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Central Governor Model

A review of Professor Noake's Revolutionary Model of Performance

(Sometime in 2004)

Part 1

Introduction

The most enduring model of endurance physiology is the Cardiovascular/Anaerobic model. Initially suggested by British physiologists A.V. Hill and associates in the mid-1920s, this model has been promoted by scientists, coaches, and athletes world-wide for nearly 80 years. This model basically posits that a lack of oxygen to working muscles is what ultimately limits exercise performance. Most adherents to this model use the terms VO₂max, lactate threshold, and running economy when discussing training or physiology; terms which are used to describe particular aspects of this model. Though this model continues to be accepted today by most runners and coaches since the 1970s an increasingly large body of research has challenged the validity of the cardiovascular/anaerobic model. However, despite the increasing volume of evidence against it, the model has persisted as the primary model of endurance physiology.

I believe the main reason the cardiovascular/anaerobic model has persisted in the face of the large body of evidence to the contrary is that no other comprehensive model of endurance physiology has been proposed. So, while the deficiencies of the cardiovascular/anaerobic model are well known, the absence of another model to replace it has allowed continued support for the cardiovascular/anaerobic model.

Recently one of the primary opponents to the cardiovascular/anaerobic model, noted researcher and author Dr. Tim Noakes, proposed a new, revolutionary, comprehensive model of endurance performance that he has termed the Hill/Noakes Central Governor Model. The existence of a physiological governor was first suggested by A.V. Hill, the same physiologist credited with the cardiovascular/anaerobic model. However, Hill's idea of a physiological governor have been overlooked or ignored by those supporting the cardiovascular/anaerobic model attributed to him and hence, remained in obscurity for many years. During a review of Hill's original work Dr. Noakes re-discovered Hill's governor theory. Intrigued by the idea, Dr. Noakes reviewed existing research and conducted new research designed to test the validity of Hill's theory. The compelling results of his research combined with the previous unexplained results of prior research convinced Dr. Noakes of the accuracy of this model. He substantially updated the model and introduced it to the physiology world as the Hill/Noakes Central Governor

Model.

I believe that his new model may well be the missing ingredient that will finally cause the abandonment of the cardiovascular/anaerobic model. As Dr. Noakes said to me, it will become increasingly difficult to continue to support the cardiovascular/anaerobic model and that at a minimum his new model clearly delineates the battle lines such that people will have to decide which side of the line they care to take. If this is correct, then in the next few years we will see a large migration of scientists, coaches, and athletes to the Central Governor Model. Undoubtedly accompanying a change in belief in the underlying physiology will be changes in accepted training methods. How significant those changes may be remains to be seen. If Dr. Noakes' model is truly the heir apparent to the cardiovascular/anaerobic model I thought it would be appropriate to review his model, its training implications, and to compare and contrast it with my power running model of performance. We begin with fatigue.

Fatigue Defined

What causes muscular fatigue? Why during the final miles of a long run or race does it become increasingly difficult to maintain a set pace? Why can't runners maintain maximum speed for an entire 100 meter sprint? Why do high ambient temperatures affect performance so dramatically, especially in the later stages of a race? These and other examples are all evidence of fatigue, but they don't tell us what is causing the fatigue. Scientists have long sought the causes of fatigue. However, before we can fully discover what causes fatigue, we have to properly define fatigue.

The traditional definition of fatigue used by physiologists is an inability to either continue a pre-defined amount of work or equal a previous level of work, despite a strong desire and effort by the subject to do so. It is common for researchers to have subjects exercise at some set work load, say a pace that initially equals 80% of VO₂max, and when the subject can no longer maintain that pace they are said to have fatigued.

While that definition is as good as far as it goes, it doesn't go far enough. Even though a subject may not be able to maintain a set work load they can continue at a lesser work load, i.e. the pace slows but the subject continues. The point being that outside of death fatigue is not absolute. A subject is not either fatigued or not fatigued. Fatigue falls on a scale, with greater or lesser amounts. The subject can always continue, albeit at a slower pace. Saying a subject is fatigued because they can't maintain a pre-determined level of output is not incorrect, but it doesn't account for the fact that the subject could continue at a new, slower pace.

Where does fatigue occur?

Now that we have established that fatigue is not an absolute event and is instead a relative event -you could even call it a pacing strategy - we have to determine where fatigue occurs. Do the muscle fibers themselves become fatigued and not contract as quickly and/or powerfully or is fatigue occurring

elsewhere and then interfering with the muscle contraction? Perhaps it is occurring in multiple locations at the same time? These are very important questions to answer accurately in order to determine the cause or causes of fatigue.

Muscles contract because they receive a signal from the brain that causes them to contract. If they do not receive the signal they don't contract. The brain controls physical activity by the signals it sends to the muscles. If a higher workload is required, the brain alters its signal and activates more fibers. If a lesser workload is desired, the brain alters the signal and reduces the number of fibers activated. This is the basic process of muscle contraction.

If the muscle fibers are the singular point of fatigue during exercise then as the muscle fibers fatigue the brain, in order to maintain work rate, would need to activate an increasing number of muscle fibers, eventually activating 100% of the fibers in order to maintain the desired workload. At the point that 100% of fibers were fatigued, then the workload would necessarily decrease despite attempts by the brain to the contrary. The fatigued fibers simply would not contract as quickly or powerfully as before, resulting in a drop in power output and a slowing of the pace. So, if fatigue is a muscular phenomenon we should see an increasing mass of muscle fiber being activated as exercise continues, cumulating in 100% fiber activation at the end of exhausting exercise.

What would cause muscle fibers to fatigue? It could be many things including a weakening of the contractile proteins within the muscle fiber, lack of oxygen within working fibers, increased muscle fiber acidity, hypoglycemia, heat buildup within a fiber causing a decrease in contractility – all of which have been pointed to as a source of fatigue - or it could be any number of other things not yet identified.

A competing theory would be that fatigue occurs elsewhere in the body and interferes with the contractile function of the muscles. For example, if the central nervous system were fatiguing during exercise the signals it produces and sends to the muscles, commanding them to contract, could be weakened or delayed resulting in a de-recruitment of muscle fibers.

To test if the muscle fibers themselves are the root location of fatigue we would only need to measure muscle activity during exhausting exercise and determine if an increasing mass of muscle fibers are being activated. This is exactly what scientists have done. Scientists can measure muscle fiber activation and studies that have done so have established that an increasing volume of muscle mass is *not* being activated during exercise and that 100% of available muscle fibers are *not* activated at the end of exhaustive endurance exercise. Instead, the number of muscle fibers activated falls during exhaustive exercise. For example, a team of researchers examined power output and muscle fiber activation during a one hour cycling time trial (1). During the course of the test the researchers intersperse 6 maximal one minute sprints. Power output and muscle activation decreased steadily from sprints 2 – 5, despite the effort of the cyclists to perform to their maximum

ability. The drop in muscle activation and power of the subjects demonstrate that central drive to the muscles was decreasing, not increasing.

Interestingly, in contrast to an increasing drop in power and muscle activation in sprints 2-5 during the 6th sprint, which was conducted in the last minute of the time trial, power output and muscle activation increased significantly. (A last minute surge by competitors at the end of a race is a commonly observed occurrence in endurance competitions, especially cycling, hence the researchers placement of the 6th sprint.) If the muscles themselves had been fatigued the cyclists would have been unable to suddenly increase power output. So the evidence points away from muscle fatigue as the source of fatigue during exercise. This is not to say that muscles don't fatigue, only that decreases in work output that define fatigue is not driven directly by muscle fiber fatigue.

If fatigue is not found primarily with the muscle fiber itself, then what causes fatigue? While the data points away from muscle fiber fatigue as the singular source of fatigue it does point to the brain as the source of fatigue. The drop in muscle activation suggests that the central drive to the muscles has decreased. The sudden return of both power output and muscle activation during the 6th and final sprint is evidence that there is at least some conscious influence of central drive. With the knowledge that the end of the sprint coincided with the end of the time trial the cyclists could consciously influence the subconscious brain to provide a final all-out effort resulting in a suddenly increased power output.

These observations from this and other studies led Dr. Noakes and his associate, Alan St. Clair Gibson, to devise a new definition of fatigue that stated

“...fatigue is actually a central (brain) perception, in fact a sensation or emotion and not a direct physical event. This stems directly from our interpretation that exhaustion results from changes in central (brain) commands to the muscles, rather than as a result of changes in the muscles themselves.”(2)

Essentially they are saying that the central nervous system (brain) reduces force output by reducing neural drive to the muscles. The reduced drive results in a reduction in the number of motor units activated during exercise. In other words, the brain itself is the source of fatigue. Additionally, the feelings of fatigue that a runner consciously senses during exercise is an emotion or sensation sent by the sub-conscious mind to the conscious mind. Though you may feel like your legs are fatigued the origination of that feeling of fatigue is your brain, not your legs.

Central Governor Model

With the definition of fatigue now centered on central control rather than something occurring within the muscles, the next topic to be addressed is why does the brain reduce its neural drive? Is the brain itself becoming fatigued or are other things influencing the brain to decrease drive to the

working muscles? How does the brain know when to de-recruit fibers? How does it determine what lower level of fiber recruitment is appropriate? How does the brain go about selecting the appropriate pace for any particular event?

There is good evidence that the brain reduces its neural drive in order to protect the body from irreversible damage. Basically the brain subconsciously monitors the status of all systems of the body, continuously computes the metabolic costs to continue at the current pace and compares that to the existing physical state. Based on this information the brain adjusts the optimum pace so that the event is completed in the most efficient manner while maintaining overall body homeostasis and a reserve of physical and mental capacity. The brain protects the body by regulating power output during any form of exercise with the ultimate goal of maintaining homeostasis and protecting life.

An example of this process would be a slower pace during events with high ambient temperatures. Runners have long known that if the outside temperature is high that the running pace will be slowed due to the heat. Previously this phenomenon has been unexplained by the cardiovascular/anaerobic model except to say that the runner has to slow down to prevent over-heating. Conversely, the Central Governor model is able to successfully explain this well-known fact. The brain calculates the build up of heat due to the high ambient temperature and then selects a slower pace requiring a lesser power output, resulting in less internal body heat being generated. In this manner the brain protects the body from the dangers of over-heating.

Research studies provide evidence of this process. In one study scientists continuously measured the heart rate of cyclists during a 104 km cycling race (3). The researchers discovered that the cyclist's heart rate, which is commonly used as a measure of exercise intensity, increased and decreased in an apparently random manner in all the subjects continuously throughout the event. These changes were not solely related to geographical changes along the race course either. During times when the course was flat the random changes in heart rate continued to occur. These findings are consistent with the brain's on-going calculations of the known remaining distance to be covered and the physical state of the cyclists and then adjusting power output (and hence pace) accordingly. The findings of this study have been confirmed in another study of professional cyclists during the three-week Tour of Spain (4).

Summary

Based on the evidence the Central Governor Model suggests fatigue is a relative condition, not an absolute one – i.e. the athlete can always continue but at a slower pace. Muscle fiber power output is not regulated by factors in the muscle itself but is continuously reset by the brain based on continuous computations of the sensory feedback it receives from all of the body's systems. Fatigue is a relative process as exercise intensity is constantly changed during exercise as the brain either recruits additional fibers to

increase power output or decreases fiber activation to decrease power output based on its calculations.

In [part 2](#) we will compare the Central Governor Model to the Power Running Model.

Reference:

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