PRECAUTIONS AGAINST HARM IN
CENTRAL NERVOUS SYSTEM OXYGEN TOXICITY

C. J. Lambertsen

Report 2.2.86

Environmental Biomedical Research Data Center
Institute for Environmental Medicine
1 John Morgan Building
University of Pennsylvania Medical Center
Philadelphia, Pennsylvania 19104-6068
PRECAUTIONS AGAINST HARM IN

CENTRAL NERVOUS SYSTEM OXYGEN TOXICITY

Oxygen Tolerance

The breathing of pure oxygen at pressures above about 1.5 atmospheres for unlimited periods will result in a progression toward development of pulmonary and some form of central nervous system oxygen toxicity. Convulsions in exposures at rest are extremely unlikely at inspired oxygen pressures less than two atmospheres. Since the pulmonary airway surfaces receive a higher \( P_2 \) exposure than do most cells within metabolizing tissues, sensations of pulmonary effect can appear early at lower inspired oxygen pressures than those required to produce convulsions. To prevent the development of detectable symptoms or signs it is necessary to avoid excessive pressure and duration of exposure, and to establish appropriate periodic interruption of high oxygen breathing, which aids in restoring tolerance. The rates of development of detectable symptoms or signs of pulmonary and central nervous system oxygen poisoning appear not to be the same, the expression of poisoning is different, and each develops more rapidly as higher inspired oxygen pressures are breathed. The graph (Figure 1) illustrates empirical borderline limitations for oxygen in decompression and therapy.
Early Warning of Oxygen Toxicity

Central nervous system effects of hyperoxia begin early, but there is no completely dependable subjective or objective warning to indicate when actual oxygen convulsions are about to occur. While twitching of the facial muscles usually occurs, it may be so rapidly followed by convulsion and loss of consciousness that it is not possible to stop the convulsion from developing. At times a convulsion, about to begin, will still generate, right after the oxygen breathing has been discontinued.

Residual Effects of Oxygen and Oxygen Toxicity

If the pressure and duration of an oxygen exposure are not sufficient to produce symptoms of central nervous or pulmonary oxygen toxicity, residual effects should not be expected. Convulsions do not produce harm in themselves, but can result in physical injury due to falling, thrashing, tongue biting and uncoordinated activity on arousal.

Prevention of Harm

Owing to the extreme rapidity of the metabolic utilization of oxygen in the cell, the \( P_{O_2} \) at a particular intracellular location should fall to natural, nontoxic levels within the few minutes of a pulmonary "oxygen washout time." Return to normal pressure of inspired oxygen is therefore the primary measure for aborting or interrupting the development of acute oxygen toxicity. There have been a few instances in man in which the toxicity has progressed to such a degree that convulsions occur a few seconds after the
removal of the oxygen mask from the subject. In such a situation the lungs could still be full of oxygen. The breath holding involved in the initial tonic phase of convulsion then results in carbon dioxide tension increase, with gross increase in brain oxygen pressure during the clonic phase of convulsion.

If a convulsion occurs, it is necessary to prevent the patient from injuring himself during the vigorous and generalized clonic contractions. Excessive force in restraint should be avoided. During the tonic phase at the onset of the convulsion the head becomes hyperextended and the lower jaw is briefly strongly depressed so that the jaws are separated. During this period of about 10 seconds, a soft but firm "bite block" such as padded tongue depressor can easily be inserted to prevent chewing of the tongue during the subsequent clonic jaw clamping.

During both the tonic and clonic phases there is severe interference with pulmonary ventilation, probably including laryngospasm as well as soft tissue oropharyngeal obstruction and incoordination of thoracic movements. It is therefore extremely important to avoid decompression during any part of the convulsion, since expanding pulmonary gas would then rupture the lung and produce a possible fatal pulmonary embolism. During about three minutes following the clonic convulsion, rhythmic breathing returns. It is at this stage that attention to maintaining the airway is especially important, and should be provided, since the patient tends to have soft tissue obstruction of the throat and has an accumulation of unswallowed, excessive salivary secretions. In
approximately three to five minutes, as breathing becomes reestablished, it is possible to proceed with decompression, if this is practical.

Following a convulsion and return to air breathing, the postconvulsive depression wears off and consciousness usually returns over a period of 5 to 10 minutes. During the state of gradually returning consciousness, the patient may be irrational and will at least require quiet reassurance and gentle restraint to prevent confused activity and self-injury. At times consciousness returns abruptly and the patient shows surprising mental clarity. Headache or nausea may occur, and muscular fatigue is to be expected. Consciousness and normal central-nervous-system function should return within a few minutes to an hour after the convulsion. Following a convulsion, severe headache may result. This can be treated with aspirin, codeine, rest and sleep.

Figure 1. The severity of pulmonary oxygen toxicity at various pressure-duration exposures. The family of rectangular hyperbolas drawn through experimental findings obtained in vital capacity to be encountered at all tolerable combinations of pressure and duration of exposure to oxygen (1,2).
PULMONARY OXYGEN TOLERANCE CURVES IN NORMAL MEN
(BASED ON VITAL CAPACITY CHANGES IN 50% OF THE SUBJECTS)

This is just generation from the master.