(SGH) levels were determined at 25-minute intervals by a radioimmunoassay technique. Since SGH secretion is induced by various stimuli, the authors were interested to study the similarity between secretion patterns during prolonged anesthesia and those found during normal sleep. Every subject had significant, transient increases in SGH levels during the first hour after induction of anesthesia, again during the fifth hour, and finally, in the last hour of anesthesia. This is not apparent in the awake state when metabolic changes and stress are avoided. On the other hand, a burst of SGH secretion is seen after the onset of deep sleep, and the authors conclude that inhibition of neocortical activity is responsible for the increased secretion of SGH found in anesthetized subjects. (*Werder, K., et al.: Growth Hormone Secretion during Long-term Anesthesia in Man, Horm. Metab. Res. 2: 309–310, 1970.*) EDITOR'S COMMENT: Although the significance of these findings is not immediately apparent, they indicate that the state of the art for quantifying endocrine function during anesthesia has come of age and an area devoid of data is ripe for study. The story must unfold eventually in the normal vounteer as well as the sick patient.

Respiration

IN-VIVO CO_2 EQUILIBRATION CURVE The authors have formulated a mathematical model to predict the effects of *in-vivo* steady-state changes of Pa_{CO_2} on plasma bicarbonate concentration and base excess as a function of blood volume (BV), packed cell volume (PCV) and interstitial fluid volume (IFV). Furthermore, the authors attempt to explain, through assistance from the model, the origin of the differences between the *in-vitro* and *in-vivo* CO_2 equilibration curves. In their opinion "CO_2 equilibration curve" is the best expression for defining the steady-state acid–base status of the blood after it has been exposed to an increase in P_{CO_2}. "CO_2 titration curve" is considered misleading, since it implies an analogy to the classical titration curve in which a known amount of acid is added and the resultant *p*H is measured. "CO_2 absorption curve" describes total CO_2 (*i.e.*, CO_2 content) taken by blood *in vitro* when P_{CO_2} is raised.

The *in-vivo* model was based on a three-compartment system: blood, interstitial fluid, and cells. The necessary values relating to buffering within these three systems, obtained from the literature, were introduced into an equation developed to express the *in-vivo* quantitative behavior of the model, *i.e.*, the rate of change of plasma HC_3^- with plasma *p*H. Analysis of the calculated data revealed the important effect of body composition (*i.e.*, BV, PCV, and IFV) upon the slope of the *in-vivo* CO_2 equilibration curve. Thus, the expected steady-state change in acid–base status in acute hypercapnia depends not only on ventilation (Pa_{CO_2}), but also on the volume of the several compartments of the body fluids into which HCO_3^- can distribute. This is further complicated by the nonbicarbonate buffer capacity of the blood. The importance of this analysis lies in its demonstration that the clinical usefulness of existing data relating HCO_3^- and Pa_{CO_2} for diagnostic purposes is sharply limited when there are concomitant disturbances in body composition. (*Dell, R. B., and Winters, R. W.: A Model for the In Vivo CO_2 Equilibration Curve, Amer. J. Physiol 219: 37, 1970.*)

OXYGEN TOXICITY IN MAN: A PROSPECTIVE STUDY In an excellent effort to elucidate the pulmonary effects of high inspired oxygen concentrations, these authors measured ventilatory variables in two comparable groups of patients who had undergone open-heart surgery. Alternate patients received either 100 per cent oxygen with IPPV or an oxygen concentration that kept arterial oxygen tension between 80 and 100 mm Hg. In the latter group of 20 patients, the highest oxygen concentration needed was 42 per cent. The mean times of exposure to these oxygen concentrations were 24 hours for the 100 per cent oxygen group and 21 hours for the "limited-oxygen" group. The investigators measured intrapulmonary right-to-left shunt, effective compliance, and the ratio of

deadspace to tidal volume. The results indicate that from the ventilatory functions measured there is no apparent adverse effect associated with ventilation with 100 per cent oxygen for 24–48 hours. The adverse pulmonary effect(s) of ventilating patients with high inspired oxygen concentrations for brief periods remain unclear. The potential therapeutic value of using high oxygen concentrations when the dangers of hypoxemia are present outweighs any disadvantage thus far postulated. (*Singer, M. M., et al.: Oxygen Toxicity in Man: Prospective Study in Patients after Open-heart Surgery, New Eng. J. Med. 283: 1473, 1970.*)

OXYGEN TOXICITY IN MAN: A PROSPECTIVE STUDY The combination of long-term artificial respiration and a high oxygen concentration in the inspired gas mixture can be responsible for various degrees of pulmonary damage, manifested as hyaline membrane formation and extensive fibrosis. Such damage, in turn, may diminish the efficiency of oxygenation through an increase in intrapulmonary right-to-left shunt. The acute effects of high inspired oxygen concentrations were studied in 20 patients who had undergone heart-valve replacement. All were ventilated mechanically for more than 24 hours postoperatively. The ventilator was set to deliver a tidal volume of 15 ml/kg at a rate that produced an arterial P_{CO_2} of 37 to 43 mm Hg. End-inspiratory pressures varied between 18 and 30 cm H_2O, and the average respiratory frequency was 8 breaths/min. In one group of nine patients the ventilator was set to deliver 83 per cent oxygen; another group of patients was given an oxygen/nitrogen mixture of 40/60 per cent. Patients in the two groups were comparable in age, sex, severity of heart disease, and surgical procedures. The authors calculated the right-to-left shunt by taking arterial and mixed venous (pulmonary artery catheter) samples following brief periods (20 minutes) of breathing of pure oxygen in the two groups. O_2 content was calculated, when necessary, from measured values of P_{O_2}, *p*H, and O_2 saturation. The authors conclude that even a brief period (24 hours) of ventilation with a high oxygen concentration is sufficient to impair the efficiency of oxygenation. This they attribute to the deleterious effects of oxygen on the pulmonary parenchyma. In the group ventilated with 83 per cent oxygen, the intrapulmonary right-to-left shunt rose from an initial average of 8 per cent to an average of 17 per cent of cardiac output during the first 24 hours; in the group ventilated with 40 per cent O_2, the shunt did not change. Since 40 per cent inspired oxygen usually produces nearly complete O_2 saturation of arterial blood, high concentrations of inspired oxygen are considered not only unnecessary but probably undesirable. It is the authors' impression that pulmonary complications, including bronchopneumonias, have been encountered less frequently since oxygen has been used in concentrations high enough to produce an arterial P_{O_2} of 100 mm Hg. (*Wolff, G., et al.: The Effect of Inspiratory Oxygen Concentrations on the Degree of Intrapulmonary Right to Left Shunt, Thoraxchirurgie 18: 356, 1970.*) EDITOR'S COMMENT: The studies by Singer *et al.* and Wolff *et al.* seem extraordinarily similar, yet their results are diametrically opposite. A full appreciation of this discrepancy can be obtained only by reading the original articles. Perhaps the levels of sedation, the degrees of muscle tonus, and the finer nuances between controlled ventilation in the paralyzed patient and assisted ventilation in the fully-awake individual may resolve the difference. At best, the cause of the difference is obscure, but it points to the danger of formulating conclusions about oxygen toxicity from clinical studies of patients with abnormal lung function.