Anesthetic Concerns in Trauma Victims Requiring Operative Intervention: The Patient Too Sick to Anesthetize

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KEYWORDS

- Multiple trauma • Injury • Shock • Resuscitation
- Massive transfusion • Damage control • Extracorporeal

Trauma is the third leading cause of death overall in the United States, and the leading cause of death among those aged 1 to 44 years. Nearly 30% of years of potential life lost before age 65 results from traumatic injury, the largest contribution of any cause of death and nearly twice that of the next leading cause, cancer.[1] Globally, the World Health Organization projects a 40% increase in global deaths caused by injury between 2002 and 2030.[2] As this burden of injury increases, anesthesiology practitioners will be challenged with the operative case management of this disease.

Case study. You are the anesthesiologist caring for an obese 66-year-old man who was involved in a motor vehicle crash; he was not wearing a seat belt. The patient presents to the operating room (OR) for emergency exploratory laparotomy. Blood pressure (BP) 83/P, heart rate 128, So_2 94% on 1.0 Fio_2 per endotracheal tube, Glasgow Coma Scale 5T, temperature 35.2°C. The patient has ecchymoses over the right face, contusions over the sternum, a flail chest, distended abdomen, and a pelvic binder in place. Crystalloids are infusing through a 14G peripheral intravenous line and the first unit of packed red blood cells (PRBC) is hanging. What are your management priorities and the interventions that you can provide to improve this patient's outcome?

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CARDIAC TRAUMA

Cardiac trauma may complicate anesthetic management of patients following injury, particularly in the patient with preexisting comorbidities on multiple medications. Traditional markers of myocardial ischemia such as electrocardiogram (ECG) changes and increased enzyme levels may be misleading in the patient who has sustained a massive energy transfer to their thorax from trauma. Blunt thoracic trauma constitutes 25% of all trauma mortalities,[3] and can have a vast range of clinical presentations. Hemodynamic instability raises a high index of suspicion for blunt myocardial injury (BMI), previously referred to as cardiac contusion, pericardial tamponade, blunt thoracic aortic injury, and myocardial infarction. Cardiac and thoracic vasculature must be evaluated during the initial trauma work-up.

Blunt traumatic aortic injury (TAI) is typically seen in rapid deceleration and crush injuries. Approximately 85% of these patients die at the scene,[4] often from concomitant nonsurvivable injuries. For those who reach the hospital, the severity of thoracic skeletal injury often may not correlate with the severity of aortic injury. TAI can be seen after a fall, and side impact in a motor vehicle crash is a major risk factor, both of which highlight the mechanism of injury: deceleration to the entire body, not just a direct blow to the chest. Despite optimal medical care, approximately 50% of patients who make it to the hospital die in the first 24 hours of hospitalization,[5] often as a result of associated trauma. The classic clinical signs and symptoms of aortic injury are chest pain, pseudocoarctation, aortic insufficiency murmur, and sternal fractures; patients may be asymptomatic from TAI and the diagnosis is made during radiologic evaluation. A patient who arrives in the emergency department (ED) after sustaining blunt thoracic trauma should have an ECG to evaluate for dysrhythmia or ECG changes. If a patient has no ECG changes on arrival then there is a lower likelihood of any cardiac injury.[6,7] If there are ECG changes, further evaluation is warranted. This evaluation should include cardiac markers (cardiac troponin T and I), with consideration of transesophageal or transthoracic echocardiography (TEE or TTE). TEE is more sensitive and specific than TTE in the detection of BMI, valvular injury, and aortic injury.[7] Pharmacologic management is aimed toward blood pressure and heart rate control, and hemodynamic stability may be best achieved with high-dose narcotic and β blockade, particularly if the patient has hemorrhage as a result of associated injuries.

Widening of the mediastinum on the chest radiograph is nonspecific and may frequently not be present in cases of severe aortic trauma. Although aortography is still held by many to be the gold standard, advancements in computed tomography angiography (CTA) have allowed it to largely replace older techniques in preoperative evaluation.[8] The other advantage of CTA is its ability to identify other significant diagnoses (eg, blunt cerebrovascular injury).

In the last 10 years endovascular stent grafting has become widely used to treat blunt thoracic aortic injury. No prospective, randomized, controlled clinical trials have yet compared stent graft placement with open repair. Meta-analyses of retrospective studies have suggested better outcomes from stent graft placement compared with open repair. Most of these studies show that mortality, paraplegia, and stroke occur less often with delayed endovascular repair.[9–11] Others have found no decrease in mortality but a decrease in neurologic injury.[12] Although there are insufficient long-term outcome data, it does seem that endovascular repair is a reasonable and effective treatment of blunt thoracic aortic injury.

Transient left ventricular apical ballooning, also known as Tako Tsubo syndrome, is a reversible cardiomyopathy described in patients with high exposure to catecholamine. The description of this syndrome, leading to refractory hypotension associated

with multiple trauma,[13] highlights an important fact: many patients who have myocardial insufficiency following injury will have complete recovery of cardiac function with resolution of traumatic illness.

There is no standard anesthetic management schema for cardiac trauma; some disease presentations have significantly different management strategies than nontrauma presentations (eg, systolic anterior motion). A pericardial window is often critical in the evaluation of a hemodynamically stable patient who is suspected of having a cardiac injury.[14] A wide variety of cardiac injuries that can occur secondary to trauma are outlined in **Table 1**.

PULMONARY TRAUMA

The trauma patient with underlying lung disease presents with 2 classes of pulmonary complications: those attributable to the traumatic injury per se, and those caused by underlying medical comorbidities that are exacerbated by the traumatic insult. Older patients with preexisting pulmonary disease are at greater risk of perioperative and postoperative pulmonary complications than young patients with healthy lungs.[30] Moreover, perioperative lung complications may be as prevalent and as predictive of mortality as perioperative cardiac complications.[31,32] There is little evidence to suggest that interventions taken by an anesthesiologist during the perioperative period might reduce postoperative pulmonary complications in patients with traumatic injury.[32] This section focuses on the 2 most common and life-threatening pulmonary problems faced by anesthesiologists during acute trauma: inability to oxygenate and inability to ventilate (**Table 2**).

Cannot Oxygenate: Hypoxia

In the intubated trauma patient receiving a high Fio_2, hypoxia results from a high fraction of pulmonary blood flow failing to perfuse ventilated portions of the lungs before reentering the systemic circulation. Although an increased shunt fraction is most commonly caused by intrapulmonary pathology, it can also be intracardiac and this may only become physiologically significant after another insult causes higher pulmonary artery pressures (ie, fat embolism syndrome (FES), pulmonary embolism (PE), acute lung injury (ALI), and aspiration pneumonitis).

Blunt chest trauma can result in pulmonary contusions and flail chest. Development of pulmonary contusions is independently associated with development of acute lung injury/acute respiratory distress syndrome (ALI/ARDS), pneumonia, and death.[33] Increasing age and medical comorbidities (congestive heart failure, cirrhosis, renal dysfunction, and others) are also independently associated with sepsis and death in thoracic trauma with rib fractures.[34] Even small pulmonary contusions can, within 24 hours, create an inflammatory cascade leading to increased pulmonary capillary permeability, decreased surfactant production, alveolar collapse, and predisposition to sepsis by inhibiting macrophage and lymphocyte immune function.[35] Moreover, clinically significant pulmonary contusions are often missed on initial chest radiography. Computed tomography (CT) of the chest is the gold standard for diagnosis. Thus, management of patients with pulmonary contusions should focus on, in addition to maintaining oxygenation, avoiding a second assault on the lungs such as aspiration, fat embolism, sepsis, or ventilator-induced lung injury. Intraoperative management strategies should include open-lung pressure-limited ventilation if hemodynamically tolerated and minimizing inspired Fio_2 in the absence of shock.[36]

Flail chest is defined as at least 2 ribs broken in 2 places causing an invagination of the chest wall with each attempt at respiration. Like pulmonary contusions, flail chest

Table 1
Traumatic cardiac injuries, typical presentation, diagnostic evaluation and treatment strategies

Injury	Diagnosis	Presentation	Treatment
Blunt myocardial injury[6,7]	Typically seen following blunt chest trauma. Presentation ranges from asymptomatic to cardiac rupture	ECG changes, wall motion abnormalities on TTE/TEE	Monitor telemetry and CE; increased risk of arrhythmia, late rupture, aneurysm formation, CHF and intracavitary thrombus
Traumatic VSD[15,16]	Typically seen after unrestrained MVC; panholosystolic murmur at left lower sternal border with precordial thrill. Rare complication of trauma and can be present with no symptoms or can result in heart failure	TTE or TEE; may have increase in cardiac enzymes (although this is an overly sensitive marker as high-energy transfer from trauma may also cause increase in enzymes). ECG changes are more predictive	Immediate or delayed surgical treatment dependent on clinical symptoms and size of defect. Multiple case studies of traumatic VSDs that were not repaired in patients who have remained healthy
Cardiac foreign bodies[17]	Traumatic versus iagtrogenic. Ranges from symptom free to cardiac tamponade or hemorrhagic shock	Chest radiograph, CT scan, echocardiography (immediate or delayed)	Surgery versus conservative treatment depending on patient's symptoms, position of object, and associated risks of embolization
Papillary muscle/ chordae tendinae rupture[18,19]	No symptoms to acute cardiogenic shock; holosystolic murmur at the apex radiating to axilla. Most common injury is to mitral valve	TTE or TEE	Surgical intervention based on hemodynamic stability and other associated injuries; mitral valve replacement versus subvalvular repair
Systolic anterior motion[20,21]	Typically seen in association with hypovolemia or anesthesia-mediated vasodilatation hypotension	Diagnosed with echocardiography; can be difficult to diagnose. Patient will either be predisposed or this will be a preinjury disease; not caused by trauma per se	Phenylephrine and fluid resuscitation; possible use of β-blockers in those with relative reductions in preload
Penetrating cardiac injury[22,23]	Pericardial tamponade to hemorrhagic shock depending on whether the pericardium is violated	FAST scan in ED has high sensitivity and specificity; pericardial window is the gold standard to confirm cardiac injury	Pericardial window, thoracotomy or median sternotomy

Cardiac tamponade[24]	Muffled heart tones, pulsus paradoxus, increased CVP, bulging neck veins, signs of shock	FAST scan in ED has high sensitivity and specificity	Delay induction of anesthesia if possible until surgeon in OR to assume direct control of the cardiac injury if hemodynamic instability
Blunt cardiac rupture or rupture of major vessels[25–28]	Varies, but typically with hypotension and hemorrhagic shock; may or may not have tamponade symptoms	High degree of suspicion is necessary; FAST, TTE or TEE	Possible need for emergency thoracotomy; immediate OR intervention
Blunt aortic injury[9–12,26,29]	Typically seen after rapid deceleration injuries. Presentation ranges from asymptomatic to hemodynamic instability	Injury identified by CTA or echocardiogram	Immediate versus delayed surgical repair. Endovascular stent placement versus primary repair

Signs and symptoms may be difficult to assess in patients with multiple injuries in a noisy/chaotic trauma bay.

Abbreviations: CE, cardiac enzymes; CHF, chronic heart failure; CT, computed tomography; CTA, computed tomographic angiography; CVP, central venous pressure; ECG, electrocardiogram; FAST, focused abdominal sonography for trauma; MVC, motor vehicle crash; TTE/TEE, transthoracic/transesophageal echocardiography; VSD, ventricular septal defect.

can lead to respiratory failure, from splinting because of chest wall pain. Adequate analgesia and effective pulmonary toilet are essential management goals. However, opiates, the mainstay of analgesia in most trauma and general surgery patients, depress the central respiratory drive, making them a suboptimal choice in the setting of flail chest. Neuraxial analgesia (epidural catheters or intercostal nerve blocks) in the setting of flail chest can be helpful, preventing intubation, and reducing duration of mechanical ventilation and time to discharge from the intensive care unit (ICU), and the incidence of nosocomial pneumonia.[37,38]

FES is an under-recognized pulmonary complication. FES has variable clinical definitions and a wide range of reported clinical incidence (0.25%–35%) in acute trauma.[39] Fat particles are identified in the pulmonary arteries in 90% of patients with skeletal trauma.[39] Major diagnostic criteria include a petechial rash, altered mental status, and respiratory insufficiency. Minor nonspecific signs include tachypnea, tachycardia, hypoxia, hypercapnea, and infiltrates on chest radiography. Severity of presentation can be fulminant or subacute and occurs 12–24 hours after injury or with surgical manipulation of long-bone fractures. Other risk factors include delayed stabilization of long-bone or pelvic fractures, male gender, age 10 to 40 years, multiple long-bone fractures, and surgical internal fixation of long-bone fractures with intramedullary reaming.[40] Because the signs and symptoms of FES are nonspecific and the treatment of FES is supportive, preventing FES and maintaining a high index of suspicion for FES is critical. Prompt fixation of pelvic and long-bone fractures may be the most effective means of avoiding FES.[41,42] If FES is present, or if there is a high suspicion of FES, manipulating long bones for definitive internal fixation may exacerbate

Table 2
Differential diagnosis and treatment of pulmonary complications of traumatic injury

	Cannot Ventilate	Cannot Oxygenate
Abnormality	High peak, high plateau pressures	Increased intrapulmonary shunt fraction
Nontraumatic causes	Auto-PEEP; CHF flare; morbid obesity; Trendelenburg position; abdominal compartment syndrome; pleural effusion	Preexisting obstructive or parenchymal lung disease (asthma, COPD); pulmonary fibrosis; bronchiectasis; pulmonary edema; aspiration pneumonitis; pulmonary embolism; TRALI
Traumatic causes	Tension pneumothorax; pulmonary contusion; ARDS (may present acutely following pulmonary contusion, aspiration or massive transfusion); gastrothorax; hemothorax	Pulmonary contusions; ARDS; pulmonary laceration; fat emboli syndrome (with long-bone fractures or massive soft tissue injury); aspiration; TRALI
Management options	• Needle decompression and tube thoracosomy if breath sounds absent and tension physiology present • Consider Auto-PEEP • Try disconnecting patient from the ventilator temporarily, allowing any trapped gas to escape • If BP and plateau pressures improve, auto-PEEP is likely present and high peak flows should be tolerated provided that plateau pressures can be kept less than 35 cm H_2O • Prolong the inspiratory/expiratory ratio to ensure that the flow reaches zero at end expiration • May require tolerating hypercapnea • Review preoperative CT scan chest, consider intraoperative ultrasound/TTE/TEE • Intraoperative bronchoscopy • Check circuit and endotracheal tube patency • Suction airways • Endotracheal bronchodilators • Consider PE or fat embolism	• Examine patient for evidence of reversible causes • Wheezing (obstructive lung disease, PE/fat emboli, CHF) • Trial of bronchodilators and/or dieresis • Moderate intraoperative fluids • Recruitment maneuvers, PEEP (prevent derecruitment) • Consider avoiding manipulation and/or intramedullary reaming of long bones • Extracorporeal support

Abbreviations: COPD, chronic obstructive pulmonary disease; PEEP, positive end-expiratory pressure.

FES, causing clinical deterioration. Treatment options in this scenario include external fixation and avoiding definitive intramedullary internal fixation until clinical signs of FES have stabilized.[43] Rapidly progressive hypoxemia and worsening pulmonary compliance should alert the anesthesiologist to the possibility of FES, which can also occur with soft tissue injury (**Fig. 1**). A differential diagnosis should include other traumatic
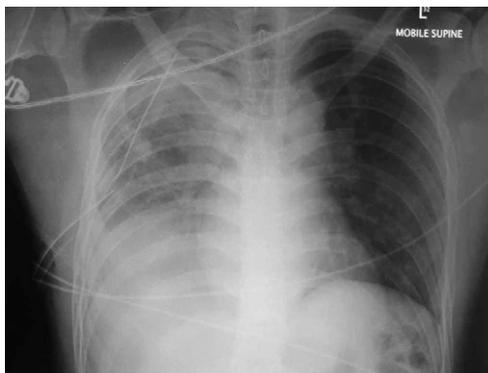
Fig. 1. Blunt thoracic trauma. Patient has right hemothorax, pneumothorax and pulmonary contusion. Hypoxia, high peak inspiratory pressure, and myocardial dysfunction are present.

causes such as pneumothorax, pulmonary contusion, severe aspiration, and volume overload from massive resuscitation.

Transfusion-related acute lung injury (TRALI) is noncardiogenic pulmonary edema resulting from immune reactivity of certain leukocyte antibodies a few hours after transfusion. Signs and symptoms appear 1 to 2 hours after transfusion and peak within 6 hours. Hypoxia, fever, dyspnea, and even fluid in the endotracheal tube may occur. There is no specific therapy other than stopping transfusion and instituting critical care supportive measures. Most patients recover in 96 hours, although TRALI is 1 of the top 3 most common causes of transfusion-related deaths.[44–46]

Cannot Ventilate: High Ventilator Pressures

Increased peak pressures and normal plateau pressures often reflect large airway obstruction from various causes; a differential diagnosis following trauma includes blood, food particles, and foreign bodies. Conversely, increased peak pressures and plateau pressures can reflect decreased pulmonary, diaphragmatic, and/or chest wall compliance (see **Table 2**). Worsening compliance is an ominous situation as it may be associated with hypoxia.

To differentiate between high peak and high plateau pressure, blow a fixed volume of air into the ventilator under high flows, with high-pressure cut-offs such that the entire tidal volume can be delivered and the inspiratory flow reaches zero before expiration. The end-inspiratory pressure at a zero flow rate is the plateau pressure, or the inspiratory pressure at the level of the alveoli. The peak pressure is simply the peak pressure generated by a given tidal volume at a given flow rate and reflects bronchial, large airway, or endotracheal tube resistance more so than plateau pressure. The peak pressure will be higher with smaller-size endotracheal tubes, or those partially occluded with blood, pulmonary debris, or secretions.

Fig. 1 is a chest radiograph of a patient with a constellation of thoracic injuries that may lead to cardiopulmonary insufficiency and progress to cardiac and respiratory collapse. When mechanical ventilation fails to achieve adequate oxygenation and/or carbon dioxide (CO_2) exchange, extracorporeal support can be a life-saving treatment option following trauma (see later discussion).

NEUROLOGIC INJURIES

Traumatic brain injury (TBI) and hemorrhagic shock are the 2 leading causes of death from trauma. Central nervous system injury is the most common cause of civilian

deaths from injury, but hemorrhage and resultant multiple organ system failure account for almost half of all deaths and almost all of the deaths that are available for secondary prevention.[47] It is not uncommon to see these devastating diseases concurrently.

Severe TBI is known to be 1 of the most important prognostic factors in patients with trauma, with mortality in 1 series of patients with head injuries about 3 times higher than in patients without relevant TBI (22.1% vs 7.3%).[48] The reasons are likely multifactorial, including interplay between the neuroendocrine system and the injured brain. A catecholamine surge occurs after TBI[49–51] that may manifest as acute hemodynamic instability and respiratory failure in the OR. Subendocardial ischemia may lead to biventricular heart failure, even in young previously healthy patients. Acute cardiopulmonary failure may be exacerbated when treated with exogenous vasoactive agents, perpetuating a cycle of ischemia. Rapidly progressive cardiopulmonary collapse with increased intracranial pressure (ICP), resulting from severe TBI, can be successfully treated with extracorporeal life support.[52,53] A 16-year-old and a 20-year-old with TBI after blunt trauma each developed cardiac failure (ejection fraction by echocardiography 20%, and 10%, respectively), ARDS, and multiorgan failure. Anticoagulation was not used for 24 hours in the 16-year-old (total extracorporeal life support [ECLS] time 4 days) and not at all in the 20-year-old patient (total ECLS time 49 hours). Both patients had full neurologic recovery.

β blockade has been shown to be protective in human studies in patients with brain injuries. Retrospective database reviews have shown neurologic improvement and a survival advantage associated with β blockade following TBI.[54–56] The largest series to date reported a significant decrease in mortality for patients with severe TBI (50% for those exposed to β blockade vs 70% for those who were not). On subgroup analysis, elderly patients with severe injury had a mortality of 28% on β-blockers compared with 60% when they did not receive them (P = .001).[54] Intraoperative initiation of β blockade in the acute management of patients with TBI has not been studied.

Non-neurologic organ dysfunction is common in patients with severe TBI and is independently associated with worse outcome.[57,58] Of 209 consecutive patients with TBI, 89% developed dysfunction of at least 1 non-neurologic organ system. Respiratory failure was the most common, followed by cardiovascular failure. Failure of the coagulation and renal systems was also seen.[57]

In the absence of ICP and cerebral oxygenation monitoring, the goals to achieve optimal cerebral perfusion and oxygenation are systolic blood pressure >90 mm Hg and Pao_2 >60 (So_2 >90%). Management of elevated ICP intraoperatively may be accomplished with mannitol (0.25–1 g/kg) with particular attention to avoidance of hypovolemia (systolic BP >90 mm Hg) and maintenance of intravascular volume status, or hypertonic saline (HTS). The use of HTS for ICP control developed from studies on small volume resuscitation in multitrauma patients in hemorrhagic shock. The subgroup with TBI showed the greatest survival benefit, while preserving or even improving hemodynamic parameters.[59] Although the use of mannitol has become commonplace, most of the discussion in the guidelines surrounds the increasing evidence for effectiveness and safety of HTS in treating increased ICP. Current research is ongoing.

Although robust data are lacking, the American Association of Neurologic Surgeons Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injuries suggest maintenance of mean arterial pressure 85 to 90 mm Hg for the first 7 days following acute spinal cord injury to improve spinal cord perfusion.[60] Maintenance of cerebral and spinal cord perfusion pressures can be a challenge in patients with exsanguinating hemorrhage, in those with acute cardiorespiratory failure as a result

of their neurologic injury, or in patients with neurogenic shock. Vasoactive agents that increase peripheral resistance and concomitantly add inotropic support may be advantageous.

EXSANGUINATION/COAGULATION ABNORMALITIES

Bleeding is the most frequent cause of preventable death after severe injury. Penetrating injury to solid organs or major vasculature, and blunt trauma resulting in high-grade pelvic fractures, significant solid organ lacerations, or massive tissue destruction may lead to exsanguination (Fig. 2). Maintaining a low mean arterial blood pressure (controlled hypotension) decreases mortality following penetrating trauma[61] and is safe following blunt trauma.[62] The Advanced Trauma Life Support Course (ATLS) now includes a far more extensive discussion on the importance of balancing the concept of limited resuscitation with excessive early crystalloid administration that may dilute blood, dislodge clot, and increase hemorrhage. A recent retrospective review did not show a difference in mortality in patients with penetrating truncal injury who received limited crystalloid resuscitation in the field[63]; larger randomized prospective trials are still needed.[64] The American Society of Anesthesiologists has published guidelines for blood transfusion and adjuvant therapies.[44] Unfortunately for the trauma patient who requires acute operative intervention, there is rarely time for preoperative assessment of possible bleeding abnormalities. The American Society of Anesthesiologists recommends transfusion to maintain hemoglobin level >6 g/dL, prothrombin time (PT) greater than 1.5 times normal or international normalized ratio greater than 2.0, an activated partial prothrombin time greater than 2 times normal, platelets 50,000 cells/mm^3, and fibrinogen <80 mg/dL as a guide to therapy, but reliance on laboratory values should not delay product infusion in a patient with acute traumatic exsanguination. Classic resuscitation strategies have been called into question by recent findings.[65]
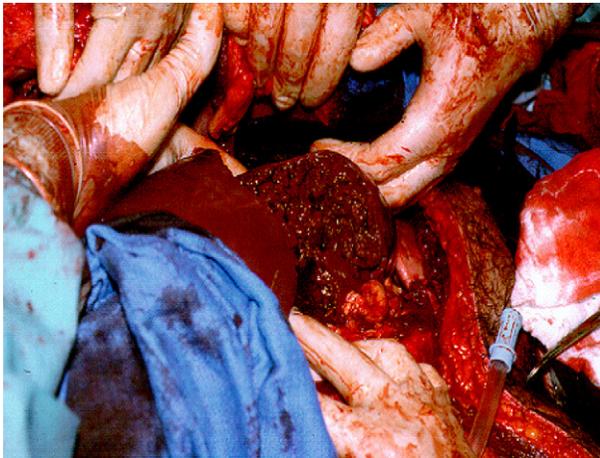
Fig. 2. High-grade liver injury following intraoperative hemorrhage control. Controlled under-resuscitation (delayed resuscitation), with mean arterial pressure of 50 to 60 mm Hg may decrease blood loss. This management strategy is challenging in a patient with traumatic brain or spinal cord injury, and in those with known preexisting cardiac or vascular disease.

At the time of arrival at the emergency department, approximately 25% of trauma patients have a detectable coagulopathy that is associated with poor outcome, and increased PT and partial thromboplastin time are independent predictors of mortality.[66,67] **Fig. 3** shows the type of injury that may lead to coagulopathy; massive resuscitation of these patients increases the risk of pulmonary and cardiac insufficiency. There is a positive correlation with injury severity score and coagulopathy, but dilution of clotting factors through infusion of intravenous fluids is not the only mechanism.[66] Coagulopathy associated with traumatic injury is the result of multiple independent but interacting mechanisms: early coagulopathy is driven by shock and requires thrombin generation from tissue injury as an initiator; initiation of coagulation occurs with activation of anticoagulant and fibrinolytic pathways. This acute coagulopathy of trauma-shock (ACoTS) is altered by subsequent events and medical therapies, including acidemia, hypothermia, and dilution. ACoTS should be considered distinct from disseminated intravascular coagulation as described in other conditions.[68,69]

The concept of damage control resuscitation attempts to address early coagulopathy and advocates transfusing earlier and with increased amounts of plasma and platelets along with the first units of red blood cells, while simultaneously minimizing crystalloid use in patients who are predicted to require a massive transfusion (MT). Patients who require MT represent a small percentage of admissions (3%), yet have a 30% to 60% mortality.[70,71] A recent review of records of 467 MT civilian trauma patients transported from the scene to 16 level 1 trauma centers between July 2005 and June 2006 correlated transfusion ratios with mortality.[72] The plasma/RBC ratio ranged from 0 to 2.89 (mean $\pm$ SD 0.56 $\pm$ 0.35) and the platelets/RBC ratio ranged from 0 to 2.5 (0.55 $\pm$ 0.50). Plasma/RBC and platelet/RBC ratios and injury severity score were predictors of death at 6 hours, 24 hours, and 30 days in multivariate logistic models. Thirty-day survival was increased in patients with high plasma/RBC ratio (>1:2) relative to those with low plasma/RBC ratio ($\leq$1:2) (low 40.4% vs high 59.6%, $P<.01$). Similarly, 30-day survival was increased in patients with high platelet/RBC ratio (>1:2) relative to those with low platelet/RBC ratio ($\leq$1:2) (low

Fig. 3. Massive right gluteal degloving injury following motorcycle crash. Coagulopathy and cardiopulmonary dysfunction are present. (Patient is in the prone position on the OR table, head to the right and lower extremities on the left.)

40.1% vs high 59.9%, $P<.01$). In patients with combat-related trauma requiring MT, the transfusion of an increased fibrinogen/RBC ratio was independently associated with improved survival to hospital discharge, primarily by decreasing the number of deaths from hemorrhage.[73] A retrospective chart review of 252 patients at a US Army combat support hospital who received MT (>10 units of RBCs in 24 hours) used the amount of fibrinogen within each blood product to calculate the fibrinogen/RBC (F/R) ratio transfused for each patient. The mean (SD) F/R ratios transfused for the low and high groups were 0.1 g/unit ($\pm$ 0.06), and 0.48 g/unit ($\pm$ 0.2), respectively ($P<.001$). Mortality was 52% and 24% in the low and high F/R ratio groups respectively ($P<.001$). Clinicians can meet this requirement by transfusing 1 unit of FFP for every 2 units of red cells, or by transfusing one 10-unit bag of cryoprecipitate for every 10 units of red cells. High concentrations of fibrinogen are available not only from cryoprecipitate but also from plasma-derived fibrinogen concentrates. Fibrinogen is also now being produced using recombinant techniques and is available in a lyophilized powder form (Pharming Group, Leiden, The Netherlands).

MT protocols in the United States are now incorporating early use of balanced resuscitation.[74,75] The optimal ratio of blood products has not yet been determined, although many agree that an FFP/PRBC transfusion ratio greater than 1:1.5 is best.[76] In patients requiring more than 8 units of blood after serious blunt injury, an FFP/PRBC transfusion ratio greater than 1:1.5 was associated with a significantly lower risk of mortality but a higher risk of acute respiratory distress syndrome. However, in a recent review of humans who received aggressive factor replacement, data collected on 806 consecutive trauma patients admitted to the intensive care unit for 2 years showed no significant difference in outcome when comparing patients who had a 1:1 PRBC/FFP ratio with those who did not receive any FFP.[77] Patients were stratified by PRBC/FFP transfusion ratio in the first 24 hours. Analyzing these patients by stepwise regression controlling for all significant variables, the PRBC/FFP ratio did not predict intensive care unit days, hospital days, or mortality even in patients who received MT ($\geq$10 U). In a more recent study, FFP was associated with higher incidence of multiple organ failure and ARDS.[78] Data were obtained from a multicenter prospective cohort study evaluating clinical outcomes in bluntly injured adults with hemorrhagic shock who required blood transfusion. Patients with isolated TBI and those not surviving beyond 48 hours were excluded. Cox proportional hazard regression models were used to estimate the outcome risks (per unit) associated with plasma-rich transfusion requirements during the initial 24 hours after injury after controlling for important confounders. There was no association with plasma-rich transfusion components and mortality or nosocomial infection. However, for every unit given, FFP was independently associated with a 2.1% and 2.5% increased risk of multiorgan failure and ARDS, respectively. Cryoprecipitate was associated with a 4.4% decreased risk of multiorgan failure (per unit), and platelets were not associated with any of the outcomes examined. When early deaths (within 48 hours) were included in the model, FFP was associated with a 2.9% decreased risk of mortality per unit transfused. Conflicting data such as that presented here, in addition to mounting evidence that transfusion of stored blood increases mortality,[79] organ dysfunction,[80] nosocomial infections[81] and resource use,[82] challenge the acute care provider to make appropriate resuscitation decisions.

Several investigators have recently described the proinflammatory characteristics of crystalloid infusion[83–86]; others have described decreased abdominal compartment syndrome and death[87] or increased ventilator and ICU-free days[88] simply by limiting the amount of crystalloid infused early after admission. Plasma has been shown to

be less inflammatory than artificial colloid, albumin, or lactated Ringer solution in an animal study of hemorrhagic shock.[85]

Adjuvant therapies to address coagulopathy include topical hemostatic dressings,[89,90] widely used in the military,[91,92] and recombinant factor VIIa.[93,94] Often cited as a concern in the use of rFVIIa, the cost of the drug has limited its use in many centers. It would seem that the optimal dose of rFVIIa is still not known. Although early studies used doses of 100–200 μg/kg after more than 10 units of PRBC,[95,96] more recent data suggest earlier administration[96] and a lower dose of VIIa (1.2 mg) is not only effective for coagulopathy[97] but is also more cost-effective than plasma, and decreases the number of blood products transfused.[96,98,99] In a 5-year retrospective chart review of coagulopathic patients with TBI, total mean charges and costs, in addition to hospital length of stay, days of mechanical ventilation, and plasma transfused, were significantly lower in the group that received rFVIIa compared with those that did not ($US 77,907 vs $108,900). There was no difference in thromboembolic complications between the groups. In another retrospective review, factor VIIa also decreased time to neurosurgical intervention (ICP monitor or craniotomy), number of units of plasma transfused, and was associated with lower mortality (33.3% in the rFVIIa group and 52.9% in controls; $P = .24$) without a difference in thromboembolic rates.[97] Administration of rVIIa should not be seen as a rescue therapy, and may be less effective when administered late in resuscitation efforts.[95] Earlier use is associated with lower rates of total blood products transfused.[94]

ORTHOPEDIC INJURIES

There is extensive debate in the trauma literature regarding timing of fraction fixation (early vs delayed)[100] and type of fixation (stabilizing vs definitive),[43,101] particularly if concurrent traumatic brain or chest injury exists.[102] Without adequate fixation, the patient cannot be mobilized; this can result in dysfunction of multiple organ systems.[103] However, overly aggressive orthopedic fixation within 24 hours of admission in multitrauma patients seems to be associated with an increased complication rate.[104] This has led to an approach that takes into account an individual patients' clinical condition (blood pressure, coagulation status, temperature, severity of injury) to guide treatment, and includes a discussion between the trauma and orthopedic surgeons, anesthesiologist, and consulting services (eg, neurosurgery).[103] Patients known to be at higher risk of perioperative complications include injury severity score greater than 40, multiple injuries in association with thoracic, abdominal or pelvic injury + shock (blood pressure <90 mm Hg), radiographic evidence of pulmonary contusion, temperature less than 35°C, and moderate-severe brain injury.[103] Pelvic fractures can be life-threatening and often require acute operative intervention for fixation or packing.[105,106] Surgery should not be delayed and damage control resuscitation may include angiographic embolization.

EXTRACORPOREAL LIFE SUPPORT

The various uses of ECLS following trauma are well known.[107–113] In addition to the cases cited earlier for ECLS following TBI, recent reports continue to illustrate the safe use of ECLS following traumatic injury. A case series by Huang and colleagues,[114] from March 2004 to October 2007, reports on 9 patients with posttraumatic ARDS who had failed conventional therapies including surgical interventions. Median time interval from trauma to ECLS was 33 hours (range 4–383 hours) and median duration of ECLS was 145 hours (range 68–456 hours). Six patients (66.7%) received additional surgeries while on ECLS; 7 patients (77.8%) were weaned and discharged. The

traumas included grade 3 or 4 liver lacerations, a grade 3 spleen laceration, and TBI. Clinicians in Germany have also had recent success with venoarterial extracorporeal membrane oxygenation in treating a series of patients with ARDS following severe chest injury in multitrauma patients.[115] One of the most exciting advances to be published recently is the use of a pumpless extracorporeal circuit, which is inherently simple, efficient, and allows for easier intrahospital and interhospital patient transport, and has the potential to allow for international transport,[116] as may be necessary to evacuate soldiers from conflict zones. The review by Flörchinger and colleagues[116] describes 10 years of experience with pumpless extracorporeal lung assist (PECLA) used in 159 patients (age range 7–78 years). Weaning was successful in 52.2% and overall survival to hospital discharge was 33.1% of patients after a mean PECLA support of 8.5 ± 6.3 days. The best outcomes were obtained in patients after trauma (n = 37).

COEXISTING DISEASES AND OUTCOME FOLLOWING TRAUMA

Increasing age has been associated with higher mortality following trauma[117,118] but until recently the role of concomitant preinjury diseases that are common with advancing age was not clear. Analysis of records of 11,142 trauma patients for a 5-year period from the trauma registry of the German Society for Trauma Surgery revealed preexisting medical conditions in 34.4% of patients.[119] Logistic regression for age-adjusted analysis showed the following preexisting conditions to be associated with increased mortality: heart disease, hepatitis/liver cirrhosis, carcinoma, obesity, and peripheral arterial occlusive disease. Previous studies have shown obesity (body mass index [BMI, calculated as weight in kilograms divided by the square of height in meters] >30 kg/m^2),[120–122] and liver disease[123–125] (independent of coagulation disturbances) to be independent risk factors for mortality. However, worse outcomes in geriatric trauma patients cannot be caused by preexisting disease solely, as the results from the German Trauma Registry confirm the independent predictive value of age in multivariable analysis. Changes in posttraumatic immune response with subsequent multiorgan failure may also be responsible for high mortality in geriatric patients.[117,126]

Table 3
Selected examples of intraoperative management of trauma-related diagnoses that vary from traditional nontrauma causes of life-threatening disorders (see text for discussion)

Medical Condition	Traditional Treatment	Trauma-Related Treatment
Systolic anterior motion	β blockade	Phenylephrine + volume
Hemorrhage	Transfusion of PRBC; fresh frozen plasma and platelets only as indicated	1:1.5:1 ratio of plasma/PRBC/platelet dose (massive) transfusion. rVIIa early
Hypotension	Volume, vasoactive agents to increase mean arterial blood pressure. Consider myocardial ischemia or exacerbation of preexisting disease (ie, CHF, valvular abnormalities)	No intervention if mean arterial blood pressure is >50 mm Hg (in absence of traumatic brain/spinal cord injury or known coronary artery disease) until hemorrhage control. Consider intraoperative TTE/TEE for traumatic cause (see Table 1)

SUMMARY

Patients with multiple severe injuries may present to the OR with little or no preoperative evaluation, and management strategies may differ from those for patients without trauma (**Table 3**). Hemorrhagic shock, gas exchange abnormalities, hemodynamic instability, and preexisting medical conditions are common. The use of damage control resuscitation, early factor replacement, and an understanding of the surgical priorities in trauma care can aid in the anesthetic management of this high-risk population.

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