The following letter is in response to the Point:Counterpoint “Increased mechanoreceptor/metaboreceptor stimulation explains the exaggerated exercise pressor reflex seen in heart failure”.

To the Editor: Debate, by its nature, serves to polarize rather than seek truth or consensus (3, 6). The mechanoreceptor reflex contributes importantly to, but cannot fully explain, the exaggerated sympathoneural response to forearm exercise observed in human heart failure, for if it did, muscle sympathetic burst frequency would increase within the first minute of isotonic handgrip, but does not and would not remain elevated during post-handgrip ischemia, yet it does—two observations that depend critically on the severity of heart failure as assessed by peak oxygen uptake, not ejection fraction (4). Middlekauff et al. (2) do not report burst frequency; they opine that arousal may also stimulate the neural response to their passive exercise. The actions of adenosine, a metabolic by-product of muscle contraction known to stimulate type III and IV muscle afferents, can be blocked by caffeine infusion at low dose (4 mg/kg). The hypothesis that such adenosine receptor antagonism affects preferentially the metaboreflex, not the mechanoreflex, of heart failure patients is supported by experiment. Increases in sympathetic burst frequency from rest were still significant within the second minute of handgrip, but caffeine abolished the sympathoneural excitation elicited by posthandgrip ischemia in the control state (5). Thus, in both experimental canine (1) and human heart failure (2, 4, 5), both metaboreceptors and mechanoreceptors are tonically active, stimulated by ischemic or nonischemic exercise at a lower threshold, and elicit greater increases in muscle sympathetic burst frequency than in healthy controls. Discordance of published findings may reflect differences in the people or experimental models studied.

REFERENCES


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