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The optimal hemodynamics management of post-cardiac arrest shock

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Patients resuscitated from cardiac arrest develop a pathophysiological state named "post–cardiac arrest syndrome." Post-resuscitation myocardial dysfunction is a common feature of this syndrome, and many patients eventually die from cardiovascular failure. Cardiogenic shock accounts for most deaths in the first 3 days, when post-resuscitation myocardial dysfunction peaks. Thus, identification and treatment of cardiovascular failure is one of the key therapeutic goals during hospitalization of post-cardiac arrest patients.

Patients with hemodynamic instability may require advanced cardiac output monitoring. Inotropes and vasopressors should be considered if hemodynamic goals are not achieved despite optimized preload. If these measures fail to restore adequate organ perfusion, a mechanical circulatory assistance device may be considered.

Adequate organ perfusion should be ensured in the absence of definitive data on the optimal target pressure goals. Hemodynamic goals should also take into account targeted temperature management and its effect on the cardiovascular function.

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Introduction

Despite initially successful resuscitation, morbidity and mortality following cardiac arrest (CA) remain high. Indeed, patients resuscitated from CA develop a pathophysiological state named "post-cardiac arrest syndrome." It is characterized by myocardial dysfunction with circulatory shock, systemic inflammation with activation of the clotting system, evolving brain injury, and a persistent precipitating pathology.[1–3] The severity of these manifestations is extremely variable, depending mainly on the duration and cause of CA (Table 1).

Post-resuscitation myocardial dysfunction is constituted by a mechanical and electrical component. The first may lead to severe impairment in myocardial contractility, while also compromising diastolic function [4]. The electrical component is responsible for cardiac dysrhythmias, and it can evolve into electrical storm in the most severe cases [4]. These components cause hemodynamic instability, which manifests as hypotension and low cardiac index, possibly leading to multiple organ failure.[5,6] Myocardial impairment is global in contrast to the regional impairment of myocardial ischemic injury due to coronary artery occlusion. Like reversible ischemic injury of coronary disease, also known as myocardial stunning, post-resuscitation myocardial dysfunction is reversible over days [7,8].

Significant myocardial dysfunction is common after CA, but the patient typically starts to recover by 2–3 days, although full recovery may take significantly longer [5,9]. Whole-body ischemia/reperfusion of CA activates immune and coagulation pathways, contributing to multiple organ failure and increasing the risk of infection [10–12]. Thus, the post–CA syndrome has many features in common with sepsis, including intravascular volume depletion, vasodilation, endothelial injury, and abnormalities of the microcirculation [13–15].

Hemodynamic monitoring after CA

Cardiogenic shock is common after resuscitation from CA, and many patients eventually die from cardiovascular causes.[5,16–18] Among patients surviving to ICU admission but subsequently dying in hospital, brain injury is the cause of death in approximately two-thirds after out-of-hospital cardiac arrest (OHCA) and approximately 25% after in-hospital CA.[16,19] Cardiovascular failure accounts for most deaths in the first 3 days, when post-resuscitation myocardial dysfunction peaks, while brain injury accounts for most of the later deaths.[16,19] Laurent and coworkers reported a reversible decline in cardiac index during the first 24 h after CA and a superimposed vasodilation lasting up to 72 h. If cardiac function failed to improve by 24 h, all patients died from multiple organ failure [5].

Every patient admitted to the intensive care should be screened for post-resuscitation myocardial dysfunction, because it is common and carries significant treatment implications. An echocardiographic evaluation (transthoracic or transesophageal) should be performed early, ideally on admission, in all patients in order to detect and quantify the degree of myocardial dysfunction [20,21]. Echocardiography may be repeated in the first few hours because the onset of post-resuscitation myocardial dysfunction may be delayed for a few hours. Myocardial dysfunction often requires inotropic support, at least transiently. Treatment may be guided by blood pressure, heart rate, urine output, rate of plasma

Table 1
Post–cardiac arrest syndrome: main features and principal therapeutic end points.

Manifestation	Therapeutic options
Myocardial dysfunction	Volume optimization by intravenous fluids Inotropes (e.g., dobutamine) Mechanical circulatory assistance devices
SIRS	Vasopressors
Brain injury	Target temperature management Active prevention and treatment of post-hypothermia fever
Persistent precipitating pathology	If cardiac cause suspected: coronary arteriography If noncardiac: treat according to specific cause

SIRS: Systemic inflammatory response syndrome.