thermia of muscles increases neuromuscular blockade due to depolarizing drugs and attenuates that due to tubocurarine. Comparable results were obtained in humans with the depolarizing drugs but results with tubocurarine were inconsistent. This was thought to be due to inadequate recordings and the inconsistency is now being eliminated. (*Zaimis, E., Cannard, T. H., and Price, H. L.: Effects of Lowered Muscle Temperature upon Neuromuscular Blockade in Man, Science 128: 34 (July 4) 1958.*)

RELAXIN While relaxin cannot influence uterine contractions of labor, alter the subjective pain of labor, or alter the normal progress of labor, relaxin can cause a reduction of blood pressure in patients with hypertension and toxemia. (*Decker, W. H., and others: Some Effects of Relaxin in Obstetrics, Obst. & Gynec. 12: 37 (July) 1958.*)

PULMONARY EDEMA Experimental pulmonary edema produced by ventilation with chlorine gas was studied in 250 rats. Morphine was found to significantly decrease the degree of pulmonary edema. Aerosols of 5 per cent silicone antifoam suspension and 25 per cent alcohol solution were less effective. Nembutal, atropine, Demerol and Phenergan were ineffective in decreasing the edema. (*Polli, J. F., and Musiker, B. S.: Effect of Morphine, Aerosol Mixtures, and Other Agents on Experimental Pulmonary Edema in Rats Following Exposure to Chlorine Gas, Exper. Med. & Surg. 16: 73, 1958.*)

PULMONARY EDEMA Experimental and clinical (cardiac catheterization) data proved that for acute pulmonary edema to develop, the presence of factors other than increased pulmonary pressure is necessary. Those factors are endogenous and exogenous and include pain, emotion, various medicinal substances, ether inhalation, trauma during cardiac surgery etc. Injection of hexamethonium in cases of acute pulmonary edema developing in mitral stenosis patients during operation resulted in rapid regression of the edema. Hexamethonium lowers the pressure in the pulmonary circulation and at the same time causes vasoconstriction, which blocks the occurrence of reflex vascular reactions. In patients under anesthesia a smaller than usual dose of hexamethonium is sufficient. It is concluded that a neurogenic factor forms the basis of acute pulmonary edema in cases of mitral stenosis; acute pulmonary edema is, therefore, a generalized pathological state with severe reflex neurogenic disturbances. (*Marinescu, V., and Ionescu-Buzhor-Karus: Mechanism of Acute Pulmonary Edema in Mitral Stenosis, Vestn. Khir. 77: 23, 1956.*)

MYASTHENIA GRAVIS If curare is given to a patient with latent myasthenia gravis during surgery, a true myasthenic crisis may occur. Maintenance of both an adequate airway and adequate ventilation is essential. Antibiotics help to prevent pneumonia. Tensilon is the antidote of choice during the initial treatment and neostigmine is indicated for maintenance. (*Warren, D., Eastwood, D., and Muller, W.: Myasthenia Gravis and Curare, Am. J. Surg. 96: 102 (July) 1958.*)

THYROID The relationship between thyroid and adrenal cortical function was studied in a group of patients with either thyrotoxicosis or primary myxedema. Plasma cortisol levels were normal in most of these patients. Infused steroids disappeared more rapidly from the plasma in thyrotoxicosis and more slowly in myxedema. Therapy of the thyroid disease returned the metabolism of the infused steroids to normal. The rate of synthesis of cortisol was reduced in myxedema and increased in thyrotoxicosis. Institution of the euthyroid state in these patients returned adrenal cortisol production to normal. These data suggest that there is a homeostatic mechanism which results in a decreased synthesis of cortisol in patients with myxedema in whom the rate of removal of cortisol by the liver is impaired, and an increased synthesis of cortisol in patients with thyrotoxicosis in whom the rate of removal of cortisol by the liver is accelerated. (*Peterson, R. E.: Influence of Thyroid on Adrenal Cortical Function, J. Clin. Invest. 37: 736 (May) 1958.*)

POSTOPERATIVE ALDOSTERONISM An adrenocortical hormone with a remarkable activity for promoting sodium