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Guidelines

# 2025 Korean Guidelines for Cardiopulmonary Resuscitation: Part 5. Cardiac arrest in special circumstances

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The 2025 update of the Korean guidelines for cardiac arrest under special circumstances incorporates new evidence and expert consensus to clarify when clinicians should modify standard resuscitation algorithms. For cardiac arrest caused by acute hyperkalemia, the guidelines suggest administering intravenous insulin with glucose; however, current evidence remains insufficient to recommend for or against routine use of sodium bicarbonate or calcium. In suspected pulmonary embolism–related cardiac arrest, thrombolytic therapy may be considered. In confirmed cases, thrombolysis, surgical embolectomy, or percutaneous mechanical thrombectomy may be appropriate, despite very low certainty of evidence. For opioid-related cardiac arrest, current evidence does not support the routine administration of naloxone in addition to standard advanced life support; however, naloxone may be administered when it is unclear whether the patient is in true cardiac arrest. The guidelines also emphasize managing cardiac arrest in the prone position. If the patient is intubated and immediate repositioning is unsafe or impractical, prone cardiopulmonary resuscitation and defibrillation may be attempted using invasive arterial pressure or end-tidal carbon dioxide monitoring to guide the timing of repositioning. Immediate supination is strongly recommended for non-intubated patients. Additional updates address drowning, severe hypothermia, pregnancy, anaphylaxis, and cardiac arrest during interventional procedures, underscoring the importance of early correction of reversible causes, appropriate airway strategies, and timely consideration of extracorporeal life support in selected cases. Overall, the 2025 recommendations highlight cautious, etiology-directed interventions and explicitly grade recommendation strength and certainty to support context-sensitive clinical decision-making in high-risk and resource-variable settings.

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## MAJOR CHANGES IN THE 2025 GUIDELINES FOR CARDIAC ARREST IN SPECIAL CIRCUMSTANCES

### Cardiac arrest caused by hyperkalemia

For patients with cardiac arrest caused by acute hyperkalemia, intravenous administration of insulin and glucose is recommended (weak recommendation, very low certainty of evidence). Current evidence is insufficient to recommend for or against the administration of intravenous bicarbonate in this setting (weak recommendation, very low certainty of evidence). Likewise, the evidence remains inadequate to support or oppose the use of intravenous calcium in patients with cardiac arrest caused by acute hyperkalemia (weak recommendation, very low certainty of evidence).

### Cardiac arrest caused by pulmonary embolism

Thrombolytics may be administered if pulmonary embolism is suspected as the cause of cardiac arrest (weak recommendation, very low certainty of evidence). When pulmonary embolism is confirmed, thrombolytics, surgical embolectomy, or percutaneous mechanical thrombectomy may be considered (weak recommendation, very low certainty of evidence).

### Cardiac arrest caused by opioid toxicity

In patients with cardiac arrest caused by opioid toxicity, existing evidence does not support administering opioid-specific therapies (e.g., naloxone) in addition to standard resuscitative care during advanced life support (ALS). However, if the rescuer is unsure whether a patient with suspected opioid toxicity is in true cardiac arrest, an opioid antagonist (e.g., naloxone) may be administered (good practice statement).

### Prone cardiopulmonary resuscitation

In patients who experience cardiac arrest in the prone position, prone cardiopulmonary resuscitation (CPR) may be attempted if the patient is intubated, and it is difficult or dangerous to turn the patient immediately to the supine position (good practice statement). Invasive arterial pressure or end-tidal carbon dioxide ($ETCO_2$) partial pressure monitoring can serve as useful indicators for monitoring perfusion pressure during prone CPR and may assist in determining when to turn the patient to the supine position for CPR (good practice statement). If a patient with cardiac arrest in the prone position does not have an advanced airway in place, it is suggested that the patient be turned to the supine position immediately for CPR (strong recommendation, low certainty of evidence). If a patient requiring defibrillation is in the prone position and cannot be turned immediately, defibrillation may be performed in this position (good practice statement).

## BACKGROUND FOR REVISION OF CARDIAC ARREST IN SPECIAL CIRCUMSTANCES

CPR in special circumstances involves adding to or modifying the treatment process of the universally recommended basic life support (BLS) and ALS by considering patient characteristics or environmental factors. To address this, the 2025 Korean guidelines for ALS reviewed the scientific evidence for items in the 2020 guidelines that lacked sufficient evidence, had missing treatment protocols, or had limitations in application within Korea, thereby deriving more objective and clearer recommendations than the previous guidelines. Since the publication of the 2020 Korean guidelines for CPR, several research findings on CPR under special circumstances, such as the management of cardiac arrest due to hyperkalemia and the use of prone CPR, have been published, and these are described in this chapter. The selection and review process for major topics is described in Part 1.

## CPR IN SPECIAL CIRCUMSTANCES

### Cardiac arrest associated with asthma

*Oxygen administration*

There are no studies comparing the effects of oxygen with those of other gases in cardiac arrest caused by asthma or chronic obstructive pulmonary disease (COPD). As the cause of cardiac arrest due to asthma or COPD is hypoxia, it is recommended to administer a high concentration of oxygen during ventilation.

*Endotracheal intubation during CPR*

Studies have shown that peak airway pressure measured in patients with severe asthma is significantly higher than that in patients with lower esophageal sphincter pressure [1]. Since lower esophageal sphincter pressure is lower than normal during cardiac arrest, the risk of gastric inflation and regurgitation is very high in cardiac arrest caused by asthma or COPD. Therefore, endotracheal intubation should be performed immediately during CPR for cardiac arrest caused by asthma or COPD.

*Ventilation support*

If ventilation is difficult in cases of cardiac arrest due to asthma or COPD, the possibility of a tension pneumothorax must be considered. As simple needle decompression may not fully resolve a

tension pneumothorax, tube thoracostomy may be necessary [2,3]. In addition, if dynamic hyperinflation is suspected during CPR, air trapping may be resolved by disconnecting the bag-mask and performing chest compressions only [4,5]. Since the harmful effects of auto–positive end-expiratory pressure persist in patients with asthma or COPD during cardiac arrest, a lower respiratory rate and tidal volume (6–8 mL/kg) than normal should be used when using a mechanical ventilator [6].

*Circulatory support*

Although there are no studies on the effect of fluid therapy on the prognosis of cardiac arrest in patients with asthma or COPD, it is reasonable to consider fluid therapy, as these patients often have insufficient dietary intake and high insensible loss. Epinephrine should be administered intravenously at a dose of 1 mg every 3 to 5 minutes, consistent with standard CPR. Extracorporeal CPR (ECPR) using an extracorporeal membrane oxygenation device may be considered for patients who do not respond to standard CPR [7].

## Cardiac arrest associated with anaphylaxis

*Airway maintenance*

Rapid, early advanced airway management is crucial. Because oropharyngeal and laryngeal edema may develop, the patient should be immediately transferred to a medical facility capable of advanced airway management. The presence of hoarseness, tongue edema, stridor, or oropharyngeal edema indicates a potential progression to a "difficult airway." Therefore, an advanced airway management plan, including surgical airway methods, must be established in advance.

*Circulation*

Epinephrine should be administered early to all patients showing signs of systemic allergic reactions, such as hypotension, airway edema, or dyspnea. The recommended dose of epinephrine is 0.2 to 0.5 mg (1:1,000) administered intramuscularly; if there is no clinical improvement, it may be repeated every 5 minutes [8,9]. If intravenous access is available, 0.05 to 0.1 mg may be administered intravenously. Subcutaneous injection is absorbed more slowly and reaches peak plasma concentration later than intramuscular injection, which may be further delayed in shock states. If the patient carries an epinephrine autoinjector prescribed by a physician due to a history of recurrent anaphylaxis, the epinephrine autoinjector should be used on the scene to administer epinephrine intramuscularly into the anterolateral aspect of the middle third of the thigh (0.3 mg for adults, 0.15 mg for children) [10].

*Fluid therapy*

Since anaphylaxis causes vasodilation and redistribution of blood volume, it can induce a shock state similar to sepsis. Therefore, it is recommended to administer at least 500 mL of balanced crystalloid or normal saline within 5 to 10 minutes to patients in shock due to anaphylaxis [11]. In some cases, additional fluid administration may be required [5].

*Oxygen administration*

If hypoxemia accompanies this condition, oxygen administration should be considered to maintain oxygen saturation between 94% and 98%.

*Vasopressors*

If the provider has experience with intravenous epinephrine injections in noncardiac arrest situations, intravenous epinephrine may be administered. An initial dose of 20 to 50 μg is given intravenously, and subsequent doses should be titrated [5]. However, because fatal cases due to overdose have been reported, monitoring of blood pressure, oxygen saturation, and electrocardiogram (ECG) is required.

*Other therapeutic agents*

If shock persists despite epinephrine administration, norepinephrine, vasopressin, or phenylephrine may be administered.

*Effect of antihistamines and steroids on anaphylaxis*

Routine use of antihistamines and steroids in the initial treatment of anaphylaxis is known to have no distinct effects. However, steroids may be considered when asthma-like symptoms persist or shock does not improve despite initial treatment [12–14].

*Extracorporeal circulatory support*

For patients with cardiac arrest due to anaphylaxis who cannot be resuscitated with standard CPR, ECPR using an extracorporeal membrane oxygenation device may be considered.

## Cardiac arrest in pregnancy

*Aortocaval compression*

In pregnant women over 20 weeks of gestation, cardiac output may decrease by 30% to 40% as a result of compression of the inferior vena cava and aorta by the uterus, which reduces venous return [15]. This phenomenon can induce hypotension or shock prior to cardiac arrest and may also lead to cardiac arrest [16,17]. In this case, the effectiveness of chest compressions is inevitably reduced even when high-quality chest compressions are per-

formed. Displacing the uterus to the left is the easiest method to avoid compressing the inferior vena cava and aorta, which may be more efficient than tilting the patient to the left, as it allows chest compressions to be performed with the patient in a supine position [18,19].

*Chest compressions*

The position, rate, and depth of the chest compressions follow the standard CPR guidelines. However, mechanical CPR is not recommended. Although recent studies have reported that the heart is displaced upward by compression of the diaphragm when the uterus is enlarged, research on appropriate hand placement is insufficient [20,21].

*Fetal delivery during maternal cardiac arrest*

If cardiac arrest is imminent or occurs in a pregnant woman, resuscitative hysterotomy or cesarean section should be performed immediately. Early delivery can improve resuscitation outcomes by relieving compression of the aorta and vena cava [22]. Several studies involving pregnant women with cardiac arrest have reported that fetal survival rates were high when cesarean section was performed successfully within 5 minutes of cardiac arrest [23]. Therefore, a cesarean section should be performed within 5 minutes when spontaneous circulation is not achieved after the first 4 minutes of CPR in pregnant women at >20 weeks of gestation or in those whose uterus is palpable above the umbilicus.

*Defibrillation*

As there is no change in transthoracic impedance during pregnancy and no reports that defibrillation affects the fetal heart, defibrillation can be performed according to standard CPR guidelines.

*Airway management*

Pregnant women are at high risk of hypoxia, have a high possibility of gastric regurgitation and aspiration, and have a high probability of failed endotracheal intubation [24,25]. While ALS guidelines recommend maintaining the airway using one of the several methods, including bag-mask ventilation, supraglottic airway device, or endotracheal intubation, depending on the rescuer's proficiency, performing endotracheal intubation as early as possible facilitates oxygenation and ventilation and prevents aspiration.

*Institutional preparedness for maternal cardiac arrest*

An important element in the CPR process for pregnant women is calling the emergency cesarean section team. Healthcare providers involved in resuscitation must clearly understand and be familiar with the fastest method of activating the cesarean section team within their working environment. Additionally, a team comprising related departments and personnel, such as obstetricians, delivery room staff, and anesthesiologists, should be organized, and education should be provided to assign clear roles to each team member so that they can perform effectively in emergency situations. Targeted temperature management in maternal cardiac arrest should be performed according to the criteria for the general adult population. However, during the hypothermia period, fetal monitoring devices should be attached, and close cooperation between obstetricians and neonatal specialists is required [26,27].

## Cardiac arrest caused by pulmonary embolism

Cardiac arrest due to pulmonary thromboembolism (PTE) is the most severe complication of venous thromboembolism, and most cases of PTE are from deep vein thrombosis [28]. The 2020 International Liaison Committee on Resuscitation (ILCOR) systematic review evaluated prognoses based on treatment modalities in cardiac arrest attributed to PTE. The principal findings and recommendations are summarized below.

*Thrombolytics*

There is evidence of very low certainty regarding survival with favorable neurological outcomes at 30 days (certainty was downgraded owing to serious imprecision and a very serious risk of bias). In a randomized controlled trial by Böttiger et al. [29], no significant difference in treatment outcomes was observed between the group using thrombolytics and the group not using them among 37 cardiac arrest patients with confirmed PTE. Similarly, one observational study by Javaudin et al. [30] reported no significant differences between the two groups. Evidence regarding 30-day survival is also of very low certainty. In one observational study by Javaudin et al. [30] and one retrospective study by Henriksson et al. [31], the participants in the intervention group demonstrated improved survival compared to those in the control group. Evidence regarding survival to hospital discharge is of very low certainty; three retrospective observational studies found no significant difference between the participants in the intervention and those in the control groups [32–34]. For return of spontaneous circulation (ROSC), the evidence is of low certainty. One of the three studies reported that the use of thrombolytics was beneficial compared with the control group [32–34]. There was evidence of very low certainty regarding 24-hour survival. While

one observational study noted no significant differences between the two groups, Janata et al. [34] reported more favorable treatment outcomes among participants in the intervention group compared to those in the control group.

*Surgical embolectomy and percutaneous mechanical thrombectomy*

No randomized controlled trials have evaluated surgical embolectomy and percutaneous mechanical thrombectomy as treatment measures for cardiac arrest caused by PTE, and the existing evidence is of very low certainty. In clinical observational studies, 30-day survival rate was higher in patients who underwent surgical embolectomy compared to those who received standard CPR. In one clinical case study, six of seven patients (85.7%) who underwent percutaneous mechanical thrombectomy achieved ROSC successfully [35].

*Korean treatment guidelines*

Thrombolytics may be administered when PTE is suspected to be the cause of cardiac arrest (weak recommendation and very low certainty of evidence). For patients in whom PTE is confirmed as the cause of cardiac arrest, thrombolytics, surgical embolectomy, or percutaneous mechanical thrombectomy may be considered (weak recommendation, very low certainty of evidence).

There are no specific concerns regarding the application of these guidelines to patients who develop cardiac arrest after a PTE has been diagnosed. However, since the diagnosis of PTE is difficult in patients undergoing CPR, further discussion on the diagnostic criteria for PTE in patients with cardiac arrest is needed. PTE cannot be diagnosed solely on the observation of right atrial and right ventricular dilation on ultrasound during CPR.

## Cardiac arrest caused by electrolyte imbalance

Electrolyte abnormalities are associated with cardiovascular emergencies and can directly cause cardiac arrest or contribute to its development. Moreover, they may impede ROSC and negatively affect the restoration of hemodynamic stability after cardiac arrest. Therefore, in patients presenting with unstable cardiovascular emergencies, early initiation of treatment modalities targeting electrolyte abnormalities should be considered in addition to standard ALS guidelines.

*Hyperkalemia*

Hyperkalemia is a potentially life-threatening condition that can lead to cardiac instability, arrhythmias, and cardiac arrest. The standard treatment for life-threatening arrhythmias due to hyperkalemia generally includes the administration of calcium, β2 agonists, and high-dose insulin therapy. However, these treatments have not been evaluated in patients experiencing cardiac arrest. Accordingly, the ILCOR systematic review evaluated prognoses by treatment modality in cardiac arrest caused by hyperkalemia, and the findings and recommendations reported in 2025 are summarized below [36]. However, as studies reporting patient-centered outcomes such as mortality are scarce, this evidence summary is organized based on studies examining changes in potassium levels and ECG changes.

(1) Evidence summary

① Insulin

In eight studies involving 645 patients (parallel studies, crossover studies, before-and-after studies, observational studies), intravenous administration of 8 to 12 IU of insulin with glucose resulted in a mean reduction in serum potassium concentration of −0.7 mmol/L (95% confidence interval [CI], −0.9 to −0.6). The certainty of the evidence is rated as low due to the risk of bias.

② β2 Agonists

In seven studies involving 87 patients (parallel studies, crossover studies, and before-and-after studies), inhaled administration of 10 to 20 mg salbutamol resulted in a mean reduction in potassium concentration of −0.9 mmol/L (95% CI, −1.2 to −0.7). The certainty of the evidence is very low, owing to the risk of bias.

In six studies involving 100 patients (parallel studies and before-and-after studies), intravenous administration of 0.5 mg salbutamol dissolved in glucose/dextrose resulted in a mean change in potassium concentration of −1.0 mmol/L (95% CI, −1.4 to −0.6). The certainty of evidence is very low due to the risk of bias and imprecision.

③ Comparison of β2 agonists and insulin

In three parallel studies involving 64 patients, intravenous administration of 0.5 mg salbutamol resulted in a mean potassium change that was −0.3 mmol/L smaller than that achieved with 10 IU insulin (95% CI, −0.5 to 0.0). The certainty of evidence is very low due to the risk of bias and imprecision.

④ Bicarbonate

In five studies involving 44 patients (before-and-after studies, parallel studies, crossover studies), intravenous administration of 50 to 390 mmol of bicarbonate resulted in a mean change in potassium concentration of −0.1 mmol/L (95% CI, −0.3 to 0.1). The certainty of the evidence is very low due to the risk of bias.

⑤ Combination of insulin and β2 agonists

In three parallel studies involving 25 patients, the combination of intravenous administration of 0.5 mg salbutamol, 10 IU insulin, and 25 or 40 g glucose resulted in a mean reduction in potassium concentration of –1.2 mmol/L (95% CI, –1.5 to –0.8). The certainty of evidence is very low due to the risk of bias and imprecision.

In three parallel studies involving 64 patients, the combination of intravenous administration of 10 IU insulin and 0.5 mg salbutamol reduced the potassium concentration by a mean of –0.22 mmol/L compared with 0.5 mg salbutamol alone (95% CI, –0.5 to 0.1). The certainty of evidence is very low due to the risk of bias and imprecision.

In three parallel studies involving 50 patients, the combination of intravenous administration of 10 IU insulin and 0.5 mg salbutamol reduced the potassium concentration by a mean of –0.5 mmol/L compared with 10 IU insulin alone (95% CI, –0.7 to –0.2). The certainty of evidence is very low due to the risk of bias and imprecision.

⑥ Effect of calcium on ECG changes

A retrospective observational study involving 111 patients found that calcium administration did not produce a statistically significant improvement in arrhythmias [37]. The certainty of evidence is very low due to the risk of bias and imprecision.

⑦ Cardiac arrest and calcium

A retrospective observational study involving 109 adults reported that calcium administration was associated with higher mortality [38]. The certainty of evidence is very low due to the risk of bias and imprecision.

(2) Korean recommendations

For patients with cardiac arrest caused by acute hyperkalemia, intravenous administration of insulin and glucose is recommended (weak recommendation, very low certainty of evidence). There is insufficient evidence to recommend for or against the administration of intravenous bicarbonate in patients with cardiac arrest caused by acute hyperkalemia (weak recommendation, very low certainty of evidence). Likewise, there is insufficient evidence to recommend for or against the administration of intravenous calcium in patients with cardiac arrest caused by acute hyperkalemia (weak recommendation, very low certainty of evidence).

*Hypokalemia*

Although the prevalence of hypokalemia (serum potassium concentration < 3.5 mmol/L) is low, it may occur due to the loss of potassium ions through the gastrointestinal tract and kidneys, and is associated with hypomagnesemia. Severe hypokalemia alters the excitability and conductivity of cardiomyocytes, and characteristic U waves and flattened T waves may appear on ECG. In patients receiving digoxin, arrhythmias can be induced (especially ventricular arrhythmias), which may lead to pulseless electrical activity (PEA) or asystole if left untreated. The treatment of hypokalemia in cardiotoxic scenarios, particularly torsades de pointes, involves slow intravenous infusion of potassium over several hours; rapid administration of potassium is not recommended [39,40]. Generally, potassium is infused at a rate of 10 mmol/hr; however, in cases of unstable arrhythmia or imminent cardiac arrest, it can be infused at a rate of 2 mmol/min for 10 minutes, followed by administration of 10 mmol over 5 to 10 minutes. If hypokalemia is accompanied by hypomagnesemia, 4 mL of 50% magnesium sulfate mixed in 10 mL of normal saline should be administered over 20 minutes before administering potassium [41].

*Hypermagnesemia*

Hypermagnesemia is defined as a serum magnesium concentration exceeding 2.2 mEq/L (1.1 mmol/L). Neurological symptoms include muscle weakness or paralysis, ataxia, drowsiness, and altered consciousness, such as confusion. Hypermagnesemia can also induce hypotension through vasodilation. In cases of very high serum magnesium concentrations, reduced consciousness, bradycardia, arrhythmias, respiratory depression, and cardiopulmonary arrest may occur [42,43]. ECG findings may include prolonged PR and QT intervals, peaked T waves, and atrioventricular block. For cardiac arrest patients due to hypermagnesemia, intravenous administration of 5 to 10 mL of 10% calcium chloride or 15 to 30 mL of 10% calcium gluconate over 2 to 5 minutes is recommended.

*Hypomagnesemia*

Hypomagnesemia is defined as a serum magnesium concentration < 1.3 mEq/L (0.6 mmol/L) and is significantly more common than hypermagnesemia. Hypomagnesemia is mainly caused by decreased absorption or loss through the kidneys or intestines (e.g., diarrhea) and may also be associated with thyroid dysfunction, medication use (e.g., pentamidine, diuretics, alcohol), malnutrition, and alcoholism. Hypomagnesemia is associated with polymorphic ventricular tachycardia, including torsades de pointes and pulseless (polymorphic) ventricular tachycardia. On the ECG, prolonged PR and QT intervals, ST-segment depression, T-wave inversion, and flattened P waves may be observed. In cas-

es of severe hypomagnesemia or seizures, 2 g (4 mL; 8 mmol) of 50% magnesium sulfate should be infused intravenously over 10 to 15 minutes; if torsades de pointes occur, the same dose may be administered over 1 to 2 minutes.

*Calcium*

Calcium electrolyte abnormality is a rare cause of cardiac arrest. In cases where hypocalcemia or hypercalcemia is suspected as a cause of cardiac arrest, empirical intravenous administration of calcium may be considered, specifically 5–10 mL of 10% calcium chloride and 15–30 mL of 10% calcium gluconate over 2 to 5 minutes. However, there are no studies demonstrating the efficacy of treating hypocalcemia or hypercalcemia during cardiac arrest.

## Cardiac arrest caused by opioid toxicity

In some countries, opioid-related emergencies, including cardiac arrest, remain significant public health concerns. Opioid antagonists (e.g., naloxone) are effective in reversing respiratory depression caused by opioid overdose and can thereby prevent progression to cardiac arrest. However, there is no established evidence regarding whether specific therapies such as naloxone should be administered in addition to standard resuscitation in cases where cardiac arrest has already occurred due to opioid overdose. This topic was previously reviewed in 2015, and the treatment recommendations remained unchanged following the 2020 evidence update [44,45]. Accordingly, the ILCOR conducted a new systematic review in 2025.

*ILCOR evidence summary: naloxone administration during cardiac arrest*

(1) Survival to discharge with favorable neurological outcome

Very low certainty was confirmed in three observational studies. In one study enrolling 218 adult out-of-hospital cardiac arrest (OHCA) patients with suspected opioid overdose, naloxone administered before ROSC was not associated with favorable neurological outcomes (adjusted odds ratio [aOR], 1.99; 95% CI, 0.34–11.55) [46]. In a subsequent analysis of the same data involving 1,807 patients with initial nonshockable rhythms not witnessed by emergency medical services (EMS), naloxone administered before vascular access was found to be associated with favorable neurological outcome (aOR, 4.61; 95% CI, 1.74–12.19) [47]. In a study that enrolled 164 adult OHCA patients with a history of drug toxicity or use, including opioids, the presence or absence of naloxone treatment was not associated with favorable neurological outcomes (26% vs. 27%, P=0.915) [48].

(2) Survival to discharge

Very low certainty was confirmed in four observational studies. In a study enrolling 8,195 OHCA patients, naloxone administration was associated with increased survival to hospital discharge (risk difference, 6.2%; 95% CI, 2.3%–10.0%) [49]. Conversely, in another OHCA registry study, when comparing 159 patients treated with naloxone with 159 who were not, naloxone was not associated with survival to discharge (aOR, 1.01; 95% CI, 0.46–2.21) [50]. Similar to favorable neurological outcome, an association between naloxone and improved survival was reported in OHCA patients with initial nonshockable rhythms not witnessed by EMS (aOR, 4.41; 95% CI, 1.78–10.97), whereas no such association was observed among OHCA patients with suspected drug overdose (aOR, 1.99; 95% CI, 0.39–10.30) [46].

(3) Return of spontaneous circulation

Very low certainty was confirmed in three observational studies. Consistent with findings from the study mentioned above, naloxone administration was associated with increased ROSC in OHCA patients (risk difference, 15.2%; 95% CI, 9.9%–20.6%) and in patients with initial nonshockable rhythms not witnessed by EMS (aOR, 2.14; 95% CI, 1.20–3.81). However, another study reported no association between naloxone administration and ROSC in OHCA patients (aOR, 0.43; 95% CI, 0.16–1.2) [50].

*Korean recommendations*

In patients experiencing cardiac arrest due to opioid toxicity, the available evidence is insufficient to support the effectiveness of administering additional opioid-specific therapies (e.g., naloxone) beyond standard resuscitative care during ALS. If the rescuer is unsure whether a patient with suspected opioid toxicity is in cardiac arrest, an opioid antagonist (e.g., naloxone) may be administered (good practice statement).

## Drowning

Drowning victims should be transported to a hospital for evaluation whenever possible. In particular, patients who require resuscitation (even if only rescue breathing) must be transported to a hospital for clinical assessment and monitoring, even if they are alert and their cardiopulmonary function is maintained normally. Because successful resuscitation or complete recovery of consciousness following prolonged submersion in cold water is rare, resuscitation efforts should be performed at the scene, and the patient should be transported to the emergency department unless death is clearly evident.

For patients who develop hypoxia during transport to the hos-

pital, supplemental oxygen should be provided to maintain an oxygen saturation of at least 94%. Endotracheal intubation may be performed if necessary (weak recommendation, very low certainty of evidence). In cases of moderate or severe acute lung injury following drowning, noninvasive ventilation may be considered as an alternative to mechanical ventilation (weak recommendation, very low certainty of evidence) [51–53]. However, noninvasive ventilation has been evaluated only in patients with a Glasgow Coma Scale score of ≥ 12 or in those who are hemodynamically stable; therefore, it may not be appropriate for all drowning victims.

### Severe hypothermia

In cases of severe hypothermia with a body temperature < 30 °C, the heart rate and respiratory rate decrease rapidly, making it difficult to determine whether cardiac arrest is present. Therefore, unless there is rigor mortis or injury incompatible with life, BLS and ALS should be continued until the patient's body temperature returns to normal. Aggressive rewarming, including invasive methods, is necessary, and early transport to a hospital is recommended compared with routine OHCA situations [54]. Removal of wet clothing to minimize ongoing heat loss, administration of warmed and humidified oxygen and intravenous fluids, and thoracic or peritoneal lavage using warm water may be necessary [55–57]. Rewarming rate can be increased using extracorporeal circulation devices [58–60]. Since patients with severe hypothermia may have accompanying conditions, such as drug toxicity, alcohol intoxication, or trauma, identifying underlying or contributing factors is necessary during rewarming therapy. If cardiac arrest occurs due to severe hypothermia, BLS and ALS should be performed according to the standard resuscitation guidelines.

### Cardiac arrest associated with electricity and lightning

Electric shock from alternating current used in homes or workplaces can induce tetanic skeletal muscle contractions, which may prevent the victim from releasing the source and result in prolonged exposure. Lightning injuries can also result in multiple casualties simultaneously. When rescuing a victim of electric shock, rescuer safety is paramount; rescue should be attempted only after safety is secured. A major cause of death from lightning is cardiac arrest, often due to ventricular fibrillation or asystole. Although cardiac function usually recovers spontaneously due to cardiac automaticity after a lightning strike, respiratory arrest may persist due to thoracic muscle spasms and suppression of the respiratory center after ROSC. Without timely respiratory support, hypoxic cardiac arrest may occur. Victims of lightning who do not develop respiratory or cardiac arrest or who respond promptly to initial intervention have a high probability of complete recovery. Therefore, in cases of multiple lightning casualties, patients with respiratory or cardiac arrest should be prioritized. If there is no evidence of breathing or circulation, BLS should be initiated immediately, and ventricular tachycardia or ventricular fibrillation should be treated using an automated external defibrillator. Cervical spine immobilization should be maintained due to the possibility of cranial and cervical spine injuries. Given the potential for significant soft tissue edema, early endotracheal intubation should be considered if airway compromise is anticipated. Adequate fluid resuscitation is also essential, as hypovolemic shock and continuous fluid loss may occur due to extensive tissue injury and necrosis.

### Cardiac arrest during percutaneous coronary intervention

If cardiac arrest occurs during percutaneous coronary intervention (PCI), the use of a mechanical CPR device is recommended [61]. Percutaneous ventricular assist devices such as intra-aortic balloon pumps, Impella (Abiomed Inc), or TandemHeart (LivaNova) may be used; however, their clinical benefit has not yet been clearly established [62]. Although several recent systematic reviews have reported the utility of ECPR, definitive recommendations remain unavailable. Therefore, the use of ECPR or other assistive devices in patients with cardiac arrest during PCI should be decided based on the patient's condition and the expertise of the specialized procedure team.

### Cardiac arrest caused by cardiac tamponade

If cardiac arrest occurs due to cardiac tamponade, emergency thoracotomy is the preferred treatment; however, ultrasound-guided pericardiocentesis may be attempted. If ultrasound is unavailable, pericardiocentesis without ultrasound guidance may be performed [63].

### Cardiac arrest following cardiac surgery

Cardiac arrest occurs in approximately 1% to 8% of patients after cardiac surgery, with common causes including ventricular fibrillation, hypovolemia, cardiac tamponade, and tension pneumothorax. Open-chest CPR may be attempted if cardiac arrest develops in the operating room, where the chest or abdomen is already open, or immediately after cardiac surgery [64,65]. If cardiac arrest occurs after cardiac surgery and the cardiac surgery team is present, direct cardiac compression can be performed following emergency resternotomy in an intensive care unit equipped with the necessary equipment. However, if emergency

resternotomy is not feasible, standard CPR must be performed without delay. In a patient treated in the intensive care unit who develops asystole or PEA and has a pacemaker inserted following cardiac surgery, pacing may be attempted immediately. In such cases, continuous hemodynamic assessment and ECG monitoring are essential. If pacing is not appropriate, ALS should be continued [66]. If cardiac arrest occurs after cardiac surgery and the patient does not respond to ALS, mechanical circulatory support may be helpful [67,68].

## Prone CPR

*Evidence summary*

In 12 adult case reports, 12 patients received prone CPR [69–80], and 8 underwent CPR after being turned to the supine position [81–87]. In 12 pediatric case reports, 11 patients received prone CPR [79,88–96], and 1 underwent CPR after being turned to the supine position [97]. Across 32 case reports (20 adults, 12 children), it is estimated that 31 patients (19 adults, 12 children) received prone CPR because repositioning to the supine position was difficult owing to head fixation or the presence of other equipment in the operating room. Only one adult patient in the intensive care unit underwent prone CPR [73].

Although objective neurological assessments of patients who survived to discharge with good neurological outcome were not performed, descriptions from eight adult cases included reports of no neurological damage [71], no cerebral damage [70], uneventful recovery [74], no damage [83], awake and oriented by day 7 [73], and no sequelae and discharged with stable neurological status [80].

In seven pediatric cases, reported descriptions included no evidence of severe brain dysfunction [88], recovery to baseline after 2 weeks [89], recovery without sequelae [90], no neurological sequelae [91], good condition [97], recovery without specific problems [79], and unchanged from preoperative status [92].

Objective assessments of good neurological outcome at 30 days or more after survival to discharge were not reported; however, two adult cases were described as having no damage [83], and being able to perform tasks and activities of daily living independently [85], as well as being able to perform tasks and activities of daily living independently [88], and recovery without sequelae [90].

Survival to hospital discharge was reported in 13 adults and 11 pediatric patients. Regarding 30-day survival or more, prone CPR was reported in one adult case [75], whereas supine CPR was reported in six cases. Five pediatric cases reported survival of 30 days or more [88,90,93,94].

Reports on the time taken to start CPR have been described in eight adult cases [69,72,73,78–80,82,85] and seven pediatric cases [79,88–90,93,94]. Prone CPR was initiated immediately in six adult cases [69,72,73,78–80], and seven pediatric cases [79,88–90,93,94]. In one adult case, the patient was immediately turned to the supine position for CPR [82], and in another adult case, the patient was turned to the supine position for CPR 6 minutes after cardiac arrest [85].

Two simulation studies reported that the time taken to turn the patient to the supine position for CPR ranged from 50±34 to 110 seconds [71,98]. One of these studies reported that the time taken to start CPR was 77±31 seconds [98].

Time to defibrillation was reported in one simulation study; the time taken to defibrillate in the prone position without chest compressions or position change was 22 seconds, whereas the time taken to turn the patient to the supine position and perform CPR and defibrillation was 108±61 seconds [98]. Notably, no adult or pediatric case report included comparable defibrillation data.

Time to ROSC was reported in all the studies. Two nonrandomized studies with a very low certainty of evidence reported arterial pressure measurements during CPR in a total of 17 patients [99,100]. Both studies reported higher systolic blood pressure during prone CPR (72 mmHg vs. 48 mmHg, $P<0.005$ [99]; 79±20 mmHg vs. 55±20 mmHg, $P=0.028$ [100]). Diastolic blood pressure was higher in only one of the two studies (34 mmHg vs. 24 mmHg, nonsignificant [99]; 17±10 mmHg vs. 13±7 mmHg, $P=0.028$ [100]). However, in the second study, data for 3 of 11 patients measured in the supine position were missing [100].

The $ETCO_2$ partial pressure during CPR was reported in five adult cases [72,74,75,80,84], and two studies reported values of 15 and 33 mmHg [75,80]. Two pediatric cases both showed $ETCO_2$ partial pressures of 10 mmHg or higher during prone CPR [94,96].

*Recommendations*

In patients who experience cardiac arrest in the prone position, prone CPR may be attempted if the patient is intubated, and it is difficult or unsafe to turn the patient to the supine position immediately (good practice statement). Invasive arterial pressure or $ETCO_2$ partial pressure monitoring can be useful indicators of perfusion pressure during prone CPR and may assist in determining the optimal timing for repositioning the patient to the supine position for CPR (good practice statement). If a patient with cardiac arrest in the prone position does not have an advanced airway in place, it is recommended that the patient be turned immediately

to the supine position for CPR (strong recommendation, low certainty of evidence). If a patient requiring defibrillation is in the prone position and cannot be turned immediately, defibrillation may be performed in the prone position (good practice statement).

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### Conflicts of interest

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### Data availability

Data sharing is not applicable as no new data were created or analyzed in this study.

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