Title: PULMONARY PRESSURE AND FLOW DURING ATELECTASIS

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Introduction. Hypoxic pulmonary vasoconstriction (HPV) occurs with atelectasis and should result in a dual response - increased pulmonary perfusion pressure (PP) and blood flow diversion from atelectatic to normoxic lung regions.[1] However, minimal flow changes,[2] transient responses or increased flows to atelectatic areas[3] have been reported. The purpose of this study was to measure the time course, direction, and magnitude of the flow and pressure responses to atelectasis with rigorous control of variables known to alter this response.

Methods. Six female mongrel dogs (19.4 ± 1.0 kg) (mean ± SE) were anesthetized with pentobarbital and paralyzed with pancuronium. Each lung was ventilated separately through a left Robertshaw endobronchial tube via a Harvard dual-piston ventilator. Blood flow to each lung was measured with electromagnetic flow probes (Micron Instruments) surgically placed around the ascending aorta and left pulmonary artery. Pulmonary arterial, left atrial and systemic arterial pressures; inspired, end-tidal and mixed expired O_2 and CO_2 tensions; and arterial and mixed venous blood gas tensions were measured. Alveolar oxygen tension and percent venous admixture (shunt) were calculated. The right lung was continuously ventilated with 100%O_2 while the left lung was either ventilated with 100%O_2 (control), unventilated (4 hours of atelectasis), or ventilated with 4%O_2 3% CO_2 93%N_2 (hypoxia). The sequence for the left lung in all studies was O_2-Hypoxia-O_2-Atelectasis-O_2-Hypoxia. Analysis of variance with Newman-Keuls test or paired 2-tailed t-test was used for statistical analysis with significance at $p<0.05$.

Results. Left lung atelectasis resulted in a reduction of the left lung blood flow from 42.8 ± 4.3% during the control phase to 24.8 ± 7.1% at 15 min. and 11.7 ± 1.4% by 60 min. which persisted for the 4 hour study period (Fig. 1). This response reduced the "expected" pulmonary shunt from 42% to 17.8 ± 2.4% at 4 hours. The percent left lung flow was significantly lower (8.2 ± 1.4%) and the resultant PaO_2 significantly higher (356 ± 38 mmHg) during the maximal response to atelectasis as compared to 15 min of hypoxia (22.5 ± 4.5%; 211 ± 21 mmHg). PP increased from the control value of 7.9 ± 0.8 mmHg to 11.0 ± 0.8 mmHg at 15 min. of atelectasis. This did not change further during the 4 hour period and was not different from that measured during hypoxia (10.6 ± 1.0 mmHg).

Discussion. Atelectasis resulted in a regional increase in pulmonary vascular resistance (PVR) causing a 75% diversion of blood from the atelectatic to the normoxic lung. The consequent reduction in "expected" shunt prevented the occurrence of systemic hypoxemia. The response measured 15 min. after the onset of atelectasis was identical to that after 15 min. of hypoxia while the maximal flow response to atelectasis at one hour was greater. This might be due to mechanical factors increasing PVR in addition to HPV. In this study, the chest was open, total flow, pH, PCO_2 and temperature were constant and arterial hypoxemia avoided. In these circumstances, the response to atelectasis of blood flow diversion and increased PP develops quickly (15 min), is maximal by one hour and is sustained for 4 hours. Alkalosis,[2] closed chest,[4] low cardiac output and/or arterial hypoxemia[4] have been associated with attenuated responses in previous reports.

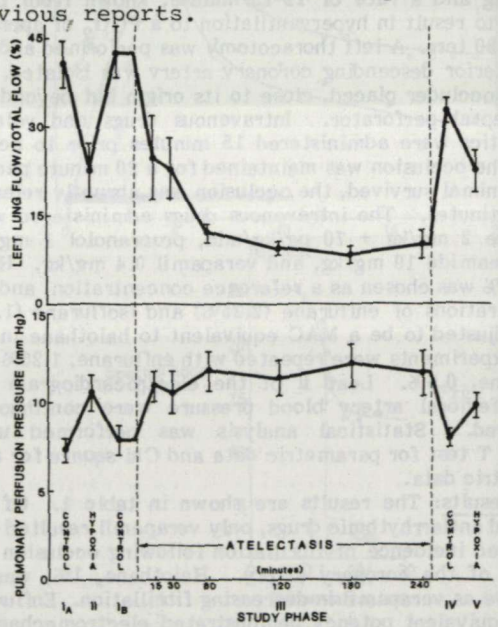

Fig. 1 Left lung blood flow as percent of total blood flow (upper) and pulmonary perfusion pressure (PP) mmHg (lower) for each study period. Atelectasis is on a time scale of 0-240 minutes. Data as mean ± SE.

References.
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