CURRENT OPINION

Sports Med 2009; 39 (9): 709-721
0112-1642/09/0009-0709/$49.95/0

Is it Time to Retire the 'Central Governor'?

Roy J. Shephard

Faculty of Physical Education and Health, University of Toronto, Toronto, Ontario, Canada

Abstract

Over the past 13 years, Noakes and his colleagues have argued repeatedly for the existence of a 'Central Governor', a specific brain centre that provides a feed-forward regulation of the intensity of vigorous effort in order to conserve homeostasis, protecting vital organs such as the brain, heart and skeletal muscle against damage from hyperthermia, ischaemia and other manifestations of catastrophic failure. This brief article reviews evidence concerning important corollaries of the hypothesis, examining the extent of evolutionary pressures for the development of such a mechanism, the effectiveness of protection against hyperthermia and ischaemia during exhausting exercise, the absence of peripheral factors limiting peak performance (particularly a plateauing of cardiac output and oxygen consumption) and proof that electromyographic activity is limiting exhausting effort. As yet, there is a lack of convincing experimental evidence to support these corollaries of the hypothesis; furthermore, some findings, such as the rather consistent demonstration of an oxygen consumption plateau in young adults, argue strongly against the limiting role of a 'Central Governor'.

1. Concept of a 'Central Governor'

An early graduate textbook[1] made a systematic review of possible factors limiting physical performance. Depending on duration of the bout of effort, critical physiological factors were, in turn, maximal anaerobic power, maximal anaerobic capacity, maximal aerobic power, maximal aerobic capacity, and reserves of food (glycogen and fat), water and minerals. In some situations, environmental constraints such as difficulty in eliminating metabolic heat could also intervene, and with any period of exercise, psychological factors (motivation, arousal and release of cerebral inhibition) could sustain or enhance an individual's performance. However, this comprehensive evaluation found no need to postulate that a specific subconscious feed-forward control mechanism was setting an upper limit to the intensity of exercise.

A more recent systematic review of the causes of fatigue during prolonged endurance cycling[2] made brief mention of the idea of such a control system, tracing its origin to Ulmer.[3] Ulmer had envisaged a feedback of information on force generation, displacement and metabolism to a central regulator, where these data were matched against motor learning and desired objectives to ensure a teleo-anticipatory optimization of the individual's metabolic rate. In developing the concept of a 'Central Governor', Noakes[4] reached back to the classic studies of Hill et al.,[5] who had postulated some mechanism (either peripherally, in cardiac muscle or in the CNS) that restricted cardiac output as the arterial oxygen saturation began to fall. Noakes envisaged the central control mechanism as limiting motoneuron output in order to conserve homeostasis and prevent tissue damage from such threats as ischaemia or hyperthermia.[4]

A long list of papers promoting the concept of a 'Central Governor' has been written by Noakes, his immediate colleagues and collaborators,[4,6-24] with a varying degree of support from some other laboratories.[3,25-40] However, most sports scientists have yet to accept the hypothesis.[41-46] In a recent debate conducted by the *Journal of Applied Physiology*,[22] seven commentators offered arguments against the hypothesis, against one brief and tepid endorsement from Carl Foster:[47]

> "… 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 CNS 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_2 transport and aerobic ATP generation."

Under the title 'The implausible governor,' a recent editorial[48] in the journal *Sportscience* (p. 9) summarized the issue thus:

> "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 are capable of 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 catastrophic malfunction in the muscles, heart, lungs or brain. Noakes believes that our brains therefore have a 'governor' that caps the drive from the brain to skeletal muscles before these catastrophes occur."

Reasoned discussion of the hypothesis has been hampered by the absence of a systematic and clearly enunciated listing of its inherent correlates. Sometimes, enthusiasm has also led to an unfortunate misrepresentation of opposing viewpoints, and/or too quick a dismissal of critical questions as 'irrelevant'.

2. Key Correlates of the 'Central Governor Hypothesis'

Review of papers written to promote the idea of a 'Central Governor' suggests several key correlates of the concept:

1. The governor has evolved in response to evolutionary pressures. Although Noakes currently argues that the hypothesis applies to all forms of exercise with the specific exception of running on a treadmill where speed is controlled by the investigator, most discussions of possible evolution have focused on species that engage in prolonged endurance activity when hunting their prey.[14,17,49]
2. The controller limits the drive to the working muscles in order to maintain homeostasis throughout the body. Thus, the brain, heart, skeletal muscle and other body organs are protected against ischaemic and thermal injury, and the lungs against pulmonary oedema.[6,7,9,49]
3. Cardiac output does not reach a ceiling value that limits the maximal intensity of exercise when measuring maximal oxygen intake or engaging in other forms of intense endurance activity.[22]
4. Oxygen consumption does not reach a plateau value that limits maximal exercise intensity. If there is neither a ceiling of cardiac output nor a plateauing of oxygen consumption, then some alternative mechanism must be limiting maximal performance, and this could be the postulated 'Central Governor'.[50,51]
5. Feed-forward regulation limits central drive to the muscles, so that electromyographic (EMG) activity is submaximal when an individual is making a peak voluntary effort.[10,52]

These several issues are examined critically below.

2.1 Potential for the Evolution of a 'Central Governor'

How strong are evolutionary pressures to develop a mechanism such as the postulated Central Governor? Many years ago, Mosso[53] noted that 3 years of learning was needed to perfect migration patterns in quails; he argued that the birds were then able to regulate their energy

expenditures over prolonged flights in such a manner as to conserve a final store of metabolites against easy capture. Marino[49] saw this observation as evidence for the evolution of a feed-forward regulator that enabled a bird to calculate its physiological requirements ahead of time, and to regulate potentially fatiguing exercise patterns accordingly. Marino[54] argued that in similar fashion, evolutionary pressures had led to the development of central regulatory mechanisms in both hunting dogs and humans, allowing prey to be run into the ground without endangering the homeostasis of the hunter. However, Hopkins[48] has recently pointed out that this scenario could be argued in several different ways – perhaps with predators risking a loss of homeostasis in order to avoid death from starvation, or the prey risking loss of homeostasis in order to avoid capture.

Animals plainly have adapted to hostile environments, given sufficient time and selective pressures. The potential for human genetic adaptation to challenging habitats was thus a major concern of the International Biological Programme (IBP).[55] Particular attention was focused on the ability of various isolated populations to undertake fatiguing work of various durations. However, the IBP found surprisingly little evidence that humans had developed unusual physiological characteristics in response to prolonged residence in extreme environments. The available findings of human anthropology, the extent of selective pressures and molecular genetics all argued against significant modification of human exercise tolerance in this way, even in small and circumscribed populations that had been exposed to rigorous conditions for many generations.

Anthropological evidence emphasized that most human groups lived at the junction of several habitats, which were exploited at different seasons. Thus, an adaptation favouring survival in one habitat would often prove a disadvantage in a second habitat exploited at other times during the year. Experimental data showed not diversity, but rather remarkably similar physiological working capacity in populations that occupied strikingly different environments.[55]

A partial explanation may lie in differences in hunting techniques between animals and humans. Although some animals capture their prey by 'running them into the ground', field studies of traditional neolithic populations suggest that their success in hunting trips, and thus any selective pressures, depends much more on intellect than brute force, running ability or tolerance of physical fatigue.[56] Even in the savannah that Marino envisaged, !Kung bushmen can survive by working only 2.2 days per week.[57] Thus, if individuals are handicapped by a poor physique, they can compensate for this by working a slightly longer week. Moreover, hunting skills such as a knowledge of the habits of game, and the ability to fashion and use weapons are acquired progressively over the adult life-span.[56] In contrast, reproduction occurs as a young adult, and this limits the potential for genetic transmission of hunting skills. Further research may yet uncover genetically acquired characteristics favouring sustained exercise in small and isolated populations, but even if they were to be demonstrated, they would be unlikely to persist in the genetic melting pot that is our 21st century.

If natural selection were a major determinant of such traits as maximal oxygen intake and heat tolerance, one would anticipate the ready identification of molecular markers of these characteristics. Further research on the human genome may yet uncover consistent genetic markers of exercise tolerance and fatigue resistance, but positive findings have to date been meagre.[58] Some 50 genes can be modified by heat exposure, but to date most interindividual differences in both exercise tolerance and thermoregulation seem phenotypic rather than genotypic.[59]

Thus, at present we lack any strong evidence for the existence of selective pressures favouring the evolution of a thermally protective 'Central Governor' in humans.

2.2 Ability of a Putative 'Central Governor' to Protect against Hyperthermia and Ischaemia

If there is indeed a 'Central Governor' with a teleo-anticipatory mechanism designed to conserve homeostasis and protect the body against such hazards as hyperthermia and ischaemia, it seemingly has a limited effectiveness. In the

African hunting dog, where natural selection is suggested as having acted strongly to develop such a governor,[49] rectal temperatures climb several degrees higher than those seen in domestic dogs. This observation has been constrained to the 'Central Governor' hypothesis by drawing upon the concept[60] that the African hunting dog focuses on conservation of water rather than regulation of its core temperature. Exercising humans also face a significant toll of deaths and dangerous episodes of hyperthermia in hot weather, particularly during American football games[61] and endurance runs.[62,63] Available data suggest that there is a far from perfect evolution of mechanisms to ensure thermal homeostasis. Rectal temperatures of those undertaking prolonged exercise commonly reach a level of 42°C (compared with the prudent ceiling of 39.5°C imposed by most Committees on Human Experimentation), and 21 US football players are known to have died of heat stroke between 1995 and 2001.[64] Nevertheless, the number of individuals who die is small relative to those at risk, and some mechanism, whether peripheral or central, at least protected the majority of the population against such fatalities.

The putative system also seems to have a less than perfect ability to protect the exerciser against myocardial ischaemia and its fatal consequences. A bout of vigorous exercise can increase the risk of sudden cardiac death as much as 50-fold.[65] Possibly, many of those who succumb to a bout of exercise had some predisposing atherosclerosis. However, even in young adults, participation in an endurance or ultra-endurance event leads to signs usually interpreted as indicative of minor myocardial damage, such as the release of cardiac troponins, and an associated depression of myocardial function of variable duration.[66,67] Myocardial fibrosis may also develop with repeated participation in such events.[68] In older adults, episodes of severe exercise-induced myocardial ischaemia are commonplace.[69]

In similar fashion, there is incomplete protection against skeletal muscle damage following not only eccentric contractions but also prolonged bouts of endurance exercise.[70] About a half of ultramarathoners show elevated levels of circulating myoglobin following a race,[71] and sometimes this progresses to acute renal failure; 19 such cases were observed in the Comrades Marathon between 1969 and 1986.[72] Recent reviews by González-Alonso[73] and Cheung and Sleivert[74] comment specifically on the potential risks of impaired brain, heart and muscle function during and immediately following marathon running.

There is also limited protection against pulmonary hypertension, and some endurance competitors show marked right ventricular dysfunction[75] and pulmonary oedema following a prolonged bout of exercise.[76,77] Zavorsky[77] reviewed 11 studies performed in 137 subjects; approximately 65% of those who had performed a prolonged bout of maximal exercise showed radiographic evidence of pulmonary oedema.

Exercise plainly leads to large increases in demand on many homeostatic systems. The limited long-term morbidity and mortality that follows a sustained bout of vigorous physical activity could perhaps be construed as evidence for some process that limits physical activity sufficiently to avoid catastrophe in most people, although there are various well known mechanisms that could account for this, without invoking the action of a specific feed-forward 'Central Governor'. Thus, Hopkins[48] suggests that the fall in local reserves of adenosine triphosphate (ATP) inevitably limits the activity of both skeletal and cardiac muscle as fatigue develops, and the negative effect of insipient pulmonary oedema upon oxygen transfer likewise serves to brake the intensity of exercise.

2.3 Absence of a Ceiling of Cardiac Output during Maximal Effort

If a ceiling of cardiac output were to be reached during maximal effort, this would point strongly to a limitation of performance by oxygen transport rather than to a 'Central Governor'. The oxygen conductance equation[78] looks at the gradient of oxygen concentration from the inspired air to the active muscles. During vigorous exercise, the main concentration gradient is

between the pulmonary and the tissue capillaries, emphasizing that the individual's peak cardiac output is the primary factor limiting oxygen transport. Furthermore, since the oxygen concentration in the tissue capillaries is low, the implication seems to be that the limitation is imposed by the pumping ability of the heart rather than a restriction of peripheral demand, as would occur with a feed-forward control of motoneuron activity.

The standard understanding of the circulation has been that as the intensity of effort is increased, cardiac output reaches a plateau, mainly because a ceiling of heart rate has been reached, but also (particularly in older adults) because there is no further increase in cardiac stroke volume. The peripheral vasculature nevertheless retains the capacity to accept a larger blood flow.[79,80] Details of the cardiovascular response depend somewhat on the individual's posture, other details of the exercise protocol such as continuous versus discontinuous testing, and the steepness of the ramp function if a progressive exercise test is used. However, typically, the characteristics observed in a person who is approaching maximal oxygen intake (an ashen grey vasoconstriction of the skin, impaired coordination of the muscles, and a clouding of consciousness progressing to collapse) strongly support a cardiac limitation of performance. Possible underlying factors include local or general myocardial ischaemia, a failure of venous return, and restrictions imposed by the pericardium.[81,82] It has sometimes been objected that a pericardial limitation is unlikely, since the stroke volume is smaller when a person is exercising in a normal, upright position than when supine or semi-supine. This is certainly true of light and moderate exercise, where most of the experimental observations have been made, but in severe and maximal exercise, the cardiac output in the upright position is equal to or slightly greater than that seen when supine.[83] A second alternative possibility, suggested by Noakes and associates, is that blood flow, venous return and cardiac output are all ultimately limited by a central restriction of muscle recruitment; this possibility is addressed below.

Noakes and Marino[22] have argued against a plateauing of stroke volume, citing as support for their view a paper that applied the acetylene rebreathing technique to ten ordinary young men, ten runners and five elite runners;[84] this investigation used a rather steep ramp function, but nevertheless it demonstrated a clear plateauing of stroke volume in 20 of the 25 subjects. A second small study of seven normal young men and seven competitive cyclists also used the acetylene rebreathing technique; again a plateauing of stroke volume was seen in normal individuals, but values for the cyclists continued to increase up to the highest heart rate tested (190 beats/min, which did not coincide with attainment of an oxygen consumption plateau).[85] Other more recent studies have pushed equally fit subjects to an oxygen consumption plateau. These observations have demonstrated convincingly that the stroke volume does indeed plateau; indeed, it tends to decline as effort is increased, this decline being exacerbated by heat stress.[42,86-89]

The second plank in this argument[22] was that if cardiac output plateaued, myocardial ischaemia would necessarily develop.[13] Myocardial ischaemia was denied on the basis of two Scandinavian papers that had suggested a reserve of coronary oxygen supply during vigorous effort.[90,91] However, the proof of this point is less than convincing. In the first of these papers,[90] the level of exercise was only moderate (a heart rate of 130–140 beats/min). In the second paper, maximal effort was said to have been reached,[91] but it is unlikely that subjects attained a true maximal effort, since they had cardiac catheters inserted into their coronary sinuses. In the first study, a switch from air to the breathing of a hypoxic gas mixture (4500 m altitude equivalent) led to a small net production of lactate by the heart muscle, indicating that coronary vasodilatation was *not* able to compensate completely for the 27% decrease in arterial oxygen content. In the second study, hypoxia was less severe (2300 m altitude equivalent), but nevertheless some of the subjects again showed a net release of lactate from the myocardium.

The myocardial ischaemia inferred from lactate release also seems likely on theoretical

grounds. Maximal effort leads to a 6-fold increase in cardiac work rate.[1] However, oxygen extraction from the coronary vessels is relatively complete even under resting conditions, so that maintenance of an adequate oxygen supply during vigorous exercise depends almost entirely upon coronary vasodilatation. There is a potential for a 5-fold increase in coronary blood flow if the cardiac output increases sufficiently to sustain systemic blood pressure.[92] During maximal effort, any overall margin of coronary oxygen transport is very slender even in a young adult, and there is plainly potential to develop at least local pockets of myocardial ischaemia.

It is worth underlining that myocardial ischaemia is not the only possible reason for the observed plateauing of cardiac output. Autonomic function, the mechanics of diastolic filling and even the restraints of the pericardium could play a role. Nevertheless, there is considerable empirical evidence that some degree of myocardial ischaemia does develop during vigorous exercise. In younger adults, this is manifested by minor changes in the electrocardiogram and a release of cardiac troponins, and in many middle-aged and older adults the electrocardiogram indicates substantial myocardial ischaemia.[69] The progressive fall in peak heart rate with both aging[93] and exposure to hypoxic environments[94] provides some support to the concept that cardiac output is restricted by local ischaemia in the cardiac pacemaker. However, it remains puzzling why the breathing of oxygen-rich mixtures does not restore the maximal heart rate of the older individual to the values seen in a young adult. Possibly, the small increase in arterial oxygen content induced by inhaling oxygen-rich mixtures is insufficient to compensate for substantial deteriorations in regional myocardial blood flow.

2.4 Absence of an Oxygen Consumption Plateau

Noakes and associates have argued that an oxygen consumption plateau is rarely seen during incremental exercise testing.[4,95] If effort is limited by a feed-forward mechanism rather than a peaking of cardiac output, this would seem a logical corollary. In a review of studies worldwide that included children and the elderly but omitted key papers such as the definitive International Biological Programme Working Party Study,[96] Noakes and St Clair Gibson[13] found that a plateau had been demonstrated in about 50% of subjects. This in itself would seem to argue strongly against a 'Central Governor' in at least half of the population. Moreover, review of the papers cited suggests that failure to demonstrate a plateau in many of the remaining subjects reflected a failure to adopt the methodology recommended by the International Working Party.[96] Problems included: (i) failure to commence a definitive ramp test in the recommended manner (close to the individual's maximal oxygen intake[78]); (ii) use of a metabolic cart with an inappropriately brief gas sampling time;[97] (iii) use of a test mode differing from that of an athlete's specialty (e.g. testing runners or rowers on a cycle ergometer);[78] (iv) activation of an inadequate muscle mass (e.g. use of a cycle ergometer in people with weak quadriceps muscles);[42,78,98] and (v) poor motivation of the individual or limitations imposed by excessive caution of the observer (particularly in young children, the elderly and those with chronic disease).[78,99] The use of a steep ramp function was perhaps the most common problem. Two examples will suffice. One such study exercised five men and one woman on a treadmill, increasing the individual's oxygen intake by as much as 7.0 mL/kg/min per stage;[100] only three of the six subjects in this report reached a typical plateau. A second study was even less successful, finding a plateau in only 12 of the 71 individuals;[101] it used a cycle ergometer rather than a treadmill as the source of exercise, and it adopted a ramp function that began with zero loading of the ergometer and increased continuously over a 12-minute protocol. However, many investigators using a more appropriate methodology have had no problems in demonstrating a clear ceiling of oxygen consumption when testing healthy adults.[41,42,44,78,89,102-105] We noted that the few elderly individuals who failed to reach an oxygen consumption plateau

could be identified by an *a priori* rating of their motivation before and during the test.[78,93]

2.5 Neuromuscular Drive and the Electromyogram

If there were a 'Central Governor', this should limit neuromuscular drive and thus EMG activity as fatigue is approached in order to conserve homeostasis and avoid catastrophic biological failure.[10,52] In effect, the peak level of oxygen consumption would be set by this centrally determined drive. Given such a system, it becomes hard to explain why the maximal oxygen intake is augmented by blood transfusion or hyperoxia; it is easy to see how such measures could boost a peripheral limitation of oxygen transport, but much harder to explain how such treatment could modify a central drive.[48]

If there were a central limitation of drive, Noakes has argued that a subject would be likely to terminate exercise before reaching full activation of available motor units in the exercising limbs.[51] A magnetic resonance imaging study estimated that between 40% and 80% of potential muscle power was used during treadmill running to exhaustion.[106] Other information on this question is limited, much being based on surface or needle EMG recordings from one or two muscles. There are many limitations to interpreting such data in terms of motor unit recruitment and the generation of force and power during relatively complicated dynamic tasks. Kayser and associates[107] compared maximal exercise to exhaustion at sea level and after 1 month of acclimatization to an altitude of 5050 m (where the sea level maximal oxygen intake was reduced by some 20%). At sea level, exhaustion was associated with an increase rather than a decrease of the integrated EMG, with a sizeable increase in arterial lactate concentration and a decrease in arterial pH, a pattern the authors considered consistent with a peripheral limitation of effort. At altitude, the changes in lactate and pH were smaller and there was no increase of the EMG signal at fatigue, leading them to suggest that in this specific situation there could have been a central limitation of muscle drive; however, it is difficult to assess the influence of other factors such as respiratory distress, weight loss, dehydration and altered acid-base balance. Further evidence to support the expectations of the 'Central Governor' hypothesis was sought in 100 km cycling time trials.[108] The peak effort over the first 4 km high-intensity epoch of the trial averaged 318 W, with a mean integrated rectus femoris EMG that was 16% of that recorded during a maximal voluntary isometric contraction. In contrast, over the final 4 km high intensity epoch, power output had dropped to 278 W, and the integrated EMG showed a similar decrease, to 11% of that seen in the maximal voluntary contraction.[108] The authors concluded that from an early stage in the trial there had been a protective limitation of muscle activation, and that this protection had increased over the course of the trial. However, the decrease in the EMG signal could also reflect in part technical factors such as changes in temperature, conductivity and displacement of the electrodes, and a close relationship between the signal and power output seems unlikely. Unfortunately, this investigation did not examine changes in the maximal voluntary contraction at the end of the trial, which might have served to show how far the decrease in EMG reflected a protective decrease of central drive. Nicol and associates[109] demonstrated a decrease in maximal voluntary contraction following participation in a marathon run, although they wisely concluded that this could reflect either a decrease of motivation or a change in central recruitment tactics.

Stronger evidence against the 'Central Governor' came from a study of soleus fatigue induced by electrical stimulation of the peripheral nerves.[110] At fatigue, maximal plantar flexion was reduced, and a testing of the Hoffman reflex demonstrated a peripheral reflex inhibition of the alpha motoneurons innervating the muscle.[110] Electrical stimulation can induce a more powerful contraction than is possible by conscious effort but, nevertheless, the end-result of prolonged stimulation was fatigue and not catastrophic injury of the muscle; in this situation, there was no centrally imposed ceiling acting to prevent

catastrophe.[111] Again, in brief ramp function tests, motor unit activation does not reach a centrally defined ceiling at a power output corresponding to the individual's maximal oxygen intake; the rectified EMG increases substantially if subjects are persuaded to exercise at an intensity 5–15% above that corresponding to the oxygen consumption plateau.[42,89] Terminal intramuscular concentrations of lactate are high during both 'supramaximal' and sustained exhausting exercise,[78] and any decrease of EMG activity at fatigue seems likely to reflect an inadequate local blood flow, and an inhibition of phosphagen regeneration as lactate and/or hydrogen ions accumulate, rather than the action of a 'Central Governor'.

3. Factors Limiting Endurance Performance

From the foregoing discussion, we may conclude that, in humans, evidence concerning the potential for evolution of a 'Central Governor' and resulting protection against hyperthermia and ischaemia is equivocal. However, a person can increase his or her neuromuscular drive substantially above the level associated with a plateauing of oxygen transport, and the

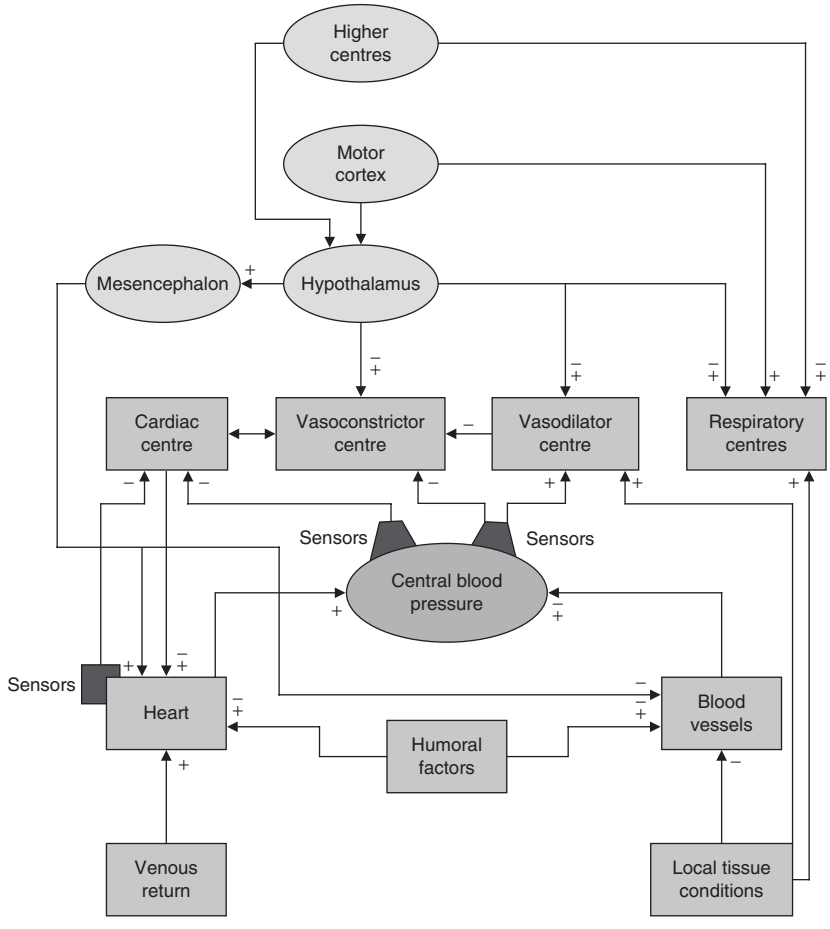

Fig. 1. Potential feedback loops regulating vigorous exercise.[1] + indicates positive feedback; – indicates negative feedback.

demonstrated ceilings of cardiac output and oxygen transport argue strongly against the 'Central Governor' hypothesis.

If there is no 'Central Governor', how is fatiguing exercise regulated? The limiting factors undoubtedly vary with the duration of activity. Noakes currently argues that the 'Central Governor' limits all forms of exercise, but much of the thinking of 'Central Governor' proponents concerns events such as a marathon or a supermarathon run, whereas critics of the hypothesis have often focused on much shorter bouts of exercise. A variety of standard texts have illustrated the many mutually redundant feedback loops that limit exercise (see for example, figure 1[1]). Among these, there is good empirical evidence for loops signalling peripheral ischaemia, hypoglycaemia and hyperthermia.[112] Plainly, there is also a potential for input from higher centres, including the motor cortex, but the effect is probably other than envisaged by Noakes and colleagues. The higher centres of an endurance athlete who is competing over a 1-mile track event call forth an initial effort to gain the desired position on the field; for most of the remaining distance, a combination of personal experience and coaching instruction hold oxygen demand just below the individual's maximal oxygen intake, at a level where a minimal accumulation of lactate in the peripheral muscles is sensed. A final sprint is begun at a distance set by coaching instruction, accumulated experience or a signal from a friend who is helping with pacing; the rate of energy expenditure is then increased so that the competitor's anaerobic capacity is fully exploited.[1,78,113] Various areas of the brain contribute to the choice of pace over an event, but the statement "only the Central Governor can explain this"[20] (p. 376) is plainly incorrect. Likewise, when determining maximal oxygen intake in a moderately fit adult, personal motivation or verbal stimulation from the monitoring physician can call forth a final extreme effort that overcomes most of the constraints imposed by feedback. Moreover, sustained effort can be limited by mental fatigue, and thus a greater perception of the required effort[114] – with a decreased cortical glucose supply contributing to this process.[112] Particularly in a hot environment, effort may be limited by both a failure of venous return, and an increase in ratings of perceived exertion at a given rate of working.[115] However, the supposed 'proof'[116] that cardiovascular factors do not limit exercise in the heat is based on moderate rather than maximal effort,[117] and it is hardly necessary to class an early reluctance[118] to exercise as hard in a hot environment as the action of an 'anticipatory Central Governor'.[116] In an older person the sensations arising from peripheral cardiac or skeletal muscle ischaemia (angina and intermittent claudication, respectively) can cause a termination of exercise. Over prolonged exercise, glycogen depletion is also undoubtedly a factor. Fatigue is postponed by dietary manipulations that increase intramuscular glycogen, and although muscle biopsies may show a residue of glycogen in some fibres at exhaustion, if the average reserves are decreased by 75%, depletion is likely complete in the most active fibres. Furthermore, the apparent absence of a depletion of Krebs cycle intermediaries in some biopsy specimens probably reflects regeneration of these substances in the interval between biopsy and freezing of the muscle sample.[119]

4. Conclusions

Over the past 13 years, a small group of investigators has argued repeatedly for the existence of a 'Central Governor' – an anticipatory feed-forward mechanism that regulates the intensity of vigorous effort with the intent of conserving homeostasis, thus protecting vital organs such as the brain, heart and skeletal muscle against hyperthermia and ischaemia. There seems mounting evidence against several key correlates of this hypothesis. It is difficult to discern evolutionary pressures that would favour the development of such a mechanism in humans. Protection of the exerciser against problems of hyperthermia and ischaemia is incomplete. Cardiac output generally reaches a plateau, perhaps in response to local oxygen lack. Most laboratories also have no difficulty in demonstrating an oxygen consumption plateau in well motivated young adults, and at least in some forms of

exercise EMG activity can increase after oxygen consumption has plateaued. Until there is convincing experimental evidence of an underlying physiological mechanism, most sports scientists will continue to express scepticism concerning the existence of a 'Central Governor'.[120]

Acknowledgements

No funding was received for the preparation of this article, and the author has no conflicts of interest that are directly relevant to the content of this article.

References

1. Shephard RJ. Physiology and biochemistry of exercise. New York (NY): Praeger Publications, 1982
2. Abbis CR, Laursen PB. Models to explain fatigue during prolonged endurance cycling. Sports Med 2005; 35: 865-98
3. Ulmer CV. Concept of an extracellular regulation of muscular metabolic rate during heavy exercise in humans by psychophysiological feedback. Experientia 1996; 52: 416-20
4. Noakes TD. How did A.V. Hill understand the VO_{2max} and the ''plateau phenomenon''? Still no clarity? Br J Sports Med 2008; 42: 574-80
5. Hill AV, Long CHN, Lupton H. Muscular exercise, lactic acid and the supply and utilisation of oxygen: parts VII-VIII. Proc Roy Soc B 1924; 97: 155-76
6. Noakes TD. Physiological models to understand exercise fatigue and the adaptations that predict or enhance athletic performance. Scand J Med Sci Sports 2000; 10: 123-45
7. Hampson DB, St Clair Gibson A, Lambert MI, et al. The influence of sensory cues on the perception of exertion during exercise and central regulation of exercise performance. Sports Med 2001; 31: 935-52
8. Kay D, Marino FE, Cannon J, et al. Evidence for neuromuscular fatigue during high intensity cycling in warm, humid conditions. Eur J Appl Physiol 2001; 84: 115-21
9. Noakes TD, Peltonen JE, Rusko HK. Evidence that a central governor regulates exercise performance during acute hypoxia and hyperoxia. J Exp Biol 2001; 204 Pt 18: 3225-34
10. Noakes TD. Linear relationship between the perception of effort and the duration of constant load exercise that remains. J Appl Physiol 2004; 96: 1571-2
11. Ansley L, Schabort EJ, St Clair Gibson A, et al. Regulation of pacing strategies during successive 4 km time trials. Med Sci Sports Exerc 2004; 38: 1819-25
12. Noakes TD, St Clair Gibson A, Lambert EV. From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans. Br J Sports Med 2004; 38: 511-4
13. Noakes TD, St Clair Gibson A. Logical limitations to the catastrophe models of fatigue during exercise in humans. Br J Sports Med 2004; 38: 648-9
14. St Clair Gibson A, Noakes TD. Evidence for complex system regulation and dynamic neural regulation if skeletal muscle recruitment during exercise in humans. Br J Sports Med 2004; 38: 797-806
15. Noakes TD, Calbet JA, Boshel R, et al. Central regulation of skeletal muscle recruitment explains the reduced maximal cardiac output during hypoxia. Am J Physiol 2004; 287: R996-9
16. Tucker R, Rauch I, Harley YXR, et al. Impaired exercise performance in the heat is associated with an anticipatory reduction in skeletal muscle recruitment. Pflüg Archiv 2004; 448: 422-30
17. Lambert M, St Clair Gibson A, Noakes TD. Complex systems model of fatigue: integrative homeostatic control of peripheral physiological systems during exercise in humans. Br J Sports Med 2005; 39: 52-62
18. Tucker R, Marle T, Lambert EV. The rate of heat storage mediates an anticipatory reduction in exercise intensity during cycling at a fixed rating of perceived exertion. J Physiol 2006; 574: 905-15
19. Noakes TD. Determining the extent of neural activation during maximal effort [abstract]. Med Sci Sports Exerc 2007; 39: 2092
20. Noakes TD. The central governor of exercise regulation applied to the marathon. Sports Med 2007; 37: 374-77
21. Noakes TD. Testing for maximum oxygen consumption has produced a brainless model of human exercise performance. Br J Sports Med 2008; 42: 551-5
22. Noakes TD, Marino FE. Maximal oxygen uptake is limited by a central nervous system governor. J Appl Physiol 2009; 106: 338-9
23. Crewe H, Tucker R, Noakes TD. The rate of increase of perceived exertion predicts the duration of exercise to fatigue at a fixed power output in different environmental conditions. Eur J Appl Physiol 2008; 103: 569-77
24. Noakes TD, Tucker R. Do we really need a central governor to explain brain regulation of exercise performance? A response to the letter of Dr Marcora. Eur J Appl Physiol 2008; 104: 933-5
25. Kayser B. Exercise starts and ends in the brain. Eur J Appl Physiol 2003; 90: 411-9
26. Abbis CR, Laursen PB. Describing and understanding pacing strategies during athletic competition. Sports Med 2008; 38: 239-52
27. Baron B, Noakes TD, Dekerle J, et al. Why does exercise terminate at the maximal lactate steady state intensity. Br J Sports Med 2008; 42: 528-33
28. Baron B, Deruelle F, Moullan F, et al. The eccentric muscle loading influences the pacing strategies during repeated downhill sprint intervals. Eur J Appl Physiol 2009; 105: 749-57
29. Castle PC, Macdonald AL, Philp A, et al. Precooling leg muscle improves intermittent sprint exercise performance in hot, humid conditions. J Appl Physiol 2006; 100: 1377-84

30. Clark SA, Bourdon PC, Schmidt W, et al. The effect of acute simulated altitude on power, performance and pacing strategies in well-trained cyclists. Eur J Appl Physiol 2007; 102: 45-55
31. Edwards AM, Mann ME, Marfell-Jones MJ, et al. Influence of moderate hydration on soccer performance: physiological responses to 45 min of outdoor match-play and the immediate subsequent performance of sport-specific and mental concentration tests. Br J Sports Med 2007; 41: 385-91
32. Eston R, Faulkner J, St Clair Gibson A, et al. The effect of antecedent fatiguing activity on the relationship between perceived exertion and physiological activity during a constant load exercise task. Psychophysiol 2007; 44: 779-86
33. Flouris AD, Cheung SS. Human conscious response to thermal input is adjusted to changes in mean body temperature. Br J Sports Med 2009; 43: 199-203
34. Joseph T, Johnson B, Battista RC, et al. Perception of fatigue during simulated competition. Med Sci Sports Exerc 2008; 40: 381-6
35. Kabitz H-J, Walker D, Schwoerer A, et al. New physiological insights into exercise-induced diaphragmatic fatigue. Resp Physiol Neurobiol 2007; 158: 88-96
36. Morante SM, Brotherhood JR. Autonomic and behavioural thermoregulation in tennis. Br J Sports Med 2008; 42: 679-85
37. Nummela AT, Heath KA, Paavolainen LM, et al. Fatigue during a 5-km running time trial. Int J Sports Med 2008; 29: 738-45
38. Racinais S, Bringard A, Puchaux K, et al. Modulation in voluntary neural drive in relation to muscle soreness. Eur J Appl Physiol 2008; 102: 439-46
39. Ross EZ, Middleton N, Shave R, et al. Corticomotor excitability contributes to neuromuscular fatigue following marathon running in man. Exp Physiol 2007; 92: 417-26
40. Thomas R, Stephens P. Prefrontal cortex oxygenation and neuromuscular responses to exhaustive exercise. Eur J Appl Physiol 2008; 102: 153-63
41. Bergh U, Ekblom B, Åstrand PO. Maximal oxygen uptake "classical" versus "contemporary" viewpoints. Med Sci Sports Exerc 2000; 32: 85-8
42. Brink-Elfegoun T, Holmberg H-C, Ekblom MN, et al. Neuromuscular and circulatory adaptations during combined arm and leg exercise with different maximal work loads. Eur J Appl Physiol 2007; 101: 603-11
43. di Prampero PE, Capelli C, Ferretti G. Positive effects of intermittent hypoxia (live high:train low) on exercise performance are/are not mediated primarily by augmented red cell volume [comments on point:counterpoint]. J Appl Physiol 2005; 99: 2453-62
44. Snell PG, Stray-Gundersen J, Levine BD, et al. Maximal oxygen uptake as a parametric measure of cardiorespiratory capacity. Med Sci Sports Exerc 2007; 39: 103-7
45. Shephard RJ. Is the measurement of maximal oxygen intake passé? Br J Sports Med 2009; 43: 83-5
46. Shephard RJ. Hard evidence for a central governor is still lacking. J Appl Physiol 2009; 106: 343
47. Foster C. Untitled. J Appl Physiol 2009; 106: 343
48. Hopkins WG. The implausible governor. Sportscience 2009; 13: 9-11
49. Marino FE. The evolutionary basis of thermoregulation and exercise performance. In: Marino FE, editor. Thermoregulation and human performance: physiological and biological aspects. Basel: Karger Publications, 2008: 1-13
50. Noakes TD. Implications of exercise testing for prediction of athletic performance: a contemporary perspective. Med Sci Sports Exerc 1988; 20: 319-30
51. Noakes TD. Maximal oxygen uptake: "classical" versus "contemporary" viewpoints – a rebuttal. Med Sci Sports Exerc 1998; 30: 1381-98
52. St Clair Gibson A, Schabort EJ, Noakes TD. Reduced neuromuscular activity and force generation during prolonged cycling. Am J Physiol 2001; 281: R187-96
53. Mosso A. Fatigue. London: George Allen & Unwin, 1915
54. Marino FE. Comparative thermoregulation and the quest for athletic supremacy. In: Marino FE, editor. Thermoregulation and human performance: physiological and biological aspects. Basel: Karger Publications, 2008: 14-25
55. Shephard RJ. Human physiological work capacity. London: Cambridge University Press, 1978
56. Godin G, Shephard RJ. Activity patterns of the Canadian Eskimo. In: Edholm O, Gunderson EK, editors. Human polar biology. Cambridge: Heinemann, 1973
57. Lee RB. Kung bushmen subsistence: an input-output analysis. In: Vayda AP, editor. Environment and cultural behavior. New York (NY): Natural History Press, 1969
58. Wolfarth B, Rankinen T, Muhlbauer S, et al. Association between a beta2-adrenergic receptor polymorphism and elite endurance performance. Metab Clin Exper 2007; 56: 1649-51
59. Lambert MI, Mann T, Dugas JP. Ethnicity and temperature regulation. In: Marino FE, editor. Thermoregulation and human performance: physiological and biological aspects. Basel: Karger Publications, 2008: 104-20
60. Taylor CR, Schmidt-Nielsen K, Dmi'el R, et al. Effect of hyperthermia on heat balance during running in the African hunting dog. Am J Physiol 1971; 220: 823-7
61. Luke AC, Bergeron MF, Roberts WO. Heat injury prevention practices in high school football. Clin J Sports Med 2007; 17: 488-93
62. Pugh LGCE, Corbett JL, Johnson RH. Rectal temperatures, weight losses and sweat rates in marathon running. J Appl Physiol 1967; 23: 347-52
63. Sutton JR, Coleman MJ, Millar AP, et al. The medical problems of mass participation in athletic competition: the "City to Surf" race. Med J Aust 1972; 2: 127-33
64. Mueller FO, Cantu RC. Twentieth annual report: Fall 1982-Spring 2002, National Center for Catastrophic Sport Injury Research. Chapel Hill (NC): University of North Carolina, National Center for Catastrophic Sport Injury Research, 2004
65. Vuori I. Exercise and sudden cardiac death: effects of age and type of activity. Sports Sci Rev 1995; 4 (2): 46-84
66. Neilan TG, Yoerger D, Douglas P, et al. Persistent and reversible cardiac dysfunction among amateur marathon runners. Eur Heart J 2006; 27: 1079-84

67. Neilan TG, Jnauzzi JL, Lee-Lewandrowski E, et al. Myocardial injury and ventricular dysfunction related to training levels among nonelite participants in the Boston marathon. Circulation 2006; 114: 2325-33
68. Whyte GP. Clinical significance of cardiac damage and changes in function after exercise. Med Sci Sports Exerc 2008; 40: 1416-23
69. Shephard RJ. Ischemic heart disease and exercise. London: Croom Helm, 1981
70. Clarkson PM. Exertional rhabdomyolysis and acute renal failure in marathon runners. Sports Med 2007; 27: 361-3
71. Schiff HB, MacSearraigh ET, Kallmeyer JC. Myoglobinuria, rhabdomyolysis and marathon running. Quart J Med 1978; 47: 463-72
72. Seedat YK, Aboo N, Naicker S, et al. Acute renal failure in the ''Comrades Marathon'' runners. Renal Failure 1989; 11: 209-12
73. González-Alonso J. Hyperthermia impairs brain, heart and muscle function in exercising humans. Sports Med 2007; 37: 371-3
74. Cheung SS, Sleivert GG. Multiple triggers for hyperthermic fatigue and exhaustion. Ex Sport Sci Rev 2004; 32: 100-6
75. Lagerche A, Prior D. Exercise: is it possible to have too much of a good thing? Heart Lung Circ 2007; 16 Suppl. 3: S102-4
76. McKechnie JK, Lear WP, Noakes TD, et al. Acute pulmonary edema in two athletes during a 90 km running race. S Afr Med J 1979; 56: 261-5
77. Zavorsky GS. Evidence of pulmonary oedema triggered by exercise in healthy humans and detected with various imaging techniques. Acta Physiol 2007; 189: 305-17
78. Shephard RJ. Aerobic fitness and health. Champaign (IL): Human Kinetics, 1994
79. Andersen P, Saltin B. Maximal perfusion of skeletal muscle in man. J Physiol 1985; 366: 233-49
80. Rowell LB. Human cardiovascular control. Oxford: Oxford University Press, 1993
81. Stray-Gundersen J, Musch TI, Haidet GC, et al. The effect of pericardiectomy on maximal oxygen consumption and maximal cardiac output in untrained dogs. Circulation Res 1986; 58: 523-30
82. Hammond HK, White FC, Bhargava V, et al. Heart size and maximal cardiac output are limited by the pericardium. Am J Physiol 1992; 263: H1675-81
83. Leyk D, Egfeld D, Hoffmann U, et al. Postural effect on cardiac output, oxygen uptake and lactate during cycle exercise of varying intensity. Eur J Appl Physiol 1994; 68: 30-5
84. Zhou B, Conlee RK, Jensen R, et al. Stroke volume does not plateau during graded exercise in elite male distance runners. Med Sci Sports Exerc 2001; 33: 1849-54
85. Gledhill N, Cox D, Jamnik R. Endurance athletes' stroke volume does not plateau: major advantage is diastolic function. Med Sci Sports Exerc 1994; 26: 1116-21
86. González-Alonso J, Calbet J. Reductions in systemic and skeletal muscle blood flow and oxygen delivery limit maximal aerobic capacity in humans. Circulation 2003; 107: 824-30
87. Mortensen SP, Dawson EA, Yoshiga CC, et al. Limitations to systemic and locomotor limb muscle oxygen delivery and uptake during maximal exercise in humans. J Physiol 2005; 566: 273-85
88. Mortensen SP, Damsgaard R, Dawson EA, et al. Restrictions in systemic and locomotor skeletal muscle perfusion, oxygen supply and VO_2 during high-intensity whole-body exercise in humans. J Physiol 2008; 586: 2621-35
89. Ekblom B. Point/counterpoint: maximal oxygen intake is not limited by a central nervous system governor. J Appl Physiol 2009; 106: 341-2
90. Kaijser L, Grubbstrom J, Berglund B. Myocardial lactate release during prolonged exercise under hypoxemia. Acta Physiol Scand 1993; 149: 427-33
91. Grubbstrom J, Berglund B, Kaijser L. Myocardial blood flow and lactate metabolism at rest and during exercise with reduced arterial oxygen content. Acta Physiol Scand 1991; 142: 567-74
92. Vatner SF, Higgins CB, Franklin D, et al. Role of tachycardia in mediating the coronary hemodynamic response to severe exercise. J Appl Physiol 1972; 32: 380-9
93. Shephard RJ. Aging, physical activity and health. Champaign (IL): Human Kinetics, 1997
94. Pugh LGCE. Physiological and medical aspects of the Himalayan Scientific and Mountaineering Expedition, 1960-1961. BMJ 1962; 2: 621-7
95. Doherty M, Nobbs I, Noakes TD. Low frequency of the ''plateau phenomenon'' during maximal exercise in elite British athletes. Eur J Appl Physiol 2003; 89: 619-23
96. Shephard RJ, Allen C, Benade AJS, et al. The maximum oxygen intake: an international reference standard of cardio-respiratory fitness. Bull WHO 1968; 38: 757-64
97. Myers J, Walsh D, Sullivan M, et al. Effect of sampling on variability and plateau in oxygen uptake. J Appl Physiol 1990; 68: 404-10
98. Shephard RJ, Bouhlel E, Vandewalle H, et al. Muscle mass as a factor limiting physical work. Eur J Appl Physiol 1988; 64: 1472-9
99. Astorino TA, Willey J, Kinnahan J, et al. Elucidating determinants of the plateau in oxygen consumption at VO_{2max}. Br J Sports Med 2005; 39: 655-60
100. Myers J, Walsh D, Buchanan N, et al. Can maximal cardiopulmonary capacity be recognized by a plateau in oxygen uptake? Chest 1989; 96: 1312-6
101. Day JR, Rossiter HB, Coats EM, et al. The maximally attainable VO_2 during exercise in humans: the peak vs maximum issue. J Appl Physiol 2003; 95: 1901-7
102. Åstrand PO, Saltin B. Maximal oxygen uptake and heart rate in various types of muscular activity. J Appl Physiol 1961; 16: 977-81
103. Hill AV, Lupton H. Muscular exercise, lactic acid, and the supply and utilisation of oxygen. Quart J Med 1923; 16: 135-71
104. Taylor HL, Buskirk ER, Henschel A. Maximal oxygen intake as an objective measure of cardio-respiratory performance. J Appl Physiol 1955; 8: 73-80

105. Wagner PD. New ideas on limitations to VO_{2max}. Exerc Sport Sci Rev 2000; 28: 10-4
106. Sloniger MA, Cureton KJ, Prior BM, et al. Lower extremity muscle activation during horizontal and uphill running. J Appl Physiol 1997; 83: 2073-9
107. Kayser B, Narici M, Binzoni T, et al. Fatigue and exhaustion in chronic hypobaric hypoxia: influence of exercising muscle mass. J Appl Physiol 1994; 76: 634-40
108. St Clair Gibson A, Lambert MI, Noakes TD. Neural control of force output during maximal and submaximal exercise. Sports Med 2001; 31: 637-50
109. Nicol C, Komi PV, Marconnet P. Fatigue effects of marathon running on neuromuscular performance. Scand J Sci Sports 1991; 1: 18-24
110. Garland SJ, McComas A. Reflex inhibition of human soleus muscle during fatigue. J Physiol 1990; 429: 17-27
111. Spriet LL, Soderlund KK, Bergstrom M, et al. Anaerobic energy release in skeletal muscle during electrical stimulation in man. J Appl Physiol 1987; 62: 611-5
112. Hargreaves M. Fatigue mechanisms determining exercise performance: integrative physiology is systems biology. J Appl Physiol 2008; 104: 1541-2
113. Tucker R, Lambert MI, Noakes TD. An analysis of pacing strategies during men's world performances in track athletics. Int J Sports Physiol Perf 2006; 1: 233-45
114. Marcora S, Staiano W, Manning V. Mental fatigue impairs physical performance in humans. J Appl Physiol 2009; 106: 857-64
115. Galloway SDR, Maughan RJ. Effect of ambient temperature on the capacity to perform prolonged cycle exercise in man. Med Sci Sports Exerc 1997; 29: 1240-9
116. Tucker R. Thermoregulation, fatigue, and exercise modality. In: Marino FE, editor. Thermoregulation and human performance: physiological and biological aspects. Basel: Karger Publications, 2008: 26-38
117. Savard GK, Nielsen B, Laszczynska J, et al. Muscle blood flow is not reduced in humans during moderate exercise and heat stress. J Appl Physiol 1988; 64: 649-57
118. Tatterson AJ, Hahn AG, Martin DT, et al. Effect of heat and humidity on time trial performance in Australian national team road cyclists. J Sci Med Sport 2000; 3: 186-93
119. Baldwin J, Snow RJ, Gibala MJ, et al. Glycogen availability does not affect the TCA cycle or TAN pools during prolonged, fatiguing exercise. J Appl Physiol 2003; 94: 2181-7
120. Weir JP, Beck TW, Cramer JT, et al. Is fatigue all in your head? A critical review of the central governor model. Br J Sports Med 2006; 40: 573-86

Correspondence: Dr *Roy J. Shephard*, PO Box 521, Brackendale, BC V0N 1H0, Canada.
E-mail: royjshep@shaw.ca