

Functional sympatholysis is a time- and intensity-dependent process in the contracting human calf muscle

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Repeated muscle contractions inhibit the sympathetically-mediated vasoconstriction within skeletal muscle that is observed at rest (*i.e.* functional sympatholysis). However, the extent to which this process evolves as a function of time and intensity of contraction is not clear.

To test this effect in human muscle, six men (24.8 ± 4.4 y) performed calf exercise at two intensities (25 and 50% maximum voluntary contraction (MVC)) and two levels of sympathetic activation (cold pressor test = CPT; control = CON). Calf exercise consisted of intermittent contractions of the left calf muscle (1 s contraction, 2 s relaxation). Four, 5-minute trials of calf exercise were performed under each of the four conditions of intensity and sympathetic activation. These 16 trials were performed over two days, 25% MVC trials were performed before 50% MVC trials, and the order of CPT and CON trials was counterbalanced. CPT involved immersing the right hand in an ice-cold slurry two minutes before exercise and throughout exercise, whereas for CON the hand was immersed in tepid water. Mean arterial pressure (MAP) and leg vascular conductance (LVC = leg blood flow/MAP) were measured at 20 s intervals during rest and 3 s intervals during exercise. Exercise LVC responses were averaged from the four trials under each condition, and the amplitudes, time constants and time delays of the four phases (growth and decay) that comprised this response were estimated using curve-fitting and empirical modeling (Reeder & Green, 2012).

At rest, MAP was greater ($P \leq 0.05$) during CPT than CON at 25% MVC (134.7 ± 6.5 vs 111.0 ± 6.8 mm Hg) and 50% MVC (131.0 ± 10.5 vs 111.2 ± 5.5 mm Hg). MAP remained unchanged throughout exercise during CPT at both intensities, whereas it increased significantly during CON to end-exercise values that were either lower than CPT at 25% MVC (117.8 ± 6.2 vs 134.1 ± 9.0 mm Hg; $P \leq 0.05$) or similar to CPT at 50% MVC (122.3 ± 6.3 vs 126.8 mm Hg). At rest, LVC was similar between CPT and CON at 25% MVC (0.023 ± 0.009 vs 0.022 ± 0.006 ml·100 ml⁻¹·min⁻¹·mm Hg⁻¹) but greater at 50% MVC (0.030 ± 0.013 vs 0.020 ± 0.006 ml·100 ml⁻¹·min⁻¹·mm Hg⁻¹; $P \leq 0.05$). At 25% MVC, the increase in LVC by the end of exercise was lower during CPT than CON at 25% MVC (0.109 ± 0.023 vs 0.125 ± 0.018 ml·100 ml⁻¹·min⁻¹·mm Hg⁻¹; $P \leq 0.05$) and this effect was due to the significant damping effect of CPT on the amplitude of the fast growth phase (0.091 ± 0.036 vs 0.128 ± 0.024 ml·100 ml⁻¹·min⁻¹·mm Hg⁻¹; $P \leq 0.05$). At 50% MVC, there was no significant difference between the change in LVC from rest to the end of exercise between CPT and CON (0.225 ± 0.037 vs 0.227 ± 0.023 ml·100 ml⁻¹·min⁻¹·mm Hg⁻¹), despite a significant blunting of the fast growth phase by CPT. At 50% MVC, CPT reduced the amplitude of the fast decay phase ($P \leq 0.05$), tended to reduce the amplitude of the slow decay phase ($P = 0.1$), and reduced the sum of the amplitudes of both decay phases ($P \leq 0.05$).

These results demonstrate that sympathetic activation prior to and during exercise damps the initial, rapid response of vasodilation (LVC) to muscle contractions at a low and high intensity. This damping was sustained at 25% MVC, suggesting that functional sympatholysis did not occur at this low intensity. By contrast, functional sympatholysis was evident at the high intensity, suggesting that it is an intensity-dependent process. Functional sympatholysis was achieved, at least partly, through the reduction in the amplitude of the rapid decay phase, demonstrating that it is initiated soon after the rapid growth phase (at ~10 s) and implicates mechanisms associated with it, such as the myogenic response.

Reeder, E.J., Green, S. (2012). Dynamic response characteristics of muscle hyperaemia: effect of exercise intensity and relation to EMG activity. *European Journal of Applied Physiology* doi 10.1007/s00421-012-2362-4.