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Editorial

Ventilation during cardiopulmonary resuscitation—*Only mostly dead!*

Recent focus of research and guidelines for management of cardiac arrest has been on the quality of chest compressions, including rate, depth, and full release, and on minimizing interruptions in compressions, measured by the compression fraction.[1–4] Some studies demonstrated an overall increase in survival by encouraging lay responders to do compressions without ventilation.[5,6] This was likely due to an increase in frequency of bystander CPR, maintaining more constant coronary perfusion pressure, and leveraging that most persons who suddenly collapse in cardiac arrest have a reservoir of oxygen in their lungs and blood that should be sufficient for 5–6 min. Some EMS systems adopted an initial approach of not pausing chest compressions for ventilation or even compression-only CPR.[7]

The study by Idris et al in this issue of *Resuscitation* should resurrect an interest in ventilation during cardiac arrest management by professional rescuers.[8] The authors reaffirm the accuracy and reliability of and then use the technology of thoracic bioimpedance measurement to determine presence or absence of ventilation during resuscitation. In this retrospective analysis of data collected at one site of the Resuscitation Outcomes Consortium for a trial comparing continuous and interrupted compressions,[9] the authors dichotomized patients into two groups - those who received ventilation during <50% of pauses and those who received ventilation during >50% of pauses. Ventilation was only counted if the impedance change correlated to a tidal volume of >250cc, the minimum considered to overcome dead space. Their analysis showed that the group with more ventilation had higher rates of ROSC, survival to hospital discharge and neurologically-intact survival. Adjusted regression analysis found that ventilation during >50% of pauses was associated with higher odds of ROSC, survival to hospital admission and good neurological outcome. Further confirming the association of ventilation with good outcomes is a dose-response relationship for 0–2 ventilations per pause.

The important and unique contribution of the Idris trial is that it uses bioimpedance changes to measure delivered lung ventilation, not just ventilation rate. While this technology was described previously,[10,11] there has been little research exploring the effects of ventilation volume and the interaction with compressions. Years ago Safar and colleagues demonstrated that chest compressions do not provide much lung ventilation[12] and more recently others have confirmed the same.[13,14] These later studies found tidal volumes of 7.5 ml and 41.5 ml, well below anatomical dead space volume. Without sufficient ventilation, oxygen stores will be exhausted and hypercapnia will lead to severe acidosis further impairing physiological function and decreasing the likelihood of resuscitation.

Aufderheide demonstrated severe hyperventilation is harmful because of elevated intrathoracic pressure leading to decreased coronary perfusion pressure, and found that ventilation rates by professional rescuers were frequently much higher than recommended by guidelines.[15] But tachy-ventilation is not always synonymous with hyper-ventilation. A recent study by Vissers et al. looked at the effect of ventilation rate on outcomes from cardiac arrest and found no difference between <10/min vs >10/min.[16] Importantly, the authors did not look at ventilation volume, as Idris does in this study.

Direct measurement of lung ventilation may inform about the recently-recognized phenomenon of intrathoracic airway closure in which lung compression and atelectasis generated by chest compressions can lead to collapse of small airways and obstruct pulmonary airflow.[17] This is observable on capnography and was verified in human cadaver models. Intrathoracic airway closure may at least in part explain why chest compressions themselves do not generate significant alveolar ventilation. While patency of the upper airway alone does not alleviate this phenomenon,[12] the provision of continuous or end expiratory positive pressure reverses the condition and leads to markedly improved alveolar ventilation.[18,19]

The study by Idris et al. should be considered thought-provoking and exploratory, as it is based on data from one site and collected by one brand of defibrillator. Expanding the analysis to include data from additional sites should be informative and will help with designing future prospective trials on the effect of ventilation during cardiac arrest management. Many questions remain unanswered about ventilation during resuscitation, leading to great variation in clinical practice.[20] These questions include when ventilation should begin, optimal minute ventilation, manner of delivery (rate, pressure, PEEP), route of delivery (mask, SGA, ETI) and optimal FIO2. But what we do now know, thanks in part to the work of Idris and his team, is that ventilation does matter, and that ventilating in less than half of the pauses (while doing 30:2 CPR) leads to worse patient outcomes.

Conflict of interest statement

Author has no conflicts of interest.

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