Neural control of blood flow to a contracting muscle: roles of central command and metaboreceptors

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There is a widespread view that sympathetic activity to contracting skeletal muscle increases, at least in part, as a function of intensity-related activation of a metaboreflex from within that muscle. However, to a large extent this view is based on evidence of sympathetic nerve recordings to inactive muscle (Victor *et al*., 1987; Mostoufi-Moab *et al*., 2000). We tested the hypothesis that muscle vasoconstrictor drive to the contracting muscle decreases during the contraction (functional sympatholysis) but increases during a period of post-exercise ischaemia (PEI) due to the metaboreflex.

Muscle sympathetic nerve activity (MSNA) was recorded in six subjects *via* tungsten microelectrodes inserted percutaneously into muscle fascicles of the common peroneal nerve. Total MSNA was calculated as the product of bursts per minute and mean burst amplitude, and expressed in arbitrary units. Subjects performed 2 min static dorsiflexions of the ankle at 10%, 30% and 50% of maximal voluntary force, with and without muscle ischaemia. Ischaemia was produced by inflation of a sphygmomanometer cuff around the thigh to 200 mmHg for the second minute of contraction and a subsequent 2 min period of rest (post-exercise ischaemia). For each intensity the change in total MSNA during each minute of contraction was determined relative to the rest period immediately prior to exercise. There was a significant effect of intensity on total MSNA ($P<0.05$), with increases (mean ± SE) during the first minute of 5 ± 2 units for the 10% contraction, 18 ± 6 units for the 30% contraction and 31 ± 10 units for the 50% contraction. Total MSNA during the second minute of contraction for the control and ischaemic conditions was not significantly different from MSNA during the first minute of these conditions ($P=0.50$). In both conditions, MSNA returned to baseline levels during the first minute of recovery. This decrease persisted throughout PEI, and an increase in MSNA levels was observed immediately following the release of ischaemia.

These data suggest that there is an intensity-dependent increase in MSNA to the contracting muscle, with central command playing a dominant role in this response. The fall in MSNA following exercise in the presence of ischaemia is in contrast to the effect reported previously in *non-contracting* muscles, in which PEI causes MSNA to remain elevated (Victor *et al*., 1987). This may suggest more complex interactions involving the metaboreflex mechanism and its role in the control of blood flow to the contracting muscle.

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Mostoufi-Moab S, Herr MD, Silber DH, Gray KS, Leuenberger UA, Sinoway LI. (2000), *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology*, **279:** 478-83.