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

PREDOMINANCE OF CENTRAL MOTOR COMMAND IN THE REGULATION OF EXERCISE

TO THE EDITOR: Neurobiological aspects of the regulation of exercise by the central nervous system (CNS) are poorly understood and have been studied mainly to understand central fatigue (i.e., a progressive decline in the drive to motoneurons) as well as prolonged strenuous exercise in humans under conditions of hyperthermia (6). The predominance of centrally originating neural signals in the perception of voluntary muscular force has been shown by data obtained in a deafferented subject who was able to accurately discriminate isometric forces solely on the basis of internal signals (3). The contribution of afferent input in sensing effort must however not be dismissed as reinforced by Amman and Secher (1). Peripheral feedback allows to modulate and calibrate the central signal of effort (2). Also, during complete paralysis by curarization, heart rate, blood pressure, and perceived effort still increase during attempted contraction of skeletal muscles, indicating that central motor command can operate independently of somatosensory inflow to the CNS (93). Finally, although Amman and Secher (1) and Marcora (4) seem to suggest that fatigue is detrimental to performance and goal-directed behavior, this may not be the case.

Fatigue may provide the cognitive system with a signal that encourages the organism to lower present goals and/or seek lower alternative strategies. Alterations of dopamine and acetylcholine influx into the prefrontal cortex that occur with prolonged task performance is adaptive in the sense that it signals the need to abandon or change the ongoing behavior in such a way to promote energy conservation (5).

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AFFERENT FEEDBACK FROM FATIGUED LOCOMOTOR MUSCLES IS IMPORTANT, BUT NOT LIMITING, FOR ENDURANCE EXERCISE PERFORMANCE

TO THE EDITOR: We have three main observations regarding this Point:Counterpoint debate (1). First, Amann, and Secher highlighted the role of afferent feedback from fatigued locomotor muscles in cardiovascular and ventilatory control during exercise. However, the role of central control cannot be ignored (5).

Second, Amann and Secher quoted the study of Löscher and colleagues (4) that provides evidence that conflicts with the former author’s conclusion that “somatosensory feedback from locomotor muscles appears to limit performance by imposing inhibitory influences on CMO...” (1). This study (4) showed that after a period of voluntary muscle contraction until exhaustion, followed immediately by muscle stimulation, subjects were nevertheless able to continue exercising voluntarily for another 85 s. Contrary to Amann and Secher’s hypothesis, voluntary muscle contraction was not prevented even when peripheral fatigue had been further developed by muscle stimulation. Hence, the level of peripheral fatigue does not appear to be accurately regulated in order to limit exercise.

Last, we agree that the five cognitive/motivational factors proposed by Marcora (1) comprise key component determinants of endurance performance. However, in our view, afferent feedback from fatigued motor muscles is also important since it acts to increase conscious awareness of bodily “discomforts.” This mechanism is a remarkable homeostatic feature in humans (2,3) and when integrated with the other five factors, allows more appropriate behavioral decisions. We believe that the complex interaction of all these aspects is crucial for endurance exercise performance.

In conclusion, although important, afferent feedback from fatigued motor muscles is not by itself the sole factor directly limiting endurance exercise performance. Fatigue is a complex process and its understanding should not be reduced to a single isolated phenomenon.

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REFLEXES, CONSCIOUSNESS, AND EVOLUTION

TO THE EDITOR: From an evolutionary point of view, it seems bizarre that the brain would limit central motor output and, thus, exercise performance (e.g., running to hunt an animal or...
to escape from predators/enemies) when locomotor muscle fatigue reaches the “critical threshold” commonly measured after various endurance performance tests (1, 5). In fact, not having anything to eat or being killed are much worse outcomes than having locomotor muscles producing <80% of maximal voluntary force. If we consider that high-intensity aerobic exercise requires only 15–20% of maximal voluntary locomotor muscle force (4), this seems an unnecessarily conservative critical threshold.

For survival, it would certainly be more beneficial if the brain limited exercise intensity and/or duration to preserve homeostasis in more vital organs such as the heart (6), or to avoid pointless energy expenditure at times (40,000 years ago) when our basic physiology evolved and food was not easily available (3). This second proposal fits with the psychobiological model of exercise performance (5). In fact, motivational intensity theory (2) predicts that humans exert effort/expand energy only when success in the task being performed (e.g., hunting an animal or, nowadays, an endurance performance test) is perceived as possible and worthwhile.

Furthermore, conscious self-regulation of exercise performance enables people to be flexible and go beyond their usual limit when the situation (e.g., a predator/enemy running after you) requires. A system based on supraspinal reflex inhibition and a critical threshold of locomotor muscle fatigue is, on the contrary, inflexible and would be detrimental to survival.

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PSYCHOBIOLOGICAL FACTORS ARE MORE IMPORTANT THAN CENTRAL FATIGUE IN LIMITING ENDURANCE PERFORMANCE

TO THE EDITOR: Amann and Secher (3) argue that the ambulatory problems observed after the fentanyl trial demonstrate that inhibitory afferent feedback from fatigued locomotor muscles (AF) is necessary to avoid the catastrophic locomotor muscle dysfunction induced by excessive exercise. This “critical threshold” is one of the cornerstones of their inhibitory AF model of exercise performance regulation (Fig. 1 in Marcora’s Counterpoint; Ref. 3). However, after the fentanyl trial, maximal voluntary locomotor muscle strength was reduced by <15%. Clearly, 85% of maximal voluntary locomotor muscle strength is more than enough to stand up and walk normally! The ambulatory problems reported by Amann and Secher are more likely due to the central effects (sickness, confusion, and impaired movement coordination) of fentanyl (a powerful opioid) and/or poorly compensated metabolic acidosis. The presence of CNS dysfunction is confirmed by the significant central fatigue (i.e., reduced neural activation during maximal voluntary contractions) measured after the fentanyl trial despite spinal blockade of AF. Interestingly, after the control and “placebo” trials there was no central fatigue despite an intact afferent pathway and significant peripheral locomotor muscle fatigue. These results (2) directly challenge Amann and colleagues’ hypothesis that inhibitory AF related to peripheral locomotor muscle fatigue is an important cause of central fatigue and, thus, endurance performance (1, 2). As proposed by Marcora, research into the mechanisms determining endurance performance should focus less on central and peripheral muscle fatigue. Instead, we should investigate more the physiological bases of those psychological constructs known to affect endurance performance such as perception of effort (4, 5) and motivation (6).

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FATIGUE—FROM MUSCLE TO BRAIN OR VICE VERSA?

TO THE EDITOR: Fatigue can be defined in several ways and depending on the emphasis that has been taken (peripheral or central), can be explained from different angles (5). This Point/Counterpoint debate nicely illustrates that depending on the “angle” the problem is attacked, each author can find clear arguments to get his statement through (1, 3). Afferent feedback (not only from muscles) is important, but there will always be an integration of the signals at the central level, where the perception of effort is computed (5).

It is therefore not only the translation of peripheral input, but also the interpretation of incoming signals that will determine fatigue. This is illustrated by experiments in which brain neurotransmission is manipulated during exercise in the heat. When the dopaminergic system is manipulated (in human and rat models) performance is improved, fatigue is postponed.
without changes in the perception of effort, but simultaneously, core and brain temperature will increase above 40°C (2, 5, 6).

Probably both authors are right because one must not forget that we are dealing with a disturbance of homeostasis of “basic” physiological systems where integrative physiology is necessary for the interpretation of incoming peripheral signals. But in this case, also nonhomeostatic pathways will be involved in the (regulation of) effort perception.

It should be noted, however, that homeostatic and nonhomeostatic pathways are probably not two completely separate (neural) systems: significant interaction at different levels might exist (4), it might therefore be interesting approach this problem from a more “gestalt” viewpoint.

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PARADIGM SHIFT TO THE PSYCHOBIOLOGICAL MODEL

TO THE EDITOR: Physiological models can explain the average speed/power during an endurance competition (3). However, they can not explain the end-spurt and many other phenomena well known to endurance athletes and their coaches but often snubbed by exercise physiologists. For instance, a detrimental effect on endurance performance is observed under mental fatigue conditions (4). The reduced endurance performance can be explained by the increased perception of effort, probably related to the increased mental effort to cycle. It is important to notice that no musculoenergetic or cardiorespiratory changes that could explain reduced performance were detected. Conversely, listening to music at fast tempo improved cycling performance by means of increased pedal cadence. It has occurred parallel to increased heart rate, thermal discomfort, and perception of effort at isocimes when compared with slow and normal tempo of a music track (4). The greater performance while listening to the fast tempo music can be explained by change in potential motivation (1), which can influence the conscious self-paced cycling strategy. These results cannot be explained by feedback models. The psychobiological model proposed by Marcora (6) provides us with a single model that can integrate both perspectives. The influence of traditional physiological factors (e.g., VO2max and heat) can be explained by their effects on perception of effort, while the influence of psychological factors such as the presence of a competitor (5) is directly explained by motivational intensity theory. Therefore, the psychobiological model should be preferred to the supraspinal reflex inhibition model proposed by Amann and Secher (6) and other physiological models of endurance performance. We need a paradigm shift that provides us with a more integrative (psychology + biology) and powerful way of explaining endurance performance.

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THE INFLUENCE OF AFFERENT FEEDBACK, PERCEIVED EXERTION AND EFFORT ON ENDURANCE PERFORMANCE

TO THE EDITOR: As highlighted in this Point:Counterpoint debate, the relationships between psychological, neurological and biological aspects of endurance performance are extremely complex. During exercise feedback from group III/IV afferents is increased in response to elevations in intramuscular bioproducts (1). It is likely that exercise performance is in some way influenced by this afferent feedback. Indeed, blockade of ascending feedback has been shown to increase central motor drive, as well as alter self-selected pacing strategy and overall performance (2, 3). Nevertheless, the complexity of endurance performance would suggest that a single model is likely not sufficient to explain this issue. Therefore, we have presented an observation and provide an explanation below.

Perceived exertion is noted as a major factor influencing the conscious regulation of submaximal power output (5, 6). However, we would like to point out the possible oversight with regards to the interchangeable use of the terms effort and exertion in this Point:Counterpoint argument (4, 5). Perceived effort, unlike perceived exertion, may be largely centrally governed, influenced by not only sensations of discomfort but also prior experience and the ability to perform the task. During submaximal exercise, increasing afferent feedback can increase perceived exertion resulting in a decrease in intensity, without altering perceived effort. Conversely, at the end of exercise perceived effort can increase in the face of intolerable perceived exertion (“end spurt”). These observations highlight the possible integration between the central and feedback models. We suggest that both models may be involved in the regulation of pace during prolonged exercise.


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ENDURANCE EXERCISE PERFORMANCE IS DETERMINED BY BOTH AUTONOMIC AND PSYCHOLOGICAL FACTORS

TO THE EDITOR: The arguments proffered by Amann and Secher (1) as well as Marcora (4) each have merit when considering the complex factors regulating exercise performance. Clearly, acute adjustments in autonomic nervous system activity are requisite for increasing cardiac output, blood pressure, and ventilation to adequately deliver oxygen to exercising muscle. Muscle work could not be sustained at all without these cardiorespiratory adjustments. While important, afferent feedback from metabolically sensitive fibers in locomotor muscle is not the sole determinant of these autonomic changes (3). Mechanically sensitive afferent fibers in muscle, central command (feedforward central cortical control), and the arterial baroreflex all contribute significantly to autonomic regulation during exercise (2, 5, 6). The fact that these neural inputs may also inhibit central motor output during strenuous exercise is not surprising. Like many “finely tuned” processes within the body, it is logical for a system to protect its performance by inhibiting outputs that could exceed its functional capacity. However, it seems naive to suggest that human consciousness does not also play a role in limiting exercise performance. Who among us has not ended an exercise session prematurely due to perceived exhaustion or overestimated task difficulty? It is unlikely that the physiological capacity for exercise is exceeded in such situations, yet exercise performance is limited. Only through learned behaviors or during activation of instinctive survival mechanisms can the physiological limits of performance be approached. A more relevant model of the features determining endurance exercise performance would include both autonomic inputs as well as psychobiological factors.

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Point:Counterpoint Comments


TO THE EDITOR: Although the original question appeared to involve the importance of muscle afferent feedback as a determinant of endurance performance, Marcora’s case appears to rest largely on his contention that afferent feedback from fatigued locomotor muscles does not contribute significantly to perception of effort during endurance exercise (2). In general support of this contention, studies using hypnosis during steady-state exercise (3, 5) have shown perceived exertion (perception of effort) can be increased or decreased without altering the actual workload, implying no change in the afferent signals arising from the working muscles. For example, when perceived exertion is increased (above the control value for the workload), cardiovascular variables also show a concomitant increase. This indicates perceived exertion can function independent of muscle afferent feedback to regulate cardiovascular responses, and subsequently influence performance. However, when perceived exertion is decreased, with afferent feedback remaining unchanged, cardiovascular variables do not decrease in line with a lower perceived exertion. In agreement with evidence presented by Amann and Secher (1), this latter finding suggests that muscle afferent input is capable of dictating the cardiovascular response, independent of (or being of greater importance than) the perception of effort. Furthermore, studies using lower-body positive pressure also support the role of muscle afferent feedback in determining exercise performance (4). In sum, muscle afferent feedback appears to contribute importantly by driving a cardiovascular response capable of meeting metabolic demand, but the cardiovascular response can certainly be modulated by the perception of effort.

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OTHER SOURCES OUTSIDE THE MUSCLE AFFERENT FEEDBACK LIMIT LOCOMOTOR PERFORMANCE IN THE HEAT

TO THE EDITOR: In addition to the direct effects that the intramuscular accumulation of selected metabolic by-products have on muscle contractility, we agree with Amann and Secher (1) that these disturbances might also limit locomotor performance via afferent feedback from fatigued muscles. This Point:Counterpoint focuses on supraspinal modulation of the motor power output, but such muscle afferents can also act at the spinal cord level. For instance, based on H-reflex recordings, we have reported that spinal loop properties are modified under fatigue [e.g., prolonged treadmill running (5)]. Additionally, we would like to emphasize that some of the cause of central fatigue can be independent of the muscle feedback. Indeed, in some circumstances, such as exercise heat stress, hyperthermia may precede the development of significant peripheral fatigue and, therefore, determine exercise capacity. Interestingly, exhaustion when exercising in a hot environment has not been linked to a reduction in cardiac output and muscle (leg) blood flow, changes in substrate utilization and availability, or to the accumulation of recognized “fatigue” substances (4). Rather, it has been reported that both trained subjects (3) and rats (6) stop exercise at the same core temperature (40°C), regardless of the initial value and the rate at which core temperature rises. This suggests that, independently of afferent feedback, the attainment of a high body temperature (i.e., critical internal temperature concept) per se is probably a major determinant of endurance exercise performance in such conditions. Additional support for this observation comes from animal studies showing that goats reduce their walking velocity on a treadmill or refuse to move when the temperature of their brain is passively increased to about 42°C (2). In summary, depending on the severity of the environmental (e.g., heat) stress, we underline that some of the neural determinants of endurance exercise performance are independent of afferent feedback from fatigued locomotor muscles.

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LIMITATION OF PERFORMANCE AND THE BRAIN: SIMPLY MORE COMPLEX

TO THE EDITOR: Obviously exercise is function of motor unit recruitment—and derecruitment—and the brain is therefore where exercise begins, is regulated and ends (3). The question is how this happens. Studies in extreme conditions like hypoxia suggest a role for muscle afferent feedback limiting exercise intensity/duration (1). But, as also suggested by Marcora (4), other mechanisms may be involved, depending on the experimental conditions. Consider high temperature, when muscle afferents are presumably not an important determinant of endurance exercise performance, as performance seems limited by the CNS when core temperature rises beyond some threshold (5) and intrinsic skeletal muscle properties are unaffected during repeated contractions performed at 43°C (6). Another example is a recent rigorously controlled study (2) showing that mouth rinsing only (no ingestion) with a carbohydrate solution (but not saccharine) activates selective brain areas, distinctive from areas activated by the sensation of sweetness. Such carbohydrate induced brain activation by mouth rinsing in fasting conditions—probably mediated by specific oral pharyngeal receptors—allowed greater power output compared to controls (7). A recent study showing that mouth rinsing with saccharin and a substantial improvement of an oropharyngeal receptors—allowed greater power output compared to controls (7).

The afferent feedbacks from fatigued locomotor muscles are likely to be different between these activities, if we agree with the model proposed by Amann and Dempsey (1). Similarly, differences exist also for the perception of discomfort and perceived peripheral fatigue between the activities (5) and this is likely to influence the pacing strategy, if we agree with the psychobiological model of Marcora (4). By comparing prolonged intermittent and continuous whole body exercises, other questions arise. For example, the respective influences of the spinal loop modulation (assessed by the H-reflex) and of the supraspinal inhibition of CMO are different in some intermittent sports (3-h tennis game) (3), but not in intermittent running (7), when compared with continuous exercise. To conclude, it seems that each type of endurance-based exercise (e.g., continuous vs. intermittent; pedalling vs. running) requires a specific combination of neuromuscular and psychobiological factors that would limit the performance in a specific way. As any of us practicing multiple sports would know, the causes of exhaustion are multi-factorial.

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CENTRAL FATIGUE IS NOT THE SOURCE BUT CAN EXPLAIN PERFORMANCE DECREMENT DUE TO AFFERENT FEEDBACK

TO THE EDITOR: The downregulation of group III/IV afferents at spinal and supraspinal levels (2) likely explains why maximal voluntary activation is lower after running than after cycling or skiing for similar intensity/distance (6). Central fatigue is not a relevant concept during endurance exercise because voluntary activation is never, in essence, maximal (3), but it can highlight the role of central regulations in performance limitation. Afferent feedback from the fatigued muscles leads to direct and indirect (i.e., disfacilitation due to presynaptic inhibition) downregulation of motoneurons. As a result, a stronger excitatory drive form the motor cortex is requested for a given motor output, inducing a higher subjective effort, which may in turn limit endurance performance (4).

As emphasized by Amann and Secher (1), arguing that muscle afferent feedback plays a significant role does not necessarily means that it is the only determining factor. Using a submaximal test until exhaustion in hypoxia/normoxia while the muscles were maintained in the same complete ischemic conditions, we showed that 1) inhibitory mechanisms from working muscles play a major role in the cessation of the exercise in hypoxia and 2) a minor but significant direct effect of inspired oxygen fraction on central nervous system could potentiate this limiting mechanism and explain why the performance was slightly depreciated in hypoxia (5).

All together, this suggests that, while afferent feedback certainly plays a critical role, the regulation of central motor command is complex and also depends on the environment (altitude, temperature) and the type of endurance exercise.

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THE IMPORTANCE OF PACING

TO THE EDITOR: Imposing different pacing strategies and thereby different levels of fatigue at the end of a race is an interesting model to study the contribution of limiting central and peripheral processes during time trial exercise (5). We have shown that even when power decreased toward the finish line because of premature fatigue, iEMG increased, suggesting that peripheral fatigue rather than a resulting restricting central motor drive limited performance in the end phase of exercise. However, this does not mean that afferent feedback is not important in the process of pacing, as shown in the fentanyl study (1).

When afferent information is blocked, subjects are not capable of distributing their energy equally over the race. Muscle afferents thus seem to be important for successful pacing. On the other hand, as stressed by Marcora et al. (6), it is also clear that other (cognitive/motivational) factors are of importance in pacing and performance, such as momentary power output, task remaining, remaining reserves (3), previous experience/learning (4), and emotions (2). A model combining all information that is relevant for performance in a specific context is necessary to decide which level of effort is chosen to be successful in a time trial or even over a longer period of time, preventing risks for insufficient recovery in periods of continuous stress (physically of mentally).

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WHAT IS THIS THING “FATIGUE” ANYWAY?

TO THE EDITOR: The scientific literature has a fairly complete discussion of why muscles fail to contract when worked to exhaustion (2). However, failure of contraction is not what most people mean when they say “I am fatigued.” Rather, they mean, “I feel tired and don’t want to continue physical/mental work.” What causes this feeling of fatigue? We propose that group III and IV sensory neurons in skeletal muscle, heart, and brain [the same afferents referred to by (1) and (6)] detect increases in specific metabolites, and signal the brain causing the cognitive sensory experience of fatigue.

We characterized this group of muscle afferent neurons in the mouse as well as another afferent group more suited to signal muscle pain (5). We determined likely molecular receptors that mediate their ability to detect muscle contraction produced metabolites.

We do not know the properties of these “fatigue” signaling afferents. Do these afferents contact the sympathetic nervous system to increase blood flow to the working muscles (thus
decreasing the metabolites) as part of the exercise pressor response (3, 4)? What changes their responsiveness (exercise, injury, nutrition, drugs, etc.)? How and what alters their signaling in the spinal cord, brain stem, cerebrum? Can inputs from these receptors alter motivation, mental states? How can these inputs best be used during strength vs. endurance exercise? Moreover, what is the real relationship between the sensation of fatigue and the ultimate failure of motor command signals? Without this knowledge it is too early to determine their real role in endurance exercise.

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AFFERENT FEEDBACK FROM LOCOMOTOR MUSCLES IS NOT RESTRICTED TO GROUP III AND IV AFFERENTS

TO THE EDITOR: A change in performance due to blocking the transmission from muscle baroreceptors and pain receptors (group III and IV afferents) to the central nervous system at the spinal level (1) does not rule out the possible substantial role of the central motor output (CMO) in regulating locomotor muscle performance during endurance exercise (2). In this response we would like to draw attention to other modes of afferent feedback, e.g., those originating from the muscle spindles (length sensitive Ia and II afferents) and the Golgi tendon organs (force sensitive group Ib afferents) that unfortunately escape this debate. Rhythmic activity of muscles during locomotor movement is regulated by a central pattern generator (CPG) that receives tonic drive (the CMO) from supraspinal centers; the intensity of the CMO determines speed and type of locomotion (4). Although the CPG can operate without motion-dependent feedback (3), it is essential for adjusting locomotion to external environmental demands and regulating phase transitions (5). For example, electrical stimulation of ankle extensor group I afferents during the extension phase in a fictive locomotion preparation (3) or in decerebrate or intact walking cats (5) increases the magnitude and duration of the ongoing activity of hindlimb extensors. This demonstrates that muscle output during locomotion depends on afferent feedback.

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TO THE EDITOR: As in most debates there is merit in both arguments, from Amann and Secher (2) and from Marcora (5). Conventional measures of central fatigue (a reduction in voluntary activation during maximal effort) (3) are not easily interpreted during sustained submaximal exercise such as cycling (6). Hence, the key argument surrounds interpretation of altered performance with muscles not being driven fully (over several pedal revolutions). Here, the initially enhanced power output during a time trial with intrathecal fentanyl is consistent with removal of a net “inhibitory” input (1), but later, Amann and Secher concede (in rebuttal) that “psychological factors contribute.” Some additional points need to be mention. During sustained activation, the gain of motoneurons decreases such that a larger descending drive is required to continue exercise. This leads to higher perceived effort for a steady motoneuron pool output. Thus the term “central motor output” can be ambiguous: does it refer to the drive to the motoneuron pool or the output from it? Further Impression derives from the term “afferent feedback.” It is clear that ischemically sensitive muscle afferents can impair descending drive to the motoneuron pool (4). However, the roles of other types of group III and IV muscle afferents (and other afferents) are poorly characterized during sustained dynamic exercise but will be exerted at segmental and cortical levels. Ultimately their influence must be integrated into the task goal. Because performance is at any instant is submaximal, both central and peripheral factors contribute to determine whether there is sufficient reserve for an end spurt.

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“TRADITIONAL” PERSPECTIVES CAN EXPLAIN THE SPRINT FINISH

TO THE EDITOR: Why is it that while the rest of the world calls a finishing sprint a “sprint finish,” a small number of exercise physiologists insist on using the term “end-spurt” (e.g., Ref. 1)? We do not refer to “spurt cyclists” nor “100-m spurers” (thank heavens!). So first a plea: let’s call it a sprint finish. Second, we believe that suggestions that peripheral mechanisms (including afferent feedback) cannot explain the finishing sprint ignore the critical power (CP) concept, which predicts that there is a finite amount of work (or distance, D’), predominantly of nonoxidative origin, that can be performed above the CP (or critical speed, CS). This, in turn, places a metabolic limit on exercise performance (2, 3, 4). Consider, for example, a 5,000-m track race performed by a runner with a CS of 5 m/s and a D’ of 300 m. Assume that the runner completes 4,600 m at 5.2 m/s, exceeding 177 m of D’ in the process. In the final lap, the runner would be able to increase speed to 7.2 m/s (sprint finish = CS/ [1−(remaining D’/400 m)] = 5/[1−(123/400)] = 7.2 m/s). It can be calculated that if the first 4,600 m were run at 5.32 m/s no acceleration would be possible, while running at 5.4 m/s would result in D’ being fully utilized after only 4,050 m (a “suicidal” pacing strategy). The CP concept is therefore integral to understanding pacing strategies. Peripheral mechanisms can and do contribute to limiting physical performance (4).

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AFFERENT MUSCULAR FEEDBACK AS A DETERMINANT OF EXERCISE ENDURANCE

TO THE EDITOR: Authors in this exchange (1, 2) raise excellent points that bear appropriately on the important issue of interest. In the interest of concision, I will limit myself to a core observation. We can profit, I believe, by envisioning effort expended across time holding the external work load (e.g., speed of a flat treadmill) constant. Assuming 1) that effort will correspond to perceptual demand, 2) that perceptual demand will increase as performance resources are depleted, and 3) that resources will be depleted across time, then one can anticipate a steady rise in effort up to one of two points. The first would be that at which performers are no longer willing to endure; the other would be that at which performers no longer can endure. Attainment of either point should yield a precipitous effort decline and performance failure. But the crucial cognitive assessment in each case would be different. In the first case, the assessment would be that endurance costs were not justified by

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endurance benefits. In the second, the assessment would be that no amount of cost would secure a benefit, i.e., that endurance was impossible. It is reasonable to assume that performers include somatic considerations (e.g., respiratory discomfort) in their cost appraisals. Insofar as performers construe afferent muscular feedback as a somatic cost, an implication is that such feedback should affect disengagement up to the point that performers cannot endure. The importance of the feedback ought to depend on the cost weighting assigned and this strikes me as an open question.

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