SOCRATES 5 (Synopsis of Cochrane Reviews applicable to Emergency Services)

P Gilligan, G Lumsden, J Jones, J Brenchley, D Hegarty, A Khan, M Shepherd, G Kitching, A Taylor, H Law

In this the fifth article of the SOCRATES series we present reviews from the Cochrane Database of Systematic Reviews relating to head and spinal trauma that the working party felt were of particular relevance to Emergency Medicine practitioners. The methods of our review and the rationale for the forming the SOCRATES working party are as have previously been published.

HEAD TRAUMA

MANNITOL FOR ACUTE TRAUMATIC BRAIN INJURY

Mannitol is sometimes dramatically effective in reversing acute brain swelling, but its effectiveness in the on-going management of severe head injury remains open to controversy. The objectives of this paper were to assess different mannitol regimes and compare with other intra-cranial pressure lowering agents.

Results

Three randomised controlled trials in patients with acute brain injury. The studies involved a total of one hundred and seventy seven participants. One trial compared mannitol therapy to ‘standard care’ (relative risk (RR) for death = 0.83; 95% CI 0.47;1.46). One trial compared mannitol to phenobarbital (RR for death = 0.85; 95% CI 0.52;1.38). One trial tested the effectiveness of pre-hospital administration of mannitol against placebo (RR for death = 1.59; 95% CI 0.44;5.79).

SOCRATES says

Mannitol therapy directed by intra-cranial pressure in acute brain injury shows a small beneficial effect compared to treatment directed by neurological signs and physiological indicators. There is insufficient data on the pre-hospital effectiveness of mannitol.


CALCIC CHANNEL BLOCKERS FOR ACUTE TRAUMATIC BRAIN INJURY

Trumatic head injury is a major cause of death and disability. Primary injury is often followed by secondary brain injury due to reduced cerebral perfusion. This may be as a consequence of raised intracranial pressure or reduced blood pressure. One suggested mechanism is that of cerebral vasospasm, thus it has been suggested that calcium blockers, which may prevent this, could be used.

Results

Four randomized control trials were found by this review, these involved 1315 participants.

SOCRATES says

At present there is insufficient evidence to support the use of calcium channel blockers in unselected patients with traumatic brain injury. There is some evidence to suggest that nimodipine may benefit those patients with traumatic subarachnoid haemorrhage.


BARBITURATES FOR ACUTE TRAUMATIC BRAIN INJURY

Following traumatic head injury raised intracranial pressure is a serious complication often leading to secondary brain injury and it is associated with a high mortality rate. Barbiturates are drugs which are believed to reduce intracranial pressure however, they also have unwanted effects such as reduction in blood pressure, which would reduce cerebral perfusion.

Results

Six trials were retrieved of which five were eligible for inclusion. One was unpublished and the data was not available. Two trials had random allocation, one had quasi-random allocation and the remaining two did not describe their methods of allocation.

SOCRATES says

Barbiturate therapy in acute traumatic head injury does not improve outcome. One in four patients suffer a fall in blood pressure. Any lowering of intracranial pressure is offset by reduced cerebral perfusion.


ANTI-EPILEPTIC DRUGS FOR PREVENTING SEIZURES FOLLOWING ACUTE TRAUMATIC BRAIN INJURY

Seizures in the period following head injury may result in a secondary brain injury as a result of increased cerebral metabolism and a rise in intracranial pressure. Antiepileptics agents would be expected to reduce this, but in addition are believed to have independent neuroprotective actions. However, they have narrow therapeutic margins and are associated with major side effects.

The purpose of this review was to determine whether the use of prophylactic antiepileptics in head injury is associated with a favourable neurological outcome.

Results

Six randomised controlled trials with a total of 1218 patients. Four trials used phenytoin, 1 phenobarbital, and 1 carbamezipine.
All the trials were in patients considered to be at high risk of seizures. Treatment was started within 24 hours in 4 of the trials. A significant reduction in seizures within the first week was demonstrated (RR 0.34 95% CI 0.21–0.54). No effect on mortality was shown (RR 1.15 95% CI 0.89–1.51).

**SOCRATES says**
There is no evidence that prophylactic anti-epileptics used after traumatic head injury reduce mortality or disability. However, there is evidence that they reduce early seizures but without reduction in late seizures. Further data is needed to establish their net benefit in this patient group.


**CORTICOSTEROIDS FOR ACUTE TRAUMATIC BRAIN INJURY**
Following traumatic brain injury there can be a rise in intracranial pressure. Corticosteroids have been shown to be beneficial in patients with cerebral oedema secondary to brain tumours and postoperatively. This has led to their use in other situations where raised intracranial pressure can occur.

**Results**
Nineteen trials were identified with a total of 2295 participants. The earliest study was in 1975 and the most recent was in 1995.

**SOCRATES says**
At present there is no demonstrable benefit in using corticosteroids in acute traumatic brain injury.


**SPINAL CORD TRAUMA**
**PHARMACOLOGICAL INTERVENTIONS FOR ACUTE SPINAL CORD INJURY**
Acute spinal cord injury affects some 40 per million of the population each year and is a devastating condition. Pharmacological treatment in the early hours of the injury is aimed at reducing the extent of permanent paralysis during the rest of the patient’s life. The objective of this paper was to review randomised trials of pharmacological therapies for acute spinal cord injury.

**Results**
A total of 10 randomised trials were included in the review. The following drugs were reviewed; methylprednisolone, GM-1 ganglioside, nimodipine, naloxone, TRH, and trilazad mesylate.

**Socrates’ says**
Methylprednisolone sodium succinate is the sole therapy to enhance sustained neurologic recovery in a Phase three randomised trial, and to have been replicated in a second trial. Further improvement in neurological recovery has been shown to occur if the maintenance therapy is extended for 48 hours.


**CONCLUSION**
In this article the SOCRATES working party have presented synopses of reviews from the Cochrane Database of Systematic Reviews relating to head and spinal trauma. In the next issue of the journal we will present those reviews we found of relevance in the areas of orthopedics and trauma.

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