Proper Handling of Soft Tissue in the Acute Phase

Joseph Leach, M.D., FACS[1]

ABSTRACT

Proper handling of soft tissue in the acute phase demands proper attention to fundamental principles in wound evaluation, preparation, and repair. Foundational to immediate wound care is appropriate hemostasis and anesthesia. Correct soft tissue cleaning techniques are important, and the surgeon should understand special situations in which contamination may be a problem, including bites. The controversial issue of antibiotic prophylaxis is discussed. Certain wounds are not amenable to closure, but, where closure is indicated, orientation and preparation of the repair are important. Regional considerations important to the face are addressed. Numerous methods of wound closure are now available, and the advantages of each are enumerated. Postoperatively, attention to patient counseling, dressings, ointment, cleaning, and scar revision help assure an optimal outcome for the traumatized tissue.

KEYWORDS: Laceration, wound, infection, cleaning, closure

INITIAL EVALUATION

When called to evaluate a soft tissue injury in the acute setting, the physician should set priorities when evaluating the patient. In most situations, the specialist in soft tissue repair will consult after another practitioner has triaged the patient. Nevertheless, before handling the soft tissue injury, it is a good idea for the physician to perform a cursory examination of the entire patient, getting a general impression of the condition of the airway, breathing, and circulation. A rapid general neurological assessment is in order, with particular attention to cervical spine and cranial integrity. As the surgeon assesses the mental status of the patient, he or she will be able to determine the ability of the patient to cooperate, the need or ability to use sedating agents, and if further neurological evaluation is needed. Slurred speech, flushed skin, or combativeness may be one of many signs indicating the presence of drugs or alcohol. In some instances, one may consider a drug screen and then either waiting until the patient is less combative or getting extra assistance. After the patient has been stabilized, one should take photographs of the wound for adequate documentation.

The head and neck area demands special consideration in the acute phase of injury. Trauma to the neck may cause laryngeal injury, which may manifest as stridor, laryngeal pain, or crepitus. There are several protocols for the evaluation and treatment of penetrating injuries to the neck, face, and temporal bone that are beyond the scope of this article. When there is suspicion that a deep critical structure is compromised, such as the upper aerodigestive tract or a vital neurovascular structure, one should follow the appropriate protocol. Even in the absence of deeply penetrating injury, trauma to the peripheral nerves must be considered. In particular, one should evaluate the seventh cranial nerves prior to the use of local or general anesthetic. Having the patient attempt to grimace adequately indi-

Facial Plastic Surgery, Volume 17, Number 4, 2001. Address for correspondence: Joseph Leach, M.D., FACS, Department of Otolaryngology-Head and Neck Surgery, University of Texas Southwestern Medical Center, 5323 Harry Hines Boulevard, Dallas, TX 75390-9035. [1]Department of Otolaryngology-Head and Neck Surgery, University of Texas Southwestern Medical Center, Dallas, TX. Tel: +1(212) 584-4662. 0736-6825,p;2001,17,04, 227,238,ftx,en;fps00401x.

cates the status of the facial nerve. If underlying bone injury is suspected, the surgeon should obtain appropriate studies to evaluate the extent of the fracture prior to extensive soft tissue repair. At times, it is more expedient to delay repair of the soft tissue injury until the facial fractures are addressed. In specific cases where there is a high-energy ballistic or avulsive facial injury, the bone should be immediately stabilized in its anatomic position. If possible, one should primarily close the existing soft tissue and debride any necrotic tissue. With large amounts of tissue loss, definitive early reconstruction of soft tissue loss with regional or microvascular flaps may be necessary.

PREPARING THE WOUND

While stabilizing and properly evaluating the patient, one should consider the need for hemostasis. When assessing a bleeding head or facial wound, simply elevate the head and neck. This will allow application of a pressure dressing. In cases where there is profuse bleeding, it is best to avoid indiscriminate clamping inside the wound. Clamping could damage an important structure such as the facial nerve or the parotid duct. Another useful method of controlling bleeding is to apply a local anesthetic containing epinephrine. This vasoconstrictive agent limits the amount of oozing from the wound. The anesthetic, typically lidocaine, allows the wound to be cleaned and inspected carefully. If profuse, uncontrolled bleeding persists despite elevation of the head, application of a pressure dressing, and instilling an epinephrine-containing agent into the wound, then other options exist. At times, hemostasis may require packing of the wound and either surgical exploration under general anesthesia or control using angiography. In rare instances, vessels in the neck need to be ligated.

ANESTHESIA

Lidocaine, ranging in strength from 0.5 to 2%, is probably the most popular local anesthetic. Lidocaine has a rapid onset of action, an acceptable duration of effectiveness, a wide margin of safety, and a low incidence of allergic sensitivity. The local agent is usually injected at the wound edge to avoid any further discomfort. It is best to use gentle pressure and a small (25 gauge or smaller) needle. Cooling the injection site with ice, buffering the agent with 10% sodium bicarbonate, or warming the anesthetic may also reduce pain at injection. Anesthetizing the wound before cleaning will allow more effective preparation. Lidocaine is usually administered with epinephrine, 1:100,000. The vasoconstrictive agent aids in hemostasis, allows a greater amount of anesthetic to be given, and prolongs the duration of the anesthetic. It has been postulated that potent vasoconstrictors such as epinephrine could cause a substantial reduction in blood supply and even necrosis, especially in areas where vascular perfusion is compromised.[1] Although epinephrine-containing anesthetics have been used successfully in all areas of the face, one might restrict their use in cases where tissue monitoring is critical or where extensive undermining of skin and soft tissue is necessary. In these cases, blanching due to undue tension or vascular compromise would be masked. Patients on beta-blockers cannot oppose alpha-receptor stimulation, and a hypertensive crisis from the epinephrine in the anesthetic solution is possible.[2] Epinephrine increases the incidence of infection in contaminated wounds in animal studies.[3] This effect is thought to be due to vasospasm. Epinephrine should therefore not be used to enhance anesthesia in contaminated wounds. Longer-lasting local agents, such as bupivicaine, may provide anesthesia for several hours. Bupivicaine has a somewhat smaller margin of safety than lidocaine, but it may be considered for the rare circumstance where repair of soft tissue injuries to the face would take several hours under local anesthesia. At times, it is best to avoid injecting local anesthetic directly into the wound. The anesthetic agent might distort an important local landmark such as the vermillion border, the hairline, or the eyebrow. In these cases, one should administer a regional block to the appropriate nerve or nerves. The supraorbital, supratrochlear, infraorbital, mental, and external nasal nerves are the branches most commonly blocked.

The use of topical anesthetic agents either in solution or gel form in the repair of soft tissue injuries has been growing in popularity over the last decade. Topical anesthetics have many potential benefits. Pain at injection sites is avoided, patient cooperation should be better, and tissue distortion from anesthesia should not be an issue. A solution containing 0.5% tetracaine, 1:2000 adrenaline, and 11.8% cocaine (TAC) has been used in the repair of lacerations since the 1980s and has been shown to be as efficacious as lidocaine infiltration.[4] Recent studies have compared the efficacy of TAC with LAT (4% lidocaine, 1:2000 adrenaline, and 0.5% tetracaine).[5,6] LAT proved itself to be as effective as TAC but also far less expensive and without the toxicity or security concerns of the cocaine mixture. Another topical agent, tetracaine-lidocaine-phenylephrine, was compared with lidocaine infiltration during repair of mucous membrane lacerations in children.[7] In this study, lidocaine infiltration performed significantly better than the topical agent. The studies cited previously were carried out in children and in uncomplicated lacerations. Whether topical anesthetics can be applied more universally to more complicated lacerations in the head and neck has yet to be studied.

Sedating children can ease laceration repair by reducing physical and psychic discomfort. Sedation does effectively reduce the amount of anxiety perceived

by both patient and parent.[8] On the other hand, sedatives increase costs, demand greater monitoring, and prolong emergency room stays.[8] Significant adverse reactions from sedatives are unusual, but the potential for serious airway or hemodynamic complications exists. Ketamine given intramuscularly has proven itself effective, as has rectal, oral, and nasal midazolam.[8,9] On occasion, it is best to perform the repair of a soft tissue injury under general anesthesia. Large, complicated lacerations in children demand ideal lighting and good cooperation. When the condition of the parotid duct or facial nerve is doubt, general anesthetic affords the surgeon the best opportunity for successful exploration. Occasionally, the patient must be transported emergently to the operating room for repair of other injuries, and the facial surgeon may execute definitive repair of the soft tissue in that setting.

CONTAMINATION AND INFECTION

There are several factors that influence wound healing, including nutrition, oxygenation, and coexisting diseases. The factor that is most under the physician's control at the time of injury, however, is infection. Infection decreases the partial oxygen pressure and increases collagenolysis.[10] Good soft tissue care minimizes the likelihood of infection by proper cleaning and good surgical technique. Typically, "clean" wounds include lacerations less than 8 hours old and surgical defects. "Dirty" wounds are those greater than 8 hours old. Conventional wisdom says that wounds older than 8 hours probably should not be closed primarily. Some areas of the body are more resistant to infection after soft tissue injury. This resistance is probably due to the increased vascularity of these areas, which results in a high oxygen tension in the wound. In the face and scalp, due to their excellent blood supply, wounds as old as 24 hours old may be closed primarily with little risk of infection.[11] Infections are uncommon when the wound is closed so that no dead space, devitalized tissue, or foreign bodies are left under the skin and the skin edges themselves are not buried.

Although it is probably true that nearly all practitioners would advocate cleaning a wound prior to closing, Hollander et al.[12] have called into question the notion that all lacerations need to be *irrigated* before repair. They cite the fact that there is no evidence that irrigation is beneficial in noncontaminated wounds and that irrigation may paradoxically increase the susceptibility to infection by expanding the volume of devitalized tissue. In a study of noncontaminated lacerations to the face and scalp, the incidence of wound infection was not significantly different between lacerations that were irrigated before primary closure and those that were not.[12] Irrigation had no impact on the eventual cosmetic appearance of the scar. Patients were excluded from the study if they had various systemic diseases or if they presented for treatment longer than 6 hours after the injury. Patients were also excluded if the wound was grossly contaminated or involved injury to fascia, muscle, or bone. Wounds that were not irrigated were cleansed with normal saline and gauze.[12] In older wounds or in those that are grossly contaminated, irrigation should be carried out. A 20-gauge angiocatheter attached to a large syringe filled with normal saline serves this purpose well. The tip of the angiocatheter can direct a fairly forceful stream of saline into the depth of the wound. If a greater amount of irrigant is needed, one can attach the angiocatheter to IV tubing and a pressurized IV bag. When using a pressure irrigating system on facial tissue it is important to use caution around the eyes. Scrubbing of road tar, soil, dirt, or grease may be necessary for abrasions. Foreign materials such as these produce tattooing if left in the wound.

Antiseptics are not indicated for the disinfection of wounds, although they are useful for prepping the surrounding skin. Antiseptics are minimally effective either in the clean or the contaminated wound because they are inactivated by clotted blood, serum, pus, and foreign debris.[13] They also have harmful effects on leukocytes and wound healing. Antiseptics containing detergents (scrub solutions) are particularly harmful to wounds.[14] In terms of tissue safety and antiseptic capabilities, chlorohexadine gluconate is a better cleaning agent than iodine, alcohol, or hexachlorophene.[15] Hydrogen peroxide is only minimally bactericidal but is toxic to fibroblasts even when diluted to 1:100.[16] For this reason, hydrogen peroxide should not be considered as a first-line wound-cleaning agent, although it is useful in the postoperative period in cleaning scabs and crusts away from incision lines. Perioperative antibiotics are usually not necessary except under certain circumstances. If there is a bite or if the wound is already infected, a broad-spectrum antibiotic such as cephalexin or amoxicillin/clavulanate is usually adequate. A history of cigarette smoking, alcoholism, diabetes, or atherosclerosis has been shown to increase the incidence of wound infection, and antibiotics should be considered for these patients.[17] Antibiotics are probably also indicated for patients who are immunocompromised or have prosthetic heart valves or prosthetic joints.

It is a good idea to consider the tetanus immunization status in any nonsurgical wound and the issue of rabies in an animal bite. If the history of tetanus immunization is unclear or fewer than three doses have been given in the past, one should consider giving tetanus immunoglobulin.[18] Booster injections of tetanus should be given every 10 years. Tetanus-prone wounds are those with heavy contamination from soil or manure, devitalized tissue, or deep puncture wounds. In the tetanus-prone wound, all patients should receive tetanus toxoid unless they have been immunized within

the past 5 years. Patients who have a history of less than two doses of toxoid in the past should be given human tetanus immunoglobulin and toxoid. In the case of rabies, it is important to ascertain the identity of the dog and whether the attack was provoked.[19] The regional incidence of rabies among the domesticated pet population is also considered. For unprovoked bites, city, county, and state health departments can provide data regarding the incidence of rabies and influence the decision to immunize.[20] Most urban areas in the United States are free of rabies, so failure to capture the attacking animal will not require rabies prophylaxis. Rabies prophylaxis is not necessary if the animal is a pet and is known to have no exposure to rabies. In other cases, identification, capture, and quarantine of the animal responsible for the attack is important. If rabies is suspected, the animal should be killed and the brain tissue studied for cytoplasmic inclusion bodies (Negri bodies). If these are present, rabies prophylaxis should be instituted.[21]

BITES

Although many types of animal may bite humans, the dog is the most common culprit. The principles applying to dog bites apply in general to other animals as well. A dog's jaws will not only lacerate tissue but will also devitalize it by crushing, tearing, or avulsing it. There have been over 60 different species of bacteria cultured from dog mouths, and many are potential human pathogens.[19]Aerobic bacterial isolates include alpha- and beta-hemolytic *Streptococcus*, *Staphylococcus aureus*, *Pasteurella multocida*, *Escherichia coli*, and *Proteus*, *Pseudomonas*, and *Moraxella* sp. Anaerobes include the species of *Bacteroides*, *Enterococcus*, *Prevotella*, and *Fusobacterium*.[19,22] The literature is unclear regarding the optimal method of treating animal bites. Controversy persists regarding the role of tissue excision, irrigation, timing of wound closure, use of antibiotics, and use of subcutaneous sutures. All animal bite wounds should be cleaned. Stucker et al.[23] recommended jet lavage with copious amounts of saline. Routine debridement, even of puncture wounds, is probably not indicated and may lead to unnecessary scarring. With fresh bites in healthy patients who lack inflammatory signs and symptoms, antibiotics are probably not necessary,[19,22,24,25] but obtaining a culture at presentation is helpful if antibiotics are indicated later. Although there are no clear guidelines as to how many hours after injury a bite may be closed, the consensus seems to be that, the fresher the bite, the more safely it may be closed. Guy and Zook[24] state that only dog bites less than 6 hours old should be closed. Other authors have found higher rates of infection if treatment was started more than 2½ hours after injury or in patients with advanced age, immunosuppression, alcoholism, or diabetic vascular disease.[26,27] If there are already signs of an infection at the time of treatment or if an infection develops after surgery, dicloxacillin and cephalexin also seem to be effective.[28] Stucker et al.[23] recommend ticarcillin/clavulanate to be given intravenously perioperatively, followed up with amoxicillin/clavulanate for 10 days. Penicillin, ampicillin, doxycycline, and ceftriaxone have also been recommended as empirical antibiotics until a definitive identification is made.[19]

The human mouth contains many aerobic and anaerobic bacterial taxa and can include various fungi, viruses, and even protozoa.[29] Many of these organisms are harmless, but others can be significant pathogens in bite wounds. Two cases have been reported of suspected AIDS transmission through human bites.[30,31] As with animal bites, authors differ on the most effective way to handle human bites. Agrawal et al.[32] repaired 36 patients with major human bite wounds to the face within 2 to 84 hours after injury. Bites that were infected clinically on gross examination were not repaired. The wounds were cleaned with chlorhexadene. No antibiotics were used, and no infections resulted. Donkor and Bankas[33] conducted a study in 30 patients who had human bites to the face in which debridement of nonvital tissue followed by primary closure was used. Patients were prescribed a course of amoxicillin and metronidazole for 7 days. No infections resulted. Chidzonga[34] reported on 22 patients with human bites to the face. The average delay in presenting to the hospital after having been bitten was 2.6 days. Treatment consisted of thorough irrigation with povidone-iodine solution, debridement of nonvital tissue, and primary closure. Ampicillin was given perioperatively. Only one patient, who had presented 14 days after injury with signs of infection already present, developed a postoperative infection.[34] The excellent blood supply of the face, use of antibiotics, and early surgical repair make infection of human bites of the face unusual, even when patients present after significant delay.

ADVANTAGES OF PRIMARY CLOSURE

Primary surgical closure of a wound minimizes the need for epithelial migration, collagen deposition, and wound contraction. With healing by primary intention, epidermal migration covers the scar within 24 hours. Healing by secondary intent relies upon the forces of fibrin clotting, fibroblast migration, collagen deposition, wound contraction, and epithelialization. This process may not be fully completed for months. Such healing is not only lengthy but it has the propensity to allow infection and distortion of local anatomic structures by contraction. As cover is provided for the wound, primary repair also has the tendency to reduce infection and inflammation. This should lead, in addition, to less redness and hyper-

trophy of the eventual scar. Not all soft tissue wounds are amenable to primary closure. Abrasions are treated best by cleaning and covering with ointment. Crush wounds are best treated by minimal debridement and allowing the tissue to heal. Small areas of avulsion should be closed, but larger avulsion injuries may require delayed closure, skin grafting, or flap coverage. Delayed primary closure or "healing by tertiary intent" is indicated for infection, need for wound contraction, or need to create a proper dermal wound bed for skin grafting. High-velocity (>1200 ft/sec) gunshot wounds and shotgun wounds are best closed in a delayed fashion. High-velocity wounds require wide debridement, drainage, and packing. Low-velocity gunshot wounds are best treated with minimal local debridement and primary closure if possible.

DESIGN AND TISSUE EXCISION

Improperly oriented wounds prognosticate a poor result, so the physician should try to close a wound parallel to the relaxed skin tension lines (RSTLs). These lines are those that follow the furrows formed when the skin is relaxed (Fig. 1). Unlike wrinkles, they are not visible. They are best seen when pinching the skin. RSTLs generally run perpendicular to the long axis of the underlying facial musculature, and scars have a better prognosis when they are parallel to these lines. If a laceration or avulsion is ragged but runs along RSTLs, the irregularities can be excised and the wound closed as a straight line. If, however, the wound runs against the RSTL it may be better to close the laceration preserving any irregularities. This has the effect of breaking up the scar into segments and making it less noticeable to the eye.

Figure 1 The relaxed skin tension lines. In general, these parallel the wrinkle lines except in the galbella, the upper crow's feet, and the mentolabial sulcus.

All obviously nonvital tissue should be excised. Such tissue would exhibit extreme blanching or cyanosis. It is best to be conservative in these assessments, however, because the face has a particularly good blood supply and wider excision than necessary carries a risk of increasing the deformity. In macerated or severely contused wounds, it is often difficult to assess tissue viability. Even tissues that appear blue or are on small pedicles may survive. One situation in which it would be wise to excise tissue would be in the case of the long, jagged wound that is oriented parallel to the RSTL. For this purpose, it is best to use sharp iris scissors or an 11 scalpel blade. Trimming wound edges is more difficult than it sounds. It is difficult to stabilize the wound edge to make a precise cut, particularly when using a scalpel. Scissors are easier to use but can cause a scalloping effect. It is best to use an assistant, who can stabilize the wound at either apex by using single hooks to evert the wound edges. When trimming, it is helpful to bevel so that the depths are wider than the surface (Fig. 2). This will cause the wound edges to evert. An exception to beveling is in hair-bearing areas where it is important to align the cut parallel to the hair follicles. When excising, the surgeon should attempt to create an ellipse that is approximately three times as long as its width. This tends to produce angles of about 30 degrees at the apices. If the excision is shorter than that, one is likely to end up with a tissue protrusion or "dog ear" at the apices of the wound (Fig. 3). Another situation where trimming is useful is in the case where the laceration is oblique (Fig. 4). These "trapdoor" injuries partially avulse the tissue with broad tangential lacerations that lift up flaps of tissue. Oblique peripheral edges may be excised and to make them perpendicular to the skin edges. When the laceration is of a curvilinear shape, the result is the so-called trapdoor flap. As the trapdoor flap heals, there is heaping up of the tissue along the concave side of the laceration. This phenomenon is probably caused by a variety of factors, including the centripetal contraction forces of the scar and obstruction of lymphatic and venous drainage. Treatment is best achieved with undermining, beveling the wound edges, and applying a pressure dressing.

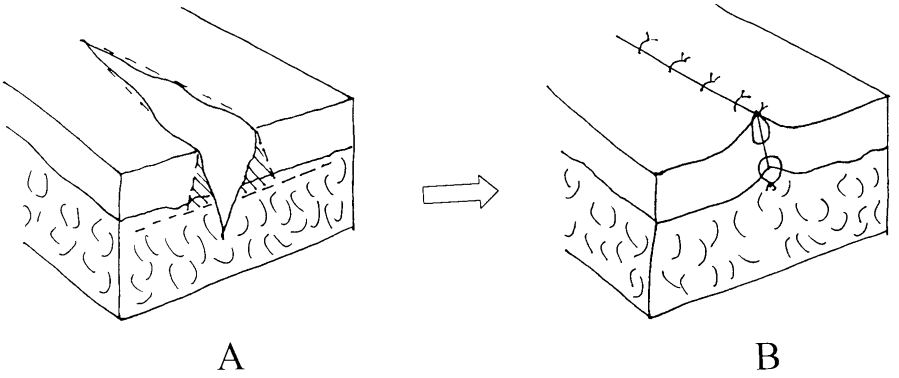

Figure 2 Beveling the tissue to allow for eversion of the suture line. A, Before closure; B, after closure.

UNDERMINING AND DRAPING

Small avulsion injuries may be closed primarily, but they should be closed without tension. Wounds closed under tension tend to heal with more scarring.[35] Elevation of the skin for a distance of 2 to 4 cm beyond the wound edge will reduce the closing tension. Undermining beyond this may compromise the viability of the skin, promote more bleeding, and create more dead space. In general, skin elevation should be carried out in the subcutaneous plane, where there is little potential for injuring underlying nerves. In the scalp or central forehead, however, undermining is easiest deep to the frontalis muscle or galea. Scissors are probably the best instrument for undermining because electrical surgical devices, lasers, and heated scalpels cause significantly greater inflammation and necrotic debris in the early phase of wound healing.[36] In some avulsion injuries there is a discrepancy between the lengths of one side of the wound and the other. In these situations, the extra tissue on the long side of the wound may be distributed along the length of the closure using the halving principle. With this technique, the first deep suture approximates the wound edges at the midpoint. The second and third sutures are placed between the midpoint and ends of the wounds. Remaining gaps are then closed until a flat, even repair is present (Fig. 5). At times it is more expedient to remove a tissue protrusion by excising a Burow's triangle (Fig. 6). This triangle can be removed from any location along the longer edge of the wound, but it is best to hide the excision in a natural camouflage line. The height of the triangle should be sufficient to produce a 30-degree angle at its apex.

Scalp lacerations tend to bleed profusely, and large bleeding vessels that do not respond to the vasoconstrictor in the anesthetic may require ligature or cautery. Shaving is usually not necessary, and surrounding hair is included in the prep. A surgical stapler is ideal for rapid closure of small to moderate lacerations.

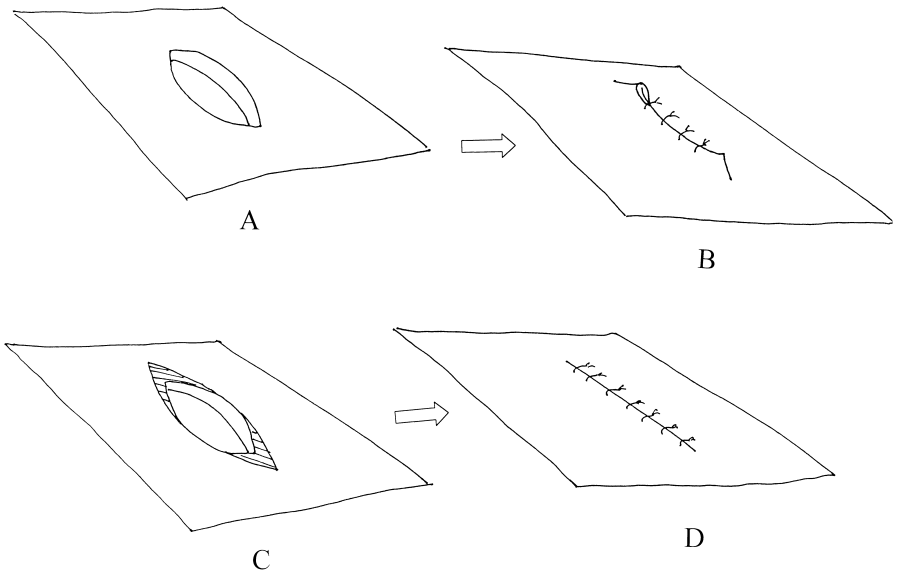

Figure 3 (A, B) Tissue protrusions result when the length of the fusiform defect is less than three times its width. (C, D) Lengthening the defect allows closure without tissue protrusion.

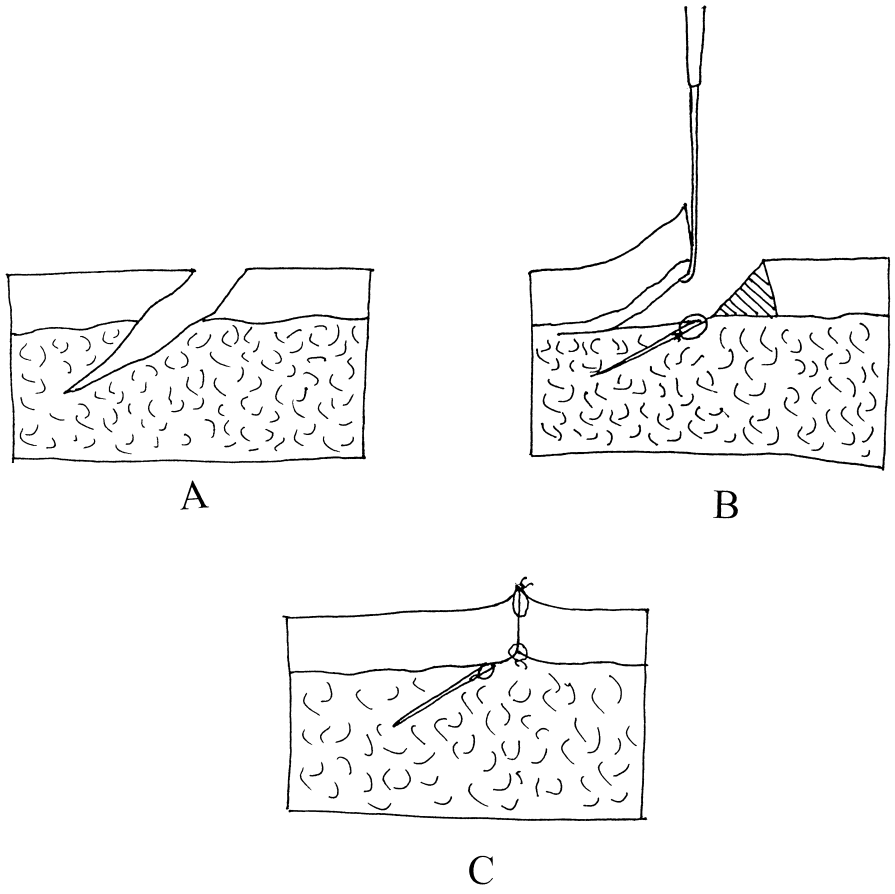

Figure 4 (A) An oblique or "trapdoor" laceration. (B) After, the tissue is undermined and the deep tissues closed, (C) the skin is beveled to allow eversion.

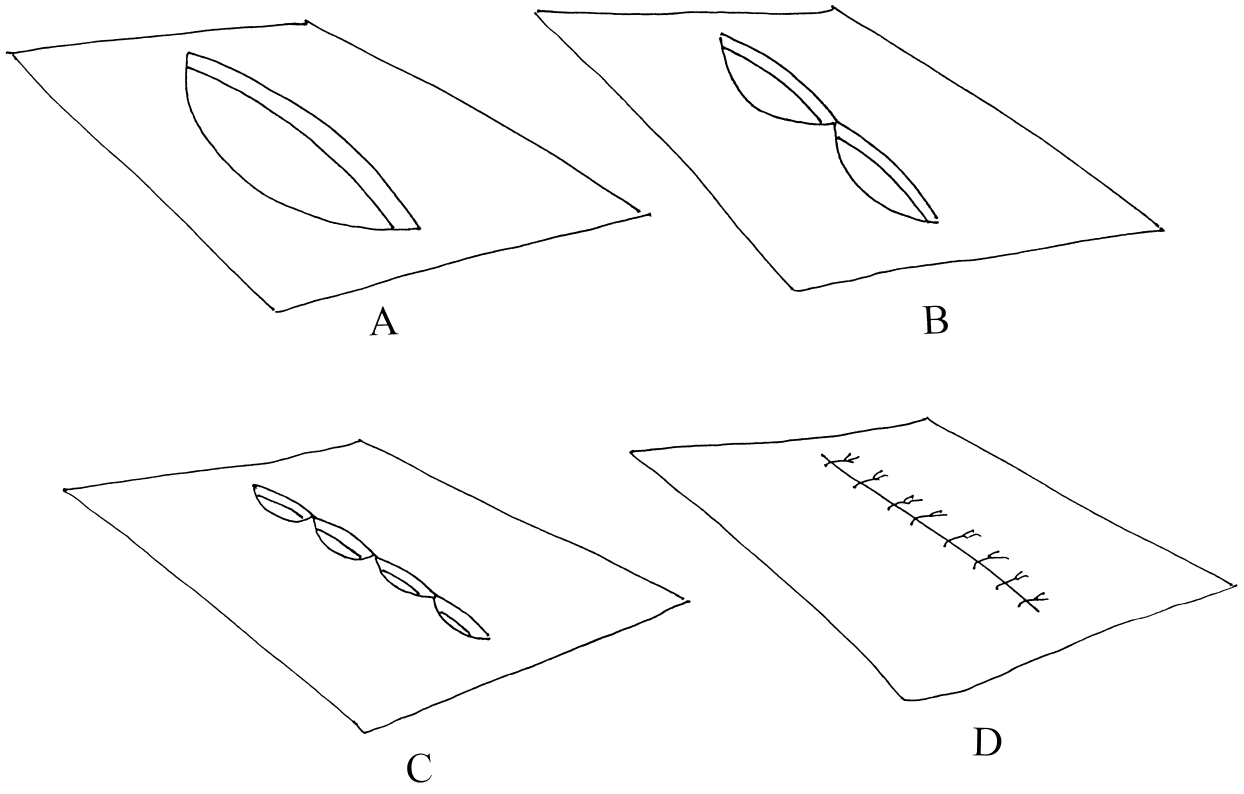

Figure 5 The halving principle. (A) A defect where one side is longer than the other. (B) A suture is placed at the midpoint to distribute the excess of the longer side evenly along the shorter side. (C) The resulting gaps are further "halved." (D) Final closure.

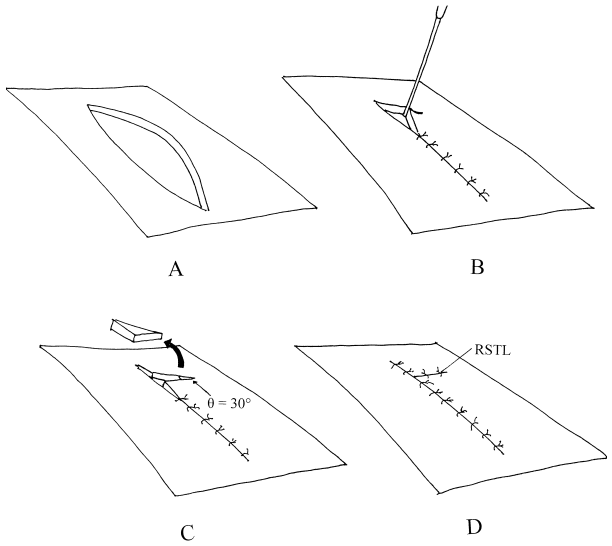

Figure 6 (A) Excision of a Burow's triangle, another method of treating a defect where one side is longer than the other. (B) Tenting excess tissue upward along the longer edge. (C) Excision of a triangle whose apex is 30 degrees. This triangle is centered along an RSTL or wrinkle line. (D) Final result.

Longer lacerations may need a few sutures placed to approximate the galea. Around the eye, it is important to assess the integrity of the lacrimal tracts. Transection of the canaliculi requires stenting for a period of weeks. Eyelid lacerations are closed in two layers. The most important eyelid landmark is the gray line, which is first approximated using a 6–0 silk suture. It is a good idea to leave the tails of the stitch long and bury them under the knots of the more inferior sutures. One may then repair the tarsal plate with fine absorbable sutures. The skin and orbicularis muscle are closed with fine nylon. Small, full-thickness avulsion injuries less than one third of the eyelid can be closed primarily. A lateral canthotomy and undermining will allow closure of defects up to one half the lid width. Around the brow, it is best not to shave the eyebrow because these hairs provide an important landmark.

When partial avulsion injuries to the ear occur, any tissue that is still attached should be preserved and reapproximated, no matter how small the pedicle. On the inside of the mouth, a running 3–0 chromic stitch is best. In the lips, the most significant landmark is the vermillion border, and this structure should be aligned before other stitches are placed. It is important to position a separate layer of stitches at the muscle fat junction on each side to avoid tethering of the lip. Some injuries to the facial nerve require repair and others do not. Transected nerve branches distal to a vertical line running through the lateral canthus usually heal spontaneously and require no neural repair. Injuries to the nerve between this line and the mastoid tip should be explored and repaired. The parotid duct empties at the level of the second upper molar tooth. One can detect transection of the duct by canalizing it through the mouth. Any injury to the duct should be repaired and stented.

SUTURING

Around the face, smaller and finer instruments will serve the surgeon well. Skin hooks are the best instruments for skin-edge handling during wound repair because they prevent maceration of the tissue. Small, single- and double-skin hooks and small Adson forceps are least traumatic. If forceps must be used, they should be approximated gently, as even fine jeweler's forceps will contuse the skin if used with too much force. Needle holders should be of the delicate type. If available, magnification is useful for revealing shelving injuries and cyanosis. Key et al.[1] used high magnification in their series of 100 facial lacerations. This revealed irregularity of wound margins, localized ischemia, or necrosis that was otherwise unapparent.

Closing dead space, skin edge alignment, and skin eversion are basic goals that should be achieved by layered closure. Each anatomic layer that has been violated should be reapproximated separately. Howes and Harvey[37] determined in 1929 that, compared with fat, it took 6 times more force to pull a suture loop out of muscle and 19 times more force to pull a suture out of fascia. Tight suturing of fat can only damage tissue within the suture loop, adding to the load of debris that must be cleared at the cellular level. For this reason, deep sutures are placed in muscle, fascia, and dermis. Burying the knot keeps the bulk of the suture material within the wound. In the dermis and epidermis, a cutting-edge needle will be less traumatic. Deep sutures obliterate dead spaces and prevent tension from developing across the epidermis. Drains are usually not necessary and should be employed in the face only in the instances of hematoma, sialocele, or abscess. Drains are large foreign bodies that can exert substantial damage to the host defenses.[38] They should be used to remove fluids that are in themselves damaging the tissue. Superficially, the skin edges should be aligned perfectly. Eversion is also important because the contractile forces of scarring will draw the wound edges inwardly in the weeks following repair. Horizontal or vertical mattress sutures can be used to evert the skin edges. The more extensive subcuticular loops of the mattress suture may, however, compromise dermal blood supply more than standard interrupted sutures. Placing sutures so that the depth is greater than the width (an egg-shaped configuration) will also help in eversion (Fig. 7). Continuous sutures are probably adequate, but one should beware of blanching in the wound edges or bunching up of the tissues. Running sutures are easier to remove and faster to put in. Irregularity of lacerations usually prevents the use of a subcuticular closure. Skin sutures that blanch

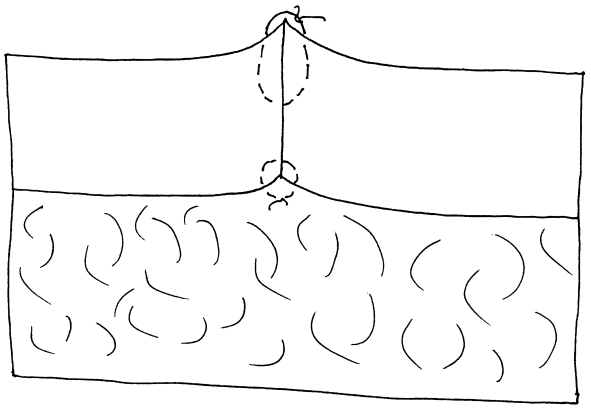

Figure 7 Placing sutures that are relatively wider at the depths than at the surface (the "egg-shaped" suture) will help evert tissue.

the skin are too tight. They should be tightened only until they lie gently on the surface. Overly tight sutures will increase the incidence of wound infection.

The material used to close a wound should elicit minimal inflammation. It should not increase the potential for infection, and it should retain its strength for an adequate period of time. The reactivity of suture material seems to be more of an issue for deep sutures. Even the least reactive suture impairs the ability of the wound to resist infection.[39] Polyglycolic acid (dexon) has been shown to cause less inflammation and potential for infection than plain and chromic gut sutures.[40] Only enough suture material to securely close a wound is necessary. More suture loops increase the risk of wound infection.[41] The configuration of the suture plays a lesser role in the development of early infection that does the chemical ingredient.[40] The infection rates in contaminated tissues with monofilament nonabsorbable sutures were higher than those closed with multifilament sutures made from the same material, but these differences were not statistically significant.[39] Subcutaneous sutures should approximate the dermal edges for about 3 weeks, until collagen formation has provided enough strength for the wound. Sutures that last much longer than this may extrude or become infected. Polyglactin (vicryl) and polyglycolic acid (dexon) sutures lose their strength in about 30 days.[42] More variable in their tensile strength retention are gut (50% at 15 days) and chromic gut (50% at 30 days).[41] Fast-absorbing gut loses 50% of its strength at 5 to 10 days AT. Polydioxanone loses strength in 56 days.[42] Based upon these data, polyglactin, polyglycolic acid, and chromic gut would seem to provide adequate tensile strength for the appropriate time. In the face, where tissue layers are not subject to vast amounts of tension, 4–0 suture is adequate for muscle and fascia repair. In the superficial layers, the material used for closure is not as critical. In fact, there have been no studies that clearly reveal any consistent clinical differences for skin wounds closed with sutures, staples, tape, or glue.[41,43] In a study of superficial facelift sutures, polypropylene and gut were found to cause the same amount of scar hypertrophy, erythema, infection, suture marks, and tissue necrosis.[44] Superficial closure requires fine approximation of the epidermis and is best accomplished with a 5-0 or 6-0 suture. Studies have reported that staples and sutures are either equivalent or the staples better in wound-closing characteristics.[45,46] In the face, however, the size of conventional staples makes it difficult to finely coapt the superficial layers. Surgical tapes (steri-strips) are useful in approximating simple superficial wounds without tension, but they can cause skin irritation.[41]

Recently, tissue adhesives have been found be an effective alternative to suturing in selected superficial facial lacerations. The adhesive is supplied as a liquid that is simply dropped onto the surface of the opposed skin edges. Each layer is allowed to dry, and three layers are recommended. Osmond et al.[43] studied the three most commonly used methods of repairing pediatric facial lacerations: nondissolving sutures, dissolving sutures, and tissue adhesive. Tissue adhesive was not only the least expensive modality but it was also the fastest to perform. Tissue adhesives were ranked first in patient preference, whereas nondissolving sutures ranked last.[43] Simon et al.[47] compared the cosmetic results of tissue adhesive versus conventional suturing. In lacerations parallel to Langer's lines, there was no difference between the groups. In lacerations running contrary to Langer's lines, on the other hand, the appearance was significantly worse for sutured lacerations.[47] Parents perceived that their children experienced less pain with tissue adhesive than with conventional suturing. In addition, the procedure took less time and the need for follow-up was eliminated. Osmond et al.[48] compared butylcyanoacrylate (hystacryl blue) and octylcyanoacrylate (dermabond) in the treatment of 4-mm superficial linear traumatic facial lacerations in children. The two groups were similar for baseline demographic and clinical characteristics. There was no difference in the 3-month cosmesis, time to complete the procedure, or pain of the procedure. The superior physical properties of octylcyanoacrylate appeared to add little benefit to the management.[48] Octylcyanocrylate is four times stronger, polymerizes more slowly, and releases less heat, which should theoretically cause less pain.[48] After polymerizing, adhesives become brittle. They should therefore not be used in skin creases or over areas of movement. In short, noncontaminated superficial facial lacerations in children, tissue adhesives will likely play an increasing role in the future.

FOLLOW-UP

After wound repair, the patient should be counseled to elevate the head above the heart to minimize edema and hematoma formation. It is a good idea to educate pa-

tients about the process of wound healing. For instance, after the sutures are removed, the scar may look acceptable but then may later become red, thick, raised, and lumpy. This phase may last for 3 to 12 months. The patient should also be warned about any residual scarring that may need revision later. Sunlight exposure is another issue to be discussed. Hyperpigmentation may occur with even minimal amounts of sunlight exposure within the first 60 days after repair. A broad brimmed hat and/or sunblock are recommended if the patient must spend any time outdoors. Re-epithelialization begins a day or two after wounding,[49] so the laceration may be washed relatively quickly. Noe and Keller[50] performed a study of 100 patients who had their wounds closed with nylon suture. The patients washed their wounds with warm soap and water twice a day beginning the morning after surgery. All healed without any disruptions, infection, or dehiscence of the wound. Staples and stitches should be taken out at 5 to 7 days to avoid punctate scars or "train tracks." At 7 to 10 days collagen cross-linking is sufficient to allow the wound to tolerate early controlled motion with little risk of disruption.[1] After 21 days the mass of collagen within the wound will reach a maximum and, along with the ingrowth of new blood vessels into the wound, will produce a typical bulky red scar.[1] Small amounts of a long-acting corticosteroid may be injected into the scar at this time to improve the appearance.

In the immediate postoperative phase, it is best to cover the wound either with ointment or some form of dressing. A scab consists of necrotic cells, fibrin, and blood products. Epidermal cells must mechanically loosen any scab with proteases, including elastase and collagenase. A scab, therefore, retards healing.[10] Occlusive dressings and ointments help prevent scab formation. Occlusive dressings allow rapid epithelialization, reduced wound pain, reduced fibrosis, reduced infections, and better cosmetic results.[51,52] The amount of necrotic and fribrinous material in occluded wounds is less than in nonoccluded wounds. A moist wound environment is more favorable in preventing dehydration of tissue, and it promotes angiogenesis, the interaction of growth factors and target cells. Fibroblasts and inflammatory cells are reduced in occluded wounds, which may explain reduced scar formation.[53] Three types of dressing are appropriate in the acute phase: polyurethane film (Op-site, Acme United Corp., Bridgeport CT; Tegaderm 3M, Eagan, MN), hydrogels (Vigilon, Bard Home Health, Berkeley Heights, NJ), and perforated plastic films (Band-Aid, Johnson & Johnson, Skillman, NJ).[54] Polyurethane film is rarely used on the face but is ideal for superficial abrasions. Because it lacks absorptive capacity, fluid accumulation under the film and leakage around the film are problems. The hydrogels consist of polyethylene oxide wafers containing 95% water sandwiched between two polyethylene films. They are used for dermabrasion wounds because they are both soothing and have absorptive properties. The hydrogels promote gram-negative bacterial growth, so an antibiotic ointment should be applied in conjunction.[55] The perforated plastic film has an absorptive pad backing that also makes it attractive for exudative wounds. A recent study by Thomas et al. compared hydrogels and dry gauze.[56] Wounds treated with the hydrogel demonstrated improved comfort and better cosmetic qualities at day 5, but by day 28 there were no significant clinical differences between the groups. A similar range of microorganisms was noted on the dressings.[56]

The necessity of applying a dressing or ointment beyond day 5 for inpatients with primarily closed facial wounds is debatable. After primary closure, epithelialization is virtually complete at 2 days. In the first 48 hours, the antibiotics in the ointment are used to prevent bacteria from entering the wound.[57] Because allergy, sensitivity, and resistant organisms are concerns with antibiotic ointments, simple petrolatum may be just as effective between days 2 and 5. Neomycin is particularly prone to cause skin sensitivity. Antibiotics are often combined together to provide a broad spectrum of antimicrobial coverage. Neomycin is effective against staphylococcal and most gram-negative bacilli except *P. aeruginosa* and obligate anaerobes.[58] Bacitracin is effective against staphylococci, streptococci, and gram-positive bacilli. Polymyxin B is active against aerobic gram-negative bacilli, including *P. aeruginosa* but not against gram-positive bacteria.[58] Polymyxin B can be nephrotoxic, neurotoxic, and ototoxic when applied over large areas.[59] Mupirocin ointment (Bactroban) has a vehicle that retards healing.[60]

Pressure in the form of surgical strips or a small dressing will help guard against the production of hypertrophic scar. Pressure may be applied for as long as 6 months. A study of 11 patients with sternal scars reported that half the scar was treated with 40 mg/ml triamcinolone injections and the other half treated with silicone gel sheets worn 12 hours per day for 12 weeks.[61] Although both techniques were effective, most of the patients preferred the silicone gel. Studies have shown that the beneficial effects of silicone sheets are not due to pressure, temperature, oxygen tension, or capillary occlusion.[62] The disadvantage of the sheet is that it is difficult to adhere to irregular surfaces. A hydrogel dressing has also been compared with silicone gel.[63] Comparable clinical improvement of the hypertrophic scars was obtained with both dressings, so it is apparently true that silicone is not a necessary component of occlusive dressings in the treatment of hypertrophic scars. During occlusive dressing therapy, there is molecular evidence for extensive connective tissue remodeling.[63] A dressing may exert its beneficial effect through hydration and occlusion, or it may affect the mediators that control fibrogenesis and fribrinolysis.[63]

Scar revision is a consideration after several months, but some centers are performing early revision of scars with selective dermabrasion or laser abrasion techniques. Dermabrasion of facial scars 4 to 8 weeks after injury frequently completely eliminates visible evidence of scarring.[64] In a study where half the scar was dermabraded and the other half treated as a control, dermabrasion at 8 weeks showed significantly greater improvement.[65] In another study, electron microscopic and immunohistochemical studies were performed on punch biopsy specimens before and after dermabrasion at 6 to 8 weeks.[66] There was an increase in collagen bundle density and size, with a tendency toward unidirectional orientation of fibers parallel to the epidermal surface AV. There is evidence that dermabrasion alters the events of primary scar formation by modifying extracellular ligand expression, thereby influencing epithelial cell interaction and reorganization of connective tissue.[64] The high-energy, pulsed CO_2 laser has demonstrated similar results. With the laser, however, the resurfaced halves of the scar showed less bleeding and crusting in comparison with the dermabraded halves.[66] There was no significant difference in terms of re-epithelialization time, postoperative erythema, and eventual clinical appearance.[66]

CONCLUSION

During a soft tissue injury, events are occurring that will affect the long-term appearance of the scar. Obviously, these events are beyond the control of the practitioner. Nevertheless, one's goal should be to make the best of the situation at hand so that the optimal outcome is achieved. The physician does the patient a great service by obtaining hemostasis, preventing infection, and making the patient comfortable. By orienting the repair properly, aligning the wound edges, and everting the tissue, the surgeon assures that the best possible scar will result. The period immediately following the repair is also important, as one guides the patient through the nuances of the early stages of wound healing. Being cognizant of the latest techniques and wound care products will help assure an acceptable result.

REFERENCES

1. Key SJ, Thomas DW, Shepherd JP. The management of soft tissue facial wounds. Br J Oral Maxillofac Surg 1995;33:76–85
2. Foster CA, Aston SJ. Propranolol-epinephrine interaction. A potential disaster. Plast Reconstr Surg 1983;72:74–77
3. Magee C, Rodeheaver GT, Edgerton MT, et al. Studies of the mechanisms by which epinephrine damages tissue defenses. J Surg Res 1977;23:126–131
4. Pryor GJ, Kilpatrick WR, Opp DR. Local anesthesia in minor lacerations: topical TAC vs lidocaine infiltration. Ann Emerg Med 1980;9:568–571
5. Ernst AA, Marvez E, Nick TG, Chin E, Wood E, Gonzaba, T. Lidocaine adrenaline tetracaine gel versus tetracaine adrenaline cocaine gel for topical anesthesia in linear scalp and facial lacerations in children aged 5 to 17 years. Pediatrics 1995; 95:255–258
6. Ernst AA, Marvez-Valls E, Nick TG, Weiss SJ. LAT (lidocaine-adrenaline-tetracaine) versus TAC (tetracaine-aderenaline-cocaine) for topical anesthesia in face and scalp lacerations. Am J Emerg Med 1995;13:151–154
7. Smith GA, Strausbaugh SD, Harbek-Weber C, Cohen DM, Shields BJ, Powers JD. Tetracaine-licocaine-phenylephrine topical anesthesia compared with lidocaine infiltration during repair of mucous membrane lacerations in children. Clin Pediatr 1998;37:405–412
8. Lawrence LM, Wright, SW. Sedation of pediatric patients for minor laceration repair: effect on length of emergency department stay and patient charges. Pediatr Emerg Care 1998; 14:393–395
9. Connors K, Terndrup TE. Nasal versus oral midazolam for sedation of anxious children undergoing laceration repair. Ann Emerg Med 1994;24:1074–1079
10. Moy LS. Management of acute wounds. Dermatol Clin 1993;11:759–766
11. Dickinson JT, Jaquiss GW, Thompson JN. Soft tissue trauma. Otolaryngol Clin North Am 1976;9:331–360
12. Hollander JE, Richman PB, Werblud M, Miller T, Heggler J, Singer AJ. Irrigation in facial and scalp lacerations: does it alter outcome? Ann Emerg Med 1998;31:73–77
13. Geronemus RG, Mertz PM, Eaglstein WH. Wound healing: the effect of topical antimicrobial agents. Arch Dermatol 1979;115:1131–1134
14. Becker G. Identification and management of the patient at high risk for wound infection. Head Neck Surg 1986;8:205
15. Peterson AF, Rosenberg A, Alatory SD. Comparative evaluation of surgical scrub preparations. Surg Gynecol Obstet 1978;146:63–65
16. Lineweaver W, Howard R, Soucy D. Topical antimicrobial toxicity. Arch Surg 1985;120:267–270
17. Cole R, Robbins K, Cohen J, et al. A predictive model for wound sepsis in oncologic surgery of the head and neck. Otolaryngol Head Neck Surg 1987;96:165–171
18. Orlando M, Saltman R. Manual of Medical Therapeutics, 25th ed. Boston, MA: Little, Brown; 1988:446–448
19. Morgan JP, Haug RH, Murphy MT. Management of facial dog bite injuries. J Oral Maxillofac Surg 1995;53:435–441
20. Krebs JW, Strine TW, Childs JE. Rabies surveillance in the United States during 1992. J Am Vet Med Assoc 1993;203: 1718–1731
21. Corey L, Hattwick M. Treatment of persons exposed to rabies. JAMA 1975;232:272–276
22. Wolff KD. Management of animal bite injuries of the face: experience with 94 patients. J Oral Maxillofac Surg 1998;56: 838–843
23. Stucker FJ, Shaw GY, Boyd S, Shockley WW. Management of animal and human bites in the head and neck. Arch Otolaryngol Head Neck Surg 1990;116:789–793
24. Guy JR, Zook EG. Successful treatment of acute head and neck dog bite wounds without antibiotics. Ann Plastic Surg 1986;17:45–58
25. Kountakis SE, Chamblee SA, Maillard AAJ, Stiernberg, CM. Animal bites to the head and neck. Ear Nose Throat J 1998;77:216–220
26. Callaham M. Dog bite wounds. JAMA 1980;244:2327–2328
27. Maimaris C, Quinton DN. Dog bite lacerations: a controlled trial of primary wound closure. Arch Emerg Med 1988;5: 156–161

28. Callaham M. Controversies in antibiotic choices for bite wounds. Ann Emerg Med 1988;17:1321–1330
29. Schuster GS. Oral flora and pathogenic organisms. Infect Dis Clin North Am 1999;13:757–774
30. News and Notes. Transmission of HIV by human bite. Lancet 1987;2:522
31. Wahn V, Kramer J, Voit T, et al. Horizontal transmission of HIV infection between two siblings. Lancet 1990;336: 502–503
32. Agrawal K, Mishra S, Panda KN. Primary reconstruction of major human bite wounds of the face. Plast Reconst Surg 1992;90:394–398
33. Donkor P, Bankas DO. A study of primary closure of human bite injuries to the face. J Oral Maxillofac Surg 1997;55: 479–481
34. Chidzonga M. Human bites of the face. S Afr Med J 1998; 88:150–152
35. Wray RC. Force required for wound closure and scar appearance. Plast Reconstr Surg 1983;72:380–382
36. Madden JE, Edlich RF, Custer JR, et al. Studies in the management of the contaminated wound. I.V.-Resistance to infection of surgical wounds made by knife, electrosurgery and laser. Am J Surg 1970;119:222–224
37. Howes EL, Harvey SC. The strength of healing wound in relation to the holding strength of the catgut suture. N Engl J Med 1929;200:1285
38. Magee C, Rodeheaver GT, Golden GT, et al. Potentiation of wound infection by surgical drains. Am J Surg 1976;131: 547–549
39. Edlich RF, Panek PH, Rodeheaver GT, Turnbull VG, Kurtz LD, Edgerton, MT. Physical and chemical configuration of sutures in the development of surgical infection. Ann Surg 1973;177:679–687
40. Edlich RF, Rodeheaver GT, Thacker JG, Winn HR, Edgerton MT. Management of soft tissue injury. Clin Plast Surg 1977;4:191–198
41. Reiter D. Methods and materials for wound closure. Otolaryngol Clin North Am 1995;28:1069–1080
42. Capperauld I, Bucknall TE. Sutures and dressings. In: Bucknall TE, Ellis H, eds. Wound Healing for Surgeons. London: Bailliere Tindall; 1984:75
43. Osmond MH, Klassen TP, Quinn JV. Economic comparison of a tissue adhesive and suturing in the repair of pediatric facial lacerations. J Pediatr 1995;126:892–895
44. Guyuron B, Vaughan C. A comparison of absorbable and nonabsorbable suture materials for skin repair. Plast Reconstr Surg 1992;89:234
45. Meiring L, Cilliers K, Barry R, et al. A comparison of a disposable skin stapler and nylon sutures for wound closure. S Afr Med J 1982;62:371
46. Rees TD. Is there a place for the staple gun in plastic surgery? Ann Plast Surg 1978;1:238
47. Simon HK, Zempsky WT, Bruns TB, Sullivan, KM. Lacerations against Langer's lines: to glue or suture? J Emerg Med 1998;16:185–189
48. Osmond MH, Quinn JV, Sutcliffe T, Jarmuske M, Klassen, TP. A randomized clinical trial comparing butylcyanoacrylate with octylcyanoacrylate in the management of selected pediatric facial lacerations. Acad Emerg Med 1999;6:171–177
49. Woodley DT, Chen JD, Kim JP, et al. Re-epithelialization. Human keratinocyte locomotion. Dermatol Clin 1993;11: 641–646
50. Noe JM, Keller M. Can stitches get wet? Plastic Reconst Surg 1987;81:82–83
51. Eaglstein WH. Occlusive dressings. J Dermatol Surg Oncol 1993;19:716–720
52. Field CK, Kerstein MD. Overview of healing in a moist environment. Am J Surg 1994;167(suppl):2S-6S
53. Linsky CB, Rovee DT, Dow, T. Effect of dressing on wound inflammation and scar tissue. In: Dineen P, Hildrick-Smith G, eds. The Surgical Wound. Philadelphia: Lea & Febiger; 1981:191
54. Brown CD, Zitelli JA. Choice of wound dressings and ointments. Otolaryngol Clin North Am 1995;28:1081–1091
55. Mertz PM, Marshall DA, Eaglstein, WH. Occlusive dressing to prevent bacterial invasion and wound infection. J Am Acad Dermatol 1985;12:662–668
56. Thomas DW, Hill M, Lewis MAO, Stephens P, Walker R, von der Weth A. Randomized clinical trial of the effect of semi-occlusive dressings on the microflora and clinical outcome of acute facial wounds. Wound Rep Reg 2000;8: 258–263
57. Schauerhamer RA, Edlich RF, Panek P, et al. Studies in the management of the contaminated wound. VII. Susceptibility of surgical wounds to postoperative surface contamination. Am J Surg 1971;122:74–77
58. Hirschmann JV. Topical antibiotics in dermatology. Arch Dermatol 1988;124:1691–1700
59. Physicians' Desk Reference, 47th ed. Montvale, NJ: Medical Economics Data; 1993:813, 818, 2187
60. Mertz PM, Dunlop BW, Eaglstein WH. The effects of Bactroban ointment on epidermal wound healing in partial thickness wounds. In: Dobson R, Leyden JJ, Noble WC, et al., eds. Bactroban. Princeton, NJ: Excerpta Medica; 1985:211
61. Sproat JE, Dalcin A, Weitauer N, Roberts RS. Hypertrophic sternal scars, silicone gel sheet versus kenalog injection treatment. Plas Reconst Surg 1992;90:988–992
62. Quinn KJ, Evans JH, Courtney JM, Gaylor JDS. Nonpressure treatment of hypertrophic scars. Burns Incl Therm Inj 1985;12:102
63. Ricketts CH, Martin L, Faria DT, Saed GM, Fivenson, DP. Cytokine mRNA changes during the treatment of hypertrophic scars with silicone and nonsilicone gel dressings. Dermatol Surg 1996;22:955–959
64. Harmon CB, Zelickson BD, Roenigk RK, et al. Dermabrasive scar revision. Immunohistochemical and utrastructural evaluation. Dermatol Surg 1995;21:503–508
65. Katz BE, Oca AG. A controlled study of the effectiveness of spot dermabrasion ('scarabrasion') on the appearance of surgical scars. J Am Acad Dermatol 1991;24:262–266
66. Nehal KS, Levine VJ, Ross B, Ashinoff R. Comparison of high-energy pulsed carbon dioxide laser resurfacing and dermabrasion in the revision of surgical scars. Dermatol Surg 1998;24:647–650