

Should we De-Emphasize or Abandon Intubation During Adult Cardiac Arrest? No!

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Although research has provided important insights into the physiology of cardiac arrest and cardiopulmonary resuscitation (CPR), our understanding of the interaction between chest compression and mechanical ventilation is limited. Despite the fact that we live in the era of "high quality chest compressions is the most important factor during CPR", it is well-known that both 'cardiac pump' and 'thoracic pump' may have a role in forward blood flow. Of course, positive intrathoracic pressure may be harmful if excessive, but proper timing of compression and ventilation may improve the circulation [1]. In fact, as positive pressure ventilation may have favorable effects, such as pulmonary recruitment, minimal thoracic volume reduction, reduced ventilation/perfusion mismatch, and improved oxygenation [2-4], it may not be so wise to deprive this treatment from our patients.

A retrospective, observational cohort study recently reported that patients who were intubated during in-hospital resuscitation were significantly less likely to survive to discharge, as well as to be discharged with good functional status than those who were not intubated [5]. In this study, 75,579 (69.9%) patients were intubated, with 71,615 (66.3% of all patients and 94.8% of those intubated) intubated within the first 15 minutes. Although multiple mechanisms were proposed to explain a potential causal relationship between tracheal intubation and poor outcomes, the authors did not report the method of post-intubation ventilation (e.g. self-inflating bag or automated ventilator). Of note, ventilation with a self-inflating bag usually results in excessive ventilation volume and rate, thus aggravating oxygenation and hemodynamics, and surprisingly, it continues to be a major limitation that is not always taken into account when interpreting the results of a study.

Although high intrathoracic pressure impairs venous return, we cannot neglect the fact that numerous studies indicate the favorable effects of 'thoracic pump' on forward blood flow. Gazmuri, *et al.* reported that increasing respiratory rate and tidal volume up to a minute-volume 10-fold higher than currently recommended had no adverse effects on coronary perfusion pressure during resuscitation in swine [6]. Cordioli, *et al.* reported that positive airway pressure associated with continuous flow insufflation of oxygen limits the loss in lung volume, enhances chest compression-induced positive intrathoracic pressure, maintains negative intrathoracic pressure during decompression, and generates more alveolar ventilation [3]. This rise in intrathoracic pressure is transmitted equally to all intrathoracic structures and squeezes out the pulmonary vessels, which increases forward blood flow, arterial oxygen partial pressure, and aortic pressure [7-9]. However, the evidence so far is inconclusive regarding the major responsible mechanism in compression-related blood flow; although both the 'cardiac pump' and 'thoracic pump' have a key role, the effect of each mechanism is highly depended on other resuscitation parameters, such as the compression depth and/or positive pressure ventilation [10]. Echocardiographic observations have shown that both pumps are operative [11,12], while the pump mechanism changes over the course of prolonged CPR; cardiac effect is an essential part of the pump at the beginning of cardiac arrest, but it fades with time, making the 'thoracic pump' the dominate mechanism after prolonged CPR [13].

As the net effects of positive pressure insufflation and chest compression are not well characterized, the balance between circulation and respiration during CPR is obviously critical and achieving the correct balance between too little and too much ventilation is of major importance for optimizing survival [14]. Theoretically, there must be an intrathoracic pressure limit at which the effect of 'thoracic pump' should be maximal. Above this limit, intrathoracic pressure would be deleterious, while under this limit, ventilation may not provide adequate blood oxygenation, increasing pulmonary vascular resistance and impairing pulmonary and systemic blood flow. Until now, there is no one-size-fits-all ventilation strategy for all cardiac arrest patients and it is difficult to obtain prospective clinical data related to this issue.

Nevertheless, the aforementioned support the findings of a recent prospective observational study which reported an association between mean airway pressure and outcome of CPR in mechanically ventilated patients, with a value of 42.5 mbar being associated with return of spontaneous circulation [15]. The strict inclusion criteria in this study clearly indicate that the effects of 'thoracic pump' cannot be de-emphasized or ignored. In patients with preserved physiology, i.e. those who receive immediate bystander CPR, the use of an automated ventilator may allow for the optimal exploitation of positive intrathoracic pressure while avoiding the common side effects observed when ventilating with a self-inflating bag. Patients with preserved physiology are those in whom ischemic contracture and relaxation of the airway smooth muscles have not yet ensued [16], allowing for the full exploitation of both pumps.

Until we have robust data, we should not de-emphasize or abandon intubation during adult cardiac arrest. Moreover, as patients with return of spontaneous circulation are admitted to the intensive care unit, potential complications may arise that affect survival rates. Therefore, prognosis involves consideration of many parameters and cannot be easily attributed to intubation per se. As proper timing of compression and ventilation seems to be the key for improving the circulation, new approaches to CPR should focus on improving perfusion by ventilation patterns associated with the effects of chest compressions.

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