

Nice (France), November Z-10, 1999



Editors

PHILIPPE EVRARD CHRISTIAN RICHELME MARC TARDIEU



MONDUZZI EDITORE

I INTERNATIONAL PROCEEDINGS DIVISION

Hyperbaric oxygenation in cerebral palsy and the brain injured child

V EPNS Congress

European Paediatric Neurology Society

Nice, France 7-10 November 1999

R. NEUBAUER

Ocean Hyperbaric Center, Lauderdale-by-the-Sea, FL (USA)

SUMMARY

Compelling evidence from around the world suggests that hyperbaric oxygenation (HBO) may play a major role in the treatment of children with cerebral palsy (CP) and brain injuries when used in the appropriate dose and time period. Its role is to reactivate idling dormant neurons and to produce a more desirable environment for the growth of neuronal tissue. Modification of the devastating effects of these problems frequently occurs. A recently completed study at McGill University in Montreal showed highly positive effects and further studies are ensuing there (1). Colombia Presbyterian in New York, as well as Cornell University are beginning trials.

INTRODUCTION

This study was performed using sequential SPECT before and during and after treatments of all patients to help identify specific areas of recoverable brain neurons. Observation on gross/fine motor and cognitive and visual, spasticity as well as videos were performed in each case. Three unusual cases with positive imaging with paralleled clinical improvements will be presented.

In Russia and Brazil, hyperbaric oxygenation has been used for many years in the early stages of the disease (2). In this study, sequential

Copyright 1999 by Monduzzi Editore S.p.A. - Bologna (Italy)

3rd EPNS Congress

European Paediatric Neurology Society

Nice, France 7-10 November 1999 SPECT (single photon emission computerized tomography) scanning was used before, during and after treatments of all patient to identify specific areas of recoverable brain neurons. Three unusual cases with positive SPECT imaging and significant clinical improvement will be presented.

MATERIALS AND METHODS

Eighty patients with CP and brain injuries have been treated with hyperbaric oxygen. The age of the patients ranged from six weeks to 14 years old. Hyperbaric oxygenation was performed in ten Vickers monoplace chambers and the pressures ranged from 1.25-1.75 atmospheres absolute (ata) for one hour each with total number of treatments varying from 4-300 exposures, along with sequential SPECT scanning following at 40, 80, 120, etc. If necessary, the parent would accompany the child in the chamber. The SPECT scan is located in-house. It is an Elscint singlehead gamma camera. The radioactive tracer used was Technetium 99, (HMPAO), Ceretec, by Amersham. The dose of the tracer was adjusted to the weight of each patient. The protocol consisted of the initial SPECT scan as a baseline prior to hyperbaric oxygenation. The second scan was done immediately following the last hyperbaric oxygen exposure. Utilizing SPECT scans, we measured changes in blood flow and metabolism since the tracer crosses the blood brain barrier. There is a strong correlation between the positive changes in SPECT imaging and the clinical condition. Videos were used during the course of the HBO treatments and all patients were assessed for visual and cognitive abilities, fine and gross motor changes, and positive changes in spasticity.

AMI (Figure 1 A&B): The patient is a 3 year old girl born 1 pound 11 ounces at 26 weeks gestation by emergency C-section due to placenta previa. Cerebral palsy developed and was manifest by severe four extremity spasticity, hyper reflexia, clonus, micro cephaly, developmental delay, and retinopathy of prematurity. Eighty-four HBOTs were administered at 1.25 - 1.5 ata/60 minutes, 1-2 per day, 5-6 days per week from 1/5/99 to 4/5/99. Baseline SPECT pre HBOT showed hypoperfusion in the basal ganglia, thalami, and occipital lobes. Repeat SPECT after 29 HBOTs showed improvement in the occipital lobes with simultaneous improvement in alertness, verbal and gross motor skills, affect, sleep, vision, cognition, and a decrease in spasticity.

JSC (Figure 2 A&B): The patient is a 15 month old, term baby boy whose delivery was complicated by prolonged cord constriction of the neck, seizures, subdural hematoma, and traumatic intubation, resulting in cerebral palsy. The patient received 35 HBOTs at 1.5 ata, 1-2 times per day, 5-6 days per week, from 5/5/99 to 5/26/99. The initial SPECT scan of 5/4/99 showed multiple marked areas of hypoperfusion which improved considerably after 35 HBOTs on repeat SPECT scan of 5/26/99. The patient experienced better gross motor and head control, standing balance, decreased spasticity, is drinking out of cup, and having increased vocalization.

CB (Figure 3 A&B): The patient is 5 years old boy delivered at 28 weeks following miscarriage of his/her twin at 12 weeks gestation, who developed early broncho-pulmonary dysplasia, sepsis, pneumonia, periventricular leukomalacia and a diagnosis of cerebral palsy. This patient re-

284



3rd EPNS Congress

European Paediatric Neurology Society

Nice, France 7-10 November 1999

Figure IA: AMI 3-D reconstruction pre-HBOT treatment Figure 1B: AMI post 29 HBOT treatments

^{3rd} EPNS Congress

European Paediatric Neurology Society

Nice, France 7-10 November 1999



Figure 2A: JSC 3-D reconstruction pre-HBOT treatment

286

Figure 2B: JSC post 35 HBOT treatments

EPNS Congress

European Paediatric Neurology Society

Nice, France 7-10 November 1999 ceived 37 HBOTs from 2/5/99 to 3/4/99 at 1.5-1.7 ata, 1-2 times per day, 5-6 days per week. The baseline SPECT scan of 2/4/99 showed marked hypoperfusion of the left frontal and bilateral parietal occipital areas which improved considerably on repeat SPECT scan after 30 HBOTs on 3-2-99. Simultaneously, spasticity decreased and the patient experienced improvement in speech, cognition, eating, sleeping, and walker assisted gait.

RESULTS AND CONCLUSIONS

In all cases, cerebral palsy has an etiologic episode which is ultimately hypoxic ischemic. Unfortunately, preemies and hypoxic infants are not given large doses of surface oxygen because of the fear of retrolental fibroplasia. Hyperbaric oxygenation has no such side effect. Recent uncovered data showed that the oxygen was not the cause, but the withdrawal from the oxygen environment. Resubmersion into the oxygen completely cured the retrolental fibroplasia (3).

There are multiple conditions causing the problems in utero, at birth or immediately post partum such as premature placental separation, amniotic fluid embolus, trauma and stroke at birth, cord around the neck, meningitis, peritonitis, and shaken baby syndrome, etc. In Russia and South America, many neonates have been taken from the delivery room into the hyperbaric chamber. With newer methods such as ultrasound, SPECT, PET (positron emission tomography) and functional MRI (magnetic resonance imaging), devastating problems may be easily identified prior to development of clinical symptoms and this is the time for intervention with hyperbaric oxygenation.

A study at McGill recently completed (and in press), was so highly positive with the above mentioned clinical features, that McGill University was given 1.8 million dollars to continue this study. Studies are being performed in the above mentioned Universities. There is a hyperbaric oxygen trust in the United Kingdom, several hundred children are currently under treatment with positive results. Results are compelling. The authors were the first to document the changes with the functional imaging, SPECT. In younger patients with dormant idling hypoxic brain areas, recoverability with pressurized oxygen is essential. The administration of hyperbaric oxygen also produces a better internal environment for the growth of new brain tissue. Observations with a developmental physiologist is encouraged to appropriately define developmental changes.

The use of hyperbaric oxygen is not new. It is the only method in which free molecular oxygen for immediate metabolic use may be delivered to the tissues and this is in direct relationship to the pressure, according to Henry's Law. Hyperbaric oxygen around the world is used primarily for air embolus, decompression illness, carbon monoxide intoxication (or poisoning), wound care, bone infections and radiation damage. More recently, the immediate neurological effects of hyperbaric oxygenation in acute stroke were reviewed by the World Federation of Neurology (WFN) and a specific protocol has been devised for its use in the acute ischemic thrombotic stroke (4). It has multiple positive effects on the injured or hypoxic brain. In the brain, there is no matrix, hence, its diffusional driving force is not limited. Therefore, molecular oxygen is made

288

immediately available without energy exchange to neuronal tissue. It reduces swelling with cerebral edema and reduces intracranial pressure. It restores the integrity of the blood brain barrier and the cell membrane, and neutralizes toxic remains. It initiates angiogenesis. Its most important activity is to reactivate idling neurons, that is, the cells that are receiving enough oxygen to exist, but not enough to fire electrically (5). In all cases of cerebral palsy and traumatic brain injury in children there is an hypoxic ischemic episode. Unfortunately, hyperbaric oxygenation is not used until all of the modalities have been tried. Ideally, it should be used as soon as the problem is diagnosed. Hopefully, these cases in this discussion will stimulate further research. The side effects are virtually negligible. In the United Kingdom, there have over 1.25 million hours of hyperbaric oxygen given without incident.

3rd EPNS Congress

European Paediatric Neurology Society

Nice, France 7-10 November 1999

REFERENCES

- (1) MAROIS P., LACROIX V., MONTGOMERY D. AMAR M., LAMBERT J., The use of Hyperbaric Oxygen Therapy in Children with Cerebral Palsy, in press, Monduzzi Editore, 1999
- (2) MACHADO, J., Clinically observed reduction of spasticity in patients with neurological diseases and in children with cerebral palsy from hyperbaric oxygen therapy, in press, Monduzzi Editore, 1999
- (3) JAMES P., Surface oxygen (80% oxygen) in premies is not the cause of retrolental fibroplasia, in press, Monduzzi Editore, 1999.
- (4) TOOLE J., World Federation of Neurology: Congress on Cerebral Ischemia. Vascular Dementia, Epilepsy and CNS Injury, May 9-13, 1998
- (5) JAIN K., Textbook of hyperbaric medicine, 3 rd revised edition USA, Hogrefe & Huber, 1999.