peated 20 minutes after atropine premedication and the arterial samples analyzed for arterial oxygen saturation. All the control arterial-blood measurements were greater than 95 per cent saturation. No significant change occurred in the arterial oxygen saturations after atropine.

Since dead space increases with lung volume, it is well when determining the effect of pharmacologic agents on the dead space to insure that the observed changes were not produced by lung volume increases. The data indicate that no significant volume changes occurred in these experiments. *Conclusion:* We were unable to demonstrate any significant difference in either tidal volume or anatomic dead space with change in body position. The injection of 0.8 mg. atropine caused a 25 per cent increase in anatomic dead space regardless of body position. Premedication with atropine was not followed by a change from the control arterial oxygen saturation measurements.

Minimum Alveolar Concentrations of Methoxyflurane, Halothane, Ether, Fluroxene, Cyclopropane and Nitrous Oxide in Man. LAWRENCE J. SAIDMAN, M.D., EDWIN S. MUNSON, M.D., EDMOND I. EGER, II, M.D., and ARTHUR A. BABAD, M.D., *Department of Anesthesia, University of California Medical Center, San Francisco, California.* The minimum alveolar concentrations (MAC) of halothane and fluroxene required to prevent movement in response to a skin incision have been previously determined (Saidman, L. J., and others: ANESTHESIOLOGY, *25:* 302, 1964; Munson, E., and others: ANESTHESIOLOGY, *26:* 143, 1965). Seventy per cent nitrous oxide reduced the MAC for halothane and for fluroxene. MAC values obtained in these studies without nitrous oxide were—halothane 0.74 per cent and fluroxene 3.4 per cent, while with nitrous oxide added they were—halothane 0.23 per cent and fluroxene 1.2 per cent. If it may be assumed that the effect of nitrous oxide was additive to that of the other agents, then a MAC of 101 per cent for nitrous oxide may be predicted. This study was performed to determine the MAC's for methoxyflurane, ether, and cyclopropane in man. *Methods:* The methods for ether and methoxyflurane studies were similar to those of the fluroxene study. Predetermined alveolar tensions of the agent being studied were held constant in surgical patients for a sufficiently long period to insure a brain tension of at least 95 per cent of that in the alveoli. When the skin incision was made the patient was observed for movement. MAC was defined as that concentration below which 50 per cent of the patients moved and above which 50 per cent failed to move. Inspired cyclopropane concentrations corrected for dilution by water vapor were measured rather than alveolar concentrations. This was considered valid since uptake of cyclopropane at the time of incision was low and balanced in part by an R/Q of less than one. *Results:* MAC for cyclopropane was 9.2 per cent, for ether 2.6 per cent, and for methoxyflurane 0.16 per cent, although more recent studies in our laboratory would indicate an ether MAC closer to 2.0 per cent. These results along with those for halothane, fluroxene, and nitrous oxide illustrate the relation between anesthetic potency and lipid solubility. The product of these two factors for each agent studied varies from 106 for cyclopropane to 174 for diethyl ether, a remarkably low range considering that these products are derived from anesthetic concentrations that vary over a 630 fold range. If this relation holds true for all general anesthetics, the average of these products (146) may be used to predict the approximate potency of any new anesthetic, given the oil solubility. *Conclusions:* The primary importance of MAC is in the establishment of what we believe are equipotent doses of anesthetic agents. With these doses comparative studies may be made of the circulatory, respiratory, metabolic or other effects of anesthetics. Thus 0.16 per cent methoxyflurane may be compared with 3.4 per cent fluroxene, each of these being equivalent anesthetic doses. (Supported in part by USPHS Grant 5R01 HE07946, 5-K3-GM-17, and 5T1-GM-63).

Halothane, Peripheral Vascular Resistance, and Respiratory Acidosis. RICHARD M. SCHLOBOUM, M.D., F. NORMAN HAMILTON, B.S., PETER J. TOMLIN, M.B., LUCIEN E. MORRIS, M.D., *Anesthesia Research Laboratories, Providence Hospital, Seattle, Washington.* Pe-