

The Improbable Central Governor of Maximal Endurance Performance

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A governor in the brain that limits drive to muscles would explain why we can't normally exercise to the point of collapse, but better explanations include self-limiting mechanisms to protect the periphery and intolerable sensations of heat and breathlessness to protect the brain. Moreover, it is reasonably clear that cardiac output and oxygen uptake are driven to maximum in intense endurance exercise, an outcome that one proposed drive-limiting governor is supposed to prevent. Until there is more evidence for such a governor or more convincing alternative explanations for the existing evidence, researchers should assume that intense exercise performance is limited by a maximum in the ability of muscles to output power. KEYWORDS: brain, control, central nervous system, effort, maximal oxygen uptake, perception, VO₂max.

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When you're trying to outrun a predator or cross the line first in an Olympic final, is your physiology truly pushed to the limit? Most exercise scientists assume it is, but for the last 10 years or so one of our number has been promoting a different view. Tim Noakes believes that our bodies have the capacity to perform exercise intense enough to cause catastrophic failure of physiology somewhere in the body. He argues that the demand for oxygen or the release of metabolites or heat by exercising muscles could result directly or indirectly in development of acute malfunction in the muscles, heart, lungs or brain. Noakes believes that our brains therefore have a "central governor" that caps the drive from the brain to skeletal muscles before a catastrophe occurs.

The concept of the governor is not new—Noakes and Frank Marino (2009a) acknowledge mention of it in 1924 by one of the great physiologists, [AV Hill](#)—but it is only Noakes and his colleagues who write articles devoted to it. Debates about the governor and related issues have featured at several conferences, including one I reported on in a [1999 issue](#) of *Sportscience*. Most recently, the role of the governor in limiting maximal oxygen uptake was the subject of a debate between Noakes and Marino (2009a) and Björn Ekblom (2009a) in the pages of the *Journal of Applied Physiology*. The debate, the commentaries that followed it

(Shephard et al., 2009) and the "last words" (Ekblom, 2009b; Noakes and Marino, 2009b) rekindled my interest in the governor model but left me feeling confused about its role. This article represents the way I came to terms with the evidence.

In an early draft of this article I argued that a governor makes little sense from an evolutionary perspective. Only an unintelligent designer would endow animals with capacities they cannot use. Furthermore, if you are being chased by a predator, it is better to push to your physiological maximum and run the risk of catastrophic collapse than to be held back by a governor and face increased risk of death. But a colleague was quick to point out that a governor could benefit the predator, if it is better to be held back and hunt again in an undamaged state than to go all out, collapse, and be in a poor condition for the next chase. Another colleague suggested that, as a predator, an animal could get the benefit of a governor, but as prey it could avoid the disadvantage by overriding the governor with fear signals from the limbic system. These arguments are interesting but by themselves unconvincing. I also found nothing useful in a search for *Noakes, governor* and *evolution* in journals, although in reviewing this article, Frank Marino drew my attention to pertinent discussions in several texts (Marino, 2008; Mosso, 1915). It's safe to say that appeal

to evolutionary principles will not provide good evidence for or against a governor, unless the development or elimination of a governor is a reproducible outcome in simulations of natural selection covering a wide range of environmental scenarios. Meantime we need other tangible evidence, and there is now enough of that to reach a reasonably confident conclusion.

The governor model is based originally on the observation that catastrophic failures in intense exercise are rare. Exercise increases the risk of sudden collapse or death with some diseases, but healthy individuals just don't seem to be able to exercise to the point of sudden collapse from muscle rigor, cardiac arrhythmia or arrest, pulmonary edema, or neuronal dysfunction resulting in disorientation or fainting. Are there better explanations than the governor for the rarity of such exercise catastrophes? I think so.

Fatigue in skeletal muscle automatically places a cap on metabolic demand: any marked fall in availability of energy (ATP) is self-limiting, so there cannot be a sufficient shortage of ATP to drive the muscle into a state like rigor mortis. A similar self-limiting process could apply to cardiac muscle: if the heart fatigues, it pumps less blood, so skeletal muscles can't maintain their power output, so the demand for blood falls, so the load on the heart falls, so the demand of heart muscle for ATP falls, so there is no development of the kind of shortage of energy that compromises the electrical activity or contractility of the heart during exercise with ischemic heart disease. Edema in the lungs is also self-limiting: as the alveolar membrane thickens with fluid exuding from the pulmonary capillaries, oxygen transfer to the blood falls, so arterial hypoxemia begins to develop, so the muscles can't maintain their power output, so their demand for blood falls, so cardiac output and pulmonary blood pressure fall, so the edema plateaus at nothing like a catastrophic level.

Neuronal dysfunction could arise from intense exercise in various ways: hypoxemia, hypocapnia, hypotension, and especially hyperthermia. The only self-limiting process that can offset any of these changes is fainting, which does not normally occur and which in any case is a catastrophic outcome in a life-or-death situation. If the governor is not involved, the animal can protect its brain and stay conscious

only by responding to the sensory stimuli that, at least in humans, evoke unpleasant perceptions. Intense exercise is reasonably unpleasant well before there is any risk of neuronal dysfunction: it's your brain's way of telling you to avoid exercise that damages muscle fibers, that uses up energy stores, and that increases the risk of injury if you miss your footing. Unpleasant perceptions of effort during exercise can therefore arise from various chemoreceptors, ergoreceptors and thermoreceptors in the body, but the perceptions most relevant to catastrophic neuronal dysfunction are probably oppressive feelings of heat and breathlessness, which build until they become imperatives to reduce exercise intensity. This protective mechanism is a governor of sorts, but importantly, it allows for the possibility of the animal pushing its physiology to maximum and holding it there until the feelings are intolerable.

So much for the better explanations, but what about specific evidence against the governor? The governor model predicts that catastrophic failures would be frequent at intensities above maximal effort. This prediction cannot be investigated directly—you can't make more than a maximal effort—but it is possible to stimulate muscles electrically to a greater extent than you can activate them consciously. Such stimulation results in fatigue, not rigor or supercontracture (Spriet et al., 1987), so the prediction of a catastrophe in skeletal muscle thus far has failed. As far as I know, excessive stimulation of heart muscle also fails to produce sustained contracture, rigor, fibrillation, or any dysfunction other than simple fatigue. Evidently muscles do have self-limiting or other built-in mechanisms to protect against catastrophic failure.

The prediction of the governor model that gets most attention from researchers is that performance is limited by drive to the muscles. It follows that the governor model is disproved if there is one common example of maxing out of a physiological system required for intense exercise. For endurance exercise of sufficient intensity, the evidence presented by Ekblom (2009a) is clearly in favor of oxygen consumption reaching a plateau consistent with a true maximum in 10 out of 10 well-trained subjects. Ekblom's data also show that the maximum in oxygen consumption appears to be due to a maximum in cardiac output, and the data of Russ Richardson (e.g., Richardson et al., 1999)

and others are consistent with fatigue developing in muscle fibers in response to a shortage of oxygen. Thus, performance appears to be limited by delivery of oxygen to muscles, and therefore not by delivery of action potentials to muscles.

Noakes is usually emphatic in his denial of the existence of a plateau in maximum oxygen uptake. His failure to mention it in the point-counterpoint debate may represent long overdue acceptance, but in rebutting Ekblom's counterpoint he asserts that "the magnitude of the VO_2max ... is determined by the mass of muscle that the brain is prepared to recruit". If I understand this assertion and the rest of the article correctly, the governor allows activation of only a certain amount of muscle mass, thereby setting the maximum of oxygen consumption. But we obviously can and do recruit more muscle in a shorter bout of more intense exercise, so VO_2max should be higher if Noakes is right, yet Ekblom found the same VO_2max in shorter and other bouts involving more muscle mass. Noakes would have to argue that the governor cuts down recruitment as the bout proceeds, but in that case, how can power output be maintained during the bout? Yes, some regulation of muscle recruitment does occur, but it is there apparently to achieve a level and pattern of activation that minimizes fatigue in a muscle that is maxed out (Enoka and Stuart, 1992). Furthermore, the governor would have to use signals from the periphery to decide on how much muscle mass to activate—otherwise it would not know that it could activate more muscle to achieve the higher VO_2max seen with blood transfusions or hyperoxia—but it is too much of a coincidence that control based on these signals results in a VO_2max that plateaus to the same value for different intensities between bouts and at different times within a bout. (In their last words, Noakes and Marino claimed that increasing oxygen delivery to muscle does not improve maximal exercise performance, but the claim was based on exercise in unusual circumstances.) The simple explanation for the physiology of intense exercise in Ekblom's studies is that oxygen consumption and power output are maxed out by a limitation in the periphery: delivery of oxygen. The implausible explanation is that oxygen consumption and power output are limited by muscle recruitment via a

governor.

In conclusion, intense exercise damages muscle, wastes energy stores, increases the risk of acute traumatic injury, and if continued long enough in the heat, increases the risk of damaging the brain. It is therefore hardly surprising that we perceive such exercise as unpleasant and that we will sometimes choose not to reach a physiologically limited maximum. But on the basis of available evidence, some healthy humans can and do reach a physiological maximum without immediate catastrophic failure of any organ, at least in intense endurance exercise. Physical performance enhancement in such exercise is evidently all about increasing the ability of muscles to output power, not about lifting the limit set by a governor.

Reviewers' Comments

Björn Ekblom found the article "in line with my views in the debate with Noakes and Marino, who refuse to accept my points about VO_2max . It seems to me they always add odd ideas without any hard evidence." He also thought it was worth calling attention to the point he made in his final word: if there were a need for a governor to protect the heart, it would surely limit the work of the heart directly via cardiovascular control rather than indirectly via control of skeletal muscle. I agree. In Ekblom's experiments the governor clearly did not limit the work of the heart, as shown by higher blood pressure at higher intensities of exercise at maximum cardiac output.

Frank Marino made several useful suggestions for improvement, which I included. He conceded that "the evidence for the governor is scarce in comparison to that supporting the classic view" but added that "evidence for the governor will come in due course". He questioned whether any studies showed evidence for a cardiac output plateau when oxygen uptake plateaued, then stated that the VO_2 plateau is possibly an experimental artifact. Finally, in his view "researchers who do not support the governor understand it only as a mechanism to explain maximal exercise, whereas the governor attempts to explain the limitations of exercise in a number of conditions and situations". I find this last point particularly hard to accept. A central governor has been put forward as a specific mechanism limiting maximal endurance performance. The executive and regulatory functions of the brain in other forms of

exercise represent a real governor that we have known about for generations. In my view, conflating these functions with the improbable central governor of maximal endurance performance leads to confusion and misattribution.

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Update 15 May 09: minor improvement to wording of Abstract.