and of more than 12 hours duration, rapid rewarming may lead to hypotension. In the latter case, rewarming is preferably by patient's own metabolic efforts at about the rate of one degree Fahrenheit per hour. (*Rees, J. R.: Accidental Hypothermia, Lancet 1: 556 (March 15) 1958.*)

LOCALIZED HYPOTHERMIA Localized cerebral hypothermia was studied in a series of 45 dog experiments with the aid of extracorporeal circulation utilizing the carotid arteries and external jugular veins. Various parameters of study were recorded in the acute stage of perfusion and, in general, were in accord with those noted in states of generalized hypothermia. Survival of dogs depended on low perfusion flows and the development of a perfusion pressure closely aligned with the systemic arterial pressure. Changes in electrocortical activity of the brain due to localized cerebral hypothermia were identical to hibernation. There was little change in the arteriovenous oxygen differences of the cerebral and systemic circulations unless the animal's body temperature at the stage of prewarming fell below a level of 34 C. (*Woodhall, B., and others: Physiologic and Pathologic Effects of Localized Cerebral Hypothermia, Ann. Surg. 147: 673 (May) 1958.*)

HYPOTHERMIA Mephentermine was injected and dogs were cooled by immersion in a cold water bath to a rectal temperature of 25 C. The incidence of hypothermic ventricular fibrillation was reduced significantly from 91 per cent in 33 control dogs to 37 per cent in 19 dogs treated with mephentermine. (*Covino, B. G.: Antifibrillary Effect of Mephentermine Sulfate (Wyamine) in General Hypothermia, J. Pharmacol. & Exper. Therap. 122: 418 (March) 1958.*)

VENTRICULAR FIBRILLATION Drug combinations were used to prevent ventricular fibrillation in dogs under hypothermia and following induced cardiac arrest. Acetylcholine was used to produce the arrest. Under hypothermia the magnesium ion increases irritability instead of decreasing it. Quinidine was the most effective agent in preventing ventricular fibrillation. (*Berman, E. J., and others: Experimental Prevention of Ventricular Fibrillation Following Hypothermia and Induced Cardiac Arrest, J. Thoracic Surg. 35: 483 (April) 1958.*)

ELECTROLYTES IN HYPOTHERMIA Dogs subjected to immersion hypothermia were studied in an effort to relate the occurrence of ventricular fibrillation to certain other observable phenomena. The studies failed to show any direct relationship between the occurrence of fibrillation and (1) serum potassium concentration or ratios of other electrolytes, (2) blood *p*H and respiratory pattern, (3) attempted prevention of hypokalemia, or (4) pretreatment with magnesium and/or insulin. No characteristic changes in the electrocardiogram presaging the occurrence of fibrillation were observed. (*Frank, E. A., and Carr, M. H.: Adaptive Changes in Hypothermia with Special Reference to Electrolyte Alterations, Experimental Study, West J. Surg. 66: 105 (March–April) 1958.*)

HYPOTHERMIA EQUIPMENT A collapsible tub is fashioned from a large sheet of heavy plastic material, folded with boxlike corners to conform to the width and length of the operating table. Stainless steel posts and rods form a rigid frame for the tub. Hypothermia blankets may be used in conjunction with the collapsible tub. When the temperature has been lowered to the proper degree, the water can be emptied quickly, the sides of the tub lowered, and there is no need to move the patient from the tub to the operating table. (*Holswade, G. R.: Collapsible Tub for Immersion Cooling on the Operating Table, Surg. Gynec. & Obst. 106: 502 (April) 1958.*)

ATARACTIC COMPLICATION Two children developed a cataleptoid status following three 4 mg. doses of Trilafon (perphenazine). (*Berry, R. V., Kamin, S. H., and Kline, A.: Trilafon Complication, U. S. Armed Forces M. J. 9: 745 (May) 1958.*)

BRAIN RESERPINE LEVELS Previous studies on rate of distribution of reser-

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-H³, 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: 231 (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 *p*H 7.37), during 10 per cent carbon dioxide rebreathing (arterial *p*H 7.23), and during voluntary hyperventilation (arterial *p*H 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 *p*H values as low as 7.06. Correction of acidosis by intravenous administration of molar sodium lactate resulted in an increased pressor response to sympathicomimetic