cerebrospinal fluid pressure generally fell as body temperature was reduced. However, the pressure varied depending upon the depth of anesthesia, anoxia, shivering, coughing on the endotracheal tube, and possibly the level of blood pressure. The idea that hypothermia is a practical method for reducing markedly increased intracranial pressure secondary to a space occupying lesion has not been substantiated. (*Lemmen, L. J., and Davis, J. S.: Studies of Cerebrospinal Fluid Pressure During Hypothermia in Intracranial Surgery, Surg. Gynec. & Obst. 106: 555 (May) 1958.*)

HYPOTHERMIA A prompt return to a normal cardiovascular status parallel with the elevation of temperature does not universally accompany rewarming following hypothermia in dogs. The time for readjustment and the mortality become more significant the more prolonged the hypothermia, even though the temperature remains stable. Animals are in a fine state of balance with restoration of normal cardiovascular hemodynamics following rewarming. However, stresses such as anesthesia may be detrimental and should be avoided. The picture of "rewarming shock," following 8 hours of hypothermia under the conditions of these experiments, was an extremely uncommon finding. Prolonged hypothermia must be avoided. The longer the period of cooling, the more complex are the deviations from normal and the more difficult the restoration to a state of normal hemodynamics. At present, there is no unequivocal experimental evidence to abandon the use of hypothermia even though undesirable sequellae and profound physiologic alterations may be expected. (*Fedor, E. J., Fisher, B., and Lee, S. H.: Rewarming Following Hypothermia of Two to Twelve Hours: I. Cardiovascular Effects, Ann. Surg. 147: 515 (April) 1958.*)

HYPOTHERMIA The response to overload of the heart and its arrest at low temperature were studied in thirteen experiments on heart-lung preparations in dogs. When the blood temperature was lowered to 32–35 C., the heart, given an increase in the influx of venous blood, showed an increase in the output of blood. At a temperature of 30 C. the frequency of heart contractions declined markedly and the amplitude of fluctuation of the arterial blood pressure increased. The total output of blood from the heart per unit of time remained constant. At a temperature of about 25 C. the frequency of heart contractions decreased to 60–70/minute, a frequency quite adequate for maintaining the blood flow. When the blood reached a temperature of 22–23 C. the heart became dilated and its insufficiency became manifest, thus leading to gradual increase of venous pressure and to further dilatation of the heart. The functional inability of the heart to pump over the total quantity of the inflowing blood progressed with further lowering in the temperature and sometimes the heart stopped at 17–23 C. Parallel with the lowering of the temperature and reduction in the frequency of heart contractions, marked changes in the electrocardiogram appeared, particularly concerning the ventricular complex. The maximum electrocardiographic changes were seen at the very lowest temperatures of the heart. (*Starkov, P. M.: Response to Overload of Heart and Cardiac Arrest at Low Temperatures, Eksper. Khir. 4: 9, 1956.*)

HYPOTHERMIA In two patients aged 19 and 4 years, a severe, not understood complication of narcosis during hypothermia arose—a massive hemorrhage into the lung parenchyma. The onset of hemorrhage was connected with introduction of the intubation tube into the lumen of the right bronchus and complete blocking off of the left lung. (*Darabinyan, T. M., and Krymskii, L. D.: Massive Hemorrhage into Lung as Complication of Narcosis by Intubation During Hypothermia, Vestn. Khir. 78: 123, 1957.*)

ACCIDENTAL HYPOTHERMIA Four patients in whom accidental hypothermia developed with rectal temperatures of 80 to 90 F. are reported. The treatment of accidental hypothermia varies with its duration, and may call for either rapid or slow rewarming. In acute hypothermia of rapid onset and less than 12 hours duration, rapid rewarming is recommended. In the chronic hypothermia of slower onset

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 Complications, U. S. Armed Forces M. J. 9: 745 (May) 1958.*)

BRAIN RESERPINE LEVELS Previous studies on rate of distribution of reser