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Response to Spindelboek et al. Oxygen and cardiac arrest: the timepoint matters

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Dear Editor,

I would like to thank Spindelboek and colleagues [1] for their letter in response to the recent editorial [2] that questions the optimal FiO_2 during advance cardiopulmonary resuscitation (CPR).

Their observational study raises some challenging hypotheses. However, I would question whether the evidence it provides is sufficient to prevent a prospective randomised control trial of normoxic verses hyperoxic inspired gas mixtures during advanced CPR. Their retrospective analysis of 145 of 1005 (14 %) patients, who they stratified into arterial oxygen tension groups, all received an FiO_2 of 1.0 from the earliest possible time point. Thus the stratification is likely to be the consequence of different cardiorespiratory pathologies in the patients. I would conjecture that it is this difference in pathology which has determined outcomes and not the arterial oxygen tension, which in this case acts merely as a surrogate

marker. This may also be reflected in differences between the groups in regard to the proportions that received atropine and thrombolysis. There are missing items of information that might also be pertinent, specifically the suspected cause of the cardiac arrest, the arterial carbon dioxide tension, the cumulative dose of adrenaline, the time to first chest compressions and the time to sustained return of spontaneous circulation (RoSC). It is also notable that approximately 70 % of the patients received 8.4 % sodium bicarbonate, which is counselled against in the most recent international guidelines. The relevance of these factors cannot easily be determined.

Theirs is the only study in adult humans that I can identify. There is, however, a significant body of antithetical evidence that supports chest compression only resuscitation as non-inferior or even superior to 30:2 cardiopulmonary resuscitation with either rescue breathing or airway adjuncts and an FiO_2 of 1.0. The probable explanation for these findings is that oxygen delivery is principally determined by circulatory flow and not oxygen content, which itself is principally determined by haemoglobin concentration and not oxygen tension. Clearly, there is a need to supply oxygen via forced ventilation after a period of cardiac arrest and chest compression only resuscitation; however, whether 100 % oxygen is beneficial over air remains speculative.

Part of the rationale that has led to the current randomised, placebo-controlled trial of adrenaline during

advanced CPR is the evidence that adrenaline may marginally improve the probability of RoSC but does not improve survival with a cerebral performance category (CPC) of 1. The same might also be true of 100 % oxygen during advanced CPR. The biological plausibility behind such speculation emerges from the evidence that hyperoxia during early reperfusion is more damaging to the brain than the heart.

In summary, the principal determinants of outcome following cardiac arrest are the cause of the cardiac arrest, the physiological reserve of the patient and the time to first effective supportive care. Time to sustained RoSC and post RoSC care, including early definitive treatment of the cause of the arrest, are also critical. I suspect the FiO_2 during resuscitation only becomes a significant factor during prolonged low flow states, the neurological outcomes from which are universally poor.

Conflicts of interest I declare that I have no competing interests.

References

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