Chapter 5

THE MANAGEMENT OF BALLISTIC WOUNDS OF SOFT TISSUE

RONALD F. BELLAMY, M.D., FACS* AND RUSS ZAJTCHUK, M.D., FACS**

'Colonel, United States Army: *Acting Chief, Department of Surgery, Walter Reed Army Medical Center. Washington. D.C. 20307-5001*
""Colonel, United States Army; Deputy Commander, Walter Reed Army Medical Center, Washington, D.C. 20307-5001

INTRODUCTION

Much of a combat surgeon's time is spent in managing wounds of the *soft tissue*s (that is, skin, fat, and skeletal muscle). Penetrating wounds account for more than 90% of combat casualties. These wounds not only have a soft-tissue component, but in about 50% of them, the **soft-tissue** wound **is** the major treatment problem. Since soft-tissue wounds rarely constitute immediate life-threatening problems, however, medical officers are responsible for assuring that potentially fatal injuries to viscera deep to the soft tissue are recognized and receive appropriate priority for care. **A** surgeon's preoccupation with soft-tissuewound management may lead to inadequate care for more seriously wounded casualties. Certainly airway, breathing, and circulatory problems must be resolved before any thought is given to soft-tissue wounds.

Deciding whether a soft-tissue wound is associated with a more serious visceral injury is not always easy. Wounds of entrance can be misleading and give no indication of the projectile's trajectory through tissue (Figures 5-1, 5-2, and 5-3). Figure 5-1 shows a wound of entrance that was made by an M43 bullet in a casualty's proximal right lateral thigh. Physical **ex**amination of the abdomen suggested an intraabdominal injury, however, which was confirmed **at** laparotomy: **surgeons** resected the casualty's ***se***verely damaged small bowel (Figure **5-2).** The roentgenogram (Figure 5-3) had failed to show the bullet's expected lodgment in the casualty's right hip. Having entered the right thigh, the bullet struck and fractured the right iliac crest, was deflected across the abdomen through the small bowel, then struck and fractured the left iliac crest, and finally was **deflected** into the casualty's left thigh, where it came to rest.

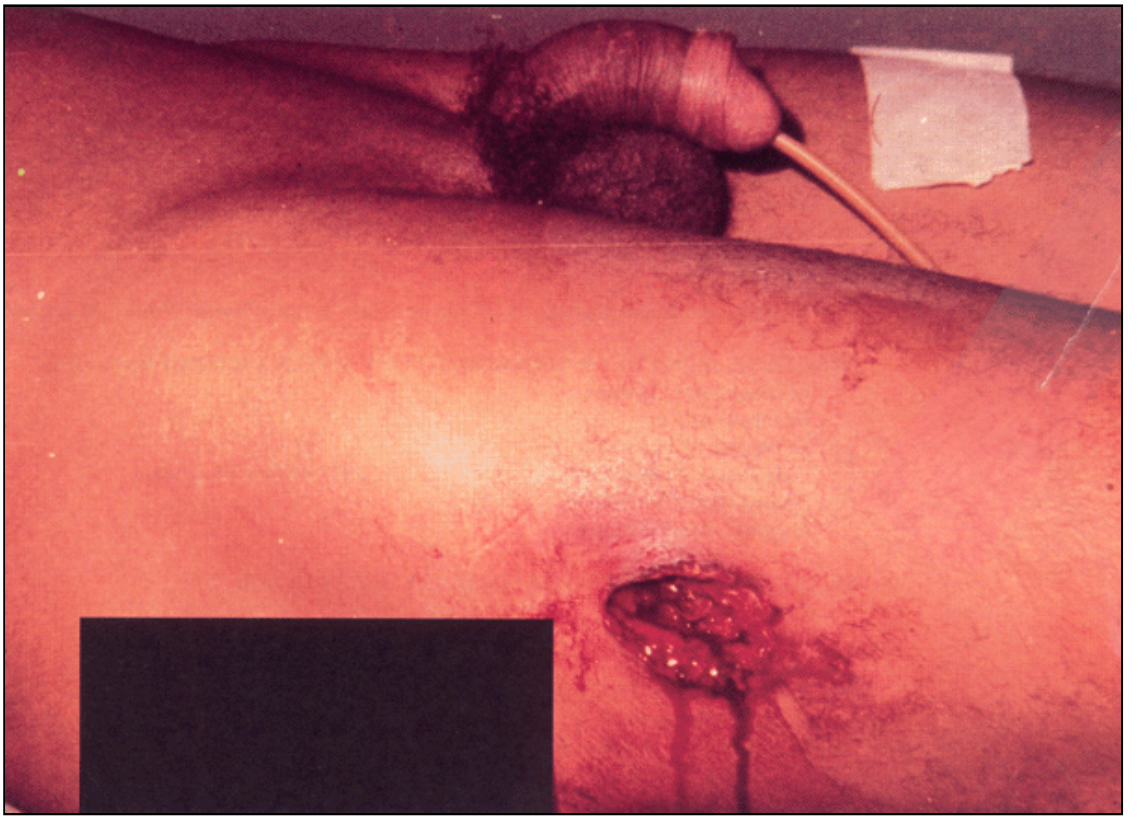

Fig. 5-1. The wound of entrance in this casualty's right hip was made by an M43 ball.
Source: Wound Dara and Munitions Effectiveness Team

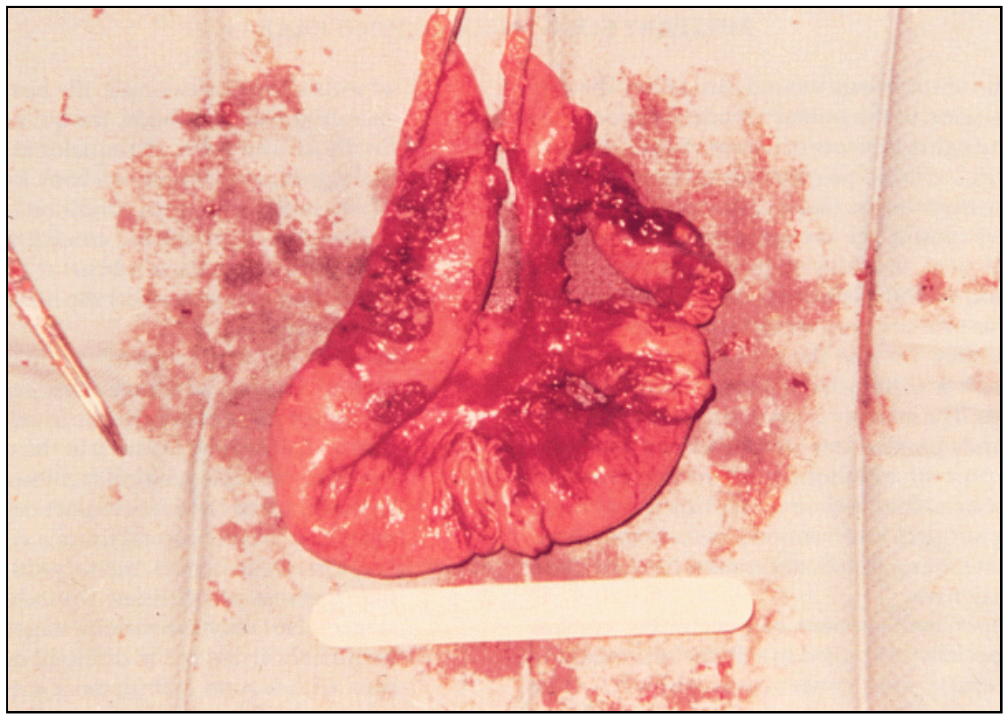

Fig. 5-2. The casualty's damaged small intestine was resected at laparotomy
Source: Wound Data and Munitions Effectiveness Team

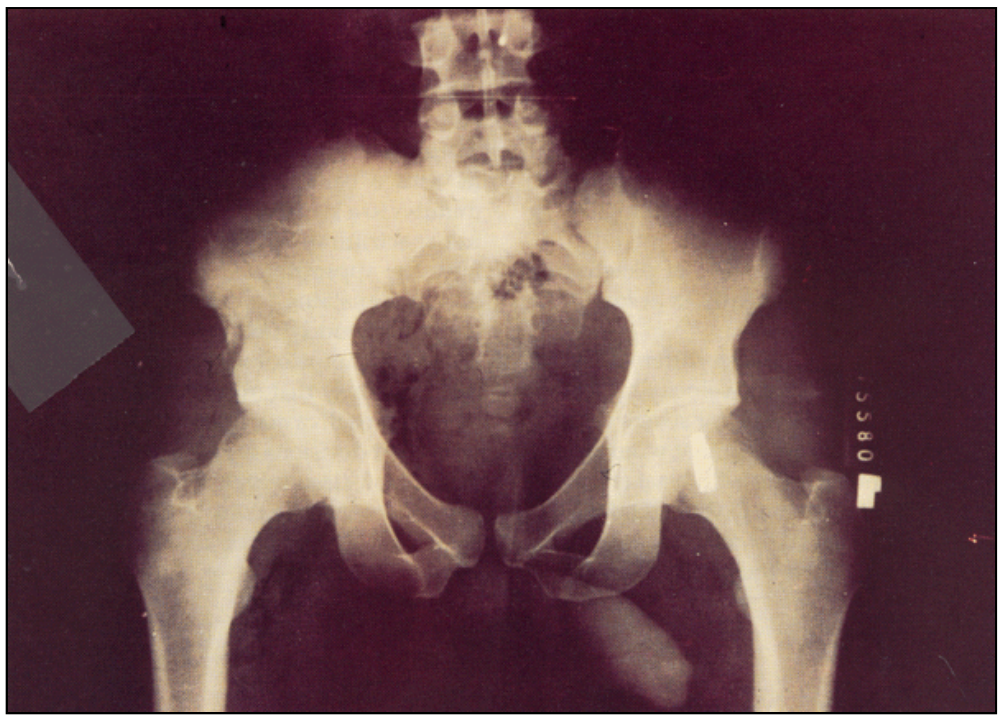

Fig. 5-3. The roentgenogram of the pelvis of the casualty shown in Figure 5-1 shows the bullet present in the soft tissue lateral to the *left* femoral head. Fractures of both iliac bones are apparent.
Source: Wound Data and Munitions Effectiveness Team

MILITARY SOFT-TISSUE WOUND CARE

The goals of soft-tissue wound care follow directly from the mission of the military medical services: to maintain the fighting power of the command. In many cases, medical officers can meet this goal by minimizing the noneffectiveness that a casualty's penetrating injuries have caused. As always in military medicine, the desired goal is achieved in the context of the greatest good for the greatest number. Infections and sepsis are the primary reasons that soft-tissue wounds fail to heal; they are also the primary reasons that a casualty with a soft-tissue wound continues to be noneffective. The military approach to managing soft-tissue wounds emphasizes simple, staged interventions that prevent infections most of the time and return most casualties to their units. Costly and time-consuming surgical interventions, even if they have perfect cosmetic and functional results, have no place during a major war.

The proper management of penetrating combat trauma, especially as applied to soft-tissue wounds, is a controversial and contentious subject. The invasiveness of military surgical management during recent wars (for example, extensive debridement) has been compared unfavorably to the the civilian approach.' Some have argued that traditional military management of penetrating trauma is unscientific and may even be dangerous, making the surgeon "a menace, doing more harm with his treatment than was done by the bullet."[2] Such criticisms usually focus upon the nature of the bullet, but there are other factors that need to be considered in understanding the nature of, and rationale for, the military approach to managing penetrating ballistic wounds.

Military and civilian management of penetrating soft-tissue trauma differ significantly in at least four ways:

- The potential for *tissue damage* differs because the weapons differ. High-velocity bullet wounds, and the potential for severe tissue damage, and multiple fragment wounds, which frequently coexist with blast effects, are much more frequent in the military.
- The potential for *wound contamination* differs since it depends upon the geographical site of wounding (for example, the heavily manured Flanders fields, the quite-clean South African veldt, the squalor at Stalingrad, or a downtown bank lobby). The casualty's physical condition (a cold, malnourished, exhausted combat soldier or a well-fed urban bank robber) at the time of wounding may also effect the likelihood of wound sepsis.
- The *timeliness of care* depends upon myriad tactical, logistic, administrative, and temporal (when mass casualties are involved) factors that severely constrain the capabilities of deployable medical facilities on a battlefield, but have less impact on a well-equipped urban hospital trauma center.
- Except in rare instances, military surgical management of soft-tissue wounds is staged. Not only are various stages of treatment carried out at different echelons of care, with wound debridement at the third echelon, and wound closure at the fourth, but different medical personnel treat the casualty at each stage.

In view of these distinctions, appropriate wound management for victims of penetrating trauma may be very different in military and civilian settings. For example, the wound management selected for an urban criminal who sustains a perforating soft-tissue wound of the thigh made by a 9-mm pistol bullet while he robs a bank, and who is transferred within 20 minutes to a trauma hospital capable of providing the full spectrum of needed care, may be very different from the appropriate wound management for a soldier who sustains a similar wound of the thigh while lying in a muddy field, and who stays in the muddy field for 12 hours before being evacuated to a hospital that is so busy that only 5 minutes can be allotted to caring for each casualty. Medical officers must understand that decisions regarding the optimum management of soft-tissue wounds sustained on the battlefield depend upon more than just the physical characteristics of the projectile or the biophysics of projectile-tissue interaction.

THE CONTAMINATED WOUND

The development of doctrine regarding the military surgeon's soft-tissue wound management (that is, staged intervention, debridement, delayed primary closure, and so forth) reflects the hard-won knowledge that battlefield wounds are probably *contaminated* (foreign material has been introduced into the wound), are likely to become *infected* (one or more pathogens has become established and is proliferating inside the

wound), and that the major threat to the casualty's recovery is *sepsis* (pyogenic and other pathogens in the wound have invaded the surrounding tissues and are elaborating their toxic substances). The medical outcome of casualties with ballistic wounds depends upon the balance between the type and amount of bacteria that contaminate the wound, the casualty's ability to resist infection, and the extent to which that resistance has been impaired.

The Mechanics of Wound Contamination

Exactly how wounds in human casualties become contaminated can only be inferred from experimental models. Unsterile bullets and fragments are undoubtedly major sources of contamination. They contaminate the wound tract with bacteria and other foreign material that they bring in as they penetrate through the casualty's clothing and skin. The aspiration that can occur during temporary cavitation contributes less significantly to the total contamination of the wound, but has been the source of considerable speculation (Figure 5-4).

During the late 1940s, researchers at Edgewood Arsenal showed that *(a)* foreign material was aspirated into their gelatin targets during the process of temporary cavitation, and *(b)* this foreign material lined the radial fissures that extended from the permanent cavity in the gelatin. This demonstration has provided a physical model explaining the propensity of battlefield wounds to become infected.[3] Theoretically, surgically excising the lining of the permanent cavity could sterilize the wound.

But if this experimental model is valid for soft-tissue wounds, the biophysical responses of gelatin and skeletal muscle to temporary cavitation must be similar. One of these researchers' most impressive findings is foreign material 3 4 inches away from the gelatin's permanent cavity. If the equivalent depth of contamination occurs in skeletal muscle, truly radical excision would be required to decontaminate a skeletal-muscle wound.

Another researcher at the Armed Forces Institute of Pathology during the 1980s investigated the extent to which the depths of contamination around the permanent cavity in skeletal muscle are equivalent to the findings in gelatin. After attaching a packet containing a solution of fluorescein to each target at its aiming point, the researcher shot M193 balls fired by an M16A1 assault rifle into 20%-gelatin blocks and the buttocks of freshly sacrificed 80–100-kg swine from a distance of about 10 m. Then he opened each target's permanent cavity transversely and illuminated it with ultraviolet light. The morphologies of the fluorescein-contaminated permanent cavities are very different. In gelatin (Figure 5-5), numerous fluorescein-contaminated fissures, some as long as 10 cm, radiate from the permanent cavity, while the contaminating fluorescence in skeletal muscle (Figure **5-6)** is not seen beyond 1 cm from the permanent cavity. Much of the partially detached muscle within the permanent cavity was contaminated, however. The therapeutic implications of this demonstration are (a) for all contamination to be removed, wound excision need not be carried deeper than **1** cm in muscle, but *(b)* all tissue within the permanent cavity should be removed.[4]

The Type and Magnitude of Bacterial Contamination

The relationship between bacterial counts and the likelihood of wound infection has been studied extensively in animal models. Researchers who injected *Staphylococcus aureus* or *Escherichia coli* into the subcutaneous tissue of guinea pigs failed to find clinically apparent infection when the inoculum contained fewer than 10^6 organisms, but found that infection was likely if the inoculum contained more than 10^7 bacteria.[5] Although this and similar studies conclude that clinically apparent infection will not develop unless the number of bacteria per unit volume of injured tissue exceeds a critical value, this seemingly clear-cut observation needs to be qualified. Certain organisms (such as group A beta-hemolytic streptococci) are especially virulent and can cause wound infection when present in much smaller numbers than 10^5 organisms per gram. Furthermore, criteria have not been established for typical battlefield contamination with obligate anaerobes and mixed aerobic-, anaerobic-, and polymicrobial bacterial contamination.

Experiments that correlate bacterial counts with the likelihood of infection suggest that it is the magnitude of bacterial contamination, not the elapsed time after wound contamination, that most importantly determines the likelihood of infection in an untreated wound. Thus, a wound that contains 10^5 aerobic organisms per gram of wounded tissue can be expected to heal without clinical infection, whether or not the wound is treated during or after an interval such as Paul L. Frederich's 6-hour golden period (which was discussed in Chapter Three). The host's normal resistance will eradicate the organisms. And conversely, this line of reasoning holds that a wound contaminated with 10^7 bacteria per gram of wounded tissue probably will become infected unless it is treated before the golden period has elapsed. The practical clinical problem is

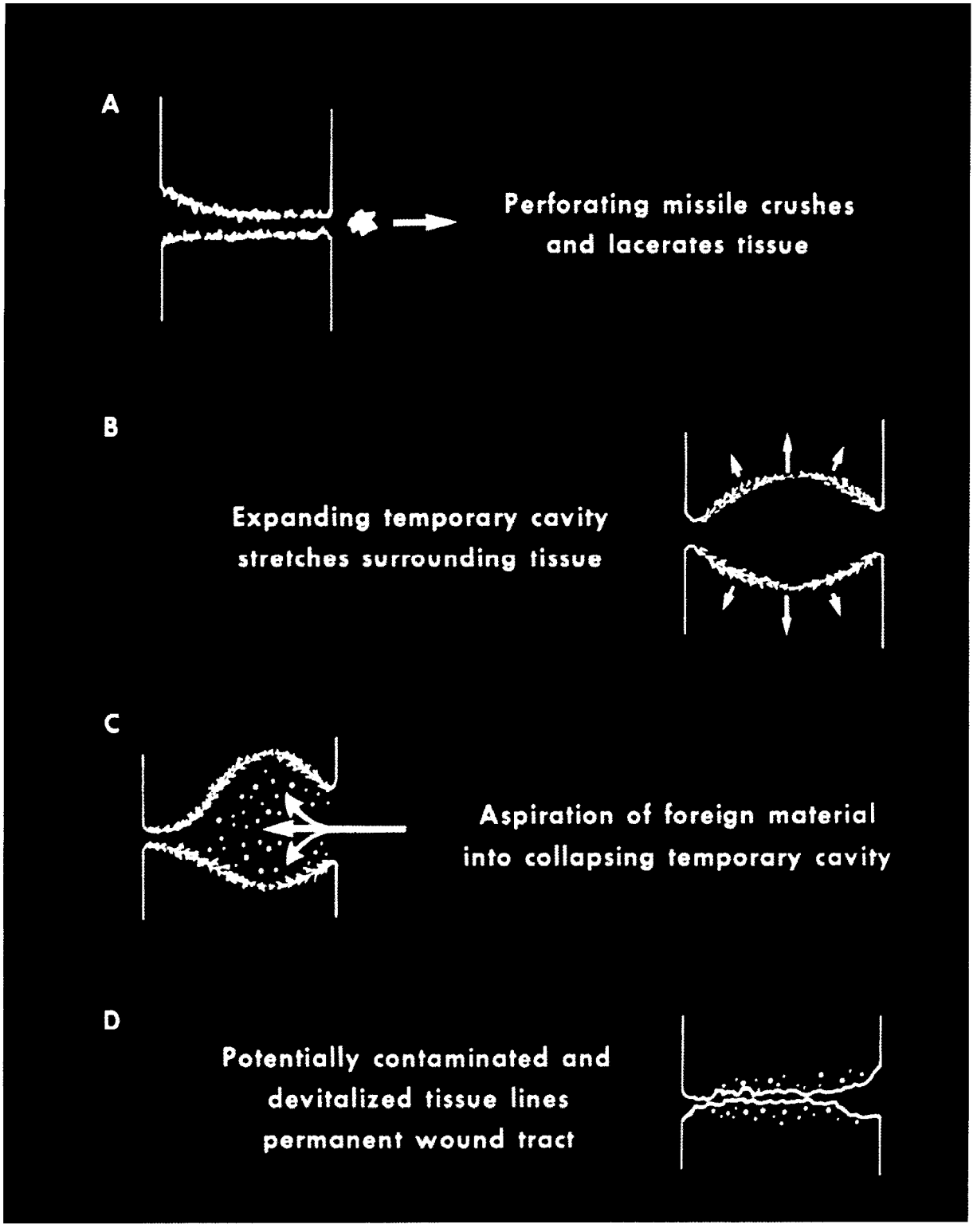

Fig. 5-4. The schematic diagram shows a mechanism by which temporary cavitation can contaminate a wound tract.

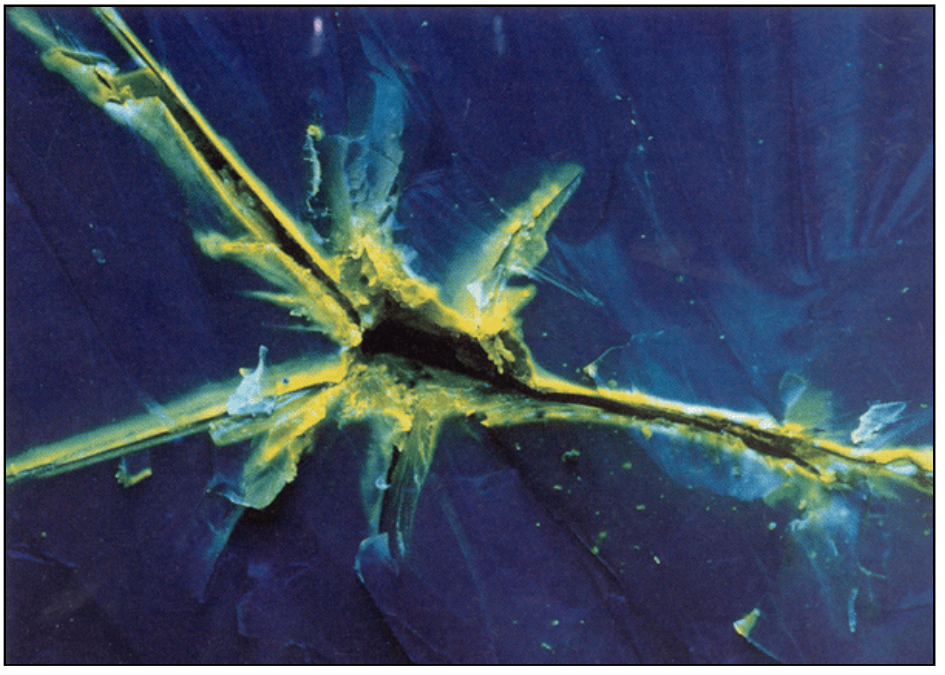

Fig. 5-5. **An** M193 ball has been fired through a packet of fluorescein dye into a gelatin block. The cracks in the gelatin made by temporary cavitation fluoresce, demonstrating the extent of "wound" contamination.

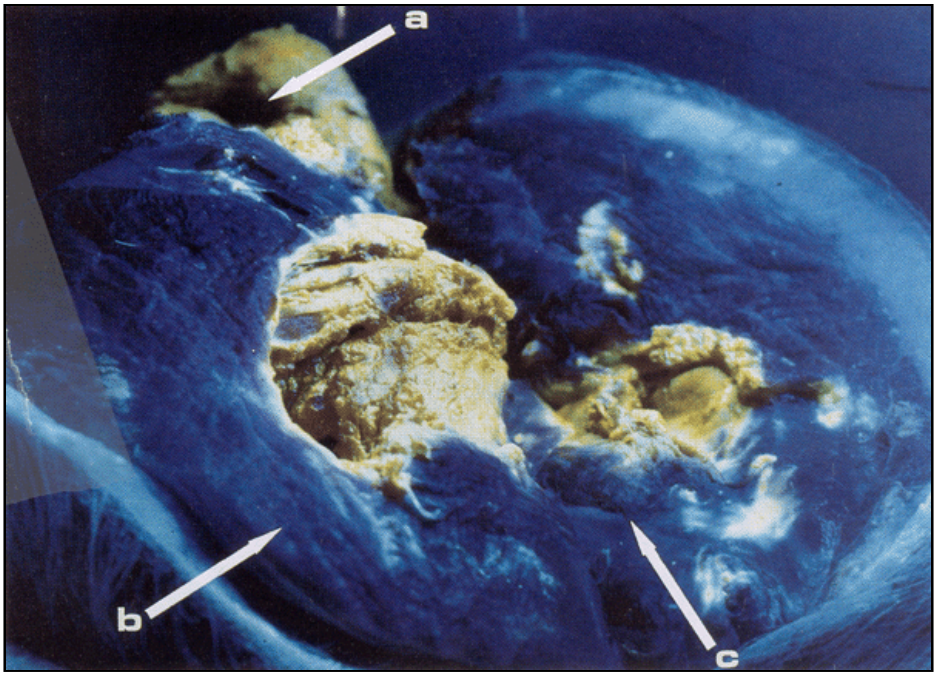

Fig. 5-6. An M193 ball has been fired through a packet of fluorescein dye into a swine's thigh. The thigh has been transected at right angles to the permanent cavity; *"a"* is a piece of detached muscle that was found in the permanent cavity; *"b"* and "c" are the uprange and downrange portions of the thigh. The tissue within and immediately adjacent to the permanent cavity shows **intense fluorescence, but there** is **no evidence of the "contaminated" radial fissures that were found in gelatin.**

that we have no way of knowing the actual duration of the golden period. In Frederich's experiments, it probably only indicated the time that was required for invasive sepsis to become apparent to him—the time after which local surgical measures to eliminate contamination would have had no effect on the infection's progression. The first goal of soft-tissue wound management is to reduce the bacterial count below the level that the casualty's resistance can normally eradicate.

The relationship between bacterial counts and infection in closed, contaminated wounds does not necessarily apply to either *(a)* open wounds or *(b)* contaminated **wuunds** that are **first** left **open and then** are closed days later. Frederich knew that open wounds can resist invasive sepsis (that develops from simple bacterial contamination) better than closed wounds can.

Experimental data elegantly confirm this clinical observation: Open soft-tissue wounds that were contaminated with bacteria at the time of wounding can safely be closed after several days have elapsed (Figure 5-7).[6] In the same study, researchers found that wounds that had been left open and then were contaminated at the time of delayed closure were at greater risk of becoming septic than wounds that had been closed initially and then were contaminated several days later. The reason for this difference remains unclear, but the obvious message is to avoid fresh contamination of wounds at the time delayed primary closure is performed. Studies such as this provide experimental support for the policy of performing delayed primary closure of contaminated wounds after 4 days.

Much clinical evidence supports the importance of bacterial counts as a determinant of the time to close soft-tissue wounds. Late in World War I, some French surgeons adopted the practice of culturing bacteria **from open** ballistic wuunds **immediately after** initial wound surgery. When the culture was inspected several days later, if fewer than five colonies of streptococci were present, the surgeons surgically closed the wound; otherwise, it was left open to close by secondary intent. Using this criterion, these surgeons obtained sepsis-free wound-healing in 90% of their cases, compared with 35% when they ignored the bacterial count.[7] More recently, surgeons demonstrated that only when the exudate from an open wound contained more than 10^6 organisms per milliliter was a clinically important infection likely.[8]

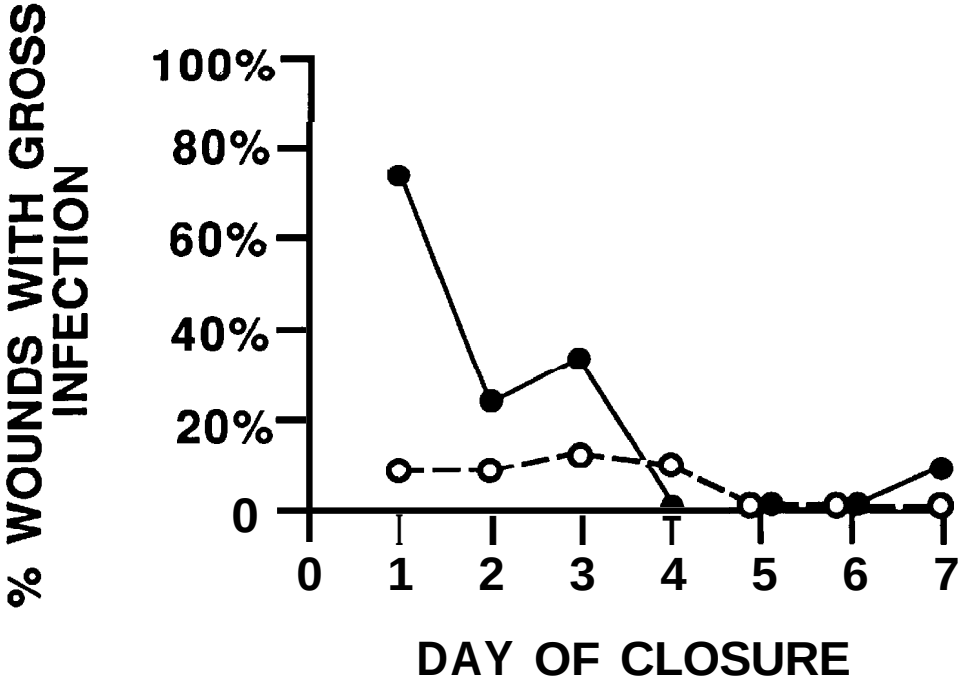

Fig. 5-7. The relationship between the time a wound was closed is plotted against the likelihood of sepsis-free healing. Wounds **that wcrc contaminated at of wounding (•); clean control wounds (○).**
Source: Drawn from data contained in Table 1 **in reference 6**

FACTORS THAT CAN MODIFY A CASUALTY'S ABILITY TO RESIST INFECTION

Many factors can modify the likelihood that a soft-tissue wound will become infected. Because a penetrating wound ruptures the cutaneous barrier, conditions within the wound (that is, *local* factors), probably rival the magnitude of contamination as determinants of wound infection. The casualty's physical condition at the time of wounding (that is, *systemic* factors) have a impact and tend to bc less important, because most combat casualties, at least initially, are not compromised hosts. Some surgical techniques also introduce the possibility that a wound will become infected, and the natural process of necrosis produces an environment that favors infection.

The vast and complicated subjects of the inflammatory and specific immune responses, their dependence upon phagocytic cells, the complement system, circulating antibodies, and cellular immunity are not unique to military medicine, **and** therefore are best **left** for specialized texts such as Howard and Simmons's *Surgical Infectious Diseases.'*

Factors Within the Wound **Tract**

Soil and Other Foreign Bodies. The permanent cavities of penetrating combat wounds can contain an incredible variety of foreign bodies. In addition to the obvious bullets or fragments, wounds can also contain bits of the casualty's clothing, vegetation, wood, and mud (Figure 5-8). Large objects—such as a tank tread or even parts of nearby casualties' bodies—can become imbedded in a wound.

Surgeons do not remove the penetrating projectile from the casualty's body during surgery 50% of casualties. Bullets and other unsterile contaminants not only cause significant wound contamination, they also potentiate additional infection by preventing tissue-to-tissue apposition. Normal host defenses can eradicate bacteria clustered in the interspace between the foreign body and the surrounding tissue, but only with difficulty. Yet this mechanism may be a less important potentiator of infection than soil.

Although previously suspected to be implicated in reducing a host's resistance to infection, soil's role in causing infections was not shown until 1974.'" In an experimental wound-infection model, skin on the backs of guinea pigs was incised and then contaminated with a known inoculum of Staphylococcus *aureus* and 5 mg of sterile soil, then closed and assessed for infection 4 days later. Researchers found that as few as 100 bacteria *per wound* could cause gross infection in the soil-contaminated animals. Eighty percent of the soil-contaminated animals developed infections, but none of the control animals (whose wounds were contaminated with bacteria but not with soil) did. The investigators found an active factor concentrated in the organic constituents of clay and the soils found in swamps, but which was absent in sand. They also showed that the soil-potentiating factor *(a)* interfered with normal leukocytic phagocytosis of bacteria, *(b)* impaired non-specific bactericidal activity in serum, and *(c)* possibly inactivated antibiotics.''

Soils differ in their ability to potentiate infection. **A** study pcrformcd during War supported this view. Researchers tested soils from ten different

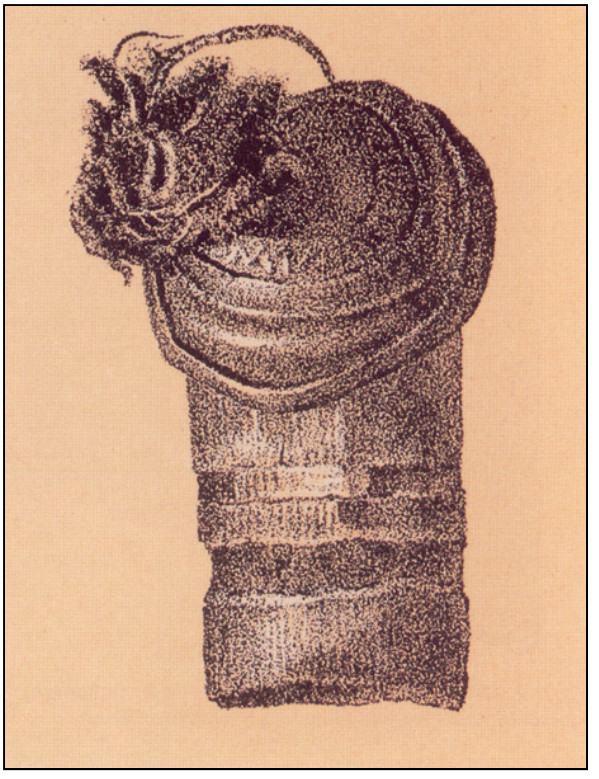

Fig. 5-8. **As** this bullet penetrated the **body** of a Civil War casualty, it carried with it **a** button and a piece of the soldier's uniform.
Source: Plate **39,** number 4151, in reference 14

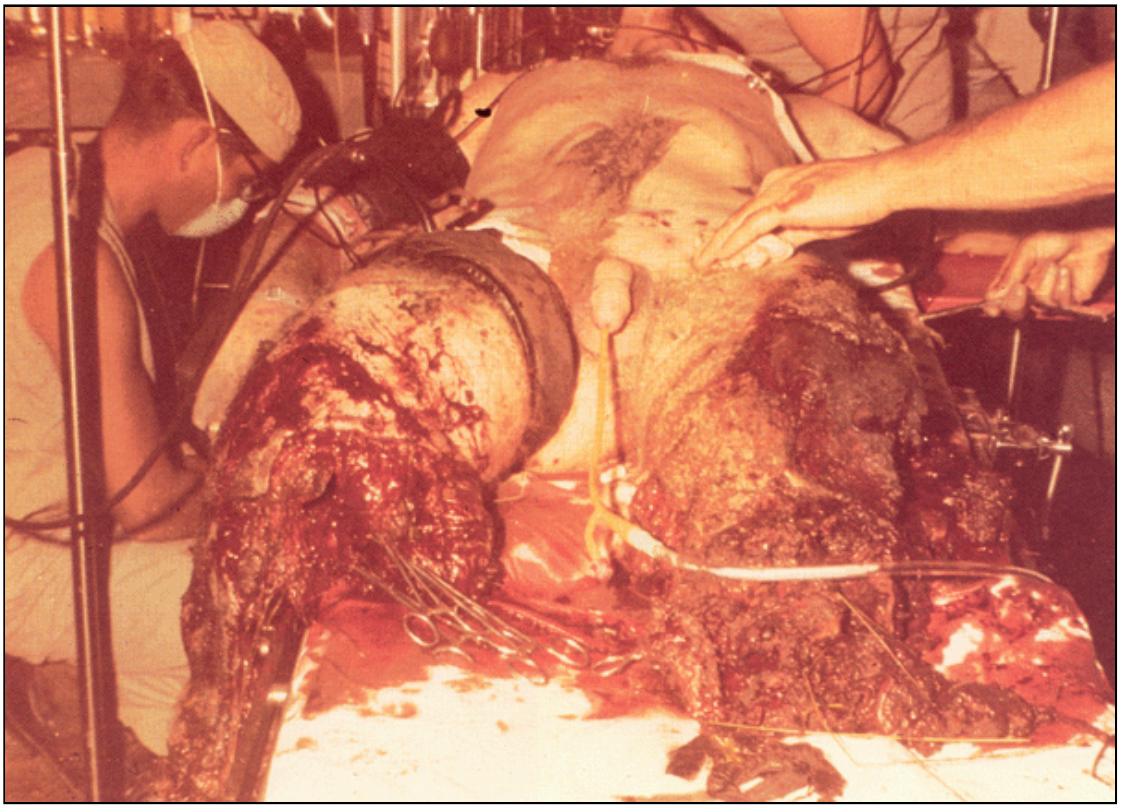

Fig. 5-9. Massive bilateral above-the-knee amputations. Note the straw and dirt that have contaminated the wounds.
Source: Wound Data and Munitions Effectiveness Team

areas of Vietnam for their ability to cause infection in an animal soft-tissue wound model. Of fifty-six animals whose wounds were contaminated with sandy soil, eighteen (32%) died of sepsis. Forty-three (90%) of forty-eight animals whose wounds were contaminated with mud or swamp soil **died.** Interestingly, similar bacterial flora were cultured from the wounds of both groups (for example, all samples contained pathogenic that the size **of** the bacterial inoculum and the virulence of the bacteria were not the only infection-producing factors.[12] This study does not mention, but its results are consistent with, the existence of an active infection-potentiating factor in soil.

An infection-potentiating factor in some soil probably explains why battlefield wounds are so much more prone than civilian penetrating traumas are to sepsis. Soldiers wounded in the battlefields of World War I were particularly at risk:

> Many **of** the patients lie in trenches until the darkness of night allows of their removal: Their clothes are infiltrated with mud, while the same shell which has caused the wound often brings down **the** side of **the trench** and **the injured** limb may be covered with soil. Again the fragment of shell is commonly fouled with soil.[13]

A subsurface detonation of random-fragmentation shells and buried antipersonnel mines will probably massively contaminate wounds (Figure 5-9). But an improved-fragmentation munition detonating in air will cause much less wound contamination. The existence of an infection-potentiating factor in soil provides an additional rationale for surgeons to excise the tissue around a wound's permanent cavity.

The potential that civilian gunshot wounds will become contaminated has recently been emphasized.[15] Interestingly, however, the one experimental study that addressed the role of foreign bodies found that there was no apparent relationship between their presence and wound sepsis,[16] probably because the contaminating conditions are so different.

Hematomas and Extravasated Blood. Blood is an excellent substrate **fnr** bacterial **growth Expanding** hematomas can impair the casualty's defenses against

infection by causing extraluminal compression of blood vessels, and therefore causing tissue ischemia. Experimental animals with bacterial contaminations of their soft-tissue hematomas had a mortality rate of 75%; animals that had either a hematoma or bacterial contamination but not both had a mortality rate of 0%. Researchers do not yet understand why infections in hematomas and extravasated blood are so markedly deleterious.[17] One possibility is that oxygen-derived free radicals form. Given conditions that probably exist in damaged muscle, the reduction of molecular oxygen, which normally results in the formation of water, can potentially generate hydrogen peroxide (H_2O_2) and two spccics of cxtrcmcly rcactivc frcc radi cals: *(a)* the superoxide anion radical ($O_2^{-}\bullet$) and *(b)* the hydroxyl radical (OH*), which is highly destructive of tissue and normally not present in the body. In the presence of ferrous iron, hydrogen peroxide and the superoxide anion radical can interact by means of the Haber-Weiss reaction to produce the hydroxyl radical:

$$O_2^{-}\bullet + H_2O_2 \xrightarrow{Fe^{++}} O_2 + OH^- + OH-$$

The proteolysis of hemoglobin located outside blood vessels can liberate large amounts of ferrous iron. Thus, the Haber-Weiss reaction could potentially be very active in damaged tissue that contains blood clots. Furthermore, the conversion of hemoglobin and myoglobin to methemoglobin and damaged muscle will produce large amounts of $O_2^{-}\bullet$. These reactions will lead to the formation of pathological amounts of the hydroxyl radical and may decrease the casualty's ability to resist infection.[18]

Dead, Injured, and Ischemic Skeletal Muscle. One of the principal lessons of the history of the surgery of soft tissue is that dead, injured, or ischemic skeletal muscle can potentiate wound infection. In a standard guinea pig wound-infection model, wounds that contained detached pieces of skeletal muscle contained ten times more S. *aureus* than control wounds did after about 4 days." Devitalized tissue impairs the bactericidal function of leukocytes, probably because the partial pressure of oxygen is low in dying tissue, and oxygen is needed for normal leukocyte function. Devitalized tissue also depresses leukocyte function *in vitro,* in the absence of bacteria, demonstrating that other factors are involved.

Muscle need not be dead to potentiate infection. Experimental studies have also shown that crushed muscle (an injury similar to that caused by the stretch of temporary cavitation) will decrease by two orders of magnitude the bacterial inoculum necessary to cause a clinically apparent infection in an animal model.[20] Studies like this provide the scientific basis for excising contused muscle from the zone of extravasation in a heavily contaminated wound.

The Casualty's Systemic Factors

Circulatory Shock. Casualties who have only soft-tissue infrequently suffer shock. Sume experimental and clinical studies show that circulatory shock during and immediately after wound contamination increases the likelihood that infection will develop, but the relationship is hard to demonstrate because the numbers are few. When shock does occur in casualties with soft-tissue wounds, visceral injuries—especially of the abdomen—are common. In these situations, the potential for bacterial seeding from the intraabdominal focus to distant sites of soft-tissue injury increases.

Malnutrition, Exhaustion, and Cold. Historically, the concomitants of septic combat wounds are malnutrition, exhaustion, and prolonged cxposurc to cold such as might typify a winter campaign." A surgeon wrote during World War I:

> The man is wounded and simultaneously inoculated with organisms [that] immediately fasten upon any dead tissue. The safety of the patient depends for the time being on his own ability to resist; and if he is collapsed from loss of much blood and is wet, cold, and starving, his leucocyte [*sic*] defense is enfeebled or absent.[22]

The casualty's resistance will clearly be impaired in such circumstances, but delayed treatment and the prevailing filthy battlefield conditions probably also contribute.

Compromised Leukocyte Response in Unconventional Warfare. Many assume that vesicant gases—such as mustard—used in chemical warfare will increase the likelihood of soft-tissue wound infections by a direct toxic effect or the systemic effect of depressing the number and action of circulating leukocytes. But the chemical warfare in World War I provides no support for this commonly held view. The nearly 900-page official World War I medical history does not mention such a synergistic interaction.[23] The very low incidence of combined injuries may partially explain the lack of such an effect; fewer than 10% of the chemical casualties had penetrating injuries as well.

While data showing a relationship between chemical agents and wound sepsis are hard to find, plentiful clinical and experimental data suggest that irradiation

adversely affects soft-tissue wound healing. Most researchers believe that an otherwise nonlethal dose of systemic irradiation (that is, less than 150 centigrays) will increase the probability of dangerous wound sepsis, although the magnitude of this possible effect remains ill defined. The presence of an open wound will increase the lethality of a given dose of irradiation, an effect that is not mediated by local infection. Early wound closure (within 2 days after a combined injury was sustained) reduced lethality from 60% to 10% in experimental animals.[24] These findings have important implications for surgeons who are managing casualties with combined injuries. Current doctrine calls for delayed primary closure of open combat wounds to be performed 4–6 days after wounding, but closing the wounds during that time period did not reduce lethality in experimental animals.

Iatrogenic Factors

Certain aspects of surgical technique increase the likelihood that soft-tissue wounds will become infected.

Dead Space. A surgeon may create a dead space (that is, a void within the wound) when excising skeletal muscle during debridement. Researchers have shown that *(a)* a wound with a dead space is twice as likely to become infected than a wound that has been opened by simple incision, *(b)* the bacterial count in a dead space increases by 100-fold, and *(c)* suturing the dead space closed did not alter the likelihood of wound infection. In fact, partial closure (for example, if a suture line dehisced) was four times more likely to become infected than a wound with a simple dead space.[25] Studies like this show that if iatrogenic dead spaces have been created, immediate closure of excised penetrating battlefield wounds will be generally unsatisfactory.

Drains. When the permanent cavity is long and difficult to expose, military surgeons have occasionally treated such wounds by simply inserting a drain along the wound tract. The usual rationale for inserting a drain is to reduce the potential for infection by assuring blood drainage. But if drains promote egress of unwanted material from the wound, they also can allow undesirable material to enter. In fact, experimental studies show that drains, regardless of their composition, actually promote wound sepsis when no fluid to be drained has collected.[26] Studies such as this suggest that not only is a drain placed through a perforating wound not an adequate substitute for debridement, but also that the drain itself can introduce bacterial contamination.

Suture Material. During most past wars, military surgery has been performed using silk and gut sutures. But sutures made of such materials may actually potentiate wound sepsis. Synthetic monofilament sutures are clearly superior and should be used in lieu of the older sutures.[27]

Dead Soft Tissue Within Combat Wounds

Military surgeons frequently justify their intervention into penetrating soft-tissue wounds as the need to remove dead or devitalized tissue to prevent infection from developing. While ballistic wounds frequently do contain dead muscle, this rationale is usually overemphasized. More often than not tissue consists of only a narrow layer that lines the permanent cavity. Two mechanisms cause muscle death: *(a)* the projectile cuts and stretches the tissue (*primary* injury), and *(b)* the vascular system is compromised either by injury to a major vessel or by compartment syndrome from a hematoma or edema (*secondary* injury). While the devitalized tissue caused by the projectile usually includes just the tissue immediately surrounding the permanent cavity, the devitalization caused by secondary injury can be extensive.

Injury to a Major Vessel. An ever-present potential exists for primary muscle injury to be exacerbated—and in fact greatly exceeded—by secondary injury if the muscle's local blood supply is disturbed either by the projectile itself or during surgery. Some muscle groups are at considerable risk of sustaining secondary injury, because the anatomy of their vascular beds features only one—or at most, several—major intramuscular vessels. Military surgeons must be aware of this potential for iatrogenic injury, and know the distribution and arrangement of the gross and intramuscular arterial supply to extremity muscles.

Compartment Syndrome. Muscles that are confined by bone and fascia in rigid anatomical compartments are at risk for secondary injury if they are subjected to a pathological increase in pressure. Intracompartmental pressure greater than 40–50 mm Hg will stop arterial flow and will cause irreversible muscle death if it persists for more than 6–8 clinical syndrome that results when tension compresses muscles invested by an osteofascial envelope—the compartment *syndrome*—is more commonly seen with blunt trauma, but bleeding within a closed compartment may cause an identical problem in penetrating injuries. Although the compartment syndrome is most commonly seen following injuries to the calf, it is also seen in the gluteal muscles—the largest muscle mass in the body and a common site for gas gangrene.[29]

Some surgeons during World War I understood

that secondary mechanisms involving vascular Compromise could cause muscle necrosis:

> [T]ake care to relieve all tension locally so as to allow of early re-establishment of the circulation. This may explain the advantage of "delayed primary suture" over "primary suture" in gunshot wounds.[30]

While neither the absolute amount of dead tissue that usually surrounds the permanent cavity of a soft tissue wound nor the relative importance of the two muscle-killing mechanisms is certain, useful data from both animal and human studies exist.

Because they *(a)* studied a large animal population and *(b)* carefully quantified the histologically dead tissue around the permanent cavities, Mendelson and Glover's landmark 1967 study is uniquely important.[16] (This is one of the most significant papers ever published on wound ballistics.) They found that the permanent cavities made by spheres and fragments were usually lined by not more than 5 mm of histologically degenerate muscle, which they measured 3–7 days after the animals were wounded. This tissue constitutes the innermost layer of the zone of extravasation. (It would be interesting to know the proportion of the zone of extravasation in relation to the total mass of damaged tissue, but since Mendelson and Glover were specifically investigating the dimensions of the permanent cavity, they did not mention the dimensions of the zone of extravasation. However, simply calculating the volumes of a hypothetical but typical wound suggests that the amount of dead tissue must be relatively small. If a 10-cm-long permanent cavity with a diameter of 1 cm and a 0.5-cm layer of dead tissue is surrounded by an additional 1-cm layer of extravasation, the volume of dead tissue is about 23 ml and the volume of the zone of extravasation is about 117 ml. Thus, only about 20% of the damaged tissue is actually dead.)

This study further suggests that, since few animals had both vascular damage and large hematomas with grossly necrotic muscle, the mechanism of death was more frequently primary injury than secondary. Furthermore, in most of these animals, clostridial infection appears to have caused the additional necrosis (beyond that attributable to the projectile). In this experimental protocol, major vascular injury was infrequent.

The researchers demonstrated that the thickness of the microscopic zone of dead tissue depended upon the projectiles' impact velocity: the higher the velocity, the larger the temporary cavity, and therefore the more extensive the primary injury. In their sample, 93% of the high-velocity (averaging 960 m/s) spheres and 77% of the high-velocity fragments caused muscle necrosis 5 mm or more thick. In contrast, only 62% of low-velocity (averaging 406 m/s) spheres and 46% of the low-velocity fragments caused muscle necrosis deeper than 5 mm.

In an earlier and less-elaborate investigation, which also spared the animals' femoral arteries, Mendelson and Glover studied skeletal-muscle death around the permanent cavities that resulted from a variety of bullet wounds and concluded: "There was no gross zone of necrosis beyond the permanent wound tract in any of the animals examined."[31] They also found that muscle death (usually associated with clostridial infection) can be massive when the arterial supply is compromised. These animal studies demonstrate conclusively that massive death of skeletal muscle is uncommon in the absence of an arterial injury.

Human-Tissue Experimentation. Although military surgeons have debrided great quantities of wounded skeletal muscle, only one published study, which was performed during the Korean War, seems to have examined the excised tissue for histological signs of death.[32] Researchers took samples from soft-tissue wounds in twelve American casualties whose wounds were caused by unspecified weapons. Surgeons had excised sixty samples of muscle that they considered to be of questionable viability from the wounds. About one-third of the samples contained muscle that was completely dead. The rest of the samples contained enough viable myocytes to have permitted the muscle to repair itself.

Whether or not battlefield surgeons can recognize dead skeletal muscle was then, and remains today, contentious. This study tested the clinical criteria for skeletal-muscle debridement — the four Cs:

- Color — dark red, red, pale, pink (normal)
- Consistency — mushy, stringy, soft, firm (normal)
- *Contractility* — its absence or presence (normal)
- *Circulation* — the absence or presence (normal) of bleeding

by correlating the surgeon's assessment at the time of debridement, performed 3–8 hours after the casualty was wounded, with the pathologist's microscopic determination of the degree of damage (Tables 5-1 through 5-4).

All criteria except color achieved statistical significance (using the chi-square test) as predictors of muscle necrosis. Nevertheless, using the four Cs raises problems of both sensitivity and specificity for the surgeon. For example, Table 5-1 shows that while all muscle that was likely to be dead had abnormal color, so did all the samples with histologically normal muscle. Similarly, Table 5-2 shows that three-fourths of the

TABLE 5-1

ASSESSMENT OF FOUR Cs: COLOR

5 x 4 contingency tables were constructed and statistical significance at the 5% level was determined by calculating a chi square statistic.

$p > 0.05$

Degree of Muscle Damage	Color (pink = normal)			
	dark red	red	pale	pink
Minimal	2	5	0	0
Slight	5	4	1	1
Moderate	14	5	0	1
Marked	5	4	0	1
Complete	8	4	0	0

Source: Reference 32

TABLE 5-2

ASSESSMENT OF FOUR Cs: CIRCULATION

5 x 4 were constructed and statistical significance at the 5% level was determined by calculating a chi square statistic.

$p < 0.001$

Degree of Muscle Damage	Circulation (+2/+3 = normal)			
	+1	+2	+3	+4
Minimal	2	3	0	2
Slight	3	3	5	0
Moderate	5	9	5	1
Marked	6	3	1	0
Complete	9	3	0	0

Source: Reference 32

TABLE 5-3

ASSESSMENT OF FOUR Cs: CONSISTENCY

5 x 4 contingency tables were constructed and statistical significance at the 5% level was determined by calculating a chi square statistic.

$p < 0.001$

Degree of Muscle Damage	Consistency (firm = normal)			
	mushy	stringy	soft	firm
Minimal	1	2	0	4
Slight	0	0	7	4
Moderate	3	1	8	8
Marked	3	3	3	1
Complete	7	4	1	0

Source: Reference 32

TABLE 5-4

ASSESSMENT OF FOUR Cs: CONTRACTILITY

5 x 4 contingency tables were constructed and statistical significance at the 5% level was determined by calculating a chi square statistic.

$p < 0.01$

Degree of Muscle Damage	Contractility (+2/+3 = normal)			
	+1	+2	+3	+4
Minimal	4	1	1	1
Slight	7	1	2	1
Moderate	9	7	2	2
Marked	9	1	0	0
Complete	12	0	0	0

Source: Reference 32

samples had little if any capillary hleeding, hut this defect was found with nearly equal frequency in both histologically dead and histologically normal muscle. Table 5-3 shows that muscle consistency was the most reliable predictor of tissue death. Contractility, in Table 5-4, was a less valuable predictor because, although contractility was diagnostic of viability when present, it was frequently absent—even in supposedly normal tissue. (A multiple-regression analysis might have improved the predictive value of the four Cs, but the original experimental data required to perform the test now no longer exist.)

Different and innovative approaches to assessing tissue viability—such as supravital staining with various dyes, measuring the transmembrane cellular electrical potential, and using ultraviolet light to demonstrate tissue-fluorescence—have been described, but none have been accepted, probably because they are impracticable. The four Cs, although perhaps lacking as predictors, certainly are practicable.

That Pvedisyose to Skeletal-Muscle Death. Many military surgeons including Raspall José Trueta have wondered whether the high prevalence of gas gangrene in wounds of the lower extremities depended upon factors other than just large muscle masses located close to the ground. In Trueta's view, ischemia of a major muscle mass was a necessary—although not a sufficient-condition for gas gangrene to develop, and the propensity for this to occur in human lower extremities depended at least partly upon the vascular anatomy of certain muscle groups.[33]

Surgeons in World War I also thought that the vascular anatomy of certain muscle groups in the lower extremities might predispose to the development of ischemic myonecrosis when vascular injury occurred. The first study to suggest this was performed near the end of World War I. During autopsies on casualties who had died of causes unrelated to injuries of the extremities, surgeons determined vascular anatomy in extremities by injecting the appropriate major artery with a barium contrast material. Muscle groups of interest were then excised and roentgenographed. On the basis of their vascular anatomy, muscle groups were divided into three categories: *(a)* those whose blood supply was derived from many sources with numerous potential anastomoses present (the deltoid, pectoralis major, and biceps brachialis muscles); *(b)* those whose blood supply was derived from one or several arteries with few potential anastomoses (the gluteus maximus, rectus femoris, and the strap muscles of the thigh); and *(c)* those whose blood supply was derived from only one artery (the gracilis and gastrocnemius muscles). The that

> vascular distribution in muscle has a most important bearing on the origin and spread of gas infection in muscle. In consequence of injury to the arterial supply, the mass of muscle to which the damaged artery is distributed in classes *(b)* and *(c)* either completely dies or becomes devitalized to a marked extent. . . . When . . . the bacilli of gas gangrene invade muscle in this condition they find a nidus suitable for their growth.[30]

The arteries that these surgeons studied were not major, named arteries such as the superficial femoral or the profunda femoral, but smaller, intramuscular vessels. A study performed at the end of World War II extended this observation. Researchers determined the vascularization of muscles in rabbit extremities that were similar in shape to the strap muscles of the human thigh. They injected dye into arteries similar to classes *(b)* and *(c)* above, after interventions such as arterial ligation, partial division of the muscle belly, and experimental gunshot wounds. They found that even small lacerations of the muscle belly—such as a penetrating projectile or surgical debridement might make—caused extensive areas of persistent devascularization (Figure 5-10).

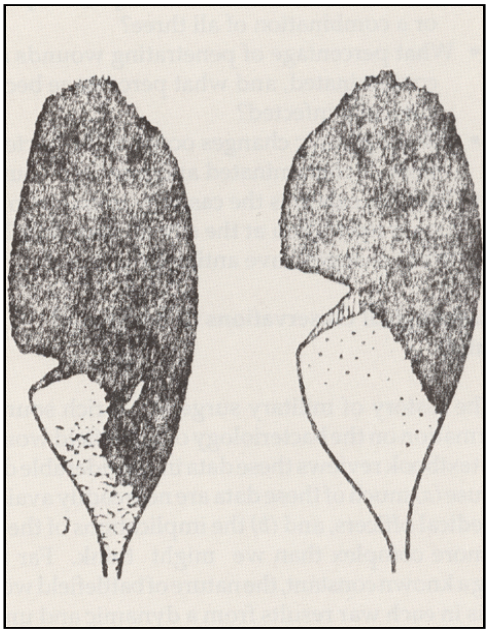

Fig. 5-10. To demonstrate tissue vascularity, a rabbit's tibialis anterior muscle was partly transected before an intraarterial dye was injected. Tissue with a normal blood flow is shown in black. The incision devitalized much of the distal portion of the muscle.
Source: **Figure 3 in reference 34**

Wnunds made in the adductor region nf the thigh (by 3/32-inch steel spheres with impact velocities ranging between 250–1,000 m/s) were usually associated with an area of unstained muscle extending 2–3 mm around the permanent cavity.

> In some cases, however, larger areas showing absence or weakness of staining were produced in the neighbourhood of the missile tract [14 mm by 8 mm]. . . . These effects are evidently due to vascular involvement, for they are too extensive to be ascribed to the direct traumatic injury of the muscular tissue by the missile. This is also shown by the fact that the areas of devascularization are usually quite asymmetrical with regard to the missile wound.[34]

Since the evidence suggested that local vasospasm contributed to producing the vascular defect, the researchers thought that direct vascular injury of intramuscular arteries was not necessary for them to become devascularized.

THE BACTERIOLOGY OF CONTAMINATED PENETRATING WOUNDS

Although many observations on the nature of the bacterial flora found in battlefield wounds have been made during the wars of this century, and our understanding of the bacteriology of penetrating wounds has been extended hy animal experimentation since World War II, some important questions remain imperfectly answered:

- What is the source of bacterial contamination? Is it the host or the battlefield environment, or is it *nosocomial* (that is, hospital-acquired) or a combination of all three?
- What percentage of penetrating wounds are contaminated, and what percentage become clinically infected?
- What dynamic changes occur in the bacterial flora of contaminated and infected wounds?
- To what extent is the casualty immune to his own microflora at the site of wounding?
- What changes have antibiotics caused?

Bacteriological Observations of Battlefield Wounds

The history of military surgery is a rich source of information on the bacteriology of battlefield wounds. This textbook reviews these data in considerable detail because *(a)* much of these data are not readily available to medical officers, and *(b)* the implications of the data are more complex than we might think. Far from being a known constant, the nature of battlefield wound sepsis in each war results from a dynamic and unique interaction among various factors: the climate, the geography, the weaponry, the tactical and operational situation, the medical treatment, and perhaps even the evolving virulence of the pathogens.

World War I—Sir Alexander Fleming. Alexander Fleming, who discovered penicillin, performed some of the earliest and most comprehensive investigations of the bacterial flora in battlefield wounds. He recognized that *(a)* the flora changed over time and *(b)* the bacteria that were important immediately after wounding were absent several weeks later, having been replaced by other organisms. He performed his first study on soft-tissue and bone injuries in late autumn, 1914 (Table 5-5).[35] Several of Fleming's observations require comment:

- All the casualties had open wounds, and the chronic bacterial growth that characterizes open wounds would have been unavoidable. The reduction of the number of casualties over time appears to have been due to the healing of some wounds and the loss to follow-up in other cases.
- Mixed flora were the norm throughout the period of observation, and the organism(s) responsible for the infection is not apparent.
- The Gram-positive anaerobic bacillus *Clostridium perfringens* (known to Fleming as *Bacillus aerogenes capsulatus*) and the streptococci (Fleming did not further specify the class; the type of hemolysis is unknown, but some of wcrc anacrobcs) were by far the most common organisms found, but they became less common as time passed. Other Gram-positive cocci predominated by the end of observation.
- Although clostridia and other anaerobic bacilli such as B. *tetani* were present in most wounds, none of these casualties developed gas gangrene.

Fleming correlated the changes that he observed in the flora with the clinical appearances of the wounds. He described three phases:

- During the first week, a foul-smelling, watery, and usually reddish-brown discharge oozed