tion at this dose level of AY 6204, 2 mg./kg., diminished after 30 to 60 minutes. Depression of arterial pressure and pulse rate due to the AY 6204 was minimal and found not to be statistically significant. Both animals who breathed spontaneously attained arterial P_{CO_2} levels between 73 and 79 mm. of mercury. The eight who were artificially ventilated did not exceed 45 mm. of mercury. Halothane levels varied widely from one dog to another but in six a steady state was maintained throughout the period of testing. *Discussion:* This particular dosage of the blocking agent was used because it had been shown to produce little hemodynamic depression. It afforded at least a twofold protection against exogenous epinephrine during halothane anesthesia in dogs. More information should be obtained about its intrinsic effects upon the cardiovascular system subjected to the stress of hemorrhage and anesthesia. (Supported in part by N.I.H. Grant GSR 1963-13.)

Apneic Oxygenation Following Respiration with Air or Nitrous Oxide-Oxygen Mixture. M. L. HELLER, M.D., T. R. WATSON, JR., M.D., and D. S. IMREDY, Ph.D., *Hitchcock Clinic and Dartmouth Medical School, Hanover, New Hampshire.* In a recent polarographic P_{O_2} study we observed that after preliminary oxygenation followed by apnea there was a marked difference in the rate of arterial deoxygenation, depending on whether the airway was open to room air or attached to an oxygen reservoir. The arterial oxygen tension fell rapidly in the former situation, whereas P_{O_2} values greater than 400 mm. of mercury were noted after five minutes of apnea when there was mass flow of oxygen down the airway (to be published in ANESTHESIOLOGY). In the present investigation apneic oxygenation was studied in man without preliminary oxygenation. *Method:* Several patients under anesthesia in preparation for surgery were ventilated first with (1) air followed by apnea, and subsequently (2) with a gas mixture of 80 per cent N_2O-20 per cent O_2, also followed by apnea. Arterial blood samples were withdrawn at one-half to one minute intervals and measured for oxygen tension with our laboratory polarograph (New Engl. J. Med. *264:* 326, 1961). *Results:* When apnea follows air breathing the arterial P_{O_2} fell rapidly to hypoxic levels no matter whether the airway was connected to a source of oxygen or open to air. It appears that when the alveolar space contains an original high nitrogen concentration there was very little mass flow of ambient gas (oxygen or nitrogen) down the airway. On the other hand, arterial oxygenation was quite different when patients were ventilated for a few minutes with an 80–20 nitrous oxide-oxygen mixture prior to apnea. In this situation when the endotracheal tube was attached to a reservoir bag filled with oxygen, the arterial oxygen tension showed no fall; or it actually demonstrated a small increase during the aventilatory period. Apparently oxygen molecules moved down the airway. *Discussion:* The underlying mechanism may be explained as follows: nitrous oxide molecules are readily taken up by the pulmonary capillary blood (until equilibrium is established) and removed from the alveolar space. This produces a lowering of the alveolar barometric pressure, and a pressure gradient is established between the airway opening and the alveoli. A mass flow of ambient gas occurs. If the atmosphere is oxygen, alveolar P_{O_2} is kept at an adequate level. When the atmosphere is air, the existing alveolar oxygen is diluted by the added nitrogen and the oxygen tension falls. This phenomenon is another physiological example of induced mass inflow of gas during apnea. However, there is a difference in the mechanism of the airway pressure gradient as described in this study in comparison with that of "diffusion oxygenation" (or more correctly "apneic oxygenation") of Draper and Whitehead (Anesth. Analg. *28:* 307, Nov. 1949). *Conclusion:* In this earlier classical description, mass flow of gas results from the difference in carbon dioxide excretion and oxygen uptake. In our present study the lowering of the alveolar barometric pressure was due to the rapid uptake of the relatively soluble nitrous oxide.

The Acid-Base "Lesion" of Bank Blood. W. S. HOWLAND, M.D., and O. SCHWEIZER, M.D., *Department of Anesthesiology, Memorial Hospital For Cancer and Allied Diseases, New York City.* For many years the deleterious

effects of exchange and massive transfusion were attributed to the ionic imbalance of bank blood. Since recent investigations have shown an inconstant relation between the ionic composition of stored blood and the untoward manifestations of the blood replacement in the patient, efforts were made to find other possible etiological factors. One of these factors is the acid-base balance of bank blood. Repeated analysis has shown that ACD preserved blood has a *p*H of 6.58–6.72, an oxygen saturation of 20.2–56.2 per cent, a P_{CO_2} of 152–210 mm. of mercury, a standard bicarbonate of 1.2–7.8 mEq./liter and a buffer base of 27–37 mEq./liter. This represents a combination of respiratory and metabolic acidosis. Since the acid-base status of patients receiving large volumes of bank blood does not reflect this increased acidity except in the presence of shock, *in vitro* experiments were conducted to determine the effectiveness of the body buffering mechanisms (Schweizer, O., and Howland, W. S.: ANESTHESIOLOGY *24:* 158, 1963). Respiratory action was stimulated by oxygenating a sample of bank blood to increase oxygen saturation and remove carbon dioxide. The addition of sodium bicarbonate to bank blood reflected the buffering mechanism of the blood, tissues and kidneys on the fixed acid. Although *in vitro* oxygenation increased the oxygen saturation, decreased the P_{CO_2} and slightly elevated the *p*H, it had no effect on metabolic acidosis. The addition of sodium bicarbonate to the oxygenated blood in amounts equivalent to the acid excess of bank blood (6 mEq./bottle of blood) resulted in elevation of the *p*H and standard bicarbonate to normal. These *in vitro* investigations revealed that both intact respiratory and renal mechanisms are essential for adequate buffering of bank blood. The *in vivo* effectiveness of these buffering mechanisms are shown in the following illustrative cases. Shock was not a factor in either patient. The first patient, who received 40 units of blood, developed a CO_2 tension of 120 mm. of mercury as a result of pulmonary insufficiency. Although the respiratory acidosis reduced the *p*H to 7.1, the adequate urinary output maintained the standard bicarbonate within normal range. In contrast, the second patient, who was transfused with 66 units of blood, showed good respiratory function but developed anuria. Acid-base balance studies revealed a *p*H of 7.08, a standard bicarbonate of 11 mEq./liter (normal 21.5–24 mEq./liter) and relatively normal values for P_{CO_2}. These two patients show that *in vivo* as well as *in vitro,* a normal acid-base balance is dependent upon adequate functioning of both the respiratory and renal mechanisms. Failure of one of these compensatory leads to severe acidosis. In view of the depressant effect of acidosis on the myocardium, it is essential to maintain satisfactory pulmonary and renal function during hemorrhage and blood replacement.

Hepatic Function and Halothane. SAMUEL I. JOSEPH, Ph.D., M.D., *City of Hope Medical Center, Duarte, California.* Liver function tests have been done in a variety of surgical patients anesthetized with halothane as the primary agent. *Method:* Eighty-four patients have been tested, both male and female and ranging in age from three to eighty-three years. The operations included general oncologic surgery, both abdominal and superficial, and thoracic surgery, including cardiac. Anesthesia consisted of a combined intravenous and inhalation technique, with the circle absorption semiclosed intratracheal method and manual ventilation, employing either the Heidbrink Vernitrol or Foregger copper kettle for the administration of halothane. Gas flows were 8 liters per minute in most cases, and not less than 4 liters per minute in any. Agents used were thiopental, succinylcholine, and nitrous oxide oxygen, plus halothane or meperidine. Liver function tests, consisting of BSP, SGOT, SGPT, alkaline phosphatase, and bilirubin determinations, were done preoperatively, at varying intervals during operation, and postoperatively. *Results:* Thirty-four patients in the halothane group having *normal* preoperative BSP values (less than 4 per cent) showed a rise in BSP retention during anesthesia and operation. The average for 12 patients after two hours was 13.3 per cent; after four hours for 18 other patients it was 20.2 per cent. These were ascertained to be statistically significant differences ($P < 0.05$). All other liver function tests in these patients remained normal. In the meperidine