

MILITARY MEDICINE

ORIGINAL ARTICLES

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The Causes of Death in Conventional Land Warfare: Implications for Combat Casualty Care Research

COL Ronald F. Bellamy, MC, USA*

A research program designed to improve health care delivery will have the greatest impact if its goals are chosen only after a comprehensive review has been made of the ways in which the existing system fails. Combat casualty care is no exception—a research program in this field must be based upon a detailed knowledge of the cause and nature of mortality and morbidity that follow injury on the battlefield. Casualty statistics have been collected during many wars and much is known about the incidence and lethality of wounds. Nevertheless, these data leave certain questions unanswered, especially when they pertain to therapy. There are several reasons why this is so.

First, because these data are specific for a given historical period with its own unique weaponry and level of medical expertise, inferences drawn from data collected in a given war may or may not be useful in predicting the outcome when circumstances are different. For instance, how does evacuation time affect the mortality associated with facial wounds? It is known that during the Korean conflict 1.3 per cent of combat casualties with facial wounds died following evacuation from the battlefield.¹⁴ Can we predict what this statistic would have been if evacuation time had been prolonged from that prevalent during the Korean conflict to that seen during World War I? In World War I, 40 percent of casualties with facial wounds died after leaving the battlefield.³ Is there any reason to believe that this statistic would have been applicable in Korea? Surely the difference between 1.3 percent and 40 percent reflects not the difference in evacuation times, but the improvement in the treatment of these wounds—especially the use of antibiotics.

Second, commonly used terms such as "killed in action" and "died of wounds" are not especially useful when one asks what specifically can be done to improve care. For instance, both the soldier who dies at a battalion aid station 10 minutes after being shot in the chest and the soldier who dies from a drug reaction during a pulmonary decortication for a chest wound inflicted 10 weeks earlier are classified as having died of thoracic wounds.

Third, existing casualty statistics lack a control group against which the efficacy of treatment can be assessed. Of course, it is doubtful that a randomized prospective study would ever be done to demonstrate the effectiveness of combat casualty care but, as will be shown in this paper, existing data can be treated in such a way as to yield what is equivalent to an untreated population.

Much useful information about the effectiveness of combat casualty care is to be found in the vast literature dealing with the surgical management of war wounds. However, more often than not, this material focuses exclusively on the problems of the select group of casualties who have survived to be evacuated from the battlefield. The emphasis is upon increasing the salvage in the died of wounds category and little attention is paid to the far larger category of casualties who have been killed in action. Thus, there is the distinct possibility that the surgical literature will stress treatment problems which may be of compelling interest in the surgical intensive care ward or operating room, but which are of minor importance when viewed in the overall context of combat casualty care.

In this paper, we will try to circumvent the aforementioned problems by analyzing the existing data in a different manner than heretofore used. A hypothetical model population of combat casualties, about which all important facts are specified, will be constructed from existing casualty statistics. Clinical judgment will then be used to decide mortality as a function of time after wounding. This idealized population may be considered an untreated control group, and the effect of combat casualty care instituted at a given time after wounding can then be assessed by reconsidering the outcome that occurred when no treatment was given. Although this analysis suffers from being greatly dependent upon a number of assumptions, the contributions—as well as the deficiencies—in present-day combat casualty care become abundantly clear.

Data Analysis

The following factors were specifically excluded from analysis:

From the Division of Combat Casualty Care, Letterman Army Institute of Research, Presidio of San Francisco, Calif. 94129.

* Chief of Division.

1. Combined conventional and nuclear/chemical/biological injury
2. Morbidity—functional outcome in survivors
3. Type of action—offensive versus defensive
4. Wounding mechanism—mix of bullet and fragmentation injuries
5. Nonbattle injuries or disease

Combined injuries were excluded from analysis because mortality may well approach 100 per cent. The type of action and the mechanism of wounding are both important considerations, the former affecting the incidence of wounds and the latter affecting lethality. A consideration of nonbattle injuries would have required that certain categories of wounding be increased by as much as one-third. Finally, morbidity has not been addressed. Morbidity needs to be considered in a separate study.

Blast and burn injuries have also been excluded from consideration, because neither occurs with sufficient frequency to affect overall mortality rates. Casualty statistics from World War II suggest that, in a population of 1,000 battle casualties, one might expect 22 blast injuries with one death⁷ and 12 burn casualties, with a mortality of 30 per cent.²

The first step in the analysis was to determine the distribution of wounds according to anatomical location. Following the procedure adopted by statisticians working with data from World War II and the Korean conflict, it was assumed that it was usually possible to recognize a single wound as the major life-threatening "hit," even though as many as one-half of all casualties have multiple

TABLE I
ANATOMICAL LOCATION OF MAJOR WOUND

Location	World War II	Korean War	Model
	Per Cent	Per Cent	Per Cent
Head	14.5	13.3	14
Face	6.6	8.4	7
Neck	2.3	2.6	2.5
Thorax	11.7	11.2	12.5
Abdomen	9.5	9.1	10
Upper Extremity	20.0	23.0	20
Lower Extremity	32.1	30.8	31.0
Multiple	3.3	1.6	3

TABLE II
PROBABILITY OF BEING KILLED IN ACTION BY A GIVEN WOUND

Location	World War II	Korean War	Model
	Head	0.59	0.56
Face	0.14	0.11	0.08
Neck	0.48	0.39	0.35
Thorax	0.44	0.40	0.38
Abdomen	0.38	0.31	0.28
Upper Extremity	0.035	0.024	0.02
Lower Extremity	0.048	0.053	0.05
Multiple	0.76	0.57	0.50

injuries. In support of this contention, in 500 consecutive autopsies performed on casualties killed in action in Vietnam, only 30 had more than one anatomical region with a potentially fatal wound.⁵

The frequency distribution of wounds as a function of their anatomical location was calculated as follows. Head wounds incurred during World War II will be considered as the specific example. The total number of battle casualties with head wounds is the sum of casualties with head wounds killed in action (KIA_H), casualties with head wounds who died of their wound (DOW_H), and all casualties with nonfatal head wounds (NFW_H). Total battle casualties will equal the sum of all those killed in action (KIA), died of wounds (DOW), and nonfatal wounds (NFW). Therefore, the percentage of all battle casualties with head wounds equals:

$$100 \cdot (KIA_H + DOW_H + NFW_H) / (KIA + DOW + NFW)$$

Since the most accessible casualty statistics give the percentage of those killed in action with head wounds ($\%KIA_H$) rather than KIA_H , the latter will be calculated from the former as:

$$KIA_H = KIA \cdot \%KIA_H / 100$$

Similar formulae can be written for DOW_H and NFW_H . The Surgeon General has recorded the following statistics for World War II:⁷

KIA	192,220	$\%KIA_H$	35.6%
DOW	20,810	$\%DOW_H$	19.7%
NFW	578,914	$\%NFW_H$	7.4%

From these data, one can calculate that the following percentage of all battle casualties during World War II sustained head wounds:

$$100 \cdot \frac{(192,220 \cdot 0.356) + (20,810 \cdot 0.197) + (578,914 \cdot 0.074)}{192,220 + 20,810 + 578,914} = 14.5$$

A similar calculation can be made for the Korean conflict, using the following statistics¹⁴:

KIA	19,353	$\%KIZ_H$	37.9%
DOW	1,957	$\%DOW_H$	25.4%
NFW	75,831	$\%NFW_H$	6.8%

Of all Korean conflict battle casualties, 13.3 per cent had head wounds.

Table I gives the frequency distribution for World War II and Korean conflict wounds calculated by the formulae for the following anatomical locations: Head, face, neck, thorax, abdomen, upper extremity, and lower extremity. A separate category has been included for multiple wounds. Casualty statistics for the hypothetical population used in this paper were taken to be the average of the World War II and Korean conflict statistics, except for wounds of the trunk.¹⁴ In keeping with the experience during the Vietnam War, when a much higher relative incidence of trunk wounds was seen,⁵ the figures for

thoracic and abdominal wounds in Table I have been adjusted upwards at the expense of extremity wounds.

A detailed description of the pathology of each individual wound was needed. For instance, in a sample population of 1,000, how many of the 140 casualties with head wounds had wounds which involved the ventricular system? This seemingly difficult task was accomplished by combining data from two sources: autopsy studies on casualties killed in action and the surgical literature on the management of war wounds. The latter source tells us about the types of wounds seen in casualties surviving to reach medical care facilities, while the former source tells us about the nature of lethal wounds. On the basis of data collected during the Vietnam War,⁵ one can expect that 56 per cent of casualties killed in action by head wounds will have penetrating wounds involving the ventricular system, basal ganglia, and/or one or more cerebral lobes. Thirty-two per cent of the casualties killed in action by craniocerebral trauma will have through-and-through perforating wounds, with loss of craniocerebral tissue. The remaining 12 per cent will have injuries to the brain stem. A similar analysis can be performed on casualties surviving to reach medical treatment facilities.⁵ Eighty percent of these casualties may be expected to have wounds in which the dura has been perforated, usually with injury to only one cerebral lobe, while the remaining 20 per cent have cranial trauma only—the dura is intact. The neurologic status of the surviving casualties will be subsequently used to predict the clinical outcome.

TABLE III

PREDICTED MORTALITY IN A HYPOTHETICAL POPULATION OF 1,000 UNTREATED COMBAT CASUALTIES

Location	0-1 hour	1-6 hours	6-24 hours	1-7 days
Head	70	11	15	27
Face	6	2	1	24
Neck	9	2	1	3
Thorax	48	11	8	33
Abdomen	28	17	16	31
Upper Extremity	4	4	3	29
Lower Extremity	15	12	10	67
Multiple	15	7	3	5
Total	195	66	57	219
Cumulative Total		261	318	537

TABLE IV

MORTALITY—RAPID EVACUATION FROM THE BATTLEFIELD

	KIA (20%)	DOW (2%)
CNS Trauma	71*	7
Hemorrhage	98	4
Other	26	5**
Total	195	16

*Including 1 cervical cord injury

**Including 2 septic shock and 1 respiratory distress syndrome

Now I have partitioned the total number of casualties with head wounds into those killed in action and those surviving to reach treatment facilities. For instance, in a sample population of 1,000 casualties, how many of the 140 with head wounds will be classified as being killed in action? Rather than directly using the appropriate casualty statistics from World War II and the Korean conflict, I have elected to modify these data in a way which I believe makes them more useful. Table II shows the fraction of casualties who were killed in action during World War II and the Korean conflict by wounds in the specified anatomical locations.¹⁴

In all categories except lower extremity wounds, the probability of being killed in action by a given wound was less in the Korean conflict than in World War II. This cannot be due to improved medical care because the killed in action category consists only of a casualties who died before reaching medical treatment facilities. It may be that the lower percentage killed in action during the Korean conflict reflects the decreased lethality of weapon systems used in that conflict. Be that as it may, I prefer to interpret the differences as being the result of the greater time spent in evacuating from the battlefield in World War II. The longer the severely injured casualty remains on the battlefield, the greater will be the probability of dying and thereby being classified as having been killed in action. If one assumes that it took twice as long to evacuate casualties in World War II compared with the Korean conflict (six to eight hours versus two to four hours)¹⁷ and a linear relation between evacuation time and mortality, one can predict how evacuation time affected survival. Specifically, a linear regression performed on these data makes possible an estimate of the immediate (< one hour) mortality for a wound of given location. Table II shows the results of these calculations, which will be used in the subsequent model development.

Of the original 140 casualties with head wounds, we can predict that 50 per cent or 70 will die immediately or shortly after wounding. Since these casualties would have died before evacuation, they may be considered to have been killed in action. Can we predict what will happen to the surviving 70 casualties, assuming that they receive no treatment on the battlefield other than wound dressing and analgesia? The outcome is predicated upon the established principles of neurological trauma surgery⁹ and the following considerations. As previously stated, about 80 per cent of the surviving casualties have wounds which have perforated the dura. The neurologic status of these casualties can be used to predict their outcome. About 20 per cent of the casualties will be comatose, and the majority of these will die within six hours if respiratory support is not instituted. About 50 per cent of the surviving casualties will have localizing neurologic findings and most will die within 24 hours, unless intracranial hematomata and necrotic tissue are removed. The majority of the remaining casualties with dural penetration will die within three days unless cerebritis and/or meningitis is treated. Finally, without local wound care, some of the casualties without dural penetration will develop life-threatening soft tissue infection within the first week.

Thus, I estimate that, without any treatment, of the 140 casualties with head wounds 70 will die within the first hour after wounding, 11 will die one to six hours after wounding, 15 will die six to 24 hours after wounding, 22 will die one to three days after wounding, and five will die three to seven days after wounding.

A similar analysis was carried out for wounds involving the other anatomical regions but, because of space constraints, only the salient features of these analyses will be considered.

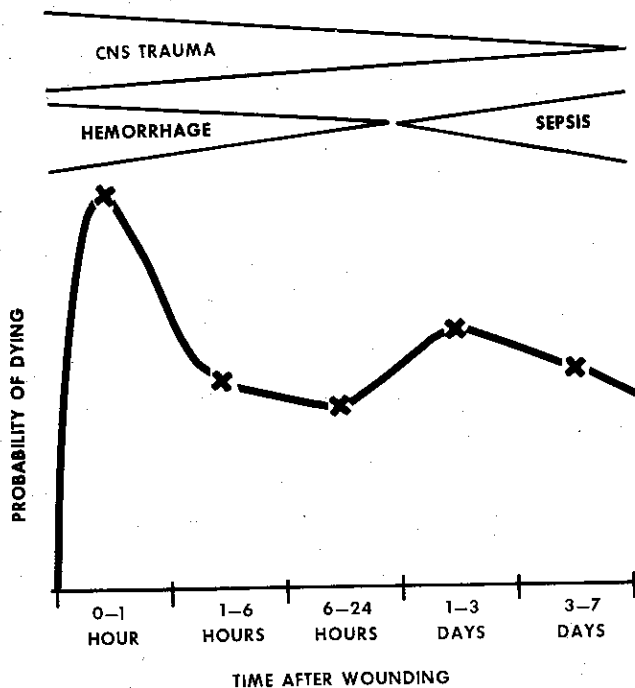


Fig. 1. Probability of dying from an untreated wound sustained in combat as a function of time after wounding.

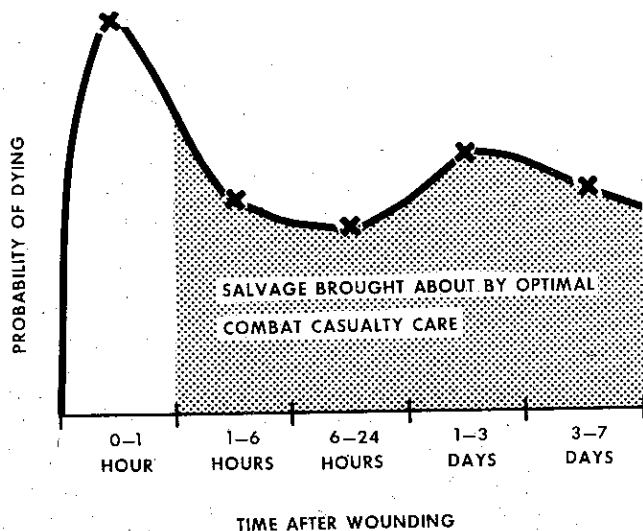


Fig. 2. Effect of optimal combat casualty care on the probability of dying after wounding. It is assumed that 20 per cent of casualties are killed in action and that two per cent of those evacuated die of wounds.

Face: Since the majority of casualties with facial wounds have mucosal lacerations, invasive oral sepsis will be a serious problem in untreated casualties.⁶ No doubt this factor contributed to the horrendous number of casualties dying of facial wounds during World War I (40 per cent).

Neck: Casualties with neck wounds will be killed by carotid or tracheal injuries or a combination of the two. Soft tissue infections in the survivors will be unusual, except in the rare casualty with an isolated esophageal injury. Occasionally, death will occur from delayed blow-out of a contused carotid artery or an isolated injury of the spinal cord.

Thorax: About one-half of these casualties will have pulmonary lacerations of varying grades of severity.⁵ The most common cause of immediate death is laceration of the heart. I assume that casualties that required emergency operation (14 per cent of wounded surviving to reach definitive care⁵) will die during the first day after wounding if not treated surgically. An unanswered question is what happens to the large number of casualties with hemopneumothoraces that are not immediately life-threatening. I have assumed that one-third develop life-threatening empyema. Twenty per cent of the total number of casualties with chest wounds have chest wall injuries without pleural penetration.¹²

Abdomen: The most common immediate cause of death is exsanguination from a lacerated liver.⁵ The colon is the single most commonly injured organ.⁵ Casualties with penetrating wounds of the abdomen who do not die of hemorrhage will almost certainly die of peritonitis. I assume that three per cent of casualties with abdominal wounds do not have peritoneal penetration.¹³ Twenty-five per cent of casualties with intraabdominal injury have associated injury to the pelvis or spine.

Upper Extremity: Traumatic amputation with associated exsanguination is the cause of death in about two per cent of these casualties.⁵ However, about four per cent will have wounds of the brachial, ulnar, or radial arteries which will prove fatal, either because of hemorrhage or because infection will be potentiated by ischemia. The major unanswered question is, what will happen to casualties with untreated soft tissue wounds? I have assumed that 20 per cent will develop life-threatening infection.¹¹

Lower Extremity: Exsanguination from traumatic amputations and/or arterial wounds is the most common immediate cause of death in this group of casualties.⁵ Vietnam casualty statistics show that about 20 per cent of casualties who survive to reach medical treatment facilities had severe injuries; i.e., open comminuted fractures, arterial injuries, nerve injuries, or amputations.⁵ I have assumed that one-third of these casualties will succumb during the first 24 hours if no treatment is given. As with upper extremity wounds, the major unanswered question is what happens to the large number of casualties with untreated soft tissue wounds. I have assumed that 20 per cent develop life-threatening infection during the first week after wounding.

Multiple: About one-half of these casualties have com-

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combined head and thoracic wounds. Most of the remainder have combined injuries of the chest and abdomen. The majority of these casualties will die during the first 24 hours.

Mortality in Untreated Combat Casualties

Table III summarizes the preceding analysis. The model predicts that about 20 percent of the casualties will die within the first hour and that 32 per cent will have died by the end of the first day. The 20 per cent figure is quite comparable to the statistic for those killed in action during the Vietnam War. It is difficult to find a comparable figure for mortality at one day. Clearly, the predicted mortality rate after the first few hours may be totally erroneous. In extremes of climate, mortality might reach 40–50 per cent at 24 hours and 70–80 per cent at one week.

Fig. 1 is a graphic representation of Table III. I have assumed that it is possible to categorize the causes of death into several basic groups such as hemorrhage, craniocerebral trauma, and sepsis. Hemorrhage and craniocerebral trauma are the dominant causes of death during the first day; sepsis is the dominant cause thereafter.

TABLE V
MORTALITY—6-HOUR EVACUATION DELAY

	KIA (26%)	DOW (4%)
CNS Trauma	82	14
Hemorrhage	143	3
Other	36	13*
Total	261	30

*Including 6 septic shock, 3 respiratory distress syndrome, 2 renal failure

TABLE VI
MORTALITY—24-HOUR EVACUATION DELAY

	KIA (32%)	DOW (8%)
CNS Trauma	97	18
Hemorrhage	175	2
Other	46	35*
Total	318	55

*Including 15 septic shock, 10 respiratory distress syndrome, 5 renal failure, 1 stress ulceration

Effect of Combat Casualty Care

How would treatment alter this dismal outcome? Let us assume a Vietnam War scenario with surviving casualties reaching medical treatment facilities within one to two hours after wounding. From Table III, we can predict that about 20 per cent of those initially wounded will have died before evacuation. Since these casualties died before they reached medical treatment facilities, they would be classified as having been killed in action (KIA). The individual causes of death, which were used to construct Table IV, have been combined into three broad categories: Central Nervous System (CNS) Trauma, Hem-

orrhage, and Other, which are shown in Table IV. Seventy-one casualties are assumed to have died of CNS trauma and 98 from hemorrhage. The category labeled "other" consists of 13 casualties with injuries to their respiratory tract (laryngeal, tracheal, sucking chest wounds, etc.), and 13 casualties with multiple combined fatal wounds. Table IV confirms what is intuitively obvious—hemorrhage is the greatest threat to life on the battlefield.

How many of the casualties that reach medical treatment facilities ultimately die? It is this group of casualties—the died of wounds (DOW)—that has traditionally been the most commonly used criterion for assessing the adequacy of combat casualty care. The important papers of Hardaway⁴ and Arnold and Cutting¹ have much information regarding this population. Given a level of medical expertise equal to that seen during the Vietnam war, one can predict that about two per cent of casualties surviving to reach medical treatment facilities, or 16 casualties from our model population of 1,000, will die of wounds. Arnold and Cutting found that craniocerebral trauma was the cause of death in 42.5 per cent, hemorrhagic shock in 23.9 per cent, septic shock in 11.7 per cent, and pulmonary insufficiency in 6.1 per cent. Burns, renal failure, spinal cord injuries, and several other conditions were the causes of death in the remaining 16 per cent of those who died of wounds. These data applied to our model population of 1,000 are also shown in Table IV, but now the "Other" category refers to casualties who have died of sepsis and/or multiple organ failure. The most important cause of death in casualties surviving to reach medical treatment facilities is craniocerebral trauma. The four casualties listed as having died of wounds in the hemorrhage category may be assumed to have exsanguinated from surgically uncontrollable bleeding from liver, pelvic, or pulmonary wounds. Coagulopathies following multiple transfusions may be expected to contribute to their demise. The immensely greater importance of hemorrhagic shock as a cause of combat mortality (102 casualties) compared with septic shock (two casualties) should not pass unnoticed.

The enormous impact of Vietnam-war-style combat casualty care (rapid evacuation and expert surgical care) is shown in Fig. 2, the shaded area represents the number of lives saved. Can these results be improved? The British experience during the Salalah, Oman, counterinsurgency operation in 1972–1973 suggests that the number of casualties dying on the battlefield can be significantly reduced by a further decrease in evacuation time.¹⁰ Although the population studied was small, less than 10 per cent of Omani casualties were killed in action. The evacuation time was about one-half the Vietnam average. In essence, the shaded area in Fig. 2 has been displaced to the left of the diagram.

Conceivably, some of those dying of wounds in the CNS, trauma and hemorrhage categories could be salvaged by improved surgical and/or anesthetic management. If one assumes that those killed in action are beyond salvage, the only remaining potential for decreasing mortality is to develop improved treatment regimens for the

TABLE VII
SITE OF EXSANGUINATION

16	Heart/ascending aorta
13	Lung/pulmonary artery
10	Liver
10	Multiple abdominal sites including mesentery, spleen, kidney, and pelvis
9	Great vessels of the thorax, principally the aorta
*9	Arteries in the lower extremity
8	Great vessels of the abdomen, especially the aorta and vena cava
*6	Amputations of the lower extremity
*3	Carotid artery (five additional carotid injuries combined with wounds of the trachea)
*2	Upper extremity amputations
*2	Arteries of the upper extremity, especially the axillary and brachial
10	Multiple sites in the chest, abdomen, and extremities

* Possibility exists for temporary control of bleeding by first aid measures

casualties dying of sepsis and multiple organ failure. Sepsis and multiple organ failure have traditionally been areas of intense interest among physicians and surgeons, and a large fraction of trauma research funding is spent on these problems and the related problems of surgical metabolism. Nevertheless, even if complete salvage of these casualties proved possible, there would only be a modest impact on combat casualty care, because sepsis and/or multiple organ failure currently cause only two to three per cent of the total combat mortality.

Effect of Delayed Evacuation

It is generally believed that the great speed with which American casualties were evacuated from the Vietnam battlefield is unlikely to be repeated in future conflicts. There is grave concern that, especially in a major European war, the lack of air superiority will prevent helicopter evacuation and, because of the paucity of tracked vehicles dedicated to medical evacuation, casualties will remain on the battlefield for many hours after wounding. The deleterious effects of delayed evacuation to treatment facilities capable of providing definitive surgical care can be estimated from Table III. A six-hour delay would increase the number of casualties who died before reaching medical treatment facilities to 26 per cent, and a 24-hour delay would mean that at least 32 per cent of the total number of casualties will have died. The latter figure no doubt underestimates the actual attrition, because the role of climatic exposure has not been considered.

Table V shows the causes of death, predicted from Table III, which might be observed when evacuation from the battlefield to treatment facilities capable of providing definitive care is delayed for six hours. The major cause of death continues to be exsanguination. The number who die of wounds in medical treatment facilities will increase because casualties will be more ill on arrival. A figure of four per cent has been assumed—this being similar to what was observed during 1944–1945 in Europe.⁷ Sepsis and multiple organ failure are an increasingly important cause of death.

Table VI shows the causes of death, predicted from Table III, which might be observed when evacuation from the battlefield to treatment facilities capable of providing definitive surgical care is delayed for 24 hours. The major cause of death continues to be exsanguination, but some of the casualties with abdominal wounds dying of shock might also be expected to have fulminating peritonitis. It is assumed that eight per cent of evacuated casualties die of their wounds. This figure is based upon the World War I statistic that, with an average evacuation time of 18 hours, eight per cent of the casualties died.⁷ At best this figure is a guess, but there can be no doubt that sepsis and multiple organ failure will become increasingly important causes of morbidity and mortality. I have tried to indicate the relative importance of the target organs in the casualties who succumb to sepsis and multiple organ failure.

The preceding analysis suggests that a delay in evacuation, although it increases the number of casualties who die of wounds, has its main effect by increasing the number of casualties who die on the battlefield. It should be noted that Tables IV, V, and VI are based on the assumption that no care other than wound dressing and administration of an analgesic took place on the battlefield. In a scenario corresponding to the experience in Vietnam, this assumption is at least partially correct, because more often than not casualties were evacuated directly to medical treatment facilities capable of providing definitive care. The battalion aid station/clearing company echelon was bypassed and the medic's prime responsibility was to arrange evacuation. If evacuation were to be delayed six hours, and certainly if the delay approached 24 hours, it is inconceivable that no care would be rendered in the field. In fact, as will be shown, a major improvement in combat casualty care can occur only if field medical care—forward resuscitation by the medic and battalion surgeon—is prompt, intensive and, most important, effective.

Consider the situation that existed in Vietnam: the only hope for a major improvement in salvage would have been to improve the care of casualties who were listed as killed in action. Would this have been possible? It is instructive to review the anatomical lesions which resulted in the 98 deaths from exsanguination in the model population shown in Table IV. These data, based upon autopsy results from 500 consecutive casualties who were killed in action, are shown in Table VII.⁵ A cursory inspection reveals that a substantial number of these casualties (marked by *) exsanguinated from arterial wounds at sites where simple first aid measures (direct pressure, pressure on the cognate artery, or application of a tourniquet) might have been expected to control hemorrhage at least temporarily. Further data from the Vietnam war point to the same conclusion.⁵ In 277 casualties who were killed in action by hemorrhage from arterial wounds, no less than 38 per cent had a site at which hemorrhage could have been controlled at least temporarily by simple first aid measures.

When evacuation is delayed, many of the casualties succumb to hemorrhage from arterial injuries which are

not immediately fatal. These deaths may be preventable. This impression is confirmed by an additional study from the Vietnam war.⁵ In a series of 224 casualties with arterial wounds who survived to reach medical treatment facilities, no less than 87 per cent were bleeding from sites easily accessible to control by first aid measures. If military antishock trousers (MAST) were available for use by the medic in the field, the potential for salvage would be even greater, since about one-third of the casualties with abdominal wounds calculated to die during the first day after wounding (20 of 61) have wounds in the lower half of the abdomen. Bleeding from sites in the pelvis and lower abdomen should be susceptible to tamponade by MAST.

The suggestion that a potential exists to increase the salvage of combat casualties who otherwise would be listed as killed in action is not original. Maughon⁸ appreciated that there was a considerable potential for increased salvage of combat casualties on the Vietnam battlefield and made a plea to that effect some 12 years ago. More recently, Rocko *et al*¹⁵ have come to a similar conclusion regarding civilian trauma victims. They have made the important observation that, in respect to control of arterial hemorrhage, the recommended first aid practice is inadequate. What is important about a "pressure dressing" is not the dressing per se, but the pressure. A big bulky dressing by itself will not stop bleeding. Of course, the majority of combat casualties have wounds of such severity that nothing short of immediate operative intervention offers any chance of survival. Furthermore, simply controlling hemorrhage may be insufficient to achieve short-term survival in a casualty who has lost 50-60 per cent of his blood volume. Nevertheless, without control of hemorrhage, other aspects of resuscitation are likely to prove futile.

It is not unrealistic to hope that intensive resuscitation in the field might save 25-30 of the casualties listed in Table V as being killed in action due to hemorrhage. Furthermore, attention to other aspects of the American College of Surgeons' advanced trauma life support program—intubation of casualties with craniocerebral trauma, evacuation of tension pneumothoraces, etc.—might double the salvage, so that the percentage dying prior to evacuation would fall from 26 per cent to 22 or even 20 per cent. The goal should be to do everything necessary to keep the casualty alive until he can be evacuated to the next treatment echelon. Paradoxically, success in this endeavor might increase the number of casualties who die of wounds—more moribund casualties will reach medical treatment facilities alive. Conversely, intensive resuscitation might improve the status of so many casualties that the overall incidence of life-threatening postinjury sequelae is reduced.

Discussion

The majority of combat deaths occur on the battlefield before evacuation to medical treatment facilities occurs. For every casualty who dies of wounds in a medical treatment facility, as many as nine have already died.

Given optimal circumstances, such as in Vietnam, neither the application of sophisticated technologies designed to improve survival of traumatized patients in surgical intensive care wards or operating rooms, nor greater success in managing the common causes of postoperative death—sepsis and multiple organ failure—will have a significant impact on improving combat casualty care.

To bring about a significant improvement in the salvage of casualties given optimal circumstances such as in Vietnam, or to prevent a drastic deterioration in care given a tactical situation in which evacuation is seriously hindered, there must be a renewed emphasis on field medical care in combat casualty care. The appropriately trained combat medic and battalion surgeon should be able to assume a position of importance equal to that of the combat surgeon.

There are several areas in which combat casualty care research can and should make a contribution toward improving forward resuscitation. First and foremost, there is a need to improve the field management of hemorrhage. The combination of simple first aid measures plus infusion of an oxygen-carrying solution and/or use of pharmacologic interventions designed to optimize cardiac output (antishock drugs) might be life saving in a surprisingly large number of casualties. The Army is sponsoring or is conducting research designed to meet some of these needs, but much remains to be done, such as finding a simple and rapid means of gaining access to the circulation of the casualty in shock. Although seemingly mundane, the practical consequences of having assured intravenous access on the battlefield cannot be exaggerated.

Second, because craniocerebral trauma is the most important cause of mortality in the definitive care setting, there is a need to determine whether or not the salvage among these casualties can (or perhaps should) be improved. Much more needs to be known about the pathophysiology of head injuries.

Third, not only is there a need to improve the management of sepsis in the combat support hospital setting, but there is a need to know whether or not the appalling toll taken by sepsis whenever definitive care is delayed can be ameliorated by steps that are within the capability of those rendering treatment on the battlefield. Is there a justification for the medic to administer antibiotics to the wounded soldier on the battlefield? Similarly, what is the natural course of untreated soft-tissue wounds? Do all such wounds need extensive surgical debridement or can lesser but equally effective measures be substituted?

Finally, there is a need to keep an open mind toward unorthodox and even outlandishly futuristic proposals for combat casualty care. It has been suggested that the best way to handle seriously wounded casualties would be to immediately place them in a rocket which would accelerate to 99.9 per cent of the speed of light.¹⁶ The casualties would return to Earth only after conditions become more civilized. Even though this might take years, the casualties' conditions would not worsen because relativistic effects would ensure that the elapsed time measured by a clock in the rocket would be only a few seconds. Although no doubt meant to be taken farcically, this

scenario really shows a brilliant insight into what needs to be done when the casualty cannot be evacuated. We need to be able to slow the casualty's biological clock. We need to be able to decrease the casualty's metabolic rate to a tiny fraction of normal without damaging organs and constituent cells. Cardiopulmonary function could then be suspended until it became possible to institute definitive treatment. Is it foolish to expect that practitioners of combat casualty care in the 21st century will employ techniques for inducing a state of suspended animation?

Summary

This paper attempts to identify areas of combat casualty care in which research may decrease the mortality of conventional land warfare. The analysis was carried out by constructing a hypothetical population of combat casualties from existing casualty statistics. Clinical judgment was used to decide mortality as a function of time after wounding, assuming either treatment was not given or treatment was given after a delay corresponding to the time required to evacuate the casualty from the battlefield to a treatment facility capable of providing definitive care.

Two evacuation scenarios are of special interest: rapid (~one hour) and delayed (~one day). The analysis predicts that 195 of 1,000 casualties will die before rapid evacuation has been completed, and 16 additional casualties will subsequently die of their wounds. Given delayed evacuation, 318 of 1,000 casualties will die before evacuation has been completed, and 55 additional casualties will die of wounds.

Exsanguination causes about 50 per cent of all deaths. Trauma to the central nervous system causes about one-third of all deaths. Sepsis/multiple organ failure causes less than 10 per cent of all deaths. For there to be a significant improvement in the results of combat casualty care, there must be a renewed emphasis on field medical care, with special attention to the management of hemorrhage on the battlefield.

References

- ¹ Arnold, K. and Cutting, R. T.: Causes of death in United States military personnel hospitalized in Vietnam. *Milit. Med.*, 143:161-164, 1978.
- ² Beebe, G. W. and DeBakey, M. E.: *Battle Casualties*. Springfield, Ill., Charles C Thomas, 192, p. 199.
- ³ Blair, V. P.: Relation of the early care to the final outcome of major face wounds in war surgery. *Milit. Surg.*, 92:12-17, 1943.
- ⁴ Hardaway, R. M.: Vietnam wound analysis. *J. Trauma*, 18:635-643, 1978.
- ⁵ Joint Technical Coordinating Group for Munitions Effectiveness. Evaluation of wound data and munitions effectiveness in Vietnam (WDMEV). Vol. I of III Vols. Final Report, December 1970, Alexandria, Va., Defense Technical Information Center (AD879516).
- ⁶ Kelly, J. F. (Ed.): Management of war injuries to the jaw and related structures. Dental Sciences Dept., Naval Medical Research Institutes, Bureau of Medicine and Surgery (Dept. of the Navy), Washington, D.C., US Government Printing Office, 1977; pp. 66-67.
- ⁷ Lada, J. (Ed.): Medical statistics in World War II. Washington, D.C., Office of the Surgeon General, Dept. of the Army, 1975.
- ⁸ Maughon, J. S.: An inquiry into the nature of wounds resulting in killed in action in Vietnam. *Milit. Med.*, 135:8-13, 1970.
- ⁹ Mierowsky, A. M. (Ed.): Neurological surgery of trauma. Washington, D.C., Office of the Surgeon General, Dept. of the Army, 1965.
- ¹⁰ Melsom, M. A., Farran, M. D., and Volkers, R. C.: Battle casualties. *Ann. R. Coll. Surg. Engl.*, 56:289-303, 1975.
- ¹¹ Mendelson, J. A. and Glover, J. L.: Sphere and shell fragment wounds of soft tissue: Experimental study. *J. Trauma*, 7:889-914, 1967.
- ¹² Office of the Surgeon General, Dept. of the Army: Surgery in World War II—Thoracic Surgery. Vol. I, Washington, D.C., US Government Printing Office, 1963, p. 64.
- ¹³ ———: Surgery in World War II—General Surgery. Vol. II, Washington, D.C., US Government Printing Office, 1955; p. 331.
- ¹⁴ Reister, F. A.: Battle casualties and medical statistics, US Army experience in the Korean War. Washington, D.C., Office of the Surgeon General, Dept. of the Army, 1973.
- ¹⁵ Rocko, J. M., Tischler, C., and Swan, K. G.: Exsanguination in public—a preventable death. *J. Trauma*, 22:635, 1982.
- ¹⁶ Snyder, K. N.: Personal communication.
- ¹⁷ Trunkey, D. D.: Overview of trauma. *Surg. Clin. North. Am.*, 62: 3-8, 1982.