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Principles of Source Control in the Early Management of Sepsis

John C. Marshall

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Abstract Source control refers to the spectrum of physical measures that are undertaken to control a focus of infection, and to facilitate restoration of optimal anatomy and physiology. These measures are classified as *drainage*—the evacuation of infected liquid through the creation of a controlled sinus or fistula, *debridement*—the physical removal of necrotic infected tissue, *device removal*, and the *definitive measures* that comprise the process of reconstruction and rehabilitation. Effective and timely source control is critical to the successful management of life-threatening infection. This article reviews the principles of diagnosis and source-control management, and their application to common infections that result in severe sepsis and septic shock.

Keywords Source control · Severe sepsis · Septic shock · Peritonitis · Intra-abdominal infection · Pancreatitis · Diverticulitis · Cholangitis · Drainage · Debridement · Surgery · Necrotizing soft-tissue infection · Empyema · Device-related infection

Introduction

The successful management of life-threatening infection is grounded in two principles: early and effective elimination of invasive infection, and successful reversal of the systemic physiologic derangements arising through the innate host response to that infection. The latter is the focus of other papers in this issue. Control of infection requires eradication of the infecting organism(s) through the use of appropriate antimicrobial agents and timely and judicious source-control measures.

Source control can be defined as any physical intervention that is undertaken to remove or eliminate a focus of invasive infection and to restore optimal anatomic function [1]. Its elements are four: 1) *drainage*—the evacuation of a discrete focus of infected fluid through its conversion to a controlled sinus or fistula; 2) *debridement*—the physical removal of devitalized infected tissue; 3) *device removal*—the removal of devices or foreign bodies that have become colonized with microorganisms, and serve as a locus of ongoing microbial seeding; and 4) *definitive reconstructive measures*—all those interventions that serve to reconstruct the damage resulting from infection or its management, and to restore the individual to an optimal state of function and well-being.

Effective source control is grounded in a basic understanding of the biologic alterations elicited by an infecting organism and the response this evokes in the host. The principles of source control are straightforward, but their successful application hinges on the integration of these principles with the specific nuances of the clinical setting.

Biologic Principles of Source Control

Source-control measures can be successfully used in one of three circumstances. First, if the infection arises from colonization of an indwelling foreign body (eg, a vascular catheter or a prosthetic hip joint), removal of the foreign body is usually sufficient to effect resolution of the infection. When the infectious process induces tissue necrosis, or when necrotic tissue becomes secondarily

J. C. Marshall (✉)
Department of Surgery, University of Toronto,
and the Li Ka Shing Knowledge Institute, St. Michael's Hospital,
4th Floor Bond Wing, Room 4-007, 30 Bond Street,
Toronto, Ontario M5B 1W8, Canada
e-mail: marshallj@smh.ca

infected, the excision of the necrotic tissue removes the bulk of the bacterial burden, and leaves vascularized tissue to which antibiotics can be delivered. Finally, innate host defenses serve to contain a focus of infection, and to isolate the infectious process from adjacent sterile tissues. The consequence of these defenses is the formation of an abscess.

Abscess Drainage: Creation of a Controlled Sinus or Fistula

An *abscess* is a well-circumscribed collection of neutrophils, bacteria, cellular debris, and tissue fluid, contained within a fibrous capsule that isolates the abscess contents from surrounding tissues. If the abscess communicates with an air-filled structure (eg, the gastrointestinal tract or bronchial tree), or if the bacteria within the abscess are gas-forming organisms, the abscess will also contain air. The initial response to local infectious challenge is the activation of a local inflammatory response [2]. Neutrophils are recruited to the site through the activity of chemokines released by resident macrophages, local blood flow is increased because of local vasodilatation secondary to increased release of nitric oxide, and capillary permeability is increased through the activity of inflammatory cytokines. These same cytokines augment the expression of tissue factor on mesothelial cells, and so activate the coagulation cascade, fed by the influx through the leaky endothelium of plasma that is rich in coagulation factors [3]. Activation of the coagulation cascade results in local generation of fibrin that becomes organized to form granulation tissue and the fibrous capsule that is characteristic of a mature abscess. Increased blood flow into the capsule of the abscess results in rim enhancement on a CT scan.

Local activation of coagulation converts a potentially invasive infection into a contained abscess, but the collection remains within the body, and can still induce a systemic inflammatory response, albeit an attenuated one. Complete resolution of the infection requires that the contained infection be externalized, a process that is termed *drainage.* Drainage converts a closed-space infection into a controlled sinus or fistula. A *sinus* is a tract between a deep-space cavity and the exterior, whereas a *fistula* is an abnormal communication between two epithelially lined surfaces. In the absence of any of the well-known factors that prevent its closure, a fistula will close spontaneously, and thus the consequence of surgical or percutaneous drainage is complete resolution with obliteration of the abscess cavity and the tract by which it was drained. The factors that prevent fistula closure include distal obstruction, the presence of a foreign body, epithelialization of the fistulous tract, uncontrolled local inflammation, malignancy along the tract, or tissue changes induced by radiation or inflammatory bowel disease. These principles underlie the success of many standard surgical procedures. For example, the tract by which an abscess is drained or a tracheostomy is created are fistulae that are kept open by deliberate placement of a foreign body—the drain or the tracheostomy tube. When the foreign body is removed, the tract will close. Similarly, a colostomy is a fistula that remains open because the tract is epithelialized by suture of the gut mucosa to the skin.

Debridement: Removal of Devitalized Tissue

Ischemic tissue is a potent growth medium for bacteria. Products of dying cells provide nutrients for bacteria, while the lack of blood flow precludes the delivery of antibiotics or host-derived immune cells or mediators. Moreover, the release of intracellular constituents from dying cells can activate host defenses and evoke an inflammatory response [4].

Debridement is the removal of ischemic or necrotic tissue. It may entail the surgical resection of a segment of gangrenous small bowel in a patient with a strangulating bowel obstruction, the removal of necrotic peripancreatic tissue in a patient with necrotizing pancreatitis, or the excision of necrotic skin, subcutaneous tissue, and muscle in a patient with necrotizing fasciitis. Typically, debridement requires surgical intervention, although the use of dressing changes to remove necrotic slough on an open wound is also an example of debridement.

Effective debridement requires that all dead tissue be removed, and that only well-vascularized healthy tissue remains. This principle guides the surgical management of infections involving necrotic tissue. In the case of an acute intestinal infarction, for example, resection of the entire ischemic segment is performed. When gut infarction occurs secondary to a low-flow state, venous thrombosis, or patchy atherosclerotic disease, it may be challenging to determine the true extent of devitalized tissue; therefore, it is advisable to perform a "second-look" procedure in 24 to 48 h to ensure complete excision, and to resect any further necrotic tissue. This principle also applies to the management of necrotizing soft-tissue infections. Resection is carried into viable tissue, indicated by the occurrence of bleeding in the wound edges. However, because the extent of tissue necrosis is often difficult to ascertain at the time of the first procedure, repeat exploration and debridement is advisable. In contrast, the release of activated pancreatic enzymes in patients with severe necrotizing pancreatitis can induce necrosis of the retroperitoneal fatty tissues which, in turn, are at risk of secondary infection. Debridement of the retroperitoneum is challenging, because it is both anatomically inaccessible and coursed by large vessels (eg, the splenic artery and vein). Aggressive early debridement of peripancreatic necrosis carries the risk of uncontrollable

hemorrhage, and case series and a single randomized controlled trial show that delayed intervention is associated with a better clinical outcome [5].

Device Removal: Elimination of Bacterial Biofilms

Invasive devices are an important locus of infection in critically ill patients. Infection arises through the capacity of certain microorganisms to create a biofilm and to establish residence on the surface of the prosthetic device. Biofilms result from the capacity of certain bacteria to elaborate an exopolymeric matrix that adheres to devices such as vascular catheters [6], urinary catheters [7], and endotracheal tubes [8], and favors the growth of the organism in a sessile state. Biofilms protect the organism from the effects of antibiotics [9] and impair host oxidative defenses [10].

Removal of a colonized device provides definitive control for device-related infection. However, the risks associated with device removal vary with the nature of the devices (Table 1). Removal or change of a urinary catheter carries minimal risk, whereas the excision of an infected vascular graft or prosthetic heart valve is a major undertaking. Thus, therapeutic decision-making must be guided by the specific risks and benefits in the particular patient.

Establishing the Need for Source Control in Life-Threatening Infection

Source-control measures are applicable to most, but not all, infections. Although the selection of appropriate antimicrobial agents hinges on establishing a definitive microbiologic diagnosis, decision-making about source control requires that an anatomic diagnosis be established. It is important that the clinician consider the site and nature of the infection responsible for the clinical syndrome of sepsis: a diagnosis of severe sepsis or septic shock is inadequate to guide decision-making about source control.

Table 1 Ease of removal of colonized foreign bodies

Urinary catheter
Temporary vascular catheter
Arterial
Venous
Endotracheal tube
Intracranial pressure monitor
Permanent vascular access devices
Prosthetic joint
Vascular graft
Prosthetic heart valve

The inciting infection may be readily apparent by history or physical examination—abdominal pain and tenderness suggest an intra-abdominal focus, whereas soft-tissue infections are typically evident on physical examination. However, radiographic imaging is generally needed to accurately define the nature and extent of the infection. A chest radiograph may reveal air beneath the diaphragm, and so suggest the presence of a perforated intra-abdominal hollow viscus (Fig. 1a); free air may also be detected on an abdominal plain film as Rigler's sign demonstrating air on both sides of the wall of a loop of bowel (Fig. 1b); CT scanning is invaluable in defining the extent to which the intestinal contents are contained or disseminated throughout the free peritoneal cavity (Fig. 1c). Ultrasonography is useful in the evaluation of the biliary tree: for example, a distended stone-filled gallbladder with pericholecystic fluid establishes a diagnosis of acute cholecystitis, and a dilated common bile duct suggests cholangitis. In general, CT provides the most information in delineating a focus of infection and guiding treatment options.

The stability of the patient and the intrinsic virulence of the infection dictate the best approach to imaging. For most infections, the treatment delay resulting from a decision to perform preoperative imaging will have minimal impact, and the benefits of properly visualizing the infectious focus will outweigh the risks associated with an increased delay. Immediate intervention is indicated in one of two circumstances. First, certain fulminant necrotizing soft-tissue infections such as those caused by *Clostridia* spp or group A streptococci spread very rapidly, and their extent is best defined by surgical exploration. Similarly, abdominal infection in an unstable, deteriorating patient is best defined by exploration. Second, in some cases, the clinical context will define the diagnosis, and so obviate the need for imaging. For example, abdominal pain and septic shock following a difficult colonic anastomosis suggest a leak and the need for urgent intervention. The decision-making process regarding the need for and selection of imaging is best made by an experienced surgeon, because these decisions define the therapeutic approach that will be used.

Timing of Source-Control Intervention

The immediate priorities in managing the patient with severe sepsis or septic shock are hemodynamic resuscitation, obtaining cultures, and initiating appropriate broad-spectrum antibiotics. Source control is an urgent intervention and, as a rule, should be instituted as soon as possible after the patient has been stabilized and an accurate anatomic diagnosis established. However, there are exceptions to this general rule.

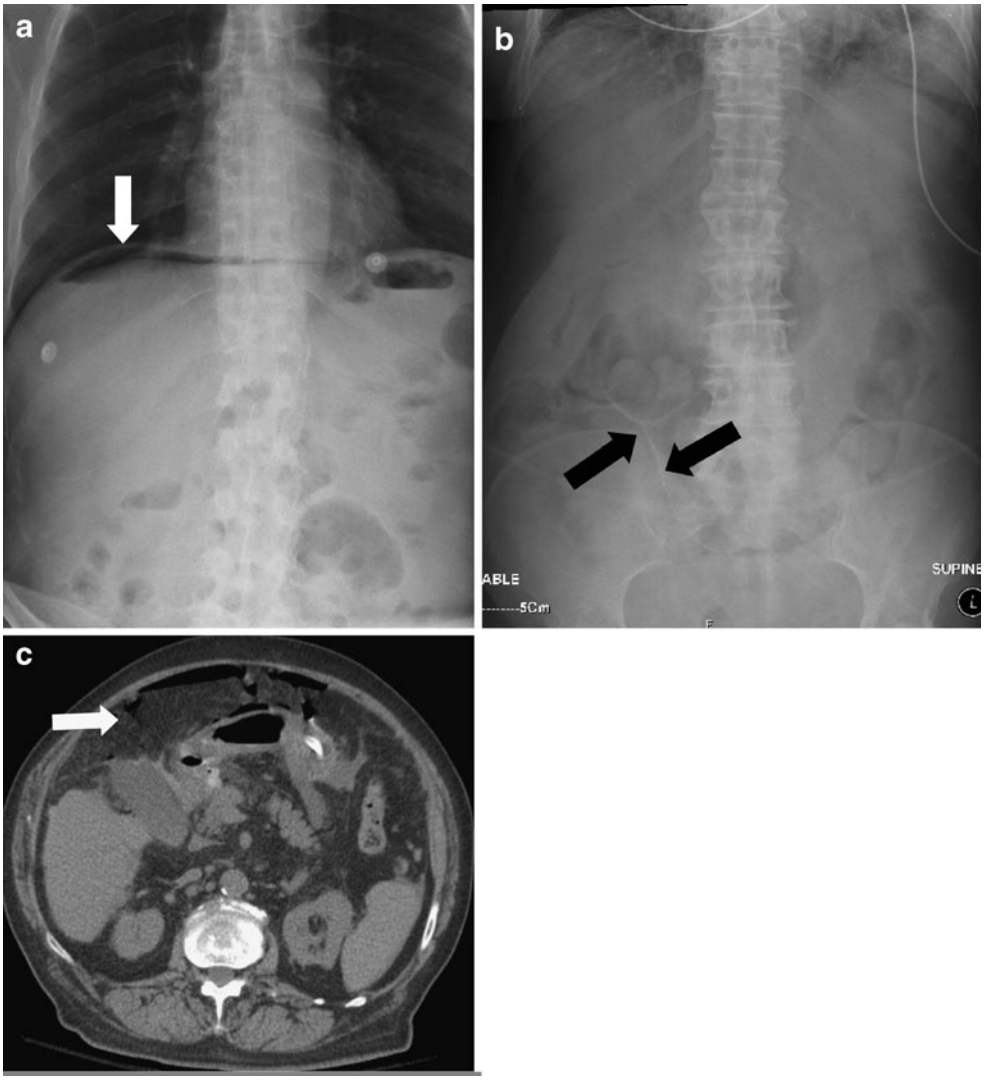

Fig. 1 **a**, Free subdiaphragmatic air (*white arrow*) in a patient with a perforated duodenal ulcer. **b**, The less common, and more subtle, sign of free intraperitoneal air is Rigler's sign, here demonstrating air on both sides of a loop of bowel in an ante-dependent position; the wall of a loop of small bowel is outlined by air within the lumen, and by air outside the lumen in the peritoneal cavity (*arrows*). **c**, Air and free intraperitoneal fluid (*arrow*) are shown on a CT scan of the same patient as in **b**; this 61-year-old woman had a perforated duodenal ulcer and extensive peritoneal soiling

Emergent intervention is indicated in necrotizing soft-tissue infections, particularly those caused by group A streptococci and *Clostridia* spp, and is associated with both improved survival and reduced tissue loss [11]. Similarly, intestinal ischemia mandates emergent intervention, which holds the possibility of restoring intestinal perfusion.

On the other hand, source control in patients with infected retroperitoneal necrosis secondary to necrotizing pancreatitis is best deferred until viable and nonviable retroperitoneal tissues have been clearly demarcated—typically at least 3 weeks after the onset of illness [12, 13]. Experience from multiple case series and a single randomized controlled trial reveals that the morbidity of early intervention outweighs the risks of delayed intervention [5]. The pancreas lies in the retroperitoneum, in intimate proximity to the splenic artery and vein; in the setting of an acute inflammatory response, surgical access to the region is challenging. With time, the tissue necrosis resulting from the leakage of activated pancreatic enzymes becomes demarcated, a process that is evident on CT scan as enhancement of the tissues surrounding the collection (Fig. 2a). Once demarcation has occurred, surgical intervention permits ready debridement of the infected necrotic tissue, and usually allows primary abdominal closure, thus obviating the need for repeat exploration.

Source Control in Specific Infections Causing Severe Sepsis

A comprehensive review of source-control options for the full spectrum of infections that can produce severe sepsis and septic shock is beyond the scope of this review article, and the reader is referred to more detailed discussions elsewhere [1, 14]. Here, we focus on the most common clinical syndromes encountered by the intensivist.

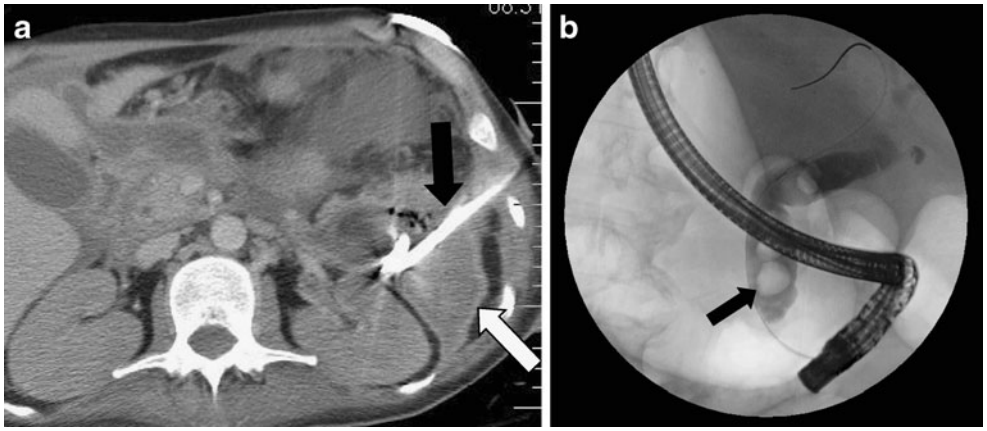

Fig. 2 **a**, Infected peripancreatic necrosis in a 37-year-old man. The *white arrow* demonstrates rim enhancement around a walled-off fluid collection that has been decompressed using a percutaneously placed drain (*black arrow*). He subsequently underwent successful operative debridement. **b**, Endoscopic removal of three obstructing gallstones causing acute cholangitis. The ampulla of Vater has been cannulated and the stones demonstrated (*black arrow*); a papillotomy permitted their removal and facilitated free drainage of the common bile duct

Intra-abdominal Infection

Intra-abdominal infections are the most common infections in critically ill patients for which source-control measures are indicated. They vary in their sites, microbiology, and optimal management strategies; however, they can be grouped into several clinical scenarios for which common source-control principles apply (Table 2).

Obstruction/Perforation of a Hollow Viscus

The gastrointestinal tract is a hollow tube, densely colonized with potentially pathogenic microorganisms, and in direct communication with two outpouchings of the gut itself—the biliary tree and pancreas and the appendix. Obstruction at any level results in an increase in intraluminal pressure which, transmitted to the intestinal wall, results in venous compression and a reduction in perfusion pressure; in addition, obstruction is associated with bacterial overgrowth in the obstructed segment. Obstruction alone, however, is rarely responsible for the activation of a systemic septic response, but rather is an indication for urgent interventions to relieve the obstruction, and prevent progression to ischemia and perforation.

Cholangitis is the exception to this general principle. Obstruction of the bile duct is typically a consequence of impaction of a gallstone, and less frequently, of obstruction of a therapeutically placed stent or of a surgical anastomosis of the bile duct to the gastrointestinal tract [15]. Pressures rise rapidly within the obstructed bile duct, and the responsible bacteria pass readily into the circulation with the result that bacteremia is common. Source control consists of ductal decompression, usually by endoscopic retrograde cholangiopancreatography (ERCP) [16] (Fig. 2b), although percutaneous transhepatic drainage or open common bile duct exploration are options when ERCP is unavailable [17].

The approach to a perforation of the gastrointestinal tract depends on whether the perforation is contained or into the free peritoneal cavity and, in the case of a contained

Table 2 Common intra-abdominal indications for source control

Obstruction/perforation of a hollow viscus
Gastric or duodenal ulcer
Appendicitis
Sigmoid diverticulitis
Acute cholecystitis
Cholangitis
Intestinal ischemia/infarction
Arterial thrombosis or embolism
Venous thrombosis
Strangulating obstruction
Low-flow state
Surgical complication
Anastomotic leak
Inadvertent enterotomy
Postoperative abscess
Solid-organ abscess
Liver
Spleen
Kidney
Gynecologic infection
Pelvic inflammatory disease
Endometritis
Retroperitoneal infection
Infected pancreatic necrosis
Pyelonephritis
Psoas abscess

perforation, whether there are liquid contents amenable to drainage. If the contents are within a well-circumscribed abscess, then drainage is possible. In general, percutaneous techniques are preferred because they accomplish the objective of creating a controlled sinus or fistula with less upset to the host [18]. On the other hand, in the absence of localization of the infectious process, operative intervention is needed to gain adequate control. Contained perforations without associated drainable fluid collections can be managed by systemic antibiotics alone. This approach—standard in the management of acute diverticulitis—is also applicable to the management of appendiceal phlegmons or contained perforation of a duodenal ulcer [19]; contained perforations such as these, however, rarely result in severe sepsis.

Perforated sigmoid diverticulitis is a relatively common cause of severe sepsis and septic shock. Source-control options depend on the consequences of the process. A localized abscess can be managed using percutaneous drainage, an approach that facilitates later management of the underlying diverticular disease by enabling an elective resection with primary anastomosis. The conventional approach to a poorly contained diverticular perforation is the so-called Hartmann procedure, consisting of surgical resection of the involved sigmoid colon, and construction of an end colostomy; the rectal stump is stapled off and left in the pelvis. This approach yields superior results to the older technique of a three-stage procedure: surgical drainage of the abscess with creation of a proximal loop colostomy, delayed sigmoid resection with anastomosis, and ultimately closure of the colostomy [20]. Several studies have suggested that resection with primary anastomosis is safe, even in the setting of free perforation with peritonitis [21, 22]. More recently, evidence has emerged suggesting that perforated sigmoid diverticulitis can be effectively managed by laparoscopic irrigation of the peritoneal cavity, without definitive management of the involved colon [23, 24].

Intestinal Ischemia/Infarction

Intestinal ischemia is a surgical emergency [25]. Rapid intervention can prevent intestinal infarction and the potential consequence of short gut syndrome. By the time that a clinical syndrome of severe sepsis has evolved, intervention is more commonly undertaken to remove gangrenous gut with the goal of saving the patient's life. Adequate source control requires the removal of all devitalized intestine. When intestinal infarction is the result of a strangulating obstruction or an arterial embolism, differentiation of viable from nonviable bowel is usually obvious, and so a single resection typically provides definitive therapy. When the viability of the bowel is less certain (eg, in patients with arterial or venous thrombosis, or ischemia resulting from a low-flow state), then resection with a planned second-look laparotomy in 24 to 48 h is advisable.

Complications of Intra-abdominal Procedures

A procedural complication should be suspected when evidence of sepsis occurs in the setting of a prior surgical procedure; such complications typically become manifest between 2 and 7 days following the intervention, although catastrophic complications such as complete dehiscence of anastomosis may present earlier [26]. Common causes include a leak from an anastomosis, an inadvertent and unrecognized injury to the gastrointestinal tract, or an infected hematoma. Optimal management depends on the extent to which the process is contained, and may range from expectant observation, through percutaneous drainage, to surgical exploration. Intra-abdominal infection arising as a complication of another procedure carries a higher risk of mortality, in part because its early manifestations are nonspecific signs of new organ dysfunction, and it is frequently misdiagnosed as volume overload, pneumonia, pulmonary embolism, or atrial fibrillation.

Solid-Organ Abscess

Abscesses of the liver or spleen are uncommon, and usually arise from hematogenous bacterial spread [27]. Percutaneous drainage is usually the preferred treatment approach, although splenectomy is often needed in the case of splenic abscesses [28].

Pancreatic Infections

Secondary infection of necrotic retroperitoneal fat or of intra-abdominal collections of pancreatic fluid is a relatively common complication of necrotizing pancreatitis of sufficient severity to warrant admission to the intensive care unit. These infections typically develop 2 to 3 weeks after the start of the episode, and are thought to occur primarily as a consequence of microbial translocation from the adjacent small bowel. As a result, the infecting flora comprises aerobic organisms; the presence of an anaerobe suggests the likelihood of a concomitant colonic perforation.

Early intervention in patients with pancreatic necrosis results in substantial morbidity and mortality, because the areas of necrosis are patchy, and so viable tissue is not sharply demarcated from nonviable tissue. The principle of debridement into bleeding tissue that guides the early management of necrotizing soft-tissue infections (see below) is not applicable in patients with retroperitoneal

necrosis from severe pancreatitis, because the area is not readily accessible anatomically, and because major vessels such as the splenic artery and vein and the superior mesenteric vein often course through, or in immediate proximity to, the area of necrosis. Debridement before viable tissues have become clearly demarcated from nonviable tissues carries a significant risk of major uncontrollable bleeding. On the other hand, retroperitoneal necrosis, and even infected retroperitoneal necrosis, is remarkably well tolerated and temporizing measures such as systemic antibiotics and percutaneous drainage, although usually not therapeutically definitive, can allow delayed surgical intervention. Both the presence of infection and the selection of appropriate antibiotics can be established through the use of CT-guided sampling of areas of suspected infected necrosis [29].

Case series and a randomized trial have established that delayed intervention in patients with infected necrosis results in enhanced survival [5, 12]. A recent Dutch randomized trial showed reduced rates of complications and organ dysfunction when percutaneous drainage was used as a strategy to facilitate delayed surgical intervention [30•]. Delayed intervention has the further advantage of obviating the need for open abdomen approaches, and so reducing the delayed morbidity associated with rehabilitation and reconstruction.

Necrotizing Soft-Tissue Infections

Uncomplicated cellulitis is an uncommon cause of severe sepsis. However, soft-tissue infections associated with tissue necrosis are life-threatening; early recognition and emergent surgical intervention are critical to a successful outcome [11, 31, 32]. The clinical presentation may suggest the urgency of the situation. Fulminant infection caused by group A streptococci or *Clostridia* spp often present dramatically with severe pain, physiologic instability, and evidence of a rapidly progressive soft-tissue infection characterized by bullae or tissue discoloration. More indolent presentations often occur, however, particularly in the polymicrobial infections arising in patients with underlying comorbidities (eg, diabetes or cirrhosis).

Clinical examination may disclose crepitus in the tissues, although this finding tends to be a late clinical sign. Air in deep tissue planes may also be evident on plain films or CT examination [33]. Tissue biopsy with frozen section has been promoted as a reliable diagnostic tool [34]; however, the simplest and most definitive diagnostic procedure is surgical exploration, looking for signs of bleeding when the tissues are cut.

Surgical debridement of all necrotic infected tissue is the mainstay of source control for a necrotizing soft-tissue infection. The initial debridement should be performed under general anesthesia to ensure surgical adequacy, and resection is carried out until the wound margins bleed readily and are clearly viable. Muscle involvement is relatively uncommon, with the result that limb salvage is often possible. Following the initial debridement, the patient should be further explored at intervals of 24 to 48 h, until it is apparent that the necrosis has been controlled. Subsequent management with a vacuum-type dressing facilitates wound care; skin grafting or other reconstructive procedures are delayed until adequate granulation tissue is present throughout the wound, and the patient is clinically stable.

Thoracic Infections

An empyema is an infection of the pleural space; its biologic evolution and management follow the same principles as those articulated above for the management of an intra-abdominal infection. Definitive diagnosis is established by culturing viable microorganisms from pleural fluid. In the earliest stages of empyema development, the empyema fluid is liquid, and readily drained through a chest tube [35]. As the infection evolves, fibrin deposition and reorganization serve not only to localize the infectious process, but also to restrict full expansion of the lung. Surgical removal of the fibrous peel—either by open decortication or by video-assisted surgery—is needed to clear the infection and re-expand the lung. Occasionally, a chronic empyema is managed by marsupialization of the empyema cavity, a procedure known as a Clagett window.

Mediastinitis may arise as a consequence of a sternotomy, as a result of a perforation of the esophagus, or as a descending infection from the neck. Access via less invasive routes is difficult, and open procedures are typically required to obtain adequate source control.

Pneumonia is not usually regarded as an infection for which source-control measures are effective. However, chest physiotherapy can be considered a form of source control, and ventilator-associated pneumonia is a device-related infection. The value of changing a colonized endotracheal tube has not been established, although evidence exists that measures that reduce the density of microbial colonization of the endotracheal tube can reduce the risk of infection [36].

Conclusions

Source-control measures are a critical component of the successful management of the septic patient. Their application has been largely the domain of the surgeon, and the evidential basis for their use is limited at best, in part because management strategies are complex, and must

often be individualized to the specific situation. Nonetheless, it is possible to articulate a series of principles that can aid the nonsurgeon in understanding the thought processes that guide their application.

First, it is essential to recognize the possibility that the clinical syndrome of severe sepsis in an individual patient may arise from an infection for which source-control measures are beneficial. Because deep tissue space infections may present subtly with the only manifestation being otherwise unexplained organ dysfunction [37], a high index of suspicion must be maintained. Moreover, the role of source control in device-related infections (eg, intravenous catheter-associated bacteremia or urinary tract infection) must be borne in mind.

As a general rule, source control is an urgent requirement that should be undertaken only once cultures have been obtained, antibiotics started, and hemodynamic stability achieved. The urgency of intervention is greater for patients with intestinal ischemia or necrotizing soft-tissue infections. Conversely, intervention is best delayed for patients with peripancreatic infections in the setting of necrotizing pancreatitis.

The specific mode of source control is dictated by the nature of the infected tissue. Drainage is appropriate for infected fluid collections, whereas debridement is required for the removal of infected solid tissues. Devices that have become colonized should be removed when removal can be done safely. In all cases, the optimal approach is that which accomplishes the objective of eliminating or exteriorizing a focus of infection with the least anatomic and physiologic upset to the patient. This principle not only minimizes the acute risk to the patient, but optimizes circumstances for subsequent reconstruction and restoration of an optimal quality of life.

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