**KL-N(1)** Respiratory muscle fatigue affects circulatory regulation during exercise

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High intensity whole body exercise elicits respiratory muscle fatigue. In healthy humans, this exercise-induced respiratory muscle fatigue does not limit the hyperventilatory response throughout exercise in healthy humans. However, it is thought that the fatiguing respiratory muscle affects circulatory regulation during exercise.

Inspiratory muscle fatigue causes a increase in muscle sympathetic nerve activity with a corresponding increase in arterial blood pressure. This sympathoexcitation occurs through respiratory muscle fatigue-induced metaboreflex. It is likely that blood flow and oxygen transport to the working limb are reduced, thereby exacerbating limb fatigue and compromising exercise performance. Clinically, respiratory muscle work could play a particularly important role in determining oxygen transport, limb muscle fatigue and, hence, exercise tolerance in patient with chronic obstructive pulmonary disease.

**KL-N(2)** Regulations of exercising muscle blood flow and arterial blood pressure - contributions of neural cardiovascular regulations –

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The systemic cardiovascular regulations well accord with metabolic demands in active skeletal muscles is critical for performing continuous exercise. Peripheral blood flow capacity within active muscle can exceed the maximal cardiac output. Therefore, some system of regulation must be interposed between the heart and the periphery to maintain homeostasis during maximal whole-body dynamic exercise. It has been postulated that the sympathetic nervous system serves to control peripheral vascular conductance and acts to prevent cardiac function from being outstripped by peripheral vasodilation. The accumulation of metabolites in active muscles causes vasodilation, while the metabolites trigger the muscle metaboreflex, thereby increasing sympathetic nerve activity. In that context, the muscle metaboreflex may play a pivotal role as a counterbalance to metabolic vasodilation. In addition, the arterial baroreflex-mediated sympathoexcitation and vasoconstriction are enhanced during activation of the muscle metaboreflex. Such modification of the arterial baroreflex function ought to be an excellent defense against systemic hypotension induced by metabolic vasodilation. Interaction between the arterial baroreflex and muscle metaboreflex would thus provide an important functional link between metabolism within active muscles and blood pressure control, which would contribute to cardiovascular regulation during exercise.

Key words: muscle metaboreflex, arterial baroreflex, integrated circulatory regulation.