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Airway Pressure Monitoring May Improve Small Airway Flow, Hemodynamics, and Tissue Oxygenation

To the Editor:

We would like to congratulate Scales and Kavanagh (1) for their insightful comments reported in the editorial accompanying the study by Grieco and colleagues (2). It is true that research on resuscitation made early gains, but recent progress has been slow because of the dispersion of researchers to aspects other than elucidating the physiology and pathophysiology of cardiac arrest and resuscitation.

Although our understanding of the interaction between chest compression and mechanical ventilation remains limited, expert opinions will probably continue to rely on flawed studies that neither report nor take into consideration, when interpreting the results, the method of postintubation ventilation (self-inflating bag or ventilator), while suggesting simultaneously that early intubation during cardiopulmonary resuscitation (CPR) does not improve, or even decreases, survival (3). Ventilation with a self-inflating bag in intubated patients usually results in excessive ventilation volume and rate, thus aggravating oxygenation and hemodynamics, and surprisingly, it continues to be a major limitation in resuscitation studies.

Cordioli and colleagues (4) demonstrated that ventilation during CPR by using currently recommended chest compression rates takes place entirely below FRC and is associated with negative intrathoracic pressures during decompression. Although the thoracic pump theory is not widely accepted among the resuscitation community, the study of Cordioli and colleagues suggests that both cardiac pump and thoracic pump have a role in forward blood flow and tissue oxygenation. In this context, the study by Grieco and colleagues (2) strengthens the evidence-based notion that the harmony between circulation and ventilation during CPR is critical. Achieving the correct balance between too little and too much ventilation is of major importance for optimizing survival, and theoretically, there must be an intrathoracic pressure limit at which the effect of a thoracic pump should be maximal. Above this limit, intrathoracic pressure would be deleterious, and under this limit, ventilation may not provide adequate blood oxygenation because of small airway closure, increasing pulmonary vascular resistance and impairing pulmonary and systemic blood flow.

Our group has recently shown 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 (5). In our patients, simultaneous positive pressure ventilation in time with each chest compression prevented a loss of intrathoracic pressure via the airway, and probably kept the small airways open. In this study, we found no difference in end-tidal carbon dioxide between survivors and nonsurvivors, probably because of the maintenance of flow in small airways and the improvement in minute-volume ventilation during CPR (6). Of note, the rise in intrathoracic pressure in mechanically ventilated patients undergoing CPR 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. Moreover, as hemodynamics may be aggravated in prolonged CPR because of vascular tone deterioration, the pressing effect of positive pressure ventilation and increased intrathoracic pressure on aortic wall may increase aortic resistance and retrograde volume loading, therefore enhancing the compression-related blood flow (5).

Collectively, the study by Grieco and colleagues and our findings highlight the favorable effects of the thoracic pump and the importance of intubation and mechanical ventilation in patients with cardiac arrest, supporting our deduction that the interplay between ventilation and chest compression during CPR is a key point to optimize outcomes (6). As proper timing of compression and ventilation seems to be the key for improving the circulation, the focus of the resuscitation community must immediately return to the elucidation of the physiology and pathophysiology of cardiac arrest and resuscitation. ■

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Reply to Chalkias and Xanthos

To the Editor:

We are very grateful to Dr. Chalkias and Dr. Xanthos for their thoughtful comments regarding the description of the phenomenon of intrathoracic airway closure reported in the *Journal* (1).

These authors recently reported an impressive series of 300 out-of-hospital patients with cardiac arrest who were resuscitated with a strategy combining rapid intubation, continuous chest compression (CC), and positive pressure ventilation delivered via a ventilator (2). The unexpectedly high percentage of return of spontaneous circulation reported in this study was significantly associated with highest mean airway pressure (Paw) measured after 3 minutes of resuscitation via an external monitor. CO_2 measured via a mainstream monitor was similar between survivors and nonsurvivors. The authors concluded that a mean Paw above 42.5 mbar was associated with a higher chance of return of spontaneous circulation.

Interestingly, the apparent negative effect of a low mean Paw during CC could be related to (or associated with) the intrathoracic airway closure we recently reported (1). In fact, the transmission of pressure generated by CC at the airway opening is limited or absent in the case of intrathoracic airway closure. Conversely, the expected beneficial effect of positive pressure delivered by the ventilator (which refers to the thoracic pump effect) can be effective only if the positive airway pressure applied at the airway opening is transmitted to the intrathoracic compartment, although this transmission will be limited by intrathoracic airway closure. The methodological difficulty of capturing the highest value of CO_2 that seems the best surrogate of alveolar CO_2 during resuscitation limits the interpretation of the lack of difference reported in their study.

Therefore, if we accept that the association between a mean Paw below 42.5 mbar and a worse prognosis reported in the study of Chalkias and colleagues might be explained by intrathoracic airway closure, several different mechanisms could still be at play. First, intrathoracic airway closure could simply be a marker of poor prognosis that also limits transmission of pressure generated by CC at the airway opening, making the calculated mean airway pressure at the mouth lower. Second, intrathoracic airway closure may have impaired the transmission of positive pressure generated by ventilation to the intrathoracic compartment, thus limiting its expected beneficial effect on the thoracic pump effect. By overcoming intrathoracic airway closure, higher mean airway pressure could be beneficial on both circulation and ventilation. Finally, one cannot exclude that the lower mean Paw associated with the lower chance of return of spontaneous circulation in the abovementioned study could be simply the reflection of less effective CC, independent of intrathoracic airway closure. These fascinating physiological discussions deserve additional observations to better understand the mechanisms at play and the evolution of airway closure along the time of resuscitation. The use of the capnogram during CC, based on the description of Grieco and colleagues, may permit us to adapt ventilator settings according to intrathoracic airway closure to balance both the beneficial and potential harmful effects of positive airway pressure during resuscitation. ■

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