EDUCATION

From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans: summary and conclusions

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It is hypothesised that physical activity is controlled by a central governor in the brain and that the human body functions as a complex system during exercise. Using feed forward control in response to afferent feedback from different physiological systems, the extent of skeletal muscle recruitment is controlled as part of a continuously altering pacing strategy, with the sensation of fatigue being the conscious interpretation of these homoeostatic, central governor control mechanisms.

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The first conclusion of this symposium is that the peripheral "limitations" or "catastrophe" models of exercise physiology are unlikely to be correct as they are unable to explain a number of common observations in the exercise sciences. The most compelling of these are reviewed.

FATIGUE AND WHOLE BODY HOMOEOSTASIS

Firstly, fatigue during any form of exercise occurs without evidence of any related failure of whole body homoeostasis as predicted by the "limitations" or "catastrophe"[1] models. Perhaps the clearest examples are the fatigue that develops in extreme hypoxia at high altitudes[2 3] and during exercise in conditions of severe heat.[4–6] In both cases, exercise terminates before there is any evidence of a catastrophic failure of either metabolic[3 5] or thermoregulatory[4–6] regulation.

For example, at extreme altitude, exercise terminates when symptoms of dyspnoea are severe; yet it is precisely the very high rates of ventilation causing that dyspnoea that maintain life by ensuring that the arterial Po_2 remains above about 30 mm Hg necessary to sustain consciousness.[7] There is no published evidence showing that homoeostasis in any other organ system is threatened during exercise at altitude; cardiovascular function in particular is entirely appropriate for the levels of exercise that are achieved,[8] whereas the low muscle lactate concentrations and submaximal cardiac output indicate that muscle hypoxia cannot be present.[9 10] In contrast, the prediction of the central governor model is that the central nervous system (CNS)[8 9] determines the work rate that can be sustained for the anticipated duration of exercise, as well as the moment at which exercise terminates, all to ensure that homoeostasis is

maintained. The decisive studies of Kayser,[2] Nybo and Nielsen,[4] and Gonzalez-Alonso *et al*[6] would now appear to confirm this interpretation, at least for exercise at altitude and in the heat.

Even during maximal exercise of short duration, which, according to the Hill cardiovascular/anaerobic/catastrophic model, is limited by a severe uncompensated metabolic acidosis, arterial blood pH seldom falls below about 7.1 and muscle pH below 6.6.[11 12] However, when isolated perfused skeletal muscle was tetanically stimulated to similar pH levels (~6.8) in vitro in the absence of an intact CNS, mechanical failure of the skeletal muscle only occurred at low temperatures (~12°C).[13 14] In contrast, at normal physiological temperatures (~32°C), force output was not affected by reduction of pH to this level.[13 14] These findings led the authors to suggest that direct reduction in force production by pH was not a significant factor in muscle fatigue at normal or raised physiological temperatures.[13 14] More probably, a developing metabolic acidosis may induce a progressively increasing homoeostatic respiratory compensation, which increases the perception of discomfort, at the same time producing a compensatory alkalosis.[16] This escalating discomfort ultimately causes the conscious decision to terminate the exercise bout.[15]

Furthermore, skeletal muscle ATP concentrations are never reduced to less than 50% of the resting value under all conditions of exercise, including maximal exercise in persons with a reduced capacity to generate ATP either oxidatively or glycolytically, or in muscles rendered ischaemic by the application of a tourniquet and forced to contract until exhaustion by external stimulation.[9] This is especially remarkable, given the small size of the total muscle ATP stores. Thus muscle ATP concentrations are as tightly regulated during exercise as they are at rest.

Yet a core teaching of the peripheral "limitations" or "catastrophe"[1] models is that physical exhaustion occurs as a direct consequence of a failure of homoeostasis—the linear model described by St Clair Gibson and Noakes.[17] Thus the presence of homoeostasis in all organ systems at the point of exhaustion is perhaps the most robust evidence supporting the hypothesis that exercise performance is regulated centrally in the brain as part of a complex dynamic system, the principal function of which is specifically to ensure that homoeostasis is maintained under all conditions of exercise. Accordingly, we have postulated that the ultimate determinant of

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this homoeostatic regulation is the CNS, which continuously alters the number of motor units that are recruited during exercise, thereby setting the total metabolic demand. Hence physical exhaustion develops and exercise terminates as part of this homoeostatic regulation rather then as a catastrophic, system-limiting event as originally hypothesised by Edwards.[1]

This new interpretation is the first to allow a more reasonable description of a number of phenomena that defy rational explanation according to the traditional "limitations" models of fatigue.[18] These include, among many others, the chronic fatigue syndrome, in which affected individuals experience evident fatigue at rest,[19] and the role of psychological and motivational factors,[20] centrally (brain) acting pharmaceutical agents,[15 21 22] hypnosis, shouting or sudden unexpected gunshots,[21] or other forms of distraction including music or premeditated deception on human exercise performance.[23 24]

RECRUITMENT OF MOTOR UNITS

The second relevant conclusion of this symposium is that the validity of the peripheral "limitations" or "catastrophe" models requires that all available motor units in the exercising limbs must be recruited at exhaustion, regardless of the duration or intensity of the activity.[9] For peripheral factors alone cannot cause the termination of exercise when a majority of the available motor units are inactive at the point of exhaustion. This question is especially relevant in the case of prolonged exercise when less than ~30% of the available motor units may be active at exhaustion in recreational athletes.[25 26] Although elite athletes are likely to recruit a greater proportion of their available motor unit reserve, there is no reason to believe that this would be by more than a maximum of 10–20% greater than that achieved by recreational athletes under the same conditions of exercise. Certainly it is unlikely to be more than 50% during prolonged exercise lasting more than an hour. Thus the question of logic requiring an answer is: why, at exhaustion during prolonged exercise, does the brain fail to recruit additional motor units so that the exercise can continue?

The prediction of the central governor model[17] is that the brain does not recruit additional motor units during prolonged exercise because such additional recruitment would threaten the capacity to maintain homoeostasis, potentially leading to, at best, the premature termination of exercise, or, at worst, organ damage, organ failure, and even perhaps death. This model also predicts that the rising perception of discomfort produced by exhausting exercise[27] progressively reduces the conscious desire to over-ride this control mechanism, which, if it were to be reduced, would lead to the recruitment of more motor units. Thus the presence of conscious over-ride would be undesirable because it would increase or maintain the exercise intensity, thereby threatening homoeostasis.

Indeed the historical, albeit subconscious assumption, that motor unit recruitment by the CNS is always maximal and hence cannot be further increased at exhaustion regardless of the nature of the activity, or its duration, or the environmental conditions under which the exercise is performed, is perhaps the single most compelling reason why the peripheral "catastrophe" models have escaped serious scientific scrutiny for the past 75 years.

But if any fewer than 100% of the available motor units in the active limbs are recruited at exhaustion, then it is not possible to presume that something other than the CNS definitely limited that particular activity.[21 28] For under such circumstances, there can be no certainty that the recruitment of even a few additional motor units would not further increase the work rate and the rates of oxygen consumption and lactate production, thereby proving that those respective rates, previously considered to be maximal, were indeed submaximal and were consequent only on incomplete recruitment of the total number of motor units in the exercising limbs.

Noakes and St Clair Gibson[9] present the evidence supporting their argument that skeletal muscle motor unit recruitment is never 100% even in supposedly maximal isometric contractions.[21 28] Neurophysiologists have long accepted that the progressive reduction in force production during sustained isometric contractions or during prolonged exercise is due principally to a progressive reduction in central neural command.[28] Thus a number of recent studies reviewed by Noakes and St Clair Gibson[9] confirm Gandevia's[28] telling conclusion that "human muscle fatigue does not simply reside in the muscle."

Why is it that exercise physiologists seem to have assumed that skeletal muscle recruitment must always be maximal during exhaustive exercise? One logical explanation could quite simply be that that is exactly how it feels; that the sensations that develop during exhausting exercise make it feel as if not a single additional muscle fibre could possibly be activated for exercise to continue at the same intensity. Another possibility is that few exercise scientists other than neurophysiologists routinely consider electromyographic evaluation to be of special value, and few use it as an indirect estimate of the extent of skeletal muscle recruitment during exercise.

Thus one of the most important intellectual advances resulting from this symposium is that the presence or absence of skeletal muscle anaerobiosis is not, as is usually argued,[29 30] even by ourselves,[31 32] the definitive test of the original cardiovascular/anaerobic/catastrophic model. Rather it is whether or not there is ever complete skeletal muscle recruitment at exhaustion. For, if there is skeletal muscle reserve at the point of physical exhaustion, then fatigue cannot be caused by a peripherally based control but must result from CNS regulation of skeletal muscle motor unit recruitment. By reviewing the evidence showing that skeletal muscle motor unit recruitment is never complete during any voluntary form of exercise, the authors believe that they have finally unearthed the crucial logical flaw in Hill's original cardiovascular/anaerobic/catastrophic model.

INTERPRETATION OF THE TERM FATIGUE

The third specific contribution of this symposium is to suggest a novel interpretation of the term fatigue. For, as St Clair Gibson and Noakes[17] have argued, if the peripheral model of "fatigue" is correct, then there is no teleological value to the potent physical sensations and emotions that arise towards the end of exercise as physical work capacity begins to decline. For the logical question is: if exercise performance is regulated peripherally in the muscles, why should these symptoms develop, as they can serve no purpose? Why should the brain be informed of peripheral events that are beyond its control, as, according to the "catastrophe" models, no response of the conscious brain can arrest the irreversible, peripheral metabolic events that progressively inhibit skeletal muscle contraction, ultimately causing the termination of exercise?

Rather the prediction of the central governor model is that, as exercise performance is centrally regulated by the CNS, then fatigue should no longer be considered a physical event but rather a sensation or emotion, separate from an overt physical manifestation—for example, the reduction in force output by the active muscles. Rather we now suggest that the physical manifestation of any increasing perception of fatigue may simply be an alteration in the subconsciously regulated pace at which the exercise is performed. Hence the novel

suggestion is that the conventional understanding of fatigue is flawed because it makes no distinction between the sensation itself and the physical expression of that sensation which, we suggest, is the alteration in the subconsciously regulated pacing strategy consequent on changing motor unit recruitment/derecruitment by the CNS.[17]

Thus the conclusion of these papers is that it may be more correct to understand fatigue as a sensory perception that results from the complex integration of physiological, biochemical, and other sensory feedback from the periphery and which may or may not be associated with any alteration in muscle force production. Thus, according to this definition, the sensations of fatigue can be present at rest when no work is being performed, as is found in the chronic fatigue syndrome[19] and other chronic diseases; or they may begin shortly after the onset of exercise, or they may peak even as the work output increases further, as in the case of the "end spurt" in the world record 10 000m running performances (fig 11 in Noakes and St Clair Gibson[9]). According to the predictions of the central governor model, the presence of fatigue at rest in chronic disease can be best understood as part of a protective mechanism to ensure that the metabolic rate cannot be elevated excessively through the recruitment of too large a muscle mass during activity, as this would overwhelm those homoeostatic mechanisms that have been impaired by the presence of disease.

This new definition raises the question of what might be the possible teleological value of the sensations of fatigue. Perhaps another insight comes from the high altitude analogy that has been so central to the development of some of these ideas.[2] It seems clear that physiological function at extreme altitude is directed to prevent the arterial Po_2 from falling below about 30 mm Hg.[7] The cost of the concerted respiratory effort to defend the Po_2 is that the Pco_2 falls, whereas pH and ventilation both rise, reaching values often exceeding those measured during maximum exercise at sea level.[33] Under these conditions, the perception of dyspnoea is extreme and even the gentlest physical activity is almost impossible. The classic statement of the first climber to summit Mount Everest without supplemental oxygen, Reinhold Meissner, describes the sensations he experienced on that summit: "As we get higher, it becomes necessary to lie down to recover our breath … at a height of 8800 meters, we can no longer keep on our feet while we rest….Breathing becomes such a strenuous business that we scarcely have strength left to go on …. I am nothing more than a single, narrow, gasping lung floating over the mists and the summits".[7]

The point is that the variable under greatest threat at extreme altitude is the arterial Po_2. Thus the maintenance of arterial Po_2 homoeostasis at extreme altitude requires that the ventilatory effort is maximised and any other factor threatening that homoeostasis, in particular physical activity, must be minimised or indeed prevented. Accordingly, the central governor model predicts that the function of the brain at altitude is actively to regulate all additional threats to homoeostasis, most especially that posed by uncontrolled physical activity. In this case, the extreme sensations of fatigue would act as a conscious deterrent to further exercise; in the words of Meissner, "we scarcely have strength left to go on".[7]

Thus one conclusion may be that it is not the physical exertion of climbing that causes the extreme symptoms that are also present at rest on the summit of Mount Everest but rather, in this specific example, it is the ventilatory effort required to maintain homoeostasis that causes such profound symptoms. If it is possible to generalise this conclusion to other examples, then we might suggest that the symptoms of fatigue may be related in some way to the biological or neural effort expended in the continuing life long battle to maintain

homoeostasis. Alternatively, the rate at which these fatigue symptoms increase during exercise may be related to the duration of the exercise that can be safely sustained at that particular exercise intensity under those specific conditions.[27]

PACING STRATEGIES

The fourth novel contribution of this symposium is to suggest that the peripheral "limitations" or "catastrophe" models of fatigue are quite unable to explain one of the most fundamental features of competitive sport, which is the rapid adoption, shortly after the onset, of different pacing strategies during exercise of very different intensities and durations.

The unambiguous observations that, we suggest, cannot be explained by the peripheral limitations model are the rapidity with which the successful pacing strategy is adopted: within about two to three seconds in a 100 m sprint and within the first few hundred metres in all longer running events.[34] Clearly these decisions cannot be based on metabolic events in the active muscles because a metabolic steady state is not achieved so expeditiously. Nor can the peripheral limitations model explain why elite athletes are able to increase their pace during the last 5–10% of the race (fig 11 in Noakes and St Clair Gibson[9]) when fatigue should be greatest because the concentration of the "poisonous" intramuscular metabolites should be the highest.

Furthermore, if fatigue is peripherally regulated by increasing accumulation of a "poisonous" metabolite, which "deactivates the enzymes for energy production and makes you slow down",[35] then it is not possible ever to run faster than one particular speed, regardless of the duration of the activity.[9] However, the clear evidence is that athletes pace themselves at myriad different speeds, both within and between different events, and that these chosen paces are highly dependent on the expected duration of the activity. In addition, the peripheral "limitations" model predicts that the slowest running speeds must occur when the concentrations of these inhibitory metabolites are the highest whereas the fastest speeds are achieved when they are at their lowest concentrations. This prediction is the precise opposite of what is found, proving that, if peripheral metabolites do indeed influence the pacing strategy, they most likely act through afferent sensory feedback to specific centres in the brain.[36]

However, the central governor model does not exclude the possibility that certain metabolites could indeed impair skeletal muscle function through a direct (peripheral) action. However, the presence of such impaired skeletal muscle function would be sensed by the central governor, which then responds appropriately. Thus, on the basis of this information, the central governor would either increase overall skeletal muscle recruitment in order to achieve the same skeletal muscle force output from a greater number of more "tired" muscle fibres, or it would slow the rate at which it usually reduces skeletal muscle recruitment during more prolonged exercise.[9] [17] However, provided that less than 100% of motor units are recruited, the central regulation of exercise performance predominates even if there is some evidence for peripheral skeletal muscle "tiredness"

Indeed an important prediction of the central governor model is that a central issue in exercise physiology becomes not fatigue as it is usually understood, but rather how the sensation of fatigue is expressed physically, through a change in pacing strategy, and how that pacing strategy is regulated in the first place.

Thus, according to the central governor model, all changes in pace, and even the termination of exercise,[2] [15] [21] [22] [28] occur as part of a regulatory strategy that is dynamic and continuously altering, and serves the teleological purpose of protecting the body from damage. This pacing strategy

What is already known on this topic

The popular model of exercise physiology predicts that exercise terminates because of a catastrophic failure of homoeostasis leading to skeletal muscle dysfunction, so called peripheral fatigue, usually as the result of either an inadequate oxygen supply to the exercising muscles or total energy depletion in the exhausted muscles.

What this study adds

This series of articles concludes that the human body functions as a complex system during exercise. Using feed forward control and in response to afferent feedback from multiple central and peripheral sensors, the brain paces the body during exercise specifically to ensure that the pre-planned activity is completed without any loss of cellular homoeostasis. Control is applied by changes in the extent of skeletal muscle recruitment during exercise and by the inhibitory effects of the increasingly disagreeable sensations of fatigue that are generated by the brain during exercise.

occurs at a subconscious level in an apparently oscillatory manner[17 36 37] whereas the ratings of perceived exertion, which in some way reflect the biological demands associated with the maintenance of homoeostasis, or even the proximity to the end point of exercise,[27] change more gradually with activity. These pacing strategies result from the CNS integration of feed forward information with afferent sensory feedback generated by metabolic and other changes in a number of peripheral organs.[36]

SIGNALLING PROPERTIES OF PERIPHERAL METABOLITES

The fifth contribution of the symposium is to propose that peripheral metabolites have signalling properties that assist in determining the continuously reset pacing strategy, and are not merely energy substrates or inert metabolic byproducts. As argued by Lambert et al,[36] the natural conclusion of each researcher examining any single metabolite is that this specific metabolite is the ultimate regulator of the complete system. But in the complex system of the central governor model, the pacing strategy is determined by the number of skeletal muscle motor units recruited by central efferent neural command consequent on neural calculations using afferent sensory inputs that detect current metabolic rate, fuel reserves, and rates of heat production among many other biological changes. Thus metabolites in skeletal muscle and elsewhere still play a manifestly important function in the new model, but they are neither the sole nor the absolute regulators of the entire complex system.

INTERACTION OF MULTIPLE PHYSIOLOGICAL PROCESSES

The sixth contribution of the symposium is therefore the suggestion that the central governor model is an example of a complex dynamic system in which multiple physiological processes in many different systems interact with each other continuously. As a result, changes in any physiological variable results from alterations in neural command or peripheral regulatory systems in response to prior system perturbation. The continuous interaction between feed forward and feedback control mechanisms in the brain and peripheral physiological systems produces a robust, self sustaining mechanism that maintains homoeostasis by ensuring that no system is ever overwhelmed or used to absolute maximal capacity. Although this control system may function perfectly in most healthy humans, the observation that some die suddenly during exercise, often from cardiac disease, indicates that the central governor is unable to predict and hence prevent every possible unfavourable outcome during exercise.

CONCLUSIONS

In summary, the symposium was designed to extend and advance the challenging ideas generated by a series of our earlier publications.[8 18 26 31 32] New ideas that have been presented here and which should stimulate further debate include the following.

(1) There is always skeletal muscle motor unit recruitment reserve in humans with an intact central nervous system who exercise voluntarily to exhaustion.[2 21 26 28 38 39] The presence of skeletal muscle recruitment reserve at exhaustion is incompatible with peripheral, linear, ''limitations'', and ''catastrophe'' models of fatigue.[17]

(2) The peripheral, linear, ''limitations'' models predict that a catastrophic system failure must precede the development of fatigue because it is the metabolic byproducts of that failure that impair skeletal muscle contractile function. However, there is little if any evidence for failure of any of the major organ systems at exhaustion in any form of voluntary exercise.[9] Rather, the more obvious conclusion is that exercise always terminates while homoeostasis is maintained. Hence the prediction of our new model is that the principal function of the central governor is to ensure that exercise terminates while homoeostasis is maintained and organ damage is prevented, an idea first mooted in 1997.[31]

(3) For the first time, the concept of linear and complex dynamic models has been applied to the study of exercise physiology. The complex model predicts that (a) pacing is a natural phenomenon of life, (b) fatigue is never absolute, and (c) exercise intensity and the activity of different metabolic systems oscillate continuously as a result of multiple interactions between all the organs that contribute to this complex system.

(4) Fatigue is a sensation that results from the conscious perception and interpretation of subconscious regulatory processes in the brain, and is therefore not the expression of a physical event. Accordingly, the study of the physical event of muscle fatigue in an isolated system, devoid of CNS control, is not likely to advance the understanding of this phenomenon. Rather, an understanding of the oscillatory pacing strategies adopted during exercise and their regulation is more likely to produce rapid advances in our understanding of how the complex system of the central governor model functions during exercise.

The challenge to exercise physiologists therefore is to examine changes in pacing strategy from a complex system perspective, rather than concentrating on any single physiological system or metabolic process when researching the causes of ''fatigue''.

Perhaps it is now finally the time to replace the concept of catastrophe with that of complexity in our future studies of human exercise physiology.

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