NARRATIVE REVIEW ARTICLE

Causes of Perioperative Cardiac Arrest: Mnemonic, Classification, Monitoring, and Actions

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Perioperative cardiac arrest (POCA) is a catastrophic complication that requires immediate recognition and correction of the underlying cause to improve patient outcomes. While the hypoxia, hypovolemia, hydrogen ions (acidosis), hypo-/hyperkalemia, and hypothermia (Hs) and toxins, tamponade (cardiac), tension pneumothorax, thrombosis (pulmonary), and thrombosis (coronary) (Ts) mnemonic is a valuable tool for rapid differential diagnosis, it does not cover all possible causes leading to POCA. To address this limitation, we propose using the preload-contractility-afterload-rate and rhythm (PCARR) construct to categorize POCA, which is comprehensive, systemic, and physiologically logical. We provide evidence for each component in the PCARR construct and emphasize that it complements the Hs and Ts mnemonic rather than replacing it. Furthermore, we discuss the significance of utilizing monitored variables such as electrocardiography, pulse oxygen saturation, end-tidal carbon dioxide, and blood pressure to identify clues to the underlying cause of POCA. To aid in investigating POCA causes, we suggest the Anesthetic care, Surgery, Echocardiography, Relevant Check and History (A-SERCH) list of actions. We recommend combining the Hs and Ts mnemonic, the PCARR construct, monitoring, and the A-SERCH list of actions in a rational manner to investigate POCA causes. These proposals require real-world testing to assess their feasibility. (Anesth Analg 2023;XXX:00–00)

GLOSSARY

A-SERCH = anesthetic care, surgery, echocardiography, and relevant check and history; **ACLS** = advanced cardiac life support; **ASA** = American Society of Anesthesiologists; **BP** = blood pressure; **CAD** = coronary artery disease; **CO** = cardiac output; **CPR** = cardiopulmonary resuscitation; **Ea** = arterial elastance; **ECG** = electrocardiography; **EDPVR** = end-diastolic pressure-volume relationship; **ESPVR** = end-systolic pressure-volume relationship; **F_IO_2** = inspired oxygen fraction; **Hs** = hypoxia, hypovolemia, hydrogen ions (acidosis), hypo-/hyperkalemia, and hypothermia; **LAST** = local anesthetic systemic toxicity; **LVOT** = left ventricle outflow tract; **MELD** = model for end-stage liver disease; **PCARR** = preload, contractility, afterload, and rate and rhythm; **PEA** = pulseless electrical activity; **PEEP** = positive end-expiratory pressure; **POCA** = perioperative cardiac arrest; **QRS** = xxxx; **QT** = xxxx; **Spo_2** = pulse oxygen saturation; **ST** = xxxx; **SV** = stroke volume; **TEE** = transesophageal echocardiography; **Ts** = toxins, tamponade (cardiac), tension pneumothorax, thrombosis (pulmonary), and thrombosis (coronary); **V/Q** = ventilation/perfusion; **V-fib** = ventricular fibrillation; **V-tach** = ventricular tachycardia

Perioperative cardiac arrest (POCA) is a devastating complication. Its incidence varies over time, geographic locations, and type of surgical cases. A Brazilian teaching hospital reported 35 cardiac arrests per 10,000 anesthetics based on data collected between 1996 and 2005.[1] The National Anesthesia Clinical Outcomes Registry reported an incidence of approximately 6 cardiac arrests per 10,000 cases based on 2010 to 2013 data.[2] For every 10,000 cases, POCA occurred in up to 4 to 5 instances in noncardiac surgery,[3,4] 73 cases in cardiac surgery,[5] 18 cases before skin incision in cardiac surgery,[6] 22 cases in pediatric surgery,[7] and 370 cases in adult liver transplantation.[8] In contrast, the median risk-adjusted in-hospital cardiac arrest incidence was 85 per 10,000 admissions.[9] Outcomes following POCA are devastating. One study reported a 30-day mortality of approximately 72% and a 30-day successful discharge rate of only 19% in surgical patients requiring resuscitation.[10]

Favorable outcomes rely on many factors, particularly the prompt recognition and correction of the underlying cause.[11] In reality, practitioners, while

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engaging in immediate and effective resuscitation, must simultaneously identify and correct the cause. Therefore, a deep understanding of and a structured approach to effectively search for potential POCA causes can promote resuscitative efforts. The widely known hypoxia, hypovolemia, hydrogen ions (acidosis), hypo-/hyperkalemia, and hypothermia (Hs) and toxins, tamponade (cardiac), tension pneumothorax, thrombosis (pulmonary), and thrombosis (coronary) (Ts) mnemonic is popularly used in the education and practice of cardiac arrest resuscitation.[12] The Hs refer to hypoxia, hypovolemia, hydrogen ions (acidosis), hypo-/hyperkalemia, and hypothermia, while the Ts refer to toxins, tamponade (cardiac), tension pneumothorax, thrombosis (pulmonary), and thrombosis (coronary).[12] This mnemonic tool is influential and has significantly contributed to cardiac arrest resuscitation; however, it does not cover all potential causes of POCA as it is not explicitly created for POCA. This mnemonic is random and lacks intuitive logic.[13] Thus, the effective investigation of POCA causes warrants further discussion.

This open-minded narrative review aims to discuss, on top of the Hs and Ts mnemonic, the additional approaches to investigate POCA causes based on a comprehensive literature review. There are 2 types of literature discussing POCA causes: 1 is case reports which is understandable as it is challenging to study POCA prospectively due to its unpredictability and rarity; the other is retrospective cohort studies based on hospital databases or registries. Consequently, our review is primarily based on case reports and cohort analyses, although we prioritize high-quality evidence when available. We note that our article focuses on POCA causes, not treatment, as no publications specifically discuss the causes responsible for POCA. For more detailed information on treatment, we recommend referring to the established guidelines for cardiac arrest resuscitation, even though they were not developed explicitly for POCA.[11,14]

DIFFERENCES BETWEEN PERIOPERATIVE, IN-HOSPITAL, AND OUT-OF-HOSPITAL CARDIAC ARREST

We define POCA as a cardiac arrest occurring in a surgical patient, from arriving in the operating room to being discharged from the postanesthesia care unit or 24 hours after surgery if admitted to the intensive care unit. POCA is distinct from in-hospital and out-of-hospital cardiac arrest (Table 1), with some highlights as follows.[15,19,20]

- Etiology: POCA is generally directly caused by adverse surgical and anesthetic events.
- Patient information: The relevant information is readily available in hospitalized surgical patients who develop POCA.
- Anesthesia and sedation: The acute mental status change is indiscoverable if POCA occurs in anesthetized or sedated patients.
- Monitoring: Patients are routinely monitored in the perioperative setting.
- Witness: POCA is usually witnessed. As a result, it is typically timely diagnosed and treated.
- Resources: Patients suffering from POCA benefit from abundant resources.

Table 1. Comparisons Between Perioperative, In-Hospital, and Out-of-Hospital Cardiac Arrest

Considerations	Perioperative cardiac arrest	In-hospital cardiac arrest	Out-of-hospital cardiac arrest
Incidence	Varying per surgery and geographic locations	290,000 per year in the United States[15]	350,000 per year in the United States[15]
Relationship with surgery and anesthetic care	Normally related	Maybe related, frequently unrelated	Normally unrelated
Witness	Normally witnessed	Frequently witnessed	Less chance of being witnessed
Monitoring	Monitored in the OR, frequently monitored outside of the OR	Monitored in the ICU, sometimes monitored outside of ICU	Normally not monitored
Time to resuscitation	Instantaneously or minimal delay	Within 5–10 min[16]	On average, approximately 20 min after the onset of cardiac arrest[15]
Airway	Frequently intubated or rapid airway securement possible	Approximately one-third of patients already intubated[15]	Airway not normally secured
Breathing	Ventilated or bag-valve-mask ventilation supplies readily available, oxygen source immediately available	Assisted ventilation supplies normally available, oxygen source normally available	Hands-only cardiopulmonary resuscitation, oxygen source delayed
Circulation	Intravenous access, drugs for circulation support, and fluid and blood products readily available	Sources for circulation support normally available, delays possible	Supportive measures normally not available
Manpower	ACLS-trained personnel normally involved timely in the resuscitative efforts	ACLS-trained personnel normally available, delays possible	ACLS-trained personnel normally not involved in the early resuscitative phase
Survival to discharge	>28%[10,16,17]	Approximately 25%[15]	8%–12%[15,18]

Abbreviations: ACLS, advanced cardiac life support; ICU, intensive care unit; OR, operating room.

- Outcomes: POCA appears to have a better survival-to-discharge rate than in-hospital and out-of-hospital cardiac arrest (Table 1).

POCA CAUSES REPORTED BY COHORT STUDIES

We summarized the POCA causes reported by cohort studies (Table 2).[1,2,4–6,8,17,21–25] We included risk factors in this summary; however, risk factors differ from causes, as a risk factor indicates an association, and association differs from causation. The summary demonstrated that the reported POCA risk factors and causes are abundantly diverse. It is overwhelming to remember all components for any practitioner. While the Hs and Ts mnemonic is a practical approach, it does not cover all possible POCA causes, which is an unignorable limitation.

In light of this discussion, we propose a physiology-based system to classify the causes of POCA to facilitate a systemic diagnostic investigation. Although the physiology underlying POCA (such as hypovolemia) may differ from the direct cause of POCA (such as massive bleeding), we argue that a physiological classification system makes investigating POCA causes much more practical, as illustrated in the following discussion.

PHYSIOLOGICAL CLASSIFICATION OF CARDIAC ARREST

We propose a classification system per the root physiological causes of cardiac arrest. The core physiological feature of cardiac arrest, disregarding any risk factors or specific direct causes, is a catastrophic reduction of cardiac output (CO). At least 1 of the 4 CO determinants—that is, preload, contractility, afterload, and heart rate and rhythm—must suffer a critical aberrant deterioration when CO plummets. Figure 1 shows the impacts of severe preload reduction, contractility impairment, and afterload increase on stroke volume. Significant CO reduction will ensue if the compensative increase in heart rate fails to ensue or is inadequate. The impact of a precipitous decrease in heart rate or severe arrhythmia on CO is also intuitive, that is, a significantly reduced number of adequate stroke volumes per minute resulting in substantial CO decline. In light of this discussion, we classify cardiac arrest according to CO's determinants: Preload crisis, Contractility crisis, Afterload crisis, and Rate and Rhythm crisis (PCARR) (Figure 2).

In the following sections, we review POCA causes in the context of the PCARR classification, with the supporting literature for each class presented. As cohort studies do not typically declare a causal effect, the following discussion is primarily based on case reports in which a cause is known or reported.

POCA DUE TO PRELOAD CRISIS

The common etiologies of preload crisis require an analysis following the path of blood flow from the peripheral circulation through the right heart, the pulmonary vasculature, and the left heart.

Hypovolemia

Massive bleeding can lead to severe hypovolemia. Overt massive bleeding is readily appreciable, while covert bleeding can delay diagnosis and is sometimes not revealed until postmortem. One example case described an otherwise healthy 26-year-old woman undergoing lumbar discectomy in the prone position to treat a prolapsed lumbar intervertebral disk at L4–5.[30] The procedure was unremarkable until 5 minutes after the discectomy when different monitor alarms sounded almost simultaneously. Cardiac arrest was speculated, and the patient turned supine for resuscitation. The abdomen appeared soft, not distended. The patient died. Postmortem found an aorta laceration about half a centimeter large at the level of discectomy and the retroperitoneal space full of blood.

Anaphylaxis can lead to severe hypovolemia. Multiple drugs, agents, and materials utilized in the perioperative environment can cause anaphylaxis, with clinical manifestations varying between cases.[31,32] Anaphylaxis can lead to vasodilation[33,34] and extravasation as evidenced by hemoconcentration,[33,35] and, thus, hypovolemia. Crucially, anaphylaxis can also reduce afterload and impair myocardial contractility.[36]

One example case described a 36-year-old primigravida undergoing an elective cesarean delivery under general anesthesia.[35] Proceedings were uneventful until immediately after extubation when she became hypotensive and dyspneic, progressing to unconsciousness. The entire body became flushed following reintubation. Transthoracic echocardiography revealed an empty left ventricle with no right ventricle dilation, pericardial effusion, or aortic dissection. Blood gas analysis suggested hemoconcentration, with hematocrit elevated from 34% to 49%. The intradermal skin test suggested that the rocuronium-sugammadex complex was the likely causative agent.

Right Heart Failure

The right heart is a crucial relay station, driving the returning systemic venous blood to flow through pulmonary vasculature and reach the left heart. Acute right heart failure can lead to preload crisis.[37] An example case described a 22-year-old woman who developed acute right heart failure and required reinstitution of cardiopulmonary bypass due to massive right heart and pulmonary thrombosis following the administration of prothrombin complex concentrates at the end of complex open-heart surgery.[37]

Table 2. The Reported Causes and Risk Factors of Perioperative Cardiac Arrest

Year (authors)	Population (data source)	Risk factors	Causes
1998 (Anthi et al)[5]	Cardiac surgery (a tertiary hospital in Grace)		• Arrhythmias preceding arrest (ventricular tachycardia/fibrillation, bradyarrhythmias, electromechanical dissociation) • Causes of cardiac arrest (myocardial infarction, tamponade, graft malfunction, unknown)
2006 (Braz et al)[1]	Any surgery (a tertiary hospital in Brazil)	Neonates, children under 1 y, elderly, male patients with ASA III or poorer physical status, emergency surgery, general anesthesia	Sepsis and multiple organ failure, trauma (motor vehicle, gunshot wound and stabling), exsanguinating hemorrhage at operation associated with primary disease, unable to wean from cardiopulmonary bypass, ruptured aneurysm: abdominal or thoracic, technical surgical complications, complications associated with cardiac surgery, complications associated to congenital heart defect, complication associated with radical cancer surgery, pulmonary embolus, perioperative myocardial infarction. The main causes of anesthesia-related cardiac arrest were respiratory events and medication-related events
2007 (Bhananker et al)[21]	Pediatric surgery (Pediatric Perioperative Cardiac Arrest Registry)		• Cardiovascular (hypovolemia associated with blood loss, electrolyte imbalance, nonhemorrhage hypovolemia, air embolism, other cardiovascular, presumed cardiovascular unclear mechanism) • Respiratory (airway obstruction—laryngospasm, airway obstruction—other, inadequate ventilation or oxygenation, inadvertent or premature extubation, difficult intubation, esophageal or endobronchial intubation, bronchospasm, pneumothorax, aspiration, other, presumed respiratory, unclear mechanism) • Medication (halothane-induced cardiovascular depression, sevoflurane-induced cardiovascular depression, other single medication, medication combination, allergic reaction, intravascular injection of local) • Equipment (central catheter, kinked or plugged endotracheal tube, peripheral intravenous catheter, breathing circuit) • Multiple events • Miscellaneous • Unknown
2010 (Ramamoorthy et al)[22]	Pediatric surgery (Pediatric Perioperative Cardiac Arrest Registry)		• Cardiovascular (myocardial ischemia, hyperkalemia, "Tet" spell, preexisting hypovolemia, sudden arrhythmia, hypovolemia-blood loss, other miscellaneous cardiovascular cause, presumed cardiovascular with unclear etiology) • Medication (inhaled anesthetic cardiovascular depression, halothane, sevoflurane, isoflurane, intravenous propofol or narcotics-related cardiovascular depression, wrong dose, medication combinations, other) • Respiratory (laryngospasm, inadequate oxygenation, difficult intubation, airway obstruction, other miscellaneous respiratory cause) • Equipment (central-line complications, breathing circuit obstruction, endotracheal tube obstruction) • Multiple events • Unknown cause
2013 (Matsusaki et al)[23]	Deceased donor liver transplantation (a tertiary hospital in the United States)	A higher MELD score and a higher serum sodium level identified as independent risk factors	Postreperfusion syndrome, pulmonary thromboembolism, hyperkalemia, uncontrolled bleeding, noncardiogenic pulmonary edema, primary nonfunctioning graft, increased intracranial pressure due to fulminant hepatic failure, or an unknown etiology

(*Continued*)

Table 2. Continued

Year (authors)	Population (data source)	Risk factors	Causes
2015 (Nunnally et al)[2]	Any surgery (National Anesthesia Clinical Outcomes Registry)	Age <1 y, age >66 y, man, ASA physical status class III/IV/V, general anesthesia, intracranial procedures, most common procedure codes—"anesthesia for intraperitoneal procedures in upper abdomen including laparoscopy; not otherwise specified" and "anesthesia for intraperitoneal procedures in lower abdomen including laparoscopy; not otherwise specified"	
2015 (Siracuse et al)[24a]	Vascular surgery (National Surgical Quality Improvement Program)	• Patient variables most predictive of postoperative cardiac arrest (dependent functional status, dialysis dependence, emergent case, and preoperative ventilator dependence) • Procedures associated with the highest risk (thoracic aortic surgery, open abdominal procedures, axillary-femoral bypass, and peripheral embolectomy) • At least 1 major complication preceded cardiac arrest (sepsis, renal failure, and myocardial infarction)	
2018 (Sobreira-Fernandes et al)[4b]	Noncardiac and nonobstetric surgery (a tertiary hospital in Portugal)		Perioperative cardiac arrests attributed to anesthesia: • High neuraxial block following positioning in the left lateral decubitus after spinal anesthesia with a combination of 10 mg bupivacaine and 10 μg fentanyl • Shock after anesthetic induction • Oversedation with a combination of 2 mg midazolam and 20 mg propofol before upper extremity locoregional anesthesia • Respiratory distress after premature endotracheal extubation • Bradycardia followed by ventricular fibrillation after administration of neostigmine • Respiratory distress following administration of fentanyl after endotracheal extubation
2020 (Fielding-Singh et al)[17]	Any surgery (the National Inpatient Sample of the Healthcare Cost and Utilization Project)	Black or missing race; cardiac, thoracic, or vascular surgery; congestive heart failure; pulmonary circulation disorders; peripheral vascular disease; end-stage renal disease; fluid and electrolyte disorders	
2021 (Smith et al)[8]	Adult liver transplantation (7 academic centers in the United States)	Extreme BMI (BMI < 20 or BMI ≥ 40), MELD score > 30, postreperfusion syndrome, living donor liver transplantation, reoperation	
2022 (Geube et al)[6c]	Cardiac surgery (a tertiary hospital in the United States)	Reduced left ventricular ejection fraction and moderate/severe pulmonary hypertension identified as independent risk factors for cardiac arrest	
2022 (Riley et al)[25]	Pediatric cardiac surgery (a scoping review)	Younger age, lower weight, extracardiac anomaly, increased surgical complexity, preoperative mechanical ventilation, preoperative nutritional status, longer cardiopulmonary bypass duration, ICU attending in-house 24/7, limited bedside nursing experience, reduced ICU staffing or hospital resources on weekends	

Abbreviations: ASA, American Society of Anesthesiologists; BMI, body mass index; ICU, intensive care unit; MELD, model for end-stage liver disease.
[a]This publication reported risk factors of cardiac arrest after vascular surgery procedures only.
[b]This publication reported anesthesia-related causes of perioperative cardiac arrest only. The causes partially attributed to anesthesia are not included in this table.
[c]This publication reported risk factors for preincision cardiac arrest in cardiac surgical patients only.

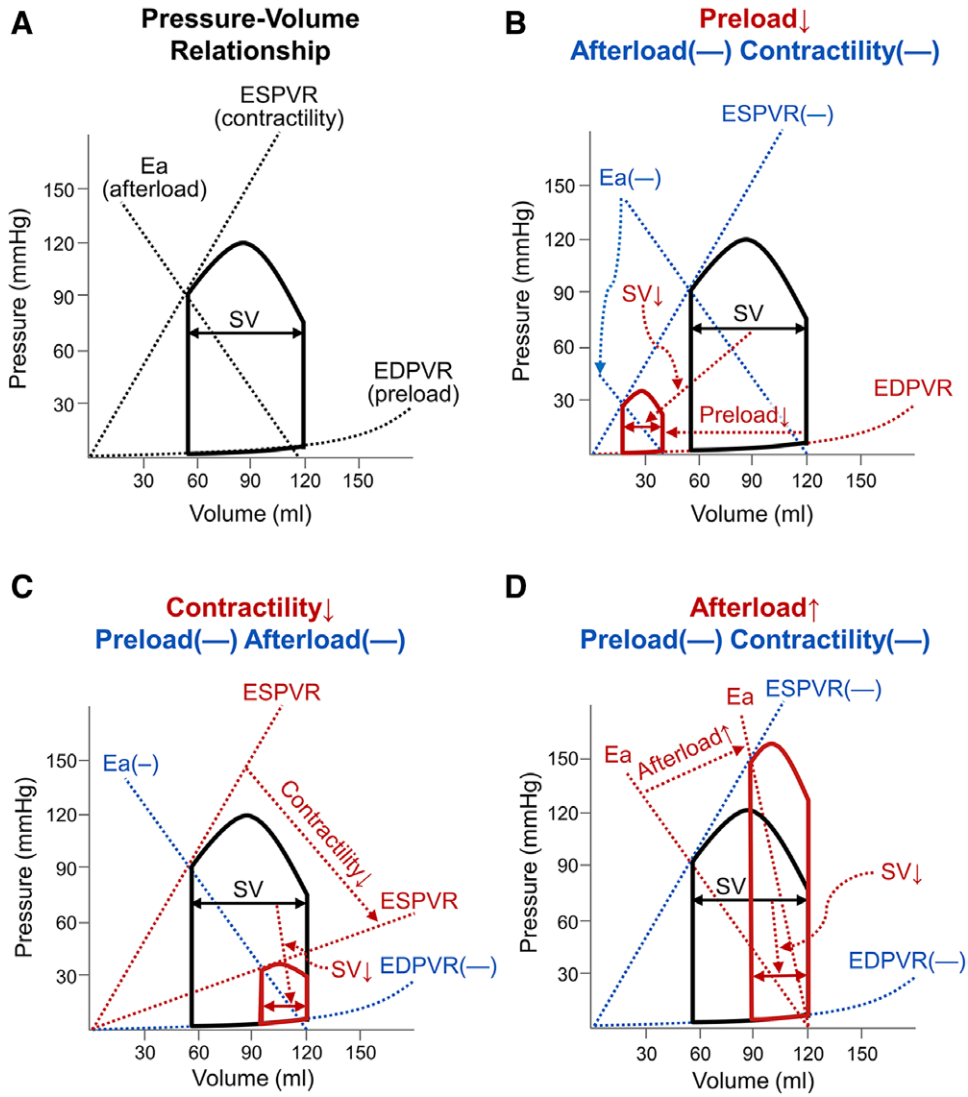


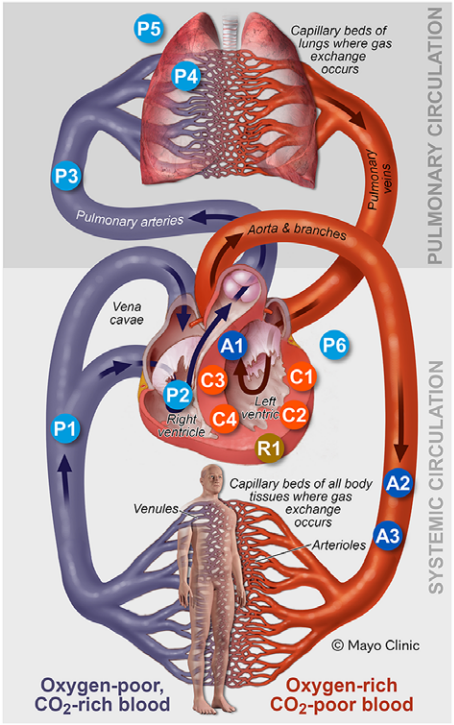
Figure 1. Impacts of significant preload decrease, contractility impairment, and afterload increase on stroke volume. The ordinate of the pressure-volume relationship plot represents the ventricular pressure, while the abscissa represents the corresponding ventricular volume. The pressure-volume relationship plot uses EDPVR to appreciate preload, ESPVR to appreciate contractility, and Ea to appreciate afterload (A). The dynamic changes in left ventricular volume and pressure during diastole can be appreciated along the EDPVR curve, with the preload defined by the point immediately before isovolumetric contraction.[26,27] Contractility can be appreciated by the slope of the curve for the ESPVR, which describes the end-systolic pressure the ventricle can develop at a given preload and afterload.[28] Afterload can be appreciated by the effective Ea—that is, the ratio of the end-systolic pressure divided by stroke volume—regarded as an afterload surrogate.[29] Based on the pressure-volume relationship analysis, it can be shown that a significant decrease in preload (B, assuming contractility and afterload remaining stable in this illustration), impairment of contractility (C, assuming preload and afterload remaining stable in this illustration), or increase of afterload (D, assuming preload and contractility remaining stable in this illustration) can all lead to significant stroke volume reduction. Ea indicates arterial elastance; EDPVR, end-diastolic pressure-volume relationship; ESPVR, end-systolic pressure-volume relationship; SV, stroke volume; ↓, decrease; ↑, increase; (—), stable.

Pulmonary Embolism

Pulmonary embolism can be caused by different emboli, as discussed below.

Pulmonary Embolism Due to Air. Multiple POCA cases were attributed to pulmonary air embolism.[38–41] One case described a 40-year-old male patient undergoing lumbar laminectomy in a prone position.[39] The procedure was unremarkable for 5.5 hours until the patient developed bradycardia and hypotension, progressing to asystole. The patient was turned supine for resuscitation to no avail. Postmortem examination 24 hours later revealed 40 mL of air in the right ventricle and air bubbles in the coronary arteries. The air may trespass from the right heart into the left heart via pulmonary vasculature or a patent foramen ovale.

Pulmonary Embolism Due to Oxygen. Oxygen can cause pulmonary embolization following surgical wound irrigation using hydrogen peroxide.[42,43] One case described a 39-year-old man undergoing



P =Preload crisis C =Contractility crisis A =Afterload crisis R =Rate & rhythm crisis

PRELOAD CRISIS

1. Hypovolemia (trauma, massive bleeding, anaphylaxis, major vein occlusion)
2. Right heart failure (myocardial ischemia, pulmonary hypertension, pulmonary embolism)
3. Pulmonary embolism (blood clot, gas [air, O_2, CO_2], surgical cement, fatty tissue, amniotic fluid)
4. Exaggerated PEEP
5. Tension pneumothorax
6. Cardiac tamponade

CONTRACTILITY CRISIS

1. Myocardial ischemia (low perfusion pressure, thrombosis, embolism [air, O_2, CO_2], spasm)
2. Myocardial hypoxia (desaturation, acute anemia)
3. Hydrogen ions (acidosis)
4. Takotsubo cardiomyopathy

AFTERLOAD CRISIS

1. LVOT obstruction (systolic anterior motion of the mitral leaflet)
2. Severe vasodilation (anaphylaxis, vasodilator overdose)
3. Severe vasoconstriction (vasoconstrictor overdose, pheochromocytoma)

RATE & RHYTHM CRISIS

1. Severe bradycardia
   Atrioventricular block
   Supraventricular tachycardia
   Atrial fibrillation
   Ventricular tachycardia & fibrillation

Potential causes

- Myocardial ischemia & hypoxia
- Hypokalemia & hyperkalemia
- Toxins & drugs
- Local anesthetic systemic toxicity
- Procedure-related stimulation
- Vasovagal response
- Hypoglycemia
- Hypothermia

Figure 2. The PCARR classification of cardiac arrest. Cardiac arrest can result from preload, contractility, afterload, or rate and rhythm crises. The causes of different crises are detailed. LVOT indicates left ventricle outflow tract; PCARR, preload-contractility-afterload-rate and rhythm; PEEP, positive end-expiratory pressure.

an elective midline suboccipital craniectomy for tumor resection in the sitting position.[43] On surgery completion, the wound was irrigated using 10 mL of 3% hydrogen peroxide. Immediately after, the patient developed a sudden decrease in $Etco_2$, blood pressure, and heart rate. Transesophageal echocardiography (TEE) noted gaseous bubbles continuously entering the right atrium. Nearly 60 mL of presumed oxygen bubbles were aspirated via the central venous catheter placed in the right subclavian vein.

Pulmonary Embolism Due to Carbon Dioxide. The carbon dioxide used during pneumoperitoneum can cause a pulmonary embolism. One case described a 40-year-old woman undergoing a laparoscopic low anterior resection and hepatic tumor resection to treat colorectal cancer with liver metastasis.[44] Pneumoperitoneum was maintained via carbon dioxide insufflation. The procedure was complicated with a right hepatic vein rupture when the patient was in the reverse Trendelenburg position. The patient progressed to cardiac arrest during emergent laparotomy for bleeding control in the neutral position. TEE supported the speculation of pulmonary carbon dioxide embolism by discovering gaseous bubbles in the right pulmonary artery.

Pulmonary Embolism Due to Thromboemboli. Pulmonary thromboembolism can cause POCA.[45] One example case described a 62-year-old woman undergoing posterior lumbar spinal fusion on a Jackson table.[46] The patient developed cardiac arrest following the position change from prone to supine after an unremarkable surgery. TEE noted a hypokinetic dilated right ventricle, a D-shaped left ventricle, and tricuspid regurgitation with a pressure gradient of 49 mm Hg. The multidetector computed tomography study noted the occlusion of both pulmonary arteries with multiple pulmonary thromboemboli.

Pulmonary Embolism Due to Surgical Cement. Surgical cement can cause a devastating pulmonary embolism. One case described a 75-year-old woman undergoing percutaneous vertebroplasty in a prone position.[47] At the time of skin closure, the patient developed sudden onset of bradycardia, hypotension, desaturation, and $Etco_2$ drop. In response, the patient was turned to the supine position for resuscitation, but to no avail. TEE showed that the right atrium and ventricle were almost filled with multiple small deposits of diffusely echogenic material, consistent with the bone cement implanted during surgery.

Pulmonary Embolism Due to Amniotic Fluid. Amniotic fluid embolism can cause POCA.[48] One case described a 23-year-old woman who became unresponsive during labor following 42 weeks of gestation.[49] She was rushed to a hospital emergency room. Shortly after arrival, cardiac arrest occurred; unfortunately,

both patient and fetus died. A peer review committee member speculated the diagnosis of amniotic fluid embolism—confirmed by an in-depth autopsy at the obstetrician's request. Crucially, the term "amniotic fluid embolism" might be a misnomer. A common pathophysiologic mechanism, that is, endogenous mediator release, might be shared among amniotic fluid embolism, anaphylaxis, and septic shock due to the striking similarities in clinical and hemodynamic findings.[50,51]

Exaggerated PEEP

Positive end-expiratory pressure (PEEP) typically does not cause POCA; however, excessive and prolonged PEEP can hinder blood transition from the right to the left heart, and in extreme cases, it can even lead to cardiac arrest in critically ill patients.[52,53] During a study examining lung recruitment and PEEP titration in patients with moderate to severe acute respiratory distress syndrome, the trial committees jointly decided to modify the recruitment maneuver and PEEP titration strategy (from maximal PEEP of 45 mm Hg down to 35 mm Hg) midtrial.[54] This decision was prompted by 3 cases of resuscitated cardiac arrest potentially associated with the initially planned excessive PEEP.[54]

It is crucial to distinguish between intrinsic PEEP, related to air trapping and dynamic hyperinflation, and PEEP applied through the ventilator.[53] One case involved a 50-year-old man who underwent a left bullectomy to address a pneumothorax resulting from a ruptured bulla.[55] The patient experienced a pulseless electrical activity (PEA) arrest while receiving 1-lung ventilation in the right lateral decubitus position. Tension pneumothorax and pulmonary embolism were ruled out, and the incident was attributed to "dynamic hyperinflation." This case serves as an example of POCA associated with intrinsic PEEP.

Tension Pneumothorax

Tension pneumothorax reduces preload among multiple physiological disturbances it incurs; in severe cases, it leads to cardiac arrest.[56,57] One case described a 76-year-old woman undergoing a right thoracotomy with a right upper lobectomy.[58] Abrupt intraoperative cardiac arrest occurred, characterized by an increased peak airway pressure, profound hypoxemia, $Etco_2$ drop, bradycardia, and a flat arterial blood pressure tracing. This crisis was attributed to a tension pneumothorax on the dependent side. A left ventricular preload crisis was suggested by elevated pulmonary artery pressure and massive jugular venous distension (ie, stalled pulmonary blood flow leading to backed-up pressure).

Cardiac Tamponade

Acute and severe cardiac tamponade compresses cardiac chambers and can cause preload crisis.[59,60] One case described a 42-year-old woman undergoing a thoracentesis for dyspnea treatment.[61] A sudden cardiac arrest developed during the procedure, likely secondary to cardiac tamponade, as the echocardiography study noted pericardial effusion and right ventricular collapsing during diastole.

POCA DUE TO CONTRACTILITY CRISIS

The following pathophysiology can lead to contractility crisis and POCA.

Myocardial Ischemia

Severe myocardial ischemia can lead to POCA. One single-center study based on the departmental TEE database identified 22 noncardiac surgical patients who experienced unexpected intraoperative cardiac arrest.[62] TEE established a preliminary cause in 19 of 22 patients, with 6 patients having acute myocardial ischemia, 9 patients having thromboembolic events, 2 patients having hypovolemia, and 2 patients having pericardial tamponade.

Hypoxemia

Severe hypoxemic events, defined as a pulse oxygen saturation (Spo_2) reading ≤85% for at least 2 consecutive minutes, may occur in up to 3.5% of patients during surgery.[63] Severe and prolonged hypoxemia can cause POCA.[64–66] A post hoc analysis of a trial involving 20,802 surgical patients suggested that the use of pulse oximetry was associated with several key benefits, including a reduction in myocardial ischemia (from 0.2% to 0.1%) and cardiac arrest (from 0.1% to 0.04%).[63,67] Hypoxemia can be secondary to many causes including, but not limited to, an inappropriately low fraction of inspired oxygen, loss of airway, bronchospasm, laryngospasm, atelectasis, pulmonary edema, pneumonia, right-to-left shunt, ventilation-to-perfusion mismatch, and tension pneumothorax.

Acidosis

Acidosis is a component of the Hs and Ts mnemonic, as evidenced by the association between metformin-induced lactic acidosis and cardiovascular collapse,[68] and the link between ethylene glycol intoxication and refractory cardiopulmonary collapse.[69] The successful utilization of high-efficiency hemodialysis during cardiopulmonary resuscitation (CPR) in patients with severe metabolic acidosis from ethylene glycol and metformin intoxications corroborates the cause-and-effect relationship between acidosis and cardiac arrest.[70]

Distinguishing between acidosis as the cause, the effect, or both in cardiac arrest scenarios can be

challenging in real-world practice. For instance, a case study involved a 66-year-old man undergoing orthotopic liver transplantation for alcohol-related end-stage liver disease.[71] The patient experienced cardiac arrest immediately after reperfusion but was resuscitated after receiving 100 mEq sodium bicarbonate. While acidosis may have played a contributory role, the authors considered it unlikely to be the sole cause.

Nonetheless, acidosis and outcomes are associated, as demonstrated by the relationship between early intra-arrest blood pH and in-hospital cardiac arrest outcomes.[72] This cohort study suggested a blood pH threshold of 7.2 to define severe acidemia during arrest and assist in profiling patients with in-hospital cardiac arrest.[72] Furthermore, it is essential to differentiate between metabolic and respiratory acidosis, as they often coexist in cases of cardiac arrest,[73] but have distinct causes and necessitate different treatment approaches.

Takotsubo Cardiomyopathy

Although Takotsubo syndrome has long been regarded as a benign disorder, cardiac arrest is relatively common in this condition and is associated with worse outcomes.[74] This relationship was highlighted in a case report of a 64-year-old woman who suffered cardiac arrest 6 hours after ureteral stenting, despite having no history of heart disease.[75] Takotsubo cardiomyopathy was diagnosed through a left ventriculogram, revealing the characteristic apical ballooning. Notably, she had a urinary tract infection before the procedure; however, sepsis was argued as an unlikely cause of her cardiac arrest.

POCA DUE TO AFTERLOAD CRISIS

The following events can lead to an afterload crisis and POCA.

Left Ventricular Outflow Tract Obstruction

Stenotic lesions can lead to sudden cardiac arrest.[76,77] The mitral valve's systolic anterior motion is a well-known POCA etiology.[78–80] One case described a 69-year-old woman undergoing an orthopedic procedure to treat her bilateral femoral neck fracture after falling out of her wheelchair.[78] The patient developed severe hypotension 5 minutes after anesthesia induction and had repeated severe hypotensive episodes during surgery. Emergent TEE noted the systolic anterior motion of the mitral valve leading to left ventricular outflow tract obstruction. Although cardiac arrest did not occur, it would be likely without appropriate intervention.

Catecholamine Surge

Patients with pheochromocytoma can experience catastrophic catecholamine surge. Peripheral vasoconstriction can lead to severe hypertension that, in turn, can result in an afterload crisis and cardiac arrest. A systematic review summarized 35 reports involving 62 patients receiving extracorporeal life support to treat intractable pheochromocytoma crises.[81] One case described a 32-year-old man who presented for a minor otolaryngology procedure under general anesthesia.[82] A few episodes of intraoperative tachycardia and hypertension occurred and were successfully treated. However, the patient developed cardiac arrest with PEA at the time of emergence. An urgent echocardiogram noted profound left ventricular failure. This patient's PEA was attributed to a left adrenal gland pheochromocytoma.

Vasopressor Overdose

Afterload increase due to inadvertent vasopressor overdose, similar to the catecholamine surge in pheochromocytoma, can also be catastrophic.[83,84] Using inotropes in hypovolemic patients or patients with hypertrophic cardiomyopathy may obstruct the left ventricle outflow tract and cause a hemodynamic crisis.[85] One case described a 56-year-old man who required endotracheal intubation after a suicide attempt.[83] During transport to the hospital, the patient experienced hypotension refractory to a 500-mL lactated Ringer's fluid bolus. Norepinephrine 5 mg was injected into the remaining 500 mL of crystalloid in the bag and titrated to effect via a roller flow control clamp. The patient developed spiking hypertension in the hospital, and, on record, the highest reading was 315/190 mm Hg. At that point, it was recognized that the prehospital norepinephrine infusion bag was running fully open. Approximately 30 seconds after infusion cessation, ventricular fibrillation developed, with a flat arterial blood pressure tracing. Classifying vasopressor overdose as an afterload crisis can be arbitrary, as certain vasopressors can also cause heart rate and rhythm crises. Whether there is a relationship between severe afterload increase and malignant arrhythmia remains to be clarified.

Severe Afterload Decline

Severe reduction in afterload can be attributed to various factors, such as anaphylaxis, accidental vasodilator overdose, and neuraxial anesthesia. Neuraxial anesthesia has been documented as a potential cause of cardiac arrest,[86,87] with decreased afterload being a contributing factor among various possible mechanisms. The profound hypotension resulting from severe afterload reduction can lead to myocardial ischemia and subsequently reduce myocardial contractility. Furthermore, in cases of anaphylaxis, the significant decrease in afterload can be further complicated by a substantial preload reduction due to venodilation or extravasation.[36]

POCA DUE TO RATE AND RHYTHM CRISIS

Arrhythmias, including severe bradycardia, atrioventricular block, supraventricular tachycardia, atrial fibrillation, and ventricular tachycardia and fibrillation, can cause POCA. The common causes of perioperative rate and rhythm crisis are as follows.

The Vasovagal Response

Various causes can elicit severe vasovagal responses in the perioperative setting.[88–90] For example, 1 case described a 39-year-old woman undergoing an elective cesarean delivery under spinal anesthesia.[91] The procedure was unremarkable until the point of placenta separation, which required repeated forceful tractions. Immediately after the placental expulsion, the patient suddenly collapsed, characterized by severe bradycardia, asystole, respiratory arrest, and loss of consciousness. The arrest was attributed to a vasovagal response triggered by uterus stretching on top of high spinal anesthesia (sensory level to alcohol swab at T3) in a pregnant woman with a history of vasovagal syncope.

Drugs

Dexmedetomidine can cause severe bradycardia and even asystole.[92] Vasopressor overdose can cause severe arrhythmia.[93] Local anesthetic systemic toxicity (LAST) can cause devastating arrhythmia and POCA.[94–106] Bupivacaine can induce severe myocardial depression[107] and cardiac toxicity, mimicking an acute non-ST segment elevation myocardial infarction.[108] One case described a 76-year-old woman undergoing outpatient TEE and atrial fibrillation cardioversion.[94] The patient was given approximately 3 doses of 25 to 30 mL 4% topical lidocaine and 2 additional doses of 20 to 30 mL 2% topical lidocaine for an approximate total of more than 3000 mg (36 mg/kg) of lidocaine. Approximately 45 minutes after lidocaine administration, she developed slurred speech, confusion, bradycardia with a heart rate in the 40 seconds, and hypoxemia with a Spo_2 of 80%, which progressed to unresponsiveness and cardiac arrest. This crisis was attributed to lidocaine toxicity. It needs to be noted that LAST can also cause myocardial contractile dysfunction.[109]

Life-Threatening Electrolyte Abnormalities

Hypokalemia can cause POCA.[110–112] One case described a 78-year-old man undergoing laparoscopic cholecystectomy.[110] The patient remained intubated and was transported to the intensive care unit after surgery for observation. One hour later, he developed tachycardia, progressing into pulseless ventricular tachycardia. Laboratory results indicated severe hypokalemia (2.4 mmol/L), whereas his preoperative serum potassium was normal (3.8 mmol/L). During intravenous potassium chloride administration, multiple episodes of ventricular fibrillation occurred. He received 210 mmol of potassium chloride within 24 hours before extubation.

Hyperkalemia can cause POCA.[113–118] One case described a 12-month-old infant undergoing elective surgical correction of severe scaphocephaly secondary to sagittal craniosynostosis.[113] The patient received a packed red blood cell transfusion due to heavy bleeding from surgery outset. After about 2 hours and 45 minutes, the patient showed electrocardiography (ECG) changes characterized by widened QRS and tall peaked T-waves, which became isoelectric in seconds. At the same time, no pulses could be detected. The serum potassium concentration was 10.1 mmol/L at the time of arrest.

Other electrolyte imbalances, including those involving sodium, magnesium, and Ca^{2+}, often play a significant role in developing cardiac arrhythmias, making them a common cause or complicating factor in resuscitation and postresuscitation care.[119] However, it is worth noting that hypercalcemia, characterized by Ca^{2+} levels >4 mmol/L, does not appear to be associated with immediately life-threatening cardiac arrhythmias or neurological complications.[120] Interestingly, a study showed that increased levels of ionized Ca^{2+} measured during CPR are linked to an enhanced likelihood of achieving the return of spontaneous circulation.[121]

Hypoglycemia

Rat studies have suggested an association between severe hypoglycemia and lethal cardiac arrhythmias.[122] Whether a causal relationship exists between hypoglycemia and cardiac arrest in humans, however, is less clear.[123] Hypoglycemia is not included in the most up-to-date guidelines as a reversible cause of cardiac arrest.[11,123,124] Some reports suggest a causal relationship between hypoglycemia and cardiac arrest.[123,125,126] One case described a 51-year-old man with Down syndrome and type I diabetes mellitus undergoing urethrotomy under spinal anesthesia.[125] The procedure was unremarkable. However, on arrival in the postanesthesia care unit, the patient suddenly lost consciousness and developed bradycardia, followed by asystole. His electrolyte and acid-base profiles were normal except for severe hypoglycemia (23 mg/dL), which was speculated as the cause of the arrest.

Hypothermia

Severe hypothermia increases the risk of fatal arrhythmia.[127] Hypothermia can cause POCA.[128,129] One case described a 61-year-old man undergoing an emergency decompressive craniectomy after head trauma.[129] Before surgery, the patient had undergone mild therapeutic hypothermia for neuroprotection. During surgery, the patient developed ventricular fibrillation, attributed to an anesthesia-related further

Table 3. Comparison Between the PCARR Construct and the Hs and Ts Mnemonica

The PCARR construct	The Hs and Ts mnemonic	Clues and comments
Preload crisis		
Hypovolemia[b]		
Trauma	Hypovolemia (not specified)	History, clinical presentation
Massive bleeding	Hypovolemia (not specified)	History, clinical presentation (overt versus covert)
Anaphylaxis	Hypovolemia (not specified)	Vasodilation, extravasation, skin rash, wheezing
Major vein occlusion	Hypovolemia (not specified)	Venous congestion
Right heart failure[c]		
Myocardial ischemia	N/A	Typical electrocardiography and/or echocardiography findings
Pulmonary hypertension	N/A	History and pulmonary blood pressure monitoring
Pulmonary embolism	Pulmonary thrombosis (not specified)	Right heart strain, echocardiography findings
Pulmonary embolism		
Blood clot	Pulmonary thrombosis (not specified)	Right heart strain, echocardiography findings
Gas (air, oxygen, carbon dioxide)	Pulmonary thrombosis (not specified)	Right heart strain, echocardiography findings, aspiration
Surgical cement	Pulmonary thrombosis (not specified)	Right heart strain, echocardiography findings
Amniotic fluid	Pulmonary thrombosis (not specified)	Obstetric setting
High intrathoracic and transpulmonary pressure		
Tension pneumothorax	Tension pneumothorax	Lack of breath sound, ultrasound and chest X-ray findings
Exaggerated positive end-expiratory pressure[c]	N/A	Airway pressure monitoring, excessively expanded breathing bag
High pericardial pressure		
Cardiac tamponade	Tamponade (cardiac)	Echocardiography findings
Contractility crisis		
Myocardial ischemia[d]		
Low perfusion pressure (hypoperfusion)[c]	N/A	Blood pressure monitoring (especially diastolic blood pressure)
Thrombosis	Thrombosis (coronary)	Electrocardiography and/or echocardiography findings
Embolism[c]	N/A	Electrocardiography and/or echocardiography findings
Spasm[c]	N/A	Typical electrocardiography finding, coronary artery bypass surgery
Myocardial hypoxia		
Hypoxemia (desaturation)[e]	Hypoxia[e]	Spo_2 monitoring, blood gas analysis, respiratory arrest
Acute anemia[c]	N/A	Acute bleeding, labs
Acid-base abnormality		
Acidosis	Hydrogen ions (acidosis)	Blood gas analysis
Acute cardiomyopathy		
Takotsubo cardiomyopathy	N/A	Echocardiography findings
Afterload crisis[f]		
Severely low afterload (severe vasodilation)		
Anaphylaxis[c]	N/A	Can also cause hypovolemia
Vasodilator overdose[c]	N/A	Inappropriate drug administration
Severely high afterload (severe vasoconstriction)		
Vasopressor overdose[c]	N/A	Inappropriate drug administration
Pheochromocytoma[c]	N/A	Severe hypertension not caused by inappropriate drug administration
Left ventricle outflow tract obstruction		
Systolic anterior motion of the mitral leaflet[c]	N/A	Typical echocardiography findings
Rate and rhythm crisis		
Severe bradycardia, atrioventricular block, supraventricular tachycardia, atrial fibrillation, ventricular tachycardia, and fibrillation		
Myocardial ischemia and hypoxia	N/A	Can also cause contractility crisis and right heart failure
Hypokalemia and hyperkalemia	Hypokalemia and hyperkalemia	Electrocardiography changes, blood gas analysis
Drugs (eg, epinephrine overdose)	Toxins	Inappropriate drug administration
LAST[g]	Toxins[h]	Local anesthetic injection may not have neurological signs and symptoms in anesthetized or sedated patients

(*Continued*)

Table 3. Continued

The PCARR construct	The Hs and Ts mnemonic	Clues and comments
Procedure-related stimulation[c]	N/A	Procedural details
Vasovagal response[c]	N/A	Abrupt onset of bradycardia most commonly related to surgical maneuvers
Hypoglycemia	N/A	Blood gas analysis, history of diabetes
Hypothermia	Hypothermia	Low central temperature

Abbreviations: Hs, hypoxia, hypovolemia, hydrogen ions (acidosis), hypo-/hyperkalemia, and hypothermia; LAST, local anesthetic systemic toxicity; N/A, not available; PCARR, preload-contractility-afterload-rate and rhythm; Spo_2, pulse oxygen saturation; Ts, toxins, tamponade (cardiac), tension pneumothorax, thrombosis (pulmonary), and thrombosis (coronary).

[a]The PCARR construct is systemic, classified, and logical.

[b]Hypovolemia can be secondary to trauma, massive bleeding, vasodilation, anaphylaxis, dehydration, or major vein occlusion; however, this is not specified in the Hs and Ts construct.

[c]Many etiologies included in the PCARR construct are not included in the Hs and Ts construct, such as right heart failure, high positive end-expiratory pressure, vasopressor overdose, and the vasovagal response.

[d]The cause of myocardial ischemia can be related to low perfusion pressure-related hypoperfusion, thrombosis, or embolism; however, this is not specified in the Hs and Ts construct.

[e]Hypoxemia refers to the low oxygen level in the blood, while hypoxia refers to the low inspired oxygen fraction or tissue oxygen level.

[f]A significant decrease in afterload can cause critically low blood pressure, leading to myocardial hypoperfusion. In contrast, a substantial increase in afterload can impede stroke volume and lead to reduced cardiac output (afterload crisis is not included in the Hs and Ts construct).

[g]The PCARR construct specifies local anesthetic systemic toxicity as one of the etiologies leading to rate and rhythm crisis, but, in contrast, the Hs and Ts construct applies the term "toxins" without specification.

[h]The toxins and tablets that cause perioperative cardiac arrest are primarily related to local anesthetics overdose or vasoactive drug overdose. Multiple drugs, contrasts, and materials can cause anaphylaxis; however, we classify anaphylaxis as preload and afterload crises.

decrease in temperature because the esophageal temperature at the time of arrest was 32.0 °C, as opposed to 33.0 °C before anesthesia induction.

Ventricular Tachycardia and Fibrillation

In the perioperative setting, pulseless ventricular tachycardia and ventricular fibrillation result from a variety of causes, including, but not limited to, direct surgical stimulation, electrocautery,[130] myocardial ischemia and infarction,[131] coronary artery occlusion,[132] graft spasm,[133] hypothermia,[134] electrolyte abnormality,[110] acidosis,[135] hypoxemia,[136] hypovolemia,[137] drugs,[83,138] and coexisting diseases.[139] Ondansetron, for example, can cause dose-dependent QT prolongation—clinically relevant when other proarrhythmic factors are present.[138]

THE HS AND TS MNEMONIC IS CONSIDERED IN THE PCARR CLASSIFICATION SYSTEM

A side-by-side comparison between the PCARR construct and the Hs and Ts mnemonic is presented in Table 3, which shows that the PCARR construct covers the components of the Hs and Ts mnemonic, although multiple components included in the PCARR construct are not included in the Hs and Ts construct (also refer to Figure 3). Compared with the Hs and Ts mnemonic, the PCARR construct is systemic, classified, and physiologically logical. However, this does not mean the Hs and Ts mnemonic is denied. Instead, the rationally combined use of both systems may further improve the investigation of POCA causes.

Clues From Clinical Monitoring

Monitoring is a significant aspect of the perioperative care. Patients' Spo_2, blood pressure, $Etco_2$, and ECG are routinely monitored during surgery. Following surgery, patients' Spo_2, blood pressure, and ECG are typically monitored in the postanesthesia recovery unit. Patients admitted to the intensive care unit after surgery are also extensively monitored.

Monitoring can provide clues about the causes of POCA (Figure 3). ECG is indispensable in diagnosing fatal arrhythmias and provides valuable information about myocardial ischemia. If there is no pulse despite normal ECG tracing, it is classified as PEA. If isolated Spo_2 changes occur before cardiac arrest, hypoxia could have caused POCA, and the causes leading to hypoxia or hypoxemia should be sought. $Etco_2$ changes do not typically cause cardiac arrest but can indicate CO changes, thus providing an early warning sign of looming arrest.[140] Severe hypotension can cause myocardial ischemia, especially in patients with coronary artery disease, while severe hypertension can cause CO reduction due to excessively high afterload (Figure 1D). If isolated changes in blood pressure precede cardiac arrest, the investigation should be directed toward understanding the causes of blood pressure changes, such as vasodilation, vasoconstriction, and CO decline. Hyperthermia and hypothermia can be determined from temperature monitoring. Excessive PEEP can be diagnosed based on respiratory pressure monitoring. An inappropriately low fraction of inspired oxygen can be diagnosed based on the gas analyzer display.

While monitoring can provide valuable insights into the underlying cause of POCA, it is essential to acknowledge that abnormal monitoring typically occurs following cardiac arrest. Therefore, it becomes crucial to promptly determine whether the monitoring offers any clues regarding the primary cause of

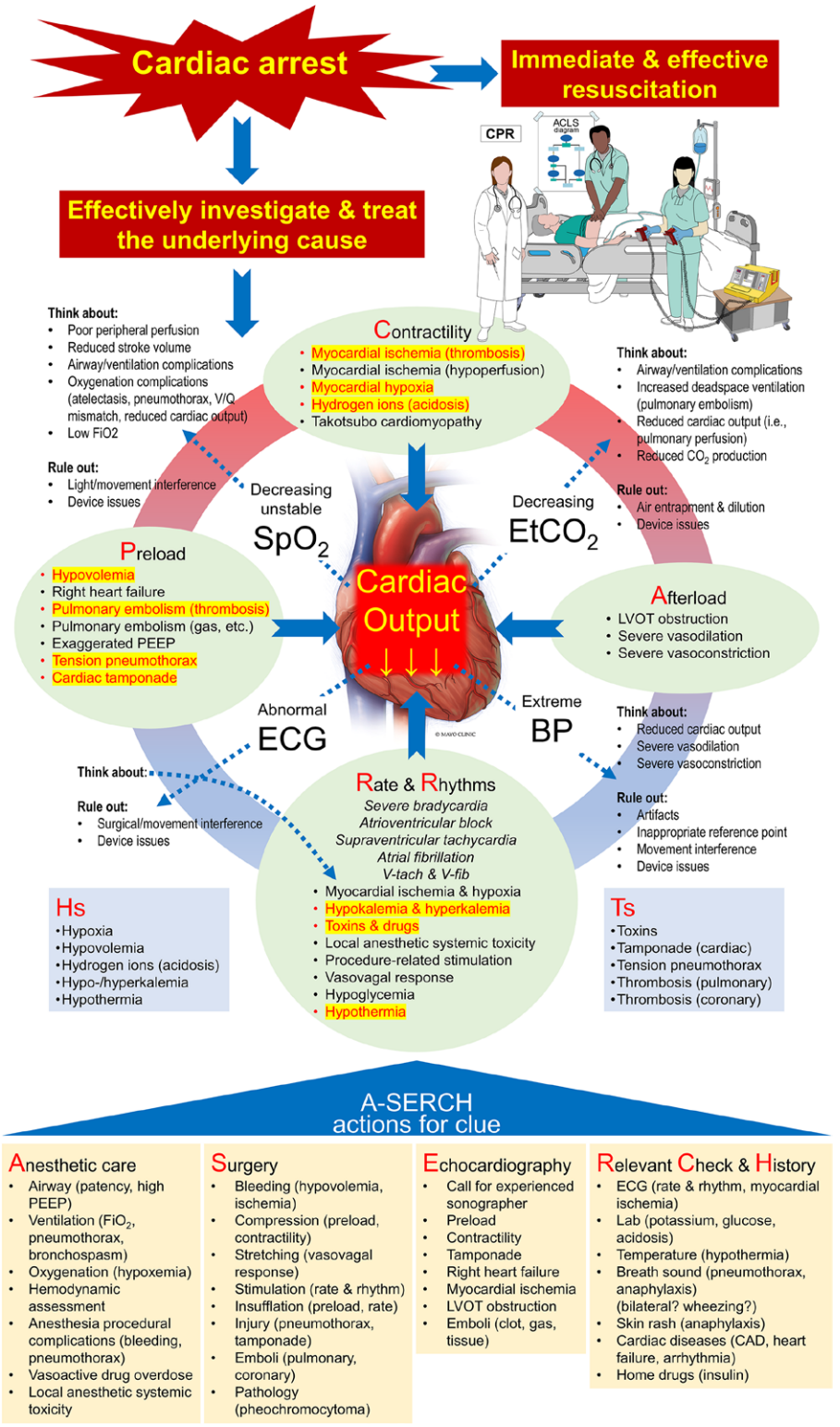


Figure 3. Integrated approaches investigating the causes leading to POCA. Once a cardiac arrest is confirmed, immediate and effective treatment is needed and follows 2 lines of simultaneous actions. One line of action is cardiopulmonary resuscitation. The other critical line of action is to investigate and treat the underlying cause. This investigation is guided by the Hs and Ts mnemonic, the PCARR classification construct, clues from monitored variables, and the A-SERCH list of actions. The components in the Hs and Ts mnemonic are highlighted in red fonts with a yellow background in the PCARR construct to emphasize their relationship. These approaches should be rationally integrated when investigating the underlying POCA causes. We emphasize that investigating the underlying causes should not interrupt cardiopulmonary resuscitation. Refer to the text for more details. A-SERCH indicates anesthetic care, surgery, echocardiography, and relevant check and history; ACLS, advanced cardiac life support; BP, blood pressure; CAD, coronary artery disease; CPR, cardiopulmonary resuscitation; ECG, electrocardiogram; FiO2, inspired oxygen fraction; Hs, hypoxia, hypovolemia, hydrogen ions (acidosis), hypo-/hyperkalemia, and hypothermia; LVOT, left ventricle outflow tract; PCARR, preload-contractility-afterload-rate and rhythm; PEEP, positive end-expiratory pressure; POCA, perioperative cardiac arrest; Spo_2, pulse oxygen saturation; Ts, toxins, tamponade (cardiac), tension pneumothorax, thrombosis (pulmonary), and thrombosis (coronary); V/Q, ventilation/perfusion; V-fib, ventricular fibrillation; V-tach, ventricular tachycardia.

the cardiac arrest (such as malignant arrhythmia, severe myocardial ischemia, hypoxemia, hypotension, hypertension, or hypothermia) or if the abnormal monitoring itself is a consequence of the cardiac arrest (such as a significant drop in $Etco_2$, desaturation, or hypotension).

Integrated Approaches Investigating POCA Causes

Effective treatment of POCA depends on 2 simultaneous actions, starting with immediate and effective CPR.[19] The second course of action is efficient and effective investigation and correction of the

underlying cause. Without correcting the underlying cause, the cardiac arrest is likely to persist or recur. However, investigating the cause is challenging due to the multitude of potential POCA causes and the stressful resuscitation environment known as the "fog of war."[141] Implementing efficient, effective, and intentional structures is essential to proactively identify underlying and propagating causes.[142]

In addition to the Hs and Ts mnemonic, the PCARR construct, and the clues from monitoring discussed above, we propose a list of actions entitled A-SERCH, representing Anesthetic care (A), Surgery (S), Echocardiography (E), and Relevant (R) Check (C) and History (H) to facilitate the investigation of POCA causes (Figure 3). A-SERCH does not stand for an absolute order of actions but allows for highlighting the priority of different actions when investigating POCA's underlying cause. Anesthetic care (A) should focus on airway, ventilation, oxygenation, continuous hemodynamic assessment, anesthetic care-related procedural complications (such as central-line placement), vasoactive drug use, and the potential for LAST. Surgery (S) should focus on bleeding, compression, stretching, stimulation, insufflation, injury, and emboli, in addition to the surgery's nature and details. Portable transthoracic and transesophageal echocardiography (E) provides valuable information related to preload and contractility—offering clues of pericardial tamponade, right heart failure, myocardial ischemia, left ventricular outflow tract obstruction, and pulmonary embolism but with no reliable information on afterload and rate and rhythm.[62,143–146] It must be emphasized that echocardiography should not interfere with standard cardiac arrest resuscitation. Also, having an experienced or trained sonographer for interpretation is warranted.[144,147] The Relevant (R) Check (C) and History (H) should focus on ECG, serum labs for electrolyte abnormalities and acidosis, temperature for hypothermia, bilateral breath sounds for pneumothorax, wheezing and skin rash for anaphylaxis, underlying cardiac diseases (especially coronary artery disease, heart failure, and arrhythmia), and home medications (especially insulin).

When investigating the underlying cause of POCA, an integrative approach that includes the Hs and Ts mnemonic, the PCARR construct, the clues from monitoring, and the A-SERCH list of actions should be used. It is essential to maintain an open mind when the cause of POCA is not immediately apparent. This combination of cognitive tools can reduce the likelihood of missing or ignoring important clues and provide a systemic approach highly relevant to POCA. However, validation of this proposal is needed in real-world practice.

SUMMARY

Cardiac arrest is a severe perioperative complication that requires constant improvement in management. While CPR has saved many lives, prompt recognition and resolution of the underlying cause are crucial in reversing the effects of cardiac arrest. The existing cognitive tool based on the Hs and Ts construct highlights some possible POCA causes but is not comprehensive. Our proposed PCARR classification construct is physiologically logical and systemically highlights the essential pathophysiology underlying POCA. Monitored variables, such as ECG, Spo_2, $Etco_2$, and blood pressure, can provide clues to the causes of POCA. The A-SERCH list of actions may also aid in effectively investigating the underlying cause. We recommend combining the Hs and Ts mnemonic, the PCARR construct, clues from monitoring, and the A-SERCH list of actions in a rational manner when investigating POCA causes.

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REFERENCES

1. Braz LG, Módolo NSP, do Nascimento P, Jr, et al. Perioperative cardiac arrest: a study of 53 718 anaesthetics over 9 yr from a Brazilian teaching hospital. *BJA*. 2006;96:569–575.
2. Nunnally ME, O'Connor MF, Kordylewski H, Westlake B, Dutton RP. The incidence and risk factors for perioperative cardiac arrest observed in the national anesthesia clinical outcomes registry. *Anesth Analg*. 2015;120:364–370.
3. Sprung J, Warner ME, Contreras MG, et al. Predictors of survival following cardiac arrest in patients undergoing noncardiac surgery: a study of 518,294 patients at a tertiary referral center. *Anesthesiology*. 2003;99:259–269.
4. Sobreira-Fernandes D, Teixeira L, Lemos TS, et al. Perioperative cardiac arrests: a subanalysis of the anesthesia-related cardiac arrests and associated mortality. *J Clin Anesth*. 2018;50:78–90.

5. Anthi A, Tzelepis GE, Alivizatos P, Michalis A, Palatianos GM, Geroulanos S. Unexpected cardiac arrest after cardiac surgery: incidence, predisposing causes, and outcome of open chest cardiopulmonary resuscitation. *Chest*. 1998;113:15–19.
6. Geube MA, Hsu A, Skubas NJ, et al. Incidence, outcomes, and risk factors for preincision cardiac arrest in cardiac surgery patients. *Anesth Analg*. 2022;135:1189–1197.
7. Bharti N, Batra YK, Kaur H. Paediatric perioperative cardiac arrest and its mortality: database of a 60-month period from a tertiary care paediatric centre. *Eur J Anaesthesiol*. 2009;26:490–495.
8. Smith NK, Zerillo J, Kim SJ, et al. Intraoperative cardiac arrest during adult liver transplantation: incidence and risk factor analysis from 7 academic centers in the United States. *Anesth Analg*. 2021;132:130–139.
9. Rasmussen TP, Riley DJ, Sarazin MV, Chan PS, Girotra S. Variation across hospitals in in-hospital cardiac arrest incidence among medicare beneficiaries. *JAMA Netw Open*. 2022;5:e2148485–e2148485.
10. Kazaure HS, Roman SA, Rosenthal RA, Sosa JA. Cardiac arrest among surgical patients: an analysis of incidence, patient characteristics, and outcomes in ACS-NSQIP. *JAMA Surg*. 2013;148:14–21.
11. Panchal AR, Bartos JA, Cabañas JG, et al; Adult Basic and Advanced Life Support Writing Group. Part 3: adult basic and advanced life support: 2020 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2020;142(16_suppl_2):S366–S468.
12. Neumar RW, Otto CW, Link MS, et al. Part 8: adult advanced cardiovascular life support: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2010;122(18_suppl_3):S729–S767.
13. Durila M. Reversible causes of cardiac arrest 4 "Ts" and 4 "Hs" can be easily diagnosed and remembered following general ABC rule, Motol University Hospital approach. *Resuscitation*. 2018;126:e7.
14. Wik L, Kramer-Johansen J, Myklebust H, et al. Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. *JAMA*. 2005;293:299–304.
15. Andersen LW, Holmberg MJ, Berg KM, Donnino MW, Granfeldt A. In-hospital cardiac arrest: a review. *JAMA*. 2019;321:1200–1210.
16. Ramachandran SK, Mhyre J, Kheterpal S, et al; American Heart Association's Get With The Guidelines-Resuscitation Investigators. Predictors of survival from perioperative cardiopulmonary arrests: a retrospective analysis of 2,524 events from the Get With The Guidelines-Resuscitation registry. *Anesthesiology*. 2013;119:1322–1339.
17. Fielding-Singh V, Willingham MD, Fischer MA, Grogan T, Benharash P, Neelankavil JP. A population-based analysis of intraoperative cardiac arrest in the United States. *Anesth Analg*. 2020;130:627–634.
18. Sasson C, Rogers MA, Dahl J, Kellermann AL. Predictors of survival from out-of-hospital cardiac arrest: a systematic review and meta-analysis. *Circ Cardiovasc Qual Outcomes*. 2010;3:63–81.
19. Moitra VK, Einav S, Thies KC, et al. Cardiac arrest in the operating room: resuscitation and management for the anesthesiologist: Part 1. *Anesth Analg*. 2018;126:876–888.
20. McEvoy MD, Thies KC, Einav S, et al. Cardiac arrest in the operating room: part 2-special situations in the perioperative period. *Anesth Analg*. 2018;126:889–903.
21. Bhananker SM, Ramamoorthy C, Geiduschek JM, et al. Anesthesia-related cardiac arrest in children: update from the Pediatric Perioperative Cardiac Arrest registry. *Anesth Analg*. 2007;105:344–350.
22. Ramamoorthy C, Haberkern CM, Bhananker SM, et al. Anesthesia-related cardiac arrest in children with heart disease: data from the Pediatric Perioperative Cardiac Arrest (POCA) registry. *Anesth Analg*. 2010;110:1376–1382.
23. Matsusaki T, Hilmi IA, Planinsic RM, Humar A, Sakai T. Cardiac arrest during adult liver transplantation: a single institution's experience with 1238 deceased donor transplants. *Liver Transpl*. 2013;19:1262–1271.
24. Siracuse JJ, Meltzer EC, Gill HL, et al. Outcomes and risk factors of cardiac arrest after vascular surgery procedures. *J Vasc Surg*. 2015;61:197–202.
25. Riley CM, Murphy LD, Mastropietro CW. Cardiac arrest in children following cardiac surgery: a scoping review of contributing factors. *World J Pediatr Congenit Heart Surg*. 2022;13:475–481.
26. Zile MR, Baicu CF, Gaasch WH. Diastolic heart failure—abnormalities in active relaxation and passive stiffness of the left ventricle. *N Engl J Med*. 2004;350:1953–1959.
27. Glantz SA, Parmley WW. Factors which affect the diastolic pressure-volume curve. *Circ Res*. 1978;42:171–180.
28. McKay RG, Miller MJ, Ferguson JJ, et al. Assessment of left ventricular end-systolic pressure-volume relations with an impedance catheter and transient inferior vena cava occlusion: use of this system in the evaluation of the cardiotonic effects of dobutamine, milrinone, posicor and epinephrine. *J Am Coll Cardiol*. 1986;8:1152–1160.
29. Chantler PD, Lakatta EG, Najjar SS. Arterial-ventricular coupling: mechanistic insights into cardiovascular performance at rest and during exercise. *J Appl Physiol*. 2008;105:1342–1351.
30. Ewah B, Calder I. Intraoperative death during lumbar discectomy. *Br J Anaesth*. 1991;66:721–723.
31. Levy JH, Adkinson NFJ. Anaphylaxis during cardiac surgery: implications for clinicians. *Anesth Analg*. 2008;106:392–403.
32. Hepner DL, Castells MC. Anaphylaxis during the perioperative period. *Anesth Analg*. 2003;97:1381–1395.
33. Mitsuhata H, Shimizu R, Yokoyama MM. Role of nitric oxide in anaphylactic shock. *J Clin Immunol*. 1995;15:277–283.
34. Schummer C, Wirsing M, Schummer W. The pivotal role of vasopressin in refractory anaphylactic shock. *Anesth Analg*. 2008;107:620–624.
35. Yamaoka M, Deguchi M, Ninomiya K, Kurasako T, Matsumoto M. A suspected case of rocuronium-sugammadex complex-induced anaphylactic shock after cesarean section. *J Anesth*. 2017;31:148–151.
36. Harper NJN, Cook TM, Garcez T, et al. Anaesthesia, surgery, and life-threatening allergic reactions: management and outcomes in the 6th National Audit Project (NAP6). *Br J Anaesth*. 2018;121:172–188.
37. Koster A, Meyer-Jark T, Schirmer U, Sandica E. Fulminant intraoperative right heart and pulmonary artery thrombosis following prothrombin complex concentrate infusion after complex open heart surgery with cardiopulmonary bypass. *A A Case Rep*. 2014;2:89–91.
38. Brown J, Rogers J, Soar J. Cardiac arrest during surgery and ventilation in the prone position: a case report and systematic review. *Resuscitation*. 2001;50:233–238.
39. Albin MS, Ritter RR, Pruett CE, Kalff K. Venous air embolism during lumbar laminectomy in the prone position: report of three cases. *Anesth Analg*. 1991;73:346–349.
40. McDouall SF, Shlugman D. Fatal venous air embolism during lumbar surgery: the tip of an iceberg? *Eur J Anaesthesiol*. 2007;24:803–805.

41. Lang SA, Duncan PG, Dupuis PR. Fatal air embolism in an adolescent with duchenne muscular dystrophy during harrington instrumentation. *Anesth Analg*. 1989;69:132–134.
42. Despond O, Fiset P. Oxygen venous embolism after the use of hydrogen peroxide during lumbar discectomy. *Can J Anaesth*. 1997;44:410–413.
43. Prabhakar H, Rath GP, Arora R, Jha M. Increased risk of venous oxygen embolism with the use of hydrogen peroxide in sitting position. *J Clin Anesth*. 2007;19:406–407.
44. Kim I-S, Jung J-W, Shin K-M. Cardiac arrest associated with carbon dioxide gas embolism during laparoscopic surgery for colorectal cancer and liver metastasis: a case report. *Korean J Anesthesiol*. 2012;63:469–472.
45. Kim SJ, Yoo KY, Lee HG, Kim WM, Jeong CW, Lee HJ. Fatal pulmonary embolism caused by thrombosis of contralateral axillary vein after arthroscopic right rotator cuff repair: a case report. *Korean J Anesthesiol*. 2010;59:S172–S175.
46. Hong B, Yoon SH, Park SY, Song S, Youn A, Hwang JG. Cardiac arrest from patient position change after spine surgery on a Jackson table. *Acute Crit Care*. 2019;34:86–91.
47. Chen HL, Wong CS, Ho ST, Chang FL, Hsu CH, Wu CT. A lethal pulmonary embolism during percutaneous vertebroplasty. *Anesth Analg*. 2002;95:1060–1062, .
48. Ecker JL, Solt K, Fitzsimons MG, MacGillivray TE. Case 40-2012. *N Engl J Med*. 2012;367:2528–2536.
49. Arnone B. Amniotic fluid embolism: a case report. *J Nurse Midwifery*. 1989;34:92–94.
50. Clark SL, Hankins GD, Dudley DA, Dildy GA, Porter TF. Amniotic fluid embolism: analysis of the national registry. *Am J Obstet Gynecol*. 1995;172(4 Pt 1):1158–67; .
51. Rudra A, Chatterjee S, Sengupta S, Nandi B, Mitra J. Amniotic fluid embolism. *Indian J Crit Care Med*. 2009;13:129–135.
52. Leithner C, Podolsky A, Globits S, et al. Magnetic resonance imaging of the heart during positive end-expiratory pressure ventilation in normal subjects. *Crit Care Med*. 1994;22:426–432.
53. Corp A, Thomas C, Adlam M. The cardiovascular effects of positive pressure ventilation. *BJA Educ*. 2021;21:202–209.
54. Investigators Writing Group for the Alveolar Recruitment for Acute Respiratory Distress Syndrome Trial. Effect of lung recruitment and titrated positive end-expiratory pressure (PEEP) vs low PEEP on mortality in patients with acute respiratory distress syndrome: a randomized clinical trial. *JAMA*. 2017;318:1335–1345.
55. Marseu K, Slinger P, de Perrot M, McRae K, Ma M. Dynamic hyperinflation and cardiac arrest during one-lung ventilation: a case report. *Canadian J Anesth*. 2011;58:396–400.
56. Roberts DJ, Leigh-Smith S, Faris PD, et al. Clinical presentation of patients with tension pneumothorax: a systematic review. *Ann Surg*. 2015;261:1068–1078.
57. Hoechter DJ, Speck E, Siegl D, Laven H, Zwissler B, Kammerer T. Tension pneumothorax during one-lung ventilation: an underestimated complication? *J Cardiothorac Vasc Anesth*. 2018;32:1398–1402.
58. Weng W, DeCrosta DJ, Zhang H. Tension pneumothorax during one-lung ventilation: a case report. *J Clin Anesth*. 2002;14:529–531.
59. Ariyarajah V, Spodick DH. Cardiac tamponade revisited: a postmortem look at a cautionary case. *Tex Heart Inst J*. 2007;34:347–351.
60. Lee C. Cardiac tamponade: a case series. *Clin Surg*. 2018;3:2051.
61. Zheng M, Kang Y, Wang T, Wei J. Cardiac tamponade complicating thoracentesis in a patient after left pneumonectomy: a case report. *Medicine (Baltimore)*. 2020;99:e19778.
62. Memtsoudis SG, Rosenberger P, Loffler M, et al. The usefulness of transesophageal echocardiography during intraoperative cardiac arrest in noncardiac surgery. *Anesth Analg*. 2006;102:1653–1657.
63. Ehrenfeld JM, Funk LM, Van Schalkwyk J, Merry AF, Sandberg WS, Gawande A. The incidence of hypoxemia during surgery: evidence from two institutions. *Canadian J Anesth*. 2010;57:888–897.
64. Keenan RL, Boyan CP. Cardiac arrest due to anesthesia: a study of incidence and causes. *JAMA*. 1985;253:2373–2377.
65. Olsson GL, Hallén B. Cardiac arrest during anaesthesia: a computer-aided study in 250,543 anaesthetics. *Acta Anaesthesiol Scand*. 1988;32:653–664.
66. Mort TC. The incidence and risk factors for cardiac arrest during emergency tracheal intubation: a justification for incorporating the ASA Guidelines in the remote location. *J Clin Anesth.* 2004;16:508–516.
67. Moller JT, Pedersen T, Rasmussen LS, et al. Randomized evaluation of pulse oximetry in 20,802 patients: I. Design, demography, pulse oximetry failure rate, and overall complication rate. *Anesthesiology*. 1993;78:436–444.
68. Pell R, Littell J, Hernandez M, Mirajkar A, Ganti L. Cardiopulmonary arrest from metformin-induced lactic acidosis. *Resusc Plus*. 2022;9:100217.
69. Rosen R, Robbins-Juarez S, Stevens J. Ethylene glycol intoxication requiring ECMO support. *Case Rep Crit Care*. 2021;2021:5545351.
70. Sahutoglu T, Sahutoglu E. Hemodialysis during cardiopulmonary resuscitation in patients with severe metabolic acidosis. *Int J Artif Organs*. 2018;41:431–436.
71. Ulukaya S, Alper I, Aydin U, Kilic M. Successful resuscitation of cardiac arrest due to postreperfusion syndrome during orthotopic liver transplantation: a case report. *Transplant Proc*. 2007;39:3527–3529.
72. Wang CH, Chang WT, Huang CH, et al. Associations between early intra-arrest blood acidaemia and outcomes of adult in-hospital cardiac arrest: a retrospective cohort study. *J Formos Med Assoc*. 2020;119:644–651.
73. Spindelboeck W, Gemes G, Strasser C, et al. Arterial blood gases during and their dynamic changes after cardiopulmonary resuscitation: a prospective clinical study. *Resuscitation*. 2016;106:24–29.
74. Gili S, Cammann VL, Schlossbauer SA, et al. Cardiac arrest in takotsubo syndrome: results from the InterTAK Registry. *Eur Heart J*. 2019;40:2142–2151.
75. Xu Y, Liu M, Li J, Rong J. Takotsubo cardiomyopathy as an overlooked cause of cardiac arrest in a patient undergoing ureteral stenting: a case report and literature review. *J Int Med Res*. 2022;50:3000605221099255.
76. Taniguchi T, Morimoto T, Shiomi H, et al; CURRENT AS Registry Investigators. Sudden death in patients with severe aortic stenosis: observations from the CURRENT AS registry. *J Am Heart Assoc*. 2018;7:e008397.
77. Weissler-Snir A, Allan K, Cunningham K, et al. Hypertrophic cardiomyopathy: related sudden cardiac death in young people in Ontario. *Circulation*. 2019; 140:1706–1716.
78. Luckner G, Margreiter J, Jochberger S, et al. Systolic anterior motion of the mitral valve with left ventricular outflow tract obstruction: three cases of acute perioperative hypotension in noncardiac surgery. *Anesth Analg*. 2005;100:1594–1598.
79. Hertel T, Banayan JM, Chaney MA, von Dossow V, Dhawan R. Systolic anterior motion of the mitral valve with left ventricular outflow tract obstruction: a rare cause of hypotension after lung transplantation. *J Cardiothorac Vasc Anesth*. 2017;31:1894–1901.
80. Smith BB, Nickels AS, Sviggum HP. A rare combination of undiagnosed hypertrophic cardiomyopathy revealed by intraoperative anaphylaxis resulting in acute left

ventricular outflow obstruction and cardiac arrest. *J Clin Anesth*. 2016;31:212–214.

81. Matteucci M, Kowalewski M, Fina D, et al. Extracorporeal life support for phaeochromocytoma-induced cardiogenic shock: a systematic review. *Perfusion*. 2020;35(1_suppl):20–28.
82. Carey M, Carter J, Nesbitt I. Phaeochromocytoma crisis presenting under anaesthesia with profound left ventricular failure—successful treatment with intra-aortic balloon pump. *J Intensive Care Soc*. 2010;11:192–195.
83. Hansel J, Ármannsson GS. Cardiac arrest due to accidental overdose with norepinephrine dissolved in crystalloid. *BMJ Case Rep*. 2020;13:e237643.
84. Borden SB, Groose MK, Robitaille MJ, Schroeder KM. Cardiac arrhythmia from epinephrine overdose in epidural test dose. *Saudi J Anaesth*. 2019;13:249–252.
85. Di Chiara A, Werren M, Badano LP, Fioretti PM. Dynamic left ventricular outflow tract obstruction: an unusual mechanism mimicking anterior myocardial infarction with cardiogenic shock. *Ital Heart J*. 2001;2:60–67.
86. Kumari A, Gupta R, Bajwa SJ, Singh A. Unanticipated cardiac arrest under spinal anesthesia: an unavoidable mystery with review of current literature. *Anesth Essays Res*. 2014;8:99–102.
87. Pollard JB. Common mechanisms and strategies for prevention and treatment of cardiac arrest during epidural anesthesia. *J Clin Anesth*. 2002;14:52–56.
88. Sprung J, Abdelmalak B, Schoenwald PK. Vasovagal cardiac arrest during the insertion of an epidural catheter and before the administration of epidural medication. *Anesth Analg*. 1998;86:1263–1265.
89. Watkins E, Dresner M, Calow C. Severe vasovagal attack during regional anaesthesia for caesarean section. *Br J Anaesth*. 2000;84:118–120.
90. Kinsella S, Tuckey J. Perioperative bradycardia and asystole: relationship to vasovagal syncope and the Bezold–Jarisch reflex. *Br J Anaesth*. 2001;86:859–868.
91. Jang Y-E, Do S-H, Song I-A. Vasovagal cardiac arrest during spinal anesthesia for cesarean section: a case report. *Korean J Anesthesiol*. 2013;64:77–81.
92. Ingersoll-Weng E, Manecke Gerard R, Thistlethwaite Patricia A. Dexmedetomidine and cardiac arrest. *Anesthesiology*. 2004;100:738–739.
93. Chelliah YR, Manninen PH. Hazards of epinephrine in transsphenoidal pituitary surgery. *J Neurosurg Anesthesiol*. 2002;14:43–46.
94. Bacon B, Silverton N, Katz M, et al. Local anesthetic systemic toxicity induced cardiac arrest after topicalization for transesophageal echocardiography and subsequent treatment with extracorporeal cardiopulmonary resuscitation. *J Cardiothorac Vasc Anesth*. 2019;33:162–165.
95. Yoshida M, Matsuda H, Fukuda I, Furuya K. Sudden cardiac arrest during cesarean section due to epidural anaesthesia using ropivacaine: a case report. *Arch Gynecol Obstet*. 2008;277:91–94.
96. Tierney KJ, Murano T, Natal B. Lidocaine-induced cardiac arrest in the emergency department: effectiveness of lipid therapy. *J Emerg Med*. 2016;50:47–50.
97. Dix SK, Rosner GF, Nayar M, et al. Intractable cardiac arrest due to lidocaine toxicity successfully resuscitated with lipid emulsion. *Crit Care Med*. 2011;39:872–874.
98. Litz RJ, Popp M, Stehr SN, Koch T. Successful resuscitation of a patient with ropivacaine-induced asystole after axillary plexus block using lipid infusion. *Anaesthesia*. 2006;61:800–801.
99. Weber F, Guha R, Weinberg G, Steinbach F, Gitman M. Prolonged pulseless electrical activity cardiac arrest after intranasal injection of lidocaine with epinephrine: a case report. *A A Pract*. 2019;12:438–440.
100. Marwick PC, Levin AI, Coetzee AR. Recurrence of cardiotoxicity after lipid rescue from bupivacaine-induced cardiac arrest. *Anesth Analg*. 2009;108:1344–1346.
101. Warren JA, Thoma RB, Georgescu A, Shah SJ. Intravenous lipid infusion in the successful resuscitation of local anesthetic-induced cardiovascular collapse after supraclavicular brachial plexus block. *Anesth Analg*. 2008;106:1578–1580, .
102. Foxall G, McCahon R, Lamb J, Hardman JG, Bedforth NM. Levobupivacaine-induced seizures and cardiovascular collapse treated with Intralipid. *Anaesthesia*. 2007;62:516–518.
103. Cordell CL, Schubkegel T, Light TR, Ahmad F. Lipid infusion rescue for bupivacaine-induced cardiac arrest after axillary block. *J Hand Surg Am*. 2010;35:144–146.
104. Harvey M, Cave G, Chanwai G, Nicholson T. Successful resuscitation from bupivacaine-induced cardiovascular collapse with intravenous lipid emulsion following femoral nerve block in an emergency department. *Emerg Med Australas*. 2011;23:209–214.
105. Vasques F, Behr AU, Weinberg G, Ori C, Di Gregorio G. A review of local anesthetic systemic toxicity cases since publication of the American Society of Regional Anesthesia Recommendations: to whom it may concern. *Reg Anesth Pain Med*. 2015;40:698–705.
106. Gitman M, Barrington MJ. Local anesthetic systemic toxicity: a review of recent case reports and registries. *Reg Anesth Pain Med*. 2018;43:124–130.
107. Aittomäki J, Liuhanen S, Sallisalmi M, Salmenperä MT, Heavner JE, Rosenberg PH. The effect of levosimendan on bupivacaine-induced severe myocardial depression in anesthetized pigs. *Reg Anesth Pain Med*. 2010;35:34–40.
108. Ryu HY, Kim JY, Lim HK, et al. Bupivacaine induced cardiac toxicity mimicking an acute non-ST segment elevation myocardial infarction. *Yonsei Med J*. 2007;48:331–336.
109. Wolfe JW, Butterworth JF. Local anesthetic systemic toxicity: update on mechanisms and treatment. *Curr Opin Anesthesiol*. 2011;24:561–566.
110. Muhammad Ali S, Shaikh N, Shahid F, Shah A, Zafar HB. Hypokalemia leading to postoperative critical arrhythmias: case reports and literature review. *Cureus*. 2020;12:e8149–e8149.
111. Struck MF, Nowak A. Cardiac arrest during elective orthopedic surgery due to moderate hypokalemia. *Middle East J Anaesthesiol*. 2011;21:435–436.
112. Shin IW, Sohn JT, Choi JY, Lee HK, Lee CH, Chung YK. Cardiac arrest due to severe hypokalemia during barbiturate coma therapy in a patient with severe acute head injury: a case report. *Korean J Anesthesiol*. 2006;50:S71–S73.
113. Buntain SG, Pabari M. Massive transfusion and hyperkalaemic cardiac arrest in craniofacial surgery in a child. *Anaesth Intensive Care*. 1999;27:530–533.
114. Baz EM, Kanazi GE, Mahfouz RA, Obeid MY. An unusual case of hyperkalaemia-induced cardiac arrest in a paediatric patient during transfusion of a "fresh" 6-day-old blood unit. *Transfus Med*. 2002;12:383–386.
115. Smith HM, Farrow SJ, Ackerman JD, Stubbs JR, Sprung J. Cardiac arrests associated with hyperkalemia during red blood cell transfusion: a case series. *Anesth Analg*. 2008;106:1062–1069.
116. Lee AC, Reduque LL, Luban NL, Ness PM, Anton B, Heitmiller ES. Transfusion-associated hyperkalemic cardiac arrest in pediatric patients receiving massive transfusion. *Transfusion*. 2014;54:244–254.
117. Woodforth IJ. Resuscitation from transfusion-associated hyperkalaemic ventricular fibrillation. *Anaesth Intensive Care*. 2007;35:110–113.

118. Lee S-H, Kim K-J, Chang C-H, Heo S-B. Cardiac arrest from acute hyperkalemia during liver surgery: a case report. *Korean J Anesthesiol*. 2010;59:S124–S127.
119. Part 10.1: life-threatening electrolyte abnormalities. *Circulation*. 2005;112(24_supplement):IV-121–IV-125.
120. Guimard C, Batard E, Lavainne F, Trewick D. Is severe hypercalcemia immediately life-threatening? *Eur J Emerg Med*. 2018;25:110–113.
121. Kim SJ, Kim HS, Hwang SO, et al; Korean Cardiac Arrest Research Consortium (KoCARC) Investigators. Ionized calcium level at emergency department arrival is associated with return of spontaneous circulation in out-of-hospital cardiac arrest. *PLoS One*. 2020;15:e0240420.
122. Reno CM, Skinner A, Bayles J, Chen YS, Daphna-Iken D, Fisher SJ. Severe hypoglycemia-induced sudden death is mediated by both cardiac arrhythmias and seizures. *Am J Physiol Endocrinol Metab*. 2018;315:E240–E249.
123. Fun JRS, Chia MYC. Hypoglycemic cardiac arrest and rapid return-of-spontaneous circulation (ROSC) with dextrose. *Am J Emerg Med*. 2020;38:1981.e1–1981.e3.
124. Monsieurs KG, Nolan JP, Bossaert LL, et al; ERC Guidelines 2015 Writing Group. European Resuscitation Council guidelines for resuscitation 2015: Section 1. Executive summary. *Resuscitation*. 2015;95:1–80.
125. Klausen HH, Cordtz J. Cardiac arrest in conjuction with hypoglycemia and spinal anesthesia. *J Clin Anesth*. 2013;25:429–430.
126. Tilahun T, Gudina G. Hypoglycemia as a cause of sudden cardiac arrest during cesarean delivery under spinal anesthesia: a case report and review of the literature. *J Med Case Rep*. 2021;15:376.
127. Dietrichs ES, Tveita T, Smith G. Hypothermia and cardiac electrophysiology: a systematic review of clinical and experimental data. *Cardiovasc Res*. 2019;115:501–509.
128. Ju MH, Kim MW. Ventricular fibrillation by hypothermia in spinal fusion surgery: a case report. *Korean J Anesthesiol*. 2009;57:647–651.
129. Park DH, Kim TW, Kim MS, et al. Cardiac arrest caused by accidental severe hypothermia and myocardial infarction during general anesthesia. *J Int Med Res*. 2021;49:300060520987945.
130. Aggarwal A, Farber NE, Kotter GS, Dhamee MS. Electrosurgery-induced ventricular fibrillation during pacemaker replacement—a unique mechanism. *J Clin Monit*. 1996;12:339–342.
131. Fogel RI, Prystowsky EN. Management of malignant ventricular arrhythmias and cardiac arrest. *Crit Care Med*. 2000;28(10 suppl):N165–N169.
132. Poon SS, George J, Obaid D, Kumar P. Myocardial infarction and ventricular fibrillation due to iatrogenic right coronary artery occlusion following tricuspid valve annuloplasty: a case report. *Eur Heart J Case Rep*. 2020;4:1–4.
133. Arps K, Chakravartti J, Hess CN, Rao SV. Ventricular fibrillation due to aortocoronary vein graft spasm during angiography. *JACC Case Rep*. 2021;3:388–391.
134. Plaisier BR. Thoracic lavage in accidental hypothermia with cardiac arrest—report of a case and review of the literature. *Resuscitation*. 2005;66:99–104.
135. Kloesel B, Ackerman MJ, Sprung J, Narr BJ, Weingarten TN. Anesthetic management of patients with Brugada syndrome: a case series and literature review. *Canadian J Anesth*. 2011;58:824–836.
136. Hollenberg SM, Dellinger RP. Noncardiac surgery: postoperative arrhythmias. *Crit Care Med*. 2000;28:N145–N150.
137. Chung E-J, Jeon Y-S, Kim H-J, et al. Torsade de pointes in liver transplantation recipient after induction of general anesthesia: a case report. *Korean J Anesthesiol*. 2014;66:80–84.
138. Brenner SM, Boucher J. Fatal cardiac arrest in 2 children: possible role of ondansetron. *Pediatr Emerg Care*. 2016;32:779–784.
139. Reid JM, Appleton PJ. A case of ventricular fibrillation in the prone position during back stabilisation surgery in a boy with Duchenne's muscular dystrophy. *Anaesthesia*. 1999;54:364–367.
140. Ornato JP, Garnett AR, Glauser FL. Relationship between cardiac output and the end-trial carbon dioxide tension. *Ann Emerg Med*. 1990;19:1104–1106.
141. Groombridge CJ, Kim Y, Maini A, Smit V, Fitzgerald MC. Stress and decision-making in resuscitation: a systematic review. *Resuscitation*. 2019;144:115–122.
142. Corazza F, Fiorese E, Arpone M, et al. The impact of cognitive aids on resuscitation performance in in-hospital cardiac arrest scenarios: a systematic review and meta-analysis. *Intern Emerg Med*. 2022;17:2143–2158.
143. Parker BK, Salerno A, Euerle BD. The use of transesophageal echocardiography during cardiac arrest resuscitation: a literature review. *J Ultrasound Med*. 2019;38:1141–1151.
144. Masoumi B, Azizkhani R, Heydari F, Zamani M, Nasr Isfahani M. The role of cardiac arrest sonographic exam (CASE) in predicting the outcome of cardiopulmonary resuscitation: a cross-sectional study. *Arch Acad Emerg Med*. 2021;9:e48.
145. Paul JA, Panzer OPF. Point-of-care ultrasound in cardiac arrest. *Anesthesiology*. 2021;135:508–519.
146. Efrimescu CI, Moorthy A, Griffin M. Rescue transesophageal echocardiography: a narrative review of current knowledge and practice. *J Cardiothorac Vasc Anesth*. 2023;37:584–600.
147. Ávila-Reyes D, Acevedo-Cardona AO, Gómez-González JF, Echeverry-Piedrahita DR, Aguirre-Flórez M, Giraldo-Diaconeasa A. Point-of-care ultrasound in cardiorespiratory arrest (POCUS-CA): narrative review article. *Ultrasound J*. 2021;13:46.