Does Muscle Metaboreflex-Induced Coronary Vasoconstriction Limit Increases in Ventricular Performance During Dynamic Exercise?

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Muscle metaboreflex activation (MMA) during dynamic exercise induces a substantial increase in cardiac work and oxygen demand via a significant increase in heart rate, ventricular contractility and afterload. This increase in cardiac work provides a potent stimulus for coronary metabolic vasodilation. However, with MMA little if any coronary vasodilation is observed due to concomitant neurogenic vasoconstriction by the increased cardiac sympathetic activity. It is unclear if this functional coronary vasoconstriction restrains the ability to increase left ventricular contractility. Using chronically instrumented, conscious dogs we measured arterial pressure, cardiac output (CO), left circumflex coronary blood flow (CxBF), and calculated coronary vascular conductance (CVC), maximal derivative of ventricular pressure (dp/dt), and preload recruitable stroke work (PRSW) at rest and during mild exercise (2mph) before and after MMA. Experiments were repeated after alpha-1 adrenergic blockade (prazosin ~50 μg/kg). Metaboreflex activation after alpha-1 adrenergic blockade caused significantly greater increases in CVC, CxBF. With the increased coronary vasodilation, significantly greater increases in CO, left ventricular dP/dt, and PRSW were observed during MMA. We conclude that the increase in cardiac sympathetic activity with metaboreflex activation limits coronary vasodilation which functionally restrains further increases in left ventricular contractility.

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