Head injury is one of the leading causes of death and irreversible brain injury within industrialised societies. With the positive impact of advertising campaigns regarding road and workplace safety, the incidence of severe head injury has significantly reduced over the past decade. However, after a head injury (the “primary injury”) occurs, many physiological processes appear including inflammation, haemorrhage, distortion of the microcirculation and development of areas of infarct; these abnormal processes lead to what is well known as “secondary injury”. Efforts towards improving neurological outcome and minimising the deleterious effects of the primary injury are the focus of neuro-critical units. Multiple advances within neuro-monitoring have occurred within the last decade but there is still a disparity between the sophistication of cerebral monitoring and the real therapeutic armamentarium capable to impact on outcome. Fundamentally, the treatment of head injury is still focused on basic principles and despite extensive world-wide investments in neuro-trauma research, basic physiological measures are the kernel of all therapy. An example of these basic therapeutic principles is to ensure blood supply to the injured brain. Blood supply will be directly related to the vessel diameter as well as to the oxygen carrying capacity of the blood, the haemoglobin level. Here is where essential aspects of head injury such as anemia have become a controversial aspect of the management of head injury, including the indication for blood transfusion.

It is well known that blood transfusions have been associated with increased mortality (1) (4) (5), impaired oxygen delivery due to use of old blood (6) (7) (8) (9), immune reactions (7) (10), bloodstream infections (1) (4) (5) and transfusion errors (1) (4) (5) (11). Whilst blood transfusions have also shown to be associated with poor neurological outcome following head injury (15), confounding factors include concomitant injuries in polytrauma patients or the need for neurosurgical intervention. Low levels of oxygen and hypotension however are markers of poor outcome in the acute management after head injury (2) (16) (17). A review found that anemia in head injury significantly increased morbidity and mortality (15). Moreover, several animal studies have shown that maintenance of a high transfusion threshold is associated with a significant increase in survival and better neurological outcomes (2) (12). Induced anemia after head injury leads to cerebral ischemia in cases where cerebral blood flow regulation is impaired and in the same side of cerebral contusions (2). Studies have shown that “secondary injury” occurs at a hematocrit of 20% consistent with blood haemoglobin level between 6-7gm/dL which is close to current transfusion practices (20) (21). The above disparity between a general approach to anaemia in the critically ill patient and abnormal events specifically in head injury patients, contribute to the lack of clarity of resuscitation goals. There is currently no data defining optimal markers of tissue perfusion or neurological outcomes in neurotrauma patients. However, critical levels of tissue oxygenation of 15mmHg, have been shown to be correlated with increased incidence of stroke and mortality (24) (25) (26) (27). Cerebral ischemia is associated with an elevated
lactate/pyruvate ratio and raised tissue concentrations of glutamate and glycerol, all markers of deranged cerebral metabolism (28). These observations suggest that transfusion thresholds in head injury patients maybe better determined by these markers of cerebral ischemia rather than assuming a generalised threshold of transfusion to be implemented in all patients.

Currently there is no evidence supporting the maintenance of anemia in head injury patients. Ethical concerns have been raised concerning trials of transfusion triggers based on differing haemoglobin concentrations where neurological outcome is the endpoint

Although some studies have correlated blood transfusion with an increase on cerebral oxygenation (6) (10) (11) (12), these studies failed to demonstrate a correlation with better neurological outcome. Only sustained and severe cerebral hypoxia has been correlated with death (6) (11). Despite the reported effects of anemia, it remains unclear what level of haemoglobin should be targeted as optimal.

Finally, a case can be made for tailoring blood transfusions to individual needs based on measures of cerebral blood flow dynamics and cerebral metabolism. The limitation of this approach is that there is no data to date showing a correlation between the introduction of advanced cerebral monitoring techniques and better neurological outcomes.

Although there is currently no pathophysiological data showing a correlation between the improvement of markers of tissue perfusion and neurological outcome, a physiological understanding of the cerebral microcirculation is needed prior to establishing transfusion thresholds in the setting of acute head injury. Current multimodality monitoring in acute head injury involves intracranial pressure-volume and cerebral metabolic responses to trauma. The integration of these parameters into a clinical management algorithm may allow patient tailored blood transfusion requirements. Meanwhile, randomised controlled trials comparing liberal versus restrictive transfusion thresholds in head injury are warranted.

References:


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