Editorial Views

Cerebral Blood Flow and Uptake of Anesthetics

Recently the National Research Council and the New York Academy of Medicine published the proceedings of a conference on the uptake and distribution of anesthetic agents. It is necessary only to peruse the table of contents to realize the breadth of disciplines encompassed—physical chemistry, pulmonary and circulatory physiology, and biochemistry. Among the chapters devoted to circulation there is a review on distribution of blood flow in the brain by Kety and another on the effects of anesthetics on cerebral blood flow by Sokoloff. Relevant studies published since that conference are those of Pierce et al. on cerebral hemodynamics and oxygenation during thiopental anesthesia at normocapnia and hypocapnia, and in this issue of Anesthesiology studies by Alexander et al. on brain uptake of nitrous oxide during thiopental anesthesia and of krypton during halothane anesthesia. Historically the measurement of cerebral hemodynamics and studies of brain uptake and distribution of anesthetics have been closely related.

In 1945 Kety and Schmidt published their classic paper describing the nitrous oxide method for determination of the rate of cerebral blood flow in conscious man. Later, during substantiation of the method, Kety examined the factors influencing uptake of nitrous oxide. He found the brain-blood partition coefficient to be 1.06 and the minimal time for equilibrium between the brain and cerebral venous blood to be somewhat less than ten minutes. The delay in equilibration could be explained either by diffusion limitation or by variation in blood flow to different regions of the brain. That the latter explanation is the correct one was subsequently demonstrated, most recently in Alexander’s paper, in which the authors further analyzed data from two recent studies of cerebral hemodynamics during general anesthesia—those of Pierce and of Wollman using halothane anesthesia. The assumption is that the chief variation in blood flow lies between gray matter, with a high perfusion rate, and a lower perfusion area comprised of white substance.

Although Kety and Schmidt demonstrated in conscious man that P$_{CO_2}$ exerts exquisite control of cerebral vascular resistance, therefore of cerebral blood flow, there remained considerable doubt whether during general anesthesia the reactivity of cerebral blood vessels to carbon dioxide was preserved. Since in the two most recent studies on cerebral blood flow no loss of reactivity was shown, Alexander states: “an anesthetist may attempt to hasten brain gas uptake or elimination by increasing pulmonary ventilation since arterial equilibration is enhanced by a rise in alveolar ventilation. Under usual clinical conditions, however, an increase in ventilation decreases arterial P$_{CO_2}$ and hence cerebral blood flow is lowered. This diminished brain blood flow could completely negate or even reverse the desirable effects produced by increased pulmonary ventilation.”
Pulmonary hyperventilation is of interest to the anesthetist for other reasons than the effect just on uptake of anesthetic gas. It may improve operating conditions, especially during neurosurgery, may reduce the amount of anesthetic drugs required for adequate surgical relaxation, and can markedly reduce jugular venous oxygenation. Rosomoff, in a recent issue of Anesthesiology, challenges the assumption that hyperventilation because of cerebral vasoconstriction results in reduction of intracranial pressure and brain volume. Utilizing a recently developed technique for the simultaneous quantitative estimation of intracranial contents, he found that during hyperventilation a decrease in intracranial blood volume was accompanied by a compensatory increase in cerebrospinal fluid volume. He concluded that controlled hyperventilation “does not reduce the volume of brain tissue, and intracranial pressure is not affected if the CO₂ tension is normal prior to the onset of ventilation.” Downes, in the same issue, reports a study of the relationship between hyperventilation and abdominal muscle relaxation. In the rat, at least, inhibition of abdominal muscle reflexes during hyperventilation was not primarily due to hypocapnia, for it occurred to the same degree when the inspired PₐCO₂ was raised during hyperventilation. Lastly, Pierce demonstrated that reduction of arterial PₐCO₂ to less than 20 mm. of mercury by passive hyperventilation during thiopental anesthesia in man was associated with a marked decrease in jugular venous oxygenation despite improvement in arterial oxygenation. It is not known whether relative hypoxia is a factor contributing to the clinical manifestations of hyperventilation; there is little evidence of residual deleterious effect.

All of this is not to say, of course, that benefit will not occur from hyperventilation during neurosurgery or, for that matter, during other operations. It certainly is preferred to hyperventilation and will probably remain an important anesthetic technique.

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References