Effects of Fentanyl Versus Sufentanil in Equianesthetic Doses on Middle Cerebral Artery Blood Flow Velocity

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SINCE 1988, there has been an ongoing discussion regarding the cerebrovascular effects of the potent synthetic opioids. A large body of literature (dating back to the 1970s) indicates that opioids produce dose-related EEG slowing along with decreases in cerebral metabolic rate and cerebral blood flow (CBF). More recent studies, including the work by Trindle et al. (page 454) suggest that these drugs can at least transiently increase CBF and/or intracranial pressure (ICP). This seems to be particularly true when the drugs are given alone, without supplementary sedatives or other anesthetics.

An unwritten caveat in medical research says that, when there are a large number of well controlled but contradictory studies, the issue in question is unlikely to be of much importance in the "messy" world of clinical medicine. Since the overwhelming bulk of clinical experience (and several clinical trials) clearly show the synthetic opioids to be safe and effective in neurosurgery, why is the finding by Trindle et al. anything more than an experimental curiosity? There are several reasons. Most importantly, it (along with other work) indicates that opioids are doing something to the brain that is unique among anesthetic agents and that is not easily explicable within the context of our current concepts of anesthetic action. If the mechanisms could be elucidated, they might provide insights into the central actions of all opioids (and perhaps other anesthetics) and into normal cerebrovascular control. For example, since the effects of opioids on CBF and ICP are not obviously dose-related, it is possible that these drugs are acting to "switch on" (or off) some vasomotor control center in the brain.

A second reason why this report by Trindle et al. is worthy of attention is that it represents only the second paper appearing in Anesthesiology that has employed the transcranial Doppler (TCD) to examine cerebrovascular pharmacology (see also Eng et al., Anesthesiology 77:872–879, 1992). Prior to this, most studies have measured CBF only under relatively steady-state conditions. While TCD is not new, it has had a difficult time achieving acceptance as a valid measure of CBF in comparison with these older, time-tested methods. It is now clear that TCD cannot provide absolute measures of CBF. However, it is also apparent that, if used in an appropriate fashion (as it was by Trindle et al.), it can provide a reasonably accurate and noninvasive assessment of relative changes in CBF, even changes that occur over a matter of seconds. For example, Aaslid et al. (Stroke 22:1148–1154, 1991) have used TCD to examine the time course of cerebral autoregulation in response to sudden changes in blood pressure—something that could never be done using traditional methods. It thus becomes possible to assess the pharmacodynamics of anesthetic effects on cerebral circulation. By recording the EEG along with TCD flow velocities (perhaps along with serum drug concentrations), it becomes possible to examine the link between drug-induced changes in electrical activity and CBF.

Whether this promise will be realized and whether such studies will yield uniquely important information remain to be demonstrated. However, it is indeed an exciting possibility.

Unintentional Hypothermia Is Associated with Postoperative Myocardial Ischemia

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AS evidenced by commercial production of devices to prevent intraoperative heat loss from patients, perioperative hypothermia is garnering increasing attention. The study by Frank et al. (page 468) provides further evidence for concern by demonstrating an apparent association between hyperthermia in the im
mediate postoperative period and myocardial ischemia. It is perhaps surprising that something so simple and potentially variable as intensive care unit admission temperature (measured orally!) would have predictive value for risk of myocardial ischemia as determined from continuous Holter monitoring, especially with a study group of only 100 patients. Not surprisingly, the occurrence of the hypothermia appears to be more frequent in older patients. However, it must be emphasized that this is only an association.

Measurement of cardiac output combined with other hemodynamic variables would have provided valuable documentation of increased myocardial work and oxygen consumption that might generate such ischemia episodes. Likewise, measurement of mixed venous oxygen saturation would have provided even more useful laboratory evidence that shivering and other thermogenic metabolic activity occurred in response to hypothermia. If ongoing studies provide further evidence of risk and also document the physiologic mechanisms involved, both the anesthesiologist and the entire surgical team will need to become actively involved in assuring better thermal care for patients. Just as we attempt to maintain homeostasis and avoid complications in other areas, assurance of quality care may mean greater attention to maintaining normothermia.

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