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

stimulation, and increased cardiac output; (c) respiratory obstruction, which increases intrathoracic pressure and elevates venous pressure; (d) anesthetics such as cyclopropane and ether, which increase the blood levels of catecholamines; (e) deep anesthesia, which causes myocardial depression and elevated venous pressure; (f) improper body position, which, if the operative site is low, increases blood hydrostatic pressure; and (g) inadequate analgesia with the central perception of pain causing peripheral circulatory stimulation. An anesthetic should be administered with knowledge of these potentials and attempts made to control each in order to minimize bleeding. Automatically, there are three sets of veins draining blood from the brain: the internal jugular veins, the emissary veins, and the vertebral plexus. It seems reasonable to believe that the jugular veins are not always the principal avenue of escape of blood from the brain. The vertebral plexus may contribute significantly and may be important in providing the means for circulatory compensation with change of posture and during deliberate hypotension in the head-up position. This provides a vascular channel of considerable capacity that in the head-up position continually keeps vascular resistance low and facilitates the flow of blood through the brain. (*Eckenhoff, J. E.: Circulatory Control in the Surgical Patient, Ann. Roy. Coll. Surg. Eng. 39: 67 (Aug.) 1966.*)

PROLONGED BED REST Five healthy adult males were studied during a 30 day bed rest experiment. Repeated tilt table tests were conducted before and after the period of recumbancy. Radioisotope blood volume determinations were made prior to, during and after the study. Results indicate that cardiovascular deconditioning occurs after 30 days of bed rest and that two weeks of ambulation is required for reconditioning. Also, blood volume decreases during the first few days of bed rest and returns toward normal at the end of the 30 day rest period. (*Vogt, F. B., and others: Tilt Table Response and Blood Volume Changes Associated with Thirty Days of Recumbency: Aerospace Med. 37: 771 (Aug.) 1966.*)

INSULIN AND PHENYLEPHERINE Cardiovascular and metabolic responses to l-phenylepherine and dl-isoproterenol were studied in acutely pancreatectomized dogs. dl-phenylepherine, in a dosage of 10 micrograms per kilogram, was significantly less effective in increasing myocardial contractile force, systolic pressure and blood glucose levels and in decreasing the heart rate in the study dogs than in the controls. Infusion of 2 micrograms per kilogram of insulin returned the responses toward normal. Recent evidence (Patel, *et al.*, J. Pharmacol. 149: 199, 1965) indicates that part of the cardiovascular actions of phenylepherine is due to release of endogenous catechol amines from intracellular stores. It is postulated that insulin is necessary for release of these stores probably by altering the membrane permeability to phenylepherine. Pancreatectomy did not alter the response to dl-isoproterenol. (*Goldberg, E., and Rosenblum, F.: Reduction in Cardiovascular and Metabolic Responses to Phenylepherine in Acutely Pancreatectomized Dogs, Amer. Heart J. 72: 482 (Oct.) 1966.*)

SICKLING Precision scale models of sickle-cell hemoglobin molecules indicate that the genetic substitution of valine for glutamic acid at the sixth position in the two beta chains allows an intramolecular hydrophobic bond to form. This changes the conformation in such a way as to allow molecular stacking. Optical rotatory dispersion studies and the results of subjection of Hb S solution to temperature change and to propane are consistent with the presence of such a bond. Examination of sickled erythrocytes in a magnetic field and in polarized light indicates that the Hb S molecules are aligned *in situ*. Filaments interpreted are hollow cables of six Hb S microfilaments have been demonstrated by electron microscopy. (*Murayama, M.: Molecular Mechanism of Red Cell "Sickling," Science 153: 145 (July) 1966.*)

BANK BLOOD Blood conserved in ACD solution was subjected to gas analysis and measurements of *p*H and oxyhemoglobin dissociation. The values were correlated with those obtained on fresh blood and blood con-