



The anticipatory regulation of performance: the physiological basis for pacing strategies and the development of a perception-based model for exercise performance

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ABSTRACT

During self-paced exercise, the exercise work rate is regulated by the brain based on the integration of numerous signals from various physiological systems. It has been proposed that the brain regulates the degree of muscle activation and thus exercise intensity specifically to prevent harmful physiological disturbances. It is presently proposed how the rating of perceived exertion (RPE) is generated as a result of the numerous afferent signals during exercise and serves as a mediator of any subsequent alterations in skeletal muscle activation levels and exercise intensity. A conceptual model for how the RPE mediates feedforward, anticipatory regulation of exercise performance is proposed, and this model is applied to previously described research studies of exercise in various conditions, including heat, hypoxia and reduced energy substrate availability. Finally, the application of this model to recent novel studies that altered pacing strategies and performance is described utilising an RPE clamp design, central nervous system drugs and the provision of inaccurate duration or distance feedback to exercising athletes.

We have previously described studies of pacing strategy using various experimental interventions, including faster and slower starts, higher temperatures, hypoxia, hyperoxia and altered energy substrate availability.¹ We have proposed the presence of a complex, regulatory system that mediates changes to skeletal muscle motor unit activation and work rate in order both to optimise performance and prevent potentially harmful changes to homeostasis. We subsequently develop a model to explain how this regulatory system might utilise the subjective rating of perceived exertion (RPE) as a means of integrating afferent information and a variety of other cues in order to achieve these objectives during exercise.

THE RPE DURING CONSTANT WORKLOAD EXERCISE: THE LIMIT VARIABLE FOR VOLITIONAL PERFORMANCE TO FATIGUE

Often measured as an index of subjective perception of effort during exercise, the RPE has been causally linked to physiological variables such as muscular force,² heart rate, ventilation, respiratory rate, oxygen uptake and blood lactate concentrations.³ Borg⁴ has stated that the RPE is the “single best indicator of physical strain”, and “integrates various information, including the many signals elicited from the peripheral working muscles and

from the central cardiovascular and respiratory functions, and from the central nervous system”. The RPE also incorporates other mediators, most notably psychological and affective components.^{5,6} The overall sensation of exertion measured during exercise is thus the conscious/verbal manifestation of the integration of these psychological and physiological cues (see Hampson *et al*⁷ for a complete review).

The biological link between the subjective sensation of effort and the physiological changes occurring during exercise is of crucial importance, because it provides a mechanism by which the RPE could, in theory, contribute to the regulation of exercise performance, the limit to exercise and pacing strategy. As described previously,¹ it has been found that an increase in RPE is linearly correlated with increases in core body temperature during exercise in hot and cool conditions, and to the increase in the α -to- β ratio suggesting reduced arousal levels.⁸⁻¹⁰ If exercise at a fixed work rate in the heat is limited due to a failure of central drive, as has been proposed,^{9,11} then the finding that arousal levels decrease progressively while RPE increases in direct proportion to body temperature invites the suggestion that if exercise was self-paced, any reduction in arousal (or increase in RPE) caused by small, incremental physiological changes, could exert effects by enforcing a reduction in the self-selected work rate.

Noakes¹² recently argued from the data of Baldwin *et al*¹³ that when exercise was performed at a constant work rate (thus negating any effect of pacing strategy) to volitional fatigue in either a glycogen-loaded or glycogen-depleted state, performance time was determined by the rate at which the RPE rose from its starting value to a maximal tolerable value. Significantly, the starting and final RPE values were similar between conditions, and the rate of increase in RPE in the glycogen-depleted state was significantly greater than in the loaded state. However, when expressed relative to the total trial duration, the RPE increased linearly and at the same rate in both conditions.¹²

This finding has two important implications. First, it reinforces the assertion that the RPE is influenced by afferent physiological feedback, as it increased more rapidly in the glycogen-depleted state. Noakes¹² suggested that the rate of increase in RPE reflected “the rate at which fuel reserves were being depleted”, although it may be more appropriate to speculate that the RPE at any stage

during exercise was dependent on the muscle glycogen content remaining at that time, and was thus higher when glycogen levels were lower. Studies of exercise performance in the heat have also found that the RPE is linearly correlated with body temperature,^{8,9} and maximal values of RPE occur at the same time as the core temperature reaches approximately 40°C,^{8,9} irrespective of the rate at which it increases. Also, interventions such as high fat diets^{14,15} and hypoxia¹⁶ result in increased effort perception and reduced time to volitional fatigue when the exercise work rate is fixed.

The volitional limit to exercise at a constant work load is thus the attainment of a subjective perception of effort, or RPE, that the exercising athlete considers to be “maximally tolerable”. That this coincides with many of the proposed physiological limits to exercise invites the possibility that the RPE is a key component of a system that exists to protect the athlete from continuing exercise for long enough to cause potential bodily harm (for example, exercising into lethal levels of hyperthermia).

Second, the observation that the RPE increased linearly but at different rates in both the glycogen-depleted and glycogen-loaded states¹² suggests that from the onset of exercise, the conscious perception of effort increases at a rate that will ensure that a maximal tolerable RPE is attained before the complete and limiting depletion of muscle glycogen stores. If exercise was ultimately limited by the depletion of energy substrates, with no anticipatory component, then it would be expected that the RPE would increase rapidly only at the end of exercise when the muscle glycogen stores were approaching critically low levels. However, this was not the case, and instead, the RPE rose at a constant rate throughout exercise in each condition.

Therefore, the rate of RPE increase, and thus the duration of exercise, may have been set from the very beginning of the trial in order to ensure that the maximal RPE occurs before any potentially harmful level of muscle glycogen depletion can occur. Afferent feedback would inform the brain of the availability of muscle glycogen, and the rate of increase in RPE would be set in an “anticipatory fashion” to ensure that the maximal RPE occurs before glycogen levels are depleted. For this reason, the RPE increases more rapidly from the onset of exercise in the glycogen-depleted state. Based on these findings, it is suggested that the RPE is not merely the direct result of afferent feedback from peripheral systems, as proposed previously,^{17,18} but plays a role in the anticipatory regulation of exercise, even when the work rate is fixed.

THE RPE DURING SELF-PACED EXERCISE: THE REGULATOR OF PERFORMANCE AND PHYSIOLOGICAL SYSTEMS

Critically, these studies^{8-10,12,13} have all examined exercise at a fixed work rate to fatigue. Few studies have examined whether the same phenomenon would occur when exercise is self-paced. It seems reasonable to suggest that rather than simply maintaining a high work rate until the RPE does rise to reach near-maximal levels, the exercising athlete would instead reduce the work rate under conditions of elevated body temperature⁸⁻¹⁰ or depleted muscle glycogen concentrations,¹⁵ giving rise to the possibility that the regulation of self-paced exercise may utilise the RPE as an important mediator of pacing strategy. As pacing strategies exist to optimise and regulate physiological changes during exercise, the RPE may thus be a key component of a regulatory system that protects against bodily harm.

To examine this possibility, Tucker *et al*¹⁹ utilised a novel “RPE clamp” model for exercise testing. In this model, the exercise work rate is free to vary, but the exercising athlete is instructed to maintain the conscious sensation of exertion at a

fixed, predetermined level. Tucker *et al*¹⁹ utilised the Borg 6 to 20 scale, and had cyclists perform RPE clamp trials at an RPE corresponding to a value of “16” on this scale. Trials were performed in three conditions: cool (15°C), normal (25°C) and hot (35°C). The novel aspect of this model is that any changes in the power output must be achieved specifically to ensure that the athlete’s RPE does not deviate from the desired value of 16. Therefore, the influence of physiological perturbations, induced by the external environment (heat, in this case), on RPE and thus exercise performance can be examined.

Using this model, it was found that during the first few minutes of exercise in the hot condition, the selected power output began to decrease more rapidly than in the cool and normal conditions. Therefore, in order to maintain the RPE at the desired level, subjects had to reduce their exercise intensity in the heat, but not in cool or normal conditions. Presumably, failure to do so would have resulted in an increase in RPE. Significantly, as with previous research in the heat,^{20,21} this altered pacing strategy occurred even though the core temperatures and heart rates were not different between the three conditions. In fact, rectal temperatures were below 38°C at the point at which the power output in the hot condition began to decrease significantly relative to the other two conditions. Instead, the only measured difference between the conditions was that the rate of heat storage in the hot condition was significantly greater than in cool or normal conditions, by virtue of the fact that the skin temperature rose significantly during the first few minutes of the trial.

The result of the significant decline in power output in the heat was that this rate of heat storage then decreased over the next 20 minutes of the trial. As a result, the heat storage in all three conditions was similar after 20 minutes of exercise, and remained this way until completion of the exercise trials. Subjects were stopped when their power output declined to 70% of its starting value, and so the time taken to complete the hot trial was significantly shorter than the trials in normal and cool conditions.

The conclusion from that study, the only such study performed using the RPE clamp model, is that exercise work rate in the heat is regulated differently based on the rate of heat storage during the first moments of exercise. In the hot condition, the exercise intensity was reduced in order to prevent the RPE from increasing above the predetermined target level, and this reduction achieved the physiological effect of reducing the rate of heat storage. As the rate of heat storage determines the rise in body temperature, the critical hyperthermia that is suggested to limit exercise was never attained. It was therefore concluded that the rating of heat storage mediates an anticipatory reduction in exercise intensity, and that this is mediated through the RPE, or the conscious sensation of exertion. Further research is required to apply this model to other interventions, including altered oxygen availability, fuel depletion and the possible use of drugs to alter sensation of fatigue during exercise.

SUMMARY

The evidence presented here suggests that the RPE may play a more significant role than simply being the integrator of afferent information from various physiological systems. Instead, the RPE may be a crucial component of the regulatory protective system that: (1) Prevents the exercising athlete from continuing exercise and causing bodily harm, by being responsible for the decision volitionally to terminate the exercise bout. That is, exercise terminates when the RPE

reaches levels that are intolerably high or uncomfortable for the exercising athlete. This coincides, or immediately precedes, potentially harmful changes in physiological function, including muscle glycogen depletion and potentially lethal heat stroke. (2) Mediates anticipatory adjustments in exercise intensity that ensure that the known exercise bout is completed safely and in optimal performance time. In this system, the RPE integrates information from various physiological systems, and adjustments in work rate are the response to the conscious RPE.

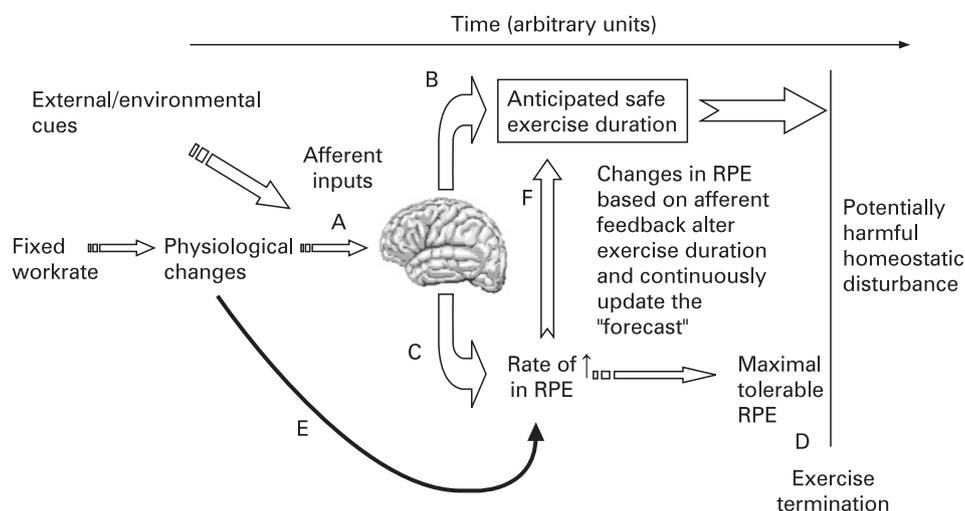
The precise mechanism by which the latter regulation of exercise performance is achieved is not known. However, based on various studies in which RPE, physiological function and performance have all been measured, we propose a model for how the conscious perception of effort regulates exercise performance under all conditions to protect the athlete and ensure optimal performance. This model is termed the “anticipatory feedback” model, because it utilises previous experience, anticipation of exercise duration/distance, and physiological feedback to regulate pacing strategy and performance. It extends the model of complex regulation of performance. It includes novel explanations for previously described performance findings, and invites research studies to elucidate further and develop the understanding of the complex regulation of exercise performance.

A PROPOSED MODEL FOR AN ANTICIPATORY SYSTEM REGULATING PERFORMANCE USING THE RATING OF PERCEIVED EXERTION

Exercise at a fixed work rate to fatigue

Figure 1 depicts a proposed model for the regulation of exercise performance to volitional exhaustion at a fixed work rate. This includes incremental exercise to exhaustion, as is typically used to measure the maximal oxygen uptake. At the onset of exercise, afferent information from various physiological systems and external/environmental cues (A) is used by the brain to forecast the duration of exercise that can be safely completed without causing harmful homeostatic derangements (B). The afferent feedback from physiological systems depends on the exercise intensity and environmental conditions, including factors such as temperature and the partial pressure of oxygen of the inspired air. Simultaneously, the initial rate of increase in RPE is set as a consequence of a subconscious anticipatory calculation of the safe exercise duration (C). The initial “setting” of exercise duration and the rate of increase in RPE represent the anticipatory component of the model.

Figure 1 Schematic diagram showing the proposed model for the anticipatory regulation of exercise performance during exercise to fatigue at a fixed work rate. RPE, rating of perceived exertion.



Because exercise terminates when the maximal tolerable RPE is attained (D), the time to exhaustion is determined by the rate of increase in RPE, which is continuously modified based on the regular integration of afferent feedback signals from numerous physiological systems, including those described previously (E). The “safe” exercise duration is thus determined by a combination of anticipatory forecasting and afferent feedback as a result of the physiological changes occurring during exercise. The maximal tolerable RPE (D) occurs before harmful changes to homeostasis can occur. Such changes include, for example, the attainment of a critically high core temperature. The high core temperature thus acts as an “off-switch”, mediated, importantly, by the RPE.

The novel aspect of this model is that physiological changes do not directly limit the ability of the athlete to perform exercise (a catastrophic model),^{22, 23} nor are they the singular cause for the generation of the RPE. Rather, afferent feedback, expected exercise duration and physiological changes during exercise are used by a central controller to forecast future physiological changes. Based on this feedback, an RPE is generated and then increased at a rate that ultimately determines the safe exercise duration. This model can be applied to the studies described in this review, which showed the influence of high temperatures^{8–11} and low muscle glycogen concentrations^{12, 13} on exercise performance to fatigue at a fixed work rate.

Model explaining performance during self-paced exercise time trials

The previous model (fig 1) is only applicable to exercise at a fixed work rate, in which the central control processes cannot alter the pacing strategy in response to experimental perturbations, but can only influence the duration of exercise. If the exercise work rate is free to vary, then experimental interventions such as changes in ambient temperature, oxygen content of the inspired air, energy substrate availability or the provision of incorrect distance feedback all alter the power output (pacing strategy), whereas the rate of increase in RPE has been found to be similar between conditions.^{13, 20, 21, 24–26}

In fig 1, the RPE is set by the brain at the onset of exercise, and the time to fatigue is determined by the rate at which the RPE rises to reach a maximal tolerable value. In contrast, self-paced exercise with a known duration would allow the work rate to be altered in response to a high or subjectively unsustainable RPE. It seems reasonable to suggest that rather

than simply maintaining a high self-selected work rate until the RPE rises to reach near-maximal levels, the exercising athlete would instead reduce the work rate in certain conditions to prevent hyperthermia or substrate depletion from occurring before the end of exercise. This would have the effect of regulating the RPE, so that it would not differ from control trials in which the exercise work rate may be maintained at higher levels.

Accordingly, the fixed work rate model in fig 1 is extended to propose that during self-paced exercise, the brain integrates afferent information from various physiological systems (described previously) to generate a conscious RPE, and then regulates the work rate to ensure that this conscious RPE does not increase excessively at any stage during exercise, because this would lead to premature exercise termination.¹²⁻¹⁹ Importantly, in order to prevent the conscious RPE from exceeding acceptable levels, there must be some expected or "acceptable" RPE at any stage during exercise, against which the conscious RPE can be continuously compared. This is termed the "template RPE" and it is proposed that it is generated as a result of previous experience and knowledge of the upcoming exercise duration.²⁷ It is important to note that this "template RPE" is a theoretical construct that cannot be measured, but, as proposed below, is necessary in order for the conscious RPE to be interpreted. In contrast, the conscious RPE is a verbalised rating, usually on the Borg scale,⁴ which is measured during exercise.

According to this model, shown in fig 2, the regulation of exercise work rate during self-paced exercise is achieved by means of a combination of feedback integration (which generates the conscious RPE) and anticipatory forecasting (which produces a "template" for the RPE against which the conscious RPE is compared).

The anticipatory component requires previous experience and training, as well as afferent input from the environment, which may include physiological inputs such as reduced muscle

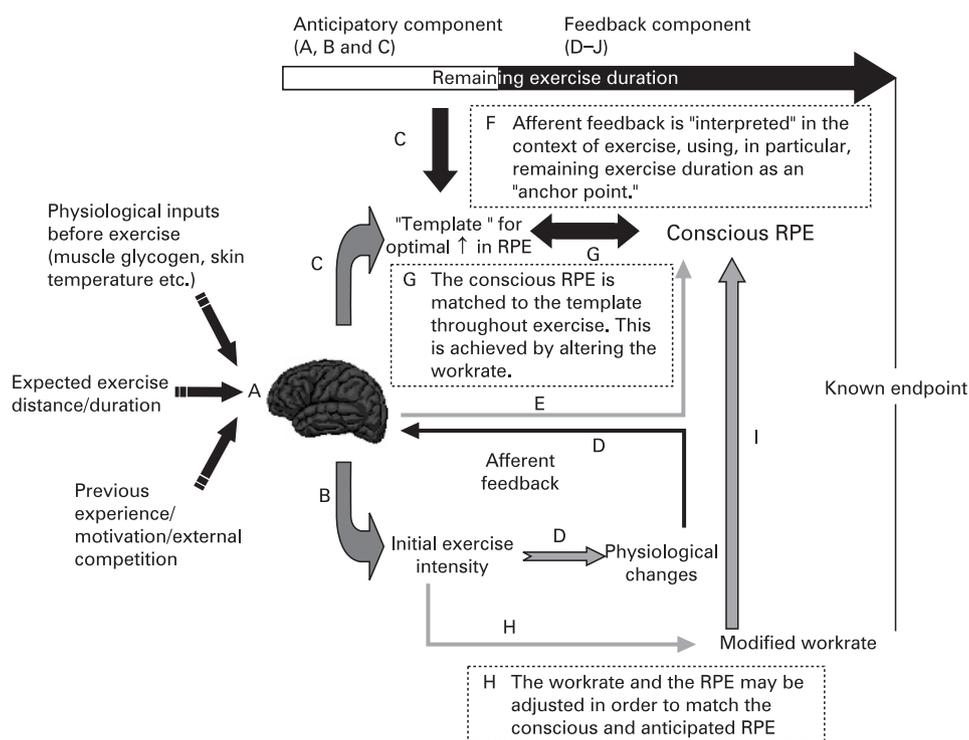
glycogen levels or increased skin temperature, as well as psychological inputs such as motivation levels, arousal and the presence of competitors (A in fig 2). In addition, at the onset of self-paced exercise, the exercising athlete is usually aware of the distance or duration of the upcoming exercise bout. Based on these factors, the exercising athlete self-selects an initial exercise intensity, which is anticipated to be optimal for the expected duration of exercise (B).

At the same time, a "template" or expectation of the desired rate of increase in the RPE during the exercise bout exists (C). That is, optimal performance requires an increase in the RPE, which ensures that a maximal tolerable RPE is reached at the moment exercise is completed, but not before. If the maximal RPE occurred before the endpoint, performance would be impaired by a reduction in the work rate or premature fatigue.¹² The "template RPE" provides a means by which the conscious RPE can be interpreted—it is a construct that represents the exercising athlete's expectation of effort perception during the exercise bout, and is based on memory or previous experience, which is updated continuously throughout exercise based on the remaining exercise duration (C).

As soon as exercise commences, changes in physiological variables, including body temperature, skin temperature, rates of heat storage, metabolite concentrations, arterial saturation levels, increased ventilatory rates and increased heart rates, and mechanical afferents from muscle generate afferent signals to the brain (D). These afferent signals are "interpreted" by the brain in the context of the exercise bout and result in the generation of a conscious RPE (E). The duration of exercise remaining is one of the key "anchors" against which the afferent feedback is interpreted (F). The conscious RPE is thus a consequence of both the duration of exercise that remains, as well as the exercise intensity, because the degree of afferent feedback is a function of the exercise work rate (D and F).

The key aspect of the present model is that the conscious RPE is then "matched" with the template RPE (or previous

Figure 2 Schematic diagram showing the proposed model for the anticipatory regulation of exercise performance during self-paced exercise. Black shading denotes input to the brain, grey shading denotes output or efferent processes. RPE, rating of perceived exertion.



experience) throughout exercise (G on fig 2). It is important to reinforce that this “template RPE” represents an RPE that the exercising athlete considers acceptable for any stage during the bout. Effectively, the athlete regulates the RPE by means of alterations in the exercise work rate to ensure that the conscious perception of effort is acceptable to them. As the RPE is strongly influenced by physiological changes, the regulation of RPE also achieves the regulation of the physiological perturbations occurring during exercise.

Adjustments to the work rate (H) would be achieved by alterations in the degree of skeletal muscle recruitment, as is supported by studies that measure electromyographical activity, a measure of skeletal muscle activation.^{21 25 28–30} Ultimately, this regulatory system will prevent harmful or limiting physiological changes from occurring before the end of exercise, while still optimising performance. The conscious RPE is an ideal candidate to perform a regulatory role, because it is a consequence of the total physiological changes occurring during exercise,¹⁸ is specific to the conditions under which exercise is being performed,⁷ and is potentially the only link between the physiological changes occurring during exercise and the athlete’s conscious awareness of these changes.

If, for example, the initial exercise work rate is excessively high, it will produce physiological changes that result in the conscious RPE being greater than that which the central controller considers optimal for a particular stage during an exercise trial. The anticipatory feedback control system would result in a reduction in motor unit recruitment and work rate (H) until the conscious RPE returns to an “acceptable” level, as shown by I on fig 2. As a result of continuous dynamic interaction between the subconscious anticipatory component and the afferent feedback component (G), power output is continuously modified in order to regulate the increase in the RPE.

Because the RPE is influenced by physiological changes,⁷ the matching of RPE to an anticipatory calculation successfully prevents catastrophic derangements to homeostasis. Therefore, only power output is ever observed to change during self-paced exercise, whereas RPE is similar in various experimental interventions. The pacing strategy is therefore a measure of how this regulatory control process allocates physiological resources and prevents the sensation of fatigue from rising excessively during exercise.

Testing the model using the RPE clamp protocol

This model (fig 2) can be evaluated using the novel RPE clamp method, described previously in the current review.¹⁹ Clamping the RPE at a constant predetermined level effectively replaces the generation of a template for an “acceptable” rise in RPE with an externally imposed “template” or expectation on the athlete (C on fig 2). The athlete must thus regulate their work rate and conscious RPE so that it remains at the predetermined (and thus expected) level (G on fig 2).

This allows two potential predictions of the anticipatory RPE model to be evaluated. First, it allows the anticipatory or forecasting component to be examined. If the initial work rate and RPE are set in a feedforward or anticipatory manner as is suggested (B and C on fig 2), then the starting power output should be similar whenever exercise is performed at a given RPE, irrespective of experimental interventions, such as increased ambient temperatures, hypoxia and dietary interventions. In contrast, if the initial work rate and RPE are the result of afferent feedback, then differences in work rate would be expected at the onset of exercise, as soon as differences between conditions are detected.

Second, the RPE clamp method allows the effects of afferent feedback on the regulation of exercise work rate to be studied. The model proposes that afferent feedback is “interpreted” by the brain (D, fig 2) and also directly influences the conscious RPE, resulting in continuous adjustments to the power output in order to return the conscious RPE to an anticipated optimal level. If the RPE is “clamped”, then power output would be reduced to prevent the conscious RPE from increasing above this predetermined level. Therefore, the hypothesis is that any intervention that results in differences in one or more physiological systems will alter the nature of the afferent feedback to the central controller and will thus result in greater changes in power output than during a control trial.

The findings of Tucker *et al*¹⁹ in applying the RPE clamp to exercise in three different environmental conditions support both these suggestions. First, it was found that the power output at the immediate onset of exercise was similar between the three conditions, suggesting that the initial selection of work rate is set in a feedforward manner (fig 2), based perhaps on previous experience and expectations of exercise, rather than an instantaneous afferent input from an elevated skin temperature in the heat.¹⁹

Furthermore, the decrease in power output over the first 10 minutes of the trial was also similar between conditions, again supporting the notion that the initial work rate is set in an anticipatory manner. However, between 10 and 30 minutes, power output decreased more rapidly in the hot (35°C) condition, resulting in a reduced trial duration compared with the other two conditions.¹⁹ Over the course of the trial, power output declined linearly in all conditions, although at different rates, which suggests that the RPE is not simply a marker of exercise intensity, because power output was falling at different rates in the different environmental conditions even though the RPE was fixed and identical between conditions, in agreement with our proposed model.

Second, the rate of heat storage was greatest in the hot condition only during the first 10 minutes of the trial. Thereafter, heat storage was similar between all three trials, despite a 20°C difference in ambient temperature between conditions. A similar rate of heat storage despite a decreased potential heat loss in the hot condition would be the result of a reduction in the rate of heat production, which can be achieved by reducing the work rate. As described, this was the case in this study,¹⁹ because the power output from 10 to 30 minutes fell more rapidly in the hot than in the temperate and cool conditions. This finding therefore supports the feedback component of the proposed model (fig 2), which posits that afferent feedback causes an increase in the conscious RPE, which, in this exercise study, forces a reduction in work rate in order to maintain the RPE at the predetermined level.

Finally, it is notable that the rapid reduction in power output in the hot condition occurred even though body temperatures were similar between the three conditions.¹⁹ This suggests that anticipatory control is present even when afferent feedback has begun to adjust the work rate, as described. There is thus no distinct transition between anticipatory and feedback regulation, but rather a continuous integration of these two components. This has been described in previous studies of self-paced exercise in the heat.²⁰

FURTHER APPLICATIONS OF THE ANTICIPATORY RPE FEEDBACK MODEL

The endspurt phenomenon

The proposed model is also able to account for the characteristically observed “endspurt” phenomenon, in which power

output or running speed increase significantly during the final stages of self-paced trials.^{20 24 26 31 32} Our model posits that changes in work rate during exercise are the result of the continuous interpretation of afferent feedback during exercise combined with an anticipatory calculation that identifies the optimal rate of increase in RPE and the initial exercise intensity. The afferent feedback is interpreted in the context of the exercise bout and influences the RPE accordingly. In particular, the distance or duration of exercise that remains is a crucial “anchor” against which the RPE is interpreted (F on fig 3).

Throughout exercise, however, there is a degree of “uncertainty”, because it can never be precisely known when the exercise bout will be completed, or what physiological demands may be incurred after an exercise bout has been completed. The interpretation of the afferent feedback and resultant regulation of work rate are therefore subject to a level of uncertainty that is greatest when exercise duration is great, and becomes progressively smaller as the known endpoint approaches. As the role of the regulatory processes is to ensure that catastrophic derangements to homeostasis do not occur, this uncertainty results in the maintenance of a motor unit and metabolic reserve throughout exercise. As the exercising athlete approaches the known endpoint, the uncertainty is reduced, and so the motor unit and metabolic reserve need not be maintained any longer, resulting in significant increases in motor unit recruitment and power output, in the characteristically observed “endspurt”.

The effects of central nervous system drugs on performance

Our model also explains the performance-enhancing effect of dopamine/noradrenaline reuptake inhibitors on exercise performance in hot conditions, as shown by Watson *et al*,³³ as well as the reported effects of amphetamines on endurance performance. These drugs are known to increase arousal and motivation and reduce the sensation of fatigue.³³ This would act at two potential sites in the proposed model, as shown in fig 3. First, the initial anticipatory setting of work rate would be altered, because arousal and motivational levels would be elevated (A), resulting in an increase in the initial power output

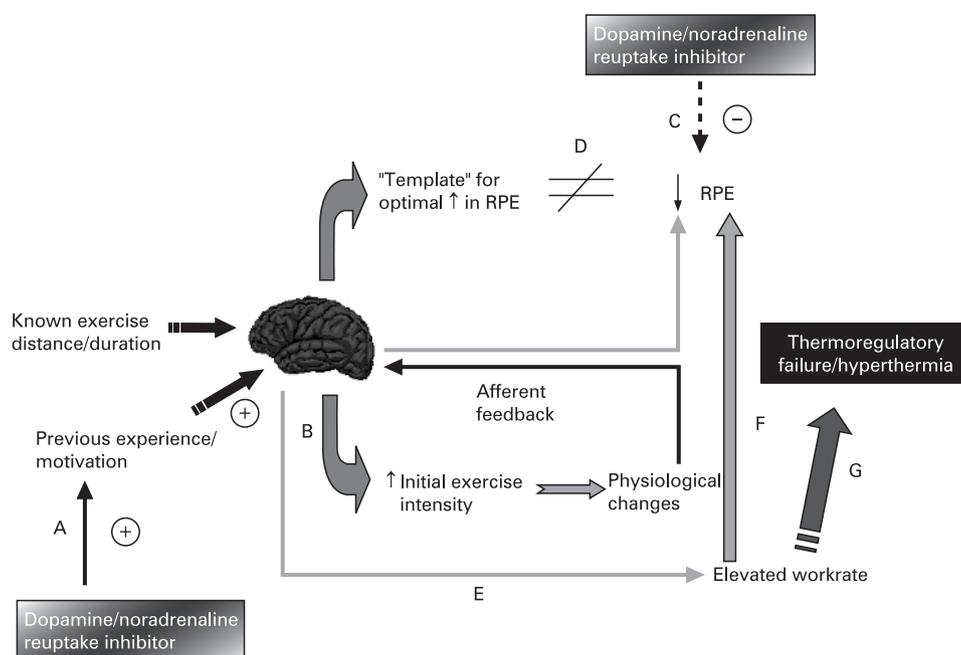
(B). Second, the conscious RPE at a given work rate would be reduced (C), resulting in a mismatch between the actual conscious RPE and the “template RPE”, which is based on previous experience (D). The consequence of this mismatch would be an increase in the work rate (E), which will elevate the conscious RPE (F) until it returns to the anticipated levels for that exercise bout. At the same time, the elevated work rate will increase heat production, causing thermoregulatory failure or hyperthermia (G).

As the purpose of this control system is to regulate exercise performance to prevent potentially harmful changes in homeostasis, a further prediction of the model would be that any improvements in performance that result from the use of these drugs will also cause a failure to regulate physiological variables compared with when exercise is performed without such drugs. Indeed, Watson *et al*³³ found a significant increase in power output during time trials in hot conditions (30°C) when the reuptake inhibitor was administered, and this was associated with a significantly elevated rectal temperature. Seven out of the nine subjects were in fact able to exceed what is usually considered the critical core temperature of 40°C without volitional fatigue, which represents an overriding of the usual control process occurring in the heat.³⁴ This observation can be accounted for by the model (G on fig 3). A similar phenomenon may explain the effects of amphetamines on exercise performance,¹² and may account for the observed effect of hypnosis on performance.

Application to studies in which misinformation is provided

Finally, the present model is also able to account for the findings of studies in which athletes are provided with incorrect or misleading feedback regarding exercise duration.^{25 35–37} Those studies, which were described earlier, are particularly interesting because they allow the anticipatory components of the model to be evaluated (A, B and C in fig 2), because they alter the expected exercise distance or duration (A, fig 2). As proposed, the anticipatory setting of the initial work rate and the generation of a template for the increase in RPE are based upon the expectation of exercise duration (B and C in fig 2).

Figure 3 Schematic diagram depicting how the performance-enhancing effects of dopamine/noradrenaline reuptake inhibitors can be explained according to the proposed model. Black shading denotes input to the brain, grey shading denotes output from the brain. (+) denotes stimulation, (–) denotes inhibition. RPE, rating of perceived exertion.



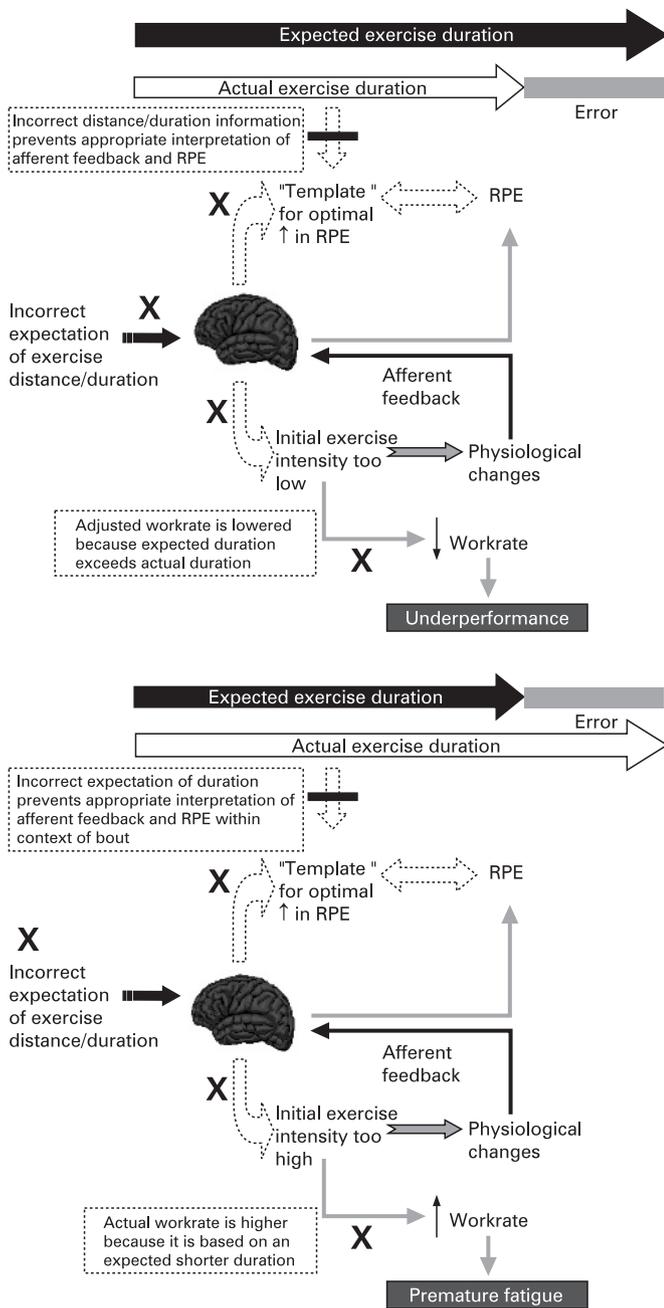


Figure 4 Schematic diagram showing how the anticipatory component of the proposed model would be influenced when athletes are incorrectly informed of exercise duration before the exercise bout commences. The top panel depicts the proposed model when the actual duration is shorter than the athlete is informed. The bottom panel depicts the proposed model when the actual duration exceeds the expected duration. Both situations result in relative underperformances (see text for details). Black shading denotes input to the brain, grey shading denotes output from the brain. Dashed white arrows depict processes that have been removed/ablated by the provision of incorrect information. RPE, rating of perceived exertion.

In addition, the continuous updating of this RPE “template” and the interpretation of afferent feedback depends on knowledge of the exercise duration that remains²⁷ (C and F, fig 2), and so incorrect expectation of distance remaining prevents the appropriate interpretation of physiological afferents. Therefore, an intervention that deceives the subject with regards to exercise duration creates numerous errors in the anticipatory

calculations and the interpretation of afferent feedback, which would alter the model as proposed in figs 4 and 5, with the errors in the anticipatory calculations denoted by crosses.

Actual duration is shorter than the expected/informed duration

If the exercising athlete is incorrectly informed about the distance of exercise before they begin, the overall effect will be to alter the initial work rate and the “template RPE”, because both are based on the expectation of exercise duration, as shown in fig 4. That is, the athlete’s conscious RPE must be compared with some expected RPE, but if the duration has been incorrectly “set”, this comparison will be incorrect. If no further distance feedback is provided during exercise, then there are two possible outcomes: First, if the actual length of exercise is shorter than what the athlete was informed, an overall underperformance will occur (fig 4, top panel). This occurs because the anticipatory setting of work rate, the optimal rate of increase in RPE and the interpretation of afferent feedback will have been based on the expectation of a longer exercise bout than is really the case. This will result in the relative underutilisation of physiological resources and an excessively large reserve will be maintained during exercise (fig 4, top panel).

Actual duration exceeds the expected/informed duration

The second possible outcome arises if the actual duration exceeds that which the athlete had been told (fig 4, bottom panel). In this case, the anticipatory setting of the initial work rate, the generation of the template for the RPE and the interpretation of feedback will have been made with the expectation of a shorter exercise bout. This will result in the maintenance of relatively greater work rates for the first part of the exercise bout in order to match the conscious RPE with the “template RPE”, until the actual duration exceeds the expected

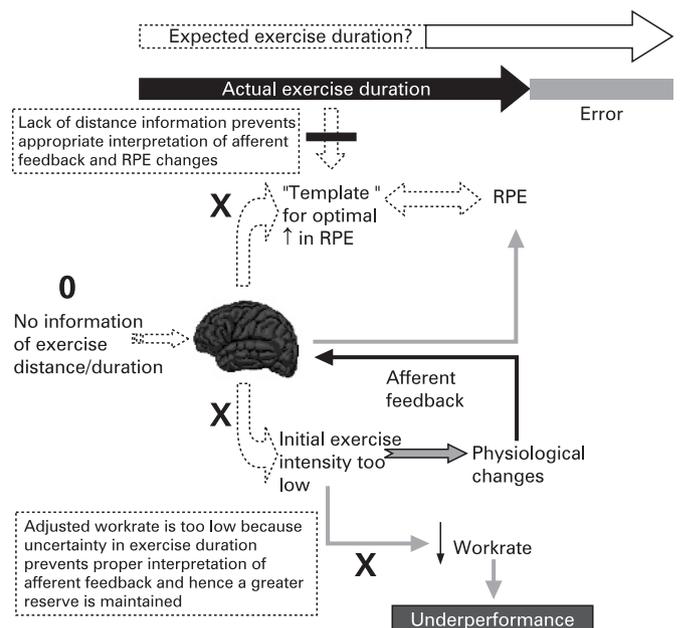


Figure 5 Schematic diagram showing how exercise performance would be impaired if no information regarding the exercise duration was provided before the commencement of exercise. Black shading denotes input to the brain, grey shading denotes output from the brain, dashed white lines depict processes that have been removed and are absent due to the lack of information. RPE, rating of perceived exertion.

duration. Thereafter, excessive afferent feedback as a result of the high initial work rates will cause premature fatigue, a rapid reduction in power output and a relative underperformance compared with what could be achieved when duration is correctly known (fig 4, bottom panel).

In support of this proposal, Ansley *et al*³⁵ found that when cyclists performed 36 s of maximal cycling after being informed that they would be cycling for only 30 s, their power output in the final 6 s was significantly lower than when they were correctly informed of the exercise duration. According to the present model, this occurs because the subjects interpreted afferent information feedback incorrectly, based on the expected duration of 30 s, and so physiological resources were incorrectly allocated from the onset of exercise. Similarly, when subjects ran on a treadmill at a fixed velocity and were deceived about the length of exercise and were instructed to run for longer than anticipated, their RPE and affect scores increased suddenly.³⁷ As described, this occurs when the exercise work rate is fixed. When it is free to vary, then the power output or running speed would be reduced.

Another study design that allows distance or duration manipulation is to inform the athlete correctly of the exercise duration before the trial, but to provide inaccurate distance feedback during the trial. Albertus *et al*²⁶ performed a study in which trained cyclists performed 20 km time trials with either correct or incorrect distance feedback every kilometre. It was found that overall performance, pacing strategies and the subjective ratings of perceived exertion were not different at any stage between trials, despite the provision of inaccurate distance splits. This suggests that the anticipatory setting of the initial work rate and the subconscious calculation of the optimal rate of increase in RPE, both of which are based on previous experience and knowledge of exercise duration (A, B and C, fig 2), are particularly important.²⁷ It also suggests that the interpretation of afferent feedback in the context of the distance remaining may have a less important role in regulating the pacing strategy under the conditions evaluated in that study, because subjects did not alter their power output in response to incorrect distance splits.²⁶

It was speculated that the exercising athlete did not take the incorrect distance information into account, but based their pacing strategy on previous experience, the initial knowledge of exercise duration and on physiological afferent inputs throughout the exercise bout (D in fig 2). This strongly supports the model that the work rate during self-paced exercise is the result, at least partly, of anticipatory calculations. As there was no experimental intervention other than the provision of incorrect distance information, the interpretation of the afferent feedback may have played less of a role. If trials are conducted in the heat, or with reduced oxygen content, for example, then continuous knowledge of the distance remaining may become more important as an anchor point in the regulation of work rate.

No duration information is provided

Finally, the model predicts that if the subject is not informed of the exercise duration before exercise begins, the initial work rate will be reduced compared with the normal situation, also resulting in an underperformance (fig 4), similar to that which would be observed when the actual duration is shorter than what is expected (fig 4, top panel, discussed previously). This occurs because of the high level of uncertainty when the endpoint is not accurately known. Ignorance of exercise duration negates the role of previous experience and training, and once the exercise bout has commenced, it prevents the

correct “interpretation” of afferent feedback. That is, the “template RPE” cannot be set and nor can afferent feedback be interpreted in the absence of an anchor point provided by knowledge of the endpoint of exercise. In the absence of this “template”, the conscious RPE cannot be interpreted, and so the overall effect will be to reduce the work rate compared with a control trial in which the athlete is aware of the duration.

Furthermore, if the athlete is informed that the end of exercise is approaching, it is expected that the work rate will increase significantly, much more than would usually be the case in an “endspurt”. This will occur because the uncertainty will have been instantly reduced from high levels to almost zero and the reserve that was maintained can then be instantly accessed to produce high work rates.

Limitations of the proposed models and measurement of physiological changes

A limitation of previous research is the use of surface electromyography techniques to measure the extent of skeletal muscle activation. As described previously, surface electromyography provides an indirect measure of skeletal muscle activation, not muscle recruitment, and numerous factors can influence the electromyography–force output relationship.^{38–39} For example, it has been argued that peripheral changes in the muscle cause the motor neuron firing rate to decrease,^{40–42} causing slower contraction and relaxation times,^{39–43} producing a reduction in electromyography that is not associated with a reduced activation level of skeletal muscle, and which is not measured using surface electromyography techniques. Also, only one active muscle was measured in three of the four studies using electromyography techniques. This may mean that other muscles are being activated or recruited differently in different conditions, but are not being detected.

At present, a number of promising techniques such as functional magnetic resonance imaging, multimodal imaging and transcranial magnetic stimulation are being investigated as methods to examine the central nervous system directly during exercise. However, at least for now, these are not feasible because of the large movement component during dynamic exercise, leaving only integrated electromyography as an indirect means to measure activation levels. As a result, it is not yet possible to conclude definitively that motor unit recruitment is altered in anticipation of physiological changes that may be harmful.

Another limitation concerns the measurement of the physiological afferents that are proposed to regulate work rate through this feedback system. For example, there is evidence that rectal temperature, which is often measured for body temperature, may show a delayed response to changes in internal and external temperature due to vascular perfusion of the rectum being lower than that of the deeper core. This may affect the ability to infer which thermoregulatory variables are being monitored in the presently proposed anticipatory regulation model, because it is the timing of the changes in work rate that is essential, and rectal temperature may not provide the necessary resolution of measurement.

Similarly, factors such as β -adrenergic activation, systemic and cerebral arterial desaturation, and the reduction in skeletal muscle adenosine triphosphate, adenosine 5'-diphosphate and total nicotinamide adenine dinucleotide, reduced form, concentrations are difficult to measure during dynamic exercise, making it difficult to establish which afferents are responsible for changes in performance when the oxygen content of the air is altered. As a result, the potential mediators of the proposed

regulatory system remain unelucidated, and future research is required to identify with certainty whether these physiological afferents are responsible for mediating the proposed anticipatory regulation.

CONCLUSION

This model incorporates anticipatory/feedforward as well as feedback components, using an expectation of exercise duration to set an initial work rate and to generate what has been termed a subconscious “template” for the rate of increase in the RPE. During exercise, afferent feedback from numerous physiological systems is responsible for the generation of the conscious RPE, which is continuously matched with the subconscious template by means of adjustments in power output. This subjective rating is biologically linked, allowing pacing strategy to be adjusted to prevent catastrophic changes in the monitored physiological variables (homeostats).

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