ministered, nor were any intravenous fluids administered. All blood samples obtained were arterial, one being taken just prior to induction, another exactly 30 minutes following induction. Surgery had started but was still superficial in approximately half of the patients at the time of the second sample. Each blood sample, immediately after being withdrawn, was added to chilled 10 per cent trichloroacetic acid and analyzed for lactate (by the method of Barker), for pyruvate (by a modification of the method of Friedeman), and for citrate (by the method of Stern) (*Colowick and Kaplan: Methods in Enzymology, vol. 3. 1957*). The mean (± standard error of the mean) blood levels of these metabolites in milligrams per cent were as follows (control levels being given first in each instance, with levels after anesthesia being given second): *Cyclopropane:* lactate 8.47 ± 1.57 to 14.71 ± 1.32; pyruvate 1.17 ± 0.10 to 1.43 ± 0.10; citrate 1.92 ± 0.14 to 2.28 ± 0.14. *Thiopental-nitrous oxide:* lactate 11.83 ± 1.40 to 7.72 ± 1.24; pyruvate 1.23 ± 0.10 to 1.12 ± 0.10; citrate 1.86 ± 0.24 to 2.00 ± 0.40. *Ether:* lactate 8.04 ± 1.48 to 18.43 ± 2.17; pyruvate 1.26 ± 0.00 to 1.65 ± 0.10; citrate 1.85 ± 0.23 to 2.00 ± 0.27. Statistically the changes in lactate during both cyclopropane as well as during ether anesthesia were significant. The changes in pyruvate associated with ether anesthesia were also significant. Changes in lactate during thiopental anesthesia were only of borderline significance. The results suggest a partial block of oxidative metabolism during ether at the pyruvate to acetyl CoA level. They also suggest decreased glycolysis during thiopental anesthesia. The rise in lactate during cyclopropane, unassociated with significant changes in pyruvate or citrate, remains unexplained. [*Supported by a Research Grant (H-3359) from the National Heart Institute of the National Institutes of Health, U. S. Public Health Service.*]

The Relationship of Respiration to Directly Measured Brain CO_2 Tension. E. P. Guy, M.D., T. N. Finley, M.D., and J. W. Severinghaus, M.D. *Department of Anesthesia, University of California Medical Center, San Francisco, California.* A P_{CO_2} electrode was modified to permit direct continuous recording of tissue P_{CO_2} *in vivo*. The electrode measures *p*H in a thin film of water separated from tissue by a membrane (Teflon) permeable to CO_2 gas but not hydrogen ions. CO_2 diffuses through this membrane, controlling the *p*H of the water film. The measured P_{CO_2} is not affected by the *p*H, pressure or flow of the sample. Response time is 1–2 minutes. The response is linear on semilog paper from 1.5 to 100 per cent CO_2. The entire tip of the electrode is about 10 mm. in diameter, the center 5 mm. of which are sensitive to P_{CO_2}. The electrode was applied to exposed cerebral cortex surface. The effect of Diamox on brain tissue P_{CO_2} and on pulmonary ventilation was studied in 7 dogs breathing oxygen spontaneously under Chloralose anesthesia. The purpose was to learn whether the respiratory center responds to changes in arterial or tissue P_{CO_2}. During the first two hours after Diamox (40 mg./kg. intravenously) we found a rise in cerebral cortex P_{CO_2} from 53 to 83 mm. Hg; a fall in alveolar P_{CO_2} from 34 to 14; a rise in arterial P_{CO_2} from 37 to 44; longitudinal sinus P_{CO_2} unchanged–54 mm. Hg. Also, for the first 10 minutes after Diamox the ventilation-tissue P_{CO_2} response curve paralleled that obtained by CO_2 breathing. The subsequent rise in cortex P_{CO_2} failed to produce further increase in ventilation. The ventilatory response to CO_2 breathing was unaltered after Diamox. Ventilation increased 2.3 times the control. The CO_2 response curve established in the control period suggests that this ventilation would result from a respiratory center P_{CO_2} increase of 12 mm. If the respiratory center were monitoring arterial P_{CO_2} directly, ventilation would have been stimulated only 1.7 times by Diamox. On the other hand, if respiratory center P_{CO_2} followed cortical P_{CO_2} respiration should have been stimulated 3.5 times. This failure of Diamox to vigorously stimulate respiration can be best explained by assuming that the respiratory center CO_2 chemoreceptor is located in tissue with a higher blood flow than cerebral cortex.

A Method of Clinically Assaying Muscle Relaxants. W. Hamelberg, M.D., J. H. Sprouse, Jr., M.D., and J. E. Mahaffey, M.D. *Department of Anesthesiology, Medical College of South Carolina, Medical College*

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Hospital, Charleston, South Carolina. All muscle relaxants are known to produce a decrease in respiratory exchange. However, the degree of respiratory depression obtained for a standard amount of muscle relaxation is believed to vary with each muscle relaxant. This phenomenon has been defined as the "respiratory-sparing" effect of muscle relaxants. In one investigation, an evaluation of the "respiratory-sparing" effect of the various muscle relaxants demonstrated that respiratory depression was more pronounced after single doses of nondepolarizing relaxants than after depolarizing relaxants (*Poulsen, H., and Hougs, W.: Acta anaesthesiol. scandinav. 1: 15, 1957*). This, however, was not true with rapidly administered single doses of succinylcholine iodide or its derivatives. These conclusions were drawn from experiments in which muscle relaxation in the extremities was compared to the observed decrease in tidal volume. A more pertinent comparison would be the decrease in tidal volume associated with a standard amount of abdominal relaxation. A method of obtaining this comparison is the basis of this report. A resistance strain gauge arch designed to measure heart contractile force was modified in such a way as to become an integral part of an abdominal retractor (*Boniface, K. J., Brodie, O. J., and Walton, R. P.: Proc. Soc. for Exp. Biol. & Med. 84: 263, 1953*). This modification made it possible to measure in grams the force exerted by the abdominal muscles of the dog before and after muscle relaxants. The change in electrical output of the strain gauge arch was amplified by a Brush electronics strain gauge analyzer with a direct inking oscillograph for recording. In pilot experiments, medium-sized mongrel dogs were lightly anesthetized with intravenous pentothal sodium, and the abdomen was opened through a midline incision. The retractor was inserted and the incision separated. A record of the force applied against the retractor was obtained. After a suitable tracing was obtained, an apneic dose of intravenous succinylcholine chloride was administered, following which a continuous record of the change in force was recorded. In 15 experiments, the force exerted on the retractor was reduced on the average from 200 Gm. to 50 Gm. In these experiments, however, no effort was made to measure the "respiratory-sparing" effect of succinylcholine chloride. The same experiment was conducted in 7 patients. The results in these patients were much the same as obtained in the animal experiments. Again, no attempt was made to establish the "respiratory-sparing" effect of succinylcholine chloride. In one patient, the apneic dose of 20 mg. of succinylcholine chloride produced a 50 per cent reduction in the force applied against the retractor. After the patient had recovered from the effects of the succinylcholine chloride and the force against the retractor returned to its previous level, the patient received 9 mg. of *d*-tubocurarine. This was followed by a 50 per cent reduction in the force applied against the retractor with only a 50 per cent decrease in the patient's tidal volume. This would tend to indicate that *d*-tubocurarine has a greater "respiratory-sparing" effect than does succinylcholine chloride in apneic doses.

Our experiments thus far with this method of measuring muscle relaxation has been satisfactory. Future work will be done to establish a "respiratory-sparing" ratio for each muscle relaxant.

Experience With Gas (Absolute) Sterilized Endotracheal Tubes. ROBERT C. HARVEY, M.D., RICHARD N. TERRY, M.D., AND RICHARD AMENT, M.D. *Department of Anesthesiology, Buffalo General Hospital, University of Buffalo School of Medicine, Buffalo, New York.* Recent outbreaks of hospital acquired staphylococcal infection has led to a review of possible sources of cross infection in hospitals. At The Buffalo General Hospital the Infection Committee questioned the reliability of existing methods of sterilization of endotracheal tubes, *i.e.*, a one-minute scrub employing 3 per cent hexachloraphine soap. It was pointed out that areas skipped in scrubbing could constitute a nosocomial infection hazard. Ethylene oxide was selected as offering maximum security for materials of low thermostability. Ethylene oxide is totally effective against all vegetating bacteria and fungi, has exceptional sporicidal activity, and is viricidal for at least the larger forms. A

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