

A3.SPINAL CORD INJURY MANAGEMENT

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Every year, 15–40 people per million suffer a traumatic spinal cord injury (TSCI). Prognosis is poor: over one third of patients leave hospital with complete paraplegia or quadriplegia and only 1 % are discharged neurologically normal. Many TSCI patients are initially admitted to an intensive care unit (ICU) but their management is variable. In brain injuries whatever is the aetiology or mechanism of injury, a cascade of pathophysiological processes is initiated resulting in neuronal damage and cell death. The primary injury occurs as a result of the initial event. However, secondary injury occurs over hours and days. Much of this secondary injury may be amendable to interventions. In TSCI although the pathophysiology seems to be similar there are no evidence-based interventions that improve outcome. There are also no drugs that improve outcome after TSCI. Neuromonitoring although is the standard of care for acute severe traumatic brain injury, this doesn't seem to be the case for TSCI. The optimum mean arterial pressure after TSCI is also unknown. The effects on the injured cord of different anaesthetics, altering PaCO₂, administering vasopressors or mannitol are also unclear.

Several reasons may account for the lack of neuromonitoring in TSCI (concerns about exacerbation of the injury, surgery is required to insert the probes, different specialties are involved in management of TSCI, difficult imaging especially after fixation). When designing pharmacological studies in TSCI patients several issues contribute to the heterogeneity of the population, which means that a big number of patients is required. Another issue is penetration of systemically administered drugs into the injury site, especially since patients with severe cervical TSCI are hypotensive with high intra-spinal pressure. It is possible that drug trials for acute TSCI fail because of inadequate drug penetration at the injury site.

We developed techniques to monitor spinal cord physiological and metabolic parameters from the surface of the injury site in patients with acute TSCI in the ICU. We monitor intra-spinal pressure (ISP) and spinal cord perfusion pressure (SCPP) as well as tissue glucose, lactate, pyruvate, glutamate and glycerol. We have shown that these parameters strongly correlate with outcome. Recently we explored the effect of fever on injury site physiology and metabolism.

We have also investigated regional spinal blood flow with the use of laser speckle contrast imaging and the effects of arterial carbon dioxide and perfusion pressure on the regional blood flow.

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