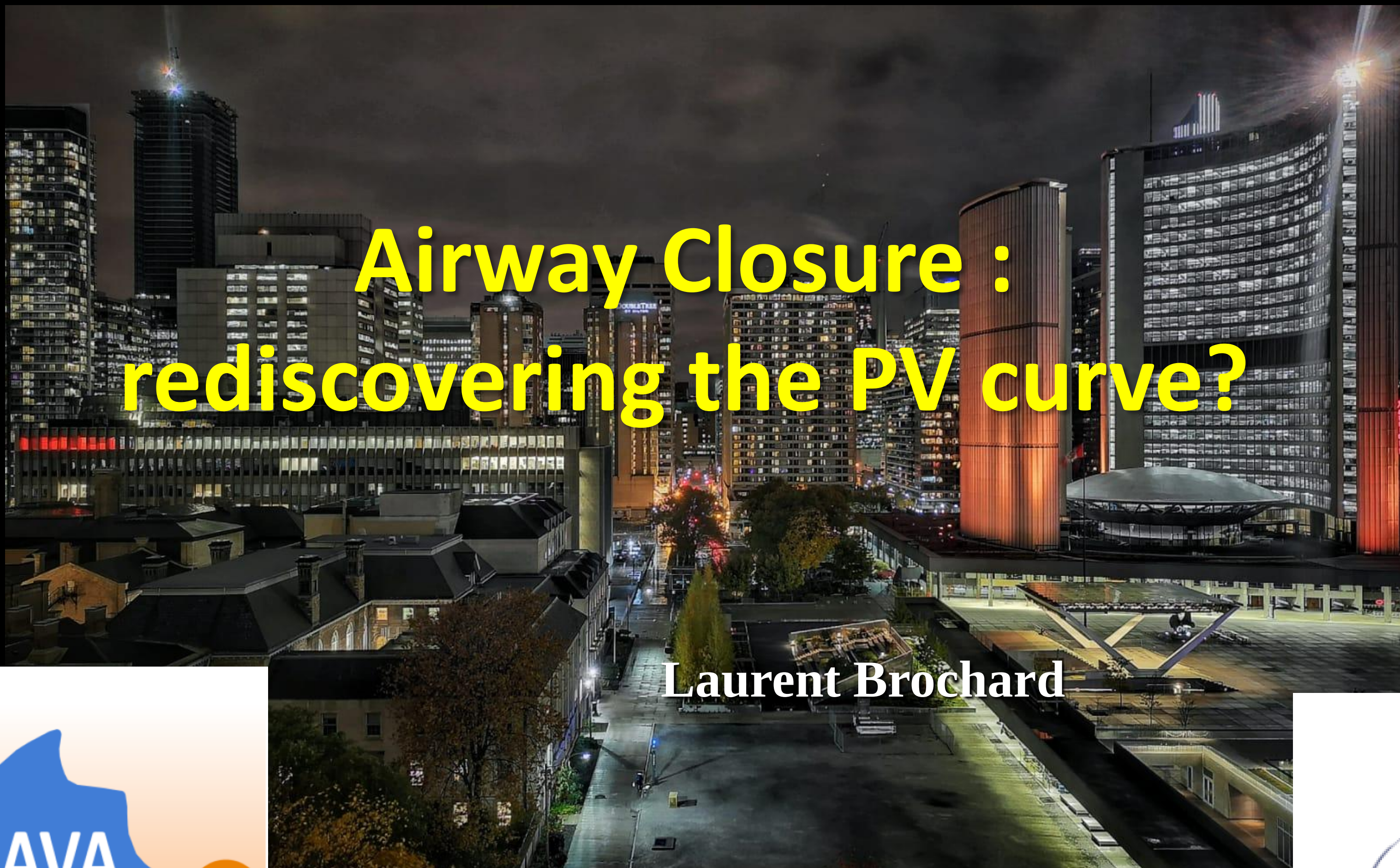


Airway Closure : rediscovering the PV curve?

Laurent Brochard

UNIVERSITY OF TORONTO

Interdepartmental Division of Critical Care Medicine

Conflicts of interest

- Research grant: Covidien Medtronic
- Research grant & equipment: Fisher Paykel
- Equipment: Maquet
- Equipment: Philips
- Equipment & patent with Universities: General Electric
- Research grant & equipment: Air Liquide

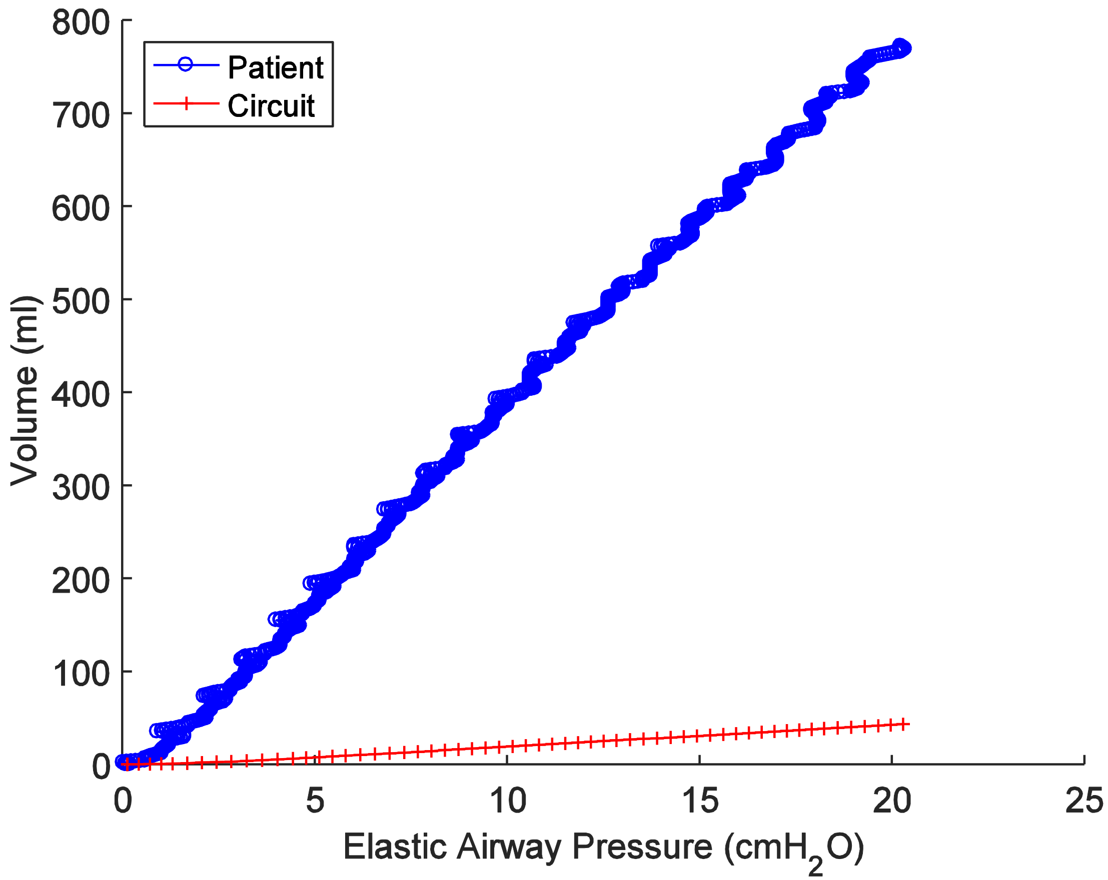
Airway Closure?

Airway Closure in Acute Respiratory Distress Syndrome: An Underestimated and Misinterpreted Phenomenon

Chen L. et al

American Journal of Respiratory and Critical Care Medicine Volume 197 Number 1 | January 1 2018

Pressure Volume curve
Majority of the patients (22/30)



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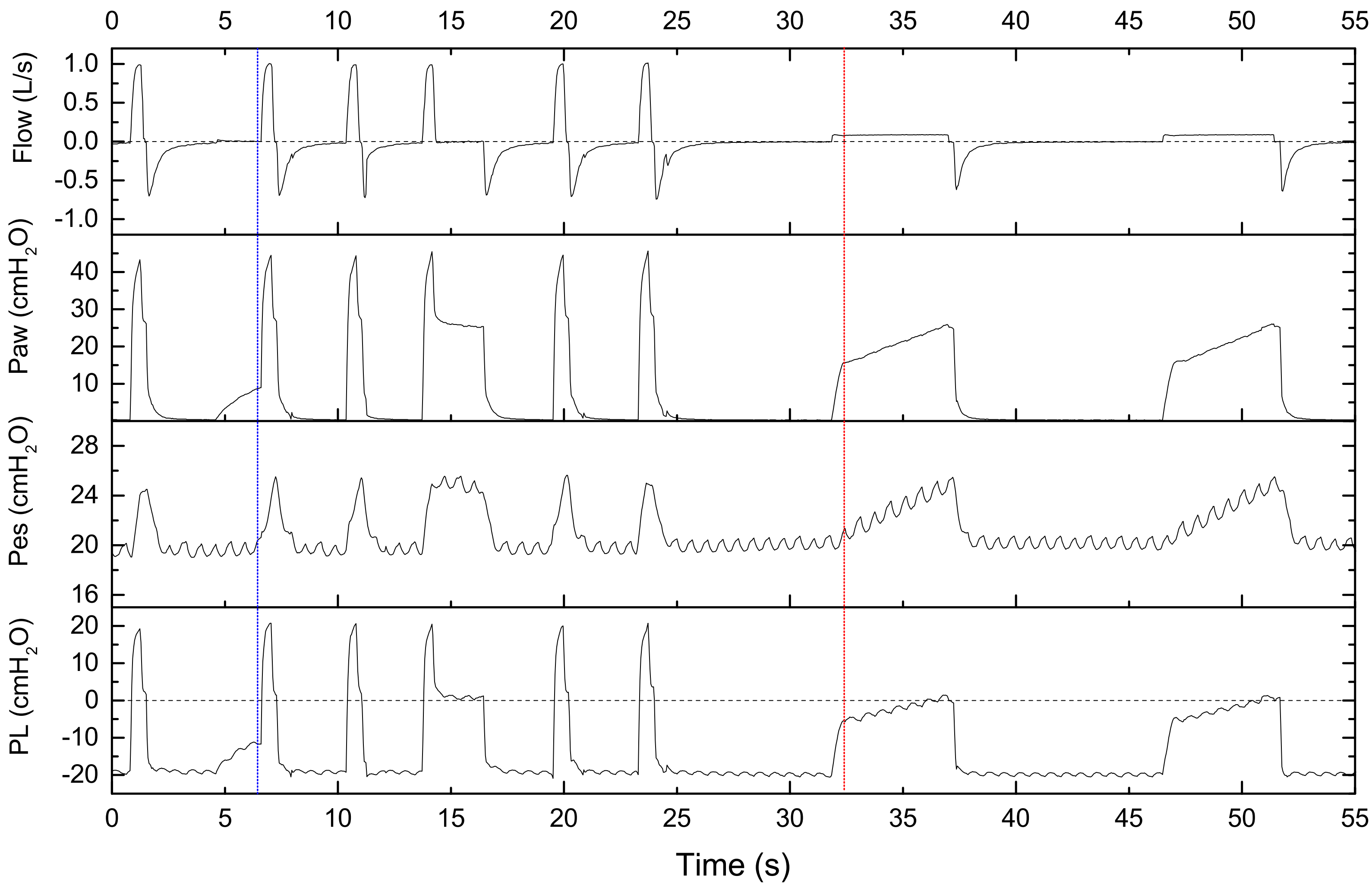
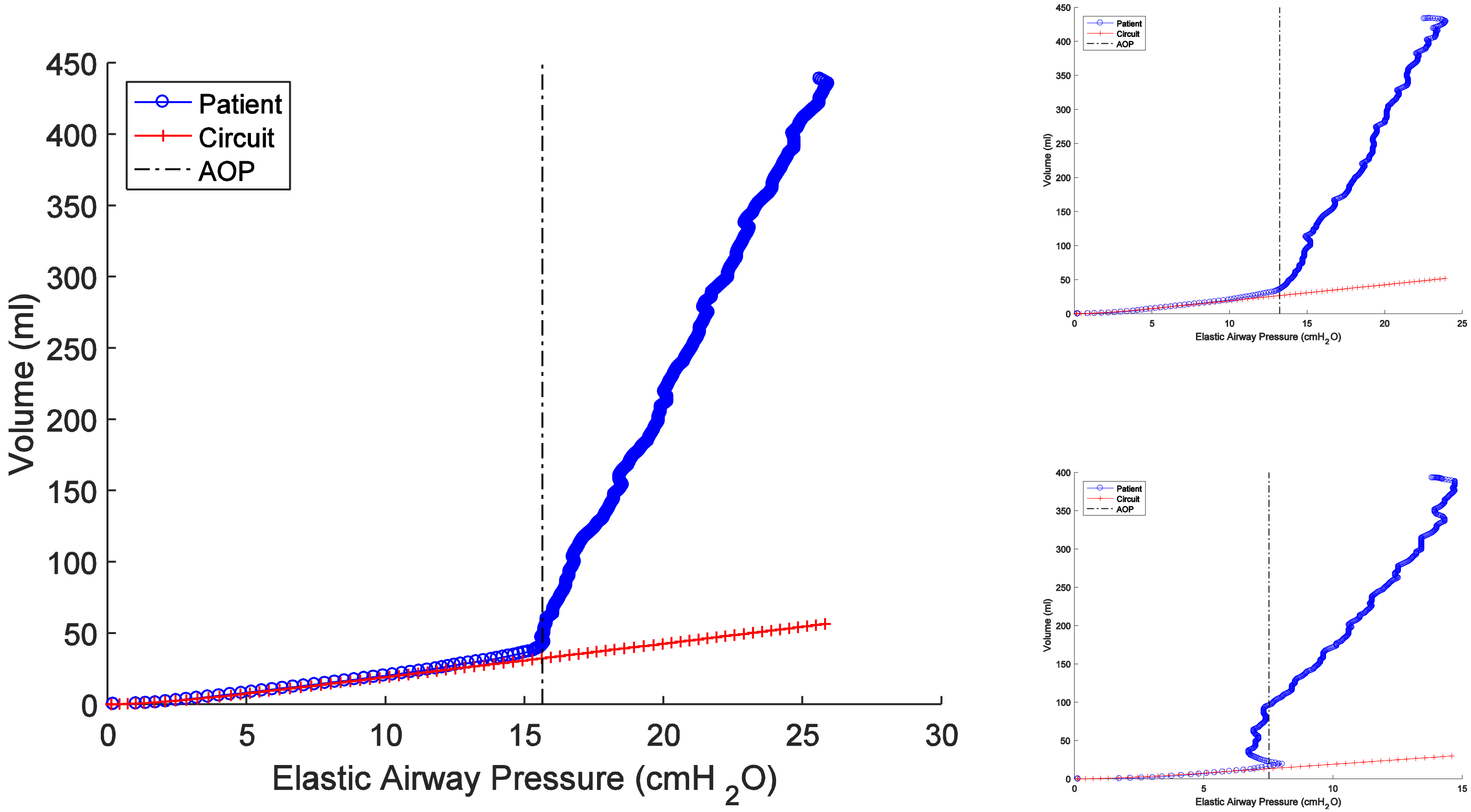


Flow (L/s)
Paw (cmH2O)
Pes (cmH2O)
PL (cmH2O)
Time (s)

Airway Opening Pressure (AOP)



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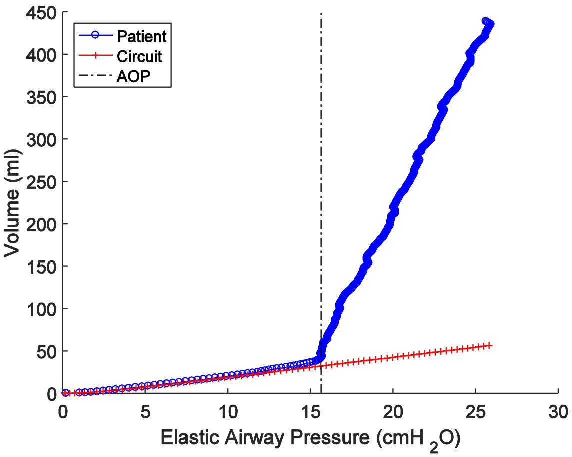
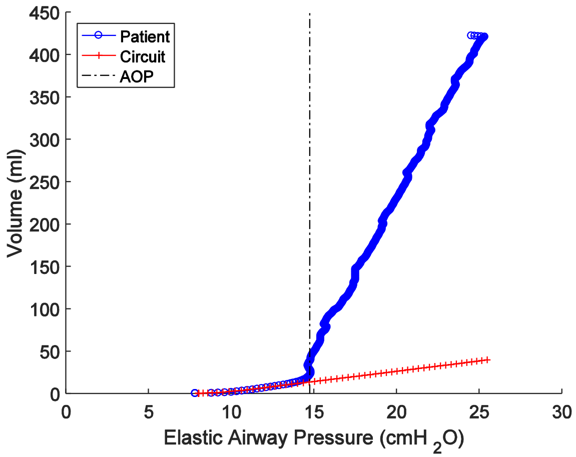
Pt#15

PEEP 0



PEEP 8



PEEP 18

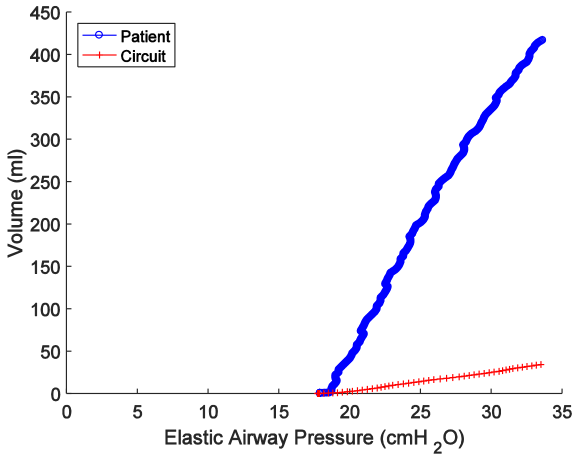


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Table 1. Characteristics and Respiratory Mechanics of Patients with Airway Closure on the Day of Study

Patient No.	Sex (*M/F*)	Age (*yr*)	Cause of ARDS	SOFA	F_{IO_2}	Pa_{O_2}/F_{IO_2} (*mm Hg*)	Pa_{CO_2} (*mm Hg*)	$\dot{V}_{E,corr}$ (*L/min*)	BMI (*kg/m²*)	Hospital Outcome	$PEEP_{low}$* (*cm H₂O*)	Total PEEP† (*cm H₂O*)	Total $PEEP_{long}$‡ (*cm H₂O*)	AOP‡ (*cm H₂O*)	Crs,st§ (*ml/cm H₂O*)	Crs,linear‖ (*ml/cm H₂O*)
1	M	53	Trauma	12	0.80	109	55	13.8	38	Survived	5	13	5	20	27	30
2	M	60	Shock	13	0.50	110	34	7.1	29	Survived	5	8	5	8	15	21
3	M	49	Pneumonia	11	0.70	97	59	15.8	23	Survived	7	16	7	19	28	54
4	M	68	Pneumonia	15	0.75	83	52	9.9	37	Survived	0	9	0	16	23	38
5	F	63	Pneumonia	16	0.60	100	51	9.6	44	Died	0	6	0	13	19	39
6	M	66	Pneumonia	15	0.90	94	51	11.5	50	Survived	0	5	0	7	40	65
7	F	45	Pneumonia	11	0.70	109	61	19.5	22	Survived	0	5	0	8	28	41
8	M	33	Pneumonia	11	0.65	120	56	8.3	32¶	Died	0	4	0	16	8	14
Mean	6M/2F	55		13	0.70	103	52	11.9	34	6S/2D	2	8	2	13	24	38
SD		12		2	0.12	12	8	4.2	10		3	4	3	5	5	10

Definition of abbreviations: AOP = airway opening pressure; ARDS = acute respiratory distress syndrome; BMI = body mass index; Crs,st = static respiratory system compliance; Crs,linear = linear portion of respiratory sytem compliance; PEEP = positive end-expiratory pressure; SOFA = sepsis-related organ failure assessment; $\dot{V}_{E,corr}$ = corrected expired volume per minute (i.e., minute ventilation times Pa_{CO_2} divided by 40 mm Hg).

*Lowest PEEP used in the study with low-flow inflation pressure–volume curves.

†Total PEEP was measured at the regular respiratory rate (20–35 breaths/min) with an end-expiratory occlusion maneuver at $PEEP_{low}$.

‡Total $PEEP_{long}$ and AOP were measured after a prolonged expiration (15–20 s) at $PEEP_{low}$.

§Crs,st was measured by occlusion maneuvers, as the tidal volume divided by the difference between the plateau pressure and total PEEP.

‖Crs,linear was measured by linear fitting on the relatively linear portion of the pressure–volume curve, where the airway pressure exceeded the AOP.

¶Patient had short stature (1.0 m).

Total = 8/30

Total PEEP = 8

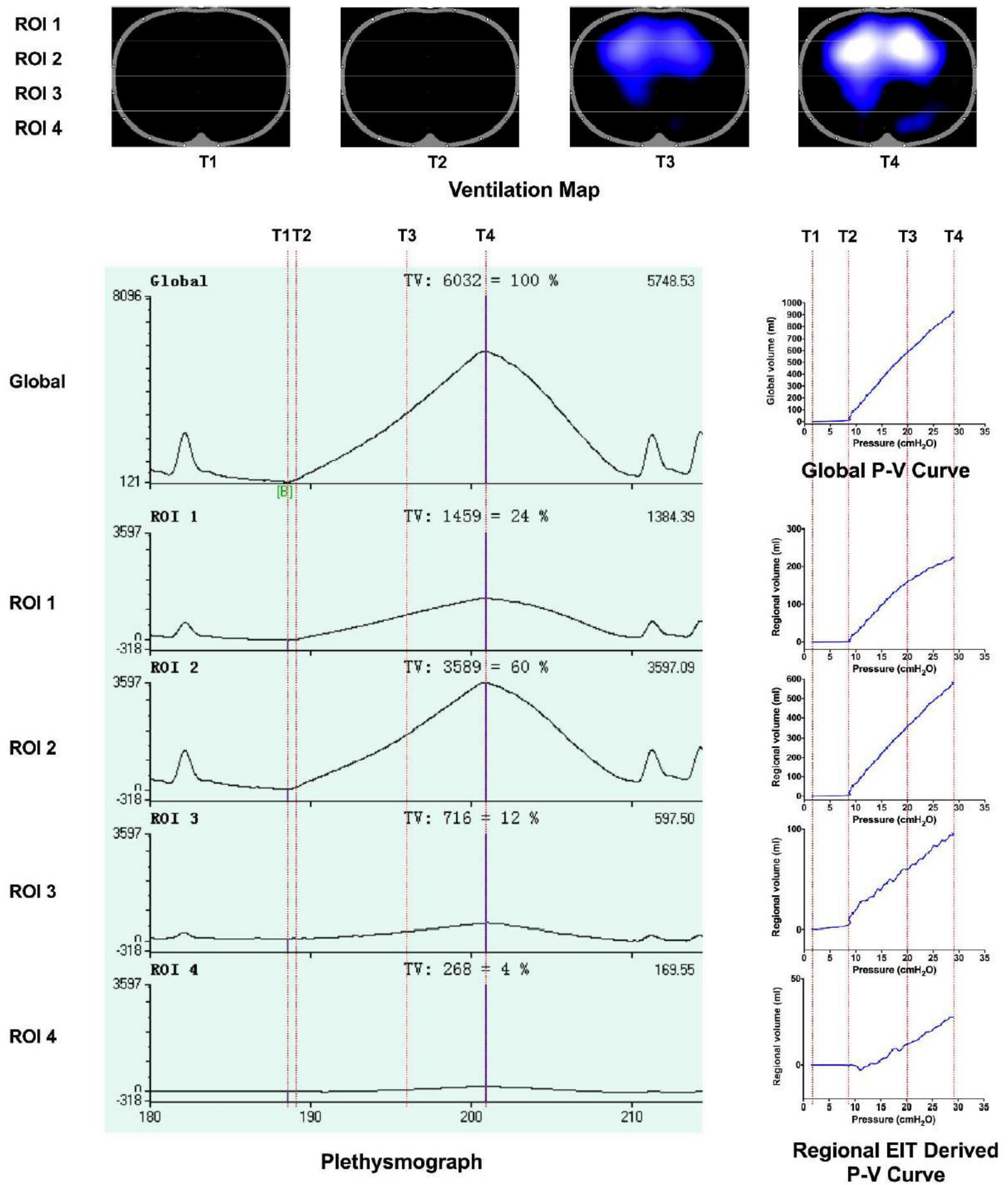
AOP = 13 [7 – 20]

EXPIRATORY FLOW LIMITATION ASSESSMENT IN ARDS PATIENTS. A REAPPRAISAL

Hodane Yonis[1], Satar Mortaza[2], Loredana Baboi[1], Alain Mercat[2, 3], Claude Guérin[1, 4, 5]

Data at the time of inclusion			
	EFL (n=13)	NFL (n=52)	P value
PaO2/FIO2 (mmHg)	144±55	165±56	0.07
PaCO2 (mmHg)	46±9	44±8	0.03
Change end expiratory lung volume (ml)	147±132	389±282	0.195
Airway reopening pattern on volume pressure curve (%)	11 (85.6)	10 (19.2)	0.00002

PEEP 5 → 21/64= 33%



Xiu-Mei Sun AJRCCM 2017

JOURNAL OF APPLIED PHYSIOLOGY
Vol. 31, No. 5, November 1971. *Printed in U.S.A.*

"Closing volume" and its relationship to gas exchange in seated and supine positions

DOUGLAS B. CRAIG, W. M. WAHBA, H. F. DON, J. G. COUTURE,
AND MARGARET R. BECKLAKE
Respiratory Division, Department of Medicine, and Department of Anesthesia,
Royal Victoria Hospital and McGill University, Montreal 112, Canada

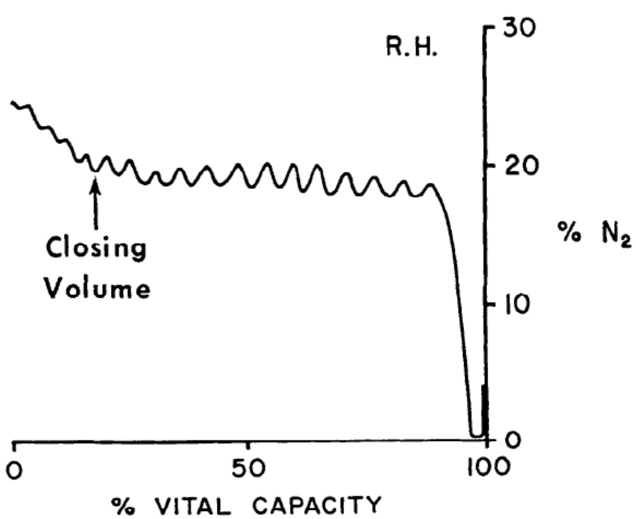
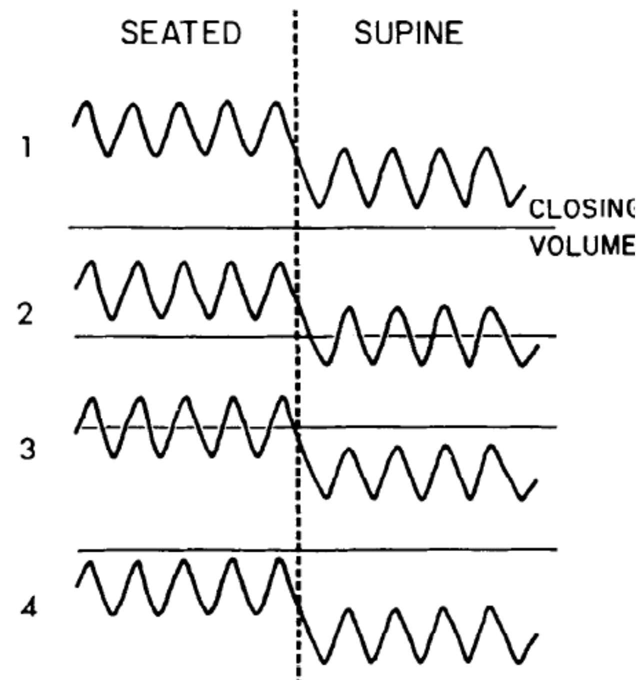


FIG. 1. Percent N_2 in expirate (at the mouth) as a function of expired volume (expressed as %VC).



Perspective on Lung Injury and Recruitment

A Skeptical Look at the Opening and Collapse Story

Rolf D. Hubmayr

Mayo Clinic, Rochester, Minnesota

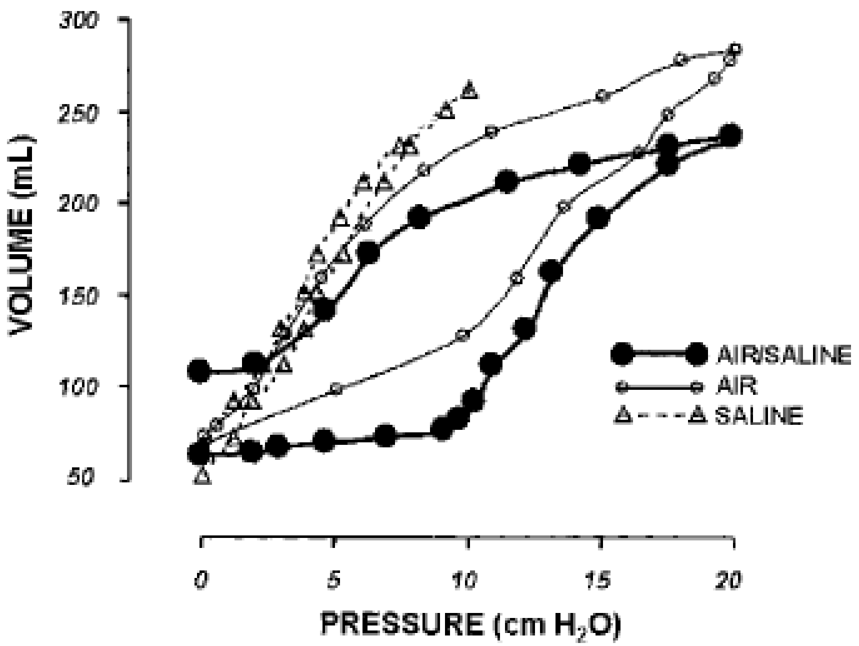
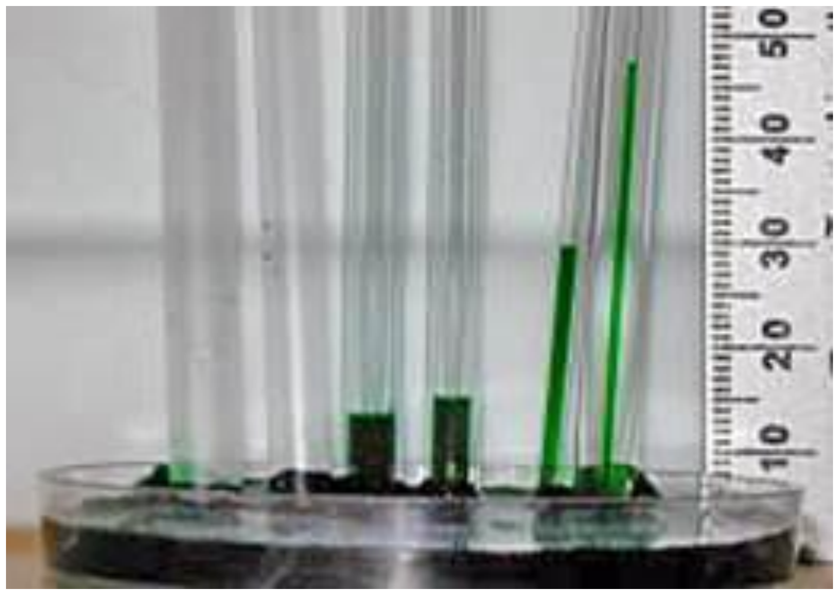
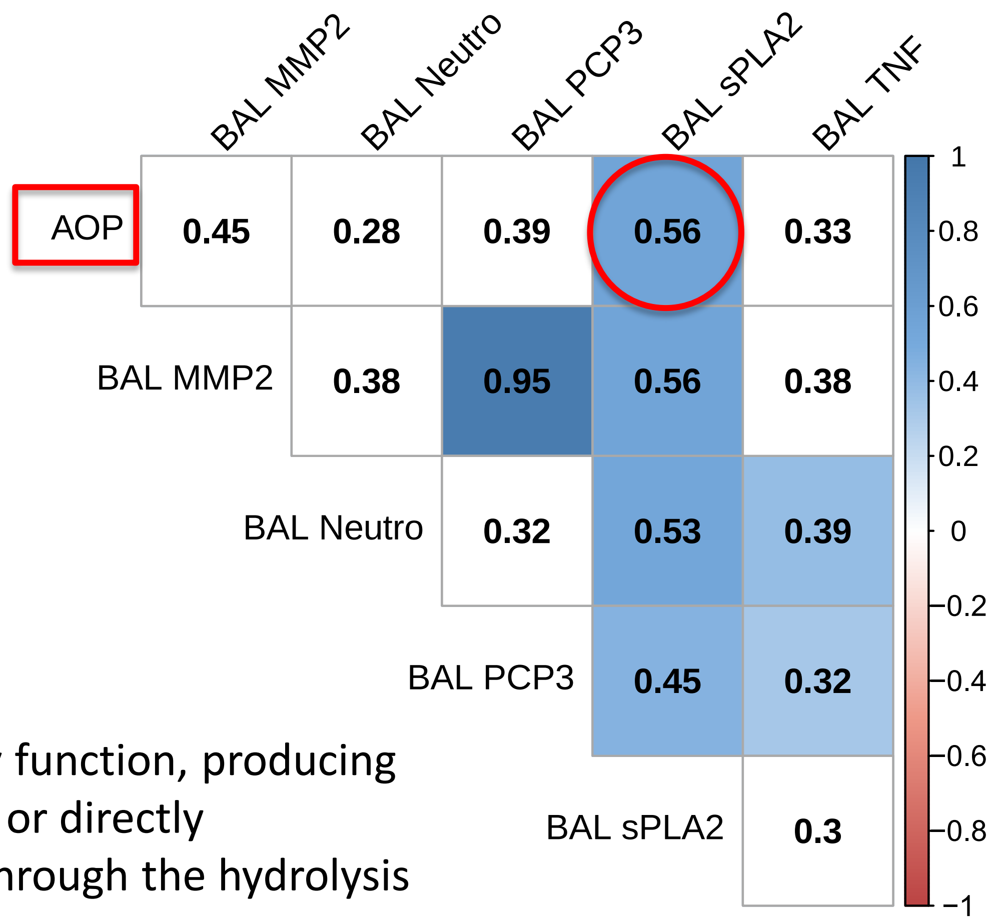


Figure 2. Pressure–volume curves of a canine caudal lobe containing air only, saline only, and a air-saline mixture. Note the high initial impedance when air is injected into a saline-filled lung. Adapted with permission from Reference 62.

Site of Airway Closure?



A role of surfactant depletion?



sPLA2s affect pulmonary function, producing inflammatory mediators or directly catabolizing surfactant through the hydrolysis of its phospholipids.

R. Coudroy, L. Chen, A Demoule et al. submitted

Consequences of Airway Closure?

- Airway (and alveolar) injury
- Reabsorption atelectasis

CRITICAL CARE

RESEARCH Open Access

Small airway remodeling in acute respiratory distress syndrome: a study in autopsy lung tissue

Maina MB Morales[1*], Ruy C Pires-Neto[1], Nicole Inforsato[1], Tatiana Lanças[1], Luiz FF da Silva[1], Paulo HN Saldiva[1], Thais Mauad[1], Carlos RR Carvalho[2], Marcelo BP Amato[2], Marisa Dolhnikoff[1]

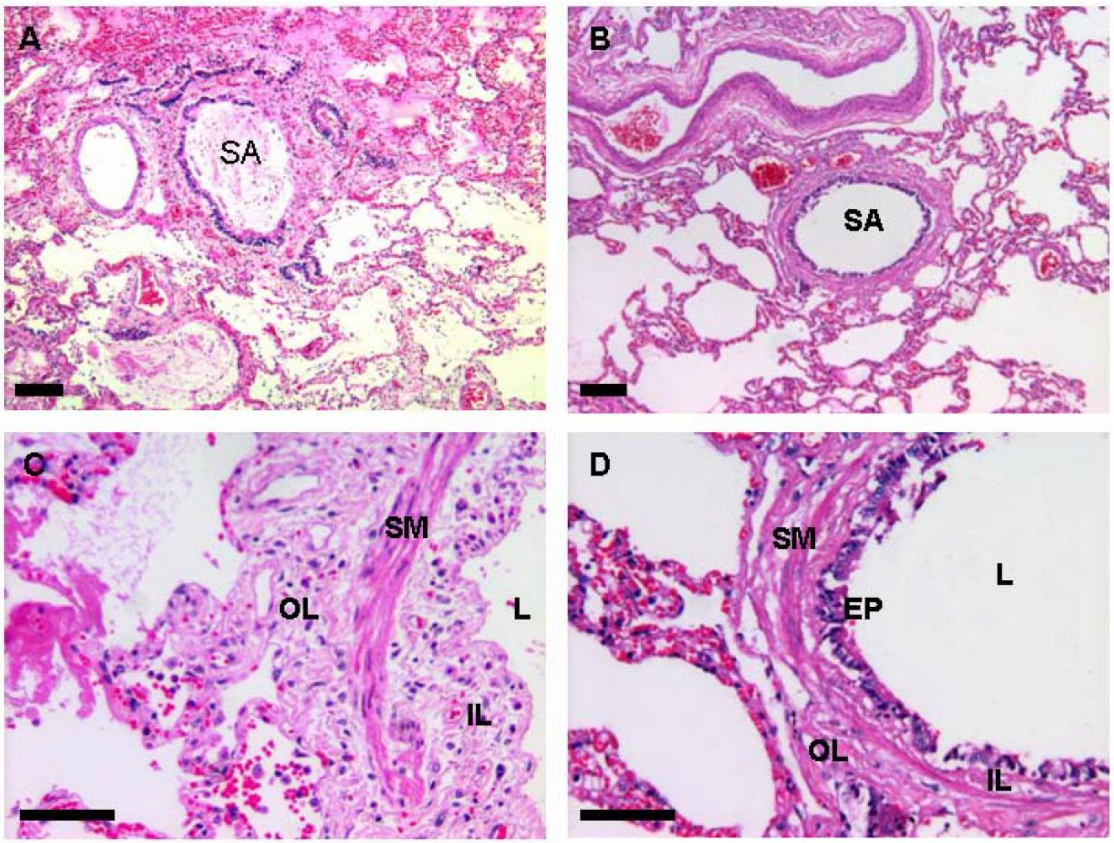
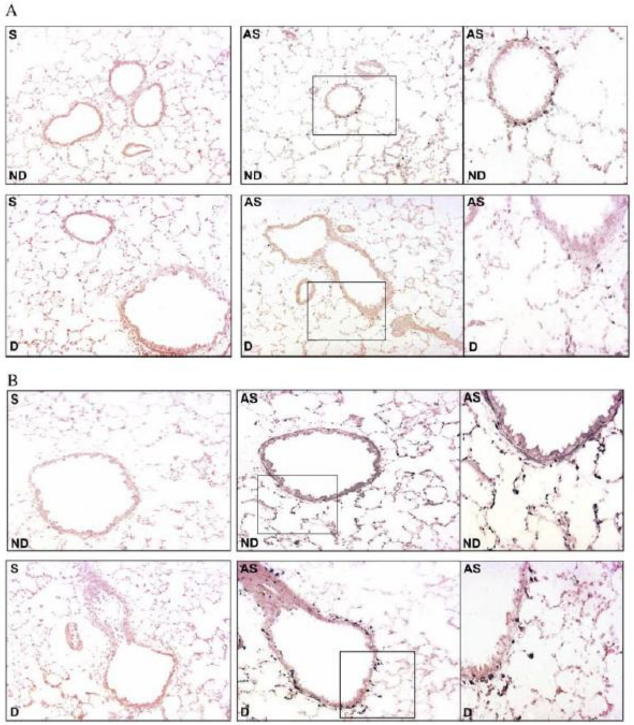


Figure 2 Lung histology from ARDS and control patients. Representative photomicrographs of distal airway and alveolar tissue from ARDS (**A** and **C**) and control (**B** and **D**) patients. ARDS lungs show extensive intra-alveolar exudate (A) and small airway thickening with mild inflammation and epithelium denudation (C). SA = airway; L = lumen; EP = epithelium; SM = smooth muscle; OL = outer layer; IL = inner layer. H&E staining. Scale bars: A and B = 100 µm, C and D = 50 µm.

Atelectasis Causes Alveolar Injury in Nonatelectatic Lung Regions

Shinya Tsuchida, Doreen Engelberts, Vanya Peltekova, Natalie Hopkins, Helena Frndova, Paul Babyn, Colin McKerlie, Martin Post, Paul McLoughlin, and Brian P. Kavanagh

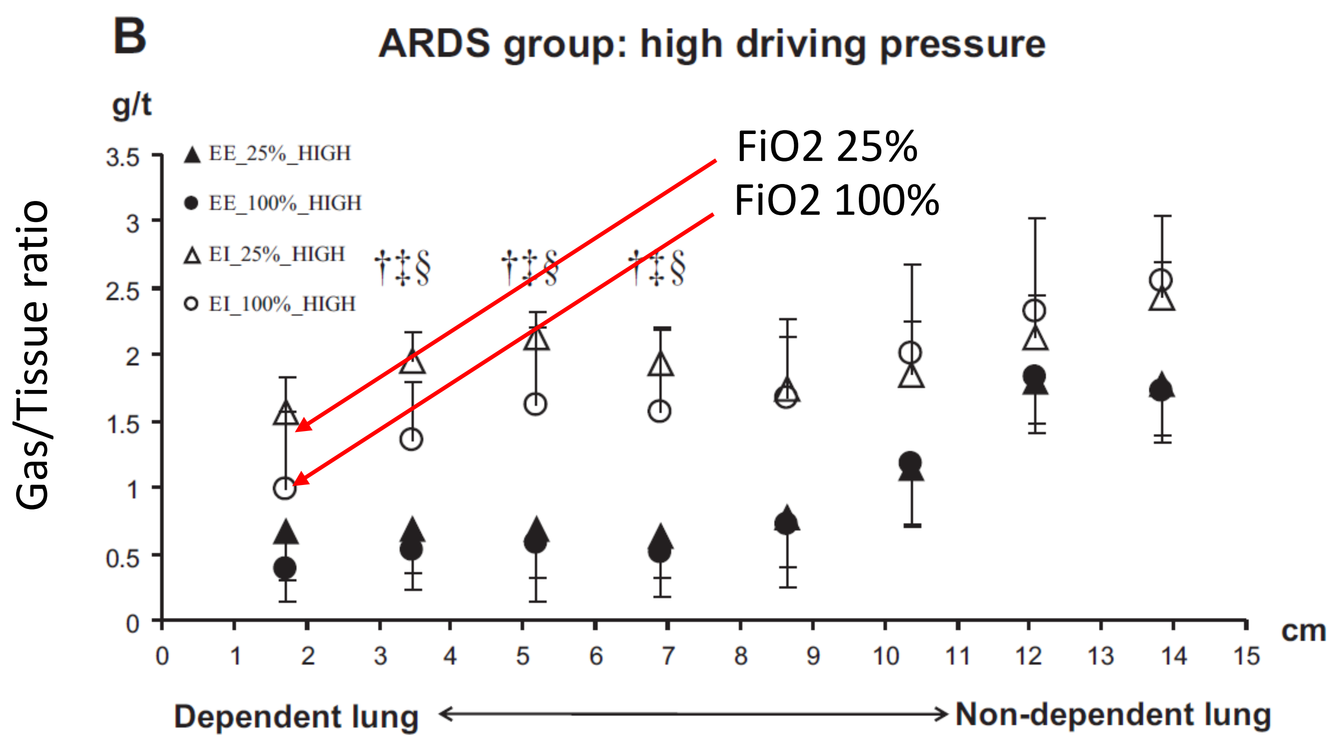
Conclusions: These data support the notion that lung injury associated with atelectasis involves trauma to the distal airways. We provide topographic and biochemical evidence that such distal airway injury is not localized solely to atelectatic areas, but is instead generalized in both atelectatic and nonatelectatic lung regions. In contrast, alveolar injury associated with atelectasis does not occur in those areas that are atelectatic but occurs instead in remote nonatelectatic alveoli.



J Appl Physiol 115: 1464–1473, 2013.
First published September 5, 2013; doi:10.1152/japplphysiol.00763.2013.

Reabsorption atelectasis in a porcine model of ARDS: regional and temporal effects of airway closure, oxygen, and distending pressure

Savino Derosa,[1,2]* João Batista Borges,[2,3]* Monica Segelsjö,[4] Angela Tannoia,[1] Mariangela Pellegrini,[1] Anders Larsson,[2] Gaetano Perchiazzi,[1] and Göran Hedenstierna[5]



Airway Closure (>5 cmH2O)

- Exists in 30 to 40% of ARDS
- From 5 to 20 cmH2O
- Probably more frequent in some populations (Obese?)
- Undetectable without a low flow PV curve
- May explain bronchiolar (and alveolar) injury by repeated stretch
- May justify PEEP set at or above AOP

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