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