Literature Briefs

JOHN W. PENDER, M.D., Editor

Briefs were submitted by Drs. Vagn Askrog (Denmark), C. M. Ballinger, Norman Bergman, Peter P. Bosomworth, M. T. Clarke, H. S. Davis, Deryck Duncalf, J. E. Eckenhoff, Martin Helrich ,G. Hohmann (Germany), J. J. Jacoby, H. Landesman, F. C. McPartland, W. H. Mannheimer, Alan Paterson, Albert O'Neil, R. E. Ponath, Alan D. Randall, H. S. Roe, Norman Rosenbaum, P. H. Sechzer, and E. A. Talmage. Briefs appearing elsewhere in this issue are a part of this column. Abstracts of Russian and Japanese literature were obtained from Exerpta Medica Foundation.

HYPERBARIC OXYGEN Diffuse myocardial infarction was produced in dogs by injection of microspheres into the coronary arteries. Changes in cardiac rhythm, rate, output, and blood pressure were noted. Animals who breathed air or oxygen had a mortality of about 75 per cent. Animals who breathed oxygen at three atmospheres pressure had a mortality of 35 per cent. This demonstrates the ability of high pressure oxygen to counteract the hypoxia produced by ischemia. (*Jacobson, J. H., II, and others: Hyperbaric Oxygenation, Arch. Surg. 89: 905 (Nov.) 1964.*)

HYPERBARIC OXYGEN Hyperbaric oxygen as contrasted with intermittent positive-pressure ventilation with oxygen has been compared for the resuscitation of mature fetal rabbits, which had been asphyxiated beyond the last gasp after delivery by cesarean section under controlled conditions. Ten of 12 rabbits treated by intermittent positive-pressure ventilation recovered, whereas none of 17 who received hyperbaric oxygenation survived. (*Cross, K. W., and others: Hyperbaric Oxygen and Intermittent Positive-Pressure Ventilation in Resuscitation of Asphyxiated Newborn Rabbits, Lancet 2: 560 (Sept. 12) 1964.*)

HYPEROXIA Mice experiments have implicated lipid peroxidation in hyperoxic hemolysis. Current studies in human beings extend these observations and demonstrate the probability that lipid peroxidation may account for lysis of erythrocytes in susceptible humans. Additional data support the concept that the formed lipid peroxides may inhibit other metabolic systems, particularly glycolysis. This would account for other manifestations of oxygen toxicity such as central nervous system changes. (*Menzel, C. E., and others: Mechanisms of In Vivo Hemolysis Induced by Hyperoxia, Aerospace Med. 35: 857 (Sep.) 1964.*)

HYPERBARIC OXYGENATION A variety of tissues from mice have been exposed to oxygen and atmospheric air at ambient and hyperbaric pressures. The epithelium of pulmonary alveoli and renal tubules was more readily damaged by hyperbaric oxygen than was that from bronchi, epididymis and prostate. The type and degree of damage varied, but it appeared that oxygen tension and not the mechanical effect of pressure was the operative factor. (*Heppleston, A. G., and Simnett, J. D.: Tissue Reaction to Hyperbaric Oxygen, Lancet 1: 1135 (May 3) 1964.*)

DYSBARISM A survey of the effects of hyperbaric exposure on 62 medical personnel exposed to 1,516 compressions and decompressions revealed no case of permanent ill effect. Pain in the ears or sinuses was the most common symptom but could often be ameliorated or avoided by the Valsalva technique of forced insufflation with the nostrils occluded. The most serious symptoms encountered were three episodes of transient homonymous hemianopsia. The classic symptoms of decompression sickness: extremity pains (the "bends), pulmonary or substernal distress (the "chokes"), and skin dysesthesias occurred only rarely, and were so mild or fleeting as to require no treatment. A further reduction in symptoms, without increase in decompression time, may be obtained by the inhalation of 100 per cent oxygen dur-