potassium salts. The total cumulative potassium deficit may be considerable and not corrected for some time after chlorothiazide is discontinued, thereby continuing the state of myocardial vulnerability and digitalis hypersensitivity. (*Richman, J. L.: Digitalis Intoxication Induced by Chlorothiazide, Bull. Tufts-New England Medical Center 6:18 (Jan.-Mar.) 1960.*)

MUSCLE RELAXANT The effects of edrophonium and choline were compared with those of the depolarizing substances acetylcholine, decamethonium, and suxamethonium in tibialis anterior muscles of cats. Both edrophonium and choline were more potent antagonists to paralysis by tubocurarine than could be accounted for by their ability to stimulate the motor end plates directly. Previous administration of benzoquinonium abolished the antagonistic action to tubocurarine of normally effective doses of edrophonium and reduced that by choline. These anti-curare compounds do not appear to act by cholinesterase inhibition, nor by an increase in the sensitivity of the motor end plates. A presynaptic mechanism of action is suggested. (*Blaber, L. C., and Bowman, W. C.: A Comparison Between the Effects of Edrophonium and Choline In the Skeletal Muscles of the Cat, Brit. J Pharmacol. 14:456 (Dec.) 1959.*)

LIGNOCAINE Lignocaine, a local anesthetic agent, in a dose of 1 to 2 mg./kg. of body weight to dogs increased cardiac output due to a rise in both heart rate and stroke volume. Arterial blood pressure was elevated as contrasted to the fall produced by procaine. Central blood volume was increased, but central venous pressure, total peripheral resistance and the ventilation perfusion ratio were decreased. In cross circulation experiments it was demonstrated that the primary site of action of lignocaine on cardiac output was central. (*Kao, F. F., and Jalar, U. H.: The Central Action of Lignocaine and its Effect on Cardiac Output, Brit. J. Pharmacol. 14: 552 (Dec.) 1959.*)

OXYGEN TOXICITY An investigation was carried out which indicated that the inhalation of oxygen at a partial pressure of 418 mm. Hg (equivalent to breathing 55 per cent oxygen at sea level) for a period of seven days was without marked effect on the general appearance, activity, and physical well being of six healthy men. The following signs of pulmonary irritation which occurred during the studies indicate that the tolerable human limitations to higher than normal oxygen concentrations may have been approached: substernal tightness, decrease in vital capacity in two subjects, and the occurrence of an area of probable atelectasis in one subject. Neither blood and urine studies, nor the measurement of pulse and respiration were of any conclusive help in determining the presence of oxygen toxicity. (*Michel, E., and others: Effect of Continuous Human Exposure to Oxygen Tension of 418 mm. Hg for 168 Hours, Aerospace Medicine 31: 138 (Feb.) 1960.*)

AUTONOMIC GANGLIA Experimental evidence supports the view that histamine 5-hydroxytryptamine and pilocarpine stimulate sympathetic ganglion cells of the cat by a mode of action different from that of acetylcholine and other nicotine-like substances. Therefore, these agents are described "non-nicotinic ganglion-stimulating substances." The ganglionic effects of these agents are abolished by depolarization of the ganglion cells but not by competitive blockade of the acetylcholine receptors. These substances seem to attach themselves to specific receptors of the ganglion cells. (*Trendelenburg, U.: Non-Nicotinic Ganglion-Stimulating Substances, Fed. Proc. 18: 1001 (Dec.) 1959.*)

CEREBROSPINAL FLUID The fate of drugs introduced directly into the cerebrospinal fluid of rabbits has been studied. Substances such as thiopental, introduced into the cerebrospinal fluid, find their way into the blood stream by either diffusion into the brain, by crossing the pia mater and the ependyma of the ventricles, or by flowing along the perivascular spaces, thence into the blood stream. A more important route, however, is by direct passage from the subarachnoid space into blood. (*Mayer, S. E, Maickel, R. P, and Brodie, B. B.: Disappearance of Various Drugs from the Cerebrospinal Fluid, J. Pharmacol. & Exper. Therap. 128: 41 (Jan.) 1960.*)