HYPERBARIC OXYGEN THERAPY FOR CEREBRAL PALSY, A POSITIVE STUDY THAT IS BOTH ILLUMINATING AND CONFUSING

Sir:

The recent Canadian multicenter hyperbaric oxygen therapy (HBOT)/cerebral palsy trial should be applauded. This study has emphatically reaffirmed the commonly reported benefit of low-pressure HBOT in cerebral palsy (1,2) and the previous McGill Pilot Trial (3). Simultaneously, it has suggested an equally effective treatment, 1.3 ATA air which has confused the medical community. The confusing equality of the two treatments may be due to a number of factors:

First, the dose of HBOT may be too high. Machado's (1) dose was 1.5 ATA oxygen/60 minutes/20 treatments, our dose was 1.5 ATA/60/40 or 80 treatments (2), the Sussex, England dose was 1.75 ATA of 95% oxygen (or 1.66 ATA 100% oxygen)/60/20 treatments, and the McGill Pilot Trial (3) was 1.75 ATA pure oxygen/60/20 treatments. Due to rapid regression of some of the patients in (3) post treatment, the protocol was increased at the strong urging of myself and Dr. Richard Neubauer to 40 treatments based on our experience with 1.5 ATA/60/40 treatments. Unfortunately, this higher dose was previously untested in cerebral palsy and may have inhibited a maximum benefit of HBOT or induced a degree of metabolic fatigue that could have suppressed test performance if testing was performed within two weeks after the 40th treatment. This is suggested by improvement in the HBOT group on 80% (4 of 5) of the GMFM subtests vs. 40 % (2 of 5) in the air group from post-intervention to 3-month follow-up testing.

Second, the air group is not a true control group; 1.3 ATA air is 27.3% oxygen. Surprisingly, this low dose of oxygen seems to be sufficient as a signaling agent (4) to cause significant improvement equal to the HBOT group. Alternatively, the combination of oxygen and pressure at 1.3 ATA altered the function of gaseous neurotransmitters such as carbon monoxide, nitric oxide, and others? to cause these improvements. To attribute the significant improvement in either group to a participation effect is unlikely; lifelong therapies and maximal stimulation have previously not had the same effect. In addition, the quantitative changes noted in this study have been documented qualitatively and with functional imaging by myself and colleagues on over 75 CP patients in the past 9 years where the parent participation effect is non-existent due to monoplace chamber treatment (5).

Third, the groups are mismatched with greater severity of injury in the HBOT group. The authors state that the greatest changes occurred in those with the lowest GMFM scores at baseline. If so, one would expect the HBOT group to show greater improvement. Unfortunately, lack of subgroup stratification by GMFM or CP subtype prevents confirmation of their statement, and thus, separation of the HBOT and air groups. At the same time, the study maybe underpowered to separate the groups. This is suggested by the results of three of the four TOVA tests which show markedly greater improvement in the HBOT group which are surprisingly statistically non-significant.

In summary, congratulations to the authors and the mothers who helped secure the study's funding. The findings reaffirm the common experience of HBOT in CP and offer surprising evidence for an equally effective treatment, hyperbaric air.

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