CRITICAL CARE

COMMENTARY

Eubaric hyperoxia: controversies in the management of acute traumatic brain injury

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See related research by Raj *et al.*, http://ccforum.com/content/17/4/R177

Abstract

Controversy exists on the role of hyperoxia in major trauma with brain injury. Hyperoxia on arterial blood gas has been associated with acute lung injury and pulmonary complications, impacting clinical outcome. The hyperoxia could be reflective of the physiological interventions following major systemic trauma. Despite the standard resuscitation of patients with acute traumatic brain injury, up to 60% demonstrate low brain oxygen upon admission to the ICU. While eubaric hyperoxia has been beneficial in experimental studies, clinical brain oxygen protocols incorporating intracranial pressure control, maintenance of cerebral perfusion pressure, and the effective use of fraction of inspired oxygen adjustments to maintain cerebral oxygenation levels >20 to 25 mmHg have demonstrated mortality reductions and improved clinical outcomes. The risk of low brain oxygen is most acute in the first 24 to 48 hours after injury. The administration of a high fraction of inspired oxygen (0.6 to 1.0) in the emergency room may be justifiable until ICU admission for the placement of invasive neurocritical care monitoring systems. Thereafter, fraction of inspired oxygen levels need to be careful titrated to prevent low brain oxygen levels.

One of the major advantages of bureaucratic institutionalized national healthcare systems is the ability to analyze treatment paradigms implemented across a wide population, providing a comprehensive overview of clinical care with the view to improve quality and outcome. In a retrospective interrogation of a prospectively collected database, Raj and colleagues demonstrate that the presence of arterial hyperoxia within the first 24 hours is not a predictive marker of neurological outcome in moderate to severe traumatic brain injury (TBI) [1].

The human has evolved into a complex adaptive system that is dynamic, exhibiting nonlinear relationships, and is acutely affected by many physiological systems [2]. Although trauma scoring systems (for example, injury severity score, revised trauma score and probability of survival) or physiological scoring methods (for example, Acute Physiology and Chronic Health Evaluation) employ a multitude of parameters, the use of a single physiological parameter – for example, arterial hyperoxia [1] – is not predictive for outcome. The hyperoxia detected could be reflective of the therapeutic intervention associated with major trauma, and the subsequent treatment and clinical course would vary after the initial arterial blood oxygen measurement since <50% of patients underwent intracranial pressure monitoring [1]. Even when intracranial pressure monitoring is implemented, a high mortality is observed. Rockswold and colleagues reported a mortality of 42% in the control group for severe TBI, similar to the publication by Raj and colleagues (39%) [1,3], but the addition of hyperoxia results in a significant relative risk reduction for mortality [3], similar to other published studies [4,5].

Early initiation of brain oxygen protocols – that is, intracranial pressure/cerebral perfusion pressure/partial pressure of brain tissue oxygen ($PbtO_2$) [5-7] and $PbtO_2$ critical care guide [4,8-10] – have shown mortality reductions [4,5] and improved patient neurological outcome [4], in contrast to delayed implementation, which may not translate into outcome benefits [11]. The addition of oxygen as a therapeutic tool would necessitate multimodality invasive neurological monitoring in all patients with severe TBI since the fraction of inspired oxygen and airway pressures (that is, positive end-expiratory pressure or pressure support) would need to be carefully titrated against $PbtO_2$ levels.

The deleterious effects of eubaric hyperoxia (that is, adult respiratory distress syndrome, atelectasis,

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ventilation/perfusion mismatch) have been postulated, which may be due to concomitant pulmonary contusion, aspiration or even high ventilator pressures [12], but have not been substantiated by clinical experience [3-5,9]. Indeed, eubaric hyperoxia – needed primarily in the first 48 hours [4] – immediately improves $PbtO_2$, reducing lactate levels and infarct size, and exhibits neuroprotective properties [13,14]. High airway pressure of barotraumas, previously implicated in lung injury [12], has been refuted by Rockswold and colleagues who demonstrated the safety and efficacy of eubaric and hyperbaric oxygen therapy in the management of acute TBI in a randomized control study. Specifically, bronchial alveolar lavage revealed no differences in interleukin levels and, rather than toxicity, microdialysis data revealed the neuroprotective effects of oxygen in both the normal and the injured brain [3].

In conclusion, one has to differentiate between hyperoxia (detected in laboratory tests) with its potential association with clinical outcome, and the use of eubaric or hyperbaric oxygen as a therapeutic tool. In the former, a single episode of hyperoxia may have shown an association with mortality as reported in this study [1], but it would rarely prove causality. In the latter, the high therapy intensity level employed with brain oxygen protocols, of which eubaric hyperoxia is critical, requires invasive monitoring with careful goal-directed oxygen titration. The emerging clinical experience demonstrates that hyperoxia is safe and beneficial to the brain, and does not injure the lung as previously feared [3].

Abbreviations

$PbtO_2$: Partial pressure of brain tissue oxygen; TBI: Traumatic brain injury.

Competing interests

PKN served as a scientific advisor for Integra Neurosciences, Plainsboro, NJ, USA, who manufactured the neuromonitoring devices between 2001 and 2010.

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