

Practical Guide

for

Intraoperative Cardiac Arrest

Japanese Society of Anesthesiologists

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1

Overview

Introduction

Overview of intraoperative cardiac arrest

Due to the aging of the population requiring surgery as well as advances in medical technology, the number of surgeries for serious cases has been increasing. According to the survey on accidental events conducted by the Japan Society of Anesthesiologists from 2009 to 2011,¹⁾ the mortality rate due to critical complications (perioperative mortality defined as death within 30 days after surgery) was 3.93/10,000 cases, while the anesthesia-related perioperative mortality rate was 0.07/10,000 cases. This perioperative mortality is significantly lower than that in 2004–2008 (5.56/10,000 cases). According to the 2014–2016 statistics, the incidence of cardiac arrest in anesthesia-managed cases has gradually decreased from 1.18/10,000 in 2014 to 1.09/10,000 in 2015, and 1.04/10,000 in 2016. Furthermore, the 30-day mortality was 0.61/10,000, 0.62/10,000, and 0.54/10,000, respectively. Causes of fatal incidents included hemorrhagic shock due to preoperative complications (26.2%), severe hemorrhage due to surgery (16.8%), multiple organ failure and sepsis (12.8%), and circulatory complications (12.0%), in that order.¹⁾ This trend is similar to the statistical trend from 2014 to 2016. Thirty-two cases of anesthesia-related perioperative mortality¹⁾ were reported, among which 6 were caused by drug administration (overdose/improper administration), 5 each by aspiration and inadequate ventilation, 4 by anesthetic overdose, and 3 by improper infusion or transfusion management. Only 1 case died due to improper airway management at the time of anesthesia induction. Deaths from nervous system accidents (brain and spinal cord) and other accidents were reported in 1 and 3 cases, respectively. Reports from institutions that were not included in the aggregation included 11 deaths due to anesthesia management, and 47 deaths in total were reported over a 3-year period. The age distribution of mortality cases shows that cardiac arrest, severe hypoxia, and perioperative mortality were the highest in newborns. The number of mortality cases decreased with age and increased again in older adults. The mortality rate per 10,000 cases was 17.0 in newborns, 10.15 in individuals aged 85 years and older, 6.05 in those aged 65–84 years, and 5.75 in infants.

According to reports of intraoperative cardiac arrest in countries outside Japan, the mortality rate was about 1.05–2.73 per 10,000 cases,²⁻⁴⁾ and the rate of perioperative cardiac arrest was 5.62–7.36 per 10,000 cases.^{5,6)} The mortality rate after cardiac arrest was 53.3–70.9%.²⁻⁹⁾ Anesthesia-related mortality was 0.01–1.12 per 10,000 cases.^{5,9,10)} Although there are various causes of cardiac arrest, many reports identified massive hemorrhage, airway problems, anaphylaxis, overdose, etc., as the cause of death.^{9,10)}

Policy of the Japan Society of Anesthesiologists

Both basic and advanced life support methods are advocated by the American Heart Association as measures for dealing with cardiac arrest outside and inside the hospital. The Japan Society of Anesthesiology has also made training in these measures required for acquiring specialist qualifications.

Based on these guidelines for cardiac arrest, it is appropriate to create a practical guide that considers intraoperative peculiarities to facilitate a prompt and appropriate response to intraoperative cardiac arrest.

Intraoperative cardiac arrest is more unusual than out-of-hospital or in-hospital incidental cardiac arrest. Vital signs are monitored, airways are controlled, infusion routes are secured, and it is often observed based on signs. Furthermore, since oxygen consumption is reduced during general anesthesia, organ protective effects may be obtained.

Although a favorable prognosis can be expected, early diagnosis of the cause and appropriate treatment are required based on the usual procedures for advanced cardiac life support (ACLS), such as early cardiopulmonary resuscitation (CPR) and early defibrillation. For cardiac arrest, it is essential that anesthesiologists, surgical clinicians, nurses, clinical engineers, and other staff involved perioperatively approach the case as a team. Ideally, the anesthesiologist will play an active role as the team leader.

Formulation of the practical guide

This practical guide was developed with the aim of improving the prognosis of patients by sharing treatment guidelines among anesthesiologists, surgical clinicians, nurses, and clinical engineers involved in the perioperative period. The contents of this guide are divided into general outline and detailed treatment. The latter lists the diagnosis and treatment methods for the pathological conditions likely to cause cardiac arrest.

Eight working group members were in charge of the formulation. The group ensured that there were no discrepancies in relation to the practical guides and guidelines already documented by the Japan Society of Anesthesiologists. There are no conflicts of interest to disclose for any of the members involved in the preparation of this practical guide.

In addition, this practical guide does not discredit medical practice that does not follow it, and is not formulated for the purpose of determining legal liability. This practical guide will be revised according to future developments in the practice of CPR.

Scope of intraoperative cardiac arrest

This practical guide is intended for perioperative anesthesia management, particularly for intraoperative cardiac arrest or crisis situations.

Basics of resuscitation in intraoperative cardiac arrest

Regardless of whether the case involves intraoperative cardiac arrest or cardiac arrest in the ward, the most important element is early detection and early initiation of CPR. Once cardiac arrest has been diagnosed, constant chest compressions are required. Compressions should be performed in the lower half of the sternum. The compression rate should be 100–120 times per minute, and the depth of compression should be 5–6 cm. The return of the thorax should be checked with each compression. If tracheal intubation or advanced airway control is not available, 30 mechanical ventilations should be given for 30 compressions. Ventilation should take about 1 second, and the rising of the victim's chest should be confirmed. Due care should be taken to avoid causing hyperventilation, and after advanced airway control, ventilation should be performed asynchronously at a rate of once every 6 seconds. Intravenous vasopressors are ineffective without chest compressions. Considering the delay in circulation time, a boost should be performed with 20 ml of physiological saline after intravenous administration.

Causes and prevention of intraoperative cardiac arrest

There are various causes of perioperative cardiac arrest, as opposed to out-of-hospital and in-hospital sudden cardiac arrest, which often involve ventricular fibrillation and ischemic heart disease. For perioperative cardiac arrest, the initial rhythms are often not ventricular fibrillation.⁵⁾ It is important to address such initial rhythms before they progress into cardiac arrest.

Causes of cardiac arrest that develop from bradycardia include surgical vagal stimulation, drug overdose (anesthetics, beta-blockers), cardiac sympathetic blockers (T1–T4), and atrioventricular block. On the other hand, typical examples of cardiac arrest due to tachycardia include hypovolemic shock due to massive bleeding, hypoxemia, anaphylaxis, and malignant hyperthermia. In hypoxemia, even if tachycardia appears early, bradycardia often occurs immediately before cardiac arrest. Ventricular and supraventricular arrhythmias change from tachycardia to ventricular fibrillation and pulseless electrical activity (PEA).

The assumed causes of intraoperative cardiac arrest include loss of airway, such as difficult airways and choking, ventilator abnormalities, severe asthma, and hypoxia due to impaired pulmonary function; deterioration of preoperative impaired cardiac function; myocardial infarction; cardiac tamponade; tension pneumothorax; anesthetic overdose; circulatory suppression due to high nerve block after spinal anesthesia; local anesthetic toxicity; anaphylaxis; pulmonary embolism; and malignant hyperthermia.

Prognosis of intraoperative cardiac arrest

In recent reports, intraoperative cardiac arrest was shown to have better prognosis than out-of-hospital cardiac arrest or cardiac arrest elsewhere in the hospital.⁶⁾ The reasons for this are that medical staff, including anesthesiologists, are nearby; monitors, airways, and infusion channels are often secured; and immediate response is possible. Ramachandran et al.⁷⁾ reported that the most common rhythm in intraoperative cardiac arrest was asystole, but its survival rate was 30.5–80%, which was higher than the resuscitation rate for in-hospital cardiac arrest (10%). Poor prognostic factors after intraoperative cardiac arrest include advanced age, high ASA physical status, and complicating infectious diseases.^{6,8,10)}

Determination of intraoperative cardiac arrest

Cardiac arrest is generally confirmed by the absence of a pulse. There are various causes of cardiac arrest, and diagnosis and treatment of the cause are required. In order to proceed to radical treatment, it is important to perform CPR until the underlying cause can be treated. Chest compressions are the most important procedure to ensure the return of spontaneous circulation; however, delays in chest compressions for intraoperative cardiac arrest have been pointed out.¹²⁾

One of the factors behind such delays in chest compressions is that the anesthesiologist tasked to give instructions for chest compressions is solely focused on the cause of circulatory collapse. Furthermore, the patient is unconscious from general anesthesia and their breathing is regulated. Moreover, most of the body is draped with a cover cloth. Depending on the surgery, the patient may be in a prone or lateral position. These factors affect the perception of cardiac arrest and, by extension, the initiation of CPR. For sudden circulatory collapse, abnormalities in the ECG (ECG), disappearance of the pressure waveform on the pulse oximeter and invasive arterial monitor, and deterioration of the end-tidal carbon dioxide monitors are useful indicators.

If it is determined that the patient is in cardiac arrest or experiencing a critical collapse, it is most important to inform the surgeon or the outpatient nurse promptly, and then ask them to cooperate in the treatment.

Monitoring

Monitoring itself is not a cure, but it is useful in diagnosing cardiac arrest, circulatory collapse, and circulatory recovery. Considering the patient's medical history, pathology, anesthesia method, and surgical procedure in the preoperative evaluation, the use of monitoring as invasive as necessary is effective for the early diagnosis and the prevention of cardiac arrest. However, the insertion of invasive monitoring should not delay resuscitation. Invasive arterial monitoring is the first choice if hemodynamics may be unstable. Central venous monitoring can provide data on central venous pressure and partial pressure of venous oxygen. This monitoring is also important as an administration route for catecholamines, etc. Pulmonary artery catheters provide cardiac output, mixed venous oxygen saturation, and are useful for assessing cardiac function. In recent years, operating rooms have been equipped with transesophageal and transthoracic echocardiography, and reports increasingly demonstrate their effectiveness in non-cardiac surgery.¹³⁾ The end-tidal carbon dioxide monitor is also used to evaluate proper circulation (quality of chest compressions) and is the most reliable monitor for airway management.^{14,15)}

Response to intraoperative cardiac arrest

When cardiac arrest is confirmed, briefly inform the situation context, request support, and immediately start chest compressions (Figure 1).¹⁶⁾ Ask the surgeon to suspend the surgery and whether there are any abnormalities in the surgical field. In case of hemorrhage, give instructions to stop the bleeding. Evaluate the ECG and prepare for cardioversion in case of ventricular fibrillation. If cardioversion is not possible,

administer adrenaline. Remember to boost with physiological saline after drug administration. If adrenaline cannot be given immediately, a vasopressor can be administered if immediately available. Administer 100% oxygen, perform manual ventilation, and check the airway pressure, ventilation volume, waveform, and the end-tidal carbon dioxide (EtCO₂) value. Cardiac arrest is preceded by a sudden decrease in EtCO₂ in cases with pulmonary embolism, and by abnormalities in EtCO₂ waveform and airway pressure in cases with airway problems. To exclude tension pneumothorax, use transthoracic ultrasonography for lung ultrasound and auscultation. Check the type and dose of the drugs that were administered immediately prior to the event. Collect blood and investigate the cause of cardiac arrest.

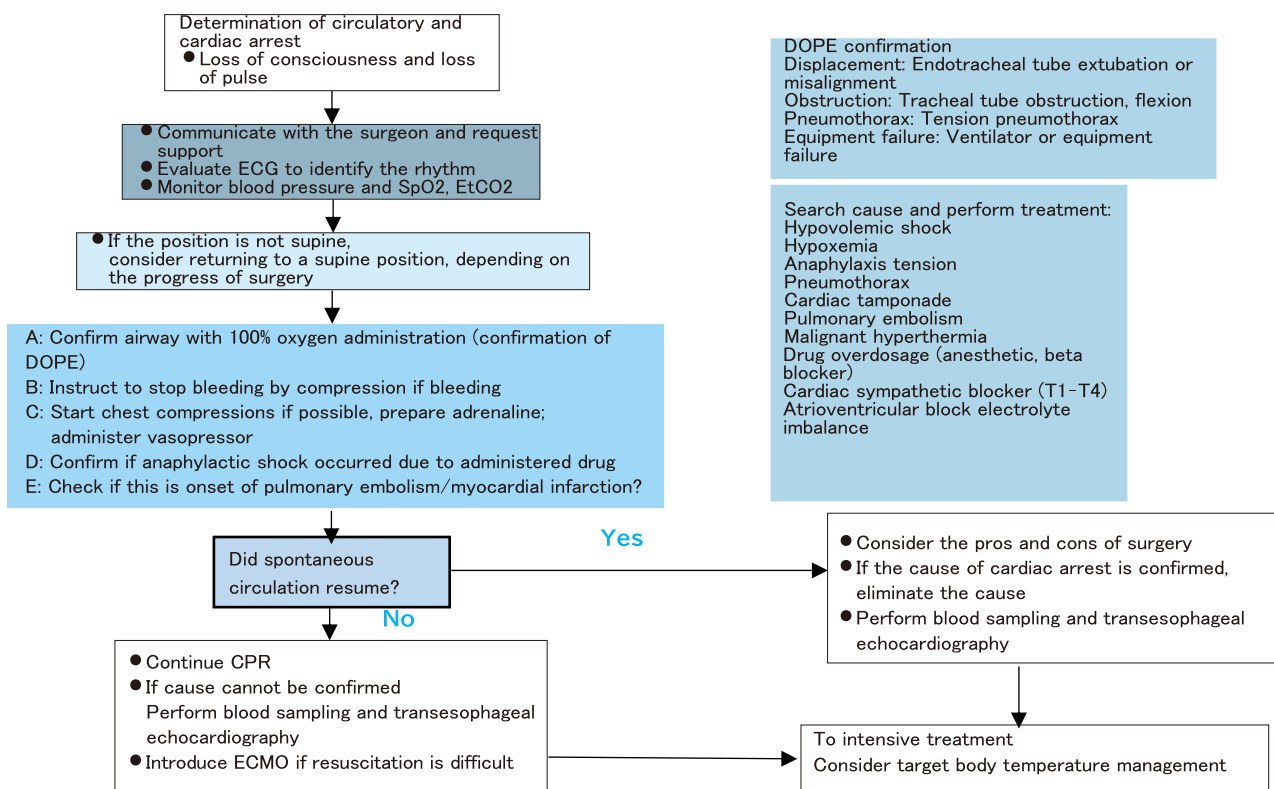


Figure 1 Initial response algorithm for intraoperative cardiac arrest. ECG, electrocardiogram; SpO₂, peripheral oxygen saturation; EtCO₂, end-tidal carbon dioxide; CPR, cardiopulmonary resuscitation; ECMO, extracorporeal membrane oxygenation.

Upon preparation, insert a 12-lead ECG and transesophageal or transthoracic echocardiography to check contraction and intracardiac volume. If cardiac arrest persists, continue treatment according to ACLS (Figure 2).

1) Compatible rhythm for cardioversion

If rhythm is suitable for cardioversion, perform the procedure immediately. For ventricular fibrillation and pulseless ventricular tachycardia, use the number of joules recommended for the device or 200 J if it is biphasic. Continue CPR for 2 minutes after cardioversion. To achieve a favorable prognosis, CPR with EtCO₂ above 20 mmHg is recommended.¹⁶⁾ Prepare 1 mg of adrenaline. If adrenaline is administered within 2 minutes of the initial cardioversion, the rate of resumption of circulation will decrease.¹⁷⁾ After 2 minutes, increase the number of joules if necessary and perform cardioversion. After the second cardioversion, administer 1 mg of adrenaline. Perform constant CPR while searching for the cause. Resume cardioversion if the rhythm is compatible with cardioversion and continues after 2 minutes. After

cardioversion, administer 300 mg of amiodarone. Lidocaine 1–1.5 mg/kg may be used as a substitute for amiodarone.

2) Incompatible rhythm for cardioversion

For PEA, 1 mg of adrenaline, or prepared ephedrine or phenylephrine, may be used. Administer the drug rapidly. Perform diagnosis and treatment of the cause at the same time as the treatment of cardiac arrest. Perform rhythm checks every 2 minutes to maintain high quality CPR.

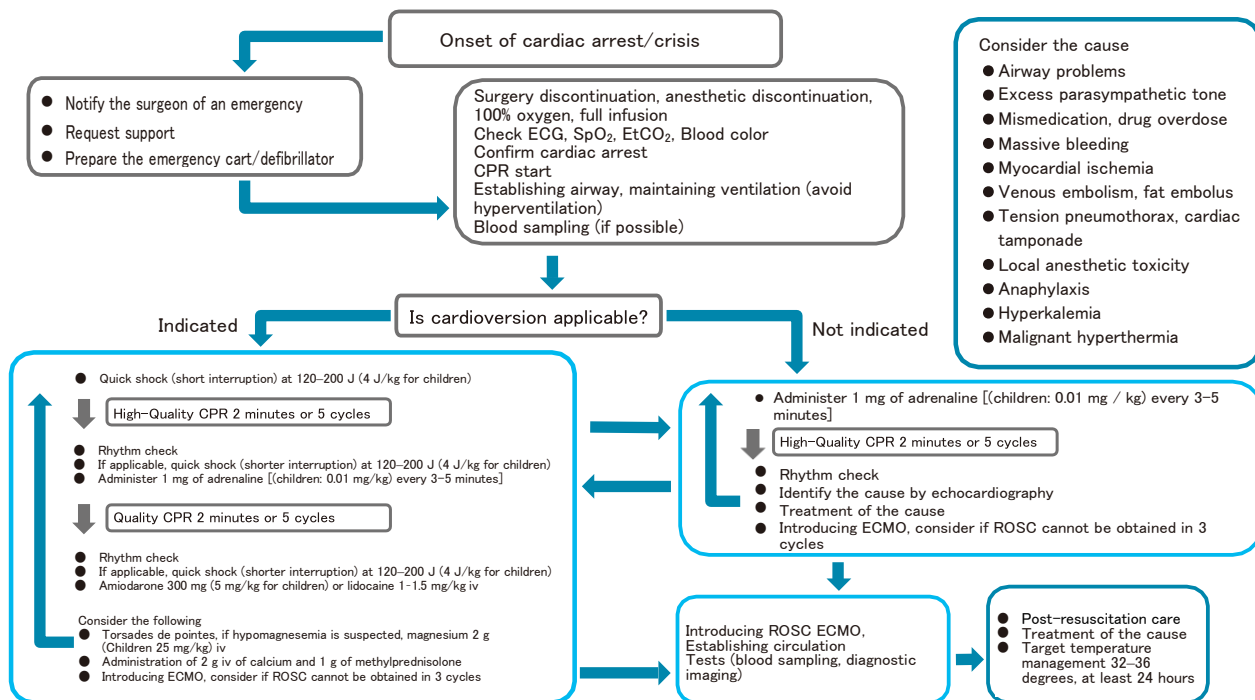


Figure 2 Algorithm for cardiac arrest treatment

ECG, electrocardiogram; EtCO₂, end-tidal carbon dioxide; SpO₂, peripheral oxygen saturation; CPR: cardiopulmonary resuscitation; ROSC: return of spontaneous circulation; ECMO, extracorporeal membrane oxygenation.

High-quality CPR : 100–120 times/min compression/chest wall returns to its original position each time/Aim for sufficient compression (EtCO₂> 20 mmHg). ROSC is unlikely at EtCO₂ < 10 mmHg. Aim for diastolic arterial pressure > 40 mmHg if invasive arterial pressure is being monitored/Do not interrupt (during artificial ventilation)/Avoid hyperventilation (primary ventilation less than 6 ml/kg, less than 10 times)

* ↓:2-minute CPR

[Revised and reprinted from 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;126:876–88]

The operating room is also advantageous for establishing extracorporeal membrane oxygenation (ECMO) at an early stage, and consideration of extracorporeal circulation CPR (ECPR) is recommended in Class IIb of the American Heart association (AHA) CPR guidelines.¹⁸⁾

In intraoperative cardiac arrest, it is highly possible to save the patient's life; thus, resuscitation must be continued without stopping.¹⁹⁾

Post-resuscitation care

When spontaneous circulation resumes, attempt to optimize respiration and circulation. Peripheral arterial oxygen saturation (SpO₂) should be maintained at 94% or higher, and partial end-tidal carbon dioxide pressure (pEtCO₂) should be maintained at 35–45 mmHg. Perform proper fluid replacement to maintain systolic blood pressure at 90 mmHg and consider the use of a vasopressor if necessary. Consider target body temperature management both during and after surgery at an early stage. Even in the AHA guidelines 2020, it is recommended to keep the temperature between 32°C and 36°C for at least 24 hours.²⁰⁾ For the purpose of thoroughly searching for the cause of cardiac arrest, perform 12-lead ECG, echocardiography, chest radiograph, and blood gas tests. If it is necessary to continue treatment for the causative disease, consult with a specialist in the appropriate department and decide on a plan according to the progress of surgery. Unless the cause of cardiac arrest is resolved, it may recur shortly afterwards (Figure 3).

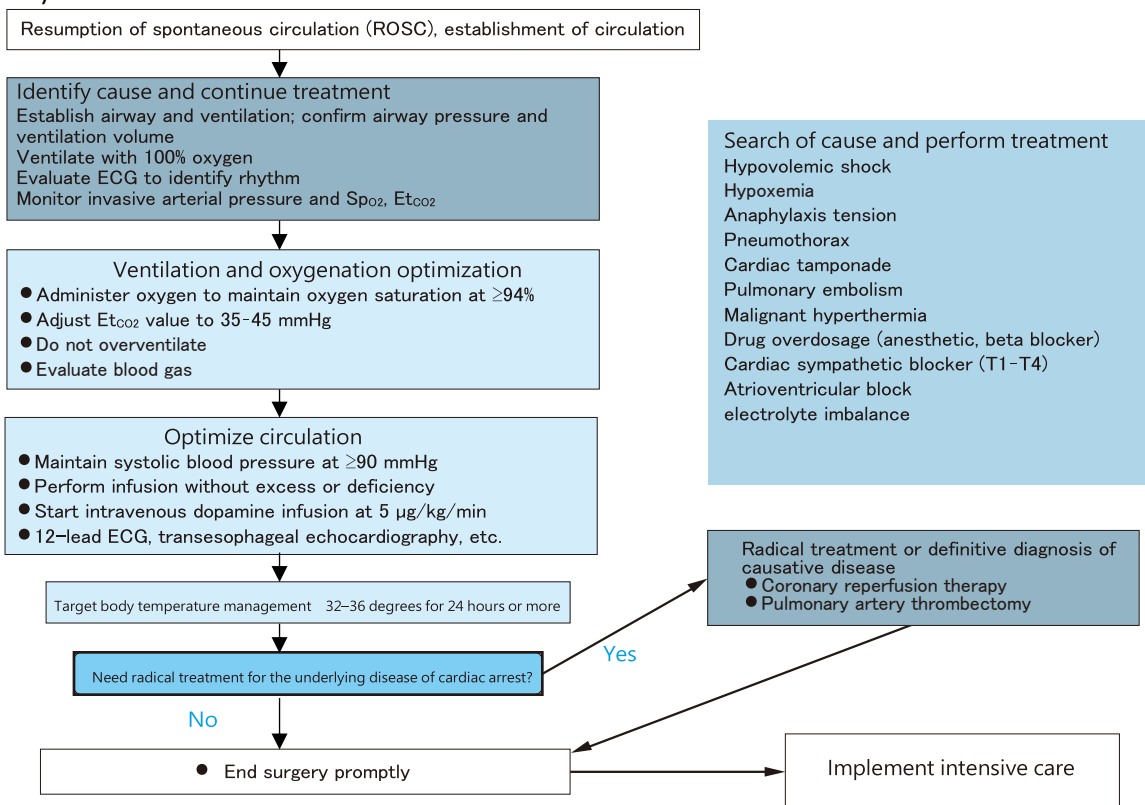


Figure 3 Post-resuscitation care algorithm

ECG, electrocardiogram; EtCO₂, end-tidal carbon dioxide; ROSC: Return of spontaneous circulation, T1–T4: 1st to 4th thoracic vertebrae level

Basic response to intraoperative cardiac arrest

1) Respiration

Ventilate with 100% oxygen. Avoid overventilation. The treatment of causes located in the airway system has a high priority. In case of airway problems, attempt oxygenation according to the DAM algorithm of the Japan Society of Anesthesiologists.²¹⁾

Once chest compressions are initiated, the development of pneumothorax should be considered, even if the cause of cardiac arrest is intrinsic. Consider veno-venous (VV) ECMO when oxygenation cannot be maintained. Mechanical ventilation with a ventilator may be used during CPR, but volume-controlled ventilation ensures better ventilation than pressure-controlled ventilation.

2) Hypovolemic shock

For hypovolemic shock, perform rapid fluid infusion. Use vasoconstrictors, such as phenylephrine, noradrenaline, and vasopressin. Compensate for the lack of circulating blood volume while administering vasoconstrictors. Do not respond by solely administering vasoconstrictors. Perform blood transfusion for anemia. For bleeding in the surgical field, instruct the surgeon to stop the bleeding through compression and prioritize vital signs.

Consider damage control surgery to avoid acidosis, abnormal coagulation, and hypothermia, which worsen prognosis.²²⁾ Respond by referring to the "Guidelines for Response to Critical Hemorrhage."²³⁾

Controlled hypotension in abdominal aortic aneurysm rupture and fluid management without diluting blood coagulation factors may be effective in managing hemorrhagic shock. However, there is no conclusion as to whether this will improve the prognosis compared to conventional treatment.²⁴⁾

3) Impaired cardiac function

(A) Impaired left cardiac function

Perform echocardiography and pulmonary artery catheterization for evaluation. Causes include ischemic heart disease, dilated cardiomyopathy, and mitral valve disease. Use catecholamines to reduce cardiac contractility. Consider preload to reduce afterload and optimize left ventricular capacity. Depending on the condition, impairment of diastolic capacity may be improved by the administration of milrinone and olprinone. If the patient is unresponsive to high concentrations of catecholamines, such as adrenaline, consider inserting an intra-aortic balloon pump (IABP) or ECMO.

(B) Right cardiac dysfunction

As with impaired left cardiac function, perform transesophageal echocardiography and pulmonary artery catheterization for evaluation. Causes of right ventricular dysfunction include decreased right ventricular contractility (right ventricular infarction, etc.), increased right ventricular volumetric load (acute tricuspid regurgitation, disruption of Fontan circulation, etc.), increased right ventricular afterload (primary pulmonary embolism hypertension, etc.), and right ventricular diastolic dysfunction (cardiac tamponade, constrictive epicarditis). Clinical findings include jugular vein engorgement, hepatic vein stasis, and transudative pleural effusion. Treatment aims to reduce right ventricular afterload, increase right ventricular contractility, and control right ventricular preload adequately. The combined use of a phosphodiesterase (PDE) III inhibitor and dobutamine is effective in reducing pulmonary vascular resistance and enhancing right ventricular contractility. If the degree of pulmonary hypertension is high, prostaglandin preparations, nitric oxide (NO) inhalation, and calcium (Ca) antagonists are effective. Vasopressin is effective in increasing pulmonary arterial pressure and lowering systemic blood pressure. If circulation cannot be maintained, ECMO reduces the load on the right ventricle.

4) Spinal anesthesia-related cardiac arrest

In cases with spinal anesthesia, bradycardia, hypotension, and cardiac arrest may occur due to drug overdosage and unexpected spread of the anesthesia level.^{25,26)} It is also known that even healthy patients can develop severe bradycardia, vasodilation, and hypotension, leading to sudden cardiac arrest. Risk factors include ① spinal anesthesia reaching at least T5, ② young healthy persons (ASA 1), ③ β -blocker use, ④ bradycardia, ⑤ head-raising position change or rapid position change, ⑥ aortocaval compression syndrome

during caesarean section. It occurs not only in spinal anesthesia but also in inappropriate subarachnoid injection during epidural anesthesia. After drug administration, check vital signs, such as blood pressure, every minute. For prophylaxis, use ephedrine or atropine.

5) Special characteristics of children

Children, particularly nursing infants, are more likely to experience cardiac arrest. There are many requests for sedation outside the operating room; thus, these patients are at high risk. Anatomically, there may be factors that make it difficult to perform airway control, such as hypertrophy of the tonsils and a relatively large tongue. Hypoxia may occur due to breathlessness and difficulty in ventilation during gradual anesthesia induction, resulting in severe bradycardia and cardiac arrest.^{27,28)} Children also have a smaller reserve until cardiac arrest as compared to adults. In addition, drug dose errors are more likely to occur. For details, refer to the “12 Peculiarities of Pediatric Perioperative Resuscitation” (p. 52–59) in this guide.

Summary

Preoperative evaluation is important because intraoperative cardiac arrest is likely to occur in patients with poor preoperative conditions. Once cardiac arrest occurs during surgery, calmly begin CPR. After resuscitation, comprehensively judge whether surgery should be discontinued or reduced. Carry out target body temperature management with post-resuscitation brain protection in mind.

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Airway Management

- Dealing with airway abnormalities is a top priority.
- For crisis management when anesthesia is introduced, refer to the Airway Management Guidelines created by the Japan Society of Anesthesiology.¹⁾ Conduct regular training using the DAM algorithm, including cricothyrotomy.
- If cardiac arrest occurs during surgery, ventilate with 100% oxygen. Ventilate manually and check ventilation by checking chest elevation, ventilation resistance, and chest auscultation. If chest compressions are appropriate, EtCO₂ should be at least 10 mmHg.²⁾ Consider pulmonary embolism in case of sudden severe hypoxemia. Establish extracorporeal membrane oxygenation (ECMO) as much as possible.^{3,4)}

Prevention

- Perform appropriate evaluation before tracheal intubation.
- Prepare a bronchofiberscope, cricothyrotomy, tracheostomy, veno-venous (VV) ECMO, etc., according to the awake intubation and prior tracheostomy plan and depending on the risk.
- At least two specialists are required in high-risk patients.
- Perform sufficient oxygenation before induction of anesthesia.
- Keep supraglottic apparatus in the anesthesia cart. Ideally, an indirect laryngoscope should be immediately available.

Response during onset

- Respond according to the algorithm procedure (Figure 1).
- Response after cardiac arrest (correspondence in the red zone)
 - In case of accidental extubation, insert a supraglottic apparatus.
 - Perform endotracheal intubation through the supraglottic apparatus and endotracheal intubation by combining supraglottic apparatus and fiberscope.
 - More than three operations by the same technician or with the same apparatus should be avoided, particularly with a direct or indirect laryngoscope.
 - Perform cricothyrotomy. Alternatively, perform tracheostomy.
 - It is recommended to use ultrasound to confirm the location of tracheostomy and cricothyroid puncture.
 - Puncture using a large-diameter intravenous needle and emergency jet ventilation should be avoided (air trapping, pneumothorax, subcutaneous emphysema, mediastinal emphysema, etc., are likely to occur).

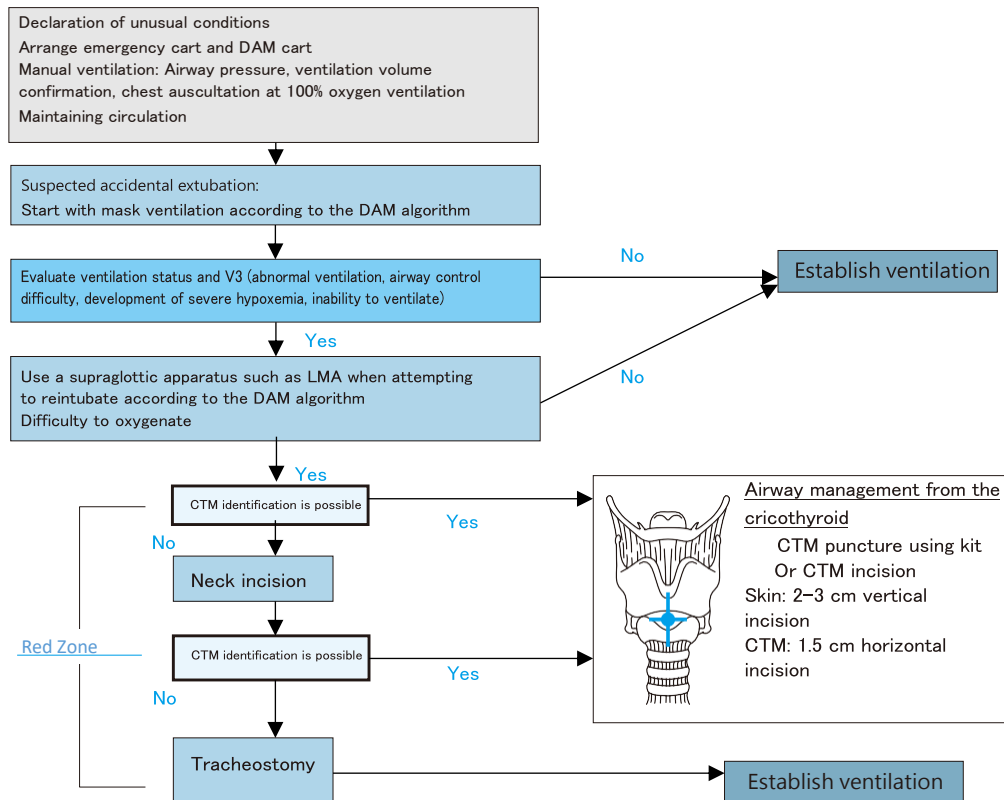


Figure 1 Algorithm for airway management during cardiac arrest

DAM: difficult airway management, LMA: laryngeal mask airway, CTM: cricothyroid membrane

● Promptly introduce ECMO if the patient's airway and respiratory function are significantly impaired.

□ Air trapping and auto-positive end-expiratory pressure

Auto-positive end-expiratory pressure (PEEP) is known as an intrinsic PEEP that can cause pulseless electrical activity (PEA).⁵⁾ In patients with severe asthma attacks or chronic obstructive pulmonary disease (COPD), the next respiration occurs before the end of exhalation, causing excessive pressure in the airways. As a result, venous return decreases.^{6,7)}

Respiratory mechanics graphics, such as pressure volume curves, are useful for analysis.⁸⁾

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Cardiac arrest due to hemorrhage

Learning items

Analysis of the causes of intraoperative cardiac arrest has identified massive bleeding as the number one cause. Response to bleeding is managed according to the "Critical Bleeding Guidelines" of the Japan Society of Anesthesiologists (Figure 1).¹⁾ If cardiac arrest occurs due to bleeding, communicate with the surgeon and respond to the best of your abilities.

Prevention

For patients at risk of massive bleeding, it is recommended to have as many thick infusion lines, at least 18 G, in the upper limbs as possible. The right upper limb is important in open heart surgery.

It is recommended to place an invasive arterial monitor for the purpose of responding to blood pressure fluctuations and performing blood sampling tests.

During cardiac arrest onset

The anesthesiologist declares an emergency and appoints a commander. The commander convenes the manpower and informs the blood transfusion department of an "emergency situation." Comprehensively evaluate the hemostasis status, hemodynamics, test data, and supply system of transfusion products; discuss with the surgeon whether to continue surgery or change the surgical procedure.

Anesthesiologist

- Instruct the surgeon to stop the bleeding by compression and gather human resources including both the surgeon and anesthesiologist.
- Administer 100% oxygen. Discontinue or reduce the dose of anesthetic.
- Administer a vasopressor drug to prevent PEA.
- Infuse extracellular fluid, artificial colloid, etc., until blood transfusion is ready.
- Administer a sufficient amount of red blood cell transfusion and fresh frozen plasma (FFP) to replace coagulation factors and platelets.
- Maintain circulatory dynamics, coagulation system, oxygen carrying capacity, body temperature control, and acid-base balance.
- In an emergency, consider blood transfusions such as O-type Rh(-) and blood that matches the cell grouping.

● Administer calcium preparations to address hypocalcemia, and warm blood transfusions and infusions to prevent hypothermia. Note that serum potassium levels rise²⁾ and calcium levels fall³⁾ during a massive blood transfusion.

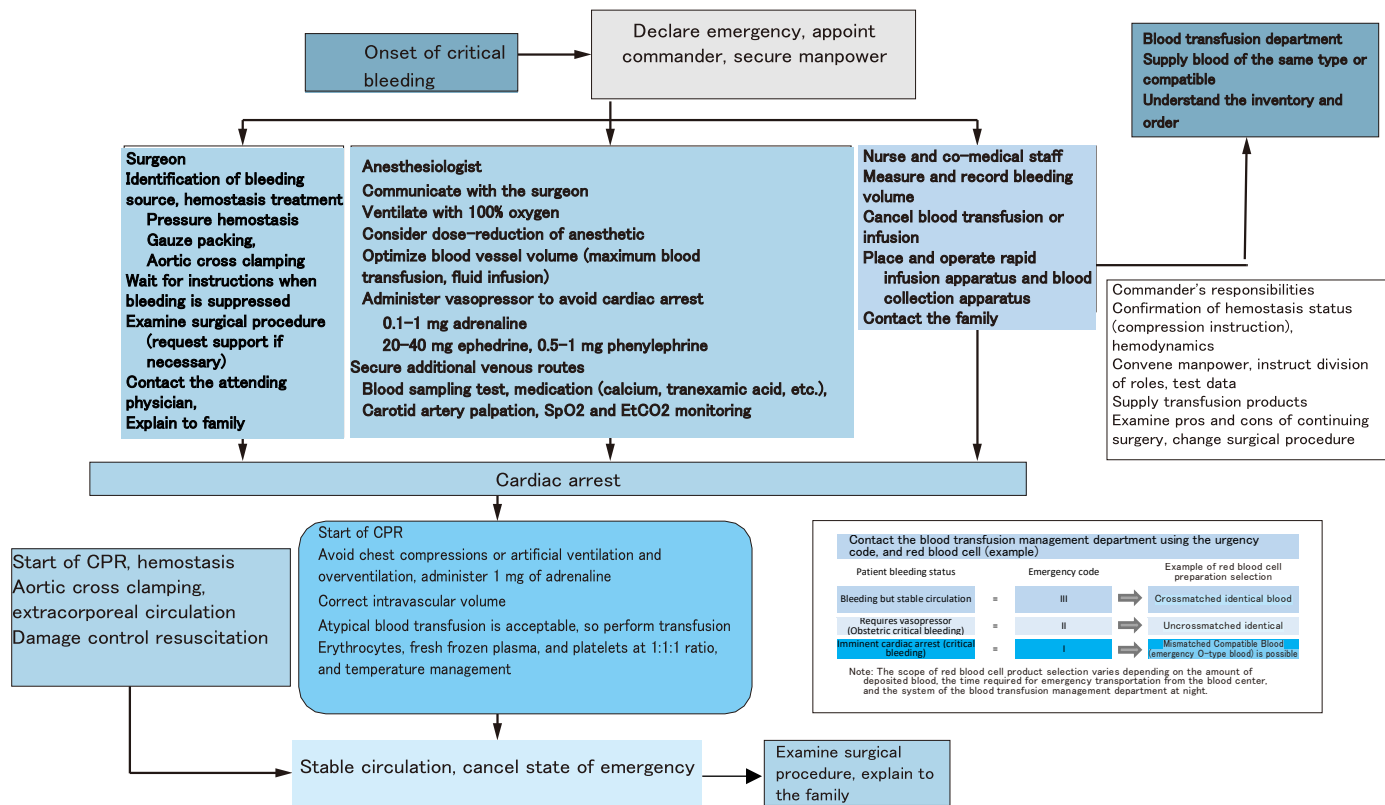


Figure 1 Algorithm for responding to critical bleeding. EtCO₂, end-tidal carbon dioxide; SpO₂, peripheral oxygen saturation; CPR: cardiopulmonary resuscitation

● Prepare and utilize a rapid blood transfusion apparatus and blood collection apparatus. At the time of bleeding, although there is a form of management that attempts to dilute coagulation factors to reduce bleeding with hypotension and small volume infusion, no clear conclusion has been reached in terms of prognosis.^{4,5)}

Surgeon

- Implement first aid measures, such as pressure hemostasis and gauze packing. In many cases, compression can provide hemodynamic stability.
- Examine whether aortic balloon closure is possible.
- Consider whether damage control resuscitation is appropriate.⁶⁾
- Assess the situation and consider explanation to the family.

Nurse

- Receive instructions for blood transfusion orders and be aware of incidents.
- Measure and record the amount of bleeding.
- Assist in blood transfusion and fluid therapy.

- Coordinate contact with the ward and family.
-

Clinical engineer

- Prepare and operate a rapid infusion apparatus and blood collection apparatus.

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4

Right-sided heart failure, pulmonary hypertension, and pulmonary embolism

Learning content

In this section, we will understand the characteristics of intraoperative cardiac arrest, the basics of initial response, and cardiac arrest associated with right-sided heart failure, pulmonary hypertension, and pulmonary embolism.

Characteristics of intraoperative cardiac arrest and basics of initial response

There are four characteristics of intraoperative cardiac arrest are found in many cases: (1) the current and past medical history are known, (2) several monitors have been attached to the patient, (3) those events occur in front of medical staffs, and (4) the effects of anesthetics and surgery must be considered.¹⁾ However, once cardiac arrest occurs, it is essential to start high-quality basic life support immediately, and especially effective chest compressions, and to perform resuscitation based on the cardiac arrest algorithm as a team. Anesthesiologists are suitable as leaders of the resuscitation team leaders.

Cardiac arrest algorithms are based on searching for the causes of cardiac arrest and providing appropriate treatment, including adrenaline bolus, defibrillation for shockable rhythms (ventricular fibrillation [VF], pulseless ventricular tachycardia [VT]).²⁾ When searching for the causes of cardiac arrest, it is important to first exclude bleeding (hypovolemia) and hypoxemia at first, together with while considering anesthetics and surgical problems.³⁻⁵⁾

During the operation, many factors may delay the detection of cardiac arrest, such as artifacts on ECG and pulse oximetry caused by surgical procedures and/or electrocautery, the absence of consciousness, draping, and the like. In general, tachycardia may imply bleeding, tension pneumothorax, cardiac tamponade, or pulmonary embolism, and bradycardia may imply hypoxemia.²⁾ However, those clues may have been modified by the effects of anesthetics. When the patient is intubated, the EtCO₂ monitor is an objective and useful indicator of cardiac arrest or return of spontaneous circulation (ROSC).²⁾

Right-sided heart failure or pulmonary hypotension

In addition to clinical findings, such as jugular vein engorgement, liver swelling, and edema, right ventricular function indicators include the right ventricular fractional area change, distance of movement of the tricuspid annulus, velocity of tricuspid annulus closure, estimated right ventricular pressure based on tricuspid regurgitation velocity, and inferior vena cava diameter. These parameters can all be acquired from echocardiography, more precisely from 3D echocardiography.⁶⁾ Right ventricle is significantly influenced by afterload, so increased pulmonary vascular resistance triggers acute right-sided heart failure.⁷⁾

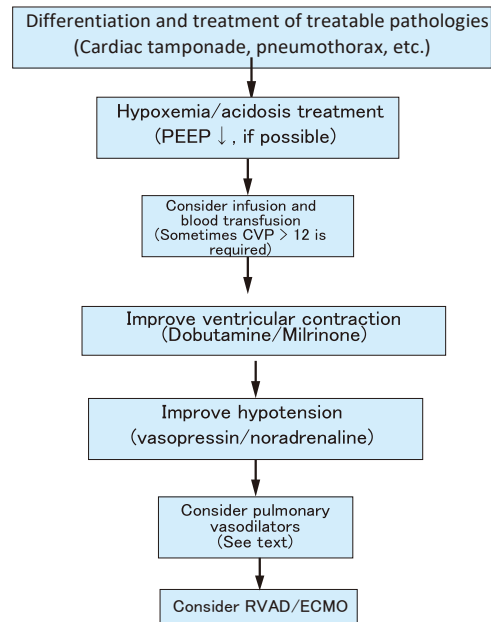


Figure 1 Algorithm for right-sided heart failure

PEEP: positive end-expiratory pressure, CVP: central venous pressure, RVAD: right ventricular assist device, ECMO: extracorporeal membrane oxygenation

In many cases, pulmonary artery catheters and echocardiography are used as intraoperative monitors. Factors that increase pulmonary vascular resistance (hypoxemia, acidosis), cardiac tamponade, tension pneumothorax, pulmonary embolism, auto-positive end-expiratory pressure (PEEP) in obstructive pulmonary disease must be differentiated from other treatable pathologies. Right-sided heart failure or pulmonary embolism can also involve bilateral ventricular failure. Cardiac tamponade in trauma patients, tension pneumothorax in laparoscopic surgery, and pulmonary thromboembolism in high-risk patients with deep vein thrombosis can have serious consequences if detection and treatment are delayed.

If there is no cardiac arrest, start treatment according to the right-sided heart failure algorithm shown in Figure 1. Treatment includes removal of the pathological causes (pathological condition), infusion, improvement of right ventricular contraction, maintenance of coronary perfusion pressure. In some cases, treatment of pulmonary hypertension should be considered with prostaglandin, endothelin antagonist, phosphodiesterase (PDE)-V inhibitor, administration of soluble guanylate cyclase stimulant, and administering nitric oxide (NO).

Extracorporeal circulation (right ventricular assist device, : extracorporeal membrane oxygenation [ECMO]) may be applied.⁸⁾ If cardiac arrest occurs, follow the adult cardiac arrest algorithm.²⁾

Pulmonary embolism

Pulmonary embolism that occurs during surgery includes thromboembolism, air embolism, and fat embolism. However, thromboembolism being is the most common. In many cases, pulmonary thromboembolism comes from deep vein thrombosis.

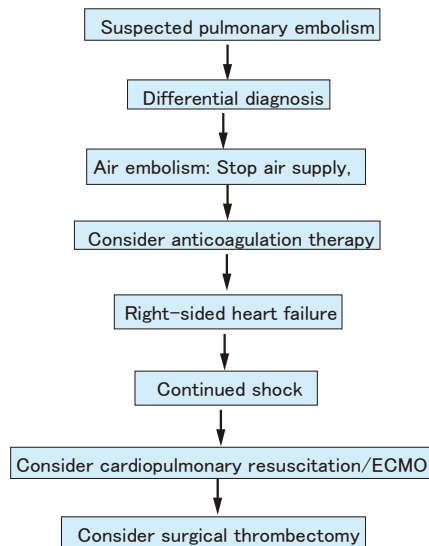


Figure 2 Treatment approach for pulmonary embolism

ECMO: Extracorporeal membrane oxygenation

In thromboembolism, in addition to the mechanical obstruction by the thrombus, humoral factors released from the thrombus and pulmonary artery spasm due to hypoxemia play important roles. Increased pulmonary artery pressure causes not only right-sided failure, but also bilateral ventricular failure. Local bronchospasm along with pulmonary embolism may exaggerate hypoxemia. Even if anticoagulant administration and intermittent pneumatic compression are used, venous thromboembolism (VTE) cannot be completely prevented completely.⁹⁾ Risk assessment of VTE includes determining the risk level (previous VTE, thrombotic predisposition, major surgery for cancer in patients over 40 years, etc.) and risk factors (previous VTE, thrombotic predisposition, lower limb paralysis, advanced age, prolonged immobility, congestive heart failure, etc.).¹⁰⁾ There are no symptoms or signs specific to pulmonary embolism, but rapidly developing hypoxemia, decreased partial end-tidal carbon dioxide pressure (pEtCO₂), hypotension, tachycardia (sometimes arrhythmia or bradycardia), and increased airway pressure due to bronchospasm may occur.

To start early treatment, it is necessary to suspect pulmonary embolism and promptly perform differential diagnosis promptly. Echocardiography is useful for diagnosis during surgery or when hemodynamics are unstable.

Air embolism can occur during laparoscopic surgery, surgery in the sitting position, central vein puncture, or minimally invasive procedures under gas insufflation. If air embolism is suspected, it is essential to stop insufflation immediately, cover the surgical field with physiological saline, and lower the patient's head.

In acute pulmonary thromboembolism (PTE), systemic management at an early stage of onset (within 1 hour of onset) can improve prognosis.¹⁰⁾ Management includes not increasing intrapleural pressure (low tidal volume, PEEP adjustment), avoiding excessive fluid load, maintaining hemodynamics with dobutamine and noradrenaline, anticoagulant therapy (unfractionated heparin 80 units/kg → 18 units/kg/h, activated partial thromboplastin time (APTT) confirmation), veno-arterial (V-A) ECMO for circulatory collapse, and considering surgical thrombectomy.¹⁰⁾ If cardiac arrest occurs, follow the adult cardiac arrest algorithm.¹⁾ The treatment approach for pulmonary embolism is shown in Figure 2.

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5

Left-sided heart failure due to intraoperative cardiogenic shock

Learning items

In this section, we will learn about the pathology of cardiogenic shock, its initial evaluation methods, and the responses to acute coronary syndrome and myocardial ischemia, as well as those to left-sided heart failure due to cardiogenic shock.

Pathology

Cardiogenic shock is a left ventricular pumping disorder associated with myocardial infarction or myocardial injury.¹⁾ Decreased left ventricular contraction reduces cardiac output (CO) and stroke volume (SV), resulting in hypotension, which further lowers coronary perfusion pressure, augments myocardial ischemia, and exacerbates myocardial damage. Furthermore, left ventricular end-diastolic pressure (LVEDP) increases due to left ventricular diastolic dysfunction, resulting in pulmonary congestion and hypoxemia, which exacerbates myocardial ischemia.

Intraoperative acute coronary syndrome and myocardial ischemia

Myocardial ischemia is the most common cause of cardiogenic shock, and acute coronary syndrome (ACS), which is the most severe form, requires a prompt response. In the initial medical treatment algorithm for ACS²⁾ in a general emergency field, chest discomfort, suggesting ischemia, is the first symptom. The initial medical treatment starts from there, but since the patient cannot speak while under general anesthesia, this condition will initially be suspected based on vital signs. Initial treatment must not be delayed, since vital signs will be continuously observed. Note the following:

- Patients with episodes of myocardial ischemia during preoperative interviews and high-risk patients, such as those with severe aortic stenosis, should be managed systemically to stabilize intraoperative vital signs.
- The depth of anesthesia should not be extremely shallow during surgery. Prevent rapid changes in hemodynamics due to insufficient analgesia or after a sudden major surgical invasion. If there is a large hemodynamic change, closely observe vital signs.
- Suspect myocardial ischemia if tachycardia, bradycardia, or hypotension with ST changes on the electrocardiogram (ECG) are observed. In general, lead II, which is used in intraoperative monitoring, is mainly useful for evaluating the right coronary artery region. Other coronary artery assessments require lead I and a 5-pole ECG.

- If ST changes or myocardial ischemia is suspected, request support or consult with the cardiology department.
- If myocardial ischemia is suspected, perform 12-lead ECG.
- If possible, simultaneously and immediately evaluate left ventricular wall motion abnormality, valve abnormality, decreased ventricular contraction, and left ventricular outflow obstruction (based on left ventricular ejection fraction, etc.), as well as decreased left ventricular diastolic function, cardiac tamponade (based on transmitral blood flow waveform, etc.) using transesophageal echocardiography or transthoracic echocardiography.
- Shock caused by left-sided heart failure is caused not only by cardiogenic shock but also by hypovolemia, cardiac tamponade, and tension pneumothorax; as such, differential diagnosis is required.
- ST elevation or a new left bundle branch block is observed on 12-lead ECG indicate ST-elevation myocardial infarction (STEMI). Immediately contact the cardiologist to complete surgery as soon as possible and prioritize reperfusion therapy.
- ST depression or T-wave inversion on 12-lead ECG indicate an unstable angina (UA) or non-STEMI (NSTEMI). Consult with a surgeon and cardiologist to decide on discontinuation, reduction, or continuation of the surgery and consider management in the intensive care unit (ICU) after surgery.
- ST-T changes that are normal or difficult to determine on 12-lead ECG indicate medium or low-risk UA. Consult with a surgeon and cardiologist to decide on discontinuation, reduction, or continuation of the surgery and consider management in the ICU after surgery.
- Avoid excessive oxygen administration during surgery. Oxygen saturation should be at 90% for STEMI and 94% for NSTEMI.
- Antiplatelet drugs, such as aspirin, and anticoagulants, such as unfractionated heparin, may promote bleeding from the surgical field; therefore, consult with the surgeon and cardiologist.
- Prophylactic administration of nitroglycerin is not effective.³⁾ Its use in patients taking therapeutic agents for hypotension and erectile dysfunction is contraindicated.²⁾
- Morphine should be administered intravenously or infused in STEMI patients.²⁾ In patients with suspected NSTEMI, intravenous administration of morphine has been reported to increase mortality and myocardial infarction incidence; thus, its administration should be performed after careful consideration.⁴⁾
- Non-steroidal anti-inflammatory drugs (excluding aspirin) may exacerbate ACS and should not be used.²⁾

Response to left-sided heart failure due to cardiogenic shock

The response to left-sided heart failure due to cardiogenic shock is shown in Figure 1.⁵⁾ Invasive monitors, such as echocardiography and pulmonary artery catheters, guide the treatment of left ventricular dysfunction. Hypovolemia induces shock in patients with decreased left ventricular function and enhances left-sided heart failure; thus, it is necessary to evaluate and optimize hypovolemia before starting drug treatment. For hypotensive patients with left ventricular heart failure and full circulating blood volume, use drugs with positive inotropic and afterload-reducing effects.^{1,5,6)} In patients with severe left ventricular diastolic dysfunction, phosphodiesterase (PDE) III inhibitors, such as milrinone, improve cardiac output, reduce the degradation of cAMP, and increase protein kinase A (PKA) activity.

Activation of PKA induces increased cardiac contraction, myocardial relaxation, and vascular smooth muscle relaxation. These effects act independently of down-regulated β -adrenergic receptors in patients with cardiac failure, resulting in increased cardiac contraction and cardiac output.

In recent years, extracorporeal life support devices such as intra-aortic balloon pumping, ventricular assist device, and extracorporeal membrane oxygenation have come to be used for left-sided heart failure, right-sided heart failure, and cardiac arrest in hospitalized patients, leading to a high probability of recovery.^{7,8)}

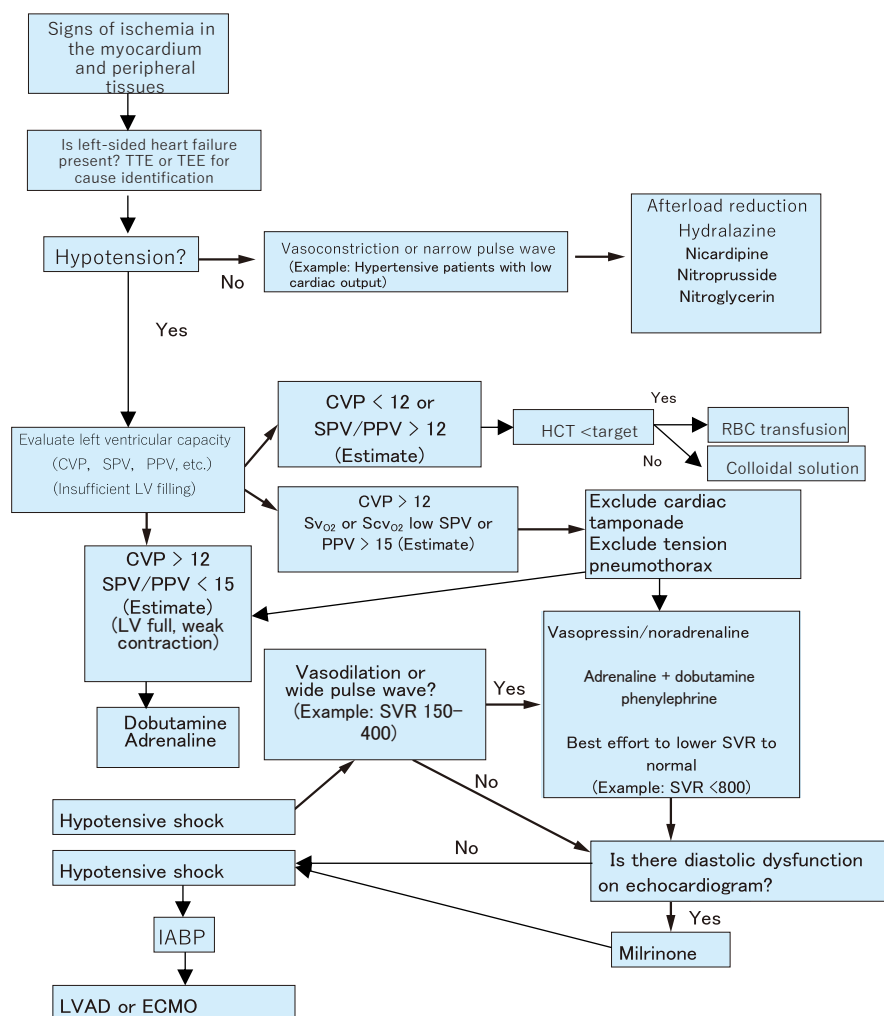


Figure 1 Treatment algorithm for left-sided heart failure due to cardiogenic shock

CVP: central venous pressure (mmHg) ECMO: extracorporeal membrane oxygenation, Hct: hematocrit, IABP: intra-aortic balloon pump, LV: left ventricular, LVAD: left ventricular assist device, PPV: pulse pressure variation (%), ScvO₂: central venous oxygen saturation, SPV: systolic pressure variation (%), SvO₂: mixed venous oxygen saturation, SVR: systemic vascular resistance (dyne · sec⁻¹ · cm⁻⁵), TEE: transthoracic echocardiography, TTE: transthoracic echocardiography [Modified and reprinted from Morita 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: 876–88]

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6

Cardiac arrest due to local anesthetic toxicity

Learning items

Nerve blocks with local anesthetics are increasingly used, which may increase the chances of local anesthetic toxicity. For the response to local anesthetic toxicity, refer to the practical guide created by the Japan Society of Anesthesiologists.¹⁾

Preparation

- At the time of peripheral nerve block, attach ECG leads and pulse oximeter and measure blood pressure at 5-minute intervals.
- Prepare a fat emulsion in the operating room to address local anesthetic toxicity.
- During peripheral nerve block, the appropriate drug dose should be used in consideration of the patient's body weight.
- The infusion should be administered in small doses after the aspiration test.

Response

If the following findings, suggesting local anesthesia poisoning, are observed, follow steps (1) through (9) (See Figure 1): numbness of tongue or lips, agitation, dizziness, difficulty in articulation, visual or hearing impairment, light-headedness, convulsions, impaired consciousness, respiratory arrest, and cardiac arrest.

- (1) Discontinue local anesthetic administration
- (2) Request support
- (3) Initiate airway management, 100% oxygen administration, and, if necessary, normal cardiopulmonary resuscitation (CPR)
- (4) Secure venous route
- (5) Administer anticonvulsants (benzodiazepines are recommended, propofol requires attention due to hypotension)
- (6) Start CPR as needed
 - Administer adrenaline as usual
 - Cardiac arrest due to local anesthetic toxicity may take time to resuscitate, but should be treated without quitting.²⁾

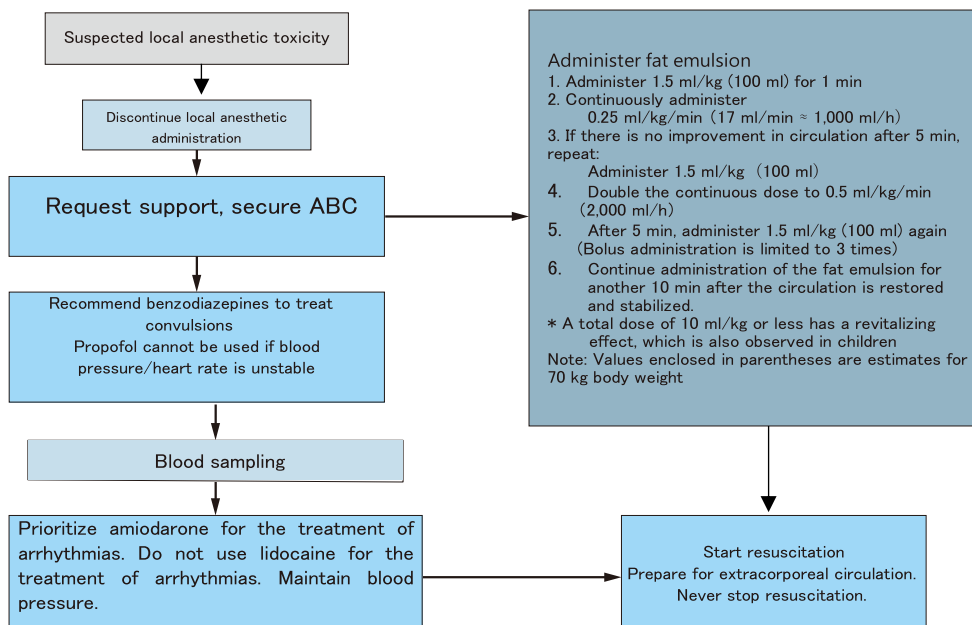


Figure 1 Algorithm for responding to local anesthetic toxicity

ABC: Airway, Breathing, Circulation

(7) Response to arrhythmia

- Give priority to amiodarone
- Do not use lidocaine

(8) Administer fat emulsion

- Administer 100 ml for 1 min, then 1000 ml/h
- After 5 min, add 100 ml if there is no improvement in circulation (up to 3 bolus doses).
- Do not administer propofol as a substitute.

(9) If the patient does not respond or barely responds to the treatment, establish extracorporeal circulation at the same time.

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7

Cardiac arrest in patients with malignant hyperthermia

Learning items

Refer to the guidelines created by the Japan Society of Anesthesiologists for the algorithm for responding to malignant hyperthermia.¹⁾ Although malignant hyperthermia is a rare disease, it progresses rapidly from onset, and appropriate measures are required. When malignant hyperthermia is suspected, it is important to administer dantrolene at an early stage.

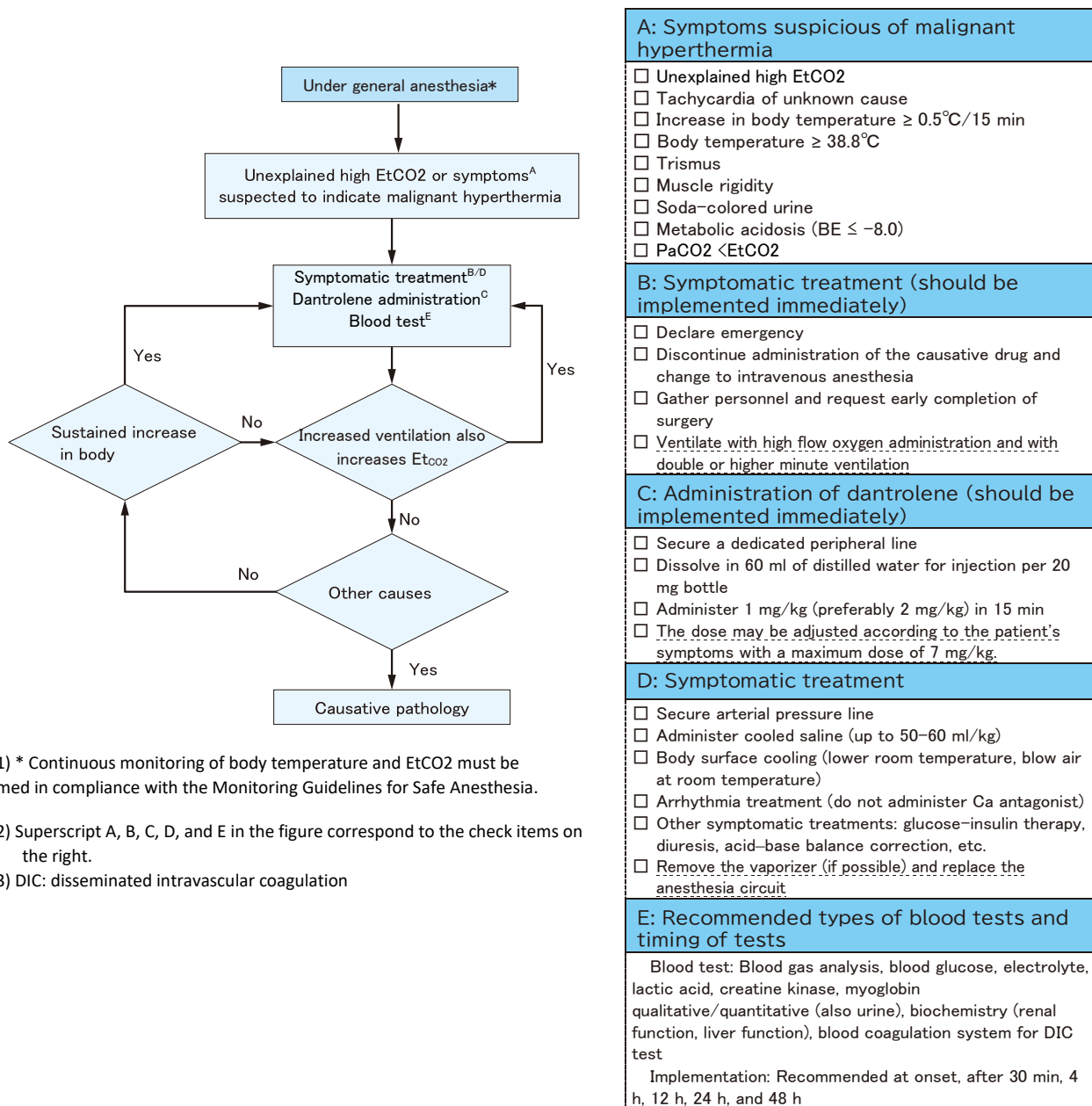
General notes

[Preparation] Identify high-risk patients. Check the stock quantity and expiration date of dantrolene in the hospital. Establish a network of pharmaceutical companies, drug wholesalers, nearby hospitals, etc., if the supply of dantrolene is insufficient.

In the event of cardiac arrest or crisis, consider the following steps in addition to normal cardiopulmonary resuscitation (CPR) (Figure 1).

- (1) Discontinue the causative volatile anesthetic and change to intravenous anesthetic
- (2) Wash out the inside of the circuit with 100% oxygen at 10 l/min or more.
- (3) Double the minute ventilation
- (4) Ask the surgeon to stop the surgery early
- (5) Administer non-depolarizing muscle relaxants
- (6) Initiate the early administration of dantrolene
 - (i) Dissolve 20 mg dantrolene in 60 ml of distilled water in a single bottle for injection (this is quite difficult to dissolve, but dissolves easily when the solution is warmed to about 40°C)²⁾
 - (ii) Administer 1–2 mg/kg within 15 minutes
- (7) Repeat the administration using decreased body temperature, decreased end-tidal CO₂ (EtCO₂), improvement of myotonia, and decreased heart rate as indicators
- (8) Administer cold physiological saline to lower body temperature (50–60 ml/kg)
- (9) To cool the body surface, expose the body surface, blow air, and sprinkle water (stop cooling when the core temperature reaches 38°C).
- (10) Symptomatic treatment

- i. Arrhythmia treatment: Calcium channel blockers should not be administered (concurrent administration with dantrolene is reported to cause cardiac arrest)



(Note 1) * Continuous monitoring of body temperature and EtCO₂ must be performed in compliance with the Monitoring Guidelines for Safe Anesthesia.

(Note 2) Superscript A, B, C, D, and E in the figure correspond to the check items on the right.

(Note 3) DIC: disseminated intravascular coagulation

Figure 1 Treatment procedure for malignant hyperthermia. EtCO₂, end-tidal CO₂; PaCO₂, partial pressure of CO₂;

- ii. Hyperkalemia treatment: ① Calcium (maximum: 2 g of calcium chloride, 3 g of calcium gluconate)/② Glucose–insulin therapy (50 ml of 50% glucose solution is administered to 10 units of insulin in 1 h)/③ Diuretic (administration of 0.5–1.0 mg/kg of furosemide)
- iii. High creatinine kinase, high myoglobinemia: ① Administer sodium bicarbonate at 1 mEq/kg/h for alkalinization of urine/② Administer up to 4,000 units of haptoglobin (estimate 2 units/kg).
- iv. Metabolic acidosis: Administer 1–2 mEq/kg of sodium bicarbonate
- (11) Insertion of open arterial pressure line

(12) Blood sampling (arterial blood gas, acid-base balance, lactic acid, electrolytes, blood glucose, creatine kinase (CK), myoglobin qualitative/quantitative) and urine test: Ideally, tests should be performed at onset and after 30 min, 4 h, 12 h, 24 h, and 48 h.

(13) Once the symptoms have improved, recurrence is possible; thus, careful management in an intensive care unit (ICU), etc., is necessary

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8

Tension pneumothorax and cardiac tamponade

In laparoscopic surgery, sudden changes in vital signs are usually small. However, in the event of cardiac arrest or other crises, such as hypotension, oxygen desaturation, or decreased tidal volume accompanied by sudden tachycardia, it is also necessary to notify the surgeon of an emergency, call for support because responding alone is difficult, and prepare a defibrillator as a treatment for cardiac arrest according to the algorithm shown in the overview. When you judge chest compression is necessary, you should start it without a hesitation. Next, consider the possible cause, while performing ventilation with 100% oxygen, fully open the infusion routes, and recheck vital signs, which will lead to a quick response. Particularly, in obstructive shocks, such as tension pneumothorax and cardiac tamponade, there is a high risk that the shock will rapidly worsen and lead to cardiac arrest, but there is also a high possibility of improvement when appropriate measures are performed. Team response training, such as simulation training, is important for appropriate management, and the role of the anesthesiologist as a team leader is of the utmost importance.

Tension pneumothorax

1) General notes

Although there have been many case reports in Japan,¹⁻⁶⁾ it is necessary to promptly diagnose and treat these cases in order to save the patient's life.⁷⁾ In recent years, there has been an increase in the number of surgeries involving pressurized endoscopy using a laparoscope, retroperitoneoscope, thoracoscope with intrapleural positive pressure, etc.⁸⁾

2) Cause, diagnosis, and treatment

(a) Cause

In the analysis of the causes of tension pneumothorax onset during the perioperative period,⁷⁾ traumatic causes, including barotrauma, were shown to be the most common (66%). This may be the reason for the rate of onset of 24.4% on both sides.

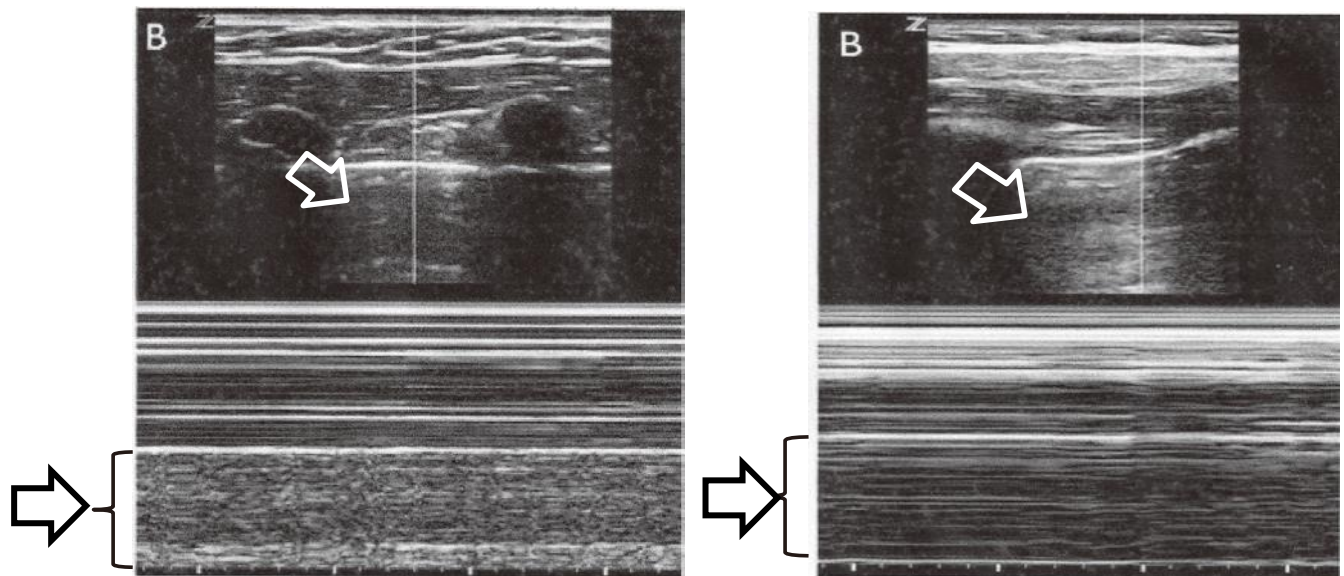
(b) Diagnosis

Table 1 summarizes the findings obtained for tension pneumothorax; however, in recent years, pulmonary echocardiography by transthoracic ultrasonography (**Figure 1**)^{9,10)} has been considered useful.

The main causes of sudden changes in intubated patients in response to hypoxemia and hypotension are summarized by the DOPE acronym, explained below. Capnography is often performed during anesthesia, and much information can be extracted from its waveform.¹¹⁾

Table 1 Symptoms of tensions pneumothorax

- Decrease in tidal volume
- Hypotension with low pulse pressure
- Tachycardia (early bradycardia as hypoxia progresses)
- ECG change: clockwise or counterclockwise electrical axis rotation
- Percussion sound
- Engorgement of jugular vein
- Laterality in chest wall movement
- Rapid rise in airway pressure
- Hypoxia
- Tracheal deviation
- Attenuation of breath sound during auscultation



(a) Normally, the movement of the pulmonary pleura can be observed with respiration, which is referred to a seashore sign in M mode echocardiography.

(b) Since there is no lung parenchyma in pneumothorax, it takes on a barcode appearance in M mode echocardiography.

Figure 1 Pulmonary echocardiography

[Reprinted from Cavayas YA, Girard M, Desjardins G, et al. Transesophageal lung ultrasonography: A novel technique for investigating hypoxemia. *Can J Anaesth* 2016;63:1266—76]

DOPE

- Displacement of endotracheal tube
- Obstruction in the endotracheal tube
- Tension Pneumothorax
- Equipment failure

(c) Treatment

As a general procedure for dealing with DOPE, it is recommended to check the waveform by capnography while evaluating the relationship between airway pressure and ventilation volume by manual ventilation. However, other forms of ventilation can also be used, since drugs need to be prepared.

In the treatment of pneumothorax, it is important to attempt oxygenation first and promptly perform deaeration from the thoracic cavity. The recommended first aid procedure is deaeration with a vertical puncture approach from the upper edge of the 3rd rib at the midline of the 2nd intercostal space. However, the approach and procedure can be difficult in cases

where the exact position cannot be identified because of changes in the organ position due to the surgical position or covering. Furthermore, delayed diagnosis leads to poor prognosis, and the highest priority must be given to emergency treatment.

Deaeration from the mediastinum should also be considered during thoracotomy. It is advisable to use surgical instruments, if available. Furthermore, even if a chest drain has been inserted, tension pneumothorax should not be excluded from the diagnosis.¹²⁾ Tension pneumothorax caused by air inflow from the drain after resumption of spontaneous respiration has also been reported;¹³⁾ hence, due care is required to monitor bleeding and lung injury after treatment. Due to time constraints, it is very important to carry out simulation training on how to deal with tension pneumothorax in order to improve prognosis.¹⁴⁾

Cardiac tamponade

1) General notes

Cardiac tamponade occurs due to various causes, as shown in [Table 2](#). The symptoms are shown in [Table 3](#), but many cases do not manifest the so-called Beck triad (jugular vein distension, hypotension, and attenuation of heart sound). The cause is often postoperative or related to catheter operation.^{15,16)}

Treatment is performed according to the algorithm for cardiac arrest (see [Figure 2 \[p. 8\]](#) in [Chapter 1 Overview: Introduction](#)), but the possibility of onset should always be kept in mind. Cardiac tamponade caused by pneumoperitoneum has also been reported.¹⁷⁾

2) Diagnosis and treatment

(a) Diagnosis

Diagnosis by transthoracic wall or transesophageal echocardiography is useful.^{15,18)}

Table 4 Determining treatment policy through scoring		
1 Cause	Malignant neoplasm	2
	Tuberculosis	2
	Recent radiotherapy	1
	Recent viral infection	1
	Persistent collection of pericardial effusion	1
	End-stage chronic renal failure	1
	Immunodeficiency	1
	Hyper/Hypothyroidism	-1
	Systemic immune disease	-1
2 Clinical symptoms	Dyspnea or tachypnea	1
	Orthopnea	3
	Hypotension (Systolic blood pressure < 95 mmHg)	0.5
	Progressive sinus tachycardia (no drugs affecting heart rate, hypothyroidism, uremia)	1
	Oliguria	1
	Paradoxical pulse (> 10 mmHg)	2
	Pericardial pain	0.5
	Pericardial friction rub	0.5
	Rapidly worsening symptoms	2

3 Imaging results	Slow disease progression	-1
	Cardiac enlargement on chest X-ray	1
	ECG changes	0.5
	ECG decrease	1
	Pericardial effusion collection (> 2 cm: diastolic)	3
	Moderate collection (1–2 cm: diastolic)	1
	Slight collection (< 1 cm) no trauma	-1
	Collapse of the right atrium (more than 1/3 of the cardiac cycle)	1
	Inferior vena cava > 2.5 cm, collapsed for more than 50% of the cardiac cycle	1.5
	Collapse of the right ventricle	2
	Collapse of the left atrium	1
	Mitral valve or tricuspid valve: Respiratory flow rate fluctuation	1
	Swinging heart	1

If the sum of scores in sections 1–3 is at least 6, perform pericardial puncture unless it is not applicable

[Reprinted from Yerdel MA, Sen O, Zor U, et al. Cardiac tamponade as a life-threatening complication of laparoscopic antireflux surgery :The real incidence and 3D anatomy of a heart injury by helical tacks. J Laparoendosc Adv Surg Tech A 2018;28:1041–6]

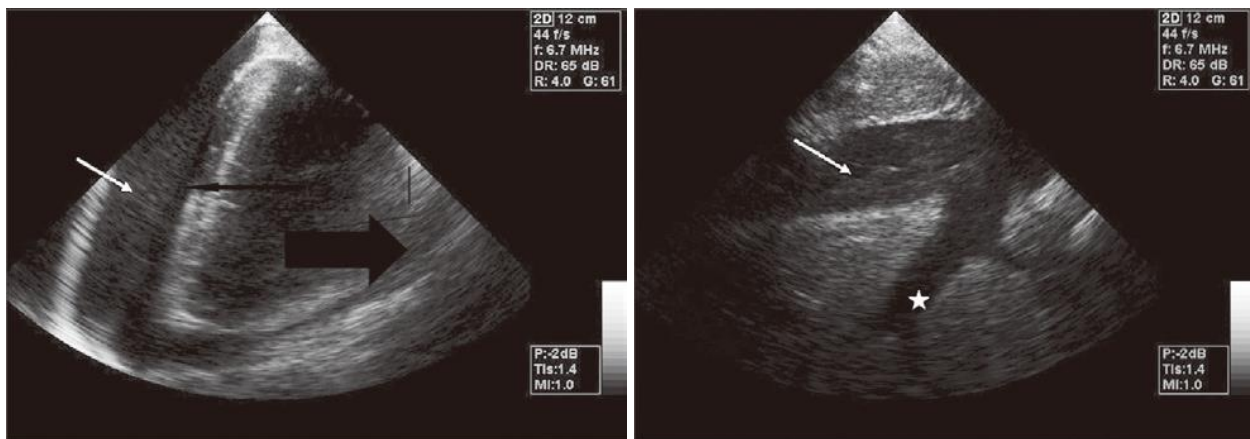
Table 2 Causes of cardiac tamponade

- | | |
|---|--|
| <ul style="list-style-type: none"> • Central venous catheter puncture, insertion, removal • Pulmonary artery catheter puncture, insertion, removal • Aortic dissection • Pericarditis | <ul style="list-style-type: none"> • Trauma • Uremia due to renal failure • Chest compressions during CPR • Malignant neoplasm |
|---|--|

Table 3 Symptoms of cardiac tamponade

- | | |
|--|--|
| <ul style="list-style-type: none"> • Hypotension • Tachycardia or PEA • Engorgement of jugular vein | <ul style="list-style-type: none"> • Decrease in pulse pressure • Attenuation of heart sound • Absent pulse even with CPR |
|--|--|

PEA: Pulseless electrical activity, CPR: Cardiopulmonary resuscitation



(a) →: Pericardial effusion collection

(b) →: Inferior vena cava dilation, ☆: Hepatic vein dilation

Figure 2 Diagnosis by transesophageal echocardiography

[Reprinted from McHugh SM, Wang X, Sullivan EA. Diagnosis of cardiac tamponade with transesophageal echocardiography following the induction of anesthesia for suspected testicular torsion. Ann Card Anaesth 2015;18:449–52]

Table 4

Apart from collection of pericardial effusion and dilation of the inferior vena cava and hepatic veins (Figure 2)

collapse of the right atrium
collapse of the right ventricle
decreased blood inflow to the left ventricle
delayed opening of the mitral valve, decreased E wave velocity of the mitral valve
prolonged isovolumic relaxation time

(b) Treatment

Drainage is performed blindly or under echocardiographic guidance, but is dangerous.¹⁹⁾ A method has been proposed in which the treatment policy is determined by a scoring system based on pathogenesis, clinical symptoms, and imaging results (Table 4).²⁰⁾

Severe chest trauma, aortic Stanford type A dissection, rupture of the ventricular free wall, and iatrogenic bleeding that cannot be controlled are all indications for emergency surgery.

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9

Cardiac arrest due to anaphylaxis

Learning content

In this section, symptoms and diagnosis of anaphylaxis, frequency of cardiac arrest due to anaphylaxis, mechanism of anaphylaxis, perioperative drugs that predispose to anaphylaxis, and response in the event of anaphylaxis will be explained.

General notes

When anaphylaxis occurs, the best efforts must be exerted to avoid cardiac arrest through prompt diagnosis and appropriate responses. The response in the event of cardiac arrest is similar to the response for cardiac arrest due to other causes.

1) Symptoms of anaphylaxis

The main symptoms of anaphylaxis are respiratory, circulatory, cutaneous, gastrointestinal, and neurological. However, gastrointestinal and neurological symptoms rarely manifest during surgery. Respiratory symptoms include increased airway pressure and decreased arterial oxygen saturation. Cardiovascular symptoms include decreased blood pressure, tachycardia, and arrhythmia. Skin symptoms include erythema and wheals. According to a recent large-scale epidemiological study in the United Kingdom (about 3.13 million subjects anesthetized in 2016), the initial symptoms of anaphylaxis are hypotension (46%), bronchospasm (18%), tachycardia (9.8%), decreased arterial oxygen saturation (4.7%), and bradycardia (3%).¹⁾

On the other hand, in terms of the incidence of symptoms that appeared during the course of anaphylaxis, regardless of frequency, the most common were hypotension (100%), rash (56%), bronchospasm or increased airway pressure (48%), tachycardia (46%), cyanosis or decreased arterial oxygen saturation (41%), and capnography waveform abnormalities (33%). Although rashes are a well-known symptom of anaphylaxis, it should be noted that no rash appeared in nearly half of the cases.

2) Diagnosis of anaphylaxis

The clinical diagnostic criteria for anaphylaxis are generally set by multiple academic societies in the United States.²⁾ Since anaphylaxis during surgery often develops after administration of a drug, it is judged that the patient was exposed to a substance that may be an allergen. In this case, anaphylaxis can be diagnosed if two or more skin symptoms, respiratory symptoms, cardiovascular symptoms, and digestive symptoms appear.

3) Cardiac arrest due to anaphylaxis

In the UK study mentioned above, 40 (15%) of the 266 patients who developed severe grade 3–5 anaphylaxis during the perioperative period had cardiac arrest.¹⁾ Thirty-one (78%) of 40 patients who had cardiac arrest were saved. In most cases (81%), the onset of cardiac arrest happened at the time

of anesthesia induction or before surgery. The electrocardiogram (ECG) waveforms during cardiac arrest were pulseless electrical activity (PEA) in 34 patients, pulseless ventricular tachycardia (VT) or ventricular fibrillation (VF) in 4 patients, and asystole in 2 patients. Arrhythmia appeared in 6 patients prior to cardiac arrest. Of the cases that had arrhythmia, 4 had bradycardia, and 2 had VF. Of the 10 patients who died of anaphylaxis during the study period, 9 had an intraoperative cardiac arrest. All ECG waveforms of the deceased patients were PEA.

Two of these 9 patients had bradycardia prior to cardiac arrest. Five of the 10 deceased patients had a history of coronary artery disease. Six patients were taking beta-blockers and angiotensin converting enzyme inhibitors (ACE-I).

In a study conducted in France from 2011 to 2012, 41 (8.4%) of 489 patients with immunoglobulin E (IgE)-related anaphylaxis (discussed below) had cardiac arrest.³⁾ This study included patients with grade 1–5 anaphylaxis, including those with mild illness, which may have led to a lower incidence of cardiac arrest compared with the UK study.

In a survey of incidental events conducted by the Japanese Society of Anesthesiologists from 2009 to 2011, 237 cases of anaphylactic shock were reported, of which 13 cases (5.5%) involved cardiac arrest. Of the 211 cases of cardiac arrest caused by intraoperative pathology, anaphylactic shock accounted for 6.2%. Thus, although cardiac arrest is not uncommon in severe anaphylaxis, anesthesiologists can respond from the bedside with the patient under full monitoring, saving lives in many cases. However, patients with a history of coronary artery disease or who are taking beta-blockers or ACE-I may be unresponsive to resuscitation, resulting in death. As such, due care is required. Furthermore, there is a high risk of death if the ECG waveform at cardiac arrest is PEA.

4) Mechanism of anaphylaxis

Anaphylaxis symptoms occur due to chemicals released from mast cells and basophils (histamine, leukotriene C4, prostaglandin D2, tryptase, chymase, renin, platelet activating factor, cytokines, chemokines, etc.). Several different pathways are involved in the stimulation of mast cells and basophils by allergens.⁴⁾ Such pathways are broadly divided into those that are related to an immunological mechanism and those that are not. Anaphylaxis caused by immunological mechanisms is further divided into those that depend on IgE and those that do not. IgG and complement cause IgE-independent anaphylaxis. On the other hand, non-immunological mechanisms involve a pathway wherein drugs directly activate mast cells and basophils.

5) Kounis syndrome

When anaphylaxis occurs, the heart is both a source of inflammatory mediators and a target organ. It has been reported that mediators released from activated mast cells and platelets induce coronary artery spasm. The acute coronary syndrome caused by such an allergic reaction is called Kounis syndrome.⁵⁾ In some cases, coronary artery spasm causes erosion in the preexistent coronary plaque, causing its rupture and leading to acute coronary syndrome. It is difficult to prove coronary artery spasm due to an allergic mechanism retrospectively, and the exact frequency of its occurrence is unknown. In some cases, it may be treated as an unexplained intraoperative cardiac arrest. Although awareness of Kounis syndrome is low in Japan, there have been some recent reports of Kounis syndrome developing during surgery.⁶⁾

6) Drugs that cause anaphylaxis

Japanese data on drugs that cause anaphylaxis during the perioperative period are scarce. Based on overseas data, it appears that many muscle relaxants and antibacterial drugs can cause anaphylaxis.^{1,3)} Muscle relaxants and antibacterial drugs are often given before the start of surgery. In particular, the onset of cardiac arrest due to anaphylaxis should be kept in mind from the introduction of anesthesia to the start of surgery. In Japan, data indicate that a large number of anaphylaxis cases are caused by muscle relaxant antagonists.^{7,8)} If this is the case, a new tracheal intubation may be required after the onset of symptoms after extubation. In addition, the onset of symptoms may occur in the recovery room or ward, so special attention is required.⁹⁾

7) Treatment of anaphylaxis

Figure 1 shows the algorithm for responding to an event of anaphylaxis during surgery.⁹⁾ If symptoms of anaphylaxis are suspected during surgery, it is important to respond early and avoid cardiac arrest. Treatment for cardiac arrest after the onset of anaphylaxis is no different from the treatment for cardiac arrest due to other causes.

8) Administration of adrenaline

Adrenaline is the first-line drug treatment for anaphylaxis. An analysis of deaths due to anaphylaxis in Canada revealed that only 22 (24%) of the 92 cases received adrenaline.¹⁰⁾ In addition, the Japan Medical Safety Research Organization analyzed 12 cases of death related to anaphylaxis caused by injectable drugs and reported that adrenaline was administered before cardiac arrest in only one case.¹¹⁾ Many physicians consider adrenaline a drug used during cardiopulmonary resuscitation (CPR) and may be reluctant to administer it before cardiac arrest. However, delays in adrenaline administration may lead to death, and if anaphylaxis is suspected, adrenaline should be administered without hesitation.^{10,11)}

Intramuscular injection of adrenaline is recommended for anaphylaxis outside the operating room.¹²⁾ On the other hand, many guidelines recommend intravenous adrenaline injection for anaphylaxis that occurs in the operating room.¹³⁻¹⁶⁾

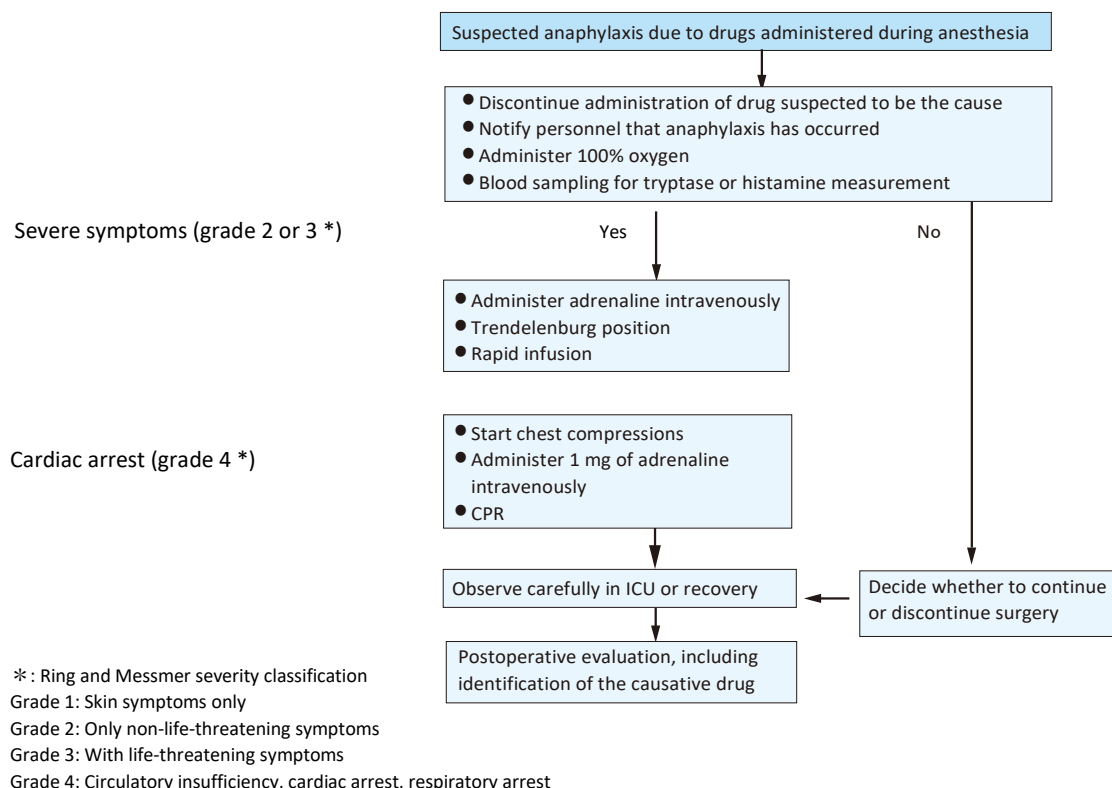


Figure 1 Response in the event of intraoperative anaphylaxis

[Modified and reprinted from Takazawa T, Mitsuhashi H, Mertes PM. Sugammadex and rocuronium-induced anaphylaxis. J Anesth 2016;30:290–7]

Table 1: Recommended dose of adrenaline for adults in foreign guidelines (excerpt from intravenous injection only)			
Guideline	Intravenous bolus administration	Intravenous continuous administration	Reference number
AAGBI	50 µg (initial dose)	Consider when a single dose is ineffective	13
	Repeated administration in severe cases		
EAACI	10–20 µg (grade 2 *)	0.05–0.1µg/kg/min	14
	100–200 µg (grade 3 *)		
SSAI	10–50 µg (mild-moderate)	0.05–0.1µg/kg/min	15
	100–1000 µg (circulatory collapse)		
ANZCA	20 µg (grade 2 *)	3 µg/min	16
	100–200 µg (grade 3 *)	Maximum 40 µg/min	

AAGBI: Association of Anaesthetists of Great Britain and Ireland
 EAACI: European Academy of Allergy and Clinical Immunology
 SSAI: The Scandinavian Society of Anesthesiology and Intensive Care
 ANZCA: The Australian and New Zealand College of Anaesthetists
 *: Ring and Messmer severity classification (see Figure)

The possible reasons for this are the following: 1. In many cases, a route for intravenous injection has already been secured in the operating room. 2. Since a monitor is attached, it is possible to administer adrenaline in small

doses while checking vital signs. The adrenaline dose for cases that do not lead to cardiac arrest vary depending on the guidelines and are shown in [Table 1](#).

Based on the guidelines of other countries, the Japanese Society of Anesthesiologists plans to suggest the following recommended doses of adrenaline in the Practical Guide to Anaphylaxis.

- Hypotension: Administer 0.2 µg/kg adrenaline intravenously.
- Circulatory collapse: Administer 0.05–0.3 mg adrenaline intravenously.
- If no venous route is available, administer 0.3 mg (0.01 mg/kg for children) adrenaline intramuscularly.
- Administer additional doses as needed. If repeated doses are required, start continuous intravenous administration.

Corticosteroids and antihistamines are frequently used in the treatment of anaphylaxis, but these drugs have no immediate effect and are positioned as second-line treatments.¹³⁻¹⁶⁾ Therefore, corticosteroids and antihistamines should not be prioritized in cases where adrenaline is required.

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10

Cardiac arrest in non-supine position

General notes

Patient monitors are often attached during the perioperative period, and staff members are often nearby. Upon recognizing cardiopulmonary arrest, resuscitation should be started as soon as possible without hesitation, but perioperative cardiopulmonary resuscitation (CPR) treatment is often restricted by surgical instruments and posture. The procedure is essentially performed according to the American Heart Association (AHA) basic life support and advanced cardiovascular life support algorithms (see Figure 2 [p. 8] in Chapter 1 Overview: Introduction).

High quality CPR treatment

High quality CPR treatment is defined by the AHA guidelines as follows:

1. Compress at a rate of 100–120 times/min
2. Compress the chest by 5–6 cm in adults and approximately 1/3 of the chest wall thickness in children and infants (5 cm and 4 cm, respectively)
3. Allow complete chest recoil after each compression
4. Avoid excessive ventilation
5. Minimize interruptions in compressions (10 seconds or less)
6. Switch compressor about every 2 min or earlier if fatigued

In practice, the operating table is narrow and long; thus, special consideration is required to ensure a proper resuscitation procedure. As an estimate, if the patient is unresponsive to the 2-min resuscitation procedure, consider returning the patient to the supine position with a stretcher while continuing the procedure. However, since it is difficult to compress the thorax during repositioning, circulation stops, and if this time is prolonged, it would be difficult to improve the patient's neurological prognosis.

Breathing often stops due to the use of muscle relaxants. Various monitors would already have been attached, and venous lines are often secured. The treatment should aim to improve the patient's condition, and it is important to avoid compromising the above-mentioned “quality CPR,” for example due to delays in confirmation of monitor values.

There are many reports of successful resumption of heartbeat in the prone and lateral positions, but this is not always easy to achieve; hence, it is important to be flexible when addressing the situation.¹⁴

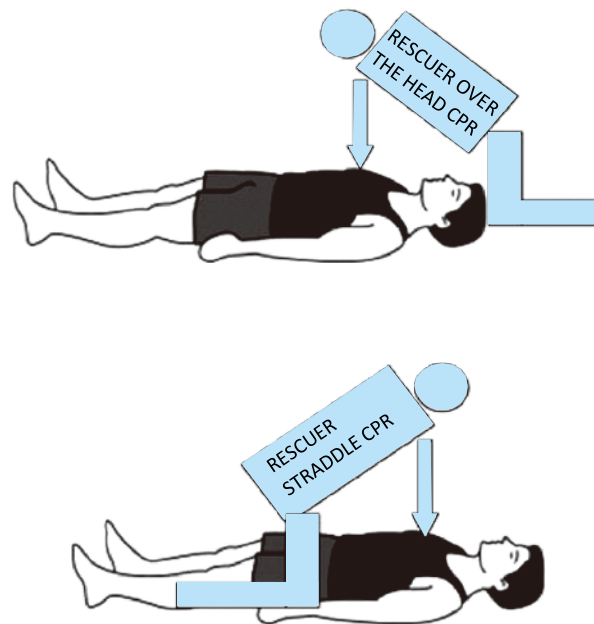


Figure 1 Chest compression approach

[Reprinted from 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 : 233–8]

Factors in CPR treatment

1) Confirmation of cardiac arrest

Resuscitation should be considered in case of bradycardic circulatory failure, even without cardiac arrest.

2) Checking the safety of the surroundings

It is often difficult to secure a safe space due to surgical and medical instruments. If the operating table is tilted, perform the procedure horizontally. Then, it is necessary to close the surgical field as soon as possible and remove the cover cloth and suspension. If it is difficult to perform chest compressions from the body side, it is necessary to kneel on top of the operating table, on both knees, and perform the procedure from the head or the foot side (Figure 1).²⁾

3) Position of the hand during compression

In high-quality CPR, both hands should be on the lower half of the sternum while the patient is in the supine position; however, it is more efficient to press on the line connecting the lower edges of the scapula in the prone position (Figure 2). If median compression is difficult due to the wound, press both sides of the wound as shown in Figure 2.³⁾

In the lateral position, compression should be performed in the same location as in the supine position, but since compressing the sternum alone will not yield sufficient cardiac output, the back should also be compressed at the same time as the chest (Figure 3).⁴⁾

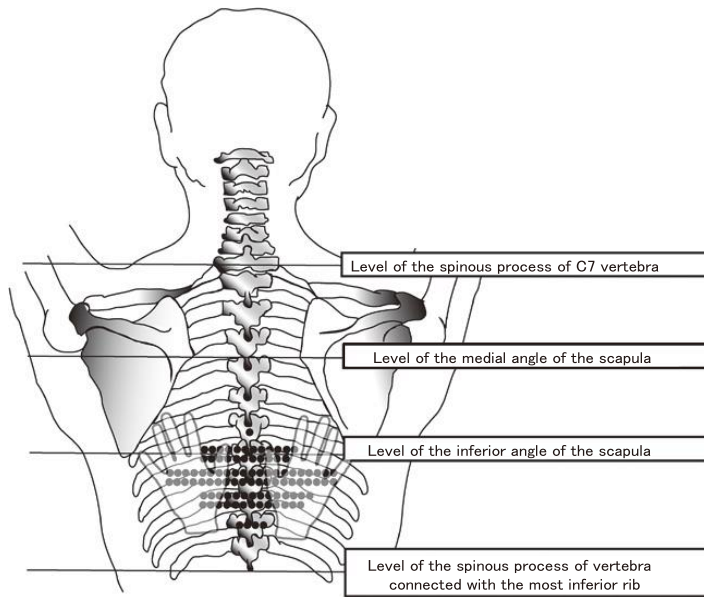


Figure 2 Position of compression for CPR

[Reprinted from Kwon MJ, Kim EH, Song IK, et al. Optimizing prone CPR: Identifying the vertebral level correlating with the largest left ventricle cross-sectional area via computed tomography scan. *Anesth Analg* 2017;124:520—3]

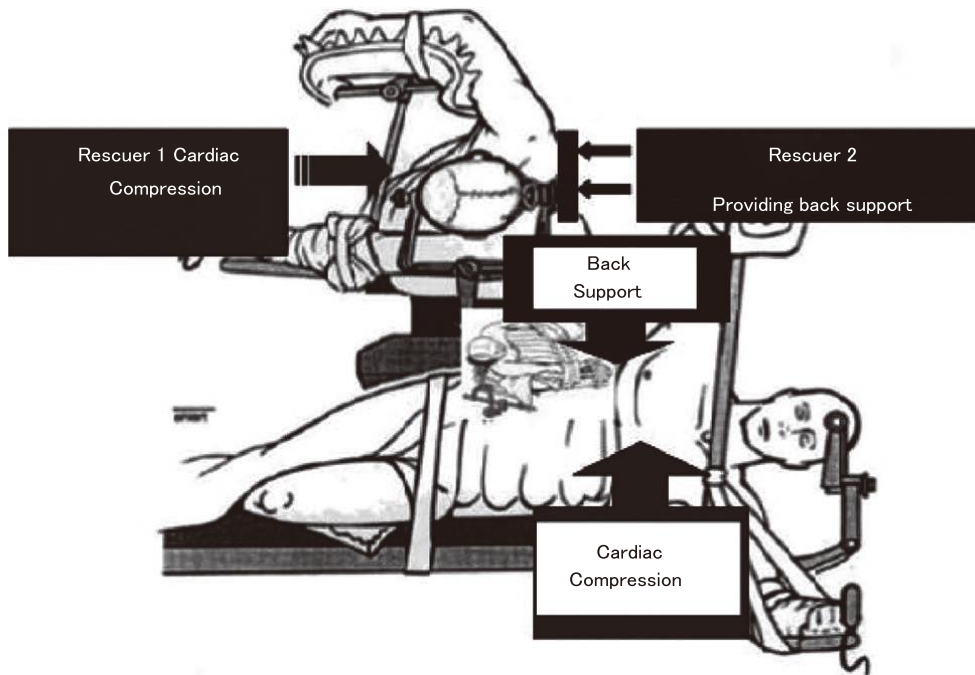


Figure 3 CPR: Compression in the lateral position

[Reprinted from 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—8]



(a) Use pillows



(b) Prone position operating table

Figure 4 Cardiopulmonary resuscitation: Prone position

[Reprinted from (a): Mazer SP, Weisfeldt M, Bai D, et al. Reverse CPR: A pilot study of CPR in the prone position. *Resuscitation* 2003;57:279–85. (b): Reprinted from Muranaka Sesakusho homepage]

It is recommended to use a back plate when the patient is in the supine position because surgical mats, body support, and specialized operating tables are flexible, which reduces the compression efficiency. Even in the prone position, efficiency is improved if there is a pillow on the sternum (Figure 4-a),⁵⁾ but this is difficult to apply on an operating table dedicated to the prone position (Figure 4-b).

4) Proper compression monitor

As for the speed of compression, refer to an ECG monitor that can monitor changes in thoracic impedance.

In terms of the actual degree of chest compressions, it is highly possible that a position-fixing mat would reduce compression efficiency. It is necessary to return the patient to the supine position as soon as possible if end-tidal carbon dioxide (EtCO₂) concentration and the pressure waveform of arterial blood are insufficient. As an estimate, proper compression should be at 20 mmHg or higher on the EtCO₂ monitor, 40 mmHg or higher diastolic arterial pressure, and 30% or higher central venous oxygen saturation.⁶⁾

5) Reduce ventilation

Normal chest compression is likely to yield about 1/4 to 1/3 of the normal cardiac output. Due to the blood flow balance, the ventilation may need to be reduced accordingly, as in the supine position.

6) Minimize interruptions

Compression is interrupted while the body is being returned to the supine position, during which the circulation stops. However, if it is determined that “quality CPR” cannot be performed, it is necessary to return the patient to the supine position promptly.

7) Defibrillation

When indicated, defibrillation is very effective and should be performed promptly, without changing body position.⁷⁾ In that case, attaching the paddle in the prone or lateral position is often difficult; as such, ideally, a defibrillation pad should be used. If possible, a defibrillation pad should be placed in advance.¹⁾

Circulation assistance

If auxiliary circulation (such as extracorporeal membrane oxygenation [ECMO]), called extracorporeal CPR (ECPR) is available, its indication for refractory cardiac arrest cases should be considered.⁴⁾

Cause search

Finding and treating the cause is difficult due to the surgical procedure and position. As such, it is necessary to return the patient's body to the supine position, and it is very important to carry out surgical simulation training in preparation for emergencies, including decision-making in such situations.⁸⁾

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11

Maternal cardiac arrest

Learning content

In this section, the resuscitation method for pregnant women experiencing cardiac arrest and the causative conditions of maternal cardiac arrest are explained, focusing on the differences from non-pregnant women.

General notes

Maternal cardiac arrest in the operating room includes perimortem cesarean delivery; women in severe conditions who go into cardiac arrest during anesthesia; and pregnant women in good conditions presented with unexpected cardiac arrest during cesarean delivery. After delivery of the baby, the resuscitation algorithm is similar to that used for a non-pregnant woman. In pregnant women after 20 weeks of gestation, left uterine displacement and perimortem cesarean delivery (PMCD) are added to the standard algorithm for non-pregnant adults (Figure 1). Causes of cardiac arrest in mothers are very different from those in non-pregnant women.

Maternal resuscitation is the priority. Although some medical tests and treatments may be disadvantageous to the fetus, provide the maximal effort to improve maternal prognosis.

Cardiopulmonary resuscitation in maternal cardiac arrest

The algorithm of maternal cardiac arrest is shown in Figure 1.¹⁾

1) Chest compressions

If cardiac arrest is diagnosed or suspected, perform chest compressions as in non-pregnant women.

2) Airway management and ventilation

Because oxygen consumption increases and functional residual capacity decreases, pregnant women are prone to hypoxemia during hypoventilation and apnea. In addition, hypoxia is often the cause of maternal cardiac arrest. Therefore, establishing ventilation is important in resuscitation of pregnant women.

Give two rescue breaths for every 30 chest compressions until an advanced airway is secured. Administer 100% oxygen. Tracheal intubation is the first choice of advanced airway. Pregnant women have impaired lower esophageal sphincter function and a higher rate of gastric regurgitation. In addition, the risk of aspiration

increases because of delayed gastric emptying during labor. In pregnant women, the pharyngeal volume decreases, and the airway mucosa is prone to bleeding.

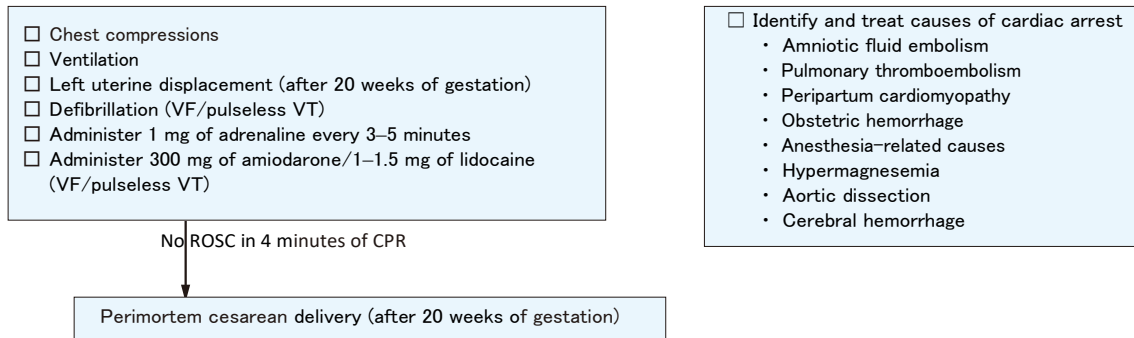


Figure 1 Algorithm of maternal cardiac arrest

VF: Ventricular fibrillation, VT: Ventricular tachycardia, ROSC: Return of spontaneous circulation

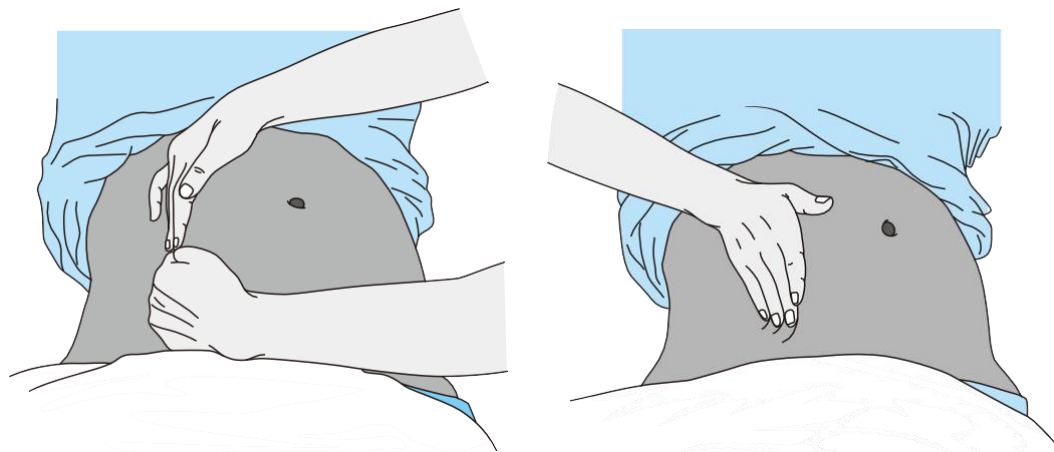


Figure 2 Manual left lateral uterine displacement

A video laryngoscope is the first choice for tracheal intubation, and smaller diameter tracheal tubes should be selected. Once advanced airway is in place, ventilate the woman every 6 seconds with continuous chest compressions.

3) Left uterine displacement

For pregnant women after 20 weeks of gestation (when the uterus is palpable at or above the level of the umbilicus), provide left uterine displacement in order to minimize the compression of the vena cava by the gravid uterus. The displacement should be maintained until the fetus is delivered. Manual uterine displacement (Figure 2) is superior to left lateral tilt of the body because the latter may interfere with effective chest compressions.²⁾

4) Defibrillation

Defibrillation in pregnant women is performed in the same manner as in non-pregnant women. Since the pregnant uterus easily conducts electricity, do not place the two pads with the uterus interposed.

5) Drugs

Administer adrenaline and antiarrhythmic drugs as in non-pregnant women.

6) Perimortem cesarean delivery

PMCD is performed primarily to improve the mother's outcome; however, the fetal prognosis is also improved if delivered from the hypoxic intrauterine environment. In pregnant women after 20 weeks of gestation, consider PMCD regardless of fetal viability.^{3,4)}

The optimal timing for commencing PMCD is yet to be established. According to a report from Japan, time from cardiac arrest to PMCD start was only 6 ± 5.7 minutes in mothers who underwent PMCD and discharged without any sequelae.⁵⁾ If the patient enters the operating room for PMCD, start the surgery as soon as possible as part of the cardiopulmonary resuscitation (CPR) procedure. Administration of anesthetics is not necessary.

In order to accomplish PMCD rapidly, multidisciplinary (obstetrics, emergency department, anesthesiology, operating room, neonatal department, etc.) discussion and pre-planning are essential. It is recommended to develop a PMCD manual and do simulation training.

Massive hemorrhage may follow return of spontaneous circulation (ROSC) during PMCD. The decision of performing PMCD must be based on the available medical resources.

Causes of maternal cardiac arrest

The major causes contributing to maternal cardiac arrest in Japan are listed below⁶⁾.

1) Amniotic fluid embolism

The cause of this devastating disease is the fetal components flowing into the maternal circulation. These induce chemical mediators and release substances which promote vasoconstriction and blood coagulation. This develops most frequently when the membranes rupture. The initial signs are hypoxemia due to pulmonary artery spasm, right-sided heart failure, arrhythmia, or convulsions due to cerebral artery spasm. After pulmonary artery spasm is relieved, it may progress to left-sided heart failure or acute respiratory distress syndrome. Massive obstetric hemorrhage follows as a result of disseminated intravascular coagulation. No specific diagnostic method has been established, and the diagnosis is made by exclusion.⁷⁾ The serum marker analyses at Hamamatsu University School of Medicine will help achieve a later diagnosis.⁸⁾ There is no specific cure. Assess hemodynamics, oxygenation, and blood coagulability (particularly fibrinogen levels) and provide symptomatic treatment.

2) Pulmonary thromboembolism

Pregnant women are in a hypercoagulable state and at high-risk of pulmonary thromboembolism.⁹⁾ In Japan, deaths from pregnancy-related pulmonary thromboembolism are most frequent during the postpartum period, but are observed also during the first trimester.¹⁰⁾

Diagnosis and treatment are similar to those for non-pregnant women. Radiation is often used for the diagnosis and treatment of pulmonary thromboembolism. Radiation has little effect on the fetus at exposure doses of 50 mGy or less.¹¹⁾ The fetal dose from a single chest computed tomography examination is estimated at < 1 mGy.¹²⁾ Iodine contrast medium is transferred to the fetus, but the development of hypothyroidism remains a theoretical concern.¹¹⁾ Unfractionated heparin is less effective in pregnant women, who require higher doses than non-pregnant women.¹³⁾ Unfractionated heparin hardly passes through the placenta.

3) Peripartum cardiomyopathy

This condition is most frequently diagnosed from the end of pregnancy to the first few weeks after giving birth. Peripartum cardiomyopathy is diagnosed when there are heart failure symptoms during pregnancy to several months after delivery, left ventricular ejection fraction is < 45%, and there are no other causes of heart failure.¹⁴⁾ Treatment is similar to that for heart failure in other conditions, including catecholamines, intra-aortic balloon pump (IABP), and extracorporeal membrane oxygenation (ECMO).

4) Obstetric hemorrhage

Obstetric hemorrhage is the prime cause of pregnancy-related death and admission to the intensive care unit (ICU) in Japan.^{6,15)} It most frequently occurs immediately after delivery. The causes include uterine atony; trauma, such as uterine rupture; retained placenta; and coagulopathy. Obstetrical hemorrhage is usually diagnosed before cardiac arrest, but retroperitoneal bleeding is difficult to diagnose by ultrasonography and is easily overlooked.

DIC-type, or uterine-type, amniotic fluid embolism is a cause of severe obstetric hemorrhage. This type of amniotic fluid embolism develops with uterine atony and incoagulable bleeding from the uterus and birth canal, without hypoxemia or circulatory collapse.⁸⁾ Other obstetric diseases that are prone to hyperfibrinolytic DIC include placental abruption and dead fetus syndrome. These diseases are characterized by fibrinogen depletion that is not proportional to the amount of bleeding. In contrast, DIC is not induced at the early phase of hemorrhage due to placenta accreta, uterine rupture, etc., but dilutional coagulopathy may follow afterwards.

Coagulation factor replacement is necessary for all types of coagulopathies; thus, administer fresh frozen plasma (FFP). Fibrinogen should be supplemented with a target of 200 mg/dl.¹⁶⁾ Fibrinogen concentrate efficiently raises fibrinogen levels. Administration of 3 g of fibrinogen is likely to increase fibrinogen by approximately 100 mg/dl.¹⁷⁾ Tranexamic acid is also effective in treating obstetric bleeding: 1 g should be administered intravenously, and if bleeding persists, an additional 1 g should be administered after 30 minutes.¹⁸⁾

5) Anesthesia-related

High spinal anesthesia and difficult airway are major causes of anesthesia-related cardiac arrest in mothers.^{19,20)} Migration of an epidural catheter can be a cause of high spinal anesthesia. When high spinal anesthesia occurs, wait for the anesthetic effect to wear off while maintaining circulation and ventilation. Difficult airway should be managed according to the guidelines.²¹⁾

6) Hypermagnesemia

Magnesium sulfate is administered as a treatment for preterm labor and preeclampsia. Overdose causes muscle weakness, impaired consciousness, respiratory arrest, and severe arrhythmia. When hypermagnesemia is suspected, consider administering 20-35 ml of 8.5% calcium gluconate or 25-50 ml of 2% calcium chloride in 2-5 minutes.²²⁾

7) Aortic dissection

Pregnancy increases the risk of aortic dissection.²³⁾ It peaks from the third trimester to immediately after delivery.²⁴⁾

8) Cerebral hemorrhage

Preeclampsia and HELLP syndrome (hemolysis, elevated liver enzyme, low platelet) are risk factors for cerebral hemorrhage.²⁵⁾

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12

Special characteristics of pediatric perioperative resuscitation

Learning content

General anesthesia is often indicated for children, and due to their anatomical and physiological characteristics, many airway, respiratory, and circulatory problems related to general anesthesia can occur. In this section we will explain the typical causes of these problems and the appropriate responses.

General notes

As in adults, physicians must do their utmost to avoid cardiac arrest in children through prompt diagnosis and appropriate response. In the event of cardiac arrest, the response should be based on the Pediatric Life Support¹⁾ guidelines in Chapter 3 of the Japan Resuscitation Council's Resuscitation Guidelines 2020. Practical guides on pediatric perioperative resuscitation include the use of intraoperative monitoring of end-tidal CO₂ (EtCO₂), invasive arterial monitors, central venous line, etc., for the purpose of determining the effect of cardiopulmonary resuscitation (CPR); CPR and defibrillation in special positions such as the prone position; as well as the application of resuscitation by thoracotomy heart massage and extracorporeal CPR (ECPR).

Causes of pediatric perioperative cardiac arrest

According to the 1998–2004 database of the US Pediatric Perioperative Cardiac Arrest (POCA) Registry²⁻⁴⁾ and a review by Flick et al. of 92,881 cases of pediatric perioperative cardiac arrest between 1998 and 2005 at one tertiary pediatric facility,⁵⁾ the main causes of pediatric perioperative cardiac arrest include hypovolemia (hemorrhage and dehydration), hyperkalemia (massive red blood cell transfusion), induction of inhalation anesthesia (laryngeal spasm, relative anesthetic excess), complications of the central venous line (venous air embolism, cardiac tamponade, tension pneumothorax), and hypoxemia due to various causes. Although relatively rare, local anesthetic toxicity, malignant hyperthermia, and anaphylaxis are also noteworthy. The following is a list of typical causative conditions.

1) Cardiac arrest during the introduction of inhalation anesthesia

Children often receive general anesthesia by breathing anesthetic gas through a mask to avoid the stress of intravenous (IV) access while awake. This type of inhaled induction can lead to cardiac arrest in the event of laryngospasm or relative overdose of anesthetic gas. If laryngeal spasm occurs during the introduction of inhalation anesthesia, immediately stop the administration of nitrous oxide and ventilate with 100% oxygen. Inhalation anesthesia may be continued if the pulse is still palpable and one is attempting to deepen the anesthetic effect. Continuous positive airway pressure may be helpful using the oropharyngeal airway when attempting ventilation. If the child is flaccid, tracheal intubation should be attempted. If the patient's tone prevents an intubation attempt and there is no infusion route, consider intramuscular injection or submental injection (local injection into the tongue muscle at the floor of the mouth, from the submental part of the extraoral cavity) of 0.02 mg/kg of atropine and 4 mg/kg of suxamethonium (maximum tolerance dose: 150 mg).⁶⁾ If an infusion route is available, a lower dose of suxamethonium (0.3–1.0 mg/kg) can relieve laryngeal spasm. Intravenous anesthesia may also be tried to relieve bronchospasm if circulation is stable. Non-depolarizing muscle relaxants can also be used, although the onset and duration of action will be longer. Due to poor circulation, chest compressions may be required to circulate the drug. If an infusion route is unavailable and cardiac arrest persists, immediately establish a bone marrow route and start CPR. Cardiac arrest due to relative excess of inhaled anesthetic drug may occur when the anesthesiologist does not notice hypovolemia (long-term fasting, laxative use, etc.) or when the depth of anesthesia is not sufficiently appreciated, and deep anesthesia is performed for a long time. The effects of anesthetics may become excessive and cause cardiac arrest in cases with hypothermia and in those with concomitant use of opioids and clonidine, and in newborns within the first month of life. Treatment involves discontinuation of nitrous oxide and inhalation anesthetics, as well as assisted ventilation with 100% oxygen. If the pulse is not palpable, chest compressions and intubation should be initiated. Intubation stimulates the sympathetic nerves and may improve hemodynamics. In addition, despite having uncertain effects, tracheal intubation may be used as an adrenaline administration route if the infusion route is unavailable (0.1 mg/kg). For bradycardia before intubation, intramuscular injection of atropine or local submental injection may be performed, but is not essential. A venous and bone marrow route should be established, following the bradycardia algorithm¹⁾ in the JRC Resuscitation Guidelines 2020.

2) Cardiac arrest due to ventriculo-peritoneal shunt dysfunction

If a child's intracranial pressure increases and causes cardiac arrest, the neurosurgeon must immediately tap her ventriculo-peritoneal (VP) shunt several times to remove cerebrospinal fluid and reduce intracranial pressure. Increased intracranial pressure limits cerebral blood flow during resuscitation. It is important to tap the VP shunt at the start of CPR. In the absence of increased intracranial pressure, one-third of the intrathoracic pressure generated during chest compressions is transmitted to intracranial pressure via the vertebral veins and the cerebrospinal fluid.⁷⁾ When the additional load of this intrathoracic pressure on the intracranial pressure increases, the cerebral blood flow during CPR decreases significantly when the intracranial pressure has already increased,⁸⁾ and even if return of spontaneous circulation (ROSC) is finally established, cerebral perfusion remains markedly reduced, resulting in a poor outcome. Consider the need to remove cerebrospinal fluid during resuscitation.

3) Cardiac arrest during posterior spondylosyndesis and craniofacial reconstruction

Hypovolemia and venous air embolism are often associated with cardiac arrest during spinal and cranial surgery. Hypovolemia may be due to underestimation of intraoperative bleeding and inadequate fluid infusion. Preload monitoring and adequate fluid infusion are also possible if a central venous line is inserted. In infants under general anesthesia, tachycardia may not occur even with decreased circulating blood volume. It has been reported that

tachycardia did not occur during hypotension, thought to be due to hypovolemia, during craniofacial surgery in children under 2 years of age.⁹⁾ It may be difficult to differentiate between decreased circulating blood volume and venous air embolism. In both cases, the arterial pressure drops sharply, resulting in pulseless electrical activity (PEA). In the case of PEA, end-tidal CO₂ (EtCO₂) disappears. Consider venous air embolism if nitrogen is found at the end of exhalation and bubbles are found by Doppler echocardiography. If the central venous pressure is elevated, this may reflect an air embolism in the pulmonary blood vessels. On the other hand, if central venous pressure is low, the circulating blood volume may decrease. Another condition associated with elevated central venous pressure and PEA-like pathology is cardiac tamponade, a complication of the central venous line. When treating venous air embolism, first inform the surgeon of the onset of air embolism, start 100% oxygen ventilation, discontinue nitrous oxide and inhalation anesthesia, block the air inlet (lower the surgical field below the heart, irrigate or cover the wound, increase the intravascular pressure in the Trendelenburg position or by fluid administration), reduce air inflow into the pulmonary circulation (place the patient in the left lateral position to trap air into the right ventricular system), and aspirate air from the central venous line. CPR and vasoactive drug administration may be required. If cardiac arrest occurs in the prone position, chest compressions should be selected in the prone position until the patient can be turned supine. Posterior compression may be effective if the body has support under the sternum. In the case of a midline posterior incision, place your hands on the ribs at both ends of the incision and compress. Aside from this method, compression can be done by placing one hand superposed with the other hand over the spine.^{10,11)} A fist or sandbag placed under the sternum has been described, but the effectiveness an object under the sternum is not clear.^{12,13)} (See Chapter 10, Cardiac arrest in non-supine position [p. 41–5]).

4) Cardiac arrest due to blood transfusion-related hyperkalemia

Children, particularly infants, are at risk of perioperative cardiac arrest due to hyperkalemia (> 6 mEq/L or electrocardiographic changes) due to massive red blood cell transfusion. Fatal or non-fatal transfusion-related hyperkalemia has been described in many case reports¹⁴⁻¹⁸⁾ and case series studies^{19,20)}. In one study, transfusion-related hyperkalemia due to stored blood was the second leading cause of perioperative cardiac arrest.²¹⁾ Furthermore, it has been reported that 19% of non-open heart surgery perioperative cardiac arrest cases in children is due to hyperkalemia.²⁰⁾ Blood transfusion-related hyperkalemia is likely to be complicated mainly by rapid massive erythrocyte transfusion, while there is little change in blood potassium concentration with routine blood transfusion.²²⁾ If the packed red blood cells administered have an older shelf life and have been irradiated to prevent graft-versus-host disease (GVHD), the risk of blood transfusion-related hyperkalemia is increased. The potassium concentration in the extracellular fluid of stored erythrocytes continues to increase at a rate of about 1 mmol/day in proportion to the storage period, averaging 38 mmol/l after 30 days, and increases up to 78.5 mmol/l within 35–42 days when stored in the CPDA-1 preservative.¹⁶⁾ Irradiation of red blood cell products may increase potassium levels by > 20 mEq/l in a single day. Rapid progression of hyperkalemia may result in ventricular arrhythmias, ventricular fibrillation (VF), or asystole. There are two treatments for hyperkalemia: removing potassium from the body or driving potassium into the cell. Removing potassium from the body is the radical option but often takes time and requires adequate perfusion of the kidney for diuretics to work, or of the intestine for sodium polystyrene (Kayexalate, Sanofi-Aventis, Malvern, PA) to be effective.

Hemodialysis is a radical option. In particular, the combined use of veno-arterial (V-A) extracorporeal membrane oxygenation (ECMO) can remove potassium from the body with hemodialysis even when the patient is in cardiac arrest.

The following methods, which move potassium into cells and suppress cytotoxicity, are acute-acting treatments.

- Intravenous calcium injection: 20 mg/kg of calcium chloride or 60 mg/kg of calcium gluconate (intravenous injection, bone marrow tract administration)
- Alkalinization: hyperventilation (immediate decrease in T wave can be observed), 1–2 mEq/kg of sodium bicarbonate (intravenous injection, bone marrow tract administration)
- Glucose-insulin therapy: 2 ml/kg of 25% glucose + 0.1 U/kg regular insulin
- Inhalation of β -agonist (administration of albuterol nebulizer) to drive potassium into cells
- Saline load + forced diuresis by furosemide administration

5) Cardiac arrest due to local anesthetic toxicity

In children, local anesthesia is usually performed under general anesthesia; thus, minor symptoms due to increased blood levels or central nervous system symptoms (excitement, confusion, muscle spasm, convulsions) are masked, making early detection difficult. The first signs of toxicity may be electrocardiogram changes that begin with PR interval prolongation, progressive bradycardia, and conduction disorders can suddenly lead to hypotension, decreased cardiac function, and asystole. When convulsions appear, it is recommended to treat the patient with benzodiazepines immediately.²³⁾ If cardiac arrest occurs, perform chest compressions and administer adrenaline. The dose of adrenaline should follow the resuscitation guidelines of the American Heart Association AHA and does not adhere to the American Local Anesthesia Society standard $< 1 \mu\text{g}/\text{kg}$.²³⁾ Amiodarone is recommended for arrhythmias. However, lidocaine and procainamide are not recommended because they have the same Na^+ channel-blocking effect as local anesthetics and additional toxic effects. Administration of fat emulsion is one of the recommended treatments for bupivacaine toxicity, mainly based on animal experiments and case reports.^{24,25)} There are also case reports of the use of fat emulsions in the treatment of children with bupivacaine^{26,27)} and ropivacaine²⁸⁾ poisoning. Rescue with fat emulsions may be effective for potential lethal cardiovascular toxicity caused not only by amide-type local anesthetics (bupivacaine, mepivacaine, and ropivacaine) but also by other drugs (haloperidol, tricyclic antidepressants, beta blockers, and calcium channel blockers).²⁹⁾

In the fat emulsion treatment protocol, a 1.5 ml/kg bolus of a 20% fat emulsion is administered over 1 min, and after hemodynamics are stabilized, it is continuously administered at 0.25 ml/kg/min for 10 min. If hemodynamics remain unstable, a 1.5 ml/kg bolus should be added and the infusion rate should be increased to 0.5 ml/kg/min. The recommended maximum amount of initial treatment is 10 ml/kg in 30 min.²³⁾ Support of patients with V-A ECMO should be considered until the local anesthetic is metabolized. Some patients have survived to complete recovery when CPR was continued during transport to a facility providing ECMO services. Propofol is not recommended as a substitute for fat emulsions.

6) Cardiac arrest due to anaphylaxis

Anaphylaxis in anesthetized children may suddenly present as circulatory collapse without redness of the skin (see Chapter 9, Cardiac arrest due to anaphylaxis: 5) Kounis syndrome [p. 38] in this guide). Mortality from immediate hypersensitivity reactions under anesthesia ranges from 3% to 9%.³⁰⁾ Symptoms of anaphylaxis

include decreased blood pressure, erythema, bronchospasm, pulmonary edema, pulmonary hypertension, arrhythmia, elevated peak inspiratory pressure, hypoxemia, stridor, urticaria, and angioedema. The most common causes of anaphylaxis in the operating room are similar in adults and children: muscle relaxants (63%), latex (14%), hypnotics (7%), antibiotics (6%), plasma substitutes (3%), and opioids (2%).³¹⁾ Treatment is adrenaline administration. If circulation is sufficient, administer a 0.01 mg/kg intramuscular injection of adrenaline once every 20 min, up to 0.5 mg, or administer a continuous intravenous infusion at 0.1–1 µg/kg/min. Rapid administration of a 20 ml/kg infusion is often required. Discontinue administration of possible allergens or eliminate allergens. Consider discontinuing surgery by reducing the dose of the anesthetic (if the anesthetic is not an allergen: not immediately after the start) or discontinuing its administration, administering 100% oxygen, or changing to the Trendelenburg position. Second-line treatments include antihistamines (administer 1–2 mg/kg of H1 blocker diphenhydramine up to 50 mg) and H2 blocker ranitidine (administer 1–2 mg/kg up to 50 mg), albuterol inhalation for wheezing, and corticosteroids (administer 2 mg/kg of methylprednisolone up to 60 mg, or 2 mg/kg of water-soluble hydrocortisone up to 100 mg). Adrenaline should be administered first. Serum tryptase levels are a useful indicator of mast cell degranulation. The serum tryptase test is time-sensitive and requires blood sampling within 6 hours. Measurement of plasma histamine may increase diagnostic accuracy, but the measurable window is narrower, requiring blood sampling within 30 min if possible, and up to 2 h at most.

7) Open-chest CPR and extracorporeal CPR

Both open-chest CPR and extracorporeal CPR (ECPR) may be more applicable in the operation room, but due to severe limitations on medical resources and the skills of surgical staff, there are currently no specific guidelines. Open-chest CPR is applied when a thoracotomy is independently required, such as for a penetrating injury. It may also be applicable in patients with marked aortic stenosis who are non-responsive to closed-chest CPR. When starting ECPR for children, specially trained staff are needed to prepare dedicated equipment and blood supplementation according to the age and body size of the patient, and to prepare the surgical insertion site of the cannula (common carotid artery, etc.). Some facilities have changed the CPR method during cannulation (60 seconds of compression + 30 seconds of interruption for insertion/cycle), and it usually takes 30–45 min to initiate ECPR in a child. It is necessary to make the decision to introduce this treatment at an early stage, ideally when the second adrenaline administration is required (about 5 min after the start of resuscitation).³²⁾

Drugs and devices frequently used during perioperative cardiac arrest

Table 1 shows the drugs frequently used during perioperative cardiac arrest. Be sure to flush saline when administering drugs via peripheral veins or a bone marrow tract during CPR. Since blood flow under the diaphragm decreases significantly during cardiac arrest, a normal saline flush should also be used when administering medications in a femorally inserted central venous catheter whose tip is below the diaphragm. Perform a normal saline flush following every drug administration. The minimum amount of normal saline solution is 0.25 ml/kg³³⁾ or 5 ml for infants, 10 ml for toddlers, and 20 ml for adolescents. In addition, there are no restrictions on drug administration from the bone marrow tract, and all drugs, such as adrenaline, adenosine, and blood products, can also be administered via the bone marrow tract. Tables 2 and 3 show the key points

about the drugs used for perioperative crisis (recommended dose for children), equipment used for pediatric perioperative cardiac arrest, and defibrillators.

Table 1 Drugs used for perioperative crisis (recommended dose for children)

Drug name	Recommended dose	Indication
ATP	Start with 0.1–0.3 mg/kg IV or IO rapid dose and increase if ineffective (maximum dose 0.3 mg/kg)	Supraventricular tachycardia
Amiodarone	First dose: 5 mg/kg IV or IO rapid administration (maximum dose 300 mg), 2nd and subsequent doses (15 min later): Same dose as above (maximum total amount 15 mg/kg) Administer 2.5–5 mg/kg IV or IO over 30 minutes. Note: Be careful of concomitant use of drugs with QT prolonging action, such as procainamide.	Ventricular fibrillation or pulseless ventricular tachycardia Ventricular tachycardia with pulse
Atropine	First dose: IV or IO 0.02 mg/kg or tracheal administration of 0.04–0.06 mg/kg, Maximum dose: 0.5 mg (children), 1 mg (adolescents). Repeated dose: same dose, maximum total amount: 1 mg (children), 3 mg (adolescents).	Symptomatic bradycardia
Calcium chloride	IV or IO of 20 mg/kg (maximum dose-2 g). Administer slowly, from central vein line if available. May need to push rapidly if pulseless from hyperkalemia.	Hyperkalemia, hypocalcemia
Dantrolene	Secure a dedicated peripheral venous route as large as possible, shake and dissolve the drug in 60 ml of distilled water for injection in a 20 mg bottle until clear, and then administer at least 1.0 mg/kg in about 15 min. If possible, it is recommended to administer 2.0 mg/kg in about 15 min. Repeat as appropriate until EtCO ₂ decreases in response to hyperventilation, muscle rigidity improves, and heart rate decreases. It can be administered up to 7.0 mg/kg. There is the possibility of relapse for 12 hours.	Malignant hyperthermia
Diphenhydramine	IV, IO, or IM administration of 1–2 mg/kg diphenhydramine (up to 50 mg) every 4–6 hours	Anaphylactic shock
Adrenaline	IV or IO of 0.01 mg/kg (maximum 1 mg) or tracheal administration of 0.1 mg/kg (maximum 2.5 mg) every 3–5 min.	Cardiac arrest
Glucose	IV or IO of 0.5–1 g/kg; newborn: 5–10 ml/kg of 10% solution; infant: 2–4 ml/kg of 25% glucose solution; adolescent: 1–2 ml/kg of 50% solution	Hypoglycemia
Hydrocortisone	IV of 2 mg/kg (up to 100 mg)	Adrenal insufficiency
Insulin	0.1 unit/kg + 2 ml/kg/30 min IV of 25% glucose solution, repeat every 30–60 min.	Hyperkalemia
20% fat emulsion	After bolus administration of 1.5 ml/kg over 1 min, continuous administration at 0.25 ml/kg/min for 10 min until circulation stabilizes. If circulation is not stable after 3–5 min, 1.5 ml/kg is administered once more over 1 min, continuous administration is increased to 0.5 ml/kg/min, and the maximum total amount is 10 ml/kg in 30 min.	Local anesthetic toxicity
Lidocaine	Administer 1 mg/kg IV or IO (maximum 100 mg) or 2–3 mg/kg via bronchus. Maintenance: 20–50 µg/kg/min IV or IO continuous administration, repeat 1 mg/kg IV or IO (maximum 100 mg) if injection is to be started more than 15 min after the initial dose.	Ventricular fibrillation or pulseless ventricular tachycardia
Magnesium sulfate	Administer 25–50 mg/kg IV or IO (maximum total 2 g), rapid administration for pulseless multifocal ventricular tachycardia or torsades de pointes, administration over 10–20 min if pulse is palpable.	Hypomagnesemia, Torsades de Pointes
Naloxone	Administer 0.1 mg/kg IV or IO, tracheal administration, or IM (maximum 20 mg) for opioid overdose; and 0.001–0.005 mg/kg IV, IO, or IM if reversing therapeutic opioid dose.	Anesthetic toxicity, resistance
Procainamide	Administer 0.25 mg/kg/min IV or IO over 30–60 min (maximum 15 mg/kg); be careful of concomitant use of drugs with QT prolonging effect, do not routinely use with amiodarone	Tachycardia
Bicarbonate	Administer 1 mEq/kg IV or IO gradually, use rapid administration during cardiac arrest due to hyperkalemia, ensuring adequate ventilation to avoid paradoxical acidosis.	Hyperkalemia
Vasopressin	Administer 0.5 units/kg IV or IO (maximum 40 units): 0.0003–0.002 units/kg/min for cardiac arrest. Continuous administration: catecholamine-resistant shock.	Cardiac arrest, catecholamine-resistant shock

IV, intravenous, IO, intraosseous; IM, intramuscular; EtCO₂: end-tidal CO₂.

Table 2 Equipment used for pediatric perioperative cardiac arrest

Equipment	Overview
Monitor	Used for early recognition of the need for resuscitation. Invasive monitoring when indicated. Use EtCO ₂ (waveform display/quantitative) for the purpose of confirming the effectiveness of chest compressions, airway maintenance, and return of spontaneous circulation
Blood products	Have sufficient replacement blood products at hand (preferably crossmatched, in the operation room if necessary).
Venous	Adequate access for monitoring and delivery of fluids and medications. IO available (useful when facing problems on induction without IV access or when unable to obtain IV access)
Crash cart	Drugs and equipment not mounted on the anesthesia cart, dosing guide (calculated for patients, height-specific, color-coded), confirmation checklist and algorithm copy, fat emulsion for local anesthetic toxicity, malignant hyperthermia kit
Defibrillator	For arrhythmia treatment that requires cardioversion or defibrillation. Be familiar with the defibrillator at your facility. Know the application of pediatric paddles. If intraoperative arrhythmia is a concern, attach a pad before draping
ECMO	Applies to treatable in-hospital cardiac arrest cases that do not respond to CPR. Start process early (prepare blood, fill circuit, prepare cannula, convene surgeon)

EtCO₂: end-tidal CO₂, IO: intraosseous, CPR: CPR, ECMO: extracorporeal membrane oxygenation

Table 3 Key points for defibrillators

Extracorporeal defibrillation	According to the JRC guidelines, the maximum dose is 4 J/kg for both the first and the following doses, but the upper limit is the adult dose. Cardioversion-compatible rhythm: ventricular fibrillation and pulseless ventricular tachycardia. The superiority whether monophasic or biphasic defibrillation is unknown. A one-time shock method, which compresses the chest immediately after cardioversion, is rational
External synchronous cardioversion	For hemodynamically unstable tachycardia (supraventricular/ventricular tachycardia), perform synchronous cardioversion at 0.5–1.0 J/kg, if unsuccessful, increase to 2 J/kg and perform again.
Open-chest defibrillation	First 2–3 J (start with the lowest energy amount of each defibrillator). Performed with thoracotomy. Use a clean paddle

JRC, Japan Resuscitation Council.

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