(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