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EDITORIAL

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Pressure, flow and Occam's Razor: a matter of "*steal*"?

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Occam's Razor, named for the fourteenth-century philosopher William of Occam, is the principle of preferring the simplest explanation of events. The principle states, literally, that entities should not be multiplied without necessity (*pluralitas non est ponenda sine necessitate*), in other words, one should not make more assumptions than are needed for an explanation. This is often called the principle of scientific 'parsimony'. The principle has played a major role in getting rid of fictitious or unnecessary elements from explanations as far back as the Middle Ages. Bertrand Russell and other logicians, for instance, eliminated traditional metaphysical concepts by employing Occam's Razor, and it may be a useful principle in intensive care today.

Mechanical ventilation is a cornerstone of the management of patients with severe traumatic brain injury (TBI). Endotracheal intubation secures the airway, and mechanical ventilation, with volume control, ensures adequate oxygenation and regulation of arterial PCO_2 tension. Failure or compromise of cerebral vasculature "autoregulation" after trauma renders the brain susceptible to fluctuations in oxygen delivery and changes in $PaCO_2$ that can exacerbate areas of ischaemia if too low and increase intracranial pressure (ICP) and therefore reduce perfusion pressure if too high. Clinical practise goals are adequate oxygenation and low normocapnia with stability, and are key to successful TBI management [1, 2].

Acute lung injury and acute respiratory distress syndrome are amongst the most common and important complications [3] after TBI and frequently pose a significant clinical challenge [4]. The contribution by Mascia et al. [5] provides useful new information to support decision making in the management of patients with both TBI and acute lung injury. The reasons for wishing to use positive end-expiratory pressure (PEEP) include improving oxygenation [6] and protecting the lung [7]. However, PEEP also increases venous pressure, and this in turn may lead to an increase in cerebral blood volume, ICP and a reduction in cerebral perfusion. Head-injury patients have traditionally been managed at 30° head-up tilt to ensure optimal venous drainage, with the head and neck alignment ensured. Mascia et al. showed that PEEP less than ICP produces a modest increase in right atrial pressure in all patients. Patients who achieved lung recruitment showed no increase in $PaCO_2$ and ICP remained stable. However, in non-recruiters alveolar hyperinflation occurred, and $PaCO_2$ increased with the consequence of an increase in ICP and transcranial Doppler velocities. Previous studies have highlighted the important relationship between PEEP and ICP, suggesting that PEEP less than ICP do not have an important effect upon ICP.

Blood flow in *both* the pulmonary vascular bed and cerebral vasculature can be described as a Starling resistor and is determined by the inflow pressure (Pi), external pressure (Pe or ICP in brain) and the outflow pressure (Pv or right atrial pressure in brain). The relationship between the three is usually classified according to zones; (a) Pe>Pi >Pv, (b) Pi >Pe >Pv and (c) Pi >Pv >Pe and has been well characterised in lung. Where focal injury occurs in the brain it is possible to have a transition from zones a to b in the centre of a lesion with locally high tissue pressure, to zone c in the peri-contusional tissue. Thus it is theoretically possible to have three Starling resistors in

parallel in a contused area of brain tissue. Experimental models show that when this occurs there is high blood flow diversion to Starling resistors with low external pressure [8]. Interestingly, high-inflow resistances make this steal phenomenon more likely and is relevant when we consider that many interventions to reduce raised ICP have as their mechanism of action by increasing cerebral vascular resistance [9, 10]. An exception is increasing arterial pressure. An increase in Pi reduces steal by decreasing flow diversion.

Thus steal is due to the difference in outflow pressures in parallel Starling resistors with high inflow resistance and can be due to Pe or Pv. Increasing Pv reduces the blood flow diversion and when Pv equals Pe (ICP in brain) flow diversion and steal is abolished, reducing heterogeneity in cerebral tissue perfusion and restoring blood flow in areas of increased tissue pressure. Increasing venous pressure decreases global cerebral blood flow and, if utilised clinically, may require augmentation of arterial pressure [8].

Therefore increasing intrathoracic pressure using PEEP may recruit lung tissue and improve maldistribution of perfusion in lung tissue, as described by Mascia et al., and may serendipitously reduce venous steal, a potential cause of tissue ischaemia in areas of injured brain with increased tissue pressure.

Patients with TBI are managed according to physiological pressure goals and not usually brain perfusion indices. These pressures are ICP and mean arterial pressure and both should have the same zero reference point. There remains debate and speculation about the effect of head up tilt on cerebral blood flow. Head-up tilt reduces venous pressure and ICP but also decreases Pi due to a reduction in hydrostatic pressure. Managing the patient with the head level (supine), increases ICP, Pi and Pv. A further increase in Pv induced by PEEP may facilitate recruitment of collapsed cerebral vascular network and correct perifocal perfusion maldistribution.

There are few clinical examples of improved cerebral perfusion with increased Pv, but recent data suggest that ischaemia remains an important problem after TBI. Further investigation is warranted in this area and investigators would do well to adhere to the principle of parsimony and reduce the unnecessarily complex hypotheses used to explain (patho)physiology.

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