Chapter 7

CUTANEOUS TRAUMA AND ITS TREATMENT

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INTRODUCTION

The skin presents itself on the front lines of defense against the outside world, affected by the harsh effects of the environment: high and low humidity and temperature, solar radiation, wind, dirt, arthropods, and infectious agents. The soldier's skin may also be assaulted by nuclear, biological, and chemical agents. Finally, skin on the feet is frequently subjected to frictional forces, which causes blisters. Dermatologists have had a traditional role in caring for skin disease resulting from many of these insults.

With the advent of dermatologic surgery, knowledge and experience in caring for wounded skin has been established. Present requirements for dermatology residency mandate knowledge of principles and practical competence in basic skin surgery, including excisional surgery with simple and layered closures. Knowledge of principles is also mandatory for scar revision, skin grafts, and local cutaneous flaps. It is a natural extension of this expertise for the dermatologist to be involved in caring for traumatic injury to the skin. Most of the dermatologists deployed during Operations Desert Shield and Desert Storm were in general medical officer positions. Those stationed at battalion aid stations were likely to receive and treat soldiers with traumatic skin injuries.

Treatment of cutaneous traumatic injuries draws on knowledge that is well established in dermatologic surgery: anatomy, anesthesia, microbiology, wound healing, surgical technique, dressings, and scar revisions. An approach based on this knowledge will be presented in this chapter. In addition, because of the prevalence of friction blisters in the military, a comprehensive review of this area will be presented.

FRICTION BLISTERS

Friction blisters are believed to be confined to humans, occurring with such frequency that they are considered one of the most common reactions to trauma.[1] Friction blisters are especially important in the military, where they can jeopardize mission performance by decreasing a soldier's work tolerance and increasing manpower loss. Friction blisters are also of significance in athletics, especially distance running, where they adversely affect performance. Finally, this type of blister can provide an important diagnostic clue to a group of inherited diseases called epidermolysis bullosa. This genodermatosis has as a hallmark blisters that are easily induced by friction.

Despite the prevalence of friction blisters, there has been a paucity of scientific study in this area. This deficiency stands in marked contrast to the well-researched classic bullous dermatoses (eg, pemphigoid, pemphigus, and dermatitis herpetiformis). The military has been an important contributor to the present fund of knowledge on friction blisters.

In the military, friction blisters have long been recognized as a significant medical problem.[2] The high incidence of friction blisters at military training centers is especially well documented. It has been estimated that 20% or more of soldiers sustain friction blisters during the early weeks of training.[3] Even experienced and well-conditioned soldiers can have a blister rate approaching 50% as a result of a long march.[4]

Friction blisters of the feet may be a short-lived medical problem, but for soldiers they are not a trivial condition. The treatment of these blisters accounts for a significant loss of time for the medical staff and the troops. The morbidity associated with friction blisters sometimes extends beyond the pain and discomfort of the lesion. One study reported that 84% of the cellulitis treated in a navy recruit population was caused by friction blisters, with an average loss of 8 days per case.[5] Of note, cellulitis composed over 17% of all recruit admissions.

Pathogenesis

To better understand the pathogenesis of friction blisters, it is first necessary to review briefly the superficial anatomy of the skin. The most superficial layer of the skin is the epidermis, consisting of several layers of cells, with the stratum corneum its outermost layer. The next layers are the stratum granulosum, followed by the stratum malpighian, and finally the stratum basale. The stratum basale is the deepest layer of the epidermis and is located adjacent to the dermis.

The histology of a friction blister has a characteristic pattern. One observes necrosis of keratinocytes in the stratum malpighian, which leads to separation of the epidermis. The cleavage plane always shows the roof of the blisters to consist of stratum corneum, stratum granulosum, and some cellular debris. The epidermis below the cleft does not show any significant cellular damage.[6]

A blister is not the only possible endpoint when friction is encountered. If the friction is applied in small amounts over a longer time (weeks to months), then the result is epidermal hypertrophy (thickening of the skin).[2] One example of this endpoint is a callus, which is known to protect against the development of friction blisters. If a significant friction force is applied acutely to areas of the body where the epidermis is not thick, then an abrasion results.[1] As a result, friction blisters typically form on the palms and soles, which have a very thick epidermis.

The most important forces in the formation of a friction blister are the dynamic shear forces. These are believed to be composed of interrelated forces consisting of vertical forces, fore and aft shear, lateral shear, and torque.[7–9] The complexity of the physics of shear force, along with the variability of gait and bone structure of the foot, make it impossible to quantify a specific force required to produce a blister.

Separation of the epidermis is determined not only by the net shear force transferred to the skin surface but also by the frictional characteristics of the skin–environment interface.[2] Knowledge of factors that can influence this frictional force is important in helping to understand and prevent the formation of friction blisters. The surface conditions of the skin influence the development of friction blisters by altering the coefficient of friction between the skin and the boot. Keeping the foot dry has been shown to reduce the formation of friction blisters by lowering the coefficient of friction.[10] Wet skin also lowers the coefficient of friction, decreasing the risk for friction blisters. Moderately moist skin, however, will tend to cause more blisters because the coefficient of friction is increased.[10–12]

Risk Factors

In addition to moderately moist skin, several other risk factors are known to influence friction blister formation. Improperly fitting or poorly designed shoes have long been recognized as a contributing factor. Several studies of basic training soldiers have shown that the majority of blister problems appear in the first 2 to 3 weeks.[13,14] A recent study done with Reserve Officer Training Corps (ROTC) cadets in initial officer basic training revealed less blister formation in officers who frequently ran long distances.[15] This same study showed no advantage to wearing broken-in boots on road marches.

Additional risk factors were identified when studying ROTC cadets at summer camp.[15] Females had a relative risk 1.6-fold greater than that of males. Cadets with a history of blisters in the 2 years before camp had an increased risk of blister formation. Cadets who reported wearing their boots less than 20 hours per week during the 2 weeks immediately before camp had an elevated risk of friction blister formation. One of the conclusions drawn from this study was that prevention of blisters primarily through conditioning the foot with a skin-thickening response was one of the most important steps that could be made in preventing friction blisters.

Another very important risk factor for friction blister formation is socks and their fiber composition.[16] Poorly fitting or worn-out socks have been identified as an important agent in the formation of friction blisters. Of even more significance is the information obtained after studying long-distance running. The results showed that an acrylic fiber sock was associated with fewer and smaller blisters when compared directly with cotton fiber socks. The Thor-lo brand of sock (manufactured by Thorneburg Hosiery Co., Inc., Statesville, N.C.) appears to have been associated with fewer blisters for army-tested soldiers when compared against the standard issued sock: acrylic wicks away moisture from the foot.[17] Socks made of natural fibers like cotton and wool absorb moisture, resulting in a harder, bulkier sock that promotes larger and more frequent blisters. It is important to note that the Thor-lo sock contains higher-density padding where frictional forces are greatest and snug-fitting, low-density padding in the arch area, to allow for better conformity. This differential in sock thickness may also be important in reduced blister formation. All acrylic socks do not possess this type of construction.

Prevention and Treatment

Prevention of friction blister formation is the most important front-line approach in dealing with this common problem.[1] Preventive measures should begin with preenlistment evaluation for congenital or acquired anomalies of the feet and skin as well as assuring adequate fit of shoes. Foot-

care lectures to recruits and recruit commanders' stressing prevention and early recognition of blisters are also important strategies.

Prevention of friction blisters can be divided into two categories: toughening the skin and reducing shear force. Currently, no adequate commercially available topical products exist to harden the skin surface. The use of tincture of benzoin has been reported anecdotally but the inconvenience and the high incidence of contact dermatitis makes this impractical.[3] The development of epidermal hypertrophy through conditioning the skin results in a decreased susceptibility to friction blisters.[15] This approach is very effective when it can be implemented.

Shear force has been decreased with several modalities. Insoles and teflon sprays successfully reduce the shearing effect, but their limited availability and considerable expense make these items impractical for large-scale military use.[3] A study of U.S. Marine Corps recruits showed that the use of an antiperspirant foot powder resulted in fewer friction blisters.[18] This outcome was seen because a dryer foot has a decreased coefficient of friction. Similar results were revealed in a study of U.S. Army soldiers that showed a reduction in friction blisters with the use of antiperspirant solutions.[19] However, the frequent finding of irritant dermatitis (38%) made this therapeutic option impractical.

The most exciting recent advances in the prevention of friction blisters have involved sock studies, as noted above. Acrylic socks with different padding densities have been shown to decrease the number and size of friction blisters in marathon runners and soldiers.[16,17]

The most common approach to the treatment of friction blisters involves first draining the blister with a sterile needle. A doughnut shape of moleskin is then cut to surround the blister, and a cap of moleskin is placed over the blister. Other aspects of treatment include daily cleaning, preferably with hydrogen peroxide, followed by the application of an antibacterial ointment. Surveillance for the development of lymphangitis or cellulitis is important. Finally, if the epidermal roof has been completely lost, then another membrane covering is needed to provide optimal conditions for wound healing. Some of the newer synthetic hydrocolloid dressings (eg, DuoDERM, manufactured by ConvaTec, Princeton, N.J.) or hydrogel dressings (eg, Vigilon, manufactured by C.R. Bard, Inc., Murray Hill, N.J.) are effective options.

CUTANEOUS TRAUMA

Cutaneous trauma treated by a dermatologist is restricted to superficial soft-tissue injuries. Anatomically, all of these lesions have in common disruption of at least the epidermis. The dermis, subcutaneous fat, and superficial fascia can also be injured. Common examples of cutaneous trauma include lacerations, burns, blisters, and traumatic tattoos from blast injuries.

Anatomy

Evaluating a wound for the extent of cutaneous damage and planning for repair rely heavily on a thorough knowledge of the injured area's anatomy. The initial evaluation should consider the possibility of underlying bone or visceral injury. In wounds confined to the skin and subcutaneous tissue, certain areas of the body contain extremely important nerves, arteries, muscles, and other specialized structures. These danger areas will be discussed in detail in this section. Discussion of the orbit, although vital, is beyond the scope of this chapter.

The face contains an abundance of superficially located structures that may be damaged by trauma. The facial nerve exits the stylohyoid foramen at the base of the skull, entering the parotid gland near the angle of the jaw, where it separates into five branches. These branches fan out across the face, ranging from the temple and forehead down to the neck and mandible. The facial nerve supplies motor nerves for the muscles of facial expression. Injury to these branches will give a noticeable deficit to facial movement.[20]

The temporal branch of the facial nerve is the branch most superficially located and easily injured. The nerve exits the parotid gland and traverses the zygomatic arch and temporal region, terminating in the occipitofrontal muscle at the forehead. The nerve is located in the superficial temporalis fascia, which is covered by a thin subcutaneous tissue layer and overlying skin. This thin protective layer is easily breached by a laceration. Once the nerve has entered the occipitofrontal muscle fascia, it arborizes sufficiently to maintain function in the event of a localized injury to the forehead.

The zygomatic and buccal branches of the facial nerve supply motor nerves to the lower eyelid, nose, and lip elevators. These nerves exit the parotid

fascia under a fairly thick cheek fat pad and emerge at the undersurface of the facial muscles they innervate. Injury of the lateral (proximal) third of their route would involve the parotid gland. This would cause extensive loss of function from these branches and possibly others. Injury along the middle third would require a deep penetration into the cheek fat pad. This injury would also threaten Stenson's duct, which traverses the parotid gland inferior to the zygoma before entering the mouth at the buccal mucosa. Localized injury of the distal third of these branches may not yield significant loss of muscle function due to the extensive branching of these nerves.

The marginal mandibular nerve exits the parotid gland and usually runs behind the ramus of the mandible. In some cases, however, it may be lateral and inferior to the ramus, exposing it to injury. The branch then runs protected by the medial surface of the mandible. It reaches the body of the mandible and, along with the facial artery, travels medially and superiorly toward the lateral commissure of the mouth. The nerve arborizes and innervates the muscles around the mouth, especially the orbicularis oris and the downward depressors of the lips. The probable points of injury are the ramus and the body of the mandible. Injury of the body of the mandible could be accompanied by profuse hemorrhage if the facial artery were severed. The cervical branch of the facial nerve innervates the platysma and has little clinical significance.[20]

The sensory innervation of the face is supplied by the trigeminal nerves. The nerves exit foramina of the skull in distinct locations. The ophthalmic branch (V_1) gives rise to the supraorbital and supratrochlear nerves, which provide sensory (afferent) function to the forehead and anterior scalp.

The infraorbital nerve of the maxillary branch (V_2) surfaces at the infraorbital foramen 1 cm below the infraorbital ridge in the midpupillary line. The nerve spreads out to innervate the lateral nose, upper lip, medial cheek, and lower eyelid. The mental nerve arises from the mandibular branch (V_3), passing through the mental foramen of the mandible approximately 1 cm above the jaw line in the same midpupillary line that aligns the supraorbital and infraorbital nerves.

The arterial supply of the face is provided mostly by the facial artery, a branch of the external carotid artery. The artery emerges from under the mandible and travels in a superior and medial direction along the lateral aspect of the mouth. The inferior and superior labial arteries branch medially to supply the lips. These arteries are on the posterior labial surfaces between the submucosa and the orbicularis oris, and they can produce significant hemorrhage. They may bleed from both sides of a laceration. The angular artery continues after the labial arteries branch off the facial artery. It lies along the side of the nose in the nasofacial sulcus and passes into the medial canthal region, where it anastomoses with the dorsal nasal artery. This latter artery is a branch of the internal carotid artery, which exits the side of the nasal bone in the medial canthal region.[20]

The auriculotemporal artery, also a branch of the external carotid artery, progresses in the preauricular fold, branching into the superficial temporal artery. Other arteries travel along with the previously named sensory nerves. The scalp is a rich anastomotic plexus of arteries and veins.

In general, any artery that is transected can be ligated without concern for ischemia of the supplied skin, provided the other anastomotic connections are intact. If a strip of skin is partially avulsed, then the blood supply may be significantly compromised.

The neck contains several vital structures that, if damaged, are beyond the expertise of the dermatologic surgeon. The trachea, thyroid gland, carotid arteries, and jugular vein are located in the anterior neck. Injury to these structures is life threatening. The lateral and posterior neck are relatively free from subcutaneous vital structures. The architecture is defined by large muscles located deep to the skin and subcutaneous tissue. The only important nerve to be aware of in this region is the spinal accessory nerve, located posterior to the upper one third of the sternocleidomastoid muscle. This nerve travels posterocaudally into the trapezius muscle. It is located on the fascia overlying the scalenus muscle. Injury to this nerve would lead to a deficit in abducting the arm laterally and fixing the scapula.

Injuries to the skin and subcutaneous tissues of the trunk that do not penetrate into pleura, peritoneum, or muscle do not approach any significant arteries or nerves; therefore, potential damage is limited. Injury to the extremities must be carefully evaluated, especially in areas of the forearm, hand, and foot. Many arteries, nerves, tendons, and joint spaces are close to the skin surface and are easily injured. Surgeons with special competence, such as orthopedic surgeons, plastic surgeons, or podiatrists, would be logical choices for definitive care of these injuries. Detailed description of this anatomy is beyond the scope of this chapter.

The arms and thighs consist of large areas of skin covering muscle tissue. Except near the elbow and knee, most important structures are well protected by these muscles.

The genitalia have tremendous psychological importance in addition to their structural importance. Injury to the urethra or other deeper structures of the penis or scrotum would best be handled by a urologist or plastic surgeon. Superficial wounds, however, may be treated like those in any other area.

Evaluation and First Aid

In emergent care, basic initial evaluation takes little time but may be lifesaving. Airway patency, breathing, and adequate circulatory system function should be evaluated first. Skin and soft-tissue injury care must be delayed until circulatory volume is adequate and internal injuries have been ruled out. With any significant facial trauma, cervical fracture should be suspected, even in the absence of symptoms.[21] Bleeding should be controlled by pressure until the patient is proved to be free of serious underlying injury. Casualties should be appropriately stabilized and triaged if further care is needed.

The types of wounds that a dermatologist would be most helpful with include abrasions, lacerations of the skin, and traumatic tattoos. Evaluation of these wounds requires a historical account of the injury. The nature of the wound, the amount of contamination that has entered the skin, the amount of delay in reaching care, and first aid rendered since the injury are ascertained. The medical status of the casualty including medications, significant medical problems such as diabetes or bleeding disorders, and tetanus immunization status are determined.

Examination of the wound proceeds with concern for loss of function, depth of injury, and cosmetic impact. Functional considerations include damage to major sensory or motor nerves, arterial supply, and muscle and tendon damage. These injuries would best be managed by a specialist with experience with such injuries. Similarly, injuries deeper than the skin and subcutaneous tissue are beyond the dermatologist's expertise. Highly specialized areas such as eyelids, hands, and genitalia would also be referred if the injury is extensive.[21]

Neurological testing of the surrounding skin may be hampered by the pain of the injury, but it needs to be thoroughly performed and documented before the area is anesthetized for debridement and closure. Knowledge of neuroanatomy is required to predict what motor nerves may be in the injured area. In an emergency, testing with standard pinpricks should be adequate for determining sensory nerve function. Frequently, small cutaneous nerves will be transected by the injury. This finding should be documented before beginning any repair of the skin, and the patient should be reassured that sensory function usually returns. Regional loss of sensory function would mandate further evaluation by an experienced specialist in this form of injury. Nerve repair could then be done immediately, or in 10 to 12 days. Nerve grafts may also be indicated.

After the neurological exam is complete, the wound should be cleaned and debrided to evaluate the wound further. Local anesthesia, regional anesthesia, or both make the process comfortable and efficient. Anesthesia will be described later.

Irrigation with saline will remove blood and clots; dirt and other foreign-body material; and gross bacterial contamination. Antibacterial agents with detergents may also be used: Betadine (povidone-iodine, manufactured by Purdue Frederick, Norwalk, Conn.), Hibiclens (chlorhexidine gluconate, manufactured by Stuart, Wilmington, Del.), or pHisoHex (hexachlorophene, manufactured by Sanofi Winthrop, New York, N.Y.). To prevent further contamination of the wound, surgical aseptic technique should be employed, including sterile drapes, gloves, instruments, and bowls for solutions. Battlefield wounds should be assumed to be contaminated from injury and reasonable means should be undertaken to prevent further contamination, thereby reducing the chance of wound infection. In the military environment, several echelons of care may be used to treat an individual patient. Each manipulation of the wound will bring another chance for bacterial contamination. Therefore, all reasonable efforts should be made to reduce such contamination.[22]

With all the clots and foreign bodies removed, careful inspection with good lighting should reveal the extent of the injury. If bleeding is present, then control by means of electrocoagulation (biterminal) or electrodesiccation (monoterminal) should be effective.[23] Bleeding from larger skin arteries may need to be controlled by ligation. The artery is first clamped with a hemostat at its bleeding point, then a loop of absorbable suture is placed around the vessel and tied firmly. The hemostat is slowly released to check for the adequacy of the ligature. For major arteries, nonabsorbable suture is recommended. Hemostasis should be careful and complete. Bleeding after surgery may lead to hematoma formation. The surgeon should be aware that the addition of epinephrine to the anesthetic solution

may temporarily constrict vessels, which may then bleed several hours later.

Many arteries travel near nerve bundles. Overzealous electrosurgery or sloppy ligation may cause permanent damage to nearby nerves. All hemostasis should be meticulous and complete, especially in wounds that are to be closed by suture.

Microbiology

It is reasonable to assume that all wounds are contaminated with bacteria and particulate matter. With aggressive wound irrigation and debridement, most of the foreign material is removed. The use of antibiotics to prevent clinically apparent infections may be helpful in some instances.[21]

From the history and examination of the wound, the approximate type and quantity of contamination can be determined. The length of time from injury to care will have a large impact on the wound. Many combat-related injuries will be delayed in receiving initial care. The healthcare delivery system in the field involves echelons of service. This contributes to multiple delays in transportation and evaluation. Most non–life-threatening soft-tissue injuries will be triaged less urgently than more serious wounds. The health of the individual also must be taken into account. Exhausted, poorly nourished soldiers who have had prolonged exposure to cold or heat may have less resistance to infection.

Bacteria that commonly have been found to cause wound infections in wartime are staphylococci (*S aureus* and *S epidermidis*), streptococci (hemolytic, nonhemolytic, and enterococci), coliforms (enteric Gram-negative rods), *Pseudomonas aeruginosa*, *Proteus* species, *Hemophilus influenza*, *Clostridium* species(*C tetanus* and *C perfringens*), and *Bacillus subtilus*. The type of organism encountered will vary with the type of contamination, climate, and the location of the wound.[24]

Animal bites may be contaminated with *Pasteurella multocida*.[25] Human bites contain a mixed flora of streptococci (group A, non–group A), *Bacteroides* species, diphtheroids, *Hemophilus influenza*, and enterococci. Soil-contaminated wounds may have multiple organisms present including clostridia, *Bacillus* species, and various Gram-negative rods. Sandy soil is less contaminated with bacteria than nonsandy soil.[26]

Traumatic wounds should be treated with prophylactic antibiotics to prevent postoperative infections. The antibiotic is best given shortly before or within 2 hours after the procedure. However, this protocol is not possible with most traumatic wounds, which are usually hours or days old and heavily contaminated. The contaminating bacteria have had an opportunity to colonize or infect the wound before any prophylactic antibiotic could be utilized. Antibiotics in these cases would be used to prevent overt infection and sepsis. The potential causes of the infection must be adequately covered. The use of antibiotics should not replace the use of proper wound-management techniques including wound toilet, debridement, and careful surgical technique. In general, penicillin, erythromycin, and cephalosporins are useful against most of the common infecting organisms.[27] The exudate from overtly infected, draining wounds should be cultured and Gram-stained before antibiotic coverage is instituted.

Contaminated wounds that are left open are much less likely to become infected than those closed primarily. The wound may be closed 4 days or more after the initial evaluation with less risk of infection and sepsis, provided wound infection is not present at the time of delayed closure. If the wound exudate contains less than 10^6 organisms per milliliter, then clinical infection is unlikely. Experiments have been performed demonstrating that an inoculum of greater than 10^6 *S aureus* bacteria was necessary to produce pus, but lower numbers of bacteria produce redness and swelling.[28] Surgeons have commented[29] that what is surprising is not that wound infection occurs, but that it does not occur more frequently.

SURGICAL INTERVENTION

The goals of surgical intervention on a traumatic wound are restoring function and achieving a cosmetically appealing result. Prevention of further morbidity due to the surgery is paramount and implicit. Through careful planning and meticulous techniques, all these objectives may be met.

Much of the planning should be preceded by evaluation of the type of wound, extent of injuries, and anatomical considerations. The first question for the dermatologist should be whether the management of the wound exceeds his or her ability; if so, appropriate consultation or referral should be made. If the dermatologist feels competent, then the planning begins.

Anesthesia

Any soft-tissue wound that is to be properly cleaned, debrided, and repaired must be anesthetized. For the wounds that a dermatologist would treat, local anesthesia, occasionally augmented with intravenous sedation, is all that should be needed. As was mentioned previously, a thorough neurological exam must precede the instillation of any anesthetic.

Because of the efficacy, safety, short duration of onset, and familiarity by dermatologists, lidocaine (1% or 2%) is the most anesthetic agent. Others have properties such as longer action (ie, bupivacaine), which may be desired. Etidocaine combines shorter duration of onset with longer duration of anesthesia.[30]

The addition of epinephrine (1:100,000–1:200,000) will control bleeding and increase the duration of anesthesia when combined with a local regional anesthetic. Caution must be used in areas where tenuous blood supply exists. Danger areas include acral skin, digital arteries, partially avulsed skin flaps, lower legs, and feet. Patients at high risk include those with significant vascular compromise due to diabetes, atherosclerosis, or other causes. If the patient is taking medications known to interact with epinephrine such as monoamine oxidase (MAO) inhibitors, propranolol, or tricyclic antidepressants, significant blood pressure elevation may result.[29]

The technique for anesthetizing the skin would be similar to that for elective soft-tissue surgery. The major difference is the pain the patient has prior to starting. Anxiety may also be much greater because of the circumstances of the injury and the pain present. Vocal anesthesia (ie, coaching) will make a tremendous impact on the success of the anesthetic experience. Reassurance, explanations, and compassion will relieve the anxiety and make the anesthetic administration easier for the patient and the physician. A recently described technique will significantly decrease the stinging effect of local anesthesia. The addition of sodium bicarbonate (1 mEq/mL) to the anesthetic solution will adjust the pH closer to the physiological range. This adjustment is accomplished by adding 1 mL of sodium bicarbonate solution for every 10 mL of anesthetic solution to be made. The solution should be used within 1 week of mixing to ensure effectiveness.[31]

Inserting a small-gauge needle (nos. 27–30) perpendicularly through the skin into the subcutaneous tissue will cause only slight discomfort. Slow instillation of anesthetic agent will decrease the pain of injection. The needle is then advanced in the subcutaneous plane to minimize the number of needle sticks. Reinsertion of the needle should be through previously anesthetized areas. Rushing will greatly increase the pain for the patient.

A variety of strategies can be used for anesthetizing a skin wound. Wounds may be quite large, and regional nerve blocks can decrease both the amount of anesthetics used and the discomfort of large amounts of local infiltration. Knowledge of the sensory innervation of the skin will allow planning of appropriate blocks. When performing a regional block, the objective is to bathe around rather than to inject directly into the nerve bundles. This technique will lessen the pain of injection and prevent possible nerve damage.

Important regional facial sensory nerve blocks include supraorbital, supratrochlear, infraorbital, and mental. Digital blocks of the fingers are also useful. Unless one has experience with regional anesthesia of the extremities, these should not be attempted.

If individual nerves are not suitable for regional block, then a field block can be utilized. Also, by injecting initially into the proximal path of the nerve supply and then advancing distally, the physician can lessen the pain of anesthesia. Local infiltration of the wound should be performed last because injection of tender tissue is more painful than injection of normal tissue.

Once the area is anesthetized, cleaning, debridement, and wound repair can proceed. Instillation of a long-acting anesthetic such as bupivacaine may give postoperative pain relief for several hours.

Surgical Technique

The decision to close the wound or to allow second-intention healing with possible delayed closure or grafting depends on several factors, including

- the length of time from injury to repair,
- the amount of contamination in the wound,
- the amount of devitalized or damaged tissue, and
- the location of the wound.

Although immediate closure offers quicker and easier repair of the defect, it greatly increases the risk of infection. Bacteria proliferate rapidly in the necrotic debris and clots of an open wound. Foreign bodies offer safe haven for these bacteria. The longer the time between injury and repair, the higher the bacterial counts. Wounds over 6 hours old

should be considered for second-intention healing or delayed repairs.

Contamination is inevitable in traumatic wounds. Relatively clean lacerations caused by glass or clean sharp objects may be cleaned of most foreign bodies and repaired. Grossly contaminated wounds, or those contacted by feces or saliva, should remain open. Devitalized tissue and necrotic skin are much more likely to harbor bacteria and, unless thoroughly excised, will lead to infection. Finally, the location of the wound or, more importantly, the adequacy of the blood supply also is considered in planning the closure. Wounds of the lower extremity are much more likely to be infected. These wounds are at high risk of infection if closed. However, due to the high volume of blood flow and generous collateral blood supply, facial and scalp lacerations are more resistant to infection and should be closed immediately.

The necrotic tissue and foreign body must be removed. Copious irrigation and direct scrubbing are very useful in preparing the wound. Irrigation should be carried out with a high-pressure system consisting of a 20- to 50-mL syringe with an 18- to 21-gauge needle. This will remove dirt and bacteria but will not embed these materials more deeply in the wound. Direct scrubbing with a sponge will remove gross material but probably does not reduce infection.

Clearly devitalized tissue should be excised carefully. Any recalcitrant foreign body should be removed also. The surface shape of the wound should be contoured to a smooth, symmetrical configuration. The sides of the wound should be at a 90° angle to the surface of the skin to prevent inversion of the scar. Reshaping should be tempered with the resultant defect's ability to close without undue tension or distortion at adjacent free edges such as eyelids, lips, or nostril rims. It may be more prudent to keep the wound small and irregular, with plans for later reconstruction, than to make a neat, but time-consuming, closure that may be both difficult to close and at greater risk for infection. Skin grafts, flaps, or reexcision with possible z-plasty, w-plasty, or v to y repairs may be done after healing is completed, under more controlled and less contaminated conditions.

Surgical debridement should remove all attached necrotic debris, devitalized tissue, and irregular jagged wound edges. On noncritical areas such as the trunk and extremities, the debridement can involve an excision around all wounded tissue. When vital structures such as nerves, muscles, and specialized structures of the face are close to the wound, then conservative debridement is recommended. When tissue vitality is uncertain, a delay of several days will allow a demarcation to develop, thus guiding further debridement.

If the wound is to be closed primarily after debridement, one should evaluate the direction and shape of the wound. Traumatic wounds often do not conform to the relaxed skin tension lines (RSTLs). Shifting the direction of closure may require a z-plasty to put the tension of closure perpendicular to these RSTLs. This additional surgery may be best delayed until after healing of the first repair. Scar revision under more controlled conditions will decrease the risk of infection.

The choice of suture materials should minimize the risk of both infection and wound dehiscence. Subcuticular sutures such as vicryl, dexon, or polydioxone (PDS) should be reserved for relatively clean wounds and those under moderate tension. High-tension closures, hematoma, and gross contamination are risk factors for infection that will be compounded by the foreign body of absorbable suture. Attention should be given to the tension of the sutures. Careful approximation must be observed, and strangulation must be avoided. Low-tension wounds may be closed with a monofilament nonabsorbable suture such as prolene or nylon. Adhesive strips are also useful in superficial or low-tension wounds; they may be used without anesthesia, and negate surface removal. They also have a low incidence of wound infection compared with percutaneous sutures.[32] As with all surgical techniques, gentle handling of the tissue is mandatory. Gentle traction with skin hooks or delicate pressure from forceps with teeth will prevent further crushing of the tissue. Hemostasis should be complete, especially if immediate closure is anticipated. For small blood vessels, light electric desiccation or electrofulguration will produce less depth of necrosis than electrocoagulation. If larger vessels are encountered, then electrocoagulation or suture ligation should be considered.

If the wound closure is delayed, then a dressing is applied. (Opinions may differ as to whether to change the dressing daily or to leave it in place for several days.) After 4 to 7 days the wound will have gone through the inflammatory stage of wound healing and will be entering into the granulation tissue stage. This highly vascular state is resistant to infection. At this time, the wound can be closed or a flap or skin graft can be placed. Some wound construction may be started at this time, and more complicated or involved closure may be attempted. Healing may be completed by second intention.

Wound Healing

The process of healing in the skin and subcutaneous tissue depends on the depth and nature of the injury. Superficial abrasions need only to restore the epidermis. Full-thickness skin lacerations, without loss of tissue, can be reapproximated easily and healed in a short period of time. Full-thickness defects of skin and subcutaneous tissue with significant tissue loss, which preclude primary closure, require a lengthy and involved process of wound repair. The defect must restore a volume of tissue before the epidermis can regrow or be grafted over the wound.

Despite these clinical differences in wound healing, many similarities are found in the biochemical and physiological stages. Several models for wound healing exist, most with three or four stages. Although these stages overlap in time and interact with the other stages, they are distinct enough in function to be separated. The classification in this chapter will include inflammation, granulation, fibroplasia and wound contraction, epidermization, and maturation.[33]

Inflammation

Inflammation initiates all the subsequent steps of wound healing. It primes the wound for the construction phases of granulation, fibroplasia, and epidermization. Many authorities break this stage into early and late phases. The early inflammatory phase begins immediately with platelet aggregation and the release of vasoactive substances. Vasoconstriction of severed vessels aids in hemostasis, and vasodilation of local intact vessels allow for the influx of plasma proteins and neutrophils into the wound.[33]

The plasma proteins involved in the formation of fibrin help to stabilize the clot and also elaborate vasoactive and other biologically active substances. The platelets and fibrin pathway generate a multitude of products including prostaglandins, leukotrienes, growth factors, and kinins. Complement activation occurs, generating products including C3a and C5a. One of the first cellular responses to all of these substances is the influx of neutrophils, whose purpose is to destroy and phagocytize bacteria and foreign-body debris.

The late inflammatory stage is characterized by the arrival of the monocyte. This critical cell arrives at the wound at about the third day and is converted to a tissue macrophage. Because it can survive low oxygen tension and pH, it is able to survive in the wound and effectively clean up what the neutrophils have not been able to finish. Besides being a highly effective scavenger, the macrophage is also a manager of early fibroblast function and proliferation. Lymphocytes are also present at this time and, although not essential, have a control function in wound healing.[33]

Granulation

The second stage of wound healing, granulation, is the beginning of reconstruction of the skin. Granulation tissue is composed of a rich plexus of new capillaries within a loose stroma of glycosaminoglycans, fibrin, fibronectin, and immature collagen. During the inflammatory phase, the fibrin clot forms a scaffolding matrix, which is then coated with fibronectin derived from serum and fibroblast sources. Activated fibroblasts and endothelial cell buds migrate over this fibronectin coating, which acts as a glue to the underlying fibrin. As the fibroblasts migrate into the fibrin matrix, they synthesize more fibronectin, glycosaminoglycans, and new collagen. Gradually, the fibrin is lysed and collagen replaces it. Endothelial cells and new capillaries also invade this matrix, driven by low oxygen and high lactate concentrations. Complete granulation tissue replacement of a deep wound may take several weeks.[33]

Fibroplasia and Wound Contraction

In the third stage of wound healing, the fibroblast undergoes a morphologic change in order to migrate into the wound. The appearance of contractile proteins in the cytoplasm provides a mechanism for motility. Because these cells greatly resemble muscle cells, they are called myofibroblasts. Within 7 days, these myofibroblasts have penetrated the matrix enough to begin exerting their contractile force on the surrounding wound edges. This force increases as more cells participate, and clinically measurable contraction takes place. This phase lasts for several weeks, even after epidermization has occurred. The clinical effect is helpful in large wounds to decrease the size of the scar and to shorten healing time. Contracted scars can cause serious morbidity in function and appearance if they are located near joints or on the face near eyelids, lips, or other movable structures.[33]

Epidermization

Epidermization begins within a few days in superficial wounds where the appendages are intact.

Hair follicles, sweat glands, and the surrounding wound edges all contribute to the advancing front of keratinocytes. In full-thickness wounds, this front must wait until granulation tissue has filled the defect, and the keratinocytes come only from the edge of the wounds.[29]

Maturation

Maturation is the final stage of wound healing. It begins when all the other stages end and is variable in time, depending on the wound. The noticeable features are the loss of redness and induration from the wound. The scar becomes softer and white or skin-colored. Histologically, the fibroplasia and hypervascularity of the wound disappear, to be replaced by a hypocellular and hypovascular scar. The collagen produced early in wound healing is more soluble with less cross-linkage. With maturation, collagen bundles are thicker and less soluble with more cross-linking.[33]

Two clinically different types of wound healing exist: primary intention and second intention. An example of primary intention healing would be a simple laceration that is reapproximated by suture. The granulation and contraction stages are minimal, and epidermization is complete within a few days. Second intention healing involves a full-thickness loss of skin volume. The stages of wound healing as described previously must all occur.[34]

Wound Dressings

The care of a wound in a military field setting will differ from care in a medical center. Resources such as time, personnel, and materials may be limited. The surgeon must pick from the available dressing materials, with the best choice based on the criteria discussed below.

Tremendous advances in the understanding of wound healing and technology have radically changed the approach to wound dressings. Whereas wounds were previously covered with dry gauze or left uncovered, it is now standard to use moist, semiocclusive dressings of ever-increasing sophistication.[35] Growth factors, bioactive dressings, and even cultured epidermal coverings are now being used in major medical centers to facilitate wound healing.[34,36] An effective dressing for wound healing should

- absorb drainage,
- prevent contamination,
- provide pressure to control bleeding,
- immobilize or splint the wound,
- prevent desiccation of tissue,
- be easy to change with minimal discomfort, and
- cushion the wound from further trauma.

In addition, the dressing should be readily available at reasonable cost.

No dressing is perfect for every wound. Grossly contaminated or actively draining wounds require that their dressings be changed more often and have greater absorption. Later, when the wound may be cleaner and have less drainage, a less-absorptive dressing needing less-frequent changes may be used.

Complications

The most common and devastating problem of traumatic wounds is infection. As previously mentioned, many factors contribute to wound infection. These wounds are at risk for much greater problems such as sepsis, gas gangrene, and necrotizing fasciitis. Early recognition and treatment of these serious conditions is vitally important. With these more involved situations, consultation with a general surgeon will be required.

Prolonged bleeding and hematomas are also probable complications of traumatic wounds. Inadequate hemostasis at the time of surgery and delayed bleeding are the likely causes. Congenital or acquired coagulopathies would be unusual but should be considered in patients with persistent bleeding. Disseminated intravascular coagulation may be accompanied by sepsis in the wounded patient. The presence of excess blood in the wound, coupled with the pressure effects on tissue from a hematoma, will increase the likelihood of infection. Early hematomas should be drained and the offending vessels controlled. If a hematoma has been present for several days, drainage will be difficult until the clot liquefies. Drainage with a large bore needle may be attempted when the clot feels more fluid. Many hematomas will resolve spontaneously.[24]

Necrosis of skin is caused by inadequate blood flow to tissue. Contributing causes include excessive tension, hematoma, infection, crush injuries, desiccation, or severe disruption of the arterial supply of the skin.

Wound dehiscence may result from infection, hematoma, or excessive tension on the wound edges. It causes delayed healing and an unattractive spread scar.[24]

Traumatic scars frequently will benefit from some form of reconstructive procedure, which traditionally falls in the realm of the plastic and reconstructive surgeon. Experienced dermatologic surgeons may be able to accomplish many of these procedures.

Many dermatologists now perform dermabrasion for the improvement of surgical or acne scarring. An irregular scar surface may be planed smooth by a brief, simple procedure that utilizes local anesthesia. Dermabrasion may be performed for blast tattoo (ie, an accidental depositing of pigmented particles within the dermis, often as the result of an explosion) to remove the pigment. A field-expedient method employs a stiff toothbrush rather than a diamond fraise. Because proper dermabrasion equipment does not exist in the field, this method allows prompt treatment of the tattoo before the pigment has been trapped by macrophages.[37,38]

Hypertrophic scars or keloids may develop on the trunk, extremities, or occasionally on the face. A nonsurgical scar improvement may be obtained by intralesional steroid injection. A concentration of 2.5 to 40 mg / mL of triamcinolone acetonide may be injected at 3- to 6-week intervals, depending on the size, location, and aggressiveness of the excess scar tissue.

Finally, many scars improve with time. In young individuals, the scar line will be red for several months to a year. Many thick scars will soften, and contracted scars will relax. Before attempting a significant surgical revision, the treatment of time should be allowed.

SUMMARY

The skin is vulnerable to injury due to environmental hazards in the battlefield environment. Extremes of weather, ultraviolet radiation, arthropod bites, chemical agents, frictional forces and lacerations from missile injury or accidents all may alter the natural barrier we depend upon to protect us from life-threatening insults. Dermatologists possess the clinical fund of knowledge and soft-tissue surgical skills to make them critical participants in the care of traumatic injuries to the skin.

Friction blisters have long been recognized as a significant medical problem in the military. The morbidity associated with friction blisters sometimes extends beyond the pain and discomfort of the lesion: secondary bacterial infection is common and often extends time lost per case to as long as a week. The most important forces in the formation of a friction blister are dynamic shear forces. Moderately moist skin, improperly fitting or poorly designed shoes, poorly fitting or worn-out socks, and wearing boots less than 20 hours per week during the 2 weeks immediately before training are all recognized risk factors for friction blisters. Prevention of friction blister formation is the most important front-line approach in dealing with this common problem and involves techniques designed to toughen the skin and reduce shear force. The development of epidermal hypertrophy through conditioning the skin, the use of an antiperspirant foot powder, and the use of acrylic socks are all important preventive strategies.

Cutaneous trauma treated by a dermatologist is restricted to superficial soft-tissue injuries. In emergent care, basic initial evaluation takes little time but may be lifesaving. Airway patency, breathing, and adequate circulatory system function should be evaluated first. Skin and soft-tissue injury care must be delayed until circulatory volume is adequate and internal injuries have been ruled out. The types of wounds that a dermatologist is helpful with include abrasions, lacerations of the skin, and traumatic tattoos. Battlefield wounds should be assumed to be contaminated from injury. Aggressive wound irrigation and debridement, and treatment with prophylactic antibiotics to prevent postoperative infections, are crucial for traumatic wounds. In general, penicillin, erythromycin, and cephalosporins are useful against most of the common infecting organisms.

The goals of surgical intervention on a traumatic wound are restoring function and achieving a cosmetically appealing result. Local anesthesia, cleansing, debriding, repair, and dressing of such injuries to the skin are all aspects of wound care for which dermatologists are well trained. They should be incorporated into the surgical care team as needed in the particular medical care setting to which they are assigned.

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