Any infection that arises immediately adjacent to the fascia may have an intra-abdominal component. Extensive exploration is warranted to assess fascial integrity. Signs of deep infection include fever, severe pain, and marked separation of wound edges.
Despite advances in medicine and surgery over the past century, postoperative wound complication remains a serious challenge. When a complication occurs, it translates into prolonged hospitalization, lost time from work, and greater cost to the patient and the health-care system.

Prevention of wound complication begins well before surgery. Requirements include:

- understanding of wound healing (see page 48) and the classification of wounds (TABLE 1, page 44)
- thorough assessment of the patient for risk factors for impaired wound healing, such as diabetes or use of corticosteroid medication (TABLE 2, page 46)
- antibiotic prophylaxis, if indicated (TABLE 3, page 47)
- good surgical technique, gentle tissue handling, and meticulous hemostasis
- placement of a drain, when appropriate
- awareness of technology that can enhance healing
- close monitoring in the postoperative period
- intervention at the first hint of abnormality.

In this article, we describe predisposing factors and preventive techniques and measures, and outline the most common wound complications, from seroma to dehiscence, including effective management strategies.

**Conditions and drugs that impair healing**

Preexisting medical conditions may limit healing, especially conditions associated with diminished delivery...
### TABLE 1 Classification of surgical wounds

<table>
<thead>
<tr>
<th>Classification of surgical wounds</th>
<th>Infection rate</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CLASS I – Clean wounds (&lt;5%)</strong></td>
<td></td>
</tr>
<tr>
<td>• Created under ideal operating room conditions</td>
<td></td>
</tr>
<tr>
<td>• Usually an elective surgical incision made under aseptic conditions</td>
<td></td>
</tr>
<tr>
<td>• Usually an uninfected wound and not predisposed to infection</td>
<td></td>
</tr>
<tr>
<td>• Uninfected wound with no inflammation</td>
<td></td>
</tr>
<tr>
<td>• No entry into oropharyngeal cavity, respiratory tract, alimentary tract, or genitourinary tract</td>
<td></td>
</tr>
<tr>
<td>• Always closed primarily and usually not drained</td>
<td></td>
</tr>
<tr>
<td><strong>CLASS II – Clean-contaminated wounds (2–10%)</strong></td>
<td></td>
</tr>
<tr>
<td>• Entry into oropharyngeal cavity, respiratory tract, alimentary tract, or genitourinary tract under controlled conditions without unusual contamination or significant spillage, e.g., hysterectomy, appendectomy</td>
<td></td>
</tr>
<tr>
<td>• Minor break in surgical technique</td>
<td></td>
</tr>
<tr>
<td>• No evidence of infection</td>
<td></td>
</tr>
<tr>
<td><strong>CLASS III – Contaminated wounds (15–20%)</strong></td>
<td></td>
</tr>
<tr>
<td>• Fresh, open, traumatic wound or injury, e.g., soft-tissue laceration, open fracture</td>
<td></td>
</tr>
<tr>
<td>• Surgical procedures that involve gross spillage from gastrointestinal tract</td>
<td></td>
</tr>
<tr>
<td>• Genitourinary or biliary tract procedures in the presence of infected urine or bile</td>
<td></td>
</tr>
<tr>
<td>• Major break in surgical technique</td>
<td></td>
</tr>
<tr>
<td>• Incisions that encounter acute, nonpurulent inflammation</td>
<td></td>
</tr>
<tr>
<td>• Infections that progress to infection within 6 hours</td>
<td></td>
</tr>
<tr>
<td><strong>CLASS IV – Dirty or infected wounds (&gt;30%)</strong></td>
<td></td>
</tr>
<tr>
<td>• Traumatic wounds more than 4 hours old</td>
<td></td>
</tr>
<tr>
<td>• Wounds that are heavily contaminated or clinically infected before surgery, e.g., tubo-ovarian abscess</td>
<td></td>
</tr>
<tr>
<td>• Neglected traumatic wounds in which devitalized tissue or foreign material is retained</td>
<td></td>
</tr>
<tr>
<td>• Incision into bowel is anticipated</td>
<td></td>
</tr>
<tr>
<td><strong>NOTE:</strong> Infection that is present at the time of surgery can increase the postoperative infection risk by an average of fourfold.</td>
<td></td>
</tr>
</tbody>
</table>

SOURCE: Centers for Disease Control and Prevention and the American College of Surgeons

Because steroids, NSAIDs, and chemotherapy agents impede wound healing, and anticoagulants may interfere with granulation, it is crucial to review the patient’s medications well in advance of surgery.

### Nutrition plays a critical role

The importance of nutrition cannot be overstated. A significant percentage of patients are thought to have some degree of nutritional deficiency preoperatively. This deficiency may alter the inflammatory response, impair collagen synthesis, and reduce the tensile strength of the wound.

Because healing requires energy, deficits in carbohydrates may limit protein utilization, and deficiencies of vitamins and micronutrients can also interfere with healing.

Obesity, too, increases the risk of postoperative wound complication. Markedly obese patients have a thick, avascular, subcutaneous layer of fat that compromises healing.

### Meticulous technique required

Good surgical technique and appropriate use of antibiotics are critical components of successful wound healing.

When placing the incision, avoid the moist, bacteria-laden subpannicular crease in the markedly obese.

During a procedure, handle tissue gently, keep it moist, and make minimal use of electrocautery to reduce tissue injury and promote healing. Keep operating time and blood loss to a minimum, and debride the wound of any foreign material and devitalized tissue.

Multiple studies have demonstrated that judicious use of prophylactic antibiotics significantly decreases the incidence of wound infection, particularly in relation to hysterectomy and vaginal procedures and when entry into bowel is anticipated. A number of prophylactic regimens are given in TABLE 3.

### Meticulous hemostasis

At the time of closure is imperative. When complete hemostasis cannot be confirmed, place a small drain in the subcutaneous space (or subfas-
cial space, if there is oozing on the muscle bed) and apply a pressure dressing to help prevent hematoma. Although a drain is not a substitute for precise hemostasis or careful surgical technique, it may be helpful when there is concern about oozing or a “wet” surface, or when the patient is markedly obese.

Some practitioners have expressed concern over the risk of bacterial migration and infection with placement of a drain, but others, including us, advocate use of a drain in the subcutaneous space to help remove residual blood, fluid, and other debris to prevent the formation of dead space and infection and promote wound closure and healing. In a small study, Gallup and associates demonstrated a decreased incidence of wound breakdown when a drain was placed.6

A closed-suction drain, such as a Jackson-Pratt or Hemovac model, helps minimize wound complication when it is placed in the subcutaneous layer. (Avoid a rubber Penrose drain because it may allow bacteria to enter the wound.) It is imperative that the drain exit the body via a separate site and not through the incision itself. We advocate removal when less than 30 mL of fluid accumulates in the reservoir over 24 hours.

Fluid within the wound does not always indicate infection

Wound collections are not necessarily indicative of infection; collections of fluid within the wound may represent a serous transudate, blood, pus, or a combination of these. If the fluid is not addressed, however, fulminant infection may be the result.

Seroma is usually painless

A seroma is a collection of wound exudates within the dead space. Seroma typically involves thin, pink, watery discharge and minimal edge separation. In some cases, there may be surrounding edema but generally little to no tenderness.

When a seroma is detected, remove the staples or stitches in the area of concern and explore the wound. It is essential to ensure fascial integrity, as serous wound drainage may be a sign of impending evisceration. After these measures are taken, cleanse and lightly pack the wound to permit drainage.

Hematoma requires identification of the source of bleeding

Hematoma represents blood or a blood clot within the tissues beneath the skin. It may be caused by persistent bleeding of a vessel, although the pressure within the wound and the pressure produced by the dressing often provide tamponade on the bleeding source, in which case the hematoma forms with no active bleeding.

Hematoma is usually caused by small bleeding vessels that were not apparent at the time of surgery or were not cauterized or

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**TABLE 2** Risk factors for poor wound healing and dehiscence

<table>
<thead>
<tr>
<th>Poor wound healing</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Advanced age</td>
</tr>
<tr>
<td>• Hypoxia/severe anemia</td>
</tr>
<tr>
<td>• Medications</td>
</tr>
<tr>
<td>- chemotherapy</td>
</tr>
<tr>
<td>- immunosuppressive drugs</td>
</tr>
<tr>
<td>- steroids</td>
</tr>
<tr>
<td>- nonsteroidal anti-inflammatory drugs</td>
</tr>
<tr>
<td>• Anticoagulants</td>
</tr>
<tr>
<td>• Poor nutritional status</td>
</tr>
<tr>
<td>• Diabetes</td>
</tr>
<tr>
<td>• Arterial-venous disease</td>
</tr>
<tr>
<td>• Dehydration</td>
</tr>
<tr>
<td>• Obesity</td>
</tr>
<tr>
<td>• Immunocompromised state</td>
</tr>
<tr>
<td>• Malignancy</td>
</tr>
<tr>
<td>• History of radiation therapy</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Abdominal wound dehiscence</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Obesity</td>
</tr>
<tr>
<td>• Malnutrition</td>
</tr>
<tr>
<td>• Marked anemia</td>
</tr>
<tr>
<td>• Advanced age</td>
</tr>
<tr>
<td>• Uncontrolled diabetes</td>
</tr>
<tr>
<td>• Pulmonary disease</td>
</tr>
<tr>
<td>• Uremia</td>
</tr>
<tr>
<td>• Malignancy</td>
</tr>
<tr>
<td>• Infection</td>
</tr>
<tr>
<td>• Abdominal distention (straining, coughing, ascites)</td>
</tr>
<tr>
<td>• History of radiation therapy</td>
</tr>
<tr>
<td>• Chemotherapy</td>
</tr>
<tr>
<td>• Use of corticosteroids</td>
</tr>
<tr>
<td>• Poor surgical technique (type of incision, type of suture, method and strength of closure, use of electrocautery in “coagulation current” setting)</td>
</tr>
</tbody>
</table>

SOURCE: Carlson,11 Cliby.12
ligated at the time of closure. For this reason, it is important to achieve good hemostasis and a “dry” wound before closing the skin.

When hematoma is suspected, open the wound enough to permit adequate exposure and identify the source of bleeding. Evacuate as much blood and clot as possible because blood is an ideal medium for bacterial growth. If active bleeding is found, use a silver nitrate applicator or handheld cautery pen to accomplish hemostasis at bedside. If bleeding is more severe, or the source cannot be visualized, consider returning to the operating room for more extensive exploration.

Once hemostasis is achieved, irrigate the wound copiously and institute local wound care.

How common is infection?
Before it is possible to address this question, it is necessary to clarify the terminology of infection. Contamination and colonization are different entities. The first refers to the presence of bacteria without multiplication. The latter describes the multiplication of bacteria in the absence of a host response. When infection is present, bacterial proliferation produces clinical signs and symptoms.

Postoperative abdominal wound infection occurs in about 5% of cases but may be more common in procedures that involve mesh. One study found a 12% incidence of wound infection, but the rate declined to 8% when antibiotic prophylaxis was instituted.

Several other studies have examined determinants of infection. For example, a large Cochrane review found no real differences in infection rate by preoperative skin preparation technique or agent, but it did observe that one study had demonstrated the superiority of chlorhexidine to other cleansing agents.

Cruse and Foord also noted the slight superiority of chlorhexidine, as well as the efficacy of clipping abdominal hair immediately before surgery.

When identifying organisms, look for the usual suspects
The offending pathogens in infection are usually endogenous flora found on the patient’s skin and within hollow organs (vagina, bowel). The organisms most commonly responsible for infection are Staphylococcus (aureus, epidermidis), enterococci, and Escherichia coli. However, the bacteria identified in the wound may not be the causative organism.

Most infections typically become clinically apparent between the fifth and 10th postoperative days, often after the patient has been discharged, although they may appear much earlier or much later. One of us (Dr. Perkins) had a patient who presented with a suppurative infection after undergoing hysterectomy for endometrial carcinoma 5 months earlier.

Cellulitis is common
Wound cellulitis, a common, non-suppurative infection of skin and underlying connective tissue, is generally not severe. The wound assumes a brawny, reddish brown appearance associated with edema, warmth, and erythema. Fever is not always present.

It is important to remember that cellulitis may surround a deeper infection. Although needle aspiration of the leading edge has been advocated, it yields a positive culture in only 20% to 40% of cases.

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Antibiotic</th>
<th>Single intravenous dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hysterectomy and uro-gynecologic procedures, including those that involve mesh</td>
<td>Cefazolin</td>
<td>1 g or 2 g</td>
</tr>
<tr>
<td></td>
<td>Clindamycin plus gentamicin, a quinolone, or aztreonam</td>
<td>600 mg plus 1.5 mg/kg, 400 mg, or 1 g, respectively</td>
</tr>
<tr>
<td></td>
<td>Metronidazole plus gentamicin or a quinolone</td>
<td>500 mg plus 1.5 mg/kg or 400 mg, respectively</td>
</tr>
</tbody>
</table>

SOURCE: American College of Obstetricians and Gynecologists

Abdominal wound infection occurs in about 5% of cases and typically becomes clinically apparent between the fifth and 10th postoperative days.
Biologic phases of wound healing—overlapping and interdependent

It was pioneering Scottish surgeon John Hunter who noted that “injury alone has in all cases the tendency to produce the disposition and means of a cure.”

Unlike the tissue regeneration that occurs primarily in lower animals, human wound healing is mediated by collagen deposition, or scarring, which provides structural support to the wound. This scarring process may itself cause a variety of clinical problems.

Wound healing is characterized by overlapping, largely interdependent phases, with no clear demarcation between them. Failure in one phase may have a negative impact on the overall outcome.

In general, wound healing involves two phases: inflammation and proliferation. Within these phases, the following processes occur: scar maturation, wound contraction, and epithelialization. These repair mechanisms are activated in response to tissue injury even when it is surgically induced.

**Inflammatory phase**
The initial response to tissue injury is inflammation, which is mediated by various amines, enzymes, and other substances. This inflammation can be further broken down into vascular and cellular responses.

The first burst of blood acts to cleanse the wound of foreign debris. It is followed by vasoconstriction, which is mediated by thromboxane 2, to decrease blood loss. Vasodilation then occurs once histamine and serotonin are released, permitting increased blood flow to the wound. The surge in blood flow accounts for the increased warmth and redness of the wound. Vasodilation also increases capillary permeability, allowing the migration of red blood cells, platelets, leukocytes, plasma, and other tissue fluids into the interstitium of the wound. This migration accounts for wound edema.

In the cellular response, which is facilitated by increased blood flow, cell migration occurs as part of an immune response. Neutrophils, the first cells to enter the wound, engage in phagocytosis of bacteria and debris. Subsequently, there is migration of monocytes, macrophages, and other cells. This nonspecific immune response is sustained by prostaglandins, aided by complement factors and cytokines. A specific immune response follows, aimed at destroying specific antigens, and involves both B- and T-lymphocytes.

**Proliferative phase**
Proliferation is characterized by the infiltration of endothelial cells and fibroblasts and subsequent collagen deposition along a previously formed fibrin network. This new, highly vascularized tissue assumes a granular appearance—hence, the term “granulation tissue.”

Collagen that is deposited in the wound undergoes maturation and remodeling, increasing the tensile strength of the wound. The process continues for months after the initial insult.

All wounds undergo some degree of contraction, but the process is more relevant in wounds that remain open or involve significant tissue loss.

Last, the external covering of the wound is restored by epithelialization.
In the absence of purulent drainage, treat cellulitis with antibiotics, utilizing sulfa-
methoxazole-trimethoprim, a cephalo-
sporin, or augmented penicillin, and apply
warm packs to the wound.

If purulent drainage is seen, or the patient
fails to improve significantly within 24 hours,
suspect an abscess or resistant organism.

Most wound infections are superficial
Approximately 75% of all wound infections
involve the skin and subcutaneous tissue lay-
ers. Superficial infection is more likely to occur
when there is an undrained hematoma, exces-
sively tight sutures, tissue trauma, or a retained
foreign material. Edema, erythema, and pain
and tenderness may be more pronounced than
with cellulitis. A low-grade fever may be pres-
ent, and incisional discharge typically occurs.

Drainage is the cornerstone of manage-
ment and requires the removal of staples
or sutures from the area. Local exploration
is mandatory, and fascial integrity must be
confirmed. If a pocket of pus is found, open
the wound liberally to determine the extent
of the pocket and permit as much evacuation
as possible. Wound culture is optional. Insti-
tute local wound care and consider adjuvant
antibiotics in selected cases.

Ensure fascial integrity
Any infection that arises immediately adja-
cent to the fascia may have an intra-abdom-
inal component, although that is unlikely.
Extensive exploration is warranted to assess
fascial integrity.

If intra-abdominal infection is suspect-
ed, order appropriate imaging.

Patients who have deep infection usually
exhibit frank, purulent discharge; fever; and
severe pain. Marked separation of wound
edges is often present as well, as is an elevat-
ed white blood cell count.

As with superficial infection, the key to
therapy is liberal exploration, drainage of
the abscess cavity, and mechanical wound
debridement. Irrigate the wound copiously
using a dilute mixture of saline and hydrogen
peroxide to remove any remaining debris.
Avoid povidone-iodine solution because it
inhibits normal tissue granulation.

The wound may be left open to heal by
secondary intention, or it may be closed sec-
donarily after 3 to 6 days, provided there is no
evidence of infection and a healthy granulat-
ing bed is present.

Consider adjuvant antibiotics, especially
when the patient is immunocompromised.

If the wound has pronounced edema and
unusual discoloration, consider a serious in-
fection such as necrotizing fasciitis.

Wound dehiscence raises risk of eviscera-
tion
Dehiscence of the abdominal incision occurs
when the various layers separate. Dehiscence
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plete fascial dehiscence or burst abdomen).
When bowel or omentum extrudes, the term **evisceration** is appropriate.

In several reviews of the literature, the incidence of dehiscence ranged from 0.4% in earlier studies to 1% to 3% in later reviews.\(^9\)\(^\text{-}12\) Despite advances in preoperative and postoperative care, suture materials, surgical technique, and antibiotics, fascial dehiscence remains a serious problem in abdominal surgery.

**What causes wound disruption?**

To a great extent, abdominal wound breakdown is a function of surgical technique and method of closure. Although the conventional wisdom is that dehiscence occurs less frequently with a transverse incision than with a vertical one, this assumption is being challenged. A small study by Hendrix and associates found no differences in the rate of dehiscence by type of incision.\(^13\) That finding suggests that the incidence of dehiscence is inversely related to the strength of closure.

Selection of the appropriate suture material also is important. In addition, use of electrocautery in the “cutting current” mode when the abdomen is opened causes less tissue injury than “coagulation current.” The latter has a greater thermal effect, thereby weakening the fascial layer.

**Patient characteristics that influence wound integrity** include comorbidities such as diabetes and malignancy, recent corticosteroid administration, and malnutrition.

Although infection may accompany superficial wound separation, its role in complete dehiscence is unclear.

Conditions that cause abdominal distention, such as severe coughing, vomiting, ileus, and ascites, may contribute to dehiscence, particularly when the closure method is less than satisfactory.

Some authors have found a greater incidence of wound disruption when multiple risk factors are present. In patients who had eight or more risk factors, wound disruption was universal.\(^11\)\(^\text{-}12\)

**Management entails debridement, irrigation, and closure**

When extrafascial dehiscence occurs, me-
Advances in wound management offer help and promise

Vacuum-assisted closure
The vacuum-assisted wound closure system is a device that speeds healing and reduces the risk of complication. It consists of a sponge dressing that can be sized to fit an open wound and connected to an apparatus that generates negative pressure. The device enhances healing by removing excess fluid and debris and decreasing wound edema.

Argenta and associates reported successful use of this system to expedite healing in three cases of wound failure.14 It can be employed in the home-health setting by nurses trained in its use.

Human acellular dermal matrix
Occasionally, breakdown of a wound creates marked fascial defects that preclude secondary closure. Synthetic materials—both absorbable and nonabsorbable varieties—have been employed to bridge the defect, but their use sometimes leads to adhesions, infection, and cutaneous fistula. These risks are of special concern when the wound is already contaminated or otherwise compromised.

One alternative is human acellular dermal matrix (AlloDerm, LifeCell Corp). Tung and colleagues described its use for repair of a fascial defect in a previously irradiated cancer patient whose postoperative course was complicated by pelvic infection.15 This dermal matrix, a basement membrane taken from cadaveric skin, promotes neovascularization and is thought to be associated with a lower incidence of infection and adhesions than is traditional mesh. It is widely used in the burn setting and in the repair of ventral hernia, but is a relatively new addition to the management of fascial defects associated with wound breakdown.

Growth factors
Wound healing is regulated by a number of entities, including cytokines and growth factors, so it is no surprise that research has turned its focus on them. In a preliminary study, investigators found that separated abdominal wounds closed faster when recombinant human platelet-derived growth factor BB was topically administered than they did when they were left open to close by secondary intention.16

Although their use is not commonplace in wound management, research suggests that growth factors may one day be helpful adjuncts in the care of wound complications.

mechanical debridement and irrigation are usually the only measures necessary before deciding how to close the wound—even if infection is present. Remove all foreign material and excise any devitalized tissue.

As for the method of closure, the choice is usually between secondary closure and leaving the wound open to heal by secondary intention. An alternative to the latter is wound closure after several days, once a healthy granulating bed develops.

Dodson and colleagues described a technique of superficial wound closure that can be performed at the bedside using local anesthesia, with little discomfort to the patient.13 Wound separation caused by a small hematoma or sterile seroma especially lends itself to this type of immediate closure.

Complete fascial dehiscence is a “catastrophic” complication
Complete dehiscence of the fascia and extrusion of intra-abdominal contents is a serious catastrophic complication that is associated with a mortality rate of about 20%. It typically occurs between the third and seventh postoperative days, although later occurrences have been reported.

Warning signs of impending evisceration include serous drainage in the absence of obvious infection, and a “popping” sensation on the part of the patient—a feeling that something is “giving way.”

If evisceration occurs, cover exposed bowel with packs soaked in saline or povidone-iodine and prepare the patient for emergency surgery. Institute both hydration and broad-spectrum antibiotics.

Before replacing the abdominal contents, thoroughly irrigate the peritoneal cavity and inspect the bowel carefully, excising any necrotic tissue.

Reapproximate the fascia using interrupted #1 or #2 monofilament suture. Also consider placing retention sutures, particularly when the patient has multiple risk fac-
tors for wound complications (FIGURE). Leave the wound open, prepared for later closure.

If the abdomen cannot be closed because of peritonitis or bowel edema, or there is an insufficient amount of fascia remaining, approximate the abdominal wall using bridging sutures over a gauze pack as a temporizing measure until reconstruction can be performed. Consultation with a plastic surgeon or trauma specialist is recommended.

### Necrotizing fasciitis:
**Worst of the worst**

Necrotizing fasciitis is a dangerous, synergistic, bacterial infection involving the fascia, subcutaneous tissue, and skin. The culprits are multiple bacterial pathogens that include *Streptococcus pyogenes*, staphylococcal species, gram-negative aerobes, and anaerobes. The infection typically originates at a localized area, spreads along the fascial planes, and ultimately causes septic thrombosis of the vessels penetrating the skin and deeper layers. The result is necrosis. The associated mortality rate is approximately 20%.

The patient who has necrotizing fasciitis typically displays severe pain; anesthetic, edematous skin; purple, necrotic wound edges; hemorrhagic bullae; and crepitus. Frank necrosis subsequently develops, with surrounding inflammation and edema, and leads to systemic toxicity, with fever, hemorrhagic bullae; and crepitus. Laboratory evaluation includes a white blood cell count. Biopsy also is recommended. If necrotizing fasciitis is present, biopsy will reveal necrosis and thrombi of vessels passing through the fascia.

Treatment of necrotizing fasciitis requires intravenous, broad-spectrum antibiotics, including penicillin, that are adjusted according to the findings of the wound culture and sensitivity test. Cardiovascular and fluid-volume support is critical, as is wide surgical debridement of all necrosed skin and fascia. The latter, in fact, is the cornerstone of therapy.

![FIGURE](image)

**Consider retention sutures for high-risk patients**

Retention sutures are placed in interrupted fashion to support the primary suture line and are carried through the full thickness of the tissue, from the abdominal wall skin through the fascia and, if possible, the peritoneum. A rubber bolster placed across each suture keeps the suture from cutting into the skin (inset).

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References