Should ketamine be used as an induction agent in traumatic brain injury?

The management of the haemodynamically unstable patient with traumatic brain injury is challenging. The airway must be secured with an endotracheal tube to protect the patient from aspiration, to allow control of oxygenation and ventilation and to facilitate further management.

The choice of intravenous induction agent is dependent upon the physiological parameters present and the experience of the clinician involved. Commonly used drugs include thiopentone, propofol, midazolam and etomidate.

Traditional teaching advocates that ketamine should be avoided as an induction agent in the presence of traumatic brain injury as it is associated with a rise in intracranial pressure. The evidence for this is not strong, however, and the potential haemodynamic benefits of this drug are well known. This article looks at the arguments for and against the use of ketamine in the patient with traumatic brain injury.

Traumatic brain injury

Traumatic brain injury is the commonest cause of death in young adults. The primary brain injury occurs at the time of trauma and is irreversible. The aim of management is to limit secondary brain injury, with a particular focus on avoidance of hypoxia and hypotension. Manley et al (2001) reported that hypotension (systolic blood pressure <90 mmHg) occurring early in the resuscitation process was associated with a significant increase in mortality, even when these periods were relatively short. Targets for cerebral perfusion pressure, mean arterial pressure and intracranial pressure must be met. Cerebral perfusion pressure must be maintained above 60 mmHg to ensure adequate delivery of oxygen to brain tissues. As cerebral perfusion pressure = mean arterial pressure – intracranial pressure, it can be seen that an increase in the intracranial pressure or a decrease in the mean arterial pressure would lead to a decrease in the cerebral perfusion pressure.

Dr P McDermott *is ST5 in Anaesthesia/Intensive Care Medicine in the Department of Anaesthetics and* **Dr J Sebastian** *is Consultant Anaesthetist in the Department of Anaesthetics, Salford Royal Foundation Trust, Salford M6 8HD*

Correspondence to: Dr P McDermott (peter.mcdermott@doctors.org.uk)

Ketamine

Ketamine is a non-competetive antagonist of the N-methyl-D-aspartate (NMDA) receptor calcium channel pore. Ketamine demonstrates several beneficial pharmacodynamic effects in the setting of hypotension. Ketamine increases sympathetic tone leading to an increase in heart rate, blood pressure and cardiac output. This is a significant advantage over other induction agents which tend to have deleterious effects on blood pressure.

The main reported negative effect of ketamine in the setting of traumatic brain injury is the associated increase in intracranial pressure, which could theoretically lead to a worsening of the secondary brain injury by reducing cerebral perfusion pressure.

The evidence

A number of small studies in the 1970s produced evidence suggesting that the use of ketamine was associated with an increase in intracranial pressure. Gardner et al (1971) reported a marked increase in CSF pressure after administration of ketamine.

More recently, Bourgoin et al (2003) reported no difference in intracranial pressure or cerebral perfusion pressure when ketamine was used as part of a sedation regimen for severe head injury patients. Albanese et al (1997) studied the effects of bolus dosing ketamine to patients who had suffered traumatic brain injury and who had a propofol infusion already running. They found that ketamine led to a significant decrease in intracranial pressure with no significant change in cerebral perfusion pressure. Himmelseher and Durieux (2005) summarized the available evidence by stating that in a sedated, ventilated patient, ketamine does not increase intracranial pressure (level III evidence) and that ketamine, when compared with opiates, reduces the need for vasopressors to maintain cerebral perfusion pressure when it is used for sedation (level II evidence).

Conclusions

The management of traumatic brain injury in the haemodynamically unstable patient presents significant challenges. Adequate brain oxygenation and perfusion must be maintained. The haemodynamic effects of ketamine make this an ideal drug to facilitate intubation in this setting. The most recent evidence suggests that ketamine does not lead to an increase in intracranial pressure and its beneficial effects on mean arterial pressure can help maintain cerebral perfusion pressure. In the end, the choice of induction agent must be made based on the available evidence, clinician experience and the clinical situation. BJHM

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Anaesthetic and critical care dilemmas are coordinated by Dr Steven Cone and Dr Matthew Henley, Specialist Registrars in Anaesthetics, Royal Free Hospital, London