Control of muscle sympathetic nerve activity to contracting muscle in humans: contributions of central command and peripheral feedback

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Purpose. The contribution of central command and the mechanoreflex in controlling muscle sympathetic nerve activity (MSNA) to a contracting limb during exercise is uncertain, as most experiments have been based on recordings of MSNA to inactive muscle. We recently showed that MSNA to the contracting muscle increases (Boulton *et al.*, 2014). Here, we tested the hypothesis that muscle vasoconstrictor drive to contracting skeletal muscle is not increased during electrical stimulation of the muscle, in which peripheral feedback is present but there is no central command to the muscle, but is increased during a voluntary contraction, when both central command and peripheral inputs are present.

Methods. MSNA was recorded in seven subjects *via* a tungsten microelectrode inserted percutaneously into a muscle fascicle of the left common peroneal nerve. Electrical stimulation (25 Hz for one minute) was performed in the *tibialis anterior* muscle through a microelectrode inserted at the motor point of the muscle. Subjects performed a series of one-minute isometric dorsiflexion contractions of the ankle (5-10% of their maximal voluntary force). A two-minute rest separated each contraction. Subjects alternated between electrically stimulated and voluntary isometric dorsiflexion contractions of the ankle for up to 10 repetitions of each. The intensity of voluntary contractions was matched to the intensity achieved during electrical stimulation. Cardiac modulation of MSNA was quantified from cross-correlation histograms constructed between the sympathetic spikes and cardiac cycles.

Results. Compared with MSNA at rest there was a significant increase (mean ± SE) of 150 ± 7% (P<0.01) in MSNA during voluntary contraction, but no change (P=0.29) during electrically stimulated contractions. MSNA during voluntary contraction was 149 ± 9% greater (P<0.01) than MSNA during electrically stimulated contractions.

Conclusions. We conclude that central command is the primary mechanism responsible for the increase in MSNA to a contracting limb during low-intensity sustained isometric lower limb exercise. Peripheral inputs appear not to contribute to the increase in MSNA to the contracting limb during exercise.

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