Revista da SOCESP
Sociedade de Cardiologia do Estado de São Paulo

POST-CARDIOPULMONARY RESUSCITATION (CPR) CARE

CUIDADOS PÓS-RESSUSCITAÇÃO CARDIOPULMONAR (RCP)

Natali Schiavo Giannetti[1]
Sergio Timerman[1,2]

1. Laboratory of Training and Simulation in Cardiovascular Emergencies of InCor – HCFMUSP, São Paulo, SP, Brazil.
2. Fellow of FACC (American College of Cardiology), FERC (European Resuscitation Council), FESC (European Society of Cardiology), FAHA (American Heart Association), FACP (American College of Physician)

Correspondence:
Natali Schiavo Giannetti
R. D. Adma Jafet, 74 Cj 84, Bela Vista, São Paulo, SP, Brazil.
natali.giannetti@incor.usp.br

Received on 07/30/2018,
Accepted on 09/15/2018

ABSTRACT

Avoid further episodes of cardiac arrest (CA). Identify and treat the causes of the patient's CPA. Provide ventilatory, hemodynamic, neurological and metabolic support. Perform therapeutic temperature modulation for all patients who have resumed spontaneous circulation. Indication of cardiac catheterization for patients with no established cause of CPA when the cause may be a coronary event.

Keywords: Cardiac Arrest; Emergencies; Patients.

RESUMO

Evitar novos episódios de parada cardiorrespiratória (PCR). Identificar e tratar as causas que levaram o paciente à PCR. Oferecer suportes ventilatório, hemodinâmico, neurológico e metabólico. Realizar a modulação terapêutica de temperatura para todos os pacientes que retornaram à circulação espontânea. Indicação de cateterismo cardíaco para pacientes sem causa estabelecida de PCR quando a causa pode ser um evento coronariano.

Descritores: *Parada Cardiorrespiratória; Emergência; Pacientes.*

INTRODUCTION

After a cardiac arrest (CA), patients who achieve return of spontaneous circulation (ROSC) are considered extremely at risk, with hospital mortality rates of approximately 63% to 90%.[1-3] We prefer using the term post-cardiopulmonary resuscitation care (post-CPR care) because care is given to patients who return to spontaneous circulation after the initial measures. The possible causes of CA must be identified and corrected immediately and early to avoid recurrence (Figure 1). Although the initial aggressor factor is ischemia, which occurs at the time of CPA, lesions occur during and after reperfusion.[4,5]

Post-CPA syndrome is defined as a complex pathophysiological state consisting of the combination of three main situations, summarized in Figure 2. Proactive treatment programs should be implemented by multidisciplinary teams in intensive care units with optimized ventilatory, hemodynamic, neurological, and metabolic supports.

VENTILATORY SUPPORT

It is important to ensure that the airways are open. Patients who are kept on bag-valve-mask ventilation during CPR must be evaluated for the need for invasive airway devices. Supraglottic devices inserted during CPR maneuvers by a definite airway, such as a tracheal tube, may need to be changed. After ROSC, some patients may regain consciousness, which is enough to maintain spontaneous ventilation. Teams should consider the risk of bronchoaspiration (Figure 3).

Hyperventilation should be avoided because it reduces cardiac output and promotes a reduction in cerebral perfusion. The optimal CO_2 levels in post-CPA are shown in Table 1.

Metabolic acidosis is common in patients after CPA. With ROSC, restoration of adequate tissue perfusion may

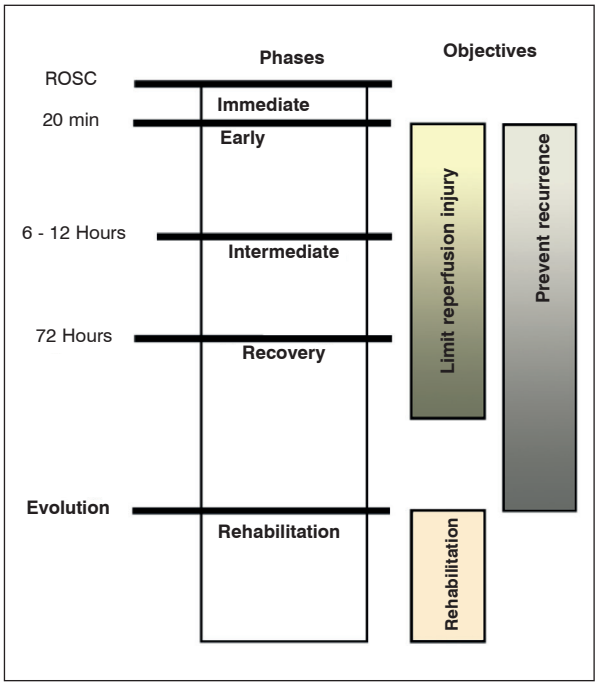

Figure 1. Phases of recovery after ROSC and goals of care.

- Brain injuries post-CPA
- Myocardial lesions and dysfunctions post-CPA
- Ischemic and reperfusional response of multiple organs

Figure 2. Post-CPA syndrome.

- Oxygen saturation of >94%
- Pulse oximetry and quantitative capnography should be monitored continuously. FiO2 should be titrated to maintain SaO2 between 94% and 96%, seeking to avoid excessive levels of oxygen due to the risk of hyperoxia lesions.

Figure 3. Goals for ventilatory support.

Table 1. Optimal levels of CO2 post CPA.

Measurement	Optimal value	Measuring mode
$PETCO_2$	35 to 40 mmHg	Capnography
$PaCO_2$	40 to 45 mmHg	Arterial blood gas test

be enough to correct acidosis. After ROSC, a hyperemic phase can last from 10 to 30 minutes, followed by a phase of low global cerebral perfusion secondary to vasoconstriction that can last for several hours. In this second phase, hyperventilation may enhance vasoconstriction, further reducing cerebral perfusion.

Acute lung injury may occur in patients after CPA and may be worsened by high concentrations of O_2. Therefore, the PaO_2/FiO_2 ratio should be carefully monitored.

HEMODYNAMIC SUPPORT

Patients who achieve ROSC should be maintained under continuous cardiac monitoring, and venous access should be reassessed. In cases where emergency intraosseous access is required, replacement by intravenous access can be attempted. If vasoactive drugs are required, central venous access should be considered.

Hypovolemia should be avoided, especially in cases of hemodynamic lability. Volume replacement should be considered to improve organic perfusion. In cases with indication of hypothermia, volume replacement should be performed with solutions at 4°C. Organic perfusion monitoring may be performed by arterial lactate and central venous saturation or mixed venous saturation.

Vasopressors are indicated in cases of shock refractory to volume replacement to maintain a mean arterial pressure of >65 mmHg and systolic blood pressure of >90 mmHg. No evidence shows the benefit of one vasopressor in relation to others (Table 2).

The search for a state of normovolemia can be a complex challenge in some patients. Acute kidney failure is frequent and should be identified early. Indications for dialysis therapy in post-CPA patients are the same indications for critically ill patients in general. Cardiac function may be depressed after CPA, and volume replacement is not always well tolerated.

Table 2. Vasopressors commonly used in the post-CPA patients who were refractory to volume expansion.

Dopamina	5 to 10 mcg/kg/minute
Noradrenalina	0.1 to 0.5 mcg/kg/minute
Epinephrine	0.1 to 0.5 mcg/kg/minute

If coronary ischemia is considered a probable cause of CPA, performing early coronary angiography should be encouraged, even in the absence of electrocardiographic or laboratory evidence of ischemia or myocardial necrosis. This is justified by the high prevalence of coronary diseases, which accounts for 65–70% of pre-hospital CPA cases and associated with a low negative predictive value of the absence of acute ischemic changes on electrocardiography (ECG), approximately 44%.[6] Approximately 26% of patients with ROSC with no ST-segment elevation on the ECG present culprit lesions to coronary angiography.[7]

Specific cases may require artificial electrical stimulation with a transcutaneous or transvenous pacemaker. In patients with refractory cardiogenic shock, the use of intra-aortic balloon or circulatory assistance devices may be necessary.

In all the patients, a 12-lead ECG should be performed. Additional tests such as echocardiography and magnetic resonance imaging may be needed to rule out structural cardiac changes. Primary electrical diseases can be investigated when coronary angiography rules out coronary disease and if no structural changes are found.

No evidence has been found to indicate the use of routine maintenance antiarrhythmic drugs in post-CPA. Detailed patient history taking must be conducted, including list of comorbidities and medications used to assess the possibility of toxicity and medications that prolong the QT interval or interfere with hydroelectrolytic control. Research on the use of illicit drugs must also be performed, and toxicological tests may be required.

In suspected cases of pulmonary embolism, the increased risk of bleeding from CPR maneuvers is not impeditive, and fibrinolytic drugs may be considered. Alternative therapies include mechanical thrombectomy and surgical embolectomy.

METABOLIC SUPPORT

Frequent glycemic monitoring is recommended, and hypoglycemia should be avoided. Control strategies and levels that require the use of insulin should be defined according to the local policy of each institution. An acceptable target is the maintenance of glycemic levels of approximately 144 mg/dL. The strategies of strict glycemic control failed to show benefits when compared to more-tolerant glycemic control strategies, probably because of the negative effects of the higher incidence of hypoglycemia.

Potassium and magnesium disorders should be promptly corrected. Potassium changes are in the list of the most frequent reversible causes of CPA. Hypokalemia is frequent, and the incidence of arrhythmias increases. Potassium levels should be maintained at >3.5 mEq/L. Hypomagnesemia may be related to increased QT interval and to the occurrence of *torsade de pointes*.

PROPHYLACTIC MEASURES

Prophylactic measures for the prevention of ventilator-associated pneumonia, stress ulcers, and venous thromboembolism should be performed.

NEUROLOGICAL SUPPORT

Neurological lesions are the major cause of mortality in patients with ROSC and accounted for 68% of deaths in

patients with CPA in an out-of-hospital setting.[8] Induction of moderate hypothermia should be considered in cases where the patient with ROSC remains comatose. Moderate hypothermia is the only consistent measure to avoid neurological reperfusion injury[9,10] (Figure 4).

Temperature control should be instituted for all patients post CPA, and fever should be intensively controlled. For these cases, the use of antipyretics is questionable. Hypothermia has more-restricted indications and should be reserved for hospitals that have well-established protocols and trained staff.[11] Hypothermia is not recommended in the pre-hospital setting.

THERAPEUTIC TEMPERATURE MODULATION

There are doubts regarding the selection of patients who can benefit from therapeutic hypothermia, identification of the ideal time to start, and the duration for maintaining hypothermia measures. Recent data from clinical trials of patients with all rhythms, identification of various adverse effects on therapy, high neurological morbidity, and mortality without specific interventions suggest that temperature is an important variable for post-7 recovery without neurological sequelae.

Thus, having a temperature variation modulated between a minimum parameter of 32°C to a maximum of 36°C is believed to possibly allow therapeutic temperature management according to the patient's hemodynamics.

Temperature modulation will depend on the specific conditions that the patient presents in the ROSC; for example, temperatures between 35°C and 36°C may be preferable for patients who present coagulation disorders that need anticoagulant therapy.

Hypothermia may be performed in patients who present seizures, brain edema, and severe head trauma. Thus, therapeutic temperature modulation is suggested as following cases: When temperature control is indicated, the temperature should be maintained at a range of 32°C to 36°C, but a target should be chosen, and little variation of the target temperature must be allowed throughout the temperature modulation.

In some subpopulations, the benefit associated with low temperatures ranging from 32°C to 34°C or higher temperatures of 36°C remain uncertain, and further studies should be performed. Temperature control is recommended for adult survivors of out-of-hospital CPA with early VF/VT rhythm and patients who remain in coma after ROSC. Temperature control is suggested for adult survivors of out-of-hospital CPA with an initial non-shockable rhythm and for patients who remain in coma after ROSC. Temperature control is suggested for adult survivors of in-hospital CPA at any initial rhythm and for patients who remain unresponsive to verbal commands after ROSC.

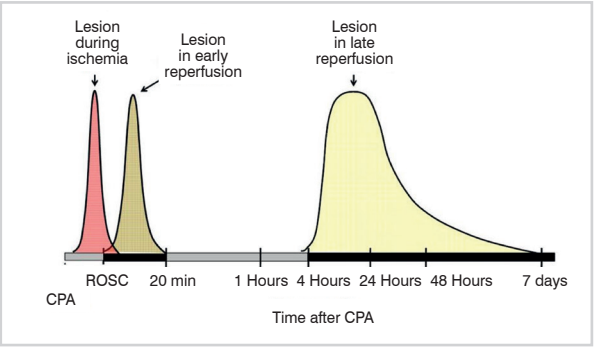

Figure 4. Times of ischemia and reperfusion injury.[9,10]

Whenever temperature modulation is initiated, maintaining the temperature for at least 24 hours is suggested. The neurological prognosis can be difficult to determine during the first 72 hours after resuscitation, as sedation, paralysis, and agitation may be confounding factors for prognostic determination.

It is important to note that the central temperature should be continuously monitored through an esophageal thermometer, bladder catheter with a temperature sensor (as long as the patient maintains a minimum urine output of 30 mL/hour), or a pulmonary artery catheter. The temperature measured through axillary, rectal, or oral thermometers is not adequate to evaluate the dynamic changes of the central temperature.

Hyperthermia should be actively avoided within 48 hours after CPA, as it is associated with increased mortality and worse outcomes.[11,13] To induce hypothermia, several methodologies can be used, from the simplest such as infusion of isotonic solution at 4°C in pressurized pockets associated with ice or cold bags on the patient's neck, armpits, and groin, to intravenous devices with a catheter intravascular feedback. An institutional protocol is needed to standardize the temperature modulation, with the suggestion in Figure 5[14]. No evidence has shown differences in results between methodologies to date, and the method should be chosen according to equipment availability and team experience.

Devices have been developed to enhance temperature control that can induce hypothermia internally by intravascular systems (positioned in the subclavian, internal jugular, or femoral veins) or externally by hydrogel plates (placed on the chest, abdomen, and roots of the limbs of patients). Both are effective, and their advantage over simpler methods consists in more precise and less labor-intensive control.

Once the selected temperature is reached, temperature variations should be avoided, as they increase the risk of complications such as coagulopathies, arrhythmias, hyperglycemia, pneumonia, sepsis, and hemorrhages.

Sedation, neuromuscular blockade, and tremor prevention medications should be part of hypothermia protocols. In the hypothermia induction protocol, comatose patients can undergo coronary angiography.

During hypothermia, some laboratory parameters may require corrections. If the patient's temperature is 33°C, the $PaCO_2$ may be 6 to 7 mmHg lower than the value obtained by the equipment. Pre-hospital therapeutic hypothermia using a rapid infusion of large volumes of ice-cold intravenous fluid immediately after ROSC is not recommended. Evidence indicates that the administration of intravenous fluid in large quantities may increase pulmonary edema and the possibility of new CPA.

The recommendation is to induce temperature modulation in post-CPA patients in a comatose state. The target temperature is between 32°C and 36°C (central temperature) for at least 24 hours. Therapeutic hypothermia can be divided into three phases, namely induction, maintenance, and reheating.

CONFLICTS OF INTEREST

The author declares that he has no conflicts of interest in this work.

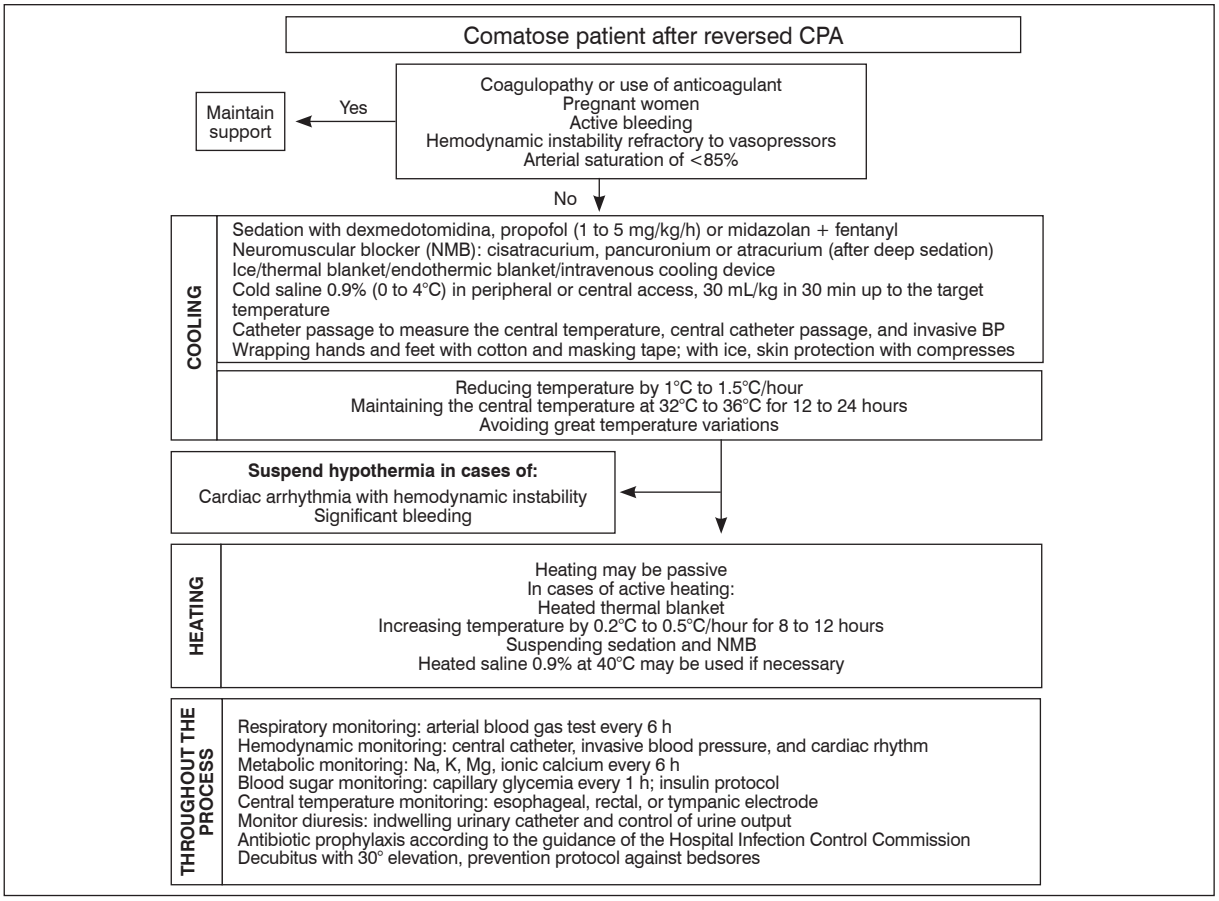

Figure 5. Temperature modulation algorithm in post-CPR patients.

AUTHORS' CONTRIBUTIONS: Each author has contributed equally in the elaboration of this publication.

REFERENCES

1. Herlitz J, Engdahl J, Svensson L, Angquist KA, Silfverstolpe J, Holmbert S. Major differences in 1-month survival between hospitals in Sweden among initial survivors of out-of-hospital cardiac arrest. Resuscitation. 2006;70:404-9.
2. Keenan SP, Dodek P, Martin C, Priestap F, Norena M, Wong H. Variation in length of intensive care unit stay after cardiac arrest: where you are is as important as who you are. Crit Care Med. 2007;35:836-41.
3. Mashiko K, Otsuka T, Shimazaki S, Kohama A, Kamishima G, Katsurada K, et al. An outcome study of outof-hospital cardiac arrest using the Utstein template: a Japanese experience. Resuscitation. 2002;55:241-6.
4. Nadkarni VM, Larkin GL, Peberdy MA, Carey SM, Kaye W, Mancini ME, et al; National Registry of Cardiopulmonary Resuscitation Investigators. First documented rhythm and clinical outcome from in-hospital cardiac arrest among children and adults. JAMA. 2006;295:50-7.
5. Stiell IG, Hébert PC, Wells GA, Vandemheen KL, Tang AS, Higginson LA, et al. Vasopressin versus epinephrine for in hospital cardiac arrest: a randomised controlled trial. Lancet. 2001;358(9276):105-9.
6. Spaulding CM, Joly LM, Rosenberg A, Monchi M, Weber SN, Dhainaut JF, et al. Immediate coronary angiography in survivors of out-of-hospital cardiac arrest. N Engl J Med. 1997;336:1629-33.
7. Radsel P, Knafelj R, Kocjancic S, Noc M. Angiographic characteristics of coronary disease and postresuscitation electrocardiograms in patients with aborted cardiac arrest outside a hospital. Am J Cardiol. 2011;108:634-8.
8. 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;30:2126-8.
9. Neumar RW. Molecular mechanisms of ischemic neuronal injury. Ann Emerg Med.2000;36(5):483–506.
10. Becker LB. New concepts in reactive oxygen species and cardiovascular reperfusion physiology. Cardiovasc Res. 2004;61(3):461–70.
11. Zeiner A, Holzer M, Sterz F, Schörkhuber W, Eisenburger P, Havel C, et al. Hyperthermia after cardiac arrest is associated with an unfavorable neurologic outcome. Arch Intern Med. 2001;161:2007–12.
12. Gonzalez MM, Timerman S, Gianotto-Oliveira R, Polastri TF, Canesin MF, Schimidt A, et al. Sociedade Brasileira de Cardiologia. I Diretriz de Ressuscitação Cardiopulmonar e Cuidados Cardiovasculares de Emergência da Sociedade Brasileira de Cardiologia. Arq Bras Cardiol. 2013;101(2Supl.3): 1-221
13. Polderman K, Malinoski D, Timerman S, Keeble T. Current Advances in the Use of Therapeutic Hypothermia. Ther Hypothermia Temp Manag. 2018;8(1):9-13.
14. Rocha TH, Vieira SR. Hipotermia terapêutica em pacientes pós-parada cardiorrespiratória: mecanismos de ação e desenvolvimento de protocolo assistencial. Rev Bras Ter Intensiva. 2010;22(2):196-205.