

of hypothermia. Increased blood viscosity and arterial hypotension play a major role in causing poor tissue perfusion. Administration of low molecular weight dextran or heparin results in improved tissue perfusion. Normal saline solution has a similar effect which is, however, shortlived. Isoproterenol administration does not produce any beneficial effects upon the microcirculation during hypothermia. (Suzuki M., and Penn, I.: *The Effect of Therapeutic Agents Upon the Microcirculation during General Hypothermia*, *Surgery* 60: 867 (Oct.) 1966.)

**RENAL BLOOD FLOW IN SHOCK** In dogs, renal blood flow averages 9.4 ml./minute/kg. Hemorrhagic hypotension reduced renal blood flow proportional to the reduced perfusion pressure with no change in renal resistance. Adrenergic blockade did not improve renal perfusion but transfusion promptly restored renal blood flow and perfusion. (Dow, R. W., and Fry, W. J.: *Hemorrhagic Shock, Changes in Renal Blood Flow and Vascular Resistance*, *Arch. Surg.* 94: 190 (Feb.) 1967.)

**DEXTRAN IN SHOCK** The hemodynamic response to a rapid infusion of dextran was evaluated in 31 patients with nonhemorrhagic shock. All but 2 patients received 500 ml. of either 6 per cent dextran 75 in saline or 10 per cent dextran-40 in 5 per cent dextrose or saline. Cardiac output (C.O.) rose from a mean of 2,937 ml./minute to a mean of 4,842 ml./minute. No significant difference was noted in the output response to the two dextran preparations. Mean arterial pressure rose after dextran from 60 to 73 mm. of mercury and pulse pressure increase from 38 to 54 mm. of mercury. Heart rate fell from an average of 103 to 95 while stroke volume rose from 31 to 56 ml./beat. Right atrial pressure rose from an average of 0.1 to 4.6 mm. of mercury while central blood volume increased an average of 319 ml. The effectiveness of dextran in augmenting cardiac output is due to an increased venous return. The beneficial hemodynamic response is also due to the rheological properties of dextran, i.e. reducing viscosity and inhibiting red cell aggregation. This latter effect should reverse red cell sludg-

ing, speed capillary flow further reducing viscosity with an overall decreased resistance to flow and an increase venous return to the heart. It is suggested that the safety and efficacy of dextran commend it as the treatment of choice in all types of hypotension and shock associated with a low or normal central venous pressure. (Cohn, J. N., and others: *Studies in Clinical Shock and Hypotension. V. Hemodynamic Effects of Dextran*, *Circulation* 35: 316 (Feb.) 1967.)

**SHOCK** The responses to volume loading with 500 ml. of 10 per cent dextran 40 followed by isoproterenol infusion were studied in 23 patients in shock. Patients were divided into three groups according to the response of cardiac index (C.I.) to dextran-40: Group I, greater than 50 per cent increase; Group II, C.I. increased 15 to 50 per cent; Group III, C.I. not changed or decreased. Following dextran infusion central venous pressure (C.V.P.) rose from 2.3 to 7.3 mm. of mercury in Group I patients. These patients also failed to respond to isoproterenol, suggesting diminished venous return as a cause for their circulatory insufficiency. C.V.P. in Group III patients also rose (7.5 to 12.5 mm. of mercury) but these patients responded dramatically to isoproterenol, suggesting myocardial insufficiency as their primary cause for shock. It is suggested that depletion or altered metabolism of endogenous catecholamines may underly this myocardial insufficiency. (Corey, J. S., and others: *Cardiovascular Function in Shock*, *Circulation* 35: 327 (Feb.) 1967.)

**BLOOD LOSS** Marked acute decreases in red blood cell mass and low absolute levels of hematocrit followed exclusive use of crystallloid solutions for priming the extracorporeal apparatus and to replace blood loss postoperatively. Sequential studies of renal, cerebral, cardiac, and hepatic function showed no significant impairment up to one week after operation. There was a marked tendency for hypovolemic shock to develop in these patients via gargantuan urination. With this, the hematocrit began to rise, exactly opposite to the tendency towards hemodilution in classical hemorrhage. An additional feature was the