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# Airway opening pressure in an obese ARDS patient: just the start of airway recruitment



Shinshu Katayama<sup>1\*</sup>, Giorgio Antonio lotti<sup>2</sup> and Ken Tonai<sup>1</sup>

To the Editor,

Small airway closure, as may occur in morbidly obese patients and acute respiratory distress syndrome (ARDS) patients, is supposed to completely interrupt the communication between the proximal airway and some alveoli that remain inflated. In this condition, the total positive end-expiratory pressure (PEEP<sub>TOT</sub>) assessed by end-expiratory occlusion has frequently been found to underestimate the real alveolar pressure. Measurement of the airway opening pressure (AOP) on a static pressure–volume (PV) curve obtained by low-flow inflation was proposed as a better alternative, enabling the correct calculation of driving pressure and the convenient setting of PEEP to at least the level of AOP [1].

In this case report, we describe a morbidly obese ARDS patient with an AOP of 12 cmH<sub>2</sub>O as measured by a slow inflation maneuver, but also with airway closure at 16 cmH<sub>2</sub>O as confirmed by respiratory mechanics' measurements and four-dimensional computed tomography (4D-CT) during dynamic changes of PEEP between 5 and 16 cmH<sub>2</sub>O.

The 51-year old female, morbidly obese patient (body mass index 56.2 kg/m<sup>2</sup> with predicted body weight 47.9 kg) was intubated for septic shock and ARDS. After stabilization, as we started to de-escalate the ventilatory

<sup>1</sup> Division of Intensive Care, Department of Anesthesiology and Intensive Care Medicine, Jichi Medical University School of Medicine, 3311-1, Yakushiji, Shimotsuke, Tochigi 329-0498, Japan support, the reduction in PEEP led to a major increase in the patient's inspiratory effort. A static inflation PV curve, which was obtained using the slow pressure-ramp method at 2 cmH<sub>2</sub>O/s [2] on a HAMILTON-G5 ventilator (Hamilton Medical, Bonaduz, Switzerland), showed an AOP of 12 cmH<sub>2</sub>O (Fig. 1). A 4D-CT at PEEP levels of 5 and 16 cmH<sub>2</sub>O showed no relevant difference in endexpiratory lung volume (EELV) or regions of ventilation between the two levels. There was no difference in peak inspiratory pressure (PIP), plateau pressure (Pplat) or esophageal pressure (Pes) between the two PEEP levels, while PEEP<sub>TOT</sub> was 15 and 18 cmH<sub>2</sub>O, respectively (Fig. 1 and Table 1).

# Discussion

In this patient, an AOP of 12 cmH<sub>2</sub>O measured on the static PV curve was lower than the PEEP<sub>TOT</sub> measured by end-expiratory occlusion. In addition, a step change in PEEP below 16 cmH<sub>2</sub>O was not associated with any relevant change in EELV. Based on these results, we would make the following remarks.

# Unchanged EELV and end-expiratory Pes (Pes,ee) despite dynamic changes in PEEP

A change in EELV must lead to a change in Pes,ee proportional to the chest-wall elastance. In our case, the absence of any change in EELV at PEEP levels between 16 and 5 cmH<sub>2</sub>O as assessed by 4D-CT is consistent with the lack of change observed in Pes,ee and the lack of any relevant temporary imbalance between inspiratory and expiratory tidal volumes (Fig. 1). The static PV curve showed an AOP of 12 cmH<sub>2</sub>O and a volume rise of less than 50 ml at an inflation pressure of 16 cmH<sub>2</sub>O (Fig. 1). We hypothesize that although some airways opened at 12



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<sup>\*</sup>Correspondence:

Shinshu Katayama

shinsyu\_k@jichi.ac.jp

<sup>&</sup>lt;sup>2</sup> Hamilton Medical AG, Bonaduz, Switzerland



**Fig. 1** Left panel: Respiratory system static pressure–volume loop by slow inflation-deflation with pressure ramps of 2 cmH<sub>2</sub>O/s. The arrow indicates the airway opening pressure (AOP). Right panel: Airway pressure (Paw), esophageal pressure (Pes), airflow, volume change and capnography during PEEP steps from 16 down to 5 cmH<sub>2</sub>O and back

Table 1 Respiratory mechanics data at PEEP 5 and 16 cmH <sub>2</sub> O		
	PEEP 5 cmH <sub>2</sub> O	PEEP 16 cmH <sub>2</sub> O
PIP, cmH <sub>2</sub> O	33	33
Pplat, cmH <sub>2</sub> O	29	29
PEEP <sub>TOT</sub> , cmH <sub>2</sub> O	15	18
PEEP <sub>1</sub> , cmH <sub>2</sub> O	10	2
∆P, cmH <sub>2</sub> O	14	11
V <sub>T</sub> , mL	300	300
Pes,ei, cmH <sub>2</sub> O	25	25
Pes,ee, cmH <sub>2</sub> O	20	20
C <sub>TOT</sub> , mL/cmH <sub>2</sub> O	21	27
Cw, mL/cmH <sub>2</sub> O	60	60
Clung, mL/cmH <sub>2</sub> O	33	50
Lung volume,ee, mL	713	730
Lung volume,ei, mL	995	1011
ΔEELV, mL	-	16

Abbreviations: Clung lung compliance,  $C_{TOT}$  Respiratory system compliance (total), *Cw* chest wall compliance (chest wall), *EELV* End-expiratory lung volume, *Lung volume*, *ee* End-expiratory lung volume, *Lung volume*, *ei* End-inspiratory lung volume, *PEEP*, Intrinsic PEEP, *PEEP*<sub>TOT</sub> Total PEEP, *PIP* Peak inspiratory pressure, *Pplat* Plateau pressure, *AP* Driving pressure, *Pes*, *ee* End-expiratory esophageal pressure, *Pes*, *ei* End-inspiratory esophageal pressure, *V*<sub>T</sub> Tidal volume Gas content volumes were calculated from computed tomography (CT) numbers analyzed from -1000 Hounsfield unit (HU) to 0 HU in each voxel [3]. Lung volume was calculated using the following formula: Gas content volume = SUM (-CT number / 1000 \* voxel volume) cmH<sub>2</sub>O, the recruitment of recruitable airways was not complete up to at least 16 cmH<sub>2</sub>O.

# AOP from static inflation PV curves with the slow pressure-ramp method

On a static PV curve obtained using low-flow inflation at 5 l/min, AOP was described as the elastic pressure corresponding to the sharp bend where the actual curve exceeds the PV curve predicted by the ventilator circuit by 4 ml [1]. The slow pressure-ramp method [2] is based on pressure and volume measurements made at the Y-piece of the ventilator circuit. By definition, this kind of measurement excludes any mechanical effect caused by the compliance of the ventilator circuit. Therefore, AOP can easily be identified as the sharp bend where the volume increases and exceeds the baseline by 4 ml, after an initial segment with increasing pressure, but no change in volume (Fig. 1).

# AOP, PEEPTOT and PEEP setting

In our case, AOP was lower than  $PEEP_{TOT}$ , although it has only been described previously as equal to or higher than  $PEEP_{TOT}$  [1]. We found a curvilinear increase in the PV curve slope above the AOP point. This feature of the inflation PV curve may not only depend on different levels of alveolar opening, but also on different levels of airway opening within the inhomogeneous lungs. We hypothesize that the AOP we detected on the static PV curve corresponded to the minimum threshold for communicating with some gas-filled but separated lung areas, while the subsequent curvature was due, at least in part, to progressive communication with the areas requiring more pressure. A PEEP<sub>TOT</sub> higher than AOP may be indicative of this condition of multiple pressure levels for airway opening that are above the lower one as assessed on the PV curve.

In conclusion, we report the new finding that, at least in morbidly obese patients,  $PEEP_{TOT}$  can be higher than AOP. In patients susceptible to small airway closure, both parameters should help us better understand the condition. However, the question remains as to which is the most relevant for calculating driving pressure or making decisions about the PEEP setting. This has never been investigated. The matter may be not so simple, especially if we consider that the recruitment of closed small airways is most likely a phenomenon stratified within a pressure range, rather than limited to a single pressure level.

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#### **Prior publication**

The images in this case report have not been previously published.

#### Authors' contributions

The author(s) read and approved the final manuscript.

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## Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

#### Declarations

# Ethics approval and consent to participate

This study was approved by the Jichi Medical University Hospital Bioethics Committee for Clinical Research, approval number A20-103. Informed consent was obtained from the patient.

#### **Consent for publication**

The patient consented to the submission of this case report to the journal.

#### **Competing interests**

GAI and SK are consultants for Hamilton Medical AG (Switzerland). SK was supported by the Japan Society for the Promotion of Science.

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