Trench entrapment: is ketamine safe to use for sedation in head injury?

Matthew Gunning, Zane Perkins, Tom Quinn

Emerg Med J 2007;24:794–795. doi: 10.1136/emj.2007.053066

A 19-year-old man was buried when the walls of the trench he was digging collapsed. Bystanders exposed the head and chest, and called the emergency services. The paramedic applied a cervical collar. Primary survey revealed a patent airway, spontaneous respirations and saturations of 99% on oxygen. Good pulses were palpated bilaterally at 90 beats/min and intravenous (iv) access was secured. The Glasgow Coma Scale (GCS) was 3/15.

The patient began groaning in response to pain, and the GCS was revised to 4/15. Pupils were 3 mm in diameter, equal and reactive to light. The prehospital doctor administered ketamine 100 mg iv to aid extrication and the patient was secured onto a spinal board.

Once in the ambulance, the patient displayed decorticate posturing in response to pain (GCS 6/15). Secondary survey excluded obvious head injury. Surgical emphysema and bruising were noted on the right side of the chest, without flail segment. The trachea was central with good breath sounds heard bilaterally, with no hyper-resonance to percussion or neck vein distension. Tracheal intubation followed rapid sequence induction (RSI) (etomidate 15 mg and suxamethonium 100 mg iv, midazolam 10 mg iv). Warmed normal saline was infused to maintain systolic arterial pressure above 100 mm Hg.

In hospital, a computed tomographic scan excluded intracranial pathology but showed a stable fracture of the transverse process of T1. Rib fractures and anterior pneumothorax were noted on the right side of the chest, which required an intercostal drain. Final diagnosis was mild hypoxic brain injury, and the patient was discharged without obvious neurological deficit.

DISCUSSION

Cerebral blood flow (CBF) is critically dependant on cerebral perfusion pressure (CPP) and oxygenation in acute head injuries. Optimal CPP is achieved by maintaining a normal mean arterial pressure (MAP) and limiting iatrogenic increases in intracranial pressure (ICP).¹ Brain tissue has high oxygen consumption and no reserves; hypoxia therefore has rapid and profound effects. Early tracheal intubation and ventilation can help prevent hypoxia and aspiration. Hypoxia and hypotension in traumatic brain injury are associated with a 75% mortality rate.² End tidal carbon dioxide should be maintained around 5 kPa, as hypercapnia causes cerebral vasodilation and increased ICP.³

Ketamine, a potent analgesic, can be used for dissociative anaesthesia in higher doses (2 mg/kg), or sedation in lower doses. It has a rapid onset and relatively short duration of action (5–10 min). Unlike other commonly used induction agents, ketamine does not suppress respiratory activity or airway reflexes; it also has a positive effect on gut motility, and vomiting after administration is uncommon. These properties make it the ideal agent when profound analgesia and sedation are required without a definitive airway in place.³

Ketamine causes increased catecholamine release and decreased norepinephrine (noradrenaline) re-uptake which results in increased heart rate, arterial pressure, and MAP. This makes it a useful analgesic for trauma patients who may already be haemodynamically compromised. A single episode of hypotension is associated with a worse outcome.²

Ketamine is an N-methyl-D-aspartate (NMDA) receptor antagonist. Head injury increases concentrations of glutamate, which induces neuronal apoptosis. Ketamine blocks the actions of glutamate on the NMDA receptor, which may protect against cellular neurotoxicity, but this has yet to be demonstrated in human studies.^{1 3 4}

Despite these benefits, the use of ketamine in patients with head injuries remains controversial. Early studies suggested that the use of ketamine may have resulted in a transient increase in ICP in a small number of patients.³ CPP was compromised only in the patients with pre-existing intracranial hypertension and obstruction to the flow of cerebral spinal fluid. This has, however, led to the persistent belief that ketamine is contraindicated in patients with traumatic head injuries. Studies done subsequently have shown, however, that the effects of ketamine on cerebral haemodynamics and ICP are in fact variable and depend on both the presence of additional anaesthetic agents and Paco₂ values.⁵

This patient was sedated with midazolam 10 mg iv, which prevents the emergence phenomenon, and ventilation was controlled artificially en route to hospital. When ketamine is used in the presence of controlled ventilation, in conjunction with anaesthetics which reduce cerebral metabolism such as γ -aminobutyric acid (GABA) receptor agonists, ICP is not increased.^{1 4}

Management of the severely head injured patient should focus on minimising secondary brain injury. Ketamine is a cardiorespiratory stable analgesic, which when used in combination with midazolam provides good sedation and has not been shown to increase ICP adversely. This, combined with the potential neuroprotective effects of NMDA antagonism, could improve neurological outcomes in brain injury. A randomised control trial to evaluate the use of ketamine in head injuries is warranted.

Authors' affiliations

Matthew Gunning, Zane Perkins, Tom Quinn, Staffordshire Ambulance Service NHS Trust, Stafford, UK

Competing interests: None declared

Informed consent was obtained for publication of the person's details in this report.

Abbreviations: CBF, cerebral blood flow; CPP, cerebral perfusion pressure; GABA, γ -aminobutyric acid; GCS, Glasgow Coma Scale; ICP, intracranial pressure; iv, intravenous; MAP, mean arterial pressure; NMDA, N-methyl-D-aspartate; RSI, rapid sequence induction

Correspondence to: Dr Matthew Gunning, CareFlight Medical Services, 44 Eastern Ave, Bilinga 4225, Queensland, Australia; MattGunning@rcsed.ac.uk

Accepted 4 August 2007

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IMAGES IN EMERGENCY MEDICINE

Fracture of an unnamed bone

Baldeep Bains, Keith Porter

A 28-year-old professional footballer presented to the emergency department having sustained an isolated direct kick to the left shin with an associated soft tissue haematoma. Radiographs were normal and his symptoms resolved over 6 weeks. Five years

later following a similar mechanism of injury, radiographs revealed a fracture of a tibio fibula synostosis occurring at the site of the previous injury (figs 1 and 2). Heterotopic ossification following soft tissue injuries is not uncommon and can present surprising radiological features.

Emerg Med J 2007;**24**:795.
doi: 10.1136/emj.2006.043356

Authors' affiliations

Baldeep Bains, Keith Porter, Selly Oak Hospital, Birmingham, UK

Correspondence to: Mr Baldeep Bains, Selly Oak Hospital, Birmingham, UK; baldeepb@hotmail.com

Competing interests: none declared

Figure 1 Anteroposterior view of heterotopic ossification of tibio fibula synostosis.

Figure 2 Fracture through the ossified tibio fibula synostosis.