tion and Papaverine, Ann. Surg. 163: 771 (May) 1966.)

VENOUS TONE The effects of various anesthetics, such as halothane, ether, methoxyfluorane, trichlorethylene, cyclopropane, thiopental and lidocaine, on the peripheral circulatory system were compared during passive tilting in human subjects. The blood content of the toe and the venous pressure at the ankle were measured simultaneously by means of a photoconductive plethysmography and an electromanometer. Generally, the blood content of the toe and the venous pressure at the ankle were increased more rapidly under general and epidural anesthesia with head-up tilting than under usual conditions. The change in the venous pressure preceded the change in the blood content of the toe. Furthermore, the administration of a muscle relaxant drug had no significant effect on the peripheral circulation of the lower extremities. These findings suggest that increase in venous distensibility occurred when the nervous system was blocked. (Hayashi, K.: Clinical Studies on the Effect of Various Anesthetics on the Vascular Volume of the Human Toe, Evaluated by a Photoconductive Plethysmograph (Japanese), Med. J. Hiroshima Univ. 13: 235, 1965.)

HEMODYNAMICS OF HEMORRHAGE

Hemodynamic and metabolic effects of hemorrhage were studied in 11 unanesthetized human volunteers. Hemorrhage of 15-20 per cent of the blood volume in 35 minutes produced a significant reduction in splanchnic blood volume, indocvanine green dve clearance and hematocrit. About one-half of the blood loss was contributed by the splanchnic viscera as the splanchnic blood volume was reduced by 40 per cent while the central blood volume was reduced by only 10 per cent. This hemorrhage, however, produced no significant change in splanchnic blood flow, oxygen consumption, A-V excess lactate production or in splanchnic vascular resistance. Cardiac output, heart rate, mean arterial blood pressure and arterial excess lactate were un-These results suggest that the splanchuic circulation functions as an important blood reservoir which can be preferentially depleted of blood in response to hemorrhage. (Price, II. L., and others: Hemodynamic and Metabolic Effects of Hemorthage in Man with Particular Reference to the Splanchnic Circulation, Circ. Res. 28: 469 (May) 1966.)

CARDIAC SHOCK The responses to vasopressors in patients with acute myocardial infarction with shock were evaluated. Control of measurements were cardiac output 2.2 liters/ o minute, mean arterial pressure (MAP) 53 mm. 5 of mercury, systemic vascular resistance (SVR) 3 27 mm. of mercury/liter/minute. amine was given to seven patients. Cardiac 8 output fell in four (19-28 per cent) and did ₹ not change in three. SVR was increased in was given to 11 patients (7 of whom had received methoxamine). Cardiac output was increased in all except one $(-8 \text{ per cent to} \bigcirc$ +57 per cent), MAP increased in all (+3 per cent to 175 per cent). Low viscosity dextran given to two patients resulted in a 145 per cent and 54 per cent increase in cardiac output. Norepinephrine is felt to be preferable to a pure vasoconstrictor (methoxamine) in patients with cardiogenic shock. (Gunnar, R. M.: Myocardial Infarction with Shock, Circulation 33: 753 (May) 1966.)

HEMORRHAGIC SHOCK One hundred heparinized dogs, in groups of four, were bled into reservoirs until a mean systolic arterial? blood pressure of 40 mm, of mercury was obtained. After 75 minutes at this blood pressure, treatment consisting of: (1) blood replacement (500-900 ml.), (2) infusion of of dextran (500 ml., or (3) hydroxyethyl starcho (500 ml.) was instituted in three of each four dogs. The fourth dog received no treatment. Survival rates were: blood infusion, 72 per cent; dextran infusion, 44 per cent; hydroxy ethyl starch, 64 per cent; and no treatment, 20 per cent. The difference in survival between dextran and hydroxyethyl starch was not statistically significant. No adverse re-∞ actions to either dextran or hydroxyethyl starcle were observed. hydroxyethyl starch is at least as effective as volume expanded as is clinical dextran. (Bal-