brovascular accidents, and for postoperative recurrences. Of patients who had a preoperative coronary occlusion less than 24 months prior to surgery, 46 per cent had a postoperative occlusion. Of patients operated on within six months of coronary occlusion, 100 per cent developed a postoperative occlusion. Although it is not possible to establish an exact period of time following an initial coronary occlusion when operation could be performed with reasonable safety, statistical analysis of the data definitely concludes that the shorter the interval from the preoperative occlusion to operation, the greater the hazard of recurrence. The data for cerebrovascular accidents appear to be in the same direction as that for coronary occlusions. Statistical analysis failed to indicate that the anesthetic agent itself played a role in the incidence of postoperative coronary occlusion or cerebrovascular accidents. Similarly, the nature of the operation could not be implicated. (*Knapp, R. B., and others: The Cerebrovascular Accident and Coronary Occlusion in Anesthesia. J. A. M. A. 182: 332 (Oct. 27) 1962.*)

IRREVERSIBLE SHOCK Hemodynamic and biochemical techniques were used to study 15 patients in severe refractory hypotension. These 15 patients failed to respond to an initial blood volume restoration and remained in a profound state of shock with a potential fatal outcome; 12 survived. Four groups of patients were discernible from this study, those with: (1) unrecognized blood-volume deficit with hyponatremia or acidosis or both, (2) severe sepsis, (3) myocardial failure, and (4) local vascular complications of extensive operations. Hyponatremia and metabolic acidosis were the two most common biochemical changes observed. The intravenous administration of corticosteroids in large doses failed to improve the condition in any patient. Continuous observation of central venous pressure, hourly urine output, skin color and capillary blood flow were more important than blood volume measurements as indices of tissue perfusion and blood volume adequacy. Postmortem examination of the patients who died indicated adequate anatomic explanation for death in every instance. Gastrointestinal bleeding was absent. (*Smith, L. L., and Moore, F. D.: Refractory Hypotension in Man. Is This Irreversible Shock? New Engl. J. Med. 267: 733 (Oct. 11) 1962.*)

SHOCK The usual cause of shock occurring only after the induction of anesthesia and the initiation of a surgical procedure is inadequate preoperative fluid replacement. The patient was maintaining a normal blood pressure only with considerable vasoconstriction. When anesthesia reduced the vasoconstriction the blood pressure declined. (*Barnett, W. O. and Hardy, J. D.: Shock and Peritonitis, Surg. Clin. N. Amer. 42: 1101 (Oct.) 1962.*)

VENOUS PRESSURE Continuous monitoring of central venous pressure is recommended as a guide for maintaining optimal blood volume. An infusion set, extension tube, and 3-way stopcock are the only other equipment required. The venous pressure indicates the relationship between blood volume and cardiac pump capacity. In shock of questionable origin, blood may be given rapidly if the venous pressure is below 6 cm.; if it is between 6 and 15 cm. blood should be given cautiously. If the venous pressure is over 14 cm. and rising cardiac failure is present; transfusion should be withheld and therapy should consist of cardiac stimulants, vasopressors, improved ventilation and correction of acidosis. (*Wilson, J. N., and others: Central Venous Pressure in Optimal Blood Volume Maintenance, Arch. Surg. 85: 563 (Oct.) 1962.*)

BUFFY COAT-POOR BLOOD Granulocytes can be removed efficiently from freshly drawn heparinized blood collected in either glass or plastic containers by passing it over a column of nylon fibers. The percentage reduction of leukocytes and platelets were 85 and 55 per cent, respectively, in a study of 82 bottles of blood. Twelve patients who had previously experienced pyrogenic transfusion reactions (because of sensitivity to leukocytes initiated by transfusions or pregnancy) were transfused. With the 82 transfusions, there were seven instances of fever but only three instances of chills. This technique eliminates differential centrifugation or sedimentation in the presence of dextran as used with other techniques to remove leukocytes. (*Greenwalt, T. J., Gajewski, M., and McKenna, J. L.: A*

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New Method for Preparing Buffy Coat-Poor Blood. Transfusion 2: 221 (July-Aug.) 1962.)

HYPOTHERMIA The dog will tolerate total cerebral ischemia for eight minutes at 37° C., for 25 minutes at 28° to 30° C., and for 60 minutes at 19° to 21° C. without evidence of permanent brain damage. The animal studies were further confirmed in two cases requiring neurosurgical intervention in man. In one instance the brain was cooled to approximately 20° C., with the body remaining at normal temperature and total cerebral occlusion being used for approximately ten minutes. The second patient was selectively cooled to a minimal level of 16° C. and circulation to the brain was completely occluded for 30 minutes. Both of these patients have recovered with no evidence of brain damage from either the brain cooling or the period of total cerebral ischemia. *(Boyd, R. J., and Connolly, J. E.: The Effect of Hypothermia in Experimental Cerebral Ischemia. Geriatrics 17: 522 (Aug.) 1962.)*

POSTOPERATIVE HYPOTHERMIA Reduction of body temperature may be of value for critically ill patients. Hypothermia is mild at 34° to 36° C., moderate at 28° to 33° C., deep at 17° to 27° C., and profound from 4° to 16° C. Bleeding from the gastrointestinal tract is reduced or stopped. Tachypnea is diminished, but effective coughing is impaired and tracheal suction is required. Renal function is satisfactory. Metabolism, cardiac output, cardiac rate, and blood pressure diminish. Defense mechanisms remain intact, but bacterial enzymatic processes and proliferative capacity are reduced. Hypothermia is a useful adjuvant in therapy. *(Hitchcock, C. R., and others: Use of Prolonged Moderate Hypothermia in Postoperative Care. Arch. Surg. 85: 549 (Oct.) 1962.)*

HYPOTHERMIA Because of the beneficial effect on the character and mortality of experimental brain injury, 21 patients with critical brain injury (thought to be incompatible with life, using standard methods of treatment) were subjected to artificial hypothermia (28° to 36° C.) for two to ten days. Nine died and 12 survived, but six of the survivors are permanent invalids with dementia. The hazards are staphylococcal pneumonia, which occurred in eight cases and contributed to all the deaths, and gastrointestinal ulceration with bleeding and perforation, which was fatal in another instance. The results in patients with clots did not differ from those without clots. Youth was the only common factor in the successful cases. Prognosis was hopeless in the presence of large fixed pupils. About one in four of these critical cases will fare well, but it is evident that a large proportion have such gross or microscopic tearing of deep cerebral structures that in the event of survival there will be severe mental and physical handicaps. *(Drake, C., and Jory, T.: Hypothermia in the Treatment of Critical Head Injury. Canad. Med. Ass. J. 87: 887 (Oct. 27) 1962.)*

CURARE AND HYPOTHERMIA Influence of lowered muscle temperature on maximal amplitude of the gastrocnemius muscle in cats was examined. Hypothermia above 23° C. had no influence; below this temperature, the maximal muscular tone shows linear reduction of about 10 per cent per degree centigrade to a temperature of 13° to 14° C., when total paralysis occurs. This course of events is reversible on rewarming. Halothane anesthesia under normothermic conditions, up to 3 volumes per cent, does not influence the reaction of muscle. Effect of *d*-tubocurarine was enhanced tenfold by increasing the halothane concentration from 1.5 volumes per cent to 3 volume per cent. Lowering the muscle temperature increases and prolongs the effect of succinylcholine. Maximal amplitude decreases in direct proportion to the lowering of temperature at a rate of 5 per cent per degree centigrade. *(Kucher, R.: Influence of Hypothermia on Neuromuscular Effects of Succinylcholine and d-Tubocurarine in the Cat. Der Anaesthesist 11: 317 (Oct.) 1962.)*

ACUTE PANCREATITIS Hypothermia is suggested for the treatment of acute hemorrhagic pancreatitis. The rationale is to reduce pancreatic activity. After producing the disease in dogs, 18 were kept normothermic, and 22 were made hypothermic (25° to 30° C.) for 12 to 24 hours. All other therapy was identical for both groups. Blood enzymes and electrolytes were similar in both groups. Con-

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