Resuscitation (2008) **76**, 333—340

available at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/resuscitation

REVIEW

Early ventilation in traumatic brain injury☆

Daniel P. Davis*

UC San Diego Department of Emergency Medicine, 200 West Arbor Drive, #8676, San Diego, CA 92103-8676, United States

Received 20 April 2007; accepted 2 August 2007

KEYWORDS
Hyperventilation;
Hypoventilation;
Intubation;
Traumatic brain injury;
Hypocapnia

Summary While airway and ventilatory compromise are significant concerns following traumatic brain injury (TBI), there is little data supporting an aggressive approach to airway management by prehospital personnel, and a growing number of reports suggesting an association between early intubation and increased mortality. Recent clinical and experimental data suggest that hyperventilation is an important contributor to these adverse outcomes in TBI patients. Various mechanisms appear to be responsible for the worsened outcomes, including hemodynamic, cerebrovascular, immunologic and cellular effects. Here, relevant experimental and clinical data regarding the impact of ventilation on TBI are reviewed. In addition, experimental data regarding potential mechanisms for the adverse effects of hyperventilation and hypocapnia on the injured brain are presented. Finally, the limited data regarding the impact of hypoventilation and hypercapnia on outcome from TBI are discussed.

Contents

☆ A Spanish translated version of the summary of this article appears as Appendix in the final online version at 10.1016/j.resuscitation.2007.08.004.

* Tel.: +1 619 543 3829.
E-mail address: davismd@cox.net.

0300-9572/$ — see front matter © 2007 Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.resuscitation.2007.08.004

Introduction

Acute traumatic brain injury (TBI) accounts for substantial morbidity and mortality.[1–3] Airway obstruction, aspiration, hypoxia and respiratory compromise are relatively common in these patients, with invasive airway management the current standard of care to enhance oxygenation and ventilation and to provide airway protection.[4–9] This includes the use of neuromuscular blocking agents as part of a rapid sequence intubation (RSI) protocol.[10–14] Success with invasive airway management has been demonstrated by both prehospital and hospital personnel; however, outcomes data supporting this approach has been conspicuously absent. In fact, an increasing number of TBI studies suggest that early intubation may be associated with worse outcomes.[15–19]

Inappropriate ventilation may be an important contributor to the adverse outcomes observed with early intubation of TBI patients. Quantitative capnometry applied in the prehospital environment has revealed a high incidence of hyperventilation among both paramedics and air medical crews.[20–29] Thomas et al. recently reported end-tidal carbon dioxide ($ETCO_2$) values of 25 mmHg or lower in one-third of patients with severe TBI transported by air medical crews with access to capnometry; over two-thirds had at least one $ETCO_2$ value of 30 mmHg or less.[21] In addition, more than three-quarters of recorded $ETCO_2$ values from the San Diego Paramedic RSI Trial were below 30 mmHg.[26] Experimental models document an adverse effect of hyperventilation on the brain through multiple mechanisms (Figure 1), and recent clinical evidence documents a detrimental effect of hyperventilation on outcome for both TBI and cardiac arrest.[26–28,30–32] Here the clinical impact and potential mechanisms related to the adverse effects of early hyperventilation on TBI are explored. In addition, the limited data regarding hypoventilation and TBI are reviewed.

Pathophysiology of hyperventilation

Hemodynamic effects

Spontaneous respiration is accomplished via a slight negative intrathoracic pressure during inspiration, which not

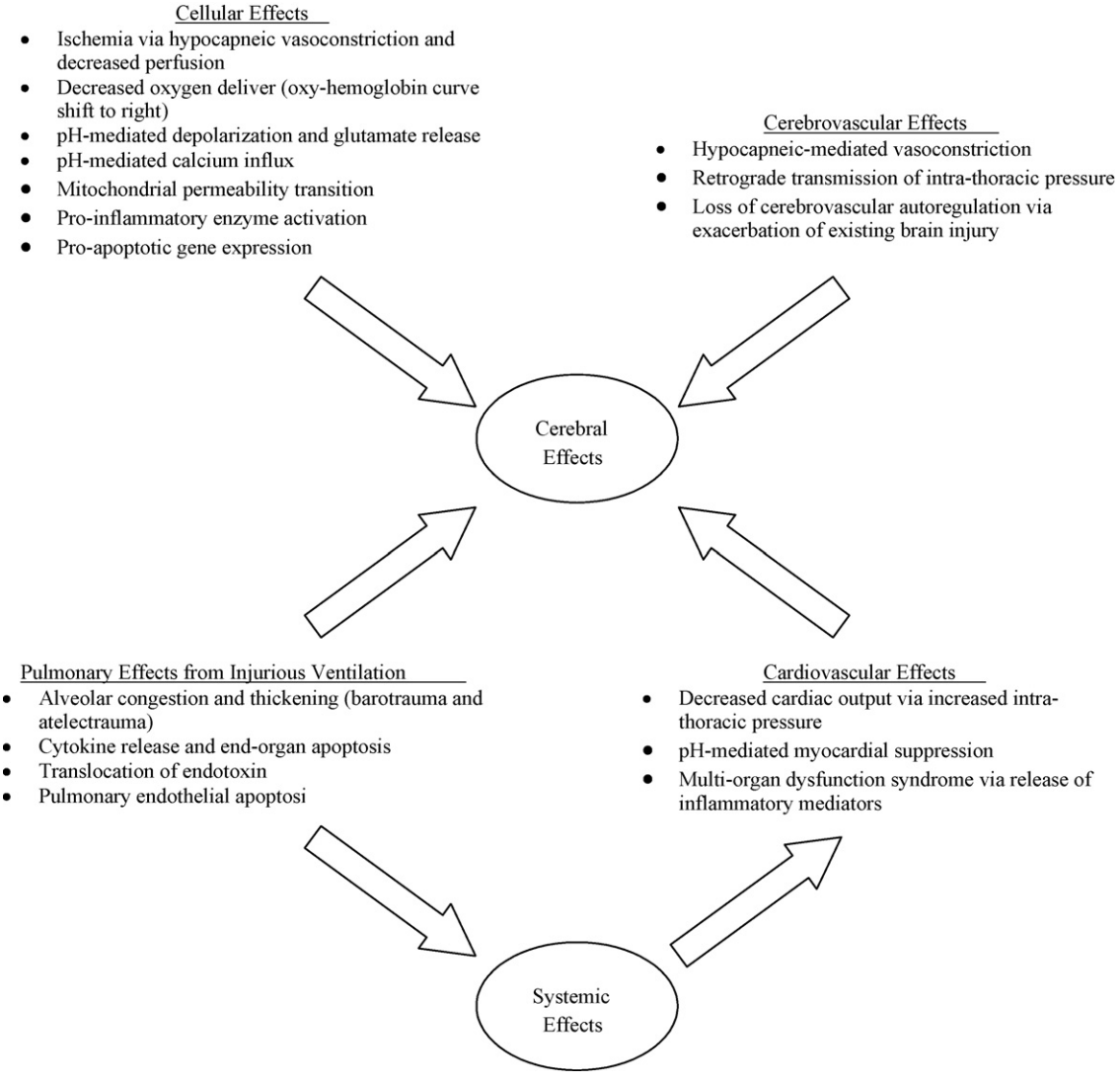

Figure 1 Schematic representing mechanisms responsible for the detrimental effects of hyperventilation on the injured brain.