# Sudden cardiac arrest

Yagi et al. monitored CBO in 20 patients with cardiac arrest by NIRS. On the arrival of patients at the emergency department, the attending physician immediately assessed whether the patient was eligible for this study after conventional advanced life support and, if eligible, measured CBO in the frontal lobe by NIRS. They found that in all patients, the cerebral blood flow waveform was in synchrony with the chest compressions. Moreover, the tissue oxygenation index increased following cardiopulmonary bypass (CPB) in patients undergoing CPB, including one patient in whom CBO was monitored using the NIRO-CCR1. In addition, although the NIRO-CCR1 could display the pulse rate (Tempo) in real-time, Tempo was not always detected, despite the detection of the cerebral blood flow waveform. This suggested that chest compressions may not have been effective, indicating that the NIRO-CCR1 also seems useful to assess the quality of CPR. This study suggests that the NIRO-CCR1 can measure CBO during CPR in patients with cardiac arrest as effectively as the NIRO-200NX; in addition, the new NIRO-CCR1 maybe even more useful, especially in prehospital fields (e.g. in an ambulance), since it is easy to carry [1)].

## Outcome

Anoxic brain injury represents the main determinant of poor outcome after cardiac arrest (CA).

Approximately 15% of deaths in a developed country are due to sudden cardiac arrest, making it the most common cause of death worldwide. Though high-quality cardiopulmonary resuscitation has improved overall survival rates, the majority of survivors remain comatose after return of spontaneous circulation secondary to hypoxic ischemic injury. Since the advent of targeted temperature management, neurologic recovery has improved substantially, but the majority of patients are left with neurologic deficits ranging from minor cognitive impairment to persistent coma. Of those who survive cardiac arrest, but die during their hospitalization, some progress to brain death and others die after withdrawal of life-sustaining treatment due to anticipated poor neurologic prognosis [2)].

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Hayman et al. sought to review the role that cerebral edema plays in neurologic outcome following cardiac arrest, to understand whether cerebral edema might be an appropriate therapeutic target for neuroprotection in patients who survive cardiopulmonary resuscitation. Articles indexed in PubMed and written in English. Following cardiac arrest, cerebral edema is a cardinal feature of brain injury and is a powerful prognosticator of neurologic outcome. Like other conditions characterized by cerebral ischemia/reperfusion, neuroprotection after cardiac arrest has proven to be difficult to achieve. Neuroprotection after cardiac arrest generally has focused on protecting neurons, not the microvascular endothelium or blood-brain barrier. Limited preclinical data suggest that strategies to reduce cerebral edema may improve neurologic outcome. Ongoing research will be necessary to determine whether targeting cerebral edema will improve patient outcomes after cardiac arrest [3)].

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Sudden cardiac arrest (SCA) is one of the leading causes of mortality and morbidity in the United States, and survivors are frequently left with severe disability. Of the 10% successfully resuscitated

from SCA, only around 10% of these live with a favorable neurologic outcome. Survivors of SCA commonly develop post-cardiac arrest syndrome (PCAS). PCAS is composed of neurologic, myocardial, and systemic injury related to inadequate perfusion and ischemia-reperfusion injury with free radical formation and an inflammatory cascade. While targeted temperature management is the cornerstone of therapy, other intensive care unit-based management strategies include monitoring and treatment of seizures, cerebral edema, and increased intracranial pressure, as well as prevention of further neurologic injury. In this review, we discuss the scientific evidence, recent updates, future prospects, and knowledge gaps in the treatment of post-cardiac arrest patients [4].

1)
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