

Neurologic Aspects of Cardiac Emergencies

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KEYWORDS

• Cardiac emergencies • Neurologic complications • Stroke • Acute brain injury

KEY POINTS

- Therapeutic hypothermia has improved neurologic outcomes after cardiac arrest, with newer prognostication models emerging.
- Hemorrhagic and ischemic stroke and transient ischemic attack are the most common neurologic complications associated with aortic dissection, ventricular assist devices and coronary artery bypass grafting.
- Neurologic complications occur in 20–40% of cases of infective endocarditis and include stroke, intracerebral hemorrhage, meningitis, brain abscess and intracranial mycotic aneurysms.
- There is a lack of data on the management of aortic dissection, ventricular assist devices, coronary artery bypass grafting and infective endocarditis in the setting of neurologic complications.
- Neurogenic stunned myocardium is an important disease entity that can mimic an acute myocardial infarction, but is a stress cardiomyopathy related to neurologic illness.

INTRODUCTION

Cardiac emergencies can be associated with a wide variety of neurologic complications and sequelae. These sequelae can be the most important factor in determining patient outcomes, including long-term functional outcomes. In this review, the important topic of cardiac arrest are discussed, with a focus on neuroprognostication and

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the emerging data, with regard to identifying more accurate predictors of neurologic outcomes in the era of therapeutic hypothermia (TH). Some of the recent controversies with regard to targeted temperature management in comatose survivors of cardiac arrest are also discussed. The next section focuses on neurologic complications associated with surgical disease and procedures, namely aortic dissection, infective endocarditis (IE), left ventricular (LV) assist devices (LVADs), and coronary artery bypass grafting (CABG). We believed that these topics should be discussed given the complexities in their management. In the final section of the review, the cause, pathogenesis, and management of neurogenic stunned myocardium (NSM), an important clinical entity, are discussed.

ANOXIC-ISCHEMIC ENCEPHALOPATHY Epidemiology

About 450,000 Americans have cardiac arrest (CA) annually.¹ Eighty percent of CAs occur at home, and mortality for such arrests is more than 90%.^{1,2} More than half the survivors have permanent brain damage of varying degrees.^{3,4} The outcomes for inhospital arrests are better, with restoration of spontaneous circulation in 44% of patients and survival to discharge in 17% of patients. Survivors of CA who remain comatose are presumed to have anoxic-ischemic encephalopathy. A wide range of neurologic outcomes is possible in such patients, ranging from brain death to good recovery.

Neuroprognostication and Predictors of Poor Outcome

The American Academy of Neurology has identified several predictors of poor neurologic outcome to assist with prognostication.⁵ These predictors include absent pupillary or corneal reflexes at day 3 after CA, absent or extensor motor responses at day $3,^{6-8}$ bilateral absent N20 responses of somatosensory evoked potentials on days 1 to $3,^{9-12}$ serum neuron-specific enolase (NSE) greater than 33 ng/mL at days 1 to $3,^{7,13}$ and the presence of myoclonus status epilepticus within 24 hours.^{7,14}

In prospective studies involving 491 patients,^{6,15–19} all 108 patients who had absent pupillary light reflex (PLR) at day 3 after CA had poor outcomes. Absent corneal reflex at 72 hours was shown to be associated with poor outcomes in 2 prospective studies.^{6,7} In another prospective study of 407 patients, myoclonic status epilepticus at 24 hours after CA was associated with no false-positive results for poor outcomes (95% confidence interval [CI], 0–14).⁷ Several prospective studies have shown that a motor response to noxious stimuli that was no better than extensor posturing at 72 hours was associated with poor outcome.^{6,7,16} However, these indicators of poor prognosis are derived from patients not treated with TH.

Several recent studies have looked into the accuracy of these predictors in the era of TH after CA. Fugate and colleagues²⁰ found that absent PLR, absent corneal reflexes, and motor response no better than extensor at day 3 remained predictive of a poor outcome in patients who received TH. Similarly, absent N20 responses were still predictive of poor outcome. However, an increased NSE level measured 1 to 3 days after CA was a less reliable predictor of poor outcome in such patients. However, pharmacologic sedation related to TH may influence prognostication of neurologic outcomes after CA. Recovery of motor responses, especially, may be delayed. A small study of 37 patients²¹ showed that 2 of 14 patients who had a motor response no better than extensor posturing on day 3 showed some recovery of consciousness on day 6. Table 1 compares the predictors of poor outcome both before and after the era of TH.

More recently, Oddo and Rossetti²² adopted a multimodal approach for outcome prediction after CA and TH. These investigators found that clinical examination, which Download English Version:

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