with a wide variety of heart diseases. The study series consisted of consecutive patients in whom cardiac outputs were determined by the Fick principle from oxygen consumption values measured during cardiac catheterization. The results provided estimates of $VO_2$/BSA based on age, sex and heart rate. Similar analyses demonstrated that A-$VO_2$ difference and arterial $O_2$ saturation were not relevant to this estimation. Knowledge of sex, age and heart rate of a subject permits estimation of $VO_2$/BSA from tables or estimation equations. (*LaFarge, C. G., and Miettinen, O. S.: The Estimation of Oxygen Consumption, Cardiovas. Res. 4: 23 (Jan.) 1970.*)

**PROPHYLACTIC DIGITALIS** The change in left ventricular output in response to rapid infusion of Tyrode's solution was less in awake dogs which had just been digitalized than in dogs which had not received digitalis. Peak cardiac output, increase over resting cardiac output, and change in output per unit change in left atrial pressure were less in the digitalized animals. The increased left atrial pressure due to the infusion caused maximal increases of both stroke volume and heart rate, to account for the increased cardiac output in the nondigitalized animals. In the dogs given digitalis, the limitation in maximum output was due to a slower maximum heart rate. (*Horwitz, L. D., and Bishop, V. S.: Effects of Ouabain on Left Ventricular Performance in Conscious Dogs, Cardiovas. Res. 4: 31 (Jan.) 1970.*)

**CEREBRAL ACIDOSIS** Bilateral craniectomy was performed in 18 beagle dogs anesthetized with pentobarbital. The right lateral ventricle was cannulated and CSF pressure monitored. Intracranial pressure was varied by inflating a balloon catheter placed in the contralateral epidural space. Direct arterial blood pressure and arterial blood *p*H were monitored by cannulation of a femoral artery. Surface *p*H of the brain (as an indirect measure of cerebral perfusion) was monitored using a *p*H glass electrode placed on the brain surface after incision of the dura. Injection of 3, 6, and 9 ml of saline solution into the balloon catheter resulted in average increases in intracranial pressure of 30, 75 to 90, and 150 mm Hg, respectively. Cerebral surface *p*H showed a mean decrease of 0.332 units with 3 ml in the balloon, a further decrease of 0.371 units with 6 ml, and a still further decrease of 0.260 units with 9 ml. Corresponding decreases in arterial blood *p*H were 0.020, 0.030, and 0.043 units. Total mean decrease in cerebral surface *p*H was 0.839 units below the final arterial blood *p*H, the former always preceding the latter. An arterial pressor response occurred in every dog but was always preceded by cerebral-surface acidosis. It is concluded that arterial hypertension in response to increased intracranial pressure is due to cerebral ischemia. (*Berman, I. R., and Rogers, L. A.: Cerebral Acidosis Following Increased Intracranial Pressure, Surg. Gynec. Obstet. 130: 483 (March) 1970.*)

**CAROTID BARORECEPTORS** The effects of changes in baroreceptor activity on the wall tension of the lateral saphenous vein of one hind limb, the splenic veins and capsule, and the resistance vessels of the other hind limb were measured in vagotomized dogs anesthetized with thiopental and chloralose. Three methods were used to alter pressure in one or both carotid sinuses: 1) both carotid sinuses were isolated vascularly and filled with oxygenated Krebs-Ringer bicarbonate solution (*p*H 7.4) from a reservoir in which the pressure could be altered easily; 2) one sinus was denervated and the contralateral sinus perfused with arterial blood at different flow rates; 3) one sinus was denervated and the innervated sinus was perfused with arterial blood at constant flow, the pressure being altered by changing the outflow resistance. The saphenous veins showed no consistent responses to changes in baroreceptor activity. A decrease in carotid sinus pressure from 180 to 100 mm Hg caused increases in venous pressure in the isovolemic spleen and in the iliac artery perfusion pressure. The same results were obtained with electrical stimulation of the carotid sinus nerve. Whereas the peak responses of the limb resistance vessels corresponded to an increase in lumbar sympathetic nerve traffic of 6 to 10 cycles/sec, the maximal splenic responses were equivalent to an increase in splenic nerve traffic of 1 to 4 cycles/sec. These