Things Are Seldom What They Seem[1]

THE MAINTENANCE of adequate cerebral perfusion is of prime concern to the anesthesiologist. In normal man, both awake and anesthetized, cerebral blood flow is exquisitely sensitive to arterial carbon dioxide tension.[2-5] The rapidity and ease with which the anesthesiologist can vary Pa_{CO_2} over a wide range make it essential that he have an understanding of the behavior of the cerebral circulation during anesthesia.

A common technique in anesthetic practice is deliberate hyperventilation. It has long been questioned whether the resulting diminution of cerebral blood flow, as well as the shift to the left of the hemoglobin dissociation curve (Bohr effect), significantly impair cerebral oxygenation. The pattern of cerebral metabolism during extreme hyperventilation [6] ($Pa_{CO_2} < 20$ mm. Hg) is markedly similar to that observed when cerebral hypoxia is known to exist.[7] Alterations in the electroencephalogram are produced when conscious man hyperventilates ($Pa_{CO_2} < 20$ mm. Hg) with air; these changes disappear when hyperventilation is continued but oxygen at three atmospheres pressure is inhaled.[8] These findings indicate that deliberate *marked* hyperventilation can result in inadequate cerebral circulation; it follows that this maneuver should be avoided.

Some surgical operations necessitate the deliberate occlusion of major vessels supplying the brain. Is there anything that the anesthesiologist can do to minimize cerebral damage? A regimen consisting of "deliberate hypercarbia and induced hypertension" [9] has been advocated; this is a technique that appears rational in view of the physiology of *normal* cerebral circulation. In this issue, of the journal, however, Soloway and his associates have presented data indicating that precisely the opposite may be true. The internal carotid and middle cerebral arteries of dogs were permanently occluded; at the time of circulatory compromise some of the animals had been hypocarbic ($Pa_{CO_2} = 25$ mm. Hg) for 30 minutes, while the remainder served as normocarbic controls. These levels of Pa_{CO_2} were maintained for two hours after ligation; the animals were then allowed to awaken. The finding that both neurologic deficits and anatomic lesions were markedly less severe in the *hypocarbic* group is most exciting. The authors' experimental design and their analysis of the data is without fault. The results appear firm: in this species, with cerebral circulatory embarrassment produced as described, anesthetized animals maintained with moderate hypocarbia for 30 minutes before and two hours after occlusion will have far less residual damage than those in which Pa_{CO_2} is normal.

Do these findings contradict the previous statement that extreme hyperventilation should be avoided? To answer this, it must be emphasized that the original discussion was confined to *normal* man. *This* paper deals with circulatory occlusion, decidedly not a normal condition, and illustrates the difficulty and perhaps the hazard of extrapolating from findings made under normal conditions to the pathologic situation.

In patients who have recently had cerebrovascular accidents, studies of regional cerebral circulation have demonstrated hyperemic foci.[10] This "over-abundant cerebral blood flow relative to the metabolic needs of the brain" was termed the "luxury perfusion syndrome." [11] Luxury perfusion represents a loss of normal vasomotor control. When Pa_{CO_2} is altered, the involved area shows little change in blood flow. On the other hand, its circulation now varies passively with blood pressure, a phenomenon not found in the surrounding normal areas.

Tissue without blood supply cannot undergo reanimation. However, in areas with collateral circulation, the abnormal physiologic response to vascular occlusion must be considered. The factors normally controlling cerebral blood flow are often inoperative at this time. In such a situation, hypocarbia-induced vasoconstriction in normal tissue could divert blood to the region of circulatory damage (in which vasoconstriction does not occur). Conversely, it

has been suggested that carbon dioxide inhalation might actually be detrimental by producing an "intracerebral steal syndrome."[10] This change in regional flow pattern may be the way in which hypocarbia exerts a protective effect.

It has been postulated that loss of normal vasomotor control is due to localized cerebral acidosis.[11] For this reason, hypocarbia may also minimize cerebral damage by aiding in the return of tissue *p*H to normal.

Although the findings of Soloway *et al.* appear quite clear, the mechanism by which hypocarbia protects the central nervous system has not been elucidated. Measurement of regional cerebral blood flow, metabolism, and tissue *p*H would be informative. The effects of *hypercarbia* in the same experimental setting would be interesting. Finally, the degree of protection afforded by hypothermia should be compared with the results obtained with normothermic hyperventilation.

Just as normal cerebral physiology cannot always be extrapolated to a pathological setting, the anesthesiologist confronted with his next carotid endarterectomy cannot directly make use of these findings. This operative procedure involves the *temporary* occlusion of a vessel in a patient whose cerebral circulation has already been compromised. If the vessel is seriously involved, little change in cerebral perfusion may result no matter what technique is used and tissue necrosis need not result. Satisfactory results have been reported by workers using a variety of approaches to the problem. It is clear that an equally careful study of the postoperative cerebral function of these patients is necessary if one technique is to be shown superior.

The paper has forcefully pointed out the need for careful study of abnormal cerebral circulatory physiology. The unexpected results shed additional light on this subject, and for this we are all grateful.

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