*ration of Apnea in Man, J. Pharmacol. & Exper. Therap. 120: 203 (June) 1957.)*

**CURARE** When topically applied to the exposed central nervous system of the cat, *d*-tubocurarine is shown to block preferentially the inhibitory synapses of the spinal cord and brain stem reticular system. This drug potentiates the action of strychnine at these sites and also the indirect development of cerebellar tetanus. (*Purpura, D. P., and Grundfest, H.: Physiological and Pharmacological Consequences of Different Synaptic Organizations in Cerebral and Cerebellar Cortex of Cat, J. Neurophysiol. 20: 194 (Sept.) 1957.*)

**NEUROMUSCULAR BLOCK** Experiments indicate that neuromuscular block produced by acetylcholine and succinylcholine is due mainly to desensitization, that is, a condition in which the end-plate has become refractory to depolarizing agents, and from which it recovers only slowly after complete withdrawal of the drug. It is suggested that this change arises from gradual transformation of the drug-receptor compound into an inactive form. (*Katz, B., and Thesleff, S.: Study of Desensitization Produced by Acetylcholine at Motor End-plate, J. Physiol. 138: 63 (Aug. 29) 1957.*)

**CURARE ANTAGONISTS** Potentiation of acetylcholine and antagonism of tubocurarine by three antagonists was investigated. The time-effect curves were casually related to the differences in the kinetics in the inhibitor-cholinesterase combination and dissociation. (*Smith, C. M., Mead, J. C., and Unna, K. R.: Antagonism of Tubocurarine; Time Course of Action of Pyridostigmine, Neostigmine and Edrophonium in Vivo and in Vitro, J. Pharmacol. & Exper. Therap. 120: 215 (June) 1957.*)

**HYPOXIA** Adrenalectomized dogs with a total preganglionic sympathetic block were made hypoxic by breathing 2.7 per cent oxygen. This group was compared with a normal group made hypoxic by breathing the 2.7 per cent oxygen. The unblocked animals showed an even greater increase in serum lactate and potassium than the blocked animals, indicating the role of epinephrine and norepinephrine in producing a more pronounced effect under the stress of hypoxia. (*Greene, N. M., and Phillips, A.: Metabolic Responses of Dogs to Hypoxia in Absence of Circulating Epinephrine and Norepinephrine, Am. J. Physiol. 189: 475 (June) 1957.*)

**STRESS ADAPTATION** Although the sympathoadrenal and adrenocortical systems exhibit many similar peripheral sites of action, one canot be substituted for the other in the regulation of adaptive reactions. Epinephrine will not restore blood pressure to normal in adrenocortical insufficiency; nor will cortical steroids prevent the postural hypotension of sympathectomy or sympathetic blockade. Following an intensive review of the action and interrelationship of the sympathoadrenal and the adrenocortical systems, the conclusion is that physiologically they appear to operate largely as a single functional unit. (*Ramey, E. R., and Goldstein, M. S.: Adrenal Cortex and Sympathetic Nervous System, Physiol. Rev. 37: 155 (April) 1957.*)

**CORTISONE** The effect of cortisone on the healing of aortic homografts was studied in dogs. The cortisone did not affect the healing or the incidence of thrombosis in the grafts. The size of the thrombus was larger in the dogs receiving cortisone. (*Kroboth, F. J., and others: Effects of Cortisone on Healing of Aortic Homografts, Surgery 42: 347 (Aug.) 1957.*)

**ALDOSTERONISM** Postoperative transient aldosteronism occurs in patients following surgery who also have sodium retention and potassium loss. By the time the sodium-potassium ratio in the urine returns to normal, the aldosterone concentration has also returned to normal. Increased production by the adrenal cortex and decreased destruction by the liver may be the cause of postoperative transient aldosteronism. (*Glaurado, J. G., and Woodruff, M. F. A.: Postoperative Transient Aldosteronism, Surgery 42: 313 (Aug.) 1957.*)

**LIGHTING** Lighting engineers suggest special filters for surgical luminaries to