Can you imagine Gatorade issuing the results from their first scientific studies of their drink and saying to people “Folks, we’ve tested the sports drinks, and we don’t have much evidence that you really NEED them. You’d most likely be fine without them, but hand over your money and buy your Gatorade at the counter anyway”? An unlikely scenario. Instead, the results of their testing was twisted to become a form of shameless endorsement for the sake of sales in subsequent years.

The two broad theories for muscle cramps

All sports-related drinks work off the same premise – they put back the electrolytes that exercise will take out. And it’s the loss of those electrolytes, the theory goes, that are responsible for the cramps during exercise. This theory, over 100 years old, is one broad category of theories for muscle cramps.

The second theory is that muscle cramps are caused by a ‘malfunction’ in the control of the muscle by the nerves – an abnormality of neuromuscular control which is caused by fatigue.

Our objective in this class is to look at these two theories, beginning with a bit of groundwork and history…

Defining cramp

Firstly, cramp has been defined as a “*spasmodic, painful, involuntary contraction of the skeletal muscle that occurs during or immediately after exercise*”.

Note that this definition applies to exercise-related cramps only, and therefore, it excludes a whole host of other possible cramps. Then of course, some cramps are what the experts call “idiopathic”, which means they have no cause (but actually means we don’t know what causes them, but it sounds better to say “idiopathic”!).

The history of cramping– THE ELECTROLYTE DEPLETION THEORY

The earliest reports of muscle cramps come from 100 years ago, when laborers in hot and humid conditions of the mines and shipyards suffered from cramps. Even that far back, the sweat could be analyzed, and it was noticed that the builders had a high chloride level in their sweat (chloride, incidentally, is one half of the salt in your sweat). The conclusion that was made was that the laborers were sweating out valuable electrolytes, causing their muscles (and nerves) to malfunction. The heat and humidity were key factors that caused this situation. It must be pointed out that no one measured the sweat of the laborers who DID NOT CRAMP, something that we’ll look at in our next post.

Later, the builders of the Hoover Dam famously recovered from cramp when they were made to drink salty milk, entrenching the theory that salt loss was the cause of cramp.

And perhaps rather surprisingly, that was it – based on those anecdotal observations, the theory which you probably hold true today, was born. That is, cramp is caused by a loss of sodium, chloride, and later calcium and magnesium were added to the mix. Heat and high humidity were blamed as additional causes, and so the term “Heat-Cramps” was created. According to this theory (**as seen by this article and the “expert” testimony**) , cramps happen because athletes exercise in the heat, lose electrolytes in their sweat, and the depletion combined with high body temperatures cause muscle cramp.

For example, take these **testimonies**:

“When a young athlete experiences heat cramps, pull him or her off the field into a cool area and gently stretch the affected muscle. “Have them drink, drink, drink, and then drink more,” says Albert C. Hergenroeder, professor of pediatrics at Baylor College of Medicine and chief of the sports medicine clinic at Texas Children’s Hospital.

“High-sodium drinks will prevent children from getting heat cramps,” says Jackie Berning, PhD, with the National Alliance for Youth Sports. “Gatorade has just enough sodium to prevent those cramps. But if you’re a heavy sweater, and you’re still getting cramps after drinking Gatorade, eat some salted pretzels or salted nuts. Those work fine.””

The problems with the serum electrolyte depletion theory

First of all, there is a key conceptual problem here, and that is that when you sweat, you don't actually reduce electrolyte concentration. That is, there are certainly electrolytes in the sweat, but when you sweat, you lose more water than electrolytes. Therefore, sweating cannot lead to a fall in electrolyte concentration.

What happened was that Gatorade (and the rest of the 'industry', it must be said) developed the theory of "salty sweaters", which is the term they gave to people who they said have abnormally high salt levels in their sweat. Small problem – no one actually knows what a salty sweater is. How much salt does there need to be in the sweat before you are placed in this group? No one knows. Recently, Professor Martin Schwellnus, widely published in this area, posed this question to scientists at the Gatorade Sports Science Institute at a conference on cramping – he received no answer.

The truth is, even the saltiest sweaters around still have plenty of electrolytes, and so the more they sweat, the more they will cause their electrolyte levels to rise, not to decrease. This is a very obvious problem that is overlooked by the electrolyte replacement advocates.…

The cramping paradox– WHY SPECIFIC MUSCLES?

The second problem is something we asked you in yesterday's post. We asked whether the depletion of serum electrolytes would be expected to cause cramps in specific muscles, or all over? Hopefully it is evident that if a cramp was caused by a loss of serum electrolytes, there is no reason for the cramp to be limited to one muscle only. Rather, you would cramp everywhere. In fact, in people who have lost a great deal of salt and have become hyponatremic (not during exercise, but clinically), we know that they cramp in ALL their muscles.

But somewhat surprisingly, exercise-associated muscle cramps ONLY happen in the muscles that have been used extensively for exercise. The afore-mentioned Prof Schwellnus found in 2004 that the quadriceps, hamstrings and calves made up 95% of cramps in the 56km Two Oceans race in Cape Town.

The lab vs. the field

One study published in 1990 showed that there was no association between potassium levels and cramps In that study cyclists rode for up to five hours. Some of the subjects did cramp, but their potassium levels were not uniformly high or low, thus showing no association between that variable and the cramps. However beyond that study (and one more that was presented at a conference but apparently not published) there is little real data out there to support or refute this hypothesis that dehydration or electrolyte disturbances cause cramps.

Study 1: Two Oceans Ultra Marathon

In a 2004 study published in the *British Journal of Sports Medicine*, Professor Schwellnus and his colleagues examined runners before and after the Two Oceans 56 km marathon in Cape Town.] Remember that many people, both scientist and personal trainer alike, will profess that cramps are caused by dehydration and/or some disturbance in the electrolytes (sodium, potassium, magnesium, etc.) So the important finding from this 2004 study was that when the crampers were compared to the controls—who were matched for body mass and finishing time—the only differences were that the crampers had *lower* sodiums and *higher* magnesiums. The problem with this is that a lower sodium concentration suggests *over*hydration and *not dehydration*, and also if magnesium deficiency is meant to cause cramps then surely the crampers should have been lower here?

The relevance of this study is that if dehydration and electrolyte disturbances really play such a large role in cramps (as they are proposed to), then the crampers should have much higher electrolyte concentrations since they would be losing fluid and causing the concentrations to rise. Yet instead we see something entirely different, first that the crampers had *lower* sodium concentrations, and second that the crampers were not really different compared to the controls.

What is also noteworthy from this study was that the crampers had an average loss of body weight of 2.9%, compared to 3.6% for the non-cramping controls. In otherwords, the people who DID NOT cramp lost more weight than the people who did. It goes further than this, because Schwellnus et al were able to measure the change in plasma volume as well – a more direct measure for what is happening to fluids. Here, they found that the crampers actually gained a small amount of 0.2% during the race. The non-cramping control subjects LOST 0.7%. So the sum effect of this data is that it suggests very strongly that cramping is not associated with dehydration, or with lower serum electrolyte levels, which is what we have had drilled into us for many years!

The follow-up study from Iron Man

Further evidence against serum electrolytes

The next year they published a study in *Medicine and Science in Sports and Exercise*, and instead of runners it was Ironman triathletes]. According to what most of us hear day in and day out, it is these ultra-distance athletes who are exercising for 10+ hours at a time that must be most susceptible to dehydration and electrolyte deficiencies. After all, they are sweating for hours on end, and the numbers tell us that with so many liters of sweat lost then they must also be losing grams and grams of “essential electrolytes” such as sodium. Below you will see the basic data on these athletes, and the important finding here is that we see the crampers and controls were the same age and were similar in mass, had similar pre to post cahnges in mass, and also finished the Ironman in similar times:

	CRAMPERS (N = 11)	CONTROLS (N = 9)
Age (years)	33.5 ± 8.8	35.4 ± 8.1
Pre-race mass (kg)	79.1 ± 5.9	77.7 ± 6.4
Post race mass (kg)	76.3 ± 5.6	74.6 ± 6.5
Body mass loss (%)	3.4 ± 1.3	3.9 ± 2.0
Total race time (min)	660.8 ± 77.9	685.7 ± 48.5

So the two groups were essentially the same in that the crampers did not spend longer in the course or lose more weight (a crude measure of dehydration). Yet again the crampers and the controls looked remarkably similar on paper—except as in the 2004 study the crampers again had a statistically significant lower sodium concentration, and, we will repeat this, that suggests they were more hydrated compared to the controls. . .yet they were cramping. Here are the data from the electrolytes in the two groups:

	CRAMPERS (N = 11)	CONTROLS (N = 9)
Sodium	140 ± 2	143 ± 3
Potassium	4.4 ± 0.06	4.2 ± 0.5
Magnesium	0.9 ± 0.2	0.8 ± 0.1

Recall that what is most often put forward as the cause of cramps is either dehydration or some electrolyte disturbance, but the data from these two studies do not support that hypothesis. Although these are field studies and we cannot assign a cause and effect relationship, this available evidence suggests that these (normal) levels of dehydration do not appear to cause cramps. If these levels of dehydration did cause cramps and were largely responsible for cramps, then what we should see is a very high incidence of cramps in all of the race finishers with the same physiological characteristics as these subjects—-or in other words, the vast majority of the race finishers.

The Muscle Fatigue and Spinal Reflex theory for Muscle Cramps

This theory has its origins in a paper published in 1997 in the *Journal of Sports Sciences*, in which Professor Martin Schwellnus and some colleagues looked at the electrolyte-dehydration issue and concluded that on the basis of insufficient evidence, a new theory was required They proposed, in this paper, that a cramp was the result of dysfunctional reflex control of the motor nerve as a result of fatigue.

A summary of the theory

But because we know it's a highly technical issue, we decided to give you the "executive summary" right at the start. For those who like the detail, read on. For those who just want the answer, here's the take-home message:

- Muscle contraction is initiated by a nerve, called the alpha motor neurone. The alpha motor neurone receives inputs from the higher brain areas (when you make conscious movements) as well as from the spinal reflexes
- These reflexes are responsible for protecting the muscle against either excessive stretching or loading – they are the muscle spindles and Golgi tendon organs, respectively
- There is evidence that fatigue causes increased firing from the muscle spindles, and decreased activity from the Golgi tendon organs
- The net result of this change in the activity of these reflexes is that the alpha motor neuron activity is increased, and the muscle thus contracts involuntarily**.**

The muscle spindle reflex

You've all probably heard of, or experienced, the classic "knee jerk" reflex, where a doctor (or a friend) taps on the knee tendon with a small hammer and you can't help but to kick out with your foot. Well, that simple test demonstrates the first important reflex.

So referring back to the knee-jerk reflex, the tapping on the knee causes your quadriceps muscle to stretch. As a result, the spindle fires, the Type Ia afferent activity to the spinal cord increases, and then the alpha motor neuron activity increases. When the alpha motor neuron fires, it causes the SAME muscle to contract, and that is why your quadriceps contract and you kick out your leg in response!

The Golgi Tendon organ reflex

Now, there is a second organ in the muscle that plays a role in reflex regulation – it's called the Golgi tendon organ. The Golgi tendon organ performs almost the opposite role to muscle spindle it monitors the tension in the muscles and tendons, and it is active when the muscle is contracted and lengthened (which puts load on the tendons). It's role is to make sure that the muscle does not contract too forcefully or under too much load. So when the muscle is placed under load (any contraction), the Golgi tendon organ fires, and it sends a signal to the spinal cord along what is called a Type Ib afferent (remember the spindles had Type Ia afferents).

So, the bottom line here is that if the Golgi tendon organ is stimulated, the end result is that muscle contraction is switched off. However, if the Golgi tendon organ is inhibited, then the alpha motor neuron activity will increase, and the muscle will contract even more – this is called "disinhibition".

So what happens with fatigue, and can it explain cramp?

In studies of muscle function and fatigue, the following has been found:

- When muscle becomes fatigued, the firing rate of the Type Ia Afferent fibers from the muscle spindle INCREASES;
- and the firing rate from the Type Ib Afferent fibers from the Golgi tendon organ DECREASES.

Remember, we previously explained that Type Ia Spindle activity will cause the muscle to contract, whereas Type Ib Golgi tendon organ activity will cause the muscle to relax. If the Golgi tendon organ is then inhibited, the muscle will contract.

Therefore, FATIGUE causes the following:

- Spindle activity increases – alpha motor activity increases – MUSCLE CONTRACTION
- Golgi tendon organ activity decreases – alpha motor activity increases – MUSCLE CONTRACTION

So you can see how fatigue could very easily explain a sustained, involuntary muscle contraction, because it switches on alpha motor neuron activity.

This theory is supported by a number of observations, which cannot be explained by the serum electrolyte depletion theory:

1. Which muscles are more likely to cramp?

This is a pretty important question. The answer, of course, is the active muscles. This theory explains why, because the fatigue, which alters the activity of these two important reflexes, is most manifest in the active muscle. The electrolyte theory doesn't explain why only the muscles being used for exercise tend to cramp – in fact, if low electrolyte levels were the cause of cramp, we'd expect generalized cramping, as occurs in clinical conditions where people lose a lot of salt and become hyponatremic. It does not happen in exercise, but the Fatigue Theory can explain it.

2. What kind of muscle cramps most often?

Here, the answer is that a muscle that crosses two joints will cramp more often. This makes sense according to the Neural Fatigue theory, because if a muscle spans two joints, then it means that the muscle is going to be in a shortened position when it contracts. Think of the calf muscle during swimming – your toes are pointed (the ankle is in plantar flexion), which means the muscle is contracting in a shortened position. When the muscle is in this position, then the activity of that Golgi tendon organ is going to reduced even more than normal. Add to this the contraction, which stimulates the muscle spindle, and the net result is that the inhibition of the motor neuron is reduced even further, predisposing one to cramp.In other words, it's actually the position of the muscle that predisposes to cramp.

3. When is cramp most likely to occur?

Here, the answer is a little less overwhelmingly in favour of the neural fatigue model, but it is still a good argument for it, and against the electrolyte theory. The answer, of course, is that cramps happen during racing and not training, and it happens only at the end of the race, when the athlete is most fatigued. One could of course argue that it's only at the end that the electrolyte levels drop to the point where it causes cramps, but we described in **Part II** that there's zero evidence for this. So the explanation now would be that the muscle becomes more and more predisposed to cramp as it fatigues.

Supporting this, Schwellnus et al found that Ironman triathletes who paced themselves poorly and tried to cycle or run faster than they were capable of (based on previous performances) were going to be the ones to cramp. In other words, if a guy was capable of a 6 hour 180km cycling leg in the IronMan, and he tried to do it in 5h45, he would cramp. Note that this has nothing to do with electrolytes – he replaces the same amount, would lose the same volume of fluid, but he cramps because his muscles are not able to do the work he is asking them to! The resulting fatigue is what causes the cramp.

4. Is there any evidence for the theory?

Note that the electrolyte theory cannot explain this finding. Even more important, with 20 seconds of passive stretching, the EMG activity goes down. This means that stretching relieves cramps. Why would this be the case if electrolytes were to blame? Surely if the cause was low electrolyte levels, then the only solution would be to replace them? But instead, the most effective treatment is stretching, and it has been shown that the alpha motor neuron activity goes down. The act of stretching restores the normal balance, because suddenly, the Golgi tendon organ activity goes up again, and the muscle eventually relaxes.

Classwork below

Before we get stuck into the actual question, I have to just step back and give a brief overview of how science evolves. I heard this analogy from a famous physicist named **Richard Feynman** – he of the Challenger explosion, and maybe the world's most famous scientist in the 1980s. It's the best analogy for science that I've heard.

Feynman said that science and research is like trying to understand the game of chess by watching just one square on the chess board! You watch that single square and based on observations, you create a series of hypotheses. So for example, your first hypothesis will be:

- All pawns move in a straight line and cannot move backwards or diagonally.

However, the more you watch, the more you measure, the more you add to your understanding. And eventually, there comes a point at which your initial hypothesis is disproved! For example, you suddenly notice that pawns can in fact move diagonally, if they are capturing an opponent's piece! *Your initial hypothesis must now be revised, or it will be incorrect*.