GROUPS III AND IV MUSCLE AFFERENTS CONTRIBUTE TO THE VENTILATORY RESPONSE TO EXERCISE

TO THE EDITOR: Our interpretation of Dr. White’s letter (10) is that he pretty much agrees that our findings suggest that thin-fiber muscle afferents in the human are significantly involved in the cardiovascular and ventilatory responses to rhythmic exercise (1) and one of the possible pathways for this effect is through their stimulation of ventilatory circuits in the medulla (3). Furthermore, we also agree, and already stated in our discussion (1), that muscle afferent feedback could also contribute to autonomic responses to exercise via multisynaptic pathways affecting central command in the general vicinity of the mesencephalic locomotor region (7, 9). However, Dr. White (10) claims that the cardiorespiratory effects of afferent blockade might also be explained by afferent-induced inhibitory effects on spinal motorneurons and their subsequent influence on “central command.” He cites older findings supporting the “muscle wisdom” hypothesis to support his argument. We believe the collective findings on this question do not support such a straightforward interpretation.

Several recent studies in humans have investigated motorneuronal excitability utilizing stimulation of the corticospinal tract at the cervicomedullary junction in order to evoke EMG responses, i.e., cervicomedullary evoked potentials (CMEPs). Taylor et al. (8) compared pre- to postexercise CMEPs and found them to be reduced immediately following a fatiguing elbow flexor muscle contraction but to recover within 2 min postexercise. However, following an identical exercise routine, muscle afferent activity was maintained via cuff inflation trapping intramuscular metabolites and the recovery of the exercise-induced CMEP was unchanged from control conditions (8). Nonetheless, effects on α-motorneuronal excitability appear to differ depending on the stimulus used to trigger the central projection of muscle afferents and/or the type of motorneurons studied. For example, Martin et al. reported that postexercise ischemia increased excitability of those motorneurons innervating elbow flexors but inhibited those innervating elbow extensors (5). Further, if the muscle afferents were stimulated by intramuscular hypertonic saline infusion into the rested muscle, both extensor and flexor muscle motor neurons were facilitated (6). Direct neuronal recordings in anesthetized cats show that chemical stimulation of group III/IV muscle afferents hyperpolarized some motorneurons and depolarized others (4). Finally, we note that all of the human studies employed fatiguing, ischemic, isometric contractions and/or artificially stimulated muscle afferents, which might or might not simulate the effects of rhythmic, nonfatiguing exercise of both flexor and extensor muscles as we employed in our study.

In conclusion, we believe the evidence to date to be inconclusive concerning the precise direction and magnitude of action of muscle afferents on α-motorneurons during rhythmic exercise (see also p. 1746–1748 in Ref. 2). More importantly, our findings showed that blockade of group III/IV muscle afferents clearly attenuated the ventilatory and cardiovascular responses to exercise (1). Accordingly, we can conclude that exercise-induced stimulation of these afferents contributes significantly to the hyperpnea regardless of whether this contribution is via stimulation of cardiorespiratory neuronal circuits in the medulla or via afferent influences on motorneuronal excitability or both. Either way, the afferents are the initiating mechanism! So, we believe the answer to Dr. White’s question is in the affirmative … at least until someone provides the next series of experiments on this elusive mystery of exercise hyperpnea.

DISCLOSURES
No conflicts of interest, financial or otherwise, are declared by the author(s).

REFERENCES
10. White MJ. I’d like it to be true, but do group III and IV muscle afferents really contribute to the ventilatory response to exercise? J Appl Physiol. 10.1152/japplphysiol.01443.2010.