Central nervous system oxygen toxicity during routine hyperbaric oxygen therapy.

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To the Editor:

We read the recent article by Hampson and Atik regarding CNS toxicity during HBO₂ (1). CNS toxicity has been a concern of specialists in HBO₂ therapy for a long time despite its low incidence. In that article, it was very interesting that the role of the equipment used in HBO₂ therapy on CNS toxicity was emphasized as a contributing factor to the condition, as being a cause of risk of toxicity. It is obvious that the incidence of CNS toxicity may depend on chamber conditions and treatment protocols. The article cites the study of Welslau in 1996 in which it was reported that out of a population of 107,264 patients, the overall incidence of CNS toxicity was 1 in 6704 (2). In that study, emergency conditions were included. In the present study, the incidence of CNS oxygen toxicity was reported at a rate of 1 in 3388 even though emergency conditions were excluded and it is clear that treatment protocol is a risk factor. The Hampson and Atik results were consistent with those of Welslau only when the same protocol was used. This point makes the importance of the treatment protocol on CNS oxygen toxicity clear. We thought this subject should be stressed more.

We would like to summarize our results of 36,500 patient treatments performed in our Hospital between 1996 and 2003. The incidence of CNS toxicity was three seizures in 36,500 patient treatments. Emergency conditions such as DCS, CO poisoning etc. were included. All treatments were carried out in a Galeazzi 17-person multiplace hyperbaric chamber. Our protocol was 90 minutes of oxygen in three 30 minute periods with 5 minutes air breaks at 2.36 ATA for routine sessions. We used US Navy recompression tables to treat DCS. We used 75 minutes of oxygen in three 25 minute periods with 5 minutes air breaks at 2.8 ATA protocol for emergency situations especially gas gangrene and CO poisoning. We used 100% of oxygen via mask for routine therapy. In emergency cases we used a hood, ventilator or mask.

The first of the three toxicity cases was a twenty-two year old man with sacral decubitus ulcers. A seizure was seen in the 30th session of HBO₂ therapy in the course; then therapy was discontinued. We transferred him to the neurology department where anticonvulsive therapy was carried out and after three days he died from status epilepticus. The second case was a fourteen-year old boy with crush syndrome of the toes of his left foot. In this case, the seizure was
observed in the 14th session of HBO2 treatment and we completed 20 sessions. Our last case, a forty-eight year old man, suffered from DCS. At 60 FSW on oxygen, a seizure occurred. None of these three patients had any risk enhancers for oxygen toxicity or any history of convulsions, and we used masks in all three of them for oxygen delivery.

The CNS oxygen toxicity incidence in our 7 years of work was therefore 1 in 12,166. This is compatible with the Hart and Strauss study and the Davis study referenced by Hampson (3,4). Our treatment protocol is the same as in the present study but for routine therapy we used a mask for oxygen administration and we think the higher incidence of CNS toxicity in the Hampson and Atik study was related to the use of the oxygen hood. Because there is no Department of Radiation Oncology in our Hospital, we rarely treat chronic radiation injured patients. We think the large number of these kinds of patients may also contribute to the high incidence of CNS oxygen toxicity in Hampson’s study. In other words, the treatment indication may be another factor that changes the incidence of CNS oxygen toxicity.

In summary, equipment and therapy indications may be important in risk of development of CNS oxygen toxicity, but we do not have definitive data. Perhaps the most important factor in CNS toxicity development is the individual patient’s predisposition.

REFERENCES