It is well known that hyperbaric oxygen therapy can have an effect on eyesight and produce visual manifestations in some patients. High levels of oxygen under pressure for extended periods of time can affect the Central Nervous System (CNS) resulting in oxygen toxicity. The visual side effects of oxygen toxicity are twitching of the eyelids and blurred vision. Less common is visual constriction and hallucinations. The reasons for these effects are uncertain but this toxicity may be the result of oxygen metabolism and the production of superoxide radicals (radicals damage cells, proteins and DNA by altering their chemical structure). Radicals are usually kept in check by cellular defense mechanisms but these may be overwhelmed in the present of such high oxygen levels. The most common defender is called superoxide dismutase, which is an antioxidant enzyme. CNS oxygen toxicity can occur quickly during oxygen exposure but can subside quickly by removing the patient from the high oxygen environment. It is impossible to predict which patient may be affected although factors such a treatment pressure and duration among others, can increase or decrease the risk. The same patient may be treated 20 times and experience no effects of oxygen toxicity until treatment number 21, and then experience no further episodes. Hyperbaric treatment can be initiated shortly after the effects subside (15 minutes) but our practice is to end the treatment and continue the next day. Another type of oxygen toxicity affects the retina of the eye, in which the patient experiences a decrease in their peripheral vision. This type is very rare and reversible once oxygen exposure is stopped.

The most common type of oxygen toxicity is lenticular or affecting the lens. This phenomenon is called progressive myopia, a condition represented by defective vision of distant objects, also called nearsightedness. The number of hyperbaric treatments administered increases its occurrence and this is usually not seen when less than 20 treatments are given. Treatments are usually 90 to 120 minute in length and given on a daily basis at 2.0 ATA or higher. The incidence is reported to be between 20 to 40% and more often seen in diabetic and elderly patient. The good news is that when hyperbaric therapy is finished the condition is reversed. Complete recovery usually takes about 6 weeks although it may take longer in some cases. This condition is always discussed with patients so they realize that is not uncommon and that it is reversible. For this reason, patients are told not to get a new eyeglass prescription until about 6 weeks after therapy has ended. For patients with presbyopia, their condition may improve. They normally have an inability to focus sharply on items that are close (like with us middle-aged people who have trouble reading). Unfortunately this improvement will also reverse itself after therapy is terminated.

Cataract formation is a potential side effect of prolonged hyperbaric therapy and is discussed with patients. It has been found that new cataracts can develop and existing cataracts may worsen. Unfortunately this condition of lens opacity (reduction in light reflective properties) does not reverse itself and may require surgery for repair.

There are ocular contraindications to hyperbaric oxygen therapy. Patients who have had placement of an orbital prosthesis called a hollow silicone orbital implant should not be exposed to changing ambient pressures. There is a potential for a pressure-induced collapse of the prosthesis in this case. Fortunately the hollow design is rarely used today. The second contraindication is an intraocular gas bubble when introduced by surgeons as an internal stent to maintain position of the retina. In this case an airspace in the eye can change in volume and cause barotrauma. However, if the

Therapists and Physicians should be aware that there are ocular contraindications to hyperbaric oxygen therapy

continued on next page

“Congratulations. You’re getting a raise. Let me know if the check clears.”
Visual Manifestations...continued from previous page

the obstetrics and gynecology community. Vidaeff and Ramin thought the technology offered insight into fetal hypoxemia and acidosis, but the technical artifacts, such as sensor-to-skin contact, acted to impede signal acquisition, and they questioned the cost effectiveness of fetal pulse oximetry.

A 2005 summation of evidence was published in the American College of Obstetricians and Gynecologists (ACOG) bulletin. These guidelines suggested that the use of fetal pulse oximetry could not be supported. Likewise, the work of Bloom and East concluded that fetal pulse oximetry was not effective in one of its primary indications, the reduction of cesarean section.

The fact that intrauterine fetal oxygen monitoring may have failed in its initial indication may lead one to think that the theory and the resulting instrumentation was a total failure. I would ask the reader to consider that, as in so many breakthrough technologies, the data gathered from intrauterine fetal pulse oximetry resulted in several major new discoveries. Simpson and James found that FSpO2 improved with fluid bolus and a tight fitting, high concentration oxygen mask on the mother, a theory difficult to support without this technology. Important to respiratory care practitioners is the influence of intrauterine pulse oximetry on the research of Zourabian and Vintzileos whose work on a transabdominal fetal pulse oximeter will eventually give real-time fetal oxygen saturation noninvasively through the mother's abdomen. If this technology is successful, then this ‘baby person’ thinks FSpO2 was a mis-step in the right direction.

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Disputed Technology...continued from page 56

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The last point to be made is that patients must be made aware of the potential effects that treatment may have on their vision. This should be an important topic in patient education and also a part of the signed patient consent. In most cases, vision changes are temporary and reversible and are not contraindications for therapy.

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