

THE EFFECTS OF SUPPLEMENTAL OXYGEN RESPIRATION
ON HEMIPLEGIC APHASIC ADULTS

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Oxygen, food and water are the basic essentials for the maintenance of life. Of these three, deprivation of oxygen, however, leads most rapidly to death.

Most tissue cells have little reserve oxygen and must be continually supplied via the circulatory system.

Deprivation of oxygen regardless of site or etiology is referred to as hypoxia.

Hypoxia is known to have effects on the central nervous system both functionally and morphologically. An acute reduction in the arterial oxygen saturation to 85% ($PO_2=50$ mmHg) decreases mental effectiveness, visual acuity, emotional stability and finer muscular coordination. Greater reduction of oxygen saturation levels creates more and more serious effects until finally circulatory failure occurs.

Acute arrest of cerebral circulation causing cerebral hypoxia produces sudden and dramatic manifestations. Loss of consciousness and EEG changes occur within seconds and within three or four minutes histological changes occur in cortical gray matter and some thalamic cells; the cerebellum is more resistant to hypoxic damage and the brain stem and cord are most resistant.

Complete return of function cannot be expected if circulatory arrest exceeds three - five minutes, and death may be expected if cerebral circulation remains static for longer periods.

Oxygen transfer in the lung to and from the blood may be evaluated from studies of arterial blood. The arterial blood is analyzed for the percent saturation of hemoglobin with oxygen and the partial pressure of oxygen in the blood.

Both normal arterial blood tension, as well as, oxygen content is important to normal body function. The oxygen extracted from the blood can be supplied only from stores of

oxygen associated with hemoglobin. On the other hand, an adequate oxygen tension is necessary for the loading of oxygen onto hemoglobin in the lungs and for diffusion of oxygen from the tissue capillaries to the cells.

In intact man normal oxygen saturation levels are between 96 and 98% and minimal normal oxygen tension is 95 mmHg.

CVA is one consequence of acute arrest of cerebral circulation. The hemiplegia secondary to CVA often results in defective respiratory function which may further reduce cerebral oxygenation.

Haas, et al among others have investigated the respiratory function in hemiplegic patients and found in general that lung volumes, mechanical performance of the thorax and the pulmonary diffusing capacity were abnormal in all subjects. Oxygen tensions and saturations were diminished at rest and decreased still further during exercise.

Clinically, if blood oxygen saturation is reduced the individual has less than a normal amount of oxygen being carried in his blood. If oxygen tension is low, the oxygen in the blood is less easily given up to tissue.

With both of these conditions present, it may be inferred that the individual with a residual right hemiplegia secondary to CVA may be suffering from a form of hypoxia.

By elevating blood oxygen saturation, it is possible to make more oxygen available to the cerebral tissue. By increasing oxygen tension, it becomes possible for a greater release of that oxygen to the tissues involved.

Supplemental oxygen can be used to correct for hypoxia which is secondary to ventilation-perfusion abnormalities.

Improved cerebral function may be expected as more oxygen becomes available to the brain and is diffused throughout the cerebral tissue.

This is not to suggest that supplemental oxygen will offer a miraculous eradication of the symptoms secondary to CVA but rather that patients will be able to function to their maximum remaining capacity rather than under the further hindrance of mild hypoxia.

Oxygen may be administered at various levels of atmospheric pressure and by different methods and both the theoretical concepts of these techniques and the clinical experiments using them appear in the literature.

The most recent study reported by Sarno and colleagues in 1972 was in agreement essentially with the over 200 patient reports where hyperbaric oxygen was administered in an attempt to improve some aspect of neurological functioning.

The preponderance of these negative results, however, should not be interpreted to mean that oxygen supply to the brain cannot be increased nor that increasing it would not be beneficial.

Using a supplemental oxygen technique rather than a hyperbaric technique, Haas in 1967 reported an increase in oxygen saturation in arterial blood from 94% to 99% in 19 left hemiplegic subjects.

Ben-Yishay that same year studied the visuomotor and motor impersistence performance of 24 left hemiplegic patients with and without supplemental oxygen and found significant improvements in 95% of the subjects.

It was the intent of this study to investigate the effects of supplemental oxygen inhalation on the verbal, visuomotor and oral neuromotor functioning of aphasic hemiplegic adults.

Twelve subjects served as their own controls in this double-blind study of the effects of supplemental oxygen respiration versus a placebo of room air on the performance of aphasic adults.

The subjects were patients in local Philadelphia hospitals and met certain criteria:

1. All were at least six weeks post CVA.
2. None had a history of previous CVA.
3. All had formal education to at least the 4th grade.
4. None had a history of pulmonary disease.
5. None had a history of psychiatric disease.
6. None had a history of hearing impairment nor presented with a hearing loss.

The subjects ranged in age from 19 years to 65 years with a mean age of 49 and a median age of 50.5 years.

The average weeks post onset of CVA was nine weeks.

Eight of these subjects' responses were used to determine the effects of 24 hours of supplemental oxygen. These eight subjects ranged in age from 19 to 65 years, with a mean age of 49.75 years and a median age of 52 years. The average weeks post onset of CVA was 7.5 weeks.

Every subject was pretested with the PICA, the Purdue Pegboard and a test of diadochokinesis. These tests were administered 48 hours prior to re-examination under a placebo condition and an experimental condition in order that a baseline be established for purposes of comparison. In the oxygen experimental condition, the subjects breathed pure oxygen at a flow rate of three liters/minute through a nasal cannula.

The placebo condition differed from the experimental condition in that subjects were breathing room air through the nasal cannula rather than oxygen. It also differed from the pretest condition where the nasal cannula was not used.

Following pretesting, the subjects were retested once under the placebo condition and again under the experimental condition. These retests took one hour each and were administered one each day for two consecutive days.

Eight of the subjects were sustained on supplemental oxygen for a twenty-four hour period and retested during the twenty-fourth hour of exposure.

The order of the placebo or experimental condition was randomized and neither the examiner nor the subject knew which condition was in effect until the entire experiment was completed. Order of the test presentation was also randomized.

The placebo was used to determine any Hawthorne effects.

The t-test for correlated samples was used to test for the significance of the difference between means for each phase of this investigation.

The results indicated that the subjects' performance on the Purdue Pegboard and on diadochokinetic tasks improved to a statistically significant degree while they were breathing supplemental oxygen for one hour as compared to their pre-test performances.

PICA overall scores also improved to a slight but statistically significant degree while the subjects were on supplemental oxygen for one hour.

Long term exposure to supplemental oxygen also demonstrated statistically significant improvement over pre-test scores but did not increase significantly the degree of improvement with the one hour exposure in any of the tasks in the test battery.

A positive placebo effect was found only in the subjects' overall performances on the PICA but not on the Purdue Pegboard nor in diadochokinetic skills.

In view of this finding it was interesting to determine whether the improvements on the PICA were modality specific. Using the t-test for correlated samples, it was found that while subjects were breathing supplemental oxygen there was significant improvement at the .01 level in the gestural modality and at the .005 level in the graphic modality. No significant change was seen in the verbal modality.

Under the placebo condition, a significant improvement at the .05 level was seen in the verbal modality and at the .01 level in the graphic modality. No significant improvement was seen in the gestural modality.

The question arises as to why oxygen did not enhance verbal performance while a placebo did. An answer to this can only be conjectured.

The placebo may have motivated subjects to perform maximally. This may account for significant improvement where it did occur. If it is assumed that oxygen enhanced respiration also offered the subject added motivation to perform maximally, there must be some explanation for this positive effect not taking place. The PICA scoring system allows for objective notation by score not only of accuracy of a response but also of the behavior of the subject as he attempts to respond. For example, an intelligible though incorrect response which does not reject the task will receive a different score (6 or 7) from one rejecting the task (5). It is suggested that under the effects of a placebo, a subject might be prone to responding to each and every item although his responses may be incorrect. Under the oxygen it is postulated that the subject's state of awareness may be increased so that he is able to recognize more clearly his deficits and inabilities to respond correctly. He might, therefore, reject or hesitate to respond to items to which he doesn't feel capable of responding correctly. This would in effect, create an illusion of poorer performance when in fact, the subject may be functioning at a higher cognitive level.

The new behavioral response may be demonstrative of movement through the stages of improvement described by Wepman 1958 as movement from one level of ability to self correct to another.

To investigate this further the number of rejections and delays were tabulated and compared under the oxygen condition and the placebo condition. It was found that a significant increase in both rejection and delay did occur with the latter behavior more than doubling in occurrence. Clearly, this is conjecture and needs further investigation.

However, within the limits of this study it may be concluded that:

1. Aphasic hemiplegic subjects made slight but statistically significant improvement in their overall communicative abilities, diadochokinetic ability and manual visuomotor skill while respiring supplemental oxygen.

2. Supplemental oxygen is effective when provided for one hour through nasal cannula at three liters/minute.

3. The effects of oxygen enhanced respiration do not improve by extending the exposure time.

4. A placebo has a slight but statistically significant effect in increasing overall communicative ability but does not affect oral neuromotor or manual visuomotor functions.

5. Finally, the potential therapeutic value of supplemental oxygen with aphasic patients should be reconsidered pending further research.