CNS-INDUCED CARDIOMYOPATHY A 49-year-old white woman with severe hypertension and bleeding from a Berry aneurysm developed acute pulmonary edema and EKG changes which progressed from initial S-T segment changes suggestive of subendocardial ischemia to a full-blown picture of an inferior-wall myocardial infarction with Q waves in II, III and AVF. Multiple foci of myocytolysis, loss of myocardial cells and loss of supporting stroma were noted at postmortem examination but the coronary arteries were normal and, on gross section, the heart appeared normal. The extensive myocardial necrosis found microscopically was unrelated to any vascular pattern and similar to that induced by large doses of exogenous catecholamines. Massive sympathetic discharge secondary to the subarachnoid bleeding may interfere with the microcirculation of the cardiac musculature, producing a neurohumoral myocarditis and congestive heart failure. (*Hammermeister, K. E., and Reichenbach, D. D.: QRS Changes, Pulmonary Edema, and Myocardial Necrosis Associated with Subarachnoid Hemorrhage, Amer. Heart J. 78: 94 (July) 1969.*) ABSTRACTER'S COMMENT: The EKG changes secondary to subarachnoid hemorrhage range from minor ST-T changes and Q-T prolongation to those of a transmural myocardial infarction, indicating a broad spectrum of myocardial damage. The anesthesiologist must use fluids judiciously and be alert for early signs of failure when confronted with these patients.

CARDIOVASCULAR STRESS Sustained high cardiac output, hyperventilation, and hypoxemia are among the severe stresses imposed upon circulatory and respiratory systems by sepsis, hemorrhage and multiple trauma. A group of 103 cases of hemorrhage and trauma, sepsis, and burns had an incidence of fatal cardiovascular and respiratory complications 15 times greater than that in a large population of general surgical patients. The cardiac index in the postshock period and in response to sepsis was significantly greater than the value in uneventful convalescence. Those who died had excess lactate values four to five times greater than the values in patients who recovered uneventfully. Tissue hypoxia, due to poor tissue perfusion and the Bohr effect, was probably the major cause of excess blood lactate. A high incidence of interstitial pneumonitis was associated with increased physiologic deadspace and pulmonary arteriovenous shunting. Artificial respiration as a means of reducing circulatory demand and improving oxygenation corrected metabolic deficits without substantial increase in blood flow. (*Rubin, J. W., and Clowes, G. H. A., Jr.: Cardiovascular Stresses in Surgery, Surg. Clin. N. Amer. 49: 489 (June) 1969.*)

SPINAL-CORD BLOOD FLOW The responses of spinal-cord blood flow (SCBF) to changes in arterial carbon dioxide tension (Pa_{CO_2}) were studied in ten goats anesthetized with nitrous oxide–oxygen. Pulmonary ventilation ($\dot{V}_E$) was controlled by a ventilator and end-tidal P_{CO_2} was monitored continuously with an infrared CO_2 analyzer and varied by altering $\dot{V}_E$. SCBF was calculated from the clearance curve of ^{133}xenon following the injection of 2 μl in saline solution into the spinal cord. Measurements of SCBF were reproducible at any given locus, but there was some variation from site to site. Mean SCBF of all animals was approximately 16.2 ml/100 g/min at a Pa_{CO_2} of 38.2 torr. SCBF decreased from 17.4 at a Pa_{CO_2} of 39.3 torr to 8.5 ml/100 g/min at a Pa_{CO_2} of 16.8 torr. Increase of Pa_{CO_2} from 37 to 59 torr increased SCBF from 13 to 19.6 ml/100 g/min. The responses of SCBF to changes in Pa_{CO_2} fell into groups of high and low sensitivity. Six sites had a mean sensitivity of 0.617 ml/100 g/min/torr. (*Smith, A. L., Pender, J. W., and Alexander, S. C.: Effects of P_{CO_2} on Spinal Cord Blood Flow, Amer. J. Physiol. 216: 1158 (May) 1969.*)

CAVAL LIGATION Serial hemodynamic measurements were made before and after inferior vena caval ligation in 13 anesthetized, artificially-ventilated dogs. Within five minutes after ligation, mean systemic arterial pressure decreased 10 per cent, cardiac output decreased 20 per cent, and inferior vena caval pressure increased from 2.6 to 28.2 mm Hg. Over the next hour all values returned to normal except inferior vena caval pressure, which remained elevated for as long as seven days.