dilution), and right auricular pressure. Arterial blood samples were analyzed for *p*H, P_{CO_2}, O_2 content, hemoglobin, hematocrit and lactic acid. After 45 minutes of anesthesia, control measurements were made, and graded arterial hemorrhage with immediate intravenous dextran replacement was carried out as follows: Initial hemorrhage of 20 per cent of estimated blood volume (10 per cent of body weight); then successive hemorrhages of 10 per cent of blood volume each, every five minutes, until hemoglobin 3 g. 100 ml. STUDY 1. *Spontaneous Respirations Versus Intermittent Positive Pressure Ventilation (IPPV) and Oxygen Versus Air.* The circulatory and metabolic effects during four types of respiration were compared: Group 1, spontaneous respiration with air; group 2, spontaneous respiration with 100 per cent O_2; group 3, IPPV with air; and group 4, IPPV with 100 per cent O_2; 7 dogs in each group. After a hemoglobin value of 3 g./100 ml. was reached, the animals were observed for 1 hour; subsequently, hemorrhage with replacement was continued until cessation of circulation. *Results:* (1) Progressive fall in arterial *p*H during spontaneous respiration with air and during spontaneous respiration with O_2 (7.37 ± 0.05 to 7.06 ± 0.10); less decrease of *p*H during IPPV with air (7.42 ± 0.07 to 7.24 ± 0.10) and no change in *p*H during IPPV with O_2 (7.51 ± 0.10 to 7.49 ± 0.10). (2) Circulatory deterioration began when arterial O_2 content had fallen below 4 volumes per cent (*i.e.*, average hemoglobin 3.5 g./100 ml. with air and 1.5 g./100 ml. with O_2); asystole occurred suddenly, approximately at 3 volumes per cent O_2, except in the IPPV with O_2 group where circulatory failure was more gradual. STUDY 2. *Adaptation to Hemodilution to 3 g./100 ml. Hemoglobin.* Ten dogs were hemodiluted as in study 1, but only to 3 g./100 ml. hemoglobin. Respiration: IPPV with 30 per cent O_2–70 per cent N_2; rate 20/minute; tidal volume adjusted to maintain end-expiratory P_{CO_2} near 40 mm. of mercury. At end of hemodilution, spontaneous respiration with air and observation for 24 hours. *Results:* (1) 7 of the 10 animals survived 24 hours. (2) *p*H fell from control 7.40 (± 0.04) to 7.30 (± 0.04) at 3 g./100 ml. hemoglobin; two hours later 7.30 (± 0.07); and 24 hours later 7.42 (± 0.07). (3) Cardiac index showed a consistant increase from control 4.34 (± 0.6) to 7.54 (± 0.7) at 3 g. 100 ml. hemoglobin; and two hours later 5.90 (± 1.30). The 3 dogs which died were those unable to maintain elevated cardiac index. (4) Lactic acid showed no consistant change and did not correlate with ability to survive for 24 hours. (5) After hemodilution to 3 g./100 ml., hemoglobin increased to 4.0 g./100 ml. at two hours and 5.5 g./100 ml. at 24 hours (average). STUDY 3. *Long-term Survival after Hemodilution to 3 g./100 ml. Hemoglobin.* Five dogs of study 2 were observed for one week; all survived and appeared normal at the end of this period. They maintained normal *p*H (7.44 ± 0.03) and blood glucose; plasma proteins were low; plasma Na and K only slightly reduced. At one week the hemoglobin had returned to approximately 8 g. 100 ml. *Conclusions:* Although these results may not apply to man, particularly those with disease, they suggest that one can be liberal with the use of dextran in treating exsanguinating hemorrhage. Hemodilution below 3 g./100 ml. hemoglobin should not be exceeded. Oxygen and controlled hyperventilation prevented acidosis and delayed onset of asystole. (Supported by U. S. Army Contract No. DA-49-193-MD-2160).

Respiratory Effects of Trichlorethylene in Man. LT. D. A. TALCOTT, MC USN, LCDR. C. P. LARSON, JR., MC USNR, and CDR. D. R. BUECHEL, MC USN, *Department of Anesthesiology, U. S. Naval Hospital, Oakland, California.* In midcollicular decerebrate cats, trichlorethylene (TCE) in 1 and 2 per cent concentration depresses respiratory responses to inhaled CO_2 and elevates the electrical stimulus threshold of the medullary respiratory center (Ngai, Farhie and Brody, Fed. Proc. 22: 186, 1963). Aside from progressive reduction in tidal volume and elevation in respiratory rate with increasing depth (Dundee and Dripps, ANESTHESIOLOGY *18:* 282, 1957), little information is available regarding the respiratory effects of TCE in man. *Methods:* To obtain additional data in man, we measured ventilation and ventilatory responses to 2 and 4 per cent inhaled CO_2 awake and at two levels of TCE anesthesia in ten healthy patients. Six patients were unpremedicated and

four patients were medicated with pentobarbital 100 mg., morphine 10 mg., and atropine 0.7 mg. per 70 kg. of body weight, given one hour prior to study. Tidal volume and respiratory rate were measured with subjects breathing from a calibrated ventimeter in a circle system and recorded with a Grass direct writing recorder. Simultaneous brachial arterial blood samples were drawn for *p*H and P_{CO_2}, using the Astrup technique. TCE was vaporized in two Vernitrol vaporizers, the approximate inspired concentration being calculated from vaporizer and diluent oxygen flows, knowing TCE vapor pressure at the existing temperature. Following control measurements, anesthesia was induced with thiopental (100 to 350 mg. intravenously) and maintained at 2 per cent TCE in O_2 via an endotracheal tube and nonrebreathing system (Stephen-Slater valve) for approximately one hour. Subjects were then temporarily returned to the circle system and ventilatory and blood-gas data obtained at this concentration. This procedure was repeated after approximately 30 minutes at 1 per cent TCE. No surgical stimulation was present during this study. *Results:* Under TCE anesthesia, tidal volume of nonpremedicated patients fell markedly (mean control—453 ml.; 1 per cent TCE—177 ml.; 2 per cent TCE—176 ml.) but the profound tachypnea which ensued (control—15/minute; 1 per cent TCE—48/minute; 2 per cent TCE—55/minute) resulted in a moderate increase in minute ventilation (control—6.7 liters/minute; 1 per cent TCE—8.3 liters/minute; 2 per cent TCE—9.3 liters/minute). However, most of this additional volume was expended in ventilating airway dead space, since no appreciable change occurred in arterial P_{CO_2} (control—35.9 mm. of mercury; 1 per cent TCE—39.2 mm. of mercury; 2 per cent TCE—37.8 mm. of mercury). In premedicated patients, tidal volume also fell (mean control—537 ml.; 1 per cent TCE—216 ml.; 2 per cent TCE—190 ml.), but the tachypnea which ensued (control—11.5/minute; 1 per cent TCE—51/minute; 2 per cent TCE—50/minute) resulted in an increased minute ventilation (control—6.1 liters/minute; 1 per cent TCE—10.8 liters/minute; 2 per cent TCE 9.7 liters/minute). Some change occurred in arterial P_{CO_2} (control—35.9 mm. of mercury; 1 per cent TCE—37.6 mm. of mercury; 2 per cent TCE—43.5 mm. of mercury), although no patient exhibited severe respiratory acidosis. TCE did not appear to alter ventilatory responses to CO_2 challenges in premedicated or unpremedicated man. *Conclusions:* These preliminary investigations suggest that under the circumstances of this study, TCE is not a respiratory depressant in man. Alveolar ventilation was adequately maintained at surgical concentrations of TCE and in contrast to data in cats, there did not appear to be any elevation in CO_2 threshold for ventilation or depression in CO_2 responsiveness.

Neuromuscular Effects of Cyclopropane. ALAN VAN POZNAK, M.D., *Associate Attending Anesthesiologist, New York Hospital and Clinical Assistant Professor of Anesthesiology in Surgery, Cornell University Medical College, New York, New York; and* JOSEPH F. ARTUSIO, JR., M.D., *Anesthesiologist-in-Chief, New York Hospital and Professor of Anesthesiology in Surgery, Cornell University Medical College, New York, New York.* Cyclopropane produces two opposing effects on mammalian nerve-muscle preparations. There is potentiation of the direct twitch which is not abolished by curarization. Despite this potentiation of direct twitch, a second and separate effect involving neuromuscular block is evident, because the indirect twitch is reduced more by the combination of cyclopropane plus *d*-tubocurarine than by *d*-tubocurarine alone. This study is concerned with the unusual picture of increasing twitch tension in the presence of increasing neuromuscular block. *Method:* Cats either anesthetized with chloralose or decerebrated were prepared for recording of twitch tension from soleus or gastrocnemius. Curarized and noncurarized muscles were examined. Electomyograms were observed for both muscles. Motor nerve terminal activity in single axon preparations was measured as the time during which repetitive discharge could be elicited by single shocks following a 10-second tetanus; the per cent depression of duration of nerve repetition induced by cyclopropane was determined. Reserpinized or chronically denervated cat preparations were also examined, as well as *in vitro* frog sartorius preparations. Inhaled cyclopropane concen-