Anoxic Brain Injury and Neural Damage: Three Case Reports

Abstract

Anoxic brain injury (ABI) is common and can occur in a wide variety of disorders. This neural injury is associated with significant and persistent cognitive impairments and poor functional outcomes, related in part to the severity of anoxia. Following ABI neuroimaging has been used diagnostically, but additional research needs to be done to predict rehabilitation outcomes. Patients with ABI have worse functional outcomes following rehabilitation that patients with traumatic brain injury (TBI). Among the different causes of brain anoxia, near drowning has the most severe prognosis. These case studies are based on the clinical observation of 3 children with ABI due to near drowning.

Introduction

Brain cells with inadequate oxygen supply will begin to die after about four minutes.\(^1,2\) Hypoxia is the term used to describe reduced oxygen supply to a tissue despite adequate perfusion of that tissue by blood, and anoxia is an extreme form of hypoxia in which the tissue is completely deprived of oxygen. Cerebral hypoxia and anoxia specifically involve the brain and when a brain injury that is a result of oxygen deprivation either due to hypoxia or anoxic mechanisms occurs it is usually termed hypoxic/anoxic injuries (HAI).

Causes
Anoxia can be caused by any event that severely interferes with the brain’s ability to receive or process oxygen. These events may be internal or external to the body. For example, a blood clot or stroke, shock and heart problems, such as cardiac arrest, interfere with cerebral perfusion. The blood flow may also be normal, however it is not carrying enough oxygen and this can happen with lung disease, lack of oxygen in the air, exposure to certain poisons or other toxins such as carbon monoxide poisoning or any event that stops a person from breathing normally like choking, suffocation or a near drowning. Severe cerebral hypoxia and anoxia is usually caused by traumatic events.

**Diagnosis**

Diagnosis of an anoxic brain injury (ABI) might include a Computed Tomography (CT) or Magnetic Resonance Imaging (MRI) scan of the head, or an Electroencephalogram (EEG) which tests brain waves and can help identify seizures and show how well the brain cells are working. A SPECT scan can also be ordered, which is a type of CT coupled with a nuclear medicine scan that examines areas of the brain for blood flow and metabolism.¹

**Treatment**

Treatment options for an ABI will depend upon the cause of the injury. Some treatments include mechanical ventilation and the use of medication to help get sufficient oxygenated blood to the brain. Sometimes to decrease the brain activity the patient’s body temperature is cooled. The idea behind this is that cooling will decrease the metabolic oxygen demand of the brain; however, the effectiveness of this treatment is unknown.² Other options include entering patients
into a rehabilitation program; the problem with some of these programs is that they are geared more towards patients who have suffered from a traumatic brain injury (TBI).

**Neuroimaging**

In cases of moderate to severe ABI, there will typically be some form of neuroimaging that will occur as part of the diagnostic clinical assessment. “Some authors argue that in the first 3 days MR findings are clearly more predictive of outcome, and this technique should be used in the early days after the event.” In the following 3 case studies, the patients received head MRI’s 3, 5, and 7 days respectively, post-injury. Some factors such as brain maturity, severity and duration of injury, and timing of imaging studies can all influence findings reported in MRI. Patients 1 and 3 both showed evidence of deficits in globi pallidi nuclei and the parietal and occipital cortical grey matter. Patient 2 showed bilateral signal abnormalities in the caudate nucleus, among other deficits. In cases with neuropsychological deficits, MRI showed evidence of structural changes in the basal ganglia, mainly in the lentiform, caudate and globi pallidi nuclei. Unfortunately, MRI is not always sensitive enough to reveal minor neurological damage, but performing an MRI sooner may provide better information. It would also be helpful to use a MR-scanner with a stronger magnetic field, such as a 3 Tesla.

In the early days of neuroimaging the main focus was still on lesion localization, mainly because there were no real quantitative methods for measuring brain pathology. Coarse linear measurements like width and length of a lesion had to be implemented by hand. While it was beneficial to know the size and location of a stroke or old contusion for example, this information was not necessarily directly applicable to the kinds of therapeutic interventions that
might be recommended during rehabilitation. Neuroimaging is an important tool that may help clarify outcomes following an ABI, but is often underutilized in neurorehabilitation.

**Neurorehabilitation**

Brain injuries result in both functional and cognitive impairment, but the physiological mechanism of the different types of injuries and how they heal differ. An ABI results in direct neuronal cell death, whereas with a TBI there is a disruption of axonal integrity. However, regardless of the type of injury, when a patient presents with either an ABI or TBI they are treated with a similar or even identical rehabilitation program. Cullen and Weisz\(^5\) indicate that ABI patients fare much worse in both functional and cognitive areas than TBI patients after completing similar rehabilitation programs. For example, in one study using the Barthel Index researchers found that 60% of patients with TBI achieved complete independence, compared to only 10% of ABI patients.\(^5\) In another study, upon discharge, patients with TBI scored significantly better on both motor and cognitive sub-scales of Functional Independence Measure (FIM) and showed greater improvement throughout the rehabilitation process than ABI patients.

Based on MRI findings, and what is referred to as DTI tactography, **Figure 1** shows an example of disrupted connectivity and its importance for neurorehabilitation. This image shows a comparison of an ABI patient, on the right, compared to an age, and sex matched normal control on the left. “Projections from the anterior aspect of the corpus callosum to the frontal lobes are distinctly abnormal.”\(^4\)\(^{(225)}\) This would imply that the patient has considerably diminished bihemispheric frontal lobe integration, which relates to reduced executive functioning, emotional control and speed of processing.\(^4\)
In these 3 case studies Pierro et al\textsuperscript{6} does not discuss neurorehabilitation but rather shows how multimodal stimulation was used as a form of rehabilitation. This stimulation was initially linked to the parent’s voice, smile and kiss every time the patient moved their head and/or arms until they generated spontaneous intentional movements.\textsuperscript{6} A black and white checkerboard (see Figure 2) measuring 100 x 100 cm, with a high color contrast and squares was set up to improve visual localization, orientation and motor control or reaching and grasping. The targets of functional recovery were visual, auditory, and tactile spatial localization. Eye/head movements, reaching, grasping, postural changes, balance and locomotion were also targets.

Current data suggests ABI patients recover at a slower rate and have worse overall functional outcomes compared to TBI patients.$^5$ Cullen and Weisz\textsuperscript{5} believe this difference in recovery is due in part to a difference in neuronal loss or in the mechanism of injury that occurs in patient with ABI versus patients with TBI. Despite unprecedented improvements in neuroimaging technology, there is very little organized research that has focused on the potential uses of advanced neuroimaging technology in a neurorehabilitation setting.$^4$

**Case Report**

This study is based on a clinical observation of three cases of children with ABI due to near drowning. The study reports recovery of functions during the first year post-injury and documents the rehabilitation process.

Case 1, a 22-month-old male suffered an anoxic event in a near drowning accident in a swimming pool. He was unconscious and without pulse or respiration when pulled from the water. The boy’s father administered cardiopulmonary resuscitation (CPR) and transported him to the hospital. It was presumed he suffered from anoxia for at least 20 minutes and upon arrival
to the hospital his Pediatric Glasgow Coma Scale (PGCS) score was 3. The patient was admitted to the Intensive Care Unit (ICU) five hours after the accident, opened his eyes on day 5, and transferred to the Department of Neurology on day 9. A head MRI was performed on day 7 and “revealed signal abnormalities in globi pallidi and in parietal regions of the cortical grey matter and subcortical white matter.”

Case 2, a 14-month-old female suffered an anoxic event in a shallow water bathtub. She was found unconscious in the water in cardiorespiratory arrest. The patient was transported to the hospital, and it was presumed that the total length of anoxia was 20 minutes and her PGCS score was 3 upon arrival. The patient was transferred to the ICU of the hospital approximately 2 hours post injury. She opened her eyes on day 7, and was transferred to the Department of Neurology on day 12. “On admission, she exhibited decorticate posturing, dystonia with opisthotonos and torsion spasms.” An MRI of the patient’s head performed 5 days post injury revealed, “bilateral signal abnormalities in caudate nucleus and putamen and at the grey matter-white matter junction; lateral ventricles had an ab extrinsecum compression and periencephalic liquoral spaces were practically absent.” An EEG showed generalized spike and wave discharges. On day 30 the patient exhibited torsion dystonia associated with severe spasticity (see Figure 3) and was in a vegetative state.

Case 3, a 15-month-old male was found in the swimming pool, unconscious with impaired respirations. The patient was transported to a local hospital where it was estimated he had suffered an anoxic event lasting about 20 minutes. His PGCS score was 3 when he arrived at the hospital. He was transferred to the ICU a few hours later. A head MRI was performed on day 3 and revealing, “signal abnormalities in globi pallidi and in bilateral parieto-occipital cortical grey matter.” On day 19 the patient was in a vegetative state and also exhibited
torsion dystonia associated with severe spasticity. At 2 months he emerged from the vegetative state. His EEG showed diffuse slowing, sharp waves and spikes in both hemispheres, but predominantly in the left.

Case 1 showed a huge increase on the coma remission scale (CRS) in the first 3 months post injury. Case 2 demonstrated only a small advancement of impaired consciousness in the 8 months post injury. Case 3 exhibited a decline in level of consciousness after 2 months post injury in spite of an initial progression (see Figure 4). Due to the worsening of dystonia, case 3 regressed back to a vegetative state. It is important to emphasize that the PGCS on arrival in the ICU and the presumed length of anoxia were similar for all three cases. The head MRIs performed showed signal abnormalities in different areas of the brain on all 3 children. The follow-up MRI scans showed no abnormalities in case 1, diffuse brain atrophy and bilateral signal abnormalities in caudate and lentiform nuclei in cases 2 and 3 (see Figure 5).

**Functional Outcomes**

Outcomes following ABI include movement and psychiatric disorders, impairments in visual perception, expression and cognition, particularly memory and poor functional outcomes, which are usually severe and most often permanent. This could be due in part to the fact that regions of the brain such as the hippocampus, basal ganglia and thalamus have high metabolic requirements and are particularly sensitive to the effect of anoxia. Analysis of neuropsychometric tests, suggest that the etiology of brain injuries may not be as important as the extent of tissue damage. Focal as well as diffuse damage can be produced by an ABI and the damage may present as structural lesions of generalized brain atrophy. When the damage is more diffuse, the rehabilitation outcome is usually worse. With focal lesions the deficits are
often specific. For example, a “focal hippocampal damage may produce distinct and severe memory impairments.”\(^7\)\(^{321}\) The long-term effects will depend on the purpose of that portion of the brain. For example, damage to the Broca’s area and the Wernicke’s area of the brain, typically causes problems with language and speech. If there was damage to the right side of the brain it may interfere with the ability to express emotions or interpret what the patients sees. Although any of these pathologies can be identified with MRI, unfortunately, neuroimaging finding were not used to help address rehabilitation outcomes.

**Prognosis**

Prognosis of ABI depends on the extent of the injury, which is determined by how long the brain lacked adequate oxygen. The longer a person is unconscious, the higher the risk for death or brain death, and the lower the chances of recovery. Mild and moderate cerebral hypoxia normally has no impact beyond the episode of hypoxia. However, severe hypoxia is another matter. The outcome will depend on the success of damage control measures, the amount of brain tissue deprived of oxygen, and the speed with which oxygen was restored to the brain. If the HAI injury was localized to a specific part of the brain, brain damage will be localized to that region. If the ABI results in a coma, the length of unconsciousness can often be used as an indication of long-term damage, the longer a coma continues the greater the possibility that the patient will remain in a vegetative state, sometimes until death. Even if the patient were to wake up, brain damage is likely to be significant enough to prevent a return to normal functioning.

ABI patients are more likely to be reliant on long-term institutional care than patients with TBI.\(^8\) The Functional Assessment Measure (FAM) is performed with patients suspected of brain injuries, upon admission, at 4-week intervals, and upon discharge, and in terms of
functional status the patients with ABI had poorer initial and discharge FAM scores that the TBI population. This could be attributed to the more global nature of injury in ABI, the existence of co-morbidities or the relatively greater cognitive impairments.8

Additional testing and research needs to be conducted to ascertain why patients with ABIs do not reach the same level of functionality and rehabilitation as patients with TBIs and to develop a more successful and tailored rehabilitation program for survivors of ABI.5

**Conclusion**

In conclusion, ABI can be caused by a variety of factors, and can affect a large number of brain regions; there are few regions or neural structures spared. While MRI or CT has not been utilized with rehabilitation both are extremely beneficial to help determine the severity and location of damage caused by an ABI. The patient in case 1 had a better outcome than cases 2 and 3 even though many of the factors were similar. Perhaps case 1 benefited from the immediate resuscitative attempts made by his father. A larger number of cases and further studies will be needed to validate this observation. Neuroimaging methods hold significant promise to better inform neurorehabilitation clinicians about the amount and degree of structural damage attributable to ABI. However, research is needed to understand cognitive and functional outcomes following ABI and why these patients fared worse than patients with TBI, even after completing similar rehabilitation programs, as well as whether specific rehabilitation techniques will result in better outcomes.
References


