Anesthesia for Trauma

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More than 100,000 deaths occur each year in the US as a direct result of trauma. Trauma is the leading overall cause of death in the age group from birth to 30 yr. Anesthesia for trauma is perhaps the highest challenge of our specialty, as we must treat critically ill patients whose history, injuries, and status are not well known to us. The surgical diagnosis is usually unknown at the time of incision, as is the nature of the procedure being undertaken. We often do not have the time to place the desired invasive monitors, and seldom have time to perform complete volume resuscitation. Nevertheless, we must anesthetize, paralyze, monitor, and resuscitate these patients while our surgical colleagues search for occult bleeding sources and remove various viscera.

In this lecture we review the basics of trauma anesthesia management, including preoperative evaluation, airway management, monitoring, induction and maintenance, fluid management, and common complications. Some new thoughts and developments in trauma therapy will be discussed.

Preoperative Evaluation and Preparation

Evaluation

The anesthesia evaluation of the acute trauma patient must always begin with the ABC: airway, breathing, circulation (not Accuse, Blame, and Criticize). Ensure these are adequate before proceeding further. Determine (with the surgeons) the nature and extent of all injuries, and obtain as much history from the patient, family, or observers as possible. History is often lacking in serious trauma patients, but much can be learned from a few simple questions (e.g., "Can you take a deep breath? Do you remember the accident?").

After the ABC part of the examination is complete, look for occult injuries (e.g., decreased breath sounds on one side, cold feet, CNS focal findings). For facial trauma, remember that maxillary fractures are often associated with basilar skull fractures and cervical spine injuries, whereas isolated mandibular fractures are usually not. For blunt chest injuries, beware of rib fractures, flail chest, cardiac or pulmonary contusion, pneumothorax, cardiac tamponade, and aortic tears or dissection (70% of these will rupture within 24 h if undiagnosed).

Preoperative investigation should include (if possible) lateral C-spine film, chest film, ECG, hematocrit(s), and urinalysis for blood. Be sure that a type and cross-match sample has been sent to the blood bank immediately. Other tests, such as CBC, coagulation profile, and electrolytes, may be ordered but should not delay emergency surgery.

Preparation, Premedication

Preoperative sedation or analgesia should not be used unless the diagnosis is certain (e.g., isolated fracture), or unless drugs are required to secure the airway. Anticholinergics to reduce secretions are acceptable if not contraindicated (e.g., tachycardia in an elderly patient). Antacids are definitely indicated if the patient is cooperative, but do not use particulate solutions such as Mylanta or Maalox. Bicitra, 15–30 mL po, 15–30 min before induction, is good aspiration prophylaxis.

In preparing the operating room, remember to warm the room to 80–85°F well in advance. Check for adequate and functioning airway equipment, drugs, fluid warmers with high flow capability, and monitors. Verify blood products availability with the blood bank before incision. When transporting to the operating room, always use supplemental oxygen, pulse oximetry, and other monitors as indicated. Bring pressor drugs with you in the unstable patient. Finally, ensure that the neck is stabilized or C-spine is cleared before moving the patient.

Airway Management

Trauma patients and all others with possible intestinal ileus must be considered to have full stomachs and, hence, are at high risk for pulmonary aspiration. Because they are often hypovolemic, they may not tolerate a "rapid-sequence induction." Furthermore, these patients may have airway or central nervous system injuries that impair their ability to breathe adequately. Their injuries or their anatomy may also make them difficult to intubate by direct laryngoscopy. Cervical

spine trauma can put them at risk for spinal cord injury during intubation. Despite the risk of aspiration or spinal cord injury, the avoidance of hypoxemia takes the highest priority at all times.

Indications for obtaining a secured airway early include poor ventilation or oxygenation, decreased or changing mental status, signs of developing airway obstruction (stridor, snoring), possible airway burns (soot in nares, singed nasal hairs), shock, or combativeness requiring sedation. Early recognition of developing obstruction requires constant attention. The patient who is apneic or obstructed requires immediate airway intervention, whereas the comatose, stable, breathing patient can afford a more deliberate approach.

If an immediate secured airway is required (apnea or obstruction), then orotracheal intubation under direct laryngoscopy is the method of first choice. Manual cervical spine stabilization should be maintained by a properly trained assistant, and cricoid pressure (Sellick's maneuver) should be applied during both intubation and mask ventilation. Although it is preferable to avoid positive-pressure mask ventilation in full-stomach patients because it increases the risk of aspiration, avoidance of hypoxia holds top priority. Proper cricoid pressure helps prevent both air insufflation into the stomach and regurgitation of stomach contents into the pharynx. Anesthetic drugs and muscle relaxants should be used only as needed.

If orotracheal intubation is not possible and an immediate airway is needed, then either transtracheal jet ventilation (TTV) or emergency cricothyroidotomy should be accomplished without delay. Using a 14-gauge IV cannula inserted through the cricoid membrane and a source of oxygen at 30 to 50 psi, TTV can maintain both adequate oxygenation and ventilation in most patients. It is an excellent way to "buy time" for establishing a more permanent, secured airway by endotracheal intubation or formal tracheostomy. A cricothyroidotomy can also be performed quickly in the apneic patient, but a surgical tracheostomy requires too much time in this situation.

In the less emergent scenario of the breathing patient who needs a secured airway, several options can be considered. Blind nasal intubation can be performed on the awake, cooperative patient, but it is rarely the method of choice in acute trauma. Stimulation of the gag reflex can cause vomiting and aspiration, and epistaxis can obscure visualization during subsequent intubation attempts. Because direct orotracheal intubation can be accomplished safely in the uncleared cervical spine using manual stabilization, there is little incentive for attempting the nasal route in these patients. Other options include fiberoptic-assisted, retrograde, and blind oral intubation using an intubating stylet. The technique selected depends on the degree of urgency, the patient's anatomy, and the nature of the injuries. Fiberoptic-assisted intubation is usually not a good choice when the pharynx is filled with blood from facial injuries.

The question of how and when to "clear" the cervical spine is important in blunt trauma patients, particularly victims of motor vehicle accidents. If the patient is apneic or in shock or has rapidly changing mental status or massive facial injuries, intubation should be performed without delay using manual stabilization. If the patient is awake and cooperative, a neurologic examination and a lateral cervical spine radiograph are indicated. If the examination is normal and the radiograph shows no injury with adequate visualization of C-7, then the cervical spine can be considered normal for purposes of airway management. If C-7 is not visualized, the radiograph can be repeated once if time permits. In the comatose or obtunded patient, the cervical spine cannot be considered cleared by single-view radiography in the absence of a neurologic examination.

Anesthetics or muscle relaxants, if needed to secure the airway, must be used with caution in trauma patients. Drugs may be required to facilitate intubation in the alert uncooperative patient, or they may be indicated to prevent dangerous increases in intracranial pressure (ICP) in the head-injury patient. If a hypnotic such as thiopental must be used in a trauma patient, the method of choice is rapid-sequence induction. Rapid-sequence minimizes the risk of aspiration, but it entails hemodynamic changes that may not be tolerated by the hypovolemic patient. A patient whose intravascular volume is depleted by 20%–30% may have a normal supine blood pressure, yet 4 to 5 mg/kg thiopental IV given to this patient may lead to cardiac arrest. Alternatives in the moderately hypovolemic patient include lower dose thiopental (1–2 mg/kg), etomidate, and ketamine. Etomidate 0.2 to 0.3 mg/kg IV decreases blood pressure less than an equipotent dose of thiopental, but, like thiopental, it is a myocardial depressant and has the additional property of adrenocortical suppression. Ketamine 1 to 2 mg/kg IV tends to maintain blood pressure by means of its sympathomimetic property, which also lends it a bronchodilating effect. However, its direct myocardial depression will be manifest in the hypovolemic patient who is already at maximum sympathetic outflow. Furthermore, ketamine is contraindicated in the head-injury patient because of its potential to increase ICP. The patient with clinical signs of shock should receive none of these hypnotic drugs. Intubation should be accomplished in this case with muscle relaxant, either alone or combined with a small dose of narcotic (e.g., fentanyl, 1–3 Φg/kg).

All drugs to facilitate intubation should be given IV, preferably via a central vein. If venous access is not available and intubation must be accomplished immediately, ketamine and succinylcholine can be given

IM. This is an undesirable alternative because absorption will be slowed in the patient who is in shock, and several minutes may elapse before acceptable intubating conditions are obtained. During this period, the airway is unprotected, and the risk of pulmonary aspiration is greatly increased.

Monitoring

All trauma patients should have minimum noninvasive monitors, including ECG, automated sphygmomanometer, stethoscope, core temperature probe (rectal or esophageal), pulse oximeter, and capnograph. Capnography is particularly useful in conjunction with arterial blood gas data, as changes in the gap between $Petco_2$ and $Paco_2$ will indicate changes in alveolar dead space, which in turn reflect changes in volume status. In addition, all but the most minor of injuries should have a Foley catheter with urometer for frequent measurement of urine output. I recommend a radial arterial catheter for all laparotomies, thoracotomies, and craniotomies, as well as peripheral injuries with significant blood loss. Of course the operation may need to begin without the arterial line if the patient is pulseless or if there is insufficient time for cannulation. The brachial and axillary arteries are also suitable for cannulation if a radial pulse cannot be palpated.

Central venous access is desirable in any case involving large blood loss or transfusion. The external or internal jugular approach is preferable, although the subclavian may also be used (with great caution–avoid pneumothorax). The femoral approach is contraindicated in the patient with potential intra-abdominal hemorrhage, but may be used for isolated limb injuries or head trauma. Central venous catheters should be of the introducer type, which allow the passage of a pulmonary artery catheter through a sheath. Pulmonary artery catheterization should be considered when there is potential for massive blood loss or transfusion, impaired left ventricular function, pulmonary edema, or sepsis. Pulmonary artery oximetry, which measures mixed venous saturation, can be helpful to evaluate the overall oxygen supply-demand relationship. Once the decision is made to place a pulmonary artery catheter, oximetry involves no increased risk and adds only $100 to the disposable costs.

Transcutaneous oxygen ($P_{tc}O_2$) is very useful in trauma patients, because it is a monitor of tissue oxygen delivery. That is, it is sensitive to both arterial oxygen content and skin perfusion, and it will reflect decreases in either. It can warn of negative trends in Pao_2 long before pulse oximetry will show any decrease in saturation, and it will also indicate decreased cardiac output or hypovolemia. Sensor temperatures of 43°C can be used on adults for more than 8 h without risk of skin burns.

Induction and Maintenance of Anesthesia

All anesthetic agents are poisons, and their therapeutic indices are generally much lower in the trauma patient. A "safe" induction dose of thiopental in a healthy patient may become lethal in the same patient after a motor vehicle accident. Selection of safe drugs and dosages is particularly difficult in the injured patient whose volume status may not be accurately known.

Although many factors must influence the choice of induction agents in the trauma patient, the most important one is usually the volume status, as follows.

A. <10% volume deficit; normotensive: Thiopental and succinylcholine by rapid sequence induction is usually safe. Maintain cricoid pressure until proper placement of endotracheal tube has been verified.
B. 10%–20% blood loss; normotensive (systolic blood pressure >100); HR <110: Ketamine (1–2 mg/kg) or etomidate (0.2–0.3 mg/kg) and succinylcholine by rapid sequence as above. Avoid ketamine in closed head injury or any potential elevation in ICP. Be aware of adrenal suppression with etomidate, although the clinical significance of this in a single dose is unclear.
C. More than 25% blood loss; hypotensive (systolic blood pressure <90) with tachycardia, respiratory distress, anuria, cold extremities: Do not use any of the above agents! These patients will "crash" with any typical anesthetic induction. If they must undergo surgery before volume resuscitation, they should be intubated quickly after succinylcholine, with or without a small dose of fentanyl (1–2 μg/kg) or midazolam (0.02–0.03 mg/kg). Remember, a survivor with recall during induction is better than a dead patient.

In any of the above scenarios, be wary of the many contraindications and side effects of succinylcholine. Despite all of its potential dangers, it is still the safest relaxant for obtaining intubating conditions rapidly in the trauma patient—including those with elevated ICP or open-eye injuries.

After anesthetic induction is complete and the airway is secured, we have not only treated the patient, we have also tested his response to drugs in a very significant way. We should therefore use the patient's response to induction to guide our choice of maintenance agents. This will range from narcotic only in the unstable, hypovolemic patient to volatile anesthetic and nitrous oxide in the very stable patient with no continuing hemorrhage. Be prepared to change the

maintenance technique at any time during the anesthetic as the patient's condition and responses change. We cannot become complacent during trauma surgery even if the patient appears to be doing well under inhalational anesthesia.

Fluid Therapy

Trauma patients are virtually always hypovolemic when we first meet them, and their blood loss is often ongoing or even worsening during anesthesia. In addition, our anesthetic agents often worsen the "functional" volume status by increasing intravascular capacity. Volume status must be continually monitored, and fluid therapy continuously varied in response to ongoing changes.

The first requirement of fluid therapy is adequate intravascular access, and in the trauma patient one can never have too much. If access cannot be obtained initially because of hypovolemia, fluids can be given through an intraosseous needle inserted into the marrow of the tibia. This method has been particularly successful in children. Central access should be established as soon as possible, subject to the considerations given above. If Military Anti-Shock Trousers are in place, they should not be deflated until fluid resuscitation is in progress and monitors are functioning.

All resuscitation fluids should be warmed to 37°C. Most trauma patients are hypothermic when they reach the hospital, and resuscitation can worsen this condition if IV fluids are not warmed. Pressurization devices should be available for large-bore IV catheters to provide rapid infusion. A satisfactory fluid-warming and infusion system should be capable of giving a unit of blood (450 mL) in 2 min.

In the trauma patient, the restoration of circulating volume is always the first priority of fluid management. It is unlikely for a previously healthy trauma patient to die acutely of anemia, but many die of hypovolemic shock. After volume status has been stabilized, the second priority is the restoration of blood oxygen-carrying capacity. Because oxygen-carrying blood substitutes are not yet commercially available, this requires the transfusion of red blood cells (RBCs). The third priority in fluid therapy is the normalization of coagulation status, which may require the transfusion of platelets, fresh-frozen plasma, or other blood components.

During initial resuscitation, the goal is adequate circulating volume. The clinician must consider the following:

(1) acute blood loss is usually underestimated;
(2) tissue losses during surgical exposure amount to 4 to 8 mL/40 kg/40 h;
(3) crystalloid replacement must be two to three times the volume of blood loss; and
(4) most anesthetics increase intravascular capacity.

These factors can yield inadequate volume replacement, which causes continued vital organ hypoperfusion, acute renal failure, worsening acidosis, and cardiovascular collapse. On the other hand, giving too much fluid can produce volume overload and mild pulmonary edema in the recovery room. In most trauma patients, it is safer to give too much fluid rather than too little; furosemide in the recovery room is preferable to cardiopulmonary resuscitation in the operating room. The first IV fluid given to trauma victims is usually crystalloid. Crystalloids can provide rapid volume replacement with balanced electrolyte solutions; they are not allergenic, immunogenic, or toxic; they quickly restore urine output, keep blood viscosity low, and present less danger of overload because they can be rapidly diuresed. On the other hand, crystalloids do not carry oxygen; they redistribute to the entire extracellular fluid volume within an hour and thereby contribute more to peripheral and pulmonary edema, can produce dilutional coagulopathies, and require larger infusion volumes than colloids (two to three times blood loss). Some studies advocated hypertonic saline as a resuscitation solution, but this technique has its own complications and is not widely accepted at present. Colloids include all blood products, high-molecular-weight dextran, albumin, and hetastarch solutions such as Hespan. Only colloids can carry oxygen or replace lost coagulation factors; they remain intravascular longer and thus create less edema, increase cardiac output more than crystalloid, and can replace that which is lost: whole blood. On the negative side, colloids can cause allergic or immune reactions; some carry risk of infection (hepatitis, HIV); they can cause electrolyte imbalances, and some have toxic effects (citrate toxicity, nephrotoxicity, cross-match problems, coagulopathy). Transfusion reactions from incompatible blood products are life-threatening complications that can occur at the worst possible time in the critical patient. The controversy between crystalloid and colloid will continue, and there are no patient outcome studies that prove either side of the argument.

Although volume loss can be corrected with either crystalloid or colloid, defects in oxygen-carrying capacity or coagulation can be corrected only by colloids. Fully crossmatched whole blood is the ideal replacement product for acute hemorrhage, but this is often not available. If partially crossmatched or O-negative blood must be used in an emergency, it may be safer to use the packed erythrocytes (PRBCs) to minimize the transfusion of plasma antibodies. When using PRBCs to improve oxygen-carrying capacity, one should not rely on an arbitrary hemoglobin threshold below which transfusion is begun. The acceptable hemoglobin value will depend on the nature of the patient's injuries or disease, general health, and the rate at which blood is being lost or is expected to

be lost. In a healthy, young patient with a traumatic amputation and no further bleeding, a hemoglobin value of 8 g/dL might be acceptable. The same patient with a rigid, swollen abdomen should be receiving transfusion at the time of incision.

The most common coagulopathy in trauma patients is dilutional thrombocytopenia, resulting from replacement of lost blood with anything other than whole blood. The platelet count should normally be kept above 70,000 mm^{-3} in the operating room. The level will remain above this value in most previously healthy patients until they have received more than one full blood volume of colloid. Platelets sequestered in the spleen and reticuloendothelial system are released during stress, thus ameliorating the decrease in platelet count caused by blood loss.

Deficiencies of fibrinogen or other coagulation factors usually appear later than thrombocytopenia, and replacement therapy should be guided by measured laboratory values such as prothrombin and partial thromboplastin time (PTT). In particular, fresh-frozen plasma should be transfused when abnormal coagulation values are measured, not according to the number of PRBC units given and never as a volume expander. During rapid, uncontrolled hemorrhage, the anesthesiologist and surgeon must anticipate specific blood component needs, allowing for the time required by the blood bank to produce the required component.

Complications

Many life-threatening intraoperative complications can occur with the major trauma patient. We shall briefly discuss only two, for which trauma patients are at particular risk. Other common complications include transfusion reactions, drug reactions, pulmonary aspiration, acute renal failure, and awareness during anesthesia.

Disseminated Intravascular Coagulation

In addition to the more common dilutional coagulopathies, trauma patients are at risk for disseminated intravascular coagulation (DIC) or "consumption coagulopathy." The hallmark of this process is generalized activation of the coagulation cascade, causing rapid consumption of platelets, fibrinogen, and Factors V and VIII. This results in diffuse, uncontrollable bleeding from the entire surgical field, possibly accompanied by diffuse intravascular thrombosis and organ ischemia. Intravascular fibrin formation activates the fibrinolytic system, causing an elevation in fibrin-split products (FSP), which in turn produces platelet dysfunction, The diagnosis of DIC is confirmed by clinical evidence of bleeding accompanied by a decreased platelet count, decreased fibrinogen level (usually <150 mg/dL), and positive fibrin-split products. Activated PTT (aPTT) is generally abnormal because of consumption of Factors V and VIII. DIC may occur without the presence of elevated FSP because these are rapidly removed from circulation by the liver.

DIC is apparently triggered by a phospholipid not normally present in the circulation: platelet Factor III, or tissue thromboplastin. Possible causes of DIC are listed according to category in Table 1. As long as phospholipid triggers circulate, the DIC process will continue. Therapy is therefore first aimed at the underlying cause; this is easier said than done. In serious trauma or burns, the damaged vascular endothelium often cannot be removed. Step two in therapy is replacement of consumed blood constituents, primarily platelets, fibrinogen, and Factors V and VIII. This requires transfusion of platelet concentrates, fresh-frozen plasma, and occasionally cryoprecipitate. The goal of replacement therapy is to maintain a platelet count of at least 100,000, a normal aPTT, and a fibrinogen level of at least 200 mg/dL. The third possible step in therapy, the use of heparin, is reserved for disorders of the coagulation cascade and is not appropriate in surgical patients.

Hypothermia

Hypothermia is almost unavoidable in trauma patients because most are hypothermic before they reach the operating room, and anesthesia further compromises their thermoregulatory mechanisms. General anesthesia lowers the threshold temperature at which hypothermic corrective responses begin. Anesthesia also reduces cutaneous vasoconstriction, which normally helps to conserve heat, and muscle relaxants block the shivering response.

Hypothermia has many physiologic effects, some of which may be beneficial in the critically ill. Metabolism decreases by 8% per °C, and reaches one half of normal values at 28°C. Oxygen demand is reduced correspondingly so that low organ perfusion may be better tolerated. Blood flow is also shifted away from the extremities and muscles, with a larger fraction perfusing the heart and brain. Decreased cardiac output accompanied by sinus bradycardia may have a beneficial effect on the myocardial oxygen supply-demand balance.

These potential benefits of hypothermia are generally outweighed by its deleterious effects in critically ill patients undergoing surgery. In addition to sinus bradycardia, the ECG may show atrial fibrillation, prolonged PR and QT intervals, and widened QRS. Below 28°C, more serious dysrhythmias ensue, including nodal rhythm, frequent premature ventricular contractions, atrioventricular block, and refractory ventricular fibrillation. Increased sympathetic tone can cause heart rate and blood pressure to increase

Table 1. Possible Causes of Disseminated Intravascular Coagulation

Disorders of Vascular Integrity
Burns
Vasculitis
Dissecting aortic aneurysm
Circulating Immune Complexes
Sepsis
Toxemia of pregnancy
Immune complex disease processes
Release of Tissue Phospholipids into Circulation
Massive crush injuries
Necrotic malignant tissue
Massive hemolysis
Obstetric complications
Amniotic fluid embolism
Fetal demise
Abruptio placentae

All causes involve a loss of localization of the hemostatic process. <ñ

under light levels of anesthesia. Myocardial contractility increases as temperature falls, reaching its maximum at 28°C. Hypoxic pulmonary vasoconstriction is attenuated by hypothermia and at 30°C is less than one half of normal response. Plasma volume is decreased because of an apparent transcapillary fluid shift, thus worsening preexisting hypovolemia. Blood viscosity is increased by both the direct effect of temperature and hemoconcentration as a result of decreased plasma volume. Of special concern in trauma patients, hypothermia causes platelet dysfunction and thrombocytopenia, with the latter resulting from platelet sequestration in the liver. Both are usually reversible by rewarming. Effects of hypothermia on other coagulation factors are unclear, but severe hypothermia is a possible cause of DIC.

Given the dangers of accidental hypothermia, particularly in trauma patients, one must make every attempt to prevent or correct it. The operating room should be kept warm, respired gases should be both warmed and humidified, all IV replacement fluids and irrigation should be warmed to 37°C, and a warming blanket should be placed between the patient and the operating table. Air-filled patient warmers, such as the Bair Hugger, may also be helpful.

Conclusions

Trauma patients often have little or no unused reserves in cardiopulmonary function. The additional stress caused by general or even regional anesthesia may be more than these patients can tolerate. To minimize anesthetic risk in the trauma victim, the anesthesiologist must know as much as possible about the injuries, resuscitation status, and any co-existing diseases the patient may have. He must understand the exact surgical procedure and its attendant risks and stresses. The anesthetic plan must account for these factors both in the drugs and the monitoring used for the case. Fluid management is especially challenging because of rapid, unpredictable changes in volume status and incomplete preoperative resuscitation. Possible complications must be anticipated, and the appropriate therapeutic options (e.g., blood products) should be made available before beginning the procedure. Above all, the anesthesiologist must maintain a high index of suspicion and avoid complacency, even when the patient appears stable.