BJA

British Journal of Anaesthesia, ■ (■): 1–11 (2018)

doi: 10.1016/j.bja.2017.11.108
Advance Access Publication Date: xxx
Review Article

REVIEW ARTICLE

Anaemia and red blood cell transfusion in intracranial neurosurgery: a comprehensive review

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Summary

Both anaemia and blood transfusion are associated with poor outcomes in the neurosurgical population. Based on the available literature, the optimal haemoglobin concentration for neurologically injured patients appears to be in the range of 9.0–10.0 g dl^{-1}, although the individual risks and benefits should be weighed. Several perioperative blood conservation strategies have been used successfully in neurosurgery, including correction of anaemia and coagulopathy, use of antifibrinolytics, and intraoperative cell salvage. Avoidance of non-steroidal anti-inflammatory drugs and starch-containing solutions is recommended given the potential for platelet dysfunction.

Keywords: anaemia; coagulation; neurosurgery; transfusion

The development of modern neurosurgery was facilitated by the evolution of general anaesthesia, intravenous fluid management, aseptic technique, and advances in neurosurgical haemostasis in the 19th and 20th centuries.[1] In 1904, pioneering American neurosurgeon Harvey Cushing described the use of a pneumatic tourniquet to reduce intraoperative bleeding from the scalp.[2] He went on to describe the utility of high frequency electrocautery in the excision of previously inaccessible intracranial tumours[3] and the use of operative cell salvage and return of autologous blood.[4] In 1946, Gardner provided an account of induced hypotension to reduce blood loss and improve neurosurgical operating conditions.[5] Cushing's landmark publication in 1911 provided a summary of neurosurgical haemostasis techniques and highlighted his appreciation for the importance of a bloodless field to achieve surgical precision, avoid haemorrhagic shock, and prevent complications of anaemia.[6] Taken for granted today, these tenets were radical in the early 20th century and not universally accepted. In particular, Cushing's recognition of the association between anaemia and poor neurosurgical outcome was ahead of its time. Although an accumulating body of evidence now supports his observation, many questions regarding haemostasis and anaemia in the neurosurgical population remain. This paper aims to review the epidemiology and clinical outcomes associated with anaemia and blood transfusion, and identify perioperative blood conservation strategies to minimise blood loss in patients undergoing intracranial neurosurgical procedures.

Editorial decision: November 30, 2017; **Accepted:** November 30, 2017

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What is the significance of anaemia in the neurosurgical population?

Although it constitutes only 2% of total body weight, the human brain has high metabolic requirements and receives 20–25% of cardiac output (CO). Total cerebral oxygen delivery is a function of cerebral blood flow (CBF) multiplied by the oxygen content of the blood (CaO_2). CBF is ordinarily autoregulated over a wide range of cerebral perfusion pressures (CPP) at approximately 50 ml 100 g^{-1} min^{-1} where CPP is determined by the mean arterial pressure (MAP) minus the cerebral venous pressure (or intracranial pressure, whichever is higher). Outside the normal physiological limits of CPP (~60–160 mm Hg), CBF is linearly dependent upon MAP. During periods of acute anaemia (reduced CaO_2), oxygen delivery is maintained via compensatory cardiovascular and cerebrovascular changes including an increase in CO, preferential distribution of CO to the cerebral circulation, increased cerebral oxygen extraction, and, importantly, an increase in CBF secondary to active cerebral vasodilatation.[7] In the setting of normovolaemic anaemia, reduced blood viscosity is a major determinant of increased CO through enhanced preload and reduced afterload.[8] Reduced viscosity may also improve the flow characteristics (i.e. rheology) of blood particularly through areas of cerebral vascular stenoses. However, these compensatory mechanisms have limitations. As cerebral oxygen utilisation becomes supply dependent, anaemia may contribute to reduced cerebral oxygen delivery and a secondary hypoxic insult to the injured brain. In healthy volunteers, anaemia-induced cognitive dysfunction is evident at haemoglobin (Hb) concentrations between 5.0 and 6.0 g dl^{-1}.[9] Following acute brain injury, impaired autoregulatory mechanisms may allow anaemia-induced cerebral dysfunction and injury at higher Hb thresholds. In a recent retrospective cohort study of patients with severe traumatic brain injury (TBI), the amount of time spent with an Hb concentration $\geq$9.0 g dl^{-1} was associated with improved neurological outcome at 6 months.[10] Similarly, in a previous cohort of patients with severe TBI, the association of anaemia (Hb<9.0 g dl^{-1}) with low brain tissue oxygen tension (<20 mm Hg) was a risk factor for poor neurological outcome.[11] In patients with subarachnoid haemorrhage (SAH), a reduction in red cell volume precedes neurological deterioration.[12] An Hb threshold >11 g dl^{-1} is associated with a reduced incidence of symptomatic cerebral vasospasm[13] and more favourable neurological outcome.[14] At higher Hb concentrations, however, the benefit of increased oxygen carrying capacity may be offset by increases in blood viscosity and reduced CBF.[15]

The incidence of anaemia in neurosurgical procedures varies depending on the population and surgery. Data from the National Surgical Quality Improvement Program reveal that the incidence of moderate to severe anaemia (i.e. haematocrit <30%) in elective cranial surgery is 2.7%.[16,17] In critically-ill neurosurgical patient populations, however, the incidence increases; at least 50% of patients with TBI[18] and 47% of patients after SAH are anaemic.[19] In the critically-ill population, anaemia may be multifactorial resulting from systemic inflammatory-mediated suppression of haematopoiesis, frequent phlebotomy, haemorrhage, and haemodynamic resuscitation-induced haemodilution. In the context of SAH, anaemia may result from deliberate hypervolaemia and haemodilution for the prevention and treatment of vasospasm. A growing body of evidence in neurosurgical patients supports anaemia as an independent predictor of cerebral vasospasm, medical complications, poor neurological outcome, and increased mortality.[14,17,20,21]

What is the significance of transfusion in the neurosurgical population?

In the setting of anaemia, transfusion of blood products can rapidly increase Hb concentrations and oxygen delivery. Overall, cohort studies including all types of neurosurgical procedures have reported a rate of allogeneic transfusion of 1.7–5.4%.[22,23] However, the reported incidence of transfusion for specific patient populations undergoing intracranial neurosurgery ranges from 10 to 95% with a much higher incidence among paediatric patients undergoing craniosynostosis surgery and cranial vault reconstruction (Table 1). In the paediatric population undergoing brain tumour resection, age <4 years, duration of surgery >270 min, and a preoperative Hb concentration <12.2 g dl^{-1} were independent predictors of allogeneic blood transfusion.[24] In the context of isolated TBI, the presence of acute traumatic coagulopathy increased the risk of transfusion by 41% and increased hospital length of stay and mortality.[25] In aneurysmal SAH, administration of packed red blood cells is strongly related to intraoperative aneurysm rupture[26] and may be as high as 25%.[27] The rate of transfusion also varies depending on the procedure, from 10% in complex skull base neurosurgical procedures[28] to 36% in patients with TBI[29] and as high as 45% in paediatric craniosynostosis surgery.[30] In the latter instance, risk factors for large volume blood loss and transfusion include syndromic craniosynostosis, pansynostosis, age <18 months, and longer duration of procedure.[31] A recent multicentre observational study of children undergoing complex cranial vault reconstruction in North America demonstrated that 95% of infants aged <24 months and 79% of children aged >24 months underwent perioperative allogeneic blood transfusion.[32] Future strategies to reduce transfusion should focus on the identification and mitigation of modifiable risk factors in these populations.

Paradoxically, while the definitive management of anaemia-induced organ dysfunction remains the transfusion of allogeneic red blood cells, transfusion itself confers an increased risk of morbidity and mortality.[33–36] Changes in immunomodulation, infectious and allergic complications, and transfusion associated lung injury and circulatory overload are all potential pathological consequences of transfusion. These complications result in an increased risk of respiratory failure, prolonged intubation, acute respiratory distress syndrome, wound infection, sepsis, cardiac events, increased hospital length of stay and death. In the setting of TBI, a clinical trial comparing Hb transfusion thresholds of 7.0 g dl^{-1} *vs* 10.0 g dl^{-1} revealed an increased risk of thromboembolic events in the latter with no significant improvement in neurological outcome.[37] Further analysis of this data demonstrated a more than two-fold increase in the risk of progressive haemorrhagic injury with a Hb transfusion threshold >10.0 g dl^{-1} when compared with a more restrictive threshold (i.e. 7.0 g dl^{-1}).[38] In patients undergoing open neurosurgical intervention for intracranial aneurysm, perioperative blood transfusion was shown to be an independent risk factor for major morbid complications.[39]

Unfortunately, the optimal trigger for Hb transfusion in neurosurgical populations remains elusive and the relative risks and benefits of anaemia and transfusion must be

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