Mechanoreceptors and central command

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STATIC EXERCISE INCREASES mean arterial pressure, heart rate, and ventilation. Central command and the exercise pressor reflex are the two neural mechanisms causing these responses to static exercise. Central command is defined as the parallel activation of the locomotor and autonomic circuits in the central nervous system that simultaneously increase motor activity as well as arterial pressure, cardiac rate, and ventilation (19). The exercise pressor reflex, the second mechanism, is evoked by the contraction-induced stimulation of thin fiber (i.e., group III and IV) muscle afferents (7).

The concept of the exercise pressor reflex has been evolving. Until recently, the thin fiber afferents evoking the reflex were thought to provide an error signal in the muscle oxygen/supply to the exercising muscles was not adequate to meet the metabolic demand of muscles (11, 13). Consequently, the exercise pressor reflex was believed to be evoked by metabolites produced by contraction, their concentration increasing in the muscle interstitial space when there was an imbalance between blood supply and demand. The increase was thought to stimulate thin fiber afferents, whose endings were also in the interstitial space of muscle. The evidence is strong that metabolites produced by contraction evoke the exercise pressor reflex. Nevertheless, the possibility that mechanical stimuli also contributed to the elicitation of the reflex was largely ignored by exercise physiologists.

Two reasons may explain why this was the case. First, there was no conceptual need to consider mechanoreception because central command was believed to be responsible for the initial cardiovascular response to exercise. Second, interaction between exercise physiologists and neurophysiologists investigating somatosensory input was limited.

One important neurophysiological contribution that should have influenced the thinking of exercise physiologists was that by the late A. S. Paintal (12), who in 1960 showed that some group III afferents in canine hindlimb muscle responded to both light nonnoxious probing of their receptive fields as well as to contraction. Unfortunately, these findings had little impact on the thinking of exercise physiologists, even though group III muscle afferents comprised part of the afferent arm of the exercise pressor reflex arc (8). Likewise, the finding in humans that involuntary contraction reflexively increased heart rate (6) within a cardiac cycle or two received little attention.

There were other findings, in humans, that deterred exercise physiologists from considering thin fiber mechanoreceptors as part of the afferent arm of the exercise pressor reflex arc. For example, exercise increased muscle sympathetic nerve activity, but only after a latency of 30–60 s (14, 17). This latency was consistent with the notion that the reflex muscle sympathetic nerve response to exercise was caused by the stimulation of metaboreceptors.

Recent evidence has suggested that thin fiber mechanoreceptors play a role in evoking the exercise pressor reflex. In healthy humans performing handgrip exercise, signal averaging techniques revealed an increase in muscle sympathetic nerve activity with a latency of 4–6 s, a finding consistent with the possibility that mechanoreceptors contributed to the reflex (5). Similarly, in anesthetized and decerebrated cats, contraction increased renal sympathetic nerve activity within 1–2 s (2, 18).

As the evidence cited above demonstrates, the role played by thin fiber mechanoreceptors in evoking the exercise pressor reflex has been controversial. Although one could stimulate these afferents selectively by stretching tendons (1) or by contracting muscles involuntarily by electrical stimulation (10), both maneuvers had drawbacks (4). A tool was needed to block discharge in mechanoreceptors, leaving metaboreceptor function intact. Such a tool could be used to reveal the role played by mechanoreceptors in the cardiovascular responses to exercise in an intact behaving preparation.

This tool was provided by Hayes et al. (3), who reported that injection of gadolinium into the arterial supply of hindlimb skeletal muscle greatly attenuated if not blocked the responses of group III mechanoreceptors to static contraction and tendon stretch. Gadolinium had no effect on the responses of group IV metaboreceptors to static contraction. It also had no effect on the responses of these thin fiber afferents to capsaicin. Moreover, gadolinium injection greatly attenuated the reflex pressor responses to static contraction (i.e., the exercise pressor reflex) and to tendon stretch (3, 15).

In this issue of American Journal of Physiology-Heart and Circulatory Physiology, Matsukawa et al. (7a) used gadolinium to investigate the role played by thin fiber mechanoreceptors in evoking the pressor-cardioaccelerator responses to static exercise in conscious cats. They found that gadolinium, injected intravenously, had no effect on the responses of these afferents. Matsukawa et al. concluded that central command and not muscle mechanoreceptors played the dominant role in causing the initial cardiovascular responses to exercise. As a control, Matsukawa et al. anesthetized the cats and examined the pressor response to muscle stretch, finding that gadolinium, injected intravenously in same dose as that used when the cats were exercising, attenuated the muscle mechanoreflex (16) (i.e., the pressor response to stretch).

The experiments by Matsukawa et al. shed new light on the role played by thin fiber mechanoreceptors in evoking the cardiovascular responses to exercise. They have been performed in conscious cats, and the importance of obtaining data in this preparation cannot be stressed too strongly. Very few, if any, laboratories in the world are capable of performing the elegant and difficult experiments described in this article. Clearly, the control experiments performed by Matsukawa et al. are more than adequate; the authors cannot be expected to meet the same standard as that met by investigators using anesthetized or decerebrated preparations. Indeed, reduced standards have been used for years in human studies of car-
diovascular control during exercise; the same standards should also apply to conscious animal studies.

Despite these impressive strengths, the findings reported by Matsukawa et al. (7a) still require qualification. Probably the most pressing is that gadolinium injection seemed to weaken the forelimb muscles, an effect that might require the cats to increase their central command to develop a tension similar to that developed before gadolinium injection. An increase in central command might have compensated for the reduced reflex pressor response evoked by the contraction-induced stimulation of thin fiber mechanoreceptors, the input of which was reduced by gadolinium. A second issue involves the effect of gadolinium on the discharge of the arterial baroreceptors. If these afferents were no longer capable of responding to increases in arterial pressure, such as those that occur during exercise, then the removal of muscle mechanoreceptor input might have been countered by the removal of baroreceptor input.

In closing, the findings reported by Matsukawa et al. (7a) are both interesting and impressive; they offer new insight into the role played by both central command and muscle mechanoreceptors in cardiovascular control during exercise. These findings should encourage further research; the issue is not settled. For example, the findings by Matsukawa et al. and those in humans, in whom muscle mechanoreceptors were reported to play an important role in the control of renal blood flow during rhythmic exercise (9, 10), need to be integrated into a cohesive theory.

REFERENCES