Last Word on Point: Counterpoint: Afferent feedback from fatigued locomotor muscles is not an important determinant of endurance exercise performance

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TO THE EDITOR: This Point:Counterpoint is about endurance performance, not central motor drive (CMD) or cardiorespiratory regulation. Therefore, the right question I have asked is whether afferent feedback from peripheral metaboreceptors (AF) affects conscious self-regulation of power output during time trials (1). Current experimental evidence suggests it does not. In fact, spinal blockade of AF does not affect average power output (fentanyl trial) unless it is complicated by a reduction in locomotor muscle strength (lidocaine trial) and consequent reduction in endurance performance (4). This is why I focused on the two spinal-blockade studies.

The inhibitory AF model proposed by Amann and colleagues (Fig. 1, Ref. 1) cannot even predict pacing strategy, i.e., changes in power output during time trials. According to this physiological model of endurance performance, CMD and power output should be high at the beginning of the time trial (low inhibitory AF) and low near the end (high inhibitory AF). On the contrary, most endurance athletes choose a conservative start and "turn on a turbo" (i.e., increase CMD to recruit additional motor units and increase power output) near the end of a race. Clearly, cognitive/ motivational factors that do not have anything to do with AF (e.g., previous experience and knowledge that the end of the race is near) have a stronger influence on conscious self-regulation of power output during endurance competitions! As discussed in my rebuttal, psychological factors also provide a plausible explanation for the paradoxical pacing strategy observed during the fentanyl trial (1).

Another important question is whether AF is one of the sensory signals processed by the brain to generate perceived exertion. Amann, Secher, and several commentators dogmatically believe that it is (1, 7), while I reviewed strong evidence suggesting that the primary sensory signals for perception of effort are corollary discharges from premotor and/or motor areas of the brain (3). I was surprised by Secher’s position on perceived exertion because he has used RPE as a subjective index of CMD during exercise for over 20 years! In fact, he demonstrated that blockade of AF does not reduce RPE even when the compensatory increase in CMD caused by lidocaine induced muscle weakness is eliminated by exercising at the same relative workload (5). This experimental finding provides strong evidence that AF is not perceived as effort during exercise.

The fact that perceived exertion is based on corollary discharges of CMD does not mean that what occurs in the periphery can not affect this conscious sensation. For example, locomotor muscle fatigue increases perception of effort because subjects have to increase CMD to cycle at the same power output with weaker locomotor muscles (4). Furthermore, perceived exertion can change without any change in CMD if neurocognitive processing of corollary discharges is affected by, for example, attentional focus (2).

In conclusion, I would like to clarify an important issue: I did not propose that psychological factors also contribute to the complex determination of endurance performance (physiological factors—endurance performance—psychological factors). My psychobiological model postulates that conscious self-regulation of power output during time trials is directly determined by cognitive/motivational factors only (1). Physiological factors (e.g., locomotor muscle fatigue) affect endurance performance indirectly if they have a significant effect on perceived exertion or the other psychological factors on which the psychobiological model is based (physiological factors—psychological factors—endurance performance). This new psychobiological model provides a single explanation for all aspects of endurance performance instead of relying on several different physiological and psychological models (6).

REFERENCES


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