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Perioperative Resuscitation and Life Support (PeRLS): An Update

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ABSTRACT

Cardiovascular collapse and arrest in the periprocedural setting and intensive care unit differ from arrests in other contexts (such as out-of-hospital or hospital ward) because clinicians almost always witness the event, and the most likely precipitating cause may be known. In comparison to other settings, the response can be timelier and more focused on treating the underlying cause(s). Since many patients deteriorate over minutes to hours, clinicians can evaluate the patient expeditiously, generate a diagnosis, and initiate appropriate treatment more rapidly than in other arrest circumstances. This iteration of Perioperative Resuscitation and Life Support (PeRLS) employs Grading of Recommendations, Assessment, Development, and Evaluations (GRADE) methodology to review the most recent evidence on preventing and managing cardiac arrest during the perioperative period. Furthermore, many of the recommendations and algorithms may also be applicable to areas outside the operating room, such as the intensive care unit and emergency room.

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Cardiac arrest during the perioperative period has an incidence of 2 to 13 per 10,000 anesthetics and an associated mortality before hospital discharge of 32 and 75%.[1–7] The incidence of perioperative cardiac arrest is higher in low-income countries compared to high-income countries.[6] Perioperative cardiac arrest can occur throughout the continuum of the perioperative period from induction of anesthesia until transfer to an inpatient unit after surgery.[1]

In 2012, a review by several authors of this article, "Anesthesia Advanced Circulatory Life Support,"[8] described the distinction between resuscitation of unresponsive individuals in the community and cardiac arrest during anesthesia, which is often witnessed and anticipated. After the American Heart Association (Dallas, Texas) published

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Abbreviations: **ACLS**, advanced cardiac life support; **ASA**, American Society of Anesthesiologists; **auto-PEEP**, auto–positive end expiratory pressure; **COI**, conflict of interest; **CPR**, cardiopulmonary resuscitation; **GRADE**, Grading of Recommendations, Assessment, Development, and Evaluations; **LAST**, local anesthetic systemic toxicity; **MH**, malignant hyperthermia; **$Paco_2$**, arterial carbon dioxide tension; **PEEP**, positive end expiratory pressure; **PeRLS**, Perioperative Resuscitation and Life Support; **PICO**, Patient, Intervention, Comparison, and Outcome; **POCUS**, point-of-care ultrasound; **PPV**, pulse pressure variation; **RCT**, randomized controlled trial; **ROSC**, return of spontaneous circulation; **TEE**, transesophageal echocardiography; **TXA**, tranexamic acid

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guidelines for managing cardiac arrest in 2015, an international group of 12 experts published a two-part review to offer an updated clinical perspective of cardiac arrest during the perioperative period.[9,10] In 2020, the American Society of Anesthesiologists' (ASA; Schaumburg, Illinois) assembled a new panel to revisit these works to update, expand, and conceive of novel ways to enhance and support resuscitation skills in the perioperative setting. To emphasize the distinction between suggestions for treating crises in the periprocedural setting and the content this group would generate, the panel was renamed the Perioperative Resuscitation and Life Support (PeRLS). This third iteration of PeRLS is based on Patient, Intervention, Comparison and Outcome (PICO) questions and summarizes the state of evidence using updated algorithms and recommendations based on the best and most recent evidence on preventing and managing cardiac arrest in these unique settings.[8–10] This article updates existing guidance in the literature to provide the most pertinent data for perioperative clinicians, and our guidance is intended to be relevant for interpreting existing cardiac arrest guidelines within the perioperative context. This guidance was conducted on behalf of the ASA. While this article focuses on the periprocedural setting, many of these algorithms may apply to other highly monitored in-hospital locations, such as the intensive care unit and the emergency room.

Materials and Methods

A panel of 17 international experts with experience in perioperative resuscitation was assembled to form a roundtable consensus body. The group reviewed published articles and generated relevant PICO questions. The evidence was assessed using GRADE methodology. GRADE provides a transparent consensus process to assess evidence and translate it into recommendations. GRADE uses the domains of study design, risk of bias, inconsistency, indirectness, imprecision, publication bias, effect size, plausible confounding effects on measured effect, and dose–response gradient to determine the certainty of evidence. Evidence certainty is classified as very low, low, moderate, or high based on specific criteria.[11] GRADE recommendations may be conditional (or weak), using the words "we suggest," or strong, using the phrase "we recommend." Further information is available in the GRADE Handbook.[12]

The group suggested new questions in addition to those already discussed in previous iterations of the project.[8–10] Overall, 97 questions relevant to perioperative resuscitation were put forward. For this project, the panel voted to prioritize 12 questions for a GRADE-based approach to the update (table 1). The panel members were then asked to create a list of outcomes of interest. Ultimately, 18 outcomes of interest were identified, which each of the panel members independently ranked by importance (table 2).

As accepted by Cochrane, based on a preliminary review of the amount and quality of the literature (S.E.), a decision was made to address 8 of the 12 PICO questions (questions 2 through 5, 7, 8, 10, and 11) without a systematic review. For these questions, eight teams of two panel members (dyads) were formed. These members were responsible for conducting a thorough independent search of the available literature for discussion and assessment. Each dyad screened the literature for articles relevant to their assigned question and the top four ranked outcomes for all PICO questions from the ranking poll (table 2). Dyads were also allowed to include literature that included lower-ranked outcomes if they believed they were particularly pertinent to the PICO question they were addressing. Recommendations were produced through an evidence-to-decision process.[13] Two members (S.E. and M.E.N.) provided methodologic support for this effort. In each case, a few studies informed the recommendation, precluding a meta-analysis. Dyads discussed the evidence with a methodologist, producing GRADE evidence profiles and evidence-to-decision tables. Recommendations were drafted and presented for voting by the complete panel. A threshold of 80% approval with

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Table 1. Summary of Patient, Intervention, Comparison, and Outcome (PICO) Questions and Recommendations Following Grading of Recommendations, Assessment, Development and Evaluations (GRADE) Methodology

Question	Recommendation	Level of Evidence
1. In perioperative patients with hypovolemia, is management with PPV assessment preferred over other therapies or no therapy?*	We suggest using PPV to guide fluid resuscitation in perioperative patients in whom hypovolemia is suspected.	Conditional recommendation, very low certainty of evidence
2. In perioperative patients during arrest, is hyperventilation preferred over other therapies or no therapy?	We suggest ventilation to a rate of 10 to 12 breaths/min and recommend against hyperventilation (RR greater than 16 breaths/min) during cardiac arrest.	Conditional recommendation, low certainty of evidence
3. In perioperative patients with symptomatic bradycardia, is atropine preferred over other therapies or no therapy?	We suggest administering atropine (0.5 mg intravenously) in severe bradycardia (heart rate of less than 40 beats/min or less than 50 beats/min with hypotension).	Conditional recommendation, low certainty of evidence
	We suggest administering atropine (1.0 mg intravenously) in severe bradycardia and suspicion of imminent or emergent arrest. Consider rapid dose escalation (*e.g.*, repeat 1.0 mg intravenously in 5 min).	Conditional recommendation, low certainty of evidence
	We recommend against administering atropine in established arrest and instead following the ACLS algorithm.†	Best practice statement
4. In perioperative patients with anaphylaxis, is epinephrine preferred over other therapies or no therapy?	We suggest administering epinephrine (50 to 100 μg intravenously or 200 to 500 μg intramuscularly) for the initial treatment of anaphylaxis. Dose escalation (*e.g.*, 100 to 300 μg) may be needed if hypotension persists after 3 to 5 min.	Conditional recommendation, very low certainty of evidence
5. In perioperative patients with anaphylaxis, is norepinephrine preferred over other therapies or no therapy?	We make no recommendation for or against norepinephrine for anaphylaxis.	No evidence
6. In perioperative patients with LAST, is lipid emulsion preferred over other therapies or no therapy?*	We recommend an infusion of lipid emulsion, 20% lipid, in perioperative patients with LAST.	Strong recommendation, low certainty of evidence
7. In perioperative patients with LAST, is epinephrine preferred over other therapies or no therapy?	We suggest administering epinephrine (initially 10 μg intravenously) for hypotension due to LAST.	Conditional recommendation, very low certainty of evidence
	We suggest administering epinephrine (initially 100 to 300 μg intravenously) for cardiac arrest due to LAST.	Conditional recommendation, very low certainty of evidence
8. In perioperative patients with MH, is dantrolene (2.5 mg/kg) preferred over other therapies or no therapy?	We recommend administering intravenous dantrolene (2.5 mg/kg) for MH.	Strong recommendation, moderate certainty of evidence
9. In perioperative patients with traumatic cardiac arrest, is permissive hypotension preferred over other therapies or no therapy?*	We suggest permissive hypotension by restricting fluid administration, targeted to a goal mean arterial pressure of 50 mmHg or a systolic blood pressure of 70 mmHg for the resuscitation of patients with traumatic cardiac arrest before definitive assessment of bleeding sources.	Conditional recommendation, very low certainty of evidence
10. In perioperative patients with high spinal, is atropine preferred over other therapies or no therapy?	We suggest administering atropine (1 mg intravenously) for high spinal cardiac arrest.	Conditional recommendation, very low certainty of evidence
11. In perioperative patients with high spinal, is epinephrine preferred over other therapies or no therapy?	For treating symptoms considered related to high spinal, we suggest administering an intravenous epinephrine bolus (100 to 200 μg) for high spinal pathophysiology in situations of arrest or when arrest appears imminent.‡	Conditional recommendation, very low certainty of evidence
12. In perioperative patients with arrest in the prone position, should we provide CPR over other therapies or no therapy?*	In perioperative patients with arrest in the prone position, we suggest CPR in the prone position over turning supine to perform classic CPR.	Conditional recommendation, very low certainty of evidence

*Supported by the Cochrane Group. †Subgroup consideration: In less urgent settings, glycopyrrolate may be equivalent. In settings where pacing is immediately available, pacing may be superior to atropine for hemodynamically significant bradycardia. ‡Note: An epinephrine infusion (starting infusion rate of 0.05 to 0.1 $\mu g \cdot kg^{-1} \cdot min^{-1}$) is best supported by the evidence for situations in which there is sufficient time to implement it.

ACLS, Advanced Cardiovascular Life Support; CPR, cardiopulmonary resuscitation; LAST, local anesthetic systemic toxicity; MH, malignant hyperthermia; PPV, pulse pressure variation; RR, respiratory rate.

70% of the members voting was established to approve each recommendation. The eight PICO questions produced nine recommendations and one best practice statement. In one instance (PICO question 5), the team made no recommendation due to lack of evidence. In another instance (PICO question 3), the team made two recommendations and one best practice statement. When the GRADE methodology was used to produce a recommendation, we indicate this with a specific recommendation (strong or conditional) and provide the level of evidence. Evidence profiles and evidence-to-decision tables are available in the Supplemental Digital Content.

Panelists disclosed potential conflicts of interest (COIs) annually, and a COI subcommittee reviewed each disclosure

Table 2. Perioperative Resuscitation Outcomes by Importance (n = 11 members)

Outcome	Average Rank*
Mortality	8.33
Neurologic outcome	8.33
ROSC	7.33
Cardiac arrest	7.17
MACE	6.33
ICU LOS	5.50
ICU admission	5.25
Time to compressions	5.00
Bradycardia events/time to resolve	4.75
Renal failure/AKI	5.25
Postoperative ventilation time	4.92
MAP ≥ 65 mmHg	4.67
Seizure	4.50
Need for additional therapies	4.50
Hemodyamic stability	4.50
Postoperative pulmonary edema/ALI	4.00
Hypotension	3.83
Congestion	3.17

*Outcomes were ranked by each panel member on a scale of 1 to 9, with 1 indicating the lowest priority and 9 indicating the highest priority.

AKI, acute kidney injury; ALI, acute lung injury; ICU, intensive care unit; LOS, length of stay; MACE, major adverse cardiovascular event; MAP, mean arterial pressure; ROSC, return of spontaneous circulation.

and provided remediation strategies for individuals with COIs. Panelists with potential financial COIs regarding a specific topic were allowed to participate in discussions but were excluded from drafting recommendations or voting on that topic.

During voting, feedback was solicited for each recommendation. All nine recommendations for the eight PICO questions passed approval thresholds. The dyads then provided materials for the drafting of rationales and key research questions that could inform recommendations. After voting, the panel collaborated on improving recommendation and rationale precision, while ensuring that no essential change was made to the underlying content. This process was used to give a voice to the collective expertise of the entire panel.

For the remaining four PICO questions (questions 1, 6, 9, and 12), a formal systematic review was conducted by the Cochrane group (Cochrane, London, United Kingdom) in collaboration with expert content input (M.F.O., M.E.N., and S.E.). A GRADE approach was then used to evaluate the evidence. The Cochrane group presented the entire panel with the results of the literature search, a meta-analysis of the data, and evidence assessment for each of the four questions. To benefit from the experience and expertise of practicing clinicians, a subgroup of the whole panel discussed the literature review, completed the evidence-to-decision process, and drafted recommendations.[14] The four PICO questions produced four recommendations. These were presented, discussed, and voted on by the panel. All four recommendations reached the approval threshold. As described above, panel feedback was incorporated for the eight non-Cochrane assisted recommendations, and the results were formatted in the same manner as described above.

Results

Table 1 lists the 12 PICO questions considered by the panel, the 13 recommendations, and the one best practice statement that emerged from the GRADE methodology. The Supplemental Digital Content provides all evidence profiles and evidence-to-decision tables.

Background

While advanced cardiac life support (ACLS) focuses on the presence of a pulse and the presenting rhythm and has been adopted by healthcare systems to manage all in-patients regardless of setting, this review highlights how resuscitation should be tailored for the periprocedural and critically ill patient populations. Bradycardia and tachycardia are common occurrences in the operating room and intensive care units (figs. 1 and 2). Cardiovascular collapse and arrest in the periprocedural setting and intensive care unit are distinct from arrests in other contexts (out-of-hospital or emergency room) because clinicians almost always witness the arrest, and the most likely precipitating cause may be known. Compared to different settings, the response can be timelier and more focused on treating the underlying cause(s). Table 3 outlines the 9 Hs and Ts of perioperative cardiac arrest. Because many patients deteriorate during minutes to hours, the clinician can evaluate the patient expeditiously, generate a diagnosis, and commence appropriate treatment more rapidly than in other arrest circumstances.

Fluid Administration

Evidence Summary. A national audit of cardiac arrests in the United Kingdom identified hemorrhage as the most common cause of arrest across all procedures (17%).[1] Fluid management is ubiquitous in anesthesiology and critical care because fluid administration can potentially increase cardiac output.

A Cochrane review that included adult, nonpregnant patients undergoing non-neurologic surgery in which the index intervention was pulse pressure variation (PPV), systolic pressure variation, or stroke volume variation assessment and the target primary outcome was fluid responsiveness assessed data through July 2023. The review included 12 studies with 405 patients. Overall, the studies suggested mid-range accuracy for the performance of dynamic indices of volume responsiveness, and the certainty of the evidence was judged as very low. For the primary

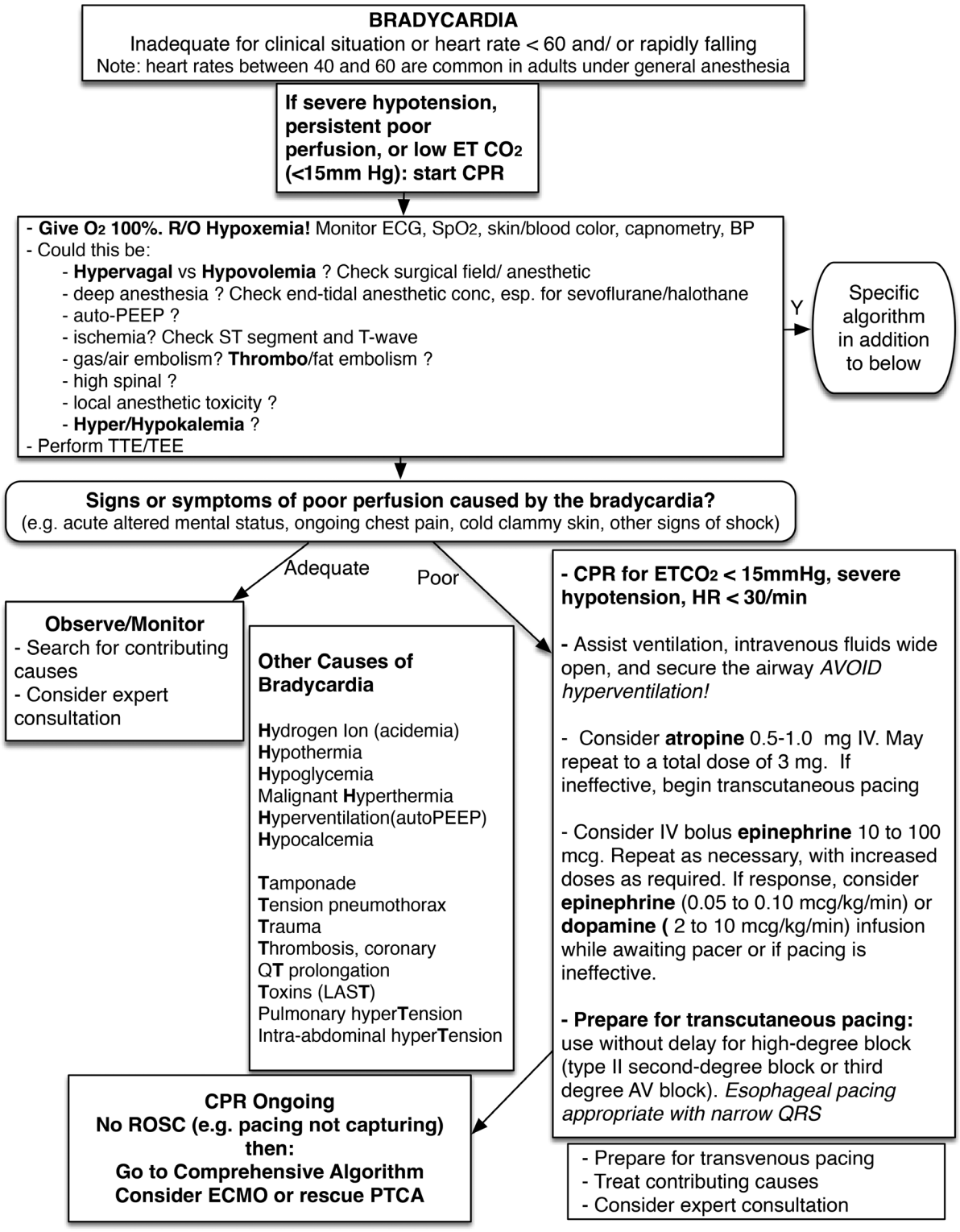

Fig. 1. Treatment algorithm for perioperative bradycardia. BP, blood pressure; CPR, cardiopulmonary resuscitation; ECG, electrocardiogram; ECMO, extracorporeal membrane oxygenation; $ETCO_2$, end-tidal carbon dioxide; HR, heart rate; PEEP, positive end-expiratory pressure; PTCA, percutaneous transluminal coronary angioplasty; R/O, rule out; ROSC, return of spontaneous circulation; TEE, transesophageal echocardiogram; TTE, transthoracic echocardiogram.

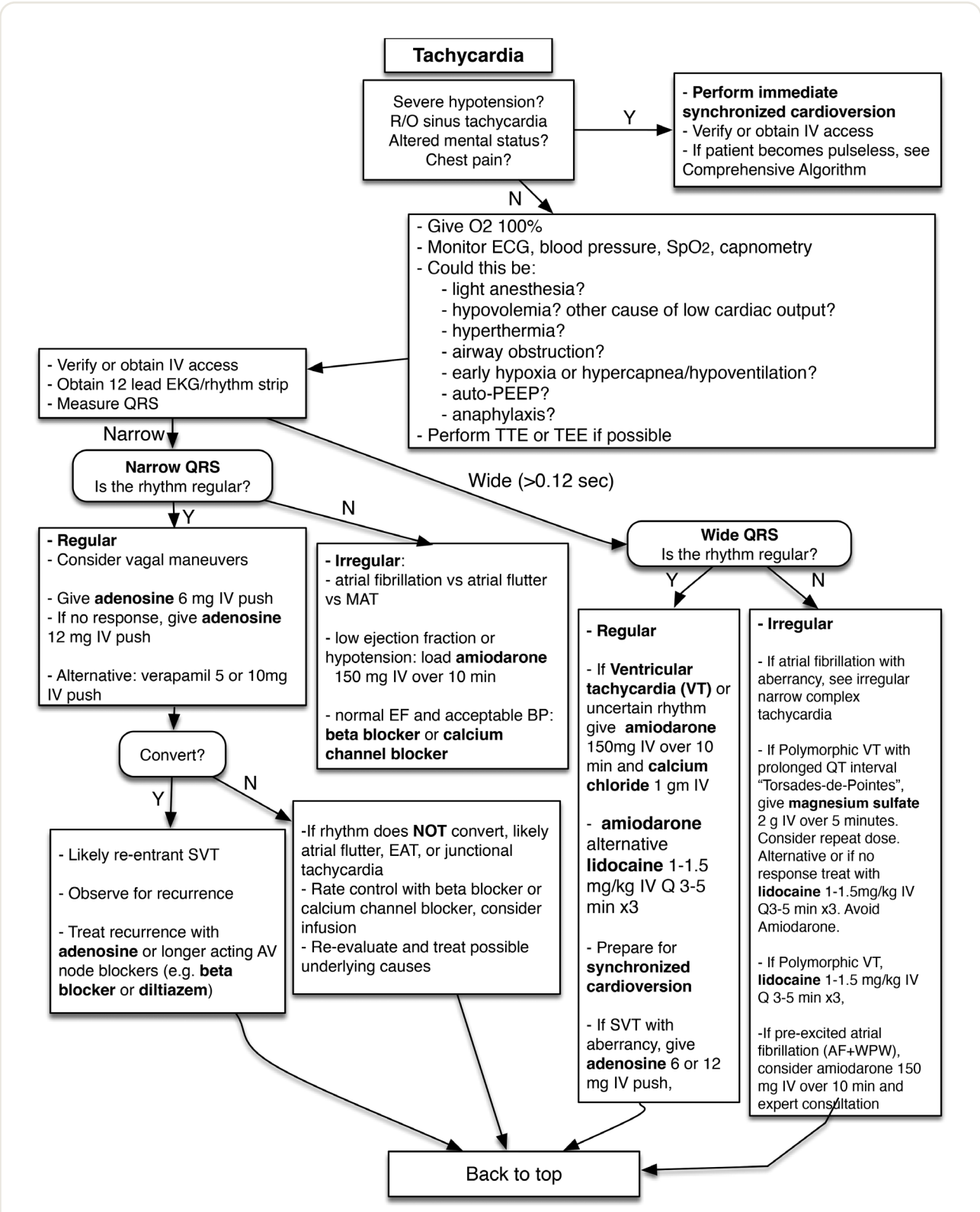

Fig. 2. Treatment algorithm for perioperative tachycardia. AF + WPW, atrial fibrillation and Wolff–Parkinson–White syndrome; BP, blood pressure; EAT, ectopic atrial tachycardia; ECG or EKG, electrocardiogram; EF, ejection fraction; IV, intravenous; MAT, multifocal atrial tachycardia; PEEP, positive end-expiratory pressure; Spo_2, oxygen saturation measured by pulse oximetry; SVT, supraventricular tachycardia; TEE, transesophageal echocardiogram; TTE, transthoracic echocardiogram.

Table 3. Perioperative Cardiac Arrest Etiologies: 9 Hs and 9 Ts

Hypoxia
Laryngospasm
Bronchospasm
Aspiration
Negative pressure pulmonary edema
Congestive heart failure
Hypovolemia
Hemorrhage
Hypervagal
Peritoneal insufflation
Surgical manipulation (eyes, gonads)
Hyper/**H**ypokalemia
AKI: hyperkalemia
Transfusion: hyperkalemia
Reperfusion (liver transplant, portal vein clamp removal, aortic or lower extremity artery clamp removal): hyperkalemia
Diuretics: hypokalemia
Hydrogen ion (acidemia)
Lactic acidosis
Acute kidney injury
Can cause pacemaker failure
Hypothermia
Massive transfusion protocol
Cold exposure
Hypoglycemia
Liver failure
Insulin overdose
Adrenal insufficiency
Malignant **H**yperthermia
Hypocalcemia
Massive transfusion
Toxins (anaphylaxis/anesthesia)
Volatile anesthetics
Neuroaxial block
Dexmedetomidine
Fentanyl
β Blockers
Medication error
Tension pneumothorax
Central line placement
Abdominal insufflation to pleural space
Thrombosis/embolus
Venous air embolism
Fat embolism
Deep vein thrombosis
Cardiac standstill
Thrombosis coronary
Acute coronary syndrome
Tamponade
Cardiac surgery
Central line placement
Trauma
Hemorrhage
Cardiac contusion
Cerebral herniation
q**T** prolongation
Hypomagnesemia (diuretics)
Hypocalcemia after transfusion
Pulmonary hyper**T**ension
Intraabdominal hyper**T**ension
Peritoneal insufflation
Ascites
Abdominal bleed
Abdominal packing

AKI, acute kidney injury.

outcome of PPV assessment for volume responsiveness, 10 studies produced a sensitivity of 82% (95% CI, 74 to 89%), and a specificity of 71% (95% CI, 55 to 83%). For stroke volume variation assessment, seven studies yielded a point estimate of sensitivity at 86% (95% CI, 81 to 90%) and specificity at 70% (95% CI, 46 to 86%).[15–21] For systolic pressure variation, one study[19] suggested a sensitivity of 81% and a specificity of 87%. The Cochrane review can be found in the Supplemental Digital Content (https://links.lww.com/ALN/E187). PPV may predict fluid responsiveness and therefore lead to more judicious fluid administration, but the evidence is very uncertain.

Panelists agreed that the evidence favors using PPV or other markers of dynamic fluid responsiveness for assessing and managing volume status in the perioperative setting for adult patients. This assessment is based on the evidence supporting PPV. The evidence-to-decision tables in the Supplemental Digital Content present the evidence assessment (https://links.lww.com/ALN/E191).

Much discussion centered on the clinical question addressed with tests of fluid responsiveness. There was equal preference for using dynamic assessment to stop fluid infusion by correctly identifying nonresponsive patients (premium on specificity) and for placing patients in whom fluid responsiveness would trigger additional infusion (premium on sensitivity).

2025 Recommendation: We suggest using PPV to guide fluid resuscitation in perioperative patients in whom hypovolemia is suspected.

Conditional recommendation, very low certainty of evidence

Additional Considerations. Measuring pulse pressure variation requires continuous arterial line blood pressure monitoring. Changes in blood pressure in the setting of fluid administration do not accurately reflect changes in cardiac output, and blood pressure changes alone may not suffice to determine fluid responsiveness.[22] Without a direct measurement of cardiac output, changes in pulse pressure (proportional to stroke volume), end-tidal carbon dioxide, or PPV can roughly guide fluid administration.[22–24] Without assessing fluid responsiveness, a bolus of fluids will increase cardiac output in only about 50% of cases.[25] Excessive fluid administration predisposes to fluid overload and venous congestion and correlates with poor outcomes after surgery and in sepsis.[26,27] Furthermore, not all fluid-responsive patients must receive fluids, especially if there is a potential for harm (*i.e.*, worsening hypoxemia, intraabdominal hypertension, cardiogenic shock).[25] The data for interventional fluid strategies is more robust in the perioperative period than in any other hospital setting.[25] Several meta-analyses of goal-directed fluid strategies in surgical patients have reported improved outcomes, including decreased mortality and intensive care unit length of stay.[28–32]

Dynamic indices of volume assessment have several limitations. They should be used with caution in certain situations, including right heart failure, an open chest, intraabdominal hypertension, and the presence of arrhythmias.[33] Given the challenges of managing fluids in patients with cardiogenic shock, PPV to guide fluid administration has been incorporated into our updated approach to managing left ventricular shock (fig. 3) and right ventricular shock because of its ease of use (fig. 4).

Preventing the Progression of Shock to Cardiac Arrest

Evidence Summary. Both left ventricular and right ventricular dysfunction can progress to shock and cardiac arrest. Echocardiography can confirm either diagnosis, and a pulmonary artery catheter (in selected cases) can guide the acute hemodynamic management of left, right, or biventricular failure. Failure to make the correct diagnosis in a hypotensive patient may result in excessive fluid administration, which stretches the ventricle, increases afterload, and exacerbates ventricular shock. Considering each ventricle as a separate organ highlights how managing left and right ventricular failures differ.

Left Ventricular Shock. Volume refractory shock with a low pulse pressure variation characterizes left ventricular shock. Our experts agree on several principles of management based on available literature. Hypovolemia in patients with poor left ventricular function should be remedied before the institution of any pharmacologic therapies. Hypotensive euvolemic or hypervolemic patients with left ventricular shock are best treated with agents that enhance inotropy (dobutamine, epinephrine, milrinone, and norepinephrine).[34] Patients with left ventricular shock with elevated systemic vascular resistance and the ability to tolerate a decrease in their blood pressure are treated with afterload-reducing agents, such as nicardipine or hydralazine. Diuretics may also be needed to reduce ventricular stretching. Patients with known significant diastolic dysfunction are often treated with a lusitropic agent such as milrinone in the acute setting. Lusitropic agents enhance ventricular relaxation, resulting in better ventricular filling, increased stroke volume, and increased cardiac output. Patients who are believed to have good potential for recovery and who do not improve with pharmacotherapy can be supported with mechanical circulatory support devices. These devices include intraaortic balloon pumps, ventricular assist devices, and extracorporeal life support (extracorporeal membrane oxygenation). Figure 3 outlines the suggested approach to managing a patient with left ventricular shock.

Right Ventricular Shock. During the perioperative period, right ventricular shock may be caused by an acute increase in right ventricular afterload (*i.e.*, venous gas, fat, cement, amniotic material, or thromboembolism). In approximately 5% of cases, acute thromboembolism causes cardiac arrest, most often pulseless electrical activity.[35,36] Not all right ventricular failures require volume to increase the "preload dependent ventricle."[37] Diuretics can offload ventricular work and promote forward flow to the left ventricle in patients whose right ventricular dysfunction results from excessive fluid administration.[37] Right ventricular shock can be managed with inhaled pulmonary vasodilators (*i.e.*, nitric oxide, epoprostenol) to reduce pulmonary vascular resistance, which reduces right ventricular afterload; inotropes to increase right ventricular contractility; and systemic arterial vasoconstrictors to maintain mean arterial pressures and vital organ perfusion (fig. 4).

Vasopressin is a vasopressor that selectively vasoconstricts the systemic circulation and spares the pulmonary circulation.[34,38,39] Vasopressin may induce pulmonary vasodilation to reduce right ventricular afterload.[38] In the case of right ventricular shock refractory to medical management, extracorporeal membrane oxygenation or a right ventricular assist device may be indicated. Table 4 lists management steps for patients with an acute pulmonary embolism (often in the setting of right ventricular shock).

Vasoplegia and Sepsis. In a large database study of surgical patients who underwent cardiopulmonary resuscitation during the perioperative period, almost 40% of patients had a preoperative diagnosis of sepsis.[40] Because there is a substantial incidence of sepsis-associated cardiomyopathy, echocardiography should be used to guide the initiation of inotropic support. Patients with vasoplegia from chronic therapy with angiotensin-converting enzyme inhibitors or angiotensin receptor blockers can be managed with infusions of vasopressin, norepinephrine, or angiotensin II.

Respiratory Rate during Cardiac Arrest

Evidence Summary. Some animal studies suggest that excessive hyperventilation might lead to worse outcomes from decreased venous return and decreased cerebral and coronary perfusion pressure.[41] The evidence on the role of hyper- *versus* hypoventilation in cardiac arrest is mixed but may slightly favor avoidance of hyperventilation. Most studies do not use $Paco_2$ to quantify hyperventilation, so ventilation rates (more than 10 breaths/min) are used as a surrogate for minute ventilation. There is some evidence that ventilation rates greater than 10 breaths/min may worsen mortality and neurologic outcomes after a cardiopulmonary arrest.[42–44] The study with the highest certainty[42] compared respiratory rates of 6 breaths/min to 12 to 16, 16 to 20, and greater than 20 breaths/min. The odds ratios for survival were 0.79 (95% CI, 0.72 to 0.87) for a respiratory rate of fewer than 6 breaths/min, compared with 1.23 (95% CI, 1.07 to 1.40) for 12 to 16 breaths/min, 1.12 (95% CI, 0.98 to 1.27) for 16 to 20 breaths/min, and 1.22 (95% CI,

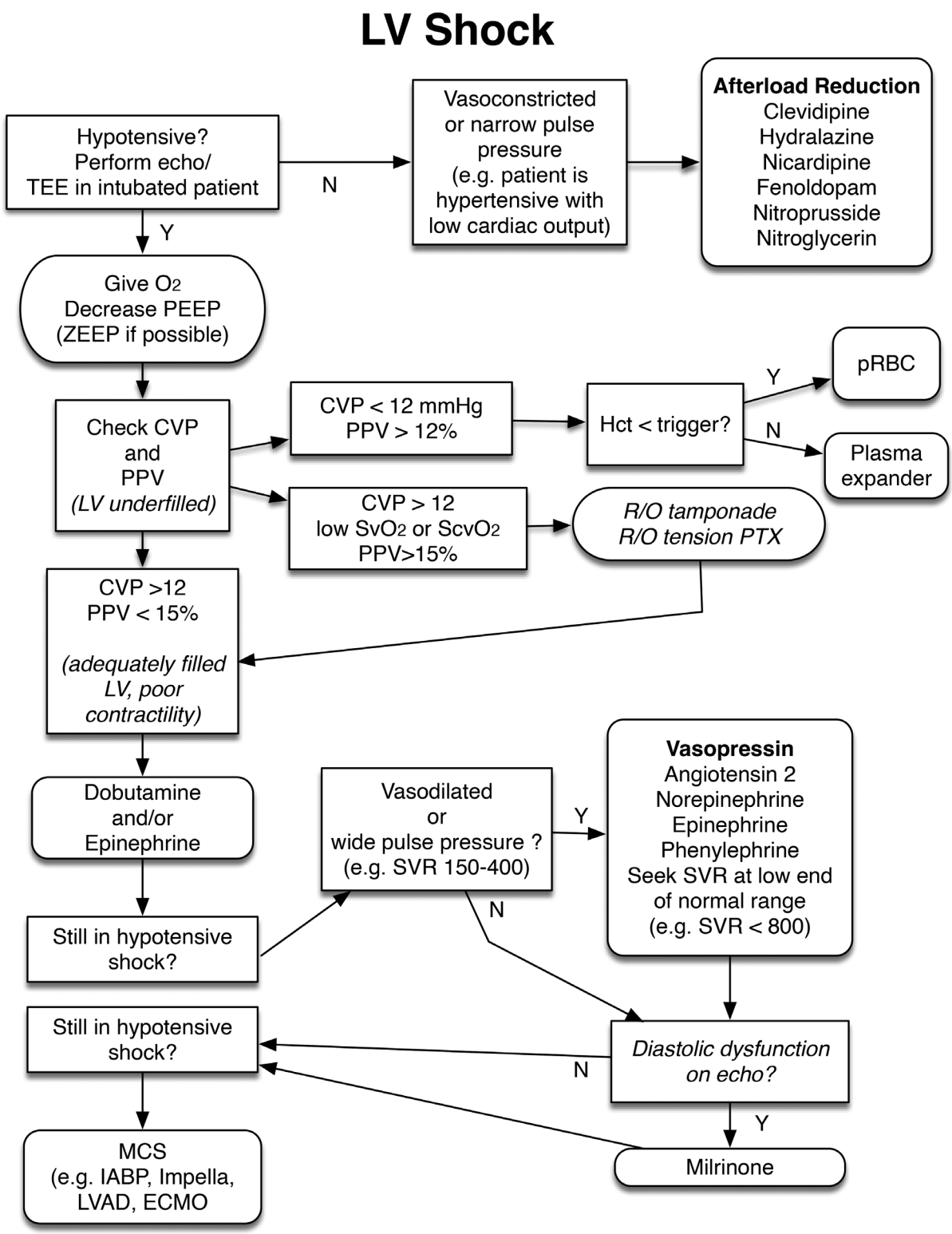

Fig. 3. Treatment algorithm of left ventricular failure with cardiogenic shock. CVP, central venous pressure; ECMO, extracorporeal membrane oxygenation; IABP, intraaortic balloon pump; LV, left ventricular; LVAD, left ventricular assist device; PEEP, positive end-expiratory pressure; PPV, pulse pressure variation; PTX, pneumothorax; SVR, systemic vascular resistance in dyne · s^{-1} · cm^{-5} · m^{-2}; TEE, transesophageal echocardiogram.

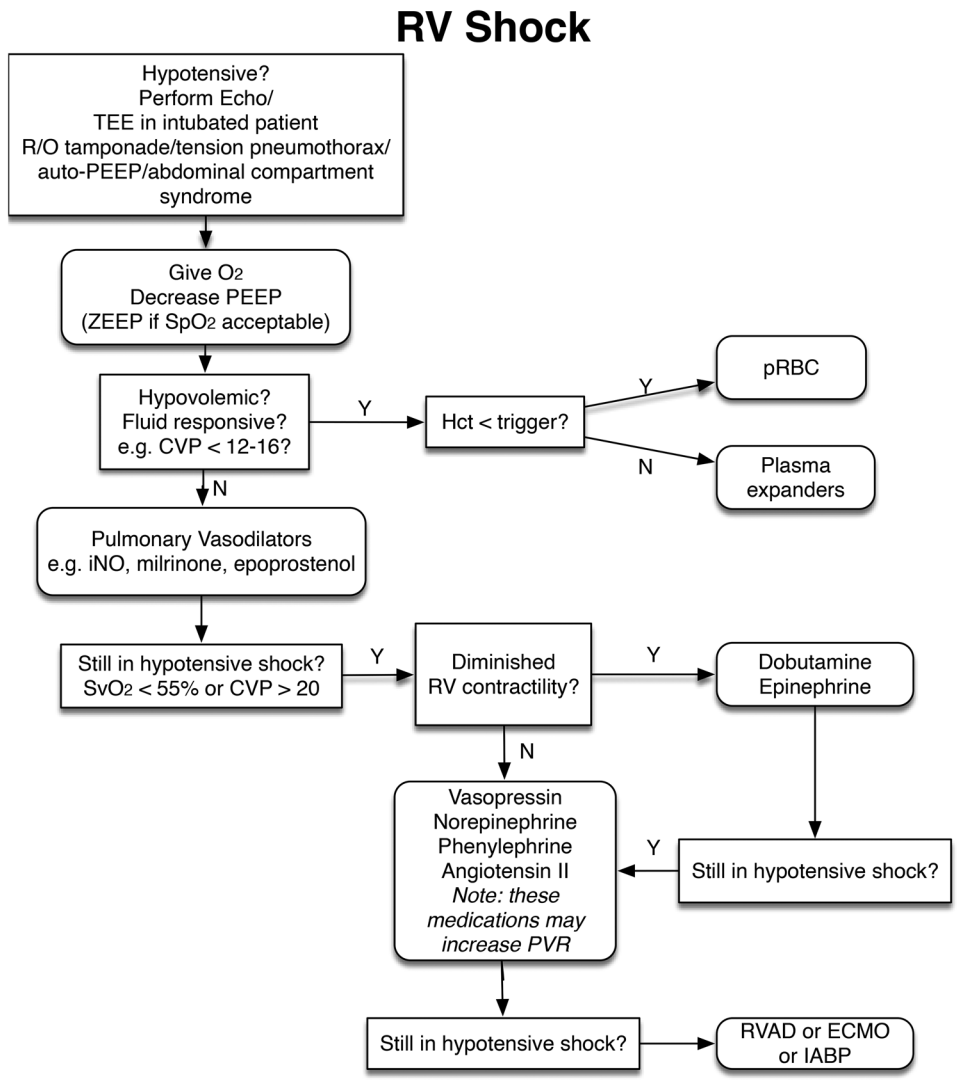

Fig. 4. Treatment algorithm of right ventricular failure with cardiogenic shock. CVP, central venous pressure; ECMO, extracorporeal membrane oxygenation; IABP, intraaortic balloon pump; iNO, inhaled nitric oxide; PEEP, positive end-expiratory pressure; PVR, pulmonary vascular resistance; RV, right ventricular; RVAD, right ventricular assist device; Svo_2, mixed venous oxygen saturation; TEE, transesophageal echocardiogram.

1.06 to 1.41) for greater than 20 breaths/min. For favorable neurologic outcomes, the values were 0.83 (95% CI, 0.66 to 1.05), 1.36 (95% CI, 1.01 to 1.84), 1.08 (95% CI, 0.79 to 1.47), and 1.30 (95% CI, 0.92 to 1.84), respectively. Assuming a baseline survival of 20% from cardiac arrest, the absolute effect estimate is 3.5 more survivors per 100 at ventilation rates of 12 to 16 breaths/min, a large effect with low certainty.

2025 Recommendation: We suggest ventilation to a rate of 10 to 12 breaths/min and suggest against hyperventilation (respiratory rate greater than 16 breaths/min) during cardiac arrest.

Conditional recommendations, low certainty of evidence

Additional Considerations. Hyperventilation can also cause incomplete exhalation, in which air is retained in the alveoli and causes auto–positive end expiratory pressure (auto-PEEP). Patients with high compliance (*e.g.*, chronic obstructive pulmonary disease), increased resistance (*e.g.*, bronchospasm), rapid respiratory rates, and large tidal volumes predispose to developing auto-PEEP. When auto-PEEP occurs, the expiratory flow waveform does not return to baseline before initiating the next breath (fig. 5). During cardiac arrest, excessive ventilation that causes auto-PEEP

Table 4. Pulmonary Embolism

Assessment	• Significant hemodynamic instability during high-risk cases • Tachycardia • Sudden decrease in $ETCO_2$ • Consider TTE/TEE
Initial management	*Prearrest* • Stop gas insufflation • Flood the surgical field • Administer 100% oxygen • Intubate for respiratory distress or hypoxemia • Place patient in Trendelenburg (head down) position • Rotate patient toward the left lateral decubitus position ○ Maintain BP (see RV shock algorithm) ○ IV fluids ○ Vasopressors ○ β-Adrenergic agents. • Consider transfer to a hyperbaric chamber if immediately available (air embolism) *Cardiac arrest* • Start CPR • Consider CPB/emergent thrombectomy
Subsequent management	• Consider RV shock algorithm

BP, blood pressure; CPB, cardiopulmonary bypass; $ETCO_2$, end-tidal carbon dioxide; IV, intravenous; RV, right ventricular; CPR, cardiopulmonary resuscitation; TEE, transesophageal echocardiography; TTE, transthoracic echocardiogram.

can reduce venous return and prevent the return of spontaneous circulation.

However, in some unique circumstances, such as increased intracranial pressure, profound acidosis, or hyperkalemia, a higher respiratory rate may be desired. Our updated approach to intubation during CPR suggests a respiratory rate of 10 to 12 breaths/min after intubation (fig. 6).

Symptomatic Bradycardia

Evidence Summary for Atropine. Sudden, severe bradycardia in the periprocedural setting can be caused by gonadal or eyeball pressure and peritoneal insufflation or other manipulations that increase vagal tone and augment the effects of vagotonic anesthetics and the sympatholysis of anesthetic agents. Atropine has been the preferred treatment for hemodynamically unstable bradycardia for decades. There are no randomized controlled trials (RCTs) of treatment with atropine for cardiac arrest preceded by bradycardia. Glycopyrrolate, catecholamines (β-agonists), pacing, and placebo have all been used as alternatives to atropine in various contexts to treat or prevent bradycardia. Randomized trials and case series identified in the literature review were oriented toward bradycardia caused by other medications (*e.g.*, repeated doses of succinylcholine, higher rates of infusion of dexmedetomidine), bradycardia associated with high neuraxial anesthetic block, and abdominal insufflation.[22] The heterogeneity among these studies precluded meta-analysis. In a trial comparing atropine (5 μg/kg) to glycopyrrolate (2.6 μg/kg), either drug resolved bradycardia 33% of the time in a standardized anesthetic using opioid infusion, while pacing resolved bradycardia 100% of the time.[45] This study was limited by severe imprecision because of its small sample size. Very-low-certainty evidence from prophylaxis trials suggests atropine's inferiority (0.01 mg/kg) to glycopyrrolate (0.005 mg/kg),[46] equivalence (0.01 mg/kg) to glycopyrrolate (0.005 mg/kg),[47] and superiority (0.5 to 0.6 mg) to saline.[48,49] All studies had very small sample sizes.

The aggregate available evidence favors neither glycopyrrolate nor atropine, and the evidence is very uncertain about the effect of atropine on bradycardia. Observations from anticholinergic use in the antagonism of neuromuscular blockade suggest a faster onset of action for atropine than glycopyrrolate.[50] These considerations lead us to recommend atropine over glycopyrrolate in situations of suspected arrest or severe bradycardia (heart rate of fewer than 40 beats/min). In less severe bradycardia, the two drugs are likely interchangeable, but consideration should be given to urgency and associated risks (*e.g.*, tachycardia in ischemic heart disease). Glycopyrrolate is associated with less tachycardia than atropine. This finding and fewer adverse central nervous system effects (delirium) may favor glycopyrrolate in less severe bradycardia.

2025 Recommendation: We suggest administering atropine (0.5 mg intravenously) in severe bradycardia (heart rate of fewer than 40 beats/min or fewer than 50 beats/min with hypotension).

Conditional recommendation, low certainty of evidence

2025 Recommendation: We suggest administering atropine (1.0 mg intravenously) in severe bradycardia and suspicion of imminent or emergent arrest. Consider rapid dose escalation (*e.g.*, repeat 1.0 mg intravenously in 5 min).

Conditional recommendation, low certainty of evidence

Best practice statement: We recommend against administering atropine in established arrest and instead following the ACLS algorithm.

Additional Considerations. In less urgent settings, glycopyrrolate (0.005 mg/kg) may be equivalent to atropine (0.01 mg/kg). In settings where pacing is immediately available, pacing may be superior to atropine for hemodynamically significant bradycardia.

Transvenous pacing is a viable alternative to medication administration, but it is not commonly available, requires specialized equipment, and may delay implementation. Under circumstances in which these feasibility concerns can be mitigated, pacing is a viable option. Transcutaneous pacing should also be considered to manage bradycardia in hypotensive patients. Figure 1 outlines

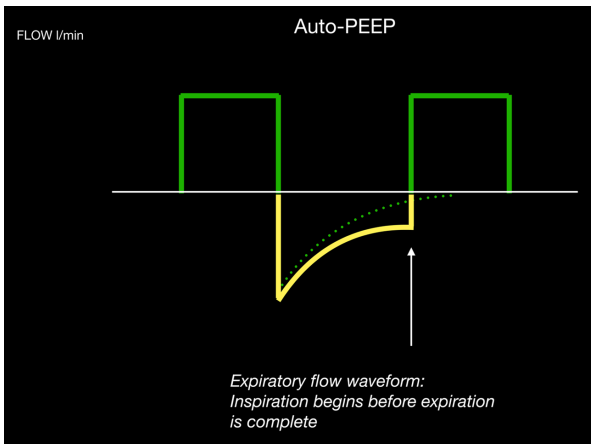

Fig. 5. Flow waveform scalar for auto-PEEP. PEEP, positive end-expiratory pressure.

an approach to symptomatic bradycardia in the perioperative period.

Anaphylaxis

Evidence Summary for Epinephrine. The clinical presentation of anaphylaxis can vary from initial nonspecific signs to rapidly progressive cardiorespiratory compromise, which could be potentially fatal. Initial signs could range from cutaneous and mucosal changes to confusion, altered sensorium, and presyncope.[51] Although seen often, bronchospasm is not consistently observed in all perioperative patients with anaphylaxis. In patients with severe, untreated anaphylaxis, rapid hemodynamic collapse ensues from vasodilation, reduction in preload, and subsequent hypovolemia and can cause myocardial ischemia, infarction, and malignant arrhythmias resulting in a state referred to as anaphylactic shock.[52,53]

The evidence for treating prearrest anaphylaxis with epinephrine is limited because of the unpredictable and life-threatening nature of this condition. There is limited evidence suggesting that in patients with anaphylaxis, treating with epinephrine before or after cardiopulmonary arrest may be associated with an increased rate of return of spontaneous circulation (ROSC; 43% with epinephrine *vs*. 4% without).[54] There is no evidence to show a decrease in mortality. Epinephrine supports the circulation through α and β sympathetic stimulation, treats bronchospasm,[55–57] and may play a role in stabilizing the immune response that drives anaphylaxis.[58,59] Because of these effects, expert opinion and guidelines support epinephrine use in anaphylaxis.[60]

2025 Recommendation: We suggest administering epinephrine (50 to 100 μg intravenously or 200 to 500 μg intramuscularly) for the initial treatment of anaphylaxis. Dose escalation (*e.g.*, 100 to 300 μg intravenously) may be needed if hypotension persists after 3 to 5 min.

Conditional recommendation, very low certainty of evidence

Evidence Summary for Norepinephrine. Norepinephrine has been proposed as a possible alternative to epinephrine in patients who do not respond to epinephrine. There are no RCTs comparing norepinephrine to epinephrine or other therapies or no therapy for perioperative anaphylaxis. No studies directly investigated norepinephrine *versus* other drugs or treatments for the management of perioperative anaphylaxis. The only evidence identified was from two clinical practice guidelines[61,62] and a case report[63] that suggest using norepinephrine as a rescue medication in patients with perioperative anaphylaxis and shock resistant to epinephrine. These suggestions were not based on RCTs but on expert opinion.

2025 Recommendation: We make no recommendation for or against norepinephrine for anaphylaxis.

No evidence

Additional Considerations. If anaphylaxis is suspected, the ongoing surgical procedure should be stopped (when possible), and potential triggers should be identified and eliminated.[64] In addition to epinephrine, fluid resuscitation of approximately 20 ml/kg will often address the hypovolemia associated with anaphylaxis.[65,66] Laboratory tests for histamine (within 2 to 15 min) and tryptase (within 30 to 120 min) should be sent to establish the diagnosis of anaphylaxis.[67–69] If frequent boluses of epinephrine are required to maintain hemodynamic stability, an epinephrine infusion (0.05 to 0.3 $\mu g \cdot kg^{-1} \cdot min^{-1}$) may be indicated.

Anaphylaxis symptomatology tends to be bimodal, and the likelihood of recrudescence is high. Therefore, once the patient with perioperative anaphylaxis has been stabilized, observation in a monitored setting is important.[70] Table 5 outlines the steps in the management of anaphylaxis.

Local Anesthetic Systemic Toxicity (LAST)

Evidence Summary for Lipid Emulsion in LAST Management. LAST continues to occur despite education, emphasis on adherence to accepted dosing limits, and the use of ultrasound guidance. The reported incidence of this iatrogenic complication varies, ranging from 1 to 10 events per 10,000 cases, depending on the exposure.[71] Recent data indicate that the risk specifically for peripheral nerve block is roughly 1.8 per 1,000, and given the likelihood of under-reporting and misdiagnosis, this rate may be underestimated.[72,73] A review of published cases found that approximately 56% of LAST cases present with cardiovascular instability, either

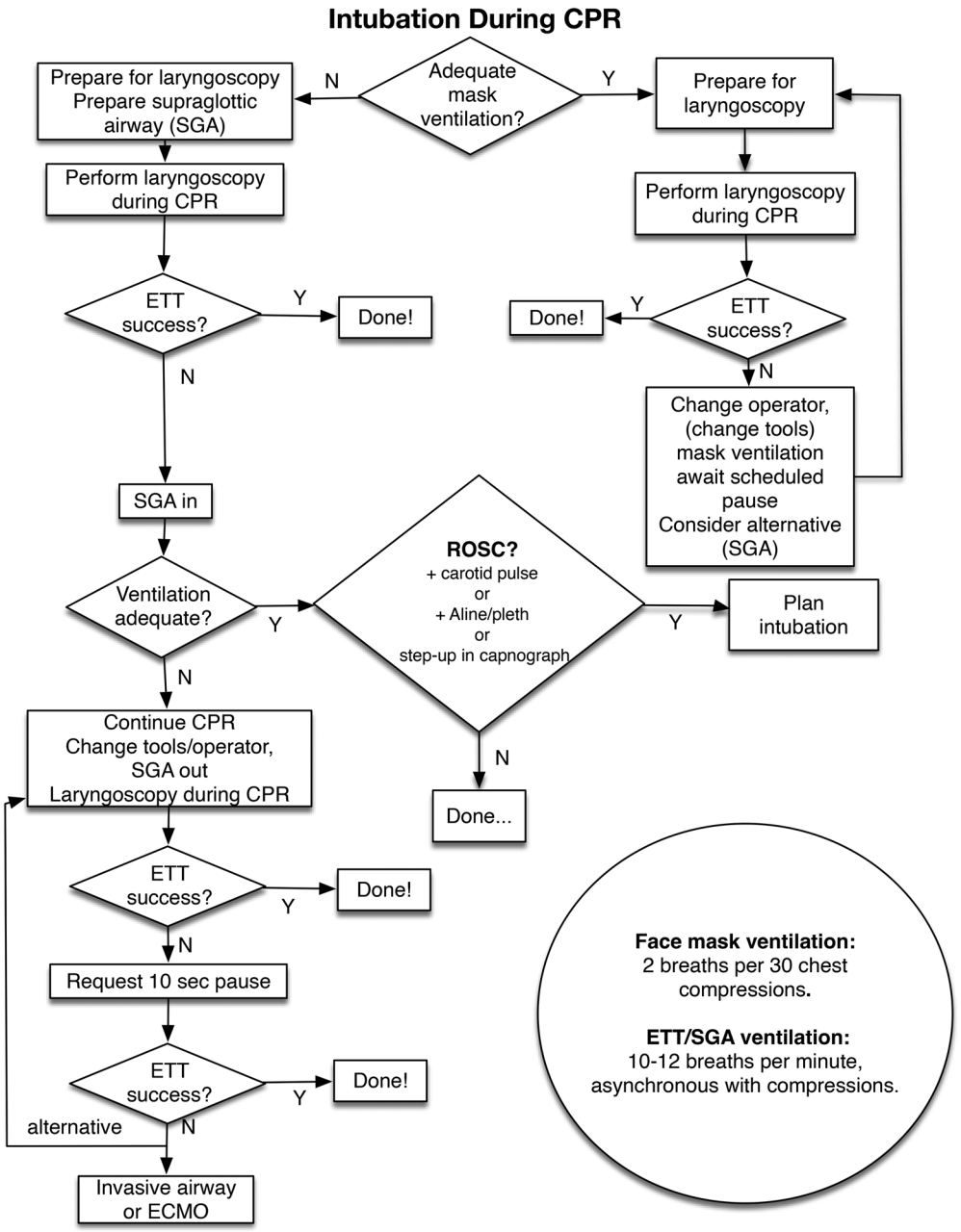

Fig. 6. Intubation during cardiac arrest. CPR, cardiopulmonary resuscitation; ECMO, extracorporeal membrane oxygenation; ETT, endotracheal tube; ROSC, return of spontaneous circulation; SGA, supraglottic airway.

alone (11%) or combined (44%) with central nervous system toxicity. Of these, 27% had bradycardia or asystole, and 13% had ventricular tachycardia/fibrillation.[74] More recent data confirm this overall frequency for cardiovascular toxicity and identified a rate of 21% for LAST events requiring cardiopulmonary resuscitation.[75]

Before 2006, treatment of LAST was largely supportive and included termination of administration of the local anesthetic, standard ACLS measures, and, if available, cardiopulmonary bypass or veno-arterial extracorporeal membrane oxygenation. Since the first published report in 2006 of the successful use of lipid emulsion in treating a patient with cardiac arrest during regional anesthesia, infusion of lipid emulsion has been increasingly used to treat LAST in various clinical situations and patient populations (see below).[76] Lipid emulsion infusion is currently included

Table 5. Anaphylaxis

Assessment	• Confusion, altered mental status/presyncope • Flushing, 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 (*e.g.*, NMBD, antibiotics, blood products, IV contrast, or latex) • Stop surgery or procedure (if possible) • Administer 100% oxygen • Intubate immediately for respiratory distress or hypoxemia • Disconnect ventilator briefly if auto-PEEP is suspected • Target systolic blood pressure ≥ 90 mmHg • Continuous arterial blood pressure monitoring • Bolus 20 ml/kg of balanced solution crystalloid • Administer 50 to 100 μg IV epinephrine in repeated doses (or 200 to 500 μg IM if no IV access present) • Administer 100 to 300 μg IV epinephrine if hypotension persists after 3 to 5 min • Start epinephrine infusion (0.05 to 0.3 $\mu g \cdot kg^{-1} \cdot min^{-1}$) for a goal SBP > 90 mmHg; ± 2 U IV vasopressin • Add vasopressin or norepinephrine infusions for patients who are hypotensive (after 3 to 5 min) despite high doses of epinephrine • Administer H1 blocker (50 mg IV diphenhydramine) • Administer H2 blocker (20 mg IV famotidine) • Administer corticosteroid (*e.g.*, 50 to 150 mg IV hydrocortisone IV or 1 to 2 mg/kg IV methylprednisolone) *Arrest* • Start CPR • 100 to 1,000 μg IV epinephrine, repeat every 3 to 5 min or replace with one dose of 40 U IV vasopressin • Continue aggressive fluid resuscitation (up to 5 l) • Disconnect ventilator briefly if auto-PEEP is suspected • Administer adjunctive therapies listed in prearrest (H1 and H2 blockers and corticosteroids) • Consider extracorporeal life support
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

CPR, cardiopulmonary resuscitation; ICU, intensive care unit; IM, intramuscular; IV, intravenous; NMBD, neuromuscular blocking drug; PEEP, positive end-expiratory pressure; SBP, systolic blood pressure; U, unit(s).

in the treatment protocols of several professional societies, including the American Heart Association, the American Society of Regional Anesthesia and Pain Medicine, and the Association of Anaesthetists of Great Britain and Ireland.[77–79]

No prospective human trials have compared the use of a lipid infusion to placebo. However, there are convincing prospective randomized animal studies, and the Cochrane Policy Institute found 7 observational human studies and 136 human case reports (with 138 cases) describing the use of a lipid emulsion to treat LAST. A complete list of these reports and a summary overview of the data are available in the Supplemental Digital Content (https://links.lww.com/ALN/E188). Lipid infusion may result in a large increase in survival. Reported survival among five noncomparative observational studies was 389 of 454 (85.7%), and among 138 individual case reports plus 2 patients from the noncomparative observational studies having sufficient information to include, the reported survival was 134 of 140 (95.7%); however, survival was only inferred in 65 of these patients. Return of spontaneous circulation was achieved in 29 of 31 (93.5%) patients in cardiac arrest. Adverse neurologic outcomes were reported in 2.1% of the cases. The noncomparative observational studies identified no serious adverse events related to lipid emulsion administration.

Although the quality of the evidence is low, the impressive benefits in survival and the relative safety of the intervention, when administered as suggested in published advisories, favor using lipid emulsion for treating LAST. The few adverse events presented in the data are likely the result of LAST rather than the treatment. LAST is a life-threatening emergency, and our strong recommendation is based on the balance between a high potential for mortality benefit and minimal risk. Adverse events identified in two published cases as specifically related to infusion of lipid emulsion during treatment of LAST (ventilation/perfusion mismatch, somnolence, altered dural sinus signal in magnetic resonance imaging) occurred during massive overdose of lipid emulsion; both patients were children, and both recovered without sequelae.[80,81] Adhering to the dosing limit (10 to 12 ml/kg lean body mass) will reduce this risk.

Numerous case reports describe the successful use of lipid emulsion for treating LAST in a variety of patients, including pediatric, geriatric, and parturients. The available data do not identify any situation or population unresponsive to the beneficial effects of lipid emulsion infusion or at a higher risk for possible complications due to this treatment. However, a theoretical concern exists for patients with familial hypertriglyceridemia. It is reasonable to follow blood triglyceride levels after treatment of LAST in these patients and those in whom lipid doses near or greater than the dosing limits were used. Most hospitals maintain a lipid emulsion supply because it is also used for parenteral nutrition. Therefore, implementing lipid emulsion use for treating LAST is unlikely to be resource intensive.

2025 Recommendation: We recommend an infusion of 20% (w/v) lipid emulsion in perioperative patients with LAST.

Strong recommendation, low certainty of evidence

Evidence Summary for Epinephrine in LAST Management. Small, randomized trials in rats investigated the role of epinephrine (alone and in combination with lipid emulsion)

in treating cardiac arrest due to LAST. Combining epinephrine and lipid emulsion resulted in better ROSC and survival than either drug alone or placebo.[82,83] However, epinephrine was associated with worse outcomes at doses of 10 μg/kg and higher.[83] A study in pigs suggested the benefit of epinephrine for survival in LAST, especially when combined with lipid infusion. Still, there are concerns that a porcine model is not a reliable surrogate for human physiology.[84,85]

Standard doses of epinephrine may be detrimental in cardiac arrest from LAST due to the drug's metabolic effect on the myocardium.[86] Limited data suggest that it may still provide benefit at lower doses than commonly used in cardiac arrest, but the evidence is very uncertain.

2025 Recommendation: We suggest administering epinephrine (initially 10 μg intravenously) for hypotension due to LAST.

Conditional recommendation, very low certainty of evidence

2025 Recommendation: We suggest administering epinephrine (initially 100 to 300 μg intravenously) for cardiac arrest due to LAST.

Conditional recommendation, very low certainty of evidence

Additional Considerations. Patients at increased risk of developing LAST include those at the extremes of age; those with preexisting heart disease, especially ischemia, and conditions associated with low cardiac output or arrhythmia; small patient size; clinical fragility; sarcopenia; those receiving local anesthetic *via* multiple routes (*e.g.*, local infiltration analgesia intravenous infusion and peripheral nerve block in a single patient); those receiving prolonged or multiple catheter infusion of local anesthetic; those having cardiac surgery; and those with chronic comorbidities that affect metabolism including carnitine deficiency.[87] A review of published cases of LAST over the past several decades indicates fewer than 50% of cases follow the classic presentation of early-onset seizure progressing to cardiac arrest. There can be a substantial delay in the onset of LAST or a complete absence of neurologic symptoms in the presence of cardiovascular symptoms.[74,88] Prevention is best accomplished by using standard safety measures for administering local anesthetic (*i.e.*, monitoring, incremental dosing, frequent aspiration, use of a tracer like epinephrine, and test dosing where appropriate), adherence to local anesthetic dosing guidelines, reducing the total dose in patients at increased risk, and use of ultrasound guidance when possible. Avoiding regional anesthesia and local anesthetics may be indicated in some cases. Recent information suggests that lidocaine (alone or in combination) has become the local anesthetic most frequently associated with LAST and that topical administration of local anesthetic (*e.g.*, topicalizing airway mucosal surfaces) is increasing in frequency as a cause of LAST.[89]

Early diagnosis and intervention are important to attenuating the progression of LAST and improving the likelihood of a favorable outcome. The initial goals in managing LAST are to establish adequate oxygenation and ventilation and to prevent seizures by administering a benzodiazepine. Early infusion of 20% lipid emulsion solution will reduce the level of local anesthetic in target tissues (brain and heart) and thereby slow or reverse the progression of toxicity and potentially prevent cardiovascular collapse.[90,91] The American Society of Regional Anesthesia and Pain Medicine (Pittsburgh, Pennsylvania) created a checklist for LAST to aid in dosing and management.[77] Propofol contains a lipid emulsion; however, the lipid content (10%) of commercial propofol formulations cannot provide sufficient lipid to change LAST. Furthermore, the high (1%) concentration of propofol prevents using large volumes, as propofol causes myocardial depression, and therefore, more than very small doses can worsen prearrest signs of toxicity and worsen hypotension.

Once clinical stabilization is accomplished, continued monitoring is paramount, as symptoms can recur after initial apparent resolution.[92] Table 6 outlines the steps in the management of LAST. Amiodarone is the drug of choice for arrhythmias from LAST, as lidocaine (a local anesthetic and antiarrhythmic) can worsen toxicity. If refractory cardiac arrest persists, extracorporeal life support may be initiated.[93]

Malignant Hyperthermia

Evidence Summary. Malignant hyperthermia (MH) is a rare pharmacogenetic disorder characterized by dysregulation of calcium within skeletal muscle after exposure to volatile anesthetics or succinylcholine. The release of calcium from the sarcoplasmic reticulum into the cytoplasm causes sustained muscle contraction that can progress to muscle necrosis and hypermetabolism and ultimately cardiac arrest. Several inherited and sporadic genetic abnormalities are associated with MH, but the most common is in the ryanodine receptor 1 (RYR1) gene that encodes the ryanodine receptor, a key channel regulating the movement of calcium within skeletal muscle cells. MH tends to occur more commonly in men and younger patients, with an incidence ranging between 1:35,000 and 1:68,000 surgical discharges.[94–96]

Dantrolene has been the preferred treatment for MH for almost four decades. Due to the sporadic and unexpected presentation of MH, no RCTs have investigated the use of dantrolene to treat MH. The available literature supporting dantrolene use comes from observational studies. The most compelling of these studies correlates a 27% absolute decrease in mortality and ventricular arrhythmias with increased use of dantrolene in a series of 338 cases

Table 6. Local Anesthetic Systemic Toxicity

Assessment	• Dynamic and progressive signs and symptoms • Neurologic instability ○ Perioral numbness, dysgeusia, auditory complaints ○ Seizures, agitation, hallucination, obtundation, coma • Cardiac instability ○ Hypertension and tachycardia (initial) ○ Progressive bradycardia, conduction delay or heart block ○ Hypotension ○ Arrhythmia
Initial management	*Prearrest* • Stop administering local anesthetic • Administer 100% oxygen and manage airway for hypoxemia and/or hypoventilation • Intubate for persistent respiratory distress or hypoxemia • Treat seizures with benzodiazepines (small doses of propofol may be used if benzodiazepines are not immediately available) • Administer IV 20% lipid emulsion 1.5 ml/kg (lean body mass) over 2 to 3 min, then infuse 0.25 ml · kg^{-1} · min^{-1} (~20 ml/min) until 10 min after ROSC • For persistent hemodynamic instability, consider a second bolus and doubling the rate of infusion (0.5 ml · kg^{-1} · min^{-1}) • Limit total lipid dose (bolus + infusion): 10 to 12 ml/kg lean body mass • Administer 10-μg IV epinephrine boluses for SBP < 90 • Consider transcutaneous or intravenous pacemakers for all symptomatic bradycardic rhythms with pulse • Consider amiodarone for persistent arrythmias • Avoid: vasopressin, calcium channel blockers, β blockers, and lidocaine *Cardiac arrest* • Start CPR (prolonged if necessary) • Continue infusion of lipid emulsion (as above) • Plan for possible VA-ECMO or other eCPR (*e.g.*, alert perfusionist) • Administer 100 of 300 μg IV epinephrine • Administer IV sodium bicarbonate if pH < 7.2 or for hyperkalemia • If no ROSC within 15 min of the first bolus of lipid emulsion, a second bolus followed by a doubling of the rate of infusion is appropriate being mindful of the total lipid dosing limit (below) • Administer 300 mg IV amiodarone for ventricular arrhythmias • Avoid: vasopressin, calcium channel blockers, β blockers, and lidocaine or procaine • Limit total lipid dose (bolus + infusion): 10 to 12 ml/kg lean body mass
Subsequent management	• Monitor for recurrence or delayed progression

CPR, cardiopulmonary resuscitation; ECG, electrocardiogram; ECMO, extracorporeal membrane oxygenation; eCPR, extracorporeal CPR; IV, intravenous; ROSC, return of spontaneous circulation; SBP, systolic blood pressure; VA, venoarterial.

of likely MH in Japan.[97] Other studies from the United States and Canada correlate mortality (15 probable MH cases),[98] various morbidities (121 likely cases),[99] and complication rates (129 likely cases) with delays in dantrolene administration.[100] Although all studies are observational, the powerful signal, consistent directionality, and underlying physiologic logic provide sufficient evidence to support dantrolene as the first-line therapy for suspected MH and even the benefit of early, rather than delayed, administration of dantrolene. This evidence leads us to strongly recommend immediate dantrolene administration in patients with diagnosed or presumed MH in the perioperative period. Using the GRADE approach, the certainty of evidence has been increased to moderate. Dantrolene likely results in reduced mortality when given for MH.

All the studies supporting our recommendation come from well-resourced healthcare settings. The costs of maintaining a ready supply of dantrolene are moderate and may not be feasible[101] in particular low- and middle-income settings where triggering anesthetic agents are used, especially when prioritizing resources for more common problems is a consideration.

2025 Recommendation: We recommend administering intravenous dantrolene (2.5 mg/kg) for MH.

Strong recommendation, moderate certainty of evidence

Additional Considerations. Sinus tachycardia and hypercapnia are the earliest presenting signs of MH, followed by generalized muscle rigidity, commonly noticed in the jaw at the outset, generalized rigidity, and hyperpyrexia. Electrolyte abnormalities, including hyperkalemia, hypocalcemia, and mixed respiratory–metabolic acidoses, are often seen on a blood gas analysis. The first step in managing MH is immediately discontinuing all triggering agents. Ongoing therapy revolves around the mitigation of progressive hyperthermia, consequent tissue injury, and its sequelae. Given the extent of skeletal muscle injury, rhabdomyolysis is common in these patients and can lead to acute renal failure and hyperkalemia. In severe cases, several complications occur

with increased temperature, including disseminated intravascular coagulation.[101,102] There is a direct correlation of mortality with an elevation in temperature.[103]

Dantrolene can be administered every 5 min until symptoms resolve. Other diagnoses should be considered if symptoms persist after 10 mg/kg administration.[101] Dantrolene should be given at maintenance doses (1 mg/kg) every 6 h for 24 h to prevent recurrence.[101,104] Older versions of dantrolene (Dantrium [Par Pharmaceutical, USA] and Revonto [US WorldMeds, LLC, USA]) require a time-consuming process to dissolve the powder in 60 ml of sterile water. A newer dantrolene formulation (Ryanodex [Eagle Pharmaceuticals, USA]) can be mixed in 5 ml of sterile water in under 1 min.[101]

In contrast to dihydropyridine calcium channel blockers, verapamil and diltiazem are contraindicated in these patients because of the risk of hyperkalemia. In addition to fluid resuscitation and dantrolene, aggressive cooling measures should be instituted, and ventilatory parameters should be optimized to mitigate complications in patients with MH. It should be noted that dantrolene is a muscle relaxant, and its use is not without potential side effects. Monitoring during and after its administration for complications that could arise from weakness (*e.g.*, respiratory failure) and MH recrudescence means significant treatment costs are associated with caring for the condition.[101]

Clinicians can also utilize MH resources on the Malignant Hyperthermia Association of the United States (Sherburne, New York) website (www.mhaus.org) in the United States and from the European Malignant Hyperthermia Group (www.emhg.org) in Europe. Table 7 outlines the steps in the management of MH.

Traumatic Cardiac Arrest

Evidence Summary. Permissive hypotension is a resuscitative strategy for patients with hemorrhagic shock that aims to maintain a lower-than-normal blood pressure until definite hemorrhage control is accomplished. This strategy could help minimize blood loss, prevent consumptive, and dilutional coagulopathy and avoid complications of aggressive fluid resuscitation while theoretically increasing the risk of end-organ dysfunction (renal/neurologic dysfunction) if not carefully executed.

Studies investigating permissive hypotension compared to standard resuscitation and restricted compared to standard fluid resuscitation in perioperative patients with hemorrhagic shock were evaluated. The search yielded 35 studies, including 9 RCTs and 17 observational studies. We identified all-cause mortality, renal failure, neurologic outcomes, and extent of hemorrhage as critical outcomes with permissive hypotension. The pooled results from all nine RCTs suggest that permissive hypotension may reduce mortality risk (relative risk, 0.68; 95% CI, 0.49 to 0.95; N = 2,858; low-certainty evidence), but the evidence is very uncertain. This finding was consistent with the pooled data from the 17 observational studies. The three RCTs that investigated this outcome showed little to no difference in renal failure. Only one RCT investigated neurologic outcomes and reported little to no difference in risk of adverse neurologic outcomes with permissive hypotension.[105] The Cochrane group's evidence assessment for permissive hypotension for perioperative patients with a traumatic cardiac arrest is found in the Supplemental Digital Content (https://links.lww.com/ALN/E189).

Similarly, there was little to no difference in blood loss between the groups in the two RCTs that reported this outcome.[105,106] Only one observational study reported lower blood transfusion rates in patients who received restricted fluid resuscitation.[107] One RCT and two observational studies identified no difference in the rate of blood product administration between the groups.[106,108,109] Two RCTs and two observational studies indicated reduced adverse events with permissive hypotension (very low certainty of evidence).[110–113]

2025 Recommendation: We suggest permissive hypotension by restricting fluid administration, targeted to a goal mean arterial pressure of 50 mmHg or systolic blood pressure of 70 mmHg, for the resuscitation of patients with traumatic cardiac arrest before definitive assessment of bleeding sources.

Conditional recommendation, very low certainty of evidence

Additional Considerations:. Traumatic cardiac arrest carries a high mortality rate, but in survivors, neurologic outcomes appear to be much better than in other causes of cardiac arrest.[114–117] This apparent paradox is likely accounted for by this population's heterogenous etiology of arrest. Uncontrolled bleeding is the leading cause of death (48%), followed by tension pneumothorax (13%), asphyxia (13%), and pericardial tamponade (10%).[118] Large systematic and nationwide reviews report an overall survival rate of 7 to 10% in a mixed group of blunt, penetrating, and unspecified presentations.[114,119,120] However, 40 to 46% of those who survive have good neurologic outcomes.[116,117,119]

In a 70-kg man, hypotension may present late and far after 1,500 ml of blood loss. In the absence of tension physiology, hypotension is observed after losing approximately 30% of circulating blood volume.[121] Resuscitative efforts in traumatic cardiac arrest should focus on immediate assessment and simultaneous treatment of reversible causes, such as pressure or tourniquet placement for external hemorrhage, or relief of tension pneumothorax or tamponade (fig. 7).[115,122] Invasive occlusion devices have fallen out of favor due to the technical difficulties of insertion (morbidity and mortality) despite the potential benefits.[123] The primary emphasis should be 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.[122,124–126] Therefore, chest compressions take a lower priority than the immediate treatment of reversible causes.

Table 7. Malignant Hyperthermia

Assessment	• Hypercapnia, precipitous rise in $ETCO_2$ • Tachycardia • Tachypnea in nonparalyzed patients • Muscle rigidity/masseter spasm • Hyperthermia
Initial management	*Ventilation* • Discontinue volatile anesthetics and switch to IV anesthetic (if necessary) • Stop procedure when feasible • Disconnect patient from ventilator and switch to manual bag ventilation from a separate source of oxygen. • Switch to a dedicated clean anesthesia ventilator or a transport or ICU ventilator (if available) • Target $ETCO_2$ of 50 to 60 mmHg *Medication* • Administer 2.5 mg/kg IV sodium dantrolene *via* largest bore IV • Repeat bolus of dantrolene, titrating to resolution of presenting symptoms (*i.e.*, tachycardia and hypercarbia [10 mg/kg suggested upper limit but give more as needed up to 30 mg/kg]) • Avoid calcium antagonists *Fever* • Place ice packs to groin, axilla, and neck • Applying cooling devices if available for targeted temperature management • Initiate cold nasogastric or peritoneal lavage when feasible • Stop cooling measures at 38°C to avoid overshooting *Hyperkalemia* • Administer 10 mg/kg IV calcium chloride • Administer 0.1 U/kg insulin • Administer 50 ml of D50W for adult or 1 ml/kg for pediatrics • Repeat as needed *Metabolic acidosis* • Administer 100 mEq of HCO_3 in adults, then titrate to pH 7.2. *Myoglobinuria with oliguria* • Place urinary catheter • Increase rate of fluid resuscitation • Initiate invasive pressure monitoring when feasible, • Consider administration of HCO_3 to neutralize urine pH • Consider intravenous mannitol *Disseminated intravascular coagulation* • Institute supportive measures **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 • Administer dantrolene 1 mg/kg every 6 h for a minimum of 24 h • 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 the Malignant Hyperthermia Association of the United States (Sherburne, New York) website https://www.mhaus.org/.
D50W, dextrose in water (50%); $ETCO_2$, end-tidal carbon dioxide; ICU, intensive care unit; IV, intravenous; MH, malignant hyperthermia; U, unit(s).

Current Brain Trauma Foundation guidelines for severe traumatic brain injury recommend maintaining an systolic blood pressure greater than or equal to 100 mmHg for patients 50 to 69 yr of age or greater than or equal to 110 mmHg for patients 15 to 49 yr of age or older than 70 yr to improve outcomes and reduce mortality.[127] These recommendations are based on a single study that did not compare patient outcomes and has not been validated in any other cohort.[128] A higher blood pressure target (*e.g.*, systolic blood pressure greater than or equal to 110 mmHg) may be preferred in brain injury and suspected increased intracranial pressure.[129]

Figure 7 shows an approach to managing traumatic cardiac (peri-) arrest. Tranexamic acid (TXA; loading dose 1 g over 10 min followed by infusion of 1 g over 8 h) increases survival from traumatic hemorrhage.[130,131] It is effective within the first 3 h after a traumatic injury. However, TXA should be initiated no later than 4 h after the injury, as late dosing is associated with higher mortality rates. While TXA should be given empirically in the cases of hypotension from hemorrhage, many clinicians will wait for confirmation of thrombolysis *via* viscoelastic testing before they give TXA. Fibrinolytic shutdown or an inhibition of the fibrinolytic system is a major concern

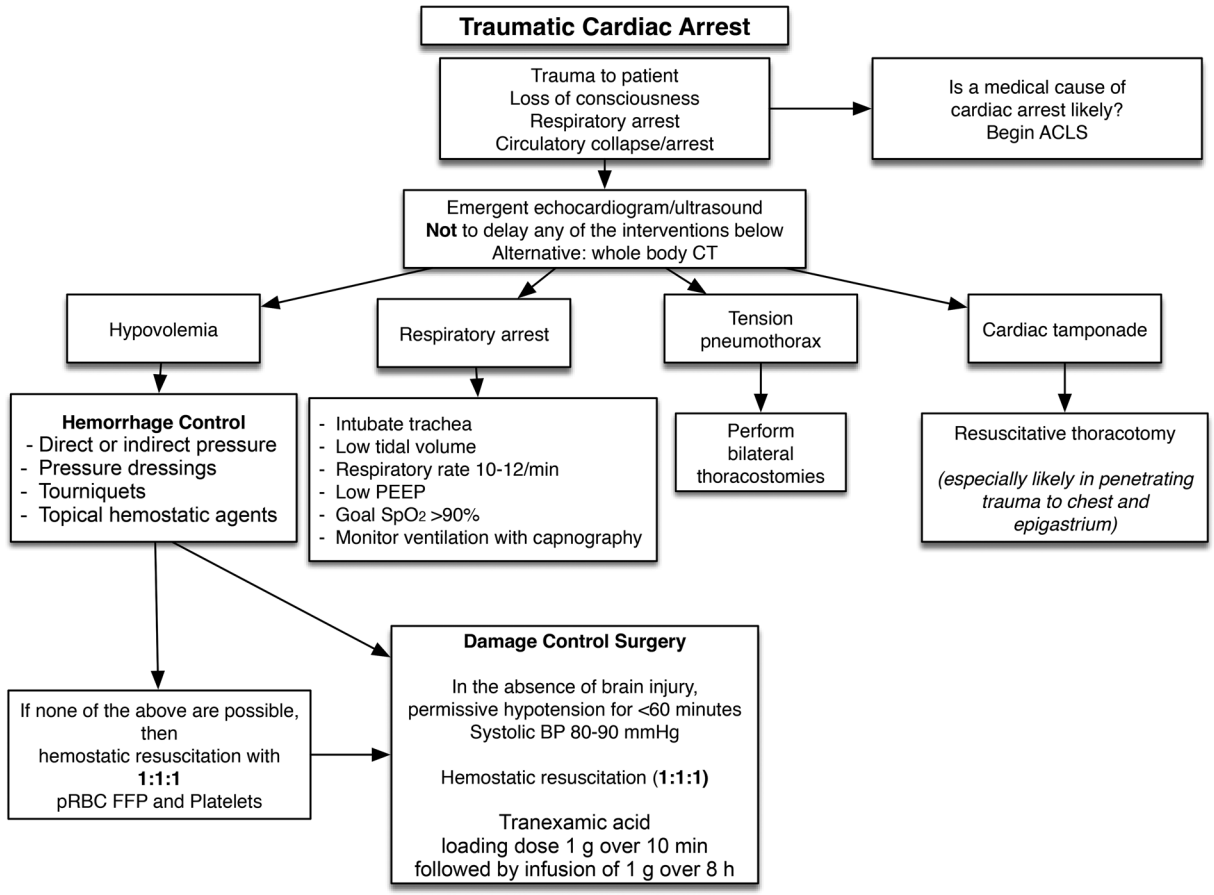

Fig. 7. Treatment algorithm for traumatic cardiac arrest. ACLS, Advanced Cardiac Life Support; CT, computerized tomography; PEEP, positive end-expiratory pressure.

after TXA, especially in the patients with peripheral vascular injuries.[132]

Cardiac Arrest with Neuraxial Blockade

Evidence Summary for Atropine in Cardiac Arrest after Neuraxial Blockade. High spinal, also known as total spinal or complete spinal anesthesia, is a rare complication of neuraxial anesthesia techniques (*i.e.*, spinal, combined spinal–epidural, and epidural). A high spinal block can cause sympathetic blockade with severe hypotension, bradycardia, cardiac arrest, and respiratory failure. The Serious Complication Repository (SCORE) Project of the Society for Obstetric Anesthesia and Perinatology (Lexington, Kentucky) found that high neuraxial block occurred in 1 of every 4,336 anesthetics in obstetric patients.[133]

Atropine has been reported as a rescue medication for cardiac arrest from neuraxial anesthesia. The evidence supporting the use of atropine in the setting of spinal arrest is limited to case reports that often describe the prearrest cardiac rhythm as nonshockable (bradycardia, asystole).[134,135] Atropine was frequently used for rescue, often in combination with other medications, such as epinephrine and ephedrine, suggesting that escalation is appropriate if initial therapies are not effective. Atropine may improve mortality, but the evidence is very uncertain. It is unclear whether atropine is preferred over other treatments, such as epinephrine, but it is a reasonable rescue option.

Paradoxical bradycardia with lower doses of atropine has been reported.[136–140] Because of these concerns, we recommend a dose of 1.0 mg. Patients after a heart transplant who may develop a high atrioventricular block with atropine administration are excluded from this recommendation. Our recommendation pertains to cardiac arrest or suspected cardiac arrest; therefore, hypotension in neuraxial anesthesia may be treated differently.

2025 Recommendation: We suggest administering atropine (1 mg intravenously) for high spinal cardiac arrest.

Conditional recommendation, very low certainty of evidence

Table 8. Cardiac Arrest and Neuraxial Anesthesia

Assessment	• Bradycardia • Hypotension • Neuromuscular weakness
Initial management	*Prearrest* • Discontinue anesthetic or sedation infusion • Administer 100% oxygen • Intubate for respiratory distress or hypoxemia • Treat bradycardia with IV atropine (1mg) or • Administer IV epinephrine 100 to 200 μg • Consider transcutaneous or transvenous pacing for all symptomatic patients *Cardiac arrest* • Start CPR • Administer 1 mg IV atropine • Administer 1 mg IV epinephrine if no response
Subsequent management	• Consider starting epinephrine infusion (0.05 to 0.1 $\mu g \cdot kg^{-1} \cdot min^{-1}$)

CPR, cardiopulmonary resuscitation; IV, intravenous.

Evidence Summary for Epinephrine in Neuraxial Blockade. The role of epinephrine in the hemodynamic support of high spinal has not been established in comparison with other drugs or therapies (*e.g.*, α-agonists, crystalloids) or no therapies. No studies directly investigated epinephrine *versus* other medications or treatments for the management of high spinal. Indirect evidence exists comparing epinephrine with other drugs to treat hypotension after the onset of spinal anesthesia. The two outcomes investigated by studies on the topic were bradycardia and hypotension. No studies addressed outcomes of critical importance (*e.g.*, mortality).

One randomized study compared epinephrine, norepinephrine, phenylephrine, and saline (0.9%) during elective caesarian deliveries under spinal anesthesia.[141] This study found similar incidences of hypotension for epinephrine, norepinephrine, and phenylephrine. All results were marked by serious or very serious imprecision. Another trial compared continuous infusions of epinephrine or phenylephrine during elective cesarean delivery under spinal anesthesia.[142] Epinephrine was associated with a reduced risk of bradycardia and hypotension, with few outcomes reported. Continuous infusions of epinephrine to treat hypotension during spinal anesthesia increased heart rates, whereas heart rates decreased with phenylephrine,[143] and severe bradycardia (heart rate less than 45 beats/min) occurred more frequently with phenylephrine (two patients) than with epinephrine (no patients). Epinephrine was effective in restoring systolic blood pressure after spinal anesthesia.

The evidence is very uncertain about the effect of epinephrine on survival. Most evidence on the use of epinephrine for the treatment of hypotension comes from pregnant individuals undergoing cesarean deliveries, and two of the studies studied prophylactic dosing.[141,142] Therefore, the effectiveness of epinephrine specifically for mitigating the effects of high spinal anesthesia is insufficiently studied, and the evidence only provides a physiologic rationale for administration in population subgroups or nonarrest settings.

2025 Recommendation: For treating symptoms considered related to high spinal, we suggest administering an intravenous epinephrine bolus (100 to 200 μg) for high spinal pathophysiology in situations of arrest or when arrest appears imminent.

Conditional recommendation, very low certainty of evidence

Additional Considerations. An epinephrine infusion (initial rate 0.05 to 0.1 $\mu g \cdot kg^{-1} \cdot min^{-1}$) is best supported by the evidence for situations with sufficient time to implement such treatment. Early identification of a high spinal block has the potential to prevent a cardiac arrest.[144] Unrecognized migration of an epidural catheter into the dural space can cause a high spinal block.[145] For a high spinal, positioning a patient in the Trendelenburg position may improve hemodynamics by improving venous return, leading to increased cardiac output; however, Trendelenburg may worsen hemodynamics because local anesthetic may spread cephalad, leading to the progression of the block.[146,147] Table 8 outlines an approach to managing neuraxial blockade and cardiac arrest.

Cardiopulmonary Resuscitation (CPR)

Figure 8 outlines our comprehensive algorithm to manage a cardiac arrest.

Evidence Summary for CPR in the Prone Position. This conditional recommendation is based on case report data and one small case series (n = 6),[148] with a high risk of bias. Cases note ROSC (16 of 22) and survival (12 of 19 to hospital discharge; 10 of 19 overall survival) with prone CPR, suggesting efficacy.[149–163] Ten case reports report neurologic outcomes, with 2 of 10 patients exhibiting neurologic injury.[150,151,154–160,163] The Cochrane group's evidence summary for CPR for perioperative patients in the prone position can be found in the Supplemental Digital Content (https://links.lww.com/ALN/E190). No data are available to compare neurologic outcomes with supine CPR. Bridging this knowledge gap is essential to inform future recommendations, as the value placed on survival might be less, and the costs associated with long-term care would be higher if poor neurologic function were more common with prone CPR than supine. The evidence is very uncertain about the effect of prone CPR on survival.

Other undesirable effects are unknown as data are lacking. The time it takes to turn a patient weighs heavily in calculating perceived risks and benefits. There are no good data to inform this discussion, but the delay is likely clinically substantial in most locations. Overall, for patients arresting in the prone position, the balance of effects likely favors prone CPR over delayed supine CPR because of the need to turn the patient. There may also be a subgroup

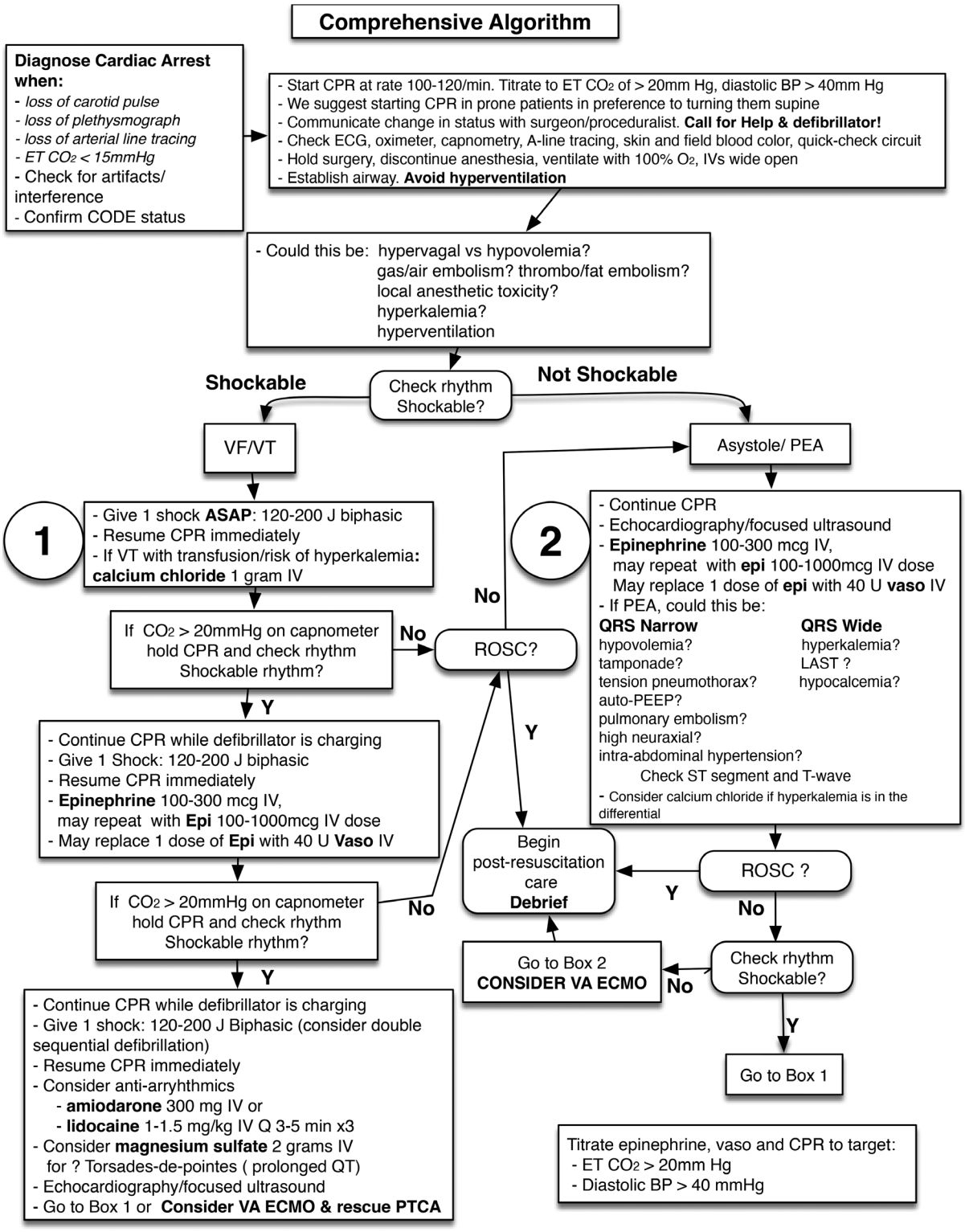

Fig. 8. Comprehensive algorithm. ASAP, as soon as possible; CPR, cardiopulmonary resuscitation; ECG, electrocardiogram; ECMO, extracorporeal membrane oxygenation; Epi, epinephrine; $ETCO_2$, end-tidal carbon dioxide; IV, intravenous; PEA, pulseless electrical activity; PTCA, percutaneous transluminal coronary angioplasty; ROSC, return of spontaneous circulation; Vaso, vasopressin; VF, ventricular fibrillation; VT, ventricular tachycardia.

effect for shockable rhythms, given that cardioversion/defibrillation may be less effective in the prone position.

2025 Recommendation: In perioperative patients with arrest in the prone position, we suggest CPR in the prone position over turning supine to perform classic CPR.

Conditional recommendation, very low certainty of evidence

Evidence Summary for Point-of-Care Ultrasound (POCUS) during CPR. Ultrasound can diagnose reversible perioperative and intensive care unit arrest etiologies such as esophageal intubation, abdominal bleeding, pneumothorax, pulmonary embolism, and tamponade during an arrest (especially with pulseless electrical activity or asystole).[164,165] Visualization of the heart also distinguishes true asystole from fine ventricular fibrillation. Several studies of ultrasound use during cardiac arrest have reported that 10 to 35% of patients with asystole have evidence of cardiac contraction.[166,167] When cardiac contraction is observed, arrest management may change, and the likelihood of survival improves.[167–169] Furthermore, POCUS may be used to assess the interventions' efficacy and the potential for the return of spontaneous circulation.[164,169–174]

Several studies have demonstrated that using POCUS during cardiac arrest increases chest compression interruptions and delays the resumption of compressions.[175,176] Obtaining external ultrasound images should not impede chest compressions; image acquisition should be limited to 10 s.[165] Placing the probe in the subxiphoid position is ideal because it does not impede hand placement for compressions. While images can be acquired during CPR, interpretation should occur after the ultrasonographer steps away from the bedside. Given the challenges in obtaining images during chest compressions, we recommend that the most experienced ultrasonographer obtain images. If transthoracic imaging is challenging (due to the presence of surgical drains, anatomical barriers, subcutaneous emphysema, and so forth), transesophageal echocardiography (TEE) can be used (without interrupting chest compressions).[165] Chest compressions can obstruct cardiac output if hands are placed over the left ventricular outflow tract, and TEE can guide hand placement during CPR.[177–179] TEE insertion has been associated with pharyngeal and esophageal injuries.[180]

Discussion

Our panel selected 12 PICO questions for which we believed there was a need to provide evidence-based recommendations based on the GRADE methodology. Although evidence was limited in all cases, we offer 13 recommendations and one best practice statement for clinicians, highlighting important areas for future research. However, our process has revealed many gaps in the available evidence and highlighted important data for interventions that cannot be addressed by human trial design, such as with malignant hyperthermia. For many of our PICO questions, there were very few studies to inform recommendations, underscoring the need for large clinical trials in anesthesia and perioperative medicine to address these questions. We also recognize that our recommendations should be considered in the context of significant heterogeneity across patient types, surgical settings, and resource availability. Given the wide variability in perioperative practices and patient physiology, the recommendations in this guidance should be integrated with standard resuscitation principles.

Our recommendations highlight the unique circumstances of perioperative and periprocedural arrest and provide specific guidance for resuscitation in this setting. They complement basic and advanced cardiac life support principles,[60] highlighting key distinctions and linking them to evidence.

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Competing Interests

Dr. Moitra is the American Society of Anesthesiologists (Schaumburg, Illinois) Liaison to the American Heart Association (Dallas, Texas), is on the board of directors of the American Board of Anesthesiology (Raleigh, North Carolina), is on the medical advisory boards of Tell Health (Chicago, Illinois) and Fize Medical (Modi'in-Maccabim-Re'ut, Israel), and is a medical malpractice expert. Dr. Stahl is a physician advisor for CurbsideMD (Cranston, Rhode Island) and is a consultant for C8Health (San Francisco, California). Dr. O'Connor is on the scientific advisory board for CLEW Medical (Netanya, Israel). Dr. Einav has received consultancy fees from Medint Medical Intelligence. Dr. Lakbar has received funding from Viatris Sante (Lyon, France). Dr. Shander has received funding from Pharmacosmos (Copenhagen, Denmark), Vifor (Zurich, Switzerland), CSL (Sydney, Australia), Lindis Care (Frankfurt, Germany), I sep (Paris, France), CSL-Vifor (Sydney, Australia), HC1, and Pfizer. Dr. Weinberg is an officer and shareholder of ResQ Pharma, Inc. (Glenview, Illinois). The other authors declare no competing interests.

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Supplemental Digital Content

Cochrane PPV, https://links.lww.com/ALN/E187
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Cochrane Hypotension Trauma, https://links.lww.com/ALN/E189
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Q1 PPV EtD Table, https://links.lww.com/ALN/E191
Q1 PPV EP, https://links.lww.com/ALN/E192
Q2 Hyperventilation EtD Table, https://links.lww.com/ALN/E193
Q2 Hyperventilation EP Table, https://links.lww.com/ALN/E194
Q3 Atropine Bradycardia EtD Table, https://links.lww.com/ALN/E195
Q3 Atropine Bradycardia EP Table, https://links.lww.com/ALN/E196
Q4 Epi Anaphylaxis EtD Table, https://links.lww.com/ALN/E197
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Q7 Epi LAST EP Table, https://links.lww.com/ALN/E204
Q8 Dantrolene MH EtD Table, https://links.lww.com/ALN/E205
Q8 Dantrolene MH EP Table, https://links.lww.com/ALN/E206
Q9 Permissive Hypotension Trauma EtD Table, https://links.lww.com/ALN/E207
Q9 Permissive Hypotension Trauma EP Table, https://links.lww.com/ALN/E208
Q10 Atropine High Spinal EtD Table, https://links.lww.com/ALN/E209
Q10 Atropine High Spinal EP Table, https://links.lww.com/ALN/E210
Q11 Epi High Spinal EtD Table, https://links.lww.com/ALN/E211
Q11 Epi High Spinal EP Table, https://links.lww.com/ALN/E212
Q12 Prone CPR EtD Table, https://links.lww.com/ALN/E213
Q12 Prone CPR EP Table, https://links.lww.com/ALN/E214

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