

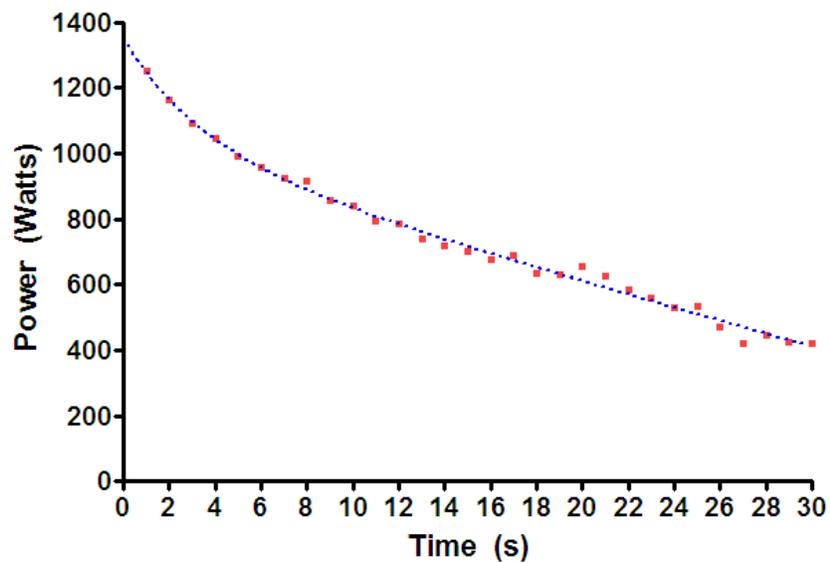
Exercise-Induced Fatigue

1. Short Term Intense Exercise (Phosphagen dependence)
2. Sustained Intense Exercise (Phosphagen and Glycolytic dependence)
3. Prolonged Exercise (Nutrient Provision and Thermoregulation dependence)



UNM

What is Fatigue?



UNM

What Contributes to Fatigue?

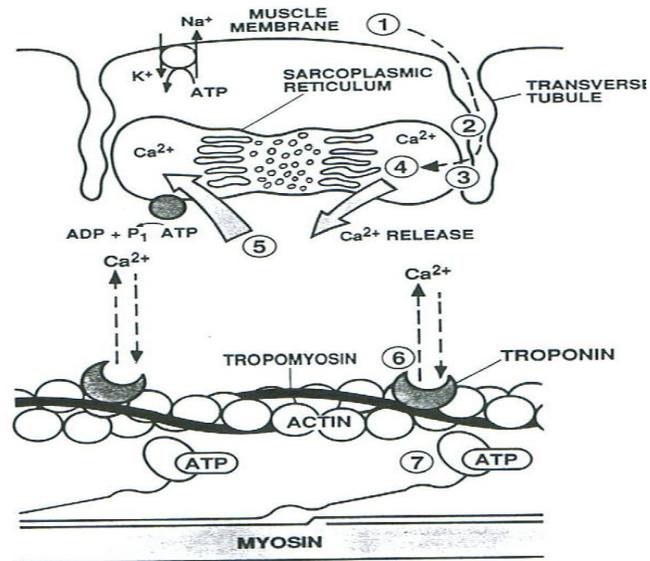


Peripheral Contributions To Exercise-Induced Fatigue

1. Short Term Intense Exercise (Phosphagen dependence)
2. Sustained Intense Exercise (Phosphagen and Glycolytic dependence)
3. Prolonged Exercise (Nutrient Provision and Thermoregulation dependence)

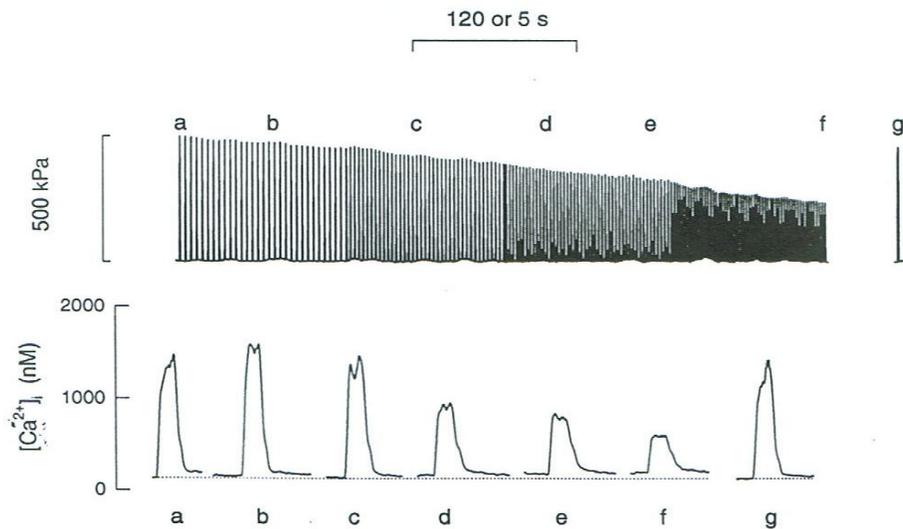


Sites of Electro-chemical Induced Muscle Fatigue

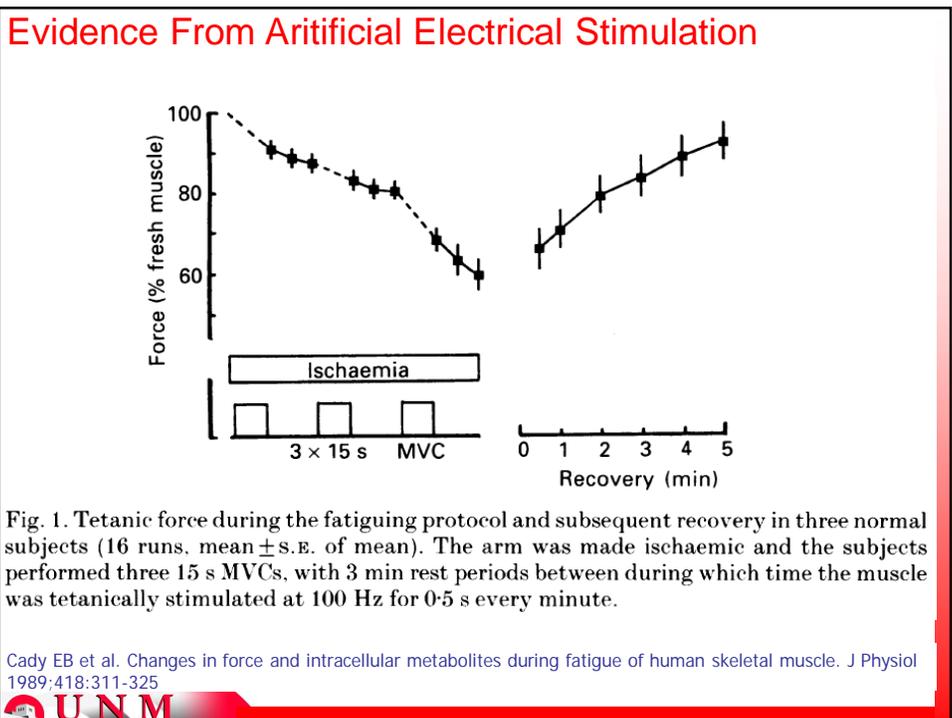
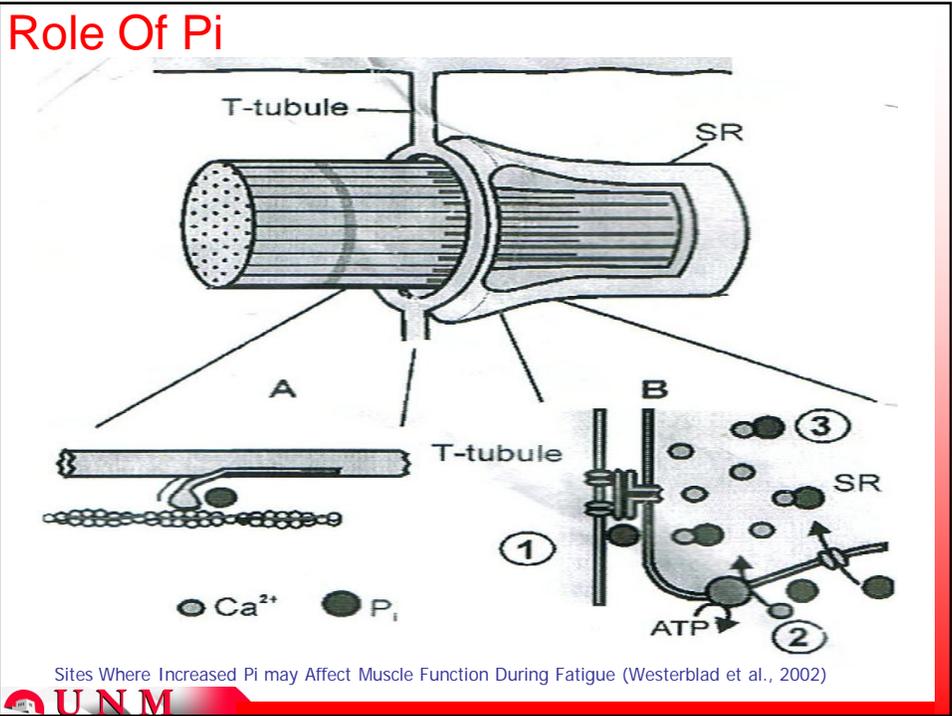


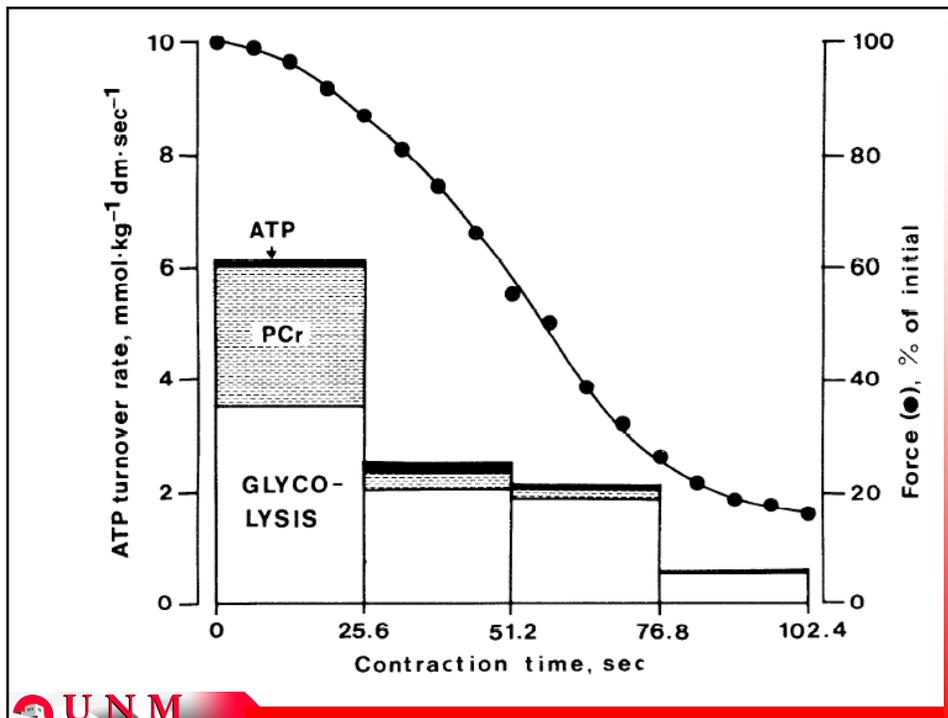
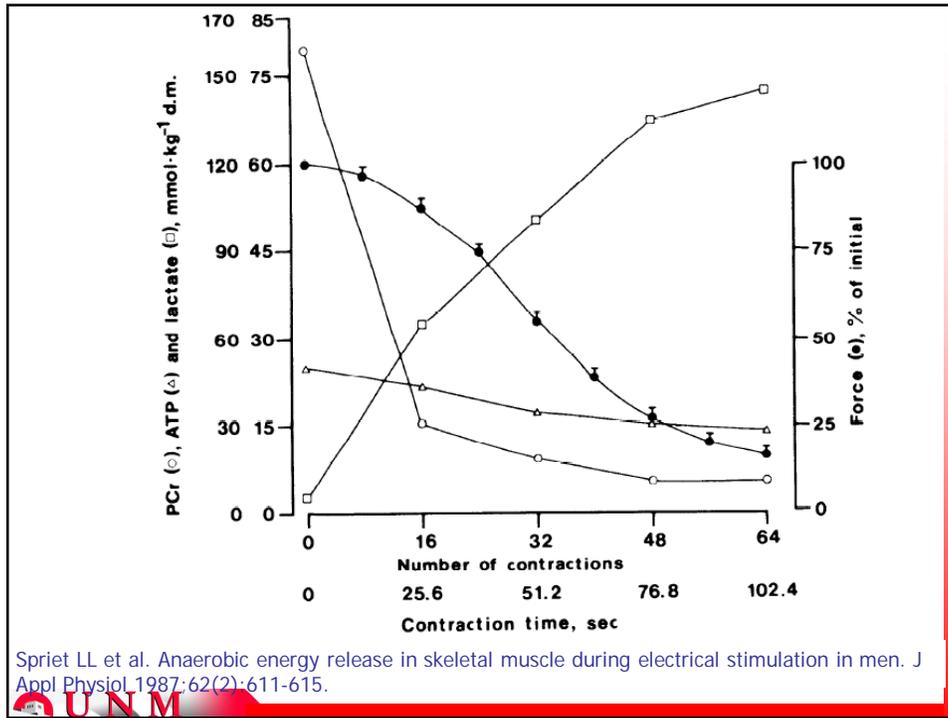
Possible Sites of Muscle Fatigue During Heavy Exercise (Fitts, 1994)

Role Of Ca²⁺



Isometric Force and Ca²⁺ during Repeated Tetanic Stimulation and Recovery (Kabbara & Allen, 1999)





Central Contributions To Exercise-Induced Fatigue

1. Short Term Intense Exercise (Central Drive, Motor unit recruitment)
2. Sustained Intense Exercise (Central processing of central & peripheral cues)
3. Prolonged Exercise (Central processing of central & peripheral cues)



UNM

Historical Perspective

Hargreaves M. Fatigue mechanisms determining exercise performance. *Integrative physiology is systems biology. J Appl Physiol.* 2008;104:1541-1542

It is important to realize that the concept of a central processing system that is involved in the exercise fatigue process is quite old!

Bainbridge FA. *Physiology of muscular exercise.* 1919, Longmans, London.

"It has long been recognized that the main seat of fatigue after muscular exercise is the central nervous system. There appear, however, to be two types of fatigue, one arising entirely within the central nervous system, the other in which fatigue of the muscles themselves is superadded to that of the nervous system."

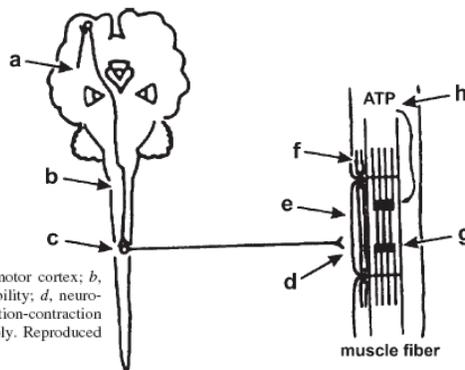


Fig. 1. Potential sites of fatigue: *a*, excitatory input to the motor cortex; *b*, excitatory drive to lower motoneuron; *c*, motoneuron excitability; *d*, neuromuscular transmission; *e*, sarcolemma excitability; *f*, excitation-contraction coupling; *g*, contractile mechanism; *h*, metabolic energy supply. Reproduced from Ref. 2 with permission.

UNM

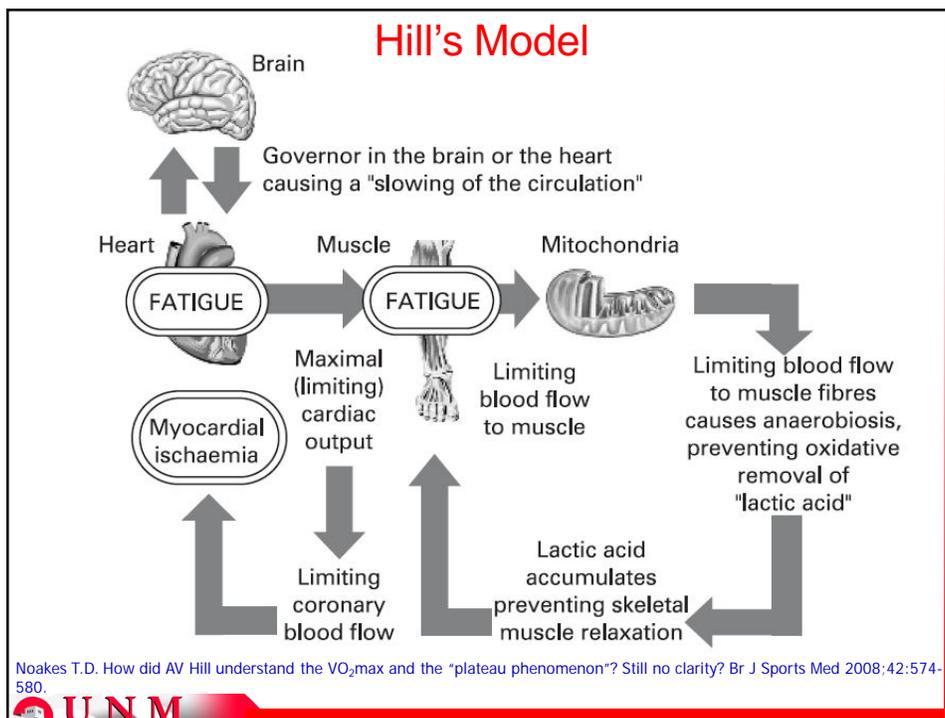
Historical Perspective, cont'd

Noakes T.D. How did AV Hill understand the VO_{2max} and the "plateau phenomenon"? Still no clarity? Br J Sports Med 2008;42:574-580.

- Hill may have been one of the first exercise physiologists to theorize a central processing "governor".
- The irony of Noakes' model is that it is named from the work of Hill, who most exercise physiologists argue provided evidence of muscle and cardio-pulmonary derived causes of fatigue during exercise!

"... it would clearly be useless for the heart to make an excessive effort if by so doing it merely produced a far lower degree of saturation of arterial blood; and we suggest that, in the body (either in the heart muscle itself or in the nervous system), there are some mechanisms which causes a slowing of the circulation as soon as a serious degree of unsaturation occurs, and vice versa. This mechanism would tend to act as a 'governor' maintaining a high degree of saturation of the blood."

Hill AV et al. Muscular exercise, lactic acid and the supply and utilization of oxygen: parts VII-VIII. Proc Royal Soc Brit 1924;97:155-176.

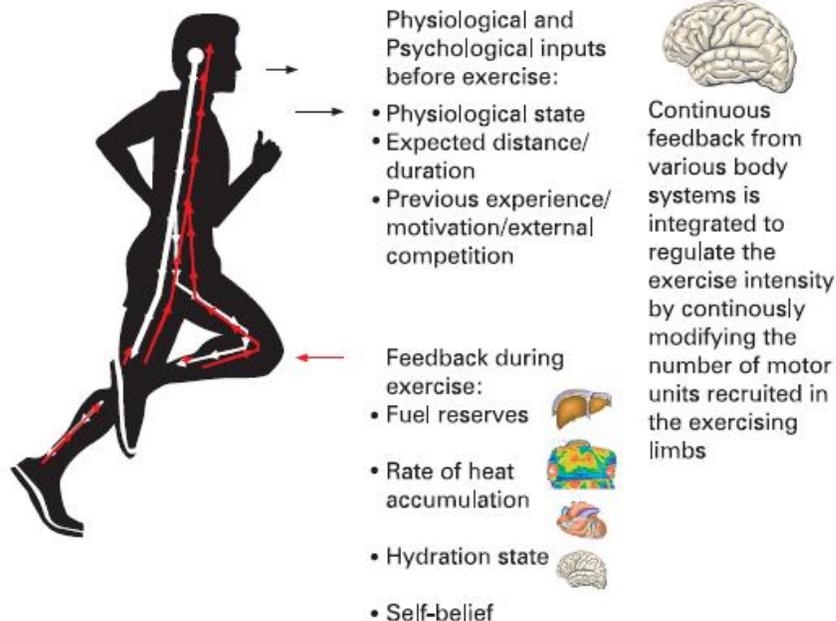


Compilation of Evidence Identified by Noakes and others

- No “catastrophic failure” in any organ/tissue during any exercise condition.
- Premature voluntary exhaustion at altitude, and during heat stress.
- Muscle ATP well preserved.
- Inability to recruit all motor units when “fatigued”.
- Why are there profound central perceptions of fatigue?
- Pacing strategies are clearly more manifested by the brain than muscle metabolism.
- Muscle function at VO_{2max} is not as compromised as previously assumed.
- Muscle blood flow is not “maximal” at VO_{2max} .
- Motor cortex has declining “sub-optimal” output during fatigue development.
- There are considerable changes in muscle afferent nerve activities during fatigue that influence both cortical and spinal level functions of the CNS.

UNM

Noakes' Central Governor Model



UNM

Anticipatory-Intense

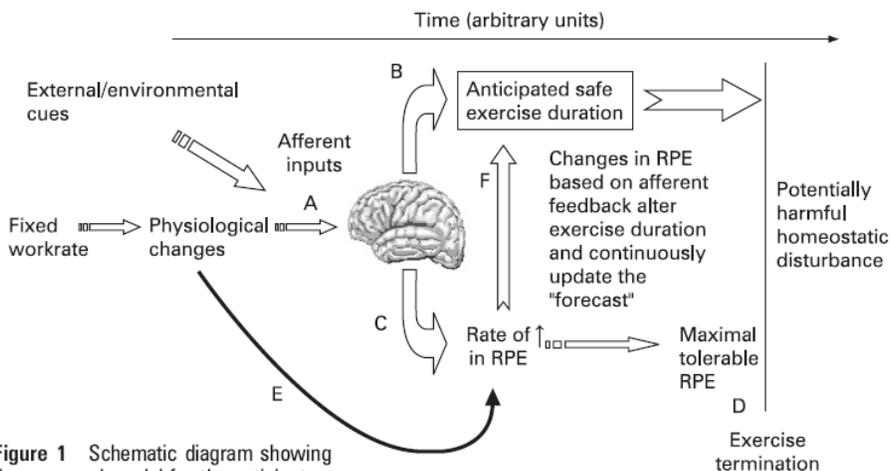


Figure 1 Schematic diagram showing the proposed model for the anticipatory regulation of exercise performance during exercise to fatigue at a fixed work rate. RPE, rating of perceived exertion.

Tucker R. The anticipatory regulation of performance: The physiological basis for Br J Sports Med. 2009;43:392-400



Self-Paced

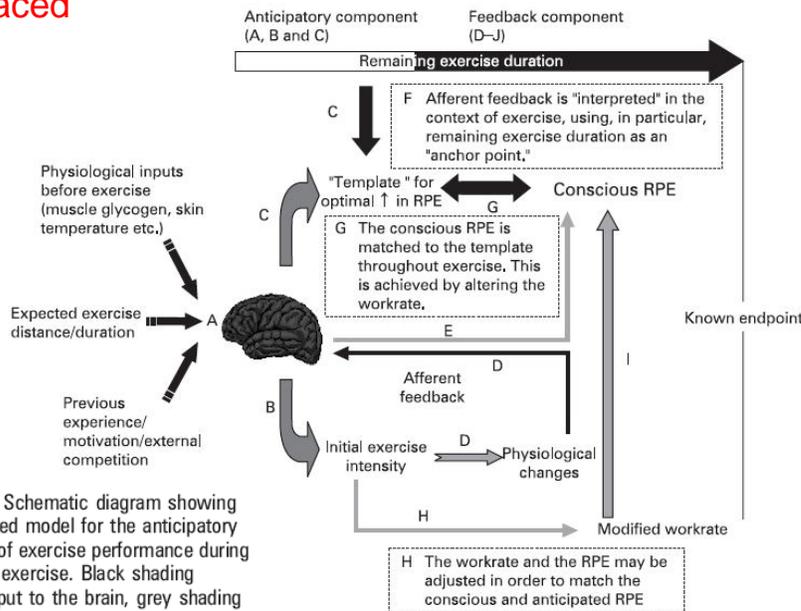


Figure 2 Schematic diagram showing the proposed model for the anticipatory regulation of exercise performance during self-paced exercise. Black shading denotes input to the brain, grey shading denotes output or efferent processes. RPE, rating of perceived exertion.



Conclusions

- There is no evidence of a “catastrophic failure” of any organ/tissue during any exercise condition in healthy humans.
- Yes, central command and some additional CNS processing exists during the process of fatigue.
- Yes, the brain/CNS is especially sensitive to the hyperthermia of exercise. Central cooling during intense exercise can delay volitional exhaustion.
- We need to be careful in distinguishing “fatigue” from the decision to end exercise at volitional exhaustion.
- Many individuals perform intense exercise to the brink of depleted phosphagen (CrP) and glycolytic (acidosis, [lactate]) capacity. How can this be anything other than muscle-based fatigue?
- We do not have the instrumentation to differentiate/quantify CNS vs. muscle roles in the instantaneous fatigue of intense exercise.
- There is no evidence of decreased motor unit recruitment in normoxia or mild hypoxia.

