

Is The Glasgow Coma Scale Getting Too Old?

Traumatic brain injury (TBI) is one of the leading causes of death from trauma worldwide. The assessment of TBI was revolutionized in 1976 when the GCS scale was first introduced. Shortly after its introduction, it was found to be predictive of outcome after brain injury. But it does have some drawbacks: it is somewhat complicated, and interrater reliability is low.

Interestingly, a number of studies have shown that the motor component of GCS is nearly as accurate as the full score in predicting survival. Thus, the Simplified Motor Score (SMS) was introduced as a possible substitute for the GCS in 2007. It was found to be equivalent for predicting survival when applied in the ED.

SMS scoring:

- Obeys commands = 2
- Localizes pain = 1
- Withdraws (or less) to pain = 0

So can this scale be validated in the field when applied by prehospital providers?

Nearly 10 years of data (almost 20,000 patients) from the Denver Health trauma registry was analyzed to

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attempt to validate SMS when used by EMS. Although the statistics were not perfect, they found that GCS and SMS were equivalent for predicting the presence of a brain injury, need for emergency intubation, need for neurosurgical intervention, and death. Interestingly, they found that both SMS and GCS were not quite as good at predicting overall outcomes as previously thought.

Bottom line: The simplified motor score is a simple system that has now been shown to be as accurate as GCS in predicting severity and outcome from head injury. To be clear, though, neither is a perfect system. They must still be combined with clinical and radiographic assessments to achieve the best accuracy. But SMS can and should be used both in-hospital and prehospital to get a quick assessment, and may help determine early intervention and need for activating the trauma team.

References:

Assessment of coma and impaired consciousness: a practical scale. Lancet 2:81-84, 1976.

Assessment and prognosis of coma after head injury. Acta Neurochir (Wien) 34:45-55, 1976.

Validation of the simplified motor score in the out-of-hospital setting for the prediction of outcomes after traumatic brain injury. Ann Emerg Med 58(5):417-425, 2011.

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Placement Of ICP Monitors By Non-Neurosurgeons

Traumatic brain injury (TBI) is a common injury worldwide, but neurosurgeons are scarce. Traditionally, neurosurgeons are the ones to place invasive monitors to watch intracranial pressure (ICP). But what about injured people who are taken to a hospital where there is no available neurosurgeon?

A group at Wichita, Kansas looked at their 10 year experience with ICP monitor placement, where it can be done by neurosurgeons, trauma surgeons or general surgical residents (under trauma surgeon supervision). A total of 63 were placed by neurosurgeons, 30 by trauma surgeons, and 464 by residents under supervision. The usual demographics, including hospital stay, were the same across groups. There were essentially no significant differences based on who placed the monitor. The abstract did not state whether the monitors were extradural or intraventricular, or both.

There were only three iatrogenic bleeds, and all occurred with resident placed monitors. None were clinically significant. Malfunction rate was about 5% across all groups. Monitors had to be replaced at some point in about 11% of all three groups. One CNS infection occurred in a patient with a resident-placed monitor.

Bottom line: With proper training and supervision, ICP monitors can be placed by just about anyone. This is particularly important in more rural locations where there are few if any neurosurgeons. But as always, this process needs to be monitored carefully by the hospital's Trauma Performance Improvement / Patient Safety program (PIPS).

Reference: Placement of intracranial pressure monitors by non-neurosurgeons: excellent outcomes can be achieved. J Trauma 73(3):558-563, 2012.

ICP Monitoring: Less Is More?

Management of severe traumatic brain injury (TBI) routinely involves monitoring and control of cerebral perfusion pressure. Monitoring is typically accomplished with an invasive monitor, with the

extraventricular drain (EVD) and fiberoptic intraparenchymal monitors (IP) being the most common.

The extraventricular drain is preferred in many centers because it not only monitors pressures, but it can also be used to drain cerebrospinal fluid (CSF) to actively try to decrease intracranial pressure (ICP). But could less really be more? Surgeons at Massachusetts General reviewed 229 patients with one of these monitors, looking at outcomes and complications. They found the following interesting results:

- There was no difference in mortality between the two monitor types
- The EVD patients did not require surgical decompression as often, possibly because of the ability to decrease ICP through drainage
- The EVD patients were monitored longer, and had a longer ICU length of stay. This was also associated with a longer hospital length of stay.
- Complications were much more common in the extraventricular drain group (31%). The most common complications were no drainage / thrombosis (15%) and malposition (10%). Hemorrhage only occurred in 1.6% of patients.
- Fiberoptic monitors had a lower complication rate (8%). The most common was malfunction leading to loss of monitoring (12%). Hemorrhage only occurred in 0.6% of patients.

Bottom line: Don't change your monitoring technique yet. Much more work needs to be done to flesh out this small retrospective study. But it should prompt us to take a critical look for better indications and contraindications for each type of monitor.

Reference: Intraparenchymal versus extracranial ventricular drain intracranial pressure monitors in traumatic brain injury: less is more? J Am Coll Surg 214(6):950-957, 2012.

Management of CSF Otorrhea / Rhinorrhea

The management of CSF leaks after trauma remains somewhat controversial. The literature is sparse, and generally consists of observational studies. However, some general guidelines are supported by large numbers of retrospectively reviewed patients.

- **Ensure that the patient actually has a CSF leak.** In most patients, this is obvious because they have clear fluid leaking from ear or nose that was not present preinjury. Here are the options when the diagnosis is less obvious (i.e. serosanguinous drainage):
 - **High resolution images of the temporal bones and skull base.** If an obvious breach is noted, especially if fluid is seen in the adjacent sinuses, then a CSF leak is extremely likely.
 - **Glucose testing.** CSF glucose is low compared to serum glucose.
 - **Beta 2 transferrin assay.** This marker is very specific to CSF. However, the test is expensive and results may take several days to a few weeks to receive. **Most leaks will have closed before the results are available, making this a poor test.**
- **Place the patient at bed rest with the head elevated.** The basic concept is to decrease intracranial pressure, which in turn should decrease the rate of leakage. This same technique is used for management of mild ICP increases after head injury.
- **Consider prophylactic antibiotics carefully.** The clinician must balance the likelihood of meningitis with the possibility of selecting resistant bacteria. If the likelihood of contamination is low and the patient is immunocompetent, antibiotics may not be needed.
- **Ear drops are probably not necessary.** They may confuse the picture when gauging resolution of the CSF leak.
- **Wait.** Most traumatic leaks will close spontaneously within 7-10 days. If it does not, a neurosurgeon or ENT surgeon should be consulted to consider surgical closure.

References:

Brodie HA, Thompson TC. Management of complications from 820 temporal bone fractures. *Am J Otol*, 1997;18:188-197.

Brodie HA. Prophylactic antibiotics for posttraumatic cerebrospinal fluid fistulas. *Arch Otolaryngol Head, Neck Surg*. 123:749-752.

Pneumocephalus And Air Transport

Everybody remembers Boyle's law, right?

$$\text{Volume of a gas} = k / \text{Pressure}$$

(where K is a constant)

Which means that, as pressure goes down, the volume of a gas increases. This is important for patients who have a pneumothorax and get on an airplane. As the plane ascends the pneumothorax gets bigger and they may have serious problems. Click here to see guidelines on flying after pneumothorax.

Well, what happens if you have air bubbles in your head (pneumocephalus)? Some patients with serious head injury may have this condition but need to be transported by air to definitive care. Most recently, this has been a consideration in military medical evacuation flights out of Afghanistan.

A paper from the US Army and Air Force studied 21 soldiers (small series) who were evacuated by air with known pneumocephalus. The volume of air was estimated by CT prior to transport, and ranged from less than 1ml to 43ml. None of the patients suffered neurologic deterioration during flight, and 3 who had external ventricular drainage (EVD) showed no significant change in intracranial pressure.

Bottom line: Only two cases of tension pneumocephalus have ever been described. Neither occurred in trauma patients. While expanding pneumothorax may be a problem during commercial flight, there is still little data on tension pneumocephalus. It works for the military because the soldiers are in a flying ICU and can be treated immediately if a problem develops. Not so in commercial aircraft, so beware! But remember, medical helicopters don't fly high enough to create tension problems in any part of the body, so they are not an issue.

Reference: Aeromedical evacuation of patients with pneumocephalus: outcomes in 21 cases. Aviation Space Env Med 79(1):30-35, 2008.

Controlling Fever In Head Injury

Fever is a well recognized side effect of head injury. Management of fever is inconsistent among physicians taking care of these patients. There is a lot of debate on the best course of action, but not so much data. Current enthusiasm for applications of hypothermia has created some reluctance to tolerate much in the way of hyperthermia. Here is my take on the currently available literature.

First, understand that there is a fundamental difference between studies that study induced hyperthermia vs those that look at spontaneous fever. This lies in the fact that the set point for temperature regulation is changed in fever, but not in hyperthermia. Therefore, it is not clear whether hyperthermia studies can truly be used to answer these questions.

Animal studies originally focused on stroke models, which showed deleterious effects from hyperthermia. TBI is very different than stroke, but some hyperthermia models did tend to show cellular damage and blood brain barrier breakdown at temperatures of 39C. However, a fever model in rats showed no outcome difference (in rats) in febrile vs normothermic animals with TBI.

A Medline search (ref 4) yielded no randomized controlled trials that could be used to guide us with regard to fever management. The lesser quality papers involved a very heterogeneous group of subjects that made it difficult to draw good conclusions. As a generalization, they found that extremes of temperature, both high and low, were probably associated with worse outcomes. One randomized prospective study showed that aggressive fever control for temperatures > 38.5C had higher mortality and more infections.

A recent meta-analysis (ref 3) found that TBI patients with fever stayed in the hospital and ICU longer. This translated into an extra \$14,000 per patient. Precise reasons for the longer stay cannot be accurately determined, but it might be expected that patients with fever would undergo time-consuming searches for

possible infectious sources.

Finally, a very recent prospective study (ref 1) at a single institution that did not try to alter temperature found that the optimum survival occurred in a group of patients whose temperatures remained between 36.5 and 38C.

Bottom line: Literature support for aggressive management of fever is poor. If there were a clear correlation with temperature maintained at or slightly below normal, we'd probably have figured it by now. Fever up to 38 degrees C probably does not need to be treated in head injured patients. However, this does not eliminate the need to continue surveillance for infectious complications.

References:

1. *The effect of spontaneous alterations in brain temperature on outcome: a prospective observational cohort study in patients with severe traumatic brain injury. J Neurotrauma 27(12):2157-2164, 2010.*
2. *Induced normothermia attenuates intracranial hypertension and reduces fever burden after severe traumatic brain injury. Neurocrit Care 11(1):82-87, 2009.*
3. *Brain injury and fever: hospital length of stay and cost outcomes. J Intensive Care Med 24(2):131-139, 2009.*
4. *The significance of altered temperature after traumatic brain injury: an analysis of investigations in experimental and human studies: part 2. Br J Neurosurg 22(4):497-507, 2008.*



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