UpToDate®
Official reprint from UpToDate®
www.uptodate.com © 2024 UpToDate, Inc. and/or its affiliates. All Rights Reserved.

Wolters Kluwer

Initial assessment and management of the adult post-cardiac arrest patient

AUTHORS: Jonathan Elmer, MD, MS, FNCS, Jon C Rittenberger, MD, MS
SECTION EDITOR: Ron M Walls, MD, FRCPC, FAAEM
DEPUTY EDITOR: Jonathan Grayzel, MD, FAAEM

All topics are updated as new evidence becomes available and our peer review process is complete.

Literature review current through: **Feb 2024.**
This topic last updated: **Nov 09, 2023.**

INTRODUCTION

Cardiac arrest affects over 600,000 people per year in North America alone [1]. Depending on the circumstances of arrest, 20 to 40 percent of adults who survive to hospital care after resuscitation from cardiac arrest are discharged alive, the majority of whom enjoy favorable functional recovery [1-8]. Advances in cardiopulmonary resuscitation (CPR) and post-cardiac arrest care delivery have improved outcomes over time [1,2].

Important interventions in the initial management of the post-cardiac arrest adult patient are reviewed here. Basic and advanced life support for adult victims of cardiac arrest, intensive care management, and secondary prevention for survivors of cardiac arrest are discussed separately. (See "Adult basic life support (BLS) for health care providers" and "Advanced cardiac life support (ACLS) in adults" and "Intensive care unit management of the intubated post-cardiac arrest adult patient" and "Overview of sudden cardiac arrest and sudden cardiac death".)

MAJOR PROBLEMS AND CARE PRIORITIES

Management of the post-cardiac arrest patient is complex and must address multiple major problems simultaneously (algorithm 1). Issues to be addressed include:

- Initial cardiopulmonary stabilization and prevention of rearrest (see 'Initial stabilization and prevention of rearrest' below)
- Identification and treatment of reversible causes of cardiac arrest (table 1) (see 'Identifying and treating reversible causes of cardiac arrest' below)
- Ongoing stabilization and prevention of brain injury (see 'Ongoing stabilization and prevention of brain injury' below)
- Early risk stratification, family communication, and disposition (see 'Early risk stratification, family communication, and disposition' below)

Immediately following resuscitation from cardiac arrest, the patient can develop severe problems due to medical comorbidities, the underlying cause of arrest, and sequelae of global ischemia-reperfusion injury. The most immediate threat to survival during the first minutes to hours is cardiovascular collapse. Interventions to optimize blood pressure and maintain brain and other end-organ perfusion (eg, boluses of intravenous [IV] fluid, vasopressors, and inotropes) can help prevent secondary injury from hypotension. Additional short-term goals during the first hours of care include optimizing oxygenation and ventilation and correcting electrolyte abnormalities. (See 'Initial stabilization and prevention of rearrest' below.)

A focused diagnostic evaluation to identify treatable causes of cardiac arrest and initiate appropriate treatments is performed concurrently with resuscitation efforts to prevent recurrent arrest and optimize outcome. Evidence supports the use of temperature management to minimize brain injury for comatose survivors of cardiac arrest. Target body temperature should be achieved within the first few hours following resuscitation. (See 'Temperature management' below.)

INITIAL STABILIZATION AND PREVENTION OF REARREST

Four in 10 patients who initially regain pulses experience at least one rearrest, and a majority of patients with return of spontaneous circulation (ROSC) develop hypotension [9-13]. Both rearrest and hypotension are associated with increased mortality. Thus, initial stabilization measures are crucial.

Circulation — Vascular access should be obtained immediately on presentation, if not already present. At least one peripheral intravenous (IV) or intraosseous (IO) catheter provides a route to administer fluids, vasopressors, and other medications. Central venous access is frequently

established during ongoing resuscitation but is rarely required urgently and can distract from more important immediate management goals.

Invasive blood pressure monitoring through an arterial catheter is useful for ongoing care but not required immediately. In the peri-arrest setting, adequate perfusion can be confirmed by peripheral pulse, sphygmomanometry, plethysmographic waveform, and end-tidal carbon dioxide ($EtCO_2$) monitoring. (See "Carbon dioxide monitoring (capnography)".)

Most patients resuscitated from cardiac arrest are preload responsive and tolerate moderate volume resuscitation. One to 2 liters of isotonic crystalloid given IV or IO via rapid bolus administration (eg, using a pressure bag) are often administered empirically during initial stabilization. A more restrictive fluid strategy is appropriate in patients with a known history of heart failure or clinical evidence of significant pulmonary edema either on physical examination (eg, difficulty oxygenating, noncompliant lungs, crackles on auscultation) or initial chest imaging.

Patients with hypotension in the peri-arrest setting require vasopressor therapy. Given the incidence of early hypotension and rearrest, we initiate vasopressors **in parallel** with fluid resuscitation in the hypotensive patient rather than sequentially. In preload responsive patients, vasopressors can be weaned rapidly. Data supporting a particular drug in this setting are lacking. Norepinephrine is generally well tolerated and effective, and it has a lower risk of tachyarrhythmia than dopamine [14]. Bolus administration of 10 to 100 mcg epinephrine given IV or IO (ie, push-dose pressor) has a rapid physiologic effect and may be used as a bridge to the initiation of other therapies (eg, norepinephrine infusion), although strong data supporting safety are lacking [15,16]. (See "Use of vasopressors and inotropes".)

Airway and breathing — Ventilation by bag-valve-mask or supraglottic airway is safe and effective in the peri-arrest setting [17-20]. For the non-intubated patient, our general practice is to defer advanced airway management until after initial cardiovascular stabilization. (See "Extraglottic devices for emergency airway management in adults".)

After ensuring adequate circulation, the adequacy of oxygenation and ventilation should be reassessed. In the non-intubated patient, confirmation of chest rise, oxygen saturation (SpO_2) >92 percent, and the absence of signs of impending airway obstruction suggest that tracheal intubation may again be deferred until any causes of arrest requiring rapid intervention are treated or excluded.

Patients who are not adequately oxygenated using a bag-valve-mask or supraglottic airway should undergo rapid sequence intubation (RSI). Most patients are comatose after resuscitation from cardiac arrest, and all are at high risk of hypotension from RSI. Although data are scant,

our practice is to reduce the dose of induction agent we administer to comatose post-arrest patients compared with that administered to responsive patients. As an example, induction with etomidate using a dose of 0.15 mg/kg (instead of 0.3mg/kg) is associated with a reduced risk of peri-intubation hypotension [21,22]. Dosing for other induction agents may be reduced in similar fashion, although available evidence does not support any specific dosing regimen. (See "Rapid sequence intubation in adults for emergency medicine and critical care".)

Recurrent arrhythmia — Patients with recurrent ventricular tachycardia (VT) or ventricular fibrillation (VF) are managed according to advanced cardiac life support (ACLS) protocols (algorithm 2). Prompt defibrillation is the mainstay of immediate management. (See "Advanced cardiac life support (ACLS) in adults".)

Amiodarone 300 mg IV/IO push or lidocaine 100 mg IV/IO push are often administered but offer no clear benefit over defibrillation alone [23]. An infusion of amiodarone or lidocaine may be initiated with the intention of preventing recurrent arrhythmia in patients with ROSC [24]. At some centers, this done for patients with at least one rearrest due to VT or VF.

When immediately available, emergency coronary revascularization or extracorporeal life support may be necessary to stabilize patients who are refractory to pharmacotherapy [25,26]. Extracorporeal life support and other mechanical circulatory support are discussed separately. (See "Extracorporeal life support in adults in the intensive care unit: Overview".)

IDENTIFYING AND TREATING REVERSIBLE CAUSES OF CARDIAC ARREST

Cardiac arrest is the final common manifestation of numerous disease processes. Identification of the cause of a specific patient's cardiac arrest requires considering a broad differential diagnosis and performing a focused but thorough history, physical examination, and diagnostic evaluation. Although cardiovascular disease is the most common cause of sudden cardiac arrest (SCA), no single cause predominates in a significant portion of patients [27]. Common etiologies for SCA are summarized in the following tables (table 2 and table 1). (See "Overview of sudden cardiac arrest and sudden cardiac death", section on 'Etiology' and "Pathophysiology and etiology of sudden cardiac arrest", section on 'Etiology of SCD'.)

Cardiac arrest sustained during trauma has different causes than nontraumatic arrest (assuming cardiac arrest did not precede the trauma) and is managed differently. Trauma management is reviewed separately. (See "Initial management of trauma in adults" and "Approach to shock in the adult trauma patient".)

History — Most patients are comatose after resuscitation and cannot provide a history of present illness or pre-existing conditions. Clinicians should check the patient's wallet and belongings for medical information and look for a medical alert bracelet. They should speak with anyone who can provide insight into the history (eg, family, friends, witnesses, emergency medical services personnel) to help determine the etiology of SCA as rapidly as possible after the return of spontaneous circulation (ROSC). The primary care physician and pharmacist may provide useful information.

Important historical details include the circumstances of collapse (eg, antecedent symptoms reported by the patient, recent illness, initial rhythm, presence of recreational drug paraphernalia at the scene) and clinical suspicion for trauma (eg, fall from standing at the time of collapse). Past medical and social history may suggest a particular arrest etiology.

Physical examination — Examination is performed concurrently with initial stabilization of the circulation and ventilation. Examination findings may suggest the etiology of arrest. A rigid abdomen suggests a surgical emergency but may be due to an air-filled stomach. Significant quantities of blood in the rectum or nasogastric tube suggest a hemorrhagic cause (eg, gastrointestinal bleeding). Extremity signs of deep venous thrombosis (eg, unilateral leg edema), intravenous (IV) drug abuse, or a source of sepsis may suggest a diagnosis. A patient with an arteriovenous fistula may have experienced hyperkalemia, leading to arrest. Bradycardia with hypotension, priapism, and other signs may suggest that collapse resulted in a high cervical spinal cord injury and respiratory arrest leading to cardiac arrest.

Baseline neurologic examination — A baseline neurologic examination should be performed to help determine the possible cause, likely clinical course, and need for neurologic interventions (eg, temperature management). The initial examination provides a baseline estimate of prognosis that can be modified based on additional information and observation [28]. Temporary avoidance, cessation, or reversal of neuromuscular blockade and sedation is necessary for a valid examination. The examination may be delayed if long-acting drugs have been administered. Train-of-four testing can be used to determine the degree of neuromuscular blockade. Train-of-four testing and the neurologic examination in the patient with depressed mental status are described separately. (See "Neuromuscular blocking agents in critically ill patients: Use, agent selection, administration, and adverse effects", section on 'Administration' and "Stupor and coma in adults", section on 'Neurologic examination'.)

Asymmetric neurologic findings are not expected following ROSC and suggest a structural intracranial lesion or acute stroke. Brainstem responses, including the pupillary, corneal, oculocephalic, gag, and cough response to stimulation, should be assessed [28]. The Glasgow Coma Scale (GCS) (table 3) or Full Outline of UnResponsiveness (FOUR) score (table 4)

should be determined, with particular attention paid to the motor score. Patients who cannot perform purposeful movements or follow basic commands generally need specialty care and targeted temperature management (TTM). (See 'Temperature management' below.)

While the initial neurologic examination is useful, many neurologic abnormalities identified early after the ROSC improve over time. As an example, pupillary light reflex is bilaterally absent in two-thirds of patients early after ROSC, of whom 30 percent enjoy a recovery with good neurologic function [29]. Initial neurologic findings alone do **not** exclude potential recovery after resuscitation from cardiac arrest [30,31].

Diagnostic tests — Diagnostic tests, including an electrocardiogram (ECG), laboratory tests, and imaging studies, are usually required to determine the etiology of cardiac arrest, confirm tracheal tube positioning, assess for trauma due to chest compressions, ascertain the function of specific organ systems, and gauge the severity of injury.

Electrocardiogram — Acute myocardial infarction, cardiomyopathy, and primary arrhythmia are common causes of cardiac arrest. Following ROSC, a 12-lead ECG should be obtained rapidly and evaluated for signs of ST-elevation myocardial infarction (STEMI; including new left bundle branch block) that requires emergency reperfusion therapy. Abnormalities of conduction intervals, electrical axis, and T-waves may provide clues to the etiology. As examples, a prolonged QTc interval may reflect a primary arrhythmia, accidental hypothermia, or an electrolyte abnormality; while signs of right heart strain (eg, right axis deviation) may be present with pulmonary embolus. (See "Electrocardiogram in the diagnosis of myocardial ischemia and infarction".)

Ischemic changes, including ST elevation on ECG obtained early after ROSC, are neither highly sensitive nor specific for acute coronary syndrome [32]. Among patients with out-of-hospital cardiac arrest, significant coronary artery disease is often present in the absence of an acute STEMI [33,34]. The incidence of coronary artery lesions is highest among patients whose presenting arrhythmia is ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) [32,35]. Thus, immediate or urgent cardiac catheterization may be needed for patients without a STEMI but who have a concerning history (antecedent chest pain or shortness of breath and cardiac risk factors) or whose presenting arrhythmia was VF or pulseless VT. (See 'Coronary revascularization' below.)

Conversely, ST elevation is present in approximately 1 in 10 patients with negative coronary angiography [36]. The false-positive rate of ECG for diagnosis of STEMI is time dependent and occurs most commonly within seven minutes of the ROSC, presumably due to transient myocardial ischemia from global hypoperfusion during cardiac arrest and administration of

high-dose epinephrine [36]. Thus, in patients who are not overtly unstable, it is reasonable to obtain a repeat ECG 5 to 10 minutes after an initial ECG obtained within minutes of ROSC that is concerning for myocardial ischemia. Given the prevalence of clinically significant lesions on cardiac catheterization in cardiac arrest patients, prompt cardiology consultation should not be delayed until a repeat ECG is obtained. If the initial ECG does not include findings concerning for myocardial ischemia, a repeat ECG obtained soon after the initial study is unnecessary, although urgent catheterization may still be appropriate based on the history (eg, shockable rhythm, no clear alternative etiology).

When the diagnosis of acute coronary syndrome is uncertain based on ECG findings, bedside echocardiography may demonstrate focal wall motion abnormalities, suggesting acute myocardial infarction. Global but not focal hypokinesis is expected following cardiac arrest. (See 'Imaging studies' below.)

Ongoing cardiogenic shock may be the sole manifestation of acute coronary syndrome in some patients with ROSC. Such patients may benefit from both percutaneous coronary intervention and mechanical circulatory support. (See "Intensive care unit management of the intubated post-cardiac arrest adult patient" and "Extracorporeal life support in adults in the intensive care unit: Overview".)

Laboratory testing — Laboratory testing may provide insight into the cause of arrest and help to determine the extent and progression of arrest-related injury to organ systems. In particular, electrolyte and acid-base disturbances require close monitoring during resuscitation and ongoing management.

The early post-arrest period is a physiologically dynamic time when patients typically receive aggressive, ongoing resuscitative efforts. After ROSC, we suggest the following laboratory tests be obtained as soon as possible and repeated serially at least every six hours during initial management:

- **Arterial blood gas** – In mechanically ventilated patients and non-intubated patients with respiratory insufficiency, an arterial blood gas is obtained as soon as possible after ROSC and repeated after initial post-arrest resuscitation efforts are implemented. Arterial vascular access is obtained routinely in comatose post-cardiac arrest patients given the need for frequent arterial blood gas measurements and the common use of vasopressor and inotropic drugs. (See "Arterial blood gases" and "Simple and mixed acid-base disorders".)

- **Electrolytes** – Basic serum electrolyte concentrations, including sodium, potassium, chloride, and bicarbonate, are obtained serially to detect abnormalities. Rapid fluctuations

in serum potassium may occur as a result of ischemia, acidosis, and catecholamine administration. Both high and low potassium can cause arrhythmias and must be treated immediately. Note that hypokalemia is often accompanied by hypomagnesaemia, which should also be corrected. (See "Treatment and prevention of hyperkalemia in adults" and "Clinical manifestations and treatment of hypokalemia in adults".)

- **Blood counts** – Blood counts are measured to detect anemia and other hematologic abnormalities. Profound anemia suggests blood loss as a factor contributing to cardiac arrest. Leukocytosis of 10,000 to 20,000 is common and may represent acute demargination of white blood cells and systemic inflammation due to global ischemia-reperfusion injury. Markedly elevated leukocytosis should prompt investigation of a cause other than cardiac arrest.

- **Troponin** – Serum troponin is measured serially to detect myocardial injury. If initial measurements are elevated, testing continues until there is clear evidence they are decreasing. Cardiac arrest, cardiopulmonary resuscitation (CPR), and defibrillation often cause mild increases in the serum troponin (troponin I 0 to 4 ng/mL, or microgram/L), but rising or higher levels suggest acute coronary artery occlusion. Using biomarkers to diagnose myocardial infarction in patients with kidney disease is discussed separately. (See "Cardiac troponins in patients with kidney disease".)

- **Lactate** – Serum lactate concentration is measured immediately after ROSC and serially thereafter. Initial lactate concentrations and the rate of lactate clearance correlate with survival [37]. Lactic acidosis, with serum lactate concentrations up to approximately 15 mmol/L, is common after cardiac arrest, but higher levels suggest ongoing intra-abdominal or muscle compartment ischemia. Lactate should clear over time after adequate perfusion is restored.

- **Toxicology** – Specific toxicology studies may be obtained in patients with a history of drug ingestion, signs of a toxicologic syndrome (eg, sympathomimetic poisoning), or clinical suspicion of poisoning. As examples, myocardial infarction or arrhythmia may be caused by acute cocaine or methamphetamine intoxication, cardiopulmonary collapse may be precipitated by massive antidepressant overdose, and sedative overdose may contribute to and prolong coma independent of any brain injury sustained during a cardiac arrest. The presence of long-acting opioids (eg, methadone) may prompt use of a reversal agent (naloxone) before neurologic testing. Evaluation and management of the unresponsive and critically ill poisoned patient is discussed separately. (See "Initial management of the critically ill adult with an unknown overdose".)

- **Kidney and liver function** – Ischemia can impair kidney and liver function, which may affect drug dosing and decisions to use IV contrast. Thus, we recommend obtaining tests for kidney function (ie, blood urea nitrogen [BUN], creatinine) and hepatic function (ie, aspartate aminotransferase [AST], alanine aminotransferase [ALT], direct and total bilirubin).

Most patients require central venous access, arterial access, and potentially other invasive procedures. Therefore, we recommend measuring the prothrombin time (PT), activated partial thromboplastin time (aPTT), and international normalized ratio (INR) as part of initial laboratory testing.

Imaging studies

- **Chest radiograph** – Obtain a chest radiograph to evaluate for pulmonary pathology and to confirm proper positioning of the tracheal tube and any central venous catheter. Pulmonary edema and evidence of aspiration are common findings after CPR but may be unrelated to the cause of arrest. Pneumothorax or mediastinal abnormalities suggestive of aortic dissection represent ongoing threats to patient survival and require immediate intervention. An enlarged aorta or concerning mediastinal findings on chest radiograph should prompt computed tomography (CT) imaging. (See "Clinical features and diagnosis of acute aortic dissection".)

- **Bedside ultrasound** – Bedside emergency ultrasound can help to identify causes of arrest that represent ongoing threats to life, including pericardial tamponade, pneumothorax, catastrophic pulmonary embolism, and intraperitoneal bleeding [38-42]. (See "Evaluation of and initial approach to the adult patient with undifferentiated hypotension and shock".)

  Ultrasound or echocardiography can be used to assess right ventricular size and function (which may be abnormal with pulmonary embolism), determine inferior vena cava diameter (which may be abnormal with hypovolemia), and assess global cardiac function. (See "Cardiac evaluation of the survivor of sudden cardiac arrest", section on 'Echocardiography'.)

- **Computed tomography** – Given the prevalence of clinically significant findings, it is our practice to err on the side of performing comprehensive CT imaging early in the care of most patients [43-46]:

  - **Head CT** – Non-contrast CT of the brain can detect early cerebral edema or intracranial hemorrhage in the comatose post-cardiac arrest patient [47-49]. Approximately 2 to 6 percent of patients have intracranial hemorrhage (usually subarachnoid hemorrhage)

identified as the cause of their arrest [27,43,47,50], altering both expected prognosis and precluding the use of anticoagulation. (See "Spontaneous intracerebral hemorrhage: Pathogenesis, clinical features, and diagnosis" and "Aneurysmal subarachnoid hemorrhage: Clinical manifestations and diagnosis" and "Nonaneurysmal subarachnoid hemorrhage".)

- **Cervical spine CT** – Fall from standing and other low-energy mechanisms of trauma at the time of arrest may result in cervical spine fracture. Among older adult patients, syncope resulting in high cervical spine fracture and associated respiratory failure may cause cardiac arrest [51]. Most patients are comatose early after ROSC and cannot provide a history. Non-contrast CT of the cervical spine is sensitive for the detection of bony injuries of the cervical spine and should be obtained when trauma, including fall from standing, cannot be excluded by history. (See "Cervical spinal column injuries in adults: Evaluation and initial management" and "Suspected cervical spine injury in adults: Choice of imaging".)

- **Chest and abdominal CT** – CT of the chest, with or without contrast, commonly identifies pathology that changes management, including pneumothorax, organ laceration, aspiration, and rib fractures from chest compressions [43-45,52]. CT of the chest with contrast is useful in cases of suspected pulmonary embolism; however, the study may be delayed if there is concern for acute kidney injury. In this setting, if the clinician strongly suspects that a massive pulmonary embolism accounts for the patient's arrest, other imaging studies (eg, transthoracic or transesophageal echocardiography, ultrasonography of the lower extremities) can be performed while treatment with empiric anticoagulation is started. The high incidence of abnormalities on chest radiograph limits the utility of ventilation/perfusion (V/Q) scanning in this population [53]. (See "Epidemiology and pathogenesis of acute pulmonary embolism in adults".)

  In patients with a markedly elevated serum lactate (>15 mmol/L), traumatic mechanism, clinical findings of peritonitis, or free intraperitoneal fluid on bedside ultrasound, CT imaging with contrast of the abdomen and pelvis is useful for determining an intra-abdominal cause of arrest. Chest compressions may cause hepatic or splenic laceration, which (although uncommon) may alter acute management [44,45].

- **Head-to-pelvis (whole-body) CT** – Several prospective cohort studies have evaluated the diagnostic yield of head-to-pelvis cross-sectional imaging after out-of-hospital

cardiac arrest. Benefits of this imaging approach include identification of both CPR-related trauma and the underlying etiology of cardiac arrest.

- In a single-center observational study of 104 patients resuscitated from cardiac arrest, head-to-pelvis imaging identified the arrest etiology in 41/104 (39 percent) and revealed time-urgent complications in 17/104 (16 percent), most commonly solid organ injury and pneumothorax [44].

- In a second observational study also with 104 subjects, head-to-pelvis imaging identified 84 (81 percent) resuscitation-related complications but only 15 (14 percent) time-critical injuries. The most common complications were rib fractures (77/104 [74 percent]) and sternal fractures (19/104 [18 percent]). Most rib fractures were not clinically significant [54].

Based on these data and the clinical importance of identifying both the etiology and sequelae of arrest, we believe it is reasonable to obtain liberal cross-sectional imaging (eg, head-to-pelvis CT with contrast) in most patients, particularly those in whom coma limits history and physical examination. Optimal timing of such imaging will vary by patient and depends on overall clinical stability (eg, perceived risk of rearrest, hypoxia, or hypotension during transport and CT) and potential for delay of other time-sensitive procedures that are clearly indicated (eg, coronary angiography for ST elevation myocardial infarction).

ONGOING STABILIZATION AND PREVENTION OF BRAIN INJURY

Respiratory considerations

Initial interventions — The clinician must ensure a patent and functioning airway. An obstructed airway can rapidly lead to recurrent cardiac arrest. If a temporizing rescue airway device (eg, laryngeal mask, laryngeal tube) was used during resuscitation, this should be replaced with a definitive airway at the earliest opportunity. The stomach should be decompressed with a gastric tube.

The details of airway management are discussed separately. (See "Rapid sequence intubation in adults for emergency medicine and critical care" and "Extraglottic devices for emergency airway management in adults" and "Overview of advanced airway management in adults for emergency medicine and critical care".)

Mechanical ventilation — Mechanical ventilation in the post-cardiac arrest patient must balance the need to reverse hypoxia and acidosis with the potential deleterious effects of hyperventilation and hyperoxia. Specifically, routine hyperventilation should not be used to compensate for metabolic acidosis. Mechanical ventilation following cardiac arrest is discussed in detail separately; a few key targets and their rationale are described below. (See "Intensive care unit management of the intubated post-cardiac arrest adult patient", section on 'Airway, ventilation, and oxygen targets' and "Mechanical ventilation of adults in the emergency department".)

The following guidelines are important for initial mechanical ventilation in the post-cardiac arrest patient:

- **Target a carbon dioxide tension ($PaCO_2$) of 40 to 50 mmHg or end-tidal carbon dioxide ($EtCO_2$) of approximately 35 to 45 mmHg.**

  Hyperventilation and the resulting hypocapnia lead to cerebral vasoconstriction that may worsen brain injury after cardiac arrest [55-58]. In a randomized trial, mild therapeutic hypercapnia ($PaCO_2$ 50 to 55 mmHg) resulted in lower concentrations of biomarkers of brain injury than targeted normocapnia ($PaCO_2$ 35 to 45 mmHg) [59]. In observational studies, $PaCO_2$ in a normal to mildly elevated range ($PaCO_2$ 35 to 55 mmHg) is associated with better outcomes than a lower $PaCO_2$ [60-62]. Significant hypercarbia may worsen acidosis.

  The Mild hypercapnia or normocapnia after out-of-hospital cardiac arrest (TAME) trial randomized 1700 patients to targeted mild therapeutic hypercapnia (PaCo2 50-55mmHg) or targeted normocapnia (PaCO2 35-45mmHg) and found no difference in functionally favorable survival using the Glasgow Outcome Scale, adverse events, or key secondary outcomes [63]. TAME corrected sample temperature to 37°C before analysis (alpha-stat method). This adjustment is made because the actual $PaCO_2$ may be lower than what is measured in the laboratory at 37°C; however, the approach has been associated with decreased cerebral blood flow after cardiac arrest in some studies [64].

  No available data support hypocapnia as a therapeutic approach after cardiac arrest, and it remains controversial whether to correct blood gas results for low temperatures while patients are hypothermic during targeted temperature management (TTM) [65]. For these reasons, the authors think that a $PaCO_2$ of 40 to 50 mmHg is a safer target than 35 mmHg at all patient temperatures.

- **Maintain oxygen saturation (SpO_2) >94 percent.**

Hypoxia must be avoided in the post-cardiac arrest patient, but hyperoxia (arterial oxygen tension [PaO_2] >300 mmHg) has also been associated with worse outcomes [66,67]. We suggest titrating the fraction of inspired oxygen (FiO_2) to the lowest value necessary to maintain SpO_2 >94 percent, or the PaO_2 to around 100 mmHg [68,69]. If the patient's core temperature is maintained at 33°C, the PaO_2 reported by the laboratory may be higher than the patient's actual PaO_2. Thus, in this clinical setting, maintaining a PaO_2 of 100 to 120 mmHg is reasonable.

Overall, published data support a strategy that avoids both severe hyperoxia and hypoxia:

According to a systematic review of 14 observational studies, hyperoxia (defined as a PaO_2 >300 mmHg) was associated with increased in-patient mortality among patients with a return of spontaneous circulation (ROSC) following cardiac arrest (odds ratio [OR] 1.4; 95% CI 1.02-1.93), although not with a poor neurologic outcome (OR 1.62; 95% CI 0.87-3.02) [70]. In a cohort of 6326 post-cardiac arrest patients, the OR for death among patients with hyperoxia (defined as PaO_2 >300 mmHg on the first arterial blood gas) was 1.8 (95% CI 1.5-2.2) compared with patients without hyperoxia [71].

A randomized trial in the prehospital and emergency department settings enrolling 428 patients compared early titration of oxygen delivery with a target oxygen saturation of 90 to 94 percent versus usual care targeting an oxygen saturation 98 to 100 percent [69]. The trial was halted early because of the COVID-19 pandemic. Nevertheless, patients in the intervention group (lower target oxygen saturation) had lower survival to hospital discharge than those in the standard care arm (38 versus 48 percent, difference -10 percent [95% CI -19 to -0.2%]) and experienced significantly more episodes of hypoxia prior to intensive care unit admission (31 versus 16 percent, difference 15.2 percent [95% CI 7-23%]).

However, not all studies support the importance of avoiding hyperoxia or maintaining higher oxygen concentrations, and the optimal oxygen level in the post-arrest patient remains an area of debate [72,73]. As an example, a randomized trial conducted in the intensive care setting compared a restrictive oxygen strategy (target PaO_2 68 to 75 mmHg) with a more permissive strategy (target PaO_2 98 to 105 mmHg) in 789 patients and reported no significant difference in the primary outcomes of death or unfavorable functional recovery at 90 days [74].

Hemodynamic considerations

Maintaining end-organ perfusion — An adequate blood pressure must be maintained in the post-cardiac arrest patient. Episodes of hypotension can cause secondary injury, in addition to

exacerbating any initial insult incurred by the brain and other organs during the arrest. Mean arterial blood pressure (MAP) should be kept above 65 mmHg to reverse the acute shock state, and preferably between 80 to 100 mmHg to optimize cerebral perfusion. This is accomplished using fluid resuscitation, vasopressors, inotropes, and possibly mechanical circulatory support.

Fluid resuscitation — Volume resuscitation with isotonic crystalloid is used to maintain adequate preload. Lactated Ringer or other balanced crystalloid solutions may avoid hyperchloremic metabolic acidosis in patients requiring large-volume infusions during initial resuscitation. Responsiveness to increased preload should be assessed serially in patients who are vasopressor dependent or show signs of inadequate organ perfusion (eg, elevated serum lactate), and any hypovolemia should be corrected. Hypotonic fluids, which can exacerbate cerebral edema, should be avoided. Rapid infusion (infusion at maximal rate using a pressure bag) of 20 to 30 mL/kg of isotonic crystalloid at 4°C is commonly used for TTM. In patients with known systolic dysfunction, a smaller volume of isotonic saline may be used. (See "Novel tools for hemodynamic monitoring in critically ill patients with shock", section on 'Volume tolerance and fluid responsiveness'.)

Vasopressor and inotrope support — Vasopressor and inotrope support can mitigate the myocardial dysfunction that is common during the first 24 to 48 hours after cardiac arrest [75,76]. There is no evidence demonstrating the superiority of any one vasopressor in the post-cardiac arrest patient. Commonly employed vasopressors include norepinephrine (0.01 to 1 mcg/kg per minute; 0.5 to 80 mcg/minute) and epinephrine (0.01 to 1 mcg/kg per minute; 1 to 80 mcg/minute). The risk of cardiac arrhythmia may be higher in patients treated with dopamine. Given these data, we use norepinephrine as the first-line vasopressor in the undifferentiated post-arrest patient. (See "Use of vasopressors and inotropes".)

In cases of cardiogenic shock (eg, global hypokinesis on echocardiogram or persistently low central venous SpO_2 despite normalized hemoglobin and MAP), inotropic support using dobutamine (2 to 10 mcg/kg per minute) or milrinone (loading dose: 50 mcg/kg over 10 minutes, then 0.375 to 0.75 mcg/kg per minute) may be helpful. Either agent may cause hypotension from vasodilation; dobutamine may cause tachyarrhythmias. Mechanical circulatory support may benefit patients with inotrope-refractory cardiogenic shock

Many post-arrest patients require vasopressor therapy. A large cohort study evaluating vasopressor support during the first 24 hours after cardiac arrest, measured by the cumulative vasopressor index, reported that 47 percent of patients receive some vasopressor support [77]. Twenty-five percent of subjects receiving vasopressors required doses of norepinephrine between 0.05 and 0.1 mcg/kg per minute. In 10 percent of subjects, a dose of norepinephrine

over 0.1 mcg/kg per minute was required. Dosing up to 0.15 mcg/kg per minute is relatively common.

Determining blood pressure goals — When determining blood pressure goals, clinicians must balance the metabolic needs of an ischemic brain with the potential for overstressing a decompensated heart. The autoregulation of cerebral perfusion is altered after cardiac arrest, and higher pressures are required to maintain cerebral blood flow [78-81]. Brain perfusion declines when the MAP falls below 80 mmHg. However, if the MAP is adequate, findings from positron emission tomography (PET) studies in post-cardiac arrest patients suggest that regional cerebral perfusion matches metabolic activity [82,83].

Not all studies support the benefit of targeting a MAP of ≥80 mmHg compared with lower targets:

- A randomized trial of 120 post-arrest patients found no difference in favorable functional outcome at six months (68 versus 62 percent, respectively) between those whose MAP was kept in the 80 to 100 mmHg range and those kept in the 65 to 75 mmHg range, although some biomarkers of brain injury were lower in the higher MAP arm [84,85]. However, enrolled patients had relatively mild brain injury and thus were likely to have experienced less alteration to normal cerebral autoregulation.

- A second randomized trial of 789 patients compared MAP targets of 63 mmHg and 77 mmHg after resuscitation from out-of-hospital cardiac arrest [86]. Although assessment of demographics and outcomes suggested that patients generally sustained mild hypoxic-ischemic brain injury and thus may have had preserved cerebrovascular autoregulation, the study found no difference in functionally favorable survival at 90 days between study arms (68 versus 66 percent, respectively). Whether these findings generalize to patients with more severe brain injuries is unclear.

Preventing arrhythmia — Antiarrhythmic drugs should be reserved for patients with recurrent or ongoing unstable arrhythmias. No data support the routine or prophylactic use of antiarrhythmic drugs after the ROSC following cardiac arrest, even if such medications were employed during the resuscitation. Determining and correcting the underlying cause of the arrhythmia (eg, electrolyte disturbance, acute myocardial ischemia, toxin ingestion) is the best intervention. (See 'Identifying and treating reversible causes of cardiac arrest' above.)

Coronary revascularization — Emergency coronary catheterization or medical reperfusion therapy is indicated for post-cardiac arrest patients with findings on the electrocardiogram (ECG) of ST-elevation myocardial infarction (STEMI) or new left bundle branch block (see "Cardiac evaluation of the survivor of sudden cardiac arrest", section on 'Coronary angiography'). The

contraindications to thrombolytic therapy are reviewed separately. (See "Acute ST-elevation myocardial infarction: The use of fibrinolytic therapy", section on 'Contraindications'.)

We believe that early coronary angiography, performed as soon as possible and prior to awakening from coma, is reasonable in patients with electrical or hemodynamic instability potentially attributable to an acute coronary syndrome after resuscitation from cardiac arrest, or for patients with evidence of cardiac ischemia (eg. elevated troponin level, history of chest pain prior to collapse). Even without coronary revascularization, medical treatments for acute coronary syndrome (eg, antiplatelet and anticoagulation therapy) may be beneficial. Based on trial results presented below, we believe it is also reasonable to delay coronary angiography in unselected post-arrest patients without ST-segment elevation in favor of early resuscitation in the intensive care unit. (See "Prognosis and treatment of cardiogenic shock complicating acute myocardial infarction".)

Regardless of ECG findings, emergency coronary catheterization and/or mechanical circulatory support may be needed for patients with ongoing hemodynamic instability. Such instability may be due to cardiogenic shock or associated with rising troponin levels or evidence of focal wall-motion abnormalities on echocardiogram. Thus, we advocate coronary artery catheterization in the post-cardiac arrest patient in the absence of an obvious non-cardiac cause of arrest, with precise timing determined by the initial ECG, trajectory of cardiac biomarker levels, presence of shock, and competing needs of ongoing resuscitation procedures. Lifesaving cardiovascular procedures should not be delayed because of coma, which may take days to resolve. Immediate discussion of these issues with an interventional cardiologist is appropriate for all patients who do not have an obvious non-cardiovascular etiology of arrest. (See "Cardiac evaluation of the survivor of sudden cardiac arrest", section on 'Coronary angiography'.)

While patients with ST segment elevation are much more likely to be treated with emergency coronary angiography, some facilities perform coronary catheterization for all patients with ROSC following out-of-hospital cardiac arrest from ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT), regardless of ECG findings, because of the high incidence of acute coronary artery occlusion in this group [33,87-89]. In one series of 435 patients, over 70 percent with VF or pulseless VT had significant lesions at catheterization [34].

Some researchers advocate even more liberal criteria for performing coronary catheterization regardless of the presenting cardiac rhythm. In a meta-analysis of 11 heterogeneous, retrospective studies involving several thousand patients, over 30 percent of post-arrest patients without ST elevation on ECG were found to have acute coronary artery occlusions, regardless of their presenting rhythm [89].

However, at a population level, both immediate and urgent coronary angiography are effective strategies for management of patients without ST elevation [90,91].

- In the Coronary Angiography after Cardiac Arrest trial (COACT), in which 552 patients without ST elevation following out-of-hospital cardiac arrest (OHCA) with an initial shockable rhythm were randomized to immediate (median time 2.3 hours) or delayed (median time 121.9 hours) coronary angiography, all with percutaneous coronary intervention performed as indicated by findings, approximately 65 percent of patients had some coronary disease, but there was no improvement in survival at 90 days with early angiography [92].

- In another multicenter trial (Angiography after Out-of-Hospital Cardiac Arrest without ST-Segment Elevation [TOMAHAWK]), 554 OHCA patients without ST elevation on their initial post-arrest ECG were randomly assigned to immediate or delayed selective coronary angiography regardless of the initial cardiac rhythm. The study found no improvement in survival and a small increase in the composite outcome of death and severe neurologic deficit in the group treated with immediate coronary angiography [93].

Several registry studies have examined the potential benefits of immediate coronary angiography compared with conservative management in subgroups stratified by severity of neurologic injury on presentation. Patients with less severe hypoxic-ischemic brain injury, as determined by presenting neurologic examination or arrest characteristics, appear most likely to benefit from emergency coronary angiography [94,95]. This effect is most pronounced among those with concomitant cardiogenic shock or ST elevations [94]. By contrast, outcomes of patients with severe hypoxic-ischemic brain injury who undergo emergency coronary angiography are not improved.

Temperature management

Principles and approach — Active control of core body temperature is an important intervention for patients who achieve ROSC and are not awake (ie, do not follow verbal commands). The role and performance of active temperature control after cardiac arrest are reviewed in detail separately; basic information about indications, contraindications, initiation, and early management is provided below. (See "Intensive care unit management of the intubated post-cardiac arrest adult patient", section on 'Active temperature control'.)

Neurologic injury is the most common cause of death after cardiac arrest [96,97]. To help prevent such injury, mild hypothermia (typically between 33 and 36°C) or active normothermia (≤37.5°C) are typically used to treat for comatose survivors. Hypothermic temperature control

immediately following cardiac arrest reduces secondary brain injury through multiple mechanisms [4,5,98-100]. Conversely, hyperthermia exacerbates neurologic injury.

Indications and contraindications — Active temperature control targeting hypothermia (33 to 36°C) or normothermia should be initiated for all patients not following commands after resuscitation from cardiac arrest. The only absolute contraindication for temperature control is an advanced directive that proscribes aggressive care or a medical scenario in which such care is not appropriate. Hypothermic temperature control may be used in pregnant or hemodynamically unstable patients and those being treated with coronary catheterization or thrombolytics [33,53,101-104]. (See "Intensive care unit management of the intubated post-cardiac arrest adult patient", section on 'Active temperature control'.)

Initiation, target temperature, and duration of treatment — In post-cardiac arrest patients, active control of the patient's core temperature should be achieved as soon as possible and maintained for at least 72 hours. Clinical data strongly support avoiding fever for at least 72 hours following cardiac arrest [4,5,98,105,106]. Regardless of the strategy and target selected, continuous core temperature monitoring with a feedback mechanism is typically necessary to control patient temperature. (See "Intensive care unit management of the intubated post-cardiac arrest adult patient", section on 'Initiation'.)

We advocate temperature control to 36°C or normothermia (≤37.5°C) for 24 hours in uncomplicated patients with evidence of mild brain injury (coma with preservation of some motor response, no malignant electroencephalographic [EEG] patterns, and no evidence of cerebral edema on computed tomography [CT] scan) [98,100,107-110]. Because hypothermia causes coagulopathy, patients with active noncompressible bleeding should generally be managed with targeted normothermia or a target temperature of 36°C rather than a lower target.

We advocate hypothermic temperature control to 33°C for at least 24 hours for patients with evidence of moderate or severe brain injury (loss of motor response or brainstem reflexes, malignant EEG patterns, or early changes on CT suggesting the development of cerebral edema) [98,100,107,108,111]. While at a population level, data from randomized controlled trials do not support the superiority of any target temperature, lower temperatures may reduce cerebral edema, seizure activity, and metabolic demand and may benefit patients with these complications.

A randomized trial of 1900 patients with coma after resuscitation from cardiac arrest compared temperature control to 33°C versus targeted normothermia maintained with a comprehensive care bundle (including core temperature monitoring, attentive fever prevention with rapidly

escalating intensity of intervention) [100]. Survival at six months did not differ by treatment arm (50 versus 48 percent, respectively). Nearly one-half of patients randomized to normothermia required use of a device for active temperature management (69 percent surface cooling, 31 percent endovascular cooling). At institutions with well-developed pathways for post-arrest care including targeted normothermia protocols, this treatment strategy is equally efficacious at a population level. Whether targeted normothermia is effective in subgroups who were excluded or infrequently enrolled from this study remains unknown.

Multiple large observational studies have shown an interaction between severity of hypoxic ischemic injury and optimal target temperature for post-arrest temperature control. Specifically, a target temperature of 33°C is associated with improved outcomes among patients with moderate to severe hypoxic ischemic injury, whether quantified by presenting neurological exam and extracerebral organ failure, EEG, or arrest characteristics. Conversely, among patients with mild hypoxic ischemic injury, a target temperature of 36°C or normothermia may be superior.

Methods of induction — When performing active temperature control, clinicians should use endovascular or surface methods to control temperature that are readily available and familiar. Even when targeted normothermia is chosen as the treatment strategy, a cooling device is required in nearly one-half of patients. Maintenance of target temperature is discussed separately, but an active feedback loop, whereby core temperature is monitored and used to modulate the intensity of cooling, is required. (See "Intensive care unit management of the intubated post-cardiac arrest adult patient", section on 'Maintenance'.)

Many patients are already mildly hypothermic (35 to 35.5°C) after the ROSC from the mixing of cooler peripheral blood with core blood [4,5]. Therefore, minimally invasive techniques can often achieve desired temperatures quickly. A brief description of methods for implementing active temperature control are described below; more detailed explanations are provided separately. (See "Intensive care unit management of the intubated post-cardiac arrest adult patient", section on 'Initiation'.)

When patients present with a core temperature above the desired target, the authors infuse 1 to 2 L of cold isotonic saline using a pressure bag while simultaneously implementing surface cooling using cooling blankets above and below the patient and ice packs applied to the axillae, groin, and neck (adjacent to major blood vessels).

Intravenous (IV) infusion of 30 mL/kg of cold (4°C [39°F]), balanced, isotonic crystalloid, using a pressure bag to increase the rate of administration, reduces the core temperature by >2°C per hour [112-114]. One liter of 4°C crystalloid infused via pressure bag over approximately 15

minutes can drop the core temperature by approximately 1°C. The rate of temperature reduction using this method is comparable with or faster than that achieved with endovascular catheters but may result in pulmonary edema and increased diuretic use [115,116].

We recommend against aggressive administration of cold crystalloid in the prehospital setting or in the first minutes after ROSC, when physiologic reserve and myocardial function may be reduced. A randomized trial of 1359 unconscious adults resuscitated from out-of-hospital cardiac arrest compared prehospital induction of hypothermia with rapid infusion of up to 2 liters of cold isotonic saline, sedation, and neuromuscular blockade versus standard prehospital post-arrest care [116]. While the intervention decreased presenting temperature by >1°C, it provided no survival benefit and was associated with higher rates of rearrest and pulmonary edema.

Patients with a history of heart failure or severely compromised kidney function, or signs of acute pulmonary edema, should **not** receive rapid infusions of fluid to induce hypothermia regardless of timing or location. Surface cooling measures or an IV cooling device should be used instead. Surface cooling methods, including ice packs, cooling blankets, and gel-adhesive pads, can reduce the core body temperature ≥1°C per hour.

When inducing hypothermic temperature control, shivering is common and may be too subtle to appreciate on visual inspection. Therefore, sedation and neuromuscular blockade are often required to facilitate cooling. (See 'Sedation and suppression of shivering' below.)

Sedation and suppression of shivering — Shivering raises body temperature and must be suppressed in patients being treated with active temperature control [109,117-119]. Failure to suppress shivering is a common reason for delays in achieving goal temperature. Therefore, we titrate sedation to shivering suppression rather than using standard sedation scales. High doses of sedatives or neuromuscular blockade are necessary to accomplish this.

A continuous infusion of propofol and fentanyl is one effective approach to sedation [120]. We start with a propofol infusion at 20 mcg/kg per minute and titrate as needed to a maximum dose of 50 mcg/kg per minute. If this is ineffective, we add fentanyl in boluses of 0.5 to 1 mcg/kg or as a continuous infusion starting between 25 and 100 mcg/hour.

In hypotensive patients, a continuous infusion of midazolam (2 to 10 mg/hour) is an effective alternative to propofol, but accumulation of this drug may interfere with subsequent neurologic evaluation [121-123]. Hypothermia causes a decrease in the metabolism and excretion of midazolam. Days may be required before the drug is cleared after the infusion is stopped [124].

Intermittent treatment with meperidine can suppress shivering, but the proconvulsant effects of its primary metabolite normeperidine make this drug unappealing, particularly in post-cardiac arrest patients. Moreover, meperidine is not recommended in patients with kidney dysfunction, which is common in patients following cardiac arrest. Dexmedetomidine has been shown to suppress the shivering threshold in healthy individuals, but its use is limited by the side effects of hypotension and bradycardia [125].

Neuromuscular blockade is highly effective at suppressing shivering. A single IV bolus dose of 1 mg/kg of rocuronium during induction of hypothermic temperature control is generally safe and well tolerated. Continuous neuromuscular blockade may be required to stop shivering but can mask seizures, which develop in a substantial percentage of post-cardiac arrest patients [4,6,126-129]. We recommend continuous EEG monitoring during continuous neuromuscular blockade. (See "Intensive care unit management of the intubated post-cardiac arrest adult patient", section on 'Neurologic considerations'.)

Temperature monitoring and rewarming — Core body temperature should be monitored continuously during active temperature management. Core temperature is a close approximation of brain temperature [130]. The gold standard for core temperature measurement is central venous temperature, but several surrogates are available. In order of preference, surrogate monitoring methods include esophageal, bladder, or rectal probes [131].

Esophageal temperature measurement is the most accurate surrogate method used to follow core temperature during the induction of hypothermic temperature control [131,132]. Bladder temperature may be erroneous if urine output falls below 0.5 mL/kg per hour. Rectal measurements may lag behind acute changes in core temperature by up to 1.5°C [131]. Many rectal temperature probes can also be placed in the esophagus, yielding a more accurate measurement. Axillary and tympanic measurements are inadequate and misleading and should not be used.

Rewarming from TTM is discussed separately. (See "Intensive care unit management of the intubated post-cardiac arrest adult patient", section on 'Rewarming'.)

Potential adverse effects — Generally, temperature control to 33 or 36°C is safe and well tolerated. Targeting a temperature of 33°C increases the risk of arrhythmia compared with targeted normothermia (24 versus 16 percent, respectively), although the clinical significance of these arrhythmias is uncertain [109]. Among unselected patients (ie, those typically excluded from clinical trials), the most significant potential adverse effect of hypothermic temperature control is impaired coagulation. Complications may be more common when targeting 33°C than 36°C. The risks of hypothermia are discussed in greater detail separately; a brief summary is

provided below. (See "Intensive care unit management of the intubated post-cardiac arrest adult patient", section on 'Adverse effects'.)

Potential adverse effects of mild hypothermia include the following:

- Mild coagulopathy
- Increased risk of infection, especially pneumonia
- Increased risk of bradyarrhythmia
- Hyperglycemia
- Hypokalemia
- Cold diuresis
- Slowed metabolism and excretion of medications

At temperatures below 35°C, clotting enzymes operate more slowly, and platelets function less effectively [133-136]. As a result, minor bleeding is seen in up to 20 percent of patients treated with hypothermia, although transfusion is rarely required [53,137]. In the event of significant bleeding (eg, hemodynamic instability, intracranial hemorrhage, noncompressible site), the target temperature is 36°C. Patients colder than this should be rewarmed to 36°C to correct cold-induced coagulopathy.

Hypothermia impairs leukocyte function. The incidence of significant infection is likely to increase if hypothermia is maintained longer than 24 hours. While an increase in infection rates has been noted in several cohorts treated with 24 hours of hypothermic temperature control [4,53,138], these infections were not associated with increased mortality.

Hypothermia slows cardiac conduction and can provoke arrhythmias, including bradycardia and QT interval prolongation [139]. Mild asymptomatic bradycardia (eg, heart rate in 40s) is common at 33°C and does not require intervention if the blood pressure is acceptable. If intervention is needed for VF or pulseless VT, animal studies report similar or improved first-shock success with defibrillation in specimens with mild hypothermia compared with those with normothermia [140,141]. Temperature control to 33°C is not associated with an increased need for vasopressor support compared with targeted normothermia or historical controls [77,109,142].

Hyperglycemia due to insulin resistance has been noted during hypothermia [139,143]. Higher doses of insulin may be needed in hyperglycemic, hypothermic patients. (See 'Glycemic control' below.)

Hypothermia leads to a "cold diuresis," which can contribute to hypovolemia, hypokalemia, hypomagnesaemia, and hypophosphatemia [144]. In addition, temperature fluctuations during

the induction of hypothermic temperature control and rewarming cause potassium to move between the extracellular and intracellular compartments [144-146]. Therefore, careful monitoring of volume status and measurement of basic electrolytes approximately every three to four hours during hypothermia is prudent. Hypokalemia is more frequently encountered in patients maintained at 33°C [98].

Hypothermia slows the metabolism and excretion of many drugs, and thus, their duration of effect may be prolonged [147-149].

OTHER EARLY INTERVENTIONS

Basic interventions — Raise the head of the bed to 30 degrees. This helps to prevent aspiration and lowers intracranial pressure. (See "Evaluation and management of elevated intracranial pressure in adults", section on 'Position'.)

Antibiotic therapy and prophylaxis — Pneumonia is common among survivors of cardiac arrest. We believe a short course of empiric antibiotics targeting community-acquired pathogens is reasonable in patients with significant aspiration or signs of disease. In the absence of such signs, we do not routinely give prophylactic antibiotics.

In a single-center, randomized trial of 198 patients resuscitated from ventricular tachycardia (VT)/ventricular fibrillation (VF) out-of-hospital cardiac arrest, treatment with two days of empiric intravenous (IV) antibiotics (amoxicillin-clavulanate) resulted in a 15 percent reduction in early ventilator associated pneumonia compared with placebo (19 versus 34, respectively) but did not reduce mortality [150]. (See "Intensive care unit management of the intubated post-cardiac arrest adult patient", section on 'Antibiotic therapy and prophylaxis'.)

Treatment of pneumonia in critically ill patients is discussed separately. (See "Treatment of hospital-acquired and ventilator-associated pneumonia in adults".)

Glycemic control — Maintain serum glucose between 140 and 180 mg/dL (7.8 and 10 mmol/L) during the period following cardiac arrest, and strive to avoid hypoglycemic episodes. Hyperglycemia is associated with worse outcomes in post-cardiac arrest patients [151,152]. (See "Glycemic control in critically ill adult and pediatric patients".)

There is no additional benefit from tight control of the serum glucose (70 to 108 mg/dL; 3.9 to 6 mmol/L) compared with more liberal management (108 to 144 mg/dL; 6 to 8.1 mmol/L) following cardiac arrest [153]. Multiple studies highlight the increased risk of hypoglycemia when lower target ranges are used [153,154].

Seizures and myoclonic jerks — Seizure activity and myoclonic jerks are common after cardiac arrest. While post-arrest myoclonus is often a marker of more severe brain injury, up to 22 percent of patients with myoclonus after cardiac arrest may recover [155-159]. Seizure monitoring and treatment following cardiac arrest is reviewed in detail separately; important aspects of acute management are discussed briefly below. (See "Intensive care unit management of the intubated post-cardiac arrest adult patient", section on 'Neurologic considerations'.)

Electroencephalogram (EEG) monitoring is recommended for comatose post-arrest patients both to guide appropriate therapy and for prognostication. EEG can distinguish between recoverable and irrecoverable patterns of post-anoxic myoclonus, while bedside examination cannot accomplish this consistently [155].

In the acute setting, it is reasonable to use a sedative with anticonvulsant properties, such as propofol or midazolam, to suppress possible seizure activity. Such patients require ongoing EEG monitoring.

The role of anticonvulsant medication for the prevention of post-arrest myoclonus, seizures, and status epilepticus is controversial. High-quality evidence is scant. Some epileptiform EEG activity may respond to treatment, and case series report favorable outcomes after aggressive therapy with anticonvulsants in certain subgroups [160,161]. A randomized controlled trial of 172 patients compared aggressive, tiered use of antiseizure medicines with standard care in patients with rhythmic and periodic EEG activity after cardiac arrest and found no difference in the primary outcome of unfavorable Cerebral Performance Category at three months (90 versus 92 percent) [162]. A post hoc analysis suggested heterogeneity of treatment effect across initial EEG findings, but the study was not designed or powered to make this comparison.

EARLY RISK STRATIFICATION, FAMILY COMMUNICATION, AND DISPOSITION

Prognosis following cardiac arrest is discussed in greater detail separately. (See "Prognosis and outcomes following sudden cardiac arrest in adults".)

Assessment of brain injury — Many cardiac arrest patients sustain a nonsurvivable brain injury, and initial illness severity is strongly associated with survival and neurologic outcome [28]. Neurologic examination and ancillary diagnostic tests (eg, brain imaging, electroencephalography [EEG]) can eventually identify patients without a chance of favorable recovery. However, during the first 72 hours after cardiac arrest, available evidence suggests no combination of clinical signs and diagnostic test results is sufficient to preclude a functionally

favorable recovery. Thus, while early risk stratification is possible, initial assessment of brain injury should not be used as a justification to limit or withdraw critical care. The neurologic assessment of post-cardiac arrest patients, including clinical evaluation and ancillary testing, is discussed in detail separately. (See "Hypoxic-ischemic brain injury in adults: Evaluation and prognosis".)

Initial family communication — Sudden cardiac arrest (SCA) is often a catastrophic, unexpected event. Depending upon the severity of brain injury, mortality for patients who survive to hospital care ranges from 20 to 90 percent [28]. At the same time, early prognostication is difficult, and premature withdrawal of life-sustaining therapies based on a perceived poor neurologic prognosis contributes to avoidable deaths [97,163].

Family communication early after cardiac arrest should focus on providing basic updates about the patient's clinical status. Thereafter, it is helpful to identify any pre-existing values and preferences the patient may have expressed regarding the acceptability of at least short-term critical care. In the absence of prior patient wishes to forego life-sustaining therapies, the anticipated clinical course can be outlined. Given the dynamic nature of post-arrest physiology, it is generally our practice to provide a broad overview during initial communication without focusing on speculative details. It is often helpful for families to anticipate at least several days of critical care; for comatose patients, the prognosis may remain unknown for at least three to five days. (See "Palliative care: Issues in the intensive care unit in adults".)

PATIENT DISPOSITION

Virtually all patients resuscitated from cardiac arrest require critical care. Depending upon the arrest etiology and success of initial resuscitative efforts, many also require emergency coronary angiography, surgery, or mechanical circulatory support prior to being transferred to the intensive care unit.

SPECIALTY CARE

Post-cardiac arrest care requires significant resources and coordination among multiple specialties. Multiple observational studies demonstrate improved short- and long-term outcomes when patients receive care at hospitals with expertise that treat larger numbers of cardiac arrest patients [164-172]. Regionalized centers for post-cardiac arrest patients have been proposed [173]. Particular hospital resources, such as cardiac catheterization facilities, are associated with more favorable outcomes. Since most hospitals see between 10 and 15 post-

cardiac arrest patients per year and some do not have continuous access to cardiac catheterization or electroencephalogram (EEG) monitoring, it is reasonable to transfer patients resuscitated from cardiac arrest to suitable tertiary care centers whenever feasible.

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Basic and advanced cardiac life support in adults".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient education: Sudden cardiac arrest (The Basics)")

SUMMARY AND RECOMMENDATIONS

- **Management algorithm and reversible causes** – We provide a management algorithm for the patient resuscitated from sudden cardiac arrest (SCA) (algorithm 1) and a table summarizing reversible causes (table 1).
- **Initial stabilization** – The most immediate threat to survival during the first minutes to hours after SCA is cardiovascular collapse. Mean arterial blood pressure (MAP) should be kept above 65 mmHg and preferably between 80 and 100 mmHg to ensure perfusion of the brain.

Most patients resuscitated from SCA respond to moderate volume resuscitation (eg, 1 to 2 L isotonic crystalloid given via rapid bolus). Less volume is given if there is a history or signs of heart failure. With hypotensive patients, vasopressor therapy is initiated simultaneously with volume resuscitation. Bolus administration of 10 to 100 mcg epinephrine may be given as a bridge to a continuous infusion (eg, norepinephrine). (See 'Initial stabilization and prevention of rearrest' above and 'Ongoing stabilization and prevention of brain injury' above.)

Additional early goals include optimizing oxygenation (oxygen saturation [SpO_2] >92 percent) and correcting electrolyte abnormalities. Ventilation using a bag-valve-mask or supraglottic airway is often sufficient, and tracheal intubation briefly deferred, while initial interventions to maintain hemodynamic stability and treat reversible causes are performed. After intubation, goals include maintaining a carbon dioxide tension ($PaCO_2$) of 40 to 50 mmHg (end-tidal carbon dioxide [$EtCO_2$] 35 to 45) and SpO_2 >94 percent.

Recurrent ventricular tachycardia (VT) or ventricular fibrillation (VF) are managed according to advanced cardiac life support (ACLS) protocols (algorithm 2).

- **History and examination** – A focused history and physical examination are performed to identify possible causes and ongoing or imminent threats to life. A broad differential diagnosis should be considered. Common etiologies, including reversible causes, for SCA are described in the attached tables (table 2 and table 1). (See 'History' above and 'Physical examination' above.)

  A baseline neurologic examination helps to determine the cause, clinical course, and need for temperature management. Cessation of neuromuscular blockade and sedation is necessary for a valid examination. Brainstem responses (pupillary, corneal, oculocephalic, gag, cough) and motor responses should be assessed. Asymmetric neurologic findings are **not** expected following return of spontaneous circulation (ROSC) and suggest a structural intracranial lesion.

- **Diagnostic testing** – Immediately following the ROSC, a 12-lead electrocardiogram (ECG) should be obtained and evaluated for signs of ST-elevation myocardial infarction (STEMI; including a new left bundle branch block), although sensitivity and specificity are limited following SCA. (See 'Electrocardiogram' above and 'Imaging studies' above and 'Laboratory testing' above.)

  Commonly performed testing on presentation includes arterial blood gas, basic electrolytes, blood counts, serum troponin, serum lactate, basic kidney and liver function studies, and possibly toxicology. Imaging studies to obtain include chest radiograph and

ultrasound, which can help to diagnose pericardial tamponade, pneumothorax, catastrophic pulmonary embolism, and intraperitoneal bleeding. Liberal use of computed tomography (CT; head, cervical spine, chest, abdomen) is encouraged given the prevalence of pathologic findings.

- **Coronary revascularization** – Emergency coronary catheterization or medical reperfusion therapy is indicated for patients with ECG findings of STEMI. Regardless of ECG findings, catheterization may be needed for patients with ongoing hemodynamic instability or rising troponin levels or evidence of focal wall-motion abnormalities on echocardiogram. Lifesaving cardiovascular procedures should never be delayed because of coma. (See 'Coronary revascularization' above.)

- **Temperature control to minimize brain injury** – To reduce the risk of neurologic injury, temperature control should be initiated after initial cardiopulmonary stabilization for all patients who are not awake (ie, do not follow verbal commands). (See 'Temperature management' above.)

  - **Mild brain injury** – For patients with evidence of mild to moderate brain injury (coma with some motor response, no malignant electroencephalographic [EEG] patterns, and no evidence of cerebral edema on CT), we advocate a target core temperature of 36°C. At hospitals with protocols and experience delivering targeted normothermia, this is an equally efficacious strategy for these patients. Patients with active noncompressible bleeding should generally be managed with a target temperature of 36°C or targeted normothermia.

  - **Moderate or severe brain injury** – For patients with evidence of severe brain injury (loss of motor response or brainstem reflexes, malignant EEG patterns, or early changes on CT suggesting cerebral edema), we advocate a target core temperature of 33°C.

  Temperature control can be initiated with infusions of cold isotonic saline and surface cooling (eg, ice packs). Shivering must be prevented, most often using sedation (eg, propofol) and neuromuscular blockade. Core temperature must be monitored continuously. Details are provided in the text. (See 'Methods of induction' above and 'Sedation and suppression of shivering' above and 'Temperature monitoring and rewarming' above.)

  Potential adverse effects of mild hypothermia include:

  - Mild coagulopathy

- Increased risk of infection, especially pneumonia
- Increased risk of bradyarrhythmia
- Hyperglycemia
- Hypokalemia
- Cold diuresis
- Slowed metabolism and excretion of medications

- **Other management** – Early care includes:
  - Raise head of bed to 30 degrees.
  - Give a short course of empiric antibiotics targeting community-acquired pathogens to patients with significant aspiration or signs of disease. (See 'Antibiotic therapy and prophylaxis' above.)
  - Maintain serum glucose between 140 and 180 mg/dL (7.8 and 10 mmol/L).
  - Perform EEG monitoring for comatose patients. Use anticonvulsants to treat epileptiform EEG activity. (See 'Seizures and myoclonic jerks' above.)
- **Specialty care and transfer** – Specialty care at high-volume centers is strongly associated with improved outcomes after SCA. Transfer patients to specialty centers when feasible.

Use of UpToDate is subject to the Terms of Use.

REFERENCES

1. Virani SS, Alonso A, Aparicio HJ, et al. Heart Disease and Stroke Statistics-2021 Update: A Report From the American Heart Association. Circulation 2021; 143:e254.
2. Andersen LW, Holmberg MJ, Berg KM, et al. In-Hospital Cardiac Arrest: A Review. JAMA 2019; 321:1200.
3. Buick JE, Drennan IR, Scales DC, et al. Improving Temporal Trends in Survival and Neurological Outcomes After Out-of-Hospital Cardiac Arrest. Circ Cardiovasc Qual Outcomes 2018; 11:e003561.
4. Hypothermia after Cardiac Arrest Study Group. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. N Engl J Med 2002; 346:549.
5. Bernard SA, Gray TW, Buist MD, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. N Engl J Med 2002; 346:557.

6. Sunde K, Pytte M, Jacobsen D, et al. Implementation of a standardised treatment protocol for post resuscitation care after out-of-hospital cardiac arrest. Resuscitation 2007; 73:29.
7. Rittenberger JC, Guyette FX, Tisherman SA, et al. Outcomes of a hospital-wide plan to improve care of comatose survivors of cardiac arrest. Resuscitation 2008; 79:198.
8. Tagami T, Hirata K, Takeshige T, et al. Implementation of the fifth link of the chain of survival concept for out-of-hospital cardiac arrest. Circulation 2012; 126:589.
9. Salcido DD, Schmicker RH, Buick JE, et al. Compression-to-ventilation ratio and incidence of rearrest-A secondary analysis of the ROC CCC trial. Resuscitation 2017; 115:68.
10. Salcido DD, Schmicker RH, Kime N, et al. Effects of intra-resuscitation antiarrhythmic administration on rearrest occurrence and intra-resuscitation ECG characteristics in the ROC ALPS trial. Resuscitation 2018; 129:6.
11. Kaji AH, Hanif AM, Thomas JL, Niemann JT. Out-of-hospital cardiac arrest: early in-hospital hypotension versus out-of-hospital factors in predicting in-hospital mortality among those surviving to hospital admission. Resuscitation 2011; 82:1314.
12. Trzeciak S, Jones AE, Kilgannon JH, et al. Significance of arterial hypotension after resuscitation from cardiac arrest. Crit Care Med 2009; 37:2895.
13. Javaudin F, Desce N, Le Bastard Q, et al. Impact of pre-hospital vital parameters on the neurological outcome of out-of-hospital cardiac arrest: Results from the French National Cardiac Arrest Registry. Resuscitation 2018; 133:5.
14. Jentzer JC, Hollenberg SM. Vasopressor and Inotrope Therapy in Cardiac Critical Care. J Intensive Care Med 2021; 36:843.
15. Weingart S. Push-dose pressors for immediate blood pressure control. Clin Exp Emerg Med 2015; 2:131.
16. Rotando A, Picard L, Delibert S, et al. Push dose pressors: Experience in critically ill patients outside of the operating room. Am J Emerg Med 2019; 37:494.
17. Lupton JR, Schmicker RH, Stephens S, et al. Outcomes With the Use of Bag-Valve-Mask Ventilation During Out-of-hospital Cardiac Arrest in the Pragmatic Airway Resuscitation Trial. Acad Emerg Med 2020; 27:366.
18. Jabre P, Penaloza A, Pinero D, et al. Effect of Bag-Mask Ventilation vs Endotracheal Intubation During Cardiopulmonary Resuscitation on Neurological Outcome After Out-of-Hospital Cardiorespiratory Arrest: A Randomized Clinical Trial. JAMA 2018; 319:779.
19. Benger JR, Kirby K, Black S, et al. Effect of a Strategy of a Supraglottic Airway Device vs Tracheal Intubation During Out-of-Hospital Cardiac Arrest on Functional Outcome: The AIRWAYS-2 Randomized Clinical Trial. JAMA 2018; 320:779.

20. Wang HE, Schmicker RH, Daya MR, et al. Effect of a Strategy of Initial Laryngeal Tube Insertion vs Endotracheal Intubation on 72-Hour Survival in Adults With Out-of-Hospital Cardiac Arrest: A Randomized Clinical Trial. JAMA 2018; 320:769.
21. Kim JM, Shin TG, Hwang SY, et al. Sedative dose and patient variable impacts on postintubation hypotension in emergency airway management. Am J Emerg Med 2019; 37:1248.
22. Smischney NJ, Nicholson WT, Brown DR, et al. Ketamine/propofol admixture vs etomidate for intubation in the critically ill: KEEP PACE Randomized clinical trial. J Trauma Acute Care Surg 2019; 87:883.
23. Kudenchuk PJ, Brown SP, Daya M, et al. Amiodarone, Lidocaine, or Placebo in Out-of-Hospital Cardiac Arrest. N Engl J Med 2016; 374:1711.
24. Panchal AR, Berg KM, Kudenchuk PJ, et al. 2018 American Heart Association Focused Update on Advanced Cardiovascular Life Support Use of Antiarrhythmic Drugs During and Immediately After Cardiac Arrest: An Update to the American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2018; 138:e740.
25. Bartos JA, Carlson K, Carlson C, et al. Surviving refractory out-of-hospital ventricular fibrillation cardiac arrest: Critical care and extracorporeal membrane oxygenation management. Resuscitation 2018; 132:47.
26. Yannopoulos D, Bartos J, Raveendran G, et al. Advanced reperfusion strategies for patients with out-of-hospital cardiac arrest and refractory ventricular fibrillation (ARREST): a phase 2, single centre, open-label, randomised controlled trial. Lancet 2020; 396:1807.
27. Chen N, Callaway CW, Guyette FX, et al. Arrest etiology among patients resuscitated from cardiac arrest. Resuscitation 2018; 130:33.
28. Rittenberger JC, Tisherman SA, Holm MB, et al. An early, novel illness severity score to predict outcome after cardiac arrest. Resuscitation 2011; 82:1399.
29. Javaudin F, Leclere B, Segard J, et al. Prognostic performance of early absence of pupillary light reaction after recovery of out of hospital cardiac arrest. Resuscitation 2018; 127:8.
30. Nolan JP, Sandroni C, Böttiger BW, et al. European Resuscitation Council and European Society of Intensive Care Medicine guidelines 2021: post-resuscitation care. Intensive Care Med 2021; 47:369.
31. Panchal AR, Bartos JA, Cabañas JG, et al. Part 3: Adult Basic and Advanced Life Support: 2020 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2020; 142:S366.

32. McFadden P, Reynolds JC, Madder RD, Brown M. Diagnostic test accuracy of the initial electrocardiogram after resuscitation from cardiac arrest to indicate invasive coronary angiographic findings and attempted revascularization: A systematic review and meta-analysis. Resuscitation 2021; 160:20.
33. Reynolds JC, Callaway CW, El Khoudary SR, et al. Coronary angiography predicts improved outcome following cardiac arrest: propensity-adjusted analysis. J Intensive Care Med 2009; 24:179.
34. Dumas F, Cariou A, Manzo-Silberman S, et al. Immediate percutaneous coronary intervention is associated with better survival after out-of-hospital cardiac arrest: insights from the PROCAT (Parisian Region Out of hospital Cardiac ArresT) registry. Circ Cardiovasc Interv 2010; 3:200.
35. Spaulding CM, Joly LM, Rosenberg A, et al. Immediate coronary angiography in survivors of out-of-hospital cardiac arrest. N Engl J Med 1997; 336:1629.
36. Baldi E, Schnaubelt S, Caputo ML, et al. Association of Timing of Electrocardiogram Acquisition After Return of Spontaneous Circulation With Coronary Angiography Findings in Patients With Out-of-Hospital Cardiac Arrest. JAMA Netw Open 2021; 4:e2032875.
37. Donnino MW, Miller J, Goyal N, et al. Effective lactate clearance is associated with improved outcome in post-cardiac arrest patients. Resuscitation 2007; 75:229.
38. Moore CL, Copel JA. Point-of-care ultrasonography. N Engl J Med 2011; 364:749.
39. Rose JS, Bair AE, Mandavia D, Kinser DJ. The UHP ultrasound protocol: a novel ultrasound approach to the empiric evaluation of the undifferentiated hypotensive patient. Am J Emerg Med 2001; 19:299.
40. Jones AE, Tayal VS, Sullivan DM, Kline JA. Randomized, controlled trial of immediate versus delayed goal-directed ultrasound to identify the cause of nontraumatic hypotension in emergency department patients. Crit Care Med 2004; 32:1703.
41. Scalea TM, Rodriguez A, Chiu WC, et al. Focused Assessment with Sonography for Trauma (FAST): results from an international consensus conference. J Trauma 1999; 46:466.
42. Kirkpatrick AW, Sirois M, Laupland KB, et al. Hand-held thoracic sonography for detecting post-traumatic pneumothoraces: the Extended Focused Assessment with Sonography for Trauma (EFAST). J Trauma 2004; 57:288.
43. Petek BJ, Erley CL, Kudenchuk PJ, et al. Diagnostic yield of non-invasive imaging in patients following non-traumatic out-of-hospital sudden cardiac arrest: A systematic review. Resuscitation 2019; 135:183.

44. Branch KRH, Strote J, Gunn M, et al. Early head-to-pelvis computed tomography in out-of-hospital circulatory arrest without obvious etiology. Acad Emerg Med 2021; 28:394.
45. Viniol S, Thomas RP, König AM, et al. Early whole-body CT for treatment guidance in patients with return of spontaneous circulation after cardiac arrest. Emerg Radiol 2020; 27:23.
46. Branch KR, Nguyen ML, Kudenchuk PJ, Johnson NJ. Head-to-pelvis CT imaging after sudden cardiac arrest: Current status and future directions. Resuscitation 2023; 191:109916.
47. Metter RB, Rittenberger JC, Guyette FX, Callaway CW. Association between a quantitative CT scan measure of brain edema and outcome after cardiac arrest. Resuscitation 2011; 82:1180.
48. Torbey MT, Selim M, Knorr J, et al. Quantitative analysis of the loss of distinction between gray and white matter in comatose patients after cardiac arrest. Stroke 2000; 31:2163.
49. Yanagawa Y, Un-no Y, Sakamoto T, Okada Y. Cerebral density on CT immediately after a successful resuscitation of cardiopulmonary arrest correlates with outcome. Resuscitation 2005; 64:97.
50. Lee KY, So WZ, Ho JSY, et al. Prevalence of intracranial hemorrhage amongst patients presenting with out-of-hospital cardiac arrest: A systematic review and meta-analysis. Resuscitation 2022; 176:136.
51. Mayà-Casalprim G, Ortiz J, Tercero A, et al. Cervical spinal cord injury by a low-impact trauma as an unnoticed cause of cardiorespiratory arrest. Eur Heart J Case Rep 2020; 4:1.
52. Jang SJ, Cha YK, Kim JS, et al. Computed tomographic findings of chest injuries following cardiopulmonary resuscitation: More complications for prolonged chest compressions? Medicine (Baltimore) 2020; 99:e21685.
53. Nielsen N, Hovdenes J, Nilsson F, et al. Outcome, timing and adverse events in therapeutic hypothermia after out-of-hospital cardiac arrest. Acta Anaesthesiol Scand 2009; 53:926.
54. Karatasakis A, Sarikaya B, Liu L, et al. Prevalence and Patterns of Resuscitation-Associated Injury Detected by Head-to-Pelvis Computed Tomography After Successful Out-of-Hospital Cardiac Arrest Resuscitation. J Am Heart Assoc 2022; 11:e023949.
55. Kågström E, Smith ML, Siesjö BK. Cerebral circulatory responses to hypercapnia and hypoxia in the recovery period following complete and incomplete cerebral ischemia in the rat. Acta Physiol Scand 1983; 118:281.
56. Bisschops LL, Hoedemaekers CW, Simons KS, van der Hoeven JG. Preserved metabolic coupling and cerebrovascular reactivity during mild hypothermia after cardiac arrest. Crit Care Med 2010; 38:1542.

57. Buunk G, van der Hoeven JG, Frölich M, Meinders AE. Cerebral vasoconstriction in comatose patients resuscitated from a cardiac arrest? Intensive Care Med 1996; 22:1191.
58. Buunk G, van der Hoeven JG, Meinders AE. Cerebrovascular reactivity in comatose patients resuscitated from a cardiac arrest. Stroke 1997; 28:1569.
59. Eastwood GM, Schneider AG, Suzuki S, et al. Targeted therapeutic mild hypercapnia after cardiac arrest: A phase II multi-centre randomised controlled trial (the CCC trial). Resuscitation 2016; 104:83.
60. McKenzie N, Williams TA, Tohira H, et al. A systematic review and meta-analysis of the association between arterial carbon dioxide tension and outcomes after cardiac arrest. Resuscitation 2017; 111:116.
61. McGuigan PJ, Shankar-Hari M, Harrison DA, et al. The interaction between arterial oxygenation and carbon dioxide and hospital mortality following out of hospital cardiac arrest: a cohort study. Crit Care 2020; 24:336.
62. Hope Kilgannon J, Hunter BR, Puskarich MA, et al. Partial pressure of arterial carbon dioxide after resuscitation from cardiac arrest and neurological outcome: A prospective multi-center protocol-directed cohort study. Resuscitation 2019; 135:212.
63. Eastwood G, Nichol AD, Hodgson C, et al. Mild Hypercapnia or Normocapnia after Out-of-Hospital Cardiac Arrest. N Engl J Med 2023; 389:45.
64. Voicu S, Deye N, Malissin I, et al. Influence of α-stat and pH-stat blood gas management strategies on cerebral blood flow and oxygenation in patients treated with therapeutic hypothermia after out-of-hospital cardiac arrest: a crossover study. Crit Care Med 2014; 42:1849.
65. Hoedemaekers C, van der Hoeven JG. Is α-stat or pH-stat the best strategy during hypothermia after cardiac arrest?*. Crit Care Med 2014; 42:1950.
66. Wang HE, Prince DK, Drennan IR, et al. Post-resuscitation arterial oxygen and carbon dioxide and outcomes after out-of-hospital cardiac arrest. Resuscitation 2017; 120:113.
67. Elmer J, Scutella M, Pullalarevu R, et al. The association between hyperoxia and patient outcomes after cardiac arrest: analysis of a high-resolution database. Intensive Care Med 2015; 41:49.
68. Zhou DW, Li ZM, Zhang SL, et al. The optimal peripheral oxygen saturation may be 95-97% for post-cardiac arrest patients: A retrospective observational study. Am J Emerg Med 2021; 40:120.
69. Bernard SA, Bray JE, Smith K, et al. Effect of Lower vs Higher Oxygen Saturation Targets on Survival to Hospital Discharge Among Patients Resuscitated After Out-of-Hospital Cardiac

Arrest: The EXACT Randomized Clinical Trial. JAMA 2022; 328:1818.

70. Wang CH, Chang WT, Huang CH, et al. The effect of hyperoxia on survival following adult cardiac arrest: a systematic review and meta-analysis of observational studies. Resuscitation 2014; 85:1142.
71. Kilgannon JH, Jones AE, Shapiro NI, et al. Association between arterial hyperoxia following resuscitation from cardiac arrest and in-hospital mortality. JAMA 2010; 303:2165.
72. Bellomo R, Bailey M, Eastwood GM, et al. Arterial hyperoxia and in-hospital mortality after resuscitation from cardiac arrest. Crit Care 2011; 15:R90.
73. Kuisma M, Boyd J, Voipio V, et al. Comparison of 30 and the 100% inspired oxygen concentrations during early post-resuscitation period: a randomised controlled pilot study. Resuscitation 2006; 69:199.
74. Schmidt H, Kjaergaard J, Hassager C, et al. Oxygen Targets in Comatose Survivors of Cardiac Arrest. N Engl J Med 2022; 387:1467.
75. Laurent I, Monchi M, Chiche JD, et al. Reversible myocardial dysfunction in survivors of out-of-hospital cardiac arrest. J Am Coll Cardiol 2002; 40:2110.
76. Ruiz-Bailén M, Aguayo de Hoyos E, Ruiz-Navarro S, et al. Reversible myocardial dysfunction after cardiopulmonary resuscitation. Resuscitation 2005; 66:175.
77. Huynh N, Kloke J, Gu C, et al. The effect of hypothermia "dose" on vasopressor requirements and outcome after cardiac arrest. Resuscitation 2013; 84:189.
78. Nishizawa H, Kudoh I. Cerebral autoregulation is impaired in patients resuscitated after cardiac arrest. Acta Anaesthesiol Scand 1996; 40:1149.
79. Sundgreen C, Larsen FS, Herzog TM, et al. Autoregulation of cerebral blood flow in patients resuscitated from cardiac arrest. Stroke 2001; 32:128.
80. van den Brule JM, Vinke E, van Loon LM, et al. Middle cerebral artery flow, the critical closing pressure, and the optimal mean arterial pressure in comatose cardiac arrest survivors-An observational study. Resuscitation 2017; 110:85.
81. van den Brule JMD, van der Hoeven JG, Hoedemaekers CWE. Cerebral Perfusion and Cerebral Autoregulation after Cardiac Arrest. Biomed Res Int 2018; 2018:4143636.
82. Rudolf J, Ghaemi M, Ghaemi M, et al. Cerebral glucose metabolism in acute and persistent vegetative state. J Neurosurg Anesthesiol 1999; 11:17.
83. Schaafsma A, de Jong BM, Bams JL, et al. Cerebral perfusion and metabolism in resuscitated patients with severe post-hypoxic encephalopathy. J Neurol Sci 2003; 210:23.
84. Jakkula P, Pettilä V, Skrifvars MB, et al. Targeting low-normal or high-normal mean arterial pressure after cardiac arrest and resuscitation: a randomised pilot trial. Intensive Care Med

2018; 44:2091.

85. Wihersaari L, Ashton NJ, Reinikainen M, et al. Neurofilament light as an outcome predictor after cardiac arrest: a post hoc analysis of the COMACARE trial. Intensive Care Med 2021; 47:39.
86. Kjaergaard J, Møller JE, Schmidt H, et al. Blood-Pressure Targets in Comatose Survivors of Cardiac Arrest. N Engl J Med 2022; 387:1456.
87. Camuglia AC, Randhawa VK, Lavi S, Walters DL. Cardiac catheterization is associated with superior outcomes for survivors of out of hospital cardiac arrest: review and meta-analysis. Resuscitation 2014; 85:1533.
88. Redfors B, Råmunddal T, Angerås O, et al. Angiographic findings and survival in patients undergoing coronary angiography due to sudden cardiac arrest in western Sweden. Resuscitation 2015; 90:13.
89. Millin MG, Comer AC, Nable JV, et al. Patients without ST elevation after return of spontaneous circulation may benefit from emergent percutaneous intervention: A systematic review and meta-analysis. Resuscitation 2016; 108:54.
90. Nikolaou NI, Netherton S, Welsford M, et al. A systematic review and meta-analysis of the effect of routine early angiography in patients with return of spontaneous circulation after Out-of-Hospital Cardiac Arrest. Resuscitation 2021; 163:28.
91. Wyckoff MH, Singletary EM, Soar J, et al. 2021 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations: Summary From the Basic Life Support; Advanced Life Support; Neonatal Life Support; Education, Implementation, and Teams; First Aid Task Forces; and the COVID-19 Working Group. Resuscitation 2021; 169:229.
92. Lemkes JS, Janssens GN, van der Hoeven NW, et al. Coronary Angiography after Cardiac Arrest without ST-Segment Elevation. N Engl J Med 2019; 380:1397.
93. Desch S, Freund A, Akin I, et al. Angiography after Out-of-Hospital Cardiac Arrest without ST-Segment Elevation. N Engl J Med 2021; 385:2544.
94. Pareek N, Beckley-Hoelscher N, Kanyal R, et al. MIRACLE2 Score and SCAI Grade to Identify Patients With Out-of-Hospital Cardiac Arrest for Immediate Coronary Angiography. JACC Cardiovasc Interv 2022; 15:1074.
95. Reynolds JC, Rittenberger JC, Toma C, et al. Risk-adjusted outcome prediction with initial post-cardiac arrest illness severity: implications for cardiac arrest survivors being considered for early invasive strategy. Resuscitation 2014; 85:1232.

96. Laver S, Farrow C, Turner D, Nolan J. Mode of death after admission to an intensive care unit following cardiac arrest. Intensive Care Med 2004; 30:2126.
97. Elmer J, Torres C, Aufderheide TP, et al. Association of early withdrawal of life-sustaining therapy for perceived neurological prognosis with mortality after cardiac arrest. Resuscitation 2016; 102:127.
98. Nielsen N, Wetterslev J, Cronberg T, et al. Targeted temperature management at 33°C versus 36°C after cardiac arrest. N Engl J Med 2013; 369:2197.
99. Sekhon MS, Ainslie PN, Griesdale DE. Clinical pathophysiology of hypoxic ischemic brain injury after cardiac arrest: a "two-hit" model. Crit Care 2017; 21:90.
100. Lascarrou JB, Merdji H, Le Gouge A, et al. Targeted Temperature Management for Cardiac Arrest with Nonshockable Rhythm. N Engl J Med 2019; 381:2327.
101. Hovdenes J, Laake JH, Aaberge L, et al. Therapeutic hypothermia after out-of-hospital cardiac arrest: experiences with patients treated with percutaneous coronary intervention and cardiogenic shock. Acta Anaesthesiol Scand 2007; 51:137.
102. Skulec R, Kovarnik T, Dostalova G, et al. Induction of mild hypothermia in cardiac arrest survivors presenting with cardiogenic shock syndrome. Acta Anaesthesiol Scand 2008; 52:188.
103. Rittenberger JC, Kelly E, Jang D, et al. Successful outcome utilizing hypothermia after cardiac arrest in pregnancy: a case report. Crit Care Med 2008; 36:1354.
104. Chauhan A, Musunuru H, Donnino M, et al. The use of therapeutic hypothermia after cardiac arrest in a pregnant patient. Ann Emerg Med 2012; 60:786.
105. Zeiner A, Holzer M, Sterz F, et al. Hyperthermia after cardiac arrest is associated with an unfavorable neurologic outcome. Arch Intern Med 2001; 161:2007.
106. Gebhardt K, Guyette FX, Doshi AA, et al. Prevalence and effect of fever on outcome following resuscitation from cardiac arrest. Resuscitation 2013; 84:1062.
107. Callaway CW, Coppler PJ, Faro J, et al. Association of Initial Illness Severity and Outcomes After Cardiac Arrest With Targeted Temperature Management at 36 °C or 33 °C. JAMA Netw Open 2020; 3:e208215.
108. Nishikimi M, Ogura T, Nishida K, et al. Outcome Related to Level of Targeted Temperature Management in Postcardiac Arrest Syndrome of Low, Moderate, and High Severities: A Nationwide Multicenter Prospective Registry. Crit Care Med 2021; 49:e741.
109. Dankiewicz J, Cronberg T, Lilja G, et al. Hypothermia versus Normothermia after Out-of-Hospital Cardiac Arrest. N Engl J Med 2021; 384:2283.

110. Granfeldt A, Holmberg MJ, Nolan JP, et al. Temperature control after adult cardiac arrest: An updated systematic review and meta-analysis. Resuscitation 2023; 191:109928.
111. Nutma S, Tjepkema-Cloostermans MC, Ruijter BJ, et al. Effects of targeted temperature management at 33 °C vs. 36 °C on comatose patients after cardiac arrest stratified by the severity of encephalopathy. Resuscitation 2022; 173:147.
112. Bernard SA, Smith K, Cameron P, et al. Induction of therapeutic hypothermia by paramedics after resuscitation from out-of-hospital ventricular fibrillation cardiac arrest: a randomized controlled trial. Circulation 2010; 122:737.
113. Kim F, Olsufka M, Longstreth WT Jr, et al. Pilot randomized clinical trial of prehospital induction of mild hypothermia in out-of-hospital cardiac arrest patients with a rapid infusion of 4 degrees C normal saline. Circulation 2007; 115:3064.
114. Kliegel A, Losert H, Sterz F, et al. Cold simple intravenous infusions preceding special endovascular cooling for faster induction of mild hypothermia after cardiac arrest--a feasibility study. Resuscitation 2005; 64:347.
115. Al-Senani FM, Graffagnino C, Grotta JC, et al. A prospective, multicenter pilot study to evaluate the feasibility and safety of using the CoolGard System and Icy catheter following cardiac arrest. Resuscitation 2004; 62:143.
116. Kim F, Nichol G, Maynard C, et al. Effect of prehospital induction of mild hypothermia on survival and neurological status among adults with cardiac arrest: a randomized clinical trial. JAMA 2014; 311:45.
117. Moore TM, Callaway CW, Hostler D. Core temperature cooling in healthy volunteers after rapid intravenous infusion of cold and room temperature saline solution. Ann Emerg Med 2008; 51:153.
118. Hostler D, Northington WE, Callaway CW. High-dose diazepam facilitates core cooling during cold saline infusion in healthy volunteers. Appl Physiol Nutr Metab 2009; 34:582.
119. Badjatia N, Strongilis E, Gordon E, et al. Metabolic impact of shivering during therapeutic temperature modulation: the Bedside Shivering Assessment Scale. Stroke 2008; 39:3242.
120. Rittenberger JC, Polderman K. Post-Arrest Management. Emergency Neurological Life Support Course. http://enlsprotocols.org/files/RCA.pdf (Accessed on February 01, 2018).
121. Chamorro C, Borrallo JM, Romera MA, et al. Anesthesia and analgesia protocol during therapeutic hypothermia after cardiac arrest: a systematic review. Anesth Analg 2010; 110:1328.
122. Rey A, Rossetti AO, Miroz JP, et al. Late Awakening in Survivors of Postanoxic Coma: Early Neurophysiologic Predictors and Association With ICU and Long-Term Neurologic Recovery.

Crit Care Med 2019; 47:85.

123. Paul M, Bougouin W, Dumas F, et al. Comparison of two sedation regimens during targeted temperature management after cardiac arrest. Resuscitation 2018; 128:204.
124. Hostler D, Zhou J, Tortorici MA, et al. Mild hypothermia alters midazolam pharmacokinetics in normal healthy volunteers. Drug Metab Dispos 2010; 38:781.
125. Callaway CW, Elmer J, Guyette FX, et al. Dexmedetomidine Reduces Shivering during Mild Hypothermia in Waking Subjects. PLoS One 2015; 10:e0129709.
126. Abend NS, Topjian A, Ichord R, et al. Electroencephalographic monitoring during hypothermia after pediatric cardiac arrest. Neurology 2009; 72:1931.
127. Rundgren M, Rosén I, Friberg H. Amplitude-integrated EEG (aEEG) predicts outcome after cardiac arrest and induced hypothermia. Intensive Care Med 2006; 32:836.
128. Krumholz A, Stern BJ, Weiss HD. Outcome from coma after cardiopulmonary resuscitation: relation to seizures and myoclonus. Neurology 1988; 38:401.
129. Nielsen N, Sunde K, Hovdenes J, et al. Adverse events and their relation to mortality in out-of-hospital cardiac arrest patients treated with therapeutic hypothermia. Crit Care Med 2011; 39:57.
130. Coppler PJ, Marill KA, Okonkwo DO, et al. Concordance of Brain and Core Temperature in Comatose Patients After Cardiac Arrest. Ther Hypothermia Temp Manag 2016; 6:194.
131. Robinson J, Charlton J, Seal R, et al. Oesophageal, rectal, axillary, tympanic and pulmonary artery temperatures during cardiac surgery. Can J Anaesth 1998; 45:317.
132. Erickson RS, Kirklin SK. Comparison of ear-based, bladder, oral, and axillary methods for core temperature measurement. Crit Care Med 1993; 21:1528.
133. Michelson AD, MacGregor H, Barnard MR, et al. Reversible inhibition of human platelet activation by hypothermia in vivo and in vitro. Thromb Haemost 1994; 71:633.
134. Reed RL 2nd, Bracey AW Jr, Hudson JD, et al. Hypothermia and blood coagulation: dissociation between enzyme activity and clotting factor levels. Circ Shock 1990; 32:141.
135. Valeri CR, Feingold H, Cassidy G, et al. Hypothermia-induced reversible platelet dysfunction. Ann Surg 1987; 205:175.
136. Jeppesen AN, Hvas AM, Duez CHV, et al. Prolonged targeted temperature management compromises thrombin generation: A randomised clinical trial. Resuscitation 2017; 118:126.
137. Jarrah S, Dziodzio J, Lord C, et al. Surface cooling after cardiac arrest: effectiveness, skin safety, and adverse events in routine clinical practice. Neurocrit Care 2011; 14:382.

138. Perbet S, Mongardon N, Dumas F, et al. Early-onset pneumonia after cardiac arrest: characteristics, risk factors and influence on prognosis. Am J Respir Crit Care Med 2011; 184:1048.
139. Polderman KH, Herold I. Therapeutic hypothermia and controlled normothermia in the intensive care unit: practical considerations, side effects, and cooling methods. Crit Care Med 2009; 37:1101.
140. Rhee BJ, Zhang Y, Boddicker KA, et al. Effect of hypothermia on transthoracic defibrillation in a swine model. Resuscitation 2005; 65:79.
141. Boddicker KA, Zhang Y, Zimmerman MB, et al. Hypothermia improves defibrillation success and resuscitation outcomes from ventricular fibrillation. Circulation 2005; 111:3195.
142. Roberts BW, Kilgannon JH, Chansky ME, et al. Therapeutic hypothermia and vasopressor dependency after cardiac arrest. Resuscitation 2013; 84:331.
143. Cueni-Villoz N, Devigili A, Delodder F, et al. Increased blood glucose variability during therapeutic hypothermia and outcome after cardiac arrest. Crit Care Med 2011; 39:2225.
144. Polderman KH, Peerdeman SM, Girbes AR. Hypophosphatemia and hypomagnesemia induced by cooling in patients with severe head injury. J Neurosurg 2001; 94:697.
145. Clifton GL, Miller ER, Choi SC, Levin HS. Fluid thresholds and outcome from severe brain injury. Crit Care Med 2002; 30:739.
146. Aibiki M, Kawaguchi S, Maekawa N. Reversible hypophosphatemia during moderate hypothermia therapy for brain-injured patients. Crit Care Med 2001; 29:1726.
147. van den Broek MP, Groenendaal F, Egberts AC, Rademaker CM. Effects of hypothermia on pharmacokinetics and pharmacodynamics: a systematic review of preclinical and clinical studies. Clin Pharmacokinet 2010; 49:277.
148. Tortorici MA, Kochanek PM, Poloyac SM. Effects of hypothermia on drug disposition, metabolism, and response: A focus of hypothermia-mediated alterations on the cytochrome P450 enzyme system. Crit Care Med 2007; 35:2196.
149. Zhou J, Poloyac SM. The effect of therapeutic hypothermia on drug metabolism and response: cellular mechanisms to organ function. Expert Opin Drug Metab Toxicol 2011; 7:803.
150. François B, Cariou A, Clere-Jehl R, et al. Prevention of Early Ventilator-Associated Pneumonia after Cardiac Arrest. N Engl J Med 2019; 381:1831.
151. Longstreth WT Jr, Diehr P, Cobb LA, et al. Neurologic outcome and blood glucose levels during out-of-hospital cardiopulmonary resuscitation. Neurology 1986; 36:1186.

152. Skrifvars MB, Pettilä V, Rosenberg PH, Castrén M. A multiple logistic regression analysis of in-hospital factors related to survival at six months in patients resuscitated from out-of-hospital ventricular fibrillation. Resuscitation 2003; 59:319.

153. Oksanen T, Skrifvars MB, Varpula T, et al. Strict versus moderate glucose control after resuscitation from ventricular fibrillation. Intensive Care Med 2007; 33:2093.

154. NICE-SUGAR Study Investigators, Finfer S, Chittock DR, et al. Intensive versus conventional glucose control in critically ill patients. N Engl J Med 2009; 360:1283.

155. Elmer J, Rittenberger JC, Faro J, et al. Clinically distinct electroencephalographic phenotypes of early myoclonus after cardiac arrest. Ann Neurol 2016; 80:175.

156. Seder DB, Sunde K, Rubertsson S, et al. Neurologic outcomes and postresuscitation care of patients with myoclonus following cardiac arrest. Crit Care Med 2015; 43:965.

157. Dhakar MB, Sivaraju A, Maciel CB, et al. Electro-clinical characteristics and prognostic significance of post anoxic myoclonus. Resuscitation 2018; 131:114.

158. Beuchat I, Sivaraju A, Amorim E, et al. MRI-EEG correlation for outcome prediction in postanoxic myoclonus: A multicenter study. Neurology 2020; 95:e335.

159. Kongpolprom N, Cholkraisuwat J. Neurological Prognostications for the Therapeutic Hypothermia among Comatose Survivors of Cardiac Arrest. Indian J Crit Care Med 2018; 22:509.

160. Barbella G, Lee JW, Alvarez V, et al. Prediction of regaining consciousness despite an early epileptiform EEG after cardiac arrest. Neurology 2020; 94:e1675.

161. Solanki P, Coppler PJ, Kvaløy JT, et al. Association of antiepileptic drugs with resolution of epileptiform activity after cardiac arrest. Resuscitation 2019; 142:82.

162. Ruijter BJ, Keijzer HM, Tjepkema-Cloostermans MC, et al. Treating Rhythmic and Periodic EEG Patterns in Comatose Survivors of Cardiac Arrest. N Engl J Med 2022; 386:724.

163. May TL, Ruthazer R, Riker RR, et al. Early withdrawal of life support after resuscitation from cardiac arrest is common and may result in additional deaths. Resuscitation 2019; 139:308.

164. Kragholm K, Malta Hansen C, Dupre ME, et al. Direct Transport to a Percutaneous Cardiac Intervention Center and Outcomes in Patients With Out-of-Hospital Cardiac Arrest. Circ Cardiovasc Qual Outcomes 2017; 10.

165. Søholm H, Wachtell K, Nielsen SL, et al. Tertiary centres have improved survival compared to other hospitals in the Copenhagen area after out-of-hospital cardiac arrest. Resuscitation 2013; 84:162.

166. Spaite DW, Bobrow BJ, Stolz U, et al. Statewide regionalization of postarrest care for out-of-hospital cardiac arrest: association with survival and neurologic outcome. Ann Emerg Med

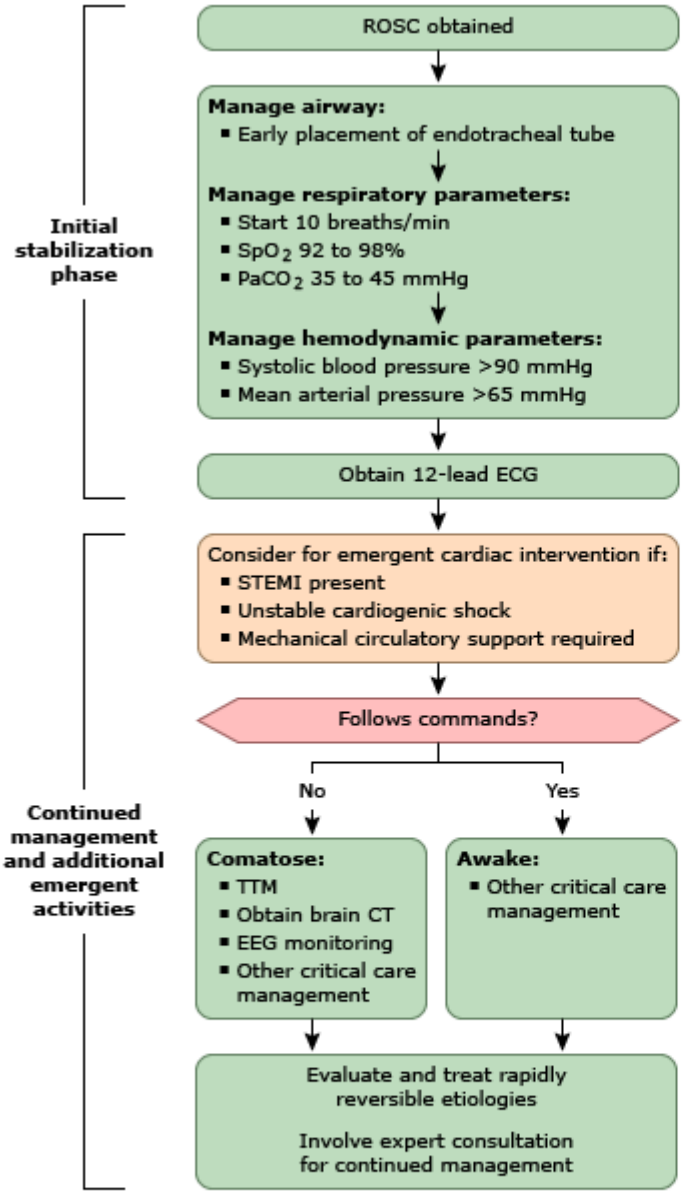
2014; 64:496.

167. Lick CJ, Aufderheide TP, Niskanen RA, et al. Take Heart America: A comprehensive, community-wide, systems-based approach to the treatment of cardiac arrest. Crit Care Med 2011; 39:26.
168. Elmer J, Callaway CW, Chang CH, et al. Long-Term Outcomes of Out-of-Hospital Cardiac Arrest Care at Regionalized Centers. Ann Emerg Med 2019; 73:29.
169. Carr BG, Kahn JM, Merchant RM, et al. Inter-hospital variability in post-cardiac arrest mortality. Resuscitation 2009; 80:30.
170. Elmer J, Rittenberger JC, Coppler PJ, et al. Long-term survival benefit from treatment at a specialty center after cardiac arrest. Resuscitation 2016; 108:48.
171. Schober A, Sterz F, Laggner AN, et al. Admission of out-of-hospital cardiac arrest victims to a high volume cardiac arrest center is linked to improved outcome. Resuscitation 2016; 106:42.
172. Callaway CW, Schmicker R, Kampmeyer M, et al. Receiving hospital characteristics associated with survival after out-of-hospital cardiac arrest. Resuscitation 2010; 81:524.
173. Berg KM, Cheng A, Panchal AR, et al. Part 7: Systems of Care: 2020 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2020; 142:S580.

Topic 13838 Version 75.0

GRAPHICS

Adult post-cardiac arrest care algorithm



Initial stabilization phase

Resuscitation is ongoing during the post-ROSC phase, and many of these activities can occur concurrently. However, if prioritization is necessary follow these steps:

- Airway management: Waveform capnography or capnometry to confirm and monitor endotracheal tube placement
- Manage respiratory parameters: Titrate FIO_2 for SpO_2 92 to 98%; start at 10 breaths/min; titrate to $PaCO_2$ of 35 to 45 mmHg
- Manage hemodynamic parameters: Administer crystalloid and/or vasopressor or inotrope for goal systolic blood pressure >90 mmHg or mean arterial pressure >65 mmHg

Continued management and additional emergent activities

These evaluations should be done concurrently so that decisions on targeted temperature management (TTM) receive high priority as cardiac interventions.

- Emergent cardiac intervention: Early evaluation of 12-lead electrocardiogram (ECG); consider hemodynamics for decision on cardiac intervention
- TTM: If patient is not following commands, start TTM as soon as possible; begin at 32 to 36°C for 24 hours by using a cooling device with feedback loop
- Other critical care management:
  - Continuously monitor core temperature (esophageal, rectal, bladder)
  - Maintain normoxia, normocapnia, euglycemia
  - Provide continuous or intermitten electroencephalogram (EEG) monitoring
  - Provide lung-protective ventilation

H's and T's

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypokalemia/**h**yperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

Reprinted with permission. Highlights of the 2020 American Heart Association Guidelines for CPR and ECC. Copyright © 2020 American Heart Association, Inc.

Graphic 129984 Version 10.0

Treatable conditions associated with cardiac arrest

Condition	Common associated clinical settings
Acidosis	Diabetes, diarrhea, drug overdose, renal dysfunction, sepsis, shock
Anemia	Gastrointestinal bleeding, nutritional deficiencies, recent trauma
Cardiac tamponade	Post-cardiac surgery, malignancy, post-myocardial infarction, pericarditis, trauma
Hyperkalemia	Drug overdose, renal dysfunction, hemolysis, excessive potassium intake, rhabdomyolysis, major soft tissue injury, tumor lysis syndrome
Hypokalemia*	Alcohol abuse, diabetes mellitus, diuretics, drug overdose, profound gastrointestinal losses
Hypothermia	Alcohol intoxication, significant burns, drowning, drug overdose, elder patient, endocrine disease, environmental exposure, spinal cord disease, trauma
Hypovolemia	Significant burns, diabetes, gastrointestinal losses, hemorrhage, malignancy, sepsis, trauma
Hypoxia	Upper airway obstruction, hypoventilation (CNS dysfunction, neuromuscular disease) pulmonary disease
Myocardial infarction	Cardiac arrest
Poisoning	History of alcohol or drug abuse, altered mental status, classic toxidrome (eg, sympathomimetic), occupational exposure, psychiatric disease
Pulmonary embolism	Immobilized patient, recent surgical procedure (eg, orthopedic), peripartum, risk factors for thromboembolic disease, recent trauma, presentation consistent with acute pulmonary embolism
Tension pneumothorax	Central venous catheter, mechanical ventilation, pulmonary disease (eg, asthma, chronic obstructive pulmonary disease), thoracentesis, thoracic trauma

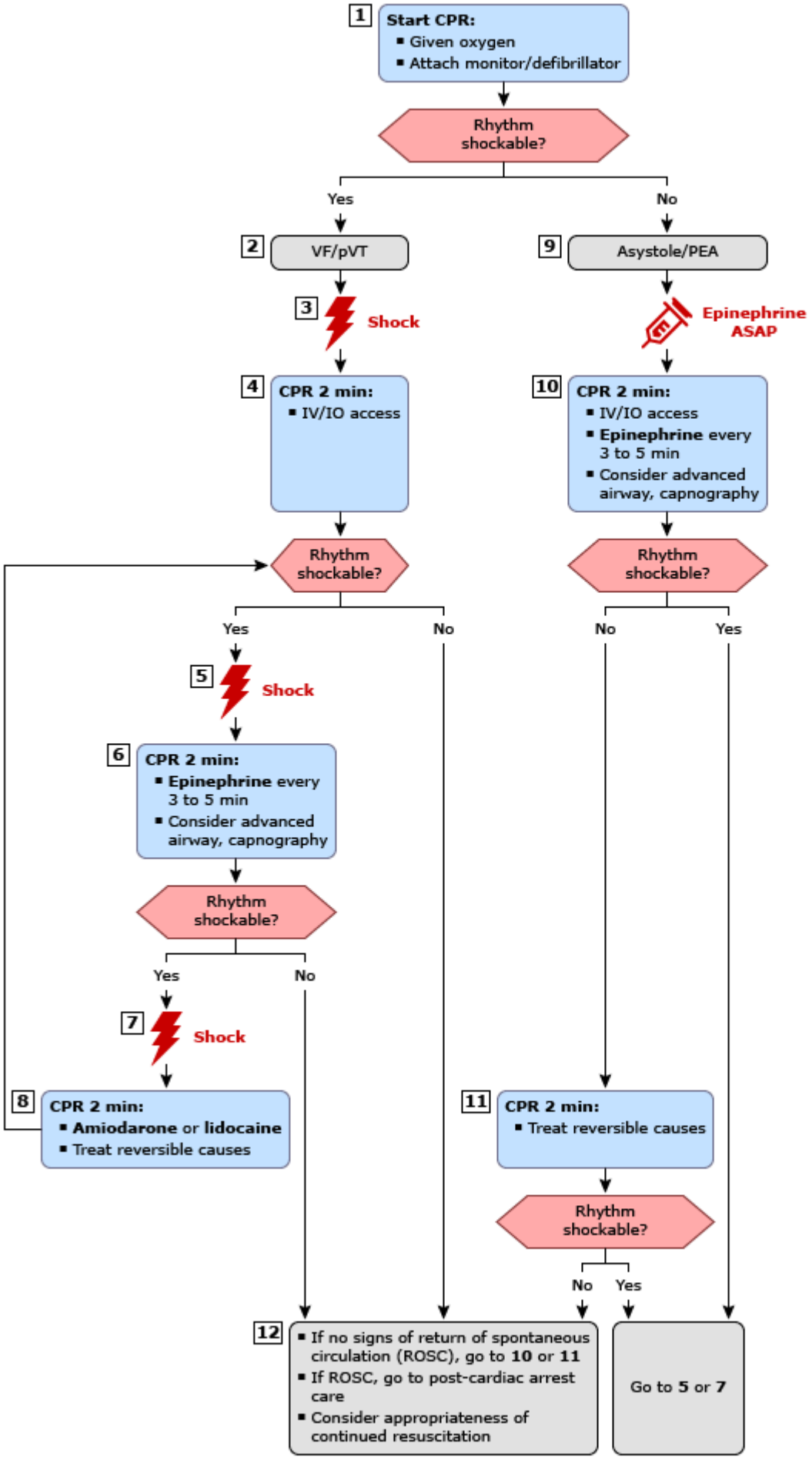
CNS: central nervous system.

* Hypomagnesemia should be assumed in the setting of hypokalemia, and both should be treated.

Adapted from: Eisenberg MS, Mengert TJ. Cardiac resuscitation. N Engl J Med 2001; 344:1304.

Graphic 52416 Version 8.0

Adult cardiac arrest algorithm



CPR quali

- Push hard (at least 2 inch
  fast (100 to 120/min) and
  chest recoil.
- Minimize interruptions in
- Avoid excessive ventilatio
- Change compressor every
  sooner if fatigued.
- If no advanced airway, 30
  ventilation ratio.
- Quantitative waveform ca
  - If $PETCO_2$ is low or decr
    CPR quality.

Shock energy for d

- **Biphasic:** Manufacturer r
  (eg, initial dose of 120 to
  unknown, use maximum
  and subsequent doses sh
  and higher doses may be
- **Monophasic:** 360 J.

Drug thera

- **Epinephrine IV/IO dos**
  1 mg every 3 to 5 minute
- **Amiodarone IV/IO dos**
  First dose: 300 mg bolus.
  Second dose: 150 mg.
  or
- **Lidocaine IV/IO dose:**
  First dose: 1 to 1.5 mg/kg
  Second dose: 0.5 to 0.75

Advanced ai

- Endotracheal intubation o
  advanced airway.
- Waveform capnography o
  confirm and monitor ET tu
- Once advanced airway in
  breath every 6 seconds (1
  with continuous chest com

Return of spontaneous c

- Pulse and blood pressure.
- Abrupt sustained increase
  (typically ≥40 mmHg).
- Spontaneous arterial pres
  intra-arterial monitoring.

Reversible ca

- **Hypovolemia**
- **Hypoxia**
- **Hydrogen ion (acidosis)**
- **Hypo-/hyperkalemia**
- **Hypothermia**
- **Tension pneumothorax**
- **Tamponade, cardiac**
- **Toxins**
- **Thrombosis, pulmonary**
- **Thrombosis, coronary**

Reprinted with permission. Highlights of the 2020 American Heart Association Guidelines for CPR and ECC. Copyright © 2020 American Heart Association, Inc.

Graphic 129983 Version 10.0

Common etiologies of cardiac arrest in adults

Cardiac
Acute coronary syndrome (most common)
Arrhythmia: Secondary to electrolyte disorder, acidosis, other process; abnormal conduction syndromes (eg, prolonged QT, Brugada)
Pericardial tamponade
Respiratory
Airway obstruction: Mucus plug, foreign body, tracheostomy decannulation
Asthma/COPD exacerbation
Pneumonia
Pulmonary embolus
Tension pneumothorax
Hemmorhage and hypovolemia
Trauma
Gastrointestinal bleeding
Abdominal aortic aneursym rupture
Intracranial hemorrhage
Profound gastrointestinal fluid loss
Drugs and poisons
Opioids
Beta blockers
Calcium-channel blockers
Benzodiazepines
Tricyclic antidepressants
Digoxin
Electrolyte disturbances
Potassium
Magnesium
Sepsis

Glasgow Coma Scale (GCS)

	Score
Eye opening	
Spontaneous	4
Response to verbal command	3
Response to pain	2
No eye opening	1
Best verbal response	
Oriented	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
No verbal response	1
Best motor response	
Obeys commands	6
Localizing response to pain	5
Withdrawal response to pain	4
Flexion to pain	3
Extension to pain	2
No motor response	1
Total	

The GCS is scored between 3 and 15, 3 being the worst and 15 the best. It is composed of three parameters: best eye response (E), best verbal response (V), and best motor response (M). The components of the GCS should be recorded individually; for example, E2V3M4 results in a GCS score of 9. A score of 13 or higher correlates with mild brain injury, a score of 9 to 12 correlates with moderate injury, and a score of 8 or less represents severe brain injury.

Graphic 81854 Version 9.0

FOUR score

Eye response
4 = eyelids open or opened, tracking, or blinking to command
3 = eyelids open but not tracking
2 = eyelids closed but open to loud voice
1 = eyelids closed but open to pain
0 = eyelids remain closed with pain
Motor response
4 = thumbs-up, fist, or peace sign
3 = localizing to pain
2 = flexion response to pain
1 = extension response to pain
0 = no response to pain or generalized myoclonus status
Brainstem reflexes
4 = pupil and corneal reflexes present
3 = one pupil wide and fixed
2 = pupil or corneal reflexes absent
1 = pupil and corneal reflexes absent
0 = absent pupil, corneal, and cough reflex
Respiration
4 = not intubated, regular breathing pattern
3 = not intubated, Cheyne-Stokes breathing pattern
2 = not intubated, irregular breathing
1 = breathes above ventilator rate
0 = breathes at ventilator rate or apnea

FOUR score: Full Outline of UnResponsiveness.

Reproduced from: Fischer M, Ruegg S, Czaplinski A, et al. Inter-rater reliability of the Full Outline of UnResponsiveness score and the Glasgow Coma Scale in critically ill patients: a prospective observational study. Crit Care 2010; 14:R64. Copyright © 2010 BioMed Central Ltd.

Graphic 62673 Version 4.0

Contributor Disclosures

Jonathan Elmer, MD, MS, FNCS Grant/Research/Clinical Trial Support: National Institutes of Health [Post-cardiac arrest care]. All of the relevant financial relationships listed have been mitigated. **Jon C Rittenberger, MD, MS** No relevant financial relationship(s) with ineligible companies to disclose. **Ron M Walls, MD, FRCPC, FAAEM** Other Financial Interest: Airway Management Education Center [Health care provider education and resources]; First Airway [Health care provider education and resources]. All of the relevant financial relationships listed have been mitigated. **Jonathan Grayzel, MD, FAAEM** No relevant financial relationship(s) with ineligible companies to disclose.

Contributor disclosures are reviewed for conflicts of interest by the editorial group. When found, these are addressed by vetting through a multi-level review process, and through requirements for references to be provided to support the content. Appropriately referenced content is required of all authors and must conform to UpToDate standards of evidence.

Conflict of interest policy

