is present for a length of time, tissue hypoxia, toxic humoral factors, and arteriolar constriction and capillary damage combine to produce "irreversible" shock and this may be accentuated by use of vasopressor drugs which act by peripheral vasoconstrictive mechanisms. Outlined are four clinical states where vasopressors may be of value: (1) peripheral circulatory failure secondary to loss of arteriolar vasomotor function; (2) central circulatory failure secondary to coronary thrombosis; (3) peripheral circulatory failure associated with suspected coronary or cerebral arteriosclerosis; (4) extreme systemic hypotension with clinical signs of inadequate coronary and/or cerebral blood flow. Humans who face supervised hemorrhage (extensive surgery) may benefit from prophylactic adnergic blockers to reduce peripheral vasoconstriction combined with vasopressors to increase cardiac output. (*Greisman, S. E.: Physiologic Basis for Vasopressor Therapy during Shock, Ann. Int. Med. 50: 1092 (May) 1959.*)

NORADRENALINE Stimulation of the cervical sympathetic nerve with an electric current caused an increase in protein-bound noradrenaline in the heart. After prolonged stimulation of the superior cervical ganglia there was a tendency to a decreased protein-bound noradrenaline content in the heart. Castration or administration of desoxycorticosterone acetate also influenced the level of the protein-bound noradrenaline in the heart. On the basis of results obtained the author postulates that binding with protein of noradrenaline in the heart is an important part of chemical mediation of sympathetic nerve stimulation. (*Barts, M. P.: Protein-bound Noradrenaline in a Rabbit Heart as a Factor of Chemical Mediation, Biokhimiya 22: 677 1957.*)

LEVARTERENOL The effectiveness of levarterenol in treatment of shock due to prolonged oligemia was studied in the dog. The survival time of animals receiving levarterenol and whole blood was significantly greater than that of animals receiving a placebo and whole blood. (*Fozzard, H. A., and Gilmore, J. P.: Use of Levarterenol in Treatment of Irreversible Hemorrhagic Shock, Am. J. Physiol: 196: 1029 (May) 1959.*)

LEVARTERENOL AND CORTISONE The response of animals to levarterenol was unchanged after the administration of hydrocortisone or aldosterone. (*Small, H. S., Weitzner, S. W., and Nahas, G. G.: Cardiovascular Effects of Levarterenol, Hydrocortisone Hemisuccinate and Aldosterone in the Dog, Am. J. Physiol. 196: 1025 (May) 1959.*)

CATECHOL AMINES Catechol amine depletion in cats was carried out by pre-treatment with reserpine and bilateral cervical sympathectomy. Isolated cardiac papillary muscle strips were prepared and subjected to repeated stimulation. Amplitude of contraction was found to be significantly lower in pretreated cats than in controls. Conclusion was that depletion of catechol amines results in depression of cardiac contractility and that under normal conditions stores of epinephrine and norepinephrine in the myocardium are released in small amounts and are necessary as humoral agents for the regulation of cardiac pacemaker and to maintain a normal state of contractility. (*Lee, W. C., and Shideman, F. E.: Role of Myocardial Catechol Amines in Cardiac Contractility, Science 129: 967 (April 10) 1959.*)

RESERPINE When the heart-lung preparation is made from a dog treated with reserpine, catechol amines such as noradrenaline and isoprenaline have a greater effect on the rate of the heart than they have in a preparation from a normal dog. Other sympathomimetic amines such as tyramine and ephedrine, on the other hand, are found to have lost their action. Since treatment with reserpine has been shown to cause the store of noradrenaline in the heart to disappear, and the infusion of noradrenaline into the preparation made from a reserpine-treated animal restores the action of tyramine, it is concluded that substances like tyramine and ephedrine normally act by liberating noradrenaline from the store, and do not act directly. (*Bejrablaya, D., Burn, J. H., and Walker, J. M.: The Action of Sympathomimetic Amines on Heart Rate in Relation to the Effect of Reserpine, Brit. J. Pharmacol. 13: 461 (Dec.) 1958.*)

DIGITALIS TOXICITY In toxic doses digitalis may act as a myocardial depressant or