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

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A classic teaching predicts that maximal exercise is limited by the capacity of the heart to pump blood to the systemic circulation since the fully dilated skeletal muscle arterial bed can alone accommodate more than the maximal cardiac output (Q) (1, 25). In contrast the Central Governor Model (CGM) proposes that the brain regulates the extent of skeletal muscle recruitment to ensure that in health, a truly maximal cardiac output is never reached (30) because this must cause myocardial ischemia (22).

The CGM has been described in detail (13, 20, 22–24, 26) but opponents (4, 5) have used only articles published between 1996 and 2001 (17–19, 21) for their disproof; more recent articles have been ignored (13, 15, 22–24, 26). In this interchange we show that recently published studies (4–6, 10) support the CGM. We will also correct misrepresentations and factual errors.

The studies of Brink-Elfegoun et al. (4, 5) and others (6, 10) conclude that a “governor” in the central nervous system (CNS) does not regulate the maximal oxygen consumption. The existence of such a governor was first proposed by Hill et al. (11) in 1924. They wished to explain why repeated bouts of maximum exercise do not produce progressive myocardial damage according to their understanding that myocardial ischemia “limits” the maximal Q and hence maximum exercise performance. To protect the ischemic heart from damage during maximal exercise, they proposed the existence of a governor: “From the point of view of a well-coordinated mechanism, some such arrangement is eminently desirable; it would clearly be useless for the heart to make an excessive effort if by so doing it merely produced a far lower degree of saturation of the arterial blood; and we suggest that, in the body (either in the heart muscle itself or in the nervous system), there is some mechanism which causes a slowing of the circulation as soon as a serious degree of unsaturation occurs, and vice versa. This mechanism would tend, to some degree, to act as a ‘governor’, maintaining a reasonably high degree of saturation of the blood” (Ref. 11, pp. 161–163). And: “It is suggested that a ‘governor’ mechanism exists, either in the heart muscle itself, or elsewhere, which tends to co-ordinate the output of the heart with the degree of saturation of the blood leaving it” (Ref. 11, p. 166). Defendants of the traditional model (4, 5, 10) should acknowledge that Hill and his colleagues proposed the original governor concept and why the traditional model (4, 5, 10) should acknowledge that Hill and his colleagues proposed the original governor concept and why the presence of a governor is essential to prevent heart damage in their model (and in all similar models).

But how does a governor function before rather than after ischemia has already developed as Hill et al. (11) incorrectly believed. For maximum exercise in health terminates before the onset of myocardial ischemia (22) and according to a majority of [but not all (8,16)] studies before the Q “plateaus” (7, 12, 30). The most effective method to prevent myocardial ischemia is to “limit” peripheral blood flow (9) (chapter 84, Figs. 84–9 and chapter 21). The CGM achieves this by regulating the amount of work that the exercising muscles may perform. This “limits” peripheral blood flow, venous return, and, as a result, the cardiac output.

Some (5, 10) have misrepresented this postulate of the CGM that: “the ultimate control of exercise performance resides in the brain’s ability to vary the work rate and metabolic demand by altering the number of skeletal muscle motor units recruited during exercise” (Ref. 23, p. 513). It is misleading to argue that the CGM claims that the “V˙O_{max} is only a consequence of the amount of work that the heart is allowed to perform” (Ref. 5, p. 604). Correctly stated, the CGM predicts that the V˙O_{max} is regulated (not limited) by the (submaximal) number of motor units recruited in the exercising limbs, a quite different meaning.

In their study Brink-Elfegoun et al. (5) found that Q, heart rate, and stroke volume were all the same during exercise at 100 and 120% V˙O_{max} whereas maximum blood pressure (BP) was significantly lower (160 ± 23 vs. 179 ± 32 mmHg) at 100% V˙O_{max}. The higher BP at the same Q indicates that total peripheral vascular resistance was increased at 120% V˙O_{max}. This can be explained by a centrally regulated BP-raising mechanism induced by exercise at 120% V˙O_{max}. The greater afterload at 120% V˙O_{max} indicates that the heart worked harder at 120% than at 100% V˙O_{max}; that is that the heart worked submaximally at 100% V˙O_{max}.

This finding is predicted by the CGM but disproves the traditional model. Furthermore, the finding that Q was not higher at 120% than at 100% V˙O_{max} although a higher mean work rate was achieved (431 ± 73 vs. 387 ± 60 W) disproves the traditional Hill model, which holds that the peak work rate is “limited” by the Q, which determines the adequacy of tissue oxygen delivery (2–6, 8, 10, 16). Instead, the CGM predicts that the higher work rate at 120% than at 100% V˙O_{max} as also reported by others (6, 10) results from the recruitment of a greater number of skeletal muscle motor units (necessary to produce a higher work rate). We know of no other explanation. Since subjects reached a higher work rate at 120% than at 100% V˙O_{max} so exercise at 100% V˙O_{max} occurred at a submaximal level of skeletal muscle recruitment as predicted by the CGM.

Furthermore, according to the calculations provided by Brink-Elfegoun et al. (5) in their Fig. 1, maximal work rate of 387 W at 100% V˙O_{max} equates to a total work output of 112,230 Nm, whereas at 120% V˙O_{max} the work rate of 431 W equates to a total work output of 94,820 Nm. Thus less total work (~17 410 Nm) was completed during exercise at 120% V˙O_{max}, although the heart worked harder. This again dissociates the amounts of work performed by the heart and the skeletal muscles. Instead the CGM predicts that the amount of work performed by the skeletal muscles (and as a consequence by the heart) is a function of the number of motor units recruited in the active muscles. Thus the extent of skeletal muscle recruitment determines the cardiac output and not the converse (20).

These findings (4, 5) invite the question: How might the CGM explain similar V˙O_{max} values during exercise at 100 and 120% V˙O_{max}? The CGM predicts that a wide range of bio-
logical signals are monitored to ensure that exercise always terminates before the loss of homeostasis in any bodily system as was the case in all these new studies (4–6, 10). This complex model (24, 26) contrasts to the reductionist model of Hill et al. (11), which holds that a single variable, the development of skeletal muscle anaerobiosis and “lactic acidosis,” alone causes the termination of maximal exercise.

A finding that cardiac function and skeletal muscle recruitment are both absolutely maximal and that homeostasis is lost at VO$_{2\text{max}}$ would disprove the CGM. These data have yet to be provided. In contrast, proponents of the Hill model need to explain how the maximal Q can occur when the heart is contracting submaximally as the data of Brink-Elfegoun et al. (5) now confirm.

But more salient is the evidence showing that exercise is regulated “in anticipation” by an intelligent, complex system (14, 15, 20, 27–29) and not by a single component such as the cardiovascular system.

REFERENCES


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COUNTERPOINT: MAXIMAL OXYGEN UPTAKE IS NOT LIMITED BY A CENTRAL NERVOUS SYSTEM GOVERNOR

Every new hypothesis can be built on theoretical calculations and ideas. This was the case when Timothy Noakes (TN) presented the Central Governor Hypothesis (CGH) in 1997. He suggested that maximal oxygen uptake (VO$_{2\text{max}}$) is limited by a central nervous command (6). However, it is the suggestion’s absolute duty to go on and prove the theory in practical experiments in one way or another. But except for re-evaluations of earlier studies, TN has not fulfilled this obligation so far. I agree that a Central Governor (CG) can terminate exercise time during maximal exercise but not limit VO$_2$ with increasing rate of work, if uphill running (R) or during combined arm+leg (CA+L) exercise are used.