atrophy of the testicles and impairment of spermatogenesis. No obvious relationship of the monoamine oxidase-inhibiting activity of the compounds to any of these changes could be detected. (*Zbinden, G., and Studer, A.: Experimental Pathology of Iproniazid and Related Compounds, Bull. New York Acad. Sc. 80: 873 (Sept. 17) 1959.*)

ANTICONVULSANT Monoamine oxidase inhibitors have a pronounced anticonvulsant effect in animals which is closely associated with elevation of brain 5-hydroxytryptamine and norepinephrine, while reserpine enhances convulsions. The association between changes in physiologically active brain amines and enhancement of or protection against experimental seizures raises a possibility that certain types of epilepsy may involve a localized dysfunction in the formation, release, or metabolism of known or unknown amines. (*Prockop, D. J., Shore, P. A. and Brodie, B. B.: Anticonvulsant Properties of Monoamine Oxidase Inhibitors, Bull. New York Acad. Sc. 80: 643 (Sept. 17) 1959.*)

PRESSOR AMINES Sympathomimetic amines can be divided into 3 main classes on the basis of their action after norepinephrine infusion. Class I contains derivatives of catecholamine. Their pressor action is diminished by prior infusion of norepinephrine (N). Class II contains some derivates of ethanolamine rather closely related to N chemically. Their pressor action is less diminished by prior infusion of N. Class III contains other derivatives of ethanolamine and derivatives of phenylethylamine. Their pressor action is markedly enhanced by a prior infusion of N. Aramine, vasoxyl and propadrine, derivatives of phenylethylamine, were found to belong to class II. Methedrine, mephine and vonedrine, derivatives of phenylethylamine, acted like class III agents with mephine having the most marked action. During an infusion of N this agent is stored in the vessel wall from which it is slowly released. It is postulated that the large amount of stored N blocks the pressor action of circulating N in some way. Class III agents appear to act by liberating N from the vessel wall. Class II agents act in this way in part, but also have a direct action on the vessel wall as do class I agents. It is suggested that the most effective means of supporting the pressor action of an N infusion which is gradually failing is to give a class III agent. Class II agents would be less effective. (*Burn, J. H. and Rand, M. J.: Fall of Blood Pressure After Noradrenaline Infusion and Its Treatment by Pressor Agents, Brit. M. J. 1: 394 (Feb. 14) 1959.*)

NOREPINEPHRINE The response to norepinephrine in 8 patients with aortic regurgitation was studied and contrasted with findings in 7 normals. A disparity of response between the two groups was most manifest in the pulmonary "capillary" and artery pressure alterations, a nearly fourfold rise to pulmonary congestion levels in the aortic regurgitation group contrasted with a 5 mm. Hg rise occurring in the normal. This small increment of left ventricular filling pressure was associated with a sizable stroke work increase in the normal, but no significant change in the aortic regurgitant group. Bradycardia and increased pulmonary arteriolar resistance, present in the normals, were absent in the aortic regurgitation group. Displacement of blood from the peripheral venous system seems the predominant if not the sole mechanism in the induction of pulmonary congestion and diminished left ventricular performance. Data acquired in four patients during peripheral venous occlusion by tourniquet supported this interpretation. Here, the pulmonary "capillary" pressure increment and the stroke work response after norepinephrine were of the same order exhibited in the normal. (*Regan, T. J., and others: Norepinephrine Induced Pulmonary Congestion in Patients with Aortic Valve Regurgitation, J. Clin. Invest. 38: 1564 (Sept.) 1959.*)

LEVARTERENOL Use of levarterenol in treatment of shock is sometimes complicated by extravasation sloughs of skin and subcutaneous tissue. Treatment has been by infiltration of the local areas of ischemia by 5–10 mg. phentolamine methane sulfonate (Regitin) with reversal of vasoconstrictive ischemic effects in 5–7 minutes. This treatment is effective only if the extravasation is discovered and treated promptly. Studies have shown mix-

tures of levarterenol and phentolamine in ratios of 4:5 up to 4:40 mg. per liter show the same pressor effect in normotensive subjects as levarterenol alone. These same mixtures when given subcutaneously do not result in ischemic necrosis. Several patients in shock treated with such mixtures displayed a satisfactory pressor response with other evidence to show that addition of phentolamine levarterenol mixtures does not impair the efficiency of the levarterenol as a pressor amine. Such mixtures are useful in preventing complication of ischemic slough during levarterenol therapy. (*Zucker, G., and Levine, J.: Pressor and Diminished Local Vasoconstrictor Effects of Levarterenol-Pentolamine Mixtures, Arch. Internal Med. 104: 607 (Oct.) 1959.*)

NOREPINEPHRINE Serious arrythmias appearing during treatment of cardiogenic shock with norepinephrine may be a function of the degree of atherosclerotic involvement of the heart and coronary circulation. This study undertook to produce gross and microscopic atherosclerotic changes and to evaluate the functional effects on the heart of epinephrine and norepinephrine. Results showed that cholesterol fed rabbits up to 38 weeks showed no significant anoxic electrocardiographic changes. Injections of epinephrine 100–500 μg. ranges produced ventricular arrythmias and on occasion ventricular fibrillation in normal and high fat fed animals. Following norepinephrine in 100–200 μg. range, tachycardias were seen only in fat fed animals, but with higher doses, 500–1,000 μg. dosage ranges similar changes were seen in both groups. Norepinephrine under these circumstances induced characteristic anoxic ST-T electrocardiographic changes, but no instances of ventricular fibrillation. Perfusion studies of isolated hearts showed decreased contractility and reduced responses to epinephrine and norepinephrine in high fat fed animals. (*Melville, K. I. and Shister, H. E.: Cardiac Responses to Epinephrine and Norepinephrine during prolonged Cholesterol and High Fat Feeding in Rabbits, Am. J. Cardiol. 4: 391 (Sept.) 1959.*)

NEUROBLASTOMATA Three patients were found, on radiological or histological evidence, to have neuroblastomas which secreted norepinephrine, normally only secreted by mature chromaffin cells or tumors arising therefrom (and postganglionic fibers of the sympathetic nervous system). Neuroblastomas though composed of cells less differentiated and mature than chromaffin cells, and not on the same direct line of development, do arise from the same primordial cell type. (*Isaacs, H., and Medalie, M.: Noradrenaline-Secreting Neuroblastoma, Brit. M. J. 1: 401 (Feb. 14) 1959.*)

POSITIVE PRESSURE BREATHING The effect of intermittent positive pressure breathing on the distribution of inspired air, and the number and types of ventilating spaces was measured in patients with pulmonary emphysema and pulmonary heart disease. Patients with pulmonary heart disease had a 100 per cent increase in functional residual capacity, and four to six spaces of ventilation. The poorly ventilated spaces comprised more than 50 per cent of the whole. During intermittent positive pressure breathing, these values decreased sharply and approached normal. Concomitantly, arterial oxygen tension was increased and carbon dioxide tension was reduced. Intermittent positive pressure breathing thus improves alveolar ventilation in these patients by improving distribution of inspired air and by decreasing the poorly ventilated spaces. (*Torres, G. E. and Lyons, H. A.: Intermittent Positive Pressure Breathing in Patients with Pulmonary Heart Disease: Its Effect on Distribution of Inspired Air, Bull. New York Acad. Med. 35: 751 (Nov.) 1959.*)

HYPERVENTILATION DURING ANESTHESIA Crile believed that the loss of consciousness alone was not sufficient to protect patients from the reflex effects of painful stimuli during surgery. Yet nitrous oxide (70 per cent) and *d*-tubocurarine has been used frequently as the sole anesthetic agents in accidents or surgical procedures. Absolute muscle flaccidity and effective control of the respiration is necessary. This combination provides no more than mere unconsciousness. In order to explain why this technique works, electroencephalographic tracings and estimation of blood *p*H were obtained during anesthesia.