Cardiac Arrest in the Operating Room: Resuscitation and Management for the Anesthesiologist: Part 1

Vivek K. Moitra, MD,* Sharon Einav, MD,† Karl-Christian Thies, MD,‡ Mark E. Nunnally, MD,§ Andrea Gabrielli, MD,‖ Gerald A. Maccioli, MD,¶ Guy Weinberg, MD,# Arna Banerjee, MD,** Kurt Ruetzler, MD,†† Gregory Dobson, MD,‡‡ Matthew D. McEvoy, MD,** and Michael F. O'Connor, MD, FCCM§§

Cardiac arrest in the operating room and procedural areas has a different spectrum of causes (ie, hypovolemia, gas embolism, and hyperkalemia), and rapid and appropriate evaluation and management of these causes require modification of traditional cardiac arrest algorithms. There is a small but growing body of literature describing the incidence, causes, treatments, and outcomes of circulatory crisis and perioperative cardiac arrest. These events are almost always witnessed, frequently known, and involve rescuer providers with knowledge of the patient and their procedure. In this setting, there can be formulation of a differential diagnosis and a directed intervention that treats the likely underlying cause(s) of the crisis while concurrently managing the crisis itself. Management of cardiac arrest of the perioperative patient is predicated on expert opinion, physiologic rationale, and an understanding of the context in which these events occur. Resuscitation algorithms should consider the evaluation and management of these causes of crisis in the perioperative setting. (Anesth Analg 2018;126:876–88)

Advanced cardiac life support (ACLS) was developed as an extension of basic life support (BLS). While ACLS was originally developed to manage patients who experienced sudden cardiac arrest in the community, it was subsequently imported into the hospital setting without adaptation or modification. Since their inception, BLS and ACLS have been intended for patients who suddenly collapse or who are found unresponsive.[1] BLS remains the foundation of ACLS, and ACLS remains organized around the electrocardiogram (ECG) and clinical signs of an (in) adequate circulation. ACLS remains focused on common cardiac causes of circulatory arrest and incorporates cardioversion, defibrillation, and pharmacotherapy to restore a spontaneous circulation.[2–4] While prior publications have described cardiac arrest and crises management in the operating room, the most recent update in ACLS prompted a review of the current literature concerning perioperative cardiac arrest and other crises.[5,6] Accordingly, the goal of this 2-part review is to offer an updated clinical perspective of cardiac arrest during the perioperative period. In the first part, we summarize the causes and outcomes of perioperative cardiac arrest, review concepts in resuscitation of the perioperative patient, and propose a set of algorithms to guide and prevent cardiac arrest during the perioperative period. In the second part, we discuss the management of special anesthesia-related and periprocedural crises.

Cardiac arrest in the perioperative setting is distinct because the arrest is almost always witnessed, and precipitating causes are often known. Compared to other settings, the response is potentially timelier, focused, and can reverse causes such as medication side effects and airway crisis.[7] Caregivers who take care of patients who undergo surgery usually know relevant medical history and witness a crisis that deteriorates over minutes to hours. Aggressive measures can be taken to support the patient to avert or delay the need for ACLS. In the era of shared decision making, the amount of escalation that is indicated when caring for a specific patient might be appropriately limited by an understanding of the patient's and family's wishes regarding heroic measures.

CONSENSUS METHODOLOGY

An international group of 12 experts in the field of perioperative resuscitation was invited to review and evaluate the evidence on management of cardiac arrest and periprocedural crises. These experts were selected on the basis of several criteria: (1) clinical experience in anesthesiology and perioperative patient management; (2) involvement in simulation training in perioperative crises and resuscitation; (3) familiarity with resuscitation guidelines; and (4) international representation (to ensure that the recommendations are easily translatable to bedside practice in multiple clinical platforms). The group communicated via e-mail, face to face, and by telephone as required. The articles selected for review were those included in the previous iteration of

From the *Columbia University, New York, New York; †Shaare Zedek Medical Center, Jerusalem, Israel; ‡Birmingham Children's Hospital, Birmingham, United Kingdom; §New York University, New York, New York; ‖University of Pennsylvania, Philadelphia, Pennsylvania; ¶Envision Healthcare, Plantation, Florida; #The University of Illinois at Chicago, Chicago, Illinois; **Vanderbilt University, Nashville, Tennessee; ††Cleveland Clinic, Cleveland, Ohio; ‡‡Dalhousie University, Nova Scotia, Canada; and §§University of Chicago, Chicago, Illinois.

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K.-C. Thies is currently affiliated with the Department of Anesthesiology, University Medical Center Greifswald, Ferdinand-Sauerbruch-Straße, Greifswald, Germany.

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Address correspondence to Vivek K. Moitra, MD, Department of Anesthesiology, PH 527-B, College of Physicians & Surgeons of Columbia University, 630 West 168th St, New York NY 10032. Address e-mail to vm2161@cumc.columbia.edu.

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these guidelines (which underwent repeat scrutiny) and relevant articles that had been published since 2012 and available on PubMed on the specific topics to be discussed. Disagreements among committee members were discussed in an attempt to reach consensus and in case of ongoing dissent adjudicated by 2 of the authors (V.K.M. and M.F.O.).

CAUSES OF PERIOPERATIVE CARDIAC ARREST

The spectrum of causes of circulatory crisis and cardiac arrest may be very different than anywhere else inside the hospital or outside it. Vagal responses to surgical manipulation, vagotonic anesthetics, sympatholysis from anesthetic agents, β-blockers, and the suppression of cardiac-accelerator fibers arising from T1 to T4 in patients undergoing neuraxial anesthesia are common causes of perioperative bradycardia.[8,9] Hypoxia associated with difficult airway management is a well-recognized cause of cardiac arrest in the operating room.[10–13] Pulseless electrical activity (PEA) from hypovolemia is a common cause of cardiac arrest in hemorrhaging patients in the operating room. The unique and broad differential diagnosis of circulatory collapse in the periprocedural period includes anesthetic conditions such as inhalational and intravenous anesthetic overdose; neuraxial blockade, local anesthetic systemic toxicity, and malignant hyperthermia; respiratory causes such as hypoxemia, auto–positive end-expiratory pressure (PEEP), and bronchospasm; and cardiovascular etiologies such as vasovagal and oculocardiac reflexes, hypovolemic shock, air embolism, increased intraabdominal pressure, transfusion and anaphylactic reactions, tension pneumothorax, pacemaker failure, prolonged QT syndrome, and electroconvulsive therapy.[10–13]

PERIOPERATIVE CARDIAC ARREST OUTCOMES

Over the past 5 years, multiple studies have reported increased survivorship after perioperative arrest compared to arrests in the general population or on inpatient hospital wards.[7,10,14–16] Another study of surgical patients reported encouraging survival statistics, with the lowest survivorship (<20%) among patients who were elderly, had higher American Society of Anesthesiologists status, emergency procedures, contaminated wounds, and high preoperative dependency.[17] Previous observations of lower survivorship after cardiac arrest at night and on weekends have been replicated.[16,18,19] Perhaps paradoxically, survivorship and neurologic outcomes from cardiac arrest are better when they occur in the postanesthesia care unit as compared to the operating room or intensive care unit. This may be related to the different etiologies leading to arrest in that setting.[16]

A recently published analysis of cardiac arrest data from the National Anesthesia Clinical Outcomes Registry revealed that the incidence of cardiac arrest associated with anesthesia is approximately 5.6 per 10,000 cases (951 arrests in 1,691,472 cases), which is considerably lower than previous estimates.[20,21] This analysis also observed that the rate of cardiac arrest increased with age and American Society of Anesthesiologists physical status. Unexpectedly, the study reported a higher rate of cardiac arrest and death among males. A recent study of patients who experienced cardiac arrest within 24 hours of surgery found that asystole was the most common cardiac arrest rhythm.[16] Survivorship after asystole in the perioperative period, however, is significantly higher (30.5%–80%) compared to survivorship after inpatient asystolic arrest (10%).[7,16,22]

PRECARDIAC ARREST CONSIDERATIONS

Surveys of anesthesiologists document lack of awareness of both basic and anesthesia-related knowledge of resuscitation and cardiac arrest.[23,24] One study documented delay in the cardioversion and defibrillation of patients with shockable rhythms in the perioperative setting.[25] To rescue a patient from crisis, caregivers must recognize the patient is in crisis and institute effective action.[26–28] Recognition that a patient is in crisis is more difficult in the periprocedural setting because the patient is sedated or under general anesthesia (precluding adequate monitoring of their mental status); their respirations are often controlled (preventing tachypnea or apnea); surgical positioning often frustrates assessment (lateral, prone, steep Trendelenburg); and large portions of their body are covered with drapes. Failure to rescue is an often-invoked "cause" of cardiac arrest and morbidity/mortality and is generally the product of hindsight bias shaping the evaluation of the care rendered.[29] While failure to rescue does occur, it almost certainly occurs less frequently than it is suggested. In many (likely most) instances, the underlying cause of crisis is so severe that the patient's demise is inevitable, even if maximal support is instituted in a timely fashion.[26,30]

Escalating Care

Escalation of care includes higher levels of monitoring and more advanced supportive measures. Decisions about higher levels of monitoring or evaluation require consideration of the patient's history, current clinical status, anesthetic, and procedure. Insertion of invasive monitors should not delay supportive care. Almost every unstable patient should be monitored with an arterial line. Central venous access is appropriate when monitoring central venous pressures or venous oxygen saturations help guide resuscitation, or when caregivers anticipate infusing vasoconstrictors over longer periods of time. Over the past decade, clinicians have increasingly performed point of care ultrasound in unstable patients to make quick diagnoses and manage a crisis.[31] The decision to escalate the level of monitoring is a clinical judgment that encompasses all relevant patient and surgical factors and is beyond the scope of these recommendations.

Clinical Progression to Shock

Anesthesiologists commonly administer titrated boluses of vasoactive drugs (ie, phenylephrine, ephedrine, vasopressin, norepinephrine, and epinephrine) to unstable patients. Often, small boluses of vasopressin (arginine vasopressin 0.5–2 units IV) may improve hemodynamics when escalating bolus doses of catecholamines have failed. The use of arginine vasopressin and its analogs in low-flow states, cardiac arrest, and hypotension refractory to catecholamines has been extensively documented.[32–35] A reasonable sequence of care for the unstable patient who is progressing toward shock is outlined in the first part of Table.

Left Ventricular Failure

Echocardiography and invasive monitors such as the pulmonary artery catheter guide the management of left ventricular (LV) failure. Hypovolemia can cause or contribute to shock in patients with poor LV function and should be remedied before institution of any pharmacologic therapies. Hypotensive euvolemic patients with LV shock are treated with inotropic

Table. Corrective Measures for Clinical Progression to Shock and a Modified Stepwise Approach for Cardiac Arrest in the Operating Room Based on American Heart Association 2010 and 2015 ACLS Guidelines and the 2008 International Liaison Committee on Resuscitation Consensus Statement on Postcardiac Arrest Syndrome

Corrective Measures for Clinical Progression to Shock	
• Recognize crisis • Call for help • Call for defibrillator • Hold procedure and reduce/hold anesthetic if feasible • Administer 1.0 Fio_2 • Confirm airway positioning and functioning • Assess oxygen source and anesthetic circuit integrity • Review $Etco_2$ trends before hemodynamic instability • Administer IV fluids wide open	
Generate a Differential Diagnosis	
• Evaluate procedure and consult with procedural colleagues • Review recently administered medications • Obtain chest radiograph perform thoracic ultrasound to rule out tension pneumothorax if airway pressures acutely increased • Obtain echocardiogram (transesophageal echocardiogram if patient is intubated or has a surgically prepped chest) to evaluate ventricular filling, ventricular function, and valvular function and to exclude pericardial tamponade (eg, FEER or similar examination) • Empiric replacement therapy with corticosteroids (in patients who have not been previously treated with steroids, 50 mg of hydrocortisone IV and 50 μg of fludrocortisone per os/ng is an appropriate dose)	
Perioperative Cardiac Arrest	
Circulation	• Check pulse for 10 s • Effective 2-rescuer CPR: 1. Minimize interruptions 2. Chest compression rate 100–120 compressions·minute^{-1} 3. Depth 2 in, full decompression, real-time feedback 4. Titrate CPR to A-line BP diastolic 40 mm Hg or $Etco_2$ 20 mm Hg • Drug therapy • Attempt CVL placement
Airway	• Bag mask ventilation until intubation • Endotracheal intubation • Difficult airway algorithm
Breathing	• Respiratory rate 10 breaths·minute^{-1} • V_T to visible chest rise • T_I 1 s • Consider ITV
Defibrillation	• Defibrillation if shockable rhythm • Repeat defibrillation every 2 min if shockable rhythm
Postcardiac Arrest	
• Invasive monitoring • Final surgical anesthetic plan • Transfer to ICU	

Abbreviations: ACLS, advanced cardiac life support; BP, blood pressure; CPR, cardiopulmonary resuscitation; CVL, central venous line; $Etco_2$, end-tidal carbon dioxide; FEER, focused echocardiographic evaluation and resuscitation; Fio_2, fraction of inspired oxygen concentration; ICU, intensive care unit; ITV, inspiratory threshold valve; IV, intravenous; T_I, inspiratory time; V_T, tidal volume.

agents and medications that reduce afterload.[36,37] In patients with known, significant diastolic dysfunction, therapy with lusitropic agents such as milrinone enhances ventricular relaxation to improve cardiac output. Increasingly, mechanical support with intraaortic balloon pumps, ventricular assist devices, and extracorporeal life support (also referred to as extracorporeal membrane oxygenation) is utilized in hospitalized patients who are believed to have good potential for recovery from severe LV shock, right ventricular (RV) shock, and cardiac arrest.[38,39] Figure 1 outlines 1 approach to manage a patient with LV shock.

RV Failure

Similar to LV shock, RV shock is best guided by a combination of invasive monitors such as the pulmonary artery catheter and/or echocardiography. In most instances, an acute rise in pulmonary vascular resistance (often in the setting of a chronic cause of pulmonary hypertension) causes and sustains RV shock.[40] A combination of inotropes, systemic arterial vasoconstrictors, and pulmonary artery vasodilators such as nitric oxide manage RV shock. In contrast to the management of LV shock, the use of systemic arterial vasoconstrictors for RV dysfunction may improve end-organ perfusion and cardiac output (Figure 2).[40,41] Administering vasopressin to enhance blood pressure may decrease the pulmonary vascular resistance-to-systemic vascular resistance ratio because vasopressin's constrictive effects spare the pulmonary vasculature compared to norepinephrine and phenylephrine.[42,43] Over the past several years, mechanical support devices, including ventricular assist devices and extracorporeal membrane oxygenation, have been

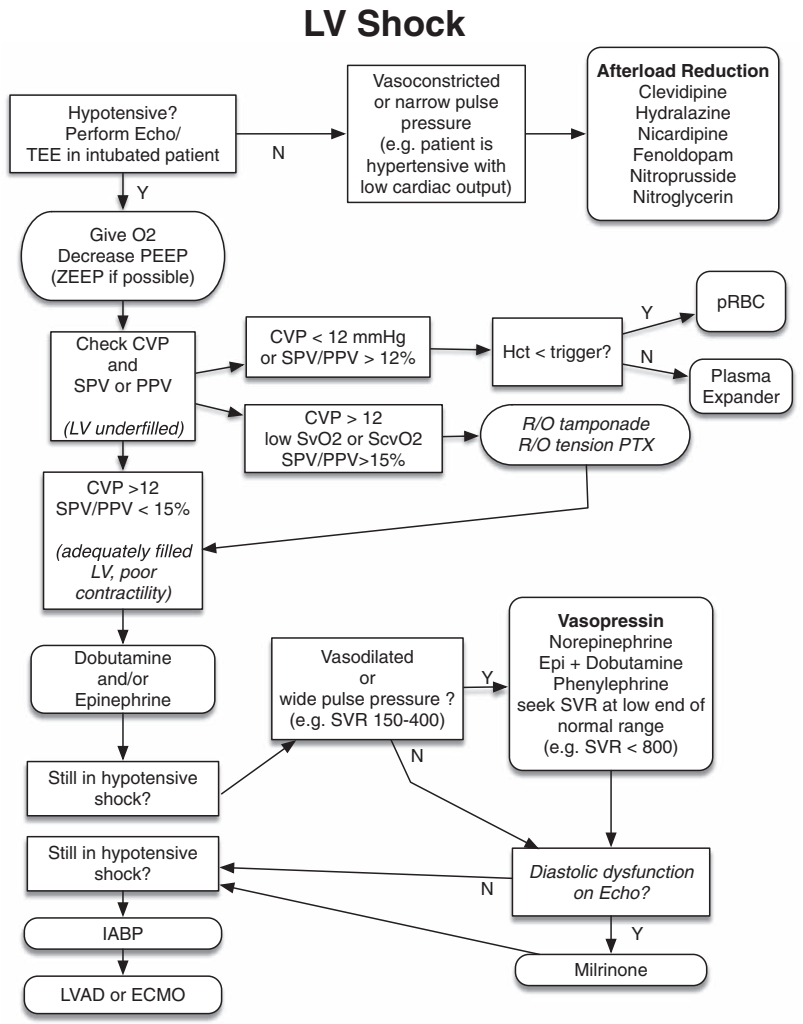

Figure 1. Treatment algorithm of LV failure with cardiogenic shock. CVP indicates central venous pressure; ECMO, extracorporeal membrane oxygenation; Hct, hematocrit; IABP, intraaortic balloon pump; LV, left ventricular; LVAD, left ventricular assist device; PEEP, positive end-expiratory pressure; PPV, pulse pressure variation; pRBC, packed red blood cell; PTX, pneumothorax; R/O, rule out; $Scvo_2$, central venous oxygen saturation; SPV, systolic pressure variation; Svo_2, mixed venous oxygen saturation; SVR, systemic vascular resistance in dyne·second$-^{1}$·cm^{-5}·m^{-2}; TEE, transesophageal echocardiography; ZEEP, zero end-expiratory pressure.

utilized with increasing frequency in patients with RV shock.[38,39]

Hypovolemia and Systolic and Pulse Pressure Variation

Hypovolemia can cause perioperative hypotension, circulatory crisis, and shock. Over the past decade, pulse pressure variation (PPV) and systolic pressure variation (SPV) have replaced central venous pressure monitoring as bedside indicators of volume responsiveness in hypotensive patients. While these measurements are most reliable in intubated, mechanically ventilated patients who are synchronous with appropriate ventilator settings (>8 mL/kg), there is a growing literature that suggests that SPV and PPV can be measured in spontaneously breathing patients with only slightly diminished reliability.[44,45] If the PPV or SPV exceeds the threshold value of 12%–15%, fluid administration or increased preload will likely increase stroke volume.[46] The plethysmographic signal from a pulse oximeter may suggest fluid responsiveness.[47] Importantly, the presence of RV shock or any of the causes of obstructive shock (auto-PEEP, cardiac tamponade, tension pneumothorax, pulmonary hypertensive crisis, and abdominal compartment syndrome) will produce elevated SPV and PPV that do "not" predict volume responsiveness.[48,49] Excessive tidal volumes (>10 mL/kg), increased residual volume and lung compliance (emphysema), and decreased chest wall compliance (third-degree chest burn, obesity, prone position) increase PPV and SPV, and criteria for volume responsiveness should be adjusted in these conditions.[50] Assessing heart–lung interactions via PPV or stroke volume variation in the setting of cardiac arrhythmias such as atrial fibrillation or frequent premature ventricular contractions is not reliable.[50]

Hypotension and PPV/SPV values of <10% suggest that hypotension and shock will not improve with fluid resuscitation. While the passive leg raise (a quick, reversible, and often easy-to-perform maneuver that raises the patient's legs to assess changes in blood pressure and hemodynamics) also predicts volume responsiveness, it is not especially practical in the operating room.[51–54] Although ultrasound assessment of inferior vena cava diameter variation with respiration may predict volume responsiveness, it is also not practical during a wide variety of operations (abdominal, cardiac, thoracic) or patient positions (lateral, prone, sitting).[55,56] Evaluation of the SVC diameter is possible with either transesophageal echocardiography or transthoracic echocardiography and is more practical in many operative settings. Esophageal Doppler assessment of aortic blood velocity is also predictive of

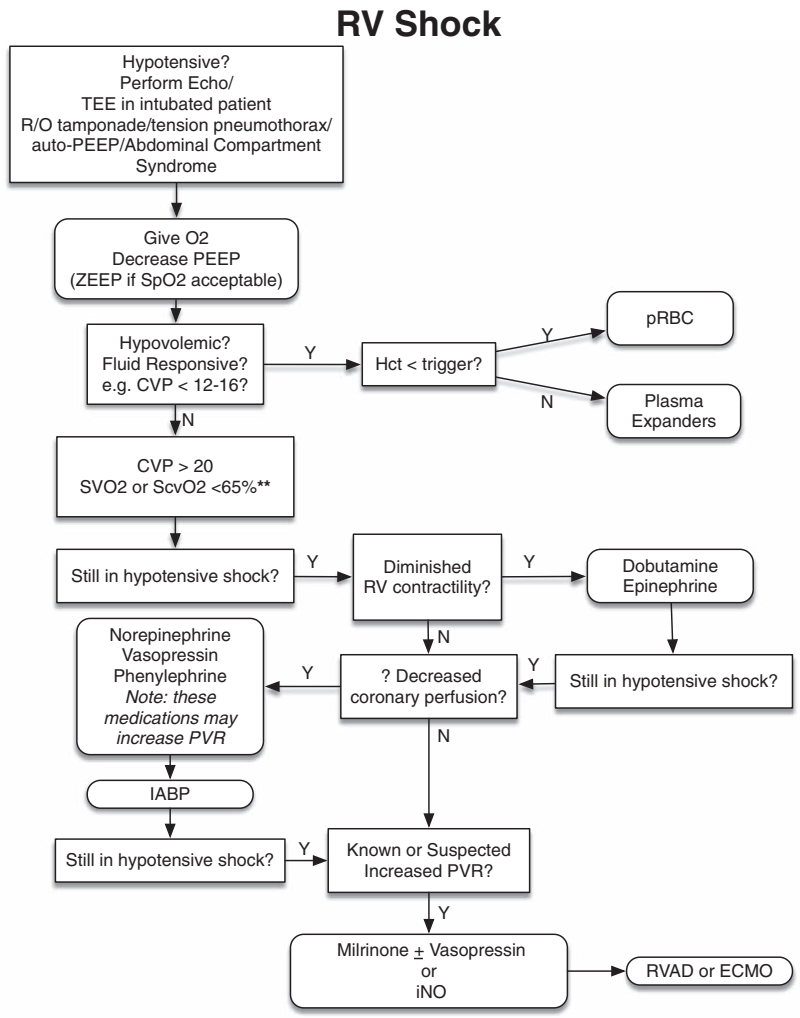

Figure 2. Treatment algorithm of RV failure with cardiogenic shock. CVP indicates central venous pressure; ECMO, extracorporeal membrane oxygenation; Hct, hematocrit; IABP, intraaortic balloon pump; iNO, inhaled nitric oxide; PEEP, positive end-expiratory pressure; pRBC, packed red blood cell; PVR, pulmonary vascular resistance; R/O, rule out; RV, right ventricular; RVAD, right ventricular assist device; $Scvo_2$, central venous oxygen saturation; Spo_2, pulse oximeter oxygen saturation; Svo_2, mixed venous oxygen saturation; TEE, transesophageal echocardiography; ZEEP, zero end-expiratory pressure.

volume responsiveness, but once again, requires instrumenting the esophagus and expertise in interpreting the data.[57] Practically speaking, a patient who is acutely and severely hypotensive should be volume resuscitated (with blood products if hemorrhage or undetected surgical bleeding is likely) as monitoring is escalated, and volume responsiveness is assessed via changes in blood pressure and heart rate.

Ventilation During Severe Shock or Cardiac Arrest

Over the past 2 decades, multiple clinical studies have demonstrated either no harm or a mortality or outcome benefit when patients with respiratory failure or acute respiratory distress syndrome are ventilated with lower tidal volumes and permissive hypercapnia; a strategy during which carbon dioxide (CO_2) levels rise and pH falls as long as the oxygen saturation stays above 90%.[58–63]

Hyperventilation is deleterious in both shock and cardiac arrest. Studies of ventilation during shock repeatedly demonstrate that the duration of increased intrathoracic pressure is proportional to the ventilation rate, tidal volume, inspiratory time, and delayed chest decompression and is inversely proportional to coronary and cerebral artery perfusion.[59,64–66] Ventilation at 20 breaths·minute^{-1} during cardiopulmonary resuscitation (CPR) is associated with significantly lower survival than ventilation at 10 breaths·minute^{-1}. BLS guidelines continue to emphasize avoiding hyperventilation during CPR and recommend higher compression-to-ventilation ratio (eg, 30:2) for victims of all ages (except newborns).[1] Even with an endotracheal tube, the respiratory rate should be 10 breaths·minute^{-1} or less, with an inspiratory time of 1 second, and the tidal volume limited to "chest rise" (approximately 500 mL in a 70-kg adult).[3] An algorithm for coordinating airway management with CPR is shown in Figure 3. Newer devices that provide a combination of automatic CPR and an airway in-line negative inspiratory valve (allowing increased venous return during chest decompression) may be associated with an increased rate of return of spontaneous circulation (ROSC), but no increase in survival to hospital discharge.[67–70]

Because positive pressure ventilation decreases venous return and hypoventilation seems to cause no harm, it is reasonable to ventilate patients in shock with the lowest ventilator settings compatible with a saturation of 90% or greater.

Auto-PEEP

Auto-PEEP, also known as intrinsic PEEP or gas trapping, is a well-described but often difficult to recognize cause of circulatory collapse and PEA.[71] Auto-PEEP occurs almost exclusively in patients with obstructive lung disease, especially asthma

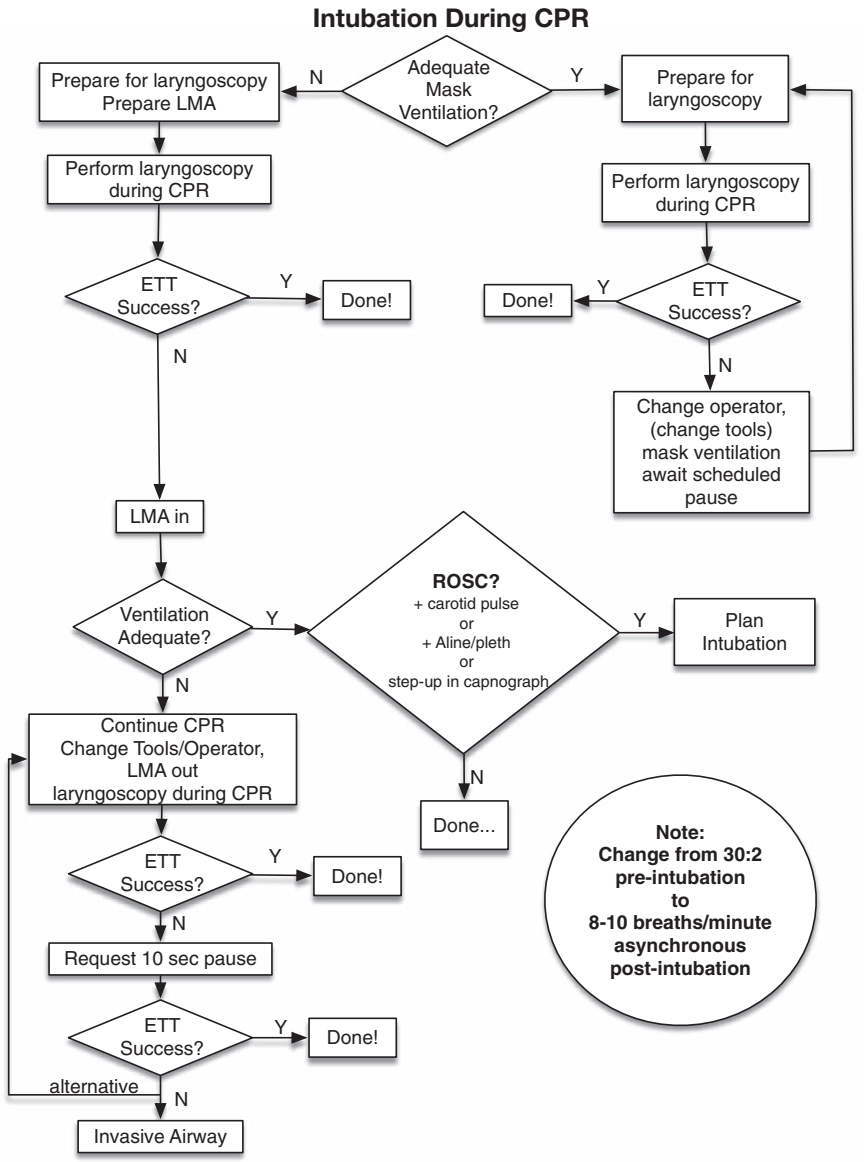

Figure 3. Intubation during CPR. CPR indicates cardiopulmonary resuscitation; ETT, endotracheal tube; LMA® , laryngeal mask airway; ROSC, return of spontaneous circulation.

and chronic obstructive pulmonary disease (emphysema). In patients with obstructive lung disease, mechanical ventilation that does not allow sufficient time for complete exhalation produces a gradual accumulation of air (volume) and pressure (end-expiratory pressure) in the alveoli. This pressure is transmitted to the pulmonary capillaries, and then to the great vessels in the thorax, where it decreases both venous return and cardiac output. Clinical reports demonstrate that as auto-PEEP increases, venous return decreases.[72,73]

The presence of auto-PEEP can be inferred whenever the expiratory flow waveform does not return to the zero baseline in between breaths. In the absence of a flow waveform display, auto-PEEP can be diagnosed by disconnecting the endotracheal tube from the ventilator for 10–20 seconds, and observing a "step-up" gain of invasive or noninvasive systolic blood pressure. Dramatic improvement in response to this maneuver should prompt maximal therapy for obstruction lung disease/bronchospasm, and mechanical ventilation with both small tidal volumes (<6 mL/kg), a low respiratory rate (<10/min), and a short inspiratory time (which will produce a paradoxical and acceptable increase in the peak inspiratory pressures). Given that auto-PEEP is an important cause of an unacceptable circulation, it should be quickly ruled out in any unstable patient. The Lazarus phenomenon, a seemingly miraculous recovery and ROSC after the discontinuation of resuscitative efforts, can diagnose circulatory collapse from auto-PEEP during resuscitation.[74]

RESCUE SEQUENCE FOR CARDIAC ARREST IN THE OPERATING ROOM

Recognizing cardiac arrest in the operating room can be more difficult than it appears to nonoperating personnel. The vast majority of alarms from sensors such as the ECG and pulse oximeter are false alarms.[75,76] Bradycardia happens relatively frequently in patients undergoing anesthesia and is often associated with hypotension from the combination of anesthesia and little or no procedural stimulation. Patients with heart rates as low as 40 beats minute^{-1} can be clinically stable and do not require intervention as long as

their blood pressure remains acceptable.[3] Finally, the combination of body habitus and pathology can render routine monitors useless. It can be difficult or impossible to obtain a reliable pulse oximetry tracing in hypothermic, hypovolemic, or vasculopathic patients.[77] Major burns or anasarca can frustrate noninvasive blood pressure, ECG, and pulse oximetry monitoring.

Features of cardiac arrest in the perioperative setting include an ECG with pulseless rhythms (ie, ventricular tachycardia, ventricular fibrillation, severe bradycardia, and asystole), loss of carotid pulse >10 seconds, loss of end-tidal CO_2 ($Etco_2$) with loss of plethysmograph, and/or loss of an arterial line tracing. Of these, loss of $Etco_2$ is perhaps the most reliable and routinely monitored indicator of circulatory crisis or cardiac arrest.

Evaluation of $Etco_2$ should be in the context of the patient's clinical status. When minute ventilation is fixed and cardiac output is low, pulmonary blood flow determines $Etco_2$. Although low $Etco_2$ values are observed in low-flow states, conditions such as air leaks with supraglottic airways, increased airway resistance (mucous plugging, bronchospasm, endotracheal tube kinking), pulmonary edema, and hyperventilation also reduce $Etco_2$.[3] Hypermetabolic states such as malignant hyperthermia or

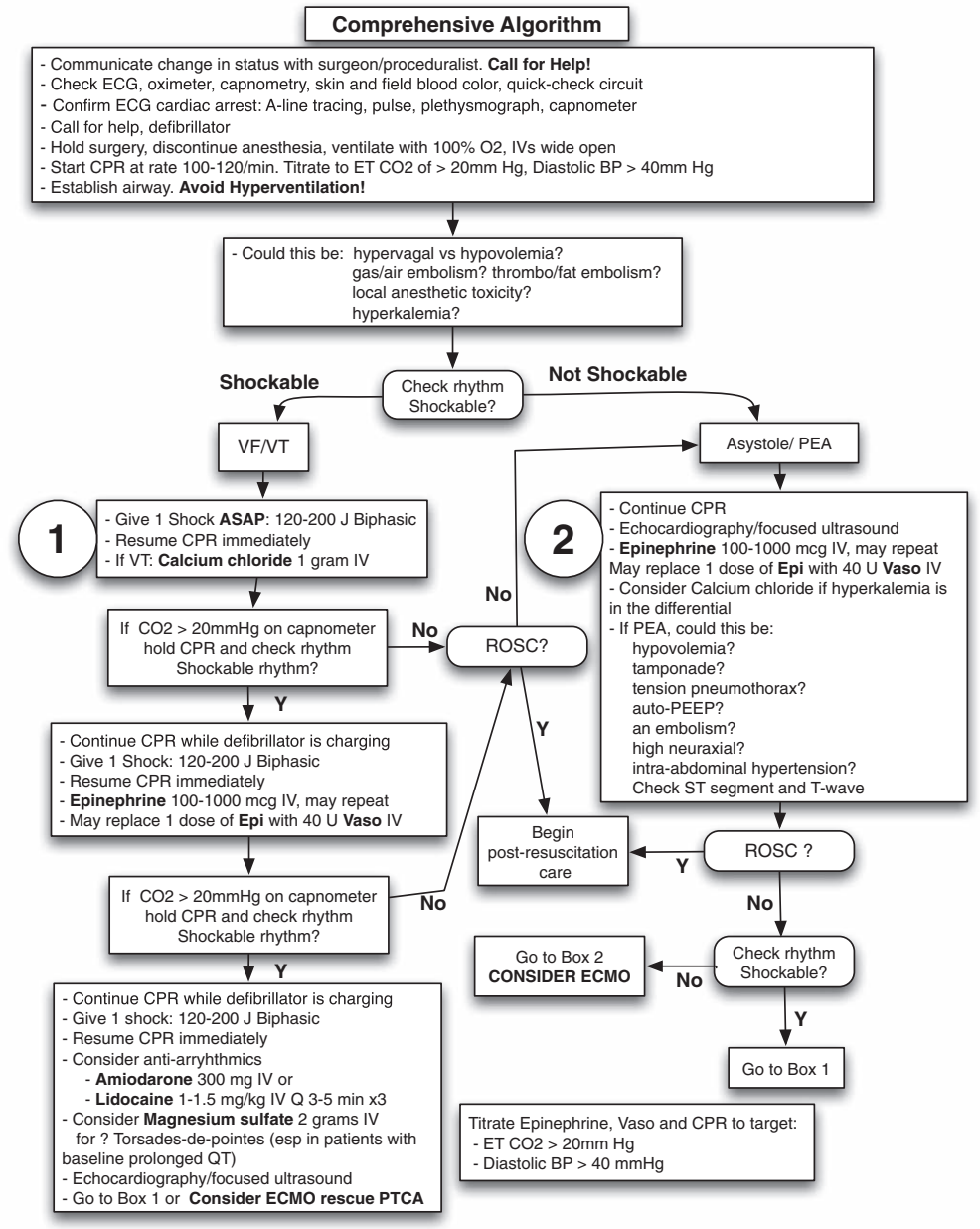

Figure 4. Comprehensive algorithm. Adaptation of the ACLS comprehensive algorithm. Rescuers are prompted to evaluate or empirically treat early for hyperkalemia. Echocardiography is especially useful in establishing the most likely cause of pulseless electrical activity and focusing resuscitation efforts. ACLS indicates advanced cardiac life support; ASAP, as soon as possible; BP, blood pressure; CPR, cardiopulmonary resuscitation; ECG, electrocardiogram; ECMO, extracorporeal membrane oxygenation; Epi, epinephrine; $Etco_2$, end-tidal carbon dioxide; IV, intravenous; PEA, pulseless electrical activity; PEEP, positive end-expiratory pressure; PTCA, percutaneous transluminal coronary angioplasty; ROSC, return of spontaneous circulation; Vaso, vasopressin; VF, ventricular fibrillation; V_T, tidal volume.

neuroleptic malignant syndrome may also increase CO_2 levels. The administration of intravenous sodium bicarbonate increases $Etco_2$ levels.

Once cardiac arrest is confirmed, CPR should be initiated without delay (Figure 4). Effective chest compression generates an $Etco_2$ close to or above 20 mm Hg, and higher $Etco_2$ values during CPR are associated with improved survival.[78] With few or no exceptions, $Etco_2$ <10 mm Hg after 20 minutes of standard ACLS is associated with failure of ROSC.[79–82] Several studies document that a relaxation (diastolic) pressure (calculated at the time of full chest decompression) of 30–40 mm Hg on an arterial tracing is associated with a higher rate of ROSC, even after prolonged CPR.[83–85] Modern defibrillators can provide real-time feedback on the quality of chest compressions, which can in turn drive timely rotation of rescuers performing CPR, and may lead to better outcomes.[86]

Table shows a stepwise approach to the evaluation and management of cardiac arrest in the OR and perioperative setting. It is based on the 2010 and 2015 American Heart Association ACLS sequence and the International Liaison Committee on Resuscitation consensus statement on postcardiac arrest syndrome. Prolonged resuscitation efforts (up to 45 minutes) in inpatients have been associated with improved survivorship.[15]

RETURN OF SPONTANEOUS CIRCULATION

Capnography is usually a more reliable indicator of ROSC than carotid or femoral arterial pulse palpation.[3] A sudden increase in $Etco_2$ (>35–40 mm Hg) suggests ROSC. Other indicators of ROSC include the presence of a palpable pulse, blood pressure, and arterial line waveforms.[3] Palpation of a pulse during chest compressions may reflect venous pulsation. If rescuers are concerned that the capnograph is malfunctioning, blowing into the sidestream CO_2 collecting tube is a quick way to evaluate this.

ACLS OPERATING ROOM ALGORITHMS

Symptomatic Bradycardia Evolving to Nonshockable Arrest

Perioperative bradycardia, asystole, and PEA have 16 causes (8 Hs and 8 Ts), that build on the differential diagnoses (6 Hs and 6 Ts) proposed by the American Heart Association[3]: hypoxemia, hypovolemia, hyper-/hypokalemia, hydrogen

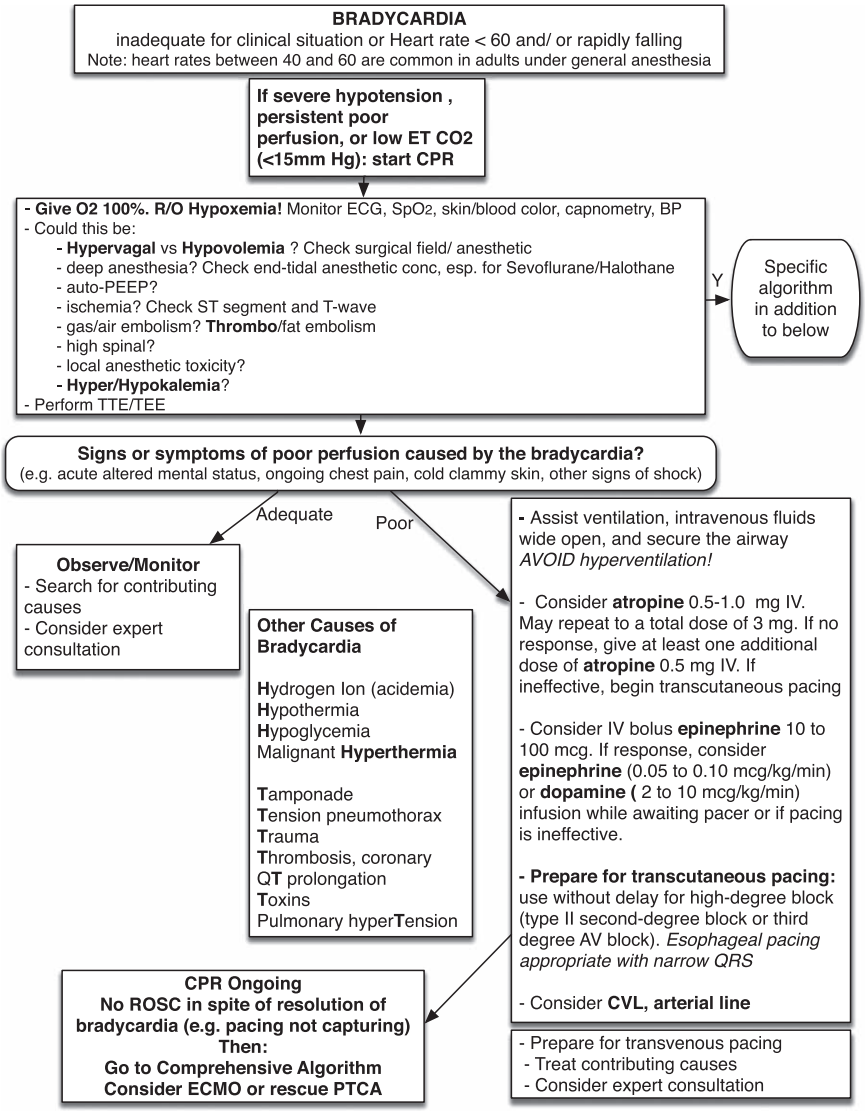

Figure 5. Bradycardia. Adaptation of ACLS algorithm for bradycardia. ACLS indicates advanced cardiac life support; AV, atrioventricular; BP, blood pressure; CPR, cardiopulmonary resuscitation; CVL, central venous line or catheter; ECG, electrocardiogram; ECMO, extracorporeal membrane oxygenation; $Etco_2$, end-tidal carbon dioxide; IV, intravenous; PEEP, positive end-expiratory pressure; PTCA, percutaneous transluminal coronary angioplasty; R/O, rule out; ROSC, return of spontaneous circulation; Spo_2, pulse oximeter oxygen saturation; TEE, transesophageal echocardiography; TTE, transthoracic echocardiography.

ion excess, hypothermia, hypoglycemia, malignant hyperthermia, hypervagal, toxins (anaphylaxis/anesthetics), tension pneumothorax, pulmonary thrombosis/embolus, coronary thrombosis, tamponade, trauma, QT prolongation, and pulmonary hypertension. These likely causes are listed alongside a suggested approach to perioperative bradycardia in Figure 5. A narrow complex QRS PEA rhythm suggests RV inflow or outflow obstruction (ie, tamponade, tension pneumothorax, auto-PEEP, myocardial ischemia, or pulmonary embolism). A wide complex QRS PEA rhythm may signify a metabolic crisis such as hyperkalemia or local anesthetic toxicity or LV pump failure.[88] Sudden, severe bradycardia in the periprocedural setting is often caused by physical manipulations that increase vagal tone and potentiated by the combination of vagotonic anesthetics and the sympatholysis that accompanies almost all anesthetics.

Treatment with atropine should be considered in any patient who does not become appropriately tachycardic in response to treatment with epinephrine or larger doses of ephedrine.[89] Reports of paradoxical bradycardia and sinus arrest have been described in patients who have received atropine doses of <1 mg. Potential mechanisms include a vagolytic-induced "stress test" of the sinus node; a vagotonic effect at the sinus node and a vagolytic effect at the atrioventricular node to cause a junctional rhythm; atropine-induced peripheral hypotension with a subsequent

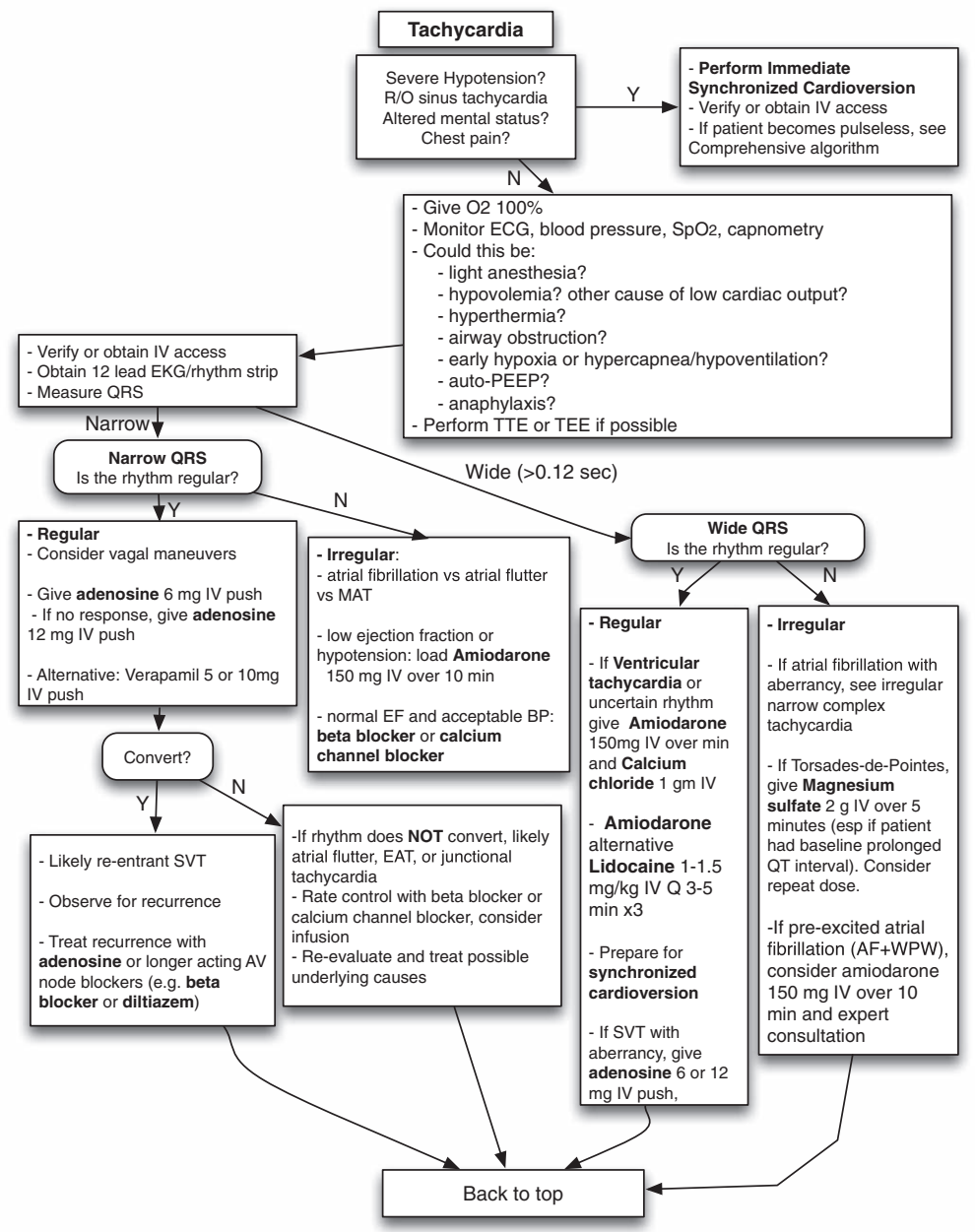

Figure 6. Tachycardia. Adaptation of the ACLS algorithm for tachycardia. ACLS indicates advanced cardiac life support; AF + WPW, atrial fibrillation and Wolff-Parkinson-White syndrome; BP, blood pressure; EAT, ectopic atrial tachycardia; ECG, electrocardiogram; EF, ejection fraction; EKG, electrocardiogram; IV, intravenous; MAT, multifocal atrial tachycardia; PEEP, positive end-expiratory pressure; R/O, rule out; Spo_2, pulse oximeter oxygen saturation; SVT, supraventricular tachycardia; TEE, transesophageal echocardiography; TTE, transthoracic echocardiography.

hypervagotonic reflex; and central nervous system vagotonia via cholinesterase inhibition.[90–93]

The different spectrum of causes of periprocedural bradycardia makes it appropriate to resort to pacing earlier in these patients. Even though we recommend it, there is no evidence to suggest any outcome benefit from the use of pacing (which may delay chest compressions) when full cardiac arrest is in progress.[94–97] Appropriate indications for emergency pacing include hemodynamically unstable bradycardia unresponsive to positive chronotropic agents; symptomatic tissue conduction dysfunction of the sinus node; Mobitz type II second-degree and third-degree block; alternating bundle branch block; or bifascicular block.[3]

Symptomatic Tachycardia Evolving to Pulseless Shockable Arrest (Ventricular Tachycardia, Ventricular Fibrillation, and Torsades De Pointes)

Hypovolemia or a significant imbalance between the depth of anesthesia and the amount of procedural stimulation are the most frequent causes of hypotension in the periprocedural setting. The 8 Hs and 8 Ts can cause a circulatory crisis that can devolve into a PEA arrest.

In general, the evolution of a malignant rhythm is an indicator of a severe process, severe cardiac comorbidities, and/or severe complications. Persistent tachycardia with hemodynamic instability can devolve into symptomatic bradycardia. Tachycardia from any cause other than sinus tachycardia that is associated with significant hypotension is an indication for immediate cardioversion (ventricular rate >150 beats·minute^{-1}).[3] Cardioversion can sometimes convert a patient into a symptomatic bradycardia, which can necessitate emergency pacing. Overdrive pacing of supraventricular or ventricular tachycardia may also be appropriate in perioperative patients, and it should be considered when the rhythm is refractory to drugs or cardioversion.[98]

Figures 4 and 6 outline the practical considerations for the management of symptomatic tachycardia in the perioperative period.

CONCLUSIONS

Cardiac arrest in the periprocedural setting is rarer than previously believed, and it arises from unique causes specific to the operating room or procedural environment. Circulatory crisis and cardiac arrest in this setting are usually managed by practitioners who are familiar with the patient, knowledgeable of the patient's medical condition, and familiar with the details of their procedure, which allows them to intervene in a directed, effective, and timely manner. Management of perioperative crisis is predicated on expert opinion and an understanding of a distinct physiologic milieu.

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Contribution: This author helped with generation, conception, writing, editing, and final approval of the content.
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Name: Sharon Einav, MD.
Contribution: This author helped with generation, conception, writing, editing, and final approval of the content.
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Name: Karl-Christian Thies, MD.
Contribution: This author helped with conception, writing, editing, and final approval of the content.
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Name: Mark E. Nunnally, MD.
Contribution: This author helped with generation, conception, writing, editing, and final approval of the content.
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Name: Andrea Gabrielli, MD.
Contribution: This author helped with conception, writing, editing, and final approval of the content.
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Name: Gerald A. Maccioli, MD.
Contribution: This author helped with conception, writing, editing, and final approval of the content.
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Name: Guy Weinberg, MD.
Contribution: This author helped with conception, writing, editing, and final approval of the content.
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Name: Arna Banerjee, MD.
Contribution: This author helped with conception, writing, editing, and final approval of the content.
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Name: Kurt Ruetzler, MD.
Contribution: This author helped with conception, writing, editing, and final approval of the content.
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Name: Gregory Dobson, MD.
Contribution: This author helped with conception, writing, editing, and final approval of the content.
Conflicts of Interest: None.
Name: Matthew D. McEvoy, MD.
Contribution: This author helped with conception, writing, editing, and final approval of the content.
Conflicts of Interest: None.
Name: Michael F. O'Connor, MD, FCCM.
Contribution: This author helped with generation, conception, writing, editing, and final approval of the content.
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This manuscript was handled by: Avery Tung, MD, FCCM.

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Cardiac Arrest in the Operating Room: Part 2—Special Situations in the Perioperative Period

Matthew D. McEvoy, MD,* Karl-Christian Thies, MD, FRCA, FERC, DEAA,† Sharon Einav, MD,‡ Kurt Ruetzler, MD,§‖ Vivek K. Moitra, MD, FCCM,¶ Mark E. Nunnally, MD, FCCM,# Arna Banerjee, MD,* Guy Weinberg, MD,** Andrea Gabrielli, MD, FCCM,†† Gerald A. Maccioli, MD, FCCM,‡‡ Gregory Dobson, MD,§§ and Michael F. O'Connor, MD, FCCM‖‖

As noted in part 1 of this series, periprocedural cardiac arrest (PPCA) can differ greatly in etiology and treatment from what is described by the American Heart Association advanced cardiac life support algorithms, which were largely developed for use in out-of-hospital cardiac arrest and in-hospital cardiac arrest outside of the perioperative space. Specifically, there are several life-threatening causes of PPCA of which the management should be within the skill set of all anesthesiologists. However, previous research has demonstrated that continued review and training in the management of these scenarios is greatly needed and is also associated with improved delivery of care and outcomes during PPCA. There is a growing body of literature describing the incidence, causes, treatment, and outcomes of common causes of PPCA (eg, malignant hyperthermia, massive trauma, and local anesthetic systemic toxicity) and the need for a better awareness of these topics within the anesthesiology community at large. As noted in part 1 of this series, these events are always witnessed by a member of the perioperative team, frequently anticipated, and involve rescuer–providers with knowledge of the patient and the procedure they are undergoing or have had. Formulation of an appropriate differential diagnosis and rapid application of targeted interventions are critical for good patient outcome. Resuscitation algorithms that include the evaluation and management of common causes leading to cardiac in the perioperative setting are presented. Practicing anesthesiologists need a working knowledge of these algorithms to maximize good outcomes. (Anesth Analg 2018;126:889–903)

From the *Department of Anesthesiology, Vanderbilt University Medical Center, Nashville, Tennessee; †Department of Anesthesiology, University Medical Centre Greifswald, Ferdinand-Sauerbruch-Straße, 17475 Greifswald, Germany; ‡Faculty of Medicine, Shaare Zedek Medical Center, Hebrew University, Jerusalem, Israel; Departments of §General Anesthesiology and ‖Outcomes Research, Cleveland Clinic, Cleveland, Ohio; ¶Department of Anesthesiology, Columbia University, College of Physicians and Surgeons, New York, New York; #Department of Anesthesiology, Perioperative Care, and Pain Medicine, New York University, New York, New York; **Department of Anesthesiology, The University of Illinois at Chicago, Chicago, Illinois; ††University of Pennsylvania, Philadelphia, Pennsylvania; ‡‡Department of Anesthesiology and Critical Care, Sheridan Healthcare, Florida; §§Department of Anesthesia, Dalhousie University, Halifax, Nova Scotia, Canada; and ‖‖Department of Anesthesia and Critical Care, University of Chicago, Chicago, Illinois.

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K.-C. Thies is currently affiliated with the Department of Anesthesiology, University Medical Center Greifswald, Ferdinand-Sauerbruch-Straße, Greifswald, Germany.

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Implication statement: The spectrum of causes of periprocedural cardiac arrest warrants specific adaptations of the advanced circulatory life support algorithms. A number of rare, life-threatening etiologies of profound hemodynamic and respiratory disturbance leading to cardiac arrest are reviewed. Good patient outcomes in these special situations can be achieved by vigilance, timely formulation of a differential diagnosis for the crisis, and adherence to best practices.

Reprints will not be available from the authors.

Address correspondence to Matthew D. McEvoy, MD, Department of Anesthesiology, Vanderbilt University Medical Center, 1301 Medical Center Dr, TVC 4648, Nashville, TN 37232. Address e-mail to matthew.d.mcevoy@vanderbilt.edu.

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Advanced cardiac life support (ACLS) was originally developed as an extension of basic life support with a focus on out-of-hospital cardiac arrest (OHCA).[1] OHCA is now recognized as a distinct entity from in-hospital cardiac arrest (IHCA), particularly in relation to more common etiology of arrest, average response rescue time, and survival.[2] As noted previously,[1] periprocedural cardiac arrest (PPCA) is different from both OHCA and medically related IHCA. The etiologies of the crisis, the perioperative team knowledge of the patient's comorbidities, the awareness of current physiological state, and the immediate rescue response time significantly improve restoration of spontaneous circulation and survival to discharge when compared to other forms of IHCA.[3–6]

In addition to these differences in clinical presentation and management, numerous studies have also demonstrated knowledge and skill deficiencies in the proper assessment and management of perioperative crises within the anesthesiology community.[7–12] Frequent and concise updates of the knowledge content necessary for managing high-stakes perioperative events is necessary for preparing anesthesiologists and perioperative teams to provide appropriate and timely care.[13,14] As noted in part 1, while previous publications have described cardiac arrest and crisis management in the operating room, the most recent update in ACLS prompted a part 1 review of the current literature concerning perioperative life-threatening crisis and cardiac arrest. Accordingly, the goal of this part 2 review is to offer an updated clinical perspective of cardiac arrest during the perioperative period. In part 1, we summarize the causes and outcomes of perioperative cardiac arrest, review concepts in resuscitation of the perioperative patient, and propose a set of algorithms to aid in the prevention and management of cardiac arrest during

the perioperative period. In this article, we discuss special anesthesia-related crises and the management thereof.

This review is focused on 8 special circumstances in the perioperative period that, while uncommon, are essential for all practicing anesthesiologists to know. The clinical scenarios presented are severe anaphylaxis, tension pneumothorax, local anesthetic systemic toxicity (LAST), malignant hyperthermia (MH), severe hyperkalemia, hypertensive crisis, trauma-related cardiac arrest, and pulmonary embolism (PE; thrombus or gas). Each scenario will be presented with a brief review of pathophysiology and epidemiology followed by recommendations on proper assessment, initial management, and subsequent management of each perioperative crisis based on a comprehensive review of the literature. The information presented in this article represents the background behind the management recommendations proposed in widely available crisis management checklists such as the Stanford and Harvard crisis checklists that are familiar to many practicing anesthesiologists.[15,16] It should be noted that these well-recognized clinical entities are presented as single cause of a life-threatening crisis and out of the clinical contest of more complex condition like septic shock or multiorgan system failure.

METHODS

An international group of 12 experts in the field of perioperative resuscitation has reviewed best available evidence on management of cardiac arrest and periprocedural crises. These experts were selected on the basis of several criteria: (1) clinical experience in anesthesiology and perioperative patient management; (2) expertise in simulation training in perioperative crises; (3) familiarity with the evidence behind current resuscitation guidelines; and (4) international representation (ensure that the recommendations are easily translatable to bedside practice in multiple clinical platforms). The group communicated via email, face-to-face meetings, and telephone. The papers selected for review were those included in the previous iteration of these guidelines[1] (which underwent repeat scrutiny) and relevant papers that had been published since 2012 and available in PubMed on the specific topics to be discussed. For part 2, disagreements among committee member were discussed as a group in an attempt to reach consensus, and in case of ongoing dissent, adjudicated by 3 of the authors (M.D.M., V.K.M., and M.F.O.).

The scenarios were chosen through a modified Delphi technique involving several rounds of input from the group. These scenarios were chosen because they represent perioperative emergencies that are likely to be immediately life threatening. Four of the topics briefly covered in a previous publication[1] were reanalyzed for a more in-depth discussion and updated knowledge (eg, severe anaphylaxis and hyperkalemia) or landmark publications (eg, trauma-related cardiac arrest). Due to constraints on length for the review article, the number of included scenarios was limited to 7. As such, the scenarios presented are not intended to be an exhaustive list.

Anaphylaxis

Pathophysiology and Epidemiology. Anaphylaxis is a severe, life-threatening systemic hypersensitivity reaction mediated by immunoglobulins IgE and IgG and accounts for about 500–1000 deaths per year in the United States.[17,18] The causative agent is usually not obvious, and assigning causality is typically complicated in the periprocedural and hospital setting, where patients are commonly exposed to multiple agents. Furthermore, anaphylactic reactions may occur with no documented prior exposure.[19] Hypersensitivity reactions are graded 1–5 corresponding to minor, low severity, life-threatening symptoms, cardiac or respiratory arrest, and death.[20,21] The overall incidence of hypersensitivity reactions is about 15 cases per 10,000 operations (95% confidence interval, 13–17 per 10,000).[22] The incidence of severe hypersensitivity reactions (grade 3–5) with life-threatening symptoms is about 2 cases per 10,000 operations.[22]

Presentation and Initial Assessment. Anaphylaxis is characterized by the rapid onset of potentially life-threatening airway, breathing, or circulatory problems. The initial symptoms are nonspecific. Rhinitis, tachycardia, confusion, altered mental status/presyncope, and skin and mucosal changes are common in the awake patient, but not always present.[23] In addition, bronchospasm is not present in all cases and does not necessarily precede cardiovascular instability. Extensive vasodilatation and increased vascular permeability lead to decreased cardiac preload with relative hypovolemia, which can in turn cause cardiovascular depression, myocardial ischemia, acute myocardial infarction, and malignant arrhythmias (anaphylactic shock).[24,25] When hemodynamic deterioration occurs rapidly and untreated, patients can experience cardiac arrest.[24,25] Evidence in the treatment of anaphylaxis is generally limited and is mostly based on case reports and extrapolations from animal models, nonfatal cases, interpretation of pathophysiology, and consensus opinion.[19]

Assessment and Initial Management Steps. When anaphylaxis is within the differential diagnosis, surgery should be interrupted, if possible, and the likely triggers of anaphylaxis should be immediately removed (eg, stopping an injection or infusion of medication or blood products).[26] Administration of epinephrine is indicated in patients with clinical features of anaphylaxis.[27,28] In the setting of signs and symptoms of severe anaphylaxis, 100–300 μg epinephrine should be given intravenously (IV) immediately with repeated and escalating doses as clinically indicated. We do not recommend to use the same epinephrine doses used in pulseless cardiac arrest (1 mg IV) if the patient maintains a cardiac rhythm with a pulse. Caution is warranted, as fatal dysrhythmias to large doses of epinephrine have been reported.[27,29] In patients without an IV line, early intramuscular administration of 300–500 μg epinephrine in the anterolateral aspect of the middle third of the thigh is recommended, with this dose being repeated every 5–15 minutes in the absence of clinical improvement.[30,31] Inhaled or subcutaneous administration of epinephrine is ineffective for severe anaphylaxis.[28] Close hemodynamic monitoring (eg, arterial blood pressure) with a goal systolic blood pressure (SBP) ≥90 mm Hg is indicated.

Immediate endotracheal intubation is critical and should not be delayed, as oropharyngeal and laryngeal edema are likely to occur rapidly.[32] If necessary, a surgical airway should be considered.[33] Initial fluid resuscitation using 20 mL/kg crystalloid infusions is indicated to treat the vasodilatory component of anaphylactic shock.[34,35]

Subsequent Assessment and Treatment Steps. If hemodynamic instability persists after initial epinephrine boluses, this drug should be continued by a carefully titrated continuous IV infusion (0.05–0.3 μg/kg/min) because the plasma half-life of epinephrine is brief (<5 minutes). If epinephrine infusion fails to restore normal hemodynamic variables, continuous infusions of vasopressin,[36,37] norepinephrine, methoxamine,[38] and metaraminol[39] may be considered. Glucagon should be considered in patients who have taken β-blockers and who are unresponsive to combined inotrope and vasopressors management.[40] Adjuvant use of antihistamines is appropriate,[41–43] and treatment with inhaled β2-adrenergic agents[27,44] and IV corticosteroids[28,45] should be considered in severe anaphylaxis. Extracorporeal life support (venous–arterial extracorporeal membrane oxygenation) has been successful in isolated cases and may be considered if clinical staff and equipment is immediately available. After stabilization, the patient should be monitored in an intensive care unit (ICU) for at least 24 hours due to the bimodal nature of severe anaphylaxis and a high risk of recrudescence. Finally, laboratory testing for histamine, tryptase, or IgE within 24 hours is indicated for diagnostic purposes.[46] Table 1 provides a full list of management steps.

Tension Pneumothorax

Epidemiology and Pathophysiology. A tension pneumothorax occurs when there is a "ball-valve effect" within the lung allowing progressive accumulation of air within the pleural space, which in turn leads to a corresponding increase in intrapleural and intrathoracic pressures. In tension pneumothorax, the intrapleural pressure is positive and exceeds the atmospheric pressure throughout the respiratory cycle. The incidence of tension pneumothorax remains poorly estimated and ranges from 1% to 3% in prehospital, major trauma, and ICU patients.[47]

The pathophysiology of tension pneumothorax differs between patients who are spontaneously breathing versus those on positive-pressure ventilation. In spontaneously breathing patients, several compensatory mechanisms likely prevent initial hemodynamic compromise. These factors include increasing respiratory rate, decreased tidal volume and negative-pressure contralateral chest excursions. These mechanisms may maintain arterial blood pressure by limiting transmitting pleural pressure to the mediastinum and contralateral hemithorax. In patients receiving positive-pressure ventilation, increased intrapleural pressure throughout the respiratory cycle produces a marked decrease in cardiac venous return, which leads to hypotension, and, if untreated, may result in cardiac arrest.[48]

Presentation and Initial Assessment. Spontaneously breathing patients with tension pneumothorax present with shortness of breath, dyspnea, tachypnea, respiratory distress, hypoxemia, and ipsilateral decreased air entry and percussion hyperresonance. In a large systematic review, the reported incidence of respiratory arrest (9%), hypotension (16%), and cardiac arrest (2%) were much lower compared to patients on positive-pressure ventilation.[49] Patients on positive-pressure ventilation usually present with

Table 1. Assessment and Management of Severe Anaphylaxis

Assessment	Confusion, altered mental status/presyncope Rash and/or rhinitis Perioral/periorbital edema, laryngeal edema, stridor Bronchospasm/dyspnea (not always present) Tachycardia Acute onset hypotension
Initial management	Prearrest Stop or remove the inciting agent or drug (eg, NMBD, antibiotics, blood products, IV contrast, or latex) If feasible, stop surgery or procedure Oxygen at F_{IO_2} of 1.0; intubate immediately for respiratory distress Watch for auto-PEEP if severe bronchospasm 100–300 μg epinephrine in repeated/escalating doses (or 300–500 μg IM if no IV access present) ±Vasopressin 2 U IV Start epinephrine infusion#8232;(0.05–0.3 μg/kg/min IV) for a goal SBP >90 mm Hg; observe for myocardial ischemia Vasopressin or norepinephrine infusions may be added in patients who are hypotensive in spite of high doses of epinephrine (eg, >2 mg IV) IV fluids/large bore access—initial treatment is bolus of 20 mL/kg IV of LR or PLA H1 blocker (50 mg diphenhydramine IV) H2 blocker (20 mg famotidine IV) ±Corticosteroid (eg, 50–150 mg hydrocortisone IV or methylprednisolone 1–2 mg/kg IV) Continuous arterial blood pressure monitoring as early as possible (systolic blood pressure ≥90 mm Hg) Arrest CPR if no carotid pulse detected for 10 s 100–1000 μg epinephrine IV, can repeat every 3–5 min or replace with 1 dose 40 U vasopressin IV If auto-PEEP suspected, disconnect the ventilator briefly Administer adjunctive therapies listed in prearrest (H1 and H2 blockers and corticosteroids) Consider extracorporeal life support in patients getting good CPR without ROSC
Subsequent management	Send blood for tryptase level to support the diagnosis Monitor in ICU for at least 24 h as there is a risk of recrudescence

Abbreviations: CPR, cardiopulmonary resuscitation; F_{IO_2}, fraction of inspired oxygen concentration; ICU, intensive care unit; IM, intramuscular; IV, intravenous; LR, lactated ringers; NMBD, neuromuscular blocking drug; PEEP, positive end-expiratory pressure; PLA, plasmalyte-A; ROSC, return of spontaneous circulation; SBP, systolic blood pressure.

hypoxemia, tachycardia, sudden onset of hypotension, subcutaneous emphysema, and ipsilateral decreased air entry. These signs are followed by circulatory collapse and subsequent cardiac arrest with pulseless electrical activity (PEA). Tension pneumothorax should always be in the differential diagnosis of a patient with acute decompensation during laparoscopic surgery.[50]

Traditionally, diagnosis relies on clinical signs and symptoms although these are unreliable (especially contralateral tracheal deviation and jugular venous distention). Thoracic ultrasonography, which is being used with increasing frequency, may be superior to chest radiography for diagnosing pneumothorax (sensitivity of approximately 80%–90% vs 50%) and can also be performed rapidly at the bedside.[51,52]

Initial Management Steps. Initial treatment should focus on maximizing oxygenation. Immediate tube thoracostomy by trained personnel is encouraged as the treatment of choice in both the ventilated and the spontaneously breathing patient.[53] However, it should be noted that in situations of high clinical suspicion of tension pneumothorax (eg, high airway pressures, unilateral breath sounds, and circulatory instability in the setting of pneumoperitoneum), immediate needle decompression would be recommended rather than delaying treatment.

Subsequent Assessment and Treatment Steps. After initial assessment and treatment, the patient should be stabilized to prevent further respiratory or cardiovascular compromise. The tube thoracostomy is left in place until the parenchymal injury that caused the tension pneumothorax has resolved. The underlying cause for the parenchymal injury needs to be ascertained. Occasionally surgical repair may be indicated. Resolution of the pneumothorax is documented with serial chest radiographs.

Local Anesthetic Systemic Toxicity

Epidemiology and Pathophysiology. While any use of local anesthetic can potentially lead to LAST, peripheral nerve block carries the highest risk, with published rates typically ranging from 1 to 10 per 10,000 qualifying this iatrogenic complication as a "rare event."[54] Nevertheless, the potential for severe, even fatal physiological sequelae demands that measures be taken to reduce the likelihood of LAST and that education/training include detection and treatment of this condition. In addition to using standard monitors and safety measures (eg, frequent aspiration during needle progression incremental injection), there is evidence that the use of ultrasound guidance can reduce the risk of LAST.[55]

Presentation and Initial Assessment. A wide range of either neurological symptoms (eg, seizure, agitation, or obtundation) or cardiovascular signs (eg, arrhythmia or conduction block, hypertension, tachycardia, or progressive hypotension and bradycardia) occur with LAST. A study of LAST episodes published from 1979 to 2009 showed that >40% of cases departed from the standard text book presentation (eg, rapid-onset seizure potentially leading to cardiac arrest).[56] In 35 of 93 patients (38%), symptoms were delayed >5 minutes, and in 10 patients (11%), cardiovascular signs occurred without a neurological prodrome. Another study from the same group indicated that there is a wide variety of clinical presentations in cases of LAST, including an increase in delayed onset (52%; >5 minutes from injection), which is likely a result of ultrasound guidance.[57]

Initial Management Steps. The initial focus in treating LAST includes managing the airway to assure adequate oxygenation and ventilation and using a benzodiazepine to suppress seizures. Early treatment of LAST by infusion of lipid emulsion 20% can prevent progression to cardiovascular compromise[58] possibly by reducing peak local anesthetic levels.[59] Propofol is cardiodepressant, and its lipid content is inadequate to confer benefit. It is important to continue monitoring even after symptoms resolve because recurrence or delayed progression can occur after an interval of apparent stability.[60]

Subsequent Assessment and Treatment Steps. If LAST progresses to cardiovascular collapse, it is important to administer high-quality cardiovascular support since improving coronary and cerebral blood flow reduces local anesthetic tissue concentrations both directly and by delivering lipid emulsion to affected sites. The main benefit of infusion of lipid emulsion in reversing LAST is accelerating redistribution of local anesthetic, rapidly shuttling drug from sites of toxicity (brain and heart) to unaffected organs (eg, liver and skeletal muscle). This scavenging effect is the result of both partitioning into the lipid phase and the direct inotropic effect of lipid emulsion infusion.[61] The direct inotropy is seen in intact rats and isolated heart without a pharmacotoxic challenge; however, during experimental LAST, it only occurs after myocardial bupivacaine content drops below a specific (eg, channel blocking) threshold. Lipid infusion also exerts a postconditioning effect that might contribute to successful resuscitation.[62] It is important to consider extracorporeal life support relatively early in those instances in which the patient does not respond to more conservative measures. Postevent monitoring should occur for at least 6 hours because cardiovascular instability can recur after initial recovery. Table 2 provides a full list of management steps.

Malignant Hyperthermia

Epidemiology and Pathophysiology. MH is an extreme reaction to volatile anesthetics and succinylcholine, which is attributed to abnormalities of skeletal muscle metabolism and calcium disposition. Its occurrence is rare, ranging between 1:62,000 and 1:500,000 anesthetics, more commonly occurring in men and younger patients, but described in a wide variety of patients.[63,64] The pathophysiology of this syndrome involves mainly cytoplasmic proteins participating in the movement of calcium within skeletal muscle, most commonly the ryanodine receptor. However, many genetic abnormalities are associated with MH, both inherited or sporadic. The syndrome is marked by extreme muscle hypermetabolism, leading to muscle necrosis, hyperpyrexia, acidosis, and in extreme cases, cardiac arrest.

Presentation and Initial Assessment. Because of its rarity, MH can be a once-in-a-career event. Mortality without dantrolene treatment is as high as 80%, but with it, it

Table 2. Assessment and Management of Local Anesthetic Systemic Toxicity

Assessment	Seizures, agitation, obtundation Tachycardia Bradycardia or new heart block All in setting of local anesthetic administration
Initial management	Prearrest Stop the administration of local anesthetic Immediate tracheal intubation and ventilation with 100% oxygen Consider transcutaneous or intravenous pacemakers for all symptomatic bradycardic rhythms with pulse If the diagnosis of local anesthetic toxicity is strongly suspected, the use of epinephrine should be avoided as it can worsen outcome 20% lipid emulsion 1.5 mL/kg IV load, then 0.25 mL/kg/min (~20 mL/min) If still with hemodynamic instability, a second bolus followed by a doubling of the rate of infusion is appropriate (0.5 mL/kg/min) Seizures should be treated with benzodiazepines. Small doses of propofol or thiopental may be used if benzodiazepines are not immediately available Cardiac arrest Immediate CPR as indicated (no carotid pulse, ECG, arterial catheter, and pulse oximeter signal) If the diagnosis of local anesthetic toxicity is strongly suspected, small doses of 10–100 μg epinephrine IV are preferable to higher doses Vasopressin is not recommended Sodium bicarbonate to maintain a pH >7.25 in patients without immediate ROSC after CPR and drug therapy If ROSC does not occur after the first bolus of lipid emulsion, a second bolus followed by a doubling of the rate of infusion is appropriate Consider therapy with H1 and H2 blockers Amiodarone is the drug of choice for ventricular arrhythmias Lidocaine should be avoided Most important, continue CPR for a prolonged period (we suggest at least 60 min) as very good neurological recovery has been reported in patients after very prolonged cardiac arrests from local anesthetic overdoses Extracorporeal life support is appropriate in circumstances where the diagnosis is certain, where ECMO is available in a timely fashion, and where there is no ROSC after a second bolus of lipid emulsion
Subsequent management	Monitor for recurrence or delayed progression

Abbreviations: CPR, cardiopulmonary resuscitation; ECG, electrocardiogram; ECMO, extracorporeal membrane oxygenation; IV, intravenous; ROSC, return of spontaneous circulation.

may be as low as 1.4%.[65] Because the time to dantrolene administration correlates with morbidity and mortality, early recognition is crucial to an effective response. The earliest signs of MH are hypercapnia and sinus tachycardia. Masseter muscle spasm, general muscle rigidity, tachypnea, and rising temperature (late) are additional common findings. Blood gas analysis can reveal respiratory and metabolic acidosis, especially when drawn from a vein draining a large muscle bed.

Initial Management Steps. When MH is suspected, all triggering agents should be immediately discontinued. Dantrolene 2.5 mg/kg IV is the key therapy for MH. Several formulations exist, and providers should be familiar with preparation and administration of a normal adult dose in anticipation of an MH event. Dantrolene should be available at all places that triggering anesthetic agents are available. Regular monitoring of arterial $Paco_2$, temperature, and lactate levels should accompany dantrolene administration, and any abnormalities should be aggressively treated with external and internal cooling, ventilation, and fluid resuscitation.

Subsequent Assessment and Treatment Steps. After initial recognition and treatment, the goals of care involve the mitigation of ongoing tissue injury, hyperthermia, and their sequelae. With extremes of temperature (median temperature, 40.3°C), disseminated intravascular coagulation may occur.[4] Other complications can occur at any temperature, but mortality correlates with temperature.[5] Rhabdomyolysis is common; if severe, it can lead to renal failure and hyperkalemia. Cooling and monitoring for these complications should continue for 72 hours after a suspected episode, because of the risk of recrudescence. Because dantrolene interferes with calcium disposition, patients should be monitored for muscle weakness. Importantly, calcium channel blockers are contraindicated in the setting of dysrhythmias. MH resources are available through expert groups in the United States (www.mhaus.org) and in Europe (www.emhg.org). Table 3 provides a full list of management steps.

Severe Hyperkalemia

Epidemiology and Pathophysiology. The exact cutoff for moderate or severe hyperkalemia is inconsistently described in the literature.[66] However, recent reports note that initiation of emergency therapies are recommended for serum potassium levels >6.0 or 6.5 or electrocardiographic (ECG) manifestations of hyperkalemia, regardless of potassium level.[67] Of note, a potassium level of ≥6.5 mmol/L occurs in only 0.1% of hospitalized patients.[68] Acidosis (primarily metabolic), for example, promotes an extracellular potassium shift; each 0.1 unit decrease in pH is accompanied by an increase of ~0.6 mmol/L in serum potassium.[69,70] The most common causes of hyperkalemia are renal pathology and drug therapy.[68,71,72] There are limited data on the prevalence of hyperkalemia in adult patients undergoing surgery and anesthesia. However,

Table 3. Assessment and Management of Malignant Hyperthermia

Assessment	Hypercapnia Tachycardia Tachypnea in nonparalyzed patients Muscle rigidity/masseter spasm Hyperthermia
Initial management	Discontinue triggering volatile anesthetics and switch from the anesthesia ventilator to manual Ambu bag ventilation from a separate source of oxygen. If available, switch to a dedicated clean anesthesia ventilator or transport or ICU ventilator when feasible. Continue $Etco_2$ monitoring Stop surgery or procedure when feasible If necessary, switch to intravenous anesthetic Sodium dantrolene: give 2.5 mg/kg or 1 mg/lb initial dose. Repeat bolus of Na dantrolene, titrating to tachycardia and hypercarbia (10 mg/kg suggested upper limit, but more may be given as needed, up to 30 mg/kg) Begin active cooling: ice packs to groin, axilla, and neck; cold intravenous solutions into the peritoneal cavity when feasible; nasogastric or peritoneal lavage when feasible Stop cooling measures at 38°C to avoid overshooting If hyperkalemia suspected by peaked ECG T waves or intraventricular conduction delay confirmed by high K serum level: 10 mg/kg calcium chloride, 0.1 U/kg insulin, 50 mL D50w for adult or 1 mL/kg for pediatrics. Repeat as necessary Metabolic acidosis: 100 mEq of $HCO3^-$ in adults, then titrate to pH 7.2. Normalize pH if confirmed rhabdomyolysis (suggested threshold, 10,000 IU/L CPK)#8232; Respiratory acidosis: treatment is controversial due to adverse hemodynamic effects of hyperventilation if low-flow state is confirmed. (We suggest an initial goal of modest permissive hypercarbia with a goal $Etco_2$ of 50–60 mm Hg) Dysrhythmias: avoid calcium antagonists after Na dantrolene, potential for worsening hyperkalemia Myoglobinuria with oliguria: place Foley catheter; increase rate of fluid resuscitation Invasive pressure monitoring when feasible, more $HCO3^-$ to neutralize urine pH, consider intravenous mannitol Supportive measures for disseminated intravascular coagulation Call for help, including the MH hotline, if feasible (www.mhaus.org), call 1–800-644-9737 or 1-800-MH-HYPER in the United States and Canada; outside the United States, call 00112094173722
Subsequent management	Monitor for recrudescence for 72 h and treat/cool as required When the crisis is resolved: consider caffeine–halothane muscle biopsy in vitro contracture test, molecular genetic testing for genetic mutation analysis for patient's relatives (sensitivity 25%)

Adapted from http://www.mhaus.org/.

Abbreviations: CPK, creatine phosphokinase; D50w, dextrose in water (50%); ECG, electrocardiogram; $Etco_2$, end-tidal carbon dioxide; ICU, intensive care unit; K, potassium; MH, malignant hyperthermia.

hyperkalemia is consistently estimated to be the cause of death in 1%–2% of cases of anesthesia-related cardiac arrests in children.[73–75]

Presentation and Initial Assessment. Clinical manifestations of this potentially life-threatening electrolyte disorder are mostly insidious and nonspecific. Thus, preoperative assessment of patients at risk should include timely blood testing. There is a common misconception that the cardiac manifestations of hyperkalemia are well known and occur in an orderly fashion. On the contrary, the cardiac clinical symptoms of hyperkalemia may randomly range from nonexistent to vertigo, chest pain, and presyncope to syncope and cardiac arrest. Physical examination may reveal bradycardia and/or bradyarrhythmia and hypotension.[76] Accompanying ECG changes include peaked T-waves, QRS widening, diminished P waves[77,78], and/or a range of arrhythmias including bradycardia,[79] atrioventricular blocks at different conduction levels,[80–82] ventricular tachycardia,[83] and ventricular fibrillation.[84–86] Absence of ECG changes should not be taken to indicate that blood potassium levels are normal; some patients with end-stage renal disease do not exhibit ECG changes in the presence of hyperkalemia because of a protective effect of calcium fluctuations.[87–89] Neurological manifestations include generalized muscle weakness and respiratory failure due to flaccid muscle paralyses.[90–92]

Abnormal ECG findings should command immediate attention and treatment when highly suggestive of severe hyperkalemia. Cardiac arrest caused by hyperkalemia has been shown to be associated with accompanying ECG changes, multiorgan system failure, and emergent admission.[93] Therefore, perioperative management of life-threatening hyperkalemia depends on whether surgery is elective or urgent and on the perioperative timing of the finding. With the widespread availability of sugammadex, succinylcholine should be avoided or used with great caution if there is any concern for the acute development of hyperkalemia (eg, patients with muscle wasting from neurological injury or those who have been immobile for days in the ICU).[94]

Initial Management Steps. The first management step is avoidance of hyperkalemia and thus postponing of elective surgical cases in the setting of this condition and avoiding succinylcholine and prolonged propofol infusions for urgent/emergent cases with known hyperkalemia.[86,95–97] Respiratory acidosis should be corrected normalizing ventilation. Acute hyperventilation is to be avoided because it can contribute to hypotension by reducing venous return. Treatment with β-2 agonists (eg, salbuterol) and glucose with insulin can be initiated to promote potassium shift toward the intracellular compartment.[98–101] Combined therapy with β-2 agonists and insulin is more effective than a single agent.[102] The literature supports administration of calcium as a membrane stabilizer when ECG changes are present.[102] In the setting of ongoing hemorrhage and blood administration (with citrate), preventative therapy with calcium may also be deemed justifiable.

Subsequent Assessment and Treatment Steps. If patient volume status is considered adequate and his or her renal

function permits, loop diuretics may be administered in the hope of inducing potassium loss. Early and aggressive correction of potassium is important to avoid deterioration to cardiac arrest.[93] Moderate quality evidence (retrospective observation) supports treatment with IV calcium chloride during adult hyperkalemic cardiac arrest.[103] The use of bicarbonate to enhance intracellular shift of potassium is controversial.[103,104] Selection bias may underlie the association of both therapies with poor cardiopulmonary resuscitation (CPR) outcomes, since both drugs are more likely to be used in critically ill patients and after prolonged CPR.[104] If hyperkalemia is considered reversible, bridging therapy with extracorporeal life support should be considered.[105] Hemodialysis should be initiated as soon as possible after return of spontaneous circulation.[106,107] There have been reports of successful outcome from hyperkalemic cardiac arrest with hemodialysis being initiated even during CPR.[108–111] Given that vascular access is often easily available in the operating room, blood purification is a pertinent option should hyperkalemic cardiac arrest occur perioperatively. Table 4 provides a full list of management steps.

Traumatic Cardiac Arrest

Epidemiology and Pathophysiology. Traumatic cardiac arrest (TCA) carries a high mortality rate, but in survivors, the neurological outcome appears to be much better than in other causes of cardiac arrest.[112,113] Uncontrolled hemorrhage is the main cause of death (48%), followed by tension pneumothorax (13%), asphyxia (13%), and pericardial tamponade (10%).[114] A large systematic review reported an overall survival rate of 3.3% in blunt and 3.7% in penetrating trauma, with good neurological outcome in 1.6% of all cases.[112]

Presentation and Initial Assessment. Patients in TCA present with loss of consciousness, agonal or absent spontaneous respiration, and absence of a femoral or carotid pulse. The prearrest state is characterized by tachycardia, tachypnea, decreased pulse pressure, and a deteriorating conscious level. Hypotension may present late and beyond 1500 mL of blood loss. Beyond this stage (class III hemorrhagic shock), peripheral pulses will become absent, and the patient left untreated will typically proceed to pulseless electrical dissociation or asystolic cardiac arrest. Resuscitative efforts in TCA should focus on immediate assessment and simultaneous treatment of the hemorrhage and surgical control of the reversible causes (Figure 1).[113,115]

Initial Management Steps. Short prehospital times are associated with increased survival rates for major trauma and TCA. The time elapsed between injury and surgical control of bleeding should be minimized. When feasible, the patient should be immediately transferred to a designated trauma center for damage control resuscitation (DCR).[116] "Scoop and run" for these patients may be a better choice for survival than engaging on a long resuscitation on the field. While anesthesiologists in many international settings may be involved with prehospital care, being prepared to manage the airway and provide aggressive fluid resuscitation on patient arrival to the emergency room is also paramount. Successful treatment of TCA requires a team approach with all measures carried out rather in parallel than sequentially. The emphasis lies on rapid treatment of all potentially reversible pathology. In cardiac arrest caused by hypovolemia, cardiac tamponade, or tension pneumothorax, chest compressions alone are unlikely to be as effective as in normovolemic cardiac arrest.[115,117–119] Therefore, chest compressions take a lower priority than the immediate treatment of reversible causes.

Ultrasonography should be used in the evaluation of the compromised trauma patient to target life-saving interventions if the cause of shock cannot be established clinically.[116,120]

Table 4. Assessment and Management of Severe Hyperkalemia

Assessment	Vertigo Chest pain Syncope Bradycardia Diminished p-waves on ECG Peaked T-waves on ECG Wide complex QRS on ECG Heart block on ECG Ventricular tachycardia Ventricular fibrillation
Initial management	Cardiac protection Administer calcium chloride or calcium gluconate 1–2 g IV. Repeat as required for ECG signs of hyperkalemia Interventions to drive potassium into intracellular space (these are temporizing interventions) Administer 1 ampule of D50 and 10 units of insulin IV Administer 50 mEq of sodium bicarbonate Administer 4–10 puffs albuterol Interventions to eliminate potassium or increase corporeal capacity Administer 20–40 mg furosemide IV, monitor urine output in response. Increase to 1–1.5 mg/kg if oliguric response Administer 30 or 60 g of kayexalate OG/NG/PR. Repeat as needed Initiate renal replacement therapy Transfuse washed pRBC (these units are hypokalemic and will avidly absorb serum potassium) In patients with hyperkalemic cardiac arrest, extracorporeal life support is appropriate while the cause of hyperkalemia is being treated and the patient is undergoing treatment to definitively lower their serum potassium
Subsequent management	Monitor serum potassium serially, continue to treat cause(s) of hyperkalemia

Abbreviations: ECG, electrocardiogram; IV, intravenous; OG, orogastric; NG, nasogastric; PR, per rectum; pRBC, packed red blood cell.

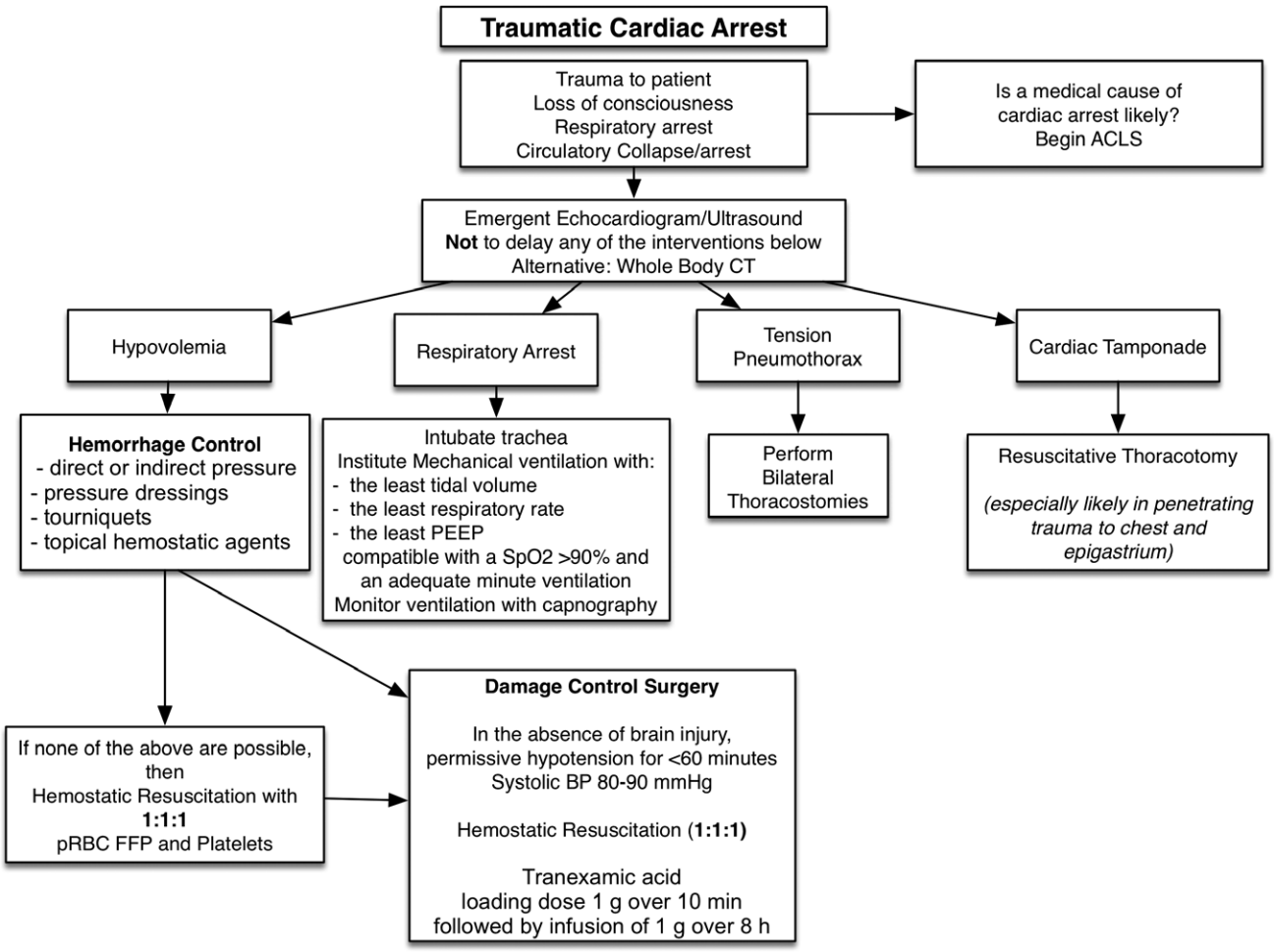

Figure 1. This figure illustrates the appropriate steps in the assessment and management of a patient experiencing traumatic cardiac arrest. ACLS indicates advanced cardiac life support; BP, blood pressure; CT, computed tomography; FFP, fresh frozen plasma; PEEP, positive end-expiratory pressure; pRBC, packed red blood cell.

Hemoperitoneum, hemothorax or pneumothorax, and cardiac tamponade can be diagnosed reliably in minutes. Early whole-body computed tomography scanning as part of the primary survey may improve outcome in major trauma.[121] Whole-body computed tomography is increasingly employed to identify the source of shock and to guide subsequent hemorrhage control. Figure 1 shows the traumatic cardiac (peri-) arrest algorithm of the European Resuscitation Council, which is based on the universal ALS algorithm.[122]

Hypovolemia. The treatment of severe hypovolemic shock has several elements. The main principle is to achieve immediate hemostasis. Temporary hemorrhage source control can be lifesaving. External hemorrhage can be treated with direct or indirect compression, pressure dressings, tourniquets, and topical hemostatic agents.[116] Noncompressible hemorrhage is more difficult to control. External splints/pressure, blood and blood products, IV fluids, and tranexamic acid (TXA) can be used during patient transport and until hemorrhage is controlled surgically. Resuscitative endovascular balloon occlusion is a promising alternative to aortic cross-clamping or manual aortic compression in patients exsanguinating from noncompressible torso injuries and can serve as a bridge to definitive hemorrhage control.[123,124] If the patient is in hypovolemic TCA, immediate restoration of the circulating blood volume with blood products is mandatory. Hyperventilation should be avoided in hypovolemic patients since positive-pressure ventilation may worsen hypotension by impeding venous return to the heart.[125] Therefore, low tidal volumes and slow respiratory rates may be associated with a more acceptable circulation.

Hypoxemia. Hypoxemia due to airway obstruction and loss of ventilator drive has been reported as the cause of 13% of all TCAs.[114] Immediate control of the airway and effective invasive ventilation can reverse hypoxic cardiac arrest. However, positive-pressure ventilation should be applied with caution to limit its deleterious effect on venous return. Oxygen should be delivered at a fraction of 1.0, and ventilation should be monitored with capnography to avoid hyperventilation.[116]

Cardiac Tamponade and Resuscitative Thoracotomy. Cardiac tamponade is the underlying cause of approximately 10% of cardiac arrests in trauma.[114] Where there is TCA and penetrating trauma to the chest or epigastrium, immediate resuscitative thoracotomy (RT; via a clamshell incision) can be life saving.[126,127] The chance of survival from cardiac injury is about 4 times higher for stab wounds than for a

gunshot wounds.[128] In 2012, an evidence review with resultant guidelines stated that RT should also be applied for 3 other categories of life-threatening injuries after arrival in hospital, which include blunt trauma with <10 minutes of prehospital CPR, penetrating torso trauma with <15 minutes of CPR, and penetrating trauma to the neck or extremity with <5 minutes of prehospital CPR.[129] The guidelines estimate survival rates of approximately 15% for RT in patients with penetrating wounds and 35% for patients with a penetrating cardiac wound. In contrast, survival from RT after blunt trauma is dismal, with reported survival rates of 0%–2%.[129,130] In the setting of the above presentation (ie, cardiac arrest with penetrating trauma), the prerequisites for a successful RT can be summarized as "4 Es rule" (4E):

(1) Expertise: RT teams must be led by a highly trained and competent health care practitioner.
(2) Equipment: adequate equipment to carry out RT and to deal with the intrathoracic findings is mandatory.
(3) Environment: ideally, RT should be carried out in an operating theatre; RT should not be carried out if there is inadequate physical access to the patient or if the receiving hospital is not easy to reach.
(4) Elapsed time: the time from loss of vital signs to commencing an RT should not be >10 minutes.

If any of the 4 criteria are not met, RT is likely less effective and exposes the team to unnecessary risks.[131]

Subsequent Management and Treatment. Damage control resuscitation is a term recently adopted in trauma resuscitation to improve outcome of uncontrolled hemorrhages. DCR combines permissive hypotension and hemostatic resuscitation with limited (damage control) surgical repair. Limited evidence and general consensus (https://www.nice.org.uk/guidance/ta74) have supported a conservative approach to IV fluid infusion, with permissive hypotension until surgical hemostasis is achieved.[132] In the absence of invasive monitoring, fluid resuscitation is titrated to maintain a radial pulse.[133,134] Hemostatic resuscitation with blood and blood products is used as primary resuscitation fluids to prevent exsanguination, dilution of hemostatic blood components, and trauma-induced coagulopathy.[135] The typical massive transfusion protocol recommends packed red blood cells, fresh frozen plasma, and platelets ratio of 1–2:1:1.[136]

Simultaneous damage control surgery and hemostatic resuscitation using massive transfusion protocol are the principles of DCR in patients with exsanguinating injuries.[116,135] Although

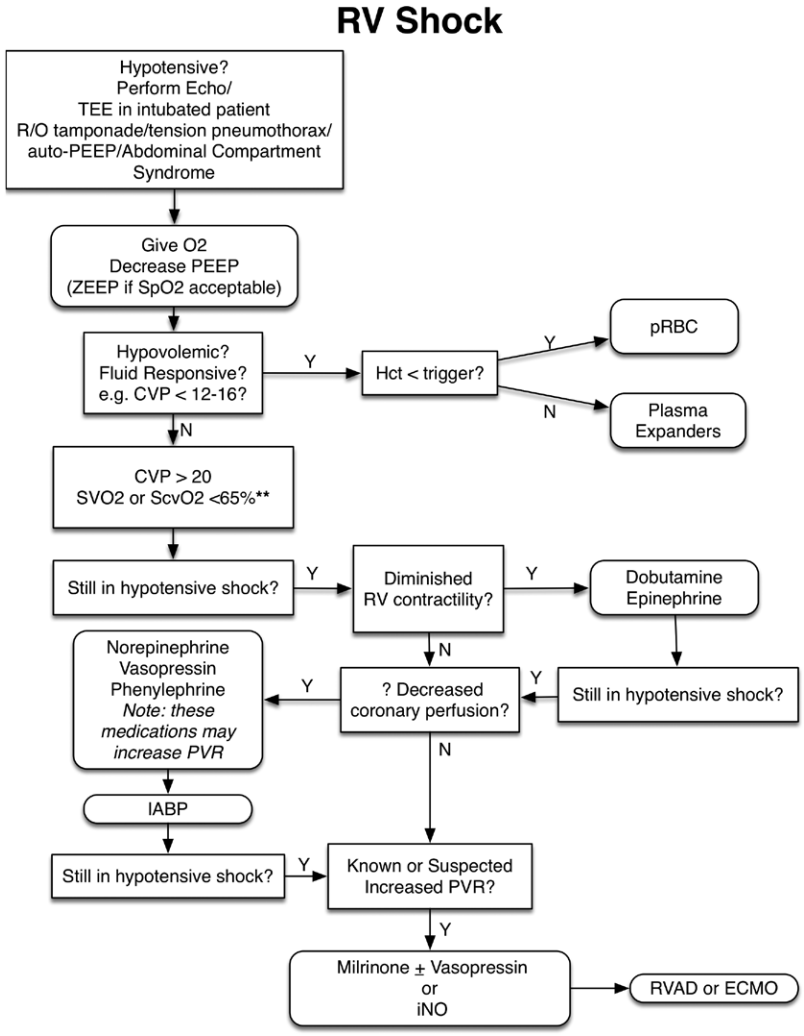

Figure 2. An assessment and management algorithm of right heart failure, which should be followed in the case of suspected thrombotic or gaseous pulmonary embolism. CVP indicates central venous pressure; ECMO, extracorporeal membrane oxygenation; Hct, hematocrit; IABP, intra-aortic balloon pump; iNO, inhaled nitric oxide; PEEP, positive end-expiratory pressure; pRBC, packed red blood cells; PVR, pulmonary vascular resistance; RV, right ventricular; RVAD, right ventricular assist device; Scvo2, central venous oxygen saturation; Spo2, peripheral oxygen saturation; SVO2, venous oxygen saturation; TEE, transesophageal echocardiography.

the evidence for permissive hypotension during resuscitation is limited, particularly with regards to blunt trauma, permissive hypotension has been endorsed in both civilian (https://www.nice.org.uk/guidance/ta74) and military care, generally aiming for an SBP of 80–90 mm Hg.[137] Caution is advised for patients with traumatic brain injury in whom a raised intracranial pressure may require a higher cerebral perfusion pressure. Specifically, the most recent Brain Trauma Foundation Guidelines for severe traumatic brain injury recommend maintaining an SBP ≥100 mm Hg for patients 50–69 years of age or ≥110 mm Hg for patients 15–49 years of age or older than 70 to improve outcomes and reduce mortality.[138]

Finally, TXA (loading dose 1 g over 10 minutes followed by infusion of 1 g over 8 hours) increases survival from traumatic hemorrhage.[139,140] It is effective when administered within the first 3 hours after trauma; however, TXA should not be started any later than 4 hours after the injury because late dosing is associated with increased mortality.

Pulmonary Embolism

Epidemiology and Pathophysiology. Thromboembolism, venous gas embolism, and fat embolism are all well-recognized complications that can occur during anesthesia and surgery. Venous thromboembolism is the most common cause of PE in periprocedural patients. Prophylaxis reduces its incidence, but cannot entirely prevent its occurrence.[141] Thromboembolism causes circulatory crisis via a combination of mechanical obstruction and the release of inflammatory mediators, both of which increase the right ventricular (RV) afterload.[142] In severe cases, the associated increase in pulmonary vascular resistance is so great that the right ventricle is unable to maintain the cardiac output. As the RV fails, it typically dilates, and the interventricular septum flattens and shifts toward the left ventricle.

Presentation and Initial Assessment. Signs of PE under general anesthesia include the following: unexplained hypotension with concurrent decrease in $Etco_2$; desaturation that is only moderately responsive to increased FIO_2; transitory bronchospasm with increased airway resistance; rapid changes of heart rhythm (often dysrhythmias or bradycardia after a transitory tachycardia); unexplained increased of central venous pressure or all pulmonary pressures; and rapid progression to nonshockable cardiac arrest (usually PEA).

Management Steps. A strategy for managing RV shock in this situation is proposed in Figure 2. In approximately 5% of cases, acute thromboembolism causes cardiac arrest, most often PEA.[143,144] Echocardiography of the patient with RV shock will typically reveal RV dilation and dysfunction, with an underfilled left ventricle.[145]

The management of intraoperative or perioperative thromboembolism is highly dependent on the procedure and patient. Therapeutic options range from supportive measures only to anticoagulation to thrombolysis.[143,144,146,147]

Epidemiology and Pathophysiology. Gas embolism is an important cause of circulatory crisis and cardiac arrest in perioperative patients. As the number of procedures in which minimally invasive techniques involving gas insufflation increases, the frequency of intraoperative gas embolisms will likely increase.[148] The risk for a venous air embolism increases when the surgical field is above the right atrium, particularly in patients with central venous pressure. The focus of hemodynamic support is on improving RV function.[52]

Common causes of gas embolism include laparoscopy, endobronchial laser procedures, central venous catheterization or catheter removal, hysteroscopy, pressurized wound irrigation, prone spinal surgery, posterior fossa surgery in the sitting position, and endoscopic retrograde cholangiopancreatography. Nonoperative cause of vascular air embolism includes direct vascular access procedures and pressurized hemoperfusion.

Management Steps. All surgical procedures at risk of venous gas embolism should be specifically monitored. Right parasternal precordial Doppler ultrasound has very high sensitivity for air embolism (88%).[149] Transesophageal echocardiography allows for recognition of air embolism size and location and assessment of ventricular function, but can be difficult or impossible to perform with some patient positions (eg, sitting) or procedures (eg, endoscopic retrograde cholangiopancreatography).[150] Massive gas embolisms in awake patients have been characterized by breathlessness, continuous coughing, arrhythmias, myocardial ischemia, acute hypotension with loss of end-tidal carbon dioxide, and cardiac arrest. Patients who survive any kind of an embolic event are likely to require continued evaluation and management for several hours in an ICU setting. Table 5 provides a full list of management steps.

Table 5. Assessment and Management of Massive or Submassive Pulmonary Embolism

Assessment	Significant hemodynamic instability or collapse during cases with high risk of thrombotic or air embolus
	$Etco_2$ that suddenly declines
	Increased airway pressures
	Consider early use of TEE to confirm diagnosis/rule out other treatable causes of pulmonary embolism
Initial management	Prearrest
	If possible, stop the infusion of the gas or ask the surgeon to flood the surgical field
	Administer 100% oxygen and intubate for significant respiratory distress or refractory hypoxemia
	Place patient in Trendelenburg (head down) position and rotate toward the left lateral decubitus position
	Maintain BP with fluid resuscitation and vasopressors/β-adrenergic agents if necessary. (See the algorithm for RV failure)
	Consider transfer to a hyperbaric chamber if immediately available
	Cardiac arrest
	Circulatory collapse should be addressed with CPR and consideration of CPB/emergent thrombectomy if available
Subsequent management	Consider the right ventricular shock algorithm

Abbreviations: BP, blood pressure; CPB, cardiopulmonary bypass; RV, right ventricular; CPR, cardiopulmonary resuscitation; TEE, transesophageal echocardiography.

Postresuscitation Management

It is beyond the scope of this article to detail the appropriate steps of postresuscitation management. Current guidelines are available that specify the appropriate management steps to maximize discharge from the hospital with a favorable neurological function as most important outcome parameters. Neurological, cardiovascular, and respiratory dysfunction are best managed in specialized ICUs where monitoring of electroencephalogram, targeted temperature management, glucose management, correction of electrolytes, and management of blood gas parameters are all promptly available.[151–153]

CONCLUSIONS

The causes, logistics, and management of periprocedural crises and arrest differ substantially from those taught in the American Heart Association ACLS guidelines. Furthermore, current evidence illustrates the need for educational updates on concerning PPCA among the anesthesiology community, including review of current evidence, use of checklists, and simulation.[11,154–157] The purpose of this review is to present the latest evidence and practical recommendations for managing 7 high-stakes perioperative events that can lead to significant circulatory disturbance and PPCA. These are core topics for all practitioners who care for patients in the periprocedural setting, but they by no means represent an exhaustive list of emergency conditions. It is incumbent upon all anesthesiologists to have a working knowledge of these clinical scenario and understanding of the current therapeutic options to maximize patient outcomes.

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