

hours duration at a temperature of 31° C., with adequate red cell replacement, showed a further decrease in the rate of erythropoiesis in the immediate postoperative period as measured by a Fe-59 half-life of 134 minutes. To establish whether the underlying cause for the decrease in rate of erythropoiesis was due to hypothermia or anesthetic agents, a small group of normothermic patients was studied in the immediate postoperative period. The latter group showed no decrease in erythropoiesis—confirming that the reduction observed in the hypothermic patient is due to the subthermic state. *Summary:* The comparison of Cr-51 and Fe-59 isotope tracer studies preoperatively and postoperatively, substantiated the fact that Fe-59 half-life was satisfactory for measurement of erythrokinetics, and in this series, the increased rate of red cell production was synonymous with a reduction in the total body hemoglobin. [The Ferrutop was supplied by E. R. Squibb & Sons. The project was supported by an Atomic Energy Commission Grant, AT(30-1) 2486, and The American Red Cross.]

**Effects of Experimental Subarachnoid Perfusion Cooling and Rewarming on the Spinal Cord.** MAURICE S. ALBIN, M.D., ROBERT J. WHITE, M.D., DAVID E. DONALD, Ph.D., COLLIN S. MACCARTY, M.D., and ALBERT FAULCONER, JR., M.D., *Mayo Clinic and Mayo Foundation, Rochester, Minnesota.* It was thought that an isolated selective technique to reduce spinal-cord temperature would provide information on the effect of cold on nervous tissue in an intact biologic preparation, provide a useful method for physiologic studies, and have clinical application in the treatment of spinal-cord trauma. *Method:* By a preferential cooling technique, 13 dogs were perfused via a plastic catheter inserted beneath and sealed in the dura following laminectomy at each site. Cold isotonic sodium chloride at 5.0° C. was used to perfuse 8 dogs from T-4 to L-5 levels, and two dogs from C-3 to L-5 levels. Three dogs served as controls: two were perfused with isotonic sodium chloride at normal cord temperatures from T-4 to L-5 levels, and one from C-3 to L-5 levels. Perfusion was achieved by gravity; flow rate was 10.0 ml./minute. Temperatures

were recorded with needle thermometers at inflow and outflow catheters and within the cord during perfusion. Rectal temperature, mean arterial pressure, and heart rate were monitored. In 7 animals, brain temperatures were taken with needle thermometers inserted through bilateral burr holes. *Results:* In the 8 dogs perfused from T-4 to L-5 levels, cord temperature was reduced to a mean of 14.4° C. at L-5 level and to a mean of 10.6° C. at T-4 level after an average of 68.3 minutes of perfusion. In the two dogs perfused from C-3 to L-5 levels, cord temperature reached a mean of 13.0° C. at L-5 level and a mean of 9.6° C. at C-3 level after an average of 40 minutes of perfusion. Cords of 4 animals previously cooled were allowed to rewarm spontaneously by immediately stopping perfusion. These cords reach preperfusion temperatures in an average of 13.2 minutes. On recooling, they reached lower temperatures sooner, averaging only 33 minutes of perfusion. In the three controls, cord temperature was unchanged after 2 hours of perfusion. Brain temperature in four cold-perfused animals declined 2.5°, 2.5°, 3.5°, and 4.0° C. respectively from levels observed before perfusion, but remained unchanged in controls. Rectal temperatures did not fluctuate significantly during perfusion in any animals. Three dogs perfused from T-4 to L-5 levels and two perfused from C-3 to L-5 levels showed significant fluctuations in both heart rate and mean arterial pressure during cooling. To test the effects of profound cooling of the cord on long-term survival, two animals were perfused from T-4 to L-5 levels for one hour; cord temperatures of 17.5° C. and 11.0° C. at L-5 and 10.5° C. and 8.5° C. at T-4 level were reached. The animals recovered completely with no subsequent neurologic damage.

**Effects of Morphine and Curare on the Respiratory Response Curve.** J. WELDON BELLVILLE, M.D., and ELLIS N. COHEN, M.D., *Department of Anesthesia, Stanford University Medical School, Palo Alto, California.* The respiratory depressant effects of morphine and curare and the combination of morphine and curare were evaluated in 8 healthy male subjects. Respiratory depression

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was defined in terms of displacement of the alveolar ventilation- $P_{CO_2}$  response curve (Seed, J. C., and others: *Arch. internat. pharmacodyn.* 116: 293, 1958). *Method:* A modification of the rebreathing method of Eckenhoff, Helrich, and Hege (*Anesthesiology* 17: 66, 1956) was employed, and the alveolar ventilation- $P_{CO_2}$  response curve was obtained automatically with the aid of a new analog computer similar to that previously described (Bellville, J. W., and Seed, J. C.: *Science* 130: 1079, 1959). All the following medications were administered under double-blind conditions: saline, morphine sulfate 5 mg., curare 9 mg., and the combination of morphine sulfate 5 mg. plus curare 9 mg. Plasma curare levels were measured in pilot studies following 6 mg. of curare intramuscularly. A micro spectrophotometric method (Elert, B. T., and Cohen, E. N.: *Amer. J. Med. Tech.*, in press) was used to determine plasma levels, which were in the order of 0.5 gamma at 20 and 40 minutes following the injection of the drug. We estimated that in the dose levels used there would be less than a 10 per cent decrease in vital capacity (Cohen, E. N., and others: *Anesthesiology* 18: 300, 1957). Following two control runs, the drug was injected intramuscularly and determinations of the respiratory response curve were made 20, 40 and 60 minutes following injection. *Results:* The mean displacements of the after-drug curves from the control curves are as follows: saline, 0.48 mm. Hg  $P_{CO_2}$ ; morphine 5 mg., 1.47 mm. Hg  $P_{CO_2}$ ; curare 9 mg., 0.68 mm. Hg  $P_{CO_2}$ ; morphine 5 mg. + curare 9 mg., 3.14 mm. Hg  $P_{CO_2}$ . From this incomplete crossover data, it is impossible to state whether there is an interaction between morphine and curare. This study is being extended to a  $3 \times 3$  factorial design in 6 of these 8 patients. That is, we will study morphine at the 5 and 10 mg. dose level, as well as curare at the 9 and 18 mg. dose level, as well as the combination of these compounds and a placebo.

**Fetal ECG and Obstetrical Blood Loss with Halothane Anesthesia.** PETER BOSOMWORTH, M.D., FRANK SIKORA, M.D., and C. MERLE WELCH, M.D., *Department of Surgery (Anesthesia) Ohio State University, Colum-*

*bus, Ohio.* Previous reports of excessive uterine blood loss during delivery under halothane anesthesia led us to a comparative study of maternal blood loss, fetal ECG, and uterine contractility. *Method:* The obstetrical patients were anesthetized with one of the following agents: halothane-oxygen-nitrous oxide, cyclopropane and oxygen, ether-oxygen-nitrous oxide, or subarachnoid block with tetracaine. One hundred and sixty-nine patients were studied with approximately equal groups for each anesthetic. The lightest plane of anesthesia was used that was compatible with forceps application and episiotomy. Only patients who had no obstetrical complications and were gravida iii or less were included in the study. Collection of blood lost from the uterus was commenced immediately after the flow of amniotic fluid had stopped. An emesis basin was placed against the perineum to collect the uterine blood and exclude the episiotomy blood loss. The blood was suctioned from the basin into a graduated cylinder or into a blood loss monitor (Critical Measurement Division of Industrial Instruments, Inc.). *Results:* The mean blood losses were as follows: halothane (47 patients) 301 cc., S.D.  $\pm 227$ ; cyclopropane (35 patients) 240 cc., S.D.  $\pm 119$ ; ether (37 patients) 337 cc., S.D.  $\pm 247$ ; and saddle block (50 patients) 251 cc., S.D.  $\pm 194$ . Using the Student *t*, a statistical comparison of blood loss during halothane anesthesia with blood loss with cyclopropane, ether, and saddle block showed no significant difference between halothane and any other agent at the 10 per cent level. Nausea and vomiting did not occur in any patient receiving halothane anesthesia, but were present in 16 per cent of the patients given cyclopropane and 12 per cent, given ether during induction of anesthesia after delivery. Awakening time was equivalent with each agent as was the duration of the third stage of labor. Ten per cent of patients given halothane and ether had a sore uterus immediately post partum, while only 4 per cent given cyclopropane lost uterine tone. The means of the infant Apgar scores ranged from 8.0 for halothane to 8.5 for saddle block, with no statistically significant differences in the score for any anesthetic agent. Fetal heart rate was recorded before,