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Invasive bacterial infections in childhood



We have greatly enjoyed reading the recently published article by Niloufar Paydar-Darian and colleagues [1]. In this retrospective study, the authors included 1460 previously healthy febrile children <21 years of age, who presented to an emergency department (ED) for evaluation of fever of <14 days' duration, who had both C-reactive protein and erythrocyte sedimentation rate obtained. Of the 1460 eligible ED encounters, the median patient age was 5.3 years and 762 (50.4%) were hospitalized. The authors showed that none of the children with an invasive bacterial infection had both a normal C-reactive protein and erythrocyte sedimentation rate result, suggesting these tests could be used to reliably exclude invasive infection before results of bacterial culture become available. However, we think that the hospitalization rate of 50.4% is quite high for previously healthy febrile children most of whom have viral infections in real-life practice. Therefore, we think that the authors should detail how did they avoided selection bias which is an error in not ensuring random sampling.

Funda Sungur Biteker, MD

Yatağan State Hospital, Department of Infectious Diseases and Clinical Microbiology, Turkey

Cem Çil, MD

Oğuzhan Çelik, MD*

Eda Özlek, MD

Bülent Özlek, MD

Aysel Gökçek, MD

Volkan Doğan, MD, Assist Prof

Muğla University, Faculty of Medicine, Department of Cardiology, Turkey

Corresponding author at: Muğla Sıtkı Kocman Üniversitesi Tıp Fakültesi, Orhaniye Mah. Haluk Özsoy Cad., 48000 Muğla, Turkey.

E-mail address: droguzhancelik@hotmail.com (O. Çelik).

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Low ventilation associated with chest compression, an old observation that requires new physiological interpretation



We read with great interest the paper of Robyn McDannold et al. about the quantification of ventilation volumes produced by chest compressions (CCs) in a convenience sample of 21 out-of-hospital cardiac arrest (OHCA) intubated patients. They assured CCs quality (accelerometer-based technology) and took a sample of delivered ventilations derived from flow sensor placed at the endotracheal tube [1].

The research question addressed in this work is whether CCs alone provide meaningful passive ventilation, which could support the low requirement for additional ventilation during the early phase of resuscitation. Median delivered tidal volume was 7.5 mL, with a maximal at 45.8 mL, remaining significantly below the average of previously estimated anatomical dead space (<120 mL), in cardiac arrest patients [2]. Interestingly, authors hypothesized that the specific anatomical features of the human thorax (i.e.; small sterno-vertebral axis) may explain the low amount of ventilation passively delivered by CCs, which is significantly different from what was quantified in animals that present a different thorax anatomy (large sterno-vertebral axis).

The presented data are consistent with the initial description by Safar et al., who reported that CCs produce an average tidal volume of 156 mL in paralyzed healthy subjects with intact circulation, but no ventilation in patients with cardiac arrest [3]. At that time, this was ascribed to airway resistance after aspiration, lung volumes loss or lung edema, leading to decreased lung compliance [4].

The concept of “intrathoracic airway closure phenomenon”, which is associated to lung volume reduction during CCs, was observed in a series of OHCA patients and may alternatively explain these observations [5]. In case of airway closure, the negative alveolar pressure generated by chest release is not transmitted to airway opening and no inspiratory flow is generated. Based on a human cadaver model, the same group showed that the application of positive pressure at the airway opening, set just above the closing pressure of the airways, increases alveolar ventilation generated during CCs [6]. More recently, the systematic analysis of CCs related oscillations magnitude observed in capnogram, obtained immediately after intubation of OHCA patients, permitted to detect intrathoracic airway closure and indirectly reflect the amount of alveolar ventilation [7]. The derived airway opening index (AOI) from oscillating CO₂ varied in that series from 0% (no oscillation indicating closure) to 100% (full oscillation indicating airway patency), with a median value around 50%. These observations are consistent with the proportion of 58% non-oscillating capnograms reported in a recent series of OHCA [8]. In accompanying bench and cadaver models experiments, high AOI reflecting airways patency was associated with a reduction in CO₂ due to the sole effect of CCs, suggesting an effective contribution of fresh gas on alveolar ventilation despite low CCs related volumes; on the opposite, no alveolar ventilation was produced during CCs if AOI was below 25%, which occurred in 20% of OHCA patients [7].

The presented results by McDannold et al. support the hypothesis that the geometry of human thorax is likely associated with lung volume reduction and intrathoracic airway closure during CCs; it may explain the low amount of ventilation generated in a substantial proportion of OHCA patients after the initial phase of resuscitation [1]. Looking at the Fig. 1, we noticed that CCs produce very minimal inspiratory flow during the decompression phase (upper part) when compression phases (lower part) reach higher peak flows. The flow tracing obtained in a cadaver model during manual CCs exhibits very similar airflow limitation during the decompression phase, despite a complete release of the positive intrathoracic pressure generated by CCs, which is prevented by adding some positive pressure above the closing pressure (Figure) [6]. It strongly suggests that the intrathoracic airway closure is the main determinant of flow limitation during CCs.

In addition, it is worth to note that in the set of measurements reported by McDannold, volume could reach 50 mL, which correspond to observations in case of high AOI and fully patent airways. Hence this series probably mixed patients with low and high AOI, meaning different levels of closure at different phase of resuscitation. As suggested by Idris, in a swine model, the decline of ventilation during CCs may be time-dependent [9].

In conclusion, the low tidal volumes generated by CCs reported after initial resuscitation, in different series of OHCA patients, interestingly comfort previous observations; new physiological data show that it is related to intrathoracic airway closure, likely occurring when the respiratory system (thorax/lung) compliance and volume have been

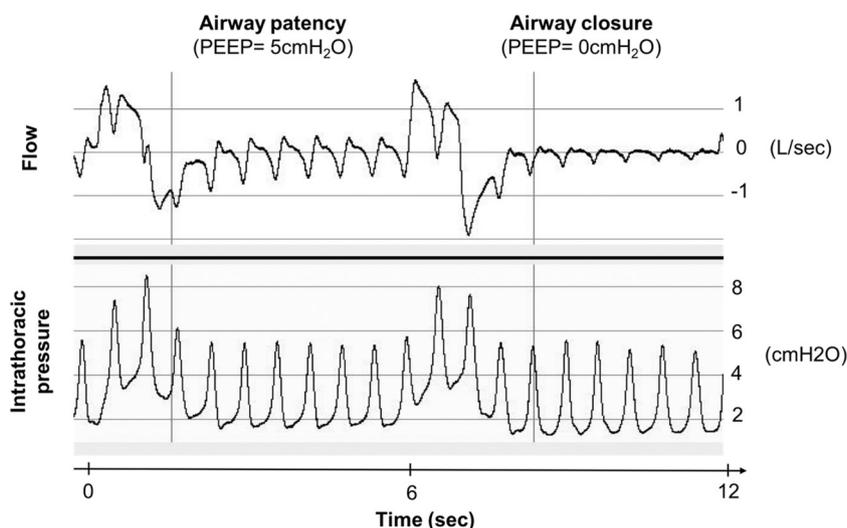


Fig. 1. Passive flow measured at the airway opening and intrathoracic pressure variation measured through an esophageal balloon, during CCs. It shows airflow limitation during the decompression phase, when positive end-expiratory pressure (PEEP) is changed from 5 to 0 cm H₂O, while intrathoracic pressure still oscillates.

decreasing under prolonged resuscitation. The analysis of CCs related oscillations on the capnogram might be a useful signal in order to detect the intrathoracic airway closure.

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Emmanuel Charbonney

University of Montreal, Department of Medicine, Montreal, Canada

Corresponding author at: Hôpital du Sacré-Coeur de Montréal, 5400

boul. Gouin Ouest, Montréal, Québec H4J 1C5, Canada.

E-mail address: emmanuel.charbonney@umontreal.ca.

Dominique Savary

SAMU74, Department of Emergency, General Hospital of Annecy, Annecy,

France

E-mail address: dsavary@ch-annecygenevois.fr.

Bilal Badat

Air Liquide Medical Systems, Antony, France

E-mail address: bilal.badat@airliquide.com.

Domenico L. Grieco

Universita Cattolica del Sacro Cuore Sede di Roma, Department of
Anesthesiology and Intensive Care Medicine, Rome, Italy

Jean-Christophe Richard

SAMU74, Department of Emergency, General Hospital of Annecy, Annecy,
France

INSERM UMR 1066, Créteil, France

the CAVIAR group

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