Why is total intravenous anesthesia preferred (over volatile) in the setting of elevated intracranial pressure? Does it improve post-operative neurological outcome?

The determinants of intracranial pressure (ICP) are described by the Monro-Kellie doctrine; that is, that the volume of an intact cranium is fixed, and the brain tissue, blood, and CSF within the cranium are in a state of volume equilibrium. An increase in any one of these components requires an equivalent decrease of another to maintain normal ICP. Cerebral blood flow (CBF) is tightly coupled to cerebral metabolic rate (CMR) and, in a normal state, autoregulates over a wide range of MAP values to maintain cerebral perfusion pressure (CPP). Additionally, CBF is altered by multiple factors including PaO_2, $PaCO_2$, blood viscosity, temperature, and drugs administered.

Intracranial hypertension may occur due to expanding mass, interference with normal CSF absorption, depressed skull fracture, excessive cerebral blood volume, or brain edema (e.g. TBI, tumour). These circumstances require a careful balance of maintaining CBF (by maintaining CPP) to avoid ischemia, and avoiding excessive CBF resulting in increases cerebral blood volume (CBV) and further increase in ICP. Additionally, increased cerebral blood volume contributes to more difficult surgical conditions in neurosurgery – that is, a less "relaxed" brain.

Total intravenous anesthesia (TIVA) is preferred over volatile in the setting of elevated ICP. Volatile anesthetics have a direct dose-dependent vasodilatory effect – they increase CBF. This is most significant for halothane; sevoflurane causes the least vasodilation. There is also a dose-dependent decrease in CMR, which leads to vasoconstriction. At doses < 0.6 MAC, this balances the vasodilatory effect and volatile anesthetics may have a net neutral effect on (or even reduction of) CBF. At doses greater than around 0.6-1 MAC, however, the vasodilatory effect is increasingly predominant, leading to increased CBF and further increased ICP. Additionally, there is uncoupling (or alteration) of CPP-CBF autoregulation, and CBF passively follows blood pressure, leading to a higher risk of ischemia, edema (from excess perfusion), or hemorrhage.

By contrast, IV anesthetic agents typically reduce both CMR and CBF. Total intravenous anesthesia is typically comprised of a combination of opioid (e.g. remifentanil) and a hypnotic (e.g. propofol). Opioids have either no effect on or cause a mild reduction of CBF, CMR, or ICP. Remifentanil in particular is neutral, and has little to no effect on blood flow dynamics. Combined with its rapid onset/offset pharmacokinetics, it makes an ideal choice for TIVA in neuroanesthesia. Hypnotic drugs (including propofol, etomidate, and barbiturates) cause a dose-related decrease in CMR and CBF.

A balanced anesthetic of a moderate dose of volatile and a rapid-acting opioid provides suitable operating conditions and does not adversely affect CBF, ICP, and CPP in a patient with normal ICP. In a patient with elevated ICP, managing ICP, CBF, and CBV is of utmost importance as post-operative neurological outcomes are improved with better control of ICP. For reasons reviewed above, TIVA is potentially the safest anesthetic to do so. However there is no evidence to show improved post-operative neurological outcomes specifically attributable to TIVA (vs volatile alone or volatile with opioid). The best choice of anesthetic is likely that which best facilitates prevention of hypotension and hypertension; allows for rapid and smooth emergence from anesthesia, and provides optimum operative conditions for the surgeon.

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