± 1.8 to 17.4 ± 2.9 mmHg and from 7.1 ± 1.8 to 13.2 ± 2.8 mmHg in the fentanyl and sufentanil groups, respectively.

The ICP zenith and MAP nadir occurred 6 and 10 min, respectively, after fentanyl and 4 and 8–15 min, respectively, after sufentanil administration.

They offered five explanations: (1) direct cerebrovascular smooth muscle dilatation, (2) an increase in the cerebral metabolic rate for oxygen, (3) systemic hypotension producing cerebral ischemia, (4) histamine release, and (5) effects of opioids on cerebral spinal fluid production and/or absorption.

They did not discuss cerebrovascular autoregulation, which may be a reasonable explanation for their findings.

Changes in cerebral perfusion pressure (CPP), within the autoregulatory range, have little effect on cerebral blood flow (CBF) because cerebral vasodilation occurs; however, this compensatory mechanism also increases cerebral blood volume (CBV) and the CBV/CBF ratio. In comatosc patients with head trauma, pharmacologic tests of cerebrovascular autoregulation of CBF, using phenylephrine or trimethaphan, resulted in a steep increase in ICP (from 20 ± 3 to 30 ± 2 mmHg) when MABP was decreased (from 120 ± 13 to 91 ± 10 mmHg) in patients with intact autoregulation, whereas in those with impaired autoregulation, decreasing MABP (from 112 ± 19 to 90 ± 17 mmHg) decreased ICP (from 16 ± 5 to 11 ± 7 mmHg). Increasing MABP in these patients did not change ICP, presumably because autoregulatory vasodilation results in a maximum 10% decrease in baseline diameter, whereas autoregulatory vasodilation may result in up to 65% increase in baseline diameter. In another study, the cerebrovascular responses to CPP changes were blunted in animals anesthetized with pentobarbital, presumably because of the cerebrovascularconstricting effect of this drug.

In summary, cerebrovascular autoregulation, intracranial clausance, and cerebrovascular effects of anesthetic drugs may result in complex interactions in patients with head trauma. The increases in ICP observed by Sperry et al. may be due to opioid-induced increased CBV in patients with compromised intracranial clausance and intact autoregulation and deserve further investigation.

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References

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In Reply—Posing an alternative mechanism for our finding, DeLima describes a study of cerebrovascular autoregulation in patients with head trauma. In patients with intact autoregulation, a decrease in blood pressure correlated with an increase in intracranial pressure (ICP). In patients with impaired autoregulation, a decrease in blood pressure did not correlate with an increase in ICP. Our study was small, and we did not believe we could legitimately separate different patient groups. However, in our results we found that there probably were two patient populations. Five of the nine patients accounted for 86% of the variation of ICP from baseline when administered fentanyl and 74% of the variation when administered sufentanil.

It is possible, although by no means proved in our study, that these two patient populations represent those described in the cited study. Further work is required to delineate this possibility.

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References

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