TITLE : CORRELATES OF PULMONARY VASCULAR RESISTANCE

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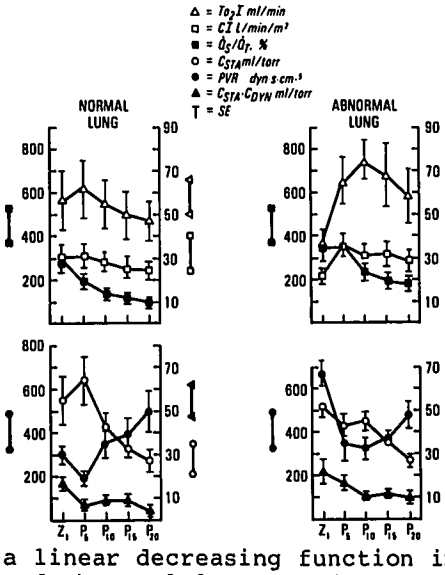
Introduction. In excised normal lungs pulmonary vascular resistance (PVR) has been described as a "U" or "J" shaped function of lung volume (LV). The trough or nadir of the "U" or "J" curve occurred at functional residual capacity (FRC). LV greater than FRC resulted in an increased total PVR, presumably due to an increase in small intra-alveolar vessel resistance, while LV less than FRC also resulted in an increased total PVR, presumably due to an increase in large extra-alveolar vessel resistance. The "U" or "J" shaped PVR curve has not been demonstrated in intact lungs nor has it been correlated with any other parameter of pulmonary function. The purpose of this experiment was to examine the PVR versus LV relationship in intact normal and abnormal lungs and correlate changes in PVR with changes in systemic oxygen transport (T_{O_2}), static and dynamic compliance and their difference (C_{STA}, C_{DYN}, C_{STA}-C_{DYN}), and intrapulmonary shunt ($\dot{Q}_s/\dot{Q}_t$).

Methods. Seven mongrel dogs were anesthetized with pentobarbital 25 mg/kg, paralyzed with pancuronium 0.1 mg/kg, tracheally intubated and mechanically ventilated with F_IO_2=1.0, tidal volume (V_t)=15 mg/kg, and a ventilatory rate so that ET_{CO_2}=5% and P_aCO_2= 40 torr. Cardiac output ($\dot{Q}_t$) was continuously measured by an electromagnetic flow probe placed around the main pulmonary artery. Pulmonary artery systolic, diastolic and wedge (P_{pas}, P_{pad}, P_{paw}), left atrial (P_{la}) and airway (P_{aw}) pressures were directly measured and following chest closure were referenced to pleural pressure (P_{pl}). Calculated were PVR=[(P_{pa}-P_{la})/$\dot{Q}_t$]79.9, T_{O_2}=$\dot{Q}_t$xC_aO_2, $\dot{Q}_s/\dot{Q}_t$ by the classic Bergren formulation, and compliance = inflated volume (18 ml/kg; held for 7 seconds)/P_{paw}-P_{pl}; the early peak P_{aw} was used for C_{DYN} and the late plateau P_{aw} was used for C_{STA}. Normal lung was converted to abnormal lung by the administration of oleic acid 0.06 ml/kg iv and 5 ml 0.1N HCL intratracheally. The experimental sequence consisted of measurement of the above values at: (Z_1) Zero end-expiratory pressure, V_t=7½ ml/kg and the chest strapped to cause a 5-8 torr increase in P_{pl}; (P_5),(P_{10}),(P_{15}) and (P_{20}) Positive end-expiratory pressure (PEEP) of 5, 10, 15, and 20 cm H_2O and V_t=15 ml/kg. Steps Z_1, P_5, P_{10}, P_{15}, and P_{20} were repeated 2 hours after oleic and HCL acid administration. All results are expressed as mean ± SE and were analyzed by paired t analysis with $p<0.05$ considered significant

Results. With a progressively increasing end-expiratory pressure (Z_1, P_5, P_{10}, P_{15}, P_{20}) as the independent variable we found that PVR was a "U" shaped function in both normal and abnormal lungs; C_{STA} was an inverted "U" shaped function in normal lungs and a decreasing function after P_{10} in abnormal lungs; C_{STA}-C_{DYN} decreased until P_5 in normal lungs and until P_{10} in abnormal lungs; $\dot{Q}_s/\dot{Q}_t$ was a linear decreasing function in both normal and abnormal lungs, and T_{O_2} was an inverted "U" shaped function in both normal and abnormal lungs (figure). Minimal PVR, maximal T_{O_2} and C_{STA} and minimal C_{STA}-C_{DYN} occurred at P_5 in normal lungs and at P_{10} in abnormal lungs while minimal $\dot{Q}_s/\dot{Q}_t$ occurred at P_{20} in both normal and abnormal lung. T_{O_2} correlated positively with $\dot{Q}_t$ in both normal and abnormal lungs.



Discussion. We have assumed in our experimental design that increasing distending pressure causes an increased lung volume. We have found that PVR is a "U" shaped function of progressively increasing end-expiratory distending pressure in both normal and abnormal in-vivo lungs which is consistent with previous in-vitro findings. In both normal and abnormal lung PVR was minimal when T_{O_2} and C_{STA} were maximal and C_{STA}-C_{DYN} were minimal. Since the distending pressure at which all of these maximal and minimal points occurred was lower in normal lung (P_5) than in abnormal lung (P_{10}) and since normal lung was more compliant than abnormal lung it appears that there is one lung volume which is optimal for ventilation, blood flow and transport of oxygen to the tissues. Attempts to find this optimal lung volume by identifying maximal C_{STA} are frequently frustrating because in clinical practice PEEP-induced changes in compliance are often small and inconclusive. Our findings strongly suggest that minimal PVR may be used as another parameter (either in combination with others or alone) by which to titrate the application of PEEP.