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 Each blood sample, immediately after being withdrawn, was added to chilled 10 per cent trichloroacetic acid and analyzed for lactate (by the method of Barker), for pyruvate (by a modification of the method of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*