TITLE: BRONCHIAL ARTERY OXYGEN TENSION INFLUENCES PULMONARY VASCULAR TONE

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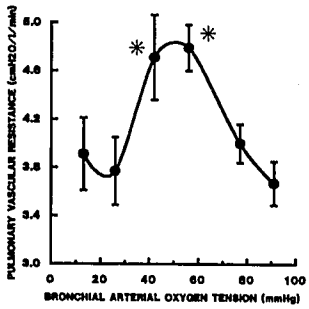
All sizes of pulmonary vessels in vitro demonstrate an hypoxic vasoconstrictor response[1]. The blood supply to the walls of pulmonary arteries is via vasa vasorum derived from the bronchial circulation[2]. The purpose of the present study was to test the hypothesis that bronchial arterial hypoxemia can influence pulmonary vascular tone.

Methods. Adult sheep were anesthetized with pentobarbital, the trachea intubated and ventilation controlled. A thoracotomy was performed and the common bronchial artery was isolated and cannulated. Blood from a recirculating hollow fibre oxygenator was diverted to perfuse the bronchial circulation at a flow such that the inflow pressure was greater than systemic.

The pulmonary artery pressure, pulmonary artery occlusion pressure and cardiac output were recorded and pulmonary vascular resistance was calculated while the lungs were ventilated with air and the bronchial blood contained oxygen ($PbrO_2$) at 13 ± 1, 26 ± 1, 42 ± 1, 56 ± 2, 77 ± 3 and 91 ± 4 mm Hg selected in random order. Data analyzed by ANOVA.

Results. During ventilation with air the alveolar, arterial and mixed venous oxygen and carbon dioxide tensions, cardiac output, pulmonary artery occlusion pressure, airway pressure and temperature were maintained constant. As the $PbrO_2$ was decreased the pulmonary vascular resistance increased until at $PO_2$ of about 50 mmHg when the resistance decreased as $PO_2$ was reduced further. (See Figure)

Conclusion. Systemic arterial hypoxemia induces a bimodal change in pulmonary vascular resistance. We hypothesise that hypoxic pulmonary vasoconstriction is responsible for the constriction, which is opposed at the lower oxygen tension by the normal mediators of systemic hypoxic vasodilation diffusing into the pulmonary smooth muscle from the vasa vasorum. This influence may be important in all forms of systemic hypoxemia, in lung transplantation and in comparing data from in vivo and vitro lungs.




This study supported in part by NIH GM29628.

References

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2. Heistad DD et al, Am J Physiol 250: H434, 1986.

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TITLE: ENDOTHELIAL CELLS ARE NOT REQUIRED FOR THE HPV RESPONSE IN ISOLATED PULMONARY ARTERIES.

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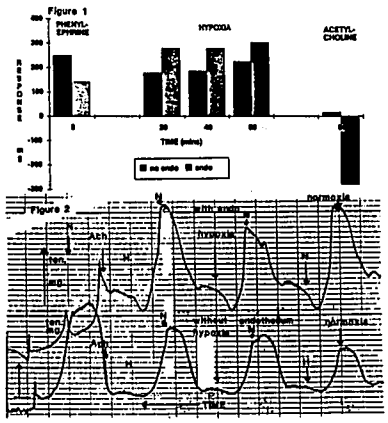
A controversy has developed as to whether[1,2] or not [3,4] the hypoxic pulmonary vasoconstrictor (HPV) response is dependent on the presence of endothelial cells(EC).

Methods. Adult female cats were anesthetized, exsanguinated and the lungs were removed. Using a microscope the intrapulmonary vessels were dissected out. These vessels were then cut into 2.5mm length,segments of equal width were paired and one of the vessels had endothelial cells removed by abrasion. Two wires were inserted into the lumen of the vessels, one wire was attached to an "L" shaped stationary holding device and the other wire was attached to a force displacement transducer. The vessels were placed in a chamber containing Earle's balanced salt solution gassed with 30%$O_2$,5%$CO_2$ balance $N_2$. Tension on the vessels was increased to a predetermined value over a 90 min stabilization period. The vessels were washed 3 times and phenylephrine was added to the bath (final concentration $1*10^{-5M}$). Increases in tension were measured in mg and were recorded on a Kipp and Zonen recorder. The gas mixture was changed to 5%$CO_2$ balance $N_2$, for a 10 min period, at which time the bath $PO_2$ was approximately 21mmHg. The gas was returned to the 30%$O_2$ mixture for 10 min. This pattern of hypoxia, normoxia was repeated 3 more times. During the last hypoxic challenge acetylcholine(Ach) was added to the bath ($1*10^{-5M}$)to check for the presence of EC.

Results. In those vessels (n=12) with no endothelium the constrictor response to PE was more rapid and larger (mean 294±45mg) compared to with endothelium (mean 141 ± 20 mg) (n=12). In the presence or absence of endothelium all the vessels responded by constricting to hypoxia. The result of the responses to PE, hypoxia and Ach. are summarized in fig. 1. The response to Ach in those vessels with EC was a significant dilatation while those with no EC did not, a typical response is shown in fig. 2. EC are not required for HPV.




Conclusion. EC may modulate the HPV and PE response but is not essential. This work supported in part by NIH GM29628.

References. 1. Johns et al., Circ Res 65:1508-1515, 1989. 2. Brashers VL et al., Clin Invest 82:1495-1502, 1988. 3. Maxson RE: Am Rev Respir Dis 139:A54,1989. 4. Marshall C et al., Anesthesiology, in press.