The Treatment of Cerebral Ischemia with Hyperbaric Oxygen (OHP)

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Abstract:
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The treatment of a patient for three and one-half months, following occlusion of the right middle cerebral artery with the associated neurological sequelae, with hyperbaric oxygen combined with methyldopa and hydrochlor-thazide is presented. Treatment scheduled was two and one-half atmospheres absolute. The treatment was interrupted after 15 treatments to rule out spontaneous remission for a period of 30 days, and no further improvement occurred until treatments were reinstituted. The dramatic return to a near normal state during treatment appears to indicate that he did benefit from therapy.

ADDITIONAL KEY WORDS cerebrovascular disease cerebral artery cranial nerve cerebral blood flow methyldopa hydrochlorothiazide deep tendon reflexes sensory EEG

Introduction
Cerebrovascular accidents from thrombotic or embolic phenomena create inadequate perfusion to a portion of the brain. The resultant decrease in perfusion causes a neuronal deficit, which appears to be due primarily to the hypoxia. Investigational works in applying hyperbaric oxygen (OHP) to this entity show it to be encouraging as a modality of adjunctive care.1-4 The ability to furnish oxygen to the ischemic part is a function of the ability to perfuse the injured area. Ischemic areas in other portions of the body have been noted to heal with repetitive exposures to hyperbaric oxygen.5-7 Therefore, there must be collaterals present or the development of a neovascular system. One controlled investigation demonstrated that a neovascular system develops in approximately three to four weeks following the onset of therapy.6 The following patient was the first patient with a cerebrovascular accident treated at the hyperbaric activity, Naval Hospital, Long Beach, California.

Case Report
A 40-year-old, right-handed Caucasian male who was an active-duty naval officer had the onset of nausea, vomiting, and malaise on September 20, 1969. The next morning he had a left hemiparesis, left facial paresis and bifrontal headache. He was hospitalized, at which time cerebral angiograms revealed an arterial block of the right middle cerebral artery (figs. 1A and B).

Improvement occurred in the patient's neurological status during the time from September 20, 1969, to October 24, 1969, when he was transferred to this facility for rehabilitation and medical discharge. He regained partial use of his left lower extremity, having a markedly spastic gait which required a leg brace and cane for him to ambulate. However, the upper extremity and facial paresis did not improve. Family history revealed that the mother died as a result of a cerebrovascular accident. Past history is relevant in that he has had essential hypertension for which he has been treated. He was maintained on methyldopa 250 mg T.I.D. and hydrochlorothiazide 50 mg/QD.
Physical Examination

At the time of admission, this Caucasian male, appearing his stated age, was normothermic with a pulse rate of 76, blood pressure of 150/100, weight 180 lb, height 5 ft 10 in. On physical examination, positive findings were limited to the neurological examination:

Cranial nerve check: There was a left facial paresis with the tongue protruding to the left.

Deep tendon reflexes:

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Positive left Babinski, negative Hoffman

Motor status: There was a completely plegic left arm, marked weakness of left hip flexion and left quadriceps and atrophy of the left hand intrinsics. He was unable to walk on his left toes or heel and there was a spastic left gait.

Sensory: There was a marked decrease of pinprick, thermal sense, deep pain, position and vibratory senses on the left.

X-rays: Cerebral arteriograms showed a thrombosis of the middle cerebral artery on the right 1 cm from the bifurcation.

EEG: There were 2 to 5 cps waves of moderately high amplitude from the right parasyylvian regions; otherwise the rhythms were from 9 to 10 cps. The EEG was interpreted as being consistent with moderately severe right parasyylvian disturbance of cortical function.

EKG: Tracings were interpreted as being within normal limits in October, 1969, and on January 14, 1970.

Hospital Course

The patient was started on physical therapy consisting of range of motion exercises and neuromuscular stimulation which he continued to receive from the time of arrival at this institution until discharge to full duties. He was maintained on methyldopa 250 mg T.I.D. and hydrochlorothiazide 50 mg per day. No improvement was noted in the left upper
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extremity or in the paresis of the left side of the face from October 24 to November 17, 1969, when hyperbaric oxygen therapy was started. Treatments were for two hours daily at 2.1 atmospheres absolute (ATA). No improvement was noted after five days of therapy. On the seventh day of therapy, he was noted to have some flexion and extension of the wrist and fingers; however, there was no abduction or adduction of the fingers. His course became one of gradual improvement until the treatments were stopped on December 15, 1969, in order to rule out a spontaneous remission. The patient at this time had the following neurological examination:

Cranial nerve check: There was resolution of the left facial paresis in that there was only mild drooping of the left corner of the mouth with slight deviation of the tongue to the left on protrusion. Palate, tongue and jaw movements were intact.

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Motor status: There was a normal gait. The patient was able to walk on his heels and toes (he had slight difficulty on the left toes). The strength in the right arm and leg was excellent. The strength of the left leg was excellent with the exception of a mild weakness of the left hip flexion. The left arm showed atrophy around the shoulder girdle and limitation of abduction of the left shoulder at 25° with marked weakness of all movements at the shoulder. There was moderate weakness of the left elbow on extension and marked weakness of the left elbow on flexion. There was a marked weakness of the left wrist flexion and extension as well as marked weakness of flexion, extension, abduction, and adduction of the fingers. He was able to oppose the left thumb to the fourth digit but unable to oppose to the fifth finger. There was marked grip weakness and he was unable to button his coat, hold eating utensils or do useful tasks with the left upper extremity.

Sensory: All modalities were found to be intact and bilaterally symmetrical.

EEG: There were 5 to 8 cps waves of moderate amplitude over the right parasyylvian regions with no spikes. This was interpreted as improving when compared with the prior EEG.

Hyperbaric oxygen therapy was started again after four weeks on January 15, 1970. Neurological examination and EEG remained the same as that of December 15, 1969, having no loss or gain. The treatments were continued for three more weeks and were stopped on February 4, 1970. He had the following neurological examination on February 10, 1970:

Cranial nerves: There was a drooping of the left corner of the mouth not readily noticeable, and slight weakness of the left upper eyelid. Palate, tongue and jaw movements were intact with no protrusion of the tongue to the left.

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Motor status: There was a mild weakness of strength at the left elbow, wrist and hand. He was able to oppose the left thumb to the fifth finger. The major weakness of the left side was at the shoulder. He was now able to abduct the arm to a 120° angle and to extend the arm to 90°. He was able to walk on his heels and toes. The patient has a normal gait; however, he states that there is some slapping of the left foot at the end of the day. He is able to hold eating utensils, button his clothes and do minor tasks with his left hand.

EEG: There were 7 to 9 cps waves of moderate amplitude over the right parasyylvian region; otherwise the rhythms over the rest of the brain were 9 to 10 cps with no spikes. This was interpreted as showing continued improvement.

He was continued on physical therapy to regain strength in the affected parts and was returned to full duty. EMG, at this time, showed a returned neural activity to all modalities. He has now been at full duties for nine months and is one year post-treatment without deterioration.

Discussion
The delivery of hyperoxygenated blood to ischemic areas to aid in their recovery is of
prime importance in the concept of hyperbaric therapy. The physiological reaction of blood vessels, however, to hyperoxygenated blood is an intense vasoconstriction. This vasoconstriction produces a decrease in cerebral blood flow.\textsuperscript{1,2} Therefore, it might be projected that the benefit from hyperoxygenated blood could be equivocal to the ischemic area. It would then seem beneficial to combine the hyperbaric therapy with a vasodilator. Recent investigations have revealed that a carbonic anhydrase inhibitor affords protection against the vasoconstriction due to hyperbaric oxygen.\textsuperscript{6-10} Reasoning at this facility, independently, concluded that by blocking the precursors to epinephrine and norepinephrine (decarboxylase inhibitor), the vasoconstriction occurring with hyperbaric oxygen could be abolished or diminished. Methyldopa, potentiated with hydrochlorthiazide, was felt to be a good choice of drugs with a low incidence of side effects. The patient had been taking this combination for some time for his essential hypertension. The response of this patient three and one-half months following the cerebrovascular accident was dramatic, but in order to rule out a spontaneous remission, OHP was stopped after 15 treatments for a period of one month. There was no loss or gain during this rest period. The marked gain noted in the second series of treatments is felt to indicate that the patient indeed did benefit from the OHP therapy. The exact mechanism in the repair of the perfusion defect in this recovery is not known, as we were reluctant to perform further arteriography in a patient who was, for all practical purposes, normal. The role of methyldopa and hydrochlorthiazide in the recovery of this patient is conjectural and the potential benefits using such combined therapy will remain to be determined.

References

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Stroke. 1971;2:247-250
doi: 10.1161/01.STR.2.3.247

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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