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)]

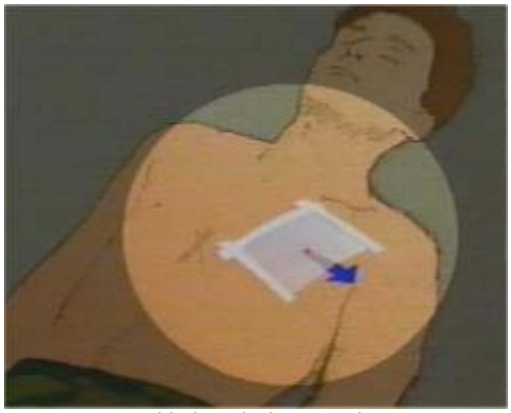
In WWII medical aidmen were originally instructed to apply an occlusive dressing

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)]

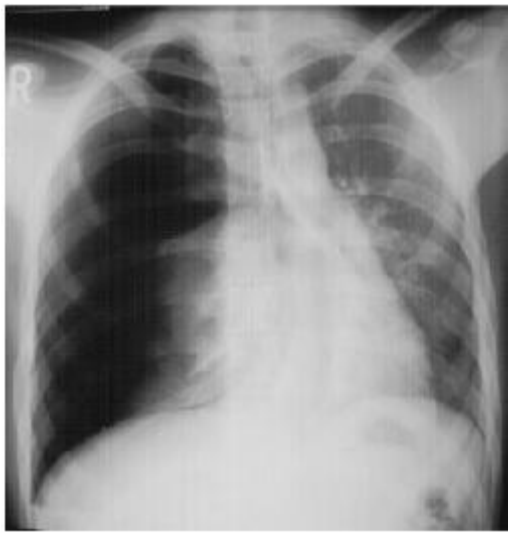
In many instances casualties with a "sucking" chest wound can tolerate the injury if the amount of air entering the wound from outside is not greater than the difference between the tidal volume (resting tidal volume in adult males is approximately 500 cubic centimeters) and the original vital capacity (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:

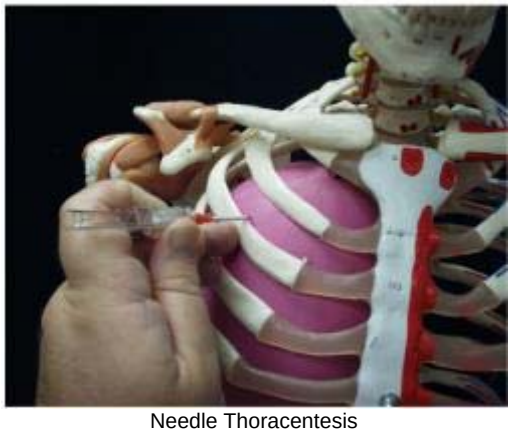
1) The preponderance of shell-fragment wounds, which resulted in a preponderance of large defects of the chest wall. For physical reasons, air could not be entrapped in the chest in such wounds. [NOTE – If true this could also explain the predominance of bullet-caused tension pneumothoraces seen in the 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.

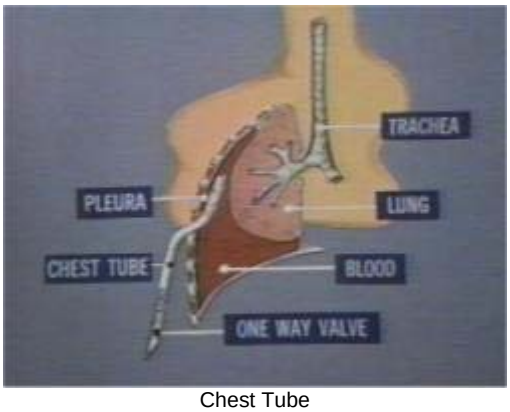


Needle Thoracentesis

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)

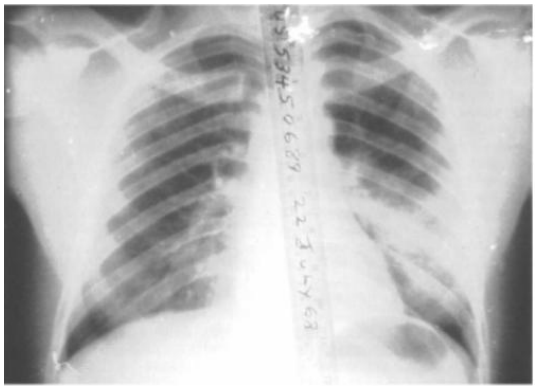
Casualties on whom a needle thoracentesis has been performed must be continually re-assessed because catheters used for this purpose can easily become occluded by blood clots and kinking.[50(p.8)] If an Asherman Chest Seal is available one could be placed over the needle to stabilize it and help prevent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