Cerebral oxygenation and the recoverable brain

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Oxygenation is the most critical function of blood flow and a sudden reduction in oxygen availability is an inevitable consequence of severe ischemic. The resulting cascade of events may result in the failure of membrane integrity of some cells and necrosis, but in the surrounding zone of tissue, less affected by hypoxia, cells survive to form the ischemic penumbra. The timing of these events is uncertain, but sufficient oxygen is available to these cells to maintain membrane ion pump mechanisms, but not enough for them to generate action potentials and therefore function as neurons. The existence of such areas has been suspected for some time based upon the nature of clinical recovery, but has now been demonstrated by SPECT imaging with a high plasma oxygen concentration under hyperbaric conditions as a tracer. A course of hyperbaric oxygen therapy frequently results in a permanent improvement in both flow and metabolism. These changes apparently represent a reversal of the changes that render neurones dormant and the activity of cells, previously undetectable by standard electrophysiological methods, can now be demonstrated. Three patients are presented in whom recoverable brain tissue has been identified using SPECT imaging and increased cerebral oxygenation under hyperbaric conditions. Improved perfusion from reoxygenation has correlated with clinical evidence of benefit especially with continued therapy. [Neurol Res 1998; 20 (Suppl 1): 533-536]

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INTRODUCTION

Stroke is the third leading cause of death in the United States and a major source of disability. It is not only devastating to the patient and the family, but also creates a large financial drain on resources. In stroke it is often stated that most recovery takes place in the first three months. However, sequential SPECT scanning has indicated that some recovery may take place spontaneously up to six months. Although stroke tissue in the center of the lesion becomes necrotic, in the surrounding zone of tissue, less affected by hypoxia, cells survive to form the ischemic penumbra'. The timing of these events is uncertain[2] but sufficient oxygen is available to these cells to maintain membrane ion pump mechanisms, but not enough for them to generate action potentials and therefore function as neurons. The existence of such areas has been suspected for some time based upon the nature of clinical recovery, but has now been demonstrated by SPECT imaging using a high plasma oxygen concentration under hyperbaric conditions[3-5]. The use of hyperbaric oxygen therapy has been described in a large series of stroke patients . Another approach to this problem has been the transposition of omental tissue which can revascularise the area to elevate oxygen delivery.

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Anoxic ischemic encephalopathy (AIE) with severe hypoxia can affect the brain at any age and may result in necrosis and death, although lesser degrees of hypoxia may not be fatal. Patients may subsequently exist in a locked-in syndrome[8] or in persistent vegetative coma. Because of the poor prognosis and the high financial costs involved it has actually been suggested that hydration and parenteral nutrition should be withheld in patients in persistent vegetative coma[9]. This condition has many causes, including near drowning, near hanging, CO poisoning, cardiac arrest, electrocution, drug overdose, surgical accidents, anesthetic mishaps and prolonged hypoglycemia. The earlier the onset of hyperbaric oxygen therapy the better the Prognosis[10] and this has been shown by Mathieu et al.' in a series of cases of near hanging. These patients are profoundly oxygen deficient with depletion of ATP and have raised intracranial pressure. Lactic acidosis is also present from anerobic glycolysis and there are many other biochemical disturbances, including the generation of oxygen free radical species, which are damaging to neurons. This phase gains momentum at about 30 min and evolves over several hours, depending upon the severity of the hypoxia. It has often been stated that patients with a Glasgow Coma Scale score of less than six have little chance of recovery after three months in coma and spontaneous recovery after prolonged coma merits extensive media coverage. However many such cases are on record with intervals of several years. Clearly, it is

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