

Characterization with EIT of regional ventilation distribution in experimental lung injury induced by unilateral ligation the of ne IRCCS Ca' Granda Economica pulmonary artery

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Introduction

Ligation of the left pulmonary artery in healthy pigs is associated with bilateral lung injury which might be prevented by inhaled CO₂. Mechanisms involved remain elusive.

Objectives: To study regional ventilation distribution in a model of unilateral ligation of the left pulmonary artery with and without administration of inhaled CO₂.

Model: LEFT PULMONARY ARTERY LIGATION

Groups: 11 pigs ventilated for 48 hours

4 Ligation group: Vt 10 ml/Kg, PEEP 5 cmH₂O, 25 bpm, FiO₂ 50%

7 Ligation + FiCO₂ 5% group: Vt 10 ml/Kg, PEEP 5 cmH₂O, 25 bpm, FiO₂ 50%, FiCO₂ 5%.

Measures at 2, 12, 24, 36 and 48 hours from artery ligation:

Distribution of ventilation measured by electrical impedance tomography

Measures at the end of the experiment:

- Histological score
- **Respiratory mechanics**
- Gas exchange

Results

At the end of the experiment animals in the Ligation group developed bilateral lung injury while inhaled CO2 prevented it (Table 1).

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	Ligation + FiCO ₂ 5% (n=7)	Ligation (n=4)	P value
Compliance (mL/cmH ₂ O)	40 [31; 44]	24 [18; 26]	0.006
Plateau Pressure (cmH ₂ O)	14 [13; 16]	23 [21; 29]	0.006
PaO ₂ /FiO ₂	520 [384; 542]	298 [141; 391]	0.039
Histological score	4 [3; 4]	8 [8; 10]	0.006



We studied distribution of ventilation by EIT as possible mechanism for contralateral lung injury (Figure 1).



The Ligation group showed higher fraction of Vt reaching the right perfused lung (Fig. 2A) from T2 to T36 compared to the Ligation + FiCO2 5% group. At T48, right and left lungs were subject to a more equal distribution of Vt, probably due to a fall in right lung compliance (Fig. 2B), consistent with development of lung injury in the perfused lung.

Conclusions

Redistribution of Vt to the perfused lung may be implied in the development of regional lung injury following ligation of contralateral pulmonary artery. Mechanisms inducing injury in the non-perfused lung remain to be elucidated.





RIGHT LEFT RIGHT LEFT Figure 1. Distribution of ventilation by EIT at T2 and T48