Acute Wounds

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Few if any “rules” govern the management of acute wounds. A basic knowledge of wound healing and a clear understanding of the potential risks and benefits of the selected therapy are paramount. This article addresses the basic concept of normal wound healing and the management of acute wounds.

Basic concepts of normal wound healing

In the past several decades, we have learned a great deal about the biochemistry, molecular biology, and cell physiology of the events that lead to a healed wound (Fig. 1). The physiologic mechanisms of wound healing are similar in different tissues with slight variations. This article covers the healing of acute wounds.

Wound healing is classically divided into three phases: the early, intermediate, and terminal phases (Fig. 2). The initial response to injury, whether mechanical, immunologic, thermal, or chemical, is a nonspecific inflammatory response. In all injuries that involve the skin, blood vessels are disrupted, resulting in bleeding. Immediately following injury, there is a period of vasoconstriction lasting 5 to 10 minutes, activation of platelets, and the coagulation cascade to produce hemostasis. The vasoconstriction in the early onset is mediated by catecholamines and prostaglandins. Platelets aggregate when exposed to the intravascular collagen and release adenosine diphosphate, which, in the presence of calcium, stimulates further platelet aggregation [1,2]. Platelet aggregation also leads to the release of cytokines, which include platelet-derived growth factor (PDGF), transforming growth factor–α (TGF-α), and transforming growth factor–β (TGF-β); these play a critical role in the later phases of wound healing. Coagulation pathways result in the production of thrombin, which catalyzes the conversion of fibrinogen to fibrin. Thrombin and fibrin contribute to hemostasis and later to other aspects of wound healing [3].

Vasoconstriction is followed by a period of vasodilatation, probably mediated by bradykinin and histamine. Thrombin contributes to the increased vascular permeability and facilitates the migration and diapedesis of leukocytes through “leaks” in the vascular endothelium in the zone of injury. Fibrin produces a scaffold for the migration of inflammatory and mesenchymal cells. Vasodilatation is also mediated by kinins, prostaglandins, and endothelial cell products [4,5]. Vasodilatation and cellular diapedesis lead to the characteristic physical signs of inflammation: erythema (rubor), heat (calor), pain (dolor), and edema.

Inflammatory phase

The predominant initial cell type is the polymorphonuclear leucocyte, which assists in wound debridement [6]. Neutrophils, macrophages, and lymphocytes are involved in the inflammatory phase of injury (Fig. 3). Chemotaxis is the movement of an organism or cell type in response to a chemical concentration gradient. Chemotactic agents contribute to the migration of leukocytes into the extravascular space. The leukocytes phagocytose injured tissue and bacteria. Neutrophils die after phagocytosis.
ing damaged tissue and bacteria; they do not play a role in the subsequent events of healing in an uncomplicated wound (Fig. 4).

Macrophages are nothing but monocytes transformed by a process mediated by serum factors and fibronectin [7–9]. The migration of macrophages in the wound bed is mediated by chemotactic factors. Macrophages are tremendously important in normal wound healing [10]. Macrophages phagocytose bacteria and dead tissue and secrete collagenases and elastases that help break down the wound matrix [11,12]. Macrophages are the primary site of cytokines that stimulate fibroblast proliferation, collagen production, and other healing processes. Macrophages are probably the most important cell in the wound healing process. Lymphocytes produce factors essential for normal wound healing [13]. They are also involved in cellular immunity and antibody production. The critical cytokines liberated are probably epidermal growth factor (EGF) and basic fibroblast...
growth factor. In the acute wound, neutrophils predominate initially, but by the third day, the macrophages predominate.

What makes acute wounds become chronic? Foreign material or bacteria can cause persistent chronic inflammation. Although the acute inflammatory phase is necessary for normal wound healing, the persistence of chronic inflammation is often deleterious [14]. The persistence of neutrophils in the wound can cause release of destructive proteolytic enzymes and free radicals, which damage tissues. The oxygen free radicals lead to persistent tissue destruction. Areas of chronic inflammation can become encapsulated, leading to granuloma formation.

**Intermediate phase**

The intermediate phase of wound healing includes mesenchymal cell chemotaxis, mesenchymal cell proliferation, angiogenesis, and epithelialization. These processes are mediated by cytokines. The cytokines that are well described include PDGF, TGF-β, EGF, acidic and basic fibroblast growth factor (aFGF, bFGF), TGF-α, tumor necrosis factor (TNF), interleukin-1 (IL-1), and insulin growth factor (IGF) (Table 1). Fibroblasts are the primary mesenchymal cells involved in wound healing. They normally reside in the dermis. Undifferentiated mesenchymal cells in the area of injury may also differentiate into fibroblasts when stimulated by chemotactic factors. PDGF is chemotactic for both fibroblasts and smooth muscle cells [15,16]. Fibronectin is a primary component of extracellular matrix and is chemotactic for fibroblasts. PDGF also acts as a potent mitogenic stimulus for both fibroblasts and smooth muscle cells, augmenting the wound’s mesenchymal cell population. Mesenchymal cell proliferation can also be stimulated by TNF, IL-1, lymphokines, and IGF [17,18].

Wound angiogenesis is stimulated by high lactate levels, acidic pH, and decreased oxygen tension (pO2) in the tissue. The capillary sprouts continue to grow and interconnect, forming patent vascular loops. Cytokines primarily involved in angiogenesis include bFGF, TGF-α, TGF-β, vascular endothelial growth factor, and prostaglandins. These stimulatory cytokines diminish when the wound is completely vascularized [19–21].
Epidermal cells constantly regenerate to provide a barrier between the external environment and the internal milieu. The process by which the epidermal cells regenerate and migrate to cover a wound is called “epithelialization.” This process is important in the healing of partial-thickness wounds, such as abrasions, superficial burns, and split-thickness skin graft donor sites. The process takes several weeks, depending on the size of the defect.

The process of epithelialization involves cellular detachment, proliferation, and differentiation. The marginal basal cells migrate toward the center of the wound as a monolayer and exhibit contact guidance [22,23]. These cells migrate until they reach cells migrating from the opposite direction, and the migration stops by “contact inhibition.” The new surface epithelial cells begin to keratinize. The re-epithelialized surface has fewer basal cells and no rete pegs. The epithelium is thicker at the wound edges than in the midportion of the re-epithelialized area.

Late phase of fibroplasia

Fibroplasia involves the production of collagen in the wound. Collagen makes up more than 50% of the protein in scar tissue [24]. Collagen is synthesized primarily by fibroblasts, beginning 3 to 5 days after the injury. The rate of collagen synthesis increases rapidly by 3 to 4 weeks, then rapidly declines. Finally, there is a balance between the rates of collagen production and collagen destruction by collagenase. Age, tension, pressure, stress, and TGF-β affect the rate of collagen production. In human skin, type I collagen makes up 80% to 90% and type III the remaining 10% to 20%. Type III collagen is seen predominantly in the early phase of wound healing. Types II and XI are seen in cartilage, type IV in basement membranes, and type V in smooth muscle cells.

Collagen consists of three polypeptide chains, each twisted into a right-hand helix. The alignment of these three chains into a triple helix is facilitated by nonhelical terminal peptide sequences. Most polypeptide chains in collagen are alpha chains. The collagen synthesis is also facilitated by hydroxylation of lysine and proline. The hydroxylation is required for covalent crosslink formation. It requires specific enzymes for lysine and proline and cofactors oxygen, vitamin C, α-ketoglutarate, and ferrous iron. The hydroxylation and crosslinking are prevented by deficiencies of vitamin C and oxygen and by corticosteroids, resulting in poor wound healing.

The collagen molecule is synthesized into the extracellular matrix in the form of procollagen. The procollagen is converted into tropocollagen, which then aggregates into collagen fibrils. This fibrin formation is facilitated by proteoglycans in the extracellular matrix.

Proteoglycans are synthesized primarily by fibroblasts and include chondroitin sulfate, dermatan sul-

<table>
<thead>
<tr>
<th>Healing function</th>
<th>Cytokine involved</th>
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<tbody>
<tr>
<td>Inflammatory cell migration</td>
<td>PDGF, TGF-β, TNF-α</td>
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<tr>
<td>Fibroblast migration</td>
<td>PDGF, TGF-β, EGF</td>
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<tr>
<td>Fibroblast proliferation</td>
<td>PDGF, TGF-β, EGF, IGF, TNF-α, IL-1</td>
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<tr>
<td>Angiogenesis</td>
<td>bFGF (FGF2), aFGF (FGF1), TGF-β, EGF, TNF-α, VEGF, IL-8, PD-ECGF</td>
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<td>Epithelialization</td>
<td>EGF, TGF-β, KGF (FGF7), bFGF (FGF2), IGF, HB-EGF</td>
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<tr>
<td>Collagen synthesis</td>
<td>PDGF, TGF-β, bFGF (FGF2), EGF</td>
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</table>

*Abbreviations:* HB-EGF, heparin-binding epidermal growth factor; IL-8, interleukin-8; KGF, keratinocyte growth factor; PD-ECGF, platelet-derived endothelial cell growth factor; VEGF, vascular endothelial growth factor.

fate, heparin, heparin sulfate, keratin sulfate, and hyaluronic acid. The biologic functions of proteoglycans are not well understood. The proteoglycans can potentiate cytokines involved in angiogenesis and cellular migration. Other key components of the wound matrix are fibronectin and other attachment proteins. Fibronectin is produced by fibroblasts and epithelial cells [25]. It aids in cellular attachment, cellular migration into the wound matrix, and binding of epithelial cells to the matrix and thus contributes to epithelialization and fibroblast migration. Another component of the connective tissue is elastin. Elastin is lacking in normal scar, which hence lacks the elastic properties of normal skin.

Wound contraction

Wound contraction begins 4 to 5 days after wounding. The process involves centripetal movement of the wound edge toward the center of the wound. Maximal wound contraction continues for 12 to 15 days, as long as the wound remains open. Wound contraction progresses at an average of 0.6 to 0.75 mm per day. The tissues that have the greatest laxity demonstrate the greatest degree of wound contraction. Therefore, wounds in areas overlying bone, such as the tibia and scalp, contract very little. Wound contraction is a major contributor to the healing of full-thickness open wounds compared with incisional wounds. Wounds with square edges contract more rapidly than circular wounds. Although wound contraction is beneficial to wound healing, contraction of large open wounds across joint surfaces can lead to limitation of motion secondary to contractures. Contractures are commonly seen in burn wounds in the submental and neck areas, axillae, elbows, and other joint surfaces.

The most accepted mechanism of wound contraction is attributed to myofibroblasts. Myofibroblasts were first described by Gabbiani et al [26] in 1971. Myofibroblasts most likely derive from normal fibroblasts in acute wounds and first appear on the third day after wounding. They persist in large numbers until about 21 days post-wounding. They are primarily found in the periphery of the wound. Experiments indicate that myofibroblasts pull the wound edges together in a “picture frame” fashion [27]. An alternative theory is that wound contraction is secondary to fibroblasts that undergo modification with stress fibers in their cytoplasm at the wound edges. Contraction is a cell-directed process that requires cell division but not collagen synthesis. PDGF and TGF-β appear to be mediators of wound contraction, whereas FGF and IGF inhibit it. Radiation and cytotoxic drugs delay contraction by inhibiting cellular activity.

Wound contraction can be detrimental in some areas, such as joint surfaces, neck, and axilla. Wound contraction can be limited by early skin grafting, full-thickness skin grafts, and splinting. Myofibroblasts disappear from the wound more frequently after a full-thickness skin graft compared with a split-thickness skin graft [28]. No pharmacologic agents inhibit wound contraction.

Scar remodeling

Scar remodeling is the terminal wound healing event. The collagen content of a healed wound is maximal at 21 days after injury. However, the bursting strength of the wound is only 15% that of normal skin (Fig. 5). Scar remodeling is a process to increase the wound’s bursting strength. By 6 weeks after the wounding, the wound bursting strength is 60% to 70% that of normal skin. By about 6 months, the wound bursting strength reaches a plateau at 80% of normal skin. The process of scar remodeling involves an increase in intra- and intermolecular crosslinks between collagen fibers. This increase in crosslinking is the major contributor to the increase in wound breaking strength. During this process, the quantity of type III collagen decreases as it is replaced by type I collagen. The quantity of water and glycosaminoglycans in the matrix also decreases. The scar remodeling continues over a period of 10 to 12 months. This is visible as the scar becomes less indurated, softer to the touch, and less red, assuming a color closer to the adjacent skin. Therefore, scar revision should not be undertaken for at least 6 to 8 months. The scar maturation process also involves an increase in col-

![Fig. 5. Tensile strength of the healing wound. (From Lawrence WT. Physiology of the acute wound. Clin Plast Surg 1998;25(3):334; with permission.)](image-url)
lagentolytic activity. The activity of collagenolytic en-
yzymes is modulated by several tissue inhibitors of
metalloproteases. Other enzymes, such as hyaluronidase, may be involved in scar remodeling [29].

Factors that impair wound healing

It is important to have a clear understanding of
the factors that may lead to impaired wound healing.
Many factors, both local and systemic, can contribute
to poor wound healing (Table 2). A diagnosis of the
factors that can impair wound healing must be es-
tablished so that healing conditions can be optimized
by controlling the systemic factors and providing
sound local wound care [30]. These factors, if not
addressed early in the management of acute wounds,
can lead to delayed healing or nonhealing wounds.
Local ischemia and tissue hypoxia should be recog-
nized both by history and clinical evaluation [31].
In infected wounds, the healing process is retarded
or delayed by a prolonged inflammatory response.
These wounds do not heal until the infection is
controlled. Foreign bodies can predispose to infec-
tion, thereby delaying healing. Foreign bodies also
prolong the inflammatory response. Radiation causes
local tissue ischemia, and incisions in a radiated bed
are slow to heal.

Extrinsic or systemic factors that impair wound
healing include diabetes mellitus, steroids, smoking,
chemotherapeutic agents, cancer, nutritional defi-
ciciencies, liver disease, chronic renal failure, and
hereditary factors. The list is by no means complete.
Uncontrolled diabetes has been demonstrated to im-
pair wound healing [32,33]. Proper control of blood
sugar and prevention or aggressive treatment of
infection are crucial. Steroids have been shown to
affect all aspects of the wound healing process [34].
The effect of steroids on wound healing can be re-
versed by administration of vitamin A [35]. Smoking
causes cutaneous vasoconstriction. Additionally, ele-
vated levels of carboxyhemoglobin can adversely
affect the oxygen-carrying capacity of blood [36].
Chemotherapeutic agents are more potent inhibitors
of wound healing when delivered preoperatively
rather than postoperatively. Therefore, any elective
surgery should be planned appropriately, and che-
motherapy should be deferred until after surgery [37,38].

Promoting wound healing in acute wounds

Improving tissue perfusion and tissue oxygenation

Adequate tissue perfusion, blood flow, and oxy-
gen levels are requisites for wound healing [39].
Tissue perfusion is essential for the delivery of ne-
cessary nutrients, including oxygen, to the healing
tissues. Oxygen is necessary for the opsonizing func-
tion of neutrophils and macrophages, the hydroxyl-
ation of prolene and lysine, and collagen synthesis
[40]. Angiogenesis in an acute wound is facilitated
by local wound hypoxia, which in turn causes wound
hyperoxia. This hypoxia–hyperoxia cycle facilitates
collagen synthesis. The rate of epithelialization is
accelerated by hyperoxia and significantly slowed by
hypoxia [41].

The control of bacterial population in wounds is
critical for successful wound healing. The ability of
leucocytes to remove bacteria depends significantly
on oxygen-dependent systems [42]. In a hypoxic
environment, wound infection rate increases, as may
be noted in an ischemic wound. A local pO2 of
30 mm Hg or greater is required for leucocytes to kill
bacteria effectively [43]. Therefore, maintaining ex-
cellent tissue perfusion and oxygen supply should
reduce the risk of wound infection and facilitate
wound healing. Patients undergoing cardiac and vas-
cular procedures appear to be at a higher risk for
wound complications, including infection, that are
attributed to wound hypoxia [44].

Tissue perfusion also correlates with the avail-
ability of oxygen to the tissues. Tissue perfusion is
affected adversely when the circulating blood volume
decreases, as seen in severe hemorrhage, dialysis pa-
ients, and cases of dehydration [45,46]. It has been
demonstrated that subcutaneous oxygen tensions are
reduced secondary to hypovolemia, reflecting less
than optimal oxygen delivery to the healing tissues.
Supplemental intravenous fluids can correct this
problem. Current evidence from clinical studies indi-
cates that either colloid or crystalloid fluid replace-
ment will support peripheral blood flow, oxygen
levels, and wound healing [47]. Other factors in

<table>
<thead>
<tr>
<th>Intrinsic factors</th>
<th>Extrinsic factors</th>
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<tr>
<td>Ischemia</td>
<td>Nutritional deficiencies</td>
</tr>
<tr>
<td>Infection</td>
<td>Diabetes mellitus</td>
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<tr>
<td>Foreign bodies</td>
<td>Chronic renal failure</td>
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<tr>
<td>Cigarette smoking</td>
<td>Steroids</td>
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<td>Venous insufficiency</td>
<td>Chemotherapeutic agents</td>
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<tr>
<td>Radiation fibrosis</td>
<td>Distant malignancies</td>
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<tr>
<td>Repeated trauma</td>
<td>Old age</td>
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<tr>
<td>Local toxins</td>
<td>Liver disease</td>
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<tr>
<td>Cancer</td>
<td>Other drugs</td>
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</table>

optimizing perfusion include warming patients and maintaining the body core temperature.

Regional blocks do not affect tissue perfusion, oxygenation, and wound healing [48]. The work done with epidural analgesia and anesthesia indicates that, although the time required for supplemental oxygen to reach the tissues is reduced after epidural anesthesia, regional vasodilatation and improved blood flow may be beneficial. Further studies are needed to document blood flow changes and wound healing outcomes.

**Supplemental oxygen**

Support of arterial oxygen tension is necessary to ensure that oxygen is available for transport to tissues. It has been demonstrated that supplemental oxygen increases oxygen tension peripherally, provided that perfusion is maintained [49,50]. Supplemental oxygen is indicated in the early postoperative period, particularly for patients who are likely to be at risk for wound healing problems (eg, those with longer surgical procedures, cardiovascular and abdominal surgeries, emergent surgeries, or severe infections, such as necrotizing fasciitis) [51]. Tissue oxygen levels are dependent on blood flow and adequate arterial oxygen content. Therefore, the importance of blood flow and tissue perfusion cannot be overemphasized.

Studies currently in progress examine the postoperative effects of supplemental oxygen therapy on wound healing outcomes. For the moment, it appears prudent to administer supplemental oxygen at doses that correct saturation levels below 94% until patients can consistently maintain acceptable Spo2 levels.

**Body core temperature regulation**

Thermoregulation may benefit wound healing and increase resistance to infection [52]. Reduction in body temperature causes cutaneous vasoconstriction, decreased tissue perfusion, and low tissue oxygen levels. The negative effects of hypothermia on resistance to infection have recently been reported. Experimental data from animals and humans indicate that wound infection rate is significantly increased in the hypothermic state compared with the normothermic state. It is likely that the increased resistance to infection associated with normothermia is due to effects on the oxidative killing mechanisms of neutrophils and macrophages and the augmentation of immune system responses [53]. Warming of inhaled gases, use of infusion fluids, and application of body warmers are some of the methods commonly used to maintain the body core temperature, in the trauma units and during and post-surgery. Modifying the wound environment by means of local application of heat may facilitate wound healing. This area of increasing study has a high potential to affect wound healing.

**Nutrition**

Adequate nutritional status influences both wound healing and immune function. Cellular proliferation, phagocytosis, and the production of matrix have tremendous energy demands. Proteins, carbohydrates, and fats are required for these processes, as are vitamins and minerals. Many enzyme systems involved in wound healing are dependent on the availability of proper nutrients. The roles of ascorbic acid (vitamin C), vitamin A, copper, and zinc are well documented [54,55]. Currently, there are no well-accepted recommendations for nutritional support to influence wound healing. Most recommendations involve correcting or replacing deficiencies.

Patients prone to poor nutrition include those with gastrointestinal diseases, such as Crohn’s disease, malabsorption, diarrhea, dialysis, alcoholism, cancer, and chronic illness, as well as elderly patients living alone. It is becoming increasingly clear that even mild nutritional deficiencies may impair wound healing [56]. Experimental and clinical data emphasize the need to evaluate the patient’s dietary history and nutritional status before surgical procedures, because the risk of wound healing impairment increases in the face of protein-energy malnutrition.

Wounds cause variable degrees of metabolic stress, based on their size and complexity. Secretions and drains are additional sources of protein loss. Sepsis is a major source of metabolic stress. Therefore, recognition of these stress factors and provision of nutritional support are crucial to optimizing wound healing. Nutritional substrates may be particularly important during acute illnesses and metabolically stressful states. Clinically, patients who received an enteral diet supplemented with arginine, RNA, omega 3, and omega 6 fatty acids for gastrointestinal surgery had significantly fewer wound healing complications [57]. Wound healing can successfully be supported by nutrition, either by enteral or by parenteral route. Whenever the patient can eat and has no malabsorption, enteral feeding is preferred. A number of factors must be considered in selecting the preferred route: gastrointestinal function, access, length of time needed for nutritional support, number of calories required per day, and activity level.

**Acute wound care**

The management of all acute wounds starts with a thorough history and physical examination. This
procedure is often difficult in an emergency room setting, especially when the patient is intoxicated and combative and the treating physician is in a rush. The initial evaluation should identify all injuries and rule out associated occult injuries. The occult injuries may be life-threatening—for instance, intra-abdominal trauma, injury to the brain and cervical spine, thoracic injuries, or airway injury. Patient safety comes first. The treating physician should identify the time and mechanism of injury, associated medical problems, tetanus immunization status, medications, and allergies. The basic question is whether the patient is stable or unstable and whether the patient needs resuscitation. Airway management should be prompt. Appropriate radiographs of the C-spine and CT scans of the head, chest, and abdomen help rule out occult injuries. Wound exploration can be performed in the emergency room or in the operating room, based on the location and complexity of the wound, the patient’s age, pain tolerance, associated injuries, and ability to cooperate, and the length of time required for the procedure.

The basic wound healing process is the same regardless of how the wound is managed. From a clinical perspective, wound healing may be separated into primary, secondary, and tertiary or delayed primary intention. Primary wound healing follows acute closure of wounds. The healing is quick with minimal scar (e.g., surgical closure of a clean laceration). Secondary wound healing occurs when the wound is allowed to heal secondarily by contraction, granulation, and epithelialization (e.g., burn wounds). In tertiary healing, or the delayed primary method, a wound left open for several days is closed by bringing the edges together.

Basic wound management has four components: evaluation and planning, wound preparation, wound closure, and wound dressing. Most acute wounds, with the exception of surgical wounds, are potentially contaminated. These wounds are cleaned by appropriate irrigation, debridement, trimming of the wound edges, if they are irregular, and excision in some cases. The decision to close an acute wound is based on (1) the age of the wound, (2) the degree of wound contamination, and (3) whether the wound is infected. Wounds that are often left open include human bite wounds, blast injuries, and severely contaminated or fecally contaminated wounds. Although these wounds are initially treated open, they are amenable to a delayed primary closure. In patients with severe injuries, wounds can be left open unless vital structures are exposed, such as major vessels, joints, and dura. The wound should be thoroughly irrigated with saline or antibiotic solution and dressed with moist, absorbent gauze dressings. Injured extremities should be splinted.

Wound debridement

Wound debridement is the most important of the four parts of wound management. It involves sharp debridement or wound excision with a knife blade or sharp, curved scissors. More recently, ultrasound and lasers have been used for wound debridement. The direction of the wound may be changed during the debridement to coincide with the lines of skin tension and achieve a better scar. Importance should be given to both cosmesis and functional outcome (e.g., in the periorbital region). Sharp debridement helps convert a dirty, contaminated wound to a clean wound, re-orient lacerations, and remove devitalized tissue. Foreign bodies on the wound surface can be removed easily using a sponge, a scrub brush, or a toothbrush. Any deeply embedded dirt is removed with a sharp blade to prevent traumatic tattoos. Occasionally, fluoroscopy may be necessary to remove deeply embedded metallic foreign bodies, such as bullet or shrapnel fragments.

Wound preparation

The most commonly used antiseptic solutions in wound preparation are hexachlorophene (pHisohex, Beyer Corp., Myerstown, PA) and complexed iodine solutions (Betadine, Novation Inc., Irving, TX). Any agents that are toxic to the wound should be avoided (Table 3). Wound irrigation with a 19-gauge

<table>
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<tr>
<th>Agent</th>
<th>Relative toxicity</th>
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<tr>
<td>Saline</td>
<td>0</td>
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<tr>
<td>Pluronic F-68</td>
<td>0</td>
</tr>
<tr>
<td>Betadine prep solution</td>
<td>1+</td>
</tr>
<tr>
<td>(povidone-iodine solution)</td>
<td></td>
</tr>
<tr>
<td>Hexachlorophene solution</td>
<td>2+</td>
</tr>
<tr>
<td>Quaternary ammonia solution</td>
<td>3+</td>
</tr>
<tr>
<td>Hydrogen peroxide</td>
<td>6+</td>
</tr>
<tr>
<td>Betadine surgical scrub</td>
<td>8+</td>
</tr>
<tr>
<td>(povidone-iodine and detergent)</td>
<td></td>
</tr>
<tr>
<td>pH2Hisohex (hexachlorophene and detergent)</td>
<td>8+</td>
</tr>
<tr>
<td>Isopropyl alcohol</td>
<td>10+</td>
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</table>

Saline (0) is the standard. The higher the number, the more caustic the agent.

needle and a 30-mL syringe provides adequate force (pressure greater than 8 psi) for an effective wound preparation. A word of caution on the use of topical antimicrobial agents is in order. Although the efficacy of topical antimicrobial agents is well documented, daily exposure to even dilute solutions of the commonly used topical agents (1% povidone-iodine, 3% hydrogen peroxide, 0%–25% acetic acid, and 0%–5% sodium hypochlorite) may reduce wound healing and wound tensile strength [58,59].

Wound closure

Superficial wounds are usually allowed to re-epithelialize by providing a moist wound healing environment. Lacerations with low to moderate contamination are closed primarily after adequate debridement. Heavily contaminated wounds should be debrided, irrigated, and packed, followed by a delayed primary closure. Simple superficial lacerations are easily amenable to Steri-strip (3M Healthcare, St. Paul, MN) closure. For deeper, more complex lacerations, a layered closure is required to achieve a good cosmetic result. A tension-free closure is important and may require undermining of the wound edges. Dead space is eliminated by closing the deeper layers with absorbable sutures. Buried dermal sutures of absorbable material can align skin edges. The skin edges can be approximated with Steri-strips, tissue glue, (Dermabond, Ethicon Inc., Johnson & Johnson, Somerville, NJ), or fast-absorbing gut or nonabsorbable suture. Whenever skin sutures are used, it is important to remove them as early as possible to avoid ugly stitch marks, especially in facial wounds.

The location, depth, and complexity of the wound, the patient’s age, the patient’s degree of cooperation, and the presence or absence of associated injuries dictate whether one uses local or general anesthesia for the wound care. Lidocaine (Xylocaine) 0.5% to 1% with 1:100,000 epinephrine is used except in the hand. The recommended dose of lidocaine is 4 to 5 mg/kg, and the dose of lidocaine with epinephrine is 7 to 10 mg/kg. Topical anesthetics that may be used include ethylene glycol spray, lidocaine spray, lidocaine and carbocaine creams, and topical cocaine for the nose.

When a soft tissue defect is too large to be closed primarily or when the primary closure results in unwanted tension and tissue distortion (eg, eyebrow, eyelid, oral commissure), either a local skin flap or a skin graft may be used. If local skin flaps are inadequate, pedicled muscle or musculocutaneous flaps or microvascular free flaps may be required to treat acute wounds, to cover exposed tendons, fractures, joints, dura, and major vessels, or for vascular prosthesis. Large, superficial, simple wounds are traditionally treated successfully with split-thickness skin grafts, 0.015 in. However, facial wounds often require full-thickness skin grafts to treat peri orbital, perioral, and nasal areas and to prevent wound contractures and secondary deformities. The pre- and postauricular and supraclavicular regions are excellent donor sites for full-thickness skin grafts. Whenever a skin graft, skin flap, or muscle flap is planned, it is better to debride the wound and carry out dressing changes for 48 to 72 hours before the final wound coverage. This delay will help ensure that the wound has been adequately debrided and any potential infection eradicated.

Use of antibiotics

Infection of traumatic wounds is related to a multitude of factors, including mechanism of injury, time elapsed since injury, wound contamination, and the presence of local or systemic factors that impair wound healing (see Table 2). All traumatic wounds are potentially contaminated, with an infection rate of 15% [60]. Infection rate is significantly reduced by thorough debridement and gentle tissue handling [61].

Research has shown that antibiotics administered preoperatively are most effective in preventing infection. Tetanus prophylaxis is key in all acute traumatic wounds (Table 4). When treating acute wounds, the decision to use antibiotic treatment is based on (1) the mechanism of injury, (2) the degree of wound contamination, (3) the patient’s delay in seeking treatment, and (4) the presence of overt signs of infection. Antibiotic treatment should not be a routine measure, especially in fresh elective surgical wounds. Systemic antibiotics should be used to treat active infections, such as cellulitis, phlebitis, and lymphangitis, grossly contaminated wounds, or active wound infection [62]. Bite wounds should be treated with antibiotics, with the choice determined by the patient’s history (Table 5).

Wound dressings

No ideal wound dressing exists. The principal function of a wound dressing is to provide an optimal healing environment. The best way to expedite healing and minimize pain and discomfort is to close
the wound. Once the wound is closed, the suture line can be left open to the air. Other popular measures to protect the skin closure include application of Steri-strips, Band-aids, and occlusive and nonocclusive dressings. In children, pain and distress related to trauma and dressing change create a particular problem. These factors have a psychologic impact on the child and family.

The beneficial effects of moist wound healing, as provided by occlusive dressings in acute wounds (surgical wounds, traumatic wounds, and burns) have been demonstrated in various studies. Regarding the use of occlusive hydrocolloid dressings in the treatment of acute wounds, evidence indicates a shorter healing time, lower frequency of infections, and greater compliance of patients, due to their minimal pain and greater ability to carry out their daily activities [63]. Data indicate that hydrocolloid dressings protect wounds equally well in adults and children and cause significantly less pain, both when in place and during removal, than do conventional absorbent gauze dressings. Therefore, hydrocolloid dressings are excellent for treating skin-graft donor sites, derm- abrased or laser-resurfaced areas, and abrasions [64]. The colloid gel produces an absorption gradient for soluble components within the exudate, thereby allowing the removal of toxic components produced by cellular and bacterial destruction. A new concept in the management of wounds is the application of negative pressure (vacuum assisted closure) therapy. It is simple to use and has many potential benefits [65], including reduction of edema, improvement in blood supply, rapid formation of granulation tissue, reduction in bacterial count, and reduction in wound size. It is an excellent technique to prepare the wound for definitive closure.

Bite wounds

The common bite wounds are secondary to dog, human, and cat bites. In zoo keepers, exotic animal bites are seen. All bite wounds should be thoroughly

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**Table 5**

<table>
<thead>
<tr>
<th>Type of wound</th>
<th>Antimicrobial of choice</th>
<th>Adult dose (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Human bite seen within 1 – 2 days</td>
<td>Penicillin or ampicillin (alternative: erythromycin)</td>
<td>0.5 – 1.0</td>
</tr>
<tr>
<td>Human bite older than 2 days</td>
<td>Dicloxacillin (alternative: erythromycin)</td>
<td>0.5</td>
</tr>
<tr>
<td>Animal bites</td>
<td>Ampicillin (penicillin for cat bites) (alternative: tetracycline)</td>
<td>0.5</td>
</tr>
<tr>
<td>Other wounds</td>
<td>Dicloxacillin (alternative: erythromycin)</td>
<td>0.5</td>
</tr>
</tbody>
</table>

These recommendations apply only to wounds without evidence of infection at the time of examination.

* Four times daily orally for 3 to 5 days.

debrided and irrigated, or excised when necessary and possible. Most bite wounds can be closed after debridement. Human bite wounds are infection prone and are better treated by delayed primary closure after thorough debridement. In all bite wounds and other puncture wounds involving the hand, one should look for penetration of the joint space. Radiographs of the hand are helpful. All bite wounds should be considered tetanus prone, and appropriate tetanus prophylaxis should be administered (see Table 4). In the United States, carnivores and scavengers, including rodents, rabbits, and squirrels, are the animals most likely to transmit rabies. The decision to give antirabies prophylaxis is based on the animal and the circumstances of the bite. The rabies prophylaxis guide is shown in Tables 6 and 7.

Edema control

Compression therapy is an essential part of the local treatment of acute wounds in the dependent areas. It is very applicable in both the upper and lower extremities, immediately following wound debridement and skin-graft procedures. Compression should be avoided whenever a local or free flap is used, because it may compromise circulation to the flap. Once the wounds are completely healed, compression garments help prevent hypertrophic scars and associated secondary deformities.

Rehabilitation and prevention

Physical therapy should be involved as early as possible in managing acute wounds involving the extremities and joint surfaces. Early ambulation, early restoration of full range of motion to the involved extremity, and prevention of joint contractures are paramount. Trauma prevention by patient and family education should not be neglected. Involvement of the social services in the diagnosis and

Table 6
Rabies postexposure prophylaxis guide

<table>
<thead>
<tr>
<th>Animal species</th>
<th>Condition of animal at time of attack</th>
<th>Treatment of exposed person(^{a})</th>
</tr>
</thead>
<tbody>
<tr>
<td>Domestic: dog and cat</td>
<td>Healthy and available for 10 days of observation</td>
<td>None, unless animal develops rabies(^{b})</td>
</tr>
<tr>
<td></td>
<td>Rabid or suspected rabid</td>
<td>RIG(^{c}) and HDCV(^{d})</td>
</tr>
<tr>
<td></td>
<td>Unknown (escaped)</td>
<td>Consult public health officials.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>If treatment is indicated, give RIG(^{c}) and HDCV(^{d})</td>
</tr>
<tr>
<td>Wild: skunk, bat, fox, coyote, raccoon, bobcat, and other carnivores</td>
<td>Regard as rabid unless proved negative by laboratory tests(^{e})</td>
<td>RIG(^{c}) and HDCV(^{d})</td>
</tr>
<tr>
<td>Other: livestock, rodents, and lagomorphs (rabbits and hares)</td>
<td>Consider individually. Local and state public health officials should be consulted on questions about the need for rabies prophylaxis. Bites of squirrels, hamsters, guinea pigs, gerbils, chipmunks, rats, mice, other rodents, rabbits, and hares almost never call for antirabies prophylaxis.</td>
<td></td>
</tr>
</tbody>
</table>

These recommendations are only a guide. When applying them, take into account the species involved, the circumstances of the bite or other exposure, the vaccination status of the animal, and the presence of rabies in the region. Local or state public health officials should be consulted if questions arise about the need for rabies prophylaxis.

**Abbreviations:** ARS, antirabies serum, equine; DEV, duck embryo vaccine; HDCV, human diploid cell rabies vaccine; RIG, rabies immune globulin.

\(^{a}\) All bites and wounds should immediately be cleansed thoroughly with soap and water. If antirabies treatment is indicated, both RIG and HDCV should be given as soon as possible, regardless of the interval from exposure.

\(^{b}\) During the usual holding period of 10 days, begin treatment with RIG and vaccine (preferably with HDCV) at first sign of rabies in a dog or cat that has bitten someone. The symptomatic animal should be killed immediately and tested.

\(^{c}\) If RIG is not available, use ARS. Do not use more than the recommended dosage.

\(^{d}\) If HDCV is not available, use DEV or other rabies vaccine. Local reactions to vaccines are common and do not contraindicate continuing treatment. Discontinue vaccine if fluorescent antibody tests of the animal are negative.

\(^{e}\) The animal should be killed and tested as soon as possible. Holding for observation is not recommended.

management of adult and child abuse cases is not to be ignored [63].

Summary

The most important factors in the management of acute wounds are the history and physical examination. The goals of wound care are fivefold: avoid further tissue damage, achieve wound closure as rapidly as possible, restore function to the injured tissue, facilitate the patient’s expedient return to normal daily activities, and restore the patient’s quality of life. The treating physician must have a good understanding of the wound healing mechanism. One must rule out all associated occult injuries that may be life threatening. Proper wound assessment and management with minimal discomfort to the patient are crucial. The primary goal is to facilitate the healing process to achieve a cosmetically pleasing and functional result.

References

[37] Lawrence WT, Talbot TL, Norton JA. Preoperative or postoperative doxoxuridine hydrochloride (Adriamycin): which is better for wound healing? Surgery 1986;100:9–12.