ELSEVIER
SAUNDERS

Surg Clin N Am 87 (2007) 73–93

SURGICAL
CLINICS OF
NORTH AMERICA

Damage Control in Trauma: Laparotomy Wound Management Acute to Chronic

Timothy C. Fabian, MD

Department of Surgery, University of Tennessee Health Science Center, 956 Court Avenue, Suite G228, Memphis, TN 38163, USA

Background

The concept of damage control laparotomy for seriously injured trauma patients was promulgated in 1983 [1]. At the Grady Memorial Hospital, Stone and colleagues documented improved outcomes in those patients who sustained intra-abdominal injury with associated blood loss by rapidly terminating the surgical procedure. They described managing life-threatening blood loss and vascular injury quickly followed by immediate closure of the abdomen without completing definitive management of bowel and other intra-abdominal injuries. Bowel ligation and packing were recommended with return to the operating room for definitive management of hollow viscus injuries after a patient's physiology had been corrected. Those clinical investigators recognized that hemorrhagic shock led to a progressive downward spiral that could be interrupted in some patients by stopping major bleeding and closing the abdomen to diminish the loss of body heat. This unremitting, cataclysmic event subsequently has been referred to as the "bloody vicious cycle" [2]. Moore and coworkers described this chain of events with major torso trauma leading to active hemorrhage progressing to metabolic acidosis, core hypothermia, and progressive coagulopathy (Fig. 1) [2].

Subsequent to this early description of rapid termination of laparotomy, Rotondo and coworkers coined the term, "damage control laparotomy" [3]. They reinforced the important concepts explored initially by Stone and demonstrated improved outcomes in the face of acidosis, hypothermia, and coagulopathy. As time has passed, all trauma centers have adopted these principles of management, and surgical techniques for acute and definitive management have been refined. Gauze packing of liver injuries and

E-mail address: tfabian@utmem.edu

doi:10.1016/j.suc.2006.09.011 surgical.theclinics.com

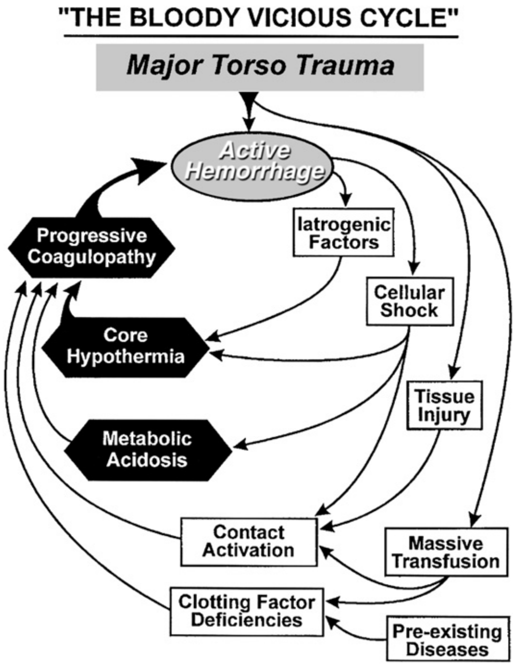

Fig. 1. Pathogenesis of the "blood vicious cycle" after severe injury is multifactorial, but progressive core hypothermia and persistent metabolic acidosis are pivotal. (*From* Moore EE. Staged laparotomy for the hypothermia, acidosis, and coagulopathy syndrome. Am J Surg 1996;172:405; with permission. © Copyright 1996 Excerpta Medica, Inc.)

pelvic injuries has become standard procedure for managing blunt and penetrating injuries. Like so many areas in medicine and surgery, techniques that have been abandoned from the past are continuing to be rediscovered, refined, and reapplied as knowledge develops combined with advances in surgical management and critical care progress.

As damage control concepts disseminated, the important entity of abdominal compartment syndrome (ACS) was recognized as a major cause of morbidity and mortality in patients who are critically injured. As management techniques have evolved, the fundamental issues of wound management have been central to the advance of damage control laparotomy. The intent of this article is to define the scenarios and pathophysiology of ACS further, consider the varied methods of management of the open abdominal wound, and address issues surrounding definitive abdominal wall closure and current techniques for reconstruction.

Compartment syndromes

The majority of cases in which the abdomen is left open after trauma laparotomy are those associated with ACS. Compartment syndromes can develop in nearly every part of the body. These include increased intracranial pressure from closed head injury, renal failure from shock-associated acute tubular necrosis, the more commonly recognized compartment syndromes of the lower extremity, and the recently recognized entity of ACS. Compartment syndromes of the lower extremity usually are associated primarily with ischemia from arterial occlusion. These include acute popliteal artery thrombosis or embolus, iliac artery occlusion, and ileofemoral acute arterial injury with shock requiring proximal clamping for vascular control. Critical warm ischemia times are highly variable in these various clinical scenarios depending on extent of collateral blood flow and the metabolic demands of the compartmental tissues. Perhaps the most important variable that has an impact on the rapidity and severity of any compartment syndrome is that of associated hemorrhagic shock combined with vascular occlusion.

Etiologies of compartment syndome

Although the anatomy of compartment syndromes is highly variable, the pathophysiology remains similar. Fig. 2 outlines some of the basic processes involved with development of compartment syndrome. Although ischemia always is involved, soft tissue injury frequently is an accompanying insult. Ischemia and soft tissue injury lead to edema and increasing tissue pressures, which cause decreasing tissue perfusion that results gradually in critical capillary closing pressures. Further cellular ischemia with edema ensues until this continuing sequence ultimately leads to tissue death. Although compartment syndromes can occur anywhere in the body, they are most prone to develop in anatomic compartments with low compliance. Conversely, they are less likely to occur when there is room for expansion. Because of the current status of global cataclysm, extremity compartment syndrome, secondary to crushing injuries, is the most common worldwide. And, although

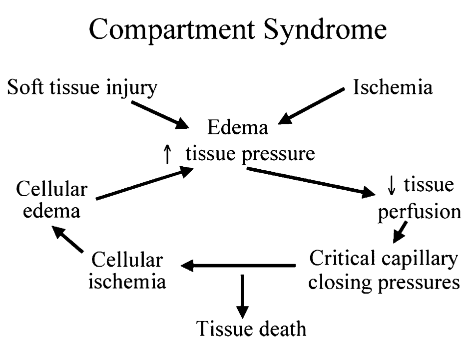

Fig. 2. The diagram illustrates the pathophysiologic processes involved with development and progression of compartment syndromes.

there is a wide variety of causes of compartment syndromes, including burn eschar, snakebites, iatrogenic sources (forearm intravenous infusion, pneumatic tourniquets, and Lloyd Davies positioning), for the purposes of this article, compartment syndromes caused by ischemia reperfusion (I/R) injury are discussed in detail.

Ischemia reperfusion injury

Endothelial damage incites the progressive cellular insult of I/R injury. In the tissue beds, the endothelial cells develop a proinflammatory phenotype and express cell surface adhesion molecules, which lead to neutrophil migration into the tissues producing cellular damage and swelling. Widened endothelial junctions simultaneously lead to increasing interstitial edema. This combination of interstitial edema and cellular swelling produces increased tissue pressures that ultimately end in a compartment syndrome. Reperfusion activates neutrophils and the production of free oxygen radicals, producing a simultaneous double-hit injury that causes damage and dysfunction of cellular membranes and further acceleration of intracellular and extracellular edema. The greatest damage occurs during this cascade of events after reperfusion. The end results of I/R are loss of membrane integrity with development of edema, leading to compromised blood flow and, ultimately, organ dysfunction and failure (Fig. 3). There have been many investigative avenues pursued to alleviate or prevent I/R injury. Although these endeavors have been successful in various animal models, there has been no significant advance relative to clinical applications.

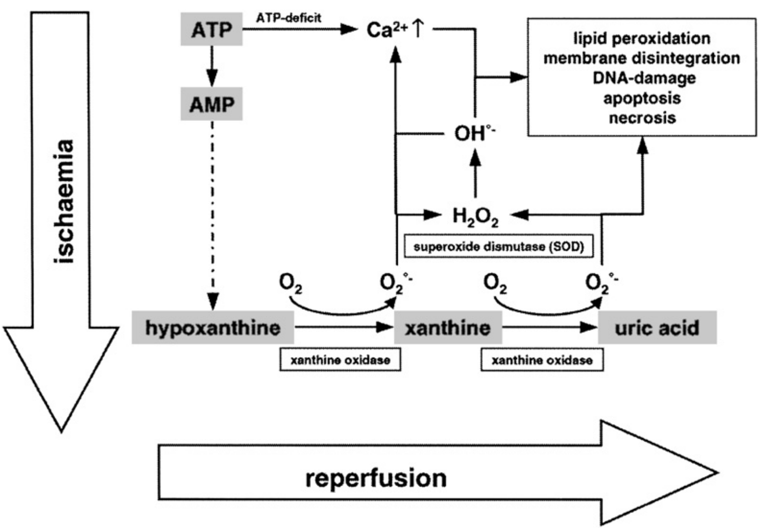

Fig. 3. Diagramatic illustration of I/R injury.

Abdominal compartment syndrome

The development of ACS can be looked on as the result of total body I/R injury. It generally begins as an insult resulting in either massive intracavitary or external hemorrhage. The hemorrhage component leads to hypoperfusion—the ischemic component of injury. In the face of prolonged shock acidosis, hypothermia and coagulopathy result. Splanchnic hypoperfusion with resultant gut mucosal acidosis, bowel edema, and hepatic ischemia follow. The gut edema is exacerbated markedly during the time of reperfusion associated with fluid resuscitation—events that visually are apparent in the operating room. The ischemia of muscle and gut leads to excessive systemic activation of the innate immune system, resulting in damage to essentially every organ, with the lung involved nearly uniformly by acute lung injury. As gut mucosal injury leads to bowel edema, there is a resultant increase in intra-abdominal hypertension, leading to the continued cycle of increasing ischemia, edema, and acidosis. The development of ACS is insidious and progressive. If not treated, ACS ends in the multiple organ dysfunction syndrome and, ultimately, death of the patient. The treatment of ACS is the same as treatment of most other compartment syndromes. The compartment must be decompressed.

Compartment syndromes result from intracompartmental hypertension. This hypertension produces dysfunction, which is inversely proportional to the compliance of the effected compartment. Compliance is represented by the formula, $C = \Delta V/\Delta P$ (C is compliance, Delta V is change in volume and Delta P is change in pressure).

Anatomic compartments with high compliance expand their volume (stretch) more readily with pressure increases than do low compliant systems. Fig. 4 illustrates this relationship conceptually. Increasing intracranial pressure from brain injury develops rapidly because of the inability to expand the cranium, whereas lower extremity compartments have a higher compliance because of limited capacity for fascial stretching to increase compartmental volume. In contrast, hypertension develops gradually in

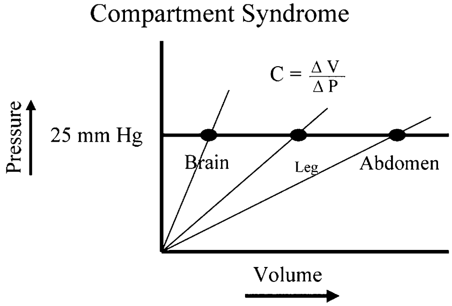

Fig. 4. The relationship of increasing volume and increasing pressure as they relate to compliance of three different body compartments.

the peritoneal cavity (abdominal compartment), as there is a greater cavitary volume compared with volume of contents. Although the figure is not meant to deal with exact relationships, it does call attention to the fact that the approximate pressure of 25 mm Hg is the critical compartment pressure for significant alterations in microvascular blood flow to the compartmental contents in essentially all parts of the body. At this pressure, therapies of either expansion of the compartment or reduction of volume (contents) have been used as the mainstay of therapy for many years. When compartment pressures in the lower extremity approach 25 mm Hg, fasciotomies are performed to expand the volume. For the brain, it is widely appreciated that at approximately a pressure of 25 mm Hg, significant alterations in flow occur, and, although diuresis has been a mainstay for many years in an attempt to decrease the intracranial swelling, frontal lobotomy also has been used to decrease the compartmental volume. In recent years, there has been a tendency toward craniectomy to increase the volume for expansion of the brain. This approach has developed simultaneously with concepts of decompressing the abdomen by leaving it open for the treatment of ACS.

Although urgent decompressive laparotomy has become the standard of care for treatment of ACS, understanding of these pathophysiologic processes has became appreciated more widely, and there has been a widespread movement toward a prophylactic approach to the management of ACS. Rather than closing the abdomen in the face of significant hemorrhage and abdominal injury, most trauma surgeons have begun leaving the abdomen open rather than risk further insult of intra-abdominal hypertension and ACS that often is lethal in this scenario.

The clinical picture of ACS is characterized by three physiologic perturbations: respiratory compromise, decreased splanchnic flow, and renal insufficiency. This pathophysiology has been delineated nicely in several laboratory experiments. In 1976, Richardson and Trinkle devised a series of experiments that were elegant in their simplicity [4]. They used an animal model of increased intra-abdominal pressure in which dogs had their peritoneal cavities insufflated with air in gradual increments. They noted that as the intra-abdominal pressure gradually went up as more air was instilled, the inspiratory pulmonary pressures rose in a gradual fashion until there was a marked increase when the intra-abdominal pressure approached 25 mm Hg. With the widespread application of laparoscopy for elective surgical procedures, 25 mm Hg is approximately the pressure at which cardiopulmonary compromise occurs. In addition, in this same model, there was a linear correlation of increasing vena caval and renal vein pressures with increased intra-abdominal pressure, and cardiac output had a linear decrease associated with increasing intra-abdominal pressures. A similar series of experiments was performed by Diebel and colleagues, in 1992 [5], in a swine model using instillation of lactated Ringer's solution to produce intra-abdominal hypertension. Systemic and splanchnic hemodynamics were investigated. It was noted that going from baseline up to 40 mm Hg in increments of 10 mm Hg that the mean arterial pressure did not change. This is similar to what is observed clinically in the scenario of ACS. Similarly, the pulmonary capillary wedge pressure did not change substantially. The cardiac output fell 30%, however, from 5.4 L per minute at baseline to 4.0 L per minute at 40 mm Hg. Also, the superior mesenteric artery flow dropped to 30% of baseline at 40 mm Hg, and, similar to the previous experiments, the drop occurred most abruptly when the intra-abdominal pressure was raised above 30 mm Hg. Using tonometry, the mucosal pH dropped from 7.21 at baseline to 6.98 at 40 mm Hg intra-abdominal pressure. These laboratory observations correlate well with what is seen in patients in operating rooms and ICUs who develop intra-abdominal hypertension and ACS.

Although ACS generally is believed to be associated with trauma, one of the first clinical descriptions of the syndrome clinically was made as a complication of ruptured abdominal aortic aneurysm repair [6]. That report documented the occurrence of ACS with ruptured aneurysms, and a subsequent report by Oelschlager and coworkers demonstrated that delaying primary closure of the abdomen resulted in improved survival [7]. Subsequently, Ivatury and colleagues documented significantly improved survival in a group of severely injured trauma patients who had mesh closure as a primary prophylactic measure versus a group of patients who had fascial closure [8]. The mortality in the prophylactic mesh group was 10.6% versus 36% in the group with the fascial closure ($P = .003$). That report and the clinical observations of surgeons in other trauma centers led to the current widespread practice of prevention of ACS by leaving the abdomen opened.

The early descriptions of ACS were from patients who had abdominal injury associated with major blood loss and shock. ACS, however, has been noted in patients who have no significant abdominal injury but require massive fluid resuscitation for extra-abdominal injuries. ACS has been seen in severe soft tissue and skeletal trauma and after burn resuscitation. Maxwell and colleagues reported seven patients who had ACS occurring after massive fluid resuscitation from extra-abdominal injuries and coined the term, "secondary ACS" [9]. It was found that emergent decompressive laparotomy significantly reduced peak inspiratory pressures, immediately yielded significant diuresis, and was associated with rapid improvements in base deficit. An important part of therapy is to administer sodium bicarbonate immediately before decompression in those patients who develop ACS. At my institution in Memphis, there were deaths associated with decompression, which was the result of a massive return of anaerobic by-products from the abdomen and lower extremities when the abdomen was opened. The patients who died became acutely hypotensive. Since institution of prophylactic sodium bicarbonate immediately before opening the abdomen, this adverse event has been avoided.

Open abdomen techniques

Although ACS has been reduced significantly since prophylactic open abdomen management has been adopted, a new set of problems has arisen, resulting in several controversial areas of management. Novel challenges, acute and chronic, have emerged. The acute problems involve managing the large open wounds and potential collateral damage, including the disastrous complication of intestinal fistulae. The chronic problem created by the successful avoidance of ACS is the challenge of definitive reconstruction of the large abdominal wall defects in those patients who recover from their acute traumatic insult. To address these difficult tasks, institutions have arrived at local approaches that are developed by trial and error techniques. Although art and science always are balanced in surgery, it seems that the approach to managing large abdominal wounds after these major traumatic events has developed with more art than science. In Memphis, this is an area where my colleagues and I certainly have learned a lot more from mistakes than from successes.

At my institution, a fairly standardized staged approach for managing the abdominal wound has been developed. The three stages are stage I—prosthetic insertion; stage —split-thickness skin grafting for a "planned ventral hernia"; and stage III—definitive reconstruction (Fig. 5). It seems that most trauma institutions have adopted similar strategies. In the remaining portion of this article, staged reconstruction methods are discussed, including alternatives to the various elements of each stage and analysis of complications associated with acute and definitive management plans.

The staged approach to the open abdomen was evaluated in 274 consecutive patients [10]. During the 8-year interval of this study, there were 2664 laparotomies performed for trauma. Thus, approximately 10% of all laparotomies were managed with open abdomen techniques because of the severity of abdominal injury, blood loss, and shock. All of these wounds

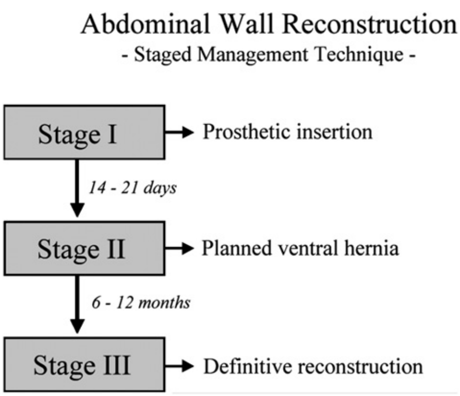

Fig. 5. This is an algorithm demonstrating the three-staged management of the open abdomen.

were incisions from xyphoid to pubic symphysis. Of the 274 open abdomen patients, there were 108 deaths (39%) within the first several days of injury resulting from complications associated with massive injury and shock.

Stage I

There are many methods described for acute management of the open abdominal defect. The technique of towel clip closure of the abdomen was used widely in the early days of damage control via rapid termination of laparotomy before definitive surgical procedures and to permit resuscitation of the clotting system, shock, and hypothermia in ICUs. Initially, surgeons used towel clipping of the fascia, whereas subsequently the technique was modified to clipping of the skin with multiple clips approximately 1 to 2 cm apart. The towel clip method was effective for rapid termination of surgery with tamponade maintained in the gauze-packed abdomen. Several patients developed ACS, however, regardless of whether or not the fascia or skin was clipped, because of the inability of the abdominal cavity to expand with increasing edema or blood and clot that developed. Thus, towel clip closure largely has been abandoned with the onset of the widespread practice of open abdominal wound management for damage control and prophylaxis against ACS.

Varying techniques have been promulgated for acute management of the open abdominal wound. Most surgeons have believed that it is important to place some type of prosthesis in the widely open abdomen to prevent evisceration and associated complications from bowels unprotected from gauze dressings. The Bogota bag was one of the early devices used and remains a practical tool. This approach is attributed to surgeons in Colombia who have had vast experience in the management of catastrophic penetrating abdominal wounds for many years. The technique uses sterile polyvinyl chloride solution bags that can be sewn to skin or fascia. Large genitourinary irrigation bags work well. X-ray cassette covers are used in a similar fashion and seem equally effective. Advantages of both are that they are widely available and inexpensive. Other materials used for acute management of the open wound include polypropylene mesh, polytetrafluoroethylene (PTFE) patches, the Wittmann Patch (Star Surgical), and absorbable meshes, including polyglycolic acid and polyglactin 910 meshes. Under ideal circumstances, omentum is inserted between the intestines and the prosthesis, but often the omentum either is inadequate or destroyed to such an extent that it is not available for coverage. I insertion of omentum in this space decreases the complication of intestinal fistula.

As part of the first stage of management, an attempt is made to pleat the prosthetic material gradually, as patients' hemodynamic status recovers to maintain midline tension to attain a potential secondary fascial closure. It has been my colleagues and my institutional policy to take patients back to the operating room within 24 to 48 hours of the initial operation for definitive repair of viscera and removal of packs. Polyglactin 910 woven mesh is inserted at that time, if it had not been placed at the primary operation. I find that attempts at pleating polyvinyl chloride have a tendency to tear the material, whereas the absorbable mesh maintains its integrity from pleating without having to replace the material at the fascia or skin edges where it has been attached. If the pleating process is begun but the patient deteriorates because of sepsis, with further swelling and abdominal distention, then occasionally the mesh is left in place, but additional mesh is sutured to expand the pleated area.

There is some disagreement as to whether or not prosthetic material should be sutured to the skin or to the fascia. The argument in favor of attaching it to the skin is that it reduces the incidence of fascial loss, which can be an important issue during definitive reconstruction. The argument for sewing it to the fascia is that there is a reduced tendency to lose abdominal domain by preventing the continuing retraction of the fascia. My general policy is to sew the prosthetic to the skin initially. If it becomes apparent that the abdomen is not be able to be closed, however, a fascial attachment is converted to, to minimize the loss of domain over the several months that pass before definitive reconstruction (Fig. 6). Using the staged approach with gradual pleating of the polyglycolic acid mesh has permitted 22% of 166 patients in my colleagues' and my experience to undergo a secondary fascial closure. Pleating also can be used with other materials that are used for acute abdominal wound management. The Wittmann Patch was designed specifically to allow gradual pleating in ICUs. The patch consists of two pieces of burr-type material that stick together like Velcro. A piece is sewn to each side of the abdomen, which allows for either expanding the abdominal cavity in the face of increasing intra-abdominal edema or for pleating to allow for gradual wound closure.

Polypropylene mesh was used extensively in the past but has fallen out of popularity because of the concern over development of enterocutaneous

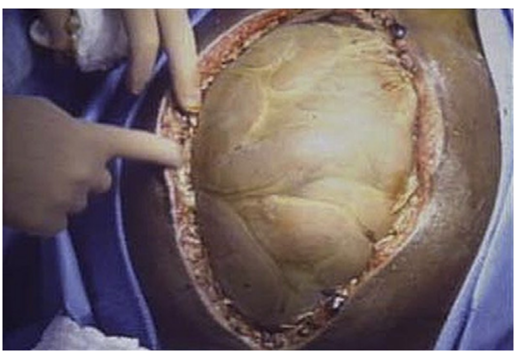

Fig. 6. The picture demonstrates placement of woven polyglactin 910 mesh for open abdomen management in stage 1.

fistulae. From a summary of 14 reports in the literature involving 128 patients, Jones and Jurkovich reported a 23% fistula rate [11]. These enterocutaneous, or perhaps more appropriately termed "enteroatmospheric" fistulas, are difficult to manage and associated with substantial morbidity and mortality. Although comparative data are not available on other materials, it is widely believed that the fistula rate is higher for polypropylene mesh than all the other techniques discussed.

An alternative technique to bridging a wound with prosthetic material is the use of the vacuum pack technique. This technique was described by Brock and coworkers in 1995 (Fig. 7) [12]. A plastic drape was inserted in the peritoneal cavity covering the viscera and on top of this was placed a surgical towel along with two sump drains covered with an adhesive-backed plastic drape attached to the skin. The sump drains are placed on continuous suction. This provided the theoretic advantages of evacuating the abdominal cavity of edema fluid, allowing for maintenance of tamponade to counteract bleeding and possible prevention of loss of domain. As opposed to my experience, in which 22% of abdomens could be closed progressively, Miller and colleagues reported being able to secondarily close approximately 80% of abdomens that were managed by a similar vacuum pack

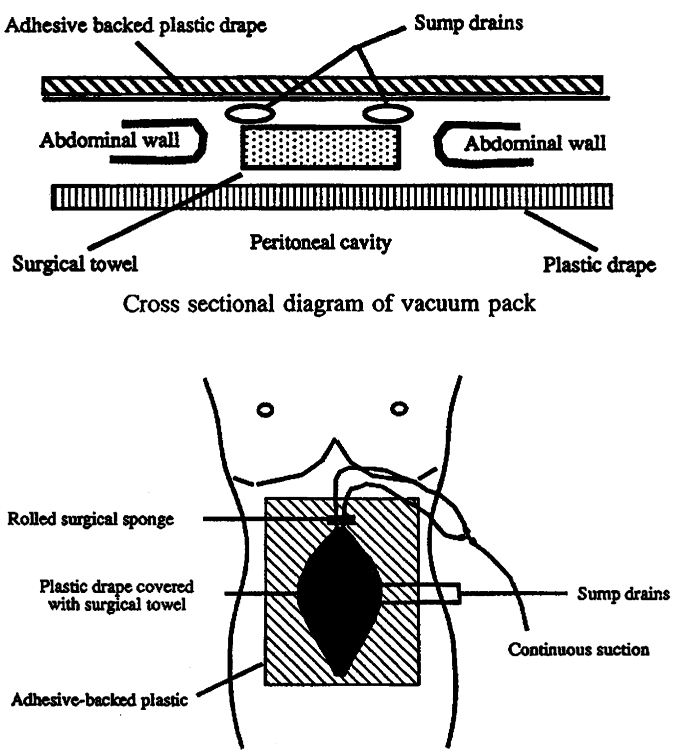

Fig. 7. Cross-section diagram of vacuum pack and completed vacuum pack.

technique [13]. It remains unclear, however, if the indications for managing patients who had the open abdomen are uniform among reported series. Institutions that are more liberal about leaving abdomens open after laparotomy for trauma likely have a much higher rate of closure than institutions that have less experience. Other factors could be reflected in referral patterns where institutions have a large number of patients who come at more prolonged intervals. Other patients may experience a greater level of shock after their injury and may have greater degrees of cellular insult prohibiting early closure resulting from prolonged time to resolution of the I/R injury. Regardless, it is important for investigators to report the total number of laparotomies in their series in addition to those managed by open abdomen so that a denominator is available to permit reasonable comparisons among institutional outcomes.

Since the early descriptions of the vacuum pack technique, commercially produced products have been developed for managing these open abdominal wounds (Kinetic Concepts, Inc.). There also is concern that the vacuum technique might promote dehiscence of intestinal suture lines in those patients who undergo intestinal repair or resection. To date, however, that complication has not been documented in the literature in association with this vacuum technique. Clinical trials would be valuable and are indicated in addressing these various techniques and products. Standard entry criteria and prospective stratification and evaluation would allow for addition of a little science to the art of open abdominal wound management.

Stage II—planned ventral hernia

When prosthetic material is inserted in stage I, patients either recover and mobilize fluids over the next several days, allowing for gradual closure of the abdomen, or develop varying degrees of multiple organ dysfunction or sepsis. In my experience, in the latter group, they rarely are able to get their fascia closed via progressive approximation. In that circumstance, the viscera granulate and adhere to the abdominal wall laterally and to the prosthesis. When absorbable mesh is used as the acute prosthetic material, granulation occurs through the mesh. Although conventional wisdom suggests that absorbable mesh would be absorbed completely and not have to be removed, that has not been my experience using the woven variety of polyglactin 910. Usually within 2 to 3 weeks, granulation occurs and a suppurative interface between mesh and viscera develops, which begins to separate the two. At this point of healthy granulation, the viscera are stuck laterally and patients are taken to an operating room. The mesh is removed easily because of the suppurative interface (Fig. 8). After removal of the mesh, a pulsatile pressure irrigation system is used to reduce colonization of the granulation tissue and a split-thickness skin graft is harvested and applied to "close" the abdominal wound.

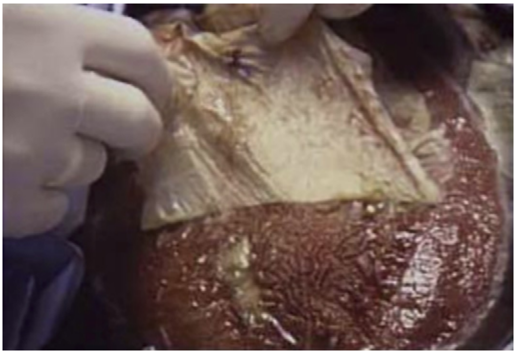

Fig. 8. The photograph demonstrates the removal of woven polyglactin 910 at stage 2 followed by placement of split-thickness skin graft for planned ventral hernia.

Intestinal fistulae

From my experience, excluding those cases of early mortality resulting from catastrophic injury, 14 fistulae developed in the 166 survivors for a fistula rate of 8.4% [10]. A root cause analysis of the fistulas was done. Ten were of small bowel origin, three were from the colon, and one was gastric. The timing of fistula development was one before insertion of the vicryl mesh resulting from suture line breakdown of a destructive small bowel injury, 10 after mesh insertion but before skin grafting, and the remaining three after split-thickness skin grafting. There were important findings that can be used to reduce fistula development in management of these large open wounds. It is important to get the skin graft on the open wound as soon as the prosthetic material is removed. When I evaluated the days of mesh insertion on patients who had and did not have fistula, those who did not have fistula had the mesh on for 18.1 days versus 26.5 for those who had fistula ($P < .04$); the longer the wound is left to granulate without coverage of the bowel, the more likely erosion of the bowel wall through the granulation tissue is to develop and instigate a fistula. One would not think that a skin graft necessarily would give much support to the wound and that this sort of coverage would not necessarily prevent a fistula. But, in my experience, it seems that skin grafting does stabilize the wound significantly. Besides bowel erosion from prolonged exposure of the uncovered, open granulating bed, I have had a few patients develop fistulae from coughing during suctioning or while on a ventilator. The presence of a skin graft seems to add a degree of integrity and reduces splitting of the bowel during sporadic increases in intraintestinal pressure. Closure of open granulating wounds also is important to decrease the proteinaceous losses through the wound and is similar to the benefit of early grafting of large surface burns. These prodigious open wounds act as major third-degree burns to the abdominal wall and produce a tremendous ongoing catabolic drain.

Regardless of how attentively the wound management care is provided, occasional fistulae develop. These can range from relatively minor irritants to life-threatening catastrophes. Sometimes, small fistulas develop on the lateral aspects of the wound. In that circumstance, wounds often granulate and contract over the top of these lateral fistulas with resolution of the fistula. Those small fistulas that do not close often can be handled as semiformal ostomies with bags placed over them until definitive reconstruction.

Those fistulas that occur away from the lateral wall in the midportion of the wound are problematic, however. For fistulae in the midst of the wound from distal small bowel, my colleagues' and my institutional approach is to constipate the patient (diphenoxylate/atropine) and to skin graft the entire wound around the fistula. Bed rotation and patient positioning are helpful adjuncts. After the graft adheres, the fistula is converted to a controlled ostomy. Although not always successful, this approach works better than might be anticipated. In contrast, midwound proximal, high-volume small bowel fistulas have significant morbidity and mortality and are the most challenging. My usual approach is to operate early to prevent the complications secondary to metabolic derangements and nutritional depletion. High-volume fistulas cannot be controlled as ostomies in these wounds. I prefer to operate within the first 7 to 10 days after they develop. Operation consists of incising the granulation tissue right at the osteum of the fistula with sharp and hemostat dissection. Generally, a relatively normal serosa is identified. Once the serosa is identified, the bowel loop is isolated gradually and resected back to nonindurated, nonedematous tissue. A primary anastomosis generally is performed. After anastomosis, an attempt is made to bury the anastomosis as deeply as possible in the abdomen beneath other bowel loops to minimize the likelihood of recurrent fistula. If recurrence develops, some fistulae may close if buried within loops of bowel, because the dehiscence is not exposed in the open wound. After resection, the large open wound is managed either with gauze abdominal dressings if the majority of the bowel is stuck, or if not, absorbable mesh once again is sewn to the skin edges laterally to maintain the viscera within the abdomen. Resection of these fistulas is an arduous task that takes many hours. It is my opinion, however, that patients do better with this early approach rather than waiting several weeks to months and having their nutritional status and overall recovery continue to erode while they are on total parenteral nutrition.

Stage III—definitive reconstruction of the abdominal wall

These giant abdominal wall defects can appear daunting (Fig. 9). An organized consistent approach, however, can provide excellent long-term outcomes. The appropriate timing for abdominal wall reconstruction is of paramount importance. If performed too early, dense adhesions make split-thickness skin graft removal difficult, often leading to multiple

Fig. 9. Photograph of a patient 11 months after hospital discharge who is ready for abdominal wall reconstruction.

enterotomies or significant bowel deserosalization. When reconstruction is delayed for an excessive amount of time, the musculature of the abdominal wall tends to contract with a progressive loss of domain. In that circumstance, when autogenous tissue reconstruction is planned, mobilization of the components of the abdominal wall is difficult, leading to repairs under tension and an increased incidence of hernia formation over time.

Many techniques are advocated for repair of these defects. The most commonly applied approaches are with the use of prosthetic materials. Other options include the application of local myofascial advancement techniques, rotational muscle flaps, and, most recently, the use of biomaterials. There are advantages and disadvantages to each of these approaches. The most obvious advantages to the use of prosthetic materials, most commonly polypropylene mesh and PTFE, are their ready availability and the fairly simple techniques of implantation. Currently, long-term comparative outcomes are not available in this population of patients who have giant defects. The approach to repair with prosthetic materials is the same as for any ventral hernia. Mobilization of skin and subcutaneous fat is required beyond the myofascial edges bilaterally. Although there is a variety of suture methods available for attaching the prosthesis, it seems that the fascia underlay technique with attachment of the prosthetic with U-sutures placed 1 to 2 cm beyond the myofascial edge is associated with better long-term results, particularly regarding recurrent buttonhole-type hernias. The disadvantages to prosthetic materials are the risks of intestinal fistula, prosthetic infection, and recurrent hernias. Whatever material is used, it is advantageous to attempt to place an interface of omentum between the mesh and the underlying bowel. In these complex cases, however, that have had multiple abdominal injuries, the omentum generally is not of good quality or quantity. The use of PTFE, including the Dualmesh variety (W.L. Gore & Associates, Flagstaff, Arizona), is proposed to be associated with lower fistula rates. A major trade-off seems to be a higher incidence of seroma developing between the prosthesis and the large skin flaps that are required for these procedures. Overall, it is difficult to provide accurate data concerning morbidity and long-term outcomes with the various reconstructive techniques because of paucity of reported data.

Although construction of muscle flaps occasionally is reported in small series, there is no large experience with either free flaps or rotational flaps in the management of these abdominal wall defects. They have the advantage of being autogenous tissue. Disadvantages include the complexity of the techniques and complications associated with those cases in which the flap fails. Larger experiences hopefully will be reported in the near future.

The component separation technique was described by Ramirez and colleagues in 1990 [14]. A modification of that original description has become my preferred method of reconstruction in the majority of these cases. The original component separation technique was a description of local myocutaneous flap advancement after extensive relaxing incisions in the abdominal wall. This involved bilateral medial mobilization of the musculofascial units of the rectus abdominus muscles. It provides autologous continuity with dynamic support of the abdominal wall. The original technique provided for 3 to 5 cm of mobilization on each side. These large abdominal wall defects, however, usually cannot be repaired with this amount of mobilization. Therefore, prosthetic material generally is required to close the defect in a tension-free manner. After recognizing this drawback of limited mobilization, my colleagues and I added a modification of Ramirez's component separation technique [15].

Modified component separation

Fig. 10 demonstrates the application of this reconstructive approach. Although it seems complicated in the diagram, it actually is straightforward.

Step 1: Begin the operation by removing the split-thickness skin graft from the underlying abdominal viscera. As discussed previously, the timing for abdominal reconstruction is extremely important to ensure optimal results. My colleagues' and my plan for reconstruction when the intra-abdominal adhesion process has matured to the point of development of filmy adhesions between loops of bowel and skin graft. This can be ascertained simply by pinching the skin graft between the fingers, and, if the bowel falls away, the time for reconstruction is at hand. If the loops of bowel remain densely adhered to the underlying skin graft, reconstruction should be delayed. Experience suggests that most patients develop these flimsy adhesions in the 6- to 12-month interval after hospital discharge. Occasionally, patients resolve the dense inflammatory responses a little sooner and occasionally somewhat later, but the dense inflammatory process usually resolves at approximately 8 to 9 months. In an operating room, after wide preparation of the abdominal wall, the skin graft is grasped between the

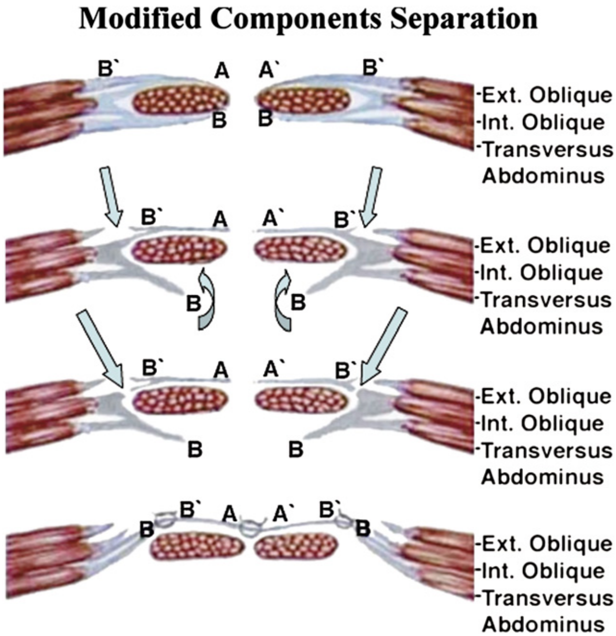

Fig. 10. Diagrammatic illustration of the modified components separation technique for reconstruction of abdominal wall defects.

fingers or with tissue forceps and sharply incised in an area that is obviously devoid of bowel. It is common for small areas of bowel to continue to have dense adhesions, so these areas are to be avoided when entering the abdomen. Once the incision is made, the flimsy adhesions are apparent and, with sharp and hemostat dissection, the skin graft is divided in its midportion from top to bottom. After this, adhesions of small bowel to the skin graft are mobilized bilaterally and dissection extends a few centimeters beyond the lateral edge of the rectus abdominus muscles bilaterally. Removal of the skin graft and satisfactory lyses of intra-abdominal adhesions generally take 45 to 60 minutes. The more dense adhesions generally are found at the medial myofascial edges bilaterally. Adhesions to the liver also generally are a little more dense then the flimsy adhesions to the loops of the bowel and caution must be exercised to avoid dissecting beneath Glisson's capsule and causing bothersome oozing of blood.

Step 2: After this mobilization, the full-thickness skin and subcutaneous fat are mobilized on each side of the wound to between the anterior and midaxillary lines, depending on the degree of abdominal wall contraction in order for lax skin closure.

Before completing description of the procedure, it is appropriate to review the anatomy of the rectus abdominus sheath. There are anterior and posterior rectus sheathes bilaterally. These are formed by the medial extensions of the fascia of the external oblique, internal oblique, and transversus abdominus muscles. The internal oblique aponeurosis splits just lateral to the rectus abdominus muscle and contributes to the anterior and posterior rectus sheaths. The external oblique fascial aponeurosis fuses with the anterior component of the internal oblique fascia, forming the anterior rectus sheath. In a similar fashion, the transverse abdominus aponeurosis and transversalis fascia fuse with the posterior element of the internal oblique aponeurosis just lateral to the lateral portion of the rectus abdominus, with this fusion producing the posterior rectus sheath. There is no posterior rectus sheath below the arcuate line. The following steps are for mobilization of the rectus abdominus myofascial flaps.

Step 3: The rectus abdominus muscle is grasped in the palm of the hand with the thumb on top and the fingers inside of the abdominal cavity. By rubbing the thumb and fingers back and forth, it is easy to identify the lateral border of the rectus abdominus muscle. The external oblique aponeurosis is divided approximately 1 cm lateral to the rectus abdominus muscle with use of hemostat to dissect the external oblique freely from the internal oblique component. The external oblique fascia then is divided superiorly and inferiorly, going over the lower costal region superiorly and down to the pubic symphysis inferiorly. In the diagram, that division is indicated by the line of incision at B′ bilaterally.

Step 4: After division of the external oblique aponeurosis, the posterior rectus fascia is separated from the rectus muscle with blunt dissection beginning at the superior aspect of the wound and continuing down below the arcuate line. In the diagram, this is indicated by the separation bilaterally at B, which indicates the medial extent of the posterior rectus fascia.

Step 5: The internal oblique component of the anterior rectus sheath is divided. This is done in a fashion similar to the division of the external oblique aponeurosis by initial sharp incision and then using hemostat dissection and cautery or knife to divide this fascia up over the lower rib cage superiorly. Inferiorly, it is imperative that the incision be stopped at the arcuate line. This can be seen from the peritoneal surface of the rectus sheath as a superiorly convex line approximately midway between umbilicus and pubic symphysis. This is the site of entry of the inferior epigastric artery into the rectus sheath and the site of spigelian hernia formation. If the internal oblique component of the anterior rectus sheath is divided below this point, a large hernia will develop, because there is no posterior fascia below the arcuate line. After these divisions of the internal and external oblique aponeuroses and mobilization of the posterior rectus sheath from the rectus muscles, mobile flaps of the rectus myofascial units have resulted bilaterally.

Step 6: The defect then is closed by suturing the medial components of the anterior rectus fascia (A to A′) (see Fig. 10), followed by closure of the medial aspect of the posterior rectus sheath to the lateral aspect of the anterior rectus sheath bilaterally (B to B′) (see Fig. 10). The suture technique used for closing these midline and lateral components of the abdominal wall is with three separate running #1 polypropylene sutures medially and bilaterally. Four flat closed suction drains then are inserted, two superiorly and two inferiorly bilaterally. The skin then is approximated in the midline to complete the procedure. (An audiovisual CD presentation of the modified components separation technique is available in the video collections of the American College of Surgeons [15a].)

This mobilization provides approximately 10 cm of medial advancement in the epigastrium, 20 cm in the midabdomen, and 8 cm in the lower abdomen. Generally, the most difficult mobilization of the myofascial units is in the superior aspect of the wound, which makes it important to carry the oblique incisions over the lower costal margin. Occasionally, a small piece of prosthetic material is required in this upper part of the incision to eliminate tension. Approximately 100 abdominal wall reconstructions have been performed at my institution using the modified component separation technique. The average follow-up of these patients is approximately 2.5 years. Long-term results have demonstrated recurrent hernias in 5% of these patients. The hernias usually are localized around one of the lateral suture lines. Results were analyzed for complications of recurrent herniation and the need for adjunctive mesh in the upper part of the wound versus timing of reconstruction. If reconstruction was performed less than 12 months after discharge, the complication rate was 7.6%, although of those who had reconstruction longer than a year after injury, complications occurred in 25% ($P = .10$). Adjunctive mesh was required in approximately 10% of my patients. The average time to reconstruction in those requiring adjunctive mesh was 20 months from the time of discharge. I believe that these are clinically relevant findings; they reinforce the concept that if reconstruction is undertaken much beyond the time of the development of loose intra-abdominal adhesions that abdominal wall contraction progresses, resulting in loss of domain of the abdominal cavity; such loss leads to repairs under increased amounts of tension.

This experience with modified component separation technique remains the largest reported for management of large abdominal wall defects associated with damage control surgery. The complication rates have been low compared with experiences with repair of large ventral hernias in general. I have had no intestinal fistulas develop when using this approach. The hernia recurrence rate of 5% is low considering the size of these hernias. This technique remains my procedure of choice for definitive reconstruction in those patients who could not be closed during their initial hospitalization. In patients who require resection of significant amounts of rectus abdominus muscle resulting from necrosis associated with their original injury, however, the modified component separation technique is not possible. Patients usually should undergo abdominal wall reconstruction at less than 1 year to avoid the loss of domain that results in repairs under tension leading to higher recurrent hernia rates. In addition, the placement of permanent mesh is avoided with the attendant morbidities of foreign body infection and occasional production of intestinal fistula.

Biomaterials for abdominal wall reconstruction

Recently there have been reports of the use of human cadaveric acellular dermis for reconstruction (Alloderm, LifeCell Corporation Branchburg, NewJersey). Guy and colleagues report using this material acutely in a one-staged approach combined with local skin flaps advanced over the acellular dermis to avoid the need for extensive reconstruction [16]. Early results in that small series are promising. This material recently has been used in my institution in wounds that are not amenable to modified component separation reconstruction technique; the use of acellular dermis has provided good early results. Long-term follow-up, however, is required with larger numbers of patients to evaluate that approach adequately. It has been found that this material acts basically as a collagen matrix and allows for tissue ingrowth. In a few cases in which the material has been excised several months after implant, it appears histologically similar to the native tissue that it replaced.

Summary

Damage control surgery has become a fundamental component of operative trauma care. It undoubtedly has saved many lives that would have been lost in the not-too-distant past. In the development of these techniques, a great deal has been learned about intra-abdominal hypertension and ACS. Prophylactic application of open abdomen techniques evolved and led to avoidance of a great deal of the organ dysfunction associated with ACS. Additionally, in years past, many wounds were closed under considerable tension in the face of contamination that resulted in a high rate of necrotizing fasciitis, intestinal fistulization, and significant mortalities attributed to those dire complications. Surgeons now are learning a great deal about management of large open abdominal wounds. A wide variety of techniques has been adopted, but as time passes, there seems to be a general consensus developing regarding acute management of these wounds. Most institutions are adopting staged techniques of management similar to that described. It is recognized that getting the open wound closed as soon as possible leads to fewer complications, including lower fistula rates. The acute use of vacuum wound dressings is promising in that it may provide for early secondary closure of a significant portion these patients. Although we are continuing to learn about acute management, there has been less study focused on optimal definitive reconstructive techniques. The modified component separation technique has provided good results, with low recurrent hernia rates and long-term functional abdominal wall dynamics. Recently, the use of biomaterials for acute and chronic reconstruction shows promise in this area.

Further focus and study in all of these areas ultimately will lead to improved outcomes in this most seriously injured cohort of trauma patients.

References

[1] Stone HH, Strom PR, Mullins RJ. Management of the major coagulopathy with onset during laparotomy. Ann Surg 1983;197:532–5.
[2] Moore EE. Staged laparotomy for the hypothermia, acidosis, and coagulopathy syndrome. Am J Surg 1996;172:405.
[3] Rotondo MF, Schwab CW, McGonigal MD, et al. 'Damage control': an approach for improved survival in exsanguinating penetrating abdominal injury. J Trauma 1993;35:375–83.
[4] Richardson JD, Trinkle JK. Hemodynamic and respiratory alterations with increased intra-abdominal pressure. J Surg Res 1976;20:401–4.
[5] Diebel LN, Dulchavsky SA, Wilson RF. Effect of increased intra-abdominal pressure on mesenteric arterial and intestinal mucosal blood flow. J Trauma 1992;33:45–9.
[6] Fietsam R Jr, Villalba M, Glover JL, et al. Intra-abdominal compartment syndrome as a complication of ruptured abdominal aortic aneurysm repair. Am Surg 1989;55:396–402.
[7] Oelschlager BK, Boyle EM Jr, Johansen K, et al. Delayed abdominal closure in the management of ruptured abdominal aortic aneurysms. Am J Surg 1997;173:411–5.
[8] Ivatury RR, Porter JM, Simon RJ, et al. Intra-abdominal hypertension after life-threatening penetrating abdominal trauma: prophylaxis, incidence, and clinical relevance to gastric mucosal pH and abdominal compartment syndrome. J Trauma 1998;44:1016–23.
[9] Maxwell RA, Fabian TC, Croce MA, et al. Secondary abdominal compartment syndrome: an underappreciated manifestation of severe hemorrhagic shock. J Trauma 1999;47:995–9.
[10] Jernigan TW, Fabian TC, Croce MA, et al. Staged management of giant abdominal wall defects: acute and long-term results. Ann Surg 2003;238349–57.
[11] Jones JW, Jurkovich GJ. Polypropylene mesh closure of infected abdominal wounds. Am Surg 1989;55:73–6.
[12] Brock WB, Barker DE, Burns RP. Temporary closure of open abdominal wounds: the vacuum pack. Am Surg 1995;61:30–5.
[13] Miller PR, Meredith JW, Johnson JC, et al. Prospective evaluation of vacuum-assisted fascial closure after open abdomen: planned ventral hernia rate is substantially reduced. Ann Surg 204;239:608–16.
[14] Ramirez OM, Ruas E, Dellon AL. "Components separation" method for closure of abdominal-wall defects: an anatomic and clinical study. Plast Reconstr Surg 1990;86:519–26.
[15] Fabian TC, Croce MA, Pritchard FE, et al. Planned ventral hernia. Staged management for acute abdominal wall defects. Ann Surg 1994;219:643–53.
[15a] American College of Surgeons. Modified components separation technique. Available at: http://www.facs.org/education/videolibrary.html.
[16] Guy JS, Miller R, Morris JA Jr, et al. Early one-stage closure in patients with abdominal compartment sybndrome: fascial replacement with human acellular dermis and bipedicle flaps. Am Surg 2003;69:1025–9.