

15. SIDE EFFECTS AND COMPLICATIONS

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Middle ear barotrauma is the most common side effect of hyperbaric oxygen (HBO₂) therapy (1,2). A review of 1,446 patients with 31,599 exposures showed an incidence of approximately 2%. It is prevented in most patients by teaching autoinflation techniques or by use of tympanostomy tubes for those who cannot autoinflate their middle ear compartment. A prospective study in patients treated with HBO₂ demonstrated that many patients develop serous otitis media during treatment. A history of eustachian tube dysfunction predicted serous otitis media (3). Pseudoephedrine has been demonstrated to be effective in preventing barotitis media in a double-blind, randomized, controlled clinical trial in underwater divers (4). Topical nasal oxymetazoline hydrochloride was ineffective in preventing middle ear barotrauma during HBO₂ therapy (5).

Sinus squeeze is seen less frequently than middle ear barotrauma (1). This second most common chamber complication usually occurs in patients with upper respiratory tract infections or allergic rhinitis. Usually a program of decongestant nasal spray, antihistamines, and/or steroid nasal spray just before compression allows therapy to continue.

Serous otitis has been reported in patients receiving HBO₂ therapy (3). Although once thought to be due to a reduced middle ear pressure by oxygen resorption, there is evidence to suggest that HBO₂ might cause a reversible derangement in a middle ear chemoreceptor reflex arc which may regulate middle ear aeration (6).

Claustrophobia, which appears to be present in about 2% of the general patient population, may cause some degree of confinement anxiety, even in a multiplace chamber. Occasionally, mild sedation is required for such individuals to continue to receive daily HBO₂ therapy (1).

Progressive myopia has been observed in some patients undergoing prolonged periods of daily HBO₂ therapy (7-10). Although the exact mechanism remains obscure, it is apparently lenticular in origin (8) and usually reverses completely within a few days to several weeks after the last therapy (7,9). Lyne (9) studied 26 patients undergoing HBO₂ therapy for more than a month. Their ages ranged from 36 to 80 yr, and four were diabetics. Treatments were at 2.5 atm abs with a 30-min compression while breathing oxygen, 60 min on oxygen at 2.5 atm abs, and a 30-min decompression breathing oxygen. Duration of the therapy series ranged from 4 to 52 wk. Pretreatment and monthly follow-up studies included refraction with and without cycloplegia, keratometry, tonometry, fundus examination, and axial length by ultrasonography. Eighteen of the 26 patients, including all 4 diabetics, developed myopia ranging from 0.5 to 5.5 diopters. After the series of HBO₂ therapies was concluded, reversal of myopia was usually rapid for the first few weeks and then continued more slowly for periods ranging from several weeks to as long as a year. No other ocular effects were found. Specifically, there were no fundus changes. No patient who started with a clear lens developed any opacities. In those patients with lens opacities present at the beginning of therapy, the opacities did not progress during therapy. No further changes were found during follow-up periods ranging from 6 mo. to 2 yr. Lyne (9) suggested that the change in refraction was due to an increased refractive index of the lens.

Twenty-five patients who received extremely prolonged treatment consisting of 150-850 daily exposures at 2-2.5 atm abs, 7 days a wk, were studied in Sweden (10). All patients but one showed myopic refractive changes. Of 15 patients with clear lens nuclei before treatment, 7 developed well-defined nuclear cataracts. The earliest that any lenticular change occurred was at

150 treatments over 4 mo. Three of the 15 developed nuclear turbidity at 150-200 treatments; 11 developed these changes after 200-850 treatments over 8-19 mo.; and in 1 patient no lenticular changes were noted. The nuclear cataracts were not reversible after cessation of these extremely prolonged therapy series.

In what appears to be an exception to the general observation that new cataracts occur only in extremely prolonged series of hyperbaric oxygen therapies, early cataract development in a 49-year-old woman who had only 48 therapies over a period of 11 weeks has been reported (11). Each therapy consisted of O₂ breathing at 2.5 atm abs for 90 min with two 5-min air breaks. Bilateral cataract formation was associated with a myopic shift that remained progressive for over 4 months after cessation of the therapy series and then stabilized at 3.25 diopters. The cataracts and associated myopic shift persisted at least until the last follow-up at 11 months post-therapy. Although the patient was not diabetic or taking steroids, her unusual susceptibility to cataract formation raises the possibility of an undetected, predisposing condition.

The above published reports (7-10), as well as extensive clinical experience in major hyperbaric centers, indicate that, with one possible exception to date (11), new cataracts do not develop within the series of 20-50 therapies that are commonly used in the United States. Even when progressive myopia does occur during a series of HBO₂ therapies, the visual acuity changes almost always reverse completely when the total number of therapies does not exceed 100. However, extension of a series beyond 100 therapies is associated with an increased risk of irreversible, refractive changes or the development of new cataracts.

Patients should be provided with this information as part of their informed consent for HBO₂ therapy. A baseline ophthalmology examination is suggested to establish preexisting lenticular opacities in patients at risk (over 50 yr of age, diabetes mellitus, irradiation therapy of the head and neck, and systemic steroid therapy). Results of recent studies are consistent with the possibility that the incidence of myopia may be greater for HBO₂ therapy at 2.4 than at 2.0 atm abs (12,13). It is also likely, though not yet demonstrated, that the lens PO₂ will be much higher when the eye is directly exposed to 100% O₂, as in an O₂ hood or monoplace chamber, than when O₂ is administered via facemask in a compressed air environment.

Other reversible effects of hyperoxia on visual function in man include contraction of peripheral vision (14,15) and the reduction in the electrical response of retinal glial cells to a light flash (16). These effects only occur in oxygen pressure-duration combinations that greatly exceed all but the most aggressive, current applications of HBO₂ therapy. Exceptions to this general rule include the development of retrolental fibroplasia following exposure of the premature retina to relatively low levels of hyperoxia (17), and a reversible loss of vision during a relatively brief oxygen exposure in an individual who had a previous history of retrobulbar neuritis (18).

Pulmonary and neurologic manifestations of oxygen poisoning are often cited as major concerns. Oxygen tolerance limits that avoid these manifestations are well defined for continuous exposures in normal men (19,20). Pulmonary symptoms are not produced by daily exposures to oxygen at 2.0 or 2.4 atm abs for 120 or 90 min, respectively. Normal human subjects (21) and patients with significant pulmonary dysfunction (adult respiratory distress syndrome) (22) treated with HBO₂ did not demonstrate alterations in pulmonary gas efficiency (pre- and post-HBO₂ arterial/alveolar ratio did not change). However, it is possible that cumulative effects of pulmonary oxygen toxicity are produced by repeated daily exposures even though individual exposures have no detectable influences on pulmonary function. Statistically significant, but quantitatively small, changes in lung expiratory function were measured in a

group of 20 patients after 2-3 weeks of breathing oxygen at 2.4 atm abs for 90 min each day (23). These changes were not associated with pulmonary symptoms or functional limitations. Similar pulmonary function measurements at weekly intervals over a period of 6 weeks did not detect significant changes in a different group of 18 patients who breathed oxygen for 90 min at 2.4 atm abs for a total of 30 therapies (24). The former group of patients (23) had normal lungs and were treated on 21 consecutive days, while the latter group (24) had a preexisting impairment in carbon monoxide diffusing capacity and were treated 5 days each week. Additional measurements are needed to investigate the possible occurrence of cumulative effects during multiweek periods of therapy.

Pulmonary barotrauma during decompression may rarely occur (25-27). Patients with airway obstruction probably are at an increased risk for pulmonary barotrauma during decompression. Significant air trapping and a history of spontaneous pneumothorax are also causes for concern and mandate a careful analysis of potential benefit from hyperbaric oxygen therapy versus the associated risk.

Early estimates of the seizure rate during therapeutic oxygen exposures at 2.0-3.0 atm abs reported a convulsion incidence of about 1 per 10,000 therapies or 0.01% (1,2,28). More recent surveys that include a total of 9957 patients from three different facilities (29-31) indicate a combined incidence of about 0.03% for a therapy protocol consisting of three 30-minute periods of oxygen breathing at 2.4-2.5 atm abs. Oxygen was delivered by head hood in the recent surveys, while the earlier studies employed a variety of delivery systems that were not always specified. Possible reasons for the differences in observed seizure rates include the potential for CO₂ accumulation in a hood and unrecognized air leaks allowed by a poorly fitting facemask.

Among 900 patients who received HBO₂ therapy for carbon monoxide poisoning, 16 or 1.8% had seizures (32). The seizure incidence for different O₂ pressures ranged from 0.3% at 2.4 atm abs (N=300) to 2.5% at 2.8-3.0 atm abs (N=600). Even when oxygen convulsions do occur, there are no residual effects if mechanical trauma can be avoided.

The United States Air Force School of Aerospace Medicine did a long-term follow-up study of 563 patients each of whom had over 20 daily HBO₂ therapies of 90 min oxygen breathing at 2.4 atm abs (1). The follow-up period was 6 mo. to 8 yr. No chronic or late effects due to HBO₂ were seen. Cataracts occurred in only two patients (a poorly controlled diabetic and a 67-yr-old man on high dose steroids).

It has recently been demonstrated that critically ill pediatric patients can be safely administered HBO₂ in a multiplace chamber by experienced personnel who use appropriate precautions (33). A group of 32 children, ranging in age from 3 days to 11 years, were mechanically ventilated while receiving HBO₂ therapy for necrotizing infections (N=21), carbon monoxide poisoning (N=9), or iatrogenic arterial air embolism (N=2). Complications included hypotension (63%), bronchospasm (34%), hemotympanum (13%), and progressive hypoxemia (6%). One child was accidentally extubated during transport.

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