

pulmonary bypass with a reduction in temperature to 10° C. hypothermia can result in hypoxic acidosis. Excess lactate increased in the mixed venous blood when the flow rate was less than 60 ml/kg./minute. However, the tissues consumed a considerable amount of oxygen when total circulatory arrest of 30 to 40 minutes was imposed. At an intravascular temperature of 10° C. the arterial-mixed venous oxygen content difference increased 10 volumes per cent and the excess lactate rose during total circulatory cessation. Although flow rates could be increased during rewarming, at the termination of the perfusion the excess lactate was still increased in the mixed venous blood and an average buffer base deficit of 8 mEq. per liter was present. This occurred in spite of the addition of sodium bicarbonate to the oxygenator to compensate for the increased hydrogen ion concentration in the blood at the end of cardiopulmonary bypass. (Neville, W. E., and others: *Relationship of Flow Rate, "Excess Lactate" and Buffer Base During Closed Chest Profound Hypothermic Perfusion with a Pump Oxygenator*, *Surgery* 55: 281 (Jan.) 1964.)

SHOCK Value of vasoexcitor-pressor drugs in treating shock has been questioned due to tissue ischemia resulting from intense constrictor effects on resistance arterioles. The more favorable excitatory effects on the more distal vessels of the capillary bed is difficult to demonstrate because of the unselective constriction produced by the amine pressors over a wide range on all muscular elements of the peripheral circulation. Vasoactive polypeptides and catecholamines activate smooth muscle through different mechanisms and may even be antagonistic at the cellular level. When rats subjected to hemorrhagic shock were given PLV-2 (Sandoz) there was an increase in survival over control animals at all blood pressure levels, especially those at lower arterial pressures. The mechanism involved has not yet been identified precisely but may be related to limited or selective vasoconstrictor action. (Hershey, S. G., and others: *Use of Pressor Drugs in Hemorrhagic Shock*, *Proc. Soc. Exp. Biol. Med.* 115: 325 (Feb.) 1964.)

VASOPRESSORS IN SHOCK Twelve patients in shock were carefully studied in terms of cardiovascular, venous and renal effects subsequent to the administration of noradrenalin, metaraminol, and angiotensin II. Cause of shock was bacterial infection in four patients, myocardial infarction in three, hypovolemia in two, and neurogenic hypotension in three. A lower cardiac output and urine flow associated with a disproportionate increase in peripheral vascular resistance were observed when angiotensin was used as compared to levarterenol and metaraminol. Use of angiotensin in treatment of shock is questioned, especially in relation to its preference over levarterenol and metaraminol. (Udhoji, V. N., and Weil, M. H.: *Circulatory Effects of Angiotensin, Levarterenol and Metaraminol in the Treatment of Shock*, *New Engl. J. Med.* 270: 501 (Mar. 5) 1964.)

ANGIOTENSIN IN SHOCK Infusion of moderate doses of angiotensin II has been shown to cause a rapid loss of relatively protein-poor fluid from the vascular system. Equipressor doses of noradrenaline produced comparable effects. The ability of angiotensin to deplete circulating plasma volume and prior evidence of endogenous production of considerable amounts of angiotensin during hemorrhagic hypotension suggest that this material could contribute to progressive circulatory deterioration in shock. A review of known cardiovascular effects of angiotensin does not reveal any which might provide a theoretical basis for its use in the treatment of shock. (Nickerson, M., and Sutter, M.: *Angiotensin in Shock*, *Canad. Med. Ass. J.* 90: 325 (Jan. 25) 1964.)

METARAMINOL SHOCK Increasing doses of metaraminol bitartrate (Aramine) in a continuous infusion were used to support blood pressure in four patients. Under such treatment a state of shock developed, characterized by intense peripheral vasoconstriction, hypotension, and anuria. In spite of an adequate fluid intake all patients showed severe hemoconcentration, and a critically low plasma volume was demonstrated in two. Treatment with plasma expanders and small doses of noradrenaline resulted in transient improvement in one patient and complete recovery from