The following letters are in response to the Point:Counterpoint series “Supraspinal locomotor centers do/do not contribute significantly to the hyperpnea of dynamic exercise in humans” that appeared in the March issue (vol. 100: 1087–1083, 2006; http://jap.physiol.org/content/vol100/issue3/2006).

To the Editor: Ventilation (VE) is controlled by the arterial tension of carbon dioxide (PaCO₂), but during exercise other mechanisms prevail. At low work rates, the moderate increase in V̇E represents “ hypoventilation” as PaCO₂ increases, whereas at 60–70% of work capacity and during intense exercise, the marked increase in V̇E restores to the resting value, or even lowers, PaCO₂ (2). Also, during intense exercise, administration of bicarbonate that eliminates the drop in pH attenuates the hyperpneic response, although PaCO₂ increases (3). However, these observations do not undermine the influence of PaCO₂. Postexercise muscle ischemia reduces V̇E, which increases again on release of the cuff(s) in close parallel with PaCO₂ (2), and a similar response is elicited by reperfusion of internal organs during surgery. However, in some subjects who experience lively pain, postexercise muscle ischemia is associated with a marked increase in V̇E (2). If no pain is experienced, neural influence from the working muscles does not influence V̇E because V̇E is not influenced by epidural anesthesia (4).

Conversely, central command has a marked influence on V̇E and the neurophysiological background is summarized elegantly by Waldrop and Iwamoto (5). In humans, the classical experiment is to perform exercise with partial neuromuscular blockade as first carried out in Germany and in the well-known study of Asmussen et al. (1). Under such circumstances, V̇E is larger than during control exercise, indicating that the will to exercise supports the first step of oxygen delivery to working skeletal muscles.

REFERENCES

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To the Editor: It is a moot point whether supraspinal locomotor centers (SLC) per se contribute significantly to exercise hyperpnea; after all, it is difficult to tell (as Dr. Waldrop concludes) because SLC and feedback drives necessarily mask each other when acting simultaneously, whereas any non-SLC mechanisms—which purportedly track gas exchange rate during exercise—are elusive at this time (as Dr. Haouzi confesses). For one thing, the dogmatic dichotomy between supraspinal feedforward and peripheral feedback “drives” to exercise hyperpnea is inevitably dead-in-the-pot as ignoratio elenchi. Glaringly missing in this futile debate is any consideration of a third, more central and equally (if not more) likely contributor to exercise hyperpnea: the ponto-medullary respiratory controller itself! More than short-term potentiation, the respiratory controller is known to be endowed with a panoply of short-term and long-term neural plasticity and gating mechanisms (1, 4, 5) that afford integral-differential calculus and logic operation capabilities like a computer. Indeed, the suggested “strategy that constrains ventilation to follow information related to gas exchange” could come from a calculated rule on the part of the respiratory controller without the need for any direct (descending or ascending) exercise inputs whatsoever, such that the contingent PaCO₂ homeostasis—far from “a main goal of the ventilatory response to exercise”—may represent a much more malleable and optimal response under varying metabolic and environmental conditions, subject to the instantaneous interaction (mutual “masking”) of the chemical and mechanical feedbacks via corresponding established afferent pathways (2, 3). The brain stem could be much smarter than we think!

REFERENCES

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To the Editor: The issue of whether what is termed “central command” contributes significantly as an essential element of the hyperpnea of dynamic exercise in humans should be considered, we believe [along with Haouzi (4)], in the context of a series of consistently demonstrated features of the normal response—in humans. If central command is assumed to operate as a proportional feedforward control mechanism throughout the exercise (4), it is difficult to see, for example, how it can account for the systematic reduction in the V̇E response amplitude to sinusoidally fluctuating work rate of increasing frequency, but fixed amplitude, actually being abolished at high frequency; i.e., despite central command still driving the workrate sinusoid to the same amplitude (1). Furthermore, reducing the ventilatory requirement for a given
work rate via proportional servocontrolled inspiratory assistance results in a proportional reduction in the intrinsic ventilatory drive necessary to maintain the close regulatory relationship to pulmonary gas exchange; i.e., “accounting for” the proportion of the overall \( V \dot{E} \) response subserved by the assistance (2, 3). This again dissociates intrinsic hyperpneic drive from central command (presumed to be unaffected, as the work rate is not changed). On these and other grounds we therefore find it difficult, along with Haouzi (4), to ascribe a significant obligatory role for central command in the control of the exercise hyperpnea in humans. Not that central command cannot, and possibly even does in some circumstances, contribute to the hyperpnea, but that more-predominant and fundamental aspects of the control are to be sought elsewhere.

REFERENCES


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To the Editor: In the recent Point:Counterpoint discussion of breathing during exercise, both authors support their views with evidence from animal experiments. However, such evidence may not necessarily apply to the control of breathing in exercising humans.

For example, biped humans do not behave as the counterpoint author Haouzi’s (5) treadmill-exercising quadruped sheep. The ventilation of human subjects walking on a treadmill follows the changes in walking pace; changes in ventilation that are absent when the exercise load is similarly varied by altering treadmill incline (3). Other such evidence for the dependence of exercise ventilation on limb movement frequency in humans has been reviewed (4).

The concept of “occlusion” of peripheral afferent information by central command that has been derived from animal experimentation, as presented by the point authors, Waldrop and Iwamoto (5), may also be misleading when applied to humans. In humans, passive leg extension immediately increases ventilation, and when that movement is actively assumed by the subject, ventilation immediately increases again, with the sum of these increases equal to the change in ventilation from rest to active exercise (1).

These human experiments suggest that both central command and peripheral afferent feedback have parts to play in the control of breathing during exercise, and one does not exclude the other. Moreover, both of these may be overruled to some extent by cognitive activity (2).

REFERENCES


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