Hypoxic–ischaemic brain injury following cardiac arrest

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Abstract

Hypoxic–ischaemic brain injury (HIBI) has become an increasingly common cause of admission to intensive care units and prolonged or permanent neurological disability. It is essential to provide an accurate prognosis to guide management. Clinical assessment is often difficult and ancillary investigation is necessary. Outcome depends on the severity of the initial insult, the effectiveness of immediate resuscitation and transfer, and the post-resuscitation management on the intensive care unit. Some patients remain profoundly impaired and others may develop complications including myoclonic epilepsy, cognitive impairment, border-zone infarction and movement disorders. The management of patients with HIBI presents considerable clinical, ethical and legal difficulties.

Keywords Coma; hypoxic–ischaemic brain injury; induced hypothermia

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Learning objectives

After reading this article you should be able to:

- understand the management of patients following HIBI
- recognise the most important prognostic factors following HIBI – clinical, neurophysiological and radiological
- understand the complications of HIBI.

Hypoxic–ischaemic brain injury (HIBI) has become increasingly common as the use of bystander cardiopulmonary resuscitation has burgeoned.[1,2] Clinical assessment is often difficult because of the effects of sedation, neuromuscular blockade, ventilation, hypothermia and inotropic management. Ancillary investigation including EEG and imaging[3] may be needed to predict an accurate prognosis.

Outcome depends on the severity of the initial insult, the effectiveness of immediate resuscitation and transfer, and the post-resuscitation management in the intensive care unit. The prognosis for HIBI is extremely poor; only a quarter of patients survive to hospital discharge, and even then there is often severe neurological or cognitive deficit.[4]

Intensive care management

Following successful resuscitation and transfer, the initial aims of management are to establish:

- haemodynamic stability
- adequate tissue oxygenation
- to prevent secondary cerebral damage due to hypotension.

After successful resuscitation there is a brief period of brain hyperaemia but this may be followed by reduced cerebral blood flow (CBF; no-reflow)[5] as a result of microvascular thrombotic occlusion leading to failure of autoregulation. It is therefore important to maintain a relatively high mean arterial blood pressure (80–100 mmHg) with crystalloid infusion or vasopressor agents. Other aspects of acute care are summarised in Table 1.

Induced hypothermia

Hypothermia causes a decrease in CBF and a consequent reduction of cerebral metabolism. Randomised trials have shown that treated patients are more likely to be discharged with no or minimal neurological damage.[6] However, hypothermia can be difficult to apply and uncomfortable, patients require sedation and a neuromuscular blocking agent (vecuronium or pancuronium) to prevent shivering, and so the technique is restricted to those patients in coma.

Assessment

The clinical effects and consequences of HIBI are highly variable. The best outcome is generally seen in patients who have had only a short period of impaired consciousness following resuscitation. Regaining purposeful motor movements and awareness with preserved memory within a few hours is usually associated with a good recovery and functional independence. However, the literature studying prognostic factors and outcome is difficult to apply because it is largely based on studies before the introduction of techniques of post-resuscitation support, including the early use of sedation and ventilation, which now prevents a full assessment of conscious level and neurological state in the immediate post-arrest period.[7,8] It is particularly important to emphasise that the use of hypothermia and sedation and neuromuscular paralysis may cause a delay in motor recovery.

If early assessment is possible the outcome of hypoxic–ischaemic brain injury worsens if:

- the patient has been in coma (i.e. unresponsive) for >6 hours
- lack of spontaneous limb movements or failure to localise painful stimuli in the initial stages
- prolonged loss of pupillary responses (provided atropine has not been administered)
- sustained conjugate eye deviation (upgaze or downgaze)
- specific forms of abnormal eye movements (e.g. upbeat and downbeat nystagmus, ping pong gaze or period alternating nystagmus)
- myoclonic seizures.

The conventional method to assess the depth of coma is the Glasgow Coma Score (GCS). This has proved valuable, robust and reproducible but was designed to monitor patients with head injury and is not thorough or detailed enough to permit accurate

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Aspects of acute care following HIBI

Intubation and ventilatory support	
Sedation	(e.g. midazolam, fentanyl) will reduce the stress of procedures and facilitate ventilation.
Neuromuscular blockade	May be necessary if therapeutic hypothermia is used
Blood sugar control	Use is controversial (hypoglycaemia may reduce cerebral blood flow)
Fever and seizures	Both increase metabolic demand on the brain, should be treated aggressively

Table 1

assessment of medical causes of coma such as hypoxic–ischaemic brain injury.

Aspects of clinical management

It is important to consider the possibility that the patient is locked-in where there is preservation of consciousness despite complete paralysis of cranial nerve and limb musculature. Any residual responsive eye movements must be intensively sought.[9]

Repeated clinical assessment may be necessary before the outcome can be predicted. Also it is necessary to wait for any effects of medication, hypothermia, sepsis and intercurrent metabolic effects to resolve. In practice, whether an oropharyngeal tube should be replaced by a cuffed tracheostomy, which makes prolonged survival more likely even if ventilatory support is reduced or withdrawn, is a critical decision. This decision therefore requires very careful consideration between intensivists, neurologists and the patient's family. However, the decision to undertake tracheostomy does not have to be made in the acute stages and can often be delayed for several days. It is important to emphasise that the decision to reduce or discontinue support cannot be rushed and all members of the team and the family must be in agreement with the management decisions.

Ancillary investigations play an important role in the assessment—EEG, SSEP and MRI can provide detailed, accurate and reliable information about the distribution and severity of hypoxic–ischaemic brain injury.

EEG: the EEG has been widely used over many years to assess the level of consciousness and to guide prognosis after hypoxic–ischaemic brain injury. The appearances are influenced by confounding factors including medication, metabolic derangements and sepsis.

A number of patterns suggest a poor prognosis:

- generalised electrical suppression
- generalised burst suppression
- unresponsive alpha, theta or delta rhythms
- periodic patterns
  - periodic lateralised epileptiform discharges (PLEDS)
  - bilateral independent PLEDS (BiPLEDS) or synchronous (BiPEDS)

Short latency somatosensory evoked potentials (SSEPs) are valuable in assessing the prognosis following HIBI. Bilateral

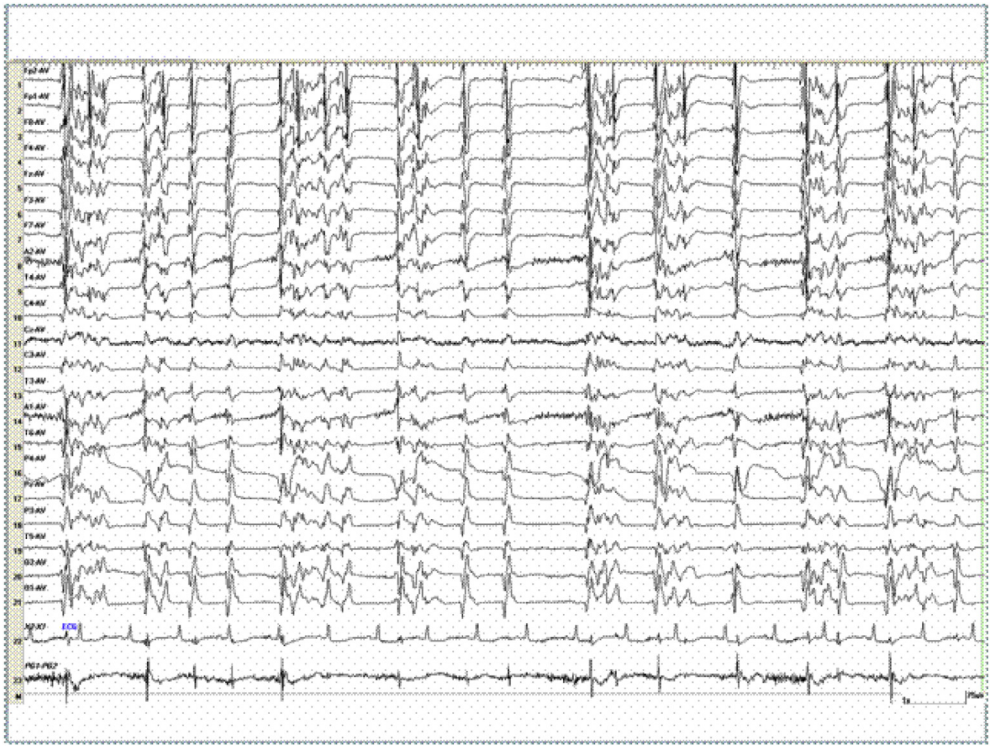

Figure 1 Post-anoxic myoclonus 3 days after cardiac arrest associated with burst-suppression pattern on video EEG. The patient had continuous twitching of his facial muscles and of his upper body and arms throughout this test. He died 2 days after this recording. Note the ongoing diffuse and bilaterally synchronous bursts of spikes/polyspikes and their association with the recorded myoclonus (bottom tracing); EEG discharges are separated by intervals of generalised attenuation of all activity lasting for 0.5–1 seconds with the pattern being non-reactive to external noxious stimulation, including suction.

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