may have the least disturbance in cerebral metabolism when they are paralyzed and ventilated, rather than otherwise.

SEPTIC SHOCK Hemodynamic measurements were made in 20 patients in septic shock. Peripheral A-V shunting, as evidenced by a high right atrial oxygen saturation, (79 to 87 per cent) was present in each of eight patients. Five patients with cardiac indices above 3.6/min/m² developed base deficits of more than 10 mEq/l, and eight patients had low oxygen consumptions (70 to 169 ml O_2/min). Eight patients with known heart disease had elevations in cerebral venous pressure and five patients with known hepatic disease had high cardiac outputs with low peripheral resistances. Mortality was 15 per cent in patients whose initial cardiac indices were greater than 2.5 l/min/m² and 83 per cent in patients with lower indices. Hemodynamics correlated poorly with clinical signs such as quality of pulses or temperature of limbs. Pulse pressure was more closely related to peripheral resistance than to cardiac output. (*Bell, H., and Thal, A.: The Peculiar Hemodynamics of Septic Shock, Postgrad. Med. 48: 106 (Oct.) 1970.*) ABSTRACTER'S COMMENT: This is additional evidence that hemodynamic studies and not just clinical impressions are necessary for the optimal management of patients with sepsis.

SHOCK Groups of dogs were subjected to hemorrhagic shock, and some also received mechanical ventilation. In the more severely shocked animals significant pulmonary injury developed most rapidly in those animals allowed to breathe spontaneously. Mechanical ventilation reduced the morphologic changes in the lungs of dogs in shock but did not prevent the development of significant pulmonary venous admixture. Ventilatory support did not alter the pulmonary injury in the dogs subjected to less severe shock, and pulmonary injury showed progression during the 24 hours after reinfusion. None of the animals in either group developed respiratory insufficiency, and the animals sacrificed 72 hours after reinfusion showed partial recovery from the morphologic and functional changes. (*Bryant, L. R., and others: Acute Respiratory Pathophysiology after Hemorrhage Shock, Surgery 68: 512 (Sept.) 1970.*)

FLUID REPLACEMENT THERAPY Mongrel dogs were anesthetized with pentobarbital and their lungs ventilated mechanically. Following cannulation of a femoral artery, the left atrium, and the main pulmonary artery (through a thoracotomy), control values for intravascular pressures, cardiac output and blood gases were obtained every ten minutes for 30 minutes. Dogs were then rapidly bled 20 ml/kg (24 to 26 per cent of estimated blood volumes) and the measurements were repeated. During the next 20 minutes, three types of volume replacement were accomplished in three groups: Ringer's lactate solution to three times the blood loss; 25 per cent Dextran 40 in buffered Ringer's lactate solution to two times the blood loss; reinfusion of previously shed blood. Measurements were made every 20 minutes for an additional two hours. Ringer's lactate solution supported circulation for 40 minutes, after which deterioration in cardiac output and blood volume appeared. Two hours after infusion, only 18 per cent of the infused volume was present intravascularly. Reinfusion of shed blood failed to restore circulatory dynamics to normal. Dextran in buffered Ringer's lactate solution provided adequate hemodynamic support for two hours after infusion, at which time 43 per cent of the infused volume remained in the circulation. (*Carey, J. S., and others: Hemodynamic Effectiveness of Colloid and Electrolyte Solutions for Replacement of Simulated Operative Blood Loss, Surg. Gynec. Obstet. 131: 679 (Oct.) 1970.*)

Neonatology

HYPOXIA AND VENTILATION Ventilation and heart rate were measured in eight healthy term infants (6 hours to 11 days old) subjected to hypoxia ($P_{A_{O_2}} = 60$ torr) at three CO_2 levels in a neutral environment. At nor-