The term “source control” encompasses a spectrum of interventions whose objective is the physical control of foci of infection and the restoration of optimal function and quality of life. Adequate source control – in conjunction with antibiotics, resuscitation and support of vital organ function – is a cornerstone of the successful management of the septic patient. It is also the oldest mode of infection control: the Edwin Smith papyrus from the seventeenth century BCE describes the role of spontaneous drainage in the treatment of an abscess of the chest wall.

The spectrum of source-control options is large, and their utility in an individual patient is heavily dependent not only on the site and nature of the inciting infection but also on the premorbid state of the patient, and the local availability of human and technological resources. Optimal decision-making requires the application of core principles in the biology of the host inflammatory response; these principles are the focus of this article.

**BIOLOGIC CONSIDERATIONS**

Infection and other stimuli evoke a response that has evolved to contain the insult, to limit damage, and to activate the necessary processes that will lead to tissue repair and sustain survival. In aggregate, these processes, which include inflammation, coagulation, and tissue repair, constitute the innate immune response.

Local tissue invasion by microorganisms results in an acute inflammatory response, characterized by the classic manifestations of rubor (redness), calor (warmth), dolor (pain), and pyrexia. The inflammatory response is initiated by the activation of pattern recognition receptors, which recognize pathogen-associated molecular patterns. The innate immune response is followed by the adaptive immune response, which is mediated by the activation of lymphocytes and antibodies.

**KEYWORDS**

- Source control
- Sepsis
- Abscess
- Surgery
- Percutaneous drainage
- Debridement
- Foreign body
pain), tumor (swelling), and functio laesa (loss of function). The consequence is an increase in local blood flow, and the influx of polymorphonuclear neutrophils, recruited through the release of chemokines such as interleukin-8, and retained following the reciprocal upregulation of adhesion molecules on the local vascular endothelium and on the infiltrating neutrophil. Coincident with this, tissue factor is expressed on the activated endothelial cells, and initiates local activation of the coagulation cascade. These processes can lead to the elimination of the invading pathogen, or to a biologic standoff, in which the organism is not eliminated, but is contained within an abscess cavity. The structure of the abscess reflects these preceding processes (Fig. 1). Activation of coagulation leads to the deposition of fibrin, which forms the wall of the abscess and contains its contents (a mixture of bacteria, infiltrating neutrophils, serum, and tissue debris). The process serves to limit bacterial spread and to isolate the abscess contents from systemic host defenses.

**Drainage**

The contents of an abscess are typically liquid, and the formation of an abscess allows the successful use of drainage, one of the 3 core elements of source control (Table 1). Successful drainage converts a closed-space abscess into a controlled sinus (a cavity that communicates with an epithelially lined surface) or fistula (an abnormal communication between 2 epithelially lined surfaces). A fistula will persist if any of several conditions is present: distal obstruction, epithelialization of the tract, a foreign body, uncontrolled infection or inflammation, malignancy, or radiation injury. In the absence of these factors, the fistulous tract will close. Thus following successful drainage of an abscess, and its conversion into a controlled fistula, the fistula is maintained open by the presence of the drainage tube, and when the tube is removed, provided that none of the other factors is present, the fistula should close (Fig. 2). The principles underlying the development and persistence or resolution of a fistula are some of the most fundamental upon which the craft of surgery is based. In addition to their use

![Fig. 1. A photomicrograph of a liver abscess illustrates the key structural features of an abscess. The abscess contents (dotted arrow) are a mixture of neutrophils, bacteria, tissue debris, and tissue fluid, colloquially called pus. Local activation of inflammation induces activation of the coagulation cascade resulting in fibrin deposition at the periphery of this process, and creating the characteristic fibrin capsule that walls off an abscess (solid arrow). From the CDC Public Health Image Library, image credit CDC/Rodney M. Donlan, PhD; Janice Carr (PHIL #7488), 2005.](image-url)
in the source-control management of an abscess, they represent the biologic basis for the success of intestinal anastomoses or stomas (epithelialization of a fistulous tract maintains its patency) or for procedures such as tracheostomy or gastrostomy, in which a foreign body enables a fistulous tract to persist.

**Debridement**

Small amounts of necrotic tissue are degraded by enzymes from phagocytic cells, and comprise one of the components of an abscess. Larger volumes of necrotic

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**Table 1**

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**Fig. 2.** Percutaneous drainage of a diverticular abscess in a patient with diverticulitis has created a colocutaneous fistula. The fistula remains open, because of the presence of a foreign body (the drain, arrows). In the absence of distal obstruction in the sigmoid colon or rectum, epithelialization of the tract, malignancy, or uncontrolled inflammation, the tract should close following drain removal, permitting an elective sigmoid resection at a later date to remove the diseased colon and prevent recurrent diverticulitis.
tissue, however, overwhelm the capacity of the host innate immune system, and so must be removed surgically. Although microbial products, interacting with pattern recognition receptors on host innate immune cells, are the best-known triggers of the inflammatory response resulting in sepsis, necrotic tissue can evoke a similar response by inducing the release of interleukin-1α (IL-1α). The mechanisms promoting the activation of an inflammatory response by noninfectious stimuli are poorly understood, and challenging to elucidate. It is known that host-derived substances such as uric acid, heat shock proteins, and oxidized phospholipids induce an inflammatory response through the activation of toll-like receptor signaling pathways. A potent systemic inflammatory response with massive neutrophil accumulation in the lung is the result of ischemia-reperfusion injury of the intestine or extremity. It is unclear whether that response results from the release of endogenous substances from the ischemic tissue, or from the secondary effects of the injury on the gastrointestinal tract, leading to the translocation of endotoxin or bacteria from the gut lumen.

It is apparent, however, that removal of necrotic tissue, and particularly infected necrotic tissue, plays an important role in eliminating the trigger of an ongoing inflammatory response. In some cases, notably gangrene of the distal extremities in patients with peripheral vascular disease, the ischemic tissue is isolated from the systemic circulation by virtue of the occlusion of the arteries and veins of the region; under these circumstances, the systemic response is minimal, and urgent intervention is not needed. In other circumstances, for example necrotizing soft tissue infections or intestinal infarction, the systemic response to the necrotic infected tissue is dramatic and life threatening, and emergent intervention is required. Yet in other infections such as infected retroperitoneal necrosis following severe acute pancreatitis, debridement of infected necrotic tissue may be necessary for the resolution of the infection, but may be safely deferred until it can be performed with minimal morbidity.

**Device Removal**

Although device-related infections causing sepsis in ambulatory individuals are uncommon, they are the most common cause of nosocomial infection in the critically ill patient, and a frequent risk factor for sepsis in hospitalized patients. Removal of the infected device is desirable, because the device typically harbours microorganisms in a biofilm, and so serves as a source of continuous bacterial shedding, and because the presence of a foreign body impairs local host defenses.

Many microorganisms are capable of living independently (in a planktonic state) or within an exopolymeric matrix known as a biofilm (in a sessile state). The transition between the 2 states is regulated by environmental factors such as nutrient availability, through microbial quorum-sensing mechanisms that provide the organism with information about the external milieu. The formation of a biofilm enables a colony of microorganisms to establish residence on an indwelling foreign body, and biofilms have been implicated in the pathogenesis and persistence of device-related infections, including those involving prosthetic joints, prosthetic heart valves, central venous catheters, urinary catheters, and endotracheal tubes. Microorganisms present within a biofilm are less sensitive to antibiotics than those in the planktonic or free state. This reduced susceptibility results not only from the physical barrier created by the biofilm but also from secondary effects of the biofilm on the local oxygen availability. The presence of a foreign body impairs neutrophil oxidative killing capacity, and so impedes innate antimicrobial host defenses.
Diagnosis

Effective source control presumes an ability to diagnose a local focus of infection that is amenable to source-control measures. The history and physical examination remain the most important initial diagnostic maneuvers. New onset of pain may aid in localizing the site of infection in a patient with systemic manifestations of sepsis. A history of a recent surgical procedure suggests an infectious complication at the surgical site, or in the surgical wound. A history of peripheral vascular disease, or recent myocardial infarction, atrial fibrillation, or invasive arteriographic procedure raises the possibility of arterial thrombosis or embolus respectively. A careful history from the patient, family, or caregivers will often enable a presumptive diagnosis to be established.

Tenderness or other local signs of inflammation are characteristic manifestations of an infectious process that may benefit from source-control measures. Invasive devices raise the possibility of a device-related infection such as catheter-related bacteremia, endocarditis, or sinusitis secondary to a nasogastric tube. Crepitus in the tissues suggests the possibility of a gas-forming necrotizing soft tissue infection.

However, physical examination alone is rarely sufficient to diagnose an infectious focus and to plan its optimal treatment. Contrast-enhanced computed tomography (CT) has emerged as the single most useful diagnostic modality, because it not only identifies a pathologic process resulting in infection but also provides information on the nature of the local infectious process: is there a discrete, walled-off abscess or diffuse nonlocalized infected fluid?; is there tissue necrosis with evidence of nonperfused tissue? CT scanning provides a means of accessing an infectious focus using percutaneous techniques.

Other radiologic techniques provide useful information in selected circumstances. A plain film of the chest or abdomen may show free intraperitoneal air, and so establish a diagnosis of a perforated viscus (typically a duodenal ulcer or sigmoid diverticulitis) (Fig. 4). Ultrasonography is particularly useful in evaluating the biliary tree.

Drainage

Drainage techniques are useful when there is a well-localized collection of infected fluid, for example, an intra-abdominal abscess, a liquid empyema, or an infected joint. The optimal approach to drainage is one that creates a controlled sinus or fistula with the least trauma to the patient. Image-guided percutaneous drainage is generally the
approach of choice; however, operating may be preferred if the collection is inaccessible to radiologic drainage, if there are multiple loculated or diffuse poorly localized collections, if percutaneous techniques have failed, if there is a significant component of infected solid tissue, or if operating can safely expedite the definitive management of the source of infection.

Percutaneous drainage is accomplished under local anesthesia using CT or ultrasound imaging. The collection is visualized, and an access route selected that avoids passing the drain through the bowel or other structures. After the abscess cavity has been entered using a finder needle, the tract is dilated using a Seldinger technique, and a flexible pigtail catheter inserted. The technique causes minimal injury to surrounding tissues; however, the small lumen of the catheter makes it susceptible to occlusion by local fibrin plugs. For the patient with multiple abscesses or a single complex multiloculated collection, several drains may be needed to accomplish adequate drainage.

The optimal surgical approach depends on the clinical context. For the patient presenting with a complex intra-abdominal process, a midline laparotomy provides optimal exposure and access, and so facilitates management. Laparoscopic approaches are an alternative to open exploration in centers where the expertise is available, and are particularly useful in the management of perforated ulcers. However, the need for pneumoperitoneum, with its adverse effects on venous return, limits the role of laparoscopy in a hemodynamically unstable patient. Anastomotic leaks or postoperative collections are usually approached through the original surgical incision, provided that exposure is adequate; a new midline incision is a useful alternative if access through the original incision is restricted. Percutaneous techniques have largely eliminated the extraperitoneal approaches classically employed for the management of subphrenic abscesses.

Open-abdomen approaches have fallen out of favor in the management of intra-abdominal infection. They have not been shown to improve outcome, and are associated with an increased risk of fistulas, and an increased nursing workload. Most surgeons leave the abdomen open only when closure is not technically possible or would result in abdominal compartment syndrome.

Fig. 4. Upright chest X-ray demonstrating free air under the right hemidiaphragm of a patient with a perforated duodenal ulcer.
Debridement

Only liquids can pass through a drainage tube; infected or necrotic solid tissue must be physically excised, a process known as debridement. This goal can be accomplished through the use of wet-to-dry dressings or chemical debriding agents, but surgery is the mainstay of therapy.

When an infectious process is of sufficient severity to result in life-threatening sepsis, surgical excision is invariably needed, although the timing of intervention may vary with the anatomic site of origin. Certain infections (notably necrotizing soft tissue infections or intestinal necrosis) follow a rapid course to a fatal outcome in the absence of intervention; emergent surgical intervention is life saving. Debridement of a necrotizing soft tissue infection requires the resources of an operating room and general anesthesia. The involved area is incised, and necrotic tissue excised back to viable tissues, identified by bleeding from the wound edges. It is often difficult to assess the extent of necrosis at the initial procedure, and so the patient should be returned to the operating room in 24 hours and on a daily basis for further debridement, until the tissue necrosis is fully controlled, and granulation tissue is forming at the wound margins. Subsequent wound management is aided by the use of a negative pressure wound vacuum device. The need for amputation in necrotizing soft tissue infections of the extremity is dictated by the amount of muscle excised, and the prospects for functional recovery; amputation can be avoided in the majority of cases.

Intestinal infarction requires the excision of the necrotic bowel, and should be performed as rapidly as possible. The demarcation between viable and nonviable bowel is usually clear if the cause of the infarction is a closed-loop obstruction or arterial embolism, but may be more difficult to determine in cases of arterial or venous thrombosis, or in infarction associated with a low-flow state. If the extent of ischemia cannot be reliably assessed at the time of the initial operation, the patient should be taken back to the operating room in 24 to 48 hours for a second-look laparotomy or laparoscopy. In an unstable patient, it is often safest to resect bowel that is visibly necrotic, leaving the ends stapled off in the peritoneal cavity, and closing the abdomen with a temporary abdominal-closure device. Further resection and anastomosis with formal abdominal closure can then be accomplished at the time of second-look laparotomy.

Device Removal

Although removal of a colonized device provides optimal source control in device-related infections, it is apparent that the morbidity of removal of a urinary or vascular catheter is significantly less than that of an infected vascular graft or heart valve (Fig. 5). In many patients, vascular access is challenging to obtain because of venous thrombosis and prior catheter use, and it may be preferable to leave the device in situ, and treat instead with a prolonged course of antibiotics.

SOURCE CONTROL IN INFECTIONS THAT ARE COMMON CAUSES OF SEPSIS

INTRA-ABDOMINAL INFECTION

Source-control decisions are often defined by multiple factors, and guided by the application of principles rather than normative data from large clinical trials. Some of those principles are discussed earlier in this article, but a few scenarios that are relatively common in patients with severe sepsis merit further comment. Decisions regarding optimal source control should be made by an experienced surgeon who can assess the relative merits of operative versus nonoperative management in an individual patient.
Gastrointestinal Perforation

Perforation of the stomach or duodenum secondary to ulcer disease, or of the sigmoid colon secondary to diverticulitis, is a common community-acquired cause of intra-abdominal infection leading to sepsis. Although it is the most common cause of the acute abdomen, appendicitis typically runs a more benign clinical course, and is rarely the cause of severe sepsis or significant clinical morbidity.

The diagnosis of a perforation of the gastrointestinal tract is typically presumptive, based on evidence of free intraperitoneal air or radiographic contrast material (Fig. 6). The need for surgery is dictated by the extent to which the procedure is localized, and the urgency of intervention by the clinical state of the patient. Thus a walled-off collection can usually be managed by percutaneous drainage, whereas evidence of diffuse peritoneal contamination identifies a situation that requires surgical intervention. The clinical state of the patient (not the findings of radiographic investigations) is the prime factor in deciding about the timing of intervention. A hemodynamically unstable patient with rapidly evolving organ dysfunction should have immediate intervention, whereas a patient who is clinically stable can be dealt with after deliberation.

In the stable patient, a walled-off perforation of a duodenal ulcer can be managed nonoperatively, because normal peritoneal defenses have accomplished the objective of creating a controlled sinus. Poor localization, suggested by diffuse pain on abdominal examination or physiologic instability, suggests that operative intervention is needed. Current approaches are to patch a duodenal ulcer defect with omentum, or excise a perforated gastric ulcer; open and laparoscopic approaches are employed.

Spontaneous colonic perforations most commonly arise in an area of diverticular disease, and originate within the sigmoid colon; resection of the sigmoid removes the focus of ongoing contamination and prevents future episodes. Nonetheless, if the perforation is localized, initial management by percutaneous drainage is the preferred approach (see Fig. 2), with definitive resection deferred to a later date. When percutaneous techniques are not possible, there are several options. Historically, a 3-stage approach was preferred, consisting of an initial laparotomy to drain the abscess and a proximal colostomy to defunction the sigmoid colon, a second procedure to resect the sigmoid and anastomose the descending colon to the rectum, and finally a third stage, the closure of the colostomy. This 3-stage approach has given way to a 2-stage approach comprising an initial laparotomy with resection of the diseased sigmoid colon and drainage of the abscess, closure of the rectal stump, and creation of an end colostomy from the end of the descending colon (the so-called...
Hartmann procedure). A small randomized trial\(^{33}\) and a synthesis of data from a series of comparative studies\(^{34}\) suggest that the 2-stage procedure results in lower rates of morbidity and mortality, particularly when the morbidity and mortality of the subsequent procedures necessary to restore intestinal continuity are considered. More recent case series suggest that resection and primary anastomosis is at least as good, if not better, than a 2-stage approach.\(^{35-37}\) Another alternative in an unstable patient is a damage-control approach, consisting of sigmoid resection alone, leaving the stapled-off ends of the colon within the abdomen. The abdomen is closed with a temporary closure device, and the patient taken back to the operating room 24 to 48 hours later to complete the anastomosis and close the abdominal wall.\(^{38}\)

**Gastrointestinal Ischemia**

Gut ischemia is a common and potentially lethal cause of an acute abdomen. Bacteria and their products can escape from the ischemic gut, and are absorbed through regional lymphatics and the peritoneal cavity. Gut ischemia can arise from arterial occlusion by embolism or thrombus, venous occlusion, or globally reduced splanchnic perfusion.\(^{39}\) The responsible mechanism dictates the optimal management approach, and can often be inferred from the clinical presentation.

Acute arterial occlusion can result from embolus or thrombosis, from vasculitis, or from compression of the arterial inflow by adhesive bands or an intestinal volvulus, leading to a closed loop obstruction (Fig. 7).\(^{40,41}\) Because of its gentle takeoff from the aorta, and lack of reliable collateral flow, the superior mesenteric artery is the most commonly affected vessel, with the result that ischemia involves the small bowel and the colon to the level of the distal transverse colon. The diagnosis is suggested by the appropriate clinical setting, reflecting risk factors such as atrial fibrillation, recent myocardial infarction, or antecedent invasive aortic angiographic procedure in the case of embolism, or severe preexisting occlusive vascular disease in the case of thrombosis, and the development of severe midgut abdominal pain, localized to the periumbilical region. Clinical features such as profound leukocytosis or acidosis are suggestive of, but neither sensitive nor specific for, the diagnosis of intestinal
ischemia. Thrombosis typically involves the orifice of the takeoff of the superior mesenteric artery, and results in ischemia or infarction that involves the small bowel from the ligament of Treitz through to the midtransverse colon. Emboli characteristically lodge distal to the first jejunal branch of the superior mesenteric artery, and thus approximately 20 to 30 cm of jejunum is spared. Venous infarction arises in patients with preexisting prothrombotic disorders, and is associated with engorgement of the affected gut that can often be identified on ultrasonography. Nonocclusive mesenteric ischemia occurs in patients with hypotension, and more commonly involves the colon.

The diagnosis is suggested by the clinical setting and is most commonly confirmed by CT scan. Clinical judgment is paramount, for radiologic investigations are not completely sensitive, and conversely, intuitively ominous findings such as portal venous gas do not necessarily imply gut ischemia. The key to successful source-control management of gut ischemia is emergent intervention to restore vascular flow or resect gangrenous intestine. The appropriate clinical setting and a high index of clinical suspicion are key to initiating the investigations and surgical interventions that can accomplish this objective.

Infected Pancreatic Necrosis

Secondary infection of necrotic pancreatic and peripancreatic tissue is a common complication of necrotizing pancreatitis of sufficient severity to warrant admission to the intensive care unit. Infection is a late complication, typically arising several weeks after the onset of illness, and involving enteric organisms that have translocated from the adjacent gastrointestinal tract.

Contemporary management of the patient with suspected infected necrosis has changed dramatically in recent decades. The diagnosis is suspected on the basis of CT findings of necrosis and gas, and confirmed by fine-needle aspiration of the involved area. Case series and a single randomized controlled trial support contemporary approaches to surgical management based on delaying intervention until the area of necrosis has become well walled-off from surrounding tissues, a process that typically is not evident for 3 or 4 weeks or longer after the onset of
the disease. Percutaneous drainage of the liquid component of a complex pancreatic infection can temporize (Fig. 8), and may in some circumstances suffice to effect resolution of, the illness.⁵⁰

**Other Intra-Abdominal Infections**

Cholangitis secondary to occlusion of the common bile duct by a gallstone produces a characteristic clinical syndrome of jaundice, fever, and right upper quadrant pain, and, in severe cases, hypotension and changes in mental status. Relief of the obstruction, usually by endoscopic retrograde pancreatography with papillotomy and stone extraction, results in rapid correction of the physiologic derangements.⁵¹ Acute cholecystitis or liver abscess is an uncommon cause of severe sepsis and septic shock; percutaneous drainage is effective initial management for both.⁵²

The management of postoperative peritonitis poses unique challenges arising from the nature of the antecedent procedure that led to the infection, the timing of diagnosis, the stability of the patient, and the skills and experience of the surgeon. Treatment must be individualized based on core principles articulated earlier in this article: the establishment of a controlled sinus or fistula by the simplest approach possible, and the removal of necrotic infected tissue.

The patient with suspected intra-abdominal infection but a nondiagnostic or negative abdominal CT scan is a common clinical scenario, and a particular source of friction between intensivists and surgeons. As a general principle, an intra-abdominal complication of sufficient severity to produce a clinical picture of severe sepsis or septic shock reflects a significant anatomic derangement that will be evident using modern CT scan techniques, and there is no credible evidence that nondirected or “blind” laparotomy is beneficial.⁵³ On the other hand, when imaging resources are not available, or in the rare situation where the clinical picture suggests a specific cause, laparotomy may be diagnostic and therapeutic. The decision is difficult, because a nontherapeutic laparotomy carries risk, and so the active involvement of an experienced surgeon is critical.

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**Fig. 8.** Percutaneous drainage of a pancreatic abscess. The use of a percutaneous drain permits decompression of a complex pancreatic composed of infected pancreatic fluid and necrotic peripancreatic fact, allowing definitive surgical management to be delayed until the abscess cavity has become well demarcated from surrounding viable tissue. (*Black arrow* identifies the abscess cavity, and the *white arrow*, the percutaneous drain).
THORACIC INFECTIONS

The role of source control in patients with pneumonia is limited. Physiotherapy may be considered a form of source control directed at mobilizing pulmonary secretions, but it has not been shown to improve prognosis or hasten the resolution of community-acquired pneumonia.\(^{54,55}\) Similarly, tracheal suctioning does not seem to alter the outcome in cases of established pneumonia, although it has been shown to reduce rates of development of ventilator-associated pneumonia in several clinical trials.\(^{56,57}\) Since ventilator-associated pneumonia is a device-related infection, minimizing the duration of intubation and mechanical ventilation, or changing a colonized endotracheal tube, are logical management strategies, although they are difficult to evaluate, and so primarily of theoretical interest.

Source-control measures are important in the management of pleural or mediastinal infections. During the early course of an empyema, the infected pleural fluid is readily controlled by percutaneous drainage using a chest tube or an image-guided catheter. With time, activation of coagulation within the cavity results in the deposition of fibrin, which can entrap the lung and prevent its full reexpansion. In the early phases of fibrinopurulent empyema, video-assisted drainage is often successful, but with further organization, formal surgical decortication becomes necessary.\(^{58}\)

Mediastinitis most commonly arises following open cardiac surgery. Although a bacteriologic diagnosis can usually be established by percutaneous sampling, adequate source control generally necessitates open surgical drainage.\(^{59}\) Mediastinitis can also be a consequence of inferior extension of infection arising in the head and neck; successful management of these infections with percutaneous catheter drainage has been reported.\(^{60}\)

NECROTIZING SOFT TISSUE INFECTIONS

Necrotizing soft tissue infections include a variety of clinical syndromes that have in common the development of necrosis of the skin, subcutaneous fat, fascia, and, in some cases, underlying muscle. The terminology applied to such infections is confusing, and includes descriptions based on the infecting organism (for example, gas gangrene caused by \textit{Clostridia}), the anatomic site involved (for example, Fournier gangrene), and the specific tissues involved (for example, necrotizing fasciitis). In reality these are overlapping syndromes, for which the key issue with respect to source control is the presence of necrosis, rather than simply inflammation. Infections with \textit{Clostridia} or group A streptococci are particularly virulent and evolve rapidly; emergent diagnosis and management are essential to minimize morbidity and mortality.

The presence of underlying necrosis may be suggested by bullae, ecchymosis, or discoloration of the overlying skin, or by crepitus on physical examination; however, these findings are not particularly sensitive. Radiographic examination may reveal air in the tissues, but this finding too lacks sensitivity and specificity for the diagnosis.\(^{61}\) Fascial thickening and edema on CT scan suggest the diagnosis,\(^{62}\) and biopsy or aspiration of the suspected area may establish a bacteriologic diagnosis. Nonetheless, it is not uncommon that the diagnosis is established through surgical exploration of the area of suspected necrosis, an approach that is sensitive and specific, and curative when positive, with only minimal additional morbidity when it is not.

The treatment of a necrotizing soft tissue infection entails wide debridement of all necrotic tissue.\(^{63}\) Because the extent of tissue necrosis may not be apparent at the initial operation, initial debridement back to bleeding tissue should be followed by
reexploration in 24 hours with further debridement as indicated. Reexploration is repeated as long as significant amounts of newly necrotic tissue are encountered.

URINARY TRACT INFECTIONS

Urinary tract infections typically carry a better prognosis than other infections that produce the clinical syndromes of severe sepsis and septic shock. Source-control measures are indicated in the event of abscess or obstruction, and in cystitis arising in association with a colonized urinary catheter.

The diagnosis of urinary tract infection is established based on clinical features, supplemented by urinalysis and culture, and appropriate imaging studies. Ultrasonography and CT scanning have emerged as the preferred diagnostic modalities for infections involving the kidney and upper urinary tract.64

Most infections of the upper urinary tract can be managed adequately by a combination of systemic antibiotics and percutaneous drainage of an obstructed ureter or renal abscess.65 The need for nephrectomy is uncommon. Cystitis is readily managed by catheter change.

SUMMARY

This brief overview of the role of source control in sepsis emphasizes the underlying principles rather than the empiric evidence from well-performed clinical studies. The reasons for this are several. First there is a paucity of high-level published evidence, with few rigorous large clinical series, and even fewer clinical trials. Second, the decision-making process in the individual patient is complex, and often not amenable to study using the design of a randomized controlled trial, for decisions involve consideration not only of the underlying disease but of the stability of the patient, the presence of comorbidities, and the prior surgical history, all factors that can heavily influence the decision to choose one therapeutic option rather than another. The scope of the topic is large, and the space limited. Interested readers are referred to more detailed discussions such as that found in the background to the recommendations on source control in the guidelines of the Surviving Sepsis Campaign.1

Source control is a core treatment modality in the management of the patient with severe sepsis or septic shock. Its optimal use assumes a comprehensive knowledge of biologic principles, the complexities of the septic response, and the range of surgical and nonsurgical options, and a combination of therapeutic aggressiveness and judicious caution in the clinician charged with making the decision. As every intensivist learns, appropriate source-control intervention can rapidly alter the course of sepsis to a more favorable direction, and suboptimal decision-making can change a difficult clinical challenge into a nightmare.

REFERENCES


