bolic acidosis in 11/13 patients. Pa_{CO_2} was 17–35 mm. Hg. The lowest Pa_{O_2} values in patients with $F_IO_2 = 1$ ranged between 40 and 145 mm. Hg. IPPB or IPPV/O_2 increased Pa_{O_2}. II. *Cardiogenic Shock with Pulmonary Edema* (nine patients). Two/9 survived. All had metabolic acidosis with hypocarbia (lowest Pa_{CO_2} value 15 mm. Hg). Pa_{O_2} values during spontaneous breathing of 100 per cent oxygen were 50–140 mm. Hg. As expected, IPPB or IPPV/$F_IO_2 = 1$ cleared pulmonary edema in most cases, and increased Pa_{O_2} (Miller, W. F., and Sproule, B. J.: Dis. Chest *35:* 469, 1959). III. *Uncomplicated Myocardial Infarction* (13 patients). All survived. Only one patient had evidence of metabolic acidosis. Pa_{CO_2} was variable. During spontaneous breathing of air, Pa_{O_2} values were 44–95 mm. Hg (in 9/13 patients, below 70 mm. Hg). During spontaneous breathing of 100 per cent oxygen, Pa_{O_2} was 154 to 550 mm. Hg. In four patients IPPB/$F_IO_2 = 1$ changed Pa_{O_2} from 295 to 430; 360 to 320; 340 to 500; and 310 to 430 mm. Hg, respectively. IV. *Miscellaneous Shock States* (seven patients). Three had oligemic shock, three septic shock, and one shock with diabetic acidosis. Two patients with oligemic shock and one with septic shock died. Blood gas changes were similar to those seen in cardiogenic shock. The lowest Pa_{O_2} values occurred in septic shock, 30 to 60 mm. Hg with IPPV/$F_IO_2 = 1$. *Comment:* Histologic changes in the lungs of subjects with shock as reported by others, include intra-alveolar and interstitial edema, hemorrhage, fibrin deposits, emboli, and thrombi. The hypoxemia observed seems to be the result of a combination of the following factors: (1) increased V_D/V_T, known to occur in oligemic shock (Gerst, P. H., Rattenborg, C., and Holaday, D. A.: J. Clin. Invest. *38:* 524, 1959), vasodilatation, hypotension (Askrog, V. F., Pender, J. W., and Eckenhoff, J. E.: ANESTHESIOLOGY *25:* 744, 1964), and cardiogenic shock (McNicol, M. W. *et al.:* Brit. Med. J. *2:* 1270, 1965); (2) V/Q mismatching; (3) diffusion block (*e.g.*, interstitial edema); (4) increased Q_S/Q_T, perhaps due to alveolar collapse from pulmonary congestion, edema, obstruction or lack of deep breaths (MacKenzie, *et al.:* Lancet *2:* 825, 1964); and (5) decrease in Q_T without change in Q_S/Q_T (decreased $P_{\bar{v}}O_2$). Hypoxemia due to factors (1) to (3), apparently predominant in uncomplicated myocardial infarction, can be corrected by simple oxygen enrichment (*e.g.*, $F_IO_2 = 0.5$). Hypoxemia due to increase in Q_S/Q_T can be partially reversed by IPPV/$F_IO_2 = 1$. Hypoxemia due to decreased Q_T needs circulatory support. The effect of these measures on tissue oxygenation is unpredictable unless cardiac output and oxygen consumption are measured simultaneously. (Supported by U. S. Army Contract No. DA-49-193-MD-2160.)

The Circulatory Effects of the Addition of Nitrous Oxide to Halothane Anesthesia in Man. N. TY SMITH, M.D., E. I. EGER, II, M.D., CHARLES E. WHITCHER, M.D., R. K. STOELTING, M.D., and T. F. WHAYNE, M.D., *Department of Anesthesia, Stanford Medical School, Palo Alto, and University of California, San Francisco, Calif.* Reports describing the circulatory effects of adding nitrous oxide to halothane anesthesia have been contradictory. Some claim stimulation; others, including clinical reports, claim depression. We have investigated this problem in nine normal unpremedicated 21-year-old male volunteer subjects. *Method:* Anesthesia was induced and maintained with halothane-oxygen. Ventilation was controlled with a fixed-volume ventilator to maintain alveolar P_{CO_2} between 30 and 35 mm. Hg. After a stable level of halothane-oxygen anesthesia had been obtained (0.8, 1.0, 1.6, or 2.0 per cent alveolar halothane concentration), the diluent was changed either to nitrous oxide/oxygen 75/25 or to air. Immediately before and 15 minutes after the change, dye-dilution cardiac outputs and occlusion plethysmograph forearm blood flows were measured, and arterial blood was withdrawn for measurement of blood gases and catecholamines by the Weil-Malherbe method. Electrocardiogram, heart rate, direct brachial arterial pressure, right atrial pressure, and external carotid artery pulse were recorded continuously. Several other parameters were calculated from these measurements. *Results:* The following are the per cent changes and standard deviations in cardiovascular variables

produced by shifting from air or oxygen to nitrous oxide at various levels of halothane anesthesia: right atrial pressure +2.11 ± 0.95 mm. Hg ($P < 0.001$); cardiac output −1.31 ± 19.96 ($P > 0.05$); mean arterial pressure +14.01 ± 16.66 ($P < 0.01$); heart rate +2.05 ± 6.94 ($P > 0.05$); stroke volume −3.35 ± 17.75 ($P > 0.05$); left ventricular minute work 13.87 ± 38.14 ($P > 0.05$); total peripheral resistance +18.24 ± 19.47 ($P < 0.001$); central blood volume +8.11 ± 12.97 ($P < 0.02$); ejection time index +1.37 ± 7.89 ($P > 0.05$); mean rate of left ventricular ejection −3.15 ± 20.49 ($P > 0.05$); left ventricular stroke work +11.93 ± 34.68 ($P > 0.05$); left ventricular stroke power +18.19 ± 31.10 ($P < 0.05$); peripheral (forearm) blood flow −20.85 ± 27.57 ($P < 0.05$); peripheral venous pressure +23.28 ± 20.86 ($P < 0.02$); venous compliance −22.87 ± 24.89 ($P < 0.05$); peripheral (forearm) resistance 67.63 ± 70.41 ($P < 0.05$). The major changes occurred at the deeper levels of halothane (1.6 and 2.0 per cent). Adding nitrous oxide to 0.8 or 1.0 per cent alveolar halothane concentrations produced a significant change (increase) in right atrial pressure only. Adding air to halothane-oxygen significantly changed heart rate only (increase). Plasma norepinephrine levels increased from 0.97 ± 0.33 to 1.33 ± 0.22 μg./ml. ($P < 0.02$) when nitrous oxide was added, whereas epinephrine levels did not change. *Discussion:* The addition of nitrous oxide to constant levels of halothane anesthesia produced significant cardiovascular effects. Contrary to previous reports, these effects were those related to peripheral vascular stimulation. The differences in results could be due to age, surgical stimulation, premedication, depth of anesthesia, and controlled ventilation. The effects appear to be explained by the release of norepinephrine, although a direct effect of nitrous oxide cannot be ruled out. The cause and site of norepinephrine release cannot be ascertained from these studies, nor can it be determined whether this release would be clinically beneficial or detrimental. The lack of response to the addition of nitrous oxide at light levels of halothane anesthesia may be due to higher initial values and to the presence of reflex vasoconstriction. The latter may have been abolished with the added depth produced by nitrous oxide, thus balancing the effects of released norepinephrine. *Conclusions:* The addition of nitrous oxide to halothane produces stimulation of the peripheral vascular system probably due to norepinephrine release. This response was absent at light levels of halothane anesthesia. (Supported by USPHS Grant GM-12527, NIH Grant 1-K3-GM-31, 757, and USPHS Grant 5 R01-HE 09746.)

Observations on the Effect of Circumferential Pneumatic Compression. JOHN STORER, M.D., W. JAMES GARDNER, M.D., and M. P. THOMAS, M.D., *Huron Road Hospital, East Cleveland, Ohio.* Circumferential pneumatic pressure (G suit effect) has been utilized for many years in the control of postural hypotension during neurosurgical procedures. Certain change circumstances suggested that this divice might effectively control intra-abdominal bleeding and be useful in management of hypovolemic shock. About 30 patients with significant intra-abdominal bleeding have been treated by this method with G suit pressures of 20 to 30 mm. Hg. The consistently beneficial effects have led to an investigation of the mechanism involved, its limitations, untoward effects, and contraindications. The study consisted of: (1) an evaluation of incisions in the aorta and arteries of mongrel dogs, and (2) a study of the G suit at varoius pressures in human volunteers. *Methods:* In a series of eight mongrel dogs, a 5.0 millimeter longitudinal incision was made in the anterior wall of the abdominal aorta isolated by a curved vascular clamp. As carotid artery pressures were recorded, the clamp was released and the G suit, previously placed, inflated to 30–40 millimeters of mercury. At the end of an hour the G suit deflated. This experiment showed consistent control of bleeding and maintenance of blood pressure during the period of G suit inflation. All dogs died following deflation of the suit. Another series of sixteen dogs was subjected to transection of the iliac artery one cm. from its origin. Eight acted as controls and the remaining eight were treated with G suit pressures of 30 mm. Hg. The control animals died promptly,