Heart rate and blood gases remained essentially unchanged. Since the cardiopulmonary function of the patient considered for this procedure is often severely compromised, these acute consequences assume importance. (*Harsanyi, P., Rius-Gauiga, J., and Moser, K.: Acute Hemodynamic Consequences of Ligation of the Inferior Vena Cava, J. Thorac. Cardiov. Surg. 57: 442 (March) 1969.*)

SHOCK With cannulae in the superior vena cava and brachial artery, the following can be measured at the bedside of the patient in shock: systemic blood pressure, central venous pressure, blood volume, cardiac output, circulation time, blood chemistries and blood gases. Seven years of clinical experiments using such measurements has lead to the following conclusions. Measurements of cardiac output were not essential to the management of patients in septic shock, although they were a valuable indicator of cardiac responses to certain agents. Circulation time correlated inversely and well with cardiac output. Defects in blood volume were present in more than half of patients in septic shock without having been suspected from other observations. The relationship between blood volume and central venous pressure was far from linear. Management of blood volume by transfusing to the maximal tolerated central venous pressure was not a safe approach because of the induction of acute pulmonary insufficiency. Severe hyponatremia occurred in more than 50 per cent of patients in septic shock in the absence of dilution by administered fluids. The dose required for complete digitalization is lower during shock because of either increased myocardial irritability or decreased excretion. Some form of cardiac arrhythmia was found in every patient. Pulmonary insufficiency with some hypoxemia, lasting several hours, was found in all patients in shock. It is common, insidious, lethal and preventable. There are no rales, distended neck veins, elevated central venous pressure or hypervolemia, but the upper-body weight increases, as does the alveolar–arterial oxygen gradient. (*Frank, E. C., and Friedman, E. W.: The Management of Shock in Man, Surg. Clin. N. Amer. 49: 471 (June) 1969.*)

NUTRITION AND RISK Survival time of acutely-starved rats is not affected adversely by surgical trauma, and the body-weight-loss curve is unaltered by trauma. The linear relationship between rapid-bleeding tolerance and body weight in the rat is unaltered by either acute or chronic undernutrition. On the other hand, tourniquet shock, which induced no mortality in control rats, induced a 42 per cent mortality in rats acutely starved to 80 per cent of initial body weight and a 100 per cent mortality in rats acutely starved to 70 per cent of initial body weight. The lethality of tourniquet shock in the acutely-starved animal is due to rapidly developing hypovolemia of overwhelming magnitude. Surgical mortality is significantly increased in severely protein-malnourished rats at 55 per cent of initial body weight, but not at 65 per cent. Saline-infusion therapy increased survival in both acutely-starved and malnourished rats. (*Pareira, M. D.: Effect of Nutrition on Surgical Risk, Surg. Clin. N. Amer. 49: 694 (June) 1969.*)

SHOCK Changes in the distribution of cardiac output following injection of endotoxin were studied in monkeys. Measurements were made during acute shock and 24 hours after the injection. Acutely, there were decreases in systolic and diastolic arterial pressures, cardiac output, and total peripheral resistance, while the heart rate remained unchanged. Twenty-four hours following the onset of shock, results were essentially the same except that heart rate had decreased. In acutely shocked animals, cardiac output to heart, adrenals, gastrointestinal tract and liver was significantly increased, while cardiac output to the brain and spleen decreased. At 24 hours there were fewer significant changes and flow was not decreased significantly to any organ. At both times there were generalized vasodilation and a decrease in total peripheral resistance that were not overcome by reflex compensatory mechanisms. The vasodilation is particularly critical in preventing maintenance of adequate blood flow to the cerebral hemispheres. (*Wyler, F., and others: Induced Regional Circulatory Changes in the Unanesthetized Monkey, Circ. Res. 24: 777 (June) 1969.*)