Literature Briefs

C. Philip Larson, Jr., M.D., Editor

Literature Briefs were submitted by Drs. A. Boutros, D. R. Bucchel, H. Cascorbi, R. B. Clark, M. I. Gold, J. Jacoby, L. M. Kitahata, W. Mannheimer, F. C. McPartland, D. H. Morrow, J. W. Pender, L. J. Saidman and A. D. Sessler.

Circulation

HALOTHANE ARRHYTHMIAS The effects of halothane upon several different types of myocardial fibers were studied to gain information about the cause of halothane-induced arrhythmias. Two per cent halothane had very little effect upon either rabbit atrial or sheep ventricular fibers except for a slight prolongation of repolarization time in the rabbit and decreased duration of action potential and shortening of the refractory period in the sheep. In contrast, the effects upon conduction tissue (sheep Purkinje fibers) were considerable, with one per cent halothane causing decreased overshoot and duration of the action potential and increase of the resting potential and conduction time. Halothane, especially in the presence of sympathomimetic amines, may produce arrhythmias because of decreased conduction velocity, shortening of the refractory period, and a pronounced disparity between the refractory periods of Purkinje and ventricular fibers. (*Hauswirth, O.: Effects of Halothane on Single Atrial, Ventricular, and Purkinje Fibers, Circ. Res. 24: 745 (May) 1969.*)

PULMONARY CIRCULATION The effects of breathing 10 per cent carbon dioxide upon the pulmonary circulation were studied in healthy eucapnic patients and convalescing hypercapnic patients. Control measurements in the eucapnic and hypercapnic groups, respectively, were: Pa_{CO_2} 38 and 55 mm Hg; mean pulmonary artery pressure (MPAP) 15 and 33 mm Hg; pulmonary vascular resistance (PVR) 1.2 and 4.8 mm Hg/l/min; Pa_{O_2} 77 and 46 mm Hg. Breathing 10 per cent CO_2 abolished hypoxemia in the hypercarbic patients and elevated MPAP and PVR in both groups. Reduction of hydrogen-ion concentration with sodium bicarbonate did not decrease MPAP. Breathing oxygen had little effect in either group. The evidence suggests the CO_2 acts on pulmonary arterioles and capillaries that are exposed to alveolar gases to increase the pulmonary vascular impedance. (*Kilburn, K. H.: Effects of Breathing 10 Per Cent Carbon Dioxide on the Pulmonary Circulation of Human Subjects, Circulation 39: 639 (May) 1969.*)

PULMONARY EMBOLI The respiratory response to localized embolization with thrombi and 42-μ glass beads was determined in closed-chest, self-respiring, anesthetized dogs. After each of 24 embolizations with thrombi, the average change in respiratory rate was −1 per cent. Following each of 14 embolizations with 42-μ glass beads, the average respiratory rate change was +9.4 per cent. The primary difference between thrombi and glass-bead embolizations was a progressive desaturation of arterial blood and a steady rise in arterial P_{CO_2} with glass-bead embolizations, whereas no such changes were noted following embolizations with thrombi. (*Daily, P. O., and Moulder, P. V.: Respiratory Response to Lobar Pulmonary Embolism in Dogs, Surgery 65: 958 (June) 1969.*)

PULMONARY EMBOLISM The natural rate of resolution of pulmonary embolism was defined in 15 patients. The patients studied exhibited definitive angiographic evidence of bilateral embolism and were treated with heparin and/or venous ligation. Sequential studies showed only minimal angiographic and hemodynamic signs of resolution at seven days. At