

propane. The others received only cyclopropane. Forty milligrams of succinylcholine were given to some patients just prior to intubation. Endobronchial intubation was carried out in 12 patients using a modified Magill catheter with a long lipped bevel as described by Bonica and Hall. The proper location of the tube was confirmed after the patient was in position for surgery, by auscultation. Thirteen patients were anesthetized, and a no. 39 double lumen Carlens catheter was put in place. All patients were in the lateral position for surgery. Measurements were made during endobronchial anesthesia under the following conditions when the Magill catheter was used: (1) before the chest incision; (2) with the chest open and the surgeon at work on the lung; (3) with the chest still open and respiration controlled after the chest procedure, and (4) after the chest was closed with the patient breathing spontaneously. The cuff was deflated, the endobronchial catheter slightly withdrawn and the cuff again inflated during procedures 3 and 4. The subjects whose trachea was intubated with the Carlens tube were studied under the following conditions during anesthesia: (1) essentially endotracheal, in that both lumina of the tube were open, before surgery began; (2) endobronchial, one lumen clamped, during surgery; (3) both lumina again open, essentially endotracheal with the chest still open and respiration controlled after the lung procedure; and (4) endotracheal after the chest was closed with the patient breathing spontaneously.

End-Expiratory Carbon Dioxide. Using the Magill tube, end-expiratory carbon dioxide was lower when endobronchial anesthesia had been discontinued (3.73 per cent) than when the patient's lungs were being ventilated with one lung (4.23 per cent). A more definite change was seen when the patient's respiration was no longer assisted (5.36 per cent) and he was breathing spontaneously with the chest closed. With the Carlens tube the results showed the same general changes. That is, end-expiratory carbon dioxide values were higher with endobronchial assisted respiration (6.15 per cent) than with endotracheal assisted respirations (4.58 per cent). Again, spontaneous respiration resulted in an increase of carbon dioxide concentration to 6.38 per cent. All values

with the Carlens tube were greater than those with the Magill tube. *Arterial Carbon Dioxide.* Arterial carbon dioxide values were close to normal throughout, using either type of catheter. No significant changes occurred between endobronchial and endotracheal respirations. *Arterial Oxygen Saturation.* With the Magill catheter, the oxygen saturations were lower with respiration through only one lung than when both were used. Endobronchial values were 91 and 92 per cent, while the values with the endotracheal tube were 98 and 99 per cent. Using the Carlens catheter endobronchial respirations showed an average oxygen saturation of 90.3 per cent, while values using both lungs were 97.7, 97.4 and 94 per cent. Pre-incision values showed some diminution of oxygen saturation with endobronchial (Magill) catheter as compared to endotracheal (Carlens) respiration. Other comparisons of results with the Carlens and Magill tubes show a lower, but not statistically significant, average oxygen saturation with the Carlens tube. These latter comparisons were under similar conditions; *i.e.*, endobronchial versus endobronchial and endotracheal versus endotracheal. Although oxygen saturation were slightly below normal during endobronchial intubation and ventilation, none were diminished sufficiently to endanger the patient. [Aided by USPHS Grant H-4308.]

Studies on Carbohydrate Metabolism During Anesthesia. NICHOLAS M. GREENE, M.D., FRANCES J. MACKAY, M.D., AND J. K. S. BELL, B.A. *Section of Anesthesiology, Yale University School of Medicine, and Department of Anesthesia, Grace-New Haven Community Hospital.* Oxidative carbohydrate metabolism was studied in 19 adult patients. None of the patients had pre-existing hepatic, endocrine, or metabolic diseases, but were unselected as to age or type of operation. Premedication consisted in 18 patients of a barbiturate (100 mg./70 kg.) and atropine or scopolamine (0.6 mg./70 kg.) 45-60 minutes before induction of anesthesia. One patient received meperidine premedication, with no apparent effect on the results. In 5 patients cyclopropane was the anesthetic, in 6 thiopental-nitrous oxide, and in 8 ether preceded by induction with nitrous oxide. No other drugs were ad-

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 (\pm 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 CO₂ 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_{CO₂} electrode was modified to permit direct continuous

recording of tissue P_{CO₂} *in vivo*. The electrode measures pH in a thin film of water separated from tissue by a membrane (Teflon) permeable to CO₂ gas but not hydrogen ions. CO₂ diffuses through this membrane, controlling the pH of the water film. The measured P_{CO₂} is not affected by the pH, 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 CO₂. The entire tip of the electrode is about 10 mm. in diameter, the center 5 mm. of which are sensitive to P_{CO₂}. The electrode was applied to exposed cerebral cortex surface. The effect of Diamox on brain tissue P_{CO₂} 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_{CO₂}. During the first two hours after Diamox (40 mg./kg. intravenously) we found a rise in cerebral cortex P_{CO₂} from 53 to 83 mm. Hg; a fall in alveolar P_{CO₂} from 34 to 14; a rise in arterial P_{CO₂} from 37 to 44; longitudinal sinus P_{CO₂} unchanged—54 mm. Hg. Also, for the first 10 minutes after Diamox the ventilation-tissue P_{CO₂} response curve paralleled that obtained by CO₂ breathing. The subsequent rise in cortex P_{CO₂} failed to produce further increase in ventilation. The ventilatory response to CO₂ breathing was unaltered after Diamox. Ventilation increased 2.3 times the control. The CO₂ response curve established in the control period suggests that this ventilation would result from a respiratory center P_{CO₂} increase of 12 mm. If the respiratory center were monitoring arterial P_{CO₂} directly, ventilation would have been stimulated only 1.7 times by Diamox. On the other hand, if respiratory center P_{CO₂} followed cortical P_{CO₂} 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 CO₂ 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*