neuromuscular block and central depression from anesthetics. The finding of a decreased inspiratory force despite an adequate minute ventilation suggests a ventilatory handicap to the patient. This may cause a decreased ability of a patient to overcome even mild airway obstructions, to cough effectively and to take the occasional deep breath essential for the maintenance of normal pulmonary compliance. Apart from studying the inspiratory force we use this measurement clinically and find it useful in supplementing our evaluation of a patient's ventilatory capacity and recovery from the effects of muscle relaxants. We have established that an inspiratory force of 20 to 25 cm. of H_2O is necessary for adequate ventilation. Our experience suggests that the inspiratory force measurement may be a valid expression of ventilatory capacity, a "vital capacity measurement in the unconscious," and a useful and simple diagnostic tool.

The Combined Use of Narcotics and Narcotic Antagonists for Premedication. HENRY M. BRUNN, JR., M.D., FRANCIS F. FOLDES, M.D., PEARL G. MCNALL, M.D., AND LUDWIG R. KOUKAL, M.D. *Department of Anesthesiology, Mercy Hospital and Section on Anesthesiology, Department of Surgery, University of Pittsburgh School of Medicine, Pittsburgh, Pa.* Forty unselected surgical patients (group 1) received intramuscularly 1.5 mg./kg. meperidine, 0.3 to 0.4 mg. scopolamine and 100 mg. of pentobarbital sodium about two hours before induction of anesthesia. Forty other patients (group 2), besides these drugs, were also given intramuscularly 0.02 mg./kg. levallorphan at the same time. At zero time, after topical anesthetization of the pharynx with 1 per cent tetracaine, pulse rate, blood pressure and respiratory rate were recorded and thiopental sodium 5.0 mg./kg. was injected in 2 minutes through the rubber sleeve of an intravenous infusion. At 2 minutes, the administration of a 4 liter to 1 liter nitrous oxide-oxygen mixture was started through a face mask. At 4 minutes, the same parameters, together with the respiratory minute volume, measured with a Bennett ventilation meter, were again recorded. At 5 minutes, 20 patients each of group 1 (subgroup 1A) and group 2 (subgroup 2A) received 0.4 mg./kg. alphaprodine and 20 others of group 1 (subgroup 1B) and group 2 (subgroup 2B) 1.0 mg./kg. meperidine, intravenously, in 30 seconds. At 8 and at 12 minutes, pulse rate, blood pressure, respiratory rate and minute volume were again recorded. If at the time of the 8 minute reading, the patient was apneic, additional 0.02 mg./kg. levallorphan was injected intravenously. The administration of 5.0 mg./kg. thiopental caused little change in pulse rate, blood pressure or respiratory rate. The only difference observed between group 1, premedicated with meperidine alone, and group 2, premedicated with meperidine plus levallorphan was an 8 per cent decrease of the respiratory rate in the former and a 5 per cent increase in the latter. Levallorphan used with the premedication offered some protection against the respiratory effects of both alphaprodine and meperidine; for example, apnea developed in 12 out of 20 patients of subgroup 1A as contrasted with 2 out of 20 in subgroup 2A. Similarly incidence of apnea was 3 and 0 in subgroups 1B and 2B respectively. The depression of the respiratory rate and respiratory minute volume in the patients who did not develop apnea after intravenous alphaprodine (subgroups 1A and 2A) or meperidine (subgroups 1B and 2B) was also less in subgroups 2A and 2B premedicated with levallorphan plus meperidine. The results presented indicate that the intramuscular injection of 0.02 mg./kg. levallorphan afforded some protection against the respiratory depressant effects of large doses of narcotic analgesics administered intravenously 2 hours later. This protection, however, was less than that obtained when identical doses of levallorphan were injected intravenously immediately prior to, or after, the intravenous administration of identical doses of narcotic analgesics.

Effects of Muscle Relaxants on the Lungs and Circulation in Man. THOMAS J. DEKORNFELD, M.D., CHUNG J. PARK, M.D., AND PETER SAFAR, M.D. *Department of Anesthesiology, Baltimore City Hospitals, Baltimore, Md.* The effects of *d*-tubocurarine (0.3 mg./kg.) and gallamine (1.5 mg./kg.) upon lung-thorax compliance, airway resistance, radial artery pressure, heart rate, electrocardio-

gram and electroencephalogram were studied in healthy adults in the absence of surgery, during (1) cyclopropane anesthesia and (2) thiopental-meperidine-N_2O hyperventilation. A Jefferson ventilator provided continuously controlled respirations by intermittent positive pressure breathing (IPPB) at a constant rate and airway pressure range before, during and after the intravenous injection of the drug. Thus the respiratory and circulatory effects of the muscle relaxants were studied without interference from changes in respiratory patterns. *d-Tubocurarine:* During cyclopropane anesthesia the injection of *d*-tubocurarine increased the compliance from 8 per cent to 10 per cent of the control value (6 patients). The minute volume changes (indicating compliance and/or resistance changes) ranged from minus 6 per cent to plus 14 per cent of the control values (15 patients). The blood pressure, pulse pressure and heart rate varied less than 10 per cent of the control and there was no change in the electrocardiographic and electroencephalographic patterns (5 patients). With thiopental-meperidine-N_2O anesthesia the injection of *d*-tubocurarine caused essentially no changes in minute volume, blood pressure, pulse pressure, heart rate, electrocardiogram and electroencephalogram (5 patients). *Gallamine:* With cyclopropane anesthesia the injection of gallamine caused a slight increase in respiratory minute volume in 3 patients and no change in 5 patients. Tachycardia occurred in all 5 patients in whom circulatory measurements were made. The rise in heart rate varied from 45 to 98 beats per minute. The mean arterial blood pressure and pulse pressure changes were minimal. One patient had an episode of bundle branch block at the peak of the tachycardia. The electrocardiogram and electroencephalogram was unchanged in all the other patients. With thiopental-meperidine-N_2O anesthesia (5 patients) the injection of gallamine caused no change in minute volume. There was a moderate increase in heart rate (23 to 25 beats per minute). The mean arterial pressure rose slightly in all patients; the pulse pressure remained essentially unchanged. There was no change in the configuration of the electrocardiogram or electroencephalogram. Thus our preliminary data indicate that the histaminic side effects of *d*-tubocurarine, described in dogs, are absent or rare in healthy anesthetized adults. Gallamine caused a very marked tachycardia during cyclopropane anesthesia, while during thiopental-meperidine-N_2O anesthesia the tachycardia was of a minor degree. Similar studies using succinylcholine and decamethonium are now in progress. (*This study was supported by the Burroughs Wellcome Co., Inc., Tuckahoe, N. Y.*)

The Effect of Reserpine on the Action of Various Vasopressors. EDMOND I. EGER, II, M.D. AND WILLIAM K. HAMILTON, M.D. *Division of Anesthesiology, State University of Iowa Medical School, Iowa City, Iowa.* The amine oxidase inhibiting property of certain vasopressors has been hypothesized as the basis by which they act. Amine oxidase inhibition results in decreased epinephrine/norepinephrine destruction with a consequent increase in catechol amine level which in turn causes a rise in blood pressure. These vasopressors, then, are dependent for their action on the presence of epinephrine and norepinephrine. The recent finding that reserpine causes the elimination of epinephrine and norepinephrine would suggest that the above vasopressors would be ineffective following reserpine administration. To test this, a series of vasopressors with proven (ephedrine) or theoretical (methamphetamine, mephentermine, methoxamine) amine oxidase inhibiting properties was compared as to pressor effect with a series of vasopressors not thought to be amine oxidase inhibitors. This was done in hyperventilated dogs anesthetized with pentobarbital. The effects were noted, the animals allowed to recover, and 5–7 days later the identical procedure was repeated following the administration of 0.4 mg./kg. of reserpine intravenously 18 hours previously. The results were as anticipated for all of the vasopressors with the exception of methoxamine. Following reserpine the pressor action of intravenous ephedrine, methamphetamine, and mephentermine was decidedly diminished. On the other hand, the blood pressure response to phenylephrine, epinephrine, and norepinephrine was unchanged or even enhanced by prior administration of reserpine. Methoxamine was the exception in the series, and