penetrating trauma). It is, however, unclear how these cricothyrotomies were deemed successful or unsuccessful. Because there is no indication in this study that autopsies were performed on those who died, it is not possible to determine whether the cricothyrotomy was an associated cause of death or even whether the procedure had been performed properly

Statistical analysis notwithstanding, the reported success rate in the above study may well be inflated because:

- Overall mortality was 55.1%
- Lack of autopsy confirmation of airway success or failure among the dead makes it unclear how many attempted cricothyrotomies were successful [Note: the authors do suggest that the procedure was not the cause of death stating "Since the RTS and GCS were significantly higher among the survivors, it appears that the high mortality rate in this series was injury-related" and was not related to the procedure.[54]]
- Success was not defined using any objective measure such as confirmation of end-tidal CO2. (i.e., it was determined subjectively by the performer of the procedure, was not independently verified, and was defined as the establishment of an effective airway and achievement of "reasonable" ventilation as determined by auscultation).

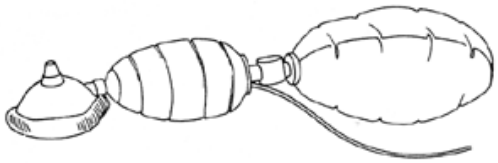
There is also reason to believe that some of the patients reported in this series did not actually need a surgical airway. Three patients, despite having an unsuccessful cricothyrotomy and experiencing associated complications, arrived at the hospital alive, surviving both their "airway obstruction" and their unsuccessful surgical airway attempt. These issues aside, the authors note that following brief training (e.g., the ATLS course) physicians are apparently capable of performing emergency cricothyrotomies in the field and their success rate without complications appears to be unrelated to medical specialty.

Certainly in the context of the failed airway, cricothyrotomy provides the ultimate fallback airway management technique and, as such, it should be in the armamentarium of the forward-deployed physician's assistant and medical officer.[48]

Equipment - Non-surgical Airway Management
Bag-Valve Mask

Although a bag-valve mask is an essential item of equipment for assisted ventilations, it is reasonable to question its utility forward of a Battalion Aid Station. In a civilian setting respiratory arrest in patients with non-traumatic injury is not uncommon and many such patients can be saved with ventilatory assistance. Further, respiratory arrest in civilian trauma patients is generally due to closed head injury; some of which can be saved with ventilatory assistance. In combat, however, trauma-associated respiratory arrest is usually due to penetrating trauma, and very few patients with such injuries can be saved even with good ventilatory assistance. As is true with advanced airway procedures there are certainly some combat-associated circumstances for which ventilatory assistance may be life-saving in the forward combat areas; these include, but are not limited to:

- Closed head injury
- Electrical shock
- Lightning injury
- Chemical warfare (particularly those involving the use of nerve agents)
- Drowning.



Bag-Valve Mask, from from United States Naval Hospital Corpsman 3 & 2 Training Manual NAVEDTRA 10669-C June 1989

Not only is the bag-valve mask unlikely to favorably improve the outcome of most combat casualties, but most providers find it difficult to use this device effectively as a sole provider. The American Heart Association in their Advanced Cardiac Life Support Course clearly state that, "a single rescuer may have difficulty providing a leakproof seal to the face while squeezing the bag adequately and maintaining an open airway." For this reason, manually operated, self-inflating bag-valve-mask units are used most effectively by at least two well-trained and experienced rescuers working together."[55] A smaller and lighter item, which is can be much more effectively used by a single person, is the pocket mask. For the majority of far-forward, point-of-wounding, circumstances the pocket mask is not only sufficient but is a superior choice.

Suction Devices

As indicated earlier, vomitus, blood, and tissue are common causes of airway obstruction in combat casualties. Most, but not all, of these common causes of obstruction can be cleared by proper casualty positioning and digital clearing of the airway. Various suctioning devices are available to aid in the clearing of liquids and small particulate matter from the airway. The ideal battlefield suction device should be:

- Lightweight
- Compact
- Durable
- Effective
- Hand operated.

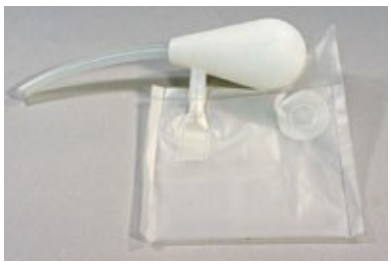
Many of the suction devices used by civilian EMS personnel are electrically operated. In addition to requiring electrical power such devices are generally

too bulky and too heavy for use by combat medics who must rely on hand-operated devices.

V-VAC™ Manual Suction Unit (Photo courtesy of Laerdal Medical Corporation)

In a recent evaluation of several commonly used, commercially available, hand-operated suction devices (along with some prototype devices and a 60-mL Toomey syringe) by 17 Special Operations Forces enlisted medical personnel, the modified Delee suction device and the V-VAC™ Manual Suction Unit (whose major drawback was its bulky, non-compressible, size) were preferred.[56]

As a field-expedient, small, simple, readily-available suction device, the 60-mL Toomey syringe, although less than ideal because it does not come with an aspirator tip, it has a narrow opening and thus is not effective at removing most thicker and particulate matter, and because it has a limited capacity, seems to be the best of the options currently available because of its ease of use and ready availability.



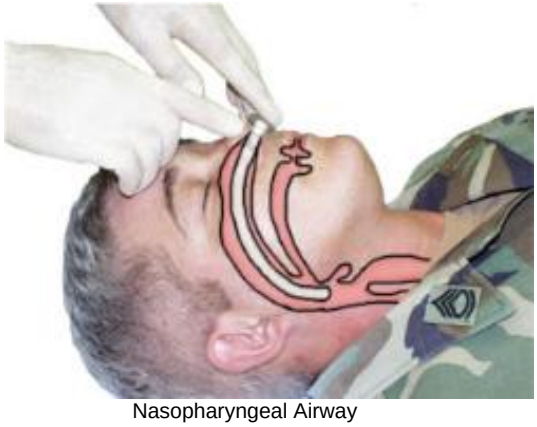
An aspirator, called the SuctionEasy™ disposable suction is now available that meets several of the characteristics of an ideal combat suction device. It is essentially a modified and enlarged infant bulb aspirator with a reservoir that can hold up to 1000 cc's of aspirated material.

It is simple to use, has no moving parts, is lightweight and reasonably compact, has an aspirator tip as large as that on most commercially available suction devices for pre-hospital use, and is capable of generating a vacuum force of approximately 100 mm Hg – more than sufficient suction to clear blood and vomit from an adult airway. This suction device might also be used in lieu of an esophageal detector device to confirm endotracheal tube placement (see following discussion below) but it has not been evaluated for this application nor has it been tested against other suction devices to determine its efficacy in the hands of military pre-hospital personnel.

Airway Adjunct

Almost by definition, any patient who can tolerate proper placement of an oropharyngeal airway needs to be intubated, and probably needs at least some assisted ventilation. In conventional combat the majority of casualties in whom an oropharyngeal airway would be indicated have severe neurological injuries or are in severe shock. Survival of such patients, even when properly managed and under relatively ideal circumstances, is unlikely.


Oropharyngeal Airway


Nasopharyngeal Airway

For these reasons, in combat, oropharyngeal airways, even if properly used, are unlikely to have much effect on morbidity or mortality. An alternative airway adjunct is the nasopharyngeal airway. This airway can be used in both responsive and unresponsive patients and, if used properly, can be safely inserted in most patients with facial trauma.

Airway Device

There are currently available a number of airway devices that do not require direct visualization of the vocal cords and are designed to be inserted blindly.[57] Earlier devices such as the esophageal obturator and esophageal gastric tube airways were single-lumen airways designed to obstruct the esophagus and direct airflow into the trachea. These devices were associated with an increased risk of death, frequently because of unrecognized tracheal instead of esophageal obstruction; they have been described a prominent trauma surgeon, Ken Mattox, as being "instruments of the devil."

Some of the newer devices are double-lumen devices that can be inserted either into the trachea or the esophagus and, if the proper ventilation tube is selected, allow for effective assisted ventilation with a bag-valve mask. The Combitube™ [http://www.life-assist.com/combitube], pharyngotracheal lumen airway [http://www.gettig.com/ptl], and the laryngeal tube airway are examples of devices that have a large-volume balloon that inflates in and seals the posterior pharynx and a distal balloon of smaller volume that inflates in the trachea or, more often, in the esophagus.[58-60]

With these devices it is critical to correctly identify which port to ventilate.[61] After confirming the location (trachea or esophagus) of the distal tip of the airway, which is best accomplished using an end-tidal CO2 detector, ventilation is provided through the proper port. In addition to helping secure a patent airway, the upper, large-volume balloon on these devices can potentially compress and control bleeding in the upper airway and thus, in some instances these devices may be the ideal airway device for some patients with maxillofacial trauma. The pharyngotracheal lumen airway was evaluated for its ability to control simu-

lated upper airway hemorrhage and was found to be effective.[62]

Another type of blind insertion device is the laryngeal mask airway. When inserted this device creates a seal around the upper portion of the airway in the posterior pharynx thus directing air into the trachea. All of these devices have been tested both by anesthesiologists in an operating room setting and by prehospital personnel in a field setting and have been found to provide effective ventilation comparable to endotracheal intubation if used properly.[63] In a study of endotracheal tube, laryngeal mask airway, and Combitube™ use by Navy Seal and Reconnaissance combat corpsmen under simulated combat conditions, insertion time for the laryngeal mask airway (22.3 seconds) was significantly shorter than the insertion times for the other devices.[64] The corpsmen easily learned how to use the Combitube™ and laryngeal mask airway, and had a similar rate of attempts for all devices (1.17 to 1.25). The authors concluded the following:

> *Simple airway devices that allow for easy learning, rapid insertion, and minimal deterioration in skill over time could significantly improve corpsmen's' ability to care for patients in combat conditions*.

It should be noted that complete airway protection is only provided in those instances in which a cuffed airway is placed in the trachea. Some of these blind insertion devices can easily be converted by a physician to a cuffed endotracheal tube in a controlled environment. Although generally safe and easy to use, these devices are associated with occasional complications. In 91W training it is the Combitube™ that is taught for advanced airway management.[65]

Devices to Confirm Endotracheal Intubation

Although endotracheal intubation is not generally indicated for most combat trauma patients (see above discussion), if it is performed, it is absolutely essential that confirmation be obtained that the trachea, and not the esophagus, has been intubated. The former gold standard of observing the tube pass through the vocal cords, confirmation of bilateral breath sounds on ventilation, and absence of ventilation sounds over the stomach is inadequate.[66] The new prehospital gold standard is capnographic confirmation of the excretion of carbon dioxide, usually with a colorimetric CO2 detector. Another commonly available, field-durable, and effective confirmation tool is the esophageal detector device, or EDD. This is a bulb-suction device that relies upon the fact that when suction is applied to the end of the endotracheal tube, the esophagus, but not the trachea, will collapse. Confirmation of tracheal intubation is obtained when the EDD is placed on the end of the ET tube, compressed, and rapidly refills when compression is released. Either, or preferably both, of these confirmation tools should be used any time endotracheal intubation is performed.[66-71]

No single method is completely adequate to confirm proper tube placement with 100% certainty so whenever possible multiple methods should be used together to achieve the highest degree of certainty possible. Unfortunately unrecognized esophageal intubation continues to be an all-too-common cause of iatro-

genic mortality in the pre-hospital setting.

Equipment for Surgical Airway Management

It is not the intent here to describe, in detail, how to perform a surgical cricothyrotomy or to list each item needed to establish a surgical airway; this information can be found in many other places.[72] The intent is to highlight critical steps, compare different methods and equipment, and assess the efficacy of these different techniques when applied by pre-hospital personnel.

Standard Surgical Airway Equipment

The basic equipment required to establish a surgical airway using the standard technique is limited and, largely for this reason, the standard technique is often preferred. Several of the required items, such as a scalpel with a # 11 blade, gauze, hemostats, needle holder, and scissors are commonly found in medic/corpsmen aid bags.

Other helpful items that are not generally carried include a tracheal dilator, a tracheal hook, and a low-pressure cuffed tracheotomy tube such as Protex or Shiley (size 5 or 6, with an 8-mm or larger internal diameter).

Although a surgical airway can be established with just a knife and an appropriately sized, cut-down, endotracheal tube (or less[73]), this procedure, which might seem easy when performed on an anesthetized goat, can go disastrously wrong when performed with improvised, inadequate, equipment under suboptimal conditions. Proper performance of this procedure is outlined well elsewhere.[72]

Establishing a surgical airway using standard surgical technique and minimal equipment can certainly be done by a trained combat medic. Unfortunately, the circumstance in which this standard surgical approach is easiest to perform in a combat setting is when the casualty is not struggling and when there is minimal to no bleeding -- conditions most commonly found in casualties who are already dead.

Cricothyrotomy. Percutaneous Dilational Cricothyrotomy

Although the Israelis[53] and others[74] have found similar complication and success rates between the standard surgical airway approach and the Seldinger technique (and found that the standard approach was faster) others have suggested that a Seldinger or modified Seldinger technique is faster, has lower complication rates and higher success rates.[75, 76] A major problem with some of these comparative studies is that the two different cricothyrotomy techniques have been evaluated using cadavers, which don't bleed, move, or have distorted airway anatomy.[72, 75]

Toye & Weinstein[76] introduced the technique of percutaneous tracheostomy in 1969. The technique was subsequently modified by Ciaglia et al.[77] in 1985 and has gained widespread acceptance among surgeons and intensivists. The advantages of this technique include a small skin incision, less soft-tissue dissection, and wire-guided, controlled placement of the airway into the trachea. Advocates of percutaneous tracheostomy cite the ease and ability with which non-surgical specialists can per-

form this technique outside the operating room.[78]

A number of percutaneous cricothyrotomy kits are marketed (such as the Pertrach™, Nu-Trach™, Melker Emergency Cricothyrotomy Catheter Set, and the Rapitrach™, to name a few). Each of these devices utilize a similar concept, that is the insertion of a needle into the trachea, through which a guidewire is inserted (Seldinger technique) followed by dilation of the puncture site with a wire-guided dilator to facilitate placement of a functional airway.

The Pertrach™ device combines the guidewire and the dilator and uses a unique, splittable needle that is divided and removed once the combined guidewire/dilator has been introduced – this simplifies the insertion process and reduces insertion time. Both an advantage and a disadvantage of the Pertrach™ device is that it uses a cuffed airway. While a cuffed airway is ideal and reduces the risk of aspiration the cuff increases the diameter of the tube that must be inserted and thus makes insertion more difficult – significant insertion force is often required with this device.

Advantages of all of these Seldinger-type devices over standard surgical dissection include less bleeding, ease of learning, and more rapid insertion. A study by Toye & Weinstein in 1986 of 100 patients treated with an early version of the Pertrach™ device revealed a total complication rate of 14%, of which 6% was due to false passage of the device, or paratracheally rather than intratracheally.[79]

An unpublished in vivo evaluation of four cricothyroidotomy devices carried out at the Institute of Surgical Research, Brooke Army Medical Center, Fort Sam Houston found that of the devices available in 1990 (the Melker device was not available at that time), the Pertrach™ was the most rapidly inserted by non-surgically-trained practitioners into a porcine model.[80] Because each of the available Seldinger-type surgical airway devices includes some type of dilator to create an opening in the cricothyroid membrane, tissue dissection is minimized.

When a cuffed airway is to be inserted, the opening through the skin and into the trachea has to be larger than the dilator. Unless an incision is made that is large enough to facilitate easy passage of the cuffed airway, significant pressure will be required to insert the cuffed airway.[81]

Use of lubrication on the airway can help to reduce insertion forces. Special Operations medics in training at the Joint Special Operations Medical Training Center in Fort Bragg, North Carolina informally assessed the Pertrach™ and Melker devices in terms of ease of use. Their primary complaint about the Pertrach™ was that significant force was required to insert the cuffed airway. They tended to prefer the Melker device because it was easier to insert. The Melker device has a significantly smaller internal diameter than the Pertrach™ and does not have a cuff.

Although a smaller uncuffed airway may be an adequate rescue airway device it is inadequate for sustained use (beyond more than a few hours) because of the increased work of breathing and because the lack of a cuff increases the risk of aspiration. When a casualty arrives at a medical treatment facility with

a small, uncuffed, airway, the airway will have to be replaced. Although this is a disadvantage, an uncuffed airway can certainly be replaced later with a cuffed airway in the more controlled setting of the medical treatment facility. For this reason it might not be unreasonable to select from among the available surgical airway devices and techniques the one that is fastest, easiest, and least risky for pre-hospital personnel to employ, even if it must later be replaced.

Previously the Army Medical Center and School at Fort Sam Houston had taught 91W combat medics to perform percutaneous cricothyrotomy using the Melker device but this has been discontinued because the lack of a cost-effective training device limited effective training. Currently combat medics are taught to perform a standard surgical cricothryoidotomy with the insertion of a small endotracheal tube.

Evacuation of Casualties with Maxillofacial Injuries

In World War II it was found that with proper forward area medical care casualties with maxillo-facial wounds could receive delayed definitive medical care without adverse consequence. The history of the activities of the 2nd Surgical Group states that, "In the average maxillo-facial case immediate surgery is not imperative and in the presence of haemostasis, clear airway, and a reasonably comfortable patient, it is wise first to reduce shock to a minimum."[3] This history also reveals that "The mean average time from [facial soft tissue] injury to first definitive treatment was eight hours" and, "The mean average time from injury to arrival at the Center [2nd Auxiliary Surgical Group] was four days." It was noted that, "Many cases will require time-consuming operative procedures…therefore surgery should be attempted only under controlled conditions."[3]

During World War II it was determined, based on experience, that casualties with serious maxillofacial injuries were best handled in evacuation hospitals, where trained oral and maxillofacial surgeons could be readily available to treat them. It was felt that maxillofacial teams were more usefully employed in the rear areas at evacuation hospitals rather than in field hospitals. It was found that if hemorrhage could be controlled and a proper airway established and maintained that casualties with maxillofacial trauma could tolerate transportation very well if kept in a prone (not supine), or sitting up and leaning forward, position. It was, however, noted that such casualties may require the attention of special attendants during evacuation.[82]

SUMMARY

Although having a patent airway is critical to survival, advanced airway management of combat casualties by medical specialists is rarely required. Penetrating trauma, the predominant form of trauma on the battlefield, infrequently produces airway obstruction and when it does, the obstruction is usually immediate, causing death before treatment by a medical specialist is available. Further, the majority of treatable airway obstruction that occurs in combat casualties can be managed by basic airway maneuvers that include proper positioning, suctioning, digital removal of debris from the airway, head tilt/jaw thrust, and insertion of a nasopharyngeal airway.

Advanced airway management, specifically intubation and cricothyrotomy, is very rarely required for combat casualties. At, or near, the point of wounding there is a high probability that these rarely performed procedures will be done incorrectly, when not indicated, or both – especially when performed by personnel with minimal training and inexperience with the techniques. All of this leads to a reasonable conclusion that the emphasis of airway management training for soldiers and medics needs to be on well-executed basic airway clearing maneuvers using either no airway adjuncts or simple, durable, lightweight, and uncomplicated equipment; perhaps the most important of these being effective suction. In addition to hemorrhage control, all soldiers should be taught proper casualty positioning and basic airway clearing techniques. Realistic simulators capable of recreating the types of airway obstruction likely to be found in battlefield casualties and able to be utilized in realistic combat scenarios are essential for effectively training pre-hospital personnel. Training therefore, and not equipment, should be the primary focus of efforts to improve the survival of combat casualties with airway obstruction. If military pre-hospital personnel are to be taught to perform advanced airway procedures such as endotracheal intubation, rapid sequence intubation, and/or cricothyrotomy, it is essential that they be properly trained, sustained, and evaluated to ensure competency. They must also be equipped with, and trained in the use of, airway rescue devices that are easy and safe to use and there must be some mechanism of oversight put into place to ensure that these procedures are being done properly and only when indicated. Further, it is important to conduct studies to determine whether overall survival of combat casualties is actually increased through the use of these procedures by pre-hospital personnel.

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Immediate Care of the Wounded

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Breathing

Breath is the bridge which connects life to consciousness, which unites your body to your thoughts.

Thich Nhat Hanh

Courage is a matter of the red corpuscle. It is oxygen that makes every attack; without oxygen in his blood to back him, a man attacks nothing-- not even a pie…

Elbert Hubbard

"Wounds of the chest, when taken as a class, are perhaps the most fatal of gunshot wounds"[1] (p.276)

Julian J. Chisolm, Confederate Surgeon

History and Epidemiology of Thoracic Injury and Respiratory Impairment in Combat Casualties

History

Throughout most of the history of warfare injuries of the chest, particularly penetrating injuries, have had a dismal outcome even for those surviving long enough to receive some form of medical attention. In 1761 John Hunter, an Army surgeon, noted that "usually little of value is done for those with chest wounds" but despite this he felt that, "something probably could be accomplished for the good of the patient." His only contribution was to suggest that hemothoraces might be treated by allowing the fluid to run out of the wound.[2]

During the Napoleonic Wars Baron Dominique Larrey observed that patients sustaining penetrating chest trauma in which the thoracic opening was larger than the glottis had significantly worse outcomes than those with smaller wounds. He, like John Hunter, recommended that casualties with penetrating chest wounds be placed with the injured side down to permit blood to drain out of the chest cavity but he went further to suggest that the wound in the chest should be closed.[2]

In his 1859 monograph on wounds of the chest in the Crimean War[3] Patrick Fraser recommended "paracentesis thoracis" without delay for chest injuries in which blood or serum accumulated without the ability to drain from an open wound in the chest and thus impeded the lungs. Fraser also noted that in most instances there was more danger associated with attempts to remove foreign bodies from the chest than there was to simply permit them to remain in situ; bullets may remain innocuous in the chest for long periods and attempts at removal may convert a relatively minor wound into a more serious penetrating wound.[3] In his monograph Fraser[3] tells the tale of a wounded soldier, who after tolerating being "poked at" for a long time by a surgeon finally inquired as to the point of the procedure. The surgeon said that he was searching for the bullet, to which the wounded man reportedly replied, "I wish you had said so earlier, because you will find it in my waistcoat pocket."

Epidemiology

The great majority of casualties, in most forms of combat throughout most of modern history, have sustained their wounds as the result of unaimed, usually indirect, fire. For this reason, differences in the location of wounds that might be related to the type of weapon used should result only from chance depending upon the mix of weapons and rate of fire, and on the relative frequency and extent of exposure of the various parts of the body. If these were the only factors influencing wound distribution the overall prevalence of thoracic trauma should then approximate the fraction of the body surface area that overlies the thorax, or about 16% (depending on how the thorax is defined). However, a significant portion of casualties with thoracic trauma are killed outright so the actual percentage of the casualty population requiring treatment for thoracic trauma is smaller, somewhere between 5% and 10%.[4][attributed to Beebe and DeBakey[5] (p.181)]

Differences in distribution and lethality do, however, exist among various weapons types, especially in relation to fatal wounds among the body regions hit. In the Korean War lethality among all those hit (killed and wounded, excluding carded-for-record-only) was highest for bullets

striking every region of the body except the upper and lower extremities. Perhaps not surprisingly this means that when small arms are commonly used (usually in close combat), the lethality of chest wounds rises because high velocity bullets tend to be lethal; especially when vital areas are hit.[6(p.44)]

As discussed in detail later, comparison of the incidence of thoracic injury in various different wars is hindered by the general lack of a clear definition of what constitutes a thoracic injury. Furthermore there are differences in incidence due to some portion of casualties never being recorded. For example, during the Crimean War, only 3.9 percent of wounds were recorded as involving the chest, 28.5 percent of these being fatal. In this same conflict chest wounds involving the lungs are recorded as only comprising 1.35% of all wounds; 79.26 percent of these being fatal.

Berry, in his historical section of the Thoracic Surgery volume of the Army Medical Department in World War II, noted that these numbers are considerably lower than predicted based upon body surface area (roughly 9% for the anterior chest and 9% for the upper back). He felt, therefore, that it is likely that during this war the majority of casualties sustaining wounds to the chest died on the battlefield, never making it into the medical system to have their wound recorded. Thoracic wounds, most especially in conflicts before World War II, tended to be highly lethal. The case fatality rate for wounds of the lung in the French Army during the Crimean war was reported to be 91.6 percent.[3]

During the Civil War only 8.1 percent of wounds involved the chest; 42.3 percent of these being penetrating. Again these low percentages are likely the result of not including most of those killed outright on the battlefield. During this war the overall case fatality rate for chest wounds was 27.8 percent and was 62.6 percent for penetrating chest wounds.[2(p.5)]

Of patients with chest wounds surviving long enough to be seen in a base hospital during the World War I, 70 percent had chest wounds caused by rifle or machine-gun bullets, 15 percent by bomb splinter, and 15 percent by shrapnel. At the front the incidence was almost the reverse with 40 percent of wounds being produced by bullets and 60 percent by other projectiles.[7(p.154)]

During World War I thoracic wounds accounted for only 2.6% of all hospital admissions and 8.2% of all hospital deaths. At 28.85%, the case fatality rate for wounds of the thorax during WWI remained quite high. It ranked as the third highest cause of hospital associated death behind wounds of the spine (55.85%) and wounds of the abdomen and pelvis (43.32%).[8] Berry, in his chapter "Historical Note" in volume one of Thoracic Surgery in The United States Army – Surgery in World War II series, notes that the reported incidence of thoracic wounds during WWI is suspect because the numbers are entirely at variance with the incidence reported in other recorded wars (far too low).

This low incidence may be because some wounds to the thorax were included with wounds of the neck and back, for which separate categories existed. A WWI German survey of causes of battlefield deaths conducted by Sauerbruch and discussed by Frank Berry in his chapter "General Considerations of Thoracic Wounds"[9 (p.64)] found that 30 percent of 300 battlefield deaths had wounds of the chest. A similar British survey conducted by Loeffler,[10]

that was also cited by Berry,[9(p.64)] found that of 469 WWI battlefield deaths 29 percent had chest injuries.

Jolly noted that during the Spanish Civil War the incidence of chest wounds varied with the type of warfare. The incidence tended to rise during war of movement and fall with positional or siege warfare, during which the thorax is naturally much less exposed. He found that these factors appeared to have a bearing on prognosis as well because in open warfare the percentage of bullet wounds, most of which are caused by the projectile entering at right angles, is high while in trench warfare there is a relatively high incidence of tangential wounds of the chest wall and of wounds caused by flying fragments. The prognosis for the former was far better than for the latter.[7(p. 150)]

Jolly also noted the effect that early mortality has on the frequency of certain thoracic injuries seen at medical treatment facilities, saying that, "Haemothorax as the result of wounds of the heart, the great vessels or the hilum of the lung is also very rare, for such cases usually succumb immediately.[7(p.150)]

In World War II casualties with wounds of the thorax accounted for 7.24 percent of all hospitalized wounded and 8.3 percent of all who died once hospitalized [DOW].[9(p.61)] Thoracic wounded casualties accounted for 9.3 percent of all KIA in World War II and thoracoabdominal wounds accounted for 0.16 percent.[9(p.69)]

First aid of chest injured casualties during WWI was limited to control of bleeding at the site of the wound combined with temporary wound closure by a thick dressing taped firmly over the wound.[2(p.8)] In World War I the indications for immediate thoracotomy were:

1) Presence of a sucking chest wound
2) A large retained foreign body
3) Severe bony injury
4) Injury to the diaphragm and/or
5) "Extensive" hemorrhage.[2(p.8)]

The case fatality rate for chest wounds significantly improved in World War II, as compared to World War I, but sadly the treatment of chest wounds at the beginning of WWII, immediately following Dunkirk, left much to be desired. Almost all casualties who received surgery for the chest wound they sustained at Dunkirk had wound repair without wound excision and no provision was made for drainage of the chest. This resulted in a large number of infections.

In his section of the series, "The Army Medical Department in World War II" Berry noted that, "Ironically a number of the casualties regarded as being too severely wounded to withstand surgery may have owed their lives to the fact that their wounds were not sutured closed and only a simple field dressing was applied.[2(p.32)] By the end of the war the cumulative case fatality rate for chest wounds had fallen significantly to 5.4%.[2(p.7)]

During the Korean War the thorax was listed as the site of wounding in 7%[6(p. 46)] of all casualties and the overall fatality rate for wounds of the thorax was 27%.[6(Table 46 p.44)] Penetrating trauma was the most common cause of death in Korean War casualties with chest wounds, accounting for 41.2% of all mechanisms of death in thoracic wounded casualties. Rib fracture(s) caused 6.3% of thoracic wound deaths and 10.9% of this category died from concussion (blast).[6(Table 48 p.45)] Not surprising the fatality rate for bullet wounds of the chest, at 34.7 percent, was much higher than the overall rate.[6(Table 46 p.44)]

The overall case fatality rate for all wounds during the Korean War reached a new low of 2.5%; significantly lower than the 4.5% rate of WWII. Not surprisingly the case fatality rate for thoracic wounded casualties remained considerably higher than the overall rate. The average case fatality rate for thoracic wounded casualties during the Korean War was 5.3%[6(Table 49 p.45)] with wounds of the thorax accounting for 21% of all casualties dying of wounds.[6(Table 45 p.43)] The case fatality rate for chest wounded casualties varied considerably between the various wounding mechanisms/agents.

When the chest wound was caused by a bullet the case fatality rate was 7.2%, when caused by a fragment it was 4.9%, and it was 5.3 percent when caused by a land mine.[6] Because the great majority of those with serious pulmonary, major thoracic vascular, or cardiac wounds die before any medical care can be provided and because the majority of those surviving initial wounding who do require a surgery only need a chest tube; the number of casualties requiring thoracic surgery in an operating room is quite small. During the Korean war thoracic surgery only accounted for 1.3% of all surgical operations in an operating room and operations for wounds of the heart, pericardium, and great vessels accounted for less than 0.1% of all surgeries.[6(p. 86)]

In Reister's Korean War data on anatomic wound location, no significant variation or major shift is apparent between body regions in the relative distributions, either by type of operation or for different periods of time during the war. Because there was no statistically significant shift of wounds from thorax and abdomen to some other body region after body armor was introduced on a limited basis it is not possible to draw any conclusions from this data as to the efficacy of body armor.[6(p.51)]

In a recent review of the Vietnam War era WDMET files [McPherson J, Feigin DS, and Bellamy RF: Prevalence of Tension Pneumothorax in Fatally Wounded Combat Casualties. DRAFT pending publication], Bellamy and colleagues found that 663 of 893 deceased combat casualties (74%) with readable post-mortem radiographs had radiographic and/or clinical evidence of a chest injury. In 9% (80) the fatal injury had occurred in the thorax. In the remainder there was fatal injury in another part of the body, usually the brain. In this WDMET review autopsy reports showed that the fatal chest injury involved the heart and/or great vessels in 2.8% (25/893) of cases. The actual number of fatal heart or great vessel wounds in this series was almost certainly greater than estimated since radiographs of such casualties, which is what was reviewed in this study, did not necessarily show evidence of mediastinal displacement or even hemothorax.

As noted in the Textbook of Military Medicine[4(p.457)] 12% of 7,500 total casualties seen at the 24th Evacuation Hospital during the Vietnam War had injuries involving the chest; 61% of these having had an intrathoracic injury. The cause of the intrathoracic injuries was fragments in 443 (81%), bullets in 76 (14%), and blunt injury in 28 (5%). No intrathoracic injuries were attributed to blast in this series.[11] Casualties with thoracic injuries commonly have other injuries as well. Eighty-five percent of casualties seen at the 24th Evacuation Hospital in Vietnam,[11] and eighty percent of chest wounded casualties seen at the Rambam Medical Center in Haifa, Israel[12] had one or more additional injuries involving another part of the body.

Most data from wars occurring within the past 35 years, with the exception of the findings of one surgeon in Lebanon,[13, 14]

indicate that a formal thoracotomy is needed in only about 10% to 20% of casualties with thoracic injuries who survive to reach a military hospital.[4(p.458)]

Epidemiology of Civilian Thoracic Trauma

Before discussing the epidemiology of thoracic trauma, whether it be civilian or military, it is essential to have a standard definition of what body area actually constitutes "the thorax." Unfortunately the epidemiology of thoracic trauma is often reported without there being included a precise definition of what constitutes a thoracic wound. This has led to confusion when comparisons are made both between civilian trauma and military trauma, and between the rates of thoracic trauma in various military conflicts.

Berry in the "Thoracic Surgery" volume of the Army Medical Department in World War II series[9(p.59)] stated that, "It is regrettable that up to this time there has been no agreement as to exactly what portion of the body constitutes the chest. Without a generally accepted definition, there has naturally been disagreement in the statistics for chest wounds." Following the Korean War, Beebe and DeBakey, in their book, "Battle Casualties"[5 (p.89)] recommend the use of Churchill's definition of the chest. Churchill defined the chest as being:

> *"…On the surface…the simplest line is one that approximately follows the lower limits of the pleural cavities. In front, this line passes from the lower end of the sternum obliquely downward along the costal margin to the 8th intercostal space. A horizontal line carried around the body to meet the corresponding point on the other side will pass approximately over the midpoint of the 11th rib and the spine of the first lumbar vertebra. The chest region as described includes that entire circumference of the trunk and is not interrupted posteriorly by a "back" or a "spine".*

Civilian epidemiology of chest trauma, like that of the military, is complicated by the fact that significant underreporting of thoracic injury can result when data is only gathered on those surviving long enough reach hospital level care. Thoracic injury, particularly penetrating thoracic injury and blunt injury involving the heart and great vessels, has a high pre-hospital mortality. Thoracic great vessel injury accounts for approximately 8% to 9% of vascular injuries, most of these being caused by penetrating mechanisms.[15]

Blunt disruption of the thoracic aorta is usually rapidly fatal prior to hospital arrival; it is estimated that only 10% to 20% of patients with these injuries survive to reach an emergency department (ED).[16, 17] Thus, if rates of thoracic trauma are based only upon it's frequency in patients who survive to be seen in a hospital, there will be significant under-reporting.

Injuries of the chest wall and lungs are common. Chest trauma has been estimated to cause 20% of all civilian traumatic deaths regardless of mechanism. This has been calculated to be about 16,000 deaths per year in the United States.[18] A cooperative study from 60 hospitals showed that among victims sustaining thoracic trauma, 50% will have chest wall injury; 10% being minor, 35% major, and the remaining 5% having flail chest injuries; injuries to the lung parenchyma are reported to occur in 26% of patients.[18] According to LoCicero and Mattox in 1989 chest trauma was the second most common cause of traumatic death in the United

States after head trauma, accounting for approximately 20% of deaths.[18]

In one study described by Orlinsky et al.[19] pulmonary complications were noted in approximately 10% of more than 3000 trauma victims. The lung is the most frequent organ involved in multiorgan failure and is usually the first organ to fail after injury.[20] Respiratory failure has the highest mortality rate compared with failure of other organ systems.[19] Jones et al. found that in traumatic chest injury, overall mortality is greater in blunt (40%) versus penetrating (20%) injuries.[21]

Physiology and Pathophysiology of Ventilation, Oxygenation, and Respiration in Combat Casualties

The focus of the following discussion is on breathing-related basic physiology and pathophysiology and on specific injuries of the chest and lungs for which there is a recommended pre-hospital treatment. Ventilation, oxygenation, and respiration are all covered. Each of these is a different but related process. The first involves the exchange of air between the lungs and the atmosphere, and consists of pulmonary ventilation (the total exchange of gasses between the lungs and the atmosphere) and alveolar ventilation (the effective ventilation of the alveoli, in which gas exchange with the blood takes place). Adequacy of ventilation is determined by assessing arterial pCO_2; the measurement of end-tidal CO_2 is now possible in the pre-hospital environment.[22] In general, however, in the pre-hospital environment assessment of ventilation is generally made by determining respiratory rate and depth. Unfortunately the importance of respiratory rate is generally underappreciated by pre-hospital personnel and it is frequently only estimated. The depth of respiration, which is also important, is similarly rarely assessed.

Oxygenation involves the saturation of hemoglobin with oxygen. Theoretically this does not require ventilation although practically it does because without ventilation oxygen cannot reach the alveoli. So long as there is a high enough partial pressure of oxygen in the lungs, and there is adequate blood flow, oxygen will move from the lungs to the peripheral tissues. Hypoxia resulting in anaerobic metabolism exists whenever the partial pressure of oxygen available at the cellular level is decreased (< 30 mm Hg). This can be due either to a low environmental partial pressure of oxygen, or due to other problems such as decreased delivery of oxygen (anemia, hemoglobinopathies, and hypotension), increased utilization of oxygen, or inability to use delivered oxygen.

The causes of hypoxia have classically been divided into four types:[23]

1) Hypoxemic
2) Anemic
3) Stagnant
4) Histiocytic[24, 25]

Histiocytic hypoxia refers to any condition in which oxygen is available but cannot be used by cellular mitochondria. This occurs in circumstances such as poisoning with cyanide and hydrogen sulfide.

In combat casualties all four of these types each of these types of hypoxia can occur singly or in combination. All types of hypoxia are discussed within this chapter but only hypoxemic (anoxic) and histiocytic hypoxia are discussed in this section; anemic and stagnant hypoxia are discussed in the “Circulation” section.

Hypoxemic hypoxia has four main causes and one much less common cause:

1) Low partial pressure of oxygen
2) Hypoventilation
3) Shunting defects
4) Perfusion defects and rarely
5) Increased oxygen transit time through the alveoli

The most easily understood is a low partial pressure of oxygen in the atmosphere such as occurs at high altitude or when there are other gasses, such as carbon dioxide, present in high concentration that displace oxygen [Dalton's Law of Partial Pressures]. Hypoventilation is one situation in which carbon dioxide can accumulate inside the alveoli and displace oxygen but, in general, the primary problem in hypoventilation is hypercarbia, not hypoxia.

When some alveoli are perfused but inadequately ventilated (shunt) oxygenated blood that has passed these alveoli mixes with properly oxygenated blood producing hypoxemic hypoxia. If there are large areas of lung that are ventilated but not perfused , e.g. a large pulmonary embolus, these alveoli contribute to dead space in which the exchange of oxygen for carbon dioxide does not occur. This can cause hypoxemic hypoxia because the CO_2 content in this dead space is higher than inhaled air and this CO_2 displaces some of the inhaled oxygen leading to a lower alveolar $PO_{2;}$ however this is not the primary reason for hypoxia in patients with pulmonary emboli as is discussed in detail later.

Finally it is at least theoretically possible that in some rare cases there can be such an increase in the distance across which intra-alveolar oxygen must diffuse to reach the passing blood that the hemoglobin in this blood remains unoxygenated resulting in hypoxemic hypoxia. This might occur in conditions such as the adult respiratory distress syndrome (ARDS) in which there is increased interstitial fluid surrounding the alveoli. Each of these causes of hypoxemic hypoxia, except increased shunt (perfusion without ventilation), is corrected by supplemental oxygen[23] so the response to supplemental oxygen can be used differentiate shunt-caused hypoxia from these other causes.

Respiration is the exchange of oxygen between the atmosphere and the cells of the body. This process includes:

1) Ventilation
2) Diffusion of oxygen from alveoli to the blood and carbon dioxide from the blood to the alveoli
3) Transport of oxygen to, and carbon dioxide from, the cells.

The last of these processes involves blood flow and problems in this area will be discussed in the "Circulation" section.

Combat trauma victims can experience difficulties in one or more of each of the above areas. Ventilation, the mechanical movement of air in and out of the chest, requires a functioning

1) Neurologic system to both generate the signal to breathe and to transmit that signal to the end-organs of ventilation
2) Bellows to move air in and out of the chest (intact diaphragm and chest wall).

Traumatic injury to the brain, the upper spinal column, or to peripheral nerves involved in the ventilation process can each disrupt normal ventilation. Injuries that disrupt the ability of the chest to generate negative intrathoracic pressure also impair ventilation. These include:

1) Blunt and penetrating trauma to the chest wall (flail chest, opening of the pleural space to the atmosphere, etc.)
2) Direct injury to the diaphragm or injuries that interfere with the proper functioning of the diaphragm, and injury to accessory muscles of respiration
3) Disruption of airflow in and out of the alveoli from injury to the trachea and/or bronchus.
4) Finally ventilation can also be disrupted by severe pain on breathing and by anything that restricts of movement of chest wall and/or diaphragm (circumferential burns etc.).

Adequate cellular oxygenation is dependent upon there being a sufficient partial pressure of oxygen in the alveoli, rapid transfer of oxygen from the alveoli into the blood stream, sufficient functional hemoglobin to carry the oxygen, and a properly working cardiovascular system to transport oxy-hemoglobin out of the lungs to the cells. The physiologic goal is to deliver sufficient oxygen to cellular mitochondria to allow aerobic metabolism. If this does not occur, for whatever reason, the cells will begin anaerobic metabolism, and will eventually die. The body normally keeps the mixed venous pO_2 around 40 mm Hg. If it falls below 30 mm Hg this indicates that anaerobic metabolism is occurring in some circulatory beds.

Although uncommon, impairment of oxygenation in combat trauma patients can be due to a lowered partial pressure of oxygen in the atmosphere. This would generally only be of clinical relevance when the partial pressure of oxygen in blood drops below 60 mmHg (roughly corresponding to 90% saturation). Partial pressures this low occur at very high altitudes (e.g. much of Afghanistan) and when other gasses are present in substantial quantity (Dalton's Law of partial pressures) that displace oxygen; a situation that can occur when carbon dioxide is produced by combustion (explosion) in an enclosed space (e.g. cave, vehicle, etc…).

In both of these combat situations supplemental oxygen would benefit combat casualties until they could be moved to an area with a higher partial pressure of oxygen. Once in an environment with a normal partial pressure of oxygen there would be no continued benefit from oxygen.

Although it is indisputable that supplemental oxygen will raise the intravascular oxygen content when hypoxia is due to one of these causes, there have been no military or even civilian, pre-hospital studies that have proven that supplemental oxygen improves survival for trauma victims. A recent study by Stockinger and McSwain[26] addressed the issue of the efficacy of pre-hospital supplemental oxygen in civilian trauma patients. They concluded that pre-hospital supplemental oxygen provides "no survival benefit" for trauma victims not requiring intubation and ventilation.

The primary cause of inadequate cellular oxygenation in battlefield casualties is an inadequate quantity of hemoglobin due to blood loss; and this cause of cellular hypoxia is only very marginally improved by supplemental oxygen. The solution to this cause of cellular hypoxia is to stop further blood loss and, when indicated, to replace blood loss with blood or another effective oxygen-carrying solution (see discussion in "Circulation" section).

There are combat-related circumstances in which supplemental oxygen may benefit casualties in the pre-hospital environment. Any situation that creates ventilation-

perfusion mismatch (alveoli that are perfused but un- (or under-) ventilated) will lead to some degree of cellular hypoxia that will be improved with supplemental oxygen. Types of combat casualties, who might have some degree of ventilation-perfusion mismatch and would therefore benefit from supplementary oxygen include patients with blast lung, pulmonary contusion, non-cardiogenic pulmonary edema, drowning, nerve agent poisoning, chemical pneumonitis (all causes including mustard agent), pulmonary anthrax or other causes of pneumonia, and the adult respiratory distress syndrome (shock lung), to name a few. Oxygen is an important element in the resuscitation of all such casualties but the incidence of such casualties on most battlefields is low.

Asthma has increased in frequency in the general population and with a recent relaxation of standards that will now allow some volunteers with a history of asthma to join the military there are an increasing number of soldiers on active duty with reactive airway disease. There are a wide variety of triggers in a combat environment that might induce bronchospasm in susceptible individuals; when severe bronchospasm occurs in such people supplemental oxygen and bronchodilators may be life-saving.

A decision regarding whether to make supplemental oxygen available in the forward battle areas, near the point of wounding, should be based upon a risk-benefit analysis that takes into consideration the probable incidence of conditions likely to benefit from the availability of oxygen. Since the cause of cellular hypoxia in most conventional combat casualties is acute blood loss, which is little benefited by supplemental oxygen, in most combat situations the "costs" probably outweigh the benefits.

Oxygen generators and compressed oxygen are both heavy, compressed gasses in general are dangerous, and oxygen is particularly hazardous in a potentially flammable/explosive environment, e.g. combat. The higher the percentage of casualties suffering from conditions likely to benefit from oxygen (high altitude or other causes of hypoxia resulting from a lowered partial pressure of oxygen) the more the equation shifts in the direction of overall benefit.

High Altitude (environmental) Hhypoxia

Although combat casualties rarely experience hypoxia as the result of a low environmental pressure of oxygen this has happened during current operations in Afghanistan where almost 50 percent of the total land area is above 2,000 meters in elevation. Many of the strategically significant mountain passes in Afghanistan lie at, or above 2,000 meters. At 4,420.7 meters, Mount Whitney, the highest mountain in the continental U.S., is lower than some of Afghanistan's important mountain passes and not much higher than most of the others.[27, 28]

It has been found during on-going operations in Afghanistan that almost all troops deploying to combat operations above 8,000 feet (2,700 meters) experience some degree of altitude related illness [10th Mountain notes from Afghanistan [http://pvtpyle.com/10th_mountain_notes_from_afghani.htm – last accessed on 29 Jan 04] – and this in otherwise healthy combatants. Altitude-related hypoxia in combat casualties, particularly when it compounds other causes of hypoxia, can be the difference between life and death. At sea level the inspired partial pressure of oxygen is 150 mm Hg; at 2,000 meters it drops to roughly 110 mm Hg; to 80 mm

Hg at 4,000 meters; and at 6,000 meters to less than 70 mm Hg.

Altitude-related hypoxia is exacerbated by the fact that the decrease in intra-alveolar oxygen pressure that occurs with increases in altitude is nonlinear – as altitude increases the intra-alveolar oxygen content drops even faster (especially at very high elevations). This is because within the alveoli, at normal body temperature, the partial pressure of water remains constant at 47 mm Hg irrespective of the partial pressure of other gasses. As a result during ascent the partial pressure of water becomes an increasingly greater portion of the total alveolar gas and displaces relatively more of the already lowered oxygen content in the alveoli.

Hypoventilation

Hypoventilation is defined as a minute ventilation (respiratory rate × tidal volume) that is inadequate to remove the carbon dioxide that is produced ($\dot{V}_{CO_2}$). When alveolar ventilation is inadequate, a not uncommon occurrence in combat casualties, carbon dioxide increases in the blood and alveolus. As carbon dioxide increases in the alveolus, there is correspondingly less room remaining for oxygen, so hypoxia occurs. Thus, the two main physiologic features of hypoventilation are an elevation in the partial pressure of CO_2 in arterial blood and improvement in arterial oxygenation (PaO_2) with even a slight a increase in the inhaled oxygen concentration (FIO_2).[23] One cause of inadequate ventilation in combat casualties is partial or complete airway obstruction.

Hypoventilation can also be caused by neurologic injury or chest trauma that either impairs ventilation or causes significant pain on breathing. Supplemental oxygen improves the hypoxia caused by hypoventilation but does not address the problem of hypercarbia. The real solution to hypoventilation is not supplemental oxygen, it is improved ventilation.

Ventilation – Perfusion (V/Q) Mismatch

When it comes to oxygenation of hemoglobin ideally each of the pulmonary alveoli would be perfused so all would be involved in the exchange of oxygen and carbon dioxide. In reality, however, some alveoli are ventilated but not perfused (dead space) and some are perfused but not ventilated (V-Q mismatch/shunt). Matching alveolar ventilation (V_A) with perfusion (Q) is a complex process, involving ventilation volume, alveolar pressure, compliance of lung and chest wall, resistance of airways, gravity, position of patient, pulmonary blood flow, and mode of ventilation.

When ventilation and perfusion are matched, $V_A/Q = 1.0$. Atelectasis, pneumonia, and pulmonary edema (low V_A/Q) all contribute to an increase in baseline shunting (unventilated but perfused alveoli) and pulmonary emboli (high V_A/Q) cause an increase in the amount of dead space (ventilated but not perfused alveoli). Shunt, in which there is mixing of oxygenated and unoxygenated blood as the pulmonary arteries bring blood back to the heart, is the only mechanism of hypoxemia in which the PaO_2 stays well below the alveolar oxygen concentration even if 100% oxygen is being delivered to the alveoli. This fact can be used to make a diagnosis of shunting in a hypoxemic patient; if hypoxia does not improve with administration of supplemental oxygen, shunting (V/Q mismatch) is likely the cause.

Anemic Hypoxia

Anemic hypoxia occurs when there is insufficient functional hemoglobin to carry the oxygen needed to meet cellular metabolic requirements. This is a common cause of hypoxemic hypoxia in combat casualties; the most common etiology being traumatic loss of hemoglobin. This cause of hypoxemic hypoxia is discussed in detail in the "Circulation" section of this monograph.

Circulatory (stagnant) Hypoxia

This form of hypoxia occurs when the body has functional, well-oxygenated, hemoglobin but is not able to deliver it to the cells at a rate fast enough to meet metabolic demands. This occurs in cardiac pump failure, which can occur in the combat casualties who have sustained blunt chest trauma, or in those with penetrating trauma who develop cardiac tamponade. This cause of hypoxia is also covered in this chapter in the "Circulation" section.

Histiocytic Hypoxia

Histiocytic hypoxia is the type of hypoxia that occurs whenever cellular mitochondria cannot use the adequate oxygen that is delivered; this can occur for a variety of reasons. Histiocytic hypoxia is an uncommon cause of significant hypoxia in the great majority of combat situations. There are, however, some specific combat circumstances in which histiocytic hypoxia should be considered as a cause of hypoxia. Cyanide toxicity is a classic example that may occur in combat casualties either as a result of exposure to gasses given off by combustion of certain types of material or, as the result of a chemical attack with a blood agent (hydrogen cyanide).[29] Although cyanide is a poor chemical weapon for use in open areas because it is difficult to achieve and sustain a lethal concentration, cyanide can be effectively used inside enclosed areas. If used, cyanide acts by disrupting the utilization of oxygen at the cellular level and in this way causes cellular hypoxia.

Perhaps the most common, although still rare, way in which combat casualties can experience histiocytic hypoxia is carbon monoxide poisoning. Most commonly this occurs not as the result of enemy action but from accidental causes such as when troops burn fuels inside poorly ventilated, enclosed spaces. The incomplete combustion of carbon fuels leads to the creation of carbon monoxide which binds to hemoglobin (creating carboxyhemoglobin) more than 200 times more tightly than oxygen and, once bound, prevents it from accepting oxygen; resulting in cellular hypoxia even when there is sufficient alveolar oxygen available.

As stated, the basic pathology in all cases of histiocytic hypoxia is an inability to effectively use oxygen that is readily available. Therefore the initial response of a casualty with histiocytic hypoxia of any cause is hyperventilation, although, particularly in the case of cyanide intoxication, apnea can result quickly. Because the problem in histiocytic hypoxia is not a lack of oxygen in the blood, in each of these cases the casualty will not initially appear cyanotic when exposed to either of these substances [The word cyanosis refers to the bluish color of the skin that occurs when there is greater than 5 grams of unoxygenated hemoglobin. It is not related to cyanide which derives its name from Prussian Blue, a dye from which hydrogen cyanide was first extracted].[30, 31] In each of these cases, if a pulse oximeter reading is obtained early in the poisoning, the reading will be normal. For this reason, unlike in most other causes of hypoxia, supplemental oxygen is of little benefit, although

if available it should be provided. In both of these situations prehospital medical personnel must:

1) Have a high index of suspicion of histiocytic hypoxia in enclosed-space casualties who are having breathing problems
2) Avoid becoming personally poisoned
3) Keep the casualty from becoming more poisoned
4) Quickly evacuate the casualty out of the area for definitive treatment because no specific treatment is currently available for either of these conditions at, or near, the point-of-wounding.

In the past some pre-hospital treatment for cyanide poisoning was available in the form of amyl nitrite ampules that create methemoglobinemia to bind cyanide but these are not now routinely provided because the threat is considered so low that more harm, as the result of misuse, than benefit would likely occur.

General Approach to the Management of a Combat Casualty withPpossible Iimpairment of Ventilation, Oxygenation, and/or Respiration

Initial Assessment and Management of the Thoracic Wounded Casualty

The pre-hospital evaluation of the thoracic wounded combat casualty is critically important. First, and most important, a rapid and accurate assessment is essential to identify immediately life threatening problems such as tension pneumothorax and critical hypoxia, so that these can be immediately addressed, and when possible, corrected. Second, while a fair number of thoracic wounded combat casualties have injuries that mandate urgent evacuation many have injuries that are either superficial or at least stable. While seriously wounded casualties may need urgent evacuation to ensure survival, the second, and larger group, may not need evacuation at all; or at least their evacuation can safely be delayed until more favorable circumstances exist. Depending upon the combat situation some of this latter group might reasonably be returned immediately to combat-related activities.

It is often difficult to determine the cause of dyspnea because it has multiple causes and there are a number of receptors that may produce the subjective feeling of shortness of breath. Key to correctly diagnosing the etiology of dyspnea in a particular patient is to identify the primary organ system that is involved. Is the primary cause respiratory, cardiovascular, both or neither. Michelson et al. in their article on the "Evaluation of the Patient with Shortness of Breath"[32] state that the cause of shortness of breath is multifactorial in 27% to 33% of dyspneic patients. They go on to note that although the standard diagnostic tools of history, physical examination, and chest radiography have been found to only be accurate 66% of the time for all diagnoses they are 81% accurate for diagnosis of the four commonest causes of dyspnea (asthma, chronic obstructive pulmonary disease, interstitial lung diseases, and cardiomyopathy);[32, 33] since none of these causes of dyspnea are likely to be the cause of dyspnea in combatants on a battlefield it is unclear how effective these traditional methods of di-

agnosing dyspnea would be in a combat setting.

History

If the thoracic injured casualty is awake and able to answer questions certain information should be sought. The presence or absence of pain and its location and relation to respiration should be determined. Difficulty breathing and whether or not the difficulty is increasing or decreasing is also important. Has the casualty coughed up blood and if so, how much? Has the casualty felt nauseated and vomited and when did he last eat? Nausea and vomiting, when present, suggest a thoraco-abdominal injury because these symptoms are uncommon in the casualty with only a thoracic wound.[34(p.417)] Was the casualty unconscious and if so for how long? Was the wound ever "sucking"? What position was the casualty in when hit and by what type of missile? The position of the patient when injured, type of missile and proximity to any explosion are important in arriving at a final decision as to the probable intra-thoracic lesion; only by knowing these factors is it possible to visualize the probable injury.[34(p.419)] All of these questions can be asked in a few moments and they often yield a surprising amount of useful information.

Physical Examination

The rib cage imposes definite limitations on the size of the thoracic cavity so any expanding space-occupying medium (e.g. air and/or blood) can seriously disrupt the normal function of the heart and lungs. Both pneumo and hemothorax can produce similar mechanical effects. By interfering with cardiac return and pulmonary expansion they reduce the volume of circulating blood and decrease oxygen saturation. This almost invariably produces an increased cardiac and respiratory rate in any severe thoracic-injured casualty. Injury to the chest wall produces pain on inspiration so to minimize pain, motion is restricted. Thus, thoracic wounded casualties usually present with a rapid pulse and rapid but shallow respirations. When a tension pneumothorax is present the signs and symptoms of fear, restlessness, distension of neck veins, extreme dyspnea and pronounced cyanosis, tend to be dramatic and unforgettable[7(p.157)]

Accurate pre-hospital evaluation of thoracic wounded casualties is difficult. In his Civil War Manual of Military Surgery, Dr. Chisolm, a Confederate surgeon, pointed out that it is often difficult to detect even serious injury to the lungs and noted that there is no one symptom that is sufficient to make the diagnosis.[1(p.281)] More recently, in 2002, Eckstein et al.[35] stated that while shortness of breath and chest pain are the most common presenting complaints of pneumothorax, the appearance of patients with pneumothorax is highly variable and can range from acutely ill with cyanosis and tachypnea to misleadingly healthy appearing. They also noted that there may be no correlation between presenting signs and symptoms and the degree of pneumothorax.[35]

This said, more recently Forsee[34(p.422)] noted that,

> *"Fortunately, most foreign objects travel in a straight line from entrance to exit, entrance to lodgement. The voluminous literature on the erratic course of missiles within the body has tended to over-emphasize the exceptional case that takes a bizarre course due to striking a rib or other bony structure. The explanation of most peculiar foreign body tracks is found by questioning the patient regarding the position he was in when injured. When the patient with a foreign body within*

> *the thorax that shows no wound of entrance other than the one over the deltoid tubercle of the arm tells the examiner that he was lying on the ground with his arm extended along the side of his head, the course of the missile is no longer mysterious...[T]he most reliable information is obtained by projecting the course of the missile."*[34(p.422)]

Although the signs and symptoms present in a thoracic wounded casualty and the physical examination may be misleading, certain clinical findings should be sought. Most important is the general appearance of the patient. If there are signs of cerebral anoxia such as unconsciousness, agitation, or irrational behavior the need for prompt and adequate therapy is great. It should be noted that cyanosis is a sign of fairly advanced anoxia so if any cyanosis is detected it is usually an indication that the casualty needs prompt resuscitation. Measures to decrease anoxia such as thoracentesis for pneumothorax and administration of oxygen should be initiated before the appearance of cyanosis. It is important, however, to recognize that when there is severe blood loss cyanosis may not be detectable due to the lowered hemoglobin content of the blood.[34(p.420)] For cyanosis to be visible there must be at least 5 grams of deoxyhemoglobin per 100 ml of blood present[36] [before cyanosis would be present in a patient with a hemoglobin of 10 more that 50% of that hemoglobin would have to be deoxygenated].

The type and character of respirations are particularly important in the assessment of thoracic wounded casualties. Assessment of ventilatory pattern can offer important clues as to the extent of injury in thoracic wounded casualties. While fear and exertion can cause an increase in the rate and depth of respiration, persisting rapid and deep respirations suggest ongoing hypoxia. Rapid and shallow respirations suggest pain on deep inspiration or inability to take a deep breath. Stridor and/or the use of the accessory muscles of respiration usually indicate some obstruction to airflow. Rapid, rattling respirations with frequent ineffectual coughs, indicate that the patient is having difficulty maintaining a clear airway[34(p.420)]

Difficulty breathing may either be due to actual "shortness of breath" or, more commonly, to being "unable to take a good breath." The former suggests hypoxia, which may be caused by any one, or a combination, of causes (hypoxemic, anemic, stagnant, and histiocytic) while the latter results from pain associated with breathing. In combat casualties without severe blood loss, "shortness of breath" is usually caused by pathology in the pulmonary parenchyma."[34(p.417)]

Some amount of hemoptysis is to be expected in almost every case of penetrating thoracic injury. But, as Fraser noted based on his experience in the Crimean War,[3] "[Expectoration of blood] is no certain indication that the [lung] has a [penetrating injury]…spitting of blood is a very deceptive diagnostic sign of lung wound." However, "When [blood] is rapidly brought up by the mouthfuls it becomes an important symptom [of penetrating injury to the lung]."[3(p.284)] While hemoptysis frequently indicates the presence of penetrating chest injury it is also commonly present when there has been blunt chest trauma or blast injury. Although the amount of blood coughed up is variable, generally the larger and more severe the injury, the greater the quantity of hemoptysis. When there is a large amount of hemoptysis the airway may become obstructed so attention must be paid to the airway.[34(p.417)]

The presence or absence of loss of consciousness and it's duration in thoracic wounded combat casualties has been reported to be helpful in both diagnosis and prognosis. According to Forsee,

> *"A short period of unconsciousness is not infrequent when injury has been caused by a high explosive shell. Longer periods, especially if preceded by consciousness, are apt to be due to cerebral anoxia and are therefore of the utmost importance, both in regard to the severity of the wound and from a prognostic standpoint. Unconsciousness of more than momentary duration means that there is cerebral concussion, severe blood loss, or cerebral damage from prolonged anoxia. Maniacal manifestations, frequently a sign of severe anoxia, may likewise be present and this oxygen-want must be combated vigorously."*[34(p.419)]

If there is either a history of, or obvious, "sucking" (exchange of air) through the chest wound it can be assumed that the pleura have been penetrated; the absence of sucking, however, says nothing about the course of the missile or the damage it may have produced.[34(p.419)]

In a pre-hospital setting assessment for certain gross physical findings may provide more accurate information regarding the status of thoracic-wounded casualties than might the more "refined" methods commonly used to evaluate such patients in civilian emergency departments. For example, checking the position of the trachea in the suprasternal notch or assessing the position of the apical impulse of the heart may be more effective at diagnosing a clinically significant hemopneumothorax than very careful, time consuming percussion and auscultation. In a more appropriate setting the latter methods are not to be ignored but the busy, noisy, and stressful pre-hospital environment is rarely conducive to an effective, meticulous, examination. In this setting minor deviations from normal are generally unimportant. Attention should primarily be paid to the patient as a whole and to his gross abnormalities.[34(p.420)]

Auscultation

Auscultation is one of the primary methods for assessing pulmonary status, even in prehospital settings, but, as will be discussed later in greater detail, auscultation is often inaccurate even when performed, by a physician. Chen and colleagues[37] in their assessment of auscultation note that optimal physical examination of the chest may be impeded by a noisy resuscitation area, an overly rapid initial examination, and inattentiveness to the task.[37] All of these are problems often present in an emergency department setting[35] and they are certainly present in the combat environment.

Penetrating injury of the chest, especially when the intercostal vessels, lung parenchyma, or pulmonary vessels are injured, generally results in a hemothorax. As noted earlier, physical findings tend to be normal in patients with a small pneumothorax.[38-40] The characteristic physical examination findings associated with a pneumothorax are more likely to be present when lung collapse is greater than 25%.[37, 41]

In their study of the efficacy of auscultation in detecting hemo and hemopneumothorax Chen et al. found that auscultation to detect these injuries, while having a fairly high specificity and positive predictive value, had a low sensitivity (58%). Their conclusion was that hemopneumothorax and hemothorax, especially when caused by gunshot wounds, are

likely to be missed by auscultation. They did, however, note that because of the fairly high specificity of auscultation, decreased breath sounds, together with overlying penetrating trauma, is a reliable indication of the need for tube thoracostomy and thus can be used to make this decision before chest radiography.[37]

Another study by Hirshberg et al., found that while auscultation may not be effective at picking up small hemo and pneumothoraces it is fairly effective (96% sensitive) at picking up larger (and more clinically important) collections of air and blood in the chest.[42] In 1990, Thomson et al.[43] reached a similar conclusion. They found that physical examination was able to detect large collections of air and fluid in the chest (96% and 94% respectively) and thus it could be used to guide management of thoracic wounded casualties. It should be noted, however, that 92% of patients in their study had sustained knife wounds making this population considerably different from that of a population of combat casualties.[43]

Use of ultrasonography to detect lung injury

Although ultrasound is not a device likely to be used at, or near, the point-of-wounding anytime in the near future, ultrasound is an effective diagnostic tool for intra-thoracic injury and it is being increasingly used far forward. A prospective study by Lichtenstein et al.[44] compared the diagnostic accuracy of lung ultrasound, auscultation, and bedside radiography with a gold standard of thoracic computed tomography. Pleural effusion, alveolar consolidation, and alveolar-interstitial syndrome were evaluated. They found that auscultation had a diagnostic accuracy of 61% for pleural effusion, 36% for alveolar consolidation, and 55% for alveolar-interstitial syndrome. Portable x-ray had a diagnostic accuracy of 47% for pleural effusion, 75% for alveolar consolidation, and 72% for alveolar-interstitial syndrome. By comparison lung ultrasonography had a diagnostic accuracy of 93% for pleural effusion, 97% for alveolar consolidation, and 95% for alveolar-interstitial syndrome.

Not only was lung ultrasound more accurate than auscultation and chest radiography but it was able to quantify the extent of lung injury. This study concluded that bedside lung ultrasonography is highly sensitive, specific, and reproducible for diagnosing the main lung pathologic entities associated with ARDS and noted that it should be considered an alternative to portable chest x-ray and chest computed tomography.[44] Ultrasound of the chest is also increasingly being used in hospital settings to make the diagnosis of pneumothorax and, as portable ultrasound devices become lighter and more durable, ultrasound is making its way to the forward edge of the battlefield.[44-48] Until a device can be developed that will give a relatively unskilled medic a simple yes-no answer about the presence or absence of pneumothorax this technology will not be of much use at the point of wounding; but perhaps this capability is not too far in the distant future.

Another technology being developed for far forward diagnosis of pneumothorax is micropower-impulse radar (MIR). This technology is being developed at the Lawrence Livermore National Labs. (**http://www.biomec.com/projects/pneumo.html**) and may result in a pneumothorax detection device that is smaller, more durable, and easier to use than ultrasound technology.

Pulse Oximetry

Today pulse oximetry is widely available for use in patient assessment. Indeed the patient's oxygen saturation is now commonly referred to as the "fifth vital sign" and the measurement of oxygenation by pulse oximetry is considered standard of care in emergency departments in the United States. Pulse oximeters have gotten smaller, lighter, and more durable; they are increasingly used in the pre-hospital environment and are being used today by some military medical personnel at, or near, the point-of-wounding.

Adequacy of oxygenation (but not of ventilation) is generally determined by a pulse oximetry reading above 90%. In most settings pulse oximetry is very effective at assessing the adequacy of oxygenation. It should be noted, however, that pulse oximetry does not directly measure the amount of oxygen bound to hemoglobin. Pulse oximetry functions by relying upon the Beer-Lambert law which states that the concentration of a light-wave absorbing substance (in the case of pulse oximetry, oxy- and deoxy-hemoglobin) in solution can be determined from the intensity of light that is transmitted through that solution, if the intensity and wavelength of incident light, the transmission path length, and the characteristic absorbance of that substance at a specific wavelength (deoxyhemoglobin absorbs at 660 nm and oxyhemoglobin absorbs at 940 nm) are known.

Because this is how oxygenation is measured pulse oximetry can give a false reading in a variety of situations. There are substances other than oxy- and deoxy-hemoglobin, such as carboxy- and methemoglobin, which absorb light at, or near, these wavelengths. When present in the blood these substances will indicate a falsely high, or low, percent hemoglobin saturation. Carboxyhemoglobin, which may be present in combat casualties who are exposed to combustion in a closed space [e.g. combat vehicles, caves, buildings, etc…], absorbs light at very nearly the same wavelength as oxyhemoglobin, giving a falsely high pulse oximetry reading. Thus when carboxyhemoglobin is present it may lead to an incorrect assumption that a combat casualty is adequately oxygenated when, in fact, this may not be the case. Methemoglobinemia, which would be unlikely to be present in combat casualties in the field, markedly increases absorbance of light at both 660 and 940 nm and produces a pulse oximeter reading that tends toward a reading of 85% saturation (regardless of true oxygen saturation). Clinically this usually means that when methemoglobin is present the pulse oximeter reading is falsely low.

Other factors, more common in combat casualties, can also influence pulse oximeter readings. These include motion artifact (some pulse oximeters are less affected by motion artifact than others), hypotension, anemia, and hypothermia. Finally, it is important to note that pulse oximetry only assesses, indirectly, the percent of hemoglobin that is saturated with oxygen; it does not confirm that oxygen is being delivered to, and consumed at, the cellular level. In cyanide poisoning, for example, the oxygen saturation measured by pulse oximetry may initially be near normal despite severe cellular hypoxia caused by cyanide's disruption of cellular oxygen metabolism.

Pre-Hospital Assessment of Thoraco-Abdominal Injury

Physical exam evidence of abdominal injury in casualties with thoraco-abdominal wounds is helpful, but not completely reliable, in making the diagnosis of thoraco-abdominal injury. Many thoracic wounds, without associated abdominal injury, cause pain and spasm of the upper abdomen while many patients with abdominal injury present with limited physical findings, especially if just solid organs have been injured.[34(p.424)] Following his experiences in the Spanish Civil War Jolly[7] also noted that abdominal rigidity often accompanies many chest wounds and that this often incorrectly suggests the presence of abdominal penetration. These diagnostic challenges notwithstanding, Jolly did note that there are some differences between the abdominal findings in patients with pure thoracic wounds and those having wounds that involve the abdomen as well. He pointed out that, "The rigidity associated with abdominal wounds usually develops bilaterally, whereas abdominal rigidity from a chest wound is usually confined to the rectus muscle of one side. He went on to say that, "Abdominal rigidity associated with [isolated] chest wounds tends to be intermittent, [with] some relaxation usually occurring with inspiration…[while]… [a]bdominal wounds are usually accompanied by vomiting or [belching].[7(p.156)]

While there are no pathognominc signs of esophageal injury pain in the area of the posterior thorax, pain radiating into the lumbar area, and/or substernal pain on swallowing all suggest injury to the esophagus.[34(p.424)]

Assessment of Shock in Thoracic Wounded Casualties

The assessment of shock in the thoracic wounded casualty may be complicated by the fact that hypoventilation in such a patient, with accumulation of excessive CO_2, may act to elevate the blood pressure. Forsee noted after World War II that "Inexperienced observers might be lulled into a false sense of security by this elevated blood pressure." He felt that because there is no one pathognomonic finding of shock the general clinical impression of an experienced medical officer may be a better guide than any specific physiologic parameters that can be measured and recorded numerically.[34(p.432)] This does not mean, however, that the physiologic parameters of pulse, blood pressure, and rate and depth of respirations should not be measured but it should rather serve as a warning not to be lulled into a false sense of security by normal appearing vital signs especially in patients with thoracic wounds.

General Management of Thoracic Wounded Casualties

Basic pre-hospital management of combat casualties with penetrating chest wounds remains largely unchanged since at least the Spanish Civil War. At the end of that war Maj. Douglas Jolly, in his book, Field Surgery in Total War, stated that, "The most useful pre-hospital treatment of the thoracic wounded casualty is the application of a large, occlusive dressing to the open pneumothorax."[7(p.158)]

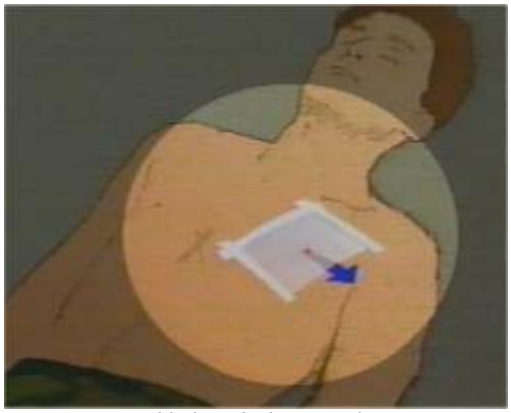
In WWII medical aidmen were originally instructed to apply an occlusive dressing

to only those wounds that were clearly sucking. Later, because it was found that penetrating chest wounds might suck only intermittently, medics were instructed to treat all chest wounds in this way. Upon reaching a battalion aid station the temporary dressing was to be replaced by a larger, Vaseline impregnated dressing, preferably one at least twice the size of the wound, held in place by adhesive tape.

If the wound was very large, large sutures were placed through the skin and tied over the dressings. These measures where felt to be effective for 5 or 6 hours, or longer. After a prolonged period, when the Vaseline-impregnated gauze dressing had become caked with blood, and was no longer pliable enough to act as a one-way valve, a needle with a flutter valve attachment was usually placed in the second interspace parasternally to provide for the escape of air and prevent development of a tension pneumothorax.[49(p.9)]

Current thoracic wound therapy for sucking chest wounds, as promulgated by The Committee on Tactical Casualty Care in the Military Medicine Chapter of the Prehospital Trauma Life Support Manual is that such wounds "Should be treated by applying a vaseline gauze during expiration, covering it with tape or a field dressing, placing the casualty in the sitting position, and monitoring for development of a tension pneumothorax .[50(p.1)]

The argument as to whether chest wound dressings should be sealed on three or four sides rages on today without a definitive answer; either is probably acceptable. Proponents of sealing on three sides [which includes the American College of Surgeons Committee on Trauma[51]] argue that the dependant side should be left open to allow air to escape on exhalation. This technique should, at least theoretically, help to prevent the development of a tension pneumothorax but there is no data to prove that it does. Proponents of sealing the dressing on four sides argue that the greater problem in applying a dressing over a chest wound in the heat of combat is getting the dressing to remain attached to the chest wall, sealed on any side at all. They feel it unlikely that the chest wall dressing, thus taped, would cause a tension pneumothorax.



3-sided Occlusive Dressing

A device called the Asherman Chest Seal™ marketed for the purpose of sealing sucking chest wounds solves the dilemma of three sides versus four. It consists of a flexible 5.5 inch diameter disc, coated with a strong adhesive, with a central hole that exits through a flexible one-way valve.

Asherman Chest Seal

The disc is designed to be applied with its opening directly over a chest wound so that any air escaping from the wound under pressure will pass through the one-way valve; air is thus prevented from entering the chest when negative intrathoracic pressure is generated by inspiration. This device is commonly used today by front line medics and corpsmen; its principle advantage over more standard methods most probably being the very effective adhesive that seals the disc to the chest wall.

It is important for forward area combat medics and corpsmen to understand that the aim of surgical treatment of thoracic wounded casualties is mainly directed toward restoring normal respiration. The main focus of forward care therefore should be to minimize the period during which hypoxia may produce cerebral damage. Once normal oxygenation and ventilation are restored, the time to surgery is generally not a matter of great importance for most thoracic-injured casualties who survive to be treated.[34(p.426)]

Oxygenation and Ventilation of Thoracic Wounded Casualties

The greatest hazard to the soldier with a thoracic wound is hypoxia. Thus, everything that can be done to increase the amount of oxygen transported by the blood is indicated. Ideally oxygen should be started on each thoracic wounded casualty as soon as it can be provided and it should be continued until the casualty has been fully evaluated. At this point the further use of oxygen can be based upon the presence or absence of a condition that will clearly benefit from it use.[34(p.437)]

Despite this general recommendation the exact role of pre-hospital oxygen, even in thoracic wounded casualties, remains to be delineated. While oxygen is used with abandon in the civilian pre-hospital setting, there are potential problems with the widespread use of oxygen in the combat zone. Increased levels of oxygen significantly increase the risk of serious fire[52-57] and widespread use of oxygen in the pre-hospital areas would create a significant logistical challenge, not the least of which is the bulk and weight of both compressed gas oxygen cylinders and oxygen generators.

Ideally oxygen would be reserved for those casualties who would obtain real benefit from its administration; specifically all those having a decreased intra-alveolar partial pressure of oxygen. As noted earlier, inadequate ventilation can lead to hypoxia that is correctable by the administration of oxygen but the fundamental problem in hypoventilation is hypercarbia and improved ventilation, even without supplemental oxygen, will correct both the hypoxia and the hypercarbia.

Inadequate ventilation, with its attendant hypercarbia, is a common problem in thoracic- wounded combat casualties. As discussed in detail later, hypoventilation in thoracic wounded combat casualties is commonly caused by pain on inspiration so adequate pain control is vital to improved ventilation in such patients.

Also discussed in detail is the important role that proper positioning plays in helping to prevent hypoventilation in thoracic-wounded combatants. Only a very small number of thoracic wounded casualties require assisted ventilation. If a thoracic wounded casualty has to be intubated and ventilated in a pre-hospital setting survival is unlikely; thus, for the great majority, penetrating thoracic injury does not, of itself, constitute a reason for intubation.

Patient Positioning for Optimal Ventilation and Respiration

Responsive patients having respiratory difficulty reflexively seek a body position that optimizes ventilation. When patients do this, health care providers should help them achieve this position and most certainly they should not interfere with their efforts in this regard. Asthmatics with acute bronchospasm, for example, seek a position that is referred to as the "Tripod" position -- sitting up, feet hanging down freely, and leaning forward slightly with hands placed on thighs and shoulders hunched up. This position optimizes ventilation by minimizing the work of breathing and maximizing the efficiency of the accessory muscles of respiration. Unless contraindicated combat casualties with breathing problems should be transported sitting up in a Semi-Fowlers position to optimize ventilation.

What is often under-appreciated is the negative impact on breathing that is caused by strapping a patient down on a backboard during transport. Simply lying supine reduces the efficiency of breathing and applying straps across the chest restricts free excursion of the chest. Concern for the cervical spine should not be allowed to over-ride optimizing ventilation/respiration, especially in the combat casualty with a penetrating injury in whom the risk for an unstable cervical spine injury is extremely low [see "Disability" section for a more detailed discuss of this issue].[58]

As indicated in the discussion about the airway, unresponsive patients should be transported in the prone (not supine) or "rescue" (lateral decubitus) position to prevent aspiration. Unresponsive patients having problems with oxygenation/respiration (not those with ventilation problems) may actually have improved oxygenation by transporting them in the prone position. Following a 1976 study that showed that placing patients with acute respiratory distress syndrome in the prone position significantly improved oxygenation this position has been used with increasing frequency in the intensive care unit treatment of such patients.[59] Possible reasons for the apparently improved oxygenation of patients in the prone position include:

1) An increase in end-expiratory lung volume[60]
2) Better ventilation-perfusion matching[61]
3) Regional changes in ventilation associated with alterations in chest-wall mechanics.[62, 63]

These studies indicate that as many as sixty to seventy percent of ARDS patients obtain improved, sometimes significantly improved, oxygenation when in the prone position. A moderately large 2001 Italian, multi-center, randomized trial[64] that compared conventional supine treatment of patients with acute lung injury or acute respiratory distress syndrome with those placed in a prone position also showed improved oxygenation in prone patients. It should, however, be noted that in this study despite improved oxygenation in the prone patients there was no difference between the two groups in overall survival.

Fluid Resuscitation of Thoracic Wounded Casualties

The immediate cause of death in most thoracic wounded casualties is hemorrhage from the heart or great vessels. Slower but persistent hemorrhage can cause delayed death; a common bleeding site being the intercostal vessels. In the past hypotensive casualties with penetrating chest wounds were aggressively fluid resuscitated but today, as discussed in detail in the "Circu-

lation" section, limited fluid resuscitation of such casualties is recommended. As Jolly noted as long ago as the Spanish Civil War, "Intravenous saline or transfusions should not be given in the First Aid Post or Classification Post. These active measures serve only to increase the outpouring of blood from the hidden vessels in the chest wall into the pleural cavity."[7(p.158)]

In 1945, Forsee pointed out that not all chest wounded casualties who are hypotensive have significant blood loss and he cautioned that, "The inexperienced are apt to institute rapid blood replacement which may be fatal to an already unbalanced cardiorespiratory system." He suggested that, "Intravenous therapy should be withheld in the thoracic casualty until it has been determined that he is suffering from blood loss."[34(p.426)]

It should, however, be noted that hypotension in the thoracic wounded combat casualty may be caused by cardiac tamponade and this condition is at least temporarily improved by fluid administration even when there has been no significant blood loss.

In the past it was suggested that when there is ongoing intrathoracic hemorrhage and a pneumothorax, the pneumothorax should not be relieved. This was based on the mistaken notion that the increased intrathoracic pressure associated with the pneumothorax would help to control bleeding. Forsee noted that, "Bleeding from the pulmonary parenchyma will stop of its own accord in the vast majority of cases, and the relatively small pressure difference that may exist in the pleural space due to a pneumothorax is not efficacious in checking a hemorrhage from one of the systemic vessels. Many more errors are made by not aspirating the chest, than by aspirating it, with the remote possibility of restarting a previously stopped hemorrhage."[65]

Interestingly it appears that as long ago as the Civil War, the benefits of sustaining (or even inducing) hypotension, in thoracic-wounded casualties were recognized. Dr. Chisolm, a senior Confederate surgeon, stated that, "It is not surprising…that the [thoracic-wounded] patient should soon become cold, pale, and faint – with feeble, small and irregular pulse, and…tendency to syncope. This is nature's effort to check further [blood] loss…"[1(p.287)]

Dr. Chisolm went so far as to suggest that "The surgeon [should try]…to induce this condition for a similar purpose…[and] [n]otwithstanding the hemorrhage, [should] open a large vein and draw away blood, if possible, to syncope."[1(p.290)] While it is clear today that it was erroneous to conclude that inducing further blood loss in bleeding, and already hypotensive, thoracic-wounded casualties would be beneficial, the basic observation that hypotension can be beneficial does now have scientific support. [See "Circulation" Section]

While vigorous saline resuscitation of thoracic wounded casualties is not advised, when the amount of bleeding into the chest is large and the patient is hypotensive, early administration of blood, when it is available, is recommended. If autotransfusion is available and there is not a thoraco-abdominal injury, larger hemothoraces, if less than 24 hours old, can be aspirated and autotransfused.[34(p.427), 66]

Relief of Pain in Chest Wounded Casualties:

Pain is almost universally present in chest injury. Key to proper pain management in the chest wounded casualty is an understanding that pain does not arise from the lung but originates in the thoracic wall. One approach to pain management in the thoracic wounded casualty is to interrupt painful stimuli near their origin with local anesthesia of the intercostal nerves that supply the painful area. Intercostal nerve block is a simple, and effective means of accomplishing pain relief in patients with chest wall pain; it is even superior to opiate analgesics and is without their adverse effects. The main risk of an intercostal nerve block is the creation of a pneumothorax. If a pneumothorax is already present, this is not a concern. If the painful segment of the thoracic wall is blocked, including nerves above and below the site of the injury, lasting pain relief usually results.

Morphine, while effective, should be administered with caution. Symptoms of cerebral anoxia, such as restlessness and agitation, may be misinterpreted as pain, resulting in the repeated administration of morphine. The situation with the highest risk of inappropriate repeated administration of morphine is when the casualty is hypotensive, with poor peripheral circulation. In such a situation, with decreased peripheral blood flow, morphine given subcutaneously or intramuscularly, may not be fully absorbed into the blood stream. This means pain will be unrelieved; a situation that frequently leads to the further administration of morphine. This may result in patients being given multiple doses of morphine without much benefit. The major hazard from such a situation, however, arises not from inadequate pain relief but from the later absorption of this depot of unabsorbed morphine once peripheral circulation is restored; when this happens the patient may become unexpectedly apneic from morphine intoxication. Excessive administration of morphine can occur during combat; the aforementioned scenario being just one such way that this can occur. During World War II four percent of patients received over one-half grain (30 milligrams) while 0.5% had one grain (60 milligrams) or more during the pre-operative period.[34(p.412-450)]

Therapeutic Interventions that may Contribute toHhypoxia - Introduction of Dead Space

Some therapeutic interventions can actually contribute to hypoxia and hypercarbia. Intubation and artificial ventilation, with its attendant use of artificial airway tubing increases dead space. This “iatrogenic” dead space is added to anatomic dead space, the conductive zone of the airways that consist of the upper airway, trachea, bronchi and bronchioles. This space has a transport function only and does not participate in gas exchange. The volume of gas that fills anatomic dead space, about 150 ml, accounts for about 30% of a normal tidal volume of 500 ml. While it takes energy to transport this dead space volume back and forth during respiration, none of the oxygen in these conductive airways is extracted. In some pathologic situations, such as pulmonary embolus, there is an increase in the amount of un-perfused but ventilated lung and these areas also contribute to dead space. Significant pathologic dead space contributes to hypoxia as described earlier. In some critical patients the energy wasted in the effort to move dead space gas back and forth can have clinical consequences.

As noted above, medical interventions, such as intubation, contribute to dead space by increasing the volume of gas in

conductive airways. In patients with otherwise healthy lungs the volume of gas contained in an endotracheal tube is generally not clinically significant but in patients with compromised respiration and pathologically increased dead space the small increase in dead space from a long endotracheal tube can be significant, especially during long term ventilation. The longer the length of tube before it is vented the higher the CO_2 and the lower the oxygen content in the lungs. For this reason, the dead space volume of connecting tubes or hoses should be as small as possible -- this means use a short tube whenever possible.

Specific Mechanisms of Combat-related Injury Associated with Impairment of Ventilation, Oxygenation, and/or Respiration and their Management

As previously noted injuries outside the thorax can impair cellular oxygenation. Neurologic injury to the head, phrenic nerve, or nerves to the accessory muscles of muscles of respiration can all impair ventilation and lead to hypercarbia and hypoxia. In addition, as emphasized earlier, loss of hemoglobin (hemorrhage) and cardiac injury can result in there being an inadequate flow of oxygenated hemoglobin to the cells. As the management of these problems is discussed elsewhere the focus of the following discussion will be on injuries to the thorax that impair ventilation and oxygenation.

Injuries to the thorax that can impair oxygenation and ventilation in combat casualties include: (1) neurologic injury (2) injury to the upper and/or lower airways (3) injury to the chest wall (4) injury involving the pleural space (5) injury to the lung parenchyma, (6) injury to structures in the mediastinum and/or (7) injury to the diaphragm. [67]

Blunt Chest Trauma (rib fractures, flail chest, pulmonary contusion)

Blunt injury as a percent of all U.S. combat-associated injury appears to be increasing. During the early part of Operation Iraqi Freedom 39% of 294 battlefield casualties transported outside of Iraq for medical care had sustained blunt/motor vehicle collision injuries.[68] Satava notes that because of the use of body armor mortality is primarily caused by direct head and neck injury or “overwhelming” force to the trunk, with thoracoabdominal injuries occurring from blunt rather than penetrating injury.[69]

There are likely several reasons for this. First, large numbers of troops are now routinely moved by motorized air, land and sea transport and injuries sustained during transport are predominantly blunt. Second, urban combat is becoming increasingly common as the percentage of the world’s population that lives in cities increases[70] and with this type of combat has come a concomitant increase in the number of crush injuries from collapsing buildings and injuries associated with falls from a height. Finally, the widespread use of body armor has significantly reduced the number of penetrating injuries to protected areas, most specifically, the chest. Kevlar also reduces the severity of blunt injury. It is widely used by athletes who are at risk of blunt chest trauma because of its ability to attenuate blunt chest injury. Although body armor use by combatants does reduce the total number of blunt trauma injuries the greatest benefit of body armor in a combat environment is in reduced penetrating injury. Because it so effectively reduces the risk of penetrating injury the overall effect of increased body

armor use is likely a relative increase in the portion of combat-associated injuries caused by blunt injury [not an increase in total number of blunt trauma injuries]. Although body armor may prevent penetrating chest injury serious blunt injury behind the armor can still occur when armor is struck by a high energy object. Body armor can thus convert what could have been a fatal penetrating injury into a behind-armor blunt injury when a combatant is struck with a high energy missile that does not penetrate.

There are also circumstances in which fragments, particularly large fragments, can cause blunt, not penetrating injury. As noted by Chisolm during the Civil War, "The lung…may be severely injured when no perforating wound exists. A blow by a spent ball, or a fragment of a shell, may make a superficial wound or bruise in the skin, and yet may shatter one or more ribs, driving the spiculae into the lung, lacerating to a greater or less extent its substance. Even without fracture of the ribs, the concussion or blow may have been sufficiently great to have caused irreparable injury to the lung."[1(p.282)]

Rib Fractures

In civilian settings, where blunt trauma dominates, rib fractures are the most common type of chest injury.[71] Although the precise incidence is unknown, studies have shown that most civilian rib fractures occur as the result of motor vehicle accidents.[71-73] The exact incidence of rib fractures among combat casualties is unknown. In World War II about 75 percent of all combat-incurred penetrating and perforating wounds of the chest were associated with compound fractures of one or, more often, several ribs.[49]

Accurate diagnosis of rib fracture(s) in the forward area is difficult but fortunately rarely important. The two important management issues in patients with presumed rib fractures are maintenance of pulmonary function and pain management. Pain management is important irrespective of whether one or more ribs are actually fractured. Oral pain medications are usually sufficient for most combatants with symptoms and findings suggestive of rib fracture(s). To the extent possible combatants with simple rib fracture(s) should be encouraged to continue their duties and deep breathing should be recommended to help prevent atelectasis and pneumonia. The general recommendation is that chest binders, belts, and other restrictive devices should not be used because they limit chest excursion (which is how they reduce pain), and promote hypoventilation with its attendant risk of atelectasis and pneumonia. In young, fit, previously healthy combatants the risk of atelectasis and pneumonia following a single, uncomplicated, rib fracture is very low so in this group the use of a rib belt may not be unreasonable if it allows them to remain functional.

Pneumonia can occur as a complication following rib fracture although this is generally not a serious risk in previously healthy combatants. Bulger et al[74]. found that among patients with rib fracture(s) admitted to a Level I trauma center pneumonia occurred in 31% of elderly versus 17% of young ($p < 0.01$) patients. More significantly the mortality from pneumonia following rib fracture was over twice as high in the elderly as in younger patients. Not surprisingly, mortality and pneumonia rates were found to increase as the number of rib fractures increased. For each additional rib fractured the odds ratio for death rose 1.19 and for pneumonia it rose 1.16 ($p < 0.001$).[74] As the average age of combatants goes up this may become an increasingly important issue in combat casualties. Many casualties with rib frac-

tures will require narcotic preparations, but care should be taken to avoid excessive analgesia with its associated sedation and hypoventilation.

If there is a reasonable question about whether the rib fracture(s) is/are accompanied by more serious injury or if there is obvious compromise of the patient's ability to cough, breathe deeply, and maintain activity, it would be advisable to evacuate the casualty to a hospital setting for more aggressive pulmonary treatment. When there is significant pain and/or when narcotic pain medications can't, or shouldn't, be administered, intercostal nerve blocks with a long-acting anesthetic such as bupivacaine with epinephrine may relieve symptoms up to 12 hours. Performance of intercostal nerve blocks obviously requires that the care provider possess the requisite skills, knowledge, experience, and equipment to perform this procedure. Pneumothorax is a potential and not rare complication of this procedure.

Pulmonary Contusion and Flail Chest

One of the more serious complications of blunt chest trauma is pulmonary contusion. In civilian settings roughly 70% of pulmonary contusions are the result of motor vehicle crashes.[75] Other causes include falls from a significant height and penetrating thoracic injury. While penetrating injury can cause pulmonary contusion it does not do so to the same degree as blunt trauma. In the Yale trauma registry seventeen percent of patients with multiple blunt trauma injuries had pulmonary contusion.[76] Pulmonary contusion, the most common pulmonary parenchymal injury in blunt chest trauma, is present in 30% to 75% of patients with significant blunt chest trauma.[77-80] By contrast, pneumothorax is present in 15% to 50% of patients with blunt chest trauma.[73] Blunt chest trauma and its related complications are responsible for up to twenty-five percent of all blunt trauma mortality.[75] In multi-trauma victims, mortality in association with pulmonary contusion can be as high as thirty-five percent.[81] The exact incidence of death in patients with isolated pulmonary contusion is difficult to know because concomitant injury is almost always present-- usually it is reported to be between 10 and 25 per cent.[82-84] With more than 200,000 cases of blunt chest trauma annually, there may be as many as 15,000 deaths due to this cause each year.[75]

Although penetrating injuries of the chest have historically been more common in combat casualties than blunt injuries, blunt injuries do occur. In civilian trauma approximately 70 per cent of blunt chest trauma is the result of motor vehicle accidents.[85-87] While blunt chest trauma is still most commonly caused by motor vehicle crashes, the increased use of restraints has decreased severe blunt chest trauma from 22.8 per cent to 9.7 per cent.[75]

Blunt chest trauma impairs ventilation by interfering with the movement of the chest wall and it often also causes contusion injury to the underlying lung resulting in ventilation-perfusion mismatch. Adult respiratory distress syndrome (ARDS) complicates 5-20 per cent of patients with pulmonary contusions.[84, 88, 89] The pulmonary contusion patient is at greatest risk of developing ARDS within the first 24 hours after injury.[84] Fortunately ARDS does not typically develop in combat casualties at, or near, the point of wounding because it takes time to develop after serious chest trauma. Acute respiratory distress syndrome is characterized by a hypoxemia that does not improve when supplemental oxygen added.[19] Mortality of ARDS remains approximately 35% despite modern intensive care. Increased

pulmonary hypertension indicates a poor prognosis in patients with trauma and respiratory failure.[19]

Rib fractures are very painful and as a result patients with rib fractures usually under-ventilate causing some degree of CO_2 retention. A flail chest, resulting from fractures of three or more contiguous ribs in two places, causes paradoxical movement of the chest wall during respiration. On inspiration, as the uninjured portion of the chest moves outward, the flail segment is drawn inward by negative intrapleural pressure; the reverse occurs during expiration. This is the so called "pendelluft" phenomenon. In the past, hypoxemia and pulmonary dysfunction associated with a flail chest were attributed to hypoventilation caused by the reduced efficiency of ventilation caused by this "pendelluft" phenomenon.[90-92] It has now been shown that the paradoxical wall motion in flail chest does not cause air movement from damaged to normal lung. Minute ventilation and O_2 uptake may actually be greater on the side of the flail segment.[67] The principal cause of hypoxia in flail chest injured patients is the pulmonary contusion that is always concomitantly present in this injury.[93] When flail chest injury is present, in addition to pulmonary contusion, there may also be intra-abdominal injury because significant traumatic forces are required to fracture multiple ribs. These associated internal injuries are the primary cause of the 5% to 7% mortality rate in patients who have multiple rib fractures.[94, 95] In some studies mortality from flail chest injuries has ranged from 10% to 50% due to the severity of the underlying injuries.[67, 94, 96]

The basic lesion in pulmonary contusion is disruption of the parenchyma. There are several mechanisms that have been proposed to explain the pathophysiology of pulmonary contusion. Wagner et al. identified four types of lesions that may be responsible for causing pulmonary contusion:[97]

- Type I lesions are caused by compression of the chest wall against the lung parenchyma – this mechanism is responsible for most cases of pulmonary contusion
- Type II lesions are caused by a shearing of lung tissue across the vertebral bodies.
- Type III lesions occur when a fractured rib, or ribs, injures the underlying lung.
- Type IV lesions occur when shearing forces across previously formed adhesions tear the pulmonary parenchyma.

Another proposed mechanism is that differential acceleration of alveoli and bronchial structures causes the alveoli to be stripped away from bronchioles. Finally, concussion from a blast wave may also cause pulmonary contusion by spalling.[98] Spalling is what happens when a concussive wave passes through a tissue-gas interface and part of the tissue at the interface is sheared off.[75, 99, 100]

Although widening of the distance between the alveolar spaces and passing blood in pulmonary capillaries can contribute to hypoxemia in patients with pulmonary contusion, serious hypoxemia is primarily due to hypoinflation.[84, 101] Ventilation in the area of the contusion may be decreased by as much as 44 percent.[102] Although not generally an issue at, or near, the point-of-wounding, atelectasis and infection can also be important contributors to hypoxia at 24 to 48 hours after injury.[101-103]

By reducing blood flow to under-ventilated areas of contused lung, hypoxic

vasoconstriction reduces shunting (ventilation-perfusion mismatch) thereby reducing the degree of hypoxia that would otherwise be present.[104-109] In uncomplicated cases, the pulmonary contusion begins to resolve within a few days and usually resolves within a week.[75, 103, 104]

Pulmonary contusion is concomitantly present in roughly 75 percent of cases of flail chest injury and when it is present morbidity and mortality are more than doubled.[82-84] Although not specific, hypoxemia is the most common finding in pulmonary contusion so when it is present contusion should be considered in the differential diagnosis.[75, 108]

Management of Flail Chest

Because it was previously felt that it was the movement of the flail segment that was the primary cause of hypoxia, earlier treatment focused on reducing the movement of this segment. First aid measures involved external methods to reduce movement of the flail segment and in-hospital treatment involved "internal stabilization" using endotracheal intubation with mechanical ventilation and positive end-expiratory pressure. Experience has shown, however, that the incidence of pulmonary infections, the number of days a patient spends in the intensive care unit, the duration of hospitalization, and mortality are all reduced when flail chest or multiple rib fractures are managed noninvasively.[110-112] Patients with flail chest should have their ventilatory status frequently assessed, adequate analgesia should be provided (to include intercostal nerve blocks), and coughing and deep breathing should be encouraged. Mechanical ventilation should be reserved for the patient with severe pulmonary failure from the underlying lung injury; early intubation is <u>not</u> indicated.

Close respiratory monitoring of patients with serious blunt chest trauma with continuous pulse oximetry and reexamination must be maintained because, as pointed out earlier, about half of all respiratory failures resulting from blunt chest trauma occur within the first few post-injury hours.[113] An elevated alveolar-arterial gradient or a low PaO_2/FiO_2 ratio on initial arterial blood gas should raise suspicion.[114] Good pulmonary toilet is important and may be achieved in a variety of ways which can/should be used in combination. These include nasotracheal suction, chest physiotherapy, postural drainage, and blow bottles are simple yet effective ways to minimize atelectasis and expel bronchial secretions.[75]

In patients with pulmonary contusion it is important to avoid over-hydration because, as pointed out earlier, hypervolemia worsens fluid extravasation into the lungs. In the absence of shock, fluids should be given judiciously but under-resuscitation should also be avoided if the patient is in shock. Rapid, and overly aggressive, fluid resuscitation with crystalloids can increase the volume of the pulmonary contusion and may even injure adjacent healthy lung.[108, 115, 116] When there is hypovolemia due to blood loss blood should be given. In the past colloid preparations have been suggested as a reasonable fluid choice for patients with blast lung and pulmonary contusion however colloids do leak out of the capillaries and there is no evidence that outcomes are improved by their use.[75, 117]

The goal of fluid replacement in thoracic injured casualties should be to restore and maintain euvolemia. The patient without extensive multiorgan injury who responds to aggressive pulmonary toilet fluids can be managed by monitoring the adequacy of end organ perfusion using clinical and laboratory data.[75]

Thoracoabdominal and Diaphragmatic Injury

Injury to the diaphragm in combat situations is almost always the result of penetrating injury but as previously stated, as the incidence of penetrating trauma of the chest has been reduced by the use of body armor, the proportion [not absolute number] of diaphragmatic injury caused by blunt trauma will likely be increased. Diaphragmatic injury caused by a penetrating missile is different from that caused by blunt injury in terms of mechanism of injury, pathophysiology, and anatomy as will be discussed later.

During World War II penetrating injuries of the diaphragm were categorized under the more general heading of "Thoracoabdominal Injuries" -- and in virtually every case, because the term "thoracoabdominal wound" was reserved for only those chest and abdominal wounds caused by a single missile, one did not exist without the other; although occasionally a missile that entered the chest could transmit sufficient force through an intact diaphragm to produce an abdominal injury this was a rare occurrence.

Between 1943 and 1945 the 2D Auxiliary Surgical Group treated a total of 903 casualties with thoracoabdominal injury; 247 of these being fatal.[34(p.414)] During this same period the 2D Auxiliary Surgical group treated 1,364 casualties with thoracic injuries so in their experience thoracoabdominal wounds accounted for almost 40% of all wounds of the chest – a surprisingly high percentage that underscores the importance of looking for this injury in all casualties with penetrating injuries of the chest. In this series artillery and mortar fire caused 590 [65.3%] of these thoracoabdominal wounds, and rifle, machine-gun, and pistol fire caused 245 [27.1%]; the remainder [7.5%] being caused by bombs, mines, and grenade fragments.[118(p.137)] In 837 [92.7%] of the cases, the penetrating missiles entered the abdomen through the thorax; in the other 66 [7.3%] cases, they entered the abdomen and then penetrated the diaphragm. In 418 [46.3%] cases, the wounds were on the right side and in 419 [46.4%] on the left side; this being a significantly different distribution of diaphragm injury than that seen in civilian blunt trauma injury (in which left side injuries predominate), as will be discussed later. In 20 [2.2%] cases both diaphragms were perforated. In the remaining 46 cases this information was not documented.

The epidemiology of penetrating injury of the diaphragm in civilian casualties is different from those occurring in combat. A study by Demetriades[119] found that of 163 penetrating injuries of the diaphragm 139 were caused by knife wounds and 24 were caused by gunshot wounds; only 75% of these patients had concomitant intra-abdominal injury making this group considerably different from combat casualties with wounds of the diaphragm in whom some degree of intra-abdominal injury was almost universal – this no doubt being due to the predominance of knife wounds in the civilian series. In this civilian series early diagnosis and treatment resulted in a 3.2% mortality rate as compared to a late mortality as high as 30% in some groups; this finding confirms the importance of early diagnosis and treatment that was observed during World War II.

When considering whether a missile might have caused a thoracoabdominal wound both anatomic and physiologic factors must be considered. Because the plural reflection generally follows the outline of the periphery of the ribs and costal cartilages any missile that reasonably could have passed from one side of this bound-

ary to the other should be assumed to have penetrated the diaphragm. But anatomy is not the only factor to consider; physiology also plays a role. The diaphragm is not simply an immobile structure that forms a consistent boundary between the chest and the abdomen; if this were so diagnosis of thoracoabdominal injury would be much simpler. The diaphragm is a domed, dynamic organ that moves a considerable distance during the respiratory cycle; these factors significantly increase its susceptibility to penetrating injury. In full expiration, any missile entering below the seventh interspace posteriorly or the fourth rib anteriorly might cause injury to the diaphragm.[118(p.106-108)]

The upper limits of the abdomen are bounded by the diaphragm, which in full expiration elevates to the level of the nipples anteriorly and the tips of the scapula posteriorly. These correspond to the level of the fourth and sixth ribs, respectively. Because the thoracic cavity extends inferiorly to the costal margins, many intra-abdominal organs are within the region of the lower chest.

The position of the casualty at the time of wounding must also be considered. Penetrating missiles entering even the uppermost portions of the chest of a prone combatant might well pass through the diaphragm causing thoracoabdominal injury.[118(p.102)]

Injury to the diaphragm can occur in both blunt and penetrating thoracoabdominal wounds. Diaphragmatic rupture is seen in 1% to 3% of patients with blunt chest trauma.[120(p.92)] The mortality rate of blunt diaphragmatic injury is high because when diaphragmatic rupture is present it is indicative of very severe injury.[120] It is generally reported that most diaphragmatic ruptures occur on the left side (9:1 left sided predominance);[120] this being typically attributed to "protection" that is afforded by the liver;[121] although this has been disputed [122] (more on this later).

In civilian trauma the incidence of diaphragmatic injury is estimated to be 1% to 6% of all patients sustaining multiple trauma.[123-125] Maddox et al. found that rupture of the diaphragm occurs in approximately 5 per cent of cases of severe blunt trauma to the trunk, and that in this group the mortality may be as high as 50 per cent.[126]

A comprehensive English literature review of 2288 civilian patients with diaphragmatic injury found that the injury was caused by penetrating trauma 53% of the time (of which 55% were knife wounds and the remainder gunshot wounds) and blunt, 47% of the time. Eighty-six percent of the cases of blunt traumatic diaphragmatic rupture occurred during a motor vehicle accident, 4% were caused by auto versus pedestrian accidents, and falls, motorcycle accidents, and crush injuries each caused 3%.[124, 127]

Sarna and Kivioja reviewed forty-one cases of blunt diaphragmatic injury and found that 71% were caused by motor vehicle accident and 10% by a fall from a height. The mortality rate in their series was 12%; with death and most of the complications being related to associated injuries and not the result of the diaphragmatic injury; splenic injury being common.[128]

Blunt, unlike penetrating, injury to the diaphragm is caused by creation of a sudden pressure differential between the pleural and peritoneal cavities. This pressure differential can force organs from one cavity through the diaphragm and into the other. A common mechanism of blunt traumatic injury is lateral impact of an occupant's chest wall during a motor ve-

hicle crash. This causes distortion of the chest wall and tears the ipsilateral diaphragm. Forces applied to the front of the abdomen, such as might occur from a high-riding safety belt during a front-on accident, can create a large radial tear in the posterolateral aspect of the diaphragm and can force abdominal contents into the chest cavity.

Most series of blunt diaphragmatic injury have demonstrated a predominance of left sided injury with 65% or more, and in some series as much as 85%, being left-sided.[125, 129-131] Right-sided ruptures, although less common, have more severe associated injuries and they generally require greater force of impact. The exact reason for this predominance of left-sided diaphragmatic injury is unclear but it has been attributed to "protection" that is afforded to the left diaphragm by the liver. In Shah et al.'s series of 980 patients with diaphragmatic rupture there were 1,000 injuries, of which 685 (68.5%) were left-sided, 242 (24.2%) right-sided, 15 (1.5%) bilateral, and 9 involved pericardial rupture (0.9%); 49 remained unclassified.[130] In a series of 20 patients with traumatic rupture of the diaphragm Anyanwu et al. found the left hemidiaphragm was ruptured in 85 per cent and the right in 15 per cent of cases.[131] Troop et al. found that sixty-four percent of the blunt diaphragm injuries in their series were left sided.[125]

Aronoff et al.[132] at the University of Texas, retrospectively reviewed 161 cases of diaphragmatic injuries and assessed the reliability of various diagnostic studies in diagnosing these injuries. Eighty-nine percent of the diaphragmatic injuries in this study were the result of penetrating trauma. In their series the left hemidiaphragm injured 60% of the time and the right 36%.

Contrary to most other studies, Waldschmidt et al., from the University of Alabama, in a retrospective study of 86 cases of diaphragmatic rupture, found no great predominance of left-sided injury, with 58% being left-sided and 39.5% being right-sided). Initial mortality in this series was approximately 16% initially, but it increased significantly to 53%-66% if diagnosis was delayed and bowel obstruction develops from incarceration of herniated viscera.[133]

It has been suggested that ruptures of the diaphragm actually do occur to both sides in almost equal frequency but because those occurring on the right side are generally more lethal, with many dying before reaching a hospital;[122] most hospital-based series appear to show a predominance of left-sided injury.[120]

Physical Examination

Unfortunately field diagnosis of thoracoabdominal injury is problematic and the best that can be hoped for is to use the tools of history, signs and symptoms, and physical examination to raise or lower the probability of thoracoabdominal injury in a patient with a penetrating chest wound.[118(p.109)] Sukul et al. in their study of 63 patients with traumatic diaphragmatic injuries found that specific signs or symptoms of diaphragmatic injury were generally absent. Diagnosis prior to operative intervention was only made in 32% of the patients and in only one case was it suspected on the basis of physical findings (peristalsis on chest examination).[134]

Diagnosis of diaphragmatic injury is difficult even for physicians with access to all the latest diagnostic tools. Diagnosis of diaphragmatic injury is made preoperatively in only 40-50% of left-sided and 0-10% of right-sided blunt diaphragmatic ruptures; perhaps this is another reason

why left sided injuries of the diaphragm predominate in most series. In 10-50% of patients, diagnosis is not made in the first 24 hours. In Shah et al.'s review of 980 patients with diaphragmatic rupture the diagnosis was made preoperatively in 43.5% of cases; in 41.3% it was made at exploration or at autopsy and in the remaining 14.6% the cases the diagnosis was delayed. The mortality was 17% in those in whom acute diagnosis was made, and the majority of the morbidity in the group that underwent operation was the result of pulmonary complications.[130]

Aronoff et al., in their review of diaphragmatic injury also found physical examination to be generally unreliable. Examination of the abdomen was unremarkable in 44% of the patients with penetrating injuries and in 55% of blunt injuries. They found physical examination to be more reliable when the injury involved the left rather than the right hemidiaphragm. Decreased breath sounds were documented in 49% of the patients with penetrating trauma and 61% of the patients with blunt trauma.[132]

Although physical examination is limited in its ability to diagnose diaphragmatic injury this diagnosis is suggested when auscultation of the chest reveals bowel sounds in the chest or when there is dullness to percussion.

Because diaphragmatic injury is rarely the immediate life-threat in multiply injured patients, the pre-hospital physical examination should, as always, focus the identification and treatment of the more common life threatening problems; specifically it should focus on identification of injuries, such as tension pneumothorax, that can be treated in a pre-hospital setting.

As previously noted clinical presentation is not always helpful in making the diagnosis of thoracoabdominal injury because thoracic wounds not involving the abdomen often cause pain and spasm of the upper abdomen, and sometimes even abdominal tenderness and rigidity. Olin[135] noted that pain in the upper abdomen and dyspnea, especially in the recumbent position, is suggestive of diaphragmatic rupture. Brewer, in his review of WWII thoracoabdominal wounds,[118] does suggest that when deep abdominal tenderness to palpation and rebound tenderness are present in a casualty with penetrating thoracic trauma, abdominal injury is likely also present because these findings are never elicited in patients with isolated chest wounds.

He also noted that while pain in the shoulder is not universally present in patients with abdominal injury, when it is present in a casualty with a penetrating chest wound [one that doesn't involve the shoulder], injury to the diaphragm should be suspected.[118(p.109)] The presence of nausea and vomiting also suggests the presence of abdominal injury because while these can sometimes occur in isolated thoracic trauma, they are not common. While the presence of normal bowel sounds some time after injury suggests that there is no hollow viscus injury they say nothing about the possibility of solid organ injury in the abdomen; in such cases active peristalisis often persists.[118(p.109)] The presence of blood in vomitus also suggests abdominal injury but this is not conclusive because the blood may have originated in the lungs. Blood in the urine also raises the likelihood of concomitant abdominal injury in the chest wounded casualty.[118(p.110)] Serial examination is essential in all patients with chest trauma who could have injury to the diaphragm and who do not undergo laparotomy.[130]

Peritoneal Lavage

Although not a procedure that can, or should, be performed at, or near, the point-of-wounding, diagnostic peritoneal lavage (DPL) can help make the diagnosis of thoraco-abdominal injury; and this procedure should be performed, when indicated, at the level of the Battalion Aid Station. The results of the DPL can be used to make triage and evacuation decisions.

Although frequently helpful it is important to note that a DPL can be negative in thoracoabdominal injury, particularly when there has been a single penetrating injury. An isolated penetrating chest wound with diaphragmatic penetration can cause bleeding into the lesser sac, which may not communicate with the rest of the peritoneal cavity and would therefore show no evidence of bleeding after a DPL. Positive DPL results, as manifested by drainage of lavage fluid out of the chest tube or greater than 10,000 RBC/mm_3 in the lavage fluid correlate with diaphragmatic injury.

Aoronoff et al. found that peritoneal lavage, when performed, was falsely negative in 36% of patients with blunt trauma, in 17% of patients with penetrating trauma, and in 23% and 18% of right and left hemidiaphragm injury, respectively. In a patient with chest tubes in place, several liters of fluid may be instilled into the abdomen at the time of lavage and chest tube output monitored to see if this fluid comes out of the chest.[132] Troop et al. suggested that the diagnosis of thoracoabdominal injury can be improved by instilling a small amount of methylene blue into peritoneal lavage fluid to make it easier to see if this fluid drains out of a chest tube. Methylene blue can also be instilled into the chest through a chest tube and its presence sought in peritoneal lavage fluid.[125]

Ultrasound

Ultrasound is increasingly being used to diagnose intra-abdominal injury and it is particularly effective at picking up intra-abdominal bleeding that is associated with blunt and penetrating solid organ injury. Diagnostic ultrasound is now sufficiently durable, lightweight, and easy to use as to be of use at the level of the battalion aid station. Ultrasound can generally easily pick up large holes in the diaphragm or herniation but it may miss small tears caused by penetrating injuries.

Treatment and Evacuation

Resuscitation of potentially thoracoabdominal-wounded casualties should focus on rapid stabilization, particularly of the casualty's respiratory status, and on rapid transport to a medical facility capable of performing resuscitative surgery on these complicated patients. Death in patients with diaphragmatic injury is almost always the result of associated injury, most specifically injuries to the spleen and the aorta, so these injuries should be the focus of resuscitative efforts. Mortality in civilian series of patients with diaphgramatic injury (mostly blunt) ranges from 15-25%.[120]

As in the management of all serious traumatic injuries, a focus on the ABCs is the cornerstone for prehospital management of patients with diaphragmatic injury. As noted earlier, diagnosis of diaphragmatic injury is difficult even for specialists who have all diagnostic tools available, so it should not be surprising that the diagnosis rarely is made in the field. Fortunately, failing to diagnose this injury in the field is unlikely to have adverse clinical consequences because no specific prehospital treatment is required for this injury. The key to improved outcomes in patients with diaphragmatic injury is maintenance of the

airway and ventilation and appropriate treatment of associated injuries.

Fluid resuscitation should be initiated in the field but should not be overly aggressive; it should be focused primarily on maintaining cerebral perfusion as defined by a normal mental status. Excessive fluid resuscitation both aggravates blood loss and increases the risk of ARDS. When serious and persisting shock is present, blood, whole blood if available, should be administered as soon possible. In the thoracoabdominal-wounded casualty it is especially important to rapidly restore as near normal a respiratory status and oxygenation as possible. The casualty should be positioned for optimal ventilation, sucking chest wounds should be closed by occlusive dressing, pneumothoraces should be alleviated by tube thoracostomy by a qualified healthcare provider as soon as practicable, and, if available, oxygen should be administered. Tube thoracostomy should be done with special caution in any patient with suspected diaphragmatic injury because of the risk of injury to herniated abdominal contents within the pleural cavity. Pain should be alleviated by adequate doses of analgesia (morphine) and/or intercostal nerve block.[118(p.112)] A nasogastric tube should be inserted and connected to low intermittent suction as soon as this is available and possible. Early antibiotic administration with an antibiotic that will provide adequate coverage for enteric bacteria is indicated for all patients with potential penetrating thoracoabdominal injury.

From the standpoint of isolated thoracic injury, within reason the length of time required to restore a thoracic-wounded casualty to near his normal physiologic status before operation is not critical. It was observed during WWII that most casualties with isolated chest wounds benefited from a short period of stabilization after cardio-respiratory abnormalities had been corrected and shock controlled. This is not the case in casualties with thoracoabdominal wounds; in such patients the time to operation is of great importance. These patients must be resuscitated as rapidly as possible and they should be operated on as soon as they appear able to tolerate surgery.[118(p.112)] Indeed, in those patients with suspected thoracoabdominal injury who persist in shock despite initial resuscitative efforts, immediate surgery is the resuscitation method of choice.[118(p.122)] In the majority the most lethal part of the thoraco-abdominal wound was the abdominal component.

Penetrating Chest Trauma

Penetrating chest trauma is the predominate form of chest injury in combat casualties. Signs and symptoms in patients with penetrating chest trauma may range from minor, even initially unrecognized by the casualty, to grossly apparent and life threatening. Casualties with penetrating chest trauma tend to divide themselves rapidly into one of three categories:

1) Those who die within less then 30 minutes
2) Those who can be managed solely by insertion of a chest tube and, by far the smallest group
3) Those who require thoracotomy.

Very few of the first group can be saved even if injured in immediate proximity to a Level I trauma center equivalent facility and in most instances the last group only becomes apparent when there is persistent and significant bleeding from a chest tube.

Insertion of a chest tube for management of penetrating chest trauma in combat casualties is not new. As long ago as the

American Civil War, surgeons treated intrathoracic collections of fluid in the pleural space by placement of a trocar into the chest. The Confederate Surgeon, J.J. Chisolm, described the indications and procedure for chest tube insertion as follows:

> *"The evil which the surgeon fears from perforating wounds [of the chest]…is that…a[n] effusion may rapidly accumulate in the thoracic cavity…Respiration becomes much embarrassed with marked dyspnoea. Percussing the side, will now give a dull, heavy sound, instead of the ordinary clear, sonorous one of health…When the surgeon has recognized such collections as rapidly forming in the chest after gunshot wounds…an early evacuation will be required…an opening for the escape of the fluid should be made at the point which nature indicates, but, in cases of excessive effusion, any broad intercostal space, between the sixth and eighth ribs of the right, or between the seventh and ninth on the left, might be the point selected…a trocar and canula, should be introduced at right angles to the chest and near the upper edge of the rib, towards its angle, in a line continuous with the posterior border of the armpit.*[1(p.279)]

Not surprisingly missiles that strike the chest and cause injury to the heart or great vessels have a high mortality. Contrarily, missiles, even high velocity missiles, that strike only lung, often cause surprisingly little serious injury. This fact, that the lung is more tolerant of wounding by high velocity bullets than other tissues, has been known for some time.[136(p.179)] In 1939, commenting on chest wounds in the Spanish Civil War, Jolly noted that, "Bullet wounds passing cleanly through the parenchyma of the lung cause little haemorrhage and, when the initial shock of injury has passed, remarkably few symptoms.[7(p.152)] Jolly did, however, go on to note that, "[I]t is the small wound of the chest caused by a tangential bullet or shell fragment, with rupture of the intercostal vessels, which is important and seen most frequently. Until at least 48 hours have passed, no such wound may be regarded as comparatively unimportant; at any time within this period it may give rise to a full haemothorax."[7(p.154)]

Jolly also noted that in the Spanish Civil War roughly half of all combat caused chest wounds do not require operation in the forward area. He went on to say that when there has been a through-and-through bullet wound with no injury to the intercostal vessels the casualty usually does fine with rest and morphine for pain. Although about 90% of surviving casualties with penetrating chest wounds have hemoptysis, in casualties with gunshot wounds of the chest bleeding generally decreases rapidly and stops within a week. Jolly stated that it is better not to attempt removal even if a bullet is retained in the lung as long as there is no extensive bleeding.

Not all of Jolly's recommendations have stood the test of time. Prior to and even during the early years of World War II it was sometimes suggested that a thoracotomy for persistent intrathoracic bleeding might be avoided by withdrawing blood from the chest via a chest tube and then replacing this blood with air under pressure. The thought was that the increased air pressure in the pleural space would decrease the rate of bleeding by decreasing the pressure differential between the bleeding vessel(s) and the pleural space.[7(p.162)] While conceptually sound, practically, the risks (primarily tension pneumothorax) outweighed any potential benefit because the reduction in blood flow gained by this procedure is minimal.

Hemo- and Pneumothorax

Hemothorax, pneumothorax, and hemopneumothorax are common complications of penetrating chest trauma. In penetrating chest injury almost always both blood and air accumulate to some degree within the pleural space. Hemothorax alone can cause hypotension and cardiopulmonary dysfunction both through hypovolemia and by compressing the lung and reducing venous return. Because the pleural space may accommodate 30% to 40% of the blood volume, the most common clinical presentation is hypovolemia.[94(p.481)] Depending upon the extent of bleeding, and the presence or absence of concomitant pathophysiology, the clinical status of a patient with hemothorax can range from stable to moribund. When massive, hemothorax may be difficult to differentiate from tension pneumothorax because both can present with diminished breath sounds, hypotension, tracheal deviation, and cardiovascular collapse. The only distinctions being that in massive hemothorax the neck veins are collapsed instead of distended and the affected side of the chest is dull to percussion instead of tympanic – but these findings are not invariably present even in tension pneumothorax so distinguishing between these two can be difficult in the field.

Because hypotension and exsanguination are the primary concern in casualties with a significant hemothorax treatment of this condition should focus on hemorrhage control and restoration of volume. Partial control of hemorrhage can be achieved by re-expansion of the lung with insertion of a chest tube but severe hemorrhage requires surgical control. As noted earlier in the "Circulation" section when hemorrhage is internal and uncontrollable aggressive volume resuscitation is not recommended.[137] As previously stated the majority of cases of hemothorax, roughly 80 to 85%, can be effectively managed solely by insertion of a chest tube. Current indications for thoracotomy, as promulgated in the Advanced Trauma Life Support Course,[51] are persistent hypotension despite aggressive fluid replacement, initial output of greater than or equal to 1500 mL of blood upon chest tube insertion, continued bleeding from chest tube greater than 200 mL/h for 2 - 4 hours, and/or need for persistent blood transfusion. Other authors have suggested similar guidelines but include as well massive continuing hemorrhage greater than 2000 mL and hemothorax in the presence of a widened mediastinum.[94, 138(p.481)] It should be noted, however, that not all trauma surgeons agree with the concept of using the initial blood loss from a chest tube, as described above, as the prime indicator of need for thoracotomy.

Mattox et al.[139 (pp.345-353)] believe that initial chest tube output alone is not as important as the rate of ongoing hemorrhage. They point out that while initially there may be up to 1500 ml of blood loss through a chest tube, if there is no further bleeding, such casualties can often be managed non-operatively. Demetriades et al. conducted a prospective study of 543 patients with stab wounds of the chest.[140] They found that the best indicators of the need for early thoracotomy were the presence of signs of cardiac or major vascular injury; e.g. tamponade, profuse bleeding, an absent or diminished peripheral pulse, and persisting shock. They suggested that the initial chest tube output, and even the rate of blood loss from the thoracotomy tube, were not reliable indicators of the need for thoracotomy; they recommended that these criteria not be used as the sole indicators for thoracotomy. They even found that some patients initially in shock could be managed non-operatively, as could even most patients with massive air leaks.

It is worth noting, however, that the Demetriaes' series was a civilian series of stab wound patients and results from this series might well not apply to a population of combat casualties with penetrating fragment or bullet wounds to the chest. In a combat zone where there is often inadequate manpower, multiple casualties to attend to, and frequently less than sophisticated diagnostic tools available, a more aggressive surgical policy is likely to produce more favorable results. However, in the event that surgery cannot be performed for some reason, it is useful to know that many patients with combat-associated penetrating chest trauma who survive to reach medical care, even those with significant and sustained blood loss, will likely do well without formal surgical intervention. Finally it should again be pointed out that while combat casualties with penetrating cardiovascular injury who survive long enough to reach medical care often have serious injury, it is quite often those with injuries to intercostal vessels that bleed the most and the longest.

In any case, in the pre hospital environment, all casualties with penetrating chest trauma who meet the ATLS criteria for thoracotomy (as defined above) should be evacuated in the "Urgent" category to a facility with a surgeon capable of performing resuscitative cardiothoracic surgery.[141(p.107)]

Pneumothorax may occur with either blunt or penetrating trauma. In combat settings the great majority are caused by penetrating injury. Blunt trauma can cause a pneumothorax by a variety of mechanisms; a sharp rib fracture can puncture a lung, air can leak into the pleural space following a tracheobronchial injury, and alveoli may burst following blast injury or other rapid compression of the chest wall.[67]

A pneumothorax may be open and "sucking" with exchange of air during respiration through the chest wall defect, it may be closed, or it may be open or closed at various times. A small portion of casualties with a closed pneumothorax go on to develop a tension pneumothorax when air, which enters the pleural cavity during inspiration, can't leave during expiration. This condition is discussed in detail later.

During World War II it was noted that all penetrating chest wounds had the potential to be "sucking" and this observation influenced the treatment recommendations for such wounds (see below). It was noted that a sucking wound sometimes sucked (or blew) constantly and sometimes only when the skin and muscles planes were in a certain alignment. Anatomy at the point of wounding frequently determined whether or not a wound sucked. It was noted that although generally a large wound is more likely to suck than a small wound, size is not necessarily the determining factor. A relatively small wound in the anterior chest wall, 2 centimeters or less in diameter, might suck constantly because in this area the muscles are thin and the rib interspaces are wide, but a larger posterior wound, through the heavy muscles of the back, might not suck at all.[142(p.5)]

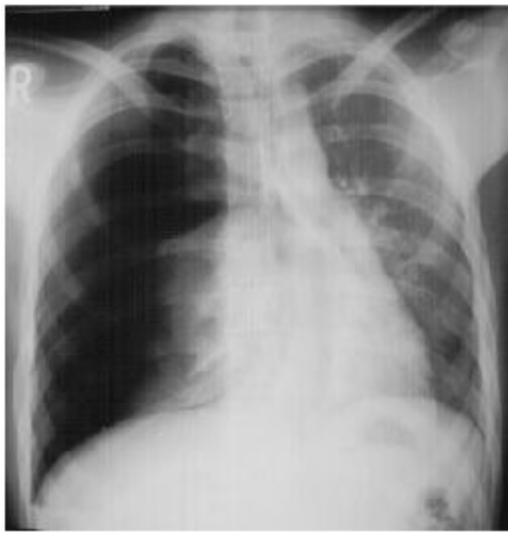
In many instances casualties with a "sucking" chest wound can tolerate the injury if the amount of air entering the wound from outside is not greater than the difference between the tidal volume (resting tidal volume in adult males is approximately 500 cubic centimeters) and the original vital capacity (which is approximately 4800 cubic centimeters in the average adult male). As Brewer and Burford note, without treatment, the outcome of a casualty with a "sucking" chest wound is determined by the size of the wound and the original vital capacity.[142(p.5)] If the size of

the traumatic opening is less than the size of the glottis, which is about 2 centimeters in diameter in the average adult male, an adequate amount of air can usually enter through the glottis to meet physiologic requirements. If the opening in the chest wall is larger and the amount of air entering the chest through the open wound during each inspiration is great enough to interfere with the tidal air requirement intake, asphyxiation is possible.[142(p.8)]

As previously noted, early in World War II aidmen were instructed to cover only "sucking" wounds with an occlusive dressing. Later in the war instruction was given that all chest wounds were to be treated in this manner because a penetrating chest wound that did not suck at one time, or in one position, might suck in another. On the battlefield the recommendation was that any type of dressing that was available could be used, even a piece of clothing.

Tension Pneumothorax

One cause of breathing difficulties in battlefield casualties is a tension pneumothorax -- a closed accumulation of air in the pleural space into which there is an opening surrounded by tissue that allows air to enter but not to escape. Tension pneumothorax has been clinically defined as "The progressive accumulation of air under pressure within the pleural cavity, with shift of the mediastinum to the opposite hemithorax and compression of the contralateral lung and great vessels…".[35(p.388)] As recently described by Domino, tension pneumothorax is a clinical, not an anatomic, diagnosis. Diagnosis is based upon the presence of a pneumothorax and a combination of some or all of the following clinical findings, (1) worsening hypotension (2) tympany on percussion of involved side (3) jugular venous distension and (4) tracheal deviation to the contralateral side. A tension pneumothorax is associated with a marked decrease in pulmonary compliance, increased peak airway pressures, hypoxemia, and hypotension that eventually leads to cardiovascular collapse. Tracheal deviation, while considered a cardinal sign of tension pneumothorax, is inconsistently present and chest wall movement and breath sounds are generally diminished or absent on the affected side in both pneumothorax and tension pneumothorax.[67]



Tension Pneumothorax

One of the most common causes of tension pneumothorax during peacetime is the use of high ventilator pressures in patients with restrictive lung disease. On the battlefield tension pneumothorax is almost always caused by penetrating trauma. While pneumothorax is quite common in combat casualties with chest injury, tension pneumothorax is very uncommon, particularly among those who survive long enough to be treated. Unfortunately, good data on the true incidence of tension pneumothorax in combat casualties is lacking. The reason for this is, as Bellamy, McPherson, and Feigin have stated, ten-

sion pneumothorax leaves little persisting physical evidence, even in the dead, and it is reversible when properly treated, having "a willow-of-the-wisp quality." Diagnosis of tension pneumothorax in the field is difficult because clinical findings such as respiratory and circulatory collapse are nonspecific.[143]

Bellamy and colleagues used autopsy chest radiographs from the Vietnam era Wound Data Munitions Effectiveness Team (WDMET) files to estimate the prevalence of tension pneumothorax in combat casualties. Somewhat surprisingly, in this review they found that eighty percent of the wounds were created by military small arms (an unexpectedly low percent of fragment-caused injury) and all were to the periphery of the lung and not the hilum (injuries of the hilum often being quickly lethal). If Bellamy et al. were able to correctly identify, from post-mortem chest radiographs (the authors themselves state that "using post-mortem chest radiographs for diagnostic purposes is fraught with the possibility of errors…"), those dying from a tension pneumothorax, then according to this study, 3%-4% of fatally wounded casualties (not all casualties) recorded in the WDMET database died from a tension pneumothorax.

During World War II traumatic tension pneumothorax was rarely seen in forward hospitals, although it was somewhat more common as a postoperative or posttherapeutic complication. Its infrequency is evident in the statistics of the 2d Auxiliary Surgical Group. Tension pneumothorax was encountered by the 2nd Auxiliary Group only 11 times in its treatment of 2,267 thoracic and thoracoabdominal injuries (0.9%).[144 (p.148)]

This infrequency of observed tension pneumothorax during World War II was explained as being likely due to the following:

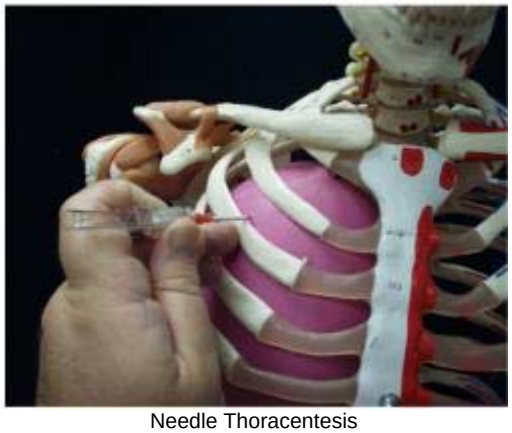
1) The preponderance of shell-fragment wounds, which resulted in a preponderance of large defects of the chest wall. For physical reasons, air could not be entrapped in the chest in such wounds. [NOTE – If true this could also explain the predominance of bullet-caused tension pneumothoraces seen in the Bellamy study. Now that improved conventional munitions, with their very small fragments, have almost completed replaced unimproved munitions it is unlikely that this explanation would be valid today].
2) The routine application of occlusive dressings tightly enough to end the sucking or blowing effect of the open chest wound but not so tightly that a tension pneumothorax was built up.
3) The almost routine use of a flapper-valve decompressive catheter in collecting stations or battalion aid stations.
4) The excellent screening of inductees in World War II, as a result of which preexistent restricting pleural adhesions, which were rather frequent in World War I soldiers, were extremely uncommon [it is unclear how standard medial screening of inductees would have picked up this condition]

It was felt that the majority of tension pneumothoraces observed during World War II were the result of continued leakage of air from lacerated pulmonary parenchyma combined with closure of the external chest wound which converted an open pneumothorax into a tension pneumothorax.[144(p.149)]

Despite representing only a relatively small percentage of all medical conditions present in combat casualties a tension pneumothorax is very important to recognize because it is one of the few, potentially fatal, combat-caused medical problems that can be treated, at least temporarily, with minimal training and equipment. While treating a tension pneumothorax is no guarantee of ultimate survival, rapid, and appropriately performed needle thoracentesis, may allow many with this condition to live long enough to reach more definitive care.

Thus this small group of combat casualties represents a significant subset of combat casualties sustaining an injury which while rapidly fatal if untreated, could be readily treated at, or near, the point-of-wounding. When compared to an estimated 20% of combat casualties who die from treatable blood loss,[145-147] and to the less than 1% who die from airway obstruction,[148] perhaps as much as a third of all potentially life saving first aid for combat casualties could be realized by effective emergency treatment of tension pneumothorax.[143] It is for this reason that early diagnosis and effective treatment of tension pneumothorax is so important. Unfortunately tension pneumothorax often causes death before treatment can be rendered and, as noted earlier, it is difficult to correctly diagnose in the field since most of its signs and symptoms are not specific. Therefore a presumptive diagnosis of tension pneumothorax should be made in every casualty with a penetrating chest injury and significant respiratory distress, particularly if there is also hypotension. Battlefield diagnosis of tension pneumothorax should not be based upon the presence of the classically described findings of decreased breath sounds, tracheal deviation, and hyperresonance to percussion, because these signs may not always be present.[149] Even if present these signs may be exceedingly difficult to appreciate on the battlefield.[149]

The practical consequence of these facts is that if the benefits of treatment are to be realized in combat casualties, rapid diagnosis must be made, and treatment rendered, at, or near, the point of wounding, by one of the least trained members of the health care team -- the combat medic. This means that the threshold for treatment must be low or this potentially treatable condition will go untreated until too late. Bellamy et al. have suggested that because of the difficulties in diagnosing tension pneumothorax in the field and because, if it is to be of use at all, needle thoracentesis must be done quickly by medical personnel nearest the point of wounding. According to Bellamy medics should be taught to do this procedure in every decompensating casualty with an apparently penetrating chest injury who does not also have a serious head injury. In some civilian settings paramedics are authorized to perform needle thoracentesis[150, 151] so it would seem that this procedure is reasonably within the capabilities of combat medics and corpsmen.

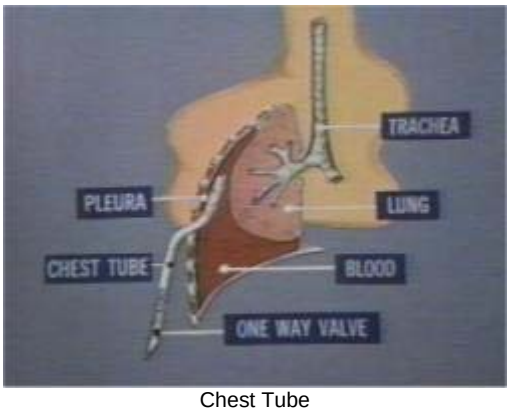


Needle Thoracentesis

Almost all casualties with penetrating chest trauma have some degree of hemo/pneumothorax and it is unlikely that the additional trauma caused by a needle

thoracostomy would significantly worsen their condition should they not actually have a tension pneumothorax.[151] The effect, however, of a low treatment threshold for tension pneumothorax would be that many casualties without this condition would also be treated, exposing them to the risks of treatment (principally creation of a pneumothorax in a casualty who does not already have one, and less often, hemothorax), without hope of benefit.

A recent civilian observational study by Cullinane et al. found that only one of the 19 trauma patients in their study who had a needle thoracentesis performed received any significant benefit from the procedure and whether even this patient would have had significant problems without the needle decompression is unclear.[152] It is important to note however that only five of these nineteen patients had sustained penetrating injury making this a very different group of patients than generally seen in combat.



Chest Tube

Definitive treatment for both pneumothorax and tension pneumothorax involves insertion of a chest tube through the fourth or fifth intercostal space, anterior axillary line – a procedure that most combat medics, and civilian paramedics, are not trained to perform. In any case tube thoracostomy is not needed for initial treatment of a tension pneumothorax – in most instances needle thoracentesis is sufficient. Finally, there is currently no data showing benefit from point-of-wounding tube thoracostomy when performed by civilian paramedics,[150, 151] and certainly none demonstrating its efficacy in a combat setting, performed by corpsmen or medics. This does not mean, however, that there is no benefit, just that there is no proof of benefit at this time.

Holcomb et al. have shown that needle thoracentesis with a 14-gauge needle rapidly relieves elevated intrapleural pressure in a swine model of traumatic tension pneumothorax.[153] Holcomb's study showed that the therapeutic effect of needle thoracentesis was sustained for 4 hours and throughout this period it was equivalent, in its ability to relieve tension, to tube thoracostomy with a 32F chest tube.[153] Minimal equipment requirement, ease and speed of performance, and low likelihood of serious complications makes needle thoracentesis the procedure of choice to relieve tension pneumothorax on the battlefield. Unfortunately, as noted above, there is no data to show that the apparent benefits of this procedure outweigh its cumulative risks. The risks, which include hemorrhage and creation of a pneumothorax, while generally of little-to-no consequence in the majority of combat casualties, may be of significance in those whose physiologic state is already precarious from blood loss and hypoxia. Thus, while training combat medics and corpsmen to perform needle thoracentesis seems reasonable an effort should be made to collect data to show that overall benefits exceed risks.

In performance of needle thoracentesis cannula length is an important consideration. [149, 154-157] The pectoral muscles, which can be very thick in young solders, must be penetrated in order to effectively treat a tension pneumothorax so the

cathter used must be sufficiently long. While difficult to appreciate in a combat situation, if there is no rush of air when the needle is inserted, it must be assumed that either the needle was too short, or that the casualty did not have a tension pneumothorax. Ranger medics currently use 10ga 3-inch needle/catheters for this procedure. (Personal communication – SFC Rob Miller, Senior Army Ranger Medic)

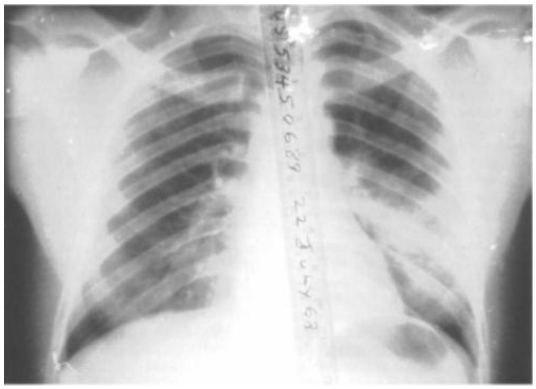
Casualties on whom a needle thoracentesis has been performed must be continually re-assessed because catheters used for this purpose can easily become occluded by blood clots and kinking.[50(p.8)] If an Asherman Chest Seal is available one could be placed over the needle to stabilize it and help prevent dislodgement as described by Allison et al.[158]

Injury to Trachea and Mainstem Bronchus

Injuries to the trachea and mainstem bronchus are extremely serious but fortunately rare. They are most often the result of penetrating lung injuries, but also occur in 1% to 3% of patients with severe blunt lung trauma.[159] In civilian series the mortality of civilian trauma patients with a tracheal injury is 30%, half of whom die within the first hour.[91, 94] Traumatic rupture of the bronchus is also rare with a similarly high case fatality rate. In Bertelsen and Howitz's study, approximately 3% of 1128 patients at autopsy had evidence of tracheobronchial injury, with most (81%) having died before reaching a hospital.[160] In civilian series penetrating injuries to the bronchus are rarely seen in the emergency department both because of the overall rarity of penetrating chest injury in this series and because most with this injury die quickly from associated great vessel injury. Charity Hospital in New Orleans reported only 22 cases in a 20-year period.[161]

Blast Lung

Although primary blast injury generally accounts for less than 3% of all combat casualties, it is essential that forward medical care providers recognize, and appropriately treat and evacuate, combat casualties with this injury. Patients exposed to underwater blast or to explosions in enclosed spaces are at much higher risk than other casualties of sustaining primary blast injury.[162] Troops exposed to blast enhanced weapons (truck/car bombs, improvised-explosive-devices (IEDs), fuel-air explosives, thermobaric weapons etc.) are also more likely to sustain primary blast injury. Primary blast injury is difficult to identify even for experienced medical personnel because there may be no external evidence of injury and, at least initially, such patients may appear relatively unharmed. Patients suspected of sustaining blast lung injury should not, if at all possible, be allowed to assist in their own evacuation because any exertion by such patients significantly increases the probability of death. Additionally, while supplemental oxygen is of little value to most combat casualties, patients with blast lung (or any cause of pulmonary contusion) often need oxygen to survive.



Chest x-ray showing small fragment wounds and *primary blast injury* of the lung resulting in pulmonary contusions demonstrated as infiltrates under the left chest wall.
From the Textbook of Military Medicine, part I, volume 5, page 302, figure 9-2.

During World War II much of the British experience with blast injury was gained through German bombings of Britain. The relative frequency of primary blast injury among civilian casualties of these bombings for a time misled U.S. Army medical officers into believing that blast injury would be a common occurrence in combat; it was not. Blunt injuries of the intact chest, with resulting contusion, were frequent, but blast injuries were uncommon in combat casualties during World War II. Early in this war these two conditions were frequently confused.[142 (p.32)]

Capt. William W. Tribby, MC[163] in his study of 1,000 battlefield deaths of U.S. Army troops in Italy, found 13 bodies in which there were no penetrating injuries and in which the cause of death was presumably blast injury; several other casualties might also have died of blast injuries, for while penetrating wounds were present, they were not sufficient in any instance, to account for the fatality.[142(p.32)] Nonetheless it was clear to Tribby that primary blast injury was an uncommon cause of death in WWII combat casualties.

Data prepared by the Medical Statistics Division, Office of the Surgeon General, Department of the Army, show 1,021 blast injuries of non-battle origin in the U.S. Army for period 1942-45, of which 48 were fatal. For the same period, there were 13,200 battle-incurred blast injuries, 140 being fatal. Of the 6,284 blast injuries occurring in 1944, 76 were fatal; 493 involved the chest, and 25 of these were fatal. The WWII naval experience with blast injury was considerably more extensive than that of the Army this no doubt being due to the fact that the principal munitions used in naval combat are large high explosive shells intended to destroy ships not people.

Circumferential Burns of the Chest Wall

Because of the increased military use of vehicles powered by hydrocarbon fuels these flammable propellants have increased the risk of burn injury during both combat and non-combat operations. The widespread use of vehicle born improvised explosive devices by enemy combatants during operation Iraqi Freedom has been a major source of combat-related burn injuries. Combustion of these fuels can cause problems with ventilation and respiration in several different ways:

- Inhalational burn injury which usually causes upper airway problems can also cause problems with ventilation and respiration
- Consumption and displacement of oxygen producing hypoxemic hypoxia
- Generation of carbon monoxide resulting in carboxyhemoglobin
- Mechanical interference of respiration by circumferential burn injury of the chest wall.

Although rare circumferential burn injury of the chest wall can cause restriction of ventilation that may critically impair the ability of a seriously burned casualty to adequately ventilate. When circumferential burn injury impairs breathing it is vital that this condition be quickly diagnosed and properly treated. Circumferential full-thickness burn injury of the chest can restrict the bellows movement of the chest resulting in a reduced tidal volume. This reduced tidal volume, particularly when combined with the frequently concomitant inhalational injury and occasionally present histiocytic hypoxia of carbon monoxide and/or cyanide intoxication, can be lethal. When this rare injury occurs treatment involves performance of an escharotomy to release the chest wall permitting

expansion of the lungs. While this procedure is not one that would be done by most pre-hospital providers, it can and, in some instances should, be done in the field by someone properly trained to diagnose and treat this condition.

Triage and Evacuation of Casualties with Oxygenation and Ventilation Problems

While some problems of oxygenation and ventilation can be definitively addressed at, or near, the point of wounding, most require evacuation to a higher level of care. Dr. Jolly noted in his book, Field Surgery in Total War, on management of combat casualties in the Spanish Civil War, that "As a general rule the only chest cases allotted to No. 1 Hospitals (the most far forward field hospital) should be those in a grave state of shock, especially those in which there is an open blowing pneumothorax. The majority should be taken to a No. 2 Hospital (where better care could be provided).[7(p.150)]

Pulmonary Embolus

Although pulmonary embolus (PE) does not appear to be a common cause of respiratory difficulty in combatants its frequency is unknown and it is likely considerably higher than reported because of the difficulty in making this diagnosis, particularly in the forward areas. Recent data on the frequency of pulmonary embolus in combat casualties returning to CONUS from Iraq and Afghanistan confirm that pulmonary embolus is a problem in combat casualties.[68, 164-166] Despite this apparent increase in PE incidence over previous conflicts which has been ascribed to there being a higher percentage of severely mangled limbs in current operations[164] the most likely reason is that current diagnostic abilities allow this diagnosis to be definitively made in living casualties although the long air evacuation of freshly wounded casualties may also contribute.

Pulmonary embolus diagnosis and treatment is not a subject that is generally addressed in combat casualty care but, as made clear by the recent high profile death of embedded NBC journalist David Blood during Operation Iraqi Freedom,[166] this condition can and does occur and it can present as a problem in the pre-hospital environment. The conditions that predisposed David Bloom to this condition were not unique to him; prolonged immobility in cramped quarters[167, 168] and relatively older age; dehydration with increased blood viscosity may have also had a role. As noted elsewhere in this chapter, demographics indicate that the average age of combatants from most first-world countries is increasing and, as also noted, long transport over great distances by motorized transport is becoming increasingly common. It is also important to note that there are an increasing number of women combatants, some of whom will be using oral contraceptives, and this population is at increased risk of pulmonary embolus.

While little can be done, in the pre-hospital environment, for the patient with a pulmonary embolus it is important that pre-hospital providers be aware of this condition. They must also be able to recognize which patients are more likely to have a PE based on risk factors and clinical presentation and they should know how to provide appropriate initial treatment (apply oxygen if available and perhaps give aspirin) and make appropriate triage and evacuation decisions for patients with this condition.

In civilian settings pulmonary embolus is a common cause of death, being the third leading cause of cardiovascular death in

North America.[169] Pulmonary embolus is an important diagnosis to make early because undiagnosed pulmonary embolism has a hospital mortality rate as high as 30%; but if properly diagnosed and treated mortality falls to near 8% and in ambulatory patients to less than 2%.[170-175] Unfortunately the diagnosis of pulmonary embolism is very difficult even for experienced clinicians with access to sophisticated diagnostic tools. Less than 35% of patients suspected by physicians of having pulmonary embolism actually have this condition.[176-178]

Although most cases of deep vein thrombosis [DVT] originate in the calf it is rare for thrombosis in the calf to lead to clinically significant pulmonary emboli; however, one-quarter of calf DVTs do extend proximally and thrombosis in large proximal veins is where most clinically significant pulmonary emboli originate. Therefore, early recognition and appropriate treatment of calf DVTs will prevent many cases of clinically significant pulmonary emboli.

Combatants in whom DVT should be considered are those who present for medical care with complaints of pain, swelling, and discoloration in a lower extremity. In this group the following history should be elicited to assess the likelihood of DVT:

1) History of recent prolonged immobilization – immobilization of lower extremity in plaster or bedridden greater than 3 days
2) History of recent lower extremity trauma
3) History of smoking
4) Strong family history of DVTs
5) Recent hospitalization within past 6 months
6) Use of oral contraceptives by women combatants

Other PE risk factors not likely to be relevant in a combatant include active cancer and major surgery within past 4 weeks. Race may also play a role in predisposing to DVT and PE. Specifically it has been found that venous thromboembolism and pulmonary embolism are much less common among Asians and Pacific Islanders in the United States than in whites and African Americans.[179]

Physical examination findings that should suggest a diagnosis of thromboembolism include the following:

1) Localized tenderness along deep venous distribution
2) Evidence of thigh and calf swelling – specifically is there greater than 3 cm of swelling on the symptomatic side than on the asymptomatic side when measured 10 cm below the tibial tuberosity?
3) Dilated superficial veins (not variscose) in the symptomatic leg
4) Pitting edema only in the symptomatic leg
5) Erythema of the symptomatic leg[180, 181]
6) Homan's sign (pain in the calf on passive dorsiflexion of the foot)
7) Edema, generalized tenderness, and warmth, may also be present but these have a low predictive value because they commonly occur in other conditions that are much more common in combatants such as musculoskeletal injury and cellulitis.

Although most combatants with many, if not most, of these clinical findings will not have deep vein thrombosis any combatant with two or more of these findings in whom an alternate diagnosis is not considered likely should be assumed to have a DVT until proven otherwise. It is important to recognize and properly treat com-

batants with symptomatic DVT because without treatment about one-half them will have recurrent venous thromboembolism within 3 months.[182, 183] A relatively high proportion of patients with recurrent episodes of venous thromboembolism end up having pulmonary emboli that are fatal (case-fatality rate over 2-fold higher).[184, 185]

Pulmonary emboli, when they do occur, cause ventilation-perfusion defects in which portions of the lung are ventilated but not perfused. When there is a moderately large pulmonary embolus the PaO_2 drops; the $PaCO_2$ also drops due to hyperventilation but the decrease is not as great as would otherwise occur because dead space is increased; pH increases secondary to hyperventilation but may not increase as much as expected because there is a concomitant lactic acidosis caused by decreased perfusion; mixed venous PO_2 falls as a result of an increase in the metabolic rate and due to decreased perfusion to some cells and because the hyperventilation-induced alkalosis reduces the amount of oxygen that can be extracted at the cellular level; increased blood flow through areas of the lung that naturally have a low VA/Q also contributes to hypoxia and finally; associated atelectasis does produce intrapulmonary shunts (V/Q mismatch) in which portions of the lung are perfused but not ventilated which worsens hypoxia. Although pulmonary emboli do produce increased dead space and cause some diffusion impairment neither of these contribute much to the hypoxia that is associated with pulmonary emboli.

Patients with pulmonary emboli present with a variety of signs and symptoms – classically they complain of chest pain and shortness of breath but the “classical” presentation is the least common. Stein et al. described three different syndromes in patients with pulmonary emboli. They define a pulmonary infarction syndrome in which patients present with pleuritic chest pain and/or hemoptysis; a syndrome of isolated dyspnea; and a syndrome of circulatory collapse.[186] They found that among patients with the pulmonary infarction syndrome, 14 of 119 (12%) had neither dyspnea nor tachypnea. Some patients with circulatory collapse did not have dyspnea, tachypnea, or pleuritic pain. Patients with the pulmonary infarction syndrome are more likely to have a PaO2 >80 mm Hg [27 of 99 (27%)], than patients with the isolated dyspnea syndrome, 2 of 19 (11%).

Pleuritic chest pain arises from pleural irritation caused by a pulmonary infarct; but pulmonary infarctions are uncommon in patients without prior cardiopulmonary disease. Patients with pulmonary emboli who have comorbid conditions tend to present with pleuritic chest pain more often than their healthier counterparts.[187] Hemoptysis occurs in only 34% of patients with pulmonary embol;.[188] and a full twelve percent of patients with pulmonary infarction syndrome, have neither dyspnea nor tachypnea.[186]

In patients with isolated dyspnea, the degree of dyspnea depends on the degree of pulmonary vascular obstruction and the patient's cardiopulmonary reserve – combat troops tend to be cardiovascularly fit so they would likely be less dyspneic. Patients with less than 50% of their pulmonary vasculature obstructed by clot are often asymptomatic.[187] The syndrome of circulatory collapse can present as transient syncope, hemodynamic instability, or frank cardiac arrest. Between 8 and 14% of patients with pulmonary emboli present with syncope.[189] Persisting right ventricular dysfunction after a large pulmonary embolus results in hemodynamic instability and severe cases present in cardiac arrest. Kurkciyan et al. have estimated that

4.5% of all cardiac arrests presenting to emergency departments are secondary to pulmonary embolus.[190]

Although one of the most common abnormalities on physical examination is tachycardia, Green and coworkers[191] found that almost 70% of patients with pulmonary emboli younger than 40 and approximately 30% of them older than 40 presented with heart rates less than 100 beats/min. Even tachypnea, which is more consistently present than tachycardia, is absent in 5% to 13% of patients with pulmonary emboli.[188, 191] A low grade temperature is present in 14% of patients with pulmonary embolus but this finding is usually not helpful in making a diagnosis of pulmonary embolus as it more commonly suggests pneumonia rather than PE.[192] Other physical examination findings common in pulmonary embolus include pleural friction rubs, rales, cyanosis, and evidence of phlebitis; but in as many as 58% of patients with proven PE the physical examination fails to reveal any clues as to the proper diagnosis.[191] To summarize, while the clinical findings of tachycardia, tachypnea, dyspnea, hemoptysis, syncope, pleuritic pain and a pleuritic friction rub may be present in patients with pulmonary emboli these findings are neither sensitive, nor specific for this condition and, as noted above, these findings are likely to be less commonly present in young combatants than in the general population.

What all this means to the medical care provider in the forward areas of the battlefield is that it is important to be aware that combatants, particularly those with risk factors as described above, are at risk for pulmonary emboli. When physical exam findings are present in such patients that suggest pulmonary embolus, such as unexplained dyspnea, tachypnea, tachycardia, and syncope; and when there are history and physical exam findings suggestive of deep vein thrombosis, it is important not to dismiss these findings or assume they are psychogenic in origin. When combat conditions permit such patients should be evacuated to a level of care capable of making this diagnosis; this generally means a facility with a CT scanner.

More important than recognizing and quickly evacuating all patients with a reasonably high probability of deep vein thrombosis and pulmonary embolus is to know how to prevent these conditions. Encouraging combatants to stretch and move their legs frequently during long trips will help prevent the formation of DVTs and keeping well hydrated has also been recommended.[193] Compression stockings have been proven to be of benefit in prevention of symptomless DVTs in air travelers[194] but this is not a practical solution for most combatants; however in those who are at high risk this might not be an unreasonable suggestion.

While not shown to be highly efficacious in preventing pulmonary emboli aspirin has been shown to have some benefit,[195] preventing four fatal pulmonary emboli per 1000 in a multi-center trial. Although not an issue in this discussion aspirin has also demonstrated efficacy in the prevention of myocardial infarction.[196] The problem with aspirin, of course, is that recommending that combatants take a drug that helps prevent clotting is not entirely satisfactory and the risk-benefit ratio for most combatants would weigh against such a recommendation. This may not be true for all, however, as some senior officers may well be at greater risk of death from pulmonary embolus or myocardial infarction than from enemy fire under certain circumstances, particularly if they have had either of these conditions in the past.

To be complete it is also important to note that emboli may not only arise from a venous thrombosis but may be caused by fat originating from the marrow of a long bone or the pelvis following a fracture. Although fractures are common in combatants there is no evidence that fat emboli are common clinical problem in combat casualties; but subclinical fat emboli almost certain do occur.[197, 198] Symptoms of fat emboli generally do not immediately follow injury but occur one to two days later so this is not a problem likely to be seen in the prehospital environment. This syndrome should, however, be suspected if typical symptoms occur in a casualty with a long bone or pelvic fracture. The classic triad of fat embolus symptoms is hypoxemia, neurologic abnormalities, and a petechial rash; but classic presentations are uncommon. The respiratory distress syndrome is the earliest, most common serious manifestation with symptoms ranging from mild, with only dyspnea and/or tachypnea, to severe and indistinguishable from the adult respiratory distress syndrome. About half of patients with fat embolism syndrome develop severe hypoxemia and require ventilatory support.[199-201]

As there is nothing that can be done for a casualty with fat embolus in the field it is only important for prehospital providers to recognize when this clinical syndrome may be present and to quickly evacuate any such patients to an appropriate level of care. The only measure of prevention relevant in the prehospital environment is to minimize movement of all fracture fragments to the fullest extent possible as manipulation of fractures has been shown to be a cause of fat emboli.

Summary

Injuries to the chest are among the most fatal to combat casualties and respiratory problems in general, because of their potential lethality, present a challenge to pre-hospital providers who must be able to sort those casualties with minor problems from those with potentially life threatening problems. In order to be able to properly diagnose respiratory problems the pre-hospital provider must understand basic respiratory physiology and pathophysiology, the difference between ventilation and oxygenation, and the common causes of dyspnea in combat casualties. They must also have a basic understanding of the epidemiology of thoracic injuries and respiratory difficulties in combatants so that they can make informed therapeutic and evacuation decisions. The diagnostic tools available to pre-hospital personnel to diagnose thoracic and respiratory problems are limited so it is very important that pre-hospital military health care providers be skilled at acquiring and properly interpreting the casualty's history, general appearance, and physical exam. Specifically they must pay particular attention to the environmental factors (altitude, presence of atmospheric contaminants), mechanism of injury, and the casualty's respiratory rate and depth and, when available, to the oxygen saturation.

Commanders, trainers, and suppliers of military prehospital personnel must utilize epidemiologic information and an understanding of the potential risks of various interventions such as needle thoracentesis for tension pneumothorax, use and application of pre-hospital oxygen, and intubation/ventilation, to make decisions about what pre-hospital procedures providers should be trained, equipped, and authorized to perform. Because of the difficulty in accurately diagnosing potentially life threatening chest and lung problems in the combat environment it is vitally important that prehospital personnel be provided simple-to-use, durable, and effective diagnostic tools for assessing ventilation problems and intra-thoracic pathology.

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Immediate Care of the Wounded

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"It will be tragic if medical historians can look back on the World War II period and write of it as a time when so much was learned and so little remembered."[1]

Beecher H. Early Care of the Seriously Wounded Man. JAMA. 1951;145(4):193-200] Henry K. Beecher - Father of the prospective, double-blind, placebo-controlled clinical trial.

"It is highly desirable that anyone engaged in war surgery should keep his ideas fluid and so be ready to abandon methods which prove unsatisfactory in favour of others which, at first, may appear revolutionary and even not free from inherent danger."[2]

Bailey H, ed. Surgery of Modern Warfare. 2nd ed. Edinburg: E & S Livingston; 1942; No. 1. – Hamilton Bailey, Reknown British Surgeon

Circulation

"…the only good thing that can be said of shock is that by enfeebling the circulation it may prevent so great a loss of blood as might otherwise occur."[3(p.9)]

J.J. Chisolm, Confederate Surgeon – Manual of Military Surgery

All bleeding stops…eventually[4]. [Attributed to Guy de Chauliac (1300-1370)]

Introduction

Exsanguination is the single most common cause of death on the battlefield; therefore the prevention of exsanguination at or near the point of wounding must be the single most important medical task. No hospital, no matter how advanced, and no surgeon, no matter how skilled, can save a wounded patient who has exsanguinated. Thus it is true, as Dr. Nicholas Senn, founder of the Association of Military Surgeons of the United States, once said, "*The fate of the wounded lies in the hands of the ones who apply the first dressing."* Most of those who die on the battlefield do so within minutes of wounding. Few of these patients with rapidly exsanguinating wounds can be saved regardless of the level of care provided; most would die even if they were wounded right outside a Level I trauma center. As noted by Col. Gray during World War I, "*The haemorrhage that takes place when a main artery is divided is usually so rapid and so copious that the wounded man dies before help can reach him."*[3(p.44)]

Furthermore, a sizeable number who could have been saved had care been immediately available die because the circumstances of combat preclude the rapid delivery of such care. An analysis of 98 Vietnam casualties who died from exsanguination revealed that nearly 20% had sustained injuries that, in ideal circumstances, could have been controlled with a tourniquet and/or appropriately applied direct pressure.[5-7] But some casualties with potentially lethal hemorrhage can be saved and it is upon this group the combat medical provider must focus.

Hemorrhagic Shock

The primary circulatory problem on the battlefield is shock; specifically hemorrhagic shock. Causes of hypovolemic shock such as massive burns and dehydration from a variety of causes, and neurogenic, cardiogenic, septic, and anaphylactic shock all occur in combat casualties but much more rarely. Hemorrhagic shock, therefore, is the primary focus of the following discussion. The other forms of shock will be briefly discussed at the end of this section.

Pathophysiology and Manifestations of Hemorrhagic Shock

A first century Roman savant, Aulus Cornelius Celsus, has been credited with describing hemorrhagic shock in vivid terms that remain accurate today.[8] He reputedly said, "When much blood is lost, the pulse becomes feeble, the skin extremely pale, the body covered with a malodorous sweat, the extremities frigid, and death occurs speedily." In the late 1800s, Samuel Gross described shock as being "a rude unhinging of the machinery of life"[9, 10] and John Warren called shock "A momentary pause in the act of death."[11] Despite great advances in hospital-level care, assessment and management of shock in the field remain little changed since at least World War II.

Types of Shock

Many different types of shock have been described. Shock is generally classified by cause or by underlying pathophysiology. Two or more types are often combined in a single patient, for example, hypovolemia may occur with septic shock and cardiogenic shock may occur when other types of shock exist. Regard-

less of the classification, the underlying defect is always inadequate tissue perfusion. Other causes of hypovolemic shock include dehydration, burns, and "third spacing" of fluid into the peritoneum or other extravascular spaces. Although the focus of the discussion herein is hypovolemic shock, other types of shock such as cardiogenic shock, neurogenic or vasogenic shock, anaphylactic and septic shock do occur in combat casualties,.

Stages of Shock

Shock is typically described as occurring in stages, although there are rarely distinct boundaries or well-defined characteristics that differentiate one stage from the next. Shock exists on a continuum that is unique in any given patient. The stages of shock, as described in most texts, are compensated, decompensated, and "irreversible" shock.

Compensated Shock

Compensated shock involves some degree of decreased tissue perfusion however the body's compensatory responses are sufficient to overcome the decrease in available intravascular volume. Cardiac output and a normal systolic blood pressure are maintained by sympathetic stimulation. In this stage the rate and depth of respirations increase, leading to decreased PCO_2 (compensatory respiratory alkalosis). This decrease is in response to decreased tissue perfusion and its resultant lactic acidosis. Sympathetic constriction of the large veins increases the amount of blood in the arterial system contributing to the maintenance of blood pressure and cardiac output (CO); essentially providing a venous capacitance autotransfusion.

Increases in heart rate (variable) and in cardiac contractility also maintain cardiac output. The quality of the pulse remains normal to somewhat weakened in strength (pulse pressure is normal to slightly decreased). Sympathetic constriction of the arteries leads to an increased peripheral vascular resistance that also helps to maintain blood pressure. This arteriolar constriction decreases capillary flow producing the cool skin so characteristic of patients in shock. Catecholamine release also produces bronchodilation, with associated improved air exchange.

The primary physical findings in compensatory shock are slightly delayed capillary refill and cool, slightly pale, skin. Other manifestations of shock may include mild to moderate thirst and depression, with no alteration in mental status. If the underlying cause remains untreated and continues unabated, the compensatory mechanisms eventually collapse. The progression of shock in the microcirculation produces a sequence of changes in capillary perfusion. Oxygen and substrate delivery to the cells supplied by these capillaries decreases, resulting in anaerobic metabolism that produces a lactic acidosis. When severe hypotension is present, oxygen delivery is markedly reduced and tissue oxygen extraction (percent of available oxygen extracted) is increased to meet demand. It should be noted that acidosis facilitates oxygen extraction at the cellular level by shifting the oxygen-hemoglobin dissociation curve to the right. For this reason shock-associated acidosis should never be treated with sodium but rather by restoring cellular perfusion.

Capillaries become "leaky" and protein-containing fluid leaks into the interstitial spaces (leaky capillary syndrome). Arte-

riovenous (AV) shunts open, particularly in the skin, kidneys and gastrointestinal tract, diminishing flow to the arterioles and capillaries in these organs. Sympathetic stimulation produces the pale, sweaty skin and rapid, thready, pulse (significantly narrowed pulse pressure) that Celsus described in the first century. It also elevates blood glucose levels and dilates coronary, cerebral, and skeletal muscle arterioles while constricting others. Blood is thus shunted to the heart, brain, and skeletal muscle with decreased capillary flow to the kidneys and abdominal viscera (autoregulation).

Decompensated Shock

Without prompt restoration of circulatory volume, compensated shock progresses to decompensated shock. Decompensated shock is manifested by a marked increase in heart rate, significantly prolonged capillary refill, and reduced blood flow to critical organs. At this stage, patients complain of marked thirst and although their mental state may remain clear, they are likely to be apathetic unless stimulated. Urine output decreases and eventually decreased blood flow to the brain alters mental status. As the shock state continues, the precapillary sphincters relax, expanding the vascular space. Postcapillary sphincters resist local effects and remain closed, causing blood to pool or stagnate in the capillary system producing capillary engorgement. Because increasing hypoxemia and acidosis lead to opening of additional venules and capillaries, the vascular space expands greatly.

With decompensated shock complete restoration of normal blood volume may be inadequate to fill the vascular "container" (this has implications for fluid resuscitation). The capillary and venule capacity may become great enough to reduce the volume of available blood for the great veins, further decreasing venous return and cardiac output. The viscera (lung, liver, kidneys and gastrointestinal mucosa) become congested due to stagnant blood flow. The respiratory system continues to attempt to compensate for the acidosis by increasing ventilation resulting in a partially compensated metabolic acidosis (decreased partial pressure of carbon dioxide [PCO_2]). Clotting mechanisms are affected leading to a hypercoagulable state that may result in disseminated intravascular coagulation. Uncorrected this stage of shock progresses to "irreversible" shock.

"Irreversible" Shock

At this point, the body is no longer able to maintain systolic pressure and both systolic and diastolic pressures begin to drop. The blood pressure may become unrecordable, the pulse will become very weak or even imperceptible, and the pulse pressure will be narrowed to such an extent that it may not be detectable with a blood pressure cuff.

"Irreversible" shock is manifested by extreme tachycardia or, in some cases, bradycardia, serious dysrhythmias, frank hypotension, and evidence of multiple organ failure. The skin is pale, cold, and clammy and capillary refill is markedly delayed. Patients in severe shock may complain bitterly of thirst, if they are able to speak. As cerebral blood flow decreases, there may be alteration in mental status manifested by apathy and such patients may appear to suffer very little. At this stage, patients may even become comatose although in previously healthy, physically fit, young adult soldiers, this tends to occur very late, if at all, and is often followed rapidly by death. In combat casualties, as a general

rule, it is dangerous to assume that shock is the cause of altered mental status before ruling out other causes such a head injury.

In "irreversible" shock, the partial pressure of oxygen (PO_2) may drop but the PCO_2 usually remains normal or low unless there is associated head or chest injury that leads to hypoventilation. Pulmonary capillaries become permeable, leading to pulmonary edema and acute respiratory failure or adult respiratory distress syndrome (ARDS). Cardiac ischemia may be present as a result of decreased hemoglobin, lower oxygen saturation, and decreased coronary perfusion secondary to diastolic hypotension. This ischemia may result in myocardial infarction and life-threatening dysrhythmias.

"Irreversible" shock is manifested by the progression of cellular ischemia and necrosis and by subsequent organ death despite restoration of oxygenation and perfusion. If capillary occlusion persists for more than 1-2 hours, the cells nourished by that capillary bed undergo irreversible changes. The unperfused cells can no longer use oxygen and metabolism stops. Hepatic failure usually occurs first, followed by renal failure, and then heart failure. Gastrointestinal bleeding and sepsis may result from mucosal necrosis. Pancreatic necrosis may lead to further clotting disorders and severe pancreatitis. Pulmonary thrombosis may produce hemorrhage and fluid loss into the alveoli, leading to death from respiratory failure.

Despite these well-described pathophysiologic changes, the irreversibility of "irreversible" shock has been questioned. The term, "irreversible shock" certainly has relevance in terms of pathophysiology but in any given patient it is simply not possible to know at what point shock has become "irreversible." Indeed, a World War II army medical team sent to Anzio to study shock in nearly 3,000 casualties made the following observation:

> *The degree of wound shock, as we saw it...precisely paralleled the blood loss. Conversely, clinical recovery from shock resulted promptly from the administration of whole blood. Although we made intensive search at the bedside of thousands of wounded men...we never found a clear case of irreversible shock, so easily spoken of in published articles on this subject...The shock we saw was caused by blood loss…it was cured by blood administration.*[12(p. 672-681)]

Individual Manifestations of Shock

Although the various stages of shock are described in terms of specific physiologic states the manifestations of shock in a particular patient are determined, in large part, by that patient's pre-existing physiologic status. Factors that influence the response to a specific shock-producing insult include (1) age and relative health (2) pre-existing physical condition, and (3) pre-existing diseases and use of medications that may impair the body's ability to mount an appropriate physiologic response. Elderly patients are less able to compensate and thus tend to develop hypotension early. Children and physically fit young adult soldiers are able to compensate longer but they tend to decompensate precipitously.

Those who provide care at or near the point of wounding need to be aware that mortally wounded soldiers may not comprehend the severity of their injuries

and may only display outward signs of shock just before death. Even vital signs may misleadingly remain within the normal range for a surprisingly long time. The military historian S.L.A. Marshall describes one such circumstance as follows, "Millsaps [started across the river with one volunteer]...The man soon began to fall behind. Millsaps asked: "Something wrong with you? He answered: "I don't think so." Then Millsaps stripped the man's jump jacket away and found six bullet holes in his upper right arm and shoulder; the soldier had not been aware of his wounds until that moment. The soldier collapsed [and died]. Millsaps continued on alone."[13(p.183)]

Identifying the Patient in Hemorrhagic Shock at or Near the Point of Wounding

Shock should be assumed in the patient who has a weak, rapid, pulse (narrow pulse pressure), and a wound consistent with a shock state. It should be noted that severe extremity wounds are often associated with shock. The Board for the Study of the Severely Wounded in World War II found that, "…[T]he greatest loss of hemoglobin occurs in wounds that involve compound fractures of long bones or traumatic amputations."[12(p.676)] Certainly it was patients with these types of wounds who survived long enough to reach the shock tents on the beach at Anzio. In fact, this study showed that the blood loss associated with compound long bone fractures was greater than that seen in traumatic amputation.

It is again worth pointing out that except for patients with head wounds, the badly injured are nearly always lucid. Importantly the degree of thirst that is present in shock is often unappreciated; substantial thirst is almost always present in severe shock.

When obtained at a single point in time, there is no single sign or symptom that can be used to reliably separate those in shock from those not; or to grade the severity of shock in an individual patient. There is simply no pathognomic sign or symptom of shock. Most important in making the diagnosis of shock is observation and repeated assessment over time. Particular attention should be given to observing the trend of the pulse and the pulse pressure (systolic minus diastolic blood pressure). A rising pulse rate, a falling blood pressure, and a narrowing pulse pressure should always alert the medical provider to the likelihood of shock, especially if associated with a cool skin, in a patient who has been in a warm environment.

Specific Tests for Shock

Blood Pressure

The general inability to use, with any beneficial effect, a single measurement of blood pressure to diagnose shock has long been recognized. Sir Zachary Cope, a pre-eminent surgeon of his day with considerable military experience in World War I, made the following observations about the utility of the blood pressure in diagnosing shock:

> *Though in general, blood loss increased with tissue damage, large blood loss might occur with small wounds... [There is] difficulty in recognizing those who had lost moderate amounts of blood, for patients who only had 70 or 80 percent of normal blood-volume might still maintain a normal blood pressure, though usually (but not always) they at the same time had a fast pulse*

> *and a pale face. A few of the patients [in shock] even presented a raised systolic blood pressure, while of those with a blood pressure of under 100 mm. Hg., some had rapid pulses, others only slightly more rapid than normal. A few, and these generally patients over fifty years, showed a pulse rate under seventy. In younger people the blood pressure was better maintained but the pulse rate tended to be faster...As a rule the blood pressure was proportional to the degree of injury and the reduction of blood volume, but occasionally extreme vasoconstriction maintained the blood pressure at a deceptive level, for example, 95 mm Hg, although in such cases the pallor was intense."*[14]

More recently Capan and Miller, in their article on resuscitation, make the comment that, "equating a normal systemic blood pressure (or pulse) with normovolemia during initial resuscitation may lead to loss of valuable time for treating the underlying hypovolemia."[15] During World War II, Henry K. Beecher said,

> *Possibly, too much attention has been given to the fact that on many occasions [patients in shock may have a normal blood pressure]...[T]his has led to a tendency to dismiss the blood pressure as a helpful sign even when it is low - a fatal error, on some occasions. More helpful than the level of the blood pressure, is the direction of its swing - a falling blood pressure, a rising pulse rate, are in most cases an urgent indication of the need for blood."*[16]

Dr. Colin Mackenzie, in his article "Anesthesia in the Shocked Patient" observes that, "It has been repeatedly shown that blood pressure and heart rate are unreliable indicators of shock in young trauma patients."[17] A recent study by Shoemaker et al. comparing invasive with non-invasive monitoring of intensive care unit (ICU) patients found that the average mean arterial pressure (MAP) initially recorded in survivors was only slightly higher than that of nonsurvivors.[18]

One final concern with relying on blood pressure to assess for shock is the accuracy of the blood pressure measurement itself. Potential problems include (1) the reliability of the provider who obtains the blood pressure, (2) the reliability of the equipment used to obtain the blood pressure, [19, 20] and (3) the ability of the equipment to perform outside of the normal physiologic range. Moskowitz and Reich note that, "The skill of the practitioner in detecting Korotkoff sounds and the speed of cuff deflation are potential sources of error in blood pressure measurement."[21] Hypotension produces hypoperfusion of the extremity with a resultant underestimation of blood pressure when assessed using the standard method.[22] Creamer et al.[23] demonstrated that the cuff pressures of patients in cardiogenic shock were poorly correlated with those obtained by direct monitoring of MAP.

Pulse Rate and Pulse Pressure

Regarding the efficacy of pulse rate and pulse pressure in the assessment of shock during World War II, The Board for the Study of the Severely Wounded noted that pulse rate is influenced by too many factors to be of great value by itself in the estimation of shock severity.[24] The Board did indicate, however, that the quality of the pulse and its upward or downward trend are most important. In a study on shock conducted by the

Board[24], the average, minimum and maximum pulse rates were the same in all degrees of shock. This surprising finding was explained as follows:

1. The tachycardia in the lesser degrees of shock may have been due in part to excitement, and

2. In some cases, the elevated pulse rate (and accompanying vasoconstriction) may have been adequate to ward off the signs of shock.

3. Further, even patients judged to be in severe shock could have a pulse rate as low as 60 beats per minute.

Of greater significance than the pulse rate was its volume, which often is decreased so far in severe shock that the pulse can no longer be felt [narrow pulse pressure]. Beecher et al. noted that as the severity of shock increases there is a significant and progressive decline in the pulse pressures as the severity of shock increases.[24]

The importance of measuring and tracking the pulse pressure of patients in shock was also emphasized by Sir Zachary Cope, who said, *"...a rapid pulse of low volume was a more constant indication of danger than a fall of blood pressure; a fall of blood pressure was sometimes a comparatively late event in circulatory failure after wounding."*[14] Occasionally patients in shock present with a normal or paradoxically decreased pulse rate. This condition has often been associated with ruptured ectopic pregnancy.[25, 26] and penetrating abdominal trauma.[27-29] but it has also been seen in hemorrhagic shock due to other causes[30-32].

Despite a long-standing recommendation to use the ability to obtain a pulse at the radial, femoral, and carotid arteries to estimate blood pressure (palpable radial pulse ≥ 80 mm Hg systolic blood pressure, palpable femoral pulse ≥ 70 mm Hg, and palpable carotid pulse ≥ 60 mm Hg systolic), there appears to be no scientific basis for this common belief. A 2000 study by Deakin and Low[33] published in the British Medical Journal compared the presence or absence of radial, femoral, and carotid pulses with intra-arterial blood pressure measured in the operating room. They found that, "The mean blood pressure and reference range obtained for each group indicate that the guidelines overestimate the systolic blood pressure associated with the number of pulses present." This author was unable to find any other studies that address the efficacy of this commonly used procedure to estimate blood pressure but empirically there appears to be a general correlation.

General Appearance and Mental State

Previously healthy, physically fit soldiers are so capable of compensating and autoregulating blood flow to the brain that even when blood loss has been massive, the usual mental lethargy that has often been described in shock may be absent and instead the patient may demonstrate unexpectedly normal mental acuity. This might easily deceive an inexperienced observer into thinking that shock is not present. Cope found *that "In shock the mental state was usu-*

ally rational…"[14] The Board for the Study of the Severely Wounded during World War II stated that assessing mental status was not useful by itself but was helpful when combined with other findings, and noted that there was some correlation between mental status and degree of shock.[24]

Orthostatic Vital Signs

Orthostatic vital signs are used specifically to assess for hypovolemia. Orthostatic vital signs consist of blood pressure and pulse measurements obtained first when the patient is lying down and then from a standing or sitting position. Although typically used to assess intravascular volume status the validity of this test is in dispute. Commonly used parameters are the following differences between lying and standing measures:

>15-20 beat-per-minute increase in pulse rate

>10-20 mm Hg decrease in systolic blood pressure

>10-15 mm Hg decrease in diastolic blood pressure

- The greater the difference, the higher the specificity and the lower the sensitivity for finding clinically significant hypovolemia

Although orthostatic vital signs are widely used to detect intravascular hypovolemia, a study by Baraff et al.[34] found that, "*No combination of orthostatic vital sign changes with a specificity of 95% or higher was sensitive enough to reliably detect an acute 450 ml blood loss.*" Koziol-McLain et al. found that many normal (euvolemic) patients had orthostatic vital signs that were consistent with hypovolemia, and they concluded that although the "tilt" test might differentiate patients with massive volume deficits from those without, attempting to do this test in critically injured patients was both unnecessary and unsafe.[35]

In a study assessing the usefulness of the tilt test in identifying significant intravascular volume depletion, Levitt et al. concluded that defining a "positive" tilt test appears to be impossible, and that although it is considered to be "the standard of care in evaluation of the hypovolemic patient," orthostatic vital signs have not been scientifically validated.[36] In a 2000 review, Dabrowski et al. questioned the accuracy of this test in diagnosing early shock when they noted that approximately 50% of normovolemic patients in the Koziol-McLain et al. study[35] exhibited positive orthostatic changes.[37]

Witting et al. found that in order to reliably separate patients with no blood loss from those with a 450-mL blood loss, the criteria for pulse had be set at a 20-point difference from lying to standing.[37] Even this sizeable difference produced a large number of false positives (normal patients identified as being abnormal). The authors also concluded that measurement of blood pressure as part of this test was without value and might actually provide misleading information.

None of this should be interpreted to mean that orthostatic vital signs are *unable* to identify many, if not most, patients in moderate to severe shock. It is, however, probably inappropriate to subject most patients in severe shock to this test because standing causes decreased perfusion of the brain and, at this stage, other findings will surely be present. If orthostatic vital signs are measured and

are grossly positive, particularly when other findings also suggest shock, the results should, most certainly, not be ignored.

Capillary Refill Test

Despite being one of the characteristic assessments in making a diagnosis of shock, delayed capillary refill has been shown to be an unreliable indicator of decreased intravascular volume. Unfortunately the efficacy of this test has not been well studied, particularly in settings where it is most relied upon, the prehospital and emergency department environments. In these settings, there is wide variability in a number of important factors including lighting, temperature, and skill level and experience of the provider making the assessment.

Furthermore, significant gender and age differences in response to this test have been shown, even in otherwise normal patients. Schriger and Baraff, in one of the few studies of this commonly used test, found that, "The upper limit of normal for adult women should be changed to 2.9 seconds and the upper limit of normal for the elderly should be changed to 4.5 seconds."[39] The authors also questioned whether the temperature dependence of the test may compromise its reliability in the prehospital setting. This study lead Knopp to make the following editorial comment, *"Given current 'normal values' and the variables of environmental temperature, age, sex and questions regarding the interpretation [which is affected by such things as lighting], one is left with the impression that at present capillary refill testing may be unreliable."*[40]

The preceding is not intended to imply that assessing for capillary refill has no place in the diagnosis of shock. It is rather to emphasize that many factors can render this test incapable of identifying early shock and to point out that capillary refill testing will over-diagnose shock, particularly in women and the elderly, and in cold, poorly lit, environments. As with orthostatic hypotension, if a patient with other signs and symptoms of shock and a mechanism of wounding consistent with shock has a positive test, e.g. delayed capillary refill, the patient is, almost certainly, in shock.

Hemoglobin Concentration Test

Although not a test that would generally be done at, or immediately near, the point of wounding, a test for hemoglobin concentration can certainly be conducted at a Battalion Aid Station. The effectiveness of this test in diagnosing shock has not been established. During World War II, Beecher found *"no early correlation between hemoglobin or hematocrit levels and circulating blood volume."*[1(p.194)]

There is some evidence, however, to suggest that an early low hemoglobin and hematocrit concentration might be of value as an indicator of acute, severe, blood loss. In a study of the effect of fluid resuscitation on hematocrit, Glick et al. found, despite the current teaching that hematocrit does not decrease immediately after acute hemorrhage, a 17% decrease in hematocrit at 15 minutes after the bleeding of splenectomized dogs that stabilized with no further drop during the remainder of the study.[41]

Knottenbelt, in a study of 1000 trauma patients, also demonstrated that, although it is traditionally taught that the body takes hours to respond to blood loss by moving fluid into the capillary circulation from interstitial spaces, patients who present with low initial he-

moglobin may have suffered significant hemorrhage.[42] In this study, a correlation between low initial hemoglobin levels and mortality in trauma patients was found. When there is significant hemorrhage and hypotension, extracellular fluid is readily drawn into capillaries, producing the low hemoglobin that is often seen on initial presentation in victims of severe trauma. This anemia is not caused by the dilutional effect of the initial fluid resuscitation bolus. Unfortunately there are also patients in hemorrhagic shock who present with hemoglobin and hematocrit concentrations within the normal range, so this test cannot be solely used to diagnose shock.

Once again, however, a patient who presents immediately after injury with a low hemoglobin/hematocrit and other signs or symptoms of shock should be presumed to be in shock. It is ill-advised to ignore positive tests, even ones that are not particularly sensitive or specific, especially when they are being applied to a population with a relatively high probability of having the condition for which the test is being conducted.

Future Tests for Shock

Based upon the finding that in experimental porcine hemorrhagic shock, changes in intestinal perfusion are the most rapid, sensitive indicators of acute blood volume loss,[43] wounded soldiers in the not-too-distant future might be given a "pill" to swallow that will monitor gut pH and will transmit this information to an external sensor that could track changes in intestinal perfusion as an early marker for shock. Someday, measurement of gut pH by prehospital personnel will allow for early, accurate, diagnosis and treatment of shock.

Fluid Resuscitation

Non-Blood Fluids

In order to have the best understanding of where we are today in terms of non-blood fluid resuscitation of patients in hemorrhagic shock, it is important to know the history of this practice. In 1832, while England was in the midst of a cholera epidemic, a 22-year-old doctor named William B. O'Shaughnessy recognized the underlying pathophysiology of cholera that was leading to death and he proposed a solution.[44, 45] He noted that, *"…the [two] indications of cure…are…to restore the blood to its natural specific gravity [and] to restore its deficient saline matters…The first of these can only be effected by absorption, by imbibition [drinking], or by the injection of aqueous fluid into the veins." "When absorption is entirely suspended…in those desperate cases,"* O'Shaughnessy recommended, *"the injection into the veins of water holding a solution of the normal salts of the blood."* Dr. Thomas Latta was the first to apply O'Shaughnessy's advice.[45] Reading the reports of doctors who had implemented his suggestions, Dr. O'Shaughnessy stated in the *Lancet* on 2 June *1832 "…the results of the practice [intravenous infusion of a saline solution]…exceed my most sanguine anticipations."*[47]

Since 1832, the benefits of non-oxygen carrying electrolyte solutions have remained undisputed when the primary need is *"to restore the blood to its natural specific gravity [and] to restore its deficient saline matters"* and [underlining added for emphasis] when the oral route, for whatever reason, is not a viable alternative.[44] The use of saline and related fluids for the treatment of hem-

orrhagic shock did not come into practice until following the 1883 recommendation of Charles E. Jennings' (a British clinician and physiologist). He stated that in "…most of the instances where [blood] transfusion is called for, I unhesitatingly advise the intravenous injection of [saline] fluid as being certain in its action, comparatively free from danger, and not requiring any special skill in its performance."[48, 49(p.193)]

In the early 1880s another British physiologist, Sidney Ringer, developed his "Ringer's Solution" that evolved into Ringers lactate, a resuscitation fluid that remains the American College of Surgeons' Advanced Trauma Life Support (ATLS) recommended trauma resuscitation fluid.[50(p.97)]

The scientific basis for infusing non-oxygen carrying fluids into patients in hemorrhagic shock has been established in numerous animal studies. When animals are exsanguinated in a controlled manner (Wigger's or modified Wigger's method) in a laboratory setting, those resuscitated with either or both crystalloid and colloidal intravenous fluids have a better probability of survival than those not similarly treated.[51(p.138-139)]

Only relatively recently have human studies been done that could establish with any certainty that trauma patients treated with intravenous fluids have a better chance of survival than those not similarly treated.[52, 53] Despite the widespread and long history of use of saline and related fluids to resuscitate patients with blood loss there has, for some time, been evidence that in some trauma victims the use of these fluids is worse than useless. Throughout the first half of the 20th Century, despite a lack of corroborative studies, military physicians with substantial experience in caring for combat casualties observed that administration of intravenous fluids to patients with uncontrolled hemorrhage was potentially detrimental.[54, 55(pp 25,189), 56] During World War I, Gordon Watson, Consulting Surgeon to the British Second Army, observed that the effects of infused saline were "too often transitory – a flash in the pan – followed by greater collapse than before."[49(p.100)] During the Spanish Civil War, Dr. Douglas Jolly noted that,

> "There is always a commendable desire in the Classification Post [read Battalion Aid Station] to do something active in cases of severe abdominal haemorrhage. Sometimes this "first-aid" takes a dangerous form – i.e. the administration of large quantities of intravenous saline or glucose. The patient has already lost so much of his circulating haemoglobin that he cannot afford to have the remainder washed into the peritoneal cavity…The presence of gum Arabic [read Dextran] in the saline makes no real difference.[55(p.189)]

Recently these early empiric observations by military medical officers have been confirmed with controlled scientific studies. In 1991, Bickell et al. demonstrated the detrimental effect of aggressive intravenous crystalloid resuscitation in pigs with uncontrolled hemorrhage.[52, 57] In 1994 this was followed up with a human trial demonstrating that patients with penetrating wounds of the torso (uncontrolled bleeding) who were resuscitated with intravenous fluids prior to arrival at a hospital fared worse than those who were not given intravenous fluids.[52]

These findings have generally been confirmed by other investigators, who also

found that animals with uncontrolled hemorrhage fare worse if aggressively resuscitated with fluids.[58-60] Some of these studies show that animals resuscitated with minimal to moderate fluids to specific physiologic endpoints do better than both those aggressively resuscitated and those not given any fluids. A study by Kim et al. showed improved survival when animals with uncontrolled bleeding were resuscitated to a pressure of 40 mm Hg with Ringers lactate and a colloidal plasma substitute as compared to resuscitation with just Ringers lactate; both groups had better survival than those given no fluid resuscitation.[60] In 1999, Soucy et al. reported improved survival in rats with tail resections when resuscitation was accomplished with a "moderate" volume (80 ml/Kg) given at a fast rate (17.8 ml/Kg/min) as compared to non-resuscitated rats (60% survival as compared with 16.7% survival).[61] Other studies have also shown the benefit of moderate volume resuscitation over no resuscitation, particularly when evacuation time to surgery is long.[62, 63]

Unfortunately because of difficulties with obtaining informed consent, the results of Bickell's human trial have not been confirmed. It should be noted that in Bickell's human trial, the prehospital transport times were very short (< 15 minutes) and the times from wounding to surgery were generally under an hour. Concerns have been expressed regarding application of the "no prehospital fluids to patients with uncontrolled truncal hemorrhage" rule when the time from wounding to surgery is hours, as is the usual case in combat.[64] The dilemma of maintaining perfusion to the brain (and other vital organs) in patients with prolonged hypovolemia, especially in those with closed head injuries, remains. Further study is required to confirm the best approach to the management of hemorrhagic shock in casualties with the prolonged transport times and long delays until definitive surgery typical of combat situations. The appropriate mean arterial pressure that would minimize ongoing blood loss yet allow for adequate organ perfusion to prevent multiorgan dysfunction has yet to be determined.[65]

The American College of Surgeons' ATLS guidelines[50] continue to recommend that initial resuscitation of patients with blood loss be done with intravenous crystalloid at a ratio of 3 mL for every 1 mL of estimated blood loss. This "three-for-one" rule is derived from the observation that most patients in hemorrhagic shock require as much as 300 mL of electrolyte solution for each 100 mL of blood loss. Applied blindly, these guidelines can result in excessive or, in some cases, inadequate fluid administration.[66] The use of bolus therapy with careful monitoring of the patient's response can moderate these extremes. Estimation of external blood loss, even by medical personnel, is notoriously inaccurate and internal blood loss cannot be estimated except during surgery.

The ATLS correlation of various physiologic parameters to specific amounts of blood loss is not well supported by good studies and really can't be used to make the 3 mL crystalloid for every 1 mL blood loss calculation.[50] In a study using splenectomized canines, Glick et al. examined the efficacy of various fluid resuscitation strategies and the effect of fluid resuscitation and hemorrhage on hematocrit concentration.[41] In the group treated with the recommended replacement of blood loss with crystalloids at a 3:1 ratio, there was a significant decrease in platelets and a significant increase in the prothrombin time com-

pared with those resuscitated at a 1:1 ratio. Although the 3:1 group did achieve a transient supranormal hemodynamic state, this had no distinct benefit over time. The conclusion of this study was that the recommended 3:1 resuscitation strategy had no advantage over a 1:1 replacement strategy even in a controlled hemorrhage model.

The following is the fluid resuscitation approach to the combat casualty in hemorrhagic shock that was recommended by Henry K. Beecher in World War II. It is based upon his own observations and those of the other members of the Board for the Study of the Severely Wounded of nearly 3,000 combat casualties, 72 of which were in severe hemorrhagic shock. It is surprisingly similar to the current recommended approach:

Resuscitation should be graded. It is important to rapidly restore a good blood color and the systolic pressure to an arbitrary level of about 85 mm of mercury in subjects whose blood pressure had previously been normal. Once these things have been accomplished, the rate of infusion of blood or blood substitutes depends on several factors:

(1) Delayed blood transfusion: If blood for transfusion or operation will not be available for a matter of hours, infusion of plasma or other blood substitute should be rapid enough only to maintain the aforementioned state (systolic B.P. of 85). More rapid administration of plasma will elevate the blood pressure to the level where bleeding will be renewed or increased, with further and perhaps disastrous loss of irreplaceable (for the time being) hemoglobin.

(2) Delayed operation: If a patient must await surgical intervention for a considerable period, even though blood may be available, there is no need to transfuse more rapidly than necessary to achieve these conditions, with one addition: when blood is available it is desirable to administer it until the skin loses the chill of shock and becomes warm. As long as the systolic blood pressure is not below a level of about 85 mm of mercury, the mucous membranes of good color, the skin warm and the pulse of good quality, there is no need to administer further blood until surgical treatment is available. To administer blood beyond the quantity necessary to achieve the condition mentioned, before surgery is available, inevitably means that more blood must be used than would otherwise be the case. Blood or plasma will leak into traumatized regions and be needlessly wasted; the hazard of an unnecessary number of transfusions will be incurred … Moreover, reasonable economy of blood means that it will be more abundantly available when need for it is urgent (systolic blood pressure below 80 mm mercury, pulse of poor quality and patient cold and pallid).

(3) Surgery available: When surgery is available, further transfusion of blood is advisable so that the rising blood pressure is achieved at the time the anesthesia is started.[1]

They felt that,

> *Complete restoration to normal blood volume or blood pressure is not a necessary preliminary to successful surgery in previously healthy young men…Operation [should be] undertaken as soon as experience has shown that the patient will tolerate it, indicated chiefly by a rising blood pressure (85 mm mercury or above), a falling pulse rate, a warm skin and good color of the mucus membranes. Delay in surgical intervention beyond the accomplishment of these things requires the constant support of the patient by blood or blood substitutes and, in the end, the use of needlessly large total quantities of these agents. Delay means that infection will have progressed. This is closely allied to a rising death rate. Some patients, more often those with penetrating wounds of the abdomen, will fail to show any response to whole blood. Generally these patients are found to have continuing concealed bleeding or wide contamination of the peritoneal cavity. The failure of response to the usual resuscitative measures calls for a critical decision: the necessity of undertaking operation in the patient in poor condition. This bold decision may be life saving."*[1]

Current Point-of-Wounding Fluid Resuscitation Guidelines

Based upon recent information (above) regarding fluid resuscitation, changes have recently been made in the curriculum for combat medics. Those changes are specifically based on input provided to the AMEDD Center & School 91W Health Care Specialist Program at Fort Sam Houston, Texas, by COL John Holcomb, the current director of the United States Army Institute for Surgical Research. This input was provided in the form of a white paper entitled, *Hypotensive resuscitation - algorithm for AMEDD combat fluid resuscitation,* which was followed by a recent publication on the same topic in the Journal of Trauma.[67]

The bases for the following recommendations are:

(1) The consensus of the 2001 Combat Fluid Resuscitation Conference, which was held at the Uniformed Services University of the Health Sciences (USUHS), Bethesda, Maryland, in June 2001[68] and

(2) Recent studies that demonstrate the value of moderate volume resuscitation, particularly when transport times may be long.[58, 63, 69-73]

There was unanimous agreement among the attendees at the 2001 Combat Fluid Resuscitation Conference that hypotensive resuscitation strategies were tactically relevant, physiologically sound and should be utilized. There was also general, although not universal, agreement on the type of fluid to utilize while performing hypotensive resuscitation, and there continues to be general agreement that a low volume fluid administration is optimal (how low remains disputed). The fluid resuscitation algorithm used in the 91W Health Care Specialist Program at Fort Sam Houston that follows is based on the following assumptions:

- The tactical situation may or may not allow for medical care

- Hemorrhage control is of paramount importance

- Stethoscopes and blood pressure cuffs are rarely available or useful to the frontline medic

- A palpable radial pulse and normal mentation are adequate and tactically relevant resuscitation endpoints to either start or stop fluid resuscitation. Both can be easily and adequately assessed in noisy and chaotic situations.

- Intravenous (IV) access is important for delivery of fluids and medication and should be obtained early on any casualty with a significant injury. Only those casualties meeting criteria for resuscitation are given fluids through a single 18-gauge saline lock. When IV access is not possible, intraosseous infusion is a reasonable alternative

- The capacity for prehospital fluid resuscitation depends on the amount, both weight and volume, of fluid that can be carried by each medic and/or soldier. Mission constraints will dictate how much fluid is available on the battlefield.

- When intravascular volume expansion is indicated, Hextend® can achieve this endpoint faster, with less fluid, and for a longer time than Ringers lactate.

Based on these assumptions, the Army Medical Department's (AMEDD's) new algorithm for fluid resuscitation[76] is as follows:

1) Superficial wounds (>50% of injured) - No immediate IV access or fluid resuscitation is required

2) Any significant extremity or truncal wound (neck, chest abdomen or pelvis), with or without obvious blood loss or hypotension, and irrespective of blood pressure:

   a. If the soldier is coherent and has a palpable radial pulse, blood loss has likely stopped. Start a saline lock; do not give any fluids but re-evaluate as frequently as the tactical situation permits.

   b. Significant blood loss from any wound *and* the soldier has no palpable radial pulse, *or* is not coherent (note: mental status changes due to blood loss only, *not* head injury):

      i. STOP THE BLEEDING using direct pressure: hands and gauze rolls, hemostatic dressings, hemostatic powder, [*QuikClot – see following discussion on hemostatic agents*], with or without adjuncts like Ace bandages—is primary when possible. Extremity injuries may require the temporary use of an effective arterial tourniquet. However, >90% of hypotensive patients suffer from truncal injuries, which are unavailable to these resuscitative measures.

      ii. After hemorrhage is controlled to the extent possible, obtain IV access and start 500 mL Hextend®.

      iii. If the casualty's mental status improves and a pal-

pable radial pulse returns, stop IV infusion, maintain saline lock, and observe for changes in vital signs.

iv. If no response is seen, give an additional 500 mL Hextend®. If a positive response is obtained, stop IV infusion, maintain saline lock, and check vital signs.

v. Titrating fluids is desirable but may not be possible given the tactical situation. Likewise, the rate of infusion is likely to be difficult to control. Based on the effective volume of Hextend® vs. Ringers lactate and coagulation concerns with increasing amounts of Hextend®, no more than 1000 mL Hextend® should be given to any one casualty (approximately 10mL/kg). This amount is the intravascular equivalent of 6 L Ringers lactate. If the casualty is still unresponsive and without a radial pulse after 1000 mL Hextend®, consideration should be given to triaging supplies and attention to more salvageable casualties.

vi. Based on response to fluids, casualties will separate themselves into responders, transient responders, and non-responders.

Responders: Casualties with a sustained response to fluids probably have had a significant blood loss but have stopped bleeding. These casualties should be evacuated at a time that is tactically judicious.

Transient and non-responders are most likely continuing to bleed. They need rapid evacuation and surgical intervention as soon as tactically feasible. If rapid evacuation is not possible, the medic may need to triage his attention, equipment and supplies, to other casualties as determined by the tactical situation. No more than 1000 ml of Hextend should be given to any one casualty.

(3) Head injuries impose special considerations. Hypotension (systolic blood pressure [SBP] < 90 mmHg) and hypoxia (SpO2 <90%) are known to exacerbate secondary brain injury. Both are exceedingly difficult to control in the initial phases of combat casualty care. Given current recommendations on the care of head injuries, we cannot at this time recommend hypotensive resuscitation, as outlined above, for the soldier with obvious head injuries. Should the combat situation allow for continuous individual patient attention, the medic can attempt to keep SBP > 90mmHg via external blood pressure monitoring and evacuate the casualty to the next higher level of care as soon as possible. The impasse for the forward-area

medic is obvious and will have to be addressed as thinking and research on these issues progress

Crystalloids versus Colloids

Although recently overshadowed by the discussion about whether IV fluids have any value at all in the prehospital setting (see above), the long-standing dispute over whether crystalloids or colloids are the better resuscitation fluid remains. The origins of this dispute go back to Ernest H. Starling and William M. Bayliss, two British physiologists, in the early 1900s.[49] Bayliss concluded that the reason saline was generally ineffective in resuscitating patients in hemorrhagic shock was because most of it leaked out of the intravascular space; this he had clearly demonstrated in his laboratory.

A modern discussion of the physiology of crystalloid and colloidal solutions can be found in Prough's article on the subject in 1996.[75] Bayliss did the first experiments with colloidal solutions and it was he who introduced into clinical practice the first colloidal resuscitation fluid, a solution of 6−7% gum acacia in normal saline.[76] James Hogan, an American contemporary of Bayliss, advocated, instead of gum acacia, the use of gelatin solution for the treatment of shock.[77] Bayliss strongly advocated the use of his new colloidal solution for the treatment of patients in hemorrhagic shock during and after World War I. Gum acacia, however, was not widely accepted by military surgeons because they were unimpressed with its efficacy for this indication.

Plasma was introduced during World War II for the treatment of shock and was widely used, although not all physicians found it equally efficacious. It is recorded in the proceedings of The Board for the Study of the Severely Wounded that,

> *The…statement of the Committee on Transfusions…that blood plasma is approximately as effective in the treatment of hemorrhagic shock as is whole blood, appears to have found origin in the conclusions drawn from laboratory experiments that were purposely designed so that the number of variables could be rigidly limited. Transference of these conclusions to a situation that introduced a number of additional variables [e.g.. combat casualties] was an error of human reasoning.*[78(p.5-7)]

Following World War II, DeBakey and Carter were quoted by Henry Beecher as having said the following about plasma:

> *Unfortunately, the early enthusiasm that accompanied this development* [use of plasma to treat hemorrhagic shock] *was so forceful that it pushed aside sound clinical judgment and led to the widespread misconception that plasma could be used as an effective and complete physiologic substitute for whole blood in the management of shock in the seriously wounded….With increasing experience in the treatment of shock, it became more and more evident that plasma could not be used as a complete substitute for whole blood.*[56]

It is perhaps because of the lack of any clearly definitive evidence of superiority of crystalloids over colloids (or vice-versa) that the argument as to which is most effective has been sustained so long and has occasionally generated

such heat (without much light). Proponents of either position can cite numerous supporting studies. What is known is that when the efficacy of these fluids in reducing death from hemorrhagic shock in trauma patients is assessed, there is no clear winner and the available evidence tilts toward crystalloids as being slightly more efficacious.

In 1989, Velanovich conducted a meta-analysis of eight randomized prospective trials comparing crystalloid with colloid resuscitation. In this analysis patients resuscitated with crystalloids had an overall 5.7% relative reduction in mortality with a 12.3% reduction among patients in trauma trials.[79] A decade later, Choi et al. conducted an extensive systematic review of the effects of isotonic crystalloids compared with colloids in fluid resuscitation but found no overall difference in mortality, pulmonary edema, or length of stay between groups.[80] Although a trend suggestive of lower mortality in favor of crystalloids emerged, it was not statistically significant. When the trauma subgroup was analyzed separately, crystalloid resuscitation was associated with lower mortality than colloid resuscitation [79]. Choi et al. cautioned that these results should be carefully interpreted and should be used primarily to direct further inquiry. The authors concluded that although inference can be drawn that crystalloids are superior to colloids in resuscitating trauma victims the data were not conclusive enough to suggest abandoning colloid administration in practice. In closing they said, "Larger well-designed randomized trials are needed to achieve sufficient power to detect potentially small differences in treatment effects if they truly exist."

Other studies have reached similar conclusions. Prough found no major clinical differences in extravascular lung water induced by either fluid and concluded that either one is suitable for treating simple hemorrhagic shock without undue concern regarding pulmonary edema. *"[I]n the absence of hypervolemia," stated Prough, "there appears to be no clinical difference in pulmonary function after administration of crystalloid or colloid solutions."*[75]

One area of controversy remains regarding the question of crystalloids versus colloids and that is in the situation of closed head injury. Which of these fluids is more likely to increase intracranial pressure? Sodium does not freely traverse the blood-brain barrier so it exerts an osmotic pressure across this barrier. For this reason an acute reduction of serum sodium will decrease plasma osmolality thereby increasing the water content of the brain. The converse is the case if there is an acute increase in serum sodium.

For some time colloidal solutions were thought to increase intracranial pressure less than crystalloid solutions because they do not cross the blood-brain barrier. Prough, however, points out that, "In fact, the choice of fluids (crystalloids or colloids) for gradual (nonresuscitative) rates of fluid infusion exerts remarkably little effect on cerebral edema after experimental head trauma."[75] What has been shown to affect brain water content in the setting of fluid resuscitation following closed head injury is the sodium concentration of the resuscitation fluid. Serum sodium concentration, rather than oncotic pressure has the greatest effect on intracranial pressure.[81-84] A general discussion regarding hypertonic saline for resuscitation of hemorrhagic shock and the effect of hypertonic fluid resuscitation on brain injured trauma victims follows in the next section.

Because there is no conclusive evidence that colloids are superior to crystalloids in terms of reducing mortality from hemorrhagic shock and, given their significantly greater cost and, in some instances associated complications, a recommendation that favors crystalloids over colloids for resuscitation seems quite reasonable. On the other hand, because colloids do tend to remain intravascular, an equivalent expansion of the intravascular space can be achieved with a smaller volume of colloid than crystalloid solution.

Pollack in his review of prehospital fluid resuscitation of the trauma patient, noted that, "A standard 1-L plastic bag of normal saline measures approximately 2744 cm^3 and weighs 1.1 kg."[85]. This led him to comment, regarding crystalloid resuscitation, *"this mode of treatment therefore can become physically burdensome on the battlefield to a medic with a limited capacity…for carrying resuscitative equipment."* Thus weight alone may recommend colloids in instances where combat medics have to carry IV fluids on their backs – perhaps.

Hypertonic Saline

Hypertonic Saline Dextran

Hypertonic saline resuscitation fluids, alone or combined with a colloid, have recently re-energized the discussion regarding which non-blood solution is most effective for resuscitating patients in hemorrhagic shock. Actually this issue is not new. Even before the entry of the United States into World War I "…*it was realized that the injection of physiologic salt solution or Ringers lactate was only temporarily effective in shock and hemorrhage and that the 'internal transfusion' accomplished by hypertonic salt solution, which withdrew fluid from the tissues and thus increased the blood volume, was equally ineffective.*"[86(p.8)] As noted in the preceding discussion, hypertonic saline solutions have been shown to reduce or at least prevent brain swelling in patients with closed head injury who receive fluid resuscitation.[87, 88]

It has been suggested that an ideal alternative to crystalloid and colloid fluids would be inexpensive, produce minimal edema, and effectively resuscitate patients in shock. Hypertonic saline, particularly if combined with a colloid, appears to have some of these characteristics. A number of studies have shown that 7.5% hypertonic saline, in relatively small volumes, is an effective resuscitation fluid for patients in hemorrhagic shock.[89-95] Some animal studies have shown that the beneficial effects of hyptertonic saline alone are not sustained, but when combined with a colloid they are longer lasting.[75, 96, 97]

The literature supports the assertion that hypertonic saline solutions are safe. Although hypertonic saline administration may result in serum sodium levels as high as 155 mEq/L patients seem to tolerate acute increases without harm and central pontine myelinolysis has not been observed in clinical trials of hypertonic resuscitation.[75]

Morbidity and Mortality Associated with Fluid Resuscitation

By far the most important study endpoint of interest for those involved in treating combat casualties is mortality. The main question about any resuscita-

tion fluid is not, "Does it improve specific physiologic parameters such as blood pressure?" but "Does it decrease mortality in patients in hemorrhagic shock?" Unfortunately there is little supporting evidence for the effect on mortality of any of the current resuscitation fluids including hypertonic saline.

Hypertonic Saline Dextran (HSD or more specifically 7.5% sodium chloride in 6% Dextran70) has been advocated as an ideal combat resuscitation fluid because several studies have shown that it increases myocardial contractility and improves redistribution of fluid from the extravascular to the vascular compartment and because considerably smaller volumes are needed to restore intravascular volume. One of the few human trials to examine the effect of hypertonic resuscitation on mortality was a 1991 prospective multi-center study that compared outcomes of trauma patients treated with normal saline with those treated with hypertonic saline dextran.[91] Both groups received equal volumes of fluid in the prehospital phase followed by standard isotonic infusions in the emergency department. Unfortunately there was no control group (i.e., no group that did not receive fluids) so a comparison of fluid vs. non-fluid resuscitation was not possible in this study. Although patients receiving hypertonic saline dextran had higher systemic blood pressures than the normal saline group on emergency department arrival there was no significant difference in overall mortality between the two groups at 24 hours. Further, there were no clinically significant complications of hypernatremia or dextran-related allergic reactions among patients receiving hypertonic saline dextran. The authors concluded that hypertonic saline dextran is safe, although further study, especially in patients with concomitant head injury, is warranted.

In assessing the efficacy of any of these resuscitation fluids it is important to differentiate between controlled hemorrhagic shock, where the bleeding has stopped or been made to stop, and uncontrolled hemorrhagic shock, where bleeding is ongoing. Gross et al. found that hypertonic saline used to treat controlled hemorrhagic shock increases blood pressure and cardiac output but when used to treat uncontrolled hemorrhagic shock causes hemodynamic deterioration and increases both bleeding and mortality.[98, 99]

In 1995 Krausz noted that, in several clinical studies in which hypertonic saline dextran or hypertonic saline alone was used to treat trauma patients, mortality was not decreased.[100] Previously he had noted that the early administration of hypertonic saline in uncontrolled hemorrhage actually increases mortality.[101] When hypertonic saline was used to treat trauma patients with a Glasgow Coma Scale (GCS) score <8 (suggesting head injury) Krausz noted a small decrease in mortality; that decrease, however, was not statistically significant. Based on his review of the studies available at that time (mid-1990s) Kraus concluded that, "The efficacy of [hypertonic saline] has not clearly been established in clinical trials…Further human trials are required to better define the patient population that would benefit most from the prehospital administration of [hypertonic saline]."[100]

Hypertonic saline has also been recommended to resuscitate burn victims (see below for a complete discussion of fluid resuscitation of burn victims at or near the point of wounding). A prospective, double-blinded controlled animal study

was conducted by Guha et al. to assess the fluid balance and hemodynamic effects of Ringers lactate, hypertonic saline, 6% hetastarch, and hypertonic saline dextran on animals with large body surface area burns.[102] This study concluded that, "Net volume loading can be reduced markedly by initial resuscitation of large body surface area burn injury using a colloid (hetastarch), and can be further reduced by use of hypertonic saline colloid." Each of the test fluids was effective at resuscitation, although more rapid resuscitation was achieved with hypertonic saline dextran; all fluids were effective within one hour. There were, however, large, significant differences, in the amount of fluid infused. In the first 8 hrs after burn, 10 mL/kg of hetastarch reduced the net fluid balance by 48%, and 10 mL/kg of hypertonic saline dextran reduced net infused volume by 74% compared with lactated Ringers solution. The authors concluded that the volume-sparing effects of hetastarch and hypertonic saline dextran must be tested and confirmed in longer-term studies before being considered clinically important.[102]

Resuscitation Fluids and Endpoints for Different Types of Trauma

No single resuscitation fluid or resuscitation protocol is ideal for every type of trauma. Patients with controlled hemorrhage differ from those with uncontrolled hemorrhage[103], those with penetrating injury are different from those with blunt injury; those with shock and closed head trauma are different from those in shock without head injury; and patients in hypovolemic shock from hemorrhage are different from those in hypovolemic shock from burn injury (see discussion below on hypovolemic shock due to burn injury).

Historically brain-injured trauma patients have been treated differently than those without brain injury. In the past, fluid restriction was recommended for brain injured patients but today this approach is recognized to cause variations in volume status and this may lead to tissue perfusion deficits. Because cerebral ischemia is one of the major causes of secondary brain injury the current focus is on aggressive maintenance of cerebral perfusion pressure (CPP). In severe head trauma maintenance of CPP at > 80 mm Hg has an associated mortality of 35−40%. Each additional 10-mm Hg decrease in CPP is associated with a 20% increase in mortality. Increasing the CPP through fluid resuscitation appears to improve morbidity and mortality.[65]

Patients with a combination of primary blast injury burn injury, and penetrating trauma present an especially unique challenge, both in terms of early diagnosis and approach to fluid resuscitation. It is difficult for even experienced professional health care providers, much less medics or corpsmen, to ascertain whether a casualty has sustained a significant primary blast injury so tailoring a specific point-of-wounding resuscitation protocol for these patients is problematic.

Casualties with significant burn injuries should receive early and relatively aggressive fluid resuscitation (see discussion below on hypovolemic shock due to burn injury) but those with primary blast injury of the lungs should be volume resuscitated with caution because they are likely to develop pulmonary edema if fluid resuscitation is too aggressive. Because of this risk Yancy

Phillips and Joan Zajtchuk, in their Textbook of Military Medicine chapter on the management of primary blast injury, recommend that, "Because the transudation of hypooncotic fluid is more likely in an injured lung, medical personnel should consider replacing the [primary blast injured] casualty's lost fluids with blood or a colloid solution rather than with a crystalloid solution."[104(p.303)] Although this seems to be a reasonable recommendation, no studies show that colloids (or blood) are any less likely than crystalloids to lead to pulmonary edema in patients with blast injury to the lung.[105]

Difficulties notwithstanding, it seems likely that each of these situations would probably best be treated with a unique fluid resuscitation protocol. Recently, Pepe and Eckstein commented that, "Evolving experience, both empiric and scientific, has demonstrated that the best treatment for 'the trauma patient' requires discrimination between the mechanism of injury, the location of anatomic involvement, and the extent, or 'staging,' of that specific process."[106]

On the battlefield, however, where there is an omnipresent threat of injury and death to both combat medic and patient, the medic does not have the time or capacity to differentiate among groups of patients, apply a different treatment for each, and/or carry several types of resuscitation fluids. Good data is needed to support the selection of a single resuscitation fluid and protocol that can be applied to all combat trauma victims. Because most combat casualties suffer primarily from hemorrhage caused by penetrating injury, without associated closed head injury, the fluid and resuscitation strategy that is most effective for this condition is likely the most appropriate choice.

Further complicating the question regarding what is the ideal combat fluid resuscitation fluid is the fact that IV fluids are not just used for the treatment of combat trauma but are more commonly used by medics to treat dehydration from a variety of causes (heat injury, diarrhea, vomiting, etc). The fact that combat casualties, unlike civilian trauma patients, may be significantly dehydrated at the time of wounding should also be taken into consideration when selecting the most appropriate resuscitation fluid.[107] Whatever resuscitation fluid is selected must, therefore, be not only safe and effective in the treatment of combat trauma, but must also be safe and effective when used to treat other, more common, conditions.

Route/Means of Intravenous Access

Although there is some controversy about the value of fluid resuscitation in the field for combat casualties, there is general agreement that it is valuable to establish vascular access at or near the point of wounding. Even if IV fluids are not required early intravascular access is still important for the following reasons:

1) Fluids and/or blood must often be given at the medical treatment facility in conjunction with hemorrhage control, and it is usually easier and faster to gain IV access in patients who are not volume depleted.

2) IV access is often necessary for purposes other than fluid administration, e.g., administration of analgesia or antibiotics.

Adequate analgesia is best achieved by titrating intravenous morphine to the level of pain, thereby minimizing both

patient discomfort and risk of opiate overdose (if morphine is given IM, especially to hypotensive and/or hypothermic casualties it is poorly absorbed from the muscle leading to repeated dosing and the risk of delayed onset of opiate overdose).

Studies by Spaite et al. have shown that civilian prehospital care providers can obtain IV access without significantly delaying evacuation, especially if access is gained during evacuation[108, 109] near the point-of-wounding access to the intravascular space is generally obtained by direct venous cannulation, usually in the upper extremities, and often in the antecubital fossa. Cannulation should always be obtained with a short 18-gauge (or larger) catheter so that large volumes of fluids and/or blood can be administered later if necessary.[110-112] As Poiseuille's equation [$Flow = \Delta P \pi r^4 / 8 \eta l$] states flow through a pipe (e.g. IV catheter) is directly proportional to the radius but inversely proportional to the length of the pipe. While the diameter of an IV catheter is by far the most important in terms of rate of flow a catheter of one inch length will have twice the flow rate of a two inch catheter with a similar diameter.

Even if clinically indicated fluid administration in the field can be problematic because care providers must then manage not only the casualty but the IV fluids and lines as well. IV management during combat/evacuation can lead to:

1) An unfortunate shift in the focus of care from the patient to the IV,

2) Difficulty ensuring that the IV line is not pulled out, and, at the very least

3) Inadequate control of the rate of fluid administration [vehicle evacuation in the forward battle areas is almost always accomplished without medical attendants using tactical, not medical, vehicles].

In summary, the average casualty who has had an IV line inserted in the field is unlikely to arrive at a medical treatment facility with a functional line, much less having received a controlled rate of fluid delivery. This is not as much of a problem as it might seem because, as discussed above, for the average combat casualty there is no evidence that volume resuscitation in the field improves survival. Under current guidance for combat medics if fluids are not to be administered, IV access can be obtained using a saline lock (see above, point-of-wounding fluid resuscitation guidelines). Without a line and a bag of IV fluids to be protected a saline lock is much more likely to remain functional during evacuation.

Another option is to obtain intraosseous access. In a recent review, Dubick and Holcomb discuss the safety, efficacy, insertion times, and flow rates associated with intraosseous vascular access.[113] Although intraosseous infusion has been used for many years in pediatric resuscitation[114], this method has recently been rediscovered as a means for prehospital fluid resuscitation in adults.[115, 116] In 1922, Drinker et al. demonstrated that red blood cells could be infused into the bloodstream through the intraosseous route.[117] In the early 1930s, this technique was demonstrated to be effective in humans [118, 119] and a few years later it was brought to the battlefield in World War II. Turkel, in 1956, commented that "Many of our fighters [in WWII]…were saved by the

intrasternal transfusion method administered to them by enlisted nonmedical but medically trained men.[120]

Today the two main routes of intraosseous access in adults are the medial malleolus and the sternum. The intramedullary space of the medial malleolus is somewhat difficult to access because of relatively thick overlying cortical bone but a device known as the bone injection gun [B.I.G.®] eliminates this problem by firing a spring-loaded hollow dart through the cortex to the correct depth.[121, 122]

The red bone marrow-containing intramedullary space of the sternum can be accessed through a flat, relatively thin bony cortex located just under the skin. This route is ideal but there is the potential risk of injury to major underlying vasculature in the mediastinum. A sternal intraosseous infusion device [F.A.S.T.®] allows for safe and rapid placement of an intraosseous needle into the sternal intramedullary space.[123-125]

With these devices an intraosseous infusion can be safely and reliably started in less than a minute. Because of relatively high resistance to flow through the intramedullary space into the intravascular space, it is not possible to administer large volumes of fluid rapidly via this route. It has, however, been previously pointed out that rapid, large-volume fluid resuscitation is harmful when bleeding has not been controlled, so this is often not an issue.

Hypertonic saline dextran has been recommended as the preferential intraosseous infusion fluid when volume resuscitation is indicated because of its ability to rapidly expand the intravascular space with minimal infused volume. Currently, there is only one study comparing intravenous and intraosseous routes of administration of hypertonic saline dextran. Chavez-Negrete et al. compared intravenous-infused and intraosseous-infused hypertonic saline dextran in patients with gastrointestinal hemorrhage.[126] Sternal intraosseous and intravenous hypertonic saline dextran were equally effective and there was no difference between the two groups in terms of blood pressure response. No complications were noted in either group.

The intraosseous route of fluid administration is generally considered safe. In 1947 Heinild et al. reviewed nearly 1,000 pediatric intraosseous infusions and found few complications.[127] More recently Orlowski et al. assessed the risks of intraosseous fluid resuscitation-induced fat and bone marrow emboli and found that although emboli did occur they were not clinically significant.[128] Other studies and reviews have confirmed the relative safety of the intraosseous route.[129-131]

A recent study by Alam et al., however, has raised some concern about a potential risk associated with intraosseous infusion of hypertonic saline dextran.[132] In this study multiple intraosseous infusions of hypertonic saline dextran to dehydrated pigs caused severe necrosis of the tibia. The clinical relevance of this finding to human trauma patients is unknown.

Any parenteral fluid access site that is obtained at, or near, the point of wounding must be replaced as soon as a new site can be established in a clean environment. When access is obtained in a combat zone, the patient's risk of developing infectious complications increases.[133] During recent combat operations in Afghanistan, several cases of sepsis were reportedly associated with

field-started intravenous lines [conversations of author with combat medical personnel]. Osteomyelitis has been reported following intraosseous infusion[134] and is a risk in field-started intraosseous infusions that are left in place longer than 24 hours.

When fluid resuscitation is indicated intraosseous infusion via either the sternal or medial malleolus routes is a reasonable option either as a backup method to the intravenous route or as a primary means of access. There are certainly other methods of obtaining access to the intravascular space but in general none are appropriate at, or near, the point of wounding.

Oral Rehydration

Oral rehydration of hypovolemic wounded soldiers should not be overlooked – simple solutions often are. In alert patients without nausea, or even in those with mild nausea, a careful oral rehydration may be reasonable.

Attendees of a 2001 workshop organized by the International Committee of the Red Cross on prehospital care for war and mine wounded agreed that any wounded patient, regardless of wound type or severity, who had not lost consciousness could be given oral fluids. No specific guidelines were developed, however, and the workshop concluded with the thought that oral rehydration is a primary area for future research.[135(p.89)]

Although there is concern about the potential for aspiration either during evacuation or surgery, no reliable scientific data confirm that drinking water actually poses such a risk. In the past decade or so the long-standing recommendation that there be a long period of preoperative fasting has been changing, particularly with regard to water.[136-140] The general consensus is that water, in any quantity, is without risk 2 hours or more before surgery. There have been suggestions that even combat casualties with abdominal wounds can have sips of water as tolerated. In sum there appears to be no good evidence to support withholding water from wounded patients and providing water seems reasonable from a physiological standpoint.

Transfusion Near the Point of Wounding

Since prior to World War I blood has been recognized as the ideal resuscitation fluid for patients in hemorrhagic shock. The reason other fluids have, at times, assumed a leading role in the resuscitation of combat casualties, particularly far forward, is because of the problems that are associated with administration of blood on the battlefield. Even with modern technology administration of blood and blood products is risky and logistically difficult. The risks and complications of transfusion therapy include hemolytic reactions; immediate ABO and delayed non-ABO reactions; allergic and anaphylactoid reactions; febrile reactions; noncardiogenic pulmonary edema; hypothermia; citrate toxicity; acid-base imbalance; hyperkalemia or hypokalemia; bacterial contamination; and transfusion-transmitted diseases such as human immunodeficiency virus (HIV), hepatitis, cytomegalovirus, West Nile virus, and Creutzfeldt-Jakob disease to name a few.[66]

In a recent review of blood transfusion medicine avoidable transfusion errors were deemed an important, if uncommon, cause of iatrogenic death and injury in U.S. hospitals.[141] Given the chaotic circumstances that often prevail in combat, it is probable that the rate of such errors is even higher when transfusions occur on the battlefield.

Fatal misidentification errors in the United States are estimated to occur in 1 in 600,000 to 1 in 800,000 transfusions and non-fatal errors occur in 1 in 12,000 to 1 in 19,000 cases.[142, 143] An error incidence of 335 per 5.5 million units of red cells transfused is reported from the United Kingdom with transfusion of an incorrect blood component accounting for almost 70% of adverse events between 1999 and 2000.[144] This study also reported 97 cases of ABO blood group incompatibility that led to immediate major complications in 29 patients and death in 4 patients.

Every medical intervention, including blood and blood product administration, has risks. The basic question is, "When the risks are balanced against the benefits, does the equation favor forward administration of blood to combat casualties at or near the point of wounding?" The Israel Defense Forces (IDF) would answer in the affirmative. Although it was generally not Israeli policy to supply blood to forward area medical units, an exception was made during the Israeli incursion into Lebanon in 1982, where heavy casualties and difficult evacuations associated with urban combat (i.e., in Beirut) were anticipated.[144] During this operation, 85% of Israeli casualties were airlifted from the battle zone and 95% received some treatment before air evacuation (almost always this included having an IV started). Every Israeli helicopter carried blood for transfusion and blood transfusions were begun in 5% (29 of 543) of patients who required an IV; 124 units of blood were administered in transit.[145]

The decision to administer blood far forward in this particular military operation was probably made because, unlike previous Arab-Israeli wars in which evacuation times to fixed hospitals were relatively short, in the Lebanon operation, evacuation times were long. As discussed in a 1999 study by Barkana et al., the IDF Medical Corps has been using blood in prehospital resuscitation of trauma patients since the 1980s.[146] In the IDF experience, time from wounding to hospital admission almost always exceeds 1 hour, and often runs 2–3 hours. The IDF uses Rh-positive type O packed red blood cells that are supplied by the central civilian blood bank and are kept in a special field refrigerator. The blood is maintained by military medical units whose members do not have any "expert" training in the maintenance of blood products. Before use, 250 mL saline is added to facilitate administration. IDF forces are somewhat unique in that physicians are stationed very far forward, often at or near the point of wounding. It is the physician who determines whether or not to administer blood, based upon ATLS guidelines.

The Barkana et al. study offered no conclusions about the efficacy of this practice because no comparison was made between casualties receiving prehospital blood with those not receiving blood.[146] The authors were able to conclude, based on their experience, that (1) blood can be administered safely at or near the point of wounding by individuals not specially trained or experienced in blood administration and (2) prehospital administration of O-positive packed red

blood cells is efficacious and indicated in selected patients.[146]

There are four major reasons to transfuse blood or blood components into a hemorrhaging patient:

1. To improve systemic oxygen transport
2. To restore critical red cell mass
3. To correct bleeding caused by dilutional thrombocytopenia, platelet dysfunction, or pathologic platelet consumption
4. To correct bleeding caused by a factor deficiency or pathologic consumption of coagulation proteins.

In short, the goals of transfusion therapy are to maintain physiologic support of the circulation and oxygen transport while avoiding the deficiencies of hemostatic factors or other physiologic deficits.[65]

Today, for reasons that are not entirely clear, blood is no longer routinely administered forward of units with surgical capability. This has not always been the case. During the Spanish Civil War, "In some classification posts [read Battalion Aid Station] blood transfusions were given to badly shocked men – usually with conserved blood but sometimes by direct transfusion."[54(p.24)]

It was during the Spanish Civil War that Dr. Norman Bethune established the first known mobile blood bank unit; it successfully delivered whole blood to casualty stations and *provided "transfusions at the front while the fighting was in progress."*[147(p.124)148] It was also observed during this war that, although the *"administration of large quantities of intravenous saline or glucose [or solutions containing gum Arabic]"* was "*dangerous*," this was not the case for blood transfusion which "…may be given in the Classification Posts [and although]…blood may drive out blood…[if] the patient is not long in reaching the operating theatre, the net result is some gain.[54(p.189)]

During World War II, the British established Field Transfusion Units, the smallest units in the British Army. These units were entirely self-contained, were fully equipped for transfusion in the field, and consisted of an officer and three enlisted men, one of whom drove the truck and was responsible for the operation of the refrigerator. These units were attached wherever they were most needed during a campaign, usually operating at field surgical units, but also at field dressing stations and casualty clearing stations.[149]

Although it is not possible to comment on the specific benefit gained by the administration of blood forward of units with surgical capability, it is clear that overall blood administration was credited with saving a large number of lives in World War II and Korea. "Prompt and liberal use of whole blood" was deemed responsible for saving the lives of innumerable casualties in World War II and the Korean War and is considered "one of the great pioneering achievements of World War II.[149]

COL Edward D. Churchill, the Consultant in Surgery, Fifth U.S. Army in World War II, in his study of whole blood and plasma and their relative role in the resuscitation of combat casualties, concluded that, *"…whole blood [is] the agent of choice in the resuscitation of the great majority of battle casualties" and that, "…whole blood [is] the only therapeutic agent that [will] prepare seriously wounded casualties for the*

surgery necessary to save life and limb."[149(p.57)]

Following World War II, Henry K. Beecher noted that, "Too much preoccupation in the clinical treatment of patients with the unsolved problems of shock can have the indirect but very closely associated result of leaving the physician without access to the therapy well proved to be effective in the treatment of shock: blood as needed."[55] He also observed that, *"The shock we saw was caused by blood loss (or loss of fractions of the blood). It was cured by blood administration."*[55]

Regarding the efficacy of blood transfusion in the field, a modern retrospective chart review by Hall et al. found that trauma patients who received packed red blood cells in the field had outcomes similar to those who did not, despite the fact that the group who received blood had significantly worse injuries (in particular more serious intra-abdominal injuries) than the group who did not receive blood.150 From these results, the authors concluded that early administration of blood may be of value, and recommended further study.

Despite overwhelming support for early blood transfusion by military surgeons with considerable experience in resuscitating combat casualties, there have been recent studies on blood transfusion that should give pause. Vincent et al., in a recent, extensive review of blood transfusion in critically ill patients, found a dose-response relationship between number of red blood cells infused and mortality.[151] All other variables being equal, receipt of a blood transfusion increased mortality risk by a factor of 1.4. This study illustrates the associations between transfusion and diminished organ function as well as between low hemoglobin level, transfusion, and mortality. The authors recommended further study of transfusion in ICU patients, as well as exploration of alternatives to transfusion.

Multiple components contained in stored blood can increase levels of pro-inflammatory mediators that may lead to neutrophil activation and promote the development of multi-organ failure.[152-154] The implication of these findings for combat casualties is unclear. Certainly, the non-trauma ICU patients in the above study by Vincent et al. are quite different from combat casualties.[151] There is also a difference between the effectiveness and side effects of stored packed red blood cells, whole blood, and fresh blood.

During World War II, it was recommended that all transfusions be given with fresh blood in order to "make up for the lack of standardized equipment and the shortage of shipping space …in evacuation hospitals." This proposal was incorporated into Circular Letter No. 108, Office of the Surgeon General, U.S. Army, dated 27 May 1943, which stated that, *"…fresh whole blood [should be administered] in general hospitals in oversea[s] theaters within 4 hours after it had been collected and for the transfusion of stored blood [it should be] collected…and used within 7 hours..."*[149(p.180)] Also during World War II, COL Elliot Cutler recommended, *"transfusion of severely wounded casualties with blood secured from lightly wounded casualties."*[149(p.192)]

Clearly fresh whole blood, and even stored whole blood, is different from packed red blood cells. These differences could certainly account, at least in part, for the apparent difference between the findings of Vincent et al. in ICU pa-

tients and the observed effects of whole blood given to combat casualties in World War II.

COL John B. Holcomb, an army surgeon, commented very favorably on the beneficial effects of whole blood transfusions given to exsanguinating combat casualties in Mogadishu, Somalia in September and October 1993. He said that whole blood had a nearly *"miraculous effect"* when given to patients who were exsanguinating.[155] [Holcomb JB, Colonel, US Army. Personal communication, August 2003]

For at least the past four decades, the general US military policy has been to use packed red blood cells for resuscitating combat casualties both because packed red blood cells are believed to be as effective as whole blood and because they have logistical advantages. Despite this stated policy, fresh whole blood has, in fact, been used by US Forces in every conflict with significant casualties since World War II, including Operation Iraqi Freedom; Army doctrine is being changed to reflect this reality.[156] In short, the experience of military surgeons in the 20th Century supports the use of whole blood in resuscitation of combat casualties.

Autologous Transfusion

One way to avoid many of the risks of blood transfusion is to use the patient's own blood. There are two ways to do this:

1. Collect the blood in advance of the need (not really an option for combat casualties) and
2. Return the patient's own lost blood back to the circulatory system through autotransfusion.

In a patient with a chest wound, this can be done by collecting blood directly from a thoracostomy tube, using a suction device, into a citrate-containing bag. The blood can then be directly reinfused through a macroaggregate filter. Another method of blood collection involves the use of a cell saver unit that collects blood into a system that

- Includes an anticoagulant and
- Uses centrifugation and a washing mechanism to provide washed red blood cells for infusion.[65]

Autotransfusion was effectively used to treat combat casualties with chest wounds during the war in Bosnia in 1992. Jevtic et al. described 19 cases of autotransfusion of blood collected from chest tubes inserted into 29 casualties with penetrating chest trauma.[157] The average quantity of returned blood delivered was 1,500 mL but in two patients, both of whom survived, ≥ 4,000 mL autotransfused blood was administered.

Although autotransfusion avoids a number of the potential risks of using donor blood (e.g., viral infection) there are several potential complications that are more common in autotransfusion. These include coagulopathy caused by excessive anticoagulant and disseminated intravascular coagulation caused by administration of activated products of coagulation and fibrinolysis. Typically, these complications are seen when >1500 mL shed blood is reinfused.

Autotransfusion of blood from the abdominal cavity can be used in the operating room, but this route of blood administration is not an option in the prehospital environment so is not discussed here.[65] Based upon the above observa-

tions, consideration should be given to increasing the emphasis at the Battalion Aid Station on autotransfusion of blood recovered from the chest via a thoracostomy tube.

Blood Substitutes

One challenge of the modern battlefield is the high degree of troop dispersion and the resulting long evacuation distances and times. In recent combat operations, the time from point of wounding to surgery has approached 24 hours. This means that wounded soldiers will have to be sustained much longer before they receive surgery. Ideally, part of this sustainment effort would be accomplished with blood administration, preferably fresh whole blood; but logistical, technical, and provider skill level issues preclude this being a viable option.

One option that is aggressively being pursued is the development and fielding of an oxygen-carrying resuscitation fluid. The search for a safe and effective oxygen-carrying resuscitation fluid has become the Holy Grail of military medicine. Millions of dollars have been spent over the past 35 years in hopes of finding a safe and efficacious blood substitute. The search actually began in the early 1600s, almost soon as it was discovered that blood circulated in the body and was vital to life.[158] In 1665, Christopher Wren tried using wine as a blood substitute, without beneficial effect. The beginnings of the modern search for an effective blood substitute began in the 1930's with Amberson, who infused stroma-free hemoglobin.[159]

The greatest efforts in this area began in the early 1970s, with the work of Rabiner et al [160] and Savitsky et al.[161] Unfortunately, despite some success, there were many problems, such as high oxygen affinity (with limited off-loading of oxygen at the cellular level), short intravascular half-life, and renal toxicity. Development in the 1980s of modified polymerized hemoglobin solutions was the start of present-day research efforts in this area. These new solutions are more stable, have better oxygen delivery characteristics, and have fewer side effects; although some problems remain.

Six companies currently have hemoglobin-based blood substitutes (Baxter [HemAssist™], Hemosol [Hemolink™, hemoglobin raffimer], Northfield [PolyHeme®], Somatogen [Optro™], Sangart [Hemospan™], and BioPure [Hemopure®, HBOC-201, or hemoglobin glutamer-250)]) that have completed patient safety studies, and some have begun efficacy trials. None of these products requires type and cross-match, all have relatively short half-lives (about 24 hours) and fairly long shelf lives, and none appears to be associated with any risk for transmission of disease. There is, however, some concern about the possibility of prion transmission by products made from bovine hemoglobin. Biopure's Hemopure® is currently approved for some indications in human patients in South Africa, and Phase III testing for US Food and Drug Administration (FDA) approval is ongoing.

Two crosslinked hemoglobin solutions are currently in production: pyridoxylated hemoglobin (PLP-Hb) and diacetylated crosslinked hemoglobin (DCLHb), and other products are in development. PLP-Hb is a dimer and DCLHb is a purified human tetrameric hemoglobin molecule. In a number of these products hemoglobin is encapsulated in liposomes; these liposome-encapsulated hemoglobins are stable during storage and have a plasma half-life of 4–20

hours without associated renal toxicity or antigenicity.[158]

Hemoglobin used in these solutions comes from (1) human red blood cells (expired donated blood), (2) bovine red blood cells, or (3) recombinant hemoglobin sources. All of these hemoglobin-based oxygen-carrying solutions have a high affinity for oxygen. Indeed, the problem has not been with these solutions binding oxygen in the lungs but with releasing that bound oxygen to the tissues that need it. Another problem with many of these solutions is that most have at least some degree of vasopressor effect secondary to binding nitric oxide (endothelial-derived relaxing factor). As noted earlier, increasing blood pressure when hemorrhage is uncontrolled simply leads to a faster rate of blood loss.

Although there was much anticipation that DCLHb would become the first of several hemoglobin-derived, oxygen-carrying blood substitutes to successfully treat hemorrhagic shock, a multicenter trial evaluating the efficacy of DCLHb was terminated when it became clear that there was a significant increase in mortality associated with the use of this solution.[162] At 28 days, 46% of those infused with DCLHb died, compared to 15% mortality in those infused with saline. The incidence of multiple organ dysfunction was also considerably higher in the DCLHb group, and the authors concluded that DCLHb was not effective for fluid resuscitation.

Another hemoglobin-based, oxygen carrying solution, which consists of polymerized tetrameric human hemoglobin, is currently undergoing final testing and appears efficacious and safe.[163, 164] A study of this polymerized human hemoglobin solution in a relatively small number of trauma patients found that this solution had a considerably lower 30-day mortality (25%) compared with an historical control of patients who refused blood transfusion for religious reasons (64.5%).[164] Final (Phase III) studies are currently underway evaluating this oxygen-carrying blood substitute in the prehospital treatment of trauma patients. If the results of this trial are favorable, this solution and/or others could have a substantial impact on the treatment of combat casualties at or near the point of wounding.

Non-hemoglobin-based oxygen carrying solutions are also being developed as possible blood substitutes; most of these are perfluorocarbon-based. Oxygen and carbon dioxide are highly soluble in perfluorocarbons, which makes these solutions able to carry oxygen to the tissues and carbon dioxide back to the lungs. In comparison to hemoglobin-based oxygen-carrying solutions, perfluorocarbons are much more effective at unloading oxygen at the tissue level. Perfluorocarbon emulsion solutions transport oxygen in a fundamentally different way than do hemoglobin based solutions. Hemoglobin based solutions exhibit a sigmoidal oxygen dissociation curve. The oxygen carrying capacity of perfluorocarbon emulsions, in contrast, is characterized by a linear relationship between PO_2 and oxygen content. Thus, sufficient oxygen delivery can only occur when patients are breathing 70 to 90% oxygen. Therefore, unlike hemoglobin-based solutions, in which supranormal levels of oxygen provide little additional benefit (and provide little added oxygen delivery to red blood cells), when perfluorocarbon emulsion solutions are used, elevated PO_2 such as that achieved with hyperbaric oxygen, is quite beneficial. Unfortunately this property means that perfluorocarbons are likely to be less valuable as a point-of-wounding resuscita-

tion fluid because oxygen, which is needed to maximize the effectiveness of these solutions, is generally not available.

Advantages of perfluorocarbons include the following:

- Some of these solutions can be stored at room temperature
- They are relatively cheap to produce
- They are completely free of biological material so they cannot be contaminated by infectious agents

Because fluorocarbons may unfavorably alter the reticuloendothelial system by long-term retention and can lower immunity [165-169] they are coated with substances, such as lecithin, which makes them soluble and reduces the extent to which they affect the immune system.

One such agent is the intravascular oxygen carrier, Oxygent™ (Alliance Pharmaceutical Corp., San Diego), which is prepared from perfluorooctyl bromide (C8F17Br) with egg yolk lecithin as the surfactant. This product, which is compatible with all blood types and has a shelf life of as long as 2 years, has moved from Phase II clinical trials to Phase III trials.[170] Preliminary data indicate that use of Oxygent obviates or minimizes the need for blood, with results being statistically significant to 3-weeks post-administration.

Although this, or another, effective blood substitute may someday be available for administration at or near the point of wounding, the promise of this prospect is unlikely to be fulfilled in the near future. When an efficacious oxygen-carrying blood substitute does become available, it will represent a major military medicine milestone that will likely have a significant impact on combat casualty survival.

Pneumatic Antishock Garment

The pneumatic antishock garment (PASG), also known as military antishock trousers (MAST), is a device that encircles the abdomen and lower extremities with air bladders that can be inflated. The concept for this device originated from the "G" suits worn by pilots of high-performance aircraft; these suits inflate during high-gravitational-pull maneuvers to prevent loss of consciousness caused by pooling of blood in the lower extremities.

The original belief was that this type of device could be used to treat shock by a mechanism of "autotransfusion" of blood from the lower extremities into the central circulation. The PASG certainly does raise the blood pressure of patients in shock, and in animal models with controlled hemorrhage it prolonged survival from otherwise fatal exsanguination [171], but it does so not by autotransfusion but by raising total peripheral vascular resistance and increasing afterload.[172, 173] Given that raising blood pressure in trauma patients with uncontrolled hemorrhage has been shown to be undesirable since at least the Spanish Civil War, it should have been a foregone conclusion that the PASG might actually worsen survival in trauma vic-

tims with on-going hemorrhage; unfortunately this was not the case.

Until relatively recently, the PASG was one of the most widely used and most-studied devices in emergency medicine and prehospital care.[174-176] Pneumatic Antishock Garments (PASGs) were a mandatory item of equipment on every ambulance in some states. Today, however, the PASG has developed such a bad reputation that it is rarely used at all. In some ways, the PASG is an excellent case study in why it is important to conduct randomized controlled human trials of all medical equipment, medical procedures, and pharmaceuticals before introducing them into widespread use. In the short span of about 25 years, the PASG went from invention, to widespread use, to obscurity.

PASG – Pneumatic Antishock Garment, From Operatiopnal Medicine 2001

Interestingly, it was one of the greatest early advocates of the PASG, Dr. Ken Mattox, who ultimately conducted the studies [177-180] that showed increased mortality in patients with penetrating abdominal injuries when the PASG was used. In patients with large-vessel involvement, survival rates were 49% for the PASG group and 65% for the control group. Not only was the PASG shown to be worse than ineffective, its use was associated, in some instances, with the development of compartment syndrome in previously normal lower extremities.[181-184] Furthermore, even during the period when its use was recommended, the PASG was known to be harmful to patients with closed head injury, thoracic injury, pregnancy, and/or pulmonary edema.

It should be noted that the Mattox and Bickell study [179] of the PASG was done in an urban environment with very short prehospital transport times. Whether the results would have been different had the transport times been longer is unknown, but there is no obvious reason why they would have been. Despite its problems, the PASG continues to be recommended by some [including this author] to apply compression over large, soft-tissue injuries in the lower extremities for hemorrhage control (PASGs reduce arterial and venous bleeding beneath the garment by decreasing vessel wound size, transmural pressure, and blood flow)[174, 176, 184, 185] to stabilize lower extremity fractures, and to help stabilize unstable pelvic fractures (see discussion below on pelvic fracture stabilization). There is some scientific support for these recommendations.[185, 186]

The limitations of the PASG prompted McCallum and Rubes in 1996 to suggest removal of the PASG from ambulances because of its "low utility and utilization."[187] Although PASGs have, in fact, been removed from most military medical equipment sets, their utility in helping to control lower extremity hemorrhage and stabilize lower extremity and pelvic fractures suggest that they should be kept available for these purposes. Prehospital medical personnel simply need to be taught that PASGs should not be used solely to treat hemorrhagic shock.

Hemmorrhage Control

"Hemorrhage, which produces such terror in the bystanders . . . should never unnerve the surgeon, who requires all of his self possession . . . to cope successfully with this ebbing away of life."[188]

Direct Pressure

Hemorrhage control is generally achieved by the application of direct pressure on the bleeding site. When possible, this is combined with elevation of the bleeding site above the level of the heart, which lowers the pressure at the point of bleeding. Digital pressure applied over proximal arterial pressure points also helps to control bleeding by lowering the pressure at the bleeding site(s). Performed correctly and, when possible, simultaneously, these hemorrhage control techniques can control significant, even brisk arterial, hemorrhage.

Until recently there has been no good method for teaching these techniques to prehospital medical personnel and so when treating hemorrhage in combat casualties, these personnel often apply insufficient pressure for an insufficient period of time. Fortunately, there has recently been increased emphasis on hemorrhage control at the 91W Health Care Specialist Program at the AMEDD Center & School at Fort Sam Houston, Texas and this has, in part, been made possible by the introduction of training devices that simulate realistic hemorrhage.[189]

Unfortunately, no matter how well this task is taught, the circumstances of combat make it unlikely, in many cases, that a medic/corpsman will be able to apply the requisite force long enough to achieve hemorrhage control. Maj. Meredith Mallory noted during a conference held at the end of the Korean War that, "*In the case of lower extremity wounds, which give rise to the most severe hemorrhage controllable by tourniquet, it has been my observation that too few doctors, much less their assistants, have a concept of the constricting pressure required about the thigh to abolish the flow of blood.*"[190] Prolonged pressure is physically demanding and prevents whoever is doing it from engaging in other tasks (like defending himself and his patient).

Although obvious pulsatile hemorrhage from a severed artery usually catches the attention of medical personnel and is dealt with promptly, if not adequately, what is often underappreciated is that casualties with large soft-tissues injuries and extensive venous ooze are also at very real risk of exsanguination -- if not in so dramatic a fashion.

During the Korean War, Artz, Sako and Howard noted that at the 46th Surgical Hospital there were 20 deaths among 138 severely wounded patients. Of these, 7 (35%) died of uncontrolled hemorrhage, 4 (20%) from uncontrolled postoperative oozing, and 3 (15%) from uncontrolled major vessel injury.[191] Patients were noted to have died from persistent oozing from massive muscle wounds. Persistent bleeding from a scalp wound can also contribute to exsanguination. One death during recent combat operations in Afghanistan was attributed to failure to control persistent venous ooze.[192]

Sustained application of direct pressure may be facilitated by a field-expedient method, that of placing a sandbag inside a clean cover and putting it directly over the top of the pressure dressing on the wound. This is basically what is done in a hospital setting after an arterial catheterization has been performed. There are, however, two problems with this recommendation:

1. A sandbag is not always available, and
2. A fully filled, full-size sandbag may be too heavy and may disperse pressure over too large an area.

A new product, named the BioHemostat®, is currently in development, which may solve both of these problems and may also be so effective as to negate, in many instances, the need for a tourniquet. Developed in 2001 by a team led by Marcus E. Carr, MD, PhD at Virginia Commonwealth University, the BioHemostat® combines a traditional pressure dressing with an attached bag that is filled with a water-absorbing polymer that can absorb, within 3 minutes, up to 1400 times its weight in blood (or water).[193]

When the bag expands, it can apply up to 90 mm Hg direct pressure, and if properly applied, will exert this force almost directly on the site of bleeding. This exerts a tourniquet-like effect on the bleeding vessel while allowing collateral vessels to continue to perfuse distal parts. Expansion of the polymer is triggered either by absorbed blood or water injected into the polymer-containing bag to cause immediate expansion. The pressure in the bag is controllable by the amount of water added. The addition of a hemostatic agent to this dressing could further enhance its' hemostatic properties.

Whether this device, or any other, will solve the problem of applying sustained focused direct pressure on the wound is unknown, but anything that could accomplish this task while freeing up the medic/corpsman would be a very useful at or near the point of wounding.

Tourniquets

A tourniquet is a constricting or compressing device that is used to control venous and arterial circulation to an extremity for a period of time. The tourniquet has been recognized as effective at controlling hemorrhage since at least the time of the Greeks [194] and it was described briefly in Roman literature but, perhaps because it lead to gangrene, it did not come into general clinical use until sometime in the mid-1500s when Ambrose Paré is credited with it's discovery as a useful medical device when accompanied by ligature of the bleeding vessel.[194] In 1628, William Harvey, an English surgeon, described the human circulatory system in detail, thus paving the way for future technological developments in hemorrhage control.

In 1718, Louis Petit, a French surgeon, developed a screw device, designed to apply pressure to specific arterial points in order to limit bleeding. It is from the French verb "tourner" (to turn), that Petit named his device a "tourniquet." Since its introduction by Paré into clinical practice, the ligature, or tourniquet, has been a standard medical item for military surgeons and has remained largely unchanged from its original form. A tourniquet on display at the National Museum of Civil War Medicine in Frederick, Maryland is remarkably similar to the one that was, until quite recently, standard Army issue to medical personnel (it still remains in the inventory).

Although a properly applied tourniquet is clearly effective at controlling distal extremity hemorrhage, the liberal use of tourniquets has long been discouraged because of their associated risks. Dr. Julian Chisolm, a senior Confederate surgeon, made the following comments about tourniquets in his book, *Manual of Military Surgery:*

> *[Tourniquets]…[u]nless very tightly applied…are of no service, as they do not control the bleeding, and if firmly applied, they act as a general ligature around the extremity, and can be used but for a short time without injury to the limb…. Should a soldier have a large artery wounded and the hemorrhage be excessive . . . the orderly should be instructed how to make a judicious finger pressure. This is much better than the tourniquet.*[195]

Following World War I, COL Gray noted the following in his book, *Early Treatment of War Wounds:*

> *When an important artery or vein has been divided…[and] if…the casualty clearing station is some distance away [from a surgical facility]…[a difficult decision must be faced regarding the application of] a tourniquet that must of necessity remain in position for many hours…. Blind grouping in the dark in a haphazard attempt to seize [a bleeding vessel] in the grasp of a pressure forceps is useless, and generally results in the loss of much additional blood. Unless the operator feels confident [in his ability to clamp a bleeding vessel] it is better to rely on a properly applied tourniquet.*[3](p.45)

Generally, hemostatic clamps have no role in the prehospital control of hemorrhage because it is rare that a hemorrhaging vessel clearly presents itself for clamping. It continues to be true, as Dr. Gray said, "Blind grouping in the dark" for a bleeding vessel is worse than useless.

It was his experience with tourniquets during the Spanish Civil War that lead Dr. Douglas Jolly to comment that, "*More limbs and lives are lost at the front from the improper use of the tourniquet than are saved by its proper use.*"[54(p.24)] During World War II, the use of tourniquets evolved as experience was gained.[196(p.427-428)] In Volume 1 of *Activities of Surgical Consultants* the following comments were made regarding the use of tourniquets in World War II:

> *Early in the war, they were used far too frequently and removed far too often. After a tourniquet had been applied by a corpsman on the battlefield, it was examined by the first medical officer who saw the casualty and frequently it was removed by this medical officer. If the tourniquet was not removed, it was the policy to loosen it routinely every 30 minutes. In some cases, death occurred from the cumulative effects of the bleeding which ensued each time the tourniquet was loosened. The fatalities usually took place during evacuation by ambulance, on trips lasting several hours, when the medical corpsman loosened the tourniquet every 30 minutes as he had been instructed to do. Many deaths which could have been avoided were thus precipitated because no facilities were at hand to control the hemorrhage which frequently followed the removal of the tourniquet. Later, the policy was reversed, and once a tourniquet had been applied, it was not loosened or removed, particularly if the casualty was in shock, until facilities were at hand for the immediate control of hemorrhage and the replacement of blood loss. It was found*

> *to be safe, when a large vessel had been damaged, to leave the tourniquet in place for periods of from 4 to 6 hours during cold weather and for somewhat shorter periods in warm weather. Rubber tubing proved more satisfactory than the Army-issue webbing tourniquet.*[196]

This last comment regarding the general ineffectiveness of the Army-issue webbing tourniquet is interesting because it seems that despite being recognized as unsatisfactory during World War II, the tourniquet has remained in the inventory to the present. It is also interesting to note that during a recent Advanced Trauma Applications for Combat Casualty Care (ATACC) conference in St. Petersburg Florida, a panel discussion on tourniquets concluded that the ½ inch surgical tubing recommended as a tourniquet during World War II was the best available field expedient tourniquet; although concerns were raised about the high tissue pressures that could be generated by the use of this tubing as a tourniquet.[197]

During World War II, MAJ Luther H. Wolff, MC, and Capt. Troglier F. Adkins, MC carried out a study on the use of tourniquets, with a particular focus on patients in shock. The results of this study formed the basis of the following regime, which was in general use when World War II ended (and which remained standard practice until quite recently):

> *Patients with tourniquets in situ should have the highest priority for transportation to the nearest hospital. The emergency medical tag should indicate that a tourniquet is in place. Marking the forehead with a red T was an additional precaution.*
>
> *An effective tourniquet should be placed on an actively bleeding extremity at the earliest possible moment.*
>
> *At the end of 2 hours, if the patient was not in shock and if all circumstances were favorable, the medical officer might cautiously loosen the tourniquet if facilities were available for immediate control of hemorrhage. If bleeding was negligible or did not recur, the tourniquet might be removed, but the patient was kept under continuous observations and the tourniquet was left in place for immediate reapplication.*
>
> *On no account should a tourniquet on a patient in shock be removed within 4 to 6 hours of its application unless the blood volume had been at least partly replaced by plasma or whole blood. After this time, the removal or loosening of the tourniquet was a matter of individual judgment. In many instances, sufficient clotting and spasm had occurred by this time to prevent further bleeding.*
>
> *When a tourniquet was in place, the temperature of the affected limb was lowered as much as possible short of actual freezing.*[196(p.427-428)]

The recommendation, during World War II, of 4 to 6 hours of tourniquet time, was empirically derived. Col Gray's opinion, based on his experiences from World War I, was that *"…about 80 per cent of limbs whose blood supply has been cut off by a tourniquet for a period of three hours…eventually come to amputation."*[3(p.45)]

A study during the Korean war of 79 major extremity vascular injuries showed that 47% were admitted with a tourniquet in place for between 40 minutes and 14 ½

hours; the average time being 4 hours.[198] Since the Korean War, a large number of studies have been done to determine the optimal maximum tourniquet time before irreversible tissue damage occurs.[199-209] Most of these studies have been done to evaluate the effect of tourniquet time on patients undergoing elective orthopedic surgery and thus patients were relatively normovolemic at the time of the tourniquet application. To what extent these studies can be applied to tourniquet use in combat casualties is unclear.

In a recent animal study of pneumatic tourniquet times and the effect of periodic episodes of reperfusion on muscle injury, Pedowitz demonstrated that 2 hours was a time threshold for tourniquet compression injury. The degree of tissue injury also depended upon cuff inflation pressure.[210] Physiologic and morphologic nerve abnormalities were caused by a 2-hour tourniquet time and necrosis was observed. With a 4-hour total tourniquet time, skeletal muscle injury beneath the cuff was significantly decreased by hourly, 10-minute reperfusion intervals.

Interestingly, this study found that if the tourniquet time was longer than 2 hours, episodes of reperfusion tended to exacerbate, not reduce, muscle injury. It is generally taught that muscles are more sensitive than nerves to long tourniquet time and that tourniquet times > 2 hours and the use of high pressure (>350 mm Hg in lower extremities and 250 mm Hg in upper extremities) increases the risk of nerve damage. It is also generally taught that if > 2 hours is required, the tourniquet should be deflated for 5 minutes during every 30 minutes of inflation time. Contradicting this recommendation is a 1999 study by Mohler et al. that demonstrated that intermittent reperfusion does not reduce neurological injury.[211] This study also showed that although tourniquet-induced muscle damage may resolve, nerve damage, if it occurs, does not.

Forward healthcare providers should be instructed that, although combat casualties may experience only minimal discomfort when the tourniquet is first applied, the great majority will begin to experience increasingly severe pain at the tourniquet site after 30−60 minutes. This pain is described as a dull, burning, aching sensation that becomes increasingly worse until the tourniquet is removed.[212] Patients requiring tourniquet placement should be given adequate analgesia prior to transport.

As previously stated, as long ago as World War II it was noted that the standard-issue 1.5-inch-wide, cotton-strap tourniquet (nonpneumatic; NSN 6515-00-383-0565), was not particularly effective.[196(p.427-428)] It is difficult even for a trained healthcare provider to achieve arterial hemostasis with this tourniquet, but when applied by the wounded soldier himself it is essentially impossible to achieve hemostasis, especially if only one hand is available for application. During the post-Korean War conference, "Recent Advances in Medicine and Surgery (19-30 April 1954) at the Army Medical Service Graduate School Walter Reed Army Medical Center, Maj. Mallory made the following comments about the standard army tourniquet:

> *Present methods are time-consuming, inaccurate as to pressure exerted and often require excessive manipulation of the injured part (especially when applied by a single person). Self-application is virtually impossible…the tourniquet to be effective must be applied before the patient reaches the aid station. This requires that it be done in or very near the battle area. This further demands that the method be very simple and rapid and*

> *applicable by anyone – characteristics not possessed by the present means…We need, critically, a better tourniquet device (underlying added for emphasis).*[213(p.61)]

During this same conference it was recommended that, *"Not infrequently, a tourniquet may be used as an emergency procedure; and, after further evaluation, bleeding may be controlled by a pressure dressing."*[198] Later during this conference LTC Hughes stated that, *"…a tourniquet should be applied only tight enough to control hemorrhage and left in place until it can be removed by a medical officer with blood or plasma expander available to resuscitate the patient. When packs or pressure dressings will suffice, the tourniquet should be removed and the pressure dressing applied only tight enough to control the hemorrhage."*[198] Interestingly this recommendation is virtually identical to that which was recently agreed to for far-forward medical care providers.

With regard to the current Army-issue tourniquet, early feedback from Navy corpsmen in support of the U.S. Marine Corps during Operation Iraqi Freedom in Spring 2003 is that once again the 1.5 x 42-inch non-pneumatic tourniquet (NSN 6515-00-383-0565) which was still issued at that time has proven ineffective. Corpsmen report that the tourniquet tended to slip around the thigh or arm while attempts were made to tighten it, causing medical personnel to resort to green slings and sticks to tighten around pressure points to stem the flow of arterial bleeding in the extremities.[214]

Calkins et al. recently evaluated available tourniquets and commented that the web-strap tourniquet is inconsistent in stopping arterial blood flow, even when applied by someone other than the injured patient him/herself.[215] Recognition of this fact has led Special Operations medics to craft their own tourniquets from triangular bandages and suitable field-expedient windlasses; it also led researchers to try to find a better tourniquet. Calkins et al. found that both a ratchet-style and Velcro bladder tourniquet were effective and could be applied singled-handedly. Despite the potential of the bladder tourniquet to develop into a "smart tourniquet," i.e., capable of periodic self deflation and re-inflation, Calkins et al. recommended the ratchet-style tourniquet for field use, primarily due to its durability, simplicity, ease-of-use, size, and weight.[215]

The revival of interest in the tourniquet in the mid-to-late 1990's led to an aggressive effort to develop, evaluate, and field an effective tourniquet that could easily be applied one-handed by a wounded soldier. The preliminary result of this endeavor was the selection of a tourniquet that came to be referred to as the Army One Handed Tourniquet. Unfortunately, while this tourniquet could be applied one-handed (even this tourniquet was not particularly easy to apply one-handed) it was not particularly effective at occluding arterial flow in the lower extremities.[216]

Other tourniquets continued to be developed and deployed. The two most widely deployed being the Combat Application Tourniquet, or CAT, and the Special Operations Forces Tactical Tourniquet, or SOFTT. In his 10 May 2005 statement to the Senate Committee on Appropriations, Defense Subcommittee, LTG Kiley, Surgeon General, United States Army, noted that since April 1, 2004 193,897 tourniquets have been issued to deploying troops; 112,697 of these tourniquets being either the Combat Application Tourniquet (CAT) and the SOFTT (Special Operations Forces Tactical Tourniquet). He went on to say that beginning April 1, 2005 all new soldiers will received spe-

cific instructions on the CAT during Basic Combat Training.[217]

Most recently developed and fielded is the Mechanical Advantage Tourniquet, or M.A.T., produced by Cybertech. Although somewhat heavier and more bulky than some of the previously fielded tourniquets this author and others feel that this tourniquet is the best of the most recently fielded tourniquets. This tourniquet is very easy to use, it can be rapidly applied with little difficulty using one-hand, and, most importantly, it is effective at occluding arterial hemorrhage in both upper and lower extremities.[218] This said, the Combat Application Tourniquet is also quite effective, and although in this author's view somewhat more difficult to use, it is lighter and less bulky.

Based upon currently available scientific data, tourniquet times in excess of 2 hours should be assumed to be associated with the loss of the distal limb. This may not be true in every instance; certainly some limbs will still be salvageable after 2 hours of tourniquet time, especially if the limb has been kept cold. In fact forward medical personnel should be advised, whenever possible, to cool limbs (while avoiding freezing of the tissue) to which a tourniquet has been applied in order to increase the possibility of limb salvage. There will also be situations where otherwise viable limbs will be lost even when the tourniquet time has been kept to less than 2 hours.

Navein and Coupland, in their 2003 Journal of Trauma article, noted that a limb occluded for less than 2 hours is almost always salvageable while a limb occluded for more than 6 hours is almost certainly not salvageable. They conclude that decision making in tourniquet application should involve viewing tourniquets in one of three ways. First, those that are "tactical tourniquets", which are applied quickly using materials immediately at hand, to control potentially lethal hemorrhage when time and circumstances do not allow for a detailed assessment and/or when manpower, or resources are not available. Such tourniquets are to be removed at the earliest possible time and the patient reassessed to determine if a tourniquet is actually needed. Second, there are those tourniquets that are "trial tourniquets"; tourniquets that have definitely been determined to be needed and have been applied in a careful manner with the intention to leave in place for up to 2 hours. Finally they describe "tourniquets of last resort". These are the tourniquets that are applied and left in place after a "trial tourniquet", which had already been left in place for two hours, is removed and bleeding continues unabated by other hemorrhage control efforts.[219]

The recommendation that is offered here is that forward healthcare providers should be given the following instructions:

1. Make every possible effort to evacuate, in less than 2 hours, all patients who require placement of a tourniquet to a facility where surgical control of hemorrhage can be provided

2. Tourniquet times in excess of 2 hours have a high associated probability of distal limb loss.

3. Although tourniquet placement may be necessary, it should always be done with an understanding that no tourniquet time is considered "safe," i.e., there is always a risk of injury any time a tourniquet is applied.

Research and development should be continued to develop and field an automatic pneumatic tourniquet that inflates to the

minimum effective inflation pressure required for hemorrhage control. If clinical studies confirm the efficacy of periodic tourniquet deflation, this new tourniquet should also be able to be set to periodically deflate and re-inflate to allow for some distal blood flow. The real barriers to fielding such a tourniquet today is that such a tourniquet should also be relatively inexpensive, very durable, and easy to use.

Other Methods of Hemorrhage Control

Fibrin Dressing

Driven by the recognition that the only place in which combat-related deaths can really be significantly reduced is in the prehospital environment, the U.S. Department of Defense medical research community has increasingly focused on prehospital care issues; in particular on hemorrhage control and management of shock. Recent well-publicized, prolonged, deaths from hemorrhage of soldiers who were in the care of medics/corpsmen (e.g., during the 1993 operation in Mogadishu, Somalia and in 2002 at Robert's Ridge during Operation Anaconda in Afghanistan) have spurred these efforts. One initiative has been the development and fielding of the dry fibrin sealant dressing. Although research in this area has recently been re-energized and now focused on the far-forward use of fibrin, the use of fibrin for hemorrhage control had begun to be explored as early as World War II.

> *When thrombin first became available, Lt. Edgar A. Bering, Jr., Mc, USN and Dr. Bailey had applied it in solution to bleeding points in several cranial and spinal operations. It did no harm…but its effect was entirely transient. Lieutenant Bering then conceived the idea of using fibrinogen, converted into fibrin foam, as a matrix…The first applications of fibrin foam were made in cases in which bleeding was difficult to control and the application of muscle was not feasible. The hemostatic effect was evident…It was agreed that the material was of extraordinary value as a hemostatic agent in neurosurgery. It…proved of great value in hemophiliacs, in controlling bleeding from traumatic lacerations, and in maintaining hemostasis during minor surgical procedures such as tooth extractions."*[85(p.364)]

Early enthusiasm for the use of thrombin to help control hemorrhage was tempered by concerns about hepatitis transmission, and in 1946, all pooled blood products were withdrawn from military stocks and the FDA removed the last human fibrinogen from the market in 1977.[220] Recently, new methods have been found to inactivate viruses in pooled plasma and more extensive purification has enabled production of safe hemostatic agents from pooled blood products.

Increased focus on improving prehospital hemorrhage control was the catalyst for development of dry fibrin sealant dressing (DFSD) by COL John Holcomb, MD. The DFSD is designed for direct application to bleeding surfaces for control of serious hemorrhage [221-225]. The dry thrombin and fibrinogen used on the dressing provide, in great excess, the last two clotting factors in the coagulation cascade so when blood from a hemorrhaging vessel comes in contact with the dressing, a strong, clot-like adhesive is quickly formed.[220] The fibrin dressing has been shown to effectively control both external[221] and internal hemorrhage[223, 224, 226-230] in animal models.

In an unblinded, randomized, prospective trial using a caprine ballistic wounding model, the DFSD reduced both total blood loss and blood loss per kilogram of body weight as compared to a standard gauze dressing. By 20 minutes post-injury, the mean blood pressure in the DFSD-treated group had returned to its pre-injury level while at the same point in time, the mean blood pressure of the gauze-dressing-treated group remained near its post-injury low.[225] All dressings in the study were applied in the same manner as conventional gauze dressings.

There is some evidence to suggest that the DFSD is more sensitive to precise application for optimal function than is the standard gauze battle dressing. In a preliminary study of the efficacy of the DFSD in a simulated land-mine injury in a caprine model done by LTC Paul Dougherty at the Joint Special Operations Medical Training Center at Fort Bragg in 1999, it appeared that the DFSD did not perform as well as a conventional dressing unless the dressing was tucked down into the depth of the wound to ensure that the fibrin was in close proximity to the point of bleeding. Although this study may not have been completed and no other discussion of optimal position of the DFSD appears in the literature, COL Holcomb, developer and strong proponent of the DFSD, admitted that, *"These agents [hemostatic dressings] are not magic; they must be placed in apposition to the major bleeding source."*[155]

During recent military operations in Iraq (Operation Iraqi Freedom) the DFSD was fielded under a special investigative protocol approved by the FDA that ceased following conclusion of major combat operations. During this approved protocol, the DFSD was fielded to Special Operations combat medics involved in ongoing combat operations in Operation Iraqi Freedom, but it was not used in any instance.

In any case, given their higher level of medical training, the field performance of this dressing when used by Special Operations Combat Medics and Special Forces medics may not be equivalent to that which may later be observed in conventional medics/corpsmen.

It is hoped that someday this dressing and other hemostatic agents will help to increase the number of survivors of combat wounding. Unfortunately the cost of the DFSD is quite high (currently $1,000 per dressing) and the price is not expected to fall below $300 per dressing[155] even when it goes into full-scale production. This high cost will likely restrict the widespread usage of this dressing and thus, even if it is highly effective, it is unlikely to have a significant impact on survival because it won't be widely available at or near the point of wounding.

QuikClot™

In 2002, a study was conducted at USUHS to evaluate the efficacy of several different, commercially available hemostatic agents in reducing blood loss after lethal uncontrolled hemorrhage in Yorkshire swine.[231] A complex groin injury that included complete division of the femoral artery and vein was inflicted, and after 5 minutes the animals were randomized to the following groups: (1) no dressing, (2) standard dressing, (3) standard dressing plus aggressive resuscitation, (4) standard dressing plus Rapid Deployment Hemostat™ (RDH, Marine Polymer Technologies], (5) standard dressing plus QuikClot™ hemostatic agent (Z-Medica), (6) standard dressing plus TraumaDEX™ (Medafor, Inc.).

In this Alam study all groups received one liter intravenous saline except Group 3, which received unlimited saline, with the goal of maintaining a systolic blood pressure >100 mm Hg. There were six animals in each group. Application of a wound dressing decreased mortality in all groups as compared to the no dressing group (Group 1), which had 83% mortality. Because of the small size of the groups, this difference was only significant for the QuikClot™ hemostatic agent, which had 0% mortality. Application of the standard dressing alone decreased mortality to 33.4%.

RDH™ is a poly-N-acetylglucosamine derived from algae that is backed by 4"x 4" gauze. QuikClot™ is a granular zeolite that adsorbs water (creating significant heat of adsorption) and promotes clot formation when it is poured onto a bleeding site. TraumaDEX™ is a powder-like agent that consists of bioinert microporous particles that absorb water and promote clotting by producing a gelling action.

In May 2002, the FDA approved QuikClot™ for clinical use. Based, in part, upon the findings in this study the United States Marine Corps decided to consider fielding QuikClot™ to medical personnel preparing to engage in impending combat operations in Iraq. Because of some concern about the potential for injury that might occur as the result of the exothermic heat of adsorption that is created when water (or blood) is added to QuikClot™, a study was done by Dr. Hasan B. Alam at USUHS to assess the degree of burn injury that might be produced during clinical use of this product.

In vitro, depending upon the amount of blood/saline and depending on the rate of addition, the temperature varied considerably. In the lab, with just the right mixture of blood, saline, and QuikClot™ the exothermic reaction reached as high as 65°C (149°F). Fortunately, in vivo, the maximum recorded temperature in tissue did not exceed 45°C (113°F). As noted by Dr. Alam, this was probably because the large volume of blood and the surrounding tissue acted as a heat sink and the distribution of QuikClot™ over a wider area minimized the amount of heat at any specific point.[232]

An interdisciplinary, multiservice panel of medical personnel chaired by COL Dave Burris was convened at USUHS in February 2003 to review Dr. Alam's findings and to make a recommendation to the U.S. Marine Corps regarding the advisability of fielding QuikClot™ to corpsmen. QuikClot™ was recommended for fielding with certain provisions. This panel concluded the following:

- QuikClot™ causes a variable amount of local tissue damage when used in animal models. Damage varies from none to as much as 5 mm into surrounding tissues. Localized burn injury to regional blood vessel walls and nerves could lead to morbidity if the casualty survives.
- In one animal study, wounds closed primarily after the use of this agent became infected. Because combat wounds should not be closed primarily, the significance of this finding is unclear.
- Given the risks of tissue damage, QuikClot™ should not be used when there is no life-threatening hemorrhage.
- QuikClot™ controls hemorrhage. The risk of tissue damage is offset by the potential to save life in otherwise uncontrollable external hemorrhage.

- QuikClot™ should only be used to treat external wounds.

- QuikClot™ should only be used after properly applied standard methods of hemorrhage control have failed.

- QuikClot™ should be an accountable item that requires reporting each time a packet is utilized.[233]

Based upon the demonstrated efficacy of QuikClot™ and upon the panel's recommendation, QuikClot™ was fielded for use to U.S. Navy corpsmen supporting the U.S. Marine Corps during combat operations in Iraq.

The following comments regarding QuikClot™ were included in an early U.S. Marine Corps Operation Iraqi Freedom After Action Report on the performance of various items of equipment including QuikClot™. [234] These comments do not reflect a comprehensive assessment of the effectiveness of QuikClot™ in treating casualties, but they are worth consideration.

2D Tank Battalion Surgeon LT Bruce Webb (USN) stated that QuikClot™ was ineffective (specifically, it was ineffective on arterial bleeding). Battalion corpsmen attempted to use QuikClot™ in three separate occasions:

- Wounded Iraqi civilian. Shot near brachial artery. QuikClotTM was applied per the instructions. The substance dried but was flaking off. Standard direct pressure applied by corpsman proved more effective on the patient.

- Iraqi civilian shot in back with punctured spine. QuikClotTM applied to severe bleeding. Pressure from bleeding sprayed QuikClotTM away. According to LT Webb, "QuikClotTM was everywhere but the wound."

- Iraqi civilian, female, shot in femoral artery. She suffered severe arterial bleeding. Patient bled out. QuikClotTM unable to be applied effectively due to pressure of blood flow from wound. Patient died.

- An LAR Marine was shot in the femoral artery. QuikClotTM was applied to the heavily bleeding wound. The pressure from the blood soon caused the QuikClotTM to be pushed out of the wound and rendered ineffective. A tourniquet was applied instead. The patient died.

It was noted that QuikClot™ may work if applied in a "buddy system" manner, with one individual applying the QuikClot™ substance while another quickly applies sterile gauze to the wound. However, applying the QuikClot™ as directed appeared to be ineffective. Direct pressure and tourniquets were used instead. It was noted in the report that the Marine Expeditionary Unit medical officer who was interviewed had a more favorable opinion of the efficacy of QuikClot™ and the author of the report recommended that further study of this item be done (unfortunately data regarding the use of this item were not collected in a rigorous fashion and thus it will not be possible, even in a retrospective way, to truly assess the efficacy of QuikClot™ when used to treat combat casualties during Operation Iraqi Freedom).

HemCon™

After Dr. Alam's initial study of available hemostatic dressings the FDA approved another hemostatic agent, the HemCon™ hemostatic dressing. HemCon™ is a chitosan-based, poly-N-acetyl-d-glucosamine

-- a naturally occurring substance. This hemostatic dressing is described by Alam et al[232] and is discussed in a review of hemostatic dressings for first responders by Neuffer et al.[235]

Pusateri et al. conducted a study of the efficacy of this product in controlling severe hemorrhage from a hepatic injury in a swine model.[236] They compared the chitosan dressing to gauze sponges in terms of ability to reduce hemorrhage and improve survival. Following induction of a large liver injury, the animals were resuscitated with Ringers lactate back to their baseline MAP at a rate of 260 mL/min (note that this is very aggressive fluid resuscitation). The dressings were applied directly to the areas of bleeding and compression was applied. The amount of post-treatment blood loss was determined by suctioning of the shed blood from the abdominal cavity.

Post-treatment blood loss was significantly reduced in the chitosan dressing group as compared to the gauze sponge group ($p<0.01$) and survival was also significantly better in the chitosan dressing group (7/8 compared to 2/7). Whether this dressing would be more effective than a properly applied standard battle dressing at controlling hemorrhage in an extremity injury in a combat casualty is unknown, however. Given that current resuscitation protocols call for limited to no volume resuscitation until hemostasis is achieved, it is unclear how this dressing would have compared to gauze if no effort had been made to raise the MAP back to pre-injury levels.

It is worth noting that Alam et. al.,[232] in their review of the various hemostatic agents considered for use by the U.S. military, concluded that, "Clearly hemostatic agents are not a substitute for, but an adjunct to, the standard treatment, and adequate training of the user will be a key factor in obtaining the desired benefit." This is most certainly true. They go on to point out that, "For the first time since the Crimean War, the KIA rate has markedly dropped below the historic 20%" with there being an overall KIA rate during Operation Iraqi Freedom of only 12.2% (citing a Washington Post article by Loeb dated 28 December 2003) – implying that the use of hemostatic dressings contributed to this decline.

While the assertion in the Alam article that the KIA rate has declined dramatically during Operation Iraqi Freedom may be true there is good reason to suspect that it is not; at least not to anywhere near the extent stated. First, at the time of their article's publication the conflict was still ongoing so the final numbers, whatever they are, have not been tabulated and analyzed. Second, this author is unaware of any systematic, comprehensive, study of battle and disease-non-battle injuries and deaths that has been conducted during this war at the time of this writing (an article in the Washington Post is anecdotal at best). Finally, and perhaps most importantly, any decline in the KIA rate, if one actually exists, is far more likely the result of the widespread use of soft and hard body armor in this conflict. None of this is to suggest that there is no value in the procurement and distribution of hemostatic agents to pre-hospital combat health care providers. It is rather intended to make clear that there is no substitute for training and for the proper application of basic first-aid procedures for hemorrhage control and to underscore that injury prevention or attenuation is far more important than the availability and use of these new hemostatic agents.

Factor VII Injection

Future methods of hemorrhage control may include the use of recombinant Factor VII and/or other procoagulants. Factor VII has long been available for use in hospital settings to treat bleeding patients with inherited or acquired coagulation factor deficiencies. The major problem that has significantly limited the use of Factor VII for hemorrhage control, even for this group of patients, has been the risk of disease transmission. Historically, Factor VII was derived from pooled blood products and was associated with a high risk of hepatitis, and more recently, HIV, transmission. Recombinant Factor VIIa (rFVIIa), which is now available, does not have these risks. It is indicated for the treatment of hemorrhage in patients with a genetic Factor VII or Factor IX deficiency (hemophilia A and B) with significant bleeding; albeit at great expense. In the United Kingdom, approximate costs of rFVIIa (converted to U.S. dollars from 2002 U.K. pounds) were $1,130 for 1.2 mg; $2,260 for 2.4 mg, and $4,520 for 4.8 mg). For treatment of severe hemorrhage in an adult, the cost of one dose exceeds $5,600.[237]

Animal studies have demonstrated the efficacy of rFVIIa in reducing mortality in lethal uncontrolled hemorrhage models.[238, 239] Recombinant Factor VIIa has recently been successfully used off-label to treat patients with exsanguinating, and otherwise uncontrollable, hemorrhage due to trauma.[240-242] Occasionally, the results have been quite remarkable despite the fact that, because the rFVIIa was given as a last resort, a number of the patients ultimately died, often from causes other than continued hemorrhage.

Not surprisingly, the major concern with administration of rFVIIa to patients without a known deficiency is the risk of thrombotic complications. Exposure of subendothelial tissue factor at the site of injury initiates the coagulation cascade, and in extensive injury this may trigger a disseminated intravascular coagulation (DIC)-like picture that consumes clotting factors and leads to sustained bleeding. Traditionally, this has been a contraindication to the use of rFVIIa. Despite these concerns, clinical experience with rFVIIa has been relatively free of serious adverse events in general and specifically there has been a low risk of serious thromboembolic complications.[241]

Today, rFVIIa is not even approved for use in U.S. hospitals to treat general trauma patients, nor is it approved for use in the prehospital setting. There is, however, some reason to believe that such treatment might someday be considered at or near the point of wounding. Given the young age and pre-existing good health of the great majority of combat casualties, it may be that rFVIIa, or a related procoagulant, could someday be safely given in the field by prehospital personnel.

Other Methods of Hemorrhage Control

Stabilization of fractures, especially pelvic fractures, is very effective at reducing or even stopping, continued bleeding. Major pelvic fracture can be associated with uncontrolled hemorrhage and mortality as high as 50%. Venous bleeding, which accounts for as much as 90% of pelvic hemorrhage, responds favorably to pelvic bone stabilization.[243]

New external pelvic stabilizers are now available that can be applied at the bedside, but such devices will almost certainly never have a role in the stabilization of pelvic fractures outside of a medical treatment facility. Stabilization of pelvic fractures can, however, be achieved with

devices that prehospital personnel either already carry, such as the PASG [181, 184, 244] or bed sheet [245], or could carry (e.g., Geneva pelvic belt, London pelvic splint, Trauma Pelvic Orthopedic Device (T-POD), etc…).

Stabilization of long-bone fractures, particularly femur fractures, can also minimize the occasionally significant blood loss often associated with these injuries. Finally, stabilization of large, soft-tissue injuries of the thigh and buttocks through bandaging and splinting can help control bleeding even when there is no associated fracture.

Other Types of Shock

As noted above, hemorrhagic shock is the primary circulatory problem of concern in the management of the combat casualty at or near the point of wounding. Other forms of shock occur in combat casualties, although they are much less common. Although disease and non-battle injury continue to be significant causes of morbidity and lost man-days in the combat zone, they are an uncommon cause of death and are outside the scope of this discussion. Therefore, the other forms of shock, i.e., hypovolemic shock from other than hemorrhage, neurogenic, cardiogenic, anaphylactic, and septic shock will only be discussed here in the context of those shock states found in combat.

Hypovolemic Shock – Non-Hemorrhagic

Burns

As noted elsewhere in this series [246], combat-related burns have historically accounted for about 3% of all combat injuries. When considering modern warfare since World War II, particularly armored/mechanized warfare, this percentage has been as high as 10.5% (1973 Yom Kippur War) [247]. During the conflict in the Falklands 14% of all UK casualties were burned and 34% of Royal Navy casualties suffered burn injuries, reflecting the high incidence of burn injury associated with naval warfare.[248]

Increased use of petroleum-powered vehicles on the modern battlefield has contributed to an increase in the number of burn injuries in land combat from both combat and non-combat causes. Although not used against U.S. forces in recent conflicts, fuel-air explosives and enhanced blast weapons are increasingly being used and will likely contribute to an increase in burn injuries in the future.

In armored warfare, a combined injury of burn, primary blast, and penetrating injury is called the "Anti-Tank Missile Syndrome." The management of combined injury is problematic because each individual mechanism may ideally require different fluid resuscitation strategies (see earlier discussion on this issue above).

Burns cause hypovolemia and shock through a variety of means. In burns, the intravascular and interstitial spaces are depleted as fluid exudes from the skin and vasoactive inflammatory mediators cause intravascular fluid to leak into surrounding tissues. All of this can happen soon after burn injury. When burns cover approximately 20% total body surface area (TBSA), hypovolemic shock may occur.

Delays in resuscitation of burn victims can be costly. It has been shown that the time to intravenous access and initiation of fluid resuscitation is a major predictor of mortality in pediatric patients with greater than 80% TBSA burns.[249] In one study, it was demonstrated that if intravenous access was not established within 2 hours from the time of injury, there was a significant increase in mortality.[250] The recommendation of this study was that if initial attempts at vascular access were unsuccessful, intraosseous access should be obtained and fluid administered.

Many different fluid resuscitation formulas for burn resuscitation have been described. These differ in the amount of crystalloid and colloid to be given, and in the tonicity of the fluid. A recent review article by Nguyen et al. notes that colloid administration with albumin or plasma protein substitutes helps maintain oncotic pressure after acute resuscitation but in randomized trials has not been shown to improve clinical outcomes.[251] According to these authors, the consensus is that the administration of colloid is unnecessary for patients with less than 40% TBSA burns and during the first 8 hours for patients with larger burns. No single fluid resuscitation protocol has proven superior at reducing the severe systemic edema that accompanies fluid resuscitation of severe burn injury; a partial list includes the following:

- Parkland Formula The most commonly used formula in adults, the Parkland formula employs crystalloid resuscitation with 4 mL/kg/%TBSA burn, using Ringers lactate in the first 24 hours post-burn.

- Brooke Formula The Brooke formula recommends 2 mL Ringers lactate/kg/% TBSA burn plus 2000 mL of 5% dextrose in water (D5W) during the first 24 hours post burn.

- Consensus Formula Promulgated by the Advanced Burn Life Support Course, the Consensus fluid formula recommends 2-4 mL Ringers lactate/kg/TBSA burn for the first few hours post-burn, with half of the calculated volume given in the first 8 hours and half over the remaining 16 hours.252

Hypertonic saline, both alone and combined with a colloid (Dextran), has also been recommended for the treatment of burn victims (see above), but because 7.5% NaCl/6% Dextran-70 is not currently FDA-approved, it will not be discussed further here.[253] In general, crystalloid resuscitation with isotonic Ringers lactate is considered the best option in the acute phase.

When, for whatever reason, intravenous or intraosseous infusion of fluids is not possible, oral rehydration of moderately burned patients with the World Health Organization Oral Rehydration Solution is an option worth considering; certainly one that should receive further study. In an Egyptian study by El-Sonbaty, an oral rehydration solution called Rehydran-n (CID Co. Giza, Egypt) was used to treat children with 10−20% TBSA burns.[254] Rehydran-n is formulated as a 5.5-gram packet, which, when dissolved in 200 mL water, gives a concentration of 90 mEq sodium, 20 mEq potassium, 30 mEq bicarbonate, 80 mEq chloride and 111 mEq glucose per liter.

All of the children in the study did well, despite developing low serum sodium levels. Because these low sodium levels were almost certainly due to the low sodium content of the Rehydran-n solution, El-Sonbaty recommended that the 5.5-gram

packet be constituted with 150 mL instead of 200 mL water to increase the sodium content to120 mEq/L. The author concluded that the advantages of this approach include (1) simplicity of use (2) low cost (3) possibility of use as a first-aid treatment until patient arrives at a hospital (4) no risk of fluid overload, and (5) avoidance of all the difficulties and complications of intravenous infusions. Oral rehydration with a suitable solution, therefore, may reasonably be recommended for moderately burned combat casualties who can tolerate oral fluids and in whom intravenous access cannot be obtained. More severely burned patients will have a burn-induced ileus and will not be able to tolerate oral fluids.

Intravascular volume status of burn patients must be re-evaluated on a frequent basis during the acute phase. Monitoring urine output closely is the best indicator of adequate resuscitation. Unfortunately, this can't be done easily or accurately in the prehospital environment. Urine output in children should be maintained at 1 mL/kg/h, whereas for adults, 0.5 mL/kg/h is sufficient. Although early and substantial fluid resuscitation is indicated for patients with significant burns, it is essential to avoid over-aggressive resuscitation, which can lead to pulmonary edema. This is especially important in casualties with inhalation burn injuries, who are at increased risk of pulmonary edema due to increased pulmonary vascular permeability.

None of the recommended burn protocols should be considered a substitute for constant reassessment of urine output and volume status within the context of the patient's overall clinical picture. An increased fluid requirement in a burned combat casualty should raise the index of suspicion for concomitant injury. If there is an increased fluid requirement occult penetrating or blunt injury, inhalation burn injury, or other causes of hemodynamic instability should be sought. Resuscitation should begin as soon as possible after the time of burn injury. Unfortunately, especially in the combat environment, delays in adequate resuscitation are, and will remain, common and this may lead to unnecessary loss of life.[249]

Cardiogenic Shock

Cardiogenic shock results when cardiac action cannot deliver a circulating blood volume adequate for tissue perfusion. The most common cause of this in the general population is myocardial ischemia or infarction. In the combat setting, cardiogenic shock in a young soldier is more likely caused by cardiac tamponade or cardiac contusion from blunt chest trauma.

Pericardial tamponade as a cause of combat-associated death was recognized and well described during the Civil War. The Confederate Surgeon General even noted that some combatants with this injury might survive stating that,

> *When the heart is injured [and yet the man lives a short period]…the pericardium soon becomes filled with blood; the action of the heart is mechanically impeded, and, sooner or later, depending upon the size of the wound and the facility for letting out blood, it ceases its pulsation. Reports of cases are not very rare in which small, oblique incised wounds of the heart have been recovered from…"*
> *255(p.281)*

The true incidence of both cardiac tamponade and cardiac contusion in modern combat casualties is unknown. There are certainly case reports of pericardial tam-

ponade found at autopsy. During World War II, the 2nd Auxiliary Surgical Group recorded that only 3.3% (75 of 2267) of those with thoracic wounds had cardiac or pericardial involvement, and in those cases where there was pericardial involvement tamponade was not present in all cases. Given that thoracic injuries only constitute 7% of all combat injuries[256] (probably less than this in current operations due to widespread use of body armor) and assuming that all of these 75 WWII casualties had pericardial tamponade this means that at most only .231% of all combat casualties in World War II had pericardial tamponade.[257]

It is noted in the above cited WWII report that, "In comparison to the frequency with which it is noted in civilian cardiac wounds, tamponade has been an infrequent finding." The absence of tamponade was attributed to the large wounding missiles used in combat, which create a hole in the pericardium so large that tamponade does not occur [257]. Whether this would be the case today, with an increased use of improved conventional munitions, is unknown. In this historical account of a busy combat surgical unit, it is worth noting that in more than 50% of the cases in which cardiac injury was ultimately diagnosed, the cardiac wound was undiagnosed prior to surgery.

When both pericardial tamponade and hemorrhagic shock are present, it is very difficult to make the proper diagnosis, even in a hospital setting. The classic triad associated with pericardial tamponade: (1) hypotension, (2) muffled heart sounds, and (3) distended neck veins (Beck's triad) are either not present (distended neck veins) or are readily attributable to hemorrhagic shock (hypotension); and finally what exactly constitutes muffled heart sounds is unclear and, in any case, accurate diagnosis of this finding in a busy emergency department, much less a combat environment, seems unlikely. The presence of *pulsus paradoxus* (>10 mm Hg decrease in systolic blood pressure during inspiration), which is sometimes found in pericardial tamponade, could help make this diagnosis. Unfortunately, given the training of the average combat medic, the available equipment, and the chaos of the combat environment, it is not likely that *pulsus paradoxus* could be used to reliably diagnose pericardial tamponade in the field.

The definitive treatment of pericardial tamponade involves thoracotomy, with opening of the pericardium and repair of the underlying injury. Pericardiocentesis, which is advocated by current ATLS guidelines, is a temporizing, not definitive, measure. The technique, as described in the ATLS manual, requires monitoring of the patient's ECG "…before, during, and after the procedure." Evidence of a "current of injury" on the ECG (extreme ST-T wave changes or widened and enlarged QRS complex) indicates that the pericardial needle should be withdrawn. [49] This cannot be done at or near the point of wounding.

Although pericardiocentesis, without early thoracotomy, can certainly be performed at or near the point of wounding, the question is, "Should it be recommended?" The ATLS pericardiocentesis recommendation assumes that a surgeon, with the ability to perform the appropriate definitive procedure, is either immediately available or can be quickly reached. This is rarely the case when combat casualties are cared for at or near the point of wounding.

Special Operations Medics are taught to perform pericardiocentesis whenever either Beck's triad [hypotension, distended neck veins, muffled heart sounds] is present or when hypotension alone is coupled

with a wound likely to cause pericardial tamponade in a patient in whom other resuscitative efforts have been unsuccessful. Unfortunately, in a combat setting, most patients who have tamponade are likely to be hypovolemic from other penetrating wounds so distended neck veins are unlikely, and, as noted above, hearing "muffled heart sounds" in a combat setting is problematic.

Is pericardiocentesis in a combat setting a reasonable recommendation for Special Operations medics who are trained to, or above, the paramedic level? Is it a reasonable recommendation for the average combat medic? Is it a reasonable recommendation for any level of provider at or near the point of wounding? These are difficult questions. Reasonable answers require an assessment of the likelihood of benefit (based upon the incidence of the condition in the population and the efficacy of the procedure) as compared to the likelihood of "cost" (risk of iatrogenic injury if done correctly or incorrectly and if indicated or not indicated).

Pericardial tamponade appears to be a very uncommon condition in combat casualties; the exact incidence is unknown. It seems likely that when it occurs, it would be rare to find all elements of Beck's triad present. It is probable that in combat casualties, pericardial tamponade would usually be combined with some degree of hemorrhagic shock and/or preexisting hypovolemia from dehydration. The incidence of hemorrhage shock, as a cause of fluid-unresponsive hypotension, is certainly much higher than the incidence of pericardial tamponade.

All of this raises the likelihood that, if the above guidelines are followed, that pericardiocentesis will be performed many times more often than required. Thus a potentially large number of patients who would not benefit from the procedure (assuming that any would benefit) would be exposed to the risks of iatrogenic injury (pneumothorax, inducing pericardial tamponade, and inducing potentially fatal cardiac arrhythmias).

If there were evidence that performing pericardiocentesis at or near the point of wounding would benefit patients with pericardial tamponade, it might still be reasonable to recommend the procedure, inherent risks notwithstanding; unfortunately there is not.

When Dr. Ken Mattox, a contemporary leading trauma surgeon, was asked whether he would recommend the performance of pericardiocentesis by combat medics at or near the point of wounding, he said that he would not. Absent almost immediate surgical correction of the underlying condition, Dr. Mattox did not feel that pericardiocentesis would improve survival (personal conversation with author circa 1999).

Without clear benefit, only the risks remain, so until clear evidence of benefit is found, pericardiocentesis should not be performed at, or near the point of wounding. A small bolus of fluid, however, may help improve cardiac output in a patient with cardiac tamponade because increasing the preload will help overcome the resistance to distention of the left ventricle.

Blunt trauma to the chest can cause dysrhythmias and/or can produce a cardiac contusion with resultant impaired cardiac function, both of which can lead to reduced cardiac output and, in some cases, cardiogenic shock. These rarely occur on the battlefield, which is fortunate because it is generally not possible for combat medics to diagnose or treat casualties with these conditions. In the proper settings,

prehospital personnel should consider cardiogenic shock in the differential of a combat casualty with hypotension.

Neurogenic Shock (Spinal Cord Shock)

In the civilian setting, the great majority of spinal column injuries are caused by blunt trauma, with about 45% being caused by motor vehicle crashes, 20% by falls, and 15% by sporting accidents. Only 15% are due to penetrating trauma.[258] In the Soviet combat experience in World War II, over 99% of all spine injuries were caused by penetrating wounds – a significant difference.[259] The exact incidence of spine injury in combat casualties is difficult to ascertain as a significant number of casualties with such an injury die at, or near, the point of wounding. In World War II only 1.5% of casualties arriving alive at a general hospital had spinal cord injuries.[260] More recently, during the Vietnam War, it was found that approximately 13% of all combat casualties have, among their injuries, a penetrating neck wound[261] – but only a relatively small percentage of these have an injury to the spine, and even a smaller number have an injury to the cord. Due to the increased use of body armor and the associated relative decrease in the numbers of penetrating combat injuries, the relative percentage of combat-associated blunt trauma spinal cord injuries from falls and vehicle and aircraft crashes is likely to rise in more modern conflicts.

Neurogenic shock, in which the normal vasomotor tone derived from sympathetic afferent nerves is lost and blood pressure drops, can occur in both partial and complete spinal cord injury from both blunt and penetrating causes. Some data suggest that neurogenic shock is more likely to occur from blunt than from penetrating injury.[258]

Traumatic spinal cord injury is associated with significant alterations in blood pressure, pulse rate, and cardiac rhythm. The immediate, acute response, to spinal cord injury is a systemic pressor response with a widened pulse pressure. This occurs within a few seconds of injury and persists for several minutes. Activation of the sympathetic nervous system and adrenal medulla is responsible for this initial, transient, pressor response.[262] This hypertensive response is often followed by a prolonged hypotensive phase (neurogenic shock) caused by an interruption of neuronal sympathetic activity.

Because the sympathetic nerves exit from above L-1, any patient with a spinal cord injury above that level could potentially have some degree of neurogenic shock. The sympathetic cardiac nerves exit the spinal cord between C-1 and T-5, so cord injury at or above T-5 may be associated with significant bradycardia that may further worsen any hypotension. Bradycardia may also be caused by a trauma-induced reflex parasympathetic discharge. Cardiovascular instability can persist for days to weeks.[263]

Sympathetic tone is most important to maintaining the blood pressure in the erect position so in the wounded supine/prone casualty, the loss of sympathetic tone has a less of an effect on tissue perfusion than might be anticipated. Most studies of neurogenic shock define hypotension as being present when systolic blood pressure is < 100 mm Hg, yet systolic blood pressure > 90 mm Hg is certainly adequate for tissue perfusion; the currently recommended threshold for volume-resuscitation of patients with uncontrolled hemorrhagic shock is systolic blood pressure < 85 mm Hg.

In a study by Zipnick et al of patients with penetrating spinal cord injuries primarily from gunshot wounds, only 5 of 75 patients (6.6%) with penetrating spinal cord injury had clear-cut, isolated, hypotension that could be attributed to neurogenic shock. Although most patients (78%) had complete spinal cord injury, only 18 (24%) were hypotensive in the field; an additional 5 patients developed hypotension after reaching the emergency department (hypotension was defined in this study as systolic blood pressure < 100 mm Hg) for a total of 23.[258] Of these, 18 had significant blood loss to explain their hypotension. Patients with cervical and upper thoracic injuries had significantly lower heart rates in the emergency department than did those with lower cord injury, but the lowest mean heart rate was 71 beats per minute.

Patients with cervical and upper thoracic spine injuries did not have a greater risk of initial hypotension; in fact, patients with lumbar cord injuries were most likely to be hypotensive, presumably because of a higher amount of blood loss. The authors noted that, *"Whereas patients with blunt spinal cord injuries are at risk for concomitant blood loss injuries, 70% of those with hypotension have neurogenic shock. This is far different in patients with penetrating spinal cord shock injuries in whom pure neurogenic shock is relatively rare."*[258]

Casualties in neurogenic shock, while suffering from the same basic underlying pathology of inadequate tissue perfusion that is present in all the other forms of shock, are different from those with some degree of hypovolemia in that there is no vasoconstriction (indeed this is the underlying problem). Replacement of vasoconstriction with vasodilation means that clinically, patients in neurogenic shock have warm, pink, dry skin instead of cold, cyanotic, moist skin. Other clues to a diagnosis of neurogenic shock, aside from hypotension with warm dry skin, are varying degrees of paralysis, and, if the spinal lesion is above C-5, some degree of respiratory difficulty that varies from abdominal (diaphragmatic) breathing to apnea.

From the perspective of the healthcare provider resuscitating the combat casualty at or near the point of wounding, the question is, "Does it matter, in terms of fluid resuscitation, if the casualty has a spinal cord injury and may have neurogenic shock?" The answer is, "Probably not." First, it should be assumed that all combat casualties, irrespective of mechanism, have ongoing hemorrhage. Zipnick et al. conclude their study with the comment that, *"Patients with early hypotension following penetrating spinal injury should be assumed to have a major blood loss injury...hypotension should not be ascribed to spinal cord injury until an exhaustive search for blood loss is completed and is clearly negative".*[258] This comment is applicable to victims of blunt trauma as well. Second, the current combat medic resuscitation protocol fluid resuscitation is dictated by mental status changes, the presence or absence of a radial pulse, and response to a fluid challenge. This protocol is reasonable for the casualty with neurogenic shock as well because the most important objective, in the short term, is to maintain adequate cerebral perfusion. Because the initial assumption should be that hypotension in a combat casualty with a possible cord injury is the result of hemorrhage, if fluid resuscitation is indicated, the type of fluid used should be the same as that recommended for hemorrhagic shock.

Given the underlying pathophysiology in neurogenic shock, it would seem reasonable to suggest using the PASG, if one is

available, because the PASG mechanically increases afterload. The problem is, as has been pointed out, that even when neurogenic shock is present, there is often concomitant hemorrhagic shock, often with uncontrolled blood loss, and the PASG is contraindicated in such a circumstance.

Furthermore the literature fails to demonstrate the efficacy of PASG to treat neurogenic shock. There is one situation in which it would still seem reasonable to recommend PASG in the management of a patient with presumed neurogenic shock, and that is in any circumstance in which such a patient would be lifted in a head-up configuration during extraction or evacuation, such as might occur during Stokes litter evacuation of a casualty onboard ship or in urban terrain. In this situation, a patient who might have been maintaining quite adequate cerebral perfusion while in the supine position could suffer a catastrophic drop in pressure when placed in a head-up position during evacuation. The PASG, by providing external compression, could counter the hypotension caused by venous pooling during a head-up evacuation.

Anaphylactic Shock

Although anaphylactic shock can certainly occur in the forward battle area from a wide range of etiologies, it is exceedingly unlikely that it would occur as a direct result of enemy action so it will not be discussed here beyond noting that it can occur as a result of medications administered to combat casualties.

Septic Shock

Although septic shock certainly may occur in combat casualties it generally occurs days after wounding and so would rarely be a cause of shock that would be treated at, or near, the point of wounding. In the case of penetrating abdominal wounds septic shock may, however, occur in less than 24 hours in some instances. In a study by Klein et al. [264] there were eighty-eight episodes of wound associated infection identified among 624 consecutively admitted battlefield casualties. Ninety-one per cent of infections occurred despite administration of a prophylactic antibiotic and in 65% of cases multiple antibiotics were being administered; so early antibiotic administration does not eliminate the risk of septic shock. In fact the practice of antibiotic wound prophylaxis may, overall, have a negative effect on battlefield wound infections by doing little-to-nothing to prevent them and by increasing the risk of infection with a resistant organism.

In a setting such as occurred during the Afghanistan conflict in the 1980's in which the International Committee of the Red Cross (ICRC) ran hospitals for the wounded that were some distance from the fighting in Pakistan, the wounded might take many days to arrive. In such a situation it is certainly possible for wounds to become infected and for septic shock to occur prior to hospital treatment. It was found in that during this conflict wounds sutured in the field tended to putrefy [265] and in this kind of a setting septic shock may be seen.

Thankfully septic shock is extremely rare on the modern battlefield due to reasonably rapid evacuation of casualties. When evacuation is much delayed, for whatever reason, septic shock certainly can occur in patients before they reach a hospital. Septic shock occurs when exotoxins (gram-positive toxic shock) or endotoxins (gram negative sepsis), enter the systemic circulation causing hypovolemia, cardiovascu-

lar depression, and systemic inflammation. Lipopolysaccharide's, contained in the cell wall of gram-negative bacteria, are one of the major causes of septic shock but gram-positive organisms can also cause septic shock and recently there has been a rise in the number of cases of sepsis in hospitalized patients caused by gram-positive organisms.[266, 267]

Hypotension in septic shock results from hypovolemia, inflammatory-mediated vasodilation, and cardiac depression. The hypovolemia of septic shock is both absolute, from gastrointestinal volume losses, sweating, and decreased oral intake, and relative from capillary leak and third spacing. Inflammatory-mediated vasodilation causes pooling of blood in the vascular bed similar to that occurring in neurogenic shock. Septic shock depresses cardiac function even early in the course of septic shock and eventually results in decreased cardiac output further worsening hypotension. Hypoxemia from capillary leak-induced acute respiratory distress syndrome (ARDS) and pneumonia and/or pulmonary aspiration, worsens the problem of hypoperfusion in septic shock. Very large volumes of fluids may be required to resuscitate a patient in septic shock. Fluid resuscitation should be continued until the mean arterial pressure is above 60 mm Hg, urine flow is adequate, and mentation is improving. Unlike most other forms of shock in which inotropic drugs that increase myocardial contractility and vasoconstricting drugs that increase systemic vascular resistance are generally either useless or contraindicated, in septic shock both may be helpful. Broad spectrum antibiotics are indicated but the management of septic shock in combat casualties usually involves the excision of necrotic and infected tissue so urgent evacuation to a surgical facility is the most important factor.

Summary

The ability to prevent exsanguination and to recognize and treat hemorrhagic shock is fundamental to quality point-of-wounding care. The single most important factor is the training of prehospital personnel of all skill levels. As important as knowing what to do for the combat casualty, is knowing what *not* to do. It is especially important to emphasize to prehospital personnel, who are by nature action-oriented, that each medical intervention carries with it some degree of risk. They must also understand that the relative value of each intervention varies with the frequency of the condition(s) for which the intervention(s) is/are being performed. If a condition is unlikely to be present in a patient, any intervention intended to treat that condition will expose the patient to risk with little hope of benefit. Because it is not possible to train prehospital personnel to make individual risk-benefit analyses at the point of wounding, their training must be protocol driven and those protocols must be evidence-based.

There must also be discipline in the system to ensure that the protocols are being followed, and this requires some degree of inspection and oversight. Today, unlike in many areas of the civilian sector, care provided by prehospital personnel in support of military operations is not monitored for quality assurance. In most instances, no one evaluates the medical decisions and interventions that are made at

or near the point of wounding. This needs to be done in both peace and war because without such evaluation, real improvements in care are unlikely to occur.
Prehospital personnel must be able to recognize quickly and reliably the patient who is in shock or likely to go into shock. As noted above, this is no easy task. To accurately diagnose shock, it is necessary to integrate a wide range of data from mechanism of injury, anatomic location of wounding, general appearance and behavior of the patient, and physiologic data gathered over time. As previously stated, there is no single test that is pathognomic for shock. Each of the generally recommended tests -- blood pressure, pulse, capillary refill, orthostatic vital signs, and hemoglobin/hematocrit -- lack both the sensitivity and specificity necessary to reliably diagnose shock at or near the point of wounding. In the proper setting, an increase in both rate and depth of respirations and increasing thirst in a combat casualty should be considered evidence of shock until proven otherwise – the value of these findings in diagnosing shock is often under-appreciated.

None of this is to suggest that any of the other tests for shock lack value. Their value lies in being used both in conjunction with other findings that would suggest shock and in being used repeatedly over time. When used to create a pattern or to define a trend, these tests can effectively be used to diagnose shock. Unfortunately in many cases, the combat lifesaver, medic or corpsman that is usually present at or near the point of wounding often has neither the training, equipment, nor time to, quickly and accurately diagnose shock. Furthermore, the circumstances of combat often render this impossible even when the capability exists. This means that prehospital personnel must be taught that when there is any question about the possibility of shock, they should manage the patient as if shock were present.

The single most important treatment for a patient in hemorrhagic shock is to stop the bleeding – *all bleeding* if possible. All else, in some instances even the airway, are of secondary importance. Without hemoglobin to carry oxygen, a patent airway is of little use. Hemorrhage control must be achieved as quickly as possible, utilizing whatever technique or techniques are both effective and medically and tactically appropriate. Ideally, methods such as direct pressure, use of pressure points, and elevation of bleeding extremities should be attempted initially.

Immediate use of a tourniquet is indicated if there is exsanguinating arterial hemorrhage in an extremity, or if the tactical circumstances preclude the application of less harmful, but more time and resource consuming, methods. The continued need for a tourniquet that has been applied as a temporizing measure *must* be re-evaluated at the earliest possible time and if other methods are capable of controlling hemorrhage, the tourniquet should be removed. The question of whether or not tourniquets should be periodically loosened to allow for some distal blood flow needs to be studied, but the experience during World War II was that this practice was implicated in enough combat deaths that it was stopped. Unless good studies show real benefit and unless training, procedures, and new equipment can ensure that the World War II experience with this practice will not be repeated, it should not be advocated.

The non-pneumatic strap tourniquet (NSN 6515-00-383-0565) that has been in the Army inventory since at least World War II *must* be removed and replaced. How many times is it necessary to prove that this tourniquet is ineffective? Finding an

effective replacement should continue to be a high priority medical research effort. Prehospital medical personnel must be trained to effectively apply tourniquets and they must be trained to fully understand the risks and implications of tourniquet application.

Ancillary methods of hemorrhage control such as the use of a dry fibrin sealant dressing, QuikClot™, the chitosin dressing, and/or the BioHemostat dressing may significantly improve the ability of prehospital personnel to control hemorrhage in a combat environment, but this needs to be proven. Most importantly, enthusiasm for these new hemorrhage control devices should not cause prehospital personnel to abandon conventional and proven methods of hemorrhage control or to preferentially use these new and, as yet, largely unproven devices. The reported case, during Operation Iraqi Freedom, of a Marine with a femoral artery injury, who was treated with QuikClot™ first and then a tourniquet is most disconcerting. Although the exact details of this incident are unknown, it appears that QuikClot™ was used as the initial hemorrhage control method, when instead a tourniquet should have been applied. Unfortunately, the Marine died. There is no way of knowing whether he would have survived had the first intervention been to apply a tourniquet, but given the available information it at least seems possible.

After all controllable hemorrhage has ceased, the next question is fluid resuscitation. Is it needed at all and if so, how much and what kind? It is appropriate, for a variety of reasons, that all combat casualties with more than trivial injuries have vascular access established. Intravascular or intraosseous access should be obtained. Intravascular cannulation should be established with a short, at least an 18-gauge or larger, catheter, and this should be set up as a saline lock that can be used for drug and/or fluid administration as required. Intraosseous access can be obtained either through the medial malleolus with the Bone Injection Gun (BIG®), the VidaPort® system, or similar devices, or through the sternum with the Pyng F.A.S.T. ® intraosseous infusion device or other similar device. These devices are generally easy to use, and a properly trained person can obtain intraosseous access in under a minute. Both drugs and fluids can be administered through the intraosseous route, but because of the resistance to flow, fluids cannot be administered as rapidly through the intraosseous route as through the intravenous route.

The current recommended fluid resuscitation protocol promulgated by COL Holcomb for the AMEDD Center & School 91W Health Care Specialist program, which is very similar to that recommended by Beecher et al. in World War II [16] seems reasonable and, in general, is supported by scientific evidence. For patients with ongoing hemorrhage that is uncontrollable in the prehospital setting, low volume, "hypotensive" resuscitation is recommended. How low, for how long, remains to be determined. The key to survival in such patients is rapid evacuation to a location where surgical control of hemorrhage can be achieved.

Although Hextend® or any other similar colloidal solution is recommended for management of hemorrhagic shock, for the reasons previously stated (weight and cube issues primarily), this recommendation may not be optimum for the following reasons:

1. There is no scientific evidence that these resuscitation fluids, when compared with normal saline or Ringers lactate, improve survival in

hemorrhagic shock (in fact there is some evidence to the contrary).

2. The efficacy and safety of these fluids in the management of dehydration of all causes (burn, heat injury, gastrointestinal loss, etc…) and for the treatment of other conditions for which intravenous fluids might be indicated (or used) is unknown.

3. These colloidal solutions are very expensive as compared to crystalloids. Because, in many instances, aggressive fluid resuscitation of combat casualties is now recognized as ill-advised, the need to carry large quantities of fluids is not as great as previously thought, so the weight and cube issue is, perhaps, not as important as it was before. However, because it is unlikely that the selection of one intravenous fluid over another will significantly alter the survival of combat casualties, this should not be allowed to become a contentious issue.

The question remains as to what role hypertonic saline dextran might play in the management of combat casualties. Hypertonic saline dextran is currently being evaluated in human trauma trials. Because of its demonstrable ability to improve outcomes in head-injured patients, plus its ability to rapidly expand the intravascular bed when given through intraosseous access and when given in small volumes, hypertonic saline dextran may ultimately be the single fluid of choice in combat casualty care. Unfortunately, many of the same concerns raised about Hextend®, Hespan®, and other colloidal solutions apply to hypertonic saline dextran as well. Until these concerns can be addressed with good scientific studies, there should be no rush to embrace this resuscitation fluid. One or more of the new oxygen-carrying, hemoglobin-based, resuscitation fluids may prove effective in the treatment of hemorrhagic shock on the battlefield. This has yet to be proven but there is great promise in these new fluids that minimize or eliminate many of the risks and logistical difficulties associated with blood transfusion while preserving oxygen-carrying capability.

The basic underlying defect in all forms of shock is inadequate tissue perfusion. The new solutions will be able to directly address this defect. It is hoped that the introduction of such a solution or solutions will not have the same effect that the introduction of saline had in causing surgeons [or in this instance, prehospital personnel] to become cavalier about hemorrhage control. Kim Pelis, in her article, *Blood Standards and Failed Fluids: Clinic, Lab, and Transfusion Solutions in London, 1868-1916*, notes that following the introduction of saline to treat blood loss, "Surgeons, with…unrestrained operative enthusiasm…spilled great quantities of blood [that was] conveniently replaced with a few to several pints of saline solution."[48] It is virtually certain that no matter how good these oxygen-carrying solutions become, they will never be as good as the patient's own blood. Most assuredly, the patient does best if his or her own blood is retained, if not by preventing hemorrhage in the first instance, then by returning it into the circulation after it has been lost.

Autotransfusion of blood drained from chest tubes appears to be done infrequently in most forward areas for a variety of reasons. Given that this procedure is demonstrably efficacious, relatively easy to do, and logistically supportable, this practice should probably be done more often when clinically indicated. Finally, there is the question of far forward administration of blood. The numerous highly favorable comments about far for-

ward early administration of blood to combat casualties during the Spanish Civil War, World War II, and the Korean War and the more recent favorable comments about the near "miraculous" effect of whole blood when used to treat casualties in Mogadishu should give rise to questions as to why blood is rarely used today forward of a surgical facility. There are most certainly significant logistical and training challenges to far forward blood administration, but surely these are no more onerous than in the past and certainly technological advances have made them less so.

With regard to the management of types of shock other than hemorrhagic, it seems reasonable that only in the instance of burn shock, and perhaps in the unlikely instance of septic shock, should the above-described recommended fluid resuscitation protocol be altered. Burn patients are unique in that they have an ongoing fluid loss that cannot be medically or surgically controlled, and they require early, aggressive, and sustained fluid resuscitation. It is probably of no major consequence, in terms of outcomes, which of the generally accepted fluid resuscitation formulas for burns is used. Ringers lactate is the recommended fluid of choice for at least the first 8 hours in all of the widely accepted resuscitation protocols. In reality, the circumstances of combat make it unlikely that it will be possible to accurately estimate the percent of TBSA burn and even if this could be done accurately by a combat medic in the heat of combat or during evacuation it is unlikely, for a variety of reasons, that it would be possible to accurately administer the calculated amount of fluid. Most importantly, prehospital personnel need to know that seriously burned patients need early and relatively aggressive fluid resuscitation and, unless there is a contraindication (i.e. concomitant pulmonary blast injury and/or inhalation burn injury) at least two liters of Ringers lactate (or normal saline if Ringers lactate is not available) should be administered quickly and, if the evacuation is long, another liter should be administered. If intravenous access can't be obtained, these fluids should be given through intraosseous access. The total amount of administered fluid must be recorded and this information transmitted to the receiving medical treatment facility. As noted above, the World Health Organization oral rehydration solution may be an option for use in moderately burned combat casualties in whom intravenous fluid is not possible for any reason. Like badly burned patients, patients in septic shock may require substantial volumes of fluid.

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Immediate Care of the Wounded

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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 nonlethal) 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]

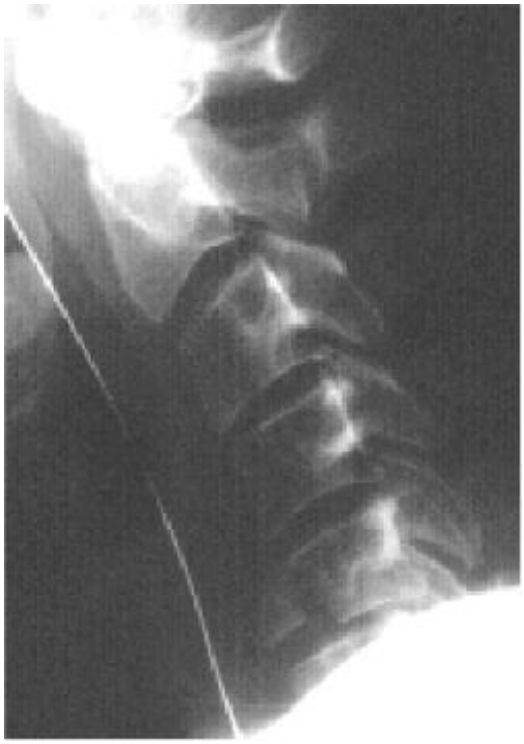
Of 4,555 cases in the WDMET database of Vietnam casualties, 614 records were coded as belonging to combat casualties with some form of penetrating neck wound (open neck wounds; superficial neck wounds; open laryngeal injury; vascular injury of the head and neck; and spinal column injury, with or without 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.

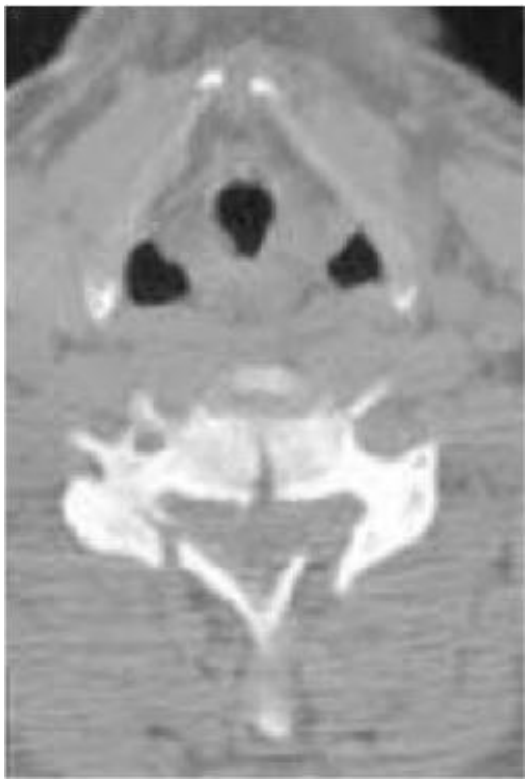
The tactical situation also caused differences in the relative frequency of cord injury and in the anatomic distribution of spine injuries in casualties presenting to hospitals. The preponderance of wounds of the thoracic spine in surviving casualties was, at least in part, due to the fact that those 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 incidence 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 NOT 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^{o} 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 prehospital 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) And 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 radiographic 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 hypotension. 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 nonpenetrating 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.

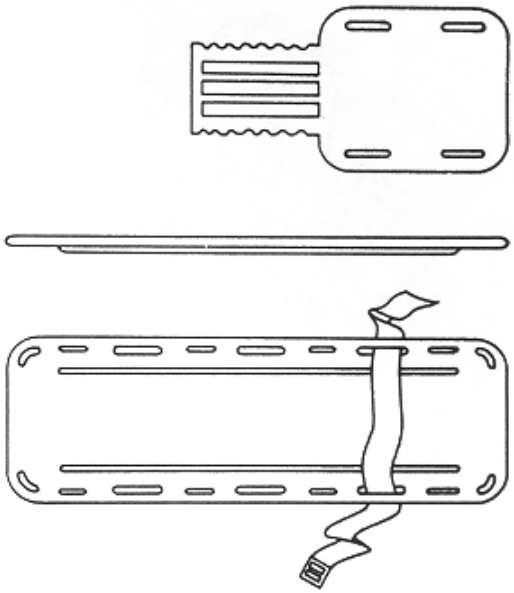
Controversy exists about the performance of cricothyrotomy by prehospital personnel.[115, 117, 118] Mortality rates are high when prehospital cricothyotomy is performed on patients with penetrating neck trauma, but it is unclear whether this is a function of the experience (or lack thereof) of the provider 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 potential 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, not 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

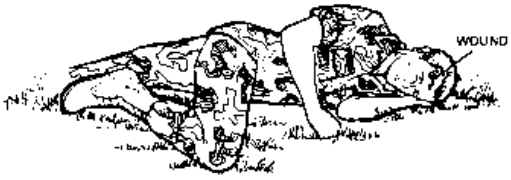


Figure 3-13. Casualty lying on side.

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 glucocorticoid 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 padding 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 all 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 <u>always</u> 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 subdural 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 subdural 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 unconsciousness and a focal neurological finding, such as pupillary asymmetry or decerebrate posturing so the evacuation decision is not difficult. Such casualties should be evacuated to a neurosurgeon as quickly as possible.

The above information is important because 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 described 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 injury.[179, 180] This rare condition typically presents in an athlete who, while still symptomatic from a previous "minor" head injury, returns to play before completely clearing his sensorium and then suffers a second head injury. This syndrome, believed to be caused by persisting cerebral vascular sensitivity, is called the "*second impact syndrome.*" A second injury then leads to cerebral autoregulatory 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 support 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 department patients can safely be sent home. They focus instead on predictive factors such as GCS < 15, focal neurologic deficits, and skull fractures. This is of little use to the care provider at or near the point of wounding, who will feel compelled to evacuate any head-injured casualty to someone who can rule out intracranial injury. In such a circumstance, the evacuation decision will be made primarily 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 remain 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 not 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 severe 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, parenterally,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 cererbral 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 a 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 not 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 appropriate 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.

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