TITLE: Influence of P\textsubscript{a}O\textsubscript{2} on blood flow to atelectatic lung


AFFILIATION: Departments of Anesthesia and Surgery, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania 19104

Introduction: Blood flow generally decreases to lung regions that become atelectatic. The principal mechanism appears to be hypoxic pulmonary vasoconstriction (HPV), stimulated by mixed venous oxygen tension (P\textsubscript{a}O\textsubscript{2}). This study examines the effects of high or low P\textsubscript{a}O\textsubscript{2}, using total extracorporeal veno-venous bypass in dogs with one lung atelectasis.

Methods: Six female dogs were anesthetized with pentobarbital, paralyzed with pancuronium, and ventilated with 100% \textsubscript{O}2 and 5 cm H\textsubscript{2}O PEEP to a Pa\textsubscript{CO}\textsubscript{2} of 40 mmHg. A Kottmeier double lumen endobronchial tube was inserted via a tracheostomy and left lung atelectasis induced. Electromagnetic flow probes were placed around the ascending aorta and left pulmonary artery. Airway, left atrial, central venous, pulmonary, and systemic arterial pressures were monitored. The bypass circuit contained a bubble oxygenator with a gas supply regulated by N\textsubscript{2}, \textsubscript{O}2, \textsubscript{CO}2 flow meters. The pump was primed with 1000 ml Normosol and 500 ml dog blood. The animal was anticoagulated with heparin as determined by activated clotting times. Total venous return was collected by cannulae in superior and inferior vena cavae and infused via a right atrial cannula.

High or low P\textsubscript{a}O\textsubscript{2} was selected in random order by adjusting the N\textsubscript{2} and \textsubscript{O}2 flows to the pump oxygenator. Measurements were repeated at normal (100 ml/min/kg) and low (50 ml/min/kg) cardiac outputs (CO). The HP\textsubscript{P} response was quantitated by left lung flow (Q\textsubscript{L}), shunt (Q\textsubscript{s}/Q\textsubscript{T}), and arterial oxygenation (Pa\textsubscript{O}\textsubscript{2}). Data were analyzed by a within subjects two factor ANOVA. Means ± SE are shown.

Results: The general experimental conditions were constant (temp = 37.1 ± 1°C, Hb = 8.1 ± 0.4 gm%, pH = 7.316 ± 0.0140, Pa\textsubscript{CO}\textsubscript{2} = 40.6 ± 1.2 mmHg, BE = 4.3 ± 0.7%). Q\textsubscript{L}/Q\textsubscript{T} % and Q\textsubscript{s} varied directly with P\textsubscript{a}O\textsubscript{2}, at both levels of cardiac output (see Figgure and Table 1). With high P\textsubscript{a}O\textsubscript{2}, diversion of blood flow away from atelectatic lung did not occur and mean Q\textsubscript{L} (40%) was nearly the flow expected for normoxic ventilated left lung (45%). Nevertheless, Pa\textsubscript{O}2 was > 300 mmHg secondary to the increased P\textsubscript{a}O\textsubscript{2}. Cardiac output did not have an effect on Pa\textsubscript{O}2, Q\textsubscript{L}/Q\textsubscript{T} % or Q\textsubscript{s} %. Mean pulmonary artery pressure (PAP) was lower with low cardiac output.

Discussion: This study shows that in atelectasis HPV varies inversely with P\textsubscript{a}O\textsubscript{2} for dogs with normal or low cardiac output. HPV was abolished by P\textsubscript{a}O\textsubscript{2} > 100 mmHg. When P\textsubscript{a}O\textsubscript{2} was reduced to 20-30 mmHg, approximately 50% of the blood flow was diverted away from the atelectatic lung. The effectiveness of HPV is demonstrated by the absence of hypoxemia when low P\textsubscript{a}O\textsubscript{2} occurs during atelectasis. These responses are consistent with those observed previously when hypoxic gas mixtures were administered.

The precise control of P\textsubscript{a}O\textsubscript{2} allowed demonstration of a greater influence of HPV than some others have reported. The atelectasis model avoids the secondary influence of P\textsubscript{a}O\textsubscript{2} on alveolar oxygen tension and ventilation/perfusion relationships which complicate interpretations.

Conclusions: Approximately 50% of blood flow is diverted away from atelectatic lung when P\textsubscript{a}O\textsubscript{2} is 20-30 mmHg. This HPV response is abolished by P\textsubscript{a}O\textsubscript{2} > 100 mmHg.