than procaine. The segments of spread for chloroprocaine and lidocaine was approximately the same, but lidocaine produced a significantly greater evenness of spread. This suggests that lidocaine is more penetrant than chloroprocaine. The duration of anesthesia was significantly greater with lidocaine than with both chloroprocaine and procaine. No serious toxic reactions occurred in the second phase of study. Procaine and chloroprocaine produced a slightly greater incidence of minimal toxic symptoms, such as tinnitus and slight nausea, than lidocaine. Further studies are planned to enlarge the samples and to include other agents.

An Assessment of Respiratory Efficiency in the Postoperative Patient. JOSEPH J. BUCKLEY, M.D., AND FREDERICK H. VAN BERGEN, M.D. *Department of Anesthesiology University of Minnesota Medical School, Minneapolis, Minnesota.* Hamilton and Devine (*Surg. Gynec. & Obst. 105: 229, 1957*) measured the end-expiratory carbon dioxide level of 100 routine recovery room patients and found that 25 per cent were ventilating inadequately. Hood *et al.* (*J. Thoracic Surg. 36: 729, 1958*), using respiratory volume and blood gas measurements, demonstrated that about 50 per cent of patients undergoing thoracic surgery had a significant depression of ventilation immediately after surgery. These findings caused us to re-evaluate the condition of recovery room patients in our institution; if respiratory depression was as prevalent as the previous studies suggested, it seemed likely that the somewhat empiric use of oxygen could accentuate the hypoventilation. Arterial blood *p*H, carbon dioxide tension and hemoglobin oxygen saturation were measured immediately after anesthesia and surgery in 23 patients breathing room air. No respiratory acidosis was found (mean *p*H 7.43, mean $P_{A_{CO_2}}$ 35.9). However, the arterial hemoglobin oxygen saturation was significantly depressed (93.8 per cent as compared to 97.1 per cent in a control group of 20 preoperative conscious patients).

In an attempt to identify the cause of this hypoxemia, 7 patients underwent "shunt studies" in which the amount of venous admixture was quantitated from blood oxygen data obtained during 100 per cent oxygen breathing (to eliminate uneven ventilation effect). The preoperative "shunt" value was found to be 3.6 per cent; the postoperative "shunt" amounted to 10.8 per cent. In addition, the administration of nasal oxygen to 16 patients at a flow rate of 5 l./minute failed to increase the hemoglobin oxygen saturation significantly (mean saturation breathing room air, 92.7 per cent; mean saturation breathing nasal oxygen, 94.5 per cent). It seems likely that the shunt (not actually measured) in most of these patients must have exceeded 15 per cent, since this is the maximum degree of venous admixture which can be offset by 50 per cent oxygen (nasal oxygen). Uneven ventilation seems an unlikely explanation in view of the data of Brattstrom (*Acta chir. scandinav. (supp. 195) 1954*). These data suggest that subtle venous admixture may follow anesthesia and surgery and may represent a postoperative complication which is difficult to recognize by ordinary clinical methods. [*Supported by research grant (H-1983C4), United States Public Health Service.*]

Respiratory Obstruction in Normothermia and Hypothermia. HAROLD F. CHASE, M.D., DAVID J. LAFIA, M.D., AND MEARL A. KILMORE, B.S. *Departments of Anesthesiology and Neurosurgery, Jefferson Medical College, Philadelphia, Pennsylvania.* This report compares physiologic responses to respiratory obstruction during normothermia and hypothermia. Dogs were anesthetized with thiopental, their tracheas intubated, and then allowed to breath a mixture of nitrous oxide and oxygen. Arterial, inferior vena caval, external jugular, endotracheal and cisternal pressures, and the electrocardiogram were recorded on a Grass polygraph. Respiration was obstructed for ten minutes by forcing the animals to breath through a 15 gauge needle. Hypothermia of 30 C. was then produced by packing the dog in ice, and the respiratory obstruction repeated. Rectal temperature was recorded by a mercury thermometer. During normothermic obstruction there was an increase in all pressures, but the elevation in endotracheal pressure was proportionately larger. The electrocardiogram showed a sinus rhythm. There was a wide fluctuation of arterial, inferior vena

caval, external jugular, and cisternal pressures, which coincided with the forceful respiratory efforts. The obstruction produced arterial hypoxia and hypercapnia. During hypothermic obstruction, pressure increases were significantly less and respiratory fluctuations dampened. The increase in endotracheal pressure and minute volume of ventilation were approximately one-half those observed during obstruction in normothermia. Nevertheless, there was no statistical difference in arterial hypoxia nor in the increase in carbon dioxide content produced by respiratory obstruction in hypothermia as compared with normothermia.

Effect of Methylphenidate Ritalin on Thiopental Ventilatory Depression. J. Gerard Converse, M.D., and Sanford Cobb, M.D. *Department of Anesthesiology, University of Miami School of Medicine at Jackson Memorial Hospital, Miami, Florida.* The effect of methylphenidate hydrochloride (Ritalin) on the ventilatory depression produced by thiopental was studied in man by CO_2 stimulus-response techniques. Patient response to a changing CO_2 stimulus was measured spirometrically during pre-drug and post-drug periods, while a constant electroencephalographic level of thiopental narcosis was maintained for both periods by adjusting the drip inflow rate of thiopental as indicated by the EEG. The changing CO_2 stimulus was provided both before and after methylphenidate injection by allowing endogenous CO_2 to accumulate in a 9-liter closed rebreathing system for eight minutes. Strength of stimulus was measured in two parameters, P_{CO_2} of end-expiratory gas and of jugular bulb blood. Magnitude of response was measured as minute alveolar ventilation. Between the two periods of CO_2 accumulation, methylphenidate 0.55 mg./kg. was given intravenously. Stimulus and response magnitudes were observed in the second, fifth and eighth minutes of each rebreathing period. By dividing the observed values of P_{CO_2} and $\dot{V}_A$ by the respective control values, changes were expressed as "P_{CO_2} Ratio" ($P_{CO_2}R$) and "alveolar ventilation Ratio" ($\dot{V}_AR$). The ratios were plotted against each other on rectangular coordinates. Post-methylphenidate curves to the left of the pre-methylphenidate curve probably indicate change of respiratory center threshold and/or sensitivity in the direction of stimulation, while additive depression may be suspected if the "test" curve lies to the right of the control curve. Studies in 10 patients in whom end-expiratory gas P_{CO_2} was the only parameter by which stimulus was quantified indicated that methylphenidate does not favorably alter the ventilatory depression produced by thiopental under the conditions of this study. It is recognized that respiratory center activity is correlated more closely with the P_{CO_2} of jugular bulb blood than with the P_{CO_2} of end-expiratory gas, and studies are in progress to obtain this more accurate stimulus evaluation. [*Supported in part by a grant from Ciba Pharmaceutical Products, Inc.*]

Spectrophotometric Method for Analysis of Blood Ether Tensions. James A. Cutter, M.D., and Benton D. King, M.D. *Department of Anesthesiology, University of Buffalo and the Edward J. Meyer Memorial Hospital, Buffalo, New York.* A micro-analytical procedure, based on the colorimetric micro-diffusion method for alcohol (*Sunshine, I., and Nenand, R.: Anal. Chem. 25: 653, 1953*), has been developed for sampling ether tensions during anesthesia, which is both simple and accurate. The method utilizes a Conway micro-diffusion cell, the interior of which is divided into two concentric compartments. The blood sample is placed in the outer compartment, and a mixture of sulfuric acid and potassium dichromate is placed in the inner compartment. The 65 per cent sulfuric acid acts as a desiccant to accelerate diffusion of the ether into the center well and the dichromate oxidizes the ether to acetic acid, during which reaction it is reduced to chromic ion with a color change from the yellow acid-dichromate complex to the green chromic ion which may be measured with precision on a spectrophotometer at wave length of 428 millimicrons. During the diffusion reaction, the Conway cell is sealed with a ground glass cover to prevent loss of ether. The diffusion is accelerated by the addition of sodium carbonate to the blood in the outer compartment and by incubation for three hours at 90 C. Accurate calibration of the method was achieved by an apparatus capable of produc-