pine have consistently demonstrated lack of correlation between pharmacological effects and tissue concentrations. Labeled reserpine was found to be present in brain and liver throughout and beyond the entire period of observable pharmacological response. The concentration in the brain varied little during the entire period of observable activity and bore no relationship to the intensity of response. It is suggested to look to some tissue other than the brain as the primary site of action of reserpine. (*Sheppard, H., and others: Brain Reserpine Levels Following Large and Small Doses of Reserpine-H[3], Proc. Soc. Exper. Biol. & Med. 97: 717 (April) 1958.*)

NEUROMYAL BLOCK Histamine administered to cats by intraarterial injection is capable of reversing a depolarizing type of neuromuscular block and of potentiating a nondepolarizing type block. Histamine injected intravenously does not produce this effect, presumably due to either dilution or plasma protein binding. No explanation for the mechanism of action of this agent is available, although it is interesting to note that the substance can cause a marked release of potassium from cells. (*Schenk, E. A. and Anderson, E. G.: Effect of Histamine on Neuromyal Blocking Agents, J. Pharmacol. & Exper. Therap. 122: 234 (Feb.) 1958.*)

DIGITALIS The pharmacological response to digitalis appears to be antagonized by potassium chloride in spite of the fact that the LD_{50} for potassium chloride is less in the presence of digitalis than it is in normal dogs. An effect due to the anion present with the potassium is possible. *Keyl, A. C.: Digitalis Antagonism, Part I, A. M. A. Arch. of Int. Med. 101: 849 (May) 1958.*)

BARBITURATE POISONING The respiratory arrest dose (RAD) of pentobarbital sodium in cats given no specific treatment was 69 mgs./Kg. of body weight. Artificial respiration alone supported life in animals until doses of barbiturate averaging 4.3 times the RAD had been given. Treatment with neosynephrine in addition to artificial ventilation sustained life until 6.7 times the RAD had been given. In another series of animals, picrotoxin reinstated spontaneous respiration up to but not beyond 2.0 times the RAD. Cardiac arrhythmias appeared in three-fifths of the animals who remained apneic during picrotoxin treatment. Convulsions were frequent. Mikedimide (Megimide) reinstated spontaneous respiration in animals who had received pentobarbital up to, but not beyond, 1.4 times the RAD. Cardiac arrhythmias appeared in one-fifth of all animals treated with Mikedimide. Convulsions appeared in two-thirds of the animals who remained apneic. (*Lavenson, G. S., Jr., Plum, F., and Swanson, A. G.: Physiological Management Compared with Pharmacological and Electrical Stimulation in Barbiturate Poisoning, J. Pharmacol. & Exper. Therap. 122: 271 (Feb.) 1958.*)

THIOPENTAL SHOCK Sudden release of large amounts of catechol-amines after administration of Pentothal in patients with pheochromocytoma has been found to precipitate a peculiar form of peripheral shock. There is tachycardia, sweating, pallor, cyanosis and absence of peripheral pulses, but there is no evidence of respiratory failure, and central arterial pressure is elevated with an extremely narrow pulse pressure. (*Marc-Aurele, J., and others: Peculiar Form of Clinical Shock, Canad. M. A. J. 78: 589 (April 15) 1958.*)

SHOCK AND ACIDOSIS Two healthy human subjects were given intravenous epinephrine during eupnea (arterial pH 7.37), during 10 per cent carbon dioxide rebreathing (arterial *p*H 7.23), and during voluntary hyperventilation (arterial pH 7.61). Increments in heart rate and arterial pressure following epinephrine injection were greatest during respiratory alkalosis and least during respiratory acidosis.

Clinical patients with septicemia and shock who had become refractory to pressor agents were studied. When refractory to the pressor effects of sympathomimetic amines, arterial blood studies demonstrated metabolic acidosis with arterial blood pH values as low as 7.06. Correction of acidosis by intravenous administration of molar sodium lactate resulted in an increased pressor response to sympathicomimetic