CROSSTALK

Last Word on the CrossTalk proposal and opposing view: There is/there is not added benefit to providing permissive hypercapnia in the treatment of ARDS

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On the face of it, we can come to a synthesis in regard to hypercapnia and ARDS.

It is clear that while HCA can limit tissue injury induced by many causes (via pH dependent and pH-independent mechanisms), there is also a clear potential for ‘off-target’ harm with acute exposure (e.g. raised ICP, pulmonary hypertension), or exposure for prolonged periods (risk of infection) or at high doses (mitochondrial inhibition).

A structured approach would be ongoing comprehensive determination of molecular, cellular and tissue impact across a range of representative pathologies; in the laboratory, much has been learned, and much painstaking discovery undoubtedly remains. In patients, such research would parallel better characterization of ARDS (which is, after all, a syndrome definition that is independent of the underlying biological processes) and short-term Phase-I studies in selected groups. In such studies, harm might be minimized by selective carbon dioxide delivery (e.g. restricted to late phase of inspiration; Brogan et al. 2004) or possibly by using THAM for pH buffering (Beitler et al. 2013).

A less desirable approach might be embarking on a so-called ‘definitive’ clinical trial. Here, large numbers of heterogeneous patients would be randomized, with recruitment based on current ‘diagnostic’ criteria, and ‘outcome’ (i.e. mortality) would be measured. Akin to the earlier studies of ‘sepsis’ (Ziegler et al. 1991; Weil, 1994) such an approach would not ‘answer’ the question – it would simply postpone it.

References


Competing interests

None declared.
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