ACID-BASE BALANCE Changes in acid-base balance in response to the administration of carbon dioxide were studied in five dogs anesthetized with chloralose. A respiratory pump was adjusted to produce a P_{CO_2} in the arterial blood of about 40 mm. of mercury. Carbon dioxide was added to the inspired gas mixture to give a concentration of approximately 12 per cent. During the period of hypercarbia the *pK* value decreased significantly and in an opposite direction to that of blood equilibrated with various carbon dioxide tensions *in vitro*. The change in *p*H with the increase in P_{CO_2} was greater than, and the change in plasma bicarbonate ion concentration less than, that predicted for the behavior of blood *in vitro*. An explanation of this difference in the behavior of the whole animal and blood *in vitro* may be offered when it is considered that both skeletal muscle and extracellular fluid have smaller buffering capacities for carbon dioxide than blood. (*Linden, R. J., and Norman, J.: The Effects of Increases in P_{CO_2} on Acid-Base Balance, J. Physiol. 185: 75P (July) 1966.*)

METABOLIC ACIDOSIS In 50 cases of acute myocardial infarction 66 per cent showed significant metabolic acidosis. Twelve of the 13 patients who died showed a significant base deficit. In patients in shock, correction of acidosis usually resulted in rise of systolic blood pressure. A significant metabolic acidosis developing within 12 hours of infarction was associated with a 28 per cent mortality. (*Neaverson, M. A.: Metabolic Acidosis in Acute Myocardial Infarction, Brit. Med. J. 2: 383 (Aug.) 1966.*)

Respiration

POSTHYPERVENTILATION APNEA Posthyperventilation apnea (PHA) was consistently demonstrated in conscious trained dogs. Although hypoxia and metabolic acidosis lowered the apneic threshold, PHA also regularly occurred during these states. PHA is due to direct effect of hypocapnea on the respiratory centers rather than to summation of vagal impulses. Evidence for this includes failure to produce PHA when arterial CO_2 tensions were maintained at normal levels during hyperventilation by addition of CO_2 to inspired gas and presence of a few breaths after hyperventilation ceased and before apnea occurred. PHA and the shift of apneic threshold with hypoxia and acidosis are more easily demonstrated in the dog than in man. (*Mitchell, R. A., Bainton, C. R., and Edelist, G.: Posthyperventilation Apnea in Awake Dogs During Metabolic Acidosis and Hypoxia, J. Appl. Physiol. 21: 1363 (July) 1966.*)

LUNG PERFUSION Distribution of lung perfusion was sudied in erect man using ^{133}Xe. The apex of the lung, where alveolar pressure exceeds pulmonary artery pressure was virtually unperfused. Further down the lung, where alveolar pressure was less than pulmonary artery pressure but greater than pulmonary venous pressure, a zone of sharply increasing flow was encountered. Toward the lung bases, where both pulmonary arterial and venous pressure exceed alveolar pressure, increase in flow was less rapid as the bases were approached. These observations in intact man confirm those previously made with isolated dogs' lungs. Pattern of perfusion was altered at both extremes of lung volume by changes in pulmonary arterial and venous pressure. (*Anthonisen, N. R., and Milic-Emili, J.: Distribution of Pulmonary Perfusion in Erect Man, J. Appl. Physiol. 21: 760 (May) 1966.*)

VENTILATION AND PERFUSION Regional subdivisions of lung volume and distribution of pulmonary ventilation and perfusion were studied using ^{133}Xe in 8 healthy conscious volunteers in the supine, prone, left and right lateral positions. In general, findings confirmed previous observations made in erect man that regional distribution of resting lung volume, pulmonary ventilation, and pulmonary perfusion are gravity-dependent and vary with vertical distance from the uppermost part of the lung. Many of the observed differences could be explained by the fact that in the positions studied, vertical dimension of the lung is much less than that in erect man. Evidence was presented that airway closure might occur at higher lung volumes in the lateral position than in the erect position. (*Kaneko, K., and others: Regional Distribution of Ven-*

tilation and Perfusion as a Function of Body Position, J. Appl. Physiol. 21: 767 (May) 1966.)

VENTILATION DISTRIBUTION Distribution of inspired gas was studied using radioactive Xenon in isolated, perfused dogs' lungs. No change in distribution of inspired gas occurred when the magnitude of blood flow to different regions of the lung was altered by large variations in pulmonary arterial and venous pressure. When the isolated lungs were suspended in air, the relative distribution of inspired gas to upper and lower lung zones was uniform. When lungs were suspended in egg-albumin foam, to simulate the gradient in pleural pressure down the intact lung, inspired gas was distributed preferentially to upper zones at low lung volumes and to lower zones at higher lung volumes. Uneven distribution of ventilation in the human lung is not caused by the uneven distribution of blood flow, but by a vertical gradient of pulmonary pressure. *(Zardini, P., and West, J. B.: Topographical Distribution of Ventilation in Isolated Lung, J. Appl. Physiol. 21: 794 (May) 1966.)*

A-A P_{CO_2} DIFFERENCE Pulmonary artery pressure was varied in anesthetized, paralyzed, artificially ventilated subjects by rapid infusion of 1,000 ml. of saline or by head-up or head-down tilt. Arterial-alveolar carbon dioxide difference varied inversely as pulmonary artery pressure and in subjects with a mean pulmonary artery pressure in the region of 20 mm. of mercury, a-A CO_2 difference was about 1 mm. of mercury, which is comparable to that in normal, conscious subjects. The increased a-A P_{CO_2} during anesthesia may be explained by a marked reduction of pulmonary capillary blood flow in the uppermost part of the lung, a reduction that is caused by a drop in pulmonary perfusion pressure. *(Askrog, V.: Changes in the (a-A) CO_2 Difference and Pulmonary Artery Pressure in Anesthetized Man, J. Appl. Physiol. 21: 1299 (July) 1966.)*

A-A P_{CO_2} DIFFERENCE When inspired gas is changed from air to oxygen in conscious subjects, increase in ventilation occurs, alveolar carbon dioxide tension falls, arterial carbon dioxide tension increases and arterial to alveolar carbon dioxide difference increases. The predominant effect is on alveolar carbon dioxide tension. Although the Haldane effect is present, the most important cause for the observed increase in a-A CO_2 tension difference is change in distribution of ventilation perfusion ratios secondary to shift of blood flow during oxygen breathing. *(Lenfant, C.: Arterial-Alveolar Difference in P_{CO_2} During Air and Oxygen Breathing, J. Appl. Physiol. 21: 1356 (July) 1966.)*

ATELECTASIS Four of 7 subjects exposed to 100 per cent oxygen at sea level and at several simulated altitudes for varying periods of time exhibited signs of pulmonary atelectasis as manifested by decreased vital capacity and/or X-ray evidence of areas of plate-like atelectasis. Individuals with alterations of pulmonary function predisposing to air trapping may have increased susceptibility to atelectasis during oxygen breathing. Absorption atelectasis occurring during oxygen breathing may be prevented by addition of 5 to 30 per cent nitrogen to the oxygen. *(Dubois, A. B., and others: Pulmonary Atelectasis in Subjects Breathing Oxygen at Sea Level or at Simulated Altitude, J. Appl. Physiol. 21: 828 (May) 1966.)*

SHUNTING Physiological shunt exceeding 8 per cent of cardiac output was uniformly observed in anesthetized, artificially ventilated subjects during hypocapnia. When carbon dioxide was added to the inspired mixture, the resulting elevation in arterial carbon dioxide tension was accompanied by an increase in arterial oxygen tension and a decrease in shunt. There changes occurred during use of both 40 per cent and 100 per cent oxygen in the inspired gas and when the cardiac output was unchanged. If cardiac output changed, then the magnitude of shunt changed in a similar direction and the effect of changing cardiac output interacted with or overrode the effect of carbon dioxide. The explanation for the observed changes is obscure. *(Michenfelder, J. D., Fowler, W. S., and Theye, R. A.: CO_2 Levels and Pulmonary Shunting in Anes-*