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Cerebral Computed Tomography in Drowning Victims

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Computed tomography in two patients who had drowned revealed bilateral basal ganglia and medial temporal lobe low densities. Postmortem examination in one case showed these areas to be necrotic, probably secondary to the hypoxia and hypotension associated with drowning.

Drowning is associated with the arrest of alveolar oxygen exchange and the development of cerebral hypoxia. Cerebral hypoxia, in turn, leads to circulatory arrest and decreased cerebral blood flow. The resultant neuropathologic changes reflect the effects of both hypoxia and ischemia. We had the opportunity to observe the computed tomographic (CT) abnormalities caused by drowning in two patients. In both, basal ganglia hypodensities were the predominant CT features.

Case Reports

Case 1

A 3-year-old boy was found after an indeterminate time in fresh water (hot tub). Cardiopulmonary assistance was instituted and initial blood gas analysis showed a pH of 6.67, P_{O_2} of 19, and a P_{CO_2} of 129. He was comatose; the pupils were fixed and dilated. Corneal reflexes, doll's eye, and caloric reflexes were absent. The initial intracranial pressure readings were close to zero.

An unenhanced CT scan about 48 hr after the accident showed bilateral low-density changes of the basal ganglia and medial aspect of the temporal lobes (figs. 1A and 1B). The lateral and third ventricles were slightly dilated. Carotid and vertebral angiography was normal. The patient remained comatose and died 6 days after the accident.

Autopsy revealed severe cerebral edema with herniation of the cerebellar tonsils, compression of the medulla, and bilateral transtentorial herniation. The periaqueductal and pontine tegmental areas were also softened. Coronal sections showed discrete encephalomalacia of the basal ganglia (fig. 1C).

Case 2

A 19-month-old girl was found in a swimming pool about 10 min after drowning. Cardiopulmonary assistance was instituted and initial blood gas analysis showed a pH of 6.6, P_{O_2} of 90, and P_{CO_2} of 90 on 100% oxygen. Initially the patient was unresponsive to pain and her pupils were 3 mm and fixed. Rare agonal spontaneous respiratory efforts were noted. Intracranial pressure readings were normal. Initial electroencephalogram tracings showed diffuse slowing without response to stimulation. Vigorous supportive care was instituted to decrease the cerebral edema. An unenhanced CT scan about 20 days after the accident showed small bilateral basal ganglia radiolucencies (fig. 2). Recovery has been limited to brainstem and diencephalic function.

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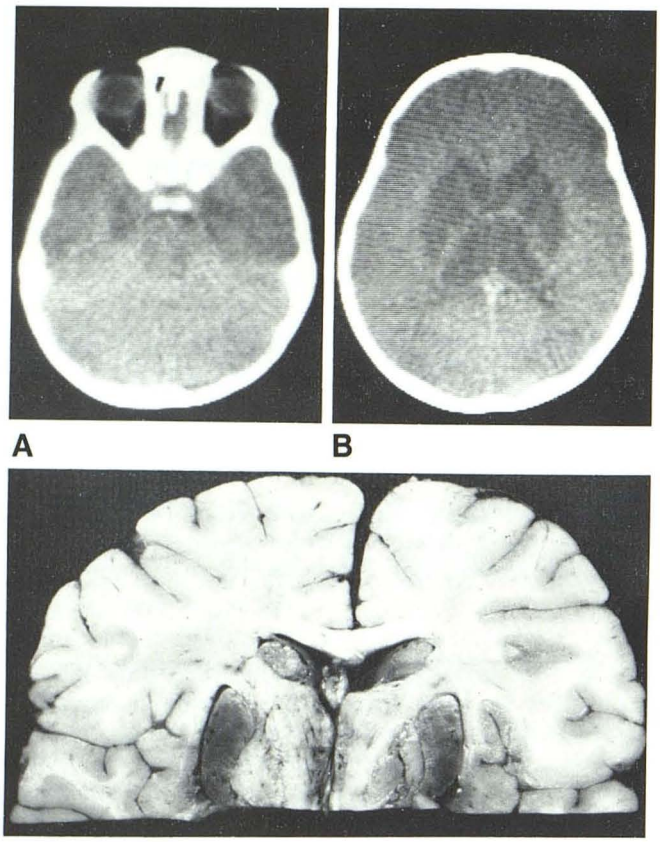

Fig. 1.—Case 1. Axial CT scans show low attenuation in medial temporal lobes and midbrain (**A**) and upper extent of basal ganglia low attenuations (**B**). **C**, Coronal section shows infarction of basal ganglia bilaterally.

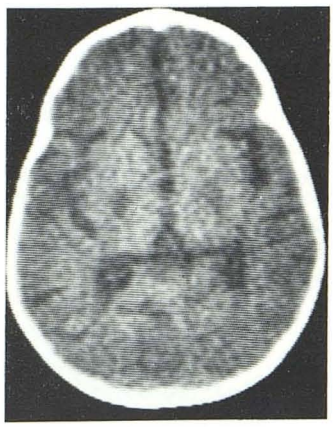

Fig. 2.—Case 2. Axial CT scan shows bilateral low-attenuation areas in posterior parts of globus pallidus.

Discussion

Because of the wide availability of CT, drowning victims very possibly will be scanned to document hypoxic changes and to identify possible organic results of the accident. Bilateral basal ganglia changes occur in various hypoxic conditions, with exposure to toxic substances, and in certain heritable disorders. Hypoxic and ischemic states may result from complications of anesthesia, hypnotic overdose, circulatory arrest, and, presumably, drowning [1–3]. Carbon monoxide, hydrogen sulfide, and cyanide poisoning produce basal ganglia lesions, predominantly in the globus pallidus, by a similar mode of action [2, 4–7].

Basal ganglia lesions of methanol intoxication involve the putamen and may be associated with hemorrhage [8, 9]. The precise mechanism is uncertain. Hepatolenticular degeneration (Wilson disease) is associated with low-density basal ganglia changes most marked in the putamen [10–12]. Similar changes have been reported in subacute necrotizing encephalomyelopathy (Leigh disease), another autosomal-recessive disorder [13].

The CT findings represent some of the noncortical results of cerebral hypoxia. In drowning decreased alveolar oxygen tension and subsequent hypoxemia (as well as obligatory circulatory failure and cerebral ischemia) result in cerebral hypoxia [1]. The mechanisms of selective brain damage is a matter of considerable debate. Myers [14] divided theories concerning the distribution of anoxic brain pathology into circulatory and inherent metabolic differences.

A border-zone concept of cerebral vascular anatomy suggests that during hypotensive episodes the border zones of major arterial territories are areas of the brain deprived of blood earliest and with the least possibility for significant collateral circulation [14]. Although the basal ganglia and medial hippocampus are not included as border-zone structures, the end-arteriole structure of basal ganglia vessels [5] and the rakelike character of vessels supplying the hippocampus [14] may be morphologically susceptible to critical reduction in blood flow during hypotension.

Considering intrinsic vascular differences of small vessels, Ames et al. [15] noted that localized areas of brain failed to reperfuse (no reflow) after hypotensive insults of as little as 7½ min. The no-reflow phenomenon suggests that the selective vulnerability of many structures in the brain, including the putamen and globus pallidus, may be related to hypoxic endothelial cell swelling and obstruction to flow.

Differences in tissue metabolism may also play a role in selective hypoxic brain damage. Tissues with higher metabolic rates may show selective damage under hypoxic conditions; indeed, Myers [14] showed accumulation of lactic acid, a metabolic by-product, in brain tissue with tissue injury. It is possible that the no-reflow phenomenon may be related to lactic acid accumulation, thus correlating vascular morphology and metabolic activity.

While the precise pathogenesis of hypoxic lesions in the basal ganglia, thalami, and hippocampi has not been established, it is relevant to emphasize that bilateral lucencies in the globus pallidus on CT suggest serious hypoxic cerebral insult. The extent of lucency may have prognostic value.

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