TITLE: **EFFECTS OF POSITIVE PRESSURE VENTILATION ON MICROHEMODYNAMICS IN ARTERIOLAR NETWORKS OF THE LUNG**

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Introduction: Positive pressure ventilation (PPV) is commonly used in general anesthesia and plays a major role in prevention and therapy of ARDS. Whereas the effects of PPV on macrohemodynamics and gas exchange are well established the local microhemodynamic changes within the lungs have not been described. Results are mainly based on indirect measurements and on histological analysis. We therefore studied the effects of PPV on pulmonary microhemodynamics within an arteriolar network in a recently developed in-vivo experimental model.

Methods: 3 rabbits were anesthetized using Thiopental (30mg/kg), Chloralose (50mg/kg) and Piritramid (0.3mg/kg), intubated and ventilated mechanically. Arterial pressure (AP), pulmonary artery pressure (PAP), left atrial pressure (LAP), cardiac output (CO) and airway pressure (AWP) were continuously monitored. After implantation of a thoracic window the pulmonary microcirculation was visualized using fluorescence video microscopy after injection of fluorescein-isothiocyanate (FITC) stained red cells. 3 arteriolar vessel trees (networks) were recorded on videotape by scanning an area of 4 x 3mm on the surface of the right upper lung lobe under different airway pressures (inspiratory/expiratory pressure: 12/3 and 8/2 mmHg). Networks were reconstructed, vessel diameters (D), red cell flux (F), red cell velocity (V), microhematocrit (H) and coefficients of variation (CV) were measured.

Results: Macrohemodynamic parameters at AWP of 8 mmHg were: PAP: 20 mmHg ; LAP: 5 mmHg ; CO: 217 ml/min; pulmonary vascular resistance (PVR): 66 mmHg/ml/min. Elevation of inspiratory AWP to 12 mmHg induced the following changes: CO -35%; PAP -10%; PVR +78%; LAP ±0%. Microhemodynamic variables in 56 precapillaries showed significant changes. In the table the microhemodynamic parameters during both levels of airway pressures (mean of 3 networks), the corresponding coefficients of variation (range over 3 networks) and the relative changes during AWP at 12 vs. 8mmHg are given.

	D	F	V	H
8mmHg	22 µm	1652 rbc/s	948 µm/s	28%
CV	12-20%	41-47%	20-37%	14-26%
12mmHg	19 µm	908 rbc/s	577 µm/s	33%
CV	13-21%	31-64%	24-48%	19-35%
12 vs. 8 mmHg	-15%	-39%	-35%	+22%
CV	10-37%	57-67%	55-68%	28-39%

Conclusion: 1) This study represents the first in vivo network analysis in the lung including measurements of pulmonary micro- and macrohemodynamics.

2) Elevation of airway pressure induced significant reduction of arteriolar microvessel diameters, red cell flux, red cell velocity and an increase of microhematocrit.

3) The increase of CV of red cell flux and red cell velocity at reduced blood flow indicates pronounced spatial heterogeneity of the local pulmonary blood flow.

4) The reduction of precapillary luminal diameters and the increase of microhematocrit might significantly contribute to the rise in pulmonary vascular resistance during positive pressure elevation.

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TITLE: MEASUREMENT OF RESPIRATORY SYSTEM ELASTANCE AND RESISTANCE DURING CONVENTIONAL MECHANICAL VENTILATION

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The elastance (E) and resistance (R) of the relaxed respiratory system, measured during sinusoidal forcing depend on respiratory frequency (f) and tidal volume (VT). As such, E and R are not completely linear [1,2,3] and may be dependent on the type of forcing flow waveform and analysis method used. In clinical settings, sinusoidal forcing is usually not available and linear analyses are common. We evaluated the errors induced when two types of non-sinusoidal waveforms, available on clinical ventilators, were used to with calculate E and R by linear analysis. Methods We measured pressure and flow (pneumotachograph, Fleisch #2) at the mouth of 5 healthy, awake humans, relaxed at functional residual capacity (FRC), during forcing with three different flow waveforms at 12 breaths/min and 750 ml VT. The waveforms used, each having inspiratory period equal to 50% of the total breath, were: 1) sinusoidal, SINE (delivered by piston pump driven by a linear motor); 2) quasi-sinusoidal, QUAS (sinusoidal inspiration, passive expiration - modified Siemens 900B); and 3) square, SQU (step inspiration, passive expiration - Siemens 900B). R and E were calculated from 3 consecutive breaths using both a linear type of analysis (Fast Fourier Transform, FFT), and a non-linear analysis, which is independent of non-linearities in respiratory system mechanical behavior. Results Table 1 shows that type of analysis had no effect on R (in $cmH_2O/l/s$) during any waveform, but E (in cmH_2O/l) during QUAS and SQU with NLA were about 10% higher than with FFT. Waveform did not affect R, but compared to sinusoidal forcing E was about 20% higher during QUAS and SQU.

TABLE 1 N=5, SE in parenthesis.

	SINE		QUAS		SQU	
	NLA	FFT	NLA	FFT	NLA	FFT
E	8.8	8.9	$10.5^{b,a}$	9.9^{b}	10.4^{b}	9.7^{b}
	(1.2)	(1.3)	(1.4)	(1.3)	(1.2)	(1.1)
R	3.2	3.22	3.37	3.11	3.74	3.12
	(0.27)	(0.29)	(0.22)	(0.23)	(0.23)	(0.25)

paired t-test ($p<0.05$): a = diff. from FFT with same type forcing; b = diff. from SINE with same analysis.

Discussion Most clinical ventilators are easily adjusted to deliver a square waveform. Such a waveform, generated by adjusting the inspiratory time to 50% of the total breath without an inspiratory pause, can be used to get a good estimate of total respiratory system R and E even if linear analysis is used. Not much more accuracy is obtained using QUAS. For studies where less than 10% error is necessary sinusoidal forcing and NLA are preferable. By means of simple ventilator adjustments the effects of clinical interventions altering R and E such as removal of retained lung secretions or rapid infusion of fluid in shock states may be quantitated. Ref. 1) The Physiologist 32(4):194, 1989. 2) Resp Physiol 78:369-382, 1989. 3) FASEB Journal 3(4):A981, 1989.