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COUNTERPOINT: AFFERENT FEEDBACK FROM FATIGUED LOCOMOTOR MUSCLES IS NOT AN IMPORTANT DETERMINANT OF ENDURANCE EXERCISE PERFORMANCE

Since Noakes’ controversial lecture on the central governor (23), brain regulation of endurance performance has been a hot topic in exercise physiology. In this series, Dr. Markus Amann argues that afferent feedback related to peripheral locomotor muscle fatigue is an important determinant of endurance performance. I have two objections to this hypothesis. First, there is no experimental evidence to support it. In fact, in two separate studies, spinal blockade of inhibitory afferent feedback from group III-IV receptors sensitive to fatigue-inducing metabolites did not improve performance in 5-K cycling time trials (5, 6). Actually, in the epidural lidocaine study, there was a significant reduction in time-trial performance (5). Amann and colleagues proposed that this net negative effect is the product of two contrasting effects of epidural lidocaine on exercise performance: 1) the negative effect mediated by the iatrogenic reduction in locomotor muscle strength, and 2) the positive effect of blocking somatosensory feedback from the legs. However, reduced locomotor muscle strength per se has a small effect on endurance performance (21). Therefore, if Amann and colleagues’ proposition is true, afferent feedback from fatigued leg muscles must have an even smaller and, thus, negligible effect on exercise performance.

My second objection to this supraspinal reflex inhibition model of endurance performance (1, 3, 4) is more theoretical. This feedback loop (Fig. 1) is attractive to physiologists because they are familiar with subconscious autonomic regulation (e.g., the exercise pressor reflex) and the mechanisms of central fatigue during maximal voluntary contractions (26). However, it is not a valid representation of what happens during the time trials chosen by Amann and colleagues to test their hypothesis (5, 6). In fact, during endurance exercise, voluntary muscle contractions are always submaximal (8) and, therefore, central fatigue is not a relevant concept (19, 26). Furthermore, during time trials, power output is consciously self-regulated by the subject, e.g., by changing gears. These are not trivial details (2), but considerations that suggest a more relevant question: does afferent feedback from fatigued locomotor muscles affect the brain processes determining conscious self-regulation of submaximal power output during time trials?

To answer this question, it is important to remind ourselves that voluntary actions like cycling as fast as possible for 5-K are the opposite of simple reflexes (13). This is why the feedback loop proposed by Amann and colleagues (1, 3, 4) can not be an adequate model of endurance performance. I propose a different approach based on the principle that conscious self-regulation, like other mental phenomena, is caused by lower-level neurobiological processes in the brain (25). Therefore, time-trial performance can be understood in psychological terms before investigating the neurobiology underlying the relevant constructs. This is why I call it the psychobiological model of exercise performance (21).

According to this model, conscious self-regulation of submaximal power output during time trials is determined primarily by the following cognitive/motivational factors:

1) Perception of effort;
2) Potential motivation;
3) Knowledge of the distance to cover;
4) Knowledge of the distance covered/remaining;
5) Previous experience/memory of perceived exertion during exercise of varying intensity and duration.

So the question now is: does afferent feedback from fatigued locomotor muscles affect these psychological constructs?

Cognitive factors 3–5 are self-explanatory, and we can assume that somatosensory feedback from the legs does not affect them. The term potential motivation refers to the maximum amount of effort that a subject is willing to exert to succeed in a task, and it varies with factors traditionally associated with motive strength, including the need for the available incentive and the value of that incentive (29). For example, in elite athletes, potential motivation would be higher when competing for an Olympic medal than during smaller-scale competitions. Again, afferent feedback related to peripheral locomotor muscle fatigue is unlikely to affect these complex psychosocial factors. This is not to say that pain generated by stimulation of group III-IV afferents in locomotor muscles does not have any motivational function. For example, severe pain would strongly motivate people to stop exercise when suffering a muscle strain. However, the leg muscle pain normally experienced during high-intensity aerobic exercise does not limit performance in healthy humans (9).

This leaves us with perception of effort, i.e., the conscious sensation of how hard, heavy, and strenuous a physical task is. The observation crucial to my Counterpoint is that, unlike pain, perceived exertion during cycling exercise is not reduced by spinal blockade of somatosensory feedback from the legs (10, 12, 15), even in conditions of high metabolic stress (17). These consistent experimental findings from various laboratories provide strong evidence that afferent feedback from fatigued locomotor muscles does not contribute significantly to perception of effort during high-intensity endurance exercise. Therefore, no change in overall time-trial performance should be expected when subjects are treated with intrathecal fentanyl, an anesthetic that does not induce leg muscle weakness.

When locomotor muscle strength is reduced by epidural anesthesia with lidocaine (10, 12, 15, 17), a compensatory increase in central neural drive to the leg muscles is required to cycle at a given submaximal power output (21). This increase in central motor drive is perceived, through its corollary discharge to sensory areas of the brain (20), as an increase in effort (10, 12, 15, 17). Therefore, lidocaine treatment should reduce time-trial performance. Importantly, these two predictions (19) have been confirmed by the spinal blockade studies performed by Amann and colleagues (5, 6). Therefore, these experiments provide support to my psychobiological model in which afferent feedback related to peripheral locomotor muscle fatigue does not play any significant role in regulating endurance performance.

Importantly, psychology provides plausible explanations for phenomena not understandable or even incompatible with the supraspinal reflex inhibition model of endurance performance (1, 3, 4). A good example is the “end spurt,” i.e., the increase in central motor drive/power-output measured at the end of intense time trials despite a very high concentration of fatigue-inducing metabolites (5–7). This phenomenon clearly shows that conscious self-regulation is more important than inhibitory afferent feedback from locomotor muscles in determining power output and performance. I suggest that this pacing strategy is chosen by most athletes because it is difficult to accurately predict at the beginning of the race how perception of effort will develop during a time trial (18). Therefore, because finishing the race is paramount, it is wise to choose a slightly conservative pace. Only near the end of the time trial, when predictions are more reliable (18), athletes know they can increase power output without risking premature exhaustion. Other examples are the different pacings at which athletes start different endurance competitions (16), the effects of severe hypoxia (7), mental fatigue (22) and social facilitation (28) on endurance performance, learning of the pacing strategy (11), and performance during low- to moderate-intensity exercise (16). The cognitive/motivational factors listed above can explain these phenomena, while afferent feedback related to metabolic fatigue can not.

Because of its predictive and explanatory power, physiologists interested in endurance performance may want to consider this alternative psychobiological model based on the principle elegantly described by Ikai and Steinhaus in this very journal almost 50 years ago: “Psychology is a special phase of brain physiology” (page 157, Ref. 14). This principle is especially relevant now that we have the technology to investigate in humans the neural correlates of psychological constructs relevant to sport performance (24, 27, 30).

REFERENCES

3. Amann M, Dempsey JA. When fatiguing cycling muscles complain, the brain insightfully responds! Physiology News 75: 13–14, 2009.


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REBUTTAL FROM AMANN AND SECHER

Dr. Marcora’s writing fails to polarise this discussion by lacking clean arguments/evidence supporting his side of the debate (i.e., muscle-afferents do not affect endurance performance). Instead, he unfortunately narrows his focus on our blocking studies (1, 2), which are, due to the detrimental concomitant impact of the spinal block on motor-unit recruitment and/or cardiorespiratory reflexes, not suitable to address peak-performance questions. These studies offer useful data to evaluate the effects of muscle afferents on CMO and the brain’s disposition to tolerate peripheral fatigue but not on exercise performance. Finally, he entirely ignores the vital role of muscle afferents in determining cardiorespiratory responses to exercise and associated consequences for performance.

However, our “antagonist” asks the right question: “do locomotor muscle-afferents affect the conscious self-regulation of CMO during time-trial exercise?” Although our work did not show improved performance in the absence of afferent feedback, it clearly answers his question by showing that CMO during time-trial exercise is upregulated in the absence of muscle afferents (1, 2).

We can only agree that psychological factors also contribute to the complex determination of the magnitude of CMO during time-trial exercise and thereby presumably to exercise performance. However, our opponent’s “psychobiological model” is highly disputable since he dismisses the fact that sensory-feedback contributes, presumably also through its indirect projection into the anterior cingulate cortex (3, 9), to central fatigue and effort perception (4, 5). Or opposition justifies his claim by comparing effort perceptions during control constant-load exercise with those during the identical exercise performed with local anesthetics (CMO increased, afferent-feed-back decreased). This is inappropriate since effort perception is likely affected by CMO and muscle afferents.

The ability to sprint at the end of a race varies considerably among athletes. The explanation is that endurance exercise depends mainly on activation of slow-twitch fibers as illustrated by their glycogen depletion, while there may be no, or just little, glycogen depletion in fast-twitch fibers (10). Accordingly, those athletes who are characterized by many, likely “trained,” fast-twitch fibers are able to turn on a “turbo,” albeit only for limited time and they need to learn for exactly how long that is. The final sprint in a long distance event is an excellent example for how the CNS takes advantage of the machinery that the muscles provide to achieve optimal performance.

We are shocked by our opponent’s statement: “... during endurance exercise, central fatigue is not a relevant concept” - especially given his recent reproduction of Angelo Mosso’s work (7) and numerous other studies (6, 8).

REFERENCES


