

BRIEFS FROM THE LITERATURE

JOHN W. PENDER, M.D., *Editor*

Briefs were submitted by Drs. C. M. Ballinger, Lee S. Binder, M. T. Clarke, J. E. Eckenhoff, Martin Helrich, J. J. Jacoby, E. J. Nelson, R. E. Ponath, William Rabenn, Alan D. Randall, and R. W. Ridley. Briefs appearing elsewhere in this issue are a part of this column.

DIFFUSION RESPIRATION Diffusion respiration carried out in dogs made apneic by succinylcholine results in arterial carbon dioxide tensions as high as 427 mm. of mercury and arterial pH readings as low as 6.48. Plasma adrenaline and noradrenaline concentrations increase progressively during diffusion respiration. In the first 30 minutes the rise is predominantly in noradrenaline; subsequently plasma adrenaline increases greatly, reaching levels up to 30 μg . per liter after 60 minutes of apnea. Ventilation rapidly reduces the arterial carbon dioxide tension and brings circulating catecholamine concentrations to near-basal levels. There is no evidence of further increase of plasma adrenaline and noradrenaline or untoward cardiac effects when ventilation is resumed and arterial carbon dioxide tension is reduced. Diastolic blood pressure falls during the first 15 minutes of diffusion respiration. Thereafter there is a marked increase in systolic pressure. After one hour of diffusion respiration blood pressure is depressed, but ventilation produces immediate, pronounced, but temporary rise in blood pressure. (Millar, R. A.: *Plasma Adrenaline and Noradrenaline During Diffusion Respiration*, *J. Physiol.* 150: 79 (Jan.) 1960.)

OXYGEN APNEA Administration of oxygen to patients with respiratory failure during acute chest infections presents problems largely unsolved. Such patients must have oxygen, yet the incautious administration of oxygen can plunge them into acidosis comparable with the most severe diabetic coma. The requirements for giving oxygen to chronic lung disease patients with acute superimposed lung disease, who are in respiratory failure, are described in 4 case histories. Oxygen

should be given continuously, but the concentration controlled so that it does not rise high enough to allow serious respiratory depression to occur. Likewise, it should not fall low enough to permit recurrence or increase of hypoxia. Rebreathing of expired air should be reduced to prevent carbon dioxide accumulation. (Campbell, E. J. M.: *Respiratory Failure—Relation Between Oxygen Concentrations of Inspired Air and Arterial Blood*, *Lancet* 2: 10 (July 2) 1960.)

INTRAGASTRIC OXYGEN Favorable effects from the administration of oxygen by gastric tube to anoxic infants has been reported previously. This phenomenon has been studied in cats, kittens and puppies anesthetized with pentobarbital. Anoxemia was generally induced by injecting 0.9 per cent sodium chloride solution at body temperature into one or both pleural cavities to collapse the underlying lung. Sometimes sufficient depression of respiration was obtained by the anesthetic. Samples of arterial and portal vein blood were obtained for analysis of oxygen content. The results indicated that intragastric oxygen conferred no definite benefit on animals exposed to it. When blood samples were analyzed at various intervals, from 2 to 20 minutes after intragastric insufflation started, no correlation could be found between the duration of treatment of oxygen and improvement in hemoglobin saturation. Under the conditions of these experiments, therefore, the use of intragastric oxygen appears to be without benefit. (Coxon, R. V.: *Effect of Intragastric Oxygen on Oxygenation of Arterial and Portal Blood in Hypoxic Animals*, *Lancet* 1: 1315 (June 18) 1960.)