Autonomic control of airways vascular and wall smooth muscle during exercise

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We defined autonomic nerves regulating bronchial blood flow (Qbr) and airway dimensions during exercise and recovery. Nine ewes (34-44 kg) trained to exercise on a treadmill were anaesthetized with intravenous thiopentone, 15 mg/kg, intubated with a cuffed endotracheal tube and ventilated with oxygen and isoflurane 2–3%. At left thoracotomy a custom-built, lightweight pulsed Doppler flow probe was mounted on the bronchial artery to measure bronchial flow. Airway dimensions i.e. airway circumference (Circbr) and wall thickness (Thbr), were respectively measured using transit-time sonomicrometer crystals attached to each side of the left main bronchus, and a single crystal tissue thickness sonomicrometer placed on the external bronchial wall. Blood pressures were measured using catheters placed in superficial cervical vessels. On recovery Group 1 (N = 5) underwent moderate exercise of 3.5 km/h, before and after cholinoceptor block; Group 2 (N = 4) underwent strenuous exercise of 7.0 km/h before and after cholinoceptor plus α-adrenoceptor block. In Group 1, Qbr and bronchial blood flow conductance (Cbr) fell to 83% ($P<0.001$) before returning toward resting values, and fell again on exercise cessation to 89% ($P<0.01$). Prior cholinoceptor blockade abolished the fall during exercise in Qbr and Cbr, but not the fall in recovery, and in late recovery Cbr rose progressively ($P<0.05$). In Group 2, Qbr and Cbr fell to 68% and 54% ($P<0.001$), respectively, and fell again in recovery. Circbr fell immediately and remained at 93% ($P<0.01$), and did not recover fully. Prior cholinoceptor/α-adrenoceptor block abolished Cbr fall and Circbr shortening during and after exercise. Therefore during central autonomic resetting in exercise the primary autonomic effect is vagus mediated constriction of airways and Cbr. In recovery sympathetic nerves constrict Cbr, and vagal activity restrains bronchial hyperaemia and delays return of airway dimensions.