attributed to the drug. The authors advocate adequate preoperative atropine (0.6 mg. with preoperative medication and again intravenously one minute prior to the start of anesthesia) the vapor concentration should not exceed 2 per cent in oxygen. (*Brennan, H. J., Hunter, A. R., and Johnstone, M.: Halothane: Clinical Assessment, Lancet 2: 453 (Sept. 7) 1597.*)

FLUOTHANE Advantages of halothane (Fluothane) as a general anesthetic agent include smooth induction, excellent relaxation, minimum of postoperative nausea and vomiting, noninflammability, and feasibility for use in a closed circuit apparatus. Its disadvantages include respiratory depression, arterial hypotension, potentiation of tubocurarine, and antagonism of suxamethonium. (*Marrett, R. H.: Halothane: Its Use in Closed Circuit, Brit. M. J. 2: 331 (August 10) 1957.*)

FLUOTHANE Halothane (trifluorochlorobromethane) has a distinct hypotensive action, due perhaps to a depression of central vasomotor centers combined with diminished cardiac output. (*Burns, T. H. S., and others: Clinical Investigations of Fluothane, Brit. M. J. 2: 483 (August 31) 1957.*)

FLUOTHANE Advantages of the drug include noninflammability and nonexplosiveness; rapid, smooth and pleasant induction; rare occurrence of coughing and breath holding; early relaxation of masseter muscles whereupon laryngoscopic stimulation of the cords results in closing of the glottis, possibly a cough, but not laryngeal spasm. Fluothane appears to be safe with small quantities of epinephrine injected subcutaneously, but this requires further investigation. Recovery from anesthesia is quick and associated with a minimum of nausea and vomiting. Disadvantages include depressed respiration, hypotension and bradycardia. Succinylcholine may be used with Fluothane with effects identical to those obtained when this relaxant is combined with other anesthetic agents. However, gallamine triethiodide and particularly *d*-tubocurarine chloride tend to aggravate the usual hypotension. Such observations suggest that Fluothane may exert some influence on the sympathetic nervous system, either at the ganglia or at some more peripheral site. (*Bryce-Smith, R., and O'Brien, H. D.: Some Observations on Fluothane, Proc. Roy. Soc. Med. 50: 193 (March) 1957.*)

ECLAMPSIA Reduction of hypertension in eclampsia to levels approximating 140/90 is recommended until pregnancy is terminated, using such vasodepressors as hydralazine or Apresoline in 10 to 50 mg. intravenous doses. The use of spinal or caudal anesthesia to reduce blood pressure is not advised, since it produces a regional rather than general vasodilatation and may enhance tissue hypoxia in areas of pooling. (*Best, B. D.: Toxemias of Pregnancy: Diagnosis and Management, Canad. M. A. J. 77: 505 (Sept. 1) 1957.*)

RESERPINE FOR TOXEMIA In 303 cases of toxemia managed with reserpine (and secondarily hydralazine) to control peripheral vasoconstriction and hypertension, good supportive measures, minimal sedation, early induction of labor in severe cases, and regional anesthesia for delivery, there were no maternal deaths and only mild side effects. The fetal salvage rate appeared to be higher than that reported by others except for the rate in eclampsia which is similar to that reported elsewhere. Reserpine effects noted in 10 per cent of the newborn were lethargy, depressed Moro reflex, nasal congestion, bradycardia, excessive secretions, and later increased frequency of bowel movements. (*Desmond, M. M., and others: Management of Toxemia of Pregnancy with Reserpine, Obst. & Gynec. 10: 140 (August) 1957.*)

APNEA Succinylcholine was repeatedly administered to 60 male patients as an adjunct to electroshock therapy. Serum cholinesterase activity was determined by ultraviolet spectrophotometry. Responsitivity to succinylcholine was found to be related to dose, esterase level, age and body weight of patient. The correlation with esterase activity appeared important. (*Kalow, W., and Gunn, D. R.: Relation Between Dose of Succinylcholine and Du-*

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. (*Parpura, 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 cannot 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