

10.4); ejection-time index (-3.6, -2.5); mean rate left ventricular ejection (-3.1, 4.9); forearm (FA) blood flow (-18.3, -6.0); FA venous pressure (35.3, 14.8); FA venous compliance (-16.1, 0.2); FA resistance (48.0, 42.4). *Summary:* Hexamethonium changed the response to  $N_2O$  from predominantly vascular to predominantly cardiac. These results indicate that: (1) the vasoconstricting action of  $N_2O$  is not a direct effect; (2) the constriction does not arise from norepinephrine released directly by nitrous oxide at the presynaptic granules.

**The Effect of Tetracaine on the Liberation of Potassium Produced by Succinylcholine.** J. E. USUBIAGA, M.D., F. MOYA, M.D., L. E. USUBIAGA, M.D., and B. VESTAL, M.S., *Department of Anesthesiology, University of Miami School of Medicine, Miami, Florida.* Depolarizing muscle relaxants liberate potassium from skeletal muscles, an effect which is antagonized by the prior administration of curare. Since local anesthetics bear a pharmacologic resemblance to curare, the effect of tetracaine on potassium liberation produced by succinylcholine was studied. *Methods:* Ten mongrel dogs were anesthetized with pentobarbital intravenously, intubated and mechanically ventilated to maintain a steady  $Pa_{CO_2}$ . Femoral arteries and veins were cannulated with loops of plastic tubing so that drugs could be injected with blood samples collected with minimal disturbances of limb circulation. Tetracaine (1 mg/kg) was injected into one femoral artery and a control injection of saline was administered in the other. Within a minute, succinylcholine (0.5-1.0 mg/kg) was administered intravenously. Pulse, arterial blood pressure, twitch tension of the anterior tibialis muscles, and plasma potassium levels in blood samples collected from the control and pretreated limbs were compared. *Results:* In six of ten dogs, intra-arterial tetracaine decreased the twitch tension of the tibialis muscle by 10 to 40 per cent. This neuromuscular paralysis was not preceded by stimulation of the muscle, nor followed by an increase in blood potassium levels. Succinylcholine given intravenously produced a complete and prolonged paralysis in both

limbs. In the untreated limb, muscle paralysis was preceded by muscle fasciculations and followed by a significant increase in plasma potassium, mean 0.45 mEq/l, SE 0.14,  $P < 0.001$ . In the extremity pretreated with tetracaine, muscle fasciculations and the increase in plasma potassium were not observed. *Summary:* Tetracaine prevented the muscle fasciculations and the rise in plasma potassium produced by succinylcholine. It also shortened the duration of muscle paralysis. The ability of local anesthetics to prevent muscle fasciculations and potassium liberation probably represents drug effects at two different sites, that is, at the motor nerve terminal and at the postsynaptic membrane. (Supported in part by USPHS General Research Support Grant FR 5363-07/1390397.)

**Depression of Antibody Production by Halothane: A Dose Response.** D. W. WINGARD, M.D., and L. J. HUMPHREY, M.D., *Department of Anesthesiology and Surgery, University of Kentucky Medical Center, Lexington, Ky.* *Methods:* The effect of halothane on antibody-producing splenic lymphocytes was determined in rats preimmunized with an intravenous injection of 0.3 ml of a 10 per cent solution of sheep erythrocytes. Three days following immunization, each test animal was exposed to an inspired concentration of 1 or 1.5 per cent halothane for one to four hours. Control observations were made in similarly treated animals that were not anesthetized. Following exposure to anesthetic or the corresponding control interval, each animal's spleen was removed and the relative number of antibody-producing splenic erythrocytes determined by the Jerne hemolysis in gel technique. *Results:* Compared with controls, the numbers of cells producing antibody were decreased to similar degrees by both halothane concentrations after two, three, and four hours of exposure. *Summary:* Although the precise mechanism responsible for the reduction in antibody-producing splenic lymphocytes by halothane has yet to be elucidated, the data support the conclusion that the duration of anesthesia should be held to a minimum in order to avoid anesthetic-induced changes in immunity.

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