Hyperoxia and acute lung injury

Aron B. Fisher and Michael F. Beers

Institute for Environmental Medicine and Department of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania

TO THE EDITOR: The review by Matute-Bello et al. (15) has misleading and potentially dangerous statements concerning the role of hyperoxia in human acute lung injury (ALI). Table 4 states “in normal human lungs, 100% oxygen has not induced lung injury...” The text states that most mammalian species develop respiratory distress and die with exposure to 100% oxygen, but the “same findings have not been reproduced in humans with normal lungs...” That statement is essentially true (fortunately) but mainly because very few studies of potentially lethal hyperoxia have been carried out in individuals with normal lungs, for obvious reasons. The one study referenced in the review (1) was carried out in patients with irreversible brain injury, which may have influenced the pulmonary response to hyperoxia, and indeed a subsequent similar study demonstrated significant lung injury (13). Studies of normal individuals exposed experimentally to 100% oxygen at normal pressure (i.e., 1 ATA) have shown evidence of tracheobronchitis and changes in vital capacity, diffusing capacity, and lung permeability (2–5, 7, 17). Inadvertent exposure of patients with normal lungs to prolonged hyperoxia resulted in clinical findings compatible with oxygen toxicity (12). Exposure of normal humans to oxygen at elevated pressure (that is, greater than 1 ATA O2) has indicated a shorter duration of exposure for equivalent pulmonary symptoms and function changes (8, 10, 16, 18). Pulmonary-related death has been reported for one patient in the normobaric O2 exposure group (12) and another with hyperbaric exposure (9). Thus evidence of lung abnormalities with severity proportional to the partial pressure of oxygen has been demonstrated in normal human lungs similar to the findings with experimental animals. It is known that different species, and indeed strains of the same species as well as individuals from the same strain, show varying sensitivity to the toxic effects of oxygen (11). Rats exposed to 0.8 ATA O2 as individuals from the same strain, show varying sensitivity to different species, and indeed strains of the same species as well to the findings with experimental animals. It is known that oxygen has been demonstrated in normal human lungs similar malalties with severity proportional to the partial pressure of oxygen. Thus evidence of lung abnor-

REFERENCES