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Hyperbaric Oxygen Therapy Leading to Recovery of a 6-Week Comatose Patient Afflicted by Anoxic Encephalopathy and Posttraumatic Edema

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Eltorai I, Montroy R. Hyperbaric oxygen therapy leading to recovery of a 6-week comatose patient afflicted by anoxic encephalopathy and posttraumatic edema. J Hyperbaric Med 1991; 6:(3):189–198.—Head injuries account for about 70,000 fatalities annually in the United States, according to 1982 statistics. The factors that contribute to mortality and coma are hypoxia, shock, edema, and increased intracranial pressure, in addition to direct cerebrovascular injuries. Hyperbaric oxygen (HBO) treatment has been used both in animal models and in humans without conclusive results. However, HBO improves cerebral anoxia and reduces intracranial pressure. A patient who was in a traumatic coma for about 6 wk and recovered with the use of HBO is reported. This result should stimulate further laboratory and clinical studies searching for lower morbidity and mortality.

posttraumatic cerebral edema, anoxic encephalopathy, coma, hyperbaric oxygen therapy

Introduction

In the United States, statistics of 1982 showed 1,974,797 deaths; of these 144,682 (7.3%) were injury related. Of these, 70,000 were due to fatal brain injury, i.e., about 48% of the total injury mortality or 22–30 per 100,000 (1). These figures have improved in the last few years, especially where excellent prehospital care and aggressive hospitalization measures are available, particularly the use of modern technology in diagnosis and management. In the prehospital phase, hypoxia and shock are two very important complications that can cause increases in intracranial pressure (ICP) and contribute to a poor outcome. Before presenting the case it is necessary to review information about brain injury.

Some Physiologic Concepts About the Brain

1. Although the brain accounts for 3% of body weight, it consumes 20–25% of the body's oxygen and glucose.

2. Glucose is the only substrate that the brain can use to produce energy, for all practical purposes.

3. Only complete oxidation of glucose in the tricarboxylic acid cycle provides the brain with adequate energy. Glucolysis by the anaerobic glucose metabolism does not meet even the basal energy requirements of the brain.

4. The brain has little capacity to store oxygen and glucose and has a minimal reserve of high-energy phosphate compounds; therefore, it depends entirely on the cardiovascular and respiratory systems for energy resources.

5. Fifteen percent of cardiac output must be delivered to the brain. When this is reduced or the substrates (O_2 or glucose) are reduced by means of hypoxemia, anemia, and hypoglycemia, the brain metabolism will suffer.

6. The brain has a constant energy output that is equivalent to almost 25 W; most of the work is electrochemical involving active transport of ions across the cell membranes to maintain conductivity and membrane excitability. Inadequate energy supply leads to electrical failure as well as cellular disintegration. Brain energy depends on the ultimate difference between energy supply and demand. In case of negative balance, structural changes occur in variable degrees and these may be reversible if minimal, or permanent if severe and prolonged. In the unconscious patient the demand is reduced. On the other hand, it is increased by epileptic seizures, excessive motor or sensory excitability, e.g., by severe pain.

Pathophysiology

We will not discuss head injuries in general, which can be consulted in two outstanding references, Cooper (2) and Tyson (3), but the factors that contribute to higher mortality and to coma.

Hypoxia

Of the 581 comatose patients entered in the pilot study of the National Coma Data Bank, 103 were considered to have been hypoxic before entering the hospital; $Pa_{O_2} < 60$ torr. A group of 47 other patients were ventilated before measurement of arterial blood gas because they were apnoeic or had slow, irregular breathing. This is in accord with the observed apnea in animals immediately after head trauma was induced. In humans, apnea and/or inefficient ventilation were noticed, possibly due to decreased respiratory drive or brain edema and increasing ICP. However, inefficient ventilation does not have to be of cerebral origin; other causes such as obstruction of the airways by blood, vomitus, foreign bodies or the tongue, and chest injuries and pulmonary edema may be contributing to the hypoxia. Resuscitation after cardiac and respiratory arrest is commonly practiced, sometimes with continued life support. This resuscitation may end in anoxic brain damage, and the survivors may remain in a coma or regain consciousness. Grosswasser et al. (4) found that the outcome in cardiopulmonary resuscitation (CPR) cases was

better than for comatose patients from craniocerebral trauma, especially in younger individuals, and the coma lasted less than 24 h. Hypoxia was found in 91% of fatal nonmissile head injuries, and is either localized to an infarcted area or diffuse. Pa_{O_2} less than 50 torr for more than 15 min leads to brain hypoxia; similarly hypotension to less than 80 mmHg may result in the same pathology. Arterial spasm and increased ICP aggravate the damage. Hypoxia, therefore, aggravates the traumatic coma in the absence of an intracranial mass (5); these authors noticed hypoxia frequently in patients who remain vegetative or severely disabled.

Shock

Shock leads to defective perfusion with lack of substrate supply to the brain. It may be complicated by vasospasm or infarction, in which case the damage may be in an arterial boundary zone. In the National Coma Data Bank, 31% of the 581 patients were in shock. The causes were multiple injuries; blood loss; cardiac arrhythmia, especially bradycardia; and brainstem failure. Hypotension was found to correlate with the poor outcome in these cases, and in some studies could be a predictor of the later increase in ICP.

Brain Edema (Swelling)

Brain edema is not an uncommon problem after head injury as seen in surgery, autopsy, and by computed tomography (CT) scan, magnetic resonance imaging (MRI), and positron emission tomography. It is defined as a specialized form of swelling in which the brain is expanded because of an increase in tissue fluid. Five different varieties have been defined by Clasen and Penn, viz: vasogenic, cytotoxic, hydrocephalic, osmotic, and ischemic, based on the etiologic factor and the biochemistry of the tissue fluid. Edema can occur per se, especially in children, and can be adjacent to a brain contusion or develop after decompression of a subdural hematoma in one or both hemispheres. It may develop as a result of hypoxia due to cardiorespiratory arrest or status epilepticus postinjury.

Increased Intracranial Pressure

When ICP is high enough, cerebral perfusion may be so inadequate as to deprive the brain of its substrate requirements, which may lead to ischemia. These changes are variable and depend on other factors, e.g., mean arterial pressure, cerebral vascular resistance, the status of pressure autoregulation, and the cerebral metabolic rate. These factors may be associated with traumatic space occupying hematoma, brain contusion, laceration, or other kinds of hemorrhage.

Hyperbaric Oxygen in Traumatic Coma After Head Injury

Traumatic cerebral pathology is extremely complicated. The pathology of the neurons and the glial cells is the determining factor for the seriousness

of the clinical consequences. Reversibility depends on the degree and duration of brain anoxia in addition to the gravity of the main injury. Anoxia is of different types: anemic, hypoxic, defective perfusion, stagnation, or histotoxic, and in brain injury one or more types get involved in cerebral damage. This can be aggravated by hyperthermia, seizures, and excessive motor and/or sensory stimulation.

One of the earliest reports is that of Fasano et al. (6), who stated: "Therefore, our first impressions are that the therapy with high-pressure oxygen administered according to the physiopathological characteristics of the anoxic state, associated with different pharmacological aids according to the relative prevalence of one of the forms of anoxia, can give promising results both from the doctrinal and the practical point of view, in the field of neurotraumatology."

Bolot et al. (7) conducted a study on 40 comatose patients including 13 head injuries. Their results were not conclusive. They reviewed the cases retrospectively.

Holbach et al. (8–11), compared a group of midbrain traumatic syndrome treated with hyperbaric oxygen (HBO) and a group without it. They concluded that the addition of HBO reversed the traumatic midbrain syndrome, especially in younger patients (1–30 yr) and in some of them it leads to a cure.

Dunn and Conolly (12) induced head injury in dogs and used different experimental conditions, and they concluded that inhaled oxygen reduced the mortality, but there was no evidence that high pressure oxygen treatment was more beneficial. Similarly, Nagao et al. (13) found increased intracranial pressures benefit only temporarily and not significantly. Sukoff et al. (14–15) reported the protective effect of HBO on experimentally induced cerebral edema, and Sukoff and Ragatz (16) found that HBO was effective in reducing ICP in conjunction with other measures. Kanshepolsky (17) induced cerebral edema in cats and demonstrated 70% survival if HBO is started 2 h after onset, compared with 38% of the untreated group.

Coe and Angyan (18) reported a helpful effect from HBO on rats' maze performance after experimental concussion. Harper et al. (19) found reduction of cerebral edema by the use of HBO. This is evidenced by a decrease in tissue acidosis and an increase in the available energy due to restoration of normal glucose metabolism (11, 20).

Miller et al. (21) found that inhalation of 100% O_2 reduced the intracranial pressure induced by an extradural balloon by 23% at normobaric O_2, 37% at 2 atm abs. This reduction was not accomplished by any significant change in the arterial Pco_2 or systemic blood pressure (BP). Miller (22) found that HBO at 3 atm abs failed to reduce intracranial pressure. According to Holbach et al., 1.5 atm abs is the optimum pressure.

Kapp et al. (23), in an experimental model in cats, found after circulatory arrest that HBO modified favorably the postischemic functional behavior and metabolic derangement.

Brown et al. (24), in a human study, concluded that ICP decreases during the pressurization phase but rebounds after the treatment or even during the treatment, without lasting effect.

Hyperbaric Oxygen and Brain Anoxia

Moor et al. (25), in an experimental circulatory arrest in dogs, found that O_2 available to the brain at 3 atm abs will only be 27% more than that normally available with air breathing at 1 atm abs and 20% more than that available with O_2 at 1 atm abs. The addition of CO_2 prearrest had little effect on the results.

Heyman et al. (26, 27) demonstrated the beneficial effect of hyperoxia with animal-induced circulatory arrest; more so with the use of hypothermia.

Lareng et al. (28) reported recovery of 2 cases of deep traumatic coma after 4 and 9 days using pressure at 2 atm abs in a multiplace chamber. In the discussion section of that paper, Deleuze mentioned complete recovery of 4 patients with deep neurosurgical coma.

Rockswold and Ford (29) presented preliminary results of a randomized study of several brain-injured patients. They concluded:

> There is considerable evidence that HBO reduces intracranial pressure by causing cerebral vasoconstriction and decreased cerebral blood flow while simultaneously supplying optimal amounts of oxygen for efficient cerebral aerobic glucose metabolism. The preliminary results of a prospective randomized clinical trial in a carefully defined group of patients with severe brain injuries treated with HBO are suggestive of a beneficial response.

These authors treated patients who were persistently unconscious with Glasgow Coma Scale (GCS) scores less than 10 within 4 h at 1.5 atm abs 100% O_2 in a Sechrist chamber. Patients with increased ICP were treated every 4 h or every 8 h and the treatment continued until either the patient recovered or was considered brain dead. Patients with a GCS of 3 expired whether treated with HBO or not. Patients with a GCS of 7–9 mostly survived (10 out of 11). However, for patients with a GCS of 6, survival rates increased with HBO. The 8 surviving patients achieved good recovery or mild-to-moderate disability.

In animal experiments, Ruiz et al. (30) induced cardiac arrest and resuscitated the animals (total time of arrest 12 min). They found no difference in the HBO group with hemodilution using Hespan, Hespan alone, and the control group with respect to survival time, cardiac function, or neurologic scoring.

A recent study by Grosswasser et al. (4) compared the rehabilitation outcome of 32 comatose patients with anoxic brain damage with 133 patients who were comatose due to head injury from a blunt instrument. Patients in the first group were comatose for periods ranging from 24 h to 18 wk. The second group was comparable. According to the literature (4), survival rate of postanoxic coma varies from 8.7 to 40%; functional recovery is rather poor,

14%. In this study, 4 out of 28 patients could go to work. The craniotraumatic coma patients took much longer to recover after months or even years, with a significant degree of functional recovery. The age and duration of the coma were significantly associated with the outcome.

Case Report

The patient was a 58-yr-old white male veteran, an incomplete quadriplegic at C5–C6 level secondary to a diving accident in 1952, and an efficient, intelligent, and active person.

On 3 November 1987, while getting into his van on a lift, he fell from the height of the van lift sustaining a right eyebrow incised wound with concussion. He was unable to remember the fall or how he got into the emergency room. While in the CT scan he became confused and nonresponsive and developed respiratory arrest. He was resuscitated and intubated with slight difficulty and he bit the tube for 2–3 min. Initial CT scan showed no bleeding and no shift. Neurologic exam showed a comatose patient on a ventilator without response to any stimuli and was classified at GCS score 3. Another CT scan showed (R) frontoparietal contusion without space-occupying mass. Patient received mannitol and decadron with i.v. fluids. On 5 November 1987, a neurologic examination revealed patient not responding to verbal stimuli, withdrawal to noxious stimuli above his cord level, and not tracking with intent visually. Roving conjugate eye movement was noted. Left pupil was miotic with minimal reaction to light. Fundi: no papilledema. EEG diffuse, slowing without focal anomaly. On 6 November 1987 he developed focal seizures described as tonic-clonic activity of the left upper limb with the eyes turned to the right and the head more or less also turned to the right. Patient was put on Dilantin therapy. Lumbar puncture showed clear cerebrospinal fluid. Patient was on the respirator and his respiratory status was stable. A I A line and a Swan-Ganz catheter was instituted. His urine output was low and BP also low. He was supported by i.v. fluids and dopamine. His urine output improved but he got hematemesis and his hematocrit dropped and necessitated blood transfusion. Peripheral hyperalimentation was started. Patient responded to noxious stimuli, turned his head and eyes but was not communicative. On 10 November 1987 he developed upper respiratory tract infection and was put on antibiotic therapy. The patient continued to be unresponsive and was considered postconcussion status plus anoxic encephalopathy. A tracheostomy was done on 18 November 1987. His EEG continued to show diffuse slowing without focal changes. On 25 November 1987 the patient was put on DNR (do not resuscitate). On 1 December 1987 he developed pneumonia, which was treated with antibiotics. The patient's neurologic status did not change and he was kept on supportive treatment until 21 December 1987, when one of us (I.E.) saw the patient. HBO was tried at 2 atm abs for 90 min using a Sechrist monoplace chamber and a Sechrist

ventilator. On the first session the patient was restless, opened his eyes, turned his head from side to side, and responded to sounds. On the second day he responded sometimes to his name.

Day 3: in addition, moving the upper limbs; Day 4 responds to names; Day 5 same; Day 6 same; Day 7 alert, responds to verbal stimuli; Day 8 same as above; Day 9 responds to name, verbal command; Day 10 as above, but looking around; Day 11 as above.

The patient continued to respond to commands at first by shaking his head or nodding until session 24 when he started talking, though only briefly, and gradually ate his meals without problems. Then he started to complain of shoulder pains and was wondering what had happened to him. He was weaned off the LP-6 respirator, and gradually his tracheostomy was plugged, which he tolerated well. He was gradually mobilized to a wheelchair and scheduled for rehabilitation.

Neurophysiologic Evaluation After Recovery Summarized the Findings

1. Patient is frustrated with his condition and angry about the quality of care he is receiving.
2. Severely depressed, although he does not acknowledge it.
3. Patient is tense and anxious about the uncertainty of his future.
4. Thought disorder; feeling alienated and distrustful of others.
5. Marked memory deficiency. The Wechsler Memory Score showed memory deficit 2 standard deviations below normal.

Two months later Minnesota Multiphasic Personality Index showed significant signs of cognitive deficit in the form of associate learning and arithmetic, reasoning, and performance ability. Four months later the patient still had difficulty with attention, could be distracted with lack of concentration, had difficulty in categorizing and association, and found it difficult to make decisions and to organize his thoughts. The patient has made excellent gains during rehabilitation, with substantial improvements in his memory but with mild-to-moderate difficulty of focusing and deficit in categorizing/association of information and in thought processing organization. MRI showed dilatation of the ventricles with brain atrophy (Fig. 1). Patient continues his rehabilitation and participates in group leisure education sessions, displaying good attention.

Discussion and Conclusion

The usefulness of HBO in the management of cerebral edema due to head injury and in hypoxia due to various reasons, especially after CPR for cardiorespiratory arrest, is still controversial. This is due to many variables under these conditions. Experimental animal models may be supportive but not conclusive. A human model of cerebral anoxia due to hanging was

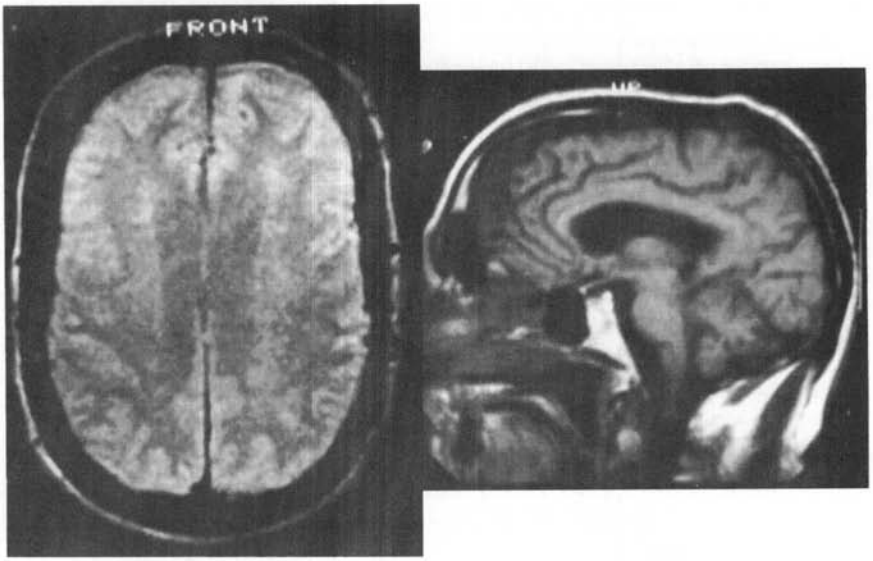

FIG. 1—MRIs showing brain atrophy.

reported by Mathieu et al. (31). Of their 170 cases of attempted suicide by hanging, 151 cases were in a coma at various levels and were treated with HBO at 2.5 atm abs: 78% recovered without neurologic residue, 5% had neurologic deficit, and 17% died. As mentioned above, HBO reduces ICP, it leads to vasoconstriction thus reducing edema, it saturates the anoxic tissues with O_2, and it favors O_2 diffusion to the neural tissue helping restore the aerobic glucose metabolism, thus reducing lactic acid accumulation and helping the neural tissue regain its functions. These investigators found that HBO enhanced the activity of the pentose shunt in the neuroglial cells, which favors neuronal recovery.

The explanation of recovery in our case of deep coma for 6 wk is mysterious. Autolysed cells of course will not recover. However, cells that are hypoxic but not dead, which can be called "parabiotic," could recover under an abundant supply of oxygen. In this case the patient was not deprived of O_2 because he was on the ventilator, but the O_2 supply may not have been enough to satisfy the need of the O_2-"hungry" cells.

Freeman (32) believes that attempts to help the brain injured should be continued. The brain has definitely some degree of plasticity, i.e., the brain may be able to reorganize or rewire by changing the patient's environment. Freeman quotes Cotman and McGaugh: "Studies on reactive synaptogenesis clearly demonstrate that the adult brain has an innate capacity to form new

synapses in a highly selective manner." They also observed: "It is clear that the adult brain has the capacity to dynamically reorganize its circuitry, and after brain damage this plasticity must be taken into account." Freeman adds, "Patients have demonstrated repeatedly that changing environment can change their ability to function. Given the correct environment, changes occur in patients even years after the brain insult." Seward et al., in an introduction to (32), said "Indeed, there is now good reason to believe that reorganization of circuitry following injury is the rule rather than the exception," and in their conclusion they state, "These observations reveal that the postsynaptic cell is a very active participant in the reinnervation process, subsequently remodeling its receptive surface during reinnervation."

Based on the citations from the literature and from this case presentation, we think it would be neither harmful nor wasteful to use HBO in management of hypoxia and/or traumatic edema of the brain because it may be life saving. However, this evidence is not sufficient to unequivocally advocate this method; further experimental and clinical work is required.

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