Mechanisms controlling airways circulation during exercise

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It has been speculated for many years that airways hyperaemia induced by exercise may be important in airways obstruction. We have reported in sheep exercising at 2.2 mph instrumented at prior thoracotomy under thiopentone (15 mg/kg i.v.)/isoflurane (2-3%) general anaesthesia with a pulsed Doppler probe mounted on the bronchial artery, and aortic pressure (Pa) catheter in superficial cervical artery[1], that during the initial, steady-state (up to 1 min 40s), and recovery phases (up to 7 min), the bronchovascular bed constricts, or is maintained, rather than dilates. The effects are intensity related; at 4 mph flow (Qbr) and conductance (Cbr) fall further to 65% and 56%, respectively ($P<0.01$). In 5 sheep we analysed the 2.2 mph responses within sheep using pharmacological block of alpha-adrenoceptors, or cholinoceptors, or both receptor groups combined. In receptor-intact sheep aortic pressure (AoP) rose from104 to 110mmHg, HR from 85 to 135 bpm, while Qbr and Cbr fell to 85% ($P<0.01$) and 81% ($P<0.001$), respectively. Following cholinoceptor block, AoP fell early then recovered, but Qbr and Cbr did not change. Following alpha-adrenoceptor block, AoP did not change but Qbr and Cbr both fell to 85% ($P<0.05$). In combined block, AoP fell slowly and although Qbr also fell slowly to 83% ($P<0.01$), the small average fall in Cbr was not significant. In this combined block state, however, the fall in bronchial flow is due to pressure gradient reduction secondary to a fall in AoP, rather than to downstream pressure rises in right atrium, pulmonary artery or pulmonary capillary/vein/left atrium[2]. Thus during and after exercise cholinoceptor vasoconstriction dominates and limits bronchovascular dilatation and airway obstruction.

1. Parsons et al., Amer. J. Resp. Crit. Care Med. 2002, 165: A339.
2. Quail et al., Arch Physiol Biochem, *in press*.

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