covery from cardioplegia and ventriculotomy. Data is presented which emphasizes the significance of neurogenic, metabolic and humoral factors in the regulation of myocardial contractility. (*Darby, T. D., and others: Influence of Cardio-Pulmonary Bypass with Cardiac Arrest and Right Ventriculotomy on Myocardial Contractile Force, Ann. Surg. 147: 596 (May) 1958.*)

CARDIOTOMY MONITORING The electrocardiogram and electroencephalogram are helpful monitors in the management of patients undergoing cardiotomy. The electrocardiogram affords immediate evidence of cardiac arrhythmias, the most serious of which are those resulting from high vagal tone or ventricular irritability. The energy output of the brain is revealed by the amplitude and frequency of the electroencephalogram tracing and is seen to decrease with deeper anesthesia, hypoxia, hypotension, hypothermia and hypercarbia. The electroencephalogram summates these effects and indicates changes during anesthesia and surgery which are not specific but indicate the need for reevaluation of the patient's essential physiological functions. (*Hale, D. E., and Moraca, P. P.: Electrocardiogram and Electroencephalogram in Elective Cardiac Arrest, J. A. M. A. 166: 1672 (April 5) 1958.*)

HYPOTHERMIA The purpose of hypothermia is to reduce body metabolism and thus diminish the need for oxygen. Hypothermia may be achieved by external surface cooling (ice water immersion, ice bags, cooling blanket); internal surface cooling (cold solutions applied to the open chest or aorta, or cold fluids circulated through the stomach or rectum); and by cooling the patient's blood externally and returning it either into his arterial or venous circulation. Shivering is inhibited by anesthetics, muscle relaxants, and chlorpromazine. Hypothermia affects many physiologic processes. (1) *Metabolism.*—Oxygen consumption decreases with temperature, but the effect of this reduction upon organ function is variable. In the liver, detoxification of drugs may be prolonged far out of proportion to diminution in oxygen consumption. (2) *Cardiovascular.*—Blood pressure and pulse rate diminishes with temperature. Blood flow and oxygenation of tissues are adequate. The electrocardiographic effects include: decreased amplitude or absent P waves, increased QRS interval, increased length of ST segment, and prolongation or inversion of T waves. Below 28 C varying degrees of heart block, ventricular extrasystoles and nodal rhythms may appear. Ventricular fibrillation is uncommon provided the heart is not manipulated or operated on and correct electrolyte balance and oxygenation is maintained. (3) *Respiration.*—Although reasonably normal respiration can persist to well below 28 C., hypothermia increases the dead space and the oxygen dissociation curve is shifted to the left. (4) *Central nervous system.*—In many, electrical cortical activity progressively diminishes until at about 18 C. electrical silence ensues. The cerebrospinal fluid pressure decreases, the brain contracts and seems to be less vulnerable to operative trauma. (5) *Renal function.*—Urinary output may be increased at 25 C. Reabsorption at the distal tubule is unchanged and excretion of water and sodium is unaltered. (*Eckenhoff, J. E.: Physiology of Hypothermia, Bull. New York Acad. Med. 34: 297 (May) 1958.*)

HYPOTHERMIA Total body cooling in man to 28–30 C. during thiopental-curare anesthesia and hyperventilation was associated with arterial blood electrolyte changes similar to those observed during respiratory alkalosis without hypothermia. Metabolic acidosis did not occur during uncomplicated hypothermia. Shivering, occlusion of major blood vessels and the transfusion of routinely collected blood (citrated) led to moderate to severe metabolic acidosis. (*Henneman, D., and others: Immediate Metabolic Response to Hypothermia in Man, J. Appl. Physiol. 12: 164 (March) 1958.*)

HYPOTHERMIA Observations of cerebrospinal fluid pressure were made in 32 patients during intracranial surgery. Anesthesia consisted of thiopental and nitrous oxide with succinylcholine. Operation began when the rectal temperature was 30 C. Cerebrospinal fluid pressure uniformly rose during the induction of anesthesia and the insertion of the endotracheal tube. The

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 sequellne 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. 1: 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