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

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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

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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

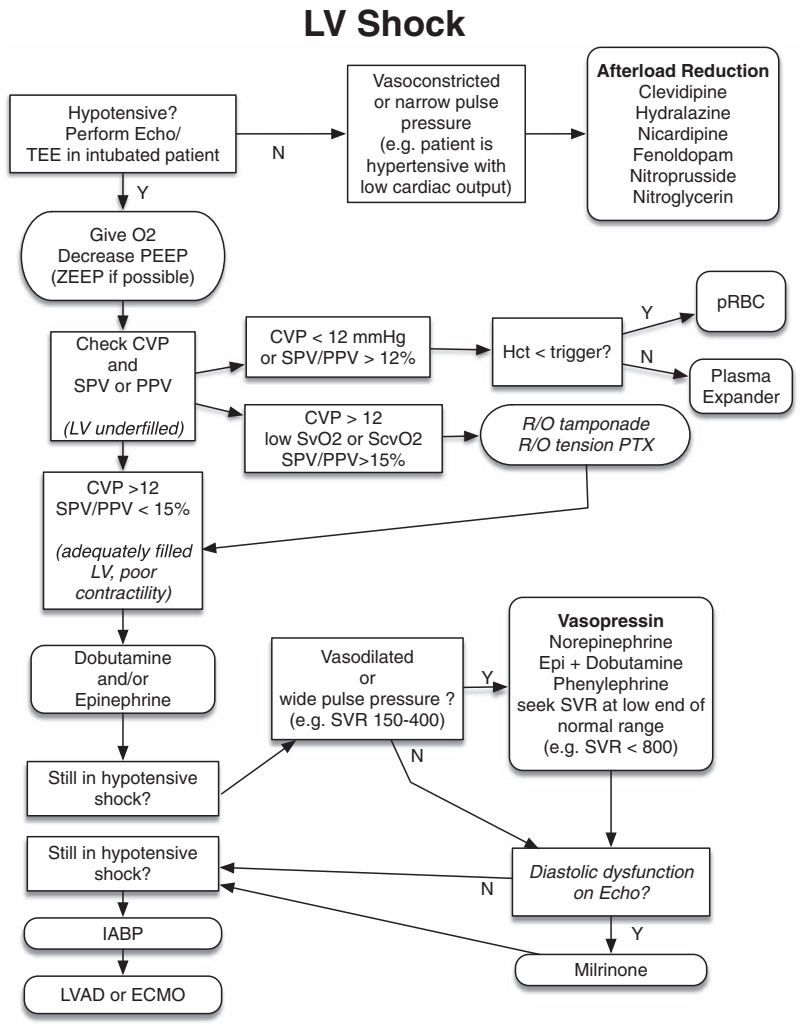

Figure 1. Treatment algorithm of LV failure with cardiogenic shock. CVP indicates central venous pressure; ECMO, extracorporeal membrane oxygenation; Hct, hematocrit; IABP, intraaortic balloon pump; LV, left ventricular; LVAD, left ventricular assist device; PEEP, positive end-expiratory pressure; PPV, 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

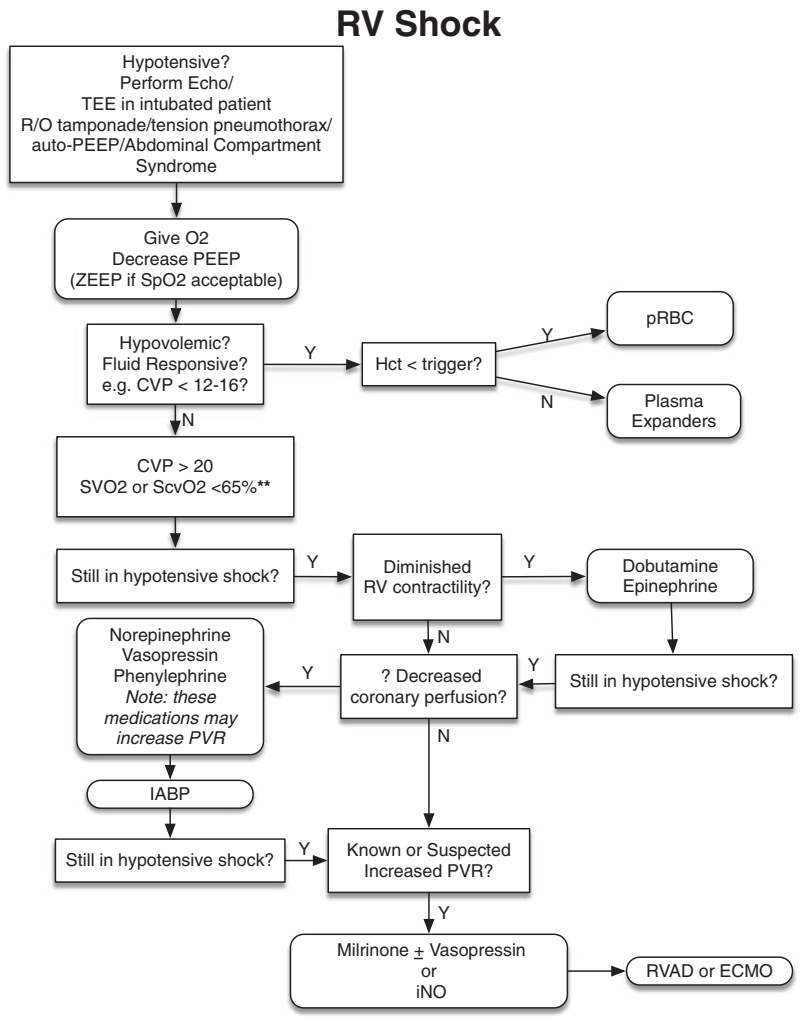

Figure 2. Treatment algorithm of RV failure with cardiogenic shock. CVP indicates central venous pressure; ECMO, extracorporeal membrane oxygenation; Hct, hematocrit; IABP, intraaortic balloon pump; iNO, inhaled nitric oxide; PEEP, positive end-expiratory pressure; pRBC, packed red blood cell; PVR, pulmonary 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

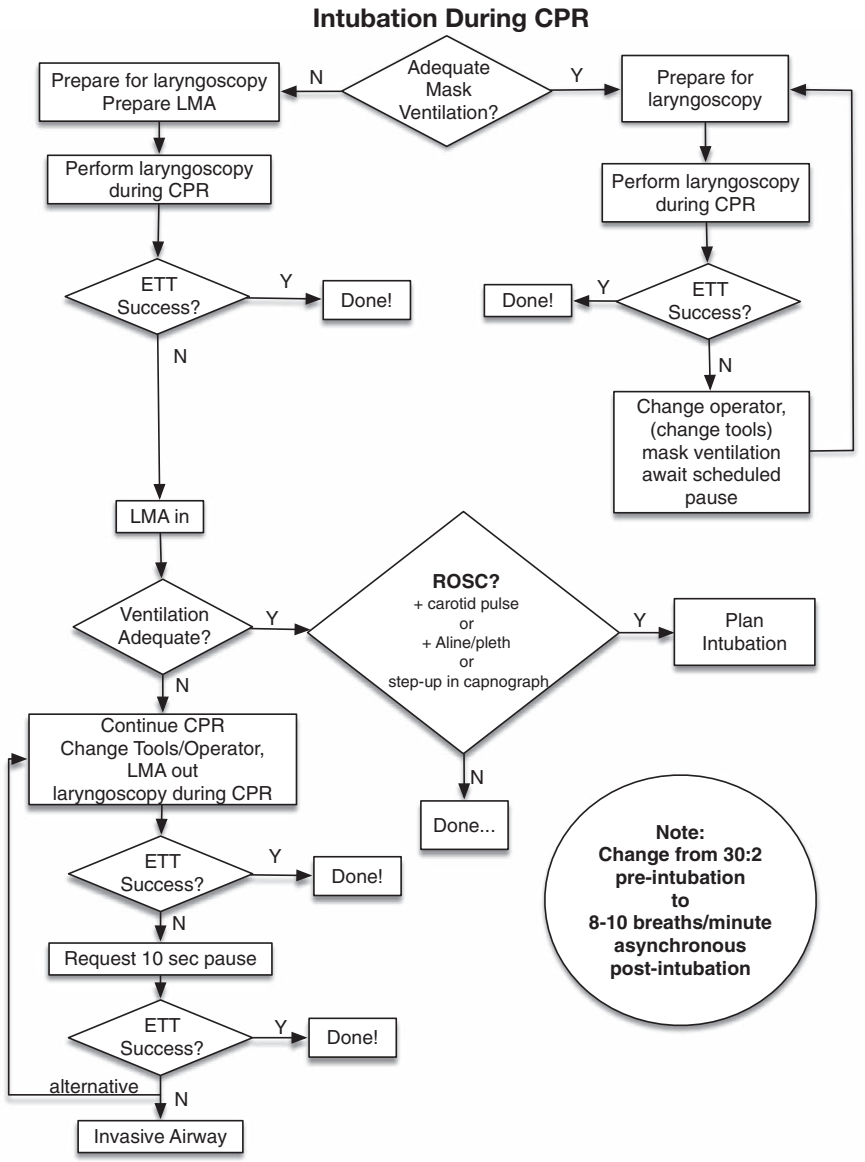

Figure 3. Intubation during CPR. CPR indicates cardiopulmonary resuscitation; ETT, endotracheal tube; LMA® , laryngeal mask airway; ROSC, return of spontaneous circulation.

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

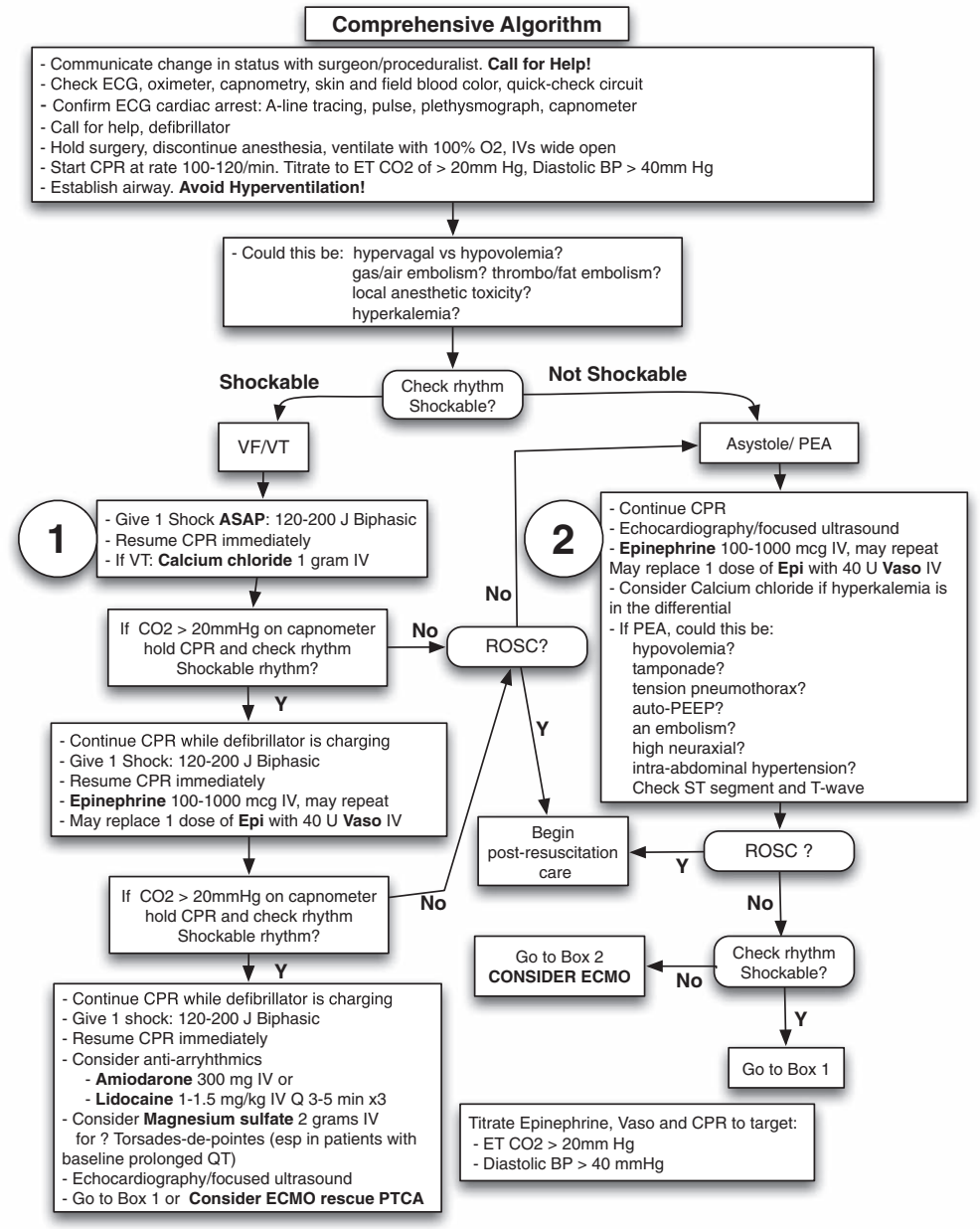

Figure 4. Comprehensive algorithm. Adaptation of the ACLS comprehensive algorithm. Rescuers are prompted to evaluate or empirically treat early for hyperkalemia. Echocardiography is especially useful in establishing the most likely cause of pulseless electrical activity and focusing resuscitation efforts. 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

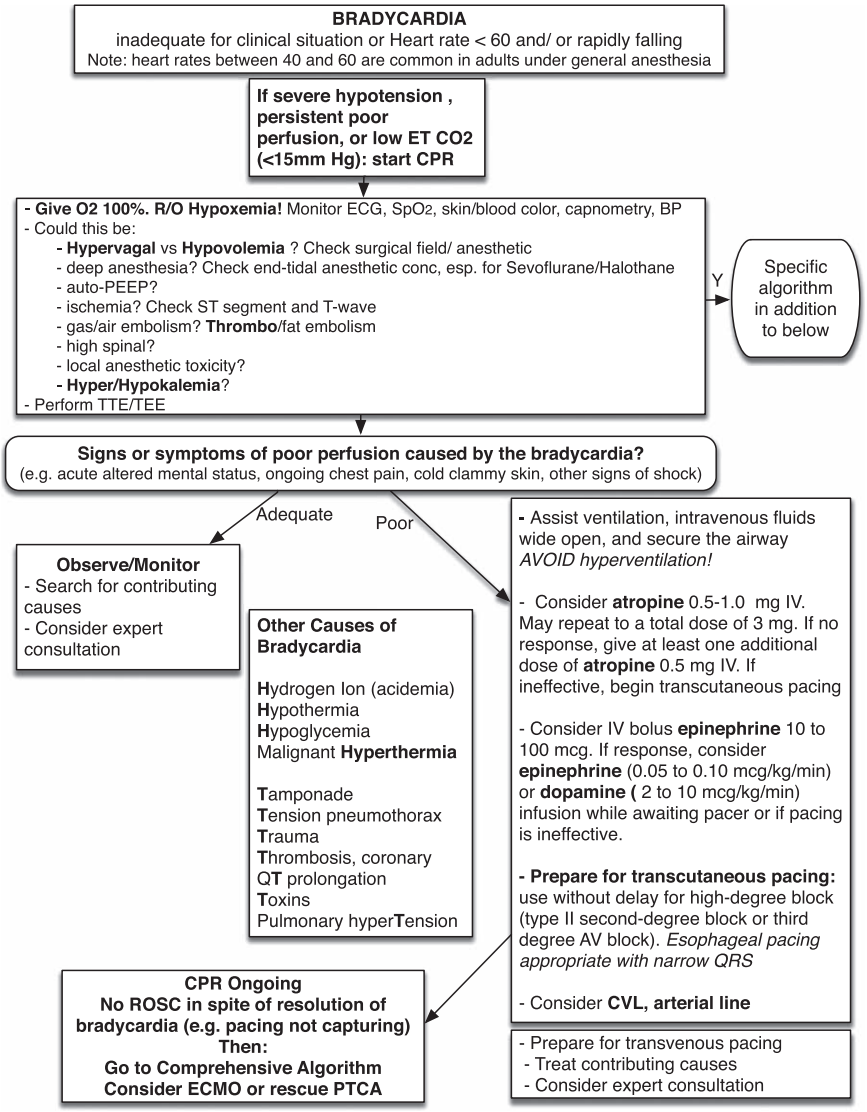

Figure 5. Bradycardia. Adaptation of ACLS algorithm for bradycardia. ACLS indicates advanced cardiac life support; AV, atrioventricular; BP, blood pressure; CPR, cardiopulmonary resuscitation; CVL, central venous line or catheter; ECG, electrocardiogram; ECMO, extracorporeal membrane oxygenation; $Etco_2$, 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

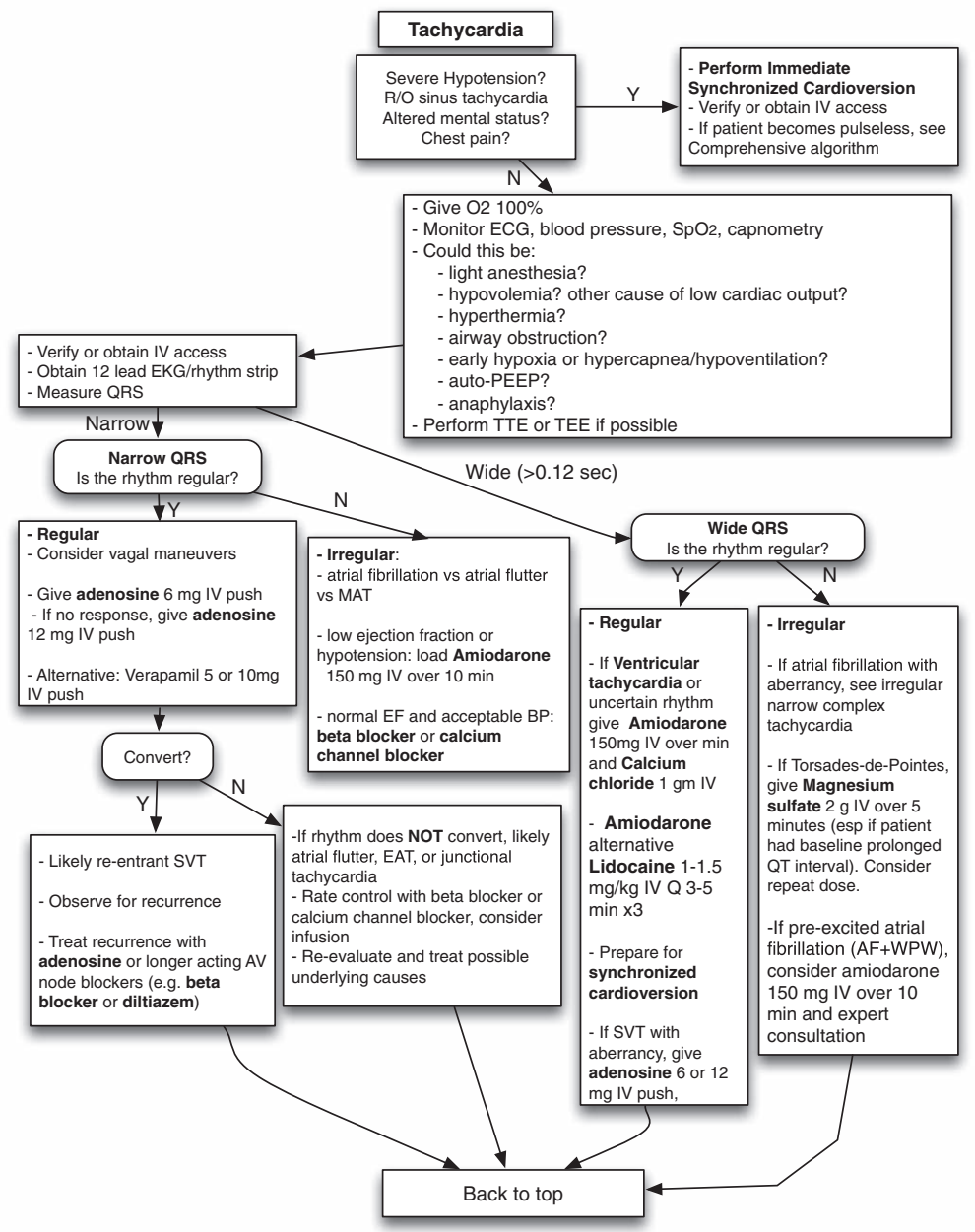

Figure 6. Tachycardia. Adaptation of the ACLS algorithm for tachycardia. ACLS indicates advanced cardiac life support; AF + WPW, atrial fibrillation and Wolff-Parkinson-White syndrome; BP, blood pressure; EAT, ectopic atrial tachycardia; ECG, electrocardiogram; EF, ejection fraction; EKG, electrocardiogram; 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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