Searching for the Limits of Human Physical Performance

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Searching for the Limits of Human Physical Performance:

The Fatigue Chronicles

By

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Cambridge
Scholars
Publishing

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This book first published 2020

Cambridge Scholars Publishing

Lady Stephenson Library, Newcastle upon Tyne, NE6 2PA, UK

British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

ISBN (10): 1-5275-4554-7
ISBN (13): 978-1-5275-4554-0

Cover: The human-powered aircraft *Daedalus* in flight across the Aegean Sea (see Chapter 11). Photograph courtesy of the Massachusetts Institute of Technology, Department of Distinctive Collections, Cambridge, Massachusetts. Reprinted with permission.

watch

Richard Feynman, 1963

Angelo Mosso, 1915

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

[illegible]

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PREFACE

The point that advances in scientific knowledge have made our lives easier, safer, and healthier seems so obvious as to hardly worth making. Indeed, what were once “miracles” have become so commonplace in the course of our everyday lives so as to no longer seem “miraculous” at all. To compile a supportive list of such achievements would simply be too extensive—and obvious--to justify the effort. The bottom line is that we enjoy the benefits of living in a highly technological society, provided by the ingenuity of human minds and the computers that support them.

But, still. One does not need to delve very far into the scientific literature to become confronted with a startling and altogether humbling reality: the answers to some of the biggest questions regarding the nature of the physical [illegible] mystery. There is, quite simply, a great deal about which we have no comprehensive understanding. Yes, we can, in a dazzling display of [illegible] of [illegible] a [illegible] moon. But we have no insight into the essence of what makes living beings different from the non-living. What does it mean to be alive? It remains an unanswered question. And we can surgically insert artificial lenses, restoring in Biblical fashion the eyesight of those blinded by cataracts, but no one knows just why our bodies deteriorate with age. What is the nature of time? What do we mean by “art” [illegible] Is “beauty” subjective or objective? What, really, is “energy”? Why do we sleep? Why do fools fall in love? No one knows. And, then, there are the Really Big Questions [illegible] why are we here? And why is there something instead of nothing?

The [illegible] book [illegible] one of [illegible] *what limits the capacity of human beings to perform physical work?* [illegible] fact, at the present time no single satisfying answer. The evolution of [illegible] of [illegible] a [illegible] basis. Those with the greater facility for moving about would be endowed with a superior ability to gain food, escape enemies, and avoid [illegible] And, [illegible] a [illegible] who [illegible] fastest or who can endure the longest in motor activities could be expected to be most favored in terms of survival and reproductive fitness. But in this development of locomotor function it is obvious that there exist limits. At the finish line of the Boston Marathon, all the runners will have “given their

> [illegible] *why?* [illegible]

[illegible]

[illegible]

[illegible]

[illegible]

Notes

[illegible]

[illegible] *History of Exercise Physiology* [illegible] *The Human Motor. Energy, Fatigue, and the Origins of Modernity* [illegible] *The Performance Cortex. How Neuroscience is Redefining Athletic Genius* [illegible]

[illegible]

[illegible] *Collected Essays on Evolution, Nature, and the Cosmos.* [illegible]

[illegible] *Everything's Relative, and Other Fables from Science and Technology.* [illegible]

CHAPTER ONE

SETTING THE STAGE:
BASIC CONCEPTS

[illegible] continues to be one of Nature's most elusive [illegible] highly-guarded secrets: what is the nature of fatigue with exercise? What factor or factors are [illegible] performance? The "usual suspects" are many, the clues abundant—although [illegible] out of [illegible].

Answers offered in the past have been couched in some variety of "[illegible] of [illegible] or [illegible] of [illegible], or [illegible] form of muscle being used, or the muscular storage of energy substrate" and [illegible] on and so forth. At the other end of the speculative spectrum, couldn't there exist a single overlying principle that governs and defines the limit of one's ability to perform muscular work? A resolution to this dilemma would bear not just small significance. Besides the inquiring minds in the exercise science laboratory, for a good number of people--military strategists, labor bosses, health professionals, athletes and their coaches—an understanding of [illegible] of [illegible] output [illegible] importance. More than a few have devoted entire careers and lost a good deal of valuable sleep in attempts to solve the riddle. They have assumed [illegible] does exist an understandable reason why certain limits of physical performance cannot be exceeded. As corollaries, too, lie the expectations [illegible] be [illegible] be [illegible] b) [illegible] of such a biological reality can in fact be revealed though human reasoning.

The traditional perspective has focused on the identification of certain elements in the linear process that provide for locomotion and the limits of some particular element ([illegible] "weakest link") that would restrain the function of the entire chain of events. More contemporary viewpoints, particularly a) the role of subconscious cerebral factors which may control exercise limits and b) the interactive features of a systems approach to biological function, have provided exciting novel approaches, but, [illegible] discussed in Chapter

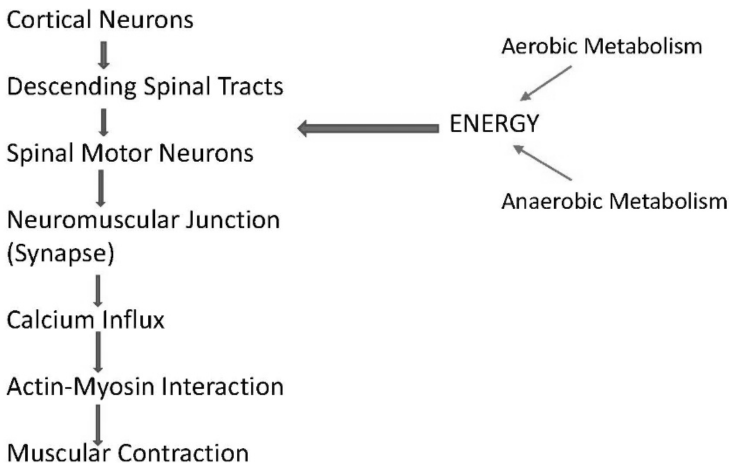
Thirteen, these intimate that an even greater complexity of the limitations of human locomotion exists than had previously been appreciated.

Before embarking on the chapters that follow, it will be useful to describe the foundations on which this search has been conducted. In this introductory chapter, then, the basic mechanisms recognized [illegible] be responsible for locomotion will be reviewed, various forms of fatigue examined, and the several hypotheses briefly presented for its critical [illegible] “leveling the playing field” for all readers [illegible] the subsequent stories of [illegible]

Mechanisms

Since the Scientific Revolution [illegible] the Seventeenth Century, the driving force in physiological research has been characterized largely by a search for mechanisms.[1] That is, this human body, just ‘how does it work? And, in contrast to the obscure identity of limiting factors [illegible] exercise capacity, here the nature of the components of the physiologic-chemical pathway that drives locomotion are known in rather exquisite detail. For our question at hand, then, this is quite convenient, since we are thus provided with a starting point [illegible] teasing out which of the components of this mechanism might best serve as candidates for the role of establishing the onset of muscle fatigue. It is worthwhile, then, to examine this operational picture, at least in an abbreviated fashion.

The mechanism underlying animal locomotion, paired down to its essentials, consists of a temporal cascade of physiological events that begins with electrical stimulation originating [illegible] the neurons of the central nervous system and eventuates in the mechanical sliding of actin-myosin filaments within the muscle fiber which effects muscular contraction (Figure 1.1). The sequence is finely-tuned by biological time keepers which control sequence of muscle contraction, thereby permitting a smoothly coordinated muscular contraction and effective propulsion of the body. The energy driving this series of events is derived from oxidation of stored carbohydrates and fat via ambient oxygen trapped by the lungs and delivered by the cardiovascular system to the skeletal muscle, as well by glycolytic anaerobic metabolic processes. It can immediately be appreciated, then, that a walk around the block calls into concert an amazingly complex but exquisitely-coordinated combination of electrical, biochemical, and mechanical processes that, in [illegible], [illegible], or [illegible] 1 [illegible] Here [illegible] more detail.



Neurological Stimulation

sodium-potassium

action potential,

neuromuscular junction

synaptic cleft

motor unit.

Chemical Activation

Acetylcholine crossing the synapse at the neuromuscular junction [illegible] the [illegible] resides on a system of internal membranes in the myofibrils of the skeletal [illegible] *sarcoplasmic reticulum.* [illegible] T-tubules, causing its release to trigger the actions of contractile proteins [illegible]. [illegible] up by the sarcoplasmic reticulum in preparation for the next contraction.

Contractile Apparatus

The mechanism of muscle contraction at the end of this cascade of events was deciphered by Hugh Huxley in Great Britain in the 1950's using an interference microscope. From his studies (and many others) the classic scenario of two protein filaments—actin and myosin—sliding past each other, increasing the region of overlap, and thereby shortening the muscle fibril has been commonly accepted. The calcium released from the sarcoplasmic reticulum is responsible for this action, causing the heads of the myosin molecule to attach [illegible] the actin by a series of cross-bridges. Each cross-bridge between the actin and myosin acts in a cyclical fashion, creating a force and then detaching, and the sliding action thereby produced shortens the length of the fibril and triggers muscle contraction. The maximum tension which is produced [illegible] considered to be governed by the amount of actin-myosin overlap. It is generally agreed that the rate of attachment of myosin [illegible] the actin filament establishes the generation of force, while the rate of detachment of these contact points accounts for the maximum shortening velocity.

Provision for Energy

No elements of the forgoing scenario could function without a [illegible]. [illegible] physiological events that eventuates in human locomotion demands energy, from the sodium-potassium pump to the secretion and uptake of synaptic neurotransmitters to the sliding of actin and myosin filaments. The currency for provision of energy for all these events resides in the chemical bonds of adenosine triphosphate (ATP), and the human body has contrived elaborate means of providing sufficient quantities of this energy source for its

Aerobic metabolism

anaerobic metabolism

glycolysis,

oxygen uptake,

Defining Fatigue

[illegible] *muscle fatigue.* [illegible]

[illegible] *peak* [illegible]

[illegible] *physiological* [illegible]
psychological barrier. [illegible]

Fatigue and Work Intensity

[illegible]

The Candidates

Clearly exercise induces a decline [illegible] maximal voluntary muscle force or pow[illegible] [illegible] [illegible] [illegible] of motor [illegible] [illegible] a [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] on [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] (MVC). This decrease in muscle contractile function has traditionally [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] how [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] repeated submaximal muscle contractile force, even at “exhaustion”, in an [illegible] [illegible] b) [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] deterioration in voluntary maximal muscle force production during the work of exercise remains unresolved. As one examines the cascade of neurophysiological events that are responsible for muscular contraction and human locomotion, there exists no lack of potential candidates. “Although it is not difficult [illegible] know when one is fatigued, [illegible] is entirely another matter [illegible] be able to identify the physiological mechanisms responsible for this condition. Although progress has been made [illegible] the study of muscle fatigue, we are largely unable to state with certainty why an individual becomes fatigued under various conditions.”[illegible]0

Such doubt notwithstanding, it is not difficult to identify and experimentally-support specific “loci” in the neuromuscular processes which eventuate in muscle force that could act as limiting factors to muscle force production. The following constitutes a brief overview. In examining each of these “candidates,” two empiric observations need to be kept in mind. First, deterioration of peak muscular force begins very early—at low work intensities—in a progressive exercise test. That is, whatever is responsible for the loss of capacity for the voluntary production of peak contractile force [illegible] acting *throughout* the exercise test, not being simply associated with performance fatigue. And, secondly, the decline in peak muscular force (as indicated as a fall in MVC) is inversely related [illegible] work intensity.

Central Fatigue

Logically, this analysis of “usual suspects” should commence in the motor [illegible] of [illegible] [illegible] of [illegible] [illegible], [illegible] [illegible] numb[illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] muscle contractile force [illegible] In fact, both factors—reduced frequency and [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] MCV [illegible] [illegible] [illegible] [illegible] of [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] respect to isometric forms of exercise, in which a single maximal

Homo sapiens.

Peripheral Neuromuscular Fatigue

in vivo

the SR when glycogen stores are depleted, etc. Considering research findings, David Allen and co-workers conclude that "low-frequency fatigue appears [illegible] be largely due to Ca^{+2}-dependent damage to the Ca^{+2} release mechanism."[13]

The "Poisoned Cell" Hypothesis

The idea that the byproducts of intensive muscular contractions serve to [illegible] one of [illegible] muscle fatigue with exercise. In 1911, the French hygienist Jules Amar wrote that fatigue was "fundamentally an intoxication; if the brain and the muscles function in a disorderly fashion as a result of excessive effort or too great a rate of exertion, the blood is no longer able to cope in its task of purification. The waste products of this intense cellular activity accumulate; the blood loaded with toxic produces fatigue in any animal into whose veins it is injected."[14] (This last comment was referring to Mosso's earlier study in which he injected a blood specimen from a fatigued dog into one who was rested, claiming that the latter then demonstrated signs of fatigue.)

In fact, the concept is intuitively attractive. During highly-intensive exercise, aerobic metabolic processes become supplemented by energy derived from anaerobic pathways (glycolysis). The latter produces a number of by-products which are recognized to impair skeletal muscle function, including hydrogen ion (H^+) from dissociation of lactic acid, lactate, ammonia, and inorganic phosphate. Most attention has focused on the accumulation of hydrogen ion, which effects a fall in muscle pH from a resting value of about 7.1 to 6.5 during highly intense exercise, such as sprinting. This level of acidosis has been demonstrated to impair ATP production while displacing Ca^{+2} within the muscle fiber. Accumulation of hydrogen ion may negatively influence glycolysis and disturb enzymatic reactions [illegible] the metabolic cell machinery.

Energy Depletion

The human exercise machine serves to convert the chemical energy in [illegible] we [illegible] oxygen inhaled [illegible] the air we breathe and supplied to the contracting muscle [illegible]. [illegible] the formation of ATP, the direct energy source for muscular contraction. [illegible] of [illegible]

[illegible]

[illegible] *peak* [illegible]

[illegible]

Exercise Fatigue and Systems Biology

[illegible]

[illegible] *reductionist* [illegible] *systems biology* [illegible] *complex system* [illegible]

[illegible] *Nature,* [illegible]

Symmorphosis

[illegible]