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, Ekbloom 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 VO2max continues in the excellent Point:Counterpoint by Noakes and Marino (6) and Ekbloom (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 VO2 reaches values during incremental exercise, which are rarely exceeded even when higher intensity exercise is performed. If VO2max 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 O2,” limiting VO2max, is supportable (2) and may be associated with an array of outcomes including changes in the iEMG-power output ratio and 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 O2 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.

Ekbloom’s (1) findings contradict the CGM of Noakes and Marino (4). Ekbloom elegantly showed that when the body is subjected to supramaximal work rates, VO2 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 VO2max is less than during a maximum voluntary contraction for the respective muscle groups, a central governor in the brain must be limiting skeletal
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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% VO2max, 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 Qmax, and not a central governor, determines VO2max. When the dog pericardium is cut, Qmax increases and so does VO2max (5). In addition, other researchers have found that whenever Qmax is diminished (through cardioselective β-blockade, atrial fibrillation, or mitral stenosis), there is a decrease in VO2max (2–3, 6). Thus O2 delivery and the pumping capacity of the heart are key determinants of VO2max.

REFERENCES


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VO2max AND EXERCISE PERFORMANCE

TO THE EDITOR: To resolve this debate, it is necessary to clearly distinguish between what limits performance and what limits VO2max 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 VO2max 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 VO2 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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TO THE EDITOR: The lack of experimental evidence ascertaining the contribution of the central nervous system to VO2max or the exact amount of muscle mass active during exercise, makes it more fruitful to focus this passionate debate (1, 4) on testable aspects of Drs. Noakes’ central governor model (CGM), which proposes “the brain regulates the extent of skeletal muscle recruitment to ensure that a truly maximal cardiac output is never achieved”(4). Drs. Noakes and Marino concede that, “a finding that cardiac function and skeletal muscle recruitment are both absolutely maximal and that homeostasis is lost at VO2max would disprove the CGM”(4). While we agree that skeletal muscle recruitment might not be absolutely maximal during exercise eliciting VO2max, compelling evidence in trained humans indicates that a plateau or drop in cardiac output (due to a fall in stroke volume) and blood flow to active muscles and brain precede the attainment of VO2max and exhaustion during incremental and constant maximal cycling (2, 3, 5,6). Importantly, cardiac output and active muscle blood flow plateau early in constant supramaximal cycling at values similar to maximal cycling, accompanying a decline in stroke volume (6). The close coupling between muscle perfusion and metabolic demand is therefore disturbed even prior to VO2max, possibly because vasoconstriction limits blood flow to muscles as cardiac function reaches its regulatory capacity (6). Thus our findings argue strongly against the CGM by supporting that the regulatory capacity of the human circulation is reached and homeostasis is lost prior to achieving VO2max.

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MAXIMAL OXYGEN UPTAKE REGULATION AS A BEHAVIORAL MECHANISM

TO THE EDITOR: The Central Governor Model [CGM (4)], suggesting that maximal oxygen uptake (V02max) is limited by a central nervous system governor, challenges the Traditional Model (TM) according to which the heart’s pumping capacity, not peripheral factors in the muscles, is the factor limiting V02max (2). From a philosophical standpoint, the CGM sug-

ments peripheral factors in the muscles, is the factor limiting a central nervous system governor, challenges the Traditional

more prevalent as a limiting V02max factor, since TM would be suggested that maximal oxygen uptake (V02max) is limited by a central nervous system governor to maximal oxygen uptake (V02max) 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 V02max during graded treadmill running at increasing work rates. These records usually show evidence of a plateau of VO2 despite increased 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 V02max 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 V02max, since the rat, after all, is able to perform incremental exercise to high intensities until VO2 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 V02max 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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EXPERIMENTAL EVIDENCE MAY INFORM THE DEBATE

TO THE EDITOR: The discussion on whether a central nervous system governor to maximal oxygen uptake (V02max) 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 V02max during graded treadmill running at increasing work rates. These records usually show evidence of a plateau of VO2 despite increased 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 V02max 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 V02max, since the rat, after all, is able to perform incremental exercise to high intensities until VO2 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 V02max 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 $VO_{2\text{max}}$ 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 $VO_{2\text{max}}$ 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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