FOCAL POINT

Because contraction is a major component of the healing process for open wounds, practitioners should know which factors enhance or inhibit contraction.

KEY FACTS

- Wound contraction is caused by a combination of movement of fibroblasts in collagen and the pulling forces of myofibroblasts on the skin edges, p. 22.
- Appropriate splinting and bandaging can eliminate pressure over wounds in certain areas and are necessary for wound contraction, p. 24.
- Drugs can be used to either stimulate or inhibit wound contraction, p. 26.
- Immobilization of wounds over flexion surfaces and in areas in which movement causes shearing and tension helps prevent wound contracture and impaired wound contraction, respectively, p. 29.
- Topical medications and dressings have been described to stimulate the growth of granulation tissue coverage over exposed bone and thus enhance wound contraction, p. 30.

Wound Contraction: Basic and Clinical Factors

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ABSTRACT: Wound contraction, the centripetal or concentric reduction in size of an open wound, is essential to second-intention healing. Wound contraction is caused by movement of fibroblasts in granulation tissue collagen and pulling forces of granulation tissue myofibroblasts on the skin edges. Contraction can result in complete and normal wound closure; however, abnormalities may cause incomplete and abnormal healing. Numerous factors affect wound contraction, including the amount of skin surrounding the wound; pressure on the wound; and medications, dressings, and surgical procedures that enhance or inhibit wound contraction. Other factors that can affect wound contraction are movement, exposed bone, infection, radiation therapy, and wound shape.

SECOND-INTENTION WOUND HEALING

Second-intention wound healing is the healing of an open wound by contraction and epithelialization.1 This method is generally used to treat wounds that are large, have extensive tissue damage, and are contaminated or infected. Primary closure, delayed primary closure, or secondary closure of these wounds is not advisable.1,2 In many cases, this practical and economical method can be used to attain wound closure as long as adequate wound care is provided.1 This healing method allows progressive, gradual debridement to ensure that only devitalized tissue is removed. It also provides optimal wound drainage.2

GRANULATION TISSUE

The basic components of granulation tissue are fibroblasts, macrophages, capillaries, and collagen.2,3 Fibroblasts in wounds originate from undifferentiated mesenchymal cells in adjacent connective tissue, primarily from the adventitia of small blood vessels. Under the influence of cytokines released by platelets and macrophages, they usually appear about the third or fourth day after injury and
advance into a wound by using the fibrin of the clot as a scaffold along which to grow. The fibroblasts have cytoplasmic extensions along their leading edge called lamellipodia, which adhere to the underlying scaffold of fibronectin-coated fibrin and collagen. By chemotaxis, haptotaxis, and contact guidance, the fibroblasts advance into the wound.2–4 Macrophages phagocytize necrotic tissue and debris. In addition, these cells are multipotential and release a large number of cytokines that direct the repair stage of healing. Macrophages remain active until the repair stage ceases.2–4

The capillaries of granulation tissue originate from the capillaries in the wound. Three days after injury, vascular buds form from preexisting vessels (primarily venules) in the wound. Macrophage- and platelet-derived cytokines and growth factors stimulate this neovascularization. The endothelial cells of these vessels are activated and begin to produce enzymes that break down the vascular basal membrane. Endothelial cells then migrate through the resulting gap and form tubular structures that grow in the direction of the wound. These structures connect with other such structures, leading to vascular loop formation.4–6 Blood flow then begins in the looplike structures. Vessels develop and differentiate, but loops with no blood flow regress.4–6 The new vasculature grows into the wound immediately behind the fibroblasts at a rate of 0.4 to 1.0 mm/day. The endothelial cells produce a plasminogen activation, resulting in fibrinolysis and breakdown of the original fibrin network in the wound. The fibrin is replaced by collagen.2

The fibroblasts deposit new collagen after they first secrete fibronectin and proteoglycans, which constitute an amorphous milieu of ground substance in the wound. This process is necessary before collagen deposition and peaks at 3 to 5 days after injury. At 4 to 5 days, tropocollagen molecules are extruded from the fibroblasts after the amino acids (i.e., proline and lysine) in the amino acid chains of the collagen have been hydroxylated. These molecules aggregate to form immature collagen fibrils near the fibroblast. Molecular bonding of the fibrils results in collagen fibers. The early type III collagen in wounds is quickly replaced by more mature type I collagen. As inter- and intramolecular bonding of collagen molecules continues, wound strength increases; and as the collagen content of the wound increases, the ground substance and fibroblast content decrease.2

The new tissue that is formed constitutes granulation tissue (Figure 1). It has a bright red granular appearance and begins to appear in a wound 3 to 6 days after injury. In small wounds it is present under a scab, whereas in large open wounds its formation is visible. The granular appearance of the tissue comes from the capillary loops or knuckles. Each granule is composed of a raised capillary loop capped primarily by fibroblasts and macrophages. Anastomosis between these new vessels and preexisting vessels within the wound tissue is considerable.2,3 Granulation tissue fills in wound depressions, serves as a barrier against invasion of underlying tissue by surface microorganisms,1 and is the seat of wound contraction.

**WOUND CONTRACTION**

**Clinical Basics**

Wound contraction is the centripetal or concentric reduction in size of an open wound (Figure 2). The full-
thickness skin that surrounds the wound is advanced inward by its margins so that the area to be covered by epithelium is reduced or eliminated. No new skin is formed in the process, which is independent of epithelialization. 2,5,7,8 In dogs, visible contraction usually begins 5 to 9 days after wound infliction. 2 In areas in which skin is loosely attached to underlying structures, wound contraction can result in complete wound closure, even with large wounds. 1,2 The rate of wound contraction varies depending on the wound location. In horses, a 400-mm² skin defect in the flank area has been reported to contract at 0.8 to 1 mm/day, whereas similar lower-limb wounds contracted at 0.2 mm/day. 7 One report 4 states that contraction begins approximately 1 week after injury and progresses at a fairly constant rate of 0.6 to 0.8 mm/day.

Wound contraction stops when contact inhibition occurs as like-tissue cells from the sides of the wound come in contact with each other or when tension from the surrounding skin equals or exceeds the force of contraction. 2,3,7 Restrictive fibrosis can also mechanically impair skin advancement. 1 Although skin surrounding a wound may be lax, contraction may stop before wound edges meet because of poor granulation tissue quality. 3 The presence of exuberant granulation tissue in a wound has been found to impede contraction in horses. 7

To assess wound contraction results, the skin around the wound can be manually pushed or gentle traction can be applied with nontraumatic forceps to determine its degree of laxity. 1,2 Skin tension is best evaluated before the wound has entered the inflammatory stage of healing when edema and enlargement of the underlying tissue occur because these changes impair skin maneuvering over the wound, especially on the limbs. 2 If skin adjacent to the wound is lax enough, unimpeded wound contraction can be expected. If a wound remains partially open when this maneuver is performed, this area may close by epithelial cell migration once contraction is complete. 7 Any deformity (e.g., joint flexion or distortion of the surrounding tissue caused by manipulating the skin edges into apposition) will remain if the wound is allowed to heal by contraction. In such cases, skin grafts or flaps are indicated for wound repair 7 (Table I).

### Cause of Contraction

Until recently, wound contraction has been attributed to the contraction of myofibroblasts. Today, fibroblasts are believed to play a major role in wound contraction. Fibroblasts move into a defect early, proliferate, and lay down collagen. As fibroblasts move within the surrounding collagen, 10,11,3,7 they reorganize it by cell membrane tractional forces, 2,10–14 thus consolidating the tissue into a smaller unit and pulling the skin with it. 10,11 This process could be likened to the tread on a toy tank or caterpillar tractor as it moves on loose carpet. The tread simulates the moving fibroblast cell membrane, and the carpet simulates the collagen-containing wound matrix. In effect, the collagen is being

### TABLE I

<table>
<thead>
<tr>
<th>Wound Type</th>
<th>Reconstructive Procedure</th>
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<tbody>
<tr>
<td>Indolent pocket wound</td>
<td>Following revision of involved tissue</td>
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<tr>
<td>Following excision of overlying nonhealing skin</td>
<td>Possible use of omental flap</td>
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<tr>
<td>Large open wound over a joint flexion surface in which contraction would cause contracture deformity</td>
<td>Skin flap or full-thickness mesh graft</td>
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<tr>
<td>Wound in which contraction will result in contractual deformity of adjacent tissue or structures (e.g., eyelids, anus)</td>
<td>Skin flap or full-thickness mesh graft</td>
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<tr>
<td>Limb wound in which contraction will not heal the wound and delicate epithelium will cover the area</td>
<td>Full-thickness mesh or strip graft</td>
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<tr>
<td>Nonhealing axillary wound in cats</td>
<td>Rotation flap or omental flap followed by closure</td>
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<tr>
<td>Irradiated wound tissue with a compromised blood supply</td>
<td>Skin flap or full-thickness mesh graft after debridement to vascularized tissue or placement of a muscle flap</td>
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*See Pavletic 1 and Swaim and Henderson 2 for detailed information on performing skin grafts and flaps in dogs and cats.*
gradually moved to the wound’s center as the fibroblasts migrate (Figure 3). A recent study of open wounds on rats showed that contraction was caused by fibroblasts; therefore, myofibroblasts were not needed for contraction. Because myofibroblasts are present in wounds, the question arises as to their function. It has been theorized that fibroblasts may develop muscle characteristics in response to demand. Development of these cells is an attempt by the fibroblasts to resist the outward tension on the wound edges (Figure 3). This activity appears to predominate later in wound contraction. Thus fibroblasts and collagen play major roles in wound contraction because of their interaction.

**CLINICAL FACTORS AFFECTING WOUND CONTRACTION**

**Amount of Surrounding Skin**

The amount of skin and its attachment to underlying tissue affects wound contraction. During second-intention healing, an abundance of skin that has loose connective tissue attachments to underlying tissue allows for closure of even large wounds (Figure 4). On dogs and cats, skin is loose and abundant on most of the body, particularly on the neck and trunk. Skin is less abundant on the limbs, tail, and head. With dogs in particular, the quantity and elasticity of skin differ among breeds. In addition, an animal’s physical condition has a bearing on the amount of loose skin that is present. For example, the trunk of a thin animal has more loose skin than does that of an obese dog.

As wounds contract, the surrounding skin is stretched, thinned, and placed under tension; but this state does not persist. Gradually, new collagen is laid down in the dermis, tension is relieved, and the dermis regains thickness. In addition, new epithelial cells are produced in areas under tension until the skin attains full thickness. This procedure is called intussusceptive growth.

Insufficient skin surrounding a wound can present problems for contraction in second-intention healing. In some areas, the skin will contract as far as possible and the remaining wound area will heal with a thin delicate epithelium. These wounds often occur on the limbs and frequently require a skin graft to provide durable tissue (Table I).

In indolent pocket wounds, sufficient skin surrounding a wound and healthy granulation tissue are present; however, wound contraction does not occur. Granulation tissue is present on the dermal surface of skin around the wound and on the underlying muscle fascia; but the two surfaces, which are not attached, form a granulation-tissue-lined pocket. Because the dermal surface tissue fails to bond with the underlying granulation tissue, the skin does not adhere to the wound margins. Thus wound contraction forces cannot advance the skin; however, as granulation tissue on the dermal surface of the skin causes contraction, the skin margins begin to arc or curl under (Figure 5). Epithelial cells from the skin migrate beneath its margin onto the dermal surface. As the granulation tissue matures and collagen deposition increases, circulation decreases and the pocket supports bacterial growth. Closure of indolent pocket wounds requires infection control,
excision of the scarred and often curled-under skin edge, and careful incision of the granulation tissue on the dermal surface of the skin. This process is necessary to preserve cutaneous circulation while allowing the skin to straighten and advance over the wound for closure. In cases in which wounds do not lend themselves to closure in this manner, the use of skin flaps or grafts may be considered (Table 1). Another option would be to use omental flaps to bring vascularized tissue into the area before closure.

**Pressure**

External pressure on a wound can affect wound contraction. Bandage pressure can splint the wound edges, pushing them centrifugally. Planimetric studies have shown that open wounds actually increase in size during the first 7 days compared with the size of the freshly created wound. This increase has been attributed to the pressure of the bandages used in treatment. After granulation tissue forms in the wound, the contractile properties of fibroblasts and myofibroblasts can overcome the bandage pressure and wound contraction can proceed. Rigid cast immobilization of open wounds over areas of potentially maximal movement (i.e., flexion surface of the tarsus) inhibits wound contraction. This inhibition has been attributed to the passive pressure of the cast against the wound. Although rigid immobilization of these wounds in extension could help prevent wound contracture deformities, cast pressure against the wound may impede healing. Thus immobilization should be provided by some means that does not place pressure on the wound (e.g., a rigid splint on the side of the limb opposite the wound; Figure 6).

Bandage pressure on a wound over a bony prominence can interfere with wound contraction. Placing additional padding in a bandage to help prevent pressure over an open wound over a bony prominence can further deter wound contraction. Additional padding in such an area increases pressure when the outer layers of the bandage are applied, especially an elastic bandage material. This pressure counteracts the action of the fibroblasts and myofibroblasts that are trying to cause wound contraction. Therefore, it is better to keep pressure off the area by redistributing it around the wound. Alleviating pressure can be accomplished by using a modified minor donut pad in the bandage. The pad can be made by folding over several layers of soft cast padding (Specialist Cast Padding, Johnson & Johnson, Raynham, MA) and cutting a hole in the center of the pad to accommodate the bony prominence (Figure 7).

Wounds on paw pads can cause wound enlargement. Most pad lac- erations would be sutured; with large pad wounds that do not lend themselves to suturing, however, open wound management is indicated. As pressure is placed on the pads when the animal bears weight, the tissue performs its normal shock-absorbing function and spreads out, causing the edges of the wound to be pushed apart. With an open wound, enlargement can result before granulation tissue forms, thereby counteracting wound contraction. Open paw pad wounds should be bandaged and a splint applied to the paw to minimize pressure on the pads that would interfere with wound contraction. A localized crutch can be applied to the paw. After applying a bandage to a paw, two metal splints—one on the palmar/plantar surface and one on the dorsal surface—should be taped to

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**Figure 5**—Indolent pocket wound. (A) The pocket of the wound is lined with smooth granulation tissue (stippled cross-section of tissue). (B) The skin margin curls under as granulation tissue on the dermal side of the skin contracts.

**Figure 6**—(A) Passive pressure from a cast over an area of potential maximal movement (tarsal flexion surface) inhibits wound contraction (small arrows). (B) Splint immobilization on the extensor surface with a light bandage on the flexion surface and around the splint helps prevent passive pressure.

**Figure 7**—(A) Bandage pressure on a wound over a bony prominence (calcaneal process) inhibits wound contraction (small arrows). (B) A modified minor donut bandage over the bony prominence helps prevent the pressure.

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2 The products and preparations mentioned in this article for wound prevention and treatment are those described in referenced articles. Other common products/preparations may not have the same mode of action or give the described results of the referenced products/preparations.
the outer bandage surface. The cups of the metal splints should face each other and extend beyond the distal end of the bandage about 1 inch. This clam shell bandage/splint places the paw in a toe-dancing posture so weight is not borne on the pads.\textsuperscript{26} The splint allows faster wound contraction (Figure 8). In cases of sutured wounds, the splint helps keep the sutures from tearing through the skin, which may occur when animals—especially large dogs—bear weight on the pad.

\textbf{Medications, Dressings, Irradiation, and Surgical Procedures}

When treating second-intention wound healing, practitioners should know which medications, dressings, irradiation, and surgical procedures enhance wound contraction and which slow wound contraction. In some cases, it is desirable to enhance contraction whereas in others contraction should be inhibited (Table II).

\textbf{Wound Contraction Stimulants/Enhancers}

New medications and dressings have become available or can be prepared (i.e., equine amnion) that stimulate wound healing. Part of the action of these products is to enhance granulation tissue formation and thus wound contraction.

Acemannan is a polydisperse $\beta$-(1,4) acetylated mannan, available in a topical hydrogel (Carravet Wound Dressing\textsuperscript{30}, Carrington Laboratories, Irving, TX) that stimulates macrophages to secrete interleukin-1 and tumor necrosis factor-\alpha. These secretions enhance fibroblast proliferation and angiogenesis (capillaries) in wounds.\textsuperscript{27} These structures are the components of granulation tissue. This medication has been found to stimulate the healing of paw pad wounds by wound contraction and epithelialization when applied as a topical gel or injected intralesionally.\textsuperscript{25} In addition, daily application of freeze-dried acemannan (CarraM Sorb M, Carrington Laboratories) has been shown to stimulate granulation tissue formation over exposed bone, which in turn results in enhanced wound contraction.\textsuperscript{28} This factor is important in treating distal limb degloving wounds.

A glycyl-L-histadyl-L-lysine tripeptide copper complex topical medication is a chemoattractant for mast cells and monocytes/macrophages. Topical tripeptide (Iamin-Vet Skin Care Gel\textsuperscript{21}, Procyte Corp., Redmond, WA) and tetrapeptide-copper–complex medications have advanced open wound healing to include the contraction portion of healing in dogs as compared with untreated controls and placebo-treated wounds, especially during the first 7 days of treatment.\textsuperscript{24} Both acemannan and tripeptide copper complex are active in stimulating granulation tissue formation and thus wound contraction.\textsuperscript{27}

Occlusive hydrogel dressings have been reported to have varying effects on granulation tissue formation and wound contraction. One dressing (Curity Conforma Gel\textsuperscript{30}, Kendall Canada, Petersborough, Ontario, Canada) used on dog limb wounds caused exuberant granulation tissue; despite this, it stimulated wound contraction on the limbs.\textsuperscript{29} Conversely, wound contraction was delayed when a hydrogel dressing (BioDres\textsuperscript{30}, DVM Pharmaceuticals, Miami, FL) was used on truncal wounds in another canine study.\textsuperscript{30} Occlusive dressings retain bacteria that are on the wound surface; therefore, bacteria cannot be absorbed into outer bandage layers and away from the wound. It would be suspected that such an environment would promote the development of wound infection; evidence suggests, however, that this is not the case and that occlusion may reduce the incidence of wound infection.\textsuperscript{31} In ad-
dition, occlusive bandages have been recommended for wounds in the repair stage of healing with a healthy bed of granulation tissue. Such tissue is a barrier against systemic infection. Topical placement of equine amnion on open wounds has been found to enhance wound contraction of trunk wounds on dogs.

The effect of pulsed electromagnetic field radiation on open wound healing has been evaluated. This therapy indicated that early wound contraction occurred in treated wounds. Five days after surgery, untreated control wounds had enlarged, indicating bandage pressure had pushed the edges apart; treated wounds had decreased in size, indicating early formation of contractile elements (i.e., granulation tissue) in wounds.

An adjustable horizontal mattress suture has been described for placing gradual tension on wound edges after granulation tissue has formed and contraction has begun, thus enhancing contraction. The technique has been used primarily on distal limb wounds with placement of a continuous intradermal monofilament suture.

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<tr>
<th>Medication, Dressing, Irradiation, or Procedure</th>
<th>Recommended Use</th>
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<tbody>
<tr>
<td><strong>Wound contraction stimulants/enhancers</strong></td>
<td></td>
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<tr>
<td>Acemannan</td>
<td>Wounds under a bandage; use with a topical antibacterial</td>
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<tr>
<td>Topical hydrogel</td>
<td></td>
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<tr>
<td>Injectable</td>
<td>Intralesional injection; days 0, 3, and 6 in wounds in which bandage loss would result in topical medication being worn or licked off (e.g., paw pad wounds)</td>
</tr>
<tr>
<td>Freeze-dried gel</td>
<td>Wounds with exposed bone under a bandage; use in combination with a topical antibacterial</td>
</tr>
<tr>
<td>Tripeptide copper complex gel</td>
<td>Wounds under a bandage; use in combination with a topical antibacterial</td>
</tr>
<tr>
<td>Occlusive hydrogel dressings</td>
<td>Limb wounds in the repair stage of healing</td>
</tr>
<tr>
<td>Equine amnion</td>
<td>Trunk wounds</td>
</tr>
<tr>
<td>Pulsed electromagnetic field radiation</td>
<td>Wounds in which enhanced contraction is indicated</td>
</tr>
<tr>
<td>Adjustable horizontal mattress suture</td>
<td>Distal limb wounds on dogs; use in combination with topical hydrogel, freeze-dried gel, or tripeptide copper complex gel</td>
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<tr>
<td>Skin stretchers</td>
<td>Large wounds on dogs; use in combination with topical hydrogel, freeze-dried gel, or tripeptide copper complex gel</td>
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<table>
<thead>
<tr>
<th>Medication, Dressing, or Procedure</th>
<th>Recommended Use and Effect of Usage</th>
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<tbody>
<tr>
<td><strong>Wound contraction inhibitors</strong></td>
<td></td>
</tr>
<tr>
<td>Corticosteroids</td>
<td>Topically on wounds around body orifices to help prevent contracture constriction</td>
</tr>
<tr>
<td>Silver sulfadiazine</td>
<td>Topical antibacterial that may prolong contraction</td>
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<tr>
<td>Mafenide acetate</td>
<td>Topical antibacterial that may prolong contraction</td>
</tr>
<tr>
<td>Hydrocolloid dressings</td>
<td>Occlusive wound dressing that may prolong contraction</td>
</tr>
<tr>
<td>Porcine small intestine submucosa</td>
<td>Topical wound dressing that may prolong contraction</td>
</tr>
<tr>
<td>Thick skin grafts and flaps</td>
<td>Early placement in wound management on large wounds over joint flexion surfaces or wounds in which open healing will result in surrounding tissue distortion</td>
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*Recommendations are based on our general experience.
in the skin edges. At the ends of the suture, an arrangement of a button and a removable fishing weight should be used to hold tension on the suture after its daily tightening. The attachment of the skin edge to the granulation tissue should not be disturbed; thus wound contraction is not disturbed but enhanced by suture tension.1,2

A skin stretching device can be used to stretch skin surrounding a wound during the course of open wound management,1 thus making skin available for the contraction process. This device consists of self-adherent skin pads, which are affixed to the skin around the wound, and elastic cables, which are used to connect the skin pads.

Wound Contraction Inhibitors

Corticosteroids, which primarily affect the inflammatory stage, retard wound healing. This slowing is carried into the early repair stage of healing with a significant delay in angiogenesis, fibroplastic proliferation, and the synthesis of proteoglycans and collagen.34,35 Thus corticosteroids impair the epithelization, wound strength, and closure of open wounds.34,35 Epithelialization and contraction are both inhibited by glucocorticoids at any time during healing.36 This factor should be considered when an animal that is being treated for an open wound must be given corticosteroids. Beer and colleagues37 have shown that higher glucocorticoid concentrations inhibit the later repair stage functions of healing. The use of corticosteroids for inhibition of granulation tissue formation and wound contraction and contracture can be beneficial. For instance, topical steroids could also be used to treat open wounds around orifices in which contraction and/or contracture could result in orifice stenosis.

Silver sulfadiazine, a topical antibacterial, is commonly used to treat open wounds, especially burns. Although this medication enhances epithelialization in the open wound healing process, it has been shown to have a negative effect on fibroblasts and thus impedes wound contraction.38 The topical burn therapy mafenide acetate has been shown to have the same effect.38 Once wound infection is controlled, the effect of these drugs on wound contraction and epithelialization is of little concern if graft or flap reconstruction is to be used. However, if open wound healing is to be employed, the contraction of such wounds may be prolonged, and the delicate epithelium may need protection during the healing process.

Factors associated with antineoplastic drugs that may have a negative effect on wound healing/contraction are reduced mesenchymal cell proliferation, thrombocytopenia and leukopenia with reduced quantities of platelets and inflammatory cell–derived growth factors, and susceptibility to infection caused by leukopenia.35 The anorexia, negative nitrogen balance, and weight loss associated with these drugs negatively affect healing. Some chemotherapeutic agents damage the gastrointestinal tract and interfere with nutrient absorption, thus decreasing the substrate available for collagen synthesis.39 These factors should be considered when treating open wounds in the presence of chemotherapeutic agents.

Radiation therapy used in the treatment of neoplasia can impair wound healing, resulting in chronic wounds. This therapy causes a progressive oblitative endarteritis of wound microvasculature, which results in tissue ischemia. In addition, it directly affects fibroblast proliferation, causing permanent damage.39 Abnormal microvasculature and fibroblasts would equate to poor granulation tissue and impaired wound contraction. In addition, decreased blood supply and fibrosis may limit the delivery of platelets and inflammatory cells to the wound. Factors available to stimulate chemotaxis, mitogenesis, and collagen synthesis are, therefore, decreased. Thus irradiated open wounds are difficult to heal by second intention because of impaired angiogenesis, wound contraction, fibroplasia, and epithelialization.1 The lack of inflammatory cells may also permit bacteria to proliferate more readily,39 also impairing wound healing.

If acute or chronic ulcers result from radiation therapy, staged debridement should be performed over a longer period than occurs with other types of wounds because necrosis and persistent infection may progress. Only visible necrotic tissue should be debrided. Debridement to bleeding edges is unreliable because of ongoing progressive endarteritis. Overly aggressive debridement may result in removal of normal tissue.2 Because open wound healing with contraction will not be optimal in irradiated tissue, skin grafts or flaps should be considered for proper healing (Table I). Flaps take their blood supply with them. However, if a graft is to be used, the wound must be debrided back to healthy vascular tissue to help ensure graft revascularization.1,42 The use of skin flaps should be the first choice for reconstruction.

In addition, muscle flaps have been described1 and could be used to take the blood supply to a chronic radiation wound if the wound is located such that one of the muscle flaps can be used on it. Skin grafts could be placed on muscle flaps. New medications that stimulate wound healing should be considered to help establish a healthy bed of granulation tissue to support a graft (see Wound Contraction Stimulants/Enhancers section).

Wound contraction can be minimized or prevented by replacing skin immediately with thick skin grafts or flaps. Skin grafts or flaps can inhibit contraction more...
effectively than can split-thickness grafts, especially if they are applied before contraction has started. When treating large open wounds over flexion surfaces, skin grafts or flaps should be considered to prevent wound contracture deformities (Table I). If joint flexion is necessary for apposition of wound edges, wound mobility will likely result in deformity because the joint is pulled into flexion as contraction takes place. Thus a skin graft or flap is indicated.

Hydrocolloid dressings are water-impermeable with a polyurethane outer covering separated from the wound by a hydrocolloid material. The hydrocolloid adheres to the skin around the wound and is dissolved over the wound itself. A canine study found that hydrocolloid dressings (DuoDerm®, ConvaTec, Princeton, NJ and Dermahéal™, E.R. Squibb & Sons, Princeton, NJ) tended to enhance wound epithelialization but slowed wound contraction. This effect was attributed to the strong adherent nature of the dressing: It adhered to the skin around the wound, splinting it such that contractile forces of the granulation tissue could not effectively contract the wound. This type of wound dressing may not be desirable on wounds in which contraction with open wound healing is preferred.

One study on rodents showed that using porcine small intestinal submucosa as a dressing for open wounds slowed wound contraction.
Movement

Wound movement can affect wound contraction. Extensive wounds over flexion surfaces of joints that heal by second intention are subject to excess scarring, joint deformity, and impaired mobility because wound contraction results in wound contracture.1–3,5,8 (Figure 9). This occurrence can be explained by a combination of two phenomena. A buckling phenomenon has been described in which the tissue of a flexion surface wound is folded together or buckled with the predominant flexion kinetics of the joint. As these fibers fold on one another, the inter- and intramolecular bonding of the fibers occurs as the tissue gluing phenomenon.9,43 (Figure 10).

With the tissue gluing phenomenon, contraction occurs, and collagen fibers become shorter in the wound and fused into a contracted mass by mucopolysaccharides of the wound’s ground substance.9,44 Immobilizing a large wound over a flexion surface in extension may help prevent such contracture deformities. A splint can help to keep the tissue stretched mechanically during the active phase of wound contraction.10 A skin flap or graft should also be considered for such wounds (Table I). Thus immobilization helps prevent wound contracture mechanically as well as providing the immobility for a graft or flap to heal in place. When a graft is healing, immobility allows vascular connections to be made between the graft and its bed.

The desired result of wound healing is for one side of the wound to heal to the opposite side. Thus wound immobilization is needed for proper tissue bonding to occur. Immobilization can play a major role in wound healing in areas of considerable movement, especially if they are to heal as open wounds. Movement interferes with wound contraction. An example of movement interfering with open wound contraction is a wound over the extensor surface of a joint (e.g., carpus or stifle) where joint flexion pulls wound edges apart (Figure 11A). Meticulous suturing, followed by casting or splinting to prevent joint flexion, is necessary for proper healing.2

Another area in which movement presents a problem is the axillary region of cats. The wound usually results from the cat manipulating one forelimb up through its collar, and the limb remains in this position for an extended time. Thus the collar exerts pressure in the axillary region and cuts into the tissue. After the collar is removed, open wound healing with wound contraction is often impeded because of the tension and shearing forces of movement as one side of the wound moves against the other as the cat walks (Figure 11B). In such cases, meticulous suture immobilization of deep wound tissue, use of an intradermal or subcutaneous suture, and finally tension-apposition sutures in the skin may allow healing. Immobilization of the limb in a Velpeau flexion bandage is also indicated. In some cases, a rotation flap from the adjacent thoracic skin can be used for wound closure2 (Table I). Omental flaps may also be used to provide vascular tissue in the area before closure, thus enhancing healing.18,19

Exposed Bone

Exposed bone in wounds is common. In small animals, bone is often exposed in distal limb degloving wounds that result from automobile trauma. Exposed bone in a wound has an indirect influence on healing by inhibiting wound contraction and epithelialization.1,2,45 Wounds with exposed bone that are to be allowed to heal by second intention require a bed of granulation tissue for healing to progress.2,45 Granulation tissue forms from the vascularized soft tissue at the periphery of a wound and gradually advances over the bone.

Means of enhancing granulation tissue coverage over bone are beneficial to the contraction aspect of second-intention healing. Topical medications and dressings have been described for this purpose. Another technique that stimulates granulation tissue to form over exposed bone entails drilling small holes in the bone (forage) to the level of the marrow cavity to allow clot formation over the bone. The clot, which is well attached at the drill holes, serves as a matrix for the fibroblast ingrowth and neovascularization of granulation tissue.1,2,47

Eschars

Eschars are composed of full-thickness degenerated skin overlying a wound, usually a burn (Figure 12). An eschar may serve as a biologic bandage to protect the wound. However, the eschar may harbor bacteria beneath it, and its edge may splint the wound edge by its attachment. Therefore, wound contraction by underlying granulation tissue is impeded. The eschar should be removed as soon as the animal is stable enough for anesthesia.1,2,46 Allowing
the wound to heal as an open wound would enable contraction to take place quickly.

**Infection**

Infection can inhibit wound healing to include the contraction of open wounds. The presence of bacterial collagenases, decreased wound pH and oxygen tension, interference with blood supply, and mechanical interference by exudate are factors contributing to the lack of healing. With infection, the inflammatory stage of healing is prolonged and the healing process is retarded. The epithelialization, contraction, and collagen deposition do not occur or occur in a limited fashion, and healing does not take place until infection is under control. When infection occurs, debridement of debris and necrotic tissue should be performed along with thorough wound lavage. Staged debridement (as needed), lavage, and protective bandaging should be done daily. Culture and sensitivity evaluation of the wound may be necessary. Topical and systemic antibiotics are indicated.

**Wound Shape**

As wounds contract, the edges move toward the center and the shape of the resulting scars depends on the original wound shape. Skin elasticity and mobility are not the same in all directions, thus skin will not move to the same extent or at the same rate. The amount of movement will depend on skin availability on different sides of the wound. On some areas of an animal (e.g., the trunk), skin may be freely available on all sides of the wound. In other areas (e.g., the limbs), skin will be relatively sparse on all sides; and in some places it may only be available on certain sides of the wound but not on others (e.g., a wound adjacent to a body structure such as an eye or anus).

When skin is equally available on all sides of a wound, triangular, rectangular, and square wounds heal centripetally, with the corners moving less than the sides as the sides move centrally. The results are a three-armed stellate scar, a double-V-shaped scar with the points of the V’s pointing toward each other and connected by a thin line, and a four-armed stellate scar, respectively. Circular wounds also heal centripetally; however, as the wound edge approaches the center of the wound, the skin will start to mechanically interfere with its own movement. The result is a crumpled, unpredictable healing pattern with a slower healing rate (Figure 13).
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mnistration of corticosteroids on wound contraction. *Ann
1. The four components of granulation tissue are
   a. lymphocytes, eosinophils, capillary loops, and collagen.
   b. fibroblasts, macrophages, capillary loops, and collagen.
   c. fibroblasts, neutrophils, capillary buds, and ground substance.
   d. basophils, eosinophils, arterioles, and tropocollagen.
   e. dermacytes, macrophages, venules, and ground substance.

2. Which of the following is not associated with fibroblasts?
   a. ground substance production
   b. collagen production
   c. wound contraction
   d. fibrin dissolution
   e. myofibroblasts formation

3. Which of the following statements regarding wound contraction is true?
   a. Contraction is the centripetal or concentric reduction in size of an open wound.
   b. New dermis and epidermis are formed as wound contraction takes place.
   c. Placement of a skin graft or flap has no effect on wound contraction.
   d. Skin tension around a wound is best evaluated after the wound has entered the inflammatory stage of healing.
   e. Wound contraction is dependent on wound epithelialization.

4. The function of fibroblasts in wound contraction is to
   a. destroy the fibrin clot to allow the myofibroblasts to cause wound contraction.
   b. provide preliminary contact guidance for myofibroblasts to enter a wound to cause wound contraction.
   c. have cell membrane movement within surrounding collagen and consolidate tissue into a smaller unit.
   d. enhance angiogenesis and provide a blood supply for new collagen.
   e. provide a base for epithelialization.

5. Which statement regarding indolent pocket wounds is false?
   a. The skin edge of the pocket everts.
   b. Circulation in the pocket decreases.
   c. Wound contraction is slowed.
   d. Skin margins begin to curl under.
   e. The pocket supports bacterial growth.

6. When bandaging an open wound over a bony prominence, the best way to prevent pressure is to
   a. apply additional padding over the wound.
   b. immobilize the wound area with a splint or cast.
   c. place a modified minor donut bandage over the area.
   d. use an occlusive hydrogel dressing to cushion the wound.
   e. use a polyurethane sponge dressing over the wound.

7. ____________ is not a wound contraction stimulant/enhancer.
   a. Acemannan
   b. A semioclusive adherent dressing
   c. An occlusive hydrogel dressing
   d. Equine amnion
e. Glycyl-L-histadyl-L-lysine tripeptide copper complex

8. Silver sulfadiazine topical medication
   a. has antibacterial properties.
   b. slows wound epithelialization.
   c. impedes wound contraction.
   d. a and b
   e. a and c

9. Wound contracture deformities occur in wounds
   a. over flexion surfaces of joints.
   b. that have excess epithelialization.
   c. over extension surfaces of joints.
   d. with irregular edges.
   e. over areas of little movement.

10. Eschars inhibit wound contraction by
    a. producing collagenases that dissolve collagen.
    b. allowing subeschar epithelialization.
    c. enhancing granulation tissue maturation.
    d. splinting the wound edges.
    e. allowing wound movement.