Pneumoperitoneum-induced very high airway opening pressure

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Argomento: Anestesia generale

Background. Airway closure is a lack of communication between proximal airways and alveoli due to airway collapse. Under this condition, alveolar pressure differs from airway pressure at end-expiration also in case of no flow, and tidal inflation starts only after a critical airway opening pressure (AOP) is overcome.

Its occurrence has been reported during laparoscopy, but pneumoperitoneum seemed not to affect AOP and esophageal pressure. We report a case series of patients who showed airway closure during surgery with AOP enormously raised by pneumoperitoneum in the Trendelenburg position.

Methods. Among the procedures of a clinical study (NCT03157479), morbidly obese patients receiving gynaecological surgery receive volume-controlled ventilation and undergo respiratory mechanics and gas dilution-derived lung volume assessment both before and after pneumoperitoneum, in the supine and Trendelenburg position respectively.

Results. Four of 25 studied patients showed airway closure after intubation (AOP range before pneumoperitoneum 7-11 cmH2O), with AOP significantly increased after pneumoperitoneum (AOP range after pneumoperitoneum 24-28 cmH2O), independently from intrinsic PEEP. Pneumoperitoneum-induced increase in AOP was accompanied by a similar change in esophageal pressure, so that transalveolar pressure and end-expiratory lung volume remained unchanged. When one patient was switched to pressure-controlled ventilation to perform a recruitment maneuver by means of step-by-step PEEP increase, no tidal ventilation was delivered until inspiratory pressure overcame AOP.

Conclusions. To our knowledge, this is the first report showing that surgical pneumoperitoneum can relevantly worsen airway closure, enormously increasing AOP and alveolar pressure at end-expiration: this yields misinterpretation of respiratory mechanics and end-expiratory alveolar pressure, which is significantly higher than measured PEEP and, ultimately, close to AOP.

Airway closure can be missed at the bedside, while this phenomenon generates a pressure threshold to inflate the lung that can reach high values. This raises concerns about the safety of pressure-controlled modes in this specific context.

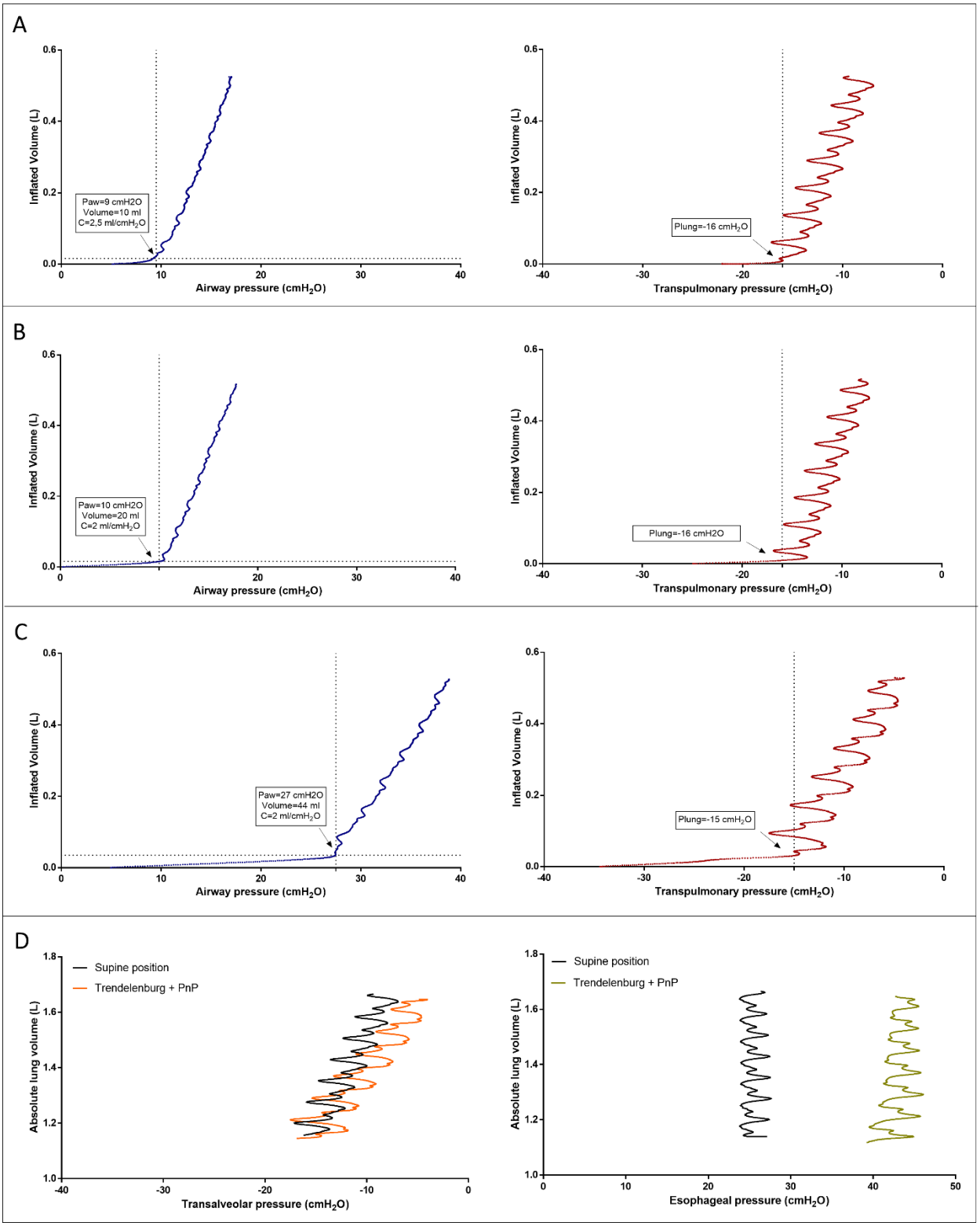

Figure 1-Tracings from one representative patient

Panel A, B, C, pictures on the left:

Airway Pressure-Volume curves during low-flow inflation before pneumoperitoneum in the supine position (starting from zero end-expiratory pressure [B] and PEEP=5 cmH2O [A]) and after pneumoperitoneum in the Trendelenburg position (starting from PEEP=5 cmH2O [C]).

In the box, we report the pressure and volume changes along with the corresponding compliance between start of inflation and AOP. Note that this compliance suggests gas compression in an occluded circuit and, hence, complete airway closure.

Panel A-B-C, pictures on the right:

Transpulmonary Pressure-Volume curves during low-flow inflation before pneumoperitoneum in the supine position (starting from zero end-expiratory pressure [B] and PEEP=5 cmH2O [A]) and after pneumoperitoneum in the Trendelenburg position (starting from PEEP=5 cmH2O [C]).

Transpulmonary pressure at which AOP is overcome (reported in the box) remains constant after pneumoperitoneum induction, as AOP increases consistently with the raise in oesophageal pressure.

Panel D

Multiple Pressure-Volume curves of the transalveolar pressure (i.e. transpulmonary pressure with patent airways, picture on the left) and the oesophageal pressure (picture on the right) recorded during low-flow inflation and plotted starting from the measured EELV, before pneumoperitoneum in the supine position and after pneumoperitoneum in the Trendelenburg position respectively.

The absence of change in the Transalveolar pressure-volume curve indicates that lung recruitment is unmodified, while the oesophageal pressure-volume curves show a shift that describes the change in the conditions of the chest wall after pneumoperitoneum: end-expiratory esophageal pressure is raised by 18 cmH_2O and chest wall compliance is reduced.

Acronyms:

Plung=transpulmonary pressure

C=compliance

Pnp=pneumoperitoneum