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

irritant. Most forms of sino-atrial block are due to increased vagal tone, and this is commonly due to digitalis intoxication. The irritant action of digitalis may be evidenced by atrial fibrillation, atrial tachycardia, and ventricular extra systoles. Depletion of potassium and excess of calcium enhance the toxic effects of digitalis. Cortisone given to the digitalized patient may cause digitalis toxicity by inducing a loss of potassium. The treatment of arrhythmias due to overdosage consists of withholding the drug, giving potassium of a chelating agent to depress serum calcium, and giving procaine amide. (*Connolly, D. C.: Arrhythmias Associated with Digitalis Therapy, Postgrad. Med. 25: 509 (May) 1959.*)

**INTERACTION OF RELAXANTS** The time course of the interaction between decamethonium or succinylcholine and tubocurarine was determined by the rabbit head-drop technique. Pretreatment with decamethonium decreased the amount of tubocurarine required to produce head-drop to an even greater extent than did pretreatment with tubocurarine itself. Succinylcholine pretreatment decreased the head-drop dose of tubocurarine. If tubocurarine were administered first and succinylcholine ten minutes later, the head-drop dose of succinylcholine was reduced. (*Smith, C. M., and Urban, C.: Interaction Between Neuromuscular Blocking Agents: Time Course of Effects as Assessed by Rabbit Head-Drop Assay, J. Pharmacol. & Exper. Therap. 125: 227 (March) 1959.*)

**NEOMYCIN APNEA** Two patients, 84 and 83 years old, developed apnea following intraperitoneal instillation of 2 Gm. of neomycin. Both patients ultimately expired, although one recovered from his apnea before death. (*Doremus, W. P.: Respiratory Arrest Following Intraperitoneal Use of Neomycin, Ann. Surg. 149: 546 (April) 1959.*)

**PRESTONAL** Prestonal (G-25178) is a shortacting muscle relaxant which has little action on blood pressure but occasionally causes tachycardia. It also possesses anticholinesterase activity. There is no antidote available. (*Hunter, A. R.: Prestonal (G-25178); A New Shortacting Muscle Relaxant, Der Anaesthesist 8: 82 (March) 1959.*)

**MEPROBAMATE INTOXICATION** A case of deep coma due to overdose of a preparation containing meprobamate is presented. Meprobamate was detected in blood, gastric washings, and urine. He was treated conservatively with vasopressor drugs and intravenous infusions. He demonstrated marked cutaneous vasodilatation. He recovered consciousness 39 hours after admission. (*Bedson, H. S.: Coma Due to Meprobamate Intoxication, Lancet 1: 288 (Feb. 7) 1959.*)

**DEXTRAN** Administration of more than 2,000 ml. of Dextran intravenously may increase the bleeding time. It increases the blood volume and causes hemodilution. Within twenty-four hours after intravenous administration Dextran is either excreted or metabolized. It is the best plasma volume expander to use, awaiting blood, and is the only one now being added to the national stockpile by the Federal Civil Defense Administration. (*Howard, J., and others: The Present Status of Dextran as a Plasma Expander, Am. J. Surg. 97: 593 (May) 1959.*)

**HYDROXYDIONE** Presuren brand of hydroxydione is a fine powder readily soluble in 0.25–0.5 per cent procaine solution. No pain occurs on rapid injection of such mixtures. In 346 patients thus anesthetized with precautions to insure emptying of the veins, the incidence of thrombosis was reduced almost to that seen with thiopental. Thrombosis tended to be painless and not extensive. In dosage of 5–8 mgm./lb. to provide basal narcosis in premedicated patients induction time with the rapid injections was three minutes. The use of this drug is advocated in anesthesia for Caesarian section and in patients with partial respiratory obstruction. (*Galley, A. H., and Lerman, L. H.: A New Technique with Hydroxydione, Brit. M. J. 1: 332 (Feb. 7) 1959.*)

**FAMILIAL DYSAUTONOMIA** This disease is a congenital condition frequently seen in siblings and characterized by specific disturbances of the nervous system, particularly the autonomic division. Striking features are: