

Acute Passive Static Stretching and Cramp Threshold Frequency

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Context: Exercise-associated muscle cramps are a common clinical problem for athletes.

Objective: To determine whether acute passive static stretching altered cramp threshold frequency (CTF) of electrically induced muscle cramps.

Design: Crossover study.

Setting: Laboratory.

Patients or Other Participants: Seventeen healthy college-aged individuals.

Intervention(s): Stretching or no stretching.

Main Outcome Measure(s): The independent variable was the static stretch versus the no-stretch condition, and the dependent variable was the CTF.

Results: The CTF increased in both the control (pretest: 18.12 ± 6.46 Hz, posttest: 19.65 ± 7.25 Hz; $P = .033$) and stretching (pretest: 18.94 ± 5.96 Hz, posttest: 20.47 ± 7.12 Hz; $P = .049$) groups. No difference between the groups was found ($t_{15} = 0.035$, $P = .97$).

Conclusions: Acute passive static stretching did not seem to increase the CTF.

Key Words: exercise-associated muscle cramps, electrically induced muscle cramps, Golgi tendon organ reflex response

Key Points

- Exercise-associated muscle cramps occur frequently in athletes, affecting as many as 67% of triathletes and 30% to 50% of marathon runners.
- Epidemiologic studies have indicated that athletes with more consistent stretching programs experienced cramps less often.
- Acute static stretching is the intervention of choice for the immediate relief of exercise-associated muscle cramps; the purported mechanism is the initiation of the Golgi tendon organ reflex response, which inhibits the α -motoneuron.

Exercise-associated muscle cramps (EAMCs) are localized, painful, and involuntary contractions of skeletal muscle that occur during or immediately postexercise and are one of the most common clinical problems reported in athletes.¹ They can hinder athletic performance and have been reported to affect 67% of triathletes and between 30% and 50% of marathon runners.² Investigations of EAMCs are difficult because the spontaneous nature of the muscular phenomenon is unknown and difficult to test in the field. To date, the most frequent method used to provoke EAMCs is electrically induced muscle cramps (EIMCs). The EIMCs are correlated with EAMCs,³ and the method has been previously validated⁴ with high intrasession and intersession reliability³ while causing little pain and soreness.⁵

Although the cause of EAMCs is unknown, the traditional mechanism behind them, as reported by Schweltnus et al,¹ is associated with dehydration. However, a paucity of empirical evidence links hypohydration to EAMCs. Miller et al⁶ and Braulick et al⁷ investigated EIMCs in the nonfatigued limbs of participants at 3% and 5% hypohydration. Neither group demonstrated an effect of hypohydration on the cramp threshold frequency (CTF).

Moreover, sodium and carbohydrate drinks are often used to prevent EAMCs before, during, and after athletic events by increasing blood plasma electrolyte concentrations. However, this notion has been challenged because gastric emptying and plasma electrolyte levels immediately after ingestion of sodium and carbohydrate drinks have not resulted in changes in blood plasma concentrations.^{8,9} (Additional reviews on dehydration and EAMCs can be found elsewhere.^{10,11})

Given the evidence refuting dehydration as a primary mechanism for EAMCs, focus has been shifted to altered neuromuscular control.^{12–14} The theory of altered neuromuscular control suggests that hyperexcitability of the motoneuron pool causes sustained α motoneuron firing.^{1,12} Specifically, the hyperexcitability results from an imbalance between increased muscle-spindle activity and reduced Golgi tendon organ feedback. Because the majority of EAMCs occur at the end of or after activity, fatigue has been implicated.¹ Fatigued muscles show increased muscle-spindle activity¹⁵ and increased baseline electromyographic (EMG) activity in animals and humans,¹⁶ suggesting persistent α -motoneuron activity.

Inconsistent stretching habits and irregular stretching times have been suggested as risk factors for EAMCs.¹ Static stretching is a common pre-event modality that has historically been used to improve sport performance or limit performance-related injury or both.¹⁷ Static stretching has been reported to alter α -motoneuron pool activity proportionally to the stretching intensity,¹⁸ as evidenced by a decreased H-reflex. Palmieri et al¹⁹ described the H-reflex as a measure of α -motoneuron pool excitability stemming from spinal afferent stimulation. In brief, low-voltage stimulation of skeletal muscle activates low-threshold spinal afferents originating in the muscle spindles. As the voltage is increased, the H-reflex amplitude (mV) rises to a peak. After the H-reflex peak amplitude is reached, increased mV stimulation results in a second EMG response: *the M-response*. As the M-response increases, the H-reflex decreases due to antidromic and orthodromic action potentials, producing a recruitment curve and the H:M ratio. The *H:M ratio* is the ratio of maximal muscle response (M-response) to motoneuron pool excitability (H-reflex peak).¹⁹

Furthermore, static stretching may cause a reduction in muscle activation as evidenced by a reduced H:M ratio²⁰ and decreased maximum voluntary contraction. The effects of static stretching on the H-reflex have been seen during, immediately after, and up to 60 minutes poststretching.^{18,21–23}

We accept the proposed mechanism of EAMC as a result of α -motoneuron pool hyperexcitability and elected to investigate static stretching as a perturbation to reduce the excitatory input from the muscle spindles to the α -motoneuron pool. To our knowledge, no authors have researched the acute effect of static stretching on CTF.

Therefore, the purpose of our study was to compare the effects of acute static stretching on CTF using a common stretching duration as recommended by the American College of Sports Medicine.²⁴ We hypothesized that acute static stretching would increase participants' CTF.

METHODS

Study Design

A randomly assigned pretest-posttest crossover design was applied to determine whether acute static stretching (SS) affected the CTF of the flexor hallucis brevis (FHB) and whether that effect was greater than in the control (no-stretching [NS]) group. Each participant acted as his or her own control by completing both conditions. The dependent variable was the CTF, which is the lowest measure of hertz (Hz) required to elicit a cramp.

Participants

A convenience sample of 17 college-aged students was recruited to participate in the study. The participants were all healthy volunteers with no known health conditions. Exclusion criteria were a self-reported injury to the dominant limb within the previous 6 months¹⁷; any self-reported metabolic, neuromuscular, or neurologic disease or disorder²⁵; or pregnancy.²⁶ This project was reviewed and approved by the institutional review board and individuals provided written consent before participating.

Procedures

Recruits were asked to avoid participating in strenuous lower body exercise within 24 hours of the scheduled session. They were instructed to report for testing on 4 occasions separated by 7 days. The first 2 sessions (days 1 and 2) were familiarization days, which allowed the participant to become accustomed to the cramping procedure before being assigned to the testing order. At the end of day 2, each participant was randomly assigned to 1 of 2 conditions: SS or NS.

Day 1 (familiarization 1) consisted of signing of consent forms, collection of anthropometric data, and identification of the dominant limb. The dominant limb was determined by asking the participant to kick an imaginary ball.⁷ Day 1 concluded with an EIMC. The second visit to the lab (familiarization 2) consisted only of 1 EIMC protocol.

Days 3 and 4 were testing days. The conditions were counterbalanced to account for an order effect between days 3 and 4. Participants began their predetermined testing order by receiving either NS on day 3 and SS on day 4 or SS on day 3 and NS on day 4. On each testing day, an EIMC was elicited to establish the pretest CTF, followed by a 10-minute break. After the break, the participant underwent the assigned condition (NS or SS), followed by a second EIMC (posttest CTF).

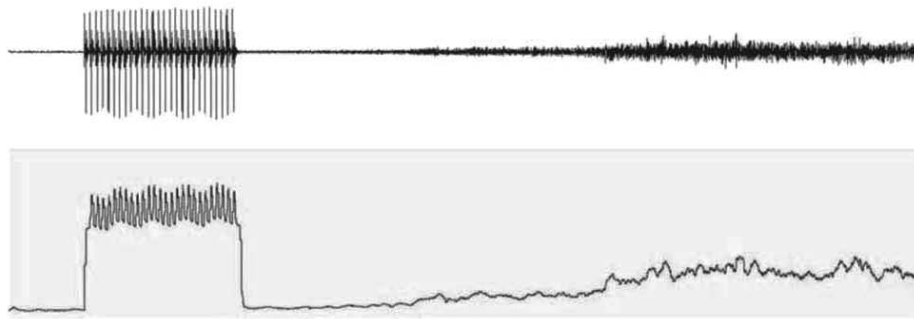
The SS protocol consisted of 3 bouts of 30-second passive static stretching of the FHB, administered by the tester, separated by 30 seconds of rest. The 3 bouts of stretching totaled 90 seconds, and the entire procedure lasted 3 minutes. The range of the stretch was determined by the participant and his or her perception of mild discomfort,²⁷ also known as the *point of discomfort* (POD). Posttest CTF was assessed immediately after the intervention ended. The CTF protocol began at 10 Hz below the first CTF: the starting Hz had no effect on CTF, thereby reducing the number of stimulations a participant experienced.²⁸ During the NS condition, the participant remained supine and relaxed for the 3 minutes between the baseline EIMC and the second EIMC. Total time was 13 minutes. Participants received 6 cramp-induction protocols during their 4 laboratory visits as follows:

- Day 1: Familiarization CTF
- Day 2: Familiarization CTF
- Day 3: Pretest CTF, NS or SS, posttest CTF
- Day 4: Pretest CTF, NS or SS, posttest CTF

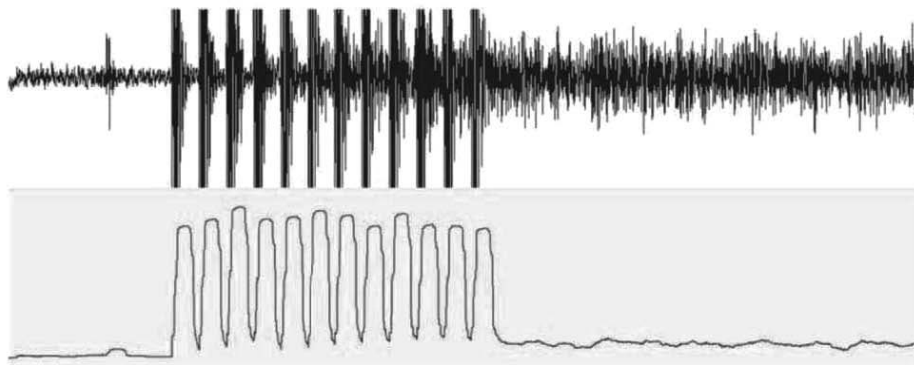
Instruments and EIMC Process

For inducing EIMCs, the FHB is the most commonly used muscle, due to the intersession and intrasession reliability, participant comfort, reproducibility of the cramp, and ease of access to the peripheral nerve.^{3–5} Participants were instructed to lay supine with the ankle and knee slightly elevated by numbered foam pads. Each participant received the same foam pads during each visit. The foam pads were used to ensure similar positions on each testing day. Participants were instructed to look at the ceiling and were given headphones and access to music to eliminate noise and distracting stimuli.⁵ To induce the cramp, we placed the 8-mm silver-silver chloride stimulating electrode (model EL 258S; Biopac Systems, Inc, Goleta, CA) slightly inferior and posterior to the medial malleolus, so that the 8-cm dispersive

Participant 96936 Pre-SS



Participant 19461 Post-SS



Participant 49789 Pre-NS

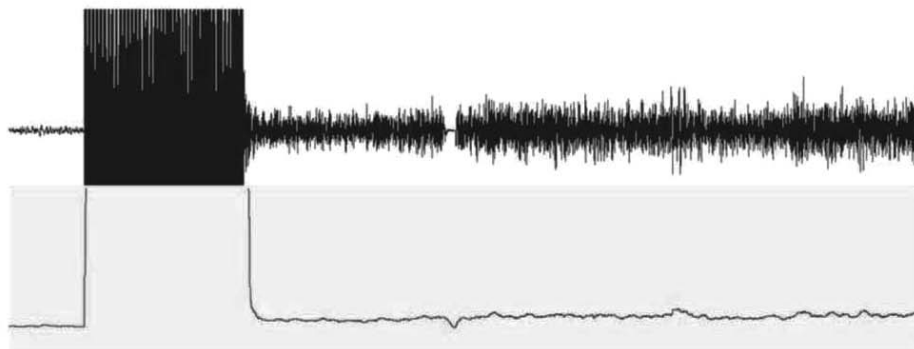


Figure 1. Electromyography and root mean square data for 3 participants. The top graph shows the raw electromyographic data at rest, stimulation, and during the electrically induced muscle cramp. The bottom graph shows the root mean square data during the same time frame.

pad was placed over the lateral malleolus. The tibial nerve was then stimulated with an 80-V, 1-millisecond electrical stimulation until full flexion of the FHB was visible. Once the appropriate tibial nerve location was identified, we marked it using a permanent marker. (Participants were asked to reapply the marking if they noticed fading.) The electrode and the dispersive pad were secured using a tight-fitting ankle brace.

To determine the CTF, we set the stimulator (model S88 REV K; Grass Instruments Company, West Warwick, RI) at 2-second trains, 80 V, and 6 pulses per second (pps or Hz). Therefore, at the start of stimulation, there would be a 1-second pause and then a 2-second stimulation period with 6 pulses of 80 V. If no cramp was elicited at 6 Hz, then 2 Hz was added, and stimulation was reapplied after a 1-minute break. The process of adding 2 Hz and giving a 1-minute

break between stimuli was continued until a cramp was elicited. The lowest value at which a cramp was elicited was termed the CTF.⁷ Similar methods are described in detail elsewhere.^{3,4,29} The stimulus was routed through a stimulation unit (model SIU8T; Grass Instruments Company). Anthropometric data were collected using a medical scale and stadiometer (model 217 stadiometer and model 869 scale; Seca Corp, Chino, CA). All EIMCs were confirmed via EMG analysis (version 4.0.0; BSL Analysis, Biopac Systems, Inc) following the previous literature^{3,30} and are reported in Figure 1 and the Appendix.

Statistical Analysis

Descriptive statistics were calculated for study participants' anthropometric values. Before performing the

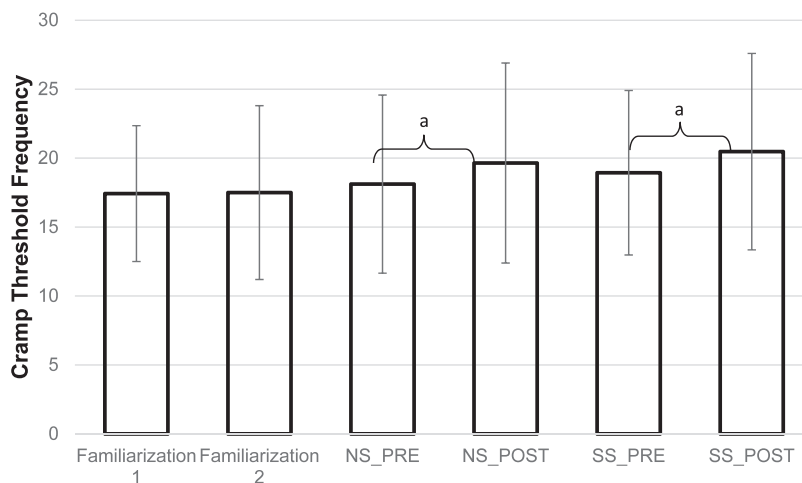


Figure 2. The mean cramp threshold frequency (CTF) for each day and condition. No-stretching (NS) condition pretest CTF = 18.12 ± 6.46 Hz, posttest CTF = 19.65 ± 7.25 Hz. Static stretching (SS) condition pretest CTF = 18.94 ± 5.96 Hz, posttest CTF = 20.47 ± 7.12 Hz. ^a Denotes differences between pretest and posttest within the NS and SS conditions ($P \leq .05$).

between-conditions statistical analysis, we conducted confirmatory modified t tests³¹ to identify whether a carryover, period effect, or order effect was present in this crossover design. Before analysis, the data from the 4 identified “crampers” (see definition in next section) were separated from the rest of the data and tested, with and without the modified t test, to verify their inclusion in the group analysis. Participants were then randomized to either NS and then SS or SS and then NS. The single-group crossover design requires 2 within-subject analyses and a between-subjects factor analysis to account for the change in treatment (NS to SS or the converse). The modified t test was also used to assess the change in CTF between the NS and SS conditions. A paired t test was calculated to determine whether statistically significant within-subject differences existed between the pretest and posttest CTF values in each condition. A 1-way analysis of variance (ANOVA) was used to evaluate the differences between the familiarization days and the pretest CTF on each testing day. The paired t test and 1-way ANOVA were determined using SPSS (version 23.0; IBM Corp, Armonk, NY) with significance set at $P < .05$. All data are presented as mean ± standard deviation.

RESULTS

All participants self-reported compliance with the instructions before the experimental days. Participants’ anthropometrics were as follows: age = 23 ± 3.3 years, height = 174.10 ± 13.60 m, and weight = 80.67 ± 24.75 kg. Of the 17 participants, 4 were self-reported “crampers.” They did not demonstrate pretest, posttest, or pretest-posttest differences from the self-reported noncrampers in either the standard t test ($P \geq .20$) or modified t test for carryover effect ($P = .16$) or a difference between treatment effects ($P = .48$). Therefore, the “crampers” were included in the group analysis. The confirmatory analysis did not identify a carryover or period effect ($t_{15} = 1.699$, $P = .11$). No within-subject effects were present between familiarization days and pretest CTFs of each condition ($P \geq .461$). Specifically, recorded CTFs were not different between familiarization days 1 and 2 (17.43 ± 4.93 Hz and 17.50 ±

6.30 Hz, respectively; $P = .68$). No difference occurred between the pretest CTFs of the NS and SS conditions (18.12 ± 6.46 Hz and 18.94 ± 5.96 Hz, respectively; $P = .702$). However, CTFs increased from pretest to posttest for both the NS and SS conditions (Figure 2). No change in CTF between the SS and NS conditions was found ($t_{15} = 0.035$, $P = .97$).

DISCUSSION

Our results show that static stretching increased CTF. However, the control condition also demonstrated an increase in CTF. No difference was evident between the conditions ($P = .97$). To our knowledge, no previous authors have examined the effects of static stretching as a preventive measure to reduce the likelihood of an EIMC as a proxy³² for EAMCs.

These data present static stretching as an ineffective intervention for decreasing the likelihood of a subsequent EIMC, as represented by no difference between the SS and NS conditions. However, static stretching and EAMC have been associated in marathon runners.¹ We are the first to directly test the effects of a stretching modality on EIMCs. An increase in CTF is thought to indicate a lower risk of EAMC occurrence because individuals prone to cramping have lower CTFs.^{3,30} Yet the CTF difference in the control (NS) condition from pretest to posttest is at odds with previously published data^{3,30} resulting from the same protocol. Miller and Knight³ reported a decrease in CTF from day 1 to day 2 in individuals with a history of cramps, whereas Stone et al³⁰ demonstrated no difference between pretest and posttest control CTFs.³⁰ Miller and Knight³ noted CTFs of 14.9 ± 1.3 Hz for “crampers,” which is consistent with our data (the average CTF for all 4 interventions was 15.275 ± 2.99 Hz). The CTF for the noncrampers was 25.5 ± 1.5 Hz, which is slightly higher than our data (20.50 ± 1.21 Hz). Stone et al³⁰ observed CTFs of 18 ± 4.9 Hz pre-exercise, of 20.0 ± 7.7 Hz pre-fatigue, and of 32.9 ± 11.7 Hz postfatigue.

Overall, the reason for the difference in the CTF standard deviation is unknown. However, one potential explanation could be due to different methods. Stone et

al³⁰ administered the highest tolerable voltage, starting at 80 V, and then started to perturb the frequency. We used the method reported by Miller and Knight,³ who did not alter the stimulus voltage. Yet we report a higher standard deviation than Miller and Knight³ despite using the same protocol; this may be due to the unknown cause of EAMCs or to the static-stretching perturbation. In the latter case, the location of the metatarsophalangeal joint may have limited the range of motion of the stretch, leading to some participants experiencing an insufficient intensity of stretch on the FHB. We designed our study to use a stretch to the POD, with the aim of inhibiting the α motoneuron pool, but the clinician administering the static-stretching condition may not have been able to reach a participant's POD. All participants were asked if they could feel a stretch of the muscles in their feet, which they confirmed. During observations of the participants, we had no obvious indication that the POD was reached, and testers were not comfortable applying additional force to the first digit due to the amount of force already applied. If the static stretch was inadequate, then the inferred mechanism may not have been tested. Moreover, new evidence emerged after the data were collected suggesting that static stretching may not elicit increased inhibition after 3 bouts of 1-minute static stretching of the gastrocnemius.³³ Another explanation from our data could be an order effect. The modified *t* test, which was used to control for the specific order used in the crossover design, provided a value of 1.71 with a *P* value of .055. It is plausible that an increase in participants could lead to an order effect, which may help explain these results.

Clinically, these data may support the use of other forms of stretching and warm-up protocols such as dynamic stretching and proprioceptive neuromuscular facilitation stretching that aim to improve performance. Static stretching may not reduce the incidence of EAMCs as previously suggested.¹

Future researchers should investigate biarticulate muscles, which are known to be more susceptible to cramping,¹ and include a measure of the H-reflex poststretch to ensure the stretching perturbation elicits the desired response. In prospective EIMC research studies, investigators should test additional intervention strategies believed to alter neuromuscular function in order to determine their effect on CTF. Testing different muscles may also offer additional insight into skeletal muscle cramping.

In conclusion, our study offers evidence that acute static stretching of the FHB, within the flexibility guidelines of the American College of Sports Medicine, had an acute effect on CTF. However, this effect was not significant when compared with the control condition. Although the same volume of stretching has been reported to cause neural, mechanical, and performance alterations in humans, it failed to result in a significant treatment effect, when compared with controls, in this study. These results are validated by similar same-day EIMC protocol differences³⁴ and day-to-day changes in participants' CTFs⁵ in studies that did not involve a perturbation. Hence, our day-to-day pattern of CTF changes mimicked those of previous researchers who did not perturb CTF. Thus, we provide evidence that acute static stretching had no effect on CTF in an EIMC model and suggest that static stretching is an ineffective intervention to acutely reduce the likelihood of future EAMCs.

Appendix. Root Mean Squares: Average Resting and Electrically Induced Muscle Cramps^a Continued on Next Page

Participant No.	Before Static Stretching		After Static Stretching	
	Resting RMS ± SD	Cramp RMS ± SD	Resting RMS ± SD	Cramp RMS ± SD
Control Condition				
19461	0.11 ± 0.02	0.75 ± 0.20	0.12 ± 0.05	0.37 ± 0.10
96936	3.69 ± 1.25	55.10 ± 29.42	3.69 ± 0.80	61.98 ± 37.21
52466	9.49 ± 2.13	33.98 ± 27.31	9.57 ± 2.22	46.32 ± 25.28
53124	3.09 ± 1.04	37.68 ± 20.44	3.87 ± 1.37	20.35 ± 52.92
86112	5.24 ± 0.98	21.45 ± 13.41	5.81 ± 2.00	58.31 ± 31.33
25517	2.82 ± 1.10	13.91 ± 10.26	3.66 ± 1.35	33.41 ± 40.57
34819	9.47 ± 2.69	20.70 ± 19.69	9.83 ± 2.83	24.41 ± 35.17
62981	6.16 ± 1.15	37.63 ± 12.75	3.16 ± 0.84	26.16 ± 46.45
14982	4.21 ± 0.97	16.01 ± 9.47	4.99 ± 1.78	26.98 ± 15.32
49789	5.64 ± 1.47	38.42 ± 32.77	4.19 ± 1.24	29.94 ± 30.83
85418	5.82 ± 1.37	26.33 ± 7.79	4.97 ± 1.34	74.41 ± 40.65
17191	12.88 ± 2.82	29.72 ± 24.95	10.76 ± 2.90	46.54 ± 33.07
38643	5.65 ± 1.39	23.64 ± 8.61	4.47 ± 1.07	45.67 ± 23.80
96613	6.61 ± 1.54	42.21 ± 28.06	9.44 ± 3.09	44.10 ± 63.62
73991	9.33 ± 1.56	27.04 ± 52.59	4.96 ± 1.74	31.95 ± 47.57
12468	7.73 ± 1.22	123.93 ± 41.68	8.44 ± 2.10	74.65 ± 34.43
85357	6.71 ± 1.80	41.05 ± 25.27	12.11 ± 3.83	25.05 ± 26.71
Average	6.16 ± 1.44	34.68 ± 21.45	6.12 ± 1.80	39.45 ± 34.41
SD	3.05 ± 0.66	26.33 ± 13.44	3.28 ± 0.96	19.88 ± 14.62

Participant No.	Before Static Stretching		After Static Stretching	
	Resting RMS ± SD	Cramp RMS ± SD	Resting RMS ± SD	Cramp RMS ± SD
Static-Stretching Condition				
19461	0.11 ± 0.02	0.75 ± 0.20	0.12 ± 0.05	0.37 ± 0.10
96936	3.69 ± 1.25	55.10 ± 29.42	3.69 ± 0.80	61.98 ± 37.21
52466	9.49 ± 2.13	33.98 ± 27.31	9.57 ± 2.22	46.32 ± 25.28
53124	3.09 ± 1.04	37.68 ± 20.44	3.87 ± 1.37	20.35 ± 52.92
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62981	6.16 ± 1.15	37.63 ± 12.75	3.16 ± 0.84	26.16 ± 46.45
14982	4.21 ± 0.97	16.01 ± 9.47	4.99 ± 1.78	26.98 ± 15.32
49789	5.64 ± 1.47	38.42 ± 32.77	4.19 ± 1.24	29.94 ± 30.83
85418	5.82 ± 1.37	26.33 ± 7.79	4.97 ± 1.34	74.41 ± 40.65
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85357	6.71 ± 1.80	41.05 ± 25.27	12.11 ± 3.83	25.05 ± 26.71
Average	6.16 ± 1.44	34.68 ± 21.45	6.12 ± 1.80	39.45 ± 34.41
SD	3.05 ± 0.66	26.33 ± 13.44	3.28 ± 0.96	19.88 ± 14.62

Abbreviations: EIMCs, exercise-induced muscle cramps; RMS, root mean square; SD, standard deviation.

^a One way to verify EIMCs is by cramp intensity. If the EIMC intensity is above 2 SDs of the resting RMS, then the EIMC is considered valid.

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