kilogram resulted in ventricular fibrillation. Clinical investigation of this vasopressor drug is proceeding. In dosages of 6.6 μg. intravenously, a marked and consistent, but short-lived (seven to 10 minutes), elevation in systolic and diastolic blood pressure is produced. A moderate slowing of the pulse rate (about 10 per minute) is noted. The respiration and mental status are unaltered under spinal analgesia. No arrhythmias have been noted under cyclopropane anesthesia. The blood pressure of three patients in shock (non-hemorrhagic) has been regulated satisfactorily with a continuous drip solution of 4.0 mg. in 500 ml. for periods up to 26 hours. [Supported in part by a grant from Ciba Pharmaceutical Products, Summit, New Jersey.]

Profound Hypothermia Induced by Direct Blood Stream Cooling. RICHARD A. THEYE, M.D., AND BRIAN DAWSON, M.B., *Section of Anesthesiology, Mayo Clinic, Rochester, Minnesota.* In 1959 Shields (*Shields, T. W., and Lewis, F. J.: Surgery 46: 164, 1959*) and Drew (*Drew, C. E., and Anderson, I. M.: Lancet 1: 748, 1959*) simultaneously and independently introduced a technique for rapidly cooling dogs down to 15 C. with prolonged periods of circulatory arrest *without* the need of an artificial oxygenator. The technique requires a left heart (left atrium–femoral artery) and a right heart (right atrium–pulmonary artery) bypass circuit. A heat exchanger in the left heart circuit provides temperature control. Cooling is started by "going on" left heart bypass. As the cooled right heart fails, right heart bypass is initiated in order to maintain a flow of oxygenated blood to the left atrium. Right and left heart bypass and cooling are continued until the desired temperature is reached. At this point both perfusions are stopped. Rewarming is accomplished by the same steps in reverse order. Application of this technique in our laboratory resulted in long term (3 weeks) survivors in 9 of 10 dogs subjected to 30 minutes of circulatory arrest at 15 C. (*Lim, R. A., and others: Surgery, in press*). Twenty-three patients have undergone intracardiac repair with this technique. Premedication was omitted and only inhalation agents were used. Two patients, each with mitral stenosis and insufficiency and arrested 50 minutes at 15 C., died within several minutes of cessation of left heart bypass and rewarming. A 7 year old patient with transposition died 6 days postoperatively without apparent central nervous system deficiencies after having been without circulation for 74 minutes at 11 C. A 6 year old patient with preoperative common atrium, complete heart block, and cardiac failure died 4 hours after leaving the operating room. All other patients survived including 12 with 13 to 19 minutes of circulatory arrest at 22 C., 5 with 30 minutes of arrest at 15 C., and 2 with 45 minutes of arrest at 15 C. In one of these two patients a pseudobulbar palsy appeared on the second postoperative day. No other significant complications were observed in the 19 survivors. In addition to this experience with the "Drew" technique, hypothermia and circulatory arrest has been carried out with the Mayo-Gibbon pump-oxygenator in 29 patients. The major advantage of circulatory arrest is a quiet, bloodless intracardiac operative field. The great hazard is neurologic damage. Björk (*J. Thor. & Cardiovasc. Surg. 40: 237, 1960*) reported abandoning the "Drew technique" after observing "death due to brain damage" in 3 of 10 patients subjected to circulatory arrest (41 minutes at 10 C., 50 minutes at 10 C., and 70 minutes at 6 C.). Gordon (*Am. J. Surg. 100: 332, 1960*) after reporting 21 clinical cases without neurologic sequelae states that the Drew technique is the "simplest, safest, and most efficient technique for intracardiac surgery at the present time." The passage of time will be required to fully assess the role of this new, provocative approach to open intracardiac surgery.

The Effect of Hemorrhagic Hypotension on Evoked Potentials in the Spinal Cord. REX J. UNDERWOOD, M.D., WILLIAM W. KRIPPAEHNE, M.D., FREDERICK P. HAUGEN, M.D., AND CLARE G. PETERSON, M.D., *Department of Anesthesiology, University of Oregon Medical School, Portland, Oregon.* Spinal cord dorsum and ventral root reflex potentials were obtained from the spinal cords of dogs by stimulating a lumbar dorsal root with single shocks just above threshold. Light pentobarbital anesthesia was used and the animals were given *d*-tubocurarine or succinylcholine to pre-

vent skeletal muscle activity. After adequate control records were obtained, the dogs were subjected to hemorrhagic shock by the method of Wiggers (*Wiggers, C. F.: Physiology of Shock.. Commonwealth Fund, New York, 1950*). The effects on the evoked potentials from the spinal cord were as follows: The axonal response from the cord dorsum was unchanged even with extreme hypotension. In several instances, the axonal response remained for 5 to 10 minutes after cessation of heartbeat. The negative potential from the cord dorsum and the polysynaptic response from the ventral root were both depressed when the blood pressure was lowered to 50 mm. Hg and still further depressed at 35 mm. Hg. Reinfusion of the withdrawn blood caused some return of these responses, but never to control levels. Both were absent when the blood pressure was less than 25 mm. Hg. The monosynaptic spike from the ventral root resulting from single shock stimulation was seen in the control period only in animals given *d*-tubocurarine. In animals not given *d*-tubocurarine, the monosynaptic spike could always be evoked by causing a conditioning stimulus to occur 3 to 6 msec. before the testing stimulus. Hypotension caused a brief increase of the spike, followed by moderate depression. Reinfusion usually caused return of the spike to control levels, followed by decreasing amplitude as the blood pressure diminished. The so-called "detonator" type of monosynaptic response from the ventral root was seen only when there was a precipitous drop in blood pressure to levels of 10 to 20 mm. Hg. The "detonator" response consisted of a marked increase in amplitude of the monosynaptic spike lasting for 1 or 2 minutes, followed by disappearance. Tentative conclusions based on the above data are that the polysynaptic pathways in the ventral root reflex and the pathways responsible for the negative potential in the cord dorsum response are both highly susceptible to depression by hemorrhagic hypotension. Further, the monosynaptic response in the ventral root reflex is made transiently hyperexcitable by hypotension, followed by severe depression. Finally, there seems to be evidence that these changes in the evoked spinal cord potentials persist, even though the blood pressure is temporarily normal following reinfusion.

A Study of Neostigmine as a Curare Antagonist. D. A. Wescott, M.D., and H. H. Bendixen, M.D., *Anesthesia Laboratory of the Harvard Medical School, Massachusetts General Hospital, Boston.* Present methods of evaluating the reversal of curare effect by neostigmine are mainly based on arbitrary clinical judgment. In an attempt to establish more objective criteria as a basis for the clinical use of neostigmine, the inspiratory force measurement as an index of the return of respiratory muscle power after curare is being investigated. The inspiratory force is defined as the maximum negative pressure exerted against the completely occluded airway. The purpose of the study is to attempt an evaluation of the routine use of neostigmine with an objective determination of optimum timing and dosage; also an investigation of the side effects of atropine/neostigmine administration, and an examination of the changes in blood gases during airway occlusion and inspiratory force measurement. Healthy adult patients undergoing abdominal operations are being studied. Premedication is with pentobarbital and atropine, anesthesia is induced with thiopental and succinylcholine is used for intubation. Maintenance is with nitrous oxide 2 liters and oxygen 1 liter, and *d*-tubocurarine in doses large enough to produce profound relaxation and apnea throughout the procedure. The radial artery is cannulated for collection of blood samples and for monitoring of the blood pressure. This is recorded during the study by a Sanborn Polyviso recorder, together with electrocardiogram and electroencephalogram. The inspiratory force is measured by a pressure transducer and also recorded by the Polyviso. At the end of the operation atropine 1 mg. is given intravenously. When a rise in pulse rate has been noted an inspiratory force measurement is recorded during a 30 second period of airway occlusion. A dose of neostigmine 1 mg. is then given intravenously and the inspiratory force measurement repeated 2 and 5 minutes later. Repeated doses of neostigmine 1 mg. are given, each dose followed by inspiratory force measurements, until no further increase in inspiratory force can be ob-