
What's New in Trauma in the Past 10 Years

Thomas Scalea, MD

The management of patients following injury has undergone a substantial evolution over the past 10 years. Our understanding of the physiology of injury and blood loss continues to be refined. This has allowed us to dispel several myths that were long-held dogmas in the area of resuscitation. Improved technology has provided us with more discriminating diagnostic modalities, allowing us to streamline the evaluation process and making important diagnoses earlier. Noninvasive or minimally invasive technology has also evolved, bringing exciting new methods of hemostasis. In addition, we are beginning to understand the physiologic and economic consequences of nontherapeutic exploratory surgery and have become much more selective in our management. As our understanding has increased, so has our ability to cull out special patients, such as older patients, and tailor management strategies specifically for them.

Injury management continues to develop quickly and profoundly. Perhaps the most important lesson we have learned over the last 10 years is that we must expand our horizons and reinvestigate those unproven beliefs that have governed many of our treatment decisions. In this chapter, we will describe some of the most important advances made in the field of trauma management over the last 10 years.

■ Hypotensive Resuscitation

During the last 50 years, resuscitation strategies have been based on the principle of early and complete restoration of forward flow. Crystalloid volume has always been administered first, with blood used when crystalloid is insufficient to immediately restore peripheral oxygen delivery. This practice is based on our understanding that it is important to limit the duration of shock following injury. Thus, patients were held in the emergency department in an attempt to completely resuscitate them, even in the face of ongoing hemorrhage.

An abundance of animal data suggests that this resuscitation strategy is, in fact, ill advised. In numerous animal models of uncontrolled hemorrhage, normalizing blood pressure before obtaining hemostasis has been shown to increase blood loss and produce recurrent hypotension.¹⁻³ Ongoing crystalloid resuscitation hemodilutes available red cells and clotting factors, leading to acute anemia and coagulopathy. The mechanism is thought to be one of simple mechanics. Hypotension slows the flow within an injured blood vessel, allowing for spontaneous hemostasis via formation of a platelet plug. The restoration of normal blood pressure risks dislodging this hemostatic clot. This then produces the cycle of hemorrhagic shock followed by resuscitation and recurrent hemorrhagic shock.

In 1994 investigators at Ben Taub Hospital in Houston published data from a randomized, prospective study suggesting that limiting fluid before attaining surgical hemostasis provided a survival advantage.³ In this trial, patients with penetrating torso trauma and hypotension were randomized in the field to a resuscitation blood pressure of 100 mm Hg versus placement of intravenous catheters and no fluid regardless of hemodynamic status. This strategy was continued in the emergency department until anesthesia induction. In this study of nearly 700 patients, there was a statistically significant survival advantage to fluid limitations.

This all-or-none strategy seems counterintuitive. It is difficult to imagine that a patient with a barely palpable blood pressure would benefit from withholding resuscitation. In addition, this study examined a very small subset of American trauma victims, those with penetrating torso trauma. The available animal data suggested that moderate hypotension may be the best goal. In addition, it would seem reasonable to continue this strategy up to the time of hemostasis, not just anesthesia induction.

We hypothesized that a mean arterial pressure of 50 mm Hg or systolic blood pressure between 70 and 80 mm Hg would be the ideal initial resuscitation goal. We then randomized all patients with hemorrhagic shock to a mean arterial pressure of 50 versus 80 mm Hg (Dutton R, personal communication). Patients were randomized at hospital admission, and the resuscitation strategy was continued until the attending surgeon deemed hemostasis to be complete. In this prospective trial, we again demonstrated no survival advantage to aggressive fluid administration.

Thus, it would seem that traditional resuscitation goals of normal blood pressure and pulse rate are, in fact, antiquated. Resuscitation strategies should be tailored to limit profound shock and provide some degree of peripheral oxygen delivery. Most importantly, the evaluation process should be truncated to the shortest possible duration in patients with active hemorrhage. Once bleeding is arrested, complete resuscitation can be accomplished and other issues investigated as appropriate.

■ Use of Ultrasound in the Evaluation of Blunt Abdominal Trauma

In the past, the primary concern in the evaluation of blunt trauma was making the diagnosis of abdominal injury. This was based on the assertion that all injuries were best served by surgical exploration and direct repair or resection. Diagnosis then in the patient with abdominal tenderness involved physical examination or peritoneal lavage. During the 1980s computed tomography (CT) scanning revolutionized the evaluation of blunt abdominal trauma. Surgeons could essentially explore the abdomen without surgery. As the resolution of CT increased, our ability to accurately diagnose solid or hollow visceral injuries increased as well.

Techniques such as physical examination and diagnostic peritoneal lavage (DPL) are easy and rapid and can be replicated. Unfortunately, physical examination is neither sensitive nor specific for the presence of abdominal injury. Neither technique can image the retroperitoneum. In a patient with a normal hematocrit, as little as 25 mL of blood in the peritoneum results in a positive DPL, assuming complete mixing of blood and fluid. Thus, laparotomy based solely on a positive DPL risks a non-therapeutic laparotomy rate of nearly 30%.⁴ Superficial injuries to the liver or spleen or trivial mesenteric injuries can easily produce a hemoperitoneum sufficient to render DPL positive.

CT scanning is both sensitive and specific for abdominal injury and images the retroperitoneum. It is, however, relatively expensive and requires significant transport time in most institutions. In addition, it uses one of the most precious resources in the emergency department, nursing hours. CT scanning requires intravenous contrast, risking the possibility of a serious contrast reaction. Finally, using CT scanning as a screening tool risks having the scanner unavailable when needed for a patient with serious traumatic brain or abdominal injury.

Thus, it seems reasonable to use a screening test that is more readily available and less expensive and is able to exclude injury in the vast majority of patients. The more sophisticated tests, such as CT scanning, can then be used more selectively. Tso and Rodriguez have described surgeon-performed ultrasound in the evaluation of blunt abdominal trauma in the United States.⁵ This technique has been used extensively in Europe. In those countries, however, surgeons are formally trained in true abdominal ultrasonography. Ultrasound in this report was used merely as a screening tool for free intraperitoneal fluid following trauma. This technique has gained popularity and is now becoming more common in the evaluation of blunt abdominal injury.

Like DPL, focused assessment with sonography for trauma (FAST) can determine the presence or absence of hemoperitoneum (Fig. 1). FAST is clearly operator-dependent, and considerable expertise is necessary to use FAST as a screening test for abdominal injury. In the hands of a skilled

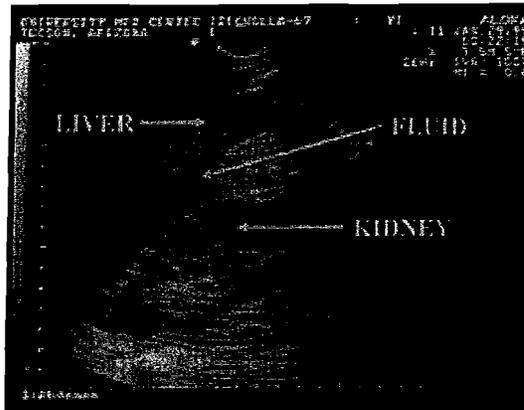


Figure 1. A positive FAST demonstrates free intraperitoneal fluid in a 20-year old man after a motor vehicle crash.

technician, it can be performed in several minutes.⁶ Like DPL, however, FAST is nonspecific and is ineffective for imaging the retroperitoneum.

The amount of abdominal fluid necessary for a positive FAST remains the subject of some debate. Clearly, more skilled operators will be able to detect smaller amounts of fluid. Many believe that several hundred milliliters of fluid are necessary to produce a positive FAST exam.⁶⁻⁸

FAST is generally performed in four areas: perisplenic, perihepatic, pelvic, and pericardial—the so-called “four Ps”. No matter which organ is injured, the perihepatic view is most commonly positive.⁹ Blood pools in Morison’s pouch, the most dependent portion of the abdomen. FAST also offers a view of the pericardium, which can be extremely helpful in some circumstances.

The utility of FAST to determine the need for laparotomy is indeterminate. McKenny et al. have encouraging data that suggest that their scoring system is accurate in determining the need for laparotomy.¹⁰ FAST has the additional advantage of being easily reproducible. Thus, a patient with a questionable FAST or only a small amount of fluid visible on ultrasound examination can be followed. If repeat ultrasound examination suggests rapidly increasing hemoperitoneum, laparotomy is probably indicated.

As with all techniques, FAST has limitations. Its ability to detect small amounts of fluid is questionable even in very skilled hands. Thus, a small bowel injury with a minimal amount of free fluid may not be discernable on the initial FAST examination. In addition, a single FAST examination is probably not capable of excluding intra-abdominal injury. A recent international consensus conference on the use of ultrasound for trauma concluded that prudent evaluation would involve a minimum of two ultrasound examinations performed at least 6 hours apart.¹¹ This requires supplementation with serial physical examinations to avoid missing intra-abdominal injury.¹²

FAST will not be able to detect visceral organ injury in the absence of hemoperitoneum. There is a subset of patients at high risk for this condition. Patients with thoracolumbar spinal fractures or pelvic fractures and patients with lower thoracic or abdominal pain or tenderness have an approximately 25% incidence of intra-abdominal injury, even with a normal FAST.¹³ Thus, these patients require CT scanning for a complete evaluation.

■ Use of Ultrasound in the Evaluation of Penetrating Trauma

FAST has recently been investigated for its utility in penetrating trauma. Because penetrating abdominal trauma may produce only a small amount of hemoperitoneum, particularly from hollow visceral injury, FAST might reasonably be expected to fail. In a prospective trial, we demonstrated that a positive FAST accurately predicts the need for surgery.⁶ A negative FAST, however, could not exclude injury in approximately one third of patients who were later found to have abdominal injuries requiring surgical repair. Thus, a positive FAST mandates exploration, while a negative FAST should prompt additional investigation. In addition, FAST has been demonstrated to be an effective means of diagnosing hemopericardium following penetrating injury around the heart.¹² A positive FAST mandates prompt sternotomy or thoracotomy for evacuation of hemopericardium and repair of the heart before cardiac tamponade becomes symptomatic.

■ CT Scanning for Traumatic Aortic Injury

Traumatic aortic injury (TAI) is potentially lethal. While over 90% of patients with TAI are dead at the scene and never reach the emergency department, a subset of patients will present hemodynamically stable who are ultimately found to have TAI.¹⁴ Unfortunately, the diagnosis of TAI can be difficult. Screening chest radiographs are notoriously insensitive in making the diagnosis. A number of chest x-ray signs have been described as being suggestive, but of those only an indistinct aortic contour is an independent predictor of TAI.^{15,16}

Unfortunately, an indistinct mediastinum is tremendously nonspecific. First, body habitus often produces an indistinct mediastinal contour, particularly as most screening chest x-rays are done in the supine anteroposterior projection. In addition, the mediastinal haziness seen with TAI is not from an aortic injury per se. Instead, mediastinal blood from concomitant venous injury or small arterial injuries is thought to produce the findings on chest x-ray. Thus, the yield of further diagnostics with abnormal chest x-rays following trauma is as low as 5%.¹⁷

In the past, angiography has been the gold standard for the diagnosis of TAI. Unfortunately, angiography is invasive and requires a potentially large contrast load. It is expensive and requires the mobilization of an entire team to carry out an investigation that has a 95% probability of being negative. Most importantly, however, it is time-consuming and delays definitive diagnosis and repair for as much as 4 or 5 hours.

Recently, we have described the use of spiral CT as a definitive test for the diagnosis of TAI.¹⁸ In our hands, a negative CT scan of the chest accurately excluded aortic injury in all cases. We are not aware of a single missed injury. In addition, CT scanning often definitively diagnoses aortic injury (Fig. 2). A diagnostic CT scan in our institution is now an indication for thoracotomy. This has reduced the time from admission to definitive surgical repair by over 50%. A small number of patients have equivocal CT scans. Patients with posterior mediastinal blood or subtle abnormalities of the aorta on CT are still best served by angiography. Using this strategy, however, we have been able to limit the number of thoracic angiograms for the diagnosis of TAI.

Traditional management of TAI has involved left posterolateral thoracotomy and direct repair of the aorta. While this results in a survival rate of over 95%, serious morbidity occurs approximately 10% of the time.¹⁴ While some morbidity, such as respiratory failure, may be serious but self-limited, others, such as the 10% incidence of paraplegia, carries with it a lifelong disability. Thus, the ideal technique would have the advantages of definitive aortic repair without the disadvantage of thoracotomy and aortic cross-clamping.

Endovascular stent-graft placement has been described for atheroscle-

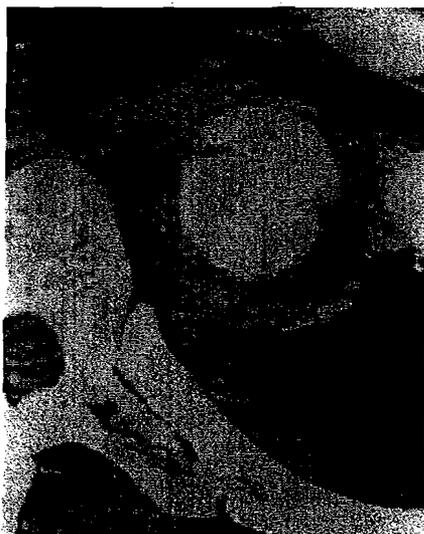


Figure 2. Helical CT scan of the chest is diagnostic for traumatic aortic injury. Patients can be operated on based solely on CT criteria.

rotic disease of the abdominal aorta as well as in acute aortic dissection. There is only one series that has reported on the use of stent-grafting for TAI.¹⁹ The results are quite encouraging, and this technique will most certainly become more common in the near future.

Some patients have serious relative contraindications to surgery. With these patients, stent-grafting is an ideal alternative. Some patients, however, can be temporized. Preoperative management of patients with TAI often involves the use of beta blockade. Limiting shear forces on the injured aorta by decreasing $\Delta p/\Delta t$ of the left ventricle may help protect the area of injury.¹⁵ There is growing evidence that definitive aortic repair may be deferred for some time if careful blood pressure control is instituted.^{15,16} This allows patients to recover and have their aortic surgery done at a time when they are more physiologically ready to accept the insult of thoracotomy and aortic repair.

■ Nonoperative Management of Blunt Solid Visceral Injury

In the past, mandatory exploration of all patients with proven solid visceral injury was advocated to prevent late sequelae such as bleeding. The advent of CT scanning has allowed identification of minor injuries that in the past were never detected. Many of these patients can be easily managed nonoperatively, and it became clear that even patients with higher-grade injuries can be managed expectantly. In addition, the risks of surgery have become more apparent. Mobilization of the liver risks dislodging clot, converting a nonbleeding hepatic injury into a life-threatening emergency. The same is true for the spleen, where mobilization can cause bleeding that had been arrested. While splenectomy is a straightforward technique, removal of the spleen does raise the concern (albeit small) of overwhelming postsplenectomy infection.

The liver is one of the most common organs injured following blunt trauma. Expectant management has become the treatment of choice for blunt hepatic injury. In a large retrospective series examining over 400 patients who were treated with the intent to manage conservatively, 98.5% of those were successfully treated nonoperatively.²⁰ Higher-grade injuries were substantially less common in this group; however, two thirds of the complications occurred in the 14% of patients with higher-grade injuries.

It has now become clear that even the development of a complication during nonoperative management, such as bleeding or bile leak, may be amenable to nonoperative techniques. In a recent series, additional minimally invasive techniques were successfully used to treat the complications of nonoperative management of hepatic injury more than 85% of the time.²¹

The success rate of nonoperative management of splenic injury has

not paralleled that of the liver. Standard approaches for nonoperative splenic injuries are bed rest and keeping the patient NPO for an undetermined period of time. Serial hematocrits and physical examinations are used to gauge the development of peritonitis and/or secondary blood loss. The cumulative adult data suggest that nonoperative management fails in the adult 10% to 15% of the time.²² The reason for so-called delayed splenic rupture is not clear. This relatively discouraging rate of secondary bleeding prompted many surgeons to continue to advocate mandatory exploration in patients with higher-grade injuries or patients with limited physiologic reserve, such as the elderly.

It would seem reasonable to think that patients with serious intraparenchymal vascular injury would be more likely to bleed. Angiography has the ability to diagnose vascular injury even if it is not actively bleeding at the time of the study. A more liberal use of angiography raises the possibility of transcatheter techniques for hemostasis as well. Transcatheter embolization is now commonly used in the management of blunt splenic injury. This technique was first described by Sclafani et al. in 1995.²³ One hundred fifty patients were studied. All patients with splenic injury underwent mandatory splenic artery angiography. The 90 patients who had a negative angiogram (no vascular injury identified regardless of the grade of injury) were observed. Only one patient failed observation. In the 60 patients with diagnosed splenic injury, hemostasis was obtained by proximal coil embolization. Proximal coil embolization presumably drops perfusion pressure to the spleen, allowing natural hemostasis. Splenic viability is maintained via collateral circulation. Using this technique, only one patient bled following transcatheter embolization. This led to a cumulative 98% splenic salvage rate, still the highest reported in the literature.

More recently, selective embolization has been advocated for the management of patients with blunt splenic injury. In this technique, a tracer wire is advanced into the splenic parenchyma until it is adjacent to the area of vascular injury. The injured splenic vessel is then directly occluded using either small particulate Gelfoam or a stainless-steel coil (Fig. 3). This technique has several potential benefits. Proximal splenic artery coils risk altering the immune function of the spleen by virtue of its relative ischemia. Selective embolization techniques infarct a very small portion of the spleen and presumably allow immune function to remain essentially normal. In our hands, selective embolization resulted in a 90–95% splenic salvage rate.²⁴

This technology has now been used in populations with progressively increasing splenic injury severity. As expected, we have now begun to define some of its limitations. A negative splenic artery study does not predict successful nonoperative management in patients with higher-grade splenic injuries. Presumably, some degree of vasospasm may exist at the time of angiography, masking the appearance of vascular injury. As

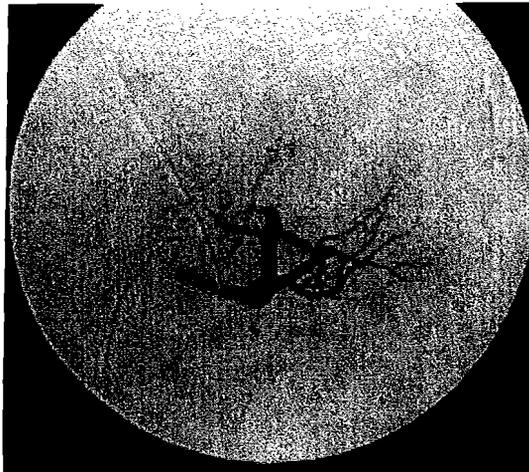


Figure 3. *Super selective embolization of the spleen following blunt trauma. A tracer wire is placed adjacent to the injury and the damaged vessel embolized.*

the splenic vasculature begins to recover, these injuries may become symptomatic.

Patients can rebleed following coil embolization. Those managed with a selective technique may develop symptomatic vascular injuries elsewhere in the spleen. We had two patients bleed following proximal splenic artery occlusion via collateral circulation. These patients had sufficient arterial pressure via the short gastric circulation to continue to bleed from their splenic vascular injuries. In our institution, the rate of bleeding following a normal angiogram is 10%; it is 8% following embolization.²⁴ Fortunately, many of these patients can be managed by so-called second-look angiography and repeated embolization. We have liberally used repeat CT scanning, even in patients who have undergone successful embolization, to identify those with delayed pseudoaneurysm formation. This strategy has resulted in a greater than 90% salvage rate, even in patients with high-grade splenic injury.

■ **Damage Control**

Traditional thinking would mandate that all injuries be repaired at the time of initial presentation. This is possible in most patients; however, a subset of patients may benefit from a staged approach to repair of their injuries.

The drug wars of the late 1980s and early 1990s produced a group of patients who presented to the nation's trauma centers with multiple injuries, often in several body cavities. Surgical therapy to repair all injuries was often quite lengthy. Despite technically successful repair of those injuries, many of these patients ultimately died. Some died acutely when

the lethal triad of acidosis, coagulopathy, and hypothermia worsened blood loss and produced cardiovascular collapse. Others died later in the intensive care unit of multiple organ failure secondary to the effects of prolonged resuscitation. In this subset of patients, initial therapy could be modified, treating life-threatening hemorrhage as the only priority. Once the hemorrhage is controlled, the patient can be further resuscitated with the goal of reversing hypothermia and coagulopathy as well as repaying the oxygen debt. Once the patient is stabilized, he or she can be returned to the operating room for more definitive repair of injuries not immediately life-threatening.

In the early 1990s, Rotondo et al. coined the term “damage control” to describe this philosophy of care.²⁵ Initially used only in the abdomen, damage control is really a set of principles that can be applied virtually anywhere in the body. The guiding principles of damage control are depicted in Table 1.

Damage control can be divided into five discrete phases. The resuscitation and evaluation phase is followed by the initial damage-control operation. Secondary resuscitation leads to a definitive surgery phase, followed by long-term reconstruction.

Phase 1

The need for damage control can often be anticipated when the patient presents to the emergency department. These patients present in extremis or may have transient hemodynamic stability, but have evidence of multicavitary injury.

All efforts in this first phase should be directed toward the rapid identification of areas of exsanguinating hemorrhage and transport to the operating room. The clinician should use only the diagnostic tests that are necessary. A chest x-ray can be obtained rapidly and often provides invaluable information. Hemothoraces can be identified or foreign bodies such as bullets located. Likewise, a plain film of the abdomen or pelvis can identify areas of hemorrhage. DPL or bedside ultrasonography can likewise identify areas of bleeding. Films should be taken and developed as the patient is transported to the operating room for hemostasis.

Table 1. *Principles of Damage Control*

-
- Only blood loss kills early.
 - GI injuries cause problems much later.
 - Everything takes longer than you think.
 - It's easy to miss an injury if you rush.
 - Hypothermia, acidosis, and coagulopathy only lead to more of the same.
 - The best place for a sick person is in the ICU.
-

Phase 2

The goal of phase 2 of damage control is to stop hemorrhage. The abdomen should be quickly explored and areas of major hemorrhage identified. A number of techniques are available to limit the amount of operative time. Only vessels that are vital to survival, such as the aorta, should be repaired. Expendable organs, such as the spleen, should be resected rather than making attempts at organ salvage. Organs that are not expendable, such as the liver, should be debrided nonanatomically to control blood loss. Major gastrointestinal contamination should be controlled by stapling the ends of the bowel and resecting the appropriate mesentery. No thought should be given to re-establishing gastrointestinal continuity. Bladder injuries should be closed in a single layer and urethral injuries either ligated or temporarily treated with external drainage. Occasionally, the patient is too unstable to tolerate even reconstruction of essential arteries. In this situation, temporary intraluminal shunts can be used to re-establish flow until the patient's resuscitation can be optimized.

Once major hemorrhage has been arrested and gastrointestinal contamination controlled, nonsurgical bleeding can be controlled with packing. The goal should be to produce sufficient pressure to stop venous bleeding. In the left or right upper quadrants, the diaphragm can be used as an organ to pack against. This generally produces sufficient pressure to stop small vessel bleeding. The pelvis can be packed in a similar manner. Topical hemostatic agents such as Gelfoam or Surgicel can be used to aid in hemostasis. In addition, the use of fibrin sealants can be helpful in the arrest of small vessel bleeding.

Abdominal closure options include a running skin closure or various synthetic materials to replace the abdominal wall. Towel clips are the fastest closure method but have the disadvantage of potentially obscuring vascular injury during angiography, which may be used in the immediate postoperative period. A running skin closure is probably preferable. Occasionally abdominal visceral edema or retroperitoneal hemorrhage precludes closing even the skin. In these cases, a "Bogata bag" can be used. A 3-L saline bag normally used for irrigation is cut open. The sterile portion is placed over the open abdomen and then sutured to the skin. Other materials, such as Goretex, may be used, but they are substantially more expensive and offer no particular advantages. Our dressing of choice is a temporary vacuum closure that uses a perforated plastic drape placed over the bowel. This allows for egress of fluid. A damp towel is placed over this, followed by damp Kerlex gauze and several Jackson Pratt drains. The dressing is completed with a sticky plastic drape that seals the abdomen. When the drains are connected to suction, the dressing materials crumple down. This temporary vacuum dressing allows for control of the serous fluid that often seeps from organs following damage-control laparotomy.

(Fig. 4). This method prevents skin breakdown and further hypothermia due to lying on wet sheets.

Phase 3: Secondary Resuscitation

The secondary resuscitation phase of a patient's care lasts between 24 and 48 hours. The goal is to restore systemic perfusion and correct coagulopathy and hypothermia as rapidly as possible. Ongoing resuscitation needs often involve the use of blood products and inotropic support as well as mechanical ventilation.

Repayment of the "oxygen debt" is central to secondary resuscitation. This nearly always requires placement of invasive hemodynamic monitoring. Patients who are seemingly stable can have substantial oxygen debt. In general, we use aggressive volume replacement to support oxygen delivery with a goal of normalizing serum lactate. The ability to normalize lactate has been shown to strongly correlate with survival, and is inversely proportional to the development of multiple organ failure.²⁶

Correcting hypothermia is equally important during secondary resuscitation. Clearly, preventing hypothermia is the best strategy, but this is often not possible in these desperately ill patients. Intravenous fluids should be warmed. Warm fluids can be used to exchange heat across the semipermeable membranes of the body, such as the peritoneal surface or the bladder or gastric mucosa. Ventilatory circuits should be warmed and ambient rewarming instituted using commercially available forced hot air blankets. For severe hypothermia, continuous arterial-venous rewarming can be employed using the commercially available Level I rewarmer. This has been shown to increase the core temperature as much as 1°F each hour.²⁷

Coagulopathy must be reversed. Ongoing coagulopathy increases bleeding and therefore transfusion needs. This serves only to perpetuate

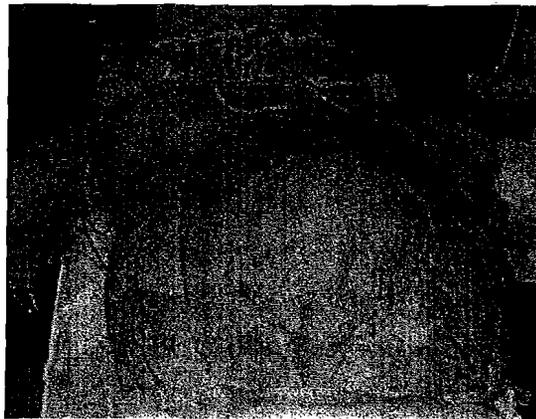


Figure 4. A temporary vacuum dressing is placed after damage-control laparotomy.

the coagulopathy. Platelets and fresh-frozen plasma should be given empirically to normalize the coagulation profile. It is also important to remember that nothing perpetuates coagulopathy as profoundly as ongoing surgical bleeding. If coagulopathy is impossible to reverse, consideration must be given to re-exploration to search for a missed injury.

During this phase, alternative means of hemostasis can be employed to supplement operative effort. Angiography can be extremely helpful in controlling ongoing blood loss, especially in areas that are not easily accessible surgically. Injuries such as deep liver lacerations may be best controlled by a combination of perihepatic packing and transcatheter embolization. There are even case reports of hepatic vein stenting for hepatic venous injury, which can be extremely difficult to control operatively.²⁸ Finally, if missiles have passed close to important vascular structures such as the aorta, diagnostic angiography can be used in lieu of direct operative exploration.

Phase 4: Definitive Surgery

Once adequately resuscitated, the patient can be returned to the operating room for re-exploration. This generally occurs between 24 and 48 hours after the initial damage-control surgery. Adequate homeostasis generally involves achieving normothermia (temperature $>36^{\circ}\text{C}$), reversal of coagulopathy (prothrombin time <15 sec, platelet count $>75,000$), and optimal cardiovascular and respiratory performance. Serum lactate should be normalized and mixed venous oxygen saturation should be above 65%. The ventilator should be weaned to an inspired oxygen content as close to 40% as possible.

In the operating room, the abdomen should first be unpacked and a search performed for missed injury. Areas such as the duodenum, pancreas, and gastroesophageal junction are notorious for being difficult to explore completely, and it is easy to miss an injury during the initial damage-control operation. The gastrointestinal tract can then be reconstructed. These patients nearly always benefit from early enteral nutrition, and a postpyloric feeding tube should be placed. A number of techniques are available, and this can be left to the surgeon's preference.

Following irrigation, the abdominal wall can be closed. Often primary closure is not possible and an abdominal wall substitute must be used. In general, we use Vicryl mesh as our fascial substitute. A more formal, vacuum-assisted closure dressing can be used, particularly if the small bowel is covered with omentum. The patient is then returned to the intensive care unit.

Phase 5: Reconstruction

If Vicryl mesh has been used, a split-thickness skin graft can be used for more formal abdominal closure following granulation of the mesh.

Occasionally, as edema resolves, the mesh can be removed and the fascia closed primarily. Another option involves muscle flap reconstruction. Options include tensor fasciae latae or a component separation technique. In general, we prefer not to burn that bridge and reserve the more complex reconstruction option for later. Lastly, skin flaps can be developed and skin at least closed, accepting the large abdominal wall hernia.

Final reconstruction should be deferred for a minimum of 6 months. At that point the abdomen can be re-explored and the abdominal wall formally reconstructed using autologous or foreign material as deemed appropriate by the surgeon.

Conclusion

It is unlikely that there will ever be a randomized prospective trial to prove the utility of damage-control techniques. It has become the treatment of choice for devastating injury in most American trauma centers. The technique is now commonly employed in areas other than the abdomen. Damage control has been used in the thorax, with blunt pelvic injury, and even with extremity injury. Even in the absence of class I data, virtually all those caring for serious injury will continue to use damage-control techniques.

■ Geriatric Trauma

The thought that elderly trauma patients should be cared for differently than younger trauma patients is a notion that has only recently been elucidated. Elderly patients fare significantly worse than younger patients with similar injuries. Elderly people have less physiologic reserve than younger patients and may be unable to mount an adequate cardiovascular response after injury. A subset of elderly patients are at special risk for death following significant blunt trauma (Table 2).

Early invasive monitoring has now been shown to improve outcome in this special subset of patients. However, when monitoring was used at the end of the normal evaluation process, mortality was not improved from its dismal 90%.²⁹ The time from patient presentation to invasive monitoring

Table 2. Geriatric Risk Factors

Pedestrian versus motor vehicle
Initial systolic blood pressure <130 mm Hg
Acidosis (pH <7.35)
Multiple long bone fractures
Head injury

was 5.5 hours. In those patients, approximately one third were in cardiogenic shock. All of them died within 24 hours. The remaining patients had evidence of low cardiac output syndrome but could be resuscitated. They all went on to develop delayed multiorgan failure.

We hypothesized that this period of nonhypotensive shock may produce an unrecoverable oxygen debt, and we sought to improve the outcome by monitoring patients earlier.²⁹ Emergency department evaluation was truncated to the smallest possible time period. Extremity x-rays were deferred and only immediately life-threatening injuries were evaluated. Head CT scanning was used for patients with coma or lateralizing signs but was not done initially in patients with a normal neurologic examination. DPL was used liberally to evaluate for abdominal injury. Time from admission to monitoring was reduced to 2.2 hours.

The same percentage of patients (approximately 30%) still presented in cardiogenic shock, but half of those patients lived. The remainder had relatively low cardiac output syndrome, but half of them also lived. There was no difference in the two groups regarding the number of injuries per patient or the distribution of injuries. Overall survival improved from 7% to 53%.

Thus, the principles that should be used when caring for elderly patients following significant injury involve the recognition that no patient is stable and that time is the enemy. Nonurgent studies should be deferred until cardiovascular stability has been established using hemodynamic monitoring.

Some of this cardiovascular dysfunction may, in fact, be due to acute cardiac ischemia. Patients with angina may be intubated and unable to report chest pain. In addition, other injuries may distract them from their chest discomfort. Finally, patients with acute blood loss may experience new-onset angina but not recognize it as pain of cardiac origin. Two new strategies may be helpful.

Perioperative beta blockade has been shown to decrease acute cardiac ischemia in patients undergoing elective surgery.³⁰ It is possible that beta blockade may play an important role in preoperative trauma patients. In addition, ACI-TIPI (acute cardiac ischemia–time sensitive predictive instrument) may help to identify patients at high risk for acute cardiac ischemia and therefore cardiovascular death.

■ The Future

The last 10 years have seen an explosion of technological advances in the care of injury. Dogmas such as resuscitation strategy and mandatory exploration for injury repair have been replaced by more physiologically based strategies. Future technology will continue to evolve rapidly, and it

is almost certain that these technological advances will be used to provide trauma care. Evaluation will become less invasive and more precise. Invasive cardiovascular monitoring will be replaced with noninvasive devices. Minimally invasive techniques will gain more popularity in the care of the injured. In addition, we will continue to define special subsets of patients who will benefit from these new techniques.

■ References

1. Capone AC, Safar P, Stezoski W, et al. Improved outcome with fluid restriction in treatment of uncontrolled hemorrhagic shock. *J Am Coll Surg* 1995;180:49-56
2. Burris D, Rhee P, Kaufmann C, et al. Controlled resuscitation for uncontrolled hemorrhagic shock. *J Trauma* 1999;46:216-233
3. Bickell WH, Wall MJ, Pepe PE, et al. Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injuries. *N Engl J Med* 1994;331:1105-1109
4. Feliciano DV, Rozycki GS. The management of penetrating abdominal trauma. *Advances in Surgery* 1995;28:1-39
5. Tso P, Rodriguez A, Cooper C, et al. Sonography in blunt abdominal trauma: a preliminary report. *J Trauma* 1992;33:39-43
6. Udobi KF, Rodriguez A, Chiu WC, Scalea TM. Role of ultrasonography in penetrating abdominal trauma: a prospective clinical study. *J Trauma* 2001;50:475-479
7. Boulanger BR, Brenneman FD, Kirkpatrick AW, et al. The indeterminate abdominal sonogram in multi-system blunt trauma. *J Trauma* 1998;45:52-56
8. Branney SW, Wolfe RE, Moore EE, et al. Quantitative sensitivity of ultrasound in detecting free intraperitoneal fluid. *J Trauma* 1995;39:375-380
9. Rozycki GS, Feliciano DV, Davis TP. Ultrasound as used in thoracoabdominal trauma. *Surg Clin North Am* 1998;78:295-310
10. McKenney MG, Martin L, Lentz K, et al. 1,000 consecutive ultrasounds for blunt abdominal trauma. *J Trauma* 1996;40:607-612
11. Scalea TM, Rodriguez A, Chiu WC, et al. Focused assessment with sonography for trauma (FAST): results from an international consensus conference. *J Trauma* 1999;46:466-472
12. Rozycki GS, Feliciano DV, Ochsner MG, et al. The role of ultrasound in patients with possible penetrating cardiac wounds: a prospective multicenter study. *J Trauma* 1999;46:543-552
13. Chiu WC, Cushing BM, Rodriguez A, et al. Abdominal injuries without hemoperitoneum: a potential limitation of focused abdominal sonography for trauma (FAST). *J Trauma* 1997;42:617-625
14. Fabian TC, Richardson JD, Croce MA, et al. Prospective study of blunt aortic injury. Multicenter trial of the American Association for the Surgery of Trauma. *J Trauma* 1997;42:374-380
15. Fabian TC, Davis KA, Gavant ML, et al. Prospective study of blunt aortic injury. Helical CT is diagnostic and antihypertensive therapy reduces rupture. *Ann Surg* 1998;5:666-677
16. Nagy K, Fabian T, Rodman G, et al. Guidelines for the diagnosis and management of blunt aortic injury: an EAST practice management guidelines work group. *J Trauma* 2000;48:1128-1143
17. Mirvis SE, Bidwell JK, Buddemeyer EU, et al. Value of chest radiography in excluding traumatic aortic rupture. *Radiology* 1987;163:487-493

18. Mirvis SE, Kathirkamuganathan S, Buell J, Rodriguez A. Use of spiral computed tomography for the assessment of blunt trauma patients with potential aortic injury. *J Trauma* 1998;45:922-930
19. Tadashi F, Yukioka T, Ishimaru S, et al. Endovascular stent grafting for the treatment of blunt thoracic aortic injury. *J Trauma* 2001;50:223-229
20. Pachter HL, Knudson MM, Esrig B, et al. Status of nonoperative management of blunt hepatic injuries in 1995: a multicenter experience with 404 patients. *J Trauma* 1996;40:31-38
21. Carrillo EH, Spain DA, Wohltmann, CD, et al. Interventional techniques are useful adjuncts in nonoperative management of hepatic injuries. *J Trauma* 1999;46:619-622
22. Sclafani SJA, Weisberg A, Scalea TM, et al. Blunt splenic injuries: nonsurgical treatment with CT, arteriography, and transcatheter arterial embolization of the splenic artery. *Radiology* 1991;181:189-196
23. Sclafani SJA, Shaftan GW, Scalea TM, et al. Nonoperative salvage of computed tomography-diagnosed splenic injuries: utilization of angiography for triage and embolization for hemostasis. *J Trauma* 1995;39:818-827
24. Haan J, Scott J, Boyd-Kranis RL, et al. Admission angiography for blunt splenic injury: advantages and pitfall [abstract]. *J Trauma* 2000;49:1172
25. Rotondo MF, Schwab CW, McGonigal MD, et al. 'Damage control': an approach for improved survival in exsanguinating penetrating abdominal injury. *J Trauma* 1993;35:375-382
26. Abramson D, Scalea TM, Hitchcock R, et al. Lactate clearance and survival following injury. *J Trauma* 1993;37:584-588
27. Gentilello LM, Cobean RA, Offner PJ, et al. Continuous arteriovenous rewarming: rapid reversal of hypothermia in critically ill patients. *J Trauma* 1992;32:316-325
28. Denton JR, Moore EE, Coldwell DM. Multimodality treatment for grade V hepatic injuries: perihaptic packing, arterial embolization, and venous stenting. *J Trauma* 1997;42:964-968
29. Scalea TM, Simon HM, Duncan AO, et al. Geriatric blunt multiple trauma: improved survival with early invasive monitoring. *J Trauma* 1990;30:129-136
30. Zaugg M, Tagliente T, Lucchinetti E, et al. Beneficial effects from beta-adrenergic blockade in elderly patients undergoing noncardiac surgery. *Anesthesiology* 1999;91:1674-1686