Insufficiency, J. A. M. A. 183: 161 (Jan. 19) 1963.)

CYANOSIS The most important factor accounting for cyanosis in primary pulmonary hypertension is uneven distribution of pulmonary capillary flow, analogous to the mechanism of arterial oxygen desaturation in multiple pulmonary emboli. Obstructive lesions of the pulmonary arteries and arterioles and small pulmonary infarcts are common in primary pulmonary hypertension. Thus, imbalance between ventilation and perfusion may be produced by mechanical block in the circulation and diversion of blood to a nonobstructed portion of the pulmonary circulation. Adequate arterialization requires that ventilation of the nonobstructed segment of the lung be increased sufficiently to maintain normal ventilation-perfusion relationships. Although total ventilation may be normal or increased, segments of lung with relative hyperventilation may result, thus allowing incompletely oxygenated blood to reach the arterial circulation. In pulmonary embolism, a decreased diffusing capacity of the lungs has been demonstrated. *(Sleeper, J. C., and others: Primary Pulmonary Hypertension, Circulation 26: 1358 (Dec.) 1962.)*

RED-CELL CHOLINESTERASE Several cases have been reported dealing with familial reduction in plasma cholinesterase. Attention in these instances was called to this defect primarily because of prolonged apnea following the administration of succinylcholine during surgery. Apart from this biochemical sensitivity to succinylcholine, these patients were healthy and free of liver disease and had normal levels of red-cell cholinesterase. The present case is presented to demonstrate the occurrence of a depletion of red-cell cholinesterase in the face of normal plasma cholinesterase activity. *(Johns, R. J.: Familial Reduction in Red-Cell Cholesterase, New Engl. J. Med. 267: 1344 (Dec. 27) 1962.)*

CAROTID BODIES Stimulation of the carotid bodies in dogs caused an increase in respiratory minute volume, and an increase, decrease or no change in heart rate. When pulmonary ventilation was controlled, slowing of the heart invariably occurred. The efferent pathway for this reflex bradycardia lies both in the vagus and sympathetic nerves. In those dogs spontaneously breathing room air or 30 per cent oxygen in nitrogen, stimulation of the carotid bodies caused vasoconstriction followed by vasodilation in the intact upper and lower limbs and in muscle. In skin and in the splanchnic vascular bed, vasoconstrictor responses predominated. In dogs artificially ventilated, vasoconstrictor responses invariably occurred in the intact limb, muscle, skin and in the splanchnic vascular bed due to an increase in sympathetic tone. The primary cardiovascular reflex effects of stimulation of the carotid bodies by hypoxic blood are bradycardia and peripheral vasoconstriction. These responses may be partly or wholly masked by mechanisms arising secondarily as a result of the concomitant reflex increase in respiratory minute volume. *(Daly, M. B., and Scott, M. J.: Analysis of the Primary Cardiovascular Reflex Effects of Stimulation of the Carotid Body Chemoreceptors in the Dog, J. Physiol. 162: 555 (Aug.) 1962.)*

CARDIAC OUTPUT Effects of variation of carbon dioxide tension on cardiac output during hyperventilation were studied in human subjects. During hypocapneic hyperventilation, in which subjects voluntarily hyperventilated with room air, a significant increase in cardiac output occurred. During isocapneic hyperventilation where sufficient carbon dioxide was added to maintain carbon dioxide tension near normal, a small increase in cardiac output occurred. During the first two minutes of hypercapneic hyperventilation, where 8.4 per cent carbon dioxide was inhaled, no increase in cardiac output occurred. The response of cardiac output to hyperventilation thus depends on the carbon dioxide content of the inspired mixture. *(McGregor, M., Donevan, R. E., and Anderson, N. M.: Influence of Carbon Dioxide and Hyperventilation on Cardiac Output in Man, J. Appl. Physiol. 17: 936 (Nov.) 1962.)*

VENTRICULAR ARRYTHMIA Intact vagus nerves are necessary for consistent induction of arrhythmia by adrenaline in anesthetized dogs although sinus bradycardia does