CARDIAC PACEMAKING Cardiac pacemaking can raise cardiac output associated with a fixed slow rate and improve splanchnic, renal and coronary blood flow. In 56 patients an implanted coil located subcutaneously which is inductively coupled to a coil overlying the skin has been used. This arrangement has the advantage of allowing rate and applied power to be varied and requiring minimal implanted material. (*Taylor, A. B.: Experience with Cardiac Pacemaking, Brit. Med. J. 2: 543 (Sept.) 1966.*)

CARDIAC OUTPUT The effect of changes in arterial *p*H and P_{CO_2} on cardiac output during halothane anesthesia was studied in normothermic dogs. Anesthesia was induced and maintained with halothane while cardiac output was measured either by dye dilution techniques or by means of a chronically implanted electromagnetic square wave flowmeter. The P_{CO_2} was maintained steady by means of a volume limited respirator while the animal was paralyzed with succinylcholine. Changes in arterial *p*H were produced by subjecting the animals to periodic bursts of hypoxia and then allowing the arterial P_{CO_2} to return to normal. After each reduction in *p*H the cardiac output was measured. Changes in P_{CO_2} were produced by adding carbon dioxide to the inspired mixture. Cardiac output was depressed linearly by linear falls in *p*H and increased parabolically by changes in arterial P_{CO_2} after allowing for those changes due to *p*H alterations. (*Tomlin, P. J.: Quantitative Effects of Changes in Arterial pH and P_{CO_2} upon Cardiac Output during Halothane Anesthesia in Dogs, J. Physiol. 185: 66P (July) 1966.*)

DIGITALIS The effects of ouabain upon the contractile properties of the intact, nonfailing human heart were studied by measuring myocardial force-velocity relations prior to and after 0.01 mg./kg. of the glycoside. Ouabain always augmented contractility but no consistent changes in cardiac output were observed. Cardiac output is controlled by many factors of which contractility is only one. Therefore in the absence of heart failure, an increase in contractility produced by a cardiac glycoside, may not increase cardiac output. However, when cardiac output is decreased as a result of decreased contractility (heart failure), digitalis will elevate output. The finding that glycosides fail to elevate cardiac output in normal subjects does not preclude a positive inotropic action. (*Sonnenblick, E. H., and others: Studies on Digitalis, Circulation 34: 532 (Sept.) 1966.*)

PULMONARY CIRCULATION A method for the measurement of the ratio of alveolar ventilation to perfusion in individual lung zones during normal steady state conditions is described in four subjects. ^{133}Xenon is used as the tracer gas. Regional count rates were recorded during a 5-minute period of constant intravenous infusion of this substance dissolved in saline and during a similar period of rebreathing into a closed circuit. For practical purposes a steady state is achieved within these time limits in normal subjects. Similar patterns of regional ventilation to perfusion ratios were recorded during quiet tidal breathing in the subjects seated upright. It was possible to correct these data to allow for the influence of re-inspired dead space gas and to compute regional ventilation to perfusion ratios where ventilation represented total gas-exchange ventilation. Regional respiratory gas concentrations were calculated from such ventilation perfusion measurements and appeared to show less regional variation than previously reported. Infusion of ^{133}xenon delivers a higher dose of radiation than do multiple single injections. This would probably prohibit repetitive infusion experiments in normal subjects for solely experimental purposes. (*Anthonisen, N. R., Dolovich, M. B., and Bates, D. V.: Steady State Measurement of Regional Ventilation to Perfusion Ratios in Normal Man, J. Clin. Invest. 45: 1349 (Aug.) 1966.*)

CEREBRAL CIRCULATION The relation between cerebral blood flow and blood CO_2 tension after rapid alterations in inspired CO_2 were studied in 13 normal male volunteers. With progressive stepwise increases and decreases in arterial CO_2 tension above the control level, jugular venous CO_2 tension correlated more exactly with cerebral blood flow

than did arterial CO_2 tension. In 16 of 19 experiments arterial CO_2 tension described a counterclockwise loop when plotted against the corresponding values for cerebral blood flow. The distinct hysteresis between arterial CO_2 tension and cerebral blood flow was evident in only one of ten experiments during stepwise reductions and return to control in the hypocapnic range. On ten occasions cerebral blood flow and arterial CO_2 tension were measured as rapidly as possible after a single breath of 28.5 per cent CO_2. These studies provided further evidence that a finite interval is required for the cerebral vessels to respond to a given elevation in arterial CO_2 tension. It is suggested that the tissue tension of carbon dioxide may be the effective regulator of cerebrovascular resistance rather than the intra-arterial tension of this gas. Thus, measurements restricted to arterial CO_2 tension during ascent of arterial CO_2 tension may overestimate and, during descent of this tension, underestimate the actual determinant of cerebrovascular resistance when these measurements are made in rapidly or continuously changing state. (*Shapiro, W., Wasserman, A. J., and Patterson, J. L., Jr.: Mechanism and Pattern of Human Cerebrovascular Regulation after Rapid Changes in Blood CO_2 Tension, J. Clin. Invest. 45: 913 (June) 1966.*)

CHEMOCEPTORS AND SLEEP During wakefulness or sleep the arterial pressure did not change in cats whose baroceptors were intact but aortic nerves were severed and carotid body chemoceptors subsequently deafferented. During deep sleep, however, there were exaggerated falls in pressure. Subsequent baroceptive denervation did not modify the hypotensive effect. Although current opinion is that chemoceptors act only in emergencies such as acute anoxia or shock, these data suggest that chemoceptors have a role in circulatory homeostasis. They may prevent hypotension or cerebral anoxia during deep sleep. (*Guazzi, M., Baccelli, G., and Zanchetti, A.: Carotid Body Chemoceptors: Physiological Role in Buffering Fall in Blood Pressure during Sleep, Science 153: 206 (July) 1966.*)

CAROTID SINUS BUFFERING The response of the systemic arterial blood pressure to changes in the transmural blood pressure in the carotid sinus area were observed in 24 conscious male subjects. It appears that in man the sensitivity of the carotid sinus buffering reflexes is maximal at arterial blood pressures below 60 to 70 mm. of mercury while it becomes minimal at pressures above normal. The carotid baroreceptors in man apparently afford protection against arterial hypotension while their buffering capacity against hypertension is almost negligible. (*Thron, H. L., Brechmann, W., and Eckert, P.: The Dependence of Arterial Blood Pressure on Transmural Pressure in the Area of the Carotid Sinus in Wake Man, Klin. Wschr. 44: 824 (July) 1966.*)

ARTERIOLAR AUTOMATICITY Direct microscopic observations of contractile activity of terminal arterioles were made in the wings of unanesthetized bats. The spontaneous activity varied widely from moment to moment and was unrelated to activities of neighboring terminal arterioles, even those from the same parent vessel. Denervation and local anesthesia did not effect spontaneous activity or vessel size. An increase in intraluminal pressure by retrograde perfusion caused a marked increase in the contractile activity of terminal arterioles in both the normal and denervated vessels. (*Wiedman, M. B.: Contractile Activity of Arterioles in the Bat Wing during Intraluminal Pressure Changes, Circ. Res. 19: 559 (Sept.) 1966.*)

ABSTRACTOR'S NOTE: This is further evidence for inherent automaticity of vascular smooth muscle. Local factors such as intraluminal pressure changes, metabolic needs, etc., produce local changes in blood flow—at least in the rat mesoappendix, in rat cremasteric muscles and in bat wings.

CIRCULATORY CONTROL An anesthetist has under his control many variables that increase wound bleeding. Among these are (a) hypercapnia, which stimulates catecholamine release, raises blood pressure, and increases cardiac output; (b) hypoxia, which leads to vascular dilatation, chemoreceptor