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CORRESPONDENCE

Non-ventilated lung airway occlusion during one-lung ventilation: a need for further research?

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To the Editor,

In the process of showing that the non-ventilated lung will collapse faster if its airway remains intentionally clamped off prior to pleural opening, Somma *et al*. seem to have introduced an "additional step" to the clinical management of one-lung ventilation (OLV) for thoracoscopic surgery.[1] Although details of the clinically relevant step are somewhat lost in a complicated Methodology section, the "study timeline" in Fig. 4 and the description of the "lung deflation optimization technique" in Fig. 7 clearly reveal that the airway of the non-ventilated lung is to be opened to air (for "one minute maximum") immediately following the initiation of OLV.[1] The proffered reason is a need during this first minute to detect "inadequate lung isolation" using spirometry loop monitoring,[1] and the reader will realize it is "gas leakage past the bronchial cuff" that must be detected. If the total volume of gas leakage were to match or exceed the "gaseous influx"[2] over ten minutes of airway occlusion, the pressure within the non-ventilated lung would not fall and subsequent lung collapse would not be facilitated. Extrapolating from the findings in a "gaseous influx" study by Moreault *et al*.,[2] this could happen with leakage of as little as 5 mL for each mechanical inspiration.

Not mentioned in the Somma *et al.* report,[1] nor in a similar Zhang *et al*. report supporting airway occlusion,[3] is the possibility that gas leakage past the bronchial cuff could also result in a life-threatening clinical emergency. If the volume of leakage over the period of airway occlusion were to be greater than the "gaseous influx", the non-ventilated lung will become progressively hyperinflated, possibly quite quickly with a larger bronchial cuff leak. Over a ten-minute period of airway occlusion, a small 20-mL leak at a respiratory rate of 15 min^{-1} would result in unilateral hyperinflation, with a likely greater than 2.0-L increase in the obstructed lung's volume. It is thus conceivable that even a moderately large leak could create a "pneumothorax-like" clinical picture possibly like that reported in patients with too-rapid insufflation of carbon dioxide into the pleural space,[4] and possibly also with similar (and similarly uncertain) underlying pathophysiologic mechanisms.[4]

What an opportunity for an interesting study on un-researched, hypothesized "acute unilateral pulmonary hyperinflation"! In a study looking at the initial clinical manifestation of tension pneumothorax in a mechanically ventilated swine model, 100-mL increments of air were introduced into the pleural space every four to five minutes via a surgically placed air-tight balloon catheter[5]: the study found that significant hypoxemia occurred early and preceded hypotension.[5] A similar model could be created for OLV with bronchial occlusion and bronchial cuff leakage. If the volume of every tidal "breath" were reduced by say 25 mL, and an equal volume injected into the airway of the non-ventilated lung distal to the bronchial

This letter is accompanied by a reply. Please see Can J Anesth 2021; this issue.

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occlusion, over a ten-minute period and a respiratory rate of 15 breaths per minute what would be the presenting clinical features? In clinical practice, if it can happen, it will!

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