those induced with thiopental. In general, infants delivered following a more prolnoged period of anesthesia and surgery tended to be more acidotic and to score less on the Apgar scale at 1 minute of age. This could be due to decreased maternal perfusion of the intervillous space resulting from compression of the inferior vena cava by the uterus with the mother in the supine position, from intermittent elevation of the central venous pressure during artificial ventilation of the mother, and from surgical manipulation. A direct effect of nitrous oxide upon the fetus cannot be ruled out. Thiopental, as used in the present study, does not appear to contribute to the depression of the newborn.

Effect of Halothane and Halothane Nitrous Oxide on the H-reflex in Man. FELIX G. FREUND, M.D., THOMAS F. HORNBEIN, M.D., WAYNE E. MARTIN, M.D., and PIERRE PARMENTIER, M.D., *University of Washington School of Medicine, Seattle, Wash.* As a clinical measure of anesthetic potency, Saidman and Eger (ANESTHESIOLOGY *25:* 302, 1964) have proposed the use of the minimum alveolar concentration of anesthetic that will prevent movement in response to painful stimulation (MAC). Since anesthetics depress synaptic transmission (Brooks and Eccles: J. Neurophysiol. *10:* 349, 1947; Somjen and Gill: J. Pharmacol. Exp. Therap. *140:* 19, 1963), we sought to correlate the change in halothane MAC resulting from the addition of nitrous oxide with changes in synaptic transmission. For this purpose we studied the H-reflex, a spinal monosynaptic reflex closely related to the Achilles tendon reflex (Magladery *et al.:* Bull. Johns Hopkins Hosp. *86:* 265, 1950; Mayer and Mawdsley: J. Neurosurg. Psychiat. *25:* 201, 1965). Electrical stimulation of the tibial nerve gives rise to two contractions of the calf muscles: the first, the M-response, is due to direct stimulation of the motor nerve fibers; the second, the H-response, is due to discharge of spinal motoneurons through stimulation of the afferent nerve fibers making synaptic connection with them. *Method:* The tibial nerve was stimulated by single shocks through needle electrodes. The magnitude of the muscle contractions was measured by the amplitude of the corresponding action potentials which were recorded by means of surface electrodes and photographed from an oscilloscope. The amplitude of the H-reflex was determined before anesthesia and at end-tidal concentrations of 1.5, 0.8 and 0.3 per cent halothane with 70 per cent N_2O-O_2, and at 1.5 and 0.8 per cent halothane-O_2. In addition, at each anesthetic level the effect on the H-reflex of increasing arterial P_{CO_2} was observed. *Results:* Six volunteers have been studied. Mean amplitude of the H-reflex was 28.6 per cent of the awake response at 0.3 per cent halothane-N_2O (MAC = 1); 27 per cent at 0.8 per cent halothane-O_2 (MAC = 1); 11.3 per cent at 1.5 per cent halothane-O_2; 8.6 per cent at 0.8 per cent halothane-N_2O; and 3.8 per cent at 1.5 per cent halothane N_2O. At each anesthetic level, increasing arterial P_{CO_2} caused an additional depression of the H-reflex. Both 0.3 per cent halothane-70 per cent N_2O-O_2 and 0.8 per cent halothane-O_2 appear to be equatable in their effect on the responsiveness to noxious stimulation (as measured by MAC) and on the amplitude of the H-reflex. Assuming that the effect of nitrous oxide is simply additive to that of halothane, the results obtained at MAC = 1 mean that 70 per cent nitrous oxide is equivalent to the addition of 0.5 per cent halothane. Thus, adding 70 per cent nitrous oxide to 0.8 per cent halothane should produce the same depression of the H-reflex as 1.3 per cent halothane, but in fact that observed depression (8.6 per cent of control) was even greater than that observed in response to 1.5 per cent halothane (11.3 per cent of control). *Conclusions:* The effect of the H-reflex of adding nitrous oxide to halothane becomes more than additive as halothane concentration increases. (Supported by UW Graduate School Initiative 171 Grant, NIH Research Career Grant 5K3-HE-9617-02, USPHS Grant HE-08866-03, and a grant from the Ayerst Company.)

The Mechanism of Action of Decamethonium. A. J. GISSEN, M.D., and WILLIAM L. NASTUK, M.D., *Presbyterian Hospital and Columbia University College of Physicians and Surgeons, New York, N. Y.* Decamethanium

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