

tion of all balloons in the small bowel was verified at laparotomy. Observations were without value in 5 patients. In two of these satisfactory inflation of the balloon could not be obtained because the tube was kinked. In two no changes in intestinal tone were apparent and motility was absent. In the remaining 7 patients, satisfactory recordings were obtained. Spinal anesthesia was administered to five. Sensory anesthesia to T6-T4 was obtained. Intraluminal pressure rose in all, the degree of rise varying from 15 to 100 per cent. Three of these patients were given thiopental intravenously in increments of 50 to 100 mg. In all intraluminal pressure decreased within 30 seconds. In one patient, the pressure remained low for several minutes but the picture was complicated by systemic hypotension. The intraluminal pressure returned promptly to former levels with the intravenous injection of phenylephrine. In one patient, the fall in pressure was transient. In the other 2 patients, after spinal anesthesia was established, the patients were given a minimal dose of thiopental, followed by curarization with 15 and 21 mg. *d*-tubocurarine, endotracheal intubation and controlled respirations with O<sub>2</sub> and subsequently N<sub>2</sub>O and O<sub>2</sub>. In both intraluminal pressure which had risen following the spinal fell below the control levels after the curare and remained low for 15 minutes, when it gradually began to increase though never reaching control levels. The final two patients were given general anesthesia. In one, anesthesia was induced with two 100 mg. doses of thiopental. Intraluminal pressure fell below control levels. Succinylcholine 40 mg. was given intravenously and the trachea was intubated. Nitrous oxide 6 l./min. and oxygen 2 l./min. was given with controlled respiration. Intraluminal pressure gradually rose and remained about 50 per cent above control levels for 5 minutes at which time 18 mg. *d*-tubocurarine was given intravenously. Intraluminal pressure then fell to about 20 per cent below control levels and remained there for 16 minutes at which time the operation began. In the last patient anesthesia was induced with 75 mg. thiopental followed by 50 mg. Intraluminal pressure declined slightly but definitely. Cyclopropane was then given for 2 minutes at which time intraluminal pressure

rose slightly. Eighteen milligrams *d*-tubocurarine were given intravenously followed in 5 minutes by an additional 9 mg. Intraluminal pressure declined rapidly to about 70 per cent of control levels with minimal evidence of intestinal motility. It remained there for 15 minutes while light cyclopropane anesthesia was continued. At this time 0.5 mg. prostigmine was given intravenously. In 3 minutes intraluminal pressure had returned to control levels or slightly above with vigorous intestinal motility. The factors that have already been described as leading to a diminished intestinal tone and motility are numerous and have not all been verified in man. Only a relatively few factors have been described as causing an increase in intestinal tone and motility. These include vagal stimulation, oxygen, prostigmine, avertin, light cyclopropane and spinal anesthesia. Our observations of the effects of spinal anesthesia, thiopental, cyclopropane, hypotension, *d*-tubocurarine, and prostigmine agree with the above categorization. Nitrous oxide, on the other hand, did not appear to be an intestinal depressant. It would appear that the effects produced by anesthetics on bowel tone is a function of dose of drug and depth of anesthesia. Even though a drug such as curare, used in relating large dosage, decreases intestinal tone, this appears limited to about 15 minutes. The effect of repetitive doses is undetermined, nor did we investigate the effects of narcotics which presumably would have a more prolonged effect. Manipulation of the bowel, as might occur during abdominal operation, might significantly affect the results as described.

**The Effect of Various Concentrations of Oxygen on the Pulmonary Circulation.** G. W. N. EGGERS, JR., M.D., AND H. W. PALEY, M.D. *Departments of Anesthesiology and Internal Medicine, University of Texas Medical Branch, Galveston, Texas.* The recent advent of cardiac catheterization has made possible the exploration of the pulmonary circulation. While many investigators have studied the effects of hypoxia and certain drugs on the pulmonary circulation (Euler, U. S., and Liljestrand, G.: *Acta. physiol. scandinav.* 12: 301, 1946; Lanari-Zabaiur, F. J., and Hamilton, W. F.: *Circulation Res.* 6: 289, 1958), only a

few have reported limited studies on the acute effects of high oxygen concentration inhalation (Barratt-Boyes, B. G., and Wood, E. H.: *J. Lab. & Clin. Med.* 51: 72, 1958). Our method of study was to place catheters in both sides of the heart and to vary the inspired oxygen concentration. Determinations of cardiac output, central blood volume, pulmonary arteriolar resistance and alterations of mean pressure in the pulmonary artery, left atrium and aorta were then made. Dogs anesthetized with pentobarbital had their tracheas intubated, and then catheters were inserted in the following sites: pulmonary artery and right ventricle via the external jugular vein, left atrium via the carotid artery, femoral artery and aorta. The catheters were inserted under fluoroscopy and their location confirmed by pressure measurements. Radioactive indicators (RISA or Diodrast) were injected into the right ventricle and timed aortic samples obtained. Cardiac output and central blood volume were calculated by the method of Stewart and Hamilton. Each dog served as his own control. The first group of 7 dogs was allowed to breathe spontaneously and comparative observations made after ten minutes of both air and 96 per cent oxygen inhalation were inconclusive and inconsistent. The indicator injection was not made during any particular phase of respiration and the order of gas administration was reversed in 3 animals. The second group of animals was paralyzed with succinylcholine and their lungs artificially ventilated. After inhalation of various oxygen mixtures for at least 30 minutes the ventilator was disconnected just prior to the indicator injection. The dilution curve determination was made within 30-40 seconds and in the end-expiratory phase of respiration. Four dogs in this group had an additional study with 50 per cent oxygen. The order of study was arbitrarily varied. Arterial blood samples for pH, oxygen, carbon dioxide and hematocrit determinations were drawn just prior to each dilution curve and all blood lost in sampling was later replaced. During the study, arterial pH values did not vary more than 0.09 pH units from the control measurements except in one instance (0.11 pH units) and carbon dioxide content did not vary more than 1.2 mM/l. from the control except in another study (3.16

mM/l.). The control values were all normal (Galla, S. J., et al.: *Anesthesiology* 20: 124, 1959). Comparison of average control central blood volumes (per cent of body weight) revealed a significant ( $p < 0.05$ ) difference: spontaneous respiration—1.16 per cent and artificial ventilation—2.12 per cent. The changes from air to 96 per cent oxygen inhalation were as follows: *cardiac output*—no consistent effect and a wide range of values, agreeing with observations of others (Howell, C. D., et al.: *Am. J. Physiol.* 196: 193, 1959); *mean pulmonary artery pressure*—decreased an average of 2.61 mm. Hg which was statistically significant ( $p < 0.05$ ), agreeing with other studies (Weil, P., et al.: *Am. J. Physiol.* 191: 453, 1957); *central blood volume and pulmonary arteriolar resistance*—no consistent effect and a wide range of values with which there are no comparable studies. Changes in mean left atrial pressure and mean aortic pressure were very small and statistically not significant. Changes associated with 50 per cent oxygen ventilation were even less consistent. Although this study did not reveal any new effects of oxygen administration on the pulmonary circulation, we believe the method of study valuable for investigation of pulmonary vascular physiology.

**Gas Chromatography as an Analytical Tool in Anesthesiology.** L. W. FABIAN, M.D., AND MARION A. CARNES, M.D. *Department of Anesthesiology, University of Mississippi Medical Center, Jackson, Mississippi.* Gas chromatography has been explored only recently for possible medical applications. This method provides rapid and accurate quantitative analysis of individual components in gas or gas-vapor mixtures and would seem particularly applicable in the field of anesthesiology. Accordingly, an investigation of the merits and over-all practicality of gas chromatography in laboratory and clinical anesthesia was undertaken. The equipment used in these studies included a Beckman GC-2 Chromatograph, a Minneapolis-Honeywell Brown Recorder, a Brown Integrator and a Sola constant voltage transformer. The use of this combination of equipment provided automatic and reproducible analyses of all components in gas mixtures within a period of 5 to 8 minutes.