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Editorial

Atelectasis: The Price of Positive Intra-Thoracic Pressure

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Editorial

During the 1952 Copenhagen polio epidemic, Bjørn Ibsen's use of Positive Pressure Ventilation (PPV) via tracheostomy solved his shortage of negative-pressure ventilators. The simplicity of applying an endotracheal tube-with full access to the thorax and abdomen-proved advantageous during surgery and intensive care. Ibsen's dramatic success rapidly transformed respiratory care worldwide. This triumph, coupled with aversion to the old devices, prompted an almost universal, uncritical shift to PPV. In this transition, the knowledge that Negative Pressure Ventilation (NPV) prevents and reverses atelectasis was largely forgotten. The history as described in reference 1 is worth reading, but the title is wrong in its suggestion that Ibsen's success led to the invention of positive pressure ventilation, for Dräger patented their Pulmotor in 1907 and already thousands of these instruments were active for ventilation at home before the first world war [1]. The Dräger Pulmomat followed in 1930-40 as its successor and from 1952 the next successor, Dräger Poliomat, was used in several countries to ventilate Polio patients [2].

Recently, Iba et al., in their Editorial in Intensive Care Medicine recognized atelectasis as a ventilator-associated event and its incidence is accepted to be high during Positive Pressure Ventilation (PPV) [3]. This is confirmed by clinical imaging and perioperative observations and is tacitly acknowledged in current guidelines. Indeed, lung-protective ventilation is now recommended even for healthy lungs during general anaesthesia, with low tidal volumes and plateau pressures to prevent ventilator-induced lung injury. Yet, immediately after extubation, patients are encouraged to take deep breaths to "re-expand" their lungs-a clear admission that atelectasis is expected after PPV, even in healthy lungs during short procedures.

A central misunderstanding concerns the interpretation of airway/alveolar pressure. There is a fundamental difference between an alveolar pressure generated by a ventilator and the same pressure produced by a brass musician. In the latter case, pressure results from coordinated contraction of expiratory intercostals and abdominal muscles, generating an equal counterpressure in the thorax and abdomen. The lung is therefore not stressed by the pressure difference. In contrast, during PPV the same airway pressure acts upon a passive thorax and relaxed diaphragm, transmitting the full pressure to lung tissue. As Dreyfuss showed, when thoracic expansion was prevented by encasing the animal's chest in plaster, no lung injury occurred-even at high airway pressures-because the damaging transpulmonary pressure difference could not develop. However, it is clear that the limitation of tidal volume as prescribed by the "Lung Protective Strategies" is based on the volutrauma concept of Dreyfuss [4].

A further fundamental distinction between PPV and NPV lies in how air is moved: blowing versus sucking. Although transpulmonary pressure ultimately determines lung inflation, the pathway by which it is established has profound mechanical

consequences. Under PPV, high airway pressures are required to overcome regional airway closure and non-uniform compliance. In contrast, during NPV the thoracic pressure is lowered externally, allowing airways to open more synchronously and producing a larger effective volume change at a much lower driving pressure. Klassen, et al., demonstrated that in an excised porcine lung for the same tidal volume, driving pressure during PPV was roughly twice that during NPV, while air leakage from a visceral leak was fivefold greater under NPV [5]. This highlights how the pattern of pressure application-within the airway or around the thorax-determines the homogeneity of ventilation.

Klassen's experiments clearly demonstrate the phenomenon of airway closure. This phenomenon has led to another misconception. Generally, it is accepted that airway pressure equals alveolar pressure and transpulmonary pressure is defined as airway pressure minus pleural pressure. This difference is sometimes reported as a negative value, but this unrealistic value only proves that there is no connection between the alveolus and the airway, described elsewhere as airway closure. Full airway closure is described by Behazin in the obese patient and is characterized by the Airway Opening Pressure (AOP): raising airway pressure will not result in flow into the lung before it reaches the AOP [6].

Recognizing atelectasis and airway closure as almost inevitable consequences of PPV should prompt renewed interest in the underlying mechanics [7,8]. High intra-thoracic pressures compress dependent regions and small airways, predisposing to collapse, "baby lung", edema and infection. Revisiting the physiological advantages of NPV may thus clarify the origins of atelectasis and airway closure and open new preventive perspectives.

Keywords: Positive Pressure Ventilation; Negative Pressure Ventilation; Airway Opening Pressure

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Conflict of Interest

Apart from their university/hospital affiliations, both authors (JvE and JPM) are members of the Exovent Developing Group, which is a UK registered charity (no. 1189967:10 Queen St Pl, London EC4R 1BE, UK.) https://exovent.org None of the authors is receiving any honoraria from this charity. On behalf of all authors, the corresponding author states that there is no conflict of interest. JPM reports payments or honoraria from Medec international outside the submitted work. JPM is a member of a Belgium registered charity ESPCOP vzw (no. 1030137921: Beukenpark 19 9930 Lievegem, BE). https://espcop.eu and co-founder of MT4L https://mt4l.com, a company developing not related airway products.

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Authors' Contributions

All authors have contributed equally to this work and have reviewed and approved the final manuscript for publication.

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