Correspondence

Capnometry in children with bronchiolitis: A pathophysiological point of view

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To the Editor

Bronchiolitis is the most common lower respiratory tract infection among infants and young children [1]. Nowadays oxygenation and clinical assessment are used to decide on hospitalization.

Jacob et al. recently explored the role of capnography upon arrival to the Emergency Department and concluded that end-tidal carbon dioxide (EtCO2) does not predict neither hospital admission nor discharge eligibility [2].

We would like to take a cue from this praiseworthy study to analyse the relationship between EtCO2 and bronchiolitis from a pathophysiological point of view.

Bronchiolitis leads to submucosal oedema and leukocytes infiltration, dysfunction of respiratory cilia, excessive mucus production, necrosis and sloughing of the respiratory epithelial cells and bronchoconstriction. Each of these contribute to airflow obstruction through the small/distal airways producing air trapping and lung hyperinflation.

On the other hand, hyperinflation and atelectasis shift tidal breathing to a less compliant portion of the pressure-volume curve making bronchiolitis a restrictive disease.

Enhanced airway resistance and reduced lung compliance increase the work of breathing (WOB).

Supra-normal WOB may produce muscle exhaustion and hypoventilation.

Hypoventilation translates into hypercapnia and augmented EtCO2.

Hyperinflation of some areas, hypoventilation of others and the compression of small arteries and capillaries by inflammatory lymphoid follicular aggregates create ventilation-perfusion mismatch.

This inequality may translate into intra-pulmonary shunt (low ventilation-perfusion ratio) or in alveolar dead-space (high ventilation-perfusion ratio) [3].

Depending on which aspects prevail, the children will be hypoxemic (shunt) or hypercapnic (dead space) or both.

Capnometry allows the monitoring of the partial pressure of CO2 (pCO2) in the respiratory gases, EtCO2 reflects the alveolar CO2. Alveolar CO2 reflects arterial pCO2 only when alveolar dead space is physiological. The presence of dead space ventilation increases arterial pCO2 and does not affect EtCO2.

Capnometry alone cannot detect death space ventilation: therefore, in bronchiolitis, it adds little to standard assessment. Measurement of EtCO2, or better measurement of the partial pressure of CO2 in mixed expired gas, and arterial pCO2, allowing rapid measurement of dead space, would enable to stratify children based on lung involvement.

In acute respiratory distress syndrome, pulmonary dead-space correlates with death [4].

Statements

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References

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