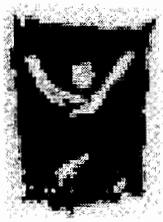

EXERCISE ASSOCIATED MUSCLE CRAMPING: INVESTIGATING A NOVEL HYPOTHESIS.

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Submitted in fulfilment of the degree
Master of Science in Medicine

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DEDICATION

Dedicated in loving memory of my Dad

" Die Erinnerung ist ein Paradies,

aus dem wir nicht vertrieben werden können."

(Jean Paul)

ABSTRACT

This thesis on Exercise Associated Muscle Cramping (EAMC) comprised three individual studies. The aims of this series of investigations were firstly to investigate the nature and prevalence of EAMC in a group of Ironman triathletes with a past history of EAMC, secondly to compare the serum electrolyte concentrations in cramping and control Ironman triathletes as well as to record the baseline electromyography (EMG) of cramping Ironman triathletes during recovery and thirdly to compare the EMG activity of cramping and control runners before, during and after a fatiguing bout of exercise.

The first study was a cross-sectional descriptive study of the Ironman triathletes who participated in the 2001 South African Ironman Triathlon (3.8km swim, 180km cycle, and 42.2km run). All prospective participants were sent a detailed questionnaire to complete and return at registration. Sixty eight and a half percent of triathletes reported a past history of EAMC and formed the 'cramp-history' group (CH, n=289). The 'cramp-history' group completed a specific section of the questionnaire detailing their cramping history (incidence of cramping, cramping severity, family history of cramping, factors associated with cramping). A significantly greater number ($p<0.01$) of the CH group cramped during running (37.1%) compared with swimming (28.9%) and cycling (15.6%), and during racing (46.1%) compared with training (18.8%). The majority of the CH group (56.5%) reported cramping in the final quarter of a race. The majority of the CH group rated their cramps as mild (77.5%), 17.4% as moderate and 3.6% as severe. Thirty eight and a half percent of the severe crampers had a family history of EAMC compared to 35.4% of the moderate and 25.2% of the mild crampers. The majority (58.4%) of the CH group associated muscle fatigue with EAMC, although electrolyte imbalances (42.9%) and dehydration (42.6%) were also perceived to be associated with cramps. These results show that there is a high lifetime prevalence of EAMC in Ironman triathletes. Furthermore, EAMC in triathletes is associated with running, racing and muscle fatigue and occurs mostly in the last quarter of a race. A positive family history of EAMC may be associated with the severe and debilitating form of cramping experienced by some triathletes during exercise.

The second study was a case control study which tested Ironman triathletes presenting with acute cramping after the 2000 South African Ironman Triathlon (cramping group, CR, n=11) and non-cramping controls (non-cramping group, CON, n=9) matched for body mass and race finishing time. All subjects were weighed during registration and immediately post-race. Baseline EMG (mV) (defined as the electrical activity in a muscle during remission from a bout of acute cramping) was recorded from the non-active control muscle (triceps) and the most severely cramping lower limb muscle (quadriceps, hamstring or calf) of the cramping group for a 10-minute period during recovery. Blood samples were drawn from both the cramping and non-cramping groups during recovery for the analysis of plasma magnesium, glucose, sodium, potassium and chloride concentrations. Haemoglobin and haematocrit were also measured. There were no significant differences between the CR and CON groups for registration or post-race body mass or percent (%) weight loss. Post-race sodium concentration was significantly higher ($p=0.01$) in the CON group than the CR group (142.7 ± 3.0 versus 139.5 ± 1.7 $mMol.L^{-1}$) but this was not clinically significant. There were no significant differences between the two groups for post-race serum electrolytes, glucose, haemoglobin concentrations or haematocrits. EMG (mV) of the cramping muscles was significantly higher ($p=0.04$, 0.04, 0.002, 0.05 respectively) than that of the control muscles of the CR group at 0, 3, 4 and 5 minutes of the 10-minute period. The findings of this study indicate that Ironman triathletes who suffer from EAMC are not dehydrated and do not have clinically significant serum electrolyte abnormalities associated with acute muscle cramping. Furthermore, the increased baseline EMG data of cramping muscles may indicate a heightened muscle activity possibly associated with muscle fatigue.

The final study recruited twenty male runners aged between 20 and 45 years. Ten runners with a recent history of EAMC formed the cramping group (CR, n=10) and were matched for age, body mass and 5km personal best race times with ten control subjects with no past experience of EAMC (control group, CON, n=10). Subjects completed a sub-maximal treadmill run at a speed of 75% of their pre-determined Peak Treadmill Running Speed to exhaustion (defined as the point at which subjects were no longer able to maintain the set treadmill pace). EMG activity (mV) was recorded from the right triceps and gastrocnemius muscles before exercise, at 30 minute intervals during exercise as well as for 20 minutes recovery. EMG activity was processed to yield amplitude and frequency spectrum data. The CR and CON group's triceps EMG amplitude (mV) remained stable throughout testing whereas the CR group's gastrocnemius EMG amplitude (mV) increased during and after exercise. The CR group's gastrocnemius EMG amplitude (mV) was consistently higher and more variable than the CON group at 60 min exercise and throughout recovery. Both the CR and CON groups' triceps and gastrocnemius frequency spectrum shifted left after exercise as a result of muscle fatigue. The CON group's frequency spectrum recovered after 20 min however, whereas the CR group's frequency shifted even further left after 20 min and had not recovered by the end of testing. These results show that cramping athletes have a higher and more variable EMG response to fatiguing exercise than non-cramping athletes and may take longer to recover from exhausting exercise than non-cramping athletes.

There is a high prevalence of Exercise Associated Muscle Cramps in Ironman triathletes which needs further investigation. EAMC is not associated with percent dehydration or clinically significant disturbances in serum electrolyte concentrations. The increased baseline EMG activity in cramping Ironman triathletes appears to support the hypothesis that EAMC is accompanied by heightened muscle activity possibly associated with muscle fatigue. The EMG response to progressive fatiguing exercise in athletes prone to cramping does not, however, support the hypothesised association between EAMC and muscle fatigue. The role of muscle fatigue in EAMC therefore remains undetermined and in need of further investigation.

DECLARATION

I, Nicole Uschi Sulzer, hereby declare that the work on which this thesis is based is my original work (except where acknowledgements indicate otherwise), and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other university. I empower the University of Cape Town to reproduce for the purpose of research either the whole or any portion of the contents in any manner whatsoever.

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To Mom, for your unconditional love, continuous support and unwavering belief in me-I could not have done it without you.

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LIST OF ABBREVIATIONS

Analysis of variance	ANOVA
Approximately	~
Beats per minute	bpm
Centimetre	cm
Chlorine	Cl
Control group	CON
Copyright	©
Correlation co-efficient	r
Cramping group	CR
Cramp history group	CH
Degrees centigrade	° C
Exercise Associated Muscle Cramps	EAMC
Electromyography	EMG
Gram	g
Grams per decilitre	g/dl
Hertz	Hz
Hour	hr
Hydrochloric acid	HCl
Integrated electromyography	IEMG
Kilogram	kg
Kilometre	km
Kilometres per hour	km/hr
Maximum voluntary contraction	MVC
Mean percentile frequency shift	MPFS
Metres per second	$m.sec^{-1}$
Millilitre	ml
Millimole	mMol
Millimole per litre	$mMol.L^{-1}$
Millivolt	mV
Minute	min
Newton metres	Nm
Non cramping group	NC

Peak treadmill running speed	PTRS
Percent	%
Plus/minus	±
Potassium	K
Probability	p
Revolutions per minute	rpm
Root mean square	RMS
Sample size	n
Second	sec
Sodium	Na
Surface electromyography	SEMG
Year	yr

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CHAPTER 1: INTRODUCTION AND SCOPE OF THE THESIS

1.1 Introduction

Exercise Associated Muscle Cramps (EAMC) can be defined as 'painful, spasmodic, involuntary contractions of skeletal muscle during or immediately after physical exercise' [70]. Muscle cramping is a common occurrence at most endurance events and the sight of athletes suffering from painful and debilitating cramping of the lower limbs is familiar to both competitors and spectators [28;49;66]. Despite the large prevalence of this painful and often frustrating condition, the exact aetiology and pathogenesis of EAMC is not well understood.

The lifetime prevalence of EAMC has been reported to be as high as 38.8% in marathon runners and 67% in triathletes [34;45]. Despite the high reported prevalence of Exercise Associated Muscle Cramping amongst triathletes, there has only been one published study on EAMC and triathletes [34]. It is clear that EAMC has not been thoroughly researched in either triathletes or Ironman triathletes and not much is known about the nature and prevalence of muscle cramping in these athletes.

There have been many theories for the aetiology of EAMC since the first reports of muscle cramping in labourers working in hot and humid conditions on steamships and in mines at the turn of the century [13;74]. The most common theory offered in published texts is that muscle cramping is associated with dehydration and abnormalities in serum electrolyte concentrations [14;15;30;51;55;64;78]. Recent evidence suggests, however, that EAMC in runners is not due to disturbances in serum electrolyte concentrations or hydration status [49;58]. The role of serum electrolyte abnormalities and hydration status in EAMC remains a contentious issue. Furthermore, although the serum electrolyte concentrations of cramping runners have been reported, no study has investigated the serum electrolyte concentrations of cramping Ironman triathletes.

Electromyography (EMG) has often been used to record the activity of cramping muscles in a laboratory setting [60;67]. Only one study has recorded the baseline EMG activity (the electrical activity in a muscle during remission from a bout of acute cramping) of cramping and control runners after an actual race [58]. There are no data on the baseline EMG activity of cramping Ironman triathletes after exercise and no study has yet compared the EMG activity of a non-exercising control muscle to the baseline EMG data of a cramping muscle after exercise.

It has been suggested that cramping is an abnormality of skeletal muscle relaxation [37]. Normal control of the alpha motor neuron at the spinal level is one of the essential physiological requirements for skeletal muscle relaxation [18]. Muscle fatigue has been shown to disrupt the homeostatic functioning of both the muscle spindle and Golgi tendon organ [31;56]. One study has reported an increased baseline EMG activity in cramping runners compared with controls after fatiguing exercise [58]. Recently a novel hypothesis for the aetiology of EAMC has suggested that EAMC is associated with muscle fatigue which alters the alpha motor neuron control at the spinal level through abnormal reflex activity [70]. This hypothesis has not yet been fully investigated.

This novel hypothesis for the aetiology of EAMC suggests that the EMG activity of cramping athletes would increase as muscle fatigue progressed and the control of the alpha motor neuron at the spinal level deteriorated. Although the EMG activity during spontaneous cramping as well as during recovery from cramping has been recorded, no study has recorded the EMG activity of athletes prone to cramping during progressive fatiguing exercise.

Exercise Associated Muscle Cramps is a condition that has not been thoroughly investigated and remains poorly understood, especially in Ironman triathletes. Most of the research on the aetiology for EAMC has focussed on the role of dehydration and serum electrolyte abnormalities but recent findings have questioned the validity of these early theories. A novel hypothesis has suggested an association between muscle fatigue and EAMC. This hypothesis offers a reasonable explanation for the aetiology of muscle cramping that affects so many exercising athletes and as such warrants further investigation.

1.2 Scope of the thesis

This thesis will begin by reviewing the literature available on Exercise Associated Muscle Cramps with an emphasis on the aetiology and pathogenesis of muscle cramping. Attention will be drawn to unanswered research questions in the current body of literature on muscle cramping in exercise.

It is apparent from the available literature that EAMC has not been thoroughly researched, especially in Ironman triathletes and not much is known about the nature and prevalence of muscle cramping in these athletes. A chapter of this thesis will thus focus on the nature and prevalence of muscle cramping in Ironman triathletes.

The role of serum electrolyte abnormalities and hydration status in EAMC remains a contentious issue. Recent studies that have investigated the serum electrolyte and hydration status of cramping athletes have focussed solely on runners. The only study that has recorded the baseline EMG activity after an actual race also focussed on cramping and control runners. This chapter will therefore focus on the serum electrolyte concentrations and hydration status of cramping and control Ironman triathletes after a race. It will also investigate the EMG activity of both a cramping and non-active control muscle in cramping Ironman triathletes after a race.

A novel hypothesis for the aetiology of EAMC has suggested an association between EAMC and muscle fatigue but this hypothesis has not yet been fully investigated. A final focus of this thesis will therefore be to investigate the EMG amplitude and frequency spectral changes of athletes prone to cramping during progressive fatiguing exercise.

The final chapter will present a summary and conclusion of the main findings of this thesis on Exercise Associated Muscle Cramping.

CHAPTER 2: LITERATURE REVIEW

2.1 Introduction

Exercise Associated Muscle Cramps (EAMC) is one of the most common problems requiring medical attention at both marathon and triathlon events [28;29;36;66]. Despite the high prevalence of this condition amongst athletes, the exact aetiology and pathogenesis of muscle cramping is not well understood.

This review will deal specifically with EAMC and will define and differentiate this condition from other causes of skeletal muscle cramping. The lifetime prevalence of EAMC amongst athletes from various disciplines will be reported and the factors associated with EAMC will be reviewed.

The 'traditional theories' for the aetiology of EAMC include the role of serum electrolyte abnormalities, hydration status, metabolic abnormalities and environmental factors in muscle cramping. Recently a novel hypothesis for the aetiology of EAMC has postulated that muscle fatigue may be associated with EAMC. These theories for the aetiology and pathogenesis of this condition will be discussed in detail.

Surface electromyography (EMG) is often used to identify muscle fatigue and has also been used to record muscle activity during cramping. The results of these investigations into muscle fatigue and cramping using the EMG technique will also be discussed.

2.2 Definition and classification of EAMC

Muscle cramping can occur as part of the general symptom complex of a variety of medical conditions and abnormalities that have recently been reviewed and are listed in Table 2.1 [69]. It is not within the scope of this review to discuss all these conditions. This review will, thus, focus solely on Exercise Associated Muscle Cramping.

Table 2.1 Classification of the causes of skeletal muscle cramps.

1. Acquired skeletal muscle cramp syndromes

1.1 Exercise Associated Muscle Cramps (EAMC)

1.2 Occupational cramps

1.3 Nocturnal calf muscle cramps

1.4 Pregnancy-associated cramps

2. Congenital abnormalities

2.1 Metabolic abnormalities

- 2.1.1 Carbohydrate metabolism-glycogen storage diseases
- 2.1.2 Fat metabolism-carnitine palmityl transferase deficiency
- 2.1.3 Purine nucleotide deficiency-muscle adenylate deaminase

2.2 Other congenital abnormalities-myotonia congenita, autosomal dominant cramping disease

3. Other acquired diseases

3.1 Endocrine diseases-thyroid disease, diabetes mellitus

3.2 Fluid and electrolyte imbalances-hyperkalaemia, hypokalaemia

3.3 Neuromuscular diseases-peripheral neuropathy, myotonic dystrophy

3.4 Other acquired diseases-liver cirrhosis, neoplastic disease, diarrhoea

3.5 Reactions to drugs or toxins-beta-agonists, ethanol, lead, strychnine

There are also other specific clinical syndromes where skeletal muscle cramps are the major symptom. These include occupational cramps, nocturnal calf muscle cramps, pregnancy-associated cramps and EAMC (Table 2.1). EAMC excludes cramps that occur in smooth muscle and in skeletal muscle at rest and also excludes muscle cramping associated with any underlying disease or use of drugs [70]. For the purpose of this review EAMC will be defined as a 'painful, spasmodic and involuntary contraction of skeletal muscle that occurs during or immediately after exercise' [70].

2.3 Lifetime prevalence of EAMC

Prevalence can be defined as the 'overall proportion of a population who suffer from a disease' [20]. The lifetime prevalence of EAMC can thus be defined as the number of athletes who have ever experienced EAMC at some time in their athletic career. The lifetime prevalence of EAMC for various activities and distances is presented in Table 2.2. It appears from the reported lifetime prevalence that the prevalence of EAMC increases as a function of the intensity and duration of the activity, with the highest lifetime prevalence of EAMC being reported in triathletes.

Table 2.2 Lifetime prevalence of EAMC (%) for various activities.

Activity	Lifetime prevalence (%)	Reference
Rugby	52%	77
Cycling	60%	21
Marathon (42.2km)	38.8%	45
Triathlon	67%	34

Despite the high reported prevalence of Exercise Associated Muscle Cramping amongst triathletes, there has only been one published study on EAMC and triathletes which documented the nature and prevalence of muscle cramping in United States Triathlon Series participants [34]. It is clear that EAMC has not been thoroughly researched in either triathletes or Ironman triathletes and not much is known about the nature and prevalence of muscle cramping in these athletes. Therefore this thesis will focus on the nature and prevalence of EAMC in Ironman triathletes.

2.4 Aetiology of EAMC

2.4.1 Historical background

The first reports of muscle cramping related to physical activity were from labourers working on steamships and in mines in hot, humid conditions at the turn of the century [13;74]. In these early reports it was noted that cramping not only occurred in the heat but that cramps were also accompanied by profuse sweating [74]. These anecdotal observations led to the still commonly accepted 'serum electrolyte' and 'dehydration' theories for the aetiology of EAMC. In the early 1950's a patient suffering from exertional muscle cramps was found to have a deficiency of myophosphorylase [50]. Several other metabolic abnormalities associated with cramping were subsequently reported which resulted in the development of the 'metabolic abnormality' theory for the aetiology of muscle cramping [2;17;38]. Case reports of cramping in extremely hot conditions have given rise to the 'environmental' theory for the aetiology of EAMC [74]. These 'traditional theories' for the aetiology of EAMC will be discussed in detail below.

2.4.2 Serum electrolyte theory

At the turn of the century it was noted that patients exposed to physical exercise in hot and humid environments developed muscle cramps and that this was associated with hyponatraemia and hypochloraemia [13;61]. Other serum electrolyte abnormalities, including hyperkaleamia, hypomagnesaemia and hypocalcaemia, have also been associated with EAMC [5;8;74;75]. The majority of these reports were based solely on anecdotal observations, however, with no proposed mechanism to explain how such imbalances in serum electrolytes could result in localised muscle cramping. Despite this, many published texts still support the role of altered serum electrolyte concentrations in EAMC [15;30;51;64;78].

Experimentally induced hyponatraemia, if accompanied by sodium loss, has been associated with generalised skeletal muscle cramping at rest [52;53]. Altered serum electrolyte concentrations caused by systemic abnormalities can result in generalised skeletal muscle cramping [69]. This is in contrast to EAMC which only occurs in the localised muscle groups involved in the repetitive contractions associated with exercise [70]. Thus results from studies which have examined the relationship between serum electrolyte abnormalities in systemic diseases and in skeletal muscle at rest may not be applicable to EAMC [70].

Only recently, two prospective cohort studies have examined the relationship between serum electrolyte abnormalities and EAMC in marathon runners [49;58]. The first study compared the serum electrolyte concentrations (sodium, potassium, calcium, phosphate) between marathon runners with acute EAMC (n=15) and control runners (n=67) before and immediately after the race [49]. This study reported no significant differences in serum electrolyte concentrations between the two groups and concluded that the results 'failed to produce any support for the suggestion that muscle cramp is the result of gross disturbances of electrolyte balance' [49].

The second study also found no significant differences in serum electrolyte concentrations (sodium, potassium, magnesium, calcium) between runners with acute EAMC (n=21) and control runners (n=22) either before an Ultra-distance run, immediately after the run or after 60 minutes of recovery [58]. This study reported a complete dissociation between EAMC and changes in serum electrolyte concentrations.

The findings of these two studies do not support an association between EAMC and abnormalities in serum electrolyte concentrations. Despite this recent evidence, the 'serum electrolyte' theory is still widely accepted and remains a contentious issue. Although the serum electrolyte concentrations of cramping runners have been reported, no study has investigated the serum electrolyte concentrations of cramping Ironman triathletes. Therefore this thesis will focus on the serum electrolyte concentrations of cramping and control Ironman triathletes.

2.4.3 Dehydration theory

Dehydration is often implicated as a cause of EAMC and is cited in many textbooks as a theory for the aetiology of muscle cramps [14;55;78]. The dehydration theory for the cause of EAMC has its roots in the early part of the twentieth century when case series reports linked cramping in mine workers to excessive sweating and presumed dehydration [74]. These were once again anecdotal observations and no actual measures of hydration status were reported.

Recently, two studies have documented the relationship between hydration status and EAMC [49;58]. In both studies body mass, blood volume and plasma volume were used as an indication of hydration status. The first study reported no significant differences in hydration status between marathon runners with EAMC (n=15) and control marathon runners (n=67) and concluded that EAMC was not associated with changes in fluid balance [49]. These findings were supported by the second study which also found no significant differences in hydration status between ultra-distance runners with EAMC (n=21) and control runners (n=22) [58].

The findings of these studies on hydration status and EAMC do not support the hypothesis of a direct relationship between dehydration and muscle cramping in runners yet this too remains a contentious issue. Furthermore, no study has compared the hydration status of cramping and control Ironman triathletes after a race. Thus the hydration status of cramping and control Ironman triathletes will also be investigated in this thesis.

2.4.4 Metabolic abnormality theory

Various inherited metabolic abnormalities are associated with skeletal muscle cramping during exercise (Table 1) [70]. Although cramping is a common symptom between these inherited metabolic abnormalities and EAMC, it is important to note that there are fundamental differences between the clinical features of these inherited metabolic abnormalities and EAMC [70]. There is little support for the hypothesis that the most common form of EAMC is caused by inherited metabolic abnormalities and this theory will not be investigated further in this thesis.

2.4.5 Environmental theory

The term 'heat cramps' was first used to describe cramps associated with physical exercise in hot and humid conditions [74]. Although EAMC is often associated with the heat, cramping has also been reported in marathon runners in cool weather [33]. It has also been reported that the development of EAMC is not directly related to an increased core temperature [49]. Furthermore, passive heating alone (at rest) does not result in EAMC and cooling does not relieve muscle cramps [70]. Indeed, exposure to extreme cold has also been associated with EAMC in swimmers [36]. It would appear that heat alone is not a direct cause of muscle cramping during exercise. This theory will not be investigated further in this thesis.

There are many theories for the aetiology of EAMC yet none have conclusive evidence. The exact aetiology of muscle cramping remains unclear and in need of further investigation.

2.5 Factors associated with EAMC

Although the exact nature of EAMC is still unclear, anecdotal observations and epidemiological studies have identified some factors that may be associated with muscle cramping.

An epidemiological study of over 1 300 marathon runners identified older age, a longer history of running, higher body mass index, shorter daily stretching time and irregular stretching habits as risk factors associated with EAMC [45]. An observation from these results is that poor stretching habits seem to be associated with an increased risk for EAMC. It is well accepted that passive stretching provides relief from muscle cramping [3;12;27;60;71].

Contracting a muscle maximally in a shortened position has often been used to induce cramping episodes [60;67]. In addition, it is well documented that the muscles most prone to cramping are those that span across two joints [45;58;69]. These are also the muscles that are often contracted in a shortened position during exercise. These two observations suggest that muscle length may also be associated with cramping [3;60].

High-intensity running (racing), subjective muscle fatigue, hill running and poor race performance were also reported to be associated with EAMC in the epidemiological study of marathon runners [45]. It has also been reported that EAMC is more common in less well-trained athletes which supports the suggestion that subjective muscle fatigue and poor race performance may be associated with EAMC [32]. It is well documented that most cramping episodes occur in the later stages of a race, usually after 30km in a standard marathon and in the late run stage of an Ironman Triathlon, which suggests that a long duration of running may also be associated with EAMC [34;45;49].

One study investigated the prevalence of a positive family history of muscle cramping in 1 383 marathon runners with EAMC [46]. This study reported that 19% of cramping marathon runners had a family history of EAMC compared with 10% of non-cramping runners who reported a positive family history of cramping [46].

It is clear that not much is known about the factors associated with EAMC, especially in triathletes. The factors associated with EAMC in Ironman triathletes are also not well documented. This thesis will focus on identifying some of the factors associated with EAMC in Ironman triathletes.

2.6 EAMC as an abnormality of skeletal muscle relaxation

It has been suggested that cramping is an abnormality of skeletal muscle relaxation [37]. Normal control of the alpha motor neuron at the spinal level is one of the essential physiological requirements for skeletal muscle relaxation [18]. The peripheral receptors, namely the muscle spindle and Golgi tendon organ, play a crucial role in the neurophysiological control of skeletal muscle.

The muscle spindle responds to changes in muscle length by increasing the firing rate of its type Ia and II alpha afferents which in turn excite the gamma efferents at the spinal level resulting in a contraction of the spindle's intrafusal fibres and shortening of the muscle [51]. The Golgi tendon organ responds to changes in muscle tension and when stimulated results in a reflex inhibition and relaxation of the muscles it innervates [78]. Muscle relaxation is thus the result of both a decreased excitatory Ia and II afferent input from the muscle spindle and an increased inhibitory Ib afferent activity from the Golgi tendon organ to the alpha motor neuron cell body [18].

2.7 Peripheral receptor activity during muscle fatigue

Muscle fatigue has been shown to disrupt the functioning of both the muscle spindle and Golgi tendon organ [31;56]. In one study fatigue to 50-60% of maximum titanic tension was induced in the isolated gastrocnemius muscle in 16 cats and the activity in the muscle spindles' type Ia and II afferents was recorded in response to a ramp stretch protocol [56]. This study reported an increased dynamic response to ramp stretch in the muscle spindles' type Ia and II fibres with fatigue [56]. A further study measured the response of the Golgi tendon organs' type Ib afferent fibres in the isolated gastrocnemius muscles of 13 cats after a ramp stretch protocol to fatigue [31]. These findings showed a significant decrease in the type Ib afferent fibres' discharge with fatigue [31].

These data suggest that in response to muscle fatigue, the excitatory muscle spindle afferents' firing rate increases while the inhibitory Golgi tendon organ afferents' activity decreases dramatically, although the exact mechanism responsible for this alteration in reflex activity is not yet known [31;56].

2.8 Electromyography (EMG) and muscle fatigue

Muscle fatigue is a complex and multi-faceted phenomenon which is influenced by both psychological and physiological factors and which, despite much research attention, remains poorly understood [47;62]. Muscle fatigue has been defined as 'the inability of a muscle or group of muscles to maintain the required force' and as 'the inability of a physiological process to continue functioning at a particular level' [47;62].

Electromyography (EMG) is a technique that is used to assess the extent of muscle activation, muscle fatigue and neural activity during muscle contraction [51;73]. The usefulness of surface EMG in the study of muscle fatigue is widely accepted [23;47;73]. Decreases in the median frequency of the power spectrum of the EMG signal, also referred to as left frequency shifts, are commonly used to identify muscle fatigue [9;42;47]. Changes in EMG amplitude are also often used to assess the degree of muscle activation as fatigue progresses [22].

It is generally accepted that EMG amplitude increases gradually with a sustained contraction at a constant force as progressively more motor units are recruited to maintain the required force output [22]. This is often followed by a decrease in the EMG amplitude when the desired force can no longer be maintained [22]. This decline in motor unit activation at certain exercise intensities has been called 'muscle wisdom' [48]. Muscle wisdom can be defined as the 'functionally appropriate reduction in motor unit firing rates which occurs during a sustained maximal voluntary contraction [48]. This phenomenon is a protective mechanism when the required force in the muscle can no longer be maintained without resulting in some form of muscle damage [22].

The majority of research into muscle fatigue has focussed on isometric contractions to fatigue [7]. These studies all report an increase in EMG amplitude and a decrease in the median frequency of the power spectrum of the EMG signal with isometric fatigue [23;25;80]. The few studies that have used endurance type exercise to induce fatigue report a decrease in the median frequency of the power spectrum of the EMG signal with fatigue but fail to mention the EMG amplitude changes with fatigue [9;23].

One study reported a decrease in gastrocnemius EMG amplitude immediately after a fatiguing marathon run which returned to pre-exercise levels after 120 minutes recovery [1]. In contrast to this finding, several studies have reported an increase in EMG amplitude during recovery from fatiguing isometric exercise [16;80;81]. One of these studies reported a decrease in the median frequency of the power spectrum of the rectus femoris and vastus lateralis muscles' EMG signal during isometric exercise which recovered to pre-exercise values after 15 minutes recovery, while another study reported that the frequency spectrum of the biceps brachii muscle continued to decrease after 10 minutes recovery from isometric exercise [16;80].

EMG is a useful tool to identify and measure muscle fatigue but caution must be taken when interpreting EMG results as the amplitude and frequency spectrum changes with fatigue are dependant on the test protocol, the type of exercise used to induce muscle fatigue and the muscles being tested.

2.9 Electromyography (EMG) and Exercise Associated Muscle Cramps

Electromyography (EMG) can also be used to measure the muscle and neural activity during muscle cramping. Numerous studies have recorded the EMG activity during spontaneous and induced muscle cramping [27;60;67].

One of the first studies to investigate the EMG activity of cramping muscles compared EMG activity during rest, voluntary contraction and induced and spontaneous muscle cramp in the rectus femoris and gastrocnemius muscles of five healthy adults [60]. This study reported that there were no apparent differences in the EMG activity of induced and spontaneous cramp, that the rate of motor unit firing was particularly high during a cramp and that the EMG activity in the cramping muscle seemed to reflect an 'abnormal hyper excitable state' [60].

Another more recent study induced cramp in the medial head of the gastrocnemius muscle in four subjects [67]. This study reported an increased EMG activity in the cramping muscle compared with a voluntary contraction in the same muscle as well as significantly higher and more variable motor unit firing rates during cramp than during voluntary contraction [67]. A further study also reported an increased EMG activity in various muscles of four cramping athletes which decreased with passive stretching [27].

One study recorded the baseline surface EMG activity in runners presenting with severe EAMC after an Ultra-endurance marathon [58]. Baseline EMG activity can be defined as the electrical activity in a muscle during remission from a bout of acute cramping. Baseline EMG activity was recorded immediately after the race and again 60 minutes later in a group of EAMC athletes (n=9) and a group of control runners (n=14) [58]. This study reported a significant decrease in the cramping group's baseline EMG activity compared to the control group during recovery.

This study also recorded the EMG activity in four runners during an acute cramp and after passive stretching [58]. They reported an increased baseline EMG activity in runners with EAMC which increased dramatically during a cramp and responded favourably to passive stretching which caused a progressive decrease in baseline EMG activity to pre-cramp values [58]. This study concluded that runners with EAMC have an increased baseline EMG activity which suggests a sustained alpha motor neuron activity and that a decrease in baseline EMG activity appears to be the best predictor of clinical recovery [58].

Although the EMG activity of cramping muscles in a laboratory setting has been recorded often, only one study has recorded the baseline EMG activity of cramping and control runners after a marathon. There are no data on the baseline EMG activity of cramping Ironman triathletes after exercise and no study has yet compared the EMG activity of a non-exercising control muscle to the baseline EMG data of a cramping muscle after exercise. Therefore, this thesis will compare the baseline EMG activity of both a cramping and non-exercising control muscle in cramping Ironman triathletes after a race.

2.10 A novel hypothesis for the aetiology of EAMC

Despite numerous theories, the aetiology of EAMC is still not fully understood. Muscle fatigue has been identified as a risk factor for EAMC and has been shown to disrupt the functioning of the peripheral receptors that play an important role in skeletal muscle relaxation [31;45;56]. Furthermore, it has been suggested that muscle cramping is an abnormality of skeletal muscle relaxation [37].

A novel hypothesis for the aetiology of EAMC has been proposed which suggests that EAMC is the result of an abnormality of alpha motor neuron control at the spinal level [70]. Muscle fatigue has been shown to disrupt the functioning of the peripheral muscle receptors by causing an increased firing rate of the muscle spindle's type Ia and II afferents as well as a decrease in the type Ib afferent activity from the Golgi tendon organ [31;56]. It is postulated that a combination of the increased excitatory activity of the muscle spindle and a reduced inhibitory effect of the Golgi tendon organ with muscle fatigue would result in a sustained alpha motor neuron activity caused by an abnormality of the alpha motor neuron control at the spinal level (Figure 2.1) [70].

This hypothesis is supported by the observation that cramping can be induced in muscles which are contracted in a shortened position [60;67]. Contracting a muscle in its shortened position would decrease the tension in the tendons of the muscle during contraction and further decrease the inhibitory afferent activity from the type Ib afferents of the Golgi tendon organ [70]. Passive stretching is the most common therapy to relieve cramping [3;12;27;60;71]. Passive stretching increases the tension in a muscle, thereby increasing the Golgi tendon organ's inhibitory activity [56]. The effectiveness of passive stretching in treating EAMC offers further support for the hypothesis that abnormal spinal reflex activity is associated with EAMC [70]. This hypothesis would also explain the increased baseline EMG activity recorded between bouts of cramping in athletes suffering from EAMC after fatiguing exercise [58].

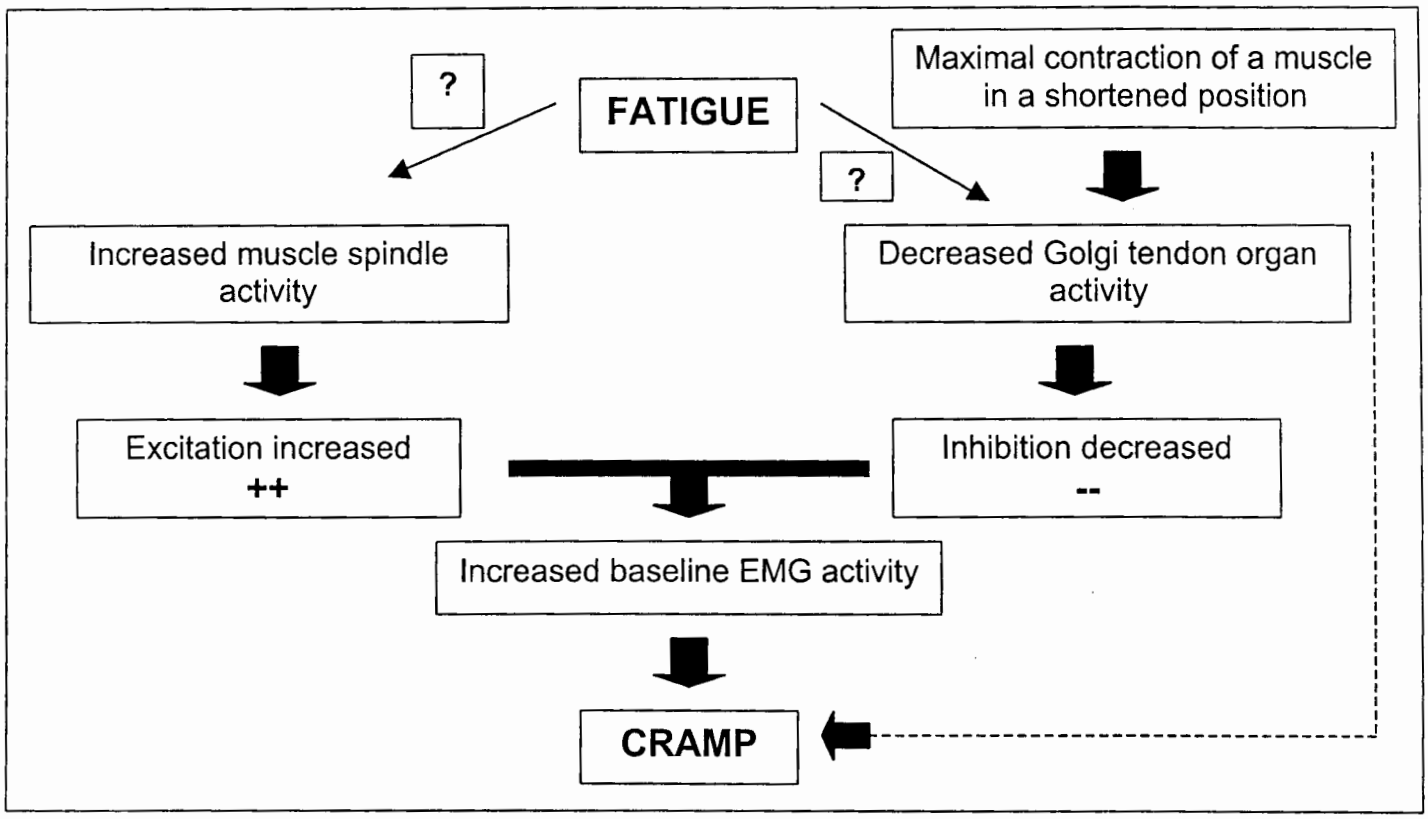

Figure 2.1 Diagrammatic representation of the postulated abnormal spinal control of motor neuron function during cramping after fatiguing exercise [70].

Unlike the existing theories for the aetiology of EAMC, this novel hypothesis for the aetiology of EAMC mentioned above is based on empirical evidence from epidemiological studies, animal experimental data on spinal reflex activity during fatigue and EMG data recorded during bouts of acute cramping after fatiguing exercise [69].

This hypothesis postulates that EAMC is associated with muscle fatigue that alters the alpha motor neuron control at the spinal level through abnormal reflex activity of the peripheral receptors [70]. It offers a plausible explanation for the aetiology of muscle cramping that affects so many exercising athletes and as such warrants further investigation.

This novel hypothesis for the aetiology of EAMC suggests that the EMG activity of cramping athletes would increase as muscle fatigue progressed and the control of the alpha motor neuron at the spinal level deteriorated. Although the EMG activity during spontaneous cramping as well as during recovery from cramping has been recorded, no study has recorded the EMG activity of athletes prone to cramping during progressive fatiguing exercise. A focus of this thesis will therefore be to compare the EMG activity of cramping and control athletes before, during and after a fatiguing bout of exercise.

2.11 Conclusion

EAMC is a condition that has not been thoroughly investigated. A recent PubMed Internet search for Exercise Associated Muscle Cramps revealed only eleven studies relevant to this condition and only one study that dealt with EAMC and Ironman triathletes specifically. Furthermore, it is apparent from a critical analysis of the literature that is available regarding EAMC that the exact aetiology and pathogenesis of muscle cramping remains a poorly understood and contentious issue and one that needs further investigation.

CHAPTER 3: THE EPIDEMIOLOGY OF EXERCISE ASSOCIATED MUSCLE CRAMPS (EAMC) IN IRONMAN TRIATHLETES

3.1 Abstract

Objectives: To investigate the nature and prevalence of Exercise Associated Muscle Cramping (EAMC) in a group of Ironman triathletes with a past history of EAMC and to compare these triathletes to a group of non-cramping triathletes. **Subjects:** All 799 triathletes who registered for the 2001 South African Ironman Triathlon were considered potential subjects. **Methods:** All prospective participants were sent a detailed questionnaire to complete and return at registration. The response rate was 95.1% (n=760). Sixty eight and a half percent of triathletes who responded to the cramping section of the questionnaire (289/422) reported a past history of EAMC and formed the 'cramp-history' (CH, n=289) group while 31.5% of triathletes (133/422) reported no previous history of EAMC and formed the 'non-cramping' group (NC, n=133). The CH group completed a specific section of the questionnaire detailing their cramping history (incidence of cramping, cramping severity, family history of cramping, factors associated with cramping). Total race times were recorded from both groups. **Results:** A significantly greater number ($p<0.01$) of the CH group cramped during running (37.1%) compared with swimming (28.9%) and cycling (15.6%), and during racing (46.1%) compared with training (18.8%). The majority of the CH group (56.5%) reported cramping in the final quarter of a race. The majority of the CH group rated their cramps as mild (77.5%), 17.4% as moderate and 3.6% as severe. Thirty eight and a half percent of the severe crampers had a family history of EAMC compared to 35.4% of the moderate and 25.2% of the mild crampers. The majority (58.4%) of the CH group associated muscle fatigue with EAMC, although electrolyte imbalances (42.9%) and dehydration (42.6%) were also perceived to be associated with cramps. The CH group had significantly slower ($p=0.02$) race times than the NC group (758.9 ± 91.5 min; 735.4 ± 98.7 min), had completed fewer Ironman Triathlons and reported less total training distance than the NC group. **Conclusions:** There is a high prevalence of EAMC amongst Ironman triathletes which is often associated with running, racing and muscle fatigue and occurs mostly in the last quarter of a race. A positive family history of cramping is associated with severe episodes of EAMC in some triathletes.

Key words: Muscle cramps, Ironman, prevalence, family history

3.2 Introduction

One of the most common problems experienced by both marathon runners and triathletes is Exercise Associated Muscle Cramps (EAMC) [34;45]. EAMC can be defined as 'painful, spasmodic, involuntary contractions of skeletal muscle during or immediately after physical exercise that is not associated with any pathophysiology' [67;70]. Despite the fact that as many as 50% of marathon runners and 67% of triathletes suffer from EAMC, most of the information available on the nature of this condition and the factors associated with it is based on anecdotal observations.

Most published texts have focussed almost exclusively on the role of dehydration and serum electrolyte abnormalities in the aetiology of muscle cramping [15;30;51;55;64;78]. Recent data indicate, however, that there is no association between EAMC and hydration status or abnormalities in serum electrolyte concentrations [49;58]. There have been very few studies that have investigated alternative aetiologies for EAMC.

In a recent epidemiological study on EAMC in 1 300 marathon runners some of the risk factors associated with skeletal muscle cramping in this population were identified [45]. The risk factors for EAMC included older age, longer history of running, higher body mass index, shorter daily stretching time, irregular stretching habits and a positive family history of cramping [45]. In another study the prevalence of EAMC as well as factors associated with EAMC in both United States Triathlon Series participants and Ironman Triathlon participants was documented [34]. These two studies form the only published data available on the nature and prevalence of EAMC in athletes and triathletes.

Despite the high reported prevalence of Exercise Associated Muscle Cramping amongst triathletes, it is clear that EAMC has not been thoroughly researched in either triathletes or Ironman triathletes and not much is known about the factors associated with EAMC in triathletes or about the nature and prevalence of muscle cramping in these athletes.

3.3 Aim of the study

The aim of this study was to document the nature and prevalence of Exercise Associated Muscle Cramping (EAMC) in a group of Ironman triathletes with a past history of EAMC and to compare them to a group of triathletes with no previous history of cramping.

3.4 Methodology

3.4.1 Pre-registration

All 799 triathletes who registered for the 2001 South African Ironman Triathlon were considered potential subjects. All prospective participants were sent a detailed explanation of the study and an informed consent form (Appendix 1). The Research and Ethics Committee of the Faculty of Health Sciences, University of Cape Town, approved the study.

Prospective participants were also sent a questionnaire including personal details (contact details, age, height, weight, gender, nationality, health status), a training log (swimming, cycling and running training distance for the current year as well as the total training distance for the last five years) and Ironman running history (amount of completed Ironman Triathlons). A detailed section containing questions specifically on cramping history was also included in the questionnaire (Appendix 2). Triathletes were asked to complete the entire questionnaire and sign the written informed consent form before arriving at registration.

3.4.2 Registration

At registration 760 questionnaires (95.1% response) were returned to the principal investigator. The collected questionnaires were examined for any missing information and/or obvious errors. Any questionnaires with errors were not included in the data collection process. There were 467 questionnaires with a completed personal details section (61.5% response), 83 questionnaires with a completed training log section (10.9% response) and 188 questionnaires with a correctly completed Ironman race history section (24.7% response).

A total of 440 triathletes completed the cramping section of the questionnaire. Eighteen of these questionnaires had missing data and/or obvious errors and were not included for analysis. The remaining 422 questionnaires (55.5% response) were divided into two groups based on whether or not triathletes reported having a past history of Exercise Associated Muscle Cramps (EAMC) during or immediately after exercise.

Triathletes with a past history of EAMC formed the 'cramp-history' group (CH group, n=289) while those triathletes with no previous history of EAMC formed the 'non-cramping' group (NC group, n=133). Only the CH group were required to complete the cramping section of the questionnaire which asked detailed questions regarding the nature and prevalence of their past cramping experiences. The specific questions included in the cramping section of the questionnaire were as follows:

i) Frequency of cramping

Triathletes were asked to report if they usually cramped during swimming, cycling and/or running. Triathletes were further asked whether they cramped during racing, training or during both racing and training.

ii) Frequency of cramping episodes

This question asked triathletes to mark at what time point during or after a race they first experienced cramping. They could choose from the beginning of the race, from the first quarter of the race, from the second quarter of the race, from the third quarter of the race, from the last quarter of the race or after the race.

iii) Muscles prone to cramping

Triathletes were asked to report which of their muscles were most prone to cramping. They could choose between the quadriceps, hamstrings, calves and/or foot muscles.

iv) Duration and severity of cramping

Triathletes were asked to estimate the duration of an average cramping bout in minutes. They were also asked to subjectively rate the severity of their cramping episodes. They could choose between 'mild' (5 minutes and you are able to continue exercising), 'moderate' (10 minutes and you are able to continue exercising) or 'severe' (15 or more minutes and you have to stop exercising).

v) Family history of cramping

Triathletes were asked to report whether they had a positive family history of EAMC. The CH group triathletes with a positive family history of EAMC were further sub-divided according to their rating of perceived cramping severity (either 'mild', 'moderate' or 'severe'). The percentage of each individual sub-group ('mild', 'moderate' and severe') with a positive family history of EAMC was then determined.

vi) Health professionals consulted about cramping

Triathletes were asked to indicate which health professional (dietician, doctor, physiotherapist, sports scientist, coach, podiatrist and/or chiropractor) they had consulted regarding their cramping.

vii) Treatment options for cramping

Triathletes were asked to report which treatment (regular stretching, increased training, decreased training, magnesium supplementation, sodium supplementation, increased fluid intake during exercise, decreased fluid intake during exercise, adequate race preparation, muscle strengthening programme and massage therapy) they had had for their cramping. Triathletes were able to choose more than one option.

viii) Factors associated with cramping

Triathletes were asked to report which factors (previous muscle injury, overhydration, dehydration, muscle twitchiness before cramping, muscle fatigue before cramping, lack of regular stretching, overstretching, increasing training load, lack of nutritional supplements, genetics, lack of electrolytes, lack of fitness and hot weather) they perceived to be associated with their cramping. Triathletes were able to choose more than one option.

3.4.3 Race finish

All race finishers reported to the Medical Tent for a medical evaluation. Total race times from both the CH and NC groups were recorded at the race finish.

3.5 Statistical analysis

All statistical analyses were performed using Statistica Version 6.0 (© StatSoft, Inc., USA) on a personal computer. Parametric data are represented by the mean ± standard deviation. Categorical data are expressed as percentages (%) of the total number in each case. Independent t-tests were used to evaluate differences between groups (CH and NC). Pearson Chi-square analyses were used to identify significant differences in the categorical data. P-values less than 0.05 were considered significant.

3.6 Results

3.6.1 Epidemiology of EAMC in Ironman triathletes

Two hundred and eighty nine of the 422 Ironman triathletes at the 2001 South African Ironman who completed the cramping section of the questionnaire reported having a past history of Exercise Associated Muscle Cramps (EAMC) during or immediately after exercise. Prevalence can be defined as the 'overall proportion of a population who suffer from a disease' [20]. The lifetime prevalence of EAMC can thus be defined as the number of athletes who have ever experienced EAMC at some time in their athletic career. The lifetime prevalence of EAMC in Ironman triathletes was 68.5%.

3.6.2 Characteristics of EAMC in Ironman triathletes

i) Frequency of cramping

Significantly more (p=0.01) of the CH group (n=289) experienced cramping during running (37.1%) than during swimming (28.9%) or cycling (15.6%). A large percent (14.8%) of the CH group reported cramping in two of the three activities while 3.2% reported cramping in all three activities. There was also a significantly greater frequency of the CH group (p=0.01) who experienced cramping during racing (46.1%) than during training (18.8%). Thirty four percent of the CH group reported cramping during both racing and training.

ii) Frequency of cramping episodes

The frequency of reported cramping episodes in the first, second, third and last quarter of a race as well as after a race in the CH group (n=289) is presented in Figure 3.1.

The majority of the CH group reported cramping in the last quarter of a race (56.5%). The percentage of triathletes who reported cramping in the last quarter of a race was significantly higher (p=0.01) than the percentage that cramped during the first (7.7%), second (5.3%) and third (30.6%) quarters of a race and was also significantly higher (p=0.01) than the percentage that cramped after (3.5%) a race. The percentage of triathletes who reported cramping in the third quarter of a race was also significantly higher (p=0.01) than the percentage that cramped during the other stages (1/4, 2/4, last quarter and after a race).

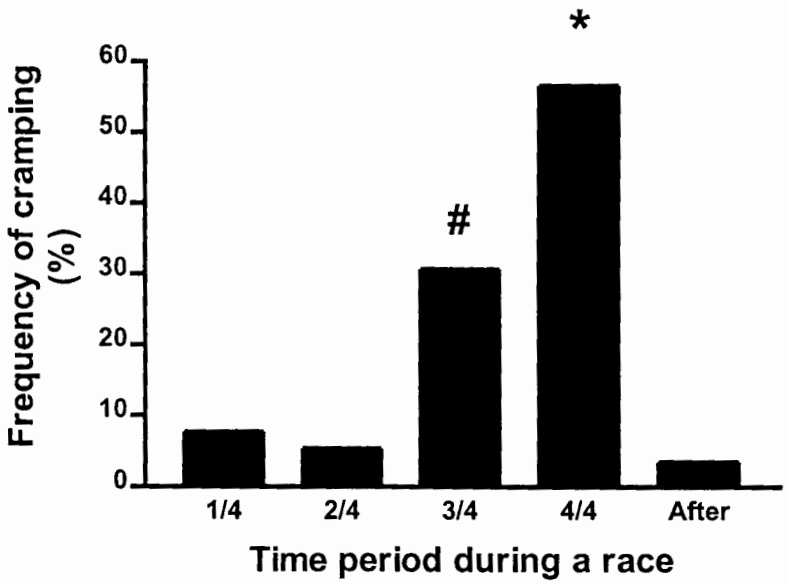

Figure 3.1 Frequency of reported cramping episodes in the CH group (n=289) during and after a race. Data are presented as a percentage (%) of the total.
* p<0.01 versus 1/4, 2/4, 3/4 and after a race
# p<0.01 versus 1/4, 2/4, last quarter and after a race

iii) Muscles prone to cramping

The majority (67.2%) of the CH group (n=289) reported cramping in the calf muscles. The hamstrings were the second most prone to cramping (45.5%), with the quadriceps third (34.7%) and the foot muscles least prone to cramping (32.4%). These differences were not significant (p=0.64).

iv) Duration and severity of cramping

The mean duration of cramping episodes in the CH group (n=289) was 4.3 ± 9.7 minutes. The majority (77.5%) of triathletes with a past history of cramping rated their cramping severity as 'mild' (5 minutes and you are able to continue exercising). This was significantly higher (p=0.01) than the 17.4% who rated their cramping as 'moderate' (10 minutes and you are able to continue exercising) and significantly higher (p=0.01) than the small 3.6% who rated their cramping as 'severe' (15 or more minutes and you have to stop exercising).

v) Family history of cramping

A relatively large percent (25.9%) of the CH group had a positive family history of Exercise Associated Muscle Cramping (EAMC).

The percentage of each cramping severity sub-group ('mild', 'moderate' or 'severe') who reported a positive family history of EAMC (n=272) is presented in Figure 3.2.

Thirty eight and a half percent of the 'severe' group (n=10) reported a positive family history of EAMC as opposed to 35.4% of the 'moderate' sub-group (n=48) and 25.2% of the 'mild' sub-group (n=214). These values were not significantly different (p=0.84). Seventeen triathletes in the CH group did not rate their cramping severity.

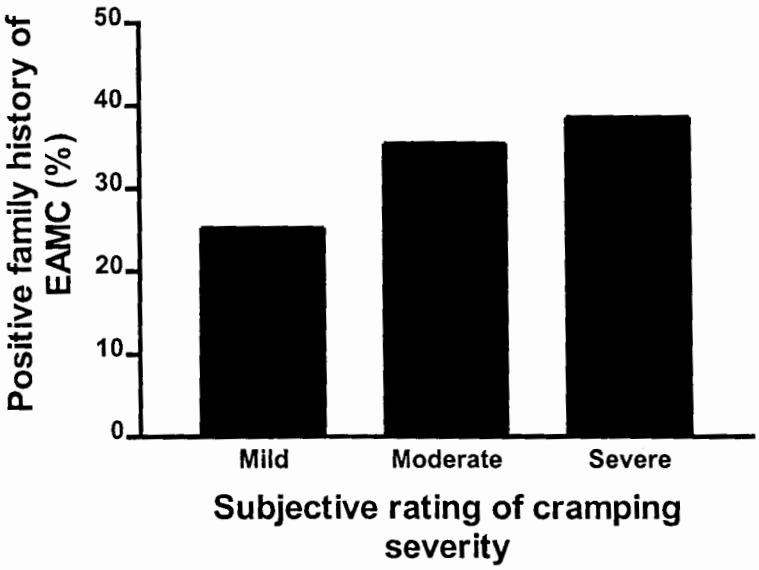

Figure 3.2 Percentage of the 'mild' (n=214), 'moderate' (n=48) and 'severe' (n=10) sub-groups of the CH group with a positive family history of EAMC. Data are presented as a percentage (%) of each individual sub-group's total.

vi) Health professionals consulted about cramping

Only 31.9% of the triathletes with a past history of cramping (n=289) sought professional advice for their cramping. Of that physiotherapists were consulted most often (12.8%) followed by doctors (6.9%). Sport scientists, coaches and dieticians were each consulted by an average of 3.5% of the CH group.

vii) Treatment options for cramping

The treatment options chosen by the CH group (n=289) are presented in Table 3.1. The majority of triathletes (51.5%) chose 'regular stretching' as a treatment for cramping. Magnesium supplements and adequate race preparation were also reported as regular treatment options (35.1% and 32.8% respectively). Massage therapy, increased fluid intake, increased training, sodium supplementation and muscle strengthening were chosen by 25.9%, 24.6%, 21.3%, 20.7% and 19.0% of the CH group respectively. Few chose decreased training and decreased fluid intake as treatment options (10.8% and 2.9% respectively).

Table 3.1 Treatment options for cramping chosen by the CH group (n=289). Results are presented in descending order and as percentages (%) of the total (n=289).

Treatment option	**% of CH group (n=289)**
Regular stretching	51.5
Magnesium supplements	35.1
Adequate race preparation	32.8
Massage therapy	25.9
Increased fluid intake	24.6
Increased training	21.3
Sodium supplements	20.7
Muscle strengthening	19.0
Decreased training	10.8
Decreased fluid intake	2.9

viii) Factors associated with cramping

The factors associated with cramping as perceived by the CH group (n=289) are presented in Table 3.2. The majority (58.4%) of the group indicated that muscle fatigue was associated with cramping. A large percentage of the group (50.8%) also associated a lack of stretching with cramping. A lack of electrolytes, muscle 'twitchiness', dehydration, lack of fitness, increased training load, hot weather and lack of supplements were chosen by an average of 40% of the CH group to be associated with muscle cramping. A relatively small percent (19.3%) indicated that a previous muscle injury was associated with cramping, 17.4% associated genetics with cramping and 15.4% reported overhydration being associated with EAMC. Only 9.8% suggested an association between overstretching and cramping.

Table 3.2 Factors associated with cramping as perceived by the CH group (n=289). Results are presented in descending order and as percentages (%) of the total (n=289).

Associated factor	**% of CH group (n=289)**
Muscle fatigue	58.4
Lack of stretching	50.8
Lack of electrolytes	42.9
Muscle twitchiness	42.6
Dehydration	42.6
Lack of fitness	41.3
Increased training load	39.3
Hot weather	36.4
Lack of supplements	36.1
Previous muscle injury	19.3
Genetics	17.4
Overhydration	15.4
Overstretching	9.8

3.6.3 Ironman race data

The CH group (n=269) were significantly slower in the present study (p=0.02) than the NC group (n=133) with an average total race time of 758.9 ± 91.5 minutes for the CH group in comparison with 735.4 ± 98.7 minutes for the NC group. The CH group (n=120) reported completing fewer Ironman Triathlons (2.9 ± 3.6 Ironman Triathlons) than the NC group (n=68) (4.1 ± 4.9 Ironman Triathlons), although this difference was not significant (p=0.07).

3.6.4 Training history

The total training distance (km) for the last year and well as the total training distance (km) for the last 5 years for swimming, cycling and running for the CH (n=63) and NC (n=20) groups is presented in Table 3.3.

There were no significant differences between the two groups for training distance, although the CH group's (n=63) training distance (km) was less than the NC group's (n=20) training distance (km) in all but one case. Only for the last year's swimming training distance (km) did the CH group (n=63) have a larger average training distance (km) than the NC (n=20) group.

Table 3.3 Training distance (km) for the last year as well as for the past five years for swimming, cycling and running for the CH (n=63) and NC (n=20) groups (mean ± standard deviation).

Variable	CH group (n=63)	NC group (n=20)	p value
Swim last year (km)	219.3 ± 121.2	192.0 ± 120.3	0.4
Swim 5 years (km)	453.5 ± 541.3	473.4 ± 534.7	0.9
Cycle last year (km)	6 134.3 ± 3 915.6	7 137.0 ± 4 723.5	0.2
Cycle 5 years (km)	12 179.9 ± 13 848.2	16 557.0 ± 16 648.3	0.3
Run last year (km)	1 780.2 ± 953.5	2 239.2 ± 1 010.9	0.1
Run 5 years (km)	4 814.1 ± 4 990.4	6 931.2 ± 5 321.6	0.1

3.7 Discussion

The main findings of this study are that there is a high lifetime prevalence of Exercise Associated Muscle Cramping (EAMC) in Ironman triathletes. Furthermore, EAMC in this group of triathletes is associated with running, racing and muscle fatigue and occurs mostly in the last quarter of a race. A positive family history of EAMC may be associated with the severe and debilitating form of cramping experienced by some triathletes during exercise.

An epidemiological study on 1 300 marathon runners reported a lifetime prevalence of EAMC of 38.8% [45]. Another study reported that 67% of 2 438 United States Triathlon Series participants complained of EAMC at some stage in their career [34]. The present study found that a similar high percentage (68.5%) of Ironman triathletes had a lifetime prevalence of EAMC.

The prevalence of cramping during running shown in this study has been reported previously in a Triathlon [34]. Exhausting stretch-shortening exercise has been reported to result in ultra-structural muscle damage and may lead to the development of muscle soreness and inflammation [57;59]. The continuous stretch-shortening cycle muscle actions and associated eccentric muscle contractions of the lower limbs inherent with running may explain the prevalence of EAMC in these muscles during running as opposed to during swimming or cycling.

The association between running (especially long-distance running) and fatigue is well accepted in the literature and recently it has been suggested that cramping may also be associated with muscle fatigue [24;26;70;83]. Muscle fatigue is known to alter the functioning of the peripheral receptors by increasing the excitatory muscle spindle activity and decreasing the inhibitory Golgi tendon organ activity, although the exact mechanism responsible for this alteration in reflex activity is not yet known [31;56].

A novel hypothesis for the aetiology of EAMC has been proposed which suggests that EAMC is the result of an abnormality of alpha motor neuron control at the spinal level [70]. It is postulated that a combination of the increased excitatory activity of the muscle spindle and a reduced inhibitory effect of the Golgi tendon organ with muscle fatigue would result in a sustained alpha motor neuron activity such as that seen in the increased baseline EMG activity between bouts of acute cramping [58]. This hypothesis may explain the higher incidence of cramping during running which is also the final stage of a Triathlon when fatigue starts to have a major effect on performance [36].

The large number of the CH group who reported cramping in two or more activities has not been reported previously but may also have been associated with muscle fatigue. The relative Ironman race inexperience of the CH group (who reported participating in an average of 2.9 ± 3.6 Ironman Triathlons) could have led to an inadequate race preparation which in turn could have caused problems with pacing during the race. Both inadequate race preparation before the race and incorrect pacing through the race could have resulted in the premature onset of muscle fatigue which may explain the prevalence of cramping in more than one activity of a race.

The prevalence of cramping during racing found in this study is in agreement with another study which reported an association between high-intensity running (racing) and cramping in an epidemiological study of 1 300 marathon runners [45]. The pressure to perform, and to perform within a certain time frame causes many athletes to push themselves either too hard or too hard too soon [59]. This may lead to an earlier onset of fatigue during racing as opposed to training and may explain the prevalence of cramping during racing reported in this population.

The present study also found that a large percentage of the CH group cramped during both racing and training. A large number (39.3%) of the CH group indicated that an increase in training load was associated with cramping. This may be the result of the fatiguing nature of the training required to prepare for such an Ultra-endurance event which demands a substantial amount of training distance in all three disciplines that make up a Triathlon. Such intensive training will invariably result in fatigue and may explain the occurrence of cramping during training for Ultra-endurance events.

Numerous studies have reported that cramping is more prevalent in the later stages of a race; in marathons it has been reported that cramping tends to occur after 30km of the race and a study of Ironman Triathlons reported that 63% of triathletes cramped in the late run [34;45;49]. The present study also found a significant prevalence of reported cramping in the last quarter of a race compared to the earlier stages of a race and showed that as a race progressed, the incidence of cramping also increased. This tends to support the hypothesis that fatigue may be associated with episodes of EAMC and further suggests that the number of cramping episodes tends to increase progressively with fatigue [70].

One study reported an increased baseline EMG activity in cramping runners immediately after completion of an Ultra-endurance run which normalised after 60 minutes recovery [58]. This suggests that the heightened muscle excitability evident during acute cramping may continue into recovery and may explain the finding in the present study that a small percentage of triathletes reported cramping after a race.

The calf muscle was found to be the muscle most prone to cramping which is supported by studies that have shown that muscles that span across two joints are most likely to cramp [45;58]. This may be due to the fact that these muscles are most often contracted in the shortened position, with less tension being produced during contraction and thus less Golgi tendon activity and therefore less inhibition of the alpha motor neurons [70]. This finding offers additional support for the novel hypothesis which suggests that an altered reflex activity may be associated with cramping [70].

The majority of triathletes in the present study described their cramping episodes as 'mild' or 'moderate', lasting between 5 and 10 minutes but not causing them to have to stop exercising. This is in contrast with the relatively small percentage of 'severe' crampers whose cramping reportedly forced them to stop exercising. These findings suggest that there may be two categories of crampers-those whose cramping does not affect their ability to exercise and those whose cramping limits their performance and interferes with their exercise. This suggestion is supported by another study which investigated the nature and prevalence of cramping in triathletes and concluded that there might be a specific group of athletes who are particularly susceptible to cramping [34].

The suggestion of a separate category of crampers is further supported by the large percentage of 'severe' crampers in this study who reported a positive family history of EAMC. This introduces the possibility that there may be a genetic component associated with severe cramping in certain athletes. This link between family history and cramping has been reported in another study which found that 19% of marathon runners who experience EAMC had a family history of muscle cramping [46]. Although many studies have researched a genetic basis for cramping associated with some type of muscle pathology, there has been no published research to date of a genetic link to EAMC specifically [4;19;39;79]. The findings of the present study suggest that a positive family history of muscle cramping may be associated with the severe and debilitating form of cramping experienced by some athletes during exercise, although more research is required in this area before any conclusive association can be made.

Only a small percentage of triathletes reported consulting a health professional for their cramping. This may have been because of the large percentage of triathletes whose cramping was described as 'mild' or 'moderate' and did not interfere with their exercise or this may have been due to the fact that there is no known definitive treatment for cramping and a lot of treatment remedies are anecdotal.

The majority of the CH group reported that they chose a regular stretching routine to avoid cramps and that a lack of stretching was associated with cramping. Passive stretching is the most common therapy to relieve cramping as it increases the tension in a muscle, thereby increasing the Golgi tendon organ's inhibitory activity which relieves the cramp [3;12;27;56;60;71]. The effectiveness of passive stretching in treating EAMC offers further support for the hypothesis that abnormal spinal reflex activity is associated with EAMC [70]. A large percentage of the CH group also listed adequate race preparation, increased training and muscle strengthening as ways to avoid cramps and that muscle fatigue and a lack of fitness were associated with EAMC. These perceptions support the hypothesis of an association between muscle fatigue and EAMC [70].

A small percentage of the CH group also mentioned a decrease in training as a treatment option for cramping. These athletes may have been training too hard and been 'over-trained' in which case a decrease in training would be necessary to prevent injury. A definite explanation is difficult, however, due to a lack of further information regarding specific training quantity and training habits.

A large percentage of the CH group reported that a lack of electrolytes and supplements as well as dehydration and hot weather were associated with EAMC and that magnesium and sodium supplements, as well as increased fluid intake, helped their cramping. This is despite a number of recent studies that have shown there is no association between serum electrolytes or percent dehydration and EAMC [49;58].

Although it appears that the focus is slowly moving away from electrolyte imbalances, dehydration and heat being the primary causes of EAMC, there are still many crampers who believe that taking supplements and drinking fluid will prevent cramps. There are also still many published texts that recommend salty food and electrolyte rich energy drinks to prevent muscle cramping [15;30]. The findings of the present study show that there is a need for greater education of athletes and further investigation into the causes and factors associated with EAMC.

The significantly slower finishing times of the CH group may be related to the relative Ironman race inexperience of this group who had completed fewer Ironman Triathlons than the NC group (although the difference in racing experience was not statistically significant). The slower race times in this group may also be explained by insufficient race preparation. Although there were no significant differences in training distance between the two groups, the CH group's training distance was consistently less than that of the NC group for all activities. Both racing inexperience and insufficient race preparation may result in premature fatigue, both of which could result in slower race times.

This cross-sectional descriptive study has some limitations. The data obtained through such a study design is purely of a descriptive nature and does not imply any cause-and-effect relationships. There is also a selective bias inherent in any questionnaire that there will be a greater response from those for whom the questions are most pertinent (such as the triathletes with a past history of cramping and the cramping questionnaire). Selective bias can also occur in the choice and wording of questions included by the investigator, although in this study certain parts of the cramping questionnaire had been validated in a previous running study [45].

A further limitation associated with any questionnaire is that the quality of the results is dependant on how comprehensively subjects answer the questions. Missing and/or incomplete data sets are frequent occurrences as are misinterpretations, misunderstandings and inappropriate answers to questions. A questionnaire such as the one used in this study provides a large amount of general information but not many details which limits the conclusions that can be drawn from the data. This questionnaire did, however, reveal some interesting findings which may serve as a starting point for future research.

The results of this study indicate that there is a high lifetime prevalence of EAMC in Ironman triathletes. EAMC is prevalent in running, racing and during the last stages of a race and may also be associated with muscle fatigue. The results of this study further suggest that a positive family history of muscle cramping may be associated with the severe and debilitating muscle cramps experienced by some athletes during exercise. Finally it appears that there is a need for triathletes in general, but cramping triathletes in particular, to be educated regarding EAMC and the factors associated with cramping during exercise.

CHAPTER 4: SERUM ELECTROLYTE CONCENTRATIONS AND BASELINE ELECTROMYOGRAPHY (EMG) OF CRAMPING AND CONTROL IRONMAN TRIATHLETES

4.1 Abstract

Objectives: To compare the serum electrolyte concentrations in cramping and control Ironman triathletes and to record the baseline electromyography (EMG) of cramping Ironman triathletes during recovery. **Subjects:** Triathletes presenting with acute cramping after the 2000 South African Ironman Triathlon (n=11) and non-cramping matched controls (n=9). **Methods:** All subjects were weighed during registration and immediately post-race. Triathletes suffering from acute Exercise Associated Muscle Cramps (EAMC) after the race formed the cramping group (CR, n=11). Non-cramping triathletes matched for race finishing time and body mass formed the control group (CON, n=9). Baseline EMG was recorded from the non-active control muscle (triceps) and the most severely cramping lower limb muscle (quadriceps, hamstring or calf) of the CR group during periods of remission from cramping. EMG (mV) was recorded at the beginning of every minute for a 10-minute period during recovery. Blood samples were drawn from both the CR and CON groups during recovery for the analysis of plasma magnesium, glucose, sodium, potassium and chloride concentrations. Haemoglobin and haematocrit were also measured. **Results:** There were no significant differences between the CR and CON groups for registration or post-race body mass or percent (%) weight loss. Post-race sodium concentration was significantly higher ($p=0.01$) in the CON group than the CR group (142.7 ± 3.0 versus 139.5 ± 1.7 $mMol.L^{-1}$) but this was not clinically significant. There were no significant differences between the two groups for post-race serum electrolytes, glucose, haemoglobin concentrations or haematocrits. EMG (mV) of the cramping muscles was significantly higher ($p=0.04$, 0.04, 0.002, 0.05 respectively) than that of the control muscles of the CR group at 0, 3, 4 and 5 minutes of the 10-minute period. There were no significant differences over time. Post-race potassium concentration was significantly correlated with EMG activity ($r=0.7$) whereas post-race plasma magnesium and sodium as well as % weight loss were not. **Conclusions:** EAMC is not associated with % dehydration or clinically significant disturbances in serum electrolyte concentrations. The increased baseline EMG activity of cramping muscles may reflect a heightened muscle activity associated with muscle fatigue.

Key words: Muscle cramps, Ironman, dehydration, serum electrolytes, EMG

4.2 Introduction

The popularity of the Triathlon (a race involving consecutive swimming, bicycling and running) is continuously increasing amongst athletes [28;36]. The Ironman Triathlon is an ultra-endurance event which includes a 3.9km swim, 180.2km bicycle ride and 42.2km run and also enjoys a large following.

Amongst the more common medical problems encountered at the Ironman Triathlons are skeletal muscle cramps [34]. Exercise Associated Muscle Cramping (EAMC) can be defined as a 'painful spasmodic involuntary contraction of skeletal muscle that occurs during or immediately after exercise' [70]. EAMC accounts for between 6% and 20% of the medical diagnoses encountered at Ironman Triathlon events [28;36]. Despite the high incidence of EAMC at ultra-endurance events, there has only been one published study on EAMC and triathletes which documented the nature and prevalence of muscle cramping in United States Triathlon Series participants [34]. It is clear that EAMC has not been thoroughly researched in Ironman triathletes.

A popular aetiology for EAMC still entertained by many athletes and coaches is that Exercise Associated Muscle Cramping (EAMC) is caused by dehydration and imbalances in serum electrolyte concentrations [14;15;30;51;55;64;78]. Recent studies show, however, that there is little support for an association between EAMC and dehydration and abnormalities in serum electrolyte concentrations in marathon runners [49;58]. Despite this recent evidence, the 'serum electrolyte' theory remains a contentious issue. Furthermore, although the serum electrolyte concentrations of cramping runners have been reported, no study has investigated the serum electrolyte concentrations of cramping Ironman triathletes.

Although the EMG activity of cramping muscles in a laboratory setting has been recorded often, only one study has recorded the baseline EMG activity (the electrical activity in a muscle during remission from a bout of acute cramping) of cramping and control runners after a marathon. This study reported that baseline EMG activity decreased significantly in the cramping group compared with the control group during the 60-minute recovery period [58]. There are no data on the baseline EMG activity of cramping Ironman triathletes after exercise and no study has yet compared the EMG activity of a non-exercising control muscle to the baseline EMG data of a cramping muscle after exercise.

4.3 Aims of the study

The aims of this study were to compare the serum electrolyte concentrations in cramping and control athletes following an Ironman Triathlon and to investigate the baseline electromyographic (EMG) status of the cramping and non-exercising control muscles of cramping Ironman triathletes during recovery from an Ironman Triathlon.

4.4 Methodology

4.4.1 Race registration

All triathletes who registered for the 2000 South African Ironman were considered potential subjects. All triathletes (irrespective of their cramping history) were informed of the study and the testing procedures at registration. Triathletes who volunteered to participate were asked to sign a written informed consent form (Appendix 3). The Research and Ethics Committee of the Faculty of Health Sciences, University of Cape Town, South Africa, approved the study.

Subjects were weighed during registration using calibrated Adamlab JPS electronic scales (Scales Inc, Brackenfell, SA) that were placed on hard, flat surfaces. All subjects were weighed in standardised clothing and shoes. Body weight was corrected for this clothing (250g) and calculated as net body mass. All other data was collected at the race finish.

4.4.2 Race finish

All race finishers were required to report to the Medical Tent for further testing and a medical evaluation. Eleven triathletes suffered from Exercise Associated Muscle Cramps (EAMC) during and/or after the race and were escorted to a specific area of the medical tent for investigation and medical treatment if necessary. They formed the 'cramping group' (CR, n=11). The diagnostic criteria for EAMC were acute muscle pain, a visibly contracted muscle (with or without fasciculation) and no history of an acute muscle tear. Non-cramping athletes were matched for gender, race finishing time and body mass. They formed the control group (CON, n=9). Due to time constraints in getting controls to the testing area, two cramping subjects did not have matched control subjects.

All subjects were weighed on entering the medical tent (Adamlab JPS electronic scales, Scales Inc, Brackenfell, SA) and their race times recorded. Triathletes were weighed in their running kit, but without shoes. Body mass was corrected for running kit (200g). Percent (%) weight loss was calculated as the difference between the initial registration body mass and final body mass divided by the initial registration mass and expressed as a percentage. This calculation was based on the assumption that percentage body mass change would approximate hydration status.

Cramping triathletes were asked to lie supine on an examination plinth and were examined by a medical doctor. Electromyographic (EMG) testing began immediately on all crampers unless medical treatment was required. Crampers requiring medical attention were first treated (mainly by active and passive stretching) until cramp- and pain-free and then tested. The control subjects were also asked to lie supine on examination plinths until their blood sample had been taken.

The South African Weather Bureau provided the weather condition details on race day.

4.4.3 Electromyography (EMG)

EMG testing of the CR group consisted of placing two disposable pre-gelled EMG electrodes (The Prometheus Group ^{TM}, USA) onto the muscle bellies of the right triceps brachii muscle (the non-active control muscle) and the most severely cramping lower limb muscle (either the quadriceps, hamstring or calf muscle). The majority of subjects experienced cramping in both legs that prevented baseline EMG activity from being recorded in an 'opposite' active muscle. If the direct site of cramping was no longer obvious, the quadriceps electrode was placed obliquely over the right vastus medialis oblique muscle belly five centimetres above the superior pole of the patella, the hamstring electrode over the right biceps femoris muscle at the site of greatest thigh girth and the calf electrode over the right gastrocnemius muscle at the site of greatest calf circumference. In cases where the site of cramping was still visible, the electrode was placed directly over the cramping area of the muscle in question.

The skin over each site was prepared by shaving the hair with a disposable razor (Dahlhausen, Koln, Germany) in a three-centimetre radius around each site. Any oil and dirt was removed with alcoholic swabs and cotton gauze swabs (Naturil, Smith and Nephew, South Africa). The two electrodes were attached to a portable EMG machine (Pathway MR 20, The Prometheus Group ^{TM}, USA) which displayed the electrical activity of the muscle as a millivolt (mV) reading.

Raw baseline EMG data from both the cramping and control muscles of the CR group were recorded manually onto a prepared data collection form (Appendix 4) from the millivolt reading on the display. Baseline EMG data is defined as the electrical activity in a muscle during remission from a bout of acute cramping. Subjects were asked to lie as still as possible for the duration of the recording. Data were recorded at the beginning of every minute for 10 minutes, starting as soon as possible after subjects were supine on the examination plinths.

4.4.4 Post-race serum electrolytes, glucose, haemoglobin concentrations and haematocrit

A 4.5ml post-race venous blood sample was drawn into lithium heparin and/or sodium fluoride and potassium oxalate vacutainer tubes from the right forearm antecubital vein from both the CR and CON group in the supine position by a medical doctor. The CON group's blood sample was taken as soon as they were supine on the examination plinths in the testing area of the medical tent. The CR group's blood sample was taken during the first break in EMG recording to prevent any interference in the triceps EMG recording. Plasma magnesium, sodium, potassium, chloride and glucose concentrations were determined from the blood samples. Blood haemoglobin concentration and haematocrit were also calculated.

Haematocrit was measured in triplicate as the packed cell volume using heparinised micro capillary tubes. The micro capillary tubes were centrifuged in a Hawksley micro-haematocrit centrifuge for 5 minutes and read with a Hawksley micro-haematocrit reader.

After the determination of haematocrit, the blood samples were centrifuged at 3 000 rpm for 10 minutes and stored at -20°C until later analysed. Plasma magnesium concentration was measured by atomic absorption spectrophotometry (Varian AA1275 atomic absorption spectrophotometer-Varian Techtron Melbourne Australia) after dilution of the plasma 1:50 with 0.1% Lanthanum chloride solution containing ~60 $mMol.L^{-1}$ HCl. Glucose concentration was analysed using a Beckman Glucose Analyser 2 (Beckman Instruments, Inc., California; USA). Plasma sodium, potassium and chloride concentrations were analysed using an EasyLyte PLUS Na/K/Cl Analyser (Medica Corporation, Bedford, MA). A standard cyanmethaemoglobin procedure using a 1 in 500 dilution of heparinised whole blood with Drabkins solution was used to measure blood haemoglobin concentrations [10].

4.4.5 Medical discharge

Once all testing had been completed a medical doctor discharged those subjects with no further symptoms. Discharge criteria were no further cramps and no pain at rest or with movement.

4.5 Statistical analysis

All statistical analyses were performed using Statistica Version 6.0 (© StatSoft, Inc., USA) on a personal computer. All data are represented by the mean ± standard deviation. Independent t-tests were used to evaluate differences between groups (CR and CON) and muscles (cramping and non-active control). A two-way analysis of variance (ANOVA) for repeated measures was performed on all data to determine whether there was an interaction effect between group and time. Significant differences ($p<0.05$) between groups or over time were further analysed by means of a Tukey post-hoc analysis to determine the site of significance. P-values less than 0.05 were considered significant.

Correlations were performed on the cramping muscle's baseline EMG data for minute 10 of recovery and the cramping subjects' post-race plasma magnesium, sodium and potassium concentrations, as well as % weight loss data. The correlation analysis calculated the correlation coefficient (r) for each of the four relationships mentioned above. The correlation coefficient represents the strength of the relationship between the chosen variables with a correlation coefficient of $r = 0.9$ being an acceptable value for predicting a relationship between two variables [82].

4.6 Results

4.6.1 Descriptive data

On race day the average temperature was 20.5 ^{O}C and the maximum temperature was 23.9 ^{O}C. The relative humidity averaged 68% for the day, with the average wind speed being 4.6 m.sec $^{-1}$.

The average age (years), registration and post-race body mass (kg), % weight loss and total race time (minutes) from the CR and CON group are presented in Table 4.1. There were no significant differences between the two groups for these variables.

Table 4.1 Age, registration and post-race body mass, % weight loss and total race time in the CR (n=11) and CON (n=9) groups (mean ± standard deviation).

Variable	CR group (n=11)	CON group (n=9)	p value
Age (years)	33.5 ± 8.8	35.4 ± 8.1	0.6
Registration body mass (kg)	79.1 ± 5.9	77.7 ± 6.4	0.6
Post-race body mass (kg)	76.3 ± 5.6	74.6 ± 6.5	0.5
% Weight loss	3.4 ± 1.3	3.9 ± 2.0	0.5
Total race time (min)	660.8 ± 77.9	685.7 ± 48.5	0.4

4.6.2 Baseline electromyographic (EMG) data

Baseline EMG (mV) activity (n=11) for both the non-active control (triceps) and cramping (calf, quadriceps or hamstring) muscles of the CR group recorded from immediate recovery (Imm.Rec.) for 10 minutes during recovery is presented in Figure 4.1.

Baseline EMG activity was significantly greater (p=0.04, 0.04, 0.002, 0.05 respectively) in the cramping compared with the control muscle at 0, 3, 4 and 5 minutes. There were no significant differences over time.

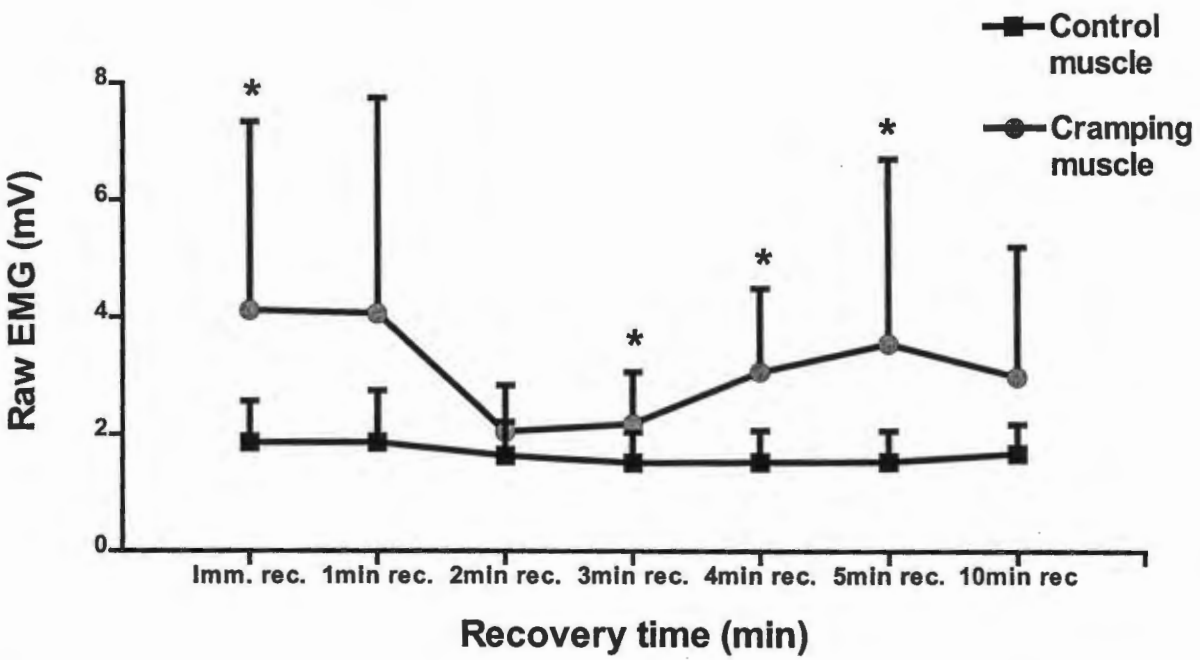

Figure 4.1 Baseline EMG (mV) for both the non-active control (n=11) and cramping (n=11) muscles of the CR group during 10 minutes of recovery. Data presented as mean ± standard deviation.
* Indicates a statistical significance of $p<0.05$.

4.6.3 Post-race serum electrolytes, glucose, haemoglobin concentrations and haematocrit

The plasma magnesium, glucose, plasma sodium, potassium and chloride concentrations ($mMol.L^{-1}$) as well as blood haemoglobin concentration (g/dl) and haematocrit (%) for both the CR (n=9) and CON (n=9) groups are presented in Table 4.2. Two of the cramping subjects refused to have their blood taken, thus the CR group's sample size for the blood analysis was 9 instead of 11.

The post-race sodium concentration was significantly higher (p=0.01) in the CON group compared with the CR group. There were no other significant differences between the two groups.

Table 4.2 Post-race plasma magnesium, glucose, plasma sodium, potassium and chloride concentrations, blood haemoglobin concentration and haematocrit in the CR (n=9) and CON (n=9) groups (mean ± standard deviation).

Variable	CR group (n=9)	CON group (n=9)	p value
Plasma magnesium ($mMol.L^{-1}$)	0.9 ± 0.2	0.8 ± 0.1	0.60
Glucose ($mMol.L^{-1}$)	7.6 ± 1.5	6.1 ± 1.5	0.07
Sodium ($mMol.L^{-1}$)	139.5 ± 1.7 *	142.7 ± 3.0	0.01
Potassium ($mMol.L^{-1}$)	4.4 ± 0.6	4.2 ± 0.5	0.50
Chloride ($mMol.L^{-1}$)	101.3 ± 2.6	103.9 ± 2.7	0.06
Haemoglobin (g/dl)	16.0 ± 0.9	15.9 ± 0.9	0.80
Haematocrit (%)	45.7 ± 3.4	45.1 ± 2.4	0.70

* Indicates a statistical significance of $p<0.05$ between the groups.

4.6.4 The relationship between baseline EMG, serum electrolytes and % weight loss

Various correlations were performed on the CR group's baseline EMG data (mV) from the cramping muscle for the last minute of the 10-minute period recorded. The correlation coefficients (r-values) obtained from these analyses are presented in Table 4.3.

There was a significant relationship (p=0.02) between the baseline EMG (mV) at minute 10 and post-race plasma potassium concentration (r = 0.7). The correlation between the baseline EMG (mV) at minute 10 and post-race plasma magnesium concentration ($mMol.L^{-1}$) showed an insignificant negative relationship however (r= -0.2). The relationship between the baseline EMG (mV) at minute 10 and post-race plasma sodium concentration ($mMol.L^{-1}$) was not significant with a correlation coefficient of r = 0.4. The negative correlation between baseline EMG (mV) at minute 10 and % weight loss was also not significant with a correlation coefficient of r = -0.4.

Table 4.3 Results from the correlations between the cramping muscle's 10-minute baseline EMG and plasma sodium and potassium concentrations and % weight loss. Data are presented as the correlation coefficient (r-value).

Correlated variable	10 minute EMG (n=9)	p value
Plasma magnesium ($mMol.L^{-1}$)	r =-0.2	0.50
Sodium ($mMol.L^{-1}$)	r = 0.4	0.30
Potassium ($mMol.L^{-1}$)	r = 0.7 *	0.02
% Weight loss	r = -0.4	0.30

* Indicates a significant correlation (p<0.05).

4.7 Discussion

The main findings of this study are that Ironman triathletes who suffer from Exercise Associated Muscle Cramping (EAMC) are not dehydrated and do not have clinically significant serum electrolyte abnormalities associated with acute muscle cramping. Furthermore, the results of the present study indicate that the baseline electromyographic (EMG) data of cramping muscles is higher and more variable than non-active control muscles during recovery from fatiguing exercise. These data appear to support the novel hypothesis that EAMC is accompanied by heightened muscle activity possibly associated with muscle fatigue.

There were no significant differences in % weight loss between the CR and CON groups in the present study. Crampers were therefore not dehydrated compared to matched controls. These results do not support the widely accepted theory that Exercise-Associated Muscle Cramping (EAMC) is related to hydration status [14;15;55;78]. The findings of the present study do, however, support recent data that suggest that cramping is not associated with dehydration in marathon runners [49;58]. A study investigating 162 Ironman triathletes also found no correlation between dehydration (% weight loss) and probability of cramping [34]. The results of the present study therefore add to the growing body of literature which shows a dissociation between dehydration and EAMC in athletes, irrespective of the type or duration of the activity.

Numerous studies have recorded an increased electromyographic (EMG) activity during an acute bout of induced and spontaneous cramping [60;67]. Only one study has focussed on baseline EMG activity in cramping subjects immediately after intensive exercise [58]. Baseline EMG is defined as the electrical activity in a muscle during remission from a bout of acute cramping. The results of the study on baseline EMG activity in cramping runners recorded a significant decrease in the cramping group's baseline EMG activity compared to the control group during 60 minutes recovery period from exercise [58].

There were no significant differences between the cramping and non-active control muscles over time in the present study. This is likely due to the relatively short 10-minute period during which EMG activity could be recorded. Subjects were reluctant to be tested for much longer than 10 minutes after the race and many of the participants were foreigners who had to return home the following day which prevented any follow-up testing.

The results of the present study did show, however, that the baseline EMG activity in the cramping muscle was consistently higher and more variable (as reflected by the large standard deviation) than that of the control muscle throughout the 10-minute period and was significantly higher than the non-active control muscle at 0, 3, 4 and 5 minutes. A novel hypothesis concerning the aetiology of EAMC suggests that muscle cramping is associated with muscle fatigue which alters the alpha motor neuron control at the spinal level, resulting in a heightened motor neuron activity which is seen as an increase in the baseline EMG recording of cramping muscles between bouts of acute cramping [70].

The present study was unique in that baseline EMG from both a cramping and non-active control muscle from the same CR group was recorded. Of importance is that only those muscles directly involved in force production during the Triathlon cramped. These results tend to indicate a local rather than a systemic cause for muscle cramping. Altered serum electrolyte concentrations caused by systemic abnormalities can result in generalised skeletal muscle cramping whereas EAMC only occurs in the localised muscle groups involved in the repetitive contractions associated with exercise [69;70].

It would appear from the results of the present study that EAMC was not associated with serum electrolyte disturbances but may be associated with muscle fatigue.

One of the most commonly accepted hypotheses for the aetiology of EAMC is an abnormality in serum electrolytes, in particular plasma magnesium, sodium, potassium and chloride due to an inadequate replacement of electrolytes during exercise [14;15;30;51;64;78]. Two recent studies compared the serum electrolyte concentrations (sodium, potassium, calcium, phosphate and plasma magnesium) in cramping and control runners both pre- and post-exercise [49;58]. Both studies reported that EAMC was not the result of abnormalities in serum electrolyte concentrations.

The CON group had a statistically significant higher sodium concentration in the present study, although this difference is not clinically significant with both groups' results falling within the standard norms for plasma sodium concentrations [72;78]. The higher plasma sodium concentration in the CON group may, however, have been associated with hydration status. The CR group had a larger post-race body mass (76.3 ± 5.6 kg versus 74.6 ± 6.5 kg) and were less dehydrated (3.4 ± 1.3 % versus 3.9 ± 2.0 %) than the CON group. This indicates that the CON group probably drank less fluid then the CR group during the race which may explain the higher plasma sodium concentration in the CON group.

There were no significant differences in post-race plasma magnesium, potassium or chloride concentrations between the CR and CON group. There were also no significant differences in post-race glucose and haemoglobin concentrations or haematocrits between the CR and CON group.

The increased baseline EMG activity recorded in the localised force-producing cramping muscles in the present study as well as the serum electrolyte concentration results in this study do not support an association between EAMC and abnormalities in serum electrolyte concentrations. It appears from the results of the present study and the results from recent studies of serum electrolyte concentrations in cramping marathon and ultra-distance runners that there is little evidence for the 'serum electrolyte theory' of EAMC [49;58].

Post-race plasma potassium concentrations were positively and significantly correlated with the baseline EMG of the CR group's cramping muscle at minute 10. Potassium concentrations have been shown to rise after ultra-endurance type exercise in comparison with pre-exercise concentrations [6;63;65]. This rise in plasma potassium concentration is usually attributed to the movement of potassium ions out of intracellular stores due to increased cell turnover and/or alterations in muscle cell membrane permeability [6]. One study reported an association between increased post-race plasma potassium concentration and muscle damage after an ultra-endurance run [63]. Although muscle damage was not measured per se, the increase in post-race plasma potassium concentration found in this study may also be the result of acute muscle damage in the cramping muscles. This is supported by the anecdotal observation that many crampers complain of muscle stiffness in the cramping muscles the day after a race. The possibility of muscle damage in cramping muscles offers an explanation for the novel finding of a positive relationship between post-race plasma potassium concentration and baseline EMG in cramping triathletes.

There were no statistically significant correlations between baseline EMG in the cramping muscle at minute 10 and post-race plasma magnesium or sodium concentrations or % weight loss. These results further support a dissociation between cramping, serum electrolyte concentrations and dehydration levels [49;58].

This case control study has some limitations. The sample size was small which was as a result of the number of cramping triathletes on the day. Body mass was recorded during race registration due to time constraints present at the start of the race. This did, however, mean that any changes that occurred in body mass between race registration and the start of the race were not recorded. The EMG equipment used in this study, although portable, did not allow for the more detailed analyses of power frequency shifts and spectral changes which provide important insight into the relative fatigue state of the muscle. Such analyses could also have shown the pattern of recovery during the 10 minutes after exhaustive exercise, thereby allowing one to draw more concrete conclusions from the data regarding the relationship between muscle fatigue and recovery in cramping muscles.

A further limitation of the study was the insufficient duration of EMG data. Although beyond the control of the investigators, the relatively short 10-minute period and the unavailability of subjects for follow-up data on the day after the race limited the scope and nature of deductions that could be made from the EMG data.

The results of this study did however show that EAMC is not associated with dehydration or clinically significant alterations in serum electrolyte concentrations in Ironman triathletes. These results do therefore not support the commonly accepted theories that EAMC is the result of dehydration and abnormalities in serum electrolyte concentrations.

In addition, the increased baseline EMG data recorded in the localised force-producing cramping muscles of the CR group appear to support the novel hypothesis that EAMC is accompanied by heightened muscle activity possibly associated with muscle fatigue.

CHAPTER 5: ELECTROMYOGRAPHIC (EMG) ACTIVITY OF CRAMPING AND CONTROL RUNNERS EXERCISED TO EXHAUSTION

5.1 Abstract

Objectives: To compare the electromyographic (EMG) activity of cramping and control runners before, during and after a fatiguing bout of exercise. **Subjects:** Twenty male runners aged between 20 and 45 years were recruited for the study. Ten runners with a recent history of Exercise Associated Muscle Cramping (EAMC) formed the 'cramping' group (CR, n=10). Ten control subjects with no past experience of EAMC were matched for age, body mass and 5km personal best race times and formed the 'control' group (CON, n=10). **Methods:** Body mass, height and percentage body fat were measured on all subjects. Subjects completed a sub-maximal treadmill run at a speed of 75% of their pre-determined Peak Treadmill Running Speed to exhaustion. Exhaustion was defined as the point at which subjects were no longer able to maintain the set treadmill pace. Surface electromyography (SEMG) was recorded from the right triceps and gastrocnemius muscles before exercise, at 30-min intervals during exercise as well as for 20 min recovery. SEMG was processed to yield amplitude and frequency spectrum data. **Results:** The CR and CON group's triceps EMG amplitude (mV) remained stable throughout testing whereas the CR group's gastrocnemius EMG amplitude (mV) increased during and after exercise. The CR group's gastrocnemius EMG amplitude (mV) was consistently higher and more variable than the CON group at 60 min exercise and throughout recovery. Both the CR and CON groups' triceps and gastrocnemius frequency spectrum shifted left after exercise as a result of muscle fatigue. The CON group's frequency spectrum recovered after 20 min however, whereas the CR group's frequency shifted even further left after 20 min and had not recovered by the end of testing. **Conclusions:** Cramping athletes have a higher and more variable EMG response to fatiguing exercise than non-cramping athletes and may take longer to recover from fatiguing exercise than non-crampers.

Key words: Muscle cramp, electromyography (EMG), muscle fatigue

5.2 Introduction

Exercise Associated Muscle Cramps can be defined as 'painful, spasmodic, involuntary contractions of skeletal muscle during or immediately after physical exercise' [70]. It is a debilitating condition suffered by a large percentage of athletes, yet the aetiology and pathogenesis of EAMC is not well understood.

Numerous studies have recorded the electromyographic (EMG) activity during an induced cramp and have compared these results to the EMG activity of the same muscle at rest (before exercise) and during voluntary contractions [27;60;67]. These studies all report heightened muscle excitability during cramp compared to both resting conditions and voluntary contractions.

Muscle relaxation is the result of both a decreased excitatory Ia afferent input from the muscle spindle and an increased inhibitory Ib afferent activity from the Golgi tendon organ to the alpha motor neuron cell body [18]. Normal control of the alpha motor neuron via the peripheral receptors is essential for muscle relaxation to occur [18]. It has been suggested that cramping is an abnormality of skeletal muscle relaxation [37].

Muscle fatigue has been shown to disrupt the functioning of both the muscle spindle and Golgi tendon organ [31;56]. In response to muscle fatigue, the muscle spindle afferent firing rate increases, while the Golgi tendon organ afferent firing rate decreases dramatically [31;56]. A combination of these two responses in fatigued muscle would result in increased alpha motor activity.

A recent study recorded the baseline EMG activity (the electrical activity in a muscle during remission from a bout of cramping) in runners presenting with severe cramps after an Ultra-endurance marathon [58]. Baseline EMG was recorded immediately after the race and again after 60 minutes of recovery in both cramping and control runners. This study reported a significant decrease in baseline EMG activity during recovery in the cramping runners compared with the control runners [58].

EMG activity has also been recorded during and between acute bouts of muscle cramp after fatiguing exercise [58]. These results showed that runners with EAMC had an increased EMG activity during a cramp as well as an increased baseline EMG activity between bouts of cramping and that a decrease in baseline EMG activity was a good predictor of clinical recovery [58].

A novel hypothesis for the aetiology of EAMC has been suggested based on the observations of an increased baseline EMG activity in cramping runners after fatiguing exercise. This hypothesis suggests that EAMC is the result of an abnormality of sustained alpha motor neuron activity caused by a disruption of the homeostatic alpha motor neuron control at the spinal level during fatiguing exercise [70]. It is postulated that muscle fatigue alters the reflex activity of the peripheral receptors by increasing the muscle spindle's excitatory afferent activity and decreasing the Golgi tendon organ's inhibitory afferent activity [31;56;70]. It is suggested that together these altered reflex activities result in heightened motor neuron activity and are responsible for the increased baseline EMG activity that has been recorded in fatigued cramping runners between bouts of acute cramping [58].

This novel hypothesis for the aetiology of EAMC suggests that the EMG activity of cramping athletes would increase as fatigue progressed and the control of the alpha motor neuron at the spinal level deteriorated. The EMG activity during induced and spontaneous bouts of cramping has been recorded on several occasions [58;60;67]. The baseline EMG activity between bouts of cramping after fatiguing exercise has also been recorded [58]. No studies, however, have recorded the EMG activity of athletes prone to cramping during progressive fatiguing exercise.

5.3 Aim of the study

The aim of this study was to compare the electromyographic (EMG) activity of cramping and control runners before, during and after a fatiguing bout of exercise.

5.4 Methodology

5.4.1 Subjects

Twenty male runners were recruited through advertisements on local running club notice boards. All runners had to have three or more years running experience and had to have an average weekly training distance of 40 kilometres or more to be eligible for inclusion into the study. Ten runners with a 'recent history of Exercise Associated Muscle Cramping' (EAMC) formed the 'cramping' group (CR, n=10). A 'recent history of cramping' was defined as having experienced EAMC in three or more races in the last year. Ten control subjects with no past experience of EAMC were matched for age, body mass and 5km personal best race times. They formed the 'control' group (CON, n=10).

All testing procedures were fully explained to the subjects before they volunteered to participate. All subjects were required to sign a written informed consent form before any testing began (Appendix 5). The Research and Ethics Committee of the Faculty of Health Sciences, University of Cape Town, South Africa, approved the study.

5.4.2 Peak Treadmill Running Speed Test

Subjects completed a brief questionnaire which included personal details and cramping history (Appendix 6). Body mass (to the nearest 100 grams) and height (to the nearest 0.1cm) were measured using a calibrated Healthometer Scale and Height Measure (Bridgeview, Illinois, USA). These measurements were taken with subjects in running shorts and without shoes. The triceps, biceps, suprailiac, subscapular, abdomen, mid-thigh and calf skinfold thickness were measured with a skinfold calliper (Holtain Ltd., Essex, U.K) according to standardised methodology [68]. All skinfold measurements were taken on the right side of the body, except for the abdominal skinfold which was recorded on the subjects' left side.

Percentage body fat was calculated from the sum of these seven skinfolds using a standardised equation [11]. Subjects were fitted with a Polar Sport Tester heart rate monitor (Polar Vantage XL, Polar Electro, Finland) which displayed subjects' resting heart rate prior to exercise.

Each subject performed a Peak Treadmill Running Speed Test on a motor driven treadmill (Quinton Instruments, Seattle, WA, USA) in order to determine their Peak Treadmill Running Speed (PTRS). All subjects completed a standardised warm-up on the treadmill for 5 minutes at a speed of 8km per hour, 0% gradient. The Peak Treadmill Running Speed Test began at a speed of 10 kilometres per hour and increased by one kilometre per hour every 60 seconds. The gradient remained at zero percent throughout the test. The test continued incrementally until subjects reached a treadmill speed that they were not able to maintain. The speed at which the test was stopped was referred to as that subject's Peak Treadmill Running Speed. Peak Treadmill Running Speed (PTRS) and maximal heart rate (Polar Vantage XL, Polar Electro, Finland) were recorded (Appendix 7).

5.4.3 Sub-maximal treadmill run

Subjects returned to the laboratory two days after the Peak Treadmill Running Speed Test for a sub-maximal treadmill run to exhaustion at a speed that equalled 75 % of their PTRS (km/hr) recorded previously. Subjects were requested to refrain from exercising for 48 hours prior to the sub-maximal run and from ingesting caffeine on the morning of the test. Pre-exercise body mass (to the nearest 100 grams) was measured using a calibrated Healthometer Scale and Height Measure (Bridgeview, Illinois, USA). Pre-exercise body mass was recorded with subjects in running shorts and without shoes (Appendix 8).

i) Surface electromyography (EMG)

EMG testing consisted of placing two electrodes (Thought Technology Triode ™ MIEPO1-00, Montreal, Canada) onto the muscle bellies of the triceps brachii and gastrocnemius muscles of all subjects. The triceps electrode was placed over the muscle belly of the right triceps brachii muscle, five centimetres above the lateral epicondyle. The calf electrode was placed over the right gastrocnemius muscle at the site of greatest calf circumference.

The skin over each site was prepared by shaving the hair with a disposable razor (Dahlhausen, Koln, Germany) in a three-centimetre radius around each site. Any oil and dirt was removed with alcohol swabs (Pro-swabs, Sekunjalo Medical, SA). The electrodes were secured with 3M Transpore dressing tape (3M, SA) and linked to the Flexcomp/DSP EMG apparatus (Thought Technology, Montreal, Canada) via a fibre-optic cable.

EMG activity was recorded with subjects sitting in a standardised position with arms hanging relaxed at their side (neutral position for the triceps) and legs outstretched with knees in full extension and ankles in a neutral position (gastrocnemius muscle's relaxed position). One minute pre-exercise resting EMG activity was recorded from both muscles before exercise began. Subjects warmed-up for five minutes on a motor driven treadmill (Quinton Instruments, Seattle, WA, USA) at a speed of 50 % of their peak treadmill running speed (PTRS) recorded previously. Once subjects were warmed-up, the sub-maximal treadmill run began. Subjects were required to run at a speed that equalled 75 % of their PTRS for 120 minutes or until they were exhausted. Exhaustion was defined as the point at which subjects were no longer able to maintain the set treadmill pace.

Subjects ran for 30-minute intervals at which time the treadmill was stopped and subjects sat in the standardised position described above while EMG activity was recorded. EMG activity from both muscles was recorded for one minute. Subjects then continued exercising at 75% of PTRS for another 30 minutes. EMG activity was recorded for 60 seconds with subjects in a seated position at each 30-minute interval throughout the run. After 120 minutes (or once subjects were exhausted) the treadmill was stopped and subjects were required to sit in the standardised position for a recovery period of 20 minutes. During this time, EMG activity was recorded every 10 minutes for a full minute.

Total time on the treadmill and heart rate at the end of the run (Polar Vantage XL, Polar Electro, Finland) were recorded. Post-exercise body mass (to the nearest 100 grams) was measured at the end of recovery using a calibrated Healthometer Scale and Height Measure (Bridgeview, Illinois, USA) and with subjects in running shorts and without shoes.

ii) EMG analysis

EMG activity was sampled at a 1 984 Hz capture rate and was passed through a 50 Hz notch filter to remove any external electrical interference to yield raw data. A five second epoch from each minute (30-35 seconds) of EMG recording was analysed. The raw EMG signals were full wave rectified and movement artefact was removed using a high pass second-order Butterworth filter (15Hz). Thereafter the data was smoothed with a low pass second-order filter (5Hz). All filtering of the integrated EMG (IEMG) data was performed using MATLAB ^{TM} gait analysis software (The Mathworks Inc., USA). EMG data was processed to yield amplitude (root mean squared) and frequency spectrum data.

The pre-exercise IEMG root mean squared (RMS) epoch data (mV) was multiplied by a conversion factor so that it equalled 1.0 mV (or 100%). Each subsequent five-second IEMG RMS epoch data from during and after exercise was normalised to this pre-exercise epoch data and the amount and direction of change in EMG amplitude (mV) was determined in relation to this baseline value.

Decreases in the frequency spectrum of the EMG signal (also referred to as left frequency shifts), due mainly to decreasing muscle fibre conduction velocity, are commonly used to identify muscle fatigue [9;44;47]. A frequency spectrum of 100% is indicative of a non-fatigued rested state in the muscle before exercise. The larger the leftward shift (or the greater the decrease in frequency spectrum away from 100%), the greater the amount of muscle fatigue. Equally a rightward shift (or increase in frequency spectrum towards 100%) reflects recovery from muscle fatigue.

The frequency spectrum for each epoch of data was assessed using a fast Fourier transformation algorithm. The pre-exercise frequency spectrum data was multiplied by a conversion factor so that it equalled 100%. Due to a technical error, a large amount of frequency shift data from the 30 and 60-minute time points during exercise was not available to be included in this analysis. The frequency spectrums from immediate and end recovery (20 minutes recovery) were normalised to the pre-exercise value of 100% and the shifts in mean percentile frequency (MPFS) were determined [41;43;44;54].

5.5 Statistical analysis

All statistical analyses were performed using Statistica Version 6.0 (© StatSoft, Inc; USA) on a personal computer. All data are represented by the mean ± standard deviation. Independent t-tests were used to evaluate differences between groups (CR and CON) and muscles (triceps and gastrocnemius). EMG data recorded at 90 and 120 minutes of the sub-maximal treadmill run were not included for statistical analysis due to the small subject numbers at these time points (n=7 and n=2 respectively). A two-way analysis of variance (ANOVA) for repeated measures was performed on all data to determine whether there was an interaction effect between group and time. Significant differences ($p<0.05$) between groups or over time were further analysed by means of a Tukey post-hoc analysis to determine the site of significance. P-values less than 0.05 were considered significant.

5.6 Results

5.6.1 Descriptive data

The average age (years), body mass (kg), height (cm), percent body fat (%), 5km personal best race times (minutes), running experience (years) and weekly training distance (km) for both the CR (n=10) and CON (n=10) groups are presented in Table 5.1. There were no significant differences between the two groups for any of the variables.

Table 5.1 Age, body mass, height, % body fat, 5km personal best race times, running experience and weekly training distance for both the CR (n=10) and CON (n=10) groups (mean ± standard deviation).

Variable	**CR group (n=10)**	**CON group (n=10)**	**p value**
Age (years)	35.1 ± 5.3	34.4 ± 7.6	0.8
Body mass (kg)	78.3 ± 18.4	72.1 ± 8.7	0.4
Height (cm)	178.5 ± 8.5	175.7 ± 7.5	0.4
Body fat (%)	17.4 ± 5.3	15.5 ± 2.9	0.3
5km personal best race time (min)	19.7 ± 4.8	17.9 ± 1.6	0.3
Running experience (years)	6.5 ± 4.3	9.1 ± 7.5	0.4
Weekly training distance (km)	63.5 ± 16.5	61.0 ± 13.3	0.7

5.6.2 Physiological variables

The physiological data collected during both the peak treadmill running speed test and sub-maximal treadmill run for both the CR (n=10) and CON (n=10) groups is presented in Table 5.2.

Physiological variables included resting heart rate (beats per minute), maximum heart rate (beats per minute), peak treadmill running speed (km/hour), total time on the treadmill (minutes), fatigue heart rate (final exercising heart rate expressed as a percentage of maximum heart rate) and change in body mass from pre- to post-exercise (kg). There were no significant differences between the two groups.

Table 5.2 Resting heart rate, maximum heart rate, peak treadmill running speed, total time on the treadmill, fatigue heart rate and change in body mass from pre- to post-exercise (kg) for the CR (n=10) and CON (n=10) groups (mean ± standard deviation).

Variable	**CR group (n=10)**	**CON group (n=10)**	**p value**
Resting heart rate (beats per minute)	56.6 ± 7.3	53.4 ± 8.9	0.4
Maximum heart rate (beats per minute)	176.3 ± 15.9	180.8 ± 11.6	0.5
Peak treadmill running speed (km/hour)	17.3 ± 2.1	18.3 ± 1.6	0.3
Total time on treadmill (min)	84.7 ± 29.4	77.3 ± 36.3	0.6
Fatigue heart rate (% of max heart rate)	94.1 ± 5.3	94.9 ± 4.9	0.7
Weight change pre-post exercise (kg)	2.1 ± 1.1	1.6 ± 1.1	0.3

5.6.3 Integrated electromyographic (IEMG) amplitude

Integrated EMG amplitude (mV) data from the non-exercising (triceps) muscle for both the CR (n=9) and CON (n=6) groups before, during and after a fatiguing bout of exercise are presented in Figure 5.1a.

There were no significant differences in the IEMG amplitude (mV) of the non-exercising muscle between the two groups at any time point during exercise (30 minutes exercise, 60 minutes exercise) or during recovery (immediate recovery, 10 minutes recovery, 20 minutes recovery). The IEMG amplitude data (mV) from the CON group (n=6) tended to be higher (p=0.06) than the IEMG amplitude data (mV) from the CR group (n=9) at 10 minutes recovery. There were no significant differences between the two groups over time. The IEMG amplitude pattern (mV) for the non-exercising muscle shown in Figure 1a remained relatively stable in both groups throughout the testing period.

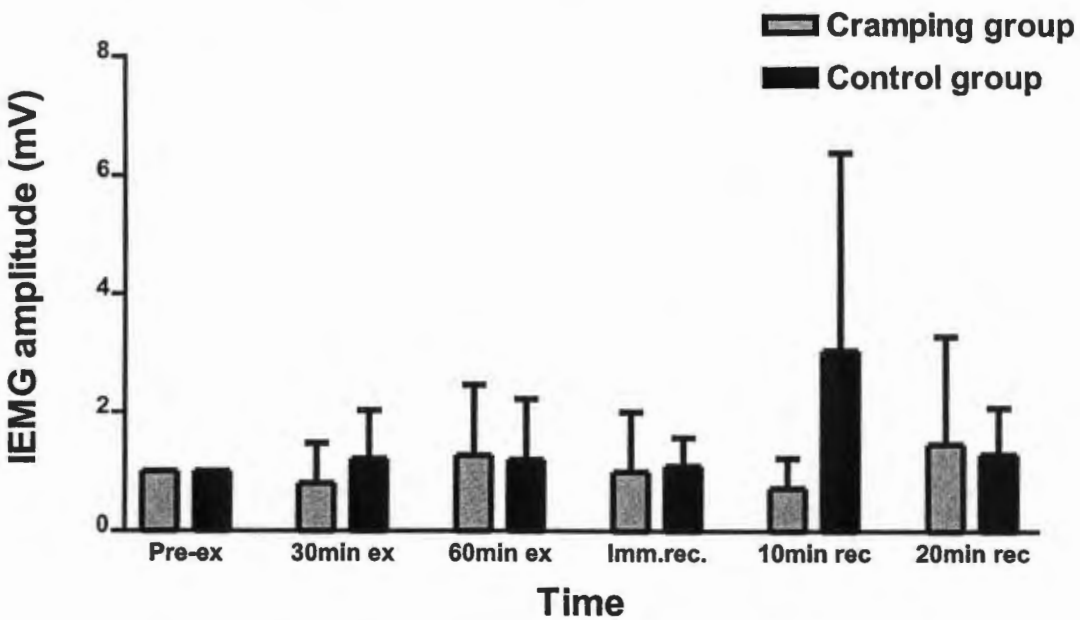

Figure 5.1a Integrated EMG amplitude (mV) from the non-exercising (triceps) muscle for both the CR (n=9) and CON (n=6) groups before, during and after a fatiguing bout of exercise. Data presented as mean ± standard deviation.

Integrated EMG amplitude (mV) data from the active (gastrocnemius) muscle for both the CR (n=9) and CON (n=5) groups before, during and after a fatiguing bout of exercise are presented in Figure 5.1b.

There were no significant differences in the active muscle's IEMG amplitude (mV) between the two groups at any time point, although the IEMG amplitude (mV) from the CR group (n=9) was consistently higher and more variable (as evidenced by the large standard deviation) than that of the CON group (n=5) at 60 minutes exercise and throughout recovery.

There were no significant differences in the active muscle over time for either group, although the active muscle's IEMG amplitude (mV) from the CR group (n=9) did increase by 320% (1.0 mV to 4.2 mV) from pre-exercise to 20 minutes recovery. This is in contrast to the IEMG amplitude (mV) from the CON group (n=5) which decreased by 20% (1.0 mV to 0.8 mV) over the same time period.

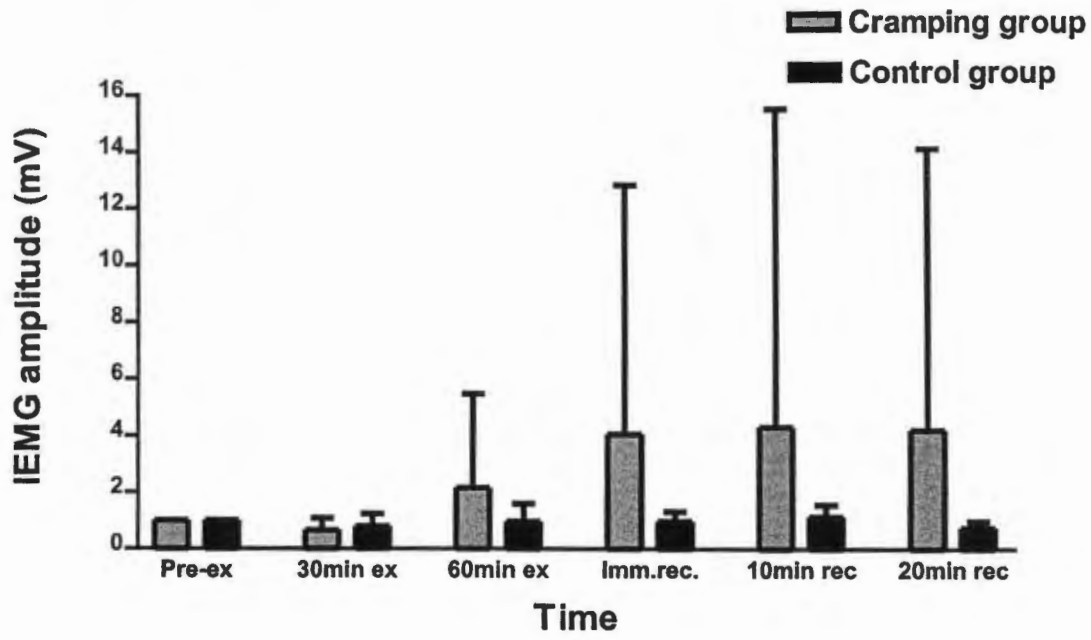

Figure 5.1b Integrated EMG amplitude (mV) from the active (gastrocnemius) muscle for both the CR (n=9) and CON (n=5) groups before, during and after a fatiguing bout of exercise. Data presented as mean ± standard deviation.

IEMG amplitude (mV) for both the triceps (n=15) and gastrocnemius (n=14) muscles for both groups combined are presented in Table 5.3.

There were no significant differences in IEMG amplitude (mV) between the two muscles at any specific time point, although the IEMG amplitude (mV) from the gastrocnemius muscle (n=14) was consistently higher than that of the triceps muscle (n=15) at 60 minutes exercise and throughout recovery. The IEMG amplitude (mV) from the gastrocnemius muscle (n=14) also showed far more variability than the triceps muscle (n=15) at the same time points as reflected by the large standard deviation in the gastrocnemius muscle's IEMG amplitude (mV). There were no significant differences between the muscles over time.

Table 5.3 IEMG amplitude (mV) from both the triceps (n=15) and gastrocnemius (n=14) muscles for both groups combined before, during and after a fatiguing bout of exercise (mean ± standard deviation).

Time point	**Triceps IEMG amplitude (mV) (n=15)**	**Gastrocnemius IEMG amplitude (mV) (n=14)**	**p value**
Pre-exercise	1.0 ± 0.0	1.0 ± 0.0	-
30min exercise	0.9 ± 0.7	0.7 ± 0.4	0.3
60min exercise	1.2 ± 1.1	1.7 ± 2.7	0.5
Immediate recovery	1.0 ± 0.8	2.9 ± 7.1	0.3
10min recovery	1.6 ± 2.5	3.2 ± 8.9	0.5
20min recovery	1.4 ± 1.5	2.9 ± 8.0	0.5

5.6.4 Frequency shifts

The percentile frequency shifts (%) from the non-exercising (triceps) muscle for both the CR (n=9) and CON (n=9) groups for immediate and end recovery (20 minutes recovery) are presented in Figure 5.2a. There were no significant differences in mean percentile frequency shifts (%) between the two groups at either time point during recovery, nor were there any significant differences over time between the groups.

Both the CR (n=9) and CON (n=8) groups' mean percentile frequency shifts (%) reflected a left shift in frequency spectrum (indicative of muscle fatigue) both immediately after exercise and after 20 minutes of recovery. Although the CON (n=8) group's mean percentile frequency shift (%) had shifted right (towards 100%) by the end of recovery (indicative of recovery from muscle fatigue), the CR (n=9) group's mean percentile frequency shift (%) had shifted even further left by the end of recovery (indicative of a worsening degree of muscle fatigue).

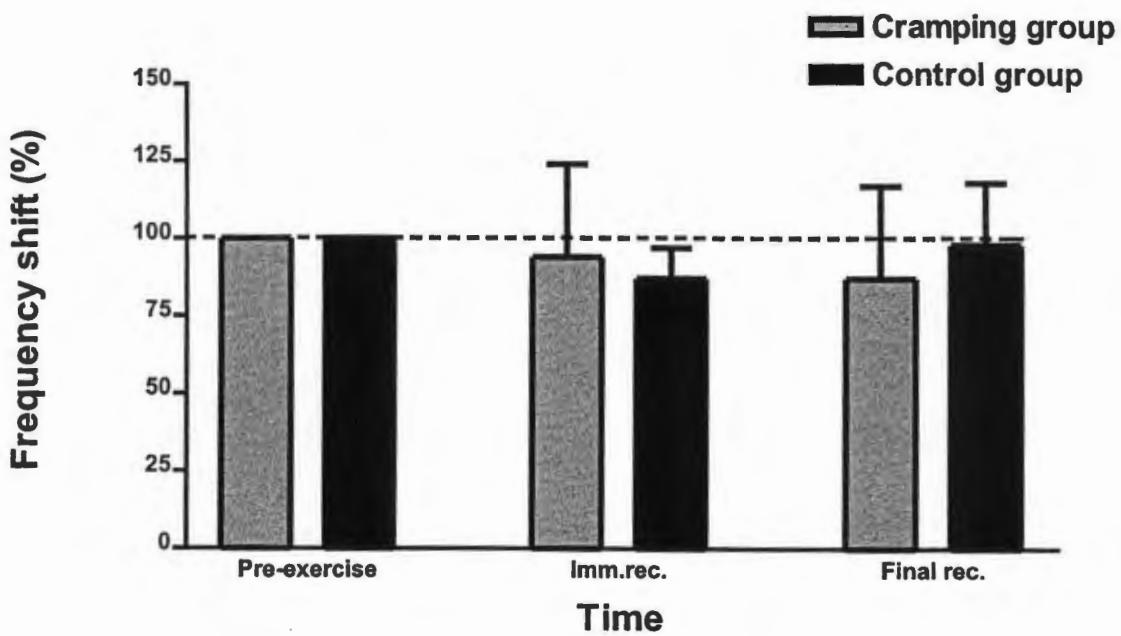

Figure 5.2a Mean percentile frequency shifts (%) from the non-exercising (triceps) muscle for both the CR (n=9) and CON (n=8) groups for immediate and end recovery. Data presented as mean ± standard deviation.

Percentile frequency shifts (%) from the active (gastrocnemius) muscle for both the CR (n=10) and CON (n=8) groups for immediate and end recovery (20 minutes recovery) are presented in Figure 5.2b. There were no significant differences in mean percentile frequency shifts (%) between the two groups at either time point during recovery, nor were there any significant differences over time between the groups.

Both the CR (n=10) and CON (n=8) groups' mean percentile frequency shifts (%) reflected a left shift in frequency spectrum (indicative of muscle fatigue) both immediately after exercise and after 20 minutes of recovery. Although the CON (n=8) group's mean percentile frequency shift (%) had shifted right (indicative of recovery) by the end of recovery, the CR (n=10) group's mean percentile frequency shift (%) had shifted further left by the end of recovery (indicative of further development of muscle fatigue).

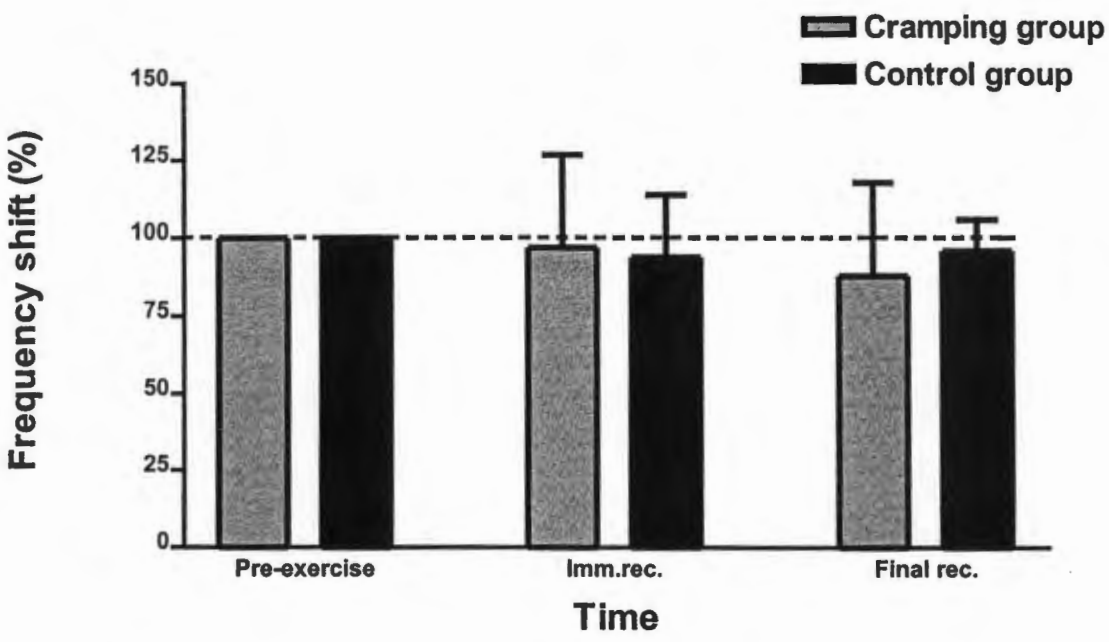

Figure 5.2b Mean percentile frequency shifts (%) from the active (gastrocnemius) muscle for both the CR (n=10) and CON (n=8) groups for immediate and end recovery. Data presented as mean ± standard deviation.

5.7 Discussion

The main findings of this study are that there are no significant differences in electromyographic (EMG) activity between cramping and control subjects before, during or after fatiguing exercise. The results of this study do suggest, however, that cramping athletes appear to have a higher and more variable EMG response to muscle fatigue and take longer to recover from fatiguing exercise than non-cramping athletes.

A unique feature of the present study was that EMG activity was recorded from both an active and non-exercising muscle during and after fatiguing exercise. Although one study has compared baseline EMG data from control and cramping subjects after an ultra-endurance run [58], no published study has previously recorded the EMG activity of a non-exercising control muscle.

As expected from a muscle not involved in actual force production during this type of exercise, the triceps muscle's EMG activity did not deviate much from the pre-exercise recording in either group and remained stable at between 1.0 and 2.0 mV throughout the testing period. The relatively high EMG activity in the control group at the 10-minute recovery data point (Figure 5.1a) was unexpected but may be explained by the commonly accepted high inter-subject variability associated with the EMG technique [43]. The trend apparent in the triceps muscle's EMG activity corresponds with data presented earlier in this thesis from the 2000 Ironman Triathlon. In that study, the baseline EMG activity in the non-exercising muscle (triceps) of the cramping group also remained stable throughout the 10-minute recovery period.

Although there were no significant differences in the active, force-producing gastrocnemius muscle's EMG activity between the CR and CON groups in this study, this muscle's mean EMG activity was consistently higher and more variable (as reflected by the large standard deviations) in the CR group than in the CON group (Figure 5.1b). The gastrocnemius muscle's EMG activity in the CR group increased throughout the testing period and had increased by 320% from the pre-exercise resting EMG value by the end of recovery. This is in contrast to the CON group whose EMG activity had decreased by 20% from the pre-exercise resting EMG value by the end of recovery.

Although there are many studies that report the effect of muscle fatigue on EMG amplitude, the majority of these studies focus on isometric contractions [7]. Two studies have reported an increase in EMG amplitude in the lower limb muscles of healthy athletes during recovery from an isometric fatigue test which they attributed to increased muscle activation and an impairment of the excitation-contraction coupling with fatiguing exercise [80;81].

One study that investigated gastrocnemius EMG activity after a marathon run reported a decrease in EMG amplitude immediately after exercise which only increased again after two hours of recovery [1]. Such a decrease in EMG amplitude is usually expected after muscle fatigue and can be explained by the phenomenon known as 'muscle wisdom'. Muscle wisdom is defined as the 'functionally appropriate reduction in motor unit firing rates which occurs during a sustained maximal voluntary contraction' [48]. The 'muscle wisdom' phenomenon is a protective mechanism which results in a decreased EMG activity when the force required from a muscle can no longer be maintained without some form of muscle damage resulting [22].

Although a few studies have shown an increased EMG activity during induced and spontaneous muscle cramping [60;67], no published study has shown an increase in EMG activity in a muscle not actually cramping. A recent study recorded the baseline EMG activity in runners presenting with severe cramps after an Ultra-endurance marathon [58]. This study reported that runners with EAMC had an increased baseline EMG activity between bouts of cramping and that there was a significant decrease in baseline EMG activity during recovery in the cramping runners compared with the control runners [58].

A novel hypothesis for the aetiology of EAMC has been suggested based on the observations of an increased baseline EMG activity in fatigued cramping athletes. This hypothesis suggests that EAMC is the result of an abnormality of sustained alpha motor neuron activity caused by a disruption of the homeostatic alpha motor neuron control at the spinal level during fatiguing exercise [70]. It is postulated that muscle fatigue alters the reflex activity of the peripheral receptors by increasing the muscle spindle's excitatory afferent

activity and decreasing the Golgi tendon organ's inhibitory afferent activity [31;56;70]. It is suggested that together these altered reflex activities result in heightened motor neuron activity and are responsible for the increased baseline EMG activity that has been recorded in fatigued cramping runners between bouts of acute cramping [58].

Although there is little evidence from the results of the present study to support an association between EAMC and muscle fatigue, the present study did show that the EMG activity of the force-producing muscle in the CR group was still elevated after 20 minutes of recovery. This is in contrast to the EMG activity in the CON group which had returned to pre-exercise values by the end of recovery. These results suggest that cramping athletes may have a heightened EMG response to muscle fatigue and may take longer to recover from fatiguing exercise than non-cramping athletes.

Muscle fatigue is a complex and multi-faceted phenomenon which, despite much research attention, remains poorly understood. Muscle fatigue can be defined as 'the inability of a muscle or group of muscles to maintain the required force' [62]. Decreases in the median frequency of the power spectrum of the EMG signal (left frequency shifts) are commonly used to identify muscle fatigue [9;42;47].

Although there were no significant differences in the frequency spectrum data between the CR and CON groups for either the triceps or gastrocnemius muscles during recovery, both groups showed a shift towards the lower frequencies (indicative of muscle fatigue) after fatiguing exercise in both muscles. Such a shift can be expected after fatiguing exercise as the gastrocnemius muscle is responsible for repeated plantar flexion of the foot during running and the triceps muscle is one of the primary extensors of the gleno-humeral joint which is constantly moving between flexion and extension during running. These results are supported by studies which have also reported a decrease in mean frequency shifts after both isometric and endurance fatigue tests [9;23;40;47].

Despite both the CR and CON groups reflecting a left shift in mean percentile frequency after fatiguing exercise, the CON group's frequency for both muscles increased towards the pre-exercise level of 100% after 20 minutes of recovery while the CR group's frequency for both muscles shifted even further left after the same amount of recovery time (Figures 5.2a and 5.2b). There are no published data on the frequency shift patterns of cramping athletes, yet several studies have reported changes in frequency shifts during recovery.

Two studies reported a decrease in the mean frequency spectrum during isometric fatigue which returned to pre-exercise values after 10-15 minutes of recovery [35;80]. Another study also recorded the frequency shifts after an isometric fatigue test but reported a continued decrease in the frequency spectrum 10 minutes after exercise [16]. This study suggested that after fatiguing exercise the electrical activity of some highly fatigable motor units may be altered and that these motor units may not be recruitable during recovery [16]. This same study further suggested that certain motor units may be more susceptible to fatigue and take longer to recover from fatigue.

One study has reported the relationship between highly fatigable fast twitch muscle fibres (Type II muscle fibres) and changes in frequency spectrum and suggests that changes in frequency shifts are related to muscle fibre composition [23]. A further study supports this by suggesting that fatigue resistance may be related to genetically determined muscle composition [62]. A tentative suggestion has been made by another study that there may be a predominance of highly fatigable fast twitch muscle fibres in people who experience muscle cramps and exertional myalgia [76].

The findings of the present study suggest that the motor units of cramping athletes may take longer to recover from fatiguing exercise than those of non-cramping athletes.

The present study has some limitations. The large variability in EMG data in the present study (as reflected by the large standard deviations) was a limitation that was not anticipated. The small sample group proved not to be sufficient to overcome the high degree of variability in the EMG data and may have resulted in a Type II statistical error (reporting no differences between variables when they actually existed). The large variability in EMG data thus limited the statistical power of the data and the strength of the conclusions that could be drawn from the available data. The relatively short recovery time of 20 minutes in this study was another limitation which restricted the conclusions that could be made about the recovery responses of the different groups to fatiguing exercise.

Although the results of the present study do not support the hypothesised association between EAMC and muscle fatigue, they do suggest that cramping athletes have a higher and more variable EMG response to fatiguing exercise than non-cramping athletes and that cramping athletes may take longer to recover from exhausting exercise than non-cramping athletes. The exact nature of the relationship between EAMC and muscle fatigue remains unclear, however, and needs further investigation.

CHAPTER 6: SUMMARY AND CONCLUSION

6.1 Summary

Exercise Associated Muscle Cramping (EAMC) is a painful and frustrating condition experienced by large numbers of athletes. Despite this high prevalence of muscle cramping amongst athletes, EAMC has not been thoroughly researched and the exact aetiology and pathogenesis of this condition is not well understood.

A high lifetime prevalence of EAMC has been reported in triathletes, yet EAMC has not been thoroughly researched in either triathletes or Ironman triathletes. This thesis documented the nature and prevalence of EAMC in a group of athletes participating in an Ironman Triathlon. This study showed that there is a high lifetime prevalence of EAMC amongst these triathletes. Furthermore, the results show that EAMC is prevalent in running, racing and during the last quarter of a race and may be associated with muscle fatigue. The results further suggest that a positive family history of muscle cramping may be associated with the severe and debilitating muscle cramps experienced by some athletes during exercise.

The role of serum electrolyte abnormalities and hydration status in EAMC is a contentious issue. Furthermore, there have been no studies on the serum electrolyte concentrations of cramping Ironman triathletes. This thesis therefore investigated the serum electrolyte concentrations in cramping and control Ironman triathletes following a race. The results show that Ironman triathletes suffering from acute EAMC are not dehydrated and do not have clinically significant serum electrolyte abnormalities. The findings of this thesis therefore do not support the commonly accepted theories that EAMC is the result of dehydration and abnormalities in serum electrolyte concentrations.

The baseline electromyographic (EMG) activity of cramping runners has been recorded after a race, but no studies have recorded the baseline EMG activity of cramping and non-exercising control muscles in cramping Ironman triathletes after a race. The results of this thesis show that cramping muscles have a higher and more variable baseline EMG activity than non-active control muscles during recovery from fatiguing exercise. These findings appear to support the novel hypothesis that EAMC is accompanied by heightened muscle activity possibly associated with muscle fatigue.

A novel hypothesis for the aetiology of EAMC has suggested an association between EAMC and muscle fatigue but this hypothesis has not yet been fully investigated. This thesis recorded the EMG amplitude and frequency spectral changes of athletes prone to cramping during progressive fatiguing exercise. Although the EMG results do not support the hypothesised association between EAMC and muscle fatigue, they do show that cramping athletes have a higher and more variable EMG response to fatiguing exercise than non-cramping athletes and that cramping athletes may take longer to recover from exhausting exercise than non-cramping athletes. However, the exact nature of the relationship between EAMC and muscle fatigue is unclear.

6.2 Conclusion

In conclusion, this thesis has shown that there is a high prevalence of Exercise Associated Muscle Cramps in Ironman triathletes which needs further investigation. Furthermore, the results of this thesis do not support the commonly accepted theories that EAMC is the result of dehydration and abnormalities in serum electrolyte concentrations. The increased baseline EMG activity in cramping Ironman triathletes after a race appears to support the novel hypothesis that EAMC is accompanied by heightened muscle activity possibly associated with muscle fatigue. The EMG response to progressive fatiguing exercise in athletes prone to cramping does not, however, support an association between EAMC and muscle fatigue. The role of muscle fatigue in EAMC therefore remains undetermined and in need of further investigation.

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APPENDICES

Appendix 1:
Ironman 2001 Study Information and Informed Consent Form

CRAMPING STUDY INFORMATION

The MRC/UCT Research Unit for Exercise Science and Sports Medicine will be conducting a research trial at the Cape Town IRONMAN Triathlon to be held in Gordons Bay on 31 March 2001. The research trial will be looking at the nature and prevalence of cramping in Ironman triathletes.

All triathletes taking part in the Triathlon will be requested to participate in the research study. Athletes will however not be coerced to take part. As a participant in this study you will receive an in-depth questionnaire to complete prior to the Triathlon. You will be requested to provide information on your personal particulars, health status, training and running history.

Registration

At race registration you will be required to report to the University of Cape Town's 'on-site' research venue in the Villa Via Hotel in Gordons Bay. There you will hand in your questionnaires to the principal investigator.
You will then be requested to donate 5ml of blood to determine your sodium and potassium levels.

Race day

Before the start of the swimming leg of the Triathlon you will be weighed to monitor your change in weight from before to after the race.

Post-race testing

At the end of the race you will be requested to enter the Medical Tent for testing and a medical examination. You will be weighed once again and requested to donate another 5ml blood sample.

2001 CRAMPING STUDY INFORMED CONSENT

I, _____, have read and fully understand the details of the study and hereby give consent to participate in the trial to be conducted by the MRC/UCT Research Unit for Exercise Science and Sports Medicine at the Cape Town Ironman Triathlon to be held in Gordons Bay on 31 March 2001.

I agree to participate in the study and understand the commitments required of me. I understand that on completion of the Triathlon, I will enter the medical tent for a medical examination and testing. I am aware that I may withdraw from the trial at any time.

Name: _____

Signature: _____

Date: _____

Investigator: _____

Signature: _____

Date: _____

Appendix 2:

Ironman 2001 Data Collection Form

A. PERSONAL PARTICULARS

(to be completed by all participants)

Event	Cape Town Ironman Triathlon		Race Number		
Surname					
First name(s)					
Postal address					
			Code		
E-mail address			Phone		
Date of birth	Y Y Y Y / M M / D D		Cell		
Height (cm)	cm		Gender	male	female
Weight (kg)	kg				
Nationality					
Country of birth			Dominant Hand	Left	Right
Do you know your blood group?	Yes	A	B	A B	O
	No	Pos	Neg		

DO YOU SUFFER FROM ANY MEDICAL CONDITIONS? e.g. Asthma, Diabetes, Heart Disease	YES	No
	IF YES, PLEASE SPECIFY.	
Are you on any medications (Over the Counter or Prescription)?	YES	No
	DETAILS	

B.MUSCLE CRAMPING QUESTIONAIRE

(to be completed by all paticipants)

1.Have you ever experienced muscle cramping during or immediately after exercise? (Please tick the box if you have)

a) in your running career ☐
b) in the last 5 years ☐
c) in the last year ☐

2.Have you ever experienced any other form of muscle cramping? (you can tick more than one box)

a) Night cramps ☐
b) Cramps during pregnancy (if applicable) ☐
c) Side stitch during exercise ☐
d) Other cramps ☐
Please specify_____

3.Please tick the box that shows which type of exercise where you are most likely to cramp? (Please tick only one box)

a) Running ☐
b) Swimming ☐
c) Cycling ☐

4.Please tick under which conditions you experience cramping

a) only in training ☐
b) only in racing ☐
c) in training and racing ☐

5. Please complete this question if you ever experienced cramping during races:

At what point during the race do you usually first experience cramping

a) From the beginning ☐
b) In the first quarter of the race ☐
c) In the second quarter of the race ☐
d) In the third quarter of the race ☐
e) In the last quarter of the race ☐
f) After the race ☐

6. Please indicate in which muscle groups do you cramp?

a) Quadriceps ☐
b) Hamstrings ☐

c) Calves ☐
d) Foot ☐
e) Other (please specify) ☐

7. Please indicate how does the cramp last for (in minutes)? ☐ minutes

8. Please indicate the severity of your cramping using the following classification (please tick one box)

a) Mild: 5 minutes and you are able to continue exercising ☐
b) Moderate: 10 minutes and you are able to continue exercising ☐
c) Severe: 15> minutes and you have to STOP exercising ☐

9. Please indicate whether there is any one in your family that has ever experienced cramping during exercise

Yes _____ No _____

10.Please indicate, by ticking the appropriate box, whether you have consulted a health professional regarding you cramping

a) Nutritionist/Dietician ☐
b) Doctor ☐
c) Physiotherapist ☐
d) Sports scientist ☐
e) Coach ☐
f) Podiatrist ☐
g) Chiropractor ☐

11.Please indicate, by ticking the appropriate box, what treatment you have had for your muscle cramping to date.

a) Regular stretching ☐
b) Increased training ☐
c) Decreased training ☐
d) Magnesium supplementation ☐
e) Sodium supplementation ☐
f) Increased fluid intake during exercise ☐
g) Decreased fluid intake during exercise ☐
h) Adequate race preparation ☐
i) Muscle strengthening program ☐
j) Massage therapy ☐

12. Which of the following factors are associated with your cramping during exercise

a) Previous muscle injury ☐
b) Overhydration ☐
c) Dehydration ☐
d) "Flu like" symptoms before the race/training ☐
e) Muscle "twitchiness" just before cramping ☐
f) Muscle fatigue just before cramping ☐
g) Not stretching the muscles regularly ☐
h) Overstretching the muscles ☐
i) Increasing your training load ☐
j) Not using nutritional supplements ☐
k) Genetic factors (family members) ☐
l) Lack of electrolytes (minerals) ☐
m) Lack of "fitness" ☐
n) Hot weather ☐

C.TRAINING LOG

(TO BE COMPLETED BY ALL PARTICIPANTS)

NOTE: If you are unsure of what training you have completed, please do not guess but rather indicate that you are unsure or can't remember.

Please complete the following tables to document your swimming, cycling and running training volume over the last 5 years.

SWIMMING

Time	Months/ year	Hrs/wk	Distance/wk
0-3 months	-		
3-6 months	-		
6-12 months	-		
1-2 years			
2-3 years			
3-4 years			
4-5 years			

CYCLING

Time	Months/ year	Hrs/wk	Distance/wk
0-3 months	-		
3-6 months	-		
6-12 months	-		
1-2 years			
2-3 years			
3-4 years			
4-5 years			

Time	Months/ year	Hrs/wk	Distance/wk
0-3 months	-		
3-6 months	-		
6-12 months	-		
1-2 years			
2-3 years			
3-4 years			
4-5 years			

D. IRONMAN RUNNING HISTORY
(TO BE COMPLETED BY ALL PARTICIPANTS)

Type of triathlon	Ironman
Have you participated in an Ironman before?	Yes No
Personal best time (hrs:min:sec)	
Year of first event	
How many events have you participated in?	

Appendix 3:

Ironman 2000 Informed Consent Form

2000 IRONMAN INFORMED CONSENT FORM

I,..... agree voluntarily to participate in a research project of the MRC/UCT Bioenergetics of Exercise Research Unit, Sport Science Institute. The project aims to gain previously unrecorded Electromyographic (EMG) data on Exercise Associated Muscle Cramping (EAMC) sufferers.

It involves recording EMG activity by means of surface electrodes placed on the cramping and triceps muscles for a maximum period of 20 minutes.

I have read this form, and I understand the procedures involved including risks and benefits. I have also had the opportunity to ask any questions, all of which have been answered to my satisfaction. I further understand that I may withdraw from the study at any time and that all data will be treated as confidential.

Subject signature.....Date.....

Investigator signature.....Date.....

Witness signature.....Date.....

Appendix 4:

Ironman 2000 Data Collection Form

IRONMAN 2000 TRIATHLON: CRAMPING STUDY

Name	**Race no**	**Cramper** **Control**
Tel no	**Age**	**Time**

- Check position, cleaned site, attach electrodes to right triceps and cramping muscle.
- Time of entry:_____

RECOVERY EMG

TIME	TRI	CRAMP
0 min		
0.30 min		
1 min		
1.30 min		
2 min		
2.30 min		
3 min		
3.30 min		
4 min		
4.30 min		
5 min		
9 min		
9.30 min		
10 min		

Time of discharge:_____

Appendix 5:

EMG and Fatigue Study Informed Consent Form

INFORMED CONSENT

I, ... agree voluntarily to participate in a research project of the MRC/UCT Bioenergetics of Exercise Research Unit, Sport Science Institute. The project aims to gain previously unrecorded data on Exercise Associated Muscle Cramping (EAMC) sufferers with respect to electromyography (EMG) activity associated with fatigue. It involves the following procedures:

1. A maximal treadmill test consisting of progressive increments in speed until exhaustion. During this test measured of body composition (height, weight, % body fat) and heart rate will also be recorded.

2. A sub-maximal test performed at 75 % of peak treadmill speed for a duration of 120 minutes. During this test EMG activity will be recorded by means of surface electrodes placed on the subjects' triceps and gastrocnemius muscle.

I have read this form, and I understand the procedures involved – including risks and benefits. I have had the opportunity to ask any questions, all of which have been answered to my satisfaction. I further understand that I may withdraw from the study at any time and that all data will be treated as confidential.

Subject signature:.....Date:.....
Investigator signature:.....Date:.....
Witness signature:.....Date:.....

Appendix 6:

Subject questionnaire

A. General Information

Name:_____

Date of birth:_____

Address:_____

_____

_____ Code:_____

Telephone number: (work) _____
(home)_____
(cell)_____

E-mail: _____

Subject number:_____

B. Training history

How long have you been running for (years)? _____

What is your average weekly training mileage (km)? _____

Best 5 km time? _____

C. History of exercise associated muscle cramps

Do you suffer from cramps associated with running? YES/NO

If yes, in 1 of the last 10 races _____
in 3 of the last 10 races _____
in 5 of the last 10 races _____
in >5 of the last 10 races _____

Do you usually experience cramps in: training _____
racing _____
both _____

In which muscle groups do you experience cramps while running?
front thigh (quads) _____
back thigh (hamstrings) _____
calf _____

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Appendix 7:

Maximum data collection form (Visit 1)

Maximal data collection session

Date: _____ Subject number: _____

Pre-test: Height: _____ Weight: _____

Tricep: _____

Bicep: _____

Subscap: _____

Supra-iliac: _____

Abdominal: _____

Thigh: _____

Calf: _____

Resting HR _____

Max test:

Time (min)	Speed (km/hr)	Achieved
0.00	10	
1.00	11	
2.00	12	
3.00	13	
4.00	14	
5.00	15	
6.00	16	
7.00	17	
8.00	18	
9.00	19	
10.00	20	

Time ended: _____

Max HR: _____ Max speed: _____

Appendix 8:

Sub-maximum data collection form (Visit 2)

Sub-max data collection

Date: _____ Subject number: _____

50%max: _____

75%max: _____

Pre-test: Weight _____

Rest EMG code _____

Warm-up: 5 min @ 50% PTRS

Test @ 75%:

Time started: _____

00.00 Start run

30.00 EMG code _____

35.00 Continue run

60.00 EMG code _____

65.00 Continue run

90.00 EMG code _____

95.00 Continue run

120.00 EMG code _____

Time ended _____

Recovery:

00.00 EMG code _____

10.00 EMG code _____

20.00 EMG code _____

Post-test BW: _____

" Wir sind mittenrein geboren,
Und sind mittenrein geraten
Uns hat keiner recht erklärt
Wie dieses Leben geht
Und wir strampeln um die Wette
Und wir bleiben auf der Strecke
Und wir stehen wieder auf
Weil dieser Weg weitergeht."

PUR Mittendrin, 2000