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Roy J. Shephard

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Comments on Point:Counterpoint: Maximal oxygen uptake is/is not limited by a central nervous system governor

HARD EVIDENCE FOR A CENTRAL GOVERNOR IS STILL LACKING!

TO THE EDITOR: Reviewing evidence for and against a Central Governor that limits an individual’s maximal oxygen intake (2, 4), I am impressed that although this hypothesis was formulated some 11 years ago, it still lacks support outside the proponent’s laboratory. I note also that, perhaps because of an inappropriate test protocol, Noakes has consistently found difficulty in reaching first base in this area of research, the demonstration of an oxygen consumption plateau. In contrast, Ekblo and colleagues (2) have had no problems in this regard.

Noakes (4) currently argues that a Central Governor is essential to prevent the development of a dangerous myocardial ischemia. However, anyone who has exercised older adults will know that a substantial proportion of such individuals manifest myocardial ischemia. Ultraendurance athletes also develop myocardial ischemia (3), and indeed may use this as a stimulus to cardiac hypertrophy. Does this imply that ultraendurance athletes and old people have carelessly broken their Central Governors?

Noakes also cites (4), with apparent approval, the argument of A. V. Hill (3) that the Governor dissuades the heart from making an excessive effort that would reduce the oxygen saturation of arterial blood. Again, the weight of current evidence is that well-motivated athletes do reduce their arterial oxygen saturations (1). Moreover, it would be hard to imagine how the mechanism postulated by Noakes could evolve, since the forces of natural selection have not focused on the ability to perform a maximal oxygen intake test.

REFERENCES

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TO THE EDITOR: The discussion regarding Noakes’ challenge to the concept of V\textsubscript{O}\textsubscript{2max} continues in the excellent Point:Counterpoint by Noakes and Marino (6) and Ekblo (3). It seems, however, that both are so engaged in an either/or argument that they have missed the point that perhaps Hill (5) was right, although forgivably too simplistic, over 80 years ago. Recent studies, including from our laboratory (4) have confirmed that V\textsubscript{O\textsubscript{2}} reaches values during incremental exercise, which are rarely exceeded even when higher intensity exercise is performed. If V\textsubscript{O}\textsubscript{2max} is limited centrally, limitations of cardiac output can as reasonably be attributed to limitations of venous return, diastolic filling time, and ventricular compliance as to losses of contractility secondary to the potential myocardial ischemia that Noakes correctly suggests must be avoided. It is also documented that failures to maintain saturation of hemoglobin during heavy exercise are common. Thus the concept of decreases in “offered \textit{O}_2” limiting V\textsubscript{O\textsubscript{2max}} is supportable (2) and may be associated with an array of outcomes including changes in the iEMG-power output ratio and the stimulated muscle performance (1). Finally, as anyone who has performed an incremental exercise test knows, this leads to the compelling “I don’t want to continue” sensation. So, yes, there must be a command coming from the central nervous system that tells the exerciser that homeostasis is becoming disturbed and that it would be advisable to stop. But, many if not most, of these “stop” signals are reasonably attributable to limitations in central O\textsubscript{2} transport and aerobic ATP generation.

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NO SUPPORT FOR CENTRAL GOVERNOR

TO THE EDITOR: Noakes and Marino (4) have not designed experiments that provide support for the central governor model (CGM). Moreover, they have not identified the specific components (e.g., stimuli, receptors, and afferent nerve fibers) involved in the negative feedback loop that supposedly protects the heart from overexertion. Ekblo’s (1) findings contradict the CGM of Noakes and Marino (4). Ekblo elegantly showed that when the body is subjected to supramaximal work rates, V\textsubscript{O\textsubscript{2}} and Q level off, but EMG activity and the work of the heart continue to increase. Noakes and Marino (4) respond that since EMG activity during combined A+L exercise at V\textsubscript{O\textsubscript{2max}} is less than during a maximum voluntary contraction for the respective muscle groups, a central governor in the brain must be limiting skeletal
muscle recruitment. Although it is true that skeletal muscle recruitment during a 1- to 2-s anaerobic “burst” is greater than during strenuous, dynamic exercise at 100% VO_{2max}, this does not show that receptors in the myocardium are sending action potentials back to a central governor in the brain, resulting in inhibition of motor units.

Ekblom (1) cites strong evidence that Q_{max} and not a central governor, determines VO_{2max}. When the dog pericardium is cut, Q_{max} increases and so does VO_{2max} (5). In addition, other researchers have found that whenever Q_{max} is diminished (through cardioselective β-blockade, atrial fibrillation, or mitral stenosis), there is a decrease in VO_{2max} (2–3, 6). Thus O_{2} delivery and the pumping capacity of the heart are key determinants of VO_{2max}.

REFERENCES


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VO_{2max} AND EXERCISE PERFORMANCE

TO THE EDITOR: To resolve this debate, it is necessary to clearly distinguish between what limits performance and what limits VO_{2max} during dynamic whole body exercise. When a plateau is achieved during an incremental test, or when “supramaximal” exercise is sustained long enough (1), it is clear that VO_{2max} is limited by the factors underlying the Fick equation, particularly stroke volume. However, it often happens that a subject decides to stop exercise before the physiological limits of the Fick equation are reached (e.g., in hypoxia). In these conditions, the maximal VO_{2} measured is proportional to exercise performance, which, in turn, is limited by the brain. So both parties of this debate are right. However, we disagree with Noakes and Marino that exercise performance is subconsciously regulated by a central governor on the basis of afferent feedback from the heart and other organs. In fact, we proposed that exercise performance is regulated by the conscious brain on the basis of potential motivation (defined as the maximum effort a person is willing to exert to satisfy a motive) and perceived exertion (5) without the need for an extra central governor (2–3). There is also strong experimental evidence that perception of effort is generated by corollary discharges of central commands to the locomotor and respiratory muscles rather than afferent sensory feedback (4). The facts that many cardiac patients can exercise past the point of myocardial ischemia (as indicated by ST segment depression) and stop because of chest pain (6) also argue against the model proposed by Noakes and Marino.

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V̇O₂max (2). From a philosophical standpoint, the CGM suggests peripheral factors in the muscles, is the factor limiting a central nervous system governor, challenges the Traditional more prevalent as a limiting V̇O₂max factor, since TM would be neuromuscular activation, independent of local muscle temper-
instance, a centrally originating progressive impairment in behavioral responses were inadequate or could not be used due to conflicting behavioral demands (5). We have shown, for peripheral thermosensors being deployed to prevent anticipated thermal insults (3), while autonomic and endocrine responses, based more on core temperature signals from central thermosensors, employed when body heat content changes because behavioral responses were inadequate or could not be used due to conflicting behavioral demands (5). We have shown, for instance, a centrally originating progressive impairment in neuromuscular activation, independent of local muscle temperature, occurring prior to temperature-induced system catastrophe (6). If our hypothesis is true, however, the CGM would be more prevalent as a limiting V̇O₂max factor, since TM would be employed only prior to system collapse. This would explain the increased support that the CGM has received (4).

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EXPERIMENTAL EVIDENCE MAY INFORM THE DEBATE
TO THE EDITOR: The discussion on whether a central nervous system governor to maximal oxygen uptake (V̇O₂max) exists (1, 5) may be informed by a series of experimental animal studies that we have performed, although admittedly, they were not designed to resolve the current debate. Nonetheless, they serve a purpose here.

We repeatedly measure V̇O₂max during graded treadmill running at increasing work rates. These records usually show evidence of a plateau of V̇O₂ despite increasing running speeds (4). Thus the plateau phenomenon is observed across species. More interestingly is perhaps that when we anesthetize and induce left ventricular myocardial infarctions in rats, we see ~40% reduced V̇O₂max and running capacity (3, 4, 6). These animals have never been exercised before, and the procedure does not involve or affect either the skeletal muscle or the central nervous system. Instead, these studies indicate a cardiac pump dysfunction as the cause of reduced V̇O₂max, since the rat, after all, is able to perform incremental exercise to high intensities until V̇O₂ levels off, despite the heart already being ischemic. It is therefore difficult to see how a central nervous system command would dictate the change in V̇O₂max by limiting skeletal muscle work in anticipation of myocardial ischemia under these conditions. Has the heart-skeletal muscle information line disentangled itself, or was it never there? Although this does not prove the non-existence of a central nervous system governor, it does put significant limitations to a model that does not include a major role of the central circulatory system.

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TO THE EDITOR: This is a rather unbalanced debate because it has been demonstrated with several experimental approaches that in health $\text{VO}_{2\text{max}}$ is limited by oxygen delivery (5). In contrast, the central governor model (CGM) is mostly supported by sophisticated reasoning, used to re-elaborate some obviations. For example, if the central nervous system does not receive sufficient oxygen then a number of physiological functions will be affected (1). Maximal exercise capacity may be limited by insufficient brain oxygenation during exercise in extreme hypoxia (2). However, even in the latter situation we do not have irrefutable evidence for central mechanisms of fatigue. It is not true that the CGM impedes “to reach a truly maximal cardiac output to prevent myocardial ischemia by limiting peripheral blood flow” (6), because maximal vasodilation at peak exercise, increases cardiac output without changing peak leg blood flow or $\text{VO}_{2\text{max}}$ in humans (4). Actually, experimental evidence indicates that the work of the heart, i.e., the variable determining myocardial oxygen demand, increases continuously during a gradual exercise to exhaustion without signs of reaching a plateau (3). Moreover, despite the ongoing discussions regarding the need of the central governor to protect the heart by blunting central command, it has been shown that the heart can protect itself very well (without need of a CGM). If the CGM was true, and is acting to prevent myocardial ischemia, then subjects with coronary syndrome would never have angina during exercise, since the CGM would cause “protective fatigue” reducing exercise intensity and ultimately preventing an infarct.

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