ifornia Medical Center at Los Angeles. Potassium superoxide is employed as a chemical source of oxygen in self-contained rescue apparatuses. It has a theoretical yield of 236 ml. O_2/g. dry weight ($4\ KO_2 + H_2O + CO_2 \rightarrow 3_{O_2} + K_2CO_3 + 2\ KOH$). The purpose of this investigation was to devise means of adapting potassium superoxide as an oxygen source for resuscitative purposes. *Method:* Initial studies indicated that the simplest design would be an adaptation of the potassium superoxide canister to mouth-to-mouth resuscitation. A 350-ml. Foregger to-and-fro canister was packed with 400 g. of KO_2 granules. The breathe-through cells of two Liston-Becker 16 CO_2 analyzers were attached to each end of the canister. The open end of each cell was fitted with an endotracheal tube adapter supplied with a side port to which a deflated clamped balloon was attached. A BOC ventimeter was inserted in the line. Two dogs were anesthetized with pentobarbital and their tracheas intubated with cuffed tubes. Each tube was fitted to the open end of a CO_2 analyzer. The continuity of the system was as follows: Dog *A* OT tube → side arm O_2 sample balloon *A* → CO_2 analyzer *A* → BOC ventimeter → KO_2 canister → CO_2 analyzer *B* → side arm O_2 sample balloon *B* → dog *B* OT tube. Thus, each dog was attached to a closed to-and-fro system employing the other dog's lungs as a rebreathing bag. CO_2 concentration delivered to each animal as well as alveolar CO_2 was recorded. Samples for oxygen analysis were obtained by clamping one endotracheal tube and releasing the sampling balloon clamp as gas was delivered from the other dog's lungs through the canister. The samples were analyzed with a Beckman D_2 analyzer. A femoral artery of each dog was cannulated. Blood samples were analyzed with a Waters Connel Oximeter. An initial sample was taken with the dogs breathing air. The thorax of dog *A* was manually compressed at a rate of 20/minute. Shortly thereafter, both dogs became apneic and inflation of dog *B*'s lungs occurred with each compression of dog *A*'s chest. Artificial respiration was continued for 40 minutes. A tidal volume of 150–350 ml. was delivered with each thoracic compression. Mechanical dead space for each animal was 100 ml. *Results:* The inspired O_2 concentration of dog *A* rose to 28 per cent within 10 minutes and 31 per cent in 40 minutes. Inspired O_2 concentrations of dog *B* were 24 per cent in 10 minutes and 29 per cent in 40 minutes. Inspired CO_2 concentration was 1.9 per cent throughout most of the experiment for dog *A* and alveolar CO_2 was 8.3 per cent. Inspired CO_2 concentration of dog *B* varied betwen 0.8 and 1.8 per cent. Alveolar CO_2 concentration varied from 3.2 to 5.8 per cent. Control arterial oxygen saturations were 81 volumes per cent (V/V) for dog *A* and 84 volumes per cent for dog *B*. After 40 minutes resuscitation through the KO_2 canister, saturation of dog *A* was 92 volumes per cent (11 volumes per cent higher than control). *Conclusion:* The data indicate that a relatively high concentration of oxygen may be delivered from a KO_2 canister during conditions of the experiment. We believe the results justify further investigation with more refined techniques.

Ventilation of the Emphysematous Patient During Anesthesia. ELWYN S. BROWN, M.D., and JAMES O. ELAM, M.D., *Department of Anesthesiology, Roswell Park Memorial Institute, Buffalo, New York.* *Method:* Nitrogen washout of the lungs was performed just prior to operation in anesthetized emphysematous patients, and in a control group of similar age with normal lungs. Following intravenous meperidine and scopolamine and topical anesthesia, the trachea was intubated. Respiratory flow rate, CO_2 and N_2 concentrations and airway pressure were recorded. Blood samples were taken from the femoral artery late in each washout-period, and CO_2 tension was measured. The first nitrogen washout was recorded while the patient breathed spontaneously. After thiopental was given and a succinylcholine drip started, tidal volume and respiratory rate were preset on the ventilator. Tidal volumes up to two liters at frequencies of 10–15 breaths per minute were employed in a series of N_2 washouts. A long expiratory period was employed to permit more complete exhalation by the emphysematous patients. *Results:* Functional residual volumes ranged from 1000 to 2000 ml. in normal men and 2500 to 5000 ml. in emphysematous men. In both the normal and the emphysematous men, 20–30 per cent of the alveolar ventilation

ventilated the lung compartment with the slowest turnover rate. Increasing the tidal volume decreased this proportion slightly. No difficulty was encountered in the emphysematous patients in reducing the alveolar and arterial P_{CO_2} to low values. No increase in the A–a gradient for CO_2 was observed. The CO_2 output increased to more than 250 per cent of that produced. The excess CO_2 output was directly proportional to the increase in the alveolar ventilation. Hypotension was observed in some patients but not in others from the increased airway pressures at large tidal volumes. *Conclusion:* This study shows that increasing tidal volume increases the rate of turnover in lung gas in emphysematous patients as effectively as it does in the lungs of the normal patient. The rate of turnover is directly proportional to the alveolar ventilation and inversely proportional to the functional residual capacity of the lung.

Interrelation of Cough Suppression and Respiratory Depression. J. R. CALVERT, M.D., J. E. STEINHAUS, M.D., PH.D., G. A. MARTIN, B.S., and J. C. MCFARLAND, B.S., *Emory University School of Medicine, Atlanta, Georgia.* Recent studies with intravenous lidocaine have suggested that the cough reflex can be depressed without marked respiratory depression in contrast to the traditional depressants. This investigation studied a series of compounds to determine if the depression of the cough reflex paralleled the depression of respiration. *Method and Results:* The first part of the study was carried out in rabbits intubated blindly after the induction of light thiopental anesthesia. The test drug was administered intravenously in doses of 2.5 mg./kg. at intervals of two minutes until spontaneous cough disappeared. At this time the minute volume was measured by a spirometer. The endotracheal tube was moved a distance of 1 cm. three times to elicit cough. If cough occurred, the administration of the test drug was continued in the same dose at two minute intervals until cough could no longer be elicited. The minute volume was again obtained. Drugs tested included morphine and meperidine from the narcotic group; thiopental, chlorpromazine, and promethazine from the phenothiazine group; tripelennamine, methapyrilene, diphenhydramine, and antazoline from the antihistamine group, and lidocaine, a local anesthetic. In addition to the minute volume, observations were made to determine the general level of central nervous system depression at the point of cough suppression. As in previous studies, thiopental and the opiates produced severe respiratory depression without satisfactory suppression of cough. The phenothiazines depressed cough with a moderate increase in minute volume, but the animals appeared quite depressed at the end of the procedure. The antihistamines depressed the cough, with an associated increase in minute volume. It was noted that many of these animals could stand upright with the endotracheal tube in place at the end of the test. Minute volume following lidocaine was not significantly changed from the normal, and central nervous system depression was intermediate between that seen with the antihistamines and the phenothiazines. The second part of the study compared the effect of morphine and lidocaine on the cough reflex and respiration of healthy patients scheduled for gynecological surgery. Induction was accomplished with thiopental (6.6 mg./kg.), and intubation, with the aid of succinylcholine (40–60 mg.). Nitrous oxide and oxygen were administered by a nonrebreathing system. Lidocaine 75 to 100 mg. or morphine 5 mg. was given intravenously every minute for four doses, then every two minutes until either the cessation of breathing or the cessation of cough and its related movements. EEG, ECG, blood pressure, minute volume and intraesophageal pressure were monitored. In six patients receiving morphine the end point was reached with doses ranging from 20 to 60 mg., and in all cases they became apneic before cough or its related movements were suppressed. At the end point of the lidocaine series all seven patients had complete suppression of cough, and their minute volumes ranged from 2.25 to 7.0 liters/minute with lidocaine dosages of 300–800 mg.

***d*-Tubocurarine and the Blood-Cerebrospinal Fluid Barrier.** ELLIS N. COHEN, M.D., *Department of Anesthesia, Stanford University Medical School, Palo Alto, California.* The existence of a blood-brain barrier to *d*-tubo-