vestigated during the period of cooling and after rewarming. Cardiac output decreased 30 per cent at 25 C. (77 F.), peripheral resistance increased 35 per cent, blood pressure decreased slightly. After rewarming blood pressure returned to normal, peripheral resistance remained increased, cardiac output tended to become normal without reaching a normal level. That the cardiac output failed to return to normal may be due to either inadequate venous return or myocardial insufficiency or both. During the cooling phase the venous pressure was very variable and after rewarming was decreased in 3 dogs and normal in 6 others. (*Lacroix, E., and Leusen, I.: Hypothermia and Hemodynamics. Arch. internat. physiol. 66: 89 (Feb.) 1958.*)

HYPOTHERMIA Artificial hypothermia increases the resistance of the organism to trauma and lack of oxygen. It strengthens the protective inhibitory mechanisms, weakens untoward reflex reactions and lowers the basal metabolism and body needs for oxygen. Sometimes, however, the opposite results occur in cases with inadequate inhibition of the CNS. In these cases sedatives and neuroplegic preparation are used. Because of this danger it is necessary to maintain a close control of the functions of circulation, pulmonary ventilation and the demand for oxygen during the period of hypothermia. The authors consider that artificial hypothermia allows a great increase in the scope of operations on the heart and major vessels. The complicated technique and the lack of its general thorough experience do not allow of its being recommended for operations which can be successfully carried out with the use of simpler and safer methods of anesthesia. (*Kupriyanov, P. A., and others: Problem of Artificial Hypothermia in Cardiac Surgery, Klin. Med. 10: 3, 1956.*)

HYPOTHERMIA The cause of thrombocytopenia and leukopenia during induced hypothermia was studied in a group of 22 dogs. The thrombocytopenia was shown to be caused by sequestration rather than destruction of platelets. This conclusion was supported by P^{32} tagging of platelets and bone marrow studies. The major site of platelet sequestration was in the portal circulation. The liver and spleen were shown to play a major role in the sequestration but other sites must also be involved. Leukocyte counts tended to parallel those of the platelets but were less consistent. (*Villalobos, T. J., and others: Cause of Thrombocytopenia and Leukopenia that Occur in Dogs During Deep Hypothermia, J. Clin. Invest. 37: 1 (Jan.) 1958.*)

HYPOTHERMIA Splenectomized dogs dogs were subjected to a hemorrhage of 35 per cent of their measured blood volume and arranged in the following three groups: (I) dogs bled in the normothermic state as controls; (II) dogs cooled to 25 C. prior to hemorrhage; and (III) dogs bled while normothermic and then cooled to 25 C. All animals in Groups I and III survived; those in Group II suffered an 80 per cent mortality. Thus, hemorrhage in an already hypothermic dog is usually fatal, while hypothermia imposed on a dog just bled is not fatal with the hemorrhage volumes employed. Compensations to hemorrhage may occur in animals of Group III during the early cooling period or "grace period." The suggested value of hypothermia as a method of treatment for hemorrhagic shock is neither confirmed nor refuted by this study, but doubt is cast upon the rationale of its use. The hypothermic dog is unable to survive a hemorrhage volume which is non-fatal to the normothermic dog. Therefore, during hypothermic surgery, blood volume deficits are to be avoided. (*Ferguson, A. T., and others: Effect of Hypothermia on Hemorrhagic Shock, Ann. Surg. 147: 281 (Mar.) 1958.*)

MEASURING BLOOD LOSS Blood loss is determined from a water filled tub containing an agitator which extracts the electrolytes from blood sponges and from suction. A conductivity bridge measures changes in conductance. A servo mechanism continuously brings the bridge into balance. The ease and accuracy of measurement with the automatic blood loss meter surpasses both the hemoglobin and