tained, or until the patient wakes up. Before and after each period of airway occlusion, arterial blood samples are taken for blood gas analysis. To date 14 patients have been studied. The average inspiratory force measurements were: before neostigmine 3 mm. Hg, and after neostigmine 23 mm. Hg. The average dose of neostigmine used was 2.3 mg., the largest dose was 3 mg. and the smallest 1 mg. The full effect of neostigmine was not present in 2 minutes, but appeared to be within 5 minutes. In one case the third milligram of neostigmine not only did not further improve the inspiratory force, but was actually followed by a decrease. No cardiac irregularities or significant blood pressure changes were seen and in only one case did the pulse rate after the last dose of neostigmine go below the rate before atropine. In 7 cases changes in blood gases during 30 seconds of airway occlusion were studied. The average fall in oxygen saturation was 12 per cent and the average rise in carbon dioxide tension was 7 mm. Hg. These patients received 30 per cent oxygen. The inspiratory force measurement appears to be a simple and safe tool for objective evaluation of reversal of curare effect, provided the period of airway occlusion does not exceed 30 seconds. For clinical purposes only a minimum of equipment is necessary, namely, a mask and a manometer registering negative pressure. With regard to the clinical use of neostigmine it deserves emphasis that in the reversal of curare effect not only must minute ventilation be restored, but the reversal must be continued in order to secure an adequate reserve of ventilatory effort.

Studies on the Combined use of Halothane and Neuromuscular Blocking Agents. B. WOLFSON, M.B., F. F. FOLDES, M.D., AND M. SOKOLL, M.D., *Department of Anesthesiology, Mercy Hospital and the Section on Anesthesiology, Department of Surgery, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania.* Although it has been stated that halothane alone frequently produces adequate muscular relaxation for abdominal surgery (*Abajian, J., Jr., Brazell, E. H., Dente, G. A., and Mills, E. L.: Anesthesiology 19: 93, 1958; Marrett, H. R.: Anaesthesia 14: 28, 1959*), in our experience, the halothane concentrations required to achieve this have almost invariably caused bradycardia, hypotension and, on occasion, tachypnea. Consequently, a study was initiated to determine which of the clinically used neuromuscular blocking agents are suitable for the production of muscular relaxation in conjunction with light halothane anesthesia. To date, three relaxants, succinylcholine, gallamine and toxiferine have been investigated. Ten patients, premedicated with pentobarbital sodium, meperidine and scopolamine, received 2.0 and 1.5 per cent halothane, vaporized in a 2 liter N_2O-2 liter O_2 mixture from a Vernitrol vaporizer, for 5 and 15 minutes respectively. Following this, tidal volume was measured with a Bennett ventilation meter and 0.6 mg./kg. succinylcholine chloride was injected intravenously. Onset of apnea and duration of apnea and respiratory depression were measured. The onset of apnea (70.5 $\pm$ 2.6 seconds) was slower, the duration of apnea (131.0 $\pm$ 11.4) was shorter and duration of respiratory depression was somewhat longer than under thiopental sodium-N_2O anesthesia. The gallamine requirements were measured in two groups of patients: One anesthetized with thiopental sodium-N_2O-O_2 and analgesics; the other primarily with halothane after induction with small doses of thiopental. Similar observations were also made with toxiferine. Two per cent halothane was administered at first in a 2 liter N_2O-2 liter O_2 mixture for 5 to 10 minutes and subsequently 300 ml. N_2O-700 ml. O_2. With the progress of anesthesia, the halothane concentration was gradually decreased to the minimal effective level. The gallamine and toxiferine requirements were about 30 and 50 per cent lower respectively in the halothane groups than in the control groups. Apnea developed following the first does of relaxant with about the same frequency (40 to 50 per cent) in all four groups; its duration, however, was generally shorter after gallamine than toxiferine. Although tidal volumes returned to about 70 to 90 per cent of control at the end of surgery, because of the character of breathing, the use of an antagonist (neostigmine 0.02 mg./kg.) was deemed advisable in about 25 per cent of the patients in whom gallamine and in 45 to 50 per cent of the patients in whom toxiferine was used. Following the use of an antagonist,

tidal volumes returned to, and were maintained at or above 95 per cent of control. Gallamine counteracted the halothane induced bradycardia and decreased the hypotension frequently seen after its use.

Clinical Evaluation of Carbocaine (Mepivacaine HCl). THOMAS Q. ZIEGLER, M.D., CHARLES GIASER, M.D., DAVID N. GOODSON, M.D., K. HORI, M.D., AND JOHN J. BONICA, M.D., *Department of Anesthesiology, Tacoma General Hospital, Tacoma, Washington.* The following evaluation of Carbocaine is a continuance of our long term assessment of local anesthetic drugs under clinical conditions. An attempt has been made to evaluate 2 per cent Carbocaine with 2 per cent lidocaine utilizing extradural blocks as the method of anesthesia. The principles promulgated in a previous publication regarding the evaluation of local anesthetics have been adhered to. *Method:* The drugs were distributed as unknowns according to random tables and the investigators followed standardized procedure. Peridural blocks were performed at L5 interspace with the catheter being advanced 15 cm. relative to the hub of the needle. In caudal blocks the catheter was also advanced 15 cm. The amount of solution was 15 ml. for peridural and 25 ml. for caudal. Epinephrine 1/200,000 was added. The solution was injected at a constant rate with the patient in the supine position. Usually 60–90 seconds was consumed for the injection. The properties of the drugs that were studied included: (1) Latency or time of onset; (2) penetrance or spread of solution; (3) duration; and (4) toxicity. Sensation was tested by pinprick; a dull pinprick was interpreted as hypalgesia; loss of a sharp sensation in the presence of pressure as analgesia and complete absence of sensation as anesthesia. Each case was recorded on a special form. The time of onset of these sensory changes at T10 level was noted and a complete survey was made fifteen minutes after initial injection. A difference in either analgesia or anesthesia of three or more segments between each side was considered to be uneven spread. Upon completion of each series the results were set out as absolute frequencies and submitted to statistical analysis. *Results:* The patients chosen for the study ranged in age from 21–66. The average age was 44. Over half of the patients were in the fourth decade. The fifth slide shows the mean latency of the two drugs. The mean latency for hypalgesia in the peridural series with Carbocaine was 6.1 minutes; for lidocaine 6.7 minutes. The latency for analgesia with Carbocaine was 10.2 minutes; for lidocaine 11.1 minutes. The latency for anesthesia with Carbocaine was 13.4 minutes; for lidocaine 11.5 minutes. The mean latency with the caudal technique for hypalgesia with Carbocaine was 7.2 minutes and with lidocaine 6.3 minutes. Analgesia was obtained in 11.7 minutes with Carbocaine and 11.5 minutes with lidocaine. Anesthesia occurred in 14 minutes with Carbocaine and 11.5 minutes with lidocaine. The above figures refer to the amount of time that it took the varying degrees of the block to reach a T10 level. Inadequacy of the block refers to failure to obtain the tested parameters at T10 level in 15 minutes. In the peridural series 25 per cent of the patients had analgesia in 4–7 minutes with both drugs. Forty-two per cent had analgesia in 11 minutes with lidocaine while 25 per cent had analgesia in 8–11 minutes with Carbocaine. Twenty-five per cent of the patients had analgesia at a T10 level in 12–15 minutes with lidocaine and 42 per cent in 12–15 minutes with Carbocaine. With both lidocaine and Carbocaine, 8.5 per cent had a latency of over 15 minutes. In the caudal series 33 per cent had analgesia at T10 level with lidocaine in 4–7 minutes; 8.5 per cent with Carbocaine. Seventeen per cent had analgesia in 8–11 minutes with lidocaine while 33 per cent had analgesia at T10 with Carbocaine. Forty-two per cent of the patients had analgesia within 12–15 minutes with both lidocaine and Carbocaine while 8.5 per cent had a latency of more than 15 minutes with Carbocaine. In the peridural series, 17 per cent of the patients had a spread of 5–9 segments with lidocaine and 8.5 per cent had a spread of 5–9 segments with Carbocaine. Forty-one per cent of the patients had a spread of 10–14 segments with lidocaine while 57 per cent had the same segmental spread with Carbocaine; 42 per cent of the patients had a spread of 15–19 segments with lidocaine and 34.5 per cent had a spread of 15–19 segments with