CATECHOL RESPONSE Responses to varying loses of noradrenaline and epinephrine were studied in intact dogs and from isolated vessels of normal and reserpinized dogs. Sodium pentobarbital did not depress the responses to noradrenaline and epinephrine in either control or reserpinized dogs when 45 mg. per kg. or less of the anesthetic were administered. When the dosage of anesthetic was raised to 60 mg. per kg. in the control animals, blood pressure fell precipitiously and amine responses became erratic. The responses to the catecholamines and the phenomenon of post-reserpine sensitivity are not significantly affected by moderate doses of pentobarbital. Most of the depression of the cardiovascular system is the result of a depression of the heart and central nervous system. The peripheral vessels retain their reactivity to the catecholamines. (*Carrier, O. J., and Holland, W. C.: Effect of Sodium Pentobarbital on the Responses to Catecholamines in Normal and Reserpinized Dogs, Canad. J. Physiol. 44: 176 (Jan.) 1966.*)

SHOCK Hemorrhagic shock with an average blood pressure of 32 mm. of mercury was induced for a maximum period of 90 minutes in anesthetized, open-chest dogs on artificial ventilation and evidence of anaerobic myocardial metabolism was studied. No significant shift to anaerobiosis occurred under the conditions prevailing. Oxygen data from the present and previous studies show that coronary sinus oxygen content and saturation during shock, while indeed falling, do not reach the level known from hypoxia experiments to be critical to the heart, *i.e.*, to initiate anaerobic metabolism. It is concluded that the available metabolic evidence does not support the hypothesis of a critical deficiency in oxygen supply to the heart as a cause of the cardiac deteriorization seen in prolonged experimental shock and considered to be important for the development of irreversibility. (*Lundsgaard-Hansen, P.: Oxygen Supply and Anaerobic Metabolism of the Heart in Experimental Hemorrhagic Shock, Ann. Surg. 163: 10 (Jan.) 1966.*)

ATP IN SHOCK There is a protective action when adenosine triphosphate is administered prior to experimental hemorrhagic shock in rats. This action is not due solely to its vasodilatory action since equivalent hypotensive amounts of both adenosine monophosphate and dibenzyline failed to afford an equivalent protection. There is a statistically significant beneficial effect when ATP is given after bleeding. It is concluded that the terminal high energy phosphate bond is responsible for this activity. (*Sharma, G. P., and Eiseman, B.: Protective Effect of ATP in Experimental Hemorrhagic Shock, Surgery 59: 66 (Jan.) 1966.*)

CATECHOLAMINES IN SHOCK Plasma catecholamine concentrations in endotoxin shock were studied in dogs employing a range of endotoxin dosage. Plasma epinephrine rose simultaneously with the onset of hypotension and then gradually returned to normal. Little or no elevations of norepinephrine were observed. A slow infusion of a large dose of endotoxin resulted in a delayed and gradual onset of hypotension with close correlation between the fall in blood pressure and the rise in plasma epinephrine. Resistance to endotoxin was found to be unrelated to the time of appearance and concentration of plasma epinephrine. Resistant and nonresistant animals exhibited the same pattern of response. Plasma epinephrine response was abolished by cervical cord section or adrenalectomy. Under these two conditions, small doses of endotoxin produced profound shock resulting in rapid death and indicating that circulating catecholamine is not necessary to initiate endotoxin shock. Although the lethal action of endotoxin was accelerated in the absence of circulating catecholamine, the precise role of these amines in the late stages of endotoxin shock requires further study. (*Spink, W. W., and others: Correlation of Plasma Catecholamine Levels with Hemodynamic Changes in Canine Endotoxin Shock, J. Clin. Invest. 45: 78 (Jan.) 1966.*)

VASOPRESSORS IN SHOCK The influence of norepinephrine, angiotensin and PLV-2 (a synthetic analogue of vasopressin) on the microcirculation in the mesoappendix was studied in rats shocked by temporary liga-

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