CENTRAL NERVOUS SYSTEM CEREBROSPINAL FLUID

Sir,—With reference to the paper by I. R. Cameron "Acid-Base Changes in Cerebrospinal Fluid" (Brit. J. Anaesth., 1969, 41, 213) it may be noted that at least two phenomena, amongst others, may be explained on the basis of a direct action of a change in concentration of hydrogen ion in the cerebrospinal fluid on chemoreceptors in the central nervous system.

In their studies of the initiation of respiration after anaesthesia accompanied by passive pulmonary hyperventilation, Utting and Gray (1962) found that under the conditions reported, spontaneous respiration started at low PaCO₂ levels in every case before consciousness was regained. In other studies relating to suitable anaesthesia for the respiratory cripple, Utting, Gray and Jackson Rees (1965) found that the first signs of respiratory effort took place when the PaCO₂ was still at a lower level than it had been on the ward before operation.

It is possible to explain the initiation of respiratory efforts at low levels of PaCO₂ on the basis that the prolonged respiratory alkalosis of hyperventilation resets the (HCO₃⁻) c.s.f. at a lower level, so that the rise of PaCO₂ and parallel rapid rise of Pco₂ c.s.f. causes a relatively much more rapid rise in (H+)+ c.s.f. with consequently earlier respiratory efforts than might otherwise be expected.

It is interesting that these investigators noted that one of their patients, not included in the series, who was a respiratory cripple showed the phenomenon of continuing to hyperventilate after anaesthesia involving passive hyperventilation. They believe that the single most important factor leading to a respiratory cripple failing to resume adequate spontaneous respiration at the end of anaesthesia is the development of hypercarbia either during the anaesthetic or after reversal of relaxant activity.

By a like chain of reasoning as above, it can be seen that the effect of hypercarbia would be to reset the (HCO₃⁻) c.s.f. of the respiratory cripple at a higher level. Then the normal increase of PaCO₂ at the end of a period of passive hyperventilation would have a smaller effect on the rise of (H⁺)+ c.s.f. with consequent diminution in the effect of this factor on the initiation of respiratory efforts.

As Semple (1965) points out, the essence of understanding these changes is to appreciate that the free diffusion of carbon dioxide is not balanced in time by the probable active transport of bicarbonate ion. This imposed time lag tends to create and perpetuate for a time, changes in the hydrogen ion concentration of the c.s.f.

T. R. Austin

REFERENCES


Dr. Austin’s letter was shown to Dr. Cameron who replied as follows:

Sir,—This is an interesting problem. It is, unfortunately, complicated by two factors. First, correlating the time course of ventilation with changes in c.s.f. (HCO₃⁻) assumes that the central respiratory receptor is responding solely to large cavity c.s.f. pH changes. At present it does not seem possible to ascribe a definite single anatomical locus to such a receptor. Second, the post-anaesthesia situation is not a steady state. Changes in c.s.f. (HCO₃⁻) may lag some time behind changes in brain c.s.f. (HCO₃⁻). The presence of a c.s.f. (HCO₃⁻) “inappropiate” to a given Pco₂ does not imply, in acute states, a similarly “inappropriate” (HCO₃⁻) in brain c.s.f. Data on these changes would be of value from several points of view; it would be of considerable interest to know the speed at which changes in large cavity (HCO₃⁻) occur, knowing the time course of these changes it should be possible to decide whether or not ventilation is following large cavity pH. Such data would be of value only if cisternal fluid were sampled, since lumbar fluid changes would lag even further behind.

I. R. Cameron
London

PLACENTAL TRANSFER OF ALCURONIUM

Sir,—I was encouraged by the support given by Dr. Thomas and his colleagues (Brit. J. Anaesth., 1969, 41, 297) to earlier efforts (Crawford and Rudofsky, 1965a, b; Crawford, 1965b) to extend Paton’s concept of the “slug of drug effect” (Paton, 1960) to placental transfer. I wonder if your contributors actually went further without drawing attention to the fact. It has been suggested (Finster et al., 1966) that if an intravenous injection coincides with the beginning of a uterine contraction, comparatively little of the injection material will be presented to the placental site for transfer. Included in the series reported by Thomas, Clinnie and Mather was an unspecified number of emergency Caesarean sections. Were these patients in labour? And did they include patients number 11 and 12, who were given a rapid i.v. injection of alcuronium and in respect to whom no evidence of the drug was to be found in cord blood?

J. Selwyn Crawford
Birmingham

REFERENCES


