added. 103 transfusions were preceded by intravenous administration of 200 mg. tetamon (tetraethylammonium, a synthetic ganglion-blocking agent) and of these 41 showed no reaction whatsoever, 61 showed a reaction much milder than could have been expected, and in one instance the reaction was marked. Twenty other transfusions were preceded by administration of 50 mg. of hexathonid (hexamethonium iodide, a more active ganglion-blocking agent); in 12 cases no reaction was observed and in the remaining 8 cases it was mild. This activity of ganglion-blocking agents shows that excessive production of acetylcholine is one of the main pathogenetic factors in the production of transfusion reactions. (*Novachenko, N. N.: New Methods of Prevention of Transfusion Reactions Based on Their Pathogenesis, Vrac. Delo 8: 873, 1956.*)

TRANSFUSION REACTIONS The serum levels of total protein and electrophoretic protein fractions were estimated (a) in 24 patients who had nonhemolytic reactions to blood transfusion; (b) in 18 healthy blood donors; and (c) in 34 patients who had taken blood transfusion well. Subnormal total protein levels were found most often in the patients who had reacted badly to blood transfusions. Where there is no urgency, the patient's serum proteins should be determined prior to transfusion. When the serum protein, and especially serum albumin, is low, the patient should be given washed erythrocytes instead of whole blood. (*Polak, A., and Fiser-Herman, M.: Serum-Proteins in Patients with Non-Haemolytic Transfusion Reactions, Lancet 1: 1042, (May 17) 1958.*)

TRANSFUSION When faced with serious blood loss which cannot be replaced rapidly enough with intravenous transfusions, the use of intra-aortic transfusions may be lifesaving. Displacement of the intestinal tract onto the abdominal wall and use of a specially curved needle facilitate entrance into the aorta. Five cases are presented in which intra-aortic transfusion preserved life. (*Rive, H. L., and others: Intra-Aortic Transfusion, Obst. & Gynec. 11: 537 (May) 1958.*)

PARENTERAL FLUID DOSAGE A rule of thumb based on weight alone (rather than on body surface area), when combined with appraisal of daily weights and clinical appearance, including skin turgor and frequency and volume of urination, is sufficiently accurate. For infants less than 1 year of age, 60 ± 15 ml. of water per pound (132 ± 33 ml. per kilogram) is given each 24 hrs.; for children 1 to 5 years, 50 ± 15 ml. per pound (110 ± 33 ml. per kilogram) is given; for children above 5 years, approximately 40 ± 15 ml. per pound (88 ± 33 ml. per kilogram) is given. In the presence of normal renal function, isotonic sodium chloride up to one-third of the total daily fluids is well tolerated; the remaining volume consists of 5 or 10 per cent dextrose in water. (*Oliver, W. J., and others: Lack of Scientific Validity of Body Surface as Basis for Parenteral Fluid Dosage, J. A. M. A. 167: 1211 (July 5) 1958.*)

TETANUS Two identical groups of patients were treated similarly, except that one received chlorpromazine and the other phenobarbital. There was no statistically significant difference in the outcome of the treatments of the two groups. However, chlorpromazine was easier to manage than barbiturates, because it controlled the tetanus convulsions without causing loss of consciousness or of clinically noticeably depressed respiration. (*Laurence, D. R., and others: Clinical Trial of Chlorpromazine Against Barbiturates in Tetanus, Lancet 1: 987 (May 10) 1958.*)

NEUROMUSCULAR BLOCK Gastrocnemius response to sciatic stimulation was measured in anesthetized cats. The response to suxamethonium and tubocurarine differed between symmetrical muscles. Increased rates of stimulation and fatigue both increased sensitivity to blocking agents. During recovery from suxamethonium block some muscles showed a transitory failure to react to each stimulus. (*Wislicki, L.: Effects of Rate of Stimulation and of Fatigue on Response to Neuromuscular Blocking Agents, Brit. J. Pharmacol. 13: 138 (June) 1958.*)

NEUROMUSCULAR BLOCK Experimental work with cats showed that hypo-

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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. (*Marinescku, V., and Ionescku-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

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