Arterial standard bicarbonate levels increased from 23 to 28 mm/l during exposure to 60 mm Hg C_{O_2} and to near 31 mm/l during exposure to 90 mm Hg CO_2. These data indicate at least as good, and perhaps better, pH stability in an hypobaric atmosphere as has been found at sea-level pressures. (*Popelko, W. E.: Adaptation of Dogs to 60 and 90 mm Hg CO_2 at a Total Pressure of 260 mm Hg, Aerospace Med. 39: 1294 (Dec.) 1968.*)

POSTVAGAL TACHYCARDIA Vagal stimulation causes sinus arrest or atrioventricular block and fall in blood pressure. After the stimulation, the sinus rate increases to above the control value. This postvagal tachycardia could result from many mechanisms, but a direct release of catecholamines or similar substances by the vagus has been suggested, since vagal stimulation or administration of acetylcholine in atropinized preparations produces tachycardia and liberates an epinephrine-like substance. In anesthetized dogs, the right vagus was crushed in the neck and the peripheral end stimulated. Termination of stimulation consistently was followed by transient sinus tachycardia, which persisted despite maintenance of normal blood pressure, thereby eliminating reflex hypotensive mechanisms. To stimulate the vagus reflexly, the carotid sinus was compressed. Termination of this reflex vagal stimulation was followed by tachycardia. Bilateral vagotomy abolished this response. Tetraethylammonium applied to the sinus node directly blocked the vagus induced tachycardia. Postvagal tachycardia was also abolished by prior administration of reserpine and restored after norepinephrine administration. Postvagal tachycardia results from the excitation of cholinergic parasympathetic fibers, leading to liberation of catecholamines, possibly from chromaffin cells. (*Copen, D. L., Cirillo, D. P., and Vassalle, M.: Tachycardia following Vagal Stimulation, Amer. J. Physiol. 215: 696 (Sept.) 1968.*)

EEG AND ALKALOSIS The EEG was monitored continuously during all cardiac procedures, and blood gases and pH were examined at frequent intervals. In a retrotspective study of more than 1,600 cases, the EEG did not prove to be as sensitive an indicator of alkalosis as has been suggested in the literature. However, an interesting case was reported in detail: a sudden drop in P_{CO_2} from 36 to 10 mm. Hg with simultaneous appearance of "spikes" in the EEG tracing. Although alkalosis was corrected promptly, the EEG changes persisted for several days postoperatively. These findings are similar to those reported by A. Harden and B. M. Ashton (EEG Clin. Neurophysiol. 22: 128 (Feb.) 1967.) (*Passelecq, J., Arfel, G., and Dubost, C.: EEG Manifestations of Alkalosis, Anaesth. Analg. 24: 535 (Sept.) 1967.*)

MONITORING A system of measurement and display was developed to care for patients acutely ill with cardiac disease or following open-heart surgery. It is based on the use of sensing systems designed for minimal disturbances of the patient or his nurses. Information such as vascular pressures, EKG, respiration and ventilatory settings is processed by digital computer and displayed as numerical or graphic data at the bedside. Therapeutic management is eased and improved. The ventilatory measurements provide constant assurance that the patient is receiving the intended tidal and minute volumes. (*Osborn, J. J., and others: Patient Monitoring of Acutely Ill Patients by Digital Computer, Surgery 64: 1057 (Dec.) 1968.*)

HALOTHANE Cardiovascular responses to induction of anesthesia with halothane–oxygen, as well as to changes in alveolar concentration from one steady-state level to another, were studied in eight subjects. Heart rate, arterial and right atrial pressures, stroke volume, cardiac output, left ventricular minute work, and total peripheral resistance were recorded beat-to-beat. The last four variables were calculated by a ballistocardiograph–analogue computer system. Induction of anesthesia increased significantly all variables except total peripheral resistance and arterial and right atrial pressures. The last two are the variables conventionally measured during anesthesia and surgery. The response to changing concentration was delayed significantly, and decreased in magnitude late, rather than