CNS Function

INTRACRANIAL PRESSURE FOLLOWING HEAD INJURY Ventricular fluid pressure (VFP) and systemic arterial pressure (SAP) were monitored continuously in 37 patients following severe head injuries. VFP was measured from a polyethylene catheter inserted into the right lateral ventricle via a frontal burr hole and attached to a transducer. SAP was measured directly from a cannulated peripheral artery. Three groups of patients were studied: I, patients with VFP below 20 mm Hg (normal pressure); II, patients with VFP between 20 and 40 mm Hg; III, patients with VFP above 40 mm Hg. Five of nine patients in Group I died, and although they had the clinical syndrome commonly ascribed to primary brain-stem injury, none was found at autopsy. One of 12 patients in Group III has made a satisfactory recovery; three are still in coma and eight have died. Although the relationship between SAP and VFP is considered critical for maintenance of adequate cerebral blood flow, there was no constant relationship between the two; in fact, the response of SAP to a rise in VFP was unpredictable, implying a lack of reliability of blood pressure measurements as indicators of rising VFP. Lowering of VFP was attempted with osmotic diuresis, hyperventilation, or withdrawal of ventricular CSF. The response to osmotic diuresis (mannitol) varied considerably and was less successful when the elevation was chronic or pronounced. Hyperventilation and removal of ventricular CSF proved very effective. The latter proved to be the more reliable of the two. The authors conclude that simultaneous clinical monitoring of VFP and SAP is mandatory for appropriate therapy of elevated intracranial pressure and evaluation of specific therapy. (*Johnston, I. H., et al.: Intracranial Pressure Changes Following Head Injury, Lancet 2: 433–436, 1970.*)

Coagulation

DISSEMINATED INTRAVASCULAR COAGULATION (DIC) IN SEPTICEMIA This study analyzed the coagulation defects in 26 children (age range 3 days to 9 years) with septic shock, a combination of symptoms usually associated with mortality ranging from 50 to 80 per cent. DIC, or consumption coagulopathy, is known to occur in septicemia, with a singular predilection for association with hypotension or shock. It can be diagnosed by finding a combination of thrombocytopenia and reduced levels of coagulation factors II, V, and VIII, as well as fibrinogen, and fibrinolytic split products (FSP). With these criteria DIC was diagnosed in 42 per cent of the hypotensive patients on admission or at the onset of the hypotension. The most common infecting organisms were gram-negative, being isolated in 12 of the 26 patients. Therapy consisted of antibiotics, intravenous fluids, plasma expanders, whole blood, and heparin, 100 units/kg body weight intravenously every four hours. Mortality in 24 patients treated with heparin was 58 per cent. The presence of frank hypofibrinogenemia (average values below 100 mg/100 ml) was associated with very high mortality (78 per cent). (*Corrigan, J. J., Jr., and Jordan, C. M.: Heparin Therapy in Septicemia with Disseminated Intravascular Coagulation, New Eng. J. Med. 283: 778–782, 1970.*) EDITOR'S COMMENT: Despite the life-threatening situation, no comment was made regarding respiratory status or the possible contribution of persistent hypoxemia to mortality. Undoubtedly this is prompted by the common impression that an otherwise-normal child can tolerate a hypoxemic insult, even when superimposed on overwhelming sepsis. DIC plays havoc with the microcirculation, and the "shock" organ most often involved is the lung. This may explain the acute deterioration in gas exchange frequently found in these patients and the importance of early diagnosis and early intervention with ventilator support.

Endocrine Function

GROWTH HORMONE SECRETION AND ANESTHESIA Five healthy volunteers were subjected to seven hours of continuous endotracheal nitrous oxide–halothane anesthesia, during which serum growth hormone

(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