Anesthesia for adult trauma patients

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INTRODUCTION — Although the most critically injured patients are ideally transported to a designated trauma center, anesthesiologists in other hospitals may provide care for a patient who requires immediate surgical or other interventions after traumatic injury.

This topic reviews anesthetic management of adult trauma patients. Other topics address immediate management of trauma patients upon arrival to the emergency department (ED) and initial decisions regarding diagnostic, surgical, and other interventions:

●(See "Initial management of trauma in adults".)

●(See "Initial evaluation of shock in the adult trauma patient and management of NON-hemorrhagic shock".)

●(See "Overview of damage control surgery and resuscitation in patients sustaining severe injury".)

GENERAL APPROACH — A clear, simple, and organized approach to the trauma patient is used in both the emergency department (ED) and operating room (OR), including assessment of airway, breathing, circulation, disability (eg, neurologic evaluation and cervical spine stabilization), and exposure (eg, hypothermia, smoke inhalation, intoxicants) [1]. An example is the Advanced Trauma Life Support (ATLS) tool. Participation of the anesthesiologist at an early stage (eg, at the time of trauma response activation or patient arrival in the ED) provides continuity of care before and after transition to the OR [2].
(See "Initial management of trauma in adults", section on 'Primary evaluation and management'.)

Goals — Primary goals in both the ED and the OR include:

●Airway management. (See 'Airway management' below.)

●Management of hemodynamic instability. This includes management of hemorrhagic hypovolemic shock and its sequelae (eg, coagulopathy, hemodilution, hypothermia, and electrolyte and acid-base

derangements), as well as other etiologies of shock after trauma. (See 'Management of hemodynamic instability' below.)

●Lung-protective ventilation. (See 'Lung-protective ventilation' below.)

●Maintenance of normothermia. (See 'Temperature management' below.)

●Maintenance of adequate cerebral blood flow, oxygenation, and ventilation is prudent to avoid secondary brain injury. Even in the absence of overt evidence of traumatic brain injury (TBI), concussion is common in trauma patients and may be associated with significant changes in cerebral hemodynamics and metabolism [3,4]. (See "Anesthesia for patients with acute traumatic brain injury", section on 'Goals for anesthetic management'.)

●Prevention of unpleasant experiences during painful interventions (eg, by employing local or regional anesthesia, sedation, or general anesthesia). (See 'Management of general anesthesia' below.)

Cognitive aids

Checklists — Checklists are often used as a cognitive aid to guide the anesthesiology team during emergency preparations for intraoperative resuscitative care of the trauma patient. An example is noted in the table (table 1) [5,6]. (See "Operating room hazards and approaches to improve patient safety", section on 'Checklists'.)

Cognitive aids for handoffs — Cognitive aids are helpful during critically important handoffs from the ED to the OR (or interventional radiology [IR] suite), and subsequently to the intensive care unit (ICU) (table 2). The ABCDE mnemonic is an example to guide communication specific for handoff of trauma patients, in which A = **A**irway; B = **B**reathing; C = **C**irculation; D = **D**isabilities or **D**rugs; and E = **E**xposure (eg, hypothermia, smoke, intoxicants), **E**xtremity injuries, and **E**verything else (eg, name, date of birth, blood type, allergies, and medical history [if known]) (table 3). (See "Operating room hazards and approaches to improve patient safety", section on 'Formal handoff procedures'.)

PATIENT STABILIZATION

Airway management — Initial airway management for trauma patients by emergency department (ED) physicians is discussed in other topics for specific types of airway injury:

●(See "Advanced emergency airway management in adults".)

●(See "Emergency airway management in the adult with direct airway trauma".)

●(See "Management of the difficult airway for general anesthesia in adults".)

●(See "Anesthesia for burn patients", section on 'Airway management'.)

Urgent airway management in trauma patients may be challenging due to maxillofacial injury or burns, blunt or penetrating neck injury, laryngeal or major bronchial disruption, cervical spine instability, compression of the airway, bleeding due to the initial traumatic injury or multiple subsequent intubation attempts that impair direct visualization of the upper airway, or oropharyngeal and/or laryngeal edema due to burn injury. These acute injuries may create a "difficult airway," or may worsen a pre-existing anatomical predisposition to a difficult airway. The American Society of Anesthesiologists Committee on

Trauma and Emergency Preparedness has developed guidance for difficult airway management in trauma patients (algorithm 1) [7].

A clearly defined, sequential approach to a patient with airway injury or abnormality is critical, since preoxygenation may be difficult and any delay in securing the airway may lead to rapidly progressing hypoxemia. Also, prolonged efforts to secure the airway may delay definitive treatment of other life-threatening injuries [8]. Details regarding management of a difficult airway in specific trauma conditions (eg, airway disruption, oral and maxillofacial trauma, airway compression, closed head injury) are described in the tables (table 4 and table 5 and table 6 and table 7) [7]. Management in patients who may have a cervical spine injury is discussed in another topic (figure 1). (See "Anesthesia for adults with acute spinal cord injury", section on 'Airway management'.)

In a patient with life-threatening injuries or hypoxemia, inability to obtain a definitive airway is an absolute indication for emergency cricothyroidotomy or surgical tracheostomy, particularly if a "cannot ventilate, cannot intubate" scenario develops [9]. If airway injury is extensive, a joint decision to place a surgical airway distal to the site of injury may be made by the anesthesiologist and the ED physician and/or trauma surgeon. Factors influencing this decision include the specific airway injury, presence of other traumatic injuries, the patient's overall condition, clinician expertise, and types of immediately available airway equipment. (See "Emergency cricothyrotomy (cricothyroidotomy)".)

In stable patients without airway compromise, conservative airway management may be suitable. In one review, immediate establishment of a definitive airway was necessary in approximately 50 percent of patients with penetrating trauma and in 80 percent of those with blunt trauma [9]. In another review, approximately one-third of traumatized patients did not require immediate endotracheal intubation in the ED, but were instead intubated after transport to the operating room (OR) [10].

Monitoring and intravenous access — An intra-arterial catheter and a central venous catheter (CVC) are inserted in most hemodynamically unstable trauma patients undergoing general anesthesia, if not previously inserted in the ED. Two large-bore peripheral intravenous (IV) catheters (eg, 16 G or larger) can be rapidly inserted instead of or in addition to a CVC for initial administration of fluid, blood transfusions, and IV vasoactive and anesthetic agents. Although all intravascular catheters are ideally inserted before anesthetic induction, placement should not unduly delay emergency surgical intervention. If obtaining reliable IV access is difficult, intraosseous (IO) access can be rapidly and reliably achieved, and can be used for (blood and fluid) resuscitation and to administer medications (see "Intraosseous infusion") [11,12]. Additional considerations for intraoperative monitoring are discussed separately. (See "Intraoperative management of shock in adults", section on 'Intraoperative monitoring'.)

Management of hemodynamic instability — Initial resuscitation efforts in a hemodynamically unstable trauma patient may occur in the ED, interventional radiology (IR) suite, and/or OR [13]. The goal is to prevent organ damage by restoring tissue perfusion pressure, normal oxygen delivery, and adequate microcirculatory flow [14]. (See "Intraoperative management of shock in adults", section on 'Initial resuscitation' and "Initial evaluation of shock in the adult trauma patient and management of NON-hemorrhagic shock".)

Treatment of hemorrhagic shock

●**General principles** – An actively bleeding trauma patient is supported with damage control resuscitation (DCR) until hemorrhage can be arrested [15-19]. In addition to early surgical control of hemorrhage, initial strategies to limit ongoing blood loss include maintenance of a low to normal systolic blood pressure (BP) at approximately 90 mmHg (or ≤110 mmHg in older adults) and/or mean arterial pressure (MAP) at 50 to 65 mmHg. Once hemostasis has been achieved, higher BP values are targeted (eg, systolic BP ≥90 mmHg and/or MAP ≥65 mmHg). Although increasing BP indicates increasing macro-circulatory pressure, micro-circulatory flow may still be abnormal. (See 'High-dose opioid supplementation' below and "Initial management of trauma in adults", section on 'Circulation'.)

●**Administration of fluid and blood products** – Fluid administration is limited by employing dynamic parameters to assess intravascular volume status and guide fluid administration in the OR (eg, transesophageal echocardiography [TEE] to assess changes in left ventricular cavity size (movie 1) or respirophasic variation in the intra-arterial pressure waveform during positive pressure ventilation (table 8 and figure 2 and figure 3) [20-22]. Our approach combines crystalloids and colloids to replace blood loss until blood is available for transfusion. (See "Intraoperative management of shock in adults", section on 'Hypovolemic shock management'.)

For patients with severe or ongoing hemorrhage, red blood cells (RBCs) and other appropriate blood products are transfused as soon as they are available, rather than continuing administration of crystalloid or colloid [23]. Current ATLS guidelines recommend no more than 1 L of warm 0.9% saline prior to administration of blood components [1]. Availability should not rely on a full crossmatch in patients with hemorrhagic shock since uncrossmatched blood can be administered until crossmatched blood is available. A ratio of 1:1:1 or 2:1:1 (RBCs: plasma: platelet packs) is targeted for blood product transfusion [24-26]. Although this ratio mirrors the content of whole blood, superior viscoelastic maximal clot formation is achieved with transfusion of whole blood compared with 1:1:1 component transfusion [27]. For this reason, fresh whole blood has been used in the military for combat injuries, and some institutions have developed protocols for its use in civilian trauma [28-30].

Fibrinogen supplementation by administration of cryoprecipitate or fibrinogen concentrate may improve outcomes following major trauma, particularly if low fibrinogen levels are documented or strongly suspected [31-36]. The guidelines of the European Society of Anaesthesiology (ESA) and the European Task Force for Advanced Bleeding Care in Trauma suggest a target fibrinogen concentration >150 to 200 mg/dL [35,36]. Proponents argue that baseline fibrinogen concentrations are relatively low and there are no fibrinogen stores to be mobilized; thus, fibrinogen is the first procoagulant to become critically low in a hemorrhaging patient [37]. Low fibrinogen concentration <100 mg/dL or fibrinolysis evident on point-of-care laboratory tests is generally treated with cryoprecipitate or fibrinogen concentrate. (See "Acute coagulopathy associated with trauma" and "Intraoperative transfusion of blood products in adults", section on 'Indications and risks for specific blood products'.)

Information rapidly derived from intraoperative laboratory tests allows rational decision-making regarding transfusion of RBCs and other blood components. Point-of-care (POC) tests of hemostatic function allow rapid assessment of causes of coagulopathy and responses to interventions, including transfusion of blood products. The most commonly used POC tests for overall hemostatic function are thromboelastography (TEG) and an adaptation of TEG known as rotational thromboelastometry (ROTEM) [38-40]. (See "Acute coagulopathy associated with trauma", section on

'Thromboelastography' and "Intraoperative transfusion of blood products in adults", section on 'Intraoperative diagnostic testing'.)

An intraoperative blood salvage system is often used [41]. In a 2015 systematic review of patients undergoing emergency abdominal or thoracic trauma surgery (one trial; n = 44), the reduction in the use of allogeneic red blood cells in the cell salvage group was 4.7 units (95% CI 1.31-8.09 units), compared with controls [42]. (See "Surgical blood conservation: Blood salvage".)

It is critically important to warm all IV fluids and blood in order to maintain normothermia and avoid hypothermia-induced exacerbation of coagulopathy. (See 'Temperature management' below.)

●**Management of coagulopathy** – Reversal of anticoagulation and control of coagulopathy are critically important, particularly in a patient with traumatic brain injury [43]. Acute coagulopathy after severe traumatic injury has multifactorial etiologies including acidosis related to tissue injury and shock, hypothermia related to exposure and fluid administration, systemic anticoagulation with activation of Protein C and Protein S, hyperfibrinolysis from amplification of tissue plasminogen activator, platelet dysfunction following platelet activation, hemodilution due to fluid or component blood product administration, consumption of clotting factors manifesting as disseminated intravascular coagulation (DIC), and other biochemical processes [44,45]. (See "Acute coagulopathy associated with trauma", section on 'Etiology'.)

Management of coagulopathy is guided by POC tests, such as TEG or ROTEM, if available [33,34,46-50]. Turnaround is rapid with these tests, and a single tracing result provides information regarding clot initiation, kinetics of clot formation, clot strength, and fibrinolysis (figure 4 and figure 5 and table 9). (See "Acute coagulopathy associated with trauma", section on 'Thromboelastography'.).

In severely injured trauma patients, onset of hyperfibrinolysis occurs rapidly; thus, antifibrinolytic therapy (typically tranexamic acid [TXA]), is administered to trauma patients when hyperfibrinolysis is noted on POC testing, and to patients with active hemorrhage if TEG or ROTEM is unavailable [51-54]. TXA is administered as an initial 1 g IV bolus over 10 minutes with TEG-guided determination of further dosing, or followed by 1 g infusion over 8 hours if TEG is unavailable. TXA is part of "massive transfusion protocols" in most major trauma centers in the United States and in the United States military. (See "Initial management of moderate to severe hemorrhage in the adult trauma patient", section on 'Antifibrinolytic agents'.)

However, evidence suggests that there are several pathological forms of fibrinolysis after severe trauma: fibrinolysis shutdown (54 percent), hyperfibrinolysis (18 percent), and physiologic fibrinolysis (18 percent) [55,56]. Fibrinolysis shutdown is associated with a fivefold increase in mortality [57]. Some investigators caution that trauma patients should be carefully selected for TXA administration since fibrinolysis is a natural process that enables clot degradation and maintains patency of the microvasculature [58]. Exogenous inhibition of the fibrinolysis system may have an adverse effect on survival and should be guided by TEG or ROTEM. Two retrospective analyses of civilian data in severely injured patients who received TXA suggest increased mortality [59] or no benefit [60]. (See "Acute coagulopathy associated with trauma", section on 'Alterations in fibrinolysis'.)

Assessment for other causes of shock — In addition to hemorrhagic shock, a trauma patient may have other known or unrecognized causes of shock. Examples include spinal cord injury causing neurogenic

(ie, vasoplegic) shock (see "Intraoperative management of shock in adults", section on 'Neurogenic shock'), severe ischemic myocardial dysfunction causing cardiogenic shock (see "Intraoperative management of shock in adults", section on 'Cardiogenic shock management'), or tension pneumothorax, pericardial tamponade, or increased intra-abdominal pressure causing obstructive shock. (See "Intraoperative management of shock in adults", section on 'Obstructive shock management'.)

Point-of-care ultrasound (eg, the focused assessment with sonography for trauma [FAST] examination) is the standard screening examination performed by ED or other clinicians to diagnose common life-threatening injuries that may otherwise be undetected in trauma patients [61]. FAST involves assessments of the pericardium to look for hemopericardium and tamponade, and of the right flank, left flank, and pelvis to look for intraperitoneal free fluid, often with an extended evaluation looking for pneumothorax (E-FAST). (See "Emergency ultrasound in adults with abdominal and thoracic trauma".)

Ongoing resuscitation — After control of acute hemorrhage, ongoing intraoperative resuscitation includes reestablishment of normothermia and continuing assessment and treatment of coagulopathy, hypothermia, electrolyte abnormalities, elevated serum lactate level, and acid-base derangements in order to maintain hemodynamic stability [26,43,62]. Correction of metabolic acidosis is initially accomplished with adequate fluid resuscitation rather than with administration of sodium bicarbonate [63]. Continuous infusion of a vasopressor or inotropic agent may be necessary to maintain blood pressure and restore adequate tissue perfusion (table 10). (See "Intraoperative management of shock in adults", section on 'Initial interventions'.)

Lung-protective ventilation — An intraoperative lung-protective strategy is used during controlled ventilation for patients with trauma and shock [64-68]. (See "Anesthesia for open abdominal aortic surgery", section on 'Ventilation management'and "Ventilator-induced lung injury".)

Either a volume- or pressure-limited ventilation mode may be used with:

●Low tidal volumes of 6 to 8 mL/kg predicted body weight. The incidence of pulmonary complications and other adverse outcomes are lower in patients receiving such low tidal volumes compared to higher tidal volumes [67-69].

●Respiratory rate (RR) at 8 to 10 breaths/minute, with adequate expiratory time to reduce air trapping (ie, inspiratory-to-expiratory [I:E] ratio of 1:3). Mild permissive hypercapnia (eg, partial pressure of arterial carbon dioxide [$PaCO_2$] 40 to 45 mmHg) is allowed, unless the patient has metabolic acidosis or known or suspected traumatic brain injury (TBI). In such cases, a faster RR may be temporarily employed to achieve a $PaCO_2$ of 30 to 35 mmHg, in order to compensate for metabolic acidosis and/or decrease intracranial pressure (ICP). (See "Anesthesia for patients with acute traumatic brain injury", section on 'Intraoperative ventilation and oxygenation'.)

●Maintenance of a low plateau pressure ≤30 cmH_2O.

●Adjustment of the fraction of inspired O_2 (FiO_2) adjusted to maintain O_2 saturation ≥92 percent.

●Initial positive end-expiratory pressure (PEEP) at 0 cmH_2O until hemodynamic stability and control of hemorrhage and adequate resuscitation has been achieved. Subsequently, PEEP may be slowly and incrementally increased to 5 to 10 cmH_2O if tolerated without provoking hypotension, and FiO_2 is

concurrently weaned to maintain arterial saturation >90 percent. The goal is to provide an optimal balance between minimizing lung injury and preventing hemodynamic instability. In patients with hemorrhagic shock, it is particularly important to avoid high levels of PEEP and dynamic hyperinflation with development of auto-PEEP [70]. PEEP and auto-PEEP increase intrathoracic pressure, and decrease venous return, cardiac output, and systemic BP. (See "Physiologic and pathophysiologic consequences of mechanical ventilation", section on 'Hemodynamics'.)

Temperature management — Perioperative temperature management is accomplished with warming devices to maintain normothermia (temperature ≥35.5°C) in patients with trauma and shock, as discussed separately. (See "Intraoperative management of shock in adults", section on 'Temperature management'.)

MANAGEMENT OF GENERAL ANESTHESIA

General principles — Anesthetic induction and maintenance agents with minimal hemodynamic effects are selected, and doses are reduced and carefully titrated to avoid exacerbation of hypotension [71]. Patients with barely compensated or decompensated hemorrhagic shock have a lower volume of distribution for all anesthetic agents. Even after hemodynamic stability has been achieved, careful titration is necessary since the patient's clinical condition may rapidly change. For example, a trauma patient may have unrecognized bleeding into the retroperitoneum after a severe pelvic injury, or into muscle and fascial compartments after bilateral femur fractures.

Induction — The goal of induction of general anesthesia is to produce an unconscious state while maintaining adequate organ perfusion. However, induction may result in profound hypotension and/or cardiac arrest in a patient with barely compensated or decompensated hemorrhagic shock. Before beginning induction, a vasopressor infusion should be connected "in line" in the IV tubing so that it is ready for immediate administration (table 10). In a hemodynamically unstable patient, we administer a bolus dose of a vasopressor concurrently with the induction agents to prevent exacerbation of hypotension. (See "Intraoperative management of shock in adults", section on 'Induction'.)

For most trauma patients, rapid sequence induction and intubation (RSII) is indicated (see "Rapid sequence induction and intubation (RSII) for anesthesia"). Either etomidate or ketamine is typically selected as the primary induction agent for a hemodynamically unstable patient. Propofol is generally avoided since administration of an intravenous (IV) bolus may further reduce blood pressure (BP) by causing dose-dependent venous and arterial dilation and decreased contractility. However, in a hemodynamically stable trauma patient, a reduced dose of propofol 0.1 to 0.5 mg/kg may be administered. Adjuvant induction agents (eg, opioids, lidocaine, midazolam) are eliminated in hemodynamically unstable patients, or reduced if hemodynamic stability has been achieved (table 11). (See "Rapid sequence induction and intubation (RSII) for anesthesia" and "Intraoperative management of shock in adults", section on 'Induction'.)

We typically select succinylcholine as the neuromuscular blocking agent (NMBA) for RSII, administered at a dose of 1.5 mg/kg IV (or 3 to 4 mg/kg intramuscularly [IM] if IV access is not available) (table 12). This dose of succinylcholine offers the advantages of swift onset (<60 seconds), excellent intubating conditions, and brief duration of action (5 to 10 minutes) [72]. A large dose of rocuronium (eg, 1.2 mg/kg) is a reasonable alternative NMBA for RSII, particularly if sugammadex is immediately

available [73]. Compared with succinylcholine, onset of excellent intubating conditions is only slightly longer after an RSII dose of rocuronium (90 to 120 seconds), although duration of action is much longer (60 to 80 minutes) (table 12). Thus, if unexpected difficulty with the airway is encountered (eg, inability to intubate or ventilate), sugammadex is administered to rapidly reverse rocuronium effects.
(See "Rapid sequence induction and intubation (RSII) for anesthesia", section on 'Nondepolarizing NMBAs'.)

Maintenance

Inhalation anesthetic agents

●**Volatile inhalation agents** – A volatile inhalation anesthetic agent (eg, desflurane, isoflurane, sevoflurane) is typically selected for maintenance of anesthesia. Administration is initiated at a lower concentration than in healthy patients due to dose-dependent cardiovascular effects of the volatile anesthetic agents. Subsequently, the agent is carefully titrated to maintain anesthesia while avoiding hypotension that may further decrease end-organ perfusion. In patients with multiple injuries or multiple episodes of severe hemodynamic instability, agents with a low blood-gas partition coefficient (eg, desflurane, sevoflurane) are preferred to permit rapid titration. If systolic BP improves to ≥90 mmHg, the selected volatile agent may be increased to ≥0.5 minimum alveolar concentration (MAC) (table 13). (See "General anesthesia: Maintenance", section on 'Inhalation anesthesia'.)

In patients with multiple traumatic injuries that may include brain injury, the volatile agent is maintained ≤1 MAC to avoid dose-dependent increases in cerebral blood flow (CBF) and intracranial pressure (ICP). (See "Anesthesia for patients with acute traumatic brain injury", section on 'Choice of anesthetic agents'.)

Although volatile inhalation anesthetics are effective modulators of the inflammatory response after tissue injury and may have beneficial effects on organ function in humans and animal models, studies have focused on ischemia-reperfusion injury and biomarkers of organ dysfunction rather than on clinical outcomes [74-81].

●Nitrous oxide **gas** – We generally avoid nitrous oxide (N_2O) in trauma patients for several reasons [82-84] (see "Inhalation anesthetic agents: Clinical effects and uses", section on 'Nitrous oxide gas'):

•N_2O expands all gas spaces and can worsen a traumatic pneumothorax or pneumocephalus.

•In patients with TBI, N_2O may increase the cerebral metabolic rate of O_2 consumption ($CMRO_2$), and may also increase ICP.

•N_2O increases pulmonary vascular resistance and may mask myocardial depression.

•N_2O may cause apoptosis and altered immunologic responses to infection.

High-dose opioid supplementation — When systolic BP is consistently maintained ≥90 mmHg and surgical hemostasis is assured, we add doses of fentanyl during the maintenance phase of anesthesia, particularly if the patient will remain intubated and sedated with controlled ventilation in the immediate postoperative period. Fentanyl may cause beneficial dilation of the microcirculation and has minimal myocardial depressant effects [85-87].

Initially, we administer 50 to 150 mcg bolus doses of fentanyl, with close monitoring of the hemodynamic response. Additional resuscitation may be necessary during fentanyl administration (eg, additional volume or vasopressor administration). If systolic BP is maintained ≥90 mmHg, fentanyl dosing is incrementally increased until the patient tolerates a single bolus of approximately 250 mcg. Use of this high-dose opioid technique typically results in administration of a total fentanyl dose of 10 to 30 mcg/kg during the surgical procedure. However, plasma fentanyl levels and total dose vary considerably if the patient's blood volume is constantly changing due to ongoing bleeding and transfusion.

If evidence of tissue hypoperfusion (eg, elevated lactate concentration and/or base deficit) persists after these relatively high doses of fentanyl, we add another opioid to achieve additional vasodilatation. We typically select methadone as the second-line opioid when the patient's electrocardiogram (ECG) reveals a normal QT interval (<440 ms). Methadone is administered in 10 mg IV increments to a total dose of 20 to 30 mg. Hydromorphone is an alternative opioid, titrated in 0.2 to 0.4 mg increments to a total dose of approximately 2 mg. Morphine is generally avoided due to concern regarding histamine release, which may exacerbate hypotension.

Strategies to minimize risk of awareness — Since it may be unsafe to administer sufficient anesthesia during all phases of damage control surgery and other interventions, trauma patients are at risk for intraoperative awareness with postoperative recall. (See 'Damage control surgery' below and "Awareness with recall following general anesthesia", section on 'Risk factors: Anesthetic underdosing'.)

We employ neuromonitoring with processed or unprocessed electroencephalography (EEG), with alarms set to detect high EEG indices indicating possible awareness (eg, bispectral index [BIS] value >60). Although such neuromonitoring does not reliably confirm lack of awareness, this risk may be minimized during periods when anesthetic underdosing is necessary because of hemodynamic instability. (See "Awareness with recall following general anesthesia", section on 'Neuromonitoring'.)

We administer incremental doses of one or more adjuvant agents during periods of light anesthetic depth to potentially limit the traumatic effect of an intraoperative awareness event, particularly if the patient is hemodynamically unstable and unable to tolerate a volatile anesthetic agent [88]. We typically administer a benzodiazepine (eg, midazolam 1 to 4 mg or diazepam2 to 10 mg) to produce amnesia [89], and/or an opioid to decrease pain. (See 'High-dose opioid supplementation' above and "Awareness with recall following general anesthesia", section on 'Adjuvant medications'.)

Scopolamine is an anticholinergic amnestic that has been used to prevent intraoperative awareness in hemodynamically unstable patients, although data regarding dosing and effectiveness are lacking [90]. However, IV preparations of scopolamine are no longer available in the United States. In countries where IV scopolamine is available, it is avoided in patients with traumatic brain injury because it has a long half-life (4.5 hours); thus, subsequent neurologic examinations are confounded by its side effect of pupillary dilation.

POSTOPERATIVE CONSIDERATIONS — After emergency trauma surgery, most patients remain intubated and sedated with controlled ventilation (see 'High-dose opioid supplementation' above). The anesthesiologist should continuously monitor the electrocardiogram (ECG), pulse oximetry (SaO_2), and

intra-arterial blood pressure during transport to the intensive care unit (ICU) [91]. (See "Intraoperative management of shock in adults", section on 'Transport to the intensive care unit'.)

Upon arrival in the ICU, a clear, simple, and organized handoff is critically important. We prefer to use a cognitive aid such as the ABCDE communication tool (table 3). (See "Operating room hazards and approaches to improve patient safety", section on 'Formal handoff procedures'.)

Reassessment of the extent of unresolved shock is necessary shortly after arrival in the ICU. Ongoing resuscitation and management of respiratory, cardiovascular, metabolic, and immunologic consequences of traumatic injury and massive transfusion may be necessary [92]. Most trauma patients require controlled ventilation and hemodynamic support, and many require correction of critical acid-base and electrolyte abnormalities, restoration of normothermia, or efforts to minimize secondary central nervous system injury. Frequent postoperative reassessments for the possibility of missed injuries or inadequately treated pain are important after surgery for traumatic injuries. (See 'Assessment for other causes of shock' above and "Overview of inpatient management of the adult trauma patient", section on 'Consider other potential injuries'.)

SPECIAL POPULATIONS — Unique anesthetic considerations exist for certain injury-specific or patient–specific situations.

Damage control surgery — Damage control surgery may be necessary for immediately life-threatening traumatic injuries, with planned delay of definitive management of these and other non-life-threatening injuries until after appropriate resuscitation. The goals of damage control surgery are to first arrest hemorrhage and then to limit contamination (eg, due to gastrointestinal tract injury). Blood flow to the vital organs and extremities must be maintained, using temporary shunts if necessary. Indications and surgical techniques for specific damage control procedures are discussed separately. (See "Overview of damage control surgery and resuscitation in patients sustaining severe injury".)

Resuscitative endovascular balloon occlusion of the aorta — In selected patients (eg, those with non-compressible torso hemorrhage following traumatic injury), resuscitative endovascular balloon occlusion of the aorta (REBOA) is a temporizing measure to support vital organ perfusion, decrease the amount of bleeding distal to the occluded site, and provide a window of opportunity for resuscitation and definitive hemorrhage control (figure 6) [93,94]. However, REBOA does not provide definitive hemorrhage control. REBOA indications and techniques are discussed separately. (See "Endovascular methods for aortic control in trauma".)

Anesthetic management during REBOA includes insertion of an intra-arterial catheter and a central venous catheter (CVC) (see 'Monitoring and intravenous access' above). The intra-arterial catheter is placed in an upper extremity since perfusion to the lower extremity arteries will be temporarily interrupted during balloon occlusion of the aorta. Similar to intraoperative monitoring during endovascular aortic repair, transesophageal echocardiography (TEE) is particularly useful to assess changes in regional and global ventricular function as well as intravascular volume status before, during, and after balloon occlusion [94,95]. TEE can also be used to monitor position of the endovascular balloon [93]. (See "Anesthesia for endovascular aortic repair", section on 'Transesophageal echocardiography'.)

During REBOA, critical hemodynamic changes occur with balloon inflation and deflation [93-95]:

●**Inflation** – Similar to application of an aortic crossclamp during abdominal aortic aneurysm (AAA) repair, proximal aortic occlusion during REBOA increases systemic vascular resistance (SVR), blood pressure (BP), and cardiac afterload, thereby increasing cerebral and myocardial perfusion (figure 6) [94,95]. Physiologically, the increased afterload, while supporting coronary perfusion, may also increase myocardial transmural wall tension and cardiac pressure work (figure 7) [96,97]. Although published recommendations for anesthetic management in this setting are lacking, careful increases in volatile inhalation anesthetic concentration to produce some degree of vasodilation is prudent if systolic BP is higher than desired during proximal aortic occlusion. (See "Anesthesia for open abdominal aortic surgery", section on 'Management of aortic cross-clamping' and "Endovascular methods for aortic control in trauma", section on 'Inflate the balloon catheter'.)

●**Deflation** – REBOA balloon deflation is attempted when hemostasis has been achieved or to check for sources of ongoing hemorrhage [93,95]. Similar to aortic unclamping during open AAA repair, deflation of the intra-aortic balloon catheter may result in severe hypotension due to a sudden decrease in SVR, decreased preload due to venodilation, hypoxia-mediated reactive hyperemia, and decreased myocardial contractility due to metabolic (lactic) acidosis (figure 8) [93,95,97,98]. Metabolic acidosis and washout of ischemic muscle tissue may also result in hyperkalemia, malignant arrhythmias, and cardiac arrest. (See "Anesthesia for open abdominal aortic surgery", section on 'Management of aortic unclamping'.)

Clear team communication is required in preparation for deflation [93-95]. In some cases, it is clinically necessary for the surgeon to transiently, partially, or gradually deflate the balloon to permit reperfusion between occlusion periods, or to allow the anesthesiologist to increase intravascular volume and add vasopressor and/or inotropic agents as needed to avoid precipitous cardiovascular collapse after full balloon deflation. (See "Endovascular methods for aortic control in trauma", section on 'Deflate the balloon'.)

Following balloon deflation, metabolic derangements are typically present during the period of reperfusion (eg, hypoxemia, hypercarbia, acidosis, hyperkalemia, anemia, disorders of hemostasis), similar to reperfusion after aortic surgery [93-95,98]. In addition to obtaining standard point-of-care laboratory tests, serum lactate is monitored to assess successful reversal of shock as intraoperative resuscitation is completed [62,99]. (See "Anesthesia for open abdominal aortic surgery", section on 'Point-of-care testing'.)

Acute traumatic brain injury — Anesthetic management of patients with acute traumatic brain injury (TBI) is summarized in the table and is discussed separately (table 14). (See "Anesthesia for patients with acute traumatic brain injury".)

Acute traumatic spinal cord injury — Anesthetic management of acute spinal cord injury is discussed separately. (See "Anesthesia for adults with acute spinal cord injury" and "Acute traumatic spinal cord injury".)

Traumatic injury in pregnant patients — Every female trauma victim of reproductive age should be considered pregnant until proven otherwise by a definitive pregnancy test. Approximately 1 in 12 women with known pregnancy experience physical trauma, which can cause maternal and fetal morbidity or mortality [100]. Specific considerations for pregnant trauma patients include:

●**Airway management** – If intubation is necessary, we suggest a rapid sequence induction and intubation (RSII) with application of cricoid pressure, and placement of a smaller sized endotracheal tube [100,101]. Also, a nasal or orogastric tube should be placed before or after intubation to prevent aspiration of acidic gastric contents [100]. Pregnant patients have increased risk for difficulties with airway management, including a difficult intubation, as well as aspiration of gastric contents [100-102]. (See "Initial evaluation and management of pregnant women with major trauma", section on 'Airway, breathing, and ventilation'.)

●**Uterine displacement** – If the uterus is at or above the umbilicus, it should be displaced to the left (off the aortocaval vessels) to increase venous return to maximize cardiac output. This is best accomplished by placing the patient on her left side; an alternative method is placement of a wedge or rolled towel under her right hip (or under the spinal board, if appropriate) to achieve a 30˚ left lateral tilt. (See "Initial evaluation and management of pregnant women with major trauma", section on 'Uterine displacement'.)

●**Volume replacement and transfusion** – Volume replacement after trauma with blood loss should be aggressive due to the physiologic hypervolemia of pregnancy. Volume replacement is preferable to vasopressor administration to support BP (figure 9). If transfusion is indicated in a Rh-negative pregnant patient, O-negative blood should be transfused until cross-matched blood becomes available (to avoid rhesus D [Rh] alloimmunization). Anti-D (RhoGAM) immune globulin should also be administered, per standard protocols. (See "Initial evaluation and management of pregnant women with major trauma", section on 'Volume replacement' and "Prevention of Rhesus (D) alloimmunization in pregnancy".)

●**Cesarean delivery** – The fetus may be viable at ≥23 weeks gestation if delivery is likely. A multidisciplinary approach with involvement of obstetricians, neonatal intensive care unit staff, and maternal fetal medicine specialists is ideal for management of mother and fetus. In the event of maternal cardiac arrest, a cesarean delivery is recommended for viable pregnancies ≥23 weeks, if possible no later than four minutes following arrest [100]. This facilitates both maternal resuscitation and fetal salvage. (See "Initial evaluation and management of pregnant women with major trauma", section on 'Delivery'.)

Other considerations for anesthetic management of a pregnant patient who must undergo nonobstetric surgery are discussed elsewhere. (See "Management of the pregnant patient undergoing nonobstetric surgery".)

Acute intoxication — Acute intoxication is frequently associated with trauma. In one study of more than 10,000 traumatically injured patients, nearly 60 percent tested positive for at least one substance of abuse, and 37 percent tested positive for multiple substances [103].

Ethanol — Ethanol is the most common acute intoxicant in trauma patients [104]. The Alcohol Use Disorders Identification Test (AUDIT) or Substance Use Brief Screening (SUBS) questionnaires can be rapidly administered in a conscious patient, and are sensitive and specific instruments to detect preoperative alcohol use (table 15 and table 16) [105].

Pathophysiologic changes that may affect intraoperative care of patients with ethanol intoxication are summarized in the table (table 17). Anesthetic management in trauma patients with known or suspected ethanol intoxication includes the following specific considerations:

●Rapid sequence induction and intubation (RSII) is employed to prevent aspiration pneumonitis since the stomach may be full of ethanol as well as food. (See "Rapid sequence induction and intubation (RSII) for anesthesia".)

●Ethanol is a central nervous system (CNS) depressant that alters the function of ion channels at several receptor sites including N-methyl-D-aspartate (NMDA), serotonin 5-hydroxytryptamine [5-HT_3], glycine, and gamma-aminobutyric acid ($GABA_A$) sites [106,107]. During acute intoxication, dose requirements of anesthetic agents are decreased due to additive central nervous system depression [106]. For example, doses of induction agents such as propofol should be decreased or eliminated, especially if hemorrhagic or other causes of shock are evident. Also, volatile agents are carefully titrated during maintenance of general anesthesia since the minimum alveolar concentration (MAC) requirements are typically lower than those for non-intoxicated patients [106].

●With chronic ethanol use, dose requirements for general anesthetics and adjuvant sedative and opioid agents may be increased if the patient is not acutely intoxicated, due to development of enzyme induction or cross-tolerance [105,108].

●If chronic liver insufficiency is present, the onset, metabolism, and duration of action of neuromuscular blocking agents (NMBAs) may be affected. NMBAs should be titrated to effect, and administration should be guided by monitoring with a peripheral nerve stimulator. (See "Anesthesia for the patient with liver disease", section on 'Neuromuscular blocking agents'.)

●Acetaminophen doses are limited or avoided because acute hepatic failure has been associated with doses as low as 4 g per day in patients with alcoholic liver disease [106,109]. (See "Acetaminophen (paracetamol) poisoning in adults: Pathophysiology, presentation, and diagnosis".)

Other aspects of management of patients with acute ethanol intoxication are discussed separately. (See "Ethanol intoxication in adults".)

Methamphetamine and hallucinogens — Acute intoxication with hallucinogens (eg, lysergic acid diethylamide [LSD], phencyclidine [PCP], ketamine, mescaline) or methamphetamines (3,4-methylenedioxmethamphatamine [MDMA] [known as ""ecstasy""]) affects anesthetic management due to the following specific considerations (see "Intoxication from LSD and other common hallucinogens" and "MDMA (ecstasy) intoxication"):

●Sympathetic nervous system activation due to any of these agents may cause [110]:

•Severe tachycardia.

•Severely increased blood pressure. Sympathomimetic drugs such as ephedrine should be administered with extreme caution (eg, in small incremental doses of 2.5 to 5.0 mg) due to potential for an exaggerated hypertensive response or life-threatening dysrhythmias.

•Increased body temperature.

•Pupillary dilation.

●Specific effects of amphetamines that may influence anesthetic management include [110]:

•Variable overall anesthetic requirements, with typically decreased MAC requirements for volatile inhalation agents [111,112].

•Severe or malignant hyperthermia.

•Cerebral edema and seizures due to severe hyponatremia if users have ingested excessive water to compensate for profound sweating. Electrolytes should be monitored closely in patients with water intoxication. (See "Manifestations of hyponatremia and hypernatremia in adults", section on 'Osmolytes and cerebral adaptation to hyponatremia'.)

•Disseminated intravascular coagulopathy (DIC).

●PCP or LSD intoxication may cause prolonged neuromuscular blocking effects of succinylcholine due to inhibition of plasma cholinesterase activity; thus, succinylcholine is avoided if early postoperative extubation is planned [110].

●Ketamine abuse may cause respiratory depression that is additive to the depressant effects of other anesthetic agents. (See "General anesthesia: Intravenous induction agents", section on 'Drug-drug interactions' and "General anesthesia: Intravenous induction agents", section on 'Dosing'.)

Cocaine — Cocaine users may have hypertension, intracranial hemorrhage, arterial vasoconstriction or thrombus formation with myocardial, pulmonary, or peripheral arterial infarction, stroke, seizures, severe hyperthermia, rhabdomyolysis, pulmonary infarction, and ischemic bowel. (See "Cocaine: Acute intoxication".)

Laryngoscopy with endotracheal intubation or noxious surgical stimuli may precipitate severe hypertension and cardiac dysrhythmias [110,111]. Prevention and/or treatment includes increasing anesthetic depth with intravenous (IV) or inhalation agents or administration of vasodilators (eg, hydralazine 5 to 10 mg bolus doses or a continuous infusion of nitroglycerin, nicardipine, or clevidipine for patients with persistent hypertension (table 10)). Beta-blockers (eg, metoprolol, propranolol, esmolol) are relatively contraindicated due to risk of inducing unopposed alpha-adrenergic stimulation [110,113]. (See "Cocaine: Acute intoxication", section on 'Cardiovascular complications'.)

Ketamine is avoided because it may potentiate cocaine's cardiovascular toxicity or cause myocardial depression if the patient has depleted catecholamine reserves due to hemorrhagic shock [110,114]. (See "General anesthesia: Intravenous induction agents", section on 'Cardiovascular effects' and "General anesthesia: Intravenous induction agents", section on 'Drug-drug interactions'.)

Succinylcholine is avoided since plasma cholinesterase metabolizes both succinylcholine and cocaine; thus, administration of succinylcholine may result in prolonged effects of cocaine as well as prolonged neuromuscular blockade [115,116]. Also, in patients who develop hyperthermia and rhabdomyolysis, succinylcholine may worsen hyperkalemia and cause life-threatening arrhythmias. Rocuronium is a reasonable alternative to produce a more reliable neuromuscular blockade. (See "Rapid sequence induction and intubation (RSII) for anesthesia", section on 'Alternatives to succinylcholine'.)

Overall anesthetic requirements for patients with acute preinjury use of cocaine are variable, and may not be greater than in patients without stimulant intoxication [112].

Opioids — Perioperative management in trauma patients with known or suspected opioid use includes the following specific considerations:

●Acute opioid intoxication: Dose requirements for anesthetic agents are typically decreased due to profound analgesia.

●Chronic opioid use: Opioid tolerance, withdrawal symptoms (table 18), and/or opioid-induced hyperalgesia may cause difficulties with postoperative pain control [117,118].

Details regarding anesthetic management of patients with acute or chronic opioid use are discussed separately. (See "Perioperative uses of intravenous opioids in adults", section on 'Chronic opioid use' and "Perioperative uses of intravenous opioids in adults", section on 'Acute opioid intoxication'.)

Gamma hydroxybutyrate (GHB) — Gamma-hydroxybutyrate (GHB) is a CNS depressant used recreationally for intoxicant effects and surreptitiously to facilitate sexual assault, with or without co-intoxicants. This agent may cause respiratory depression, seizures, and coma. (See "Gamma hydroxybutyrate (GHB) intoxication".)

Cannabis (marijuana) and synthetic cannabinoids — Acute intoxication with cannabis (marijuana) or synthetic cannabinoids (eg, "spice", "K2") reduces parasympathetic activity with resultant tachycardia. Thus, drugs that increase heart rate (eg, ketamine, atropine, epinephrine) are generally avoided [110]. Other cannabis effects on the cardiovascular system include myocardial depression; thus, doses of anesthetic agents with myocardial depressant effects (eg, volatile inhalation agents) are carefully titrated to avoid hypotension. (See "Cannabis (marijuana): Acute intoxication".)

Cannabis smoking has been reported to cause respiratory depression that is additive to the depressant effects of other anesthetic agents. Smoking marijuana may also cause bronchospasm that exacerbates underlying pulmonary disease (eg, asthma, bronchitis), as well as airway obstruction due to acute upper airway edema airway obstruction during laryngoscopy and endotracheal intubation [110,119,120].

Synthetic cannabinoids also have cardiovascular and respiratory effects including tachycardia and respiratory depression that is additive to the depressant effects of other anesthetic agents. Unlike cannabis, synthetic cannabinoids have significant potential to cause serious and life-threatening toxicity including coma, seizures, severe or malignant hyperthermia, rhabdomyolysis, and acute kidney injury [121]. (See "Synthetic cannabinoids: Acute intoxication".)

SUMMARY AND RECOMMENDATIONS

●Specific goals for trauma patients in the emergency department (ED) and the operating room (OR) include (see 'Goals'above):

•Airway management – A clearly defined, sequential approach to a patient with airway injury or abnormality is critical, since delay in securing the airway may lead to rapidly progressing hypoxemia (algorithm 1 and table 4 and table 5 and table 6 and table 7 and figure 1). (See 'Airway management' above.)

•Management of hemodynamic instability – Resuscitation of hypotensive patients to a targeted systolic blood pressure (BP) ≥90 mmHg is the primary goal until hemostasis has been achieved. Other goals for patients with hemorrhagic hypovolemic shock and its sequelae include management of coagulopathy,

hemodilution, hypothermia, electrolyte abnormalities, and acid-base derangements, as well as management of coexisting etiologies of shock after trauma. (See 'Management of hemodynamic instability' above.)

•Lung-protective ventilation – We employ low tidal volumes of 6 to 8 mL/kg, low plateau pressure ≤30 cmH_2O, and initial positive end-expiratory pressure (PEEP) at 0 cmH_2O. When the patient is hemodynamically stable, we incrementally increase PEEP to 5 to 10 cmH_2O and concurrently wean FiO_2 to maintain arterial oxygenation. (See 'Lung-protective ventilation' above.)

•Maintenance of normothermia – We employ warming devices to maintain temperature ≥35.5°C. (See 'Temperature management' above.)

•Prevention of unpleasant experiences – We employ strategies to minimize risk of awareness since it may be unsafe to administer sufficient anesthesia during all phases of trauma surgery. (See 'Strategies to minimize risk of awareness' above.)

●We employ cognitive aids such as a checklist to guide emergency preparations for intraoperative resuscitative care (table 1) and critically important handoffs from the ED to the OR to the intensive care unit (ICU) (table 3). (See 'Cognitive aids' above and 'Postoperative considerations' above.)

●An intra-arterial catheter and a central venous catheter (CVC) are usually inserted in hemodynamically unstable patients, ideally before anesthetic induction. However, insertion should not unduly delay emergency surgical intervention and large-bore peripheral intravenous catheters may be used rather than a CVC. (See 'Monitoring and intravenous access' above.)

●Anesthetic induction and maintenance agents with minimal hemodynamic effects are selected, and doses are reduced and carefully titrated to avoid exacerbation of hypotension. (See 'General principles' above.)

●A rapid sequence induction and intubation (RSII) technique with either etomidate or ketamine is typically employed; propofol is avoided in hypotensive patients (table 19). We typically select succinylcholine 1.5 mg/kg as the neuromuscular blocking agent (NMBA) for RSII; rocuronium 1.2 mg/kg is a reasonable alternative, particularly if sugammadex is immediately available (table 12). (See 'Induction' above.)

●We typically employ a volatile inhalation anesthetic agent (eg, desflurane, isoflurane, sevoflurane) for maintenance of anesthesia, administered at a lower concentration than in healthy patients due to dose-dependent cardiovascular effects, and titrated to maintain anesthesia while avoiding hypotension. We generally avoid nitrous oxide. (See 'Inhalation anesthetic agents' above.)

●When systolic BP is consistently maintained ≥90 mmHg and surgical hemostasis is assured, we add fentanyl in 50 to 150 mcg increments to a total dose of 10 to 30 mcg/kg during the surgical procedure, in order to beneficially dilate the microcirculation. If evidence of tissue hypoperfusion persists (eg, elevated lactate concentration and/or base deficit), we add another opioid such as methadone or hydromorphone to achieve additional vasodilatation. (See 'High-dose opioid supplementation' above.)

●In the postoperative period, most patients remain intubated and sedated with controlled ventilation. The electrocardiogram (ECG), pulse oximetry (SaO_2), and intra-arterial blood pressure are continuously

monitored during transport. A formal handoff process (table 3) and reassessment of the extent of unresolved shock are necessary upon arrival in the ICU. (See 'Postoperative considerations' above.)

●Injury-specific or patient–specific situations that require additional anesthetic considerations include (see 'Special populations' above):

•Use of resuscitative endovascular balloon occlusion of the aorta (REBOA) (see 'Resuscitative endovascular balloon occlusion of the aorta' above)

•Acute traumatic brain injury (see "Anesthesia for patients with acute traumatic brain injury")

•Acute traumatic spinal cord injury (see "Anesthesia for adults with acute spinal cord injury" and "Acute traumatic spinal cord injury")

•Traumatic injury in pregnant patients (see 'Traumatic injury in pregnant patients' above)

•Acute intoxication (eg, ethanol, methamphetamine, hallucinogens, cocaine, opioids, gamma hydroxybutyrate, cannabis [marijuana], synthetic cannabinoids) (see 'Acute intoxication' above)

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