Perceived Pain Responses to Foam Rolling Associate with Basal Heart Rate Variability

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Background: Foam rolling (FR) is a self-myofascial release technique with unclear effects on autonomic functioning, indexed by heart rate variability (HRV). FR can be perceived as painful or relaxing, which may explain interindividual HRV responses.

Purpose: To determine if acute FR alters resting HRV. A secondary aim was to determine if perceived pain during FR would predict HRV responses.

Setting: Academic institution.

Methods: In a randomized, crossover design, healthy adults (50% female) performed total body FR or control on separate days. Perceived pain ratings were obtained following FR of each muscle group and summed to generate an overall perceived pain rating. Seated measures of the mean RR interval and the natural logarithm of the root-mean square of successive RR interval differences (LnRMSSD, a parasympathetic HRV index) were obtained at 5-10 min pre-, 5-10 min post-, and 25-30 min post-FR.

Results: No effects were observed for RR interval (p = .105–.561) or LnRMSSD (p = .110–.129). All effect sizes ranged from trivial–small (0.00–0.26). Changes in RR interval (r = 0.220–0.228, p = .433–.488) and LnRMSSD (r = 0.013–0.256, p = .376–.964) were not associated with pain scale sum. Baseline LnRMSSD was associated with pain scale sum (r = -0.663; p = .001).

Conclusion: FR did not systematically alter HRV, nor did perceived pain ratings predict HRV responses. Those with lower pre-FR HRV reported higher perceived pain during FR. Basal cardiac autonomic activity may, therefore, influence pain sensitivity to FR in healthy adults.

KEYWORDS: self-myofascial release; cardiac autonomic; rolling massage

INTRODUCTION

The physiological stress response is partly regulated by the sympathetic (i.e., fight or flight) and parasympathetic (i.e., rest and digest) branches of the autonomic nervous system, which helps to maintain homeostasis through the coordination of diverse physiological systems.[1] Heart rate variability (HRV), defined as the variation in time between successive heartbeats, is a simple, non-invasive, and cost-effective tool to evaluate autonomic functioning as a means to gain insight into stress recovery and physical activity readiness.[2] Depressed HRV reflects autonomic imbalance shifted in the sympathetic direction. Short-term reductions in HRV have been associated with stress and fatigue,[3] whereas chronically suppressed values are associated with various pathological conditions[4] and greater all-cause mortality risk.[5] Contrastingly, higher HRV is associated with superior health and longevity.[6] As such, regular (e.g., daily) tracking of HRV is becoming increasingly popular in healthy,[7] athletic,[8] and clinical populations alike.[9] Moreover, there is great interest in lifestyle behaviors and interventional strategies to mitigate physical stress via activation of the parasympathetic nervous system, reflected by an increase in HRV.[10]

Massage is an intervention conventionally used to decrease muscle tension[11] that has shown promise as a means to modulate autonomic stress via stimulation of cardiac-parasympathetic activity under resting[12,13] or postexercise conditions.[14] However, massage may be inaccessible for many due to prohibitive costs or exorbitant time requirements. Whether more readily available bodywork alternatives may similarly reduce physiological stress, reflected

by increased parasympathetic activity and thus HRV, remains to be determined.

Self-myofascial release techniques are commonly used by athletes and wellness enthusiasts as a means to enhance recovery through a reduction in pain and inflammation.[15] Foam rolling is among the most popular forms of self-myofascial release, and involves a series of body weight exercises intended to apply compressive forces to a specific muscle or group of muscles using a high-density foam cylinder. Previous research has provided evidence for the efficacy of foam rolling as a means to increase joint range of motion[16] and reduce delayed onset muscle soreness.[17] However, research on the cardiac autonomic influence of foam rolling is limited and has produced mixed results. Though evidence for an increase in parasympathetic activity following foam rolling has been observed,[18] other studies have shown no significant effects.[19] One possible explanation for these inconsistent findings may be that interindividual cardiac autonomic responses to muscular trigger point compressions may be influenced by pain sensation,[20] where foam rolling may reduce HRV among pain-sensitive individuals via nociceptor-mediated sympathetic activation.[21] However, associations between cardiac autonomic activity and perceived pain ratings in response to foam rolling have yet to be investigated.

Given the similarities between passive massage and self-myofascial release using a foam roller, an improved understanding of whether foam rolling may serve as a cost- and time-effective alternative to reduce stress via autonomic modulation is warranted. The purpose of the present study was to determine if an acute foam rolling session affects resting cardiac parasympathetic activity, measured using HRV. A secondary aim was to evaluate the hypothesis that subjective ratings of pain during the foam rolling session would predict HRV responses.

METHODS

Participants

The smallest worthwhile change threshold commonly used in HRV-guided exercise training interventions is ± 0.5 of the baseline standard deviation.[22] To detect an effect size of 0.5 with 80% power and α of 0.05, it was determined that a minimum total of 10 subjects was required (G*Power software version 3.1.9.4; Franz Faul, Heinrich-Heine-Universität, Düsseldorf, Germany). Thus, 15 healthy and recreationally active young adults were recruited for this study via convenience sampling. One subject was excluded due to an irregular heart rhythm. Thus, 14 volunteers were included in the final analysis (50% female; age = 25.86 ± 2.13 yrs.; height = 172.04 ± 12.82 cm, weight = 84.16 ± 16.90 kg). All participants reported having performed foam rolling exercise within the past 30 days and were free from cardiovascular, metabolic, and orthopedic disorders. Subjects reported not taking any heart rate-altering medications (e.g., β-blockers or β2-2 agonists). The study was approved by the University's Institutional Review Board. All study procedures, risks, and benefits were explained to the participants and written informed consent was obtained prior to participation.

Study Design

Using a randomized cross-over design, participants reported to the laboratory for two visits, each of which was separated by a minimum of 48 hrs but no more than seven days, and were scheduled at the same time of day to control for diurnal variation in HRV.[23] Participants performed a bout of foam rolling or simulated control in random order. Perceived pain ratings were obtained throughout the foam rolling intervention and summed intraindividually. Electrocardiographic (ECG) recordings were performed for 10 min immediately before and for 30 min after each condition.

Procedures

Intervention

The intervention consisted of an acute foam rolling session lasting ~7 min that was performed using a moderate density foam roller (AXIS Standard 91.4 cm × 15.2 cm, OPTP, Minneapolis, MN). The single-session foam rolling intervention targeted major muscle groups throughout the body in the following sequence, performed for one series: 1) lower posterior leg, 2) knee flexors, 3) knee extensors, 4) elbow extensors, 5) latissimus dorsi, and 6) upper back. Subjects started the foam rolling session on the right side of the body and proceeded to roll the specified muscle group for 30 sec before switching to their left side. For

each muscle, participants were instructed to roll back and forth at a self-selected pace in a direction parallel to the muscle architecture. Constant tension was applied throughout and the process was repeated for each group of muscles. For the control condition, participants were asked to simulate the foam rolling exercises without the use of the foam roller. The simulation involved performing movements and isometric holds in positions that mimic the foam rolling exercises for the same time duration to control for the effects of physical activity on changes in heart rate parameters. For example, subjects would hold a push-up position while balancing on their left leg to mimic foam rolling the right knee extensors.

Perceived pain

Immediately after foam rolling an individual muscle group, participants were asked to provide a perceived pain rating relative to passive sitting using the Wong-Baker Faces Pain Scale.[24] The pain scale included scores ranging from 0 to 10, with higher values indicating greater pain. Muscle site pain ratings were added together for the pain scale sum.

Heart rate variability

Upon arrival, participants were taken to a quiet, temperature-controlled laboratory where they were outfitted with a clinically-validated, single-lead ECG sensor (Bittium Faros 180, 1000 Hz, Oulu, Finland).[25] Participants remained quiet and still, and breathed spontaneously throughout data acquisition. R-R intervals were extracted from ECG recordings in 5 min segments immediately before the intervention (T1, following a 5-min stabilization period), 5-10 min post- (T2), and 25-30 min post-foam rolling or control (T3). R-R data were exported to Kubios™ software (University of Kuopio, Finland) for manual inspection and filtering of artifact and ectopic beats. The mean RR interval was obtained and the natural logarithm of the root-mean square of successive R-R interval differences (LnRMSSD) was computed as the vagally-mediated HRV parameter for analysis.[26] Participants were instructed to avoid any food or drink for a minimum of 2 hrs prior to each visit, and to avoid caffeine and exercise the day of each trial. Vigorous muscle-damaging exercise, such as resistance training, was discouraged for 48 hrs prior to each visit to avoid the influence of delayed-onset muscle soreness on perceived pain ratings.

Statistical Analysis

Normality of the standardized residuals for RR interval and RMSSD were assessed with Shapiro-Wilks Tests. Natural logarithm (Ln) transformations were applied to the nonnormally distributed RMSSD values (p <.05). Linear mixed models were used to examine variation in RR interval and Ln-RMSSD. Condition (Foam Roll vs. Control) was included as a fixed effect, time (T1 vs. T2 vs. T3) as a repeated fixed effect, condition x time as an interaction effect, and subject identification as a random effect. Hedges' g effect sizes were calculated to determine standardized differences between mean values.[27] Effect sizes (ESs) were qualitatively interpreted as follows: <0.20 = trivial, <0.60 = small, <1.20 = moderated, <2.0 = large, and >2.0 = very large.[28] Associations between pain scale sum and cardiac autonomic parameters (baseline and relative changes from baseline) were quantified with Pearson product-moment correlations. Statistical significance was set at $p < .05$. Statistical procedures were carried out using JMP 13 (SAS Institute Inc., Cary, North Carolina, USA).

RESULTS

Model Effects

RR interval and LnRMSSD means and standard deviations are provided in Table 1. No significant model effects were observed for RR interval (p = .105–.561) or LnRMSSD (p = .110–.129). All ESs ranged from trivial – small (ES = 0.00–0.26). Moreover, visual inspection of individual RR interval and LnRMSSD values showed no evidence of a responder vs. nonresponder phenomenon (Figure 1).

Associations

Baseline LnRMSSD was significantly associated with pain scale sum (r = -0.663; p = .001) whereas baseline RR interval was not (r = -0.394, p = .163) (Figure 2). No associations were observed between changes in RR interval (r = 0.220–0.228, p = .433–.488) or LnRMSSD (r = 0.013–0.256, p = .376–.964) and pain scale sum in response to foam rolling.

TABLE 1. Mean and Standard Deviation for:
TABLE 1(a). Condition

	Control	*Foam Roll*	*p, ES*
RR Interval (ms)	857.7 ± 141.9	858.1 ± 109.5	.56, 0.00
LnRMSSD	3.83 ± 0.54	3.95 ± 0.53	.11, 0.22

TABLE 1(b). Time

	T1	*T2*	*T3*	*T1 vs. T2 p, ES*	*T1 vs. T3 p, ES*	*T2 vs. T3 p, ES*
RR Interval (ms)	861.2 ± 137.8	842.9 ± 122.6	860.8 ± 119.9	.12, -0.14	.95, 0.00	.21, 0.14
LnRMSSD	3.93 ± 0.55	3.87 ± 0.54	3.86 ± 0.53	.24, -0.11	.10, -0.13	.88, -0.02

TABLE 1(c). Condition × Time

	T1	*T2*	*T3*	*T1 vs. T2 p, ES*	*T1 vs. T3 p, ES*	*T2 vs. T3 p, ES*
RR Interval (ms)						
Foam Roll	873.2 ± 117.3	842.9 ± 112.2	858.1 ± 104.9	.14, -0.26	.80, -0.13	.79, 0.14
Control	849.3 ± 159.3	842.9 ± 136.4	863.8 ± 138.6	.99, -0.04	.96, 0.09	.76, 0.15
Foam Roll vs. Control *p*, ES	.66, -0.17	1.00, 0.00	1.00, 0.05			
LnRMSSD						
Foam Roll	4.03 ± 0.52	3.88 ± 0.56	3.94 ± 0.55	.11, -0.27	.65, -0.16	.85, 0.10
Control	3.84 ± 0.59	3.87 ± 0.52	3.77 ± 0.52	.99, 0.05	.83, -0.12	.49, -0.19
Foam Roll vs. Control *p*, ES	.29, -0.33	.99, -0.02	.40, -0.31			

LnRMSSD = natural logarithm of the root mean square of successive R-R interval differences; T1 = time point 1 (baseline); T2 = time point 2 (5–10 min post-foam rolling or control); T3 = time point 3 (25–30 min post-foam rolling or control); ES = effect size.

DISCUSSION

In this randomized, crossover study, we observed no effect of an acute foam rolling session on resting RR interval or vagally-mediated HRV in young and recreationally active men and women. Meanwhile, a significant, inverse relationship between pain scale sum and baseline LnRMSSD was observed, portending to a link between activity of the Vagus nerve and the perception of pain. Collectively, these findings highlight that self-myofascial release using a foam roller is an ineffective means of modulating autonomic stress via cardiac parasympathetic stimulation, but that resting vagal activity appears to influence pain perception.

Contrary to our hypothesis, a total body foam rolling session did not affect indices of cardiac autonomic activity. Initial research in this area demonstrated reductions in heart rate, blood pressure, and stress hormones (e.g., cortisol), concomitant with an increase in HRV following passive massage techniques.[29] Building upon these findings, a recent investigation provided evidence for greater parasympathetic modulation, indexed by increased normalized high frequency spectral power ($p < .01$) and reduced systolic and diastolic blood pressure, for up to 30 min following total body foam rolling in 15 healthy young adults.[18] Another study reported improvements in cardiovascular health markers (i.e., improved endothelial function

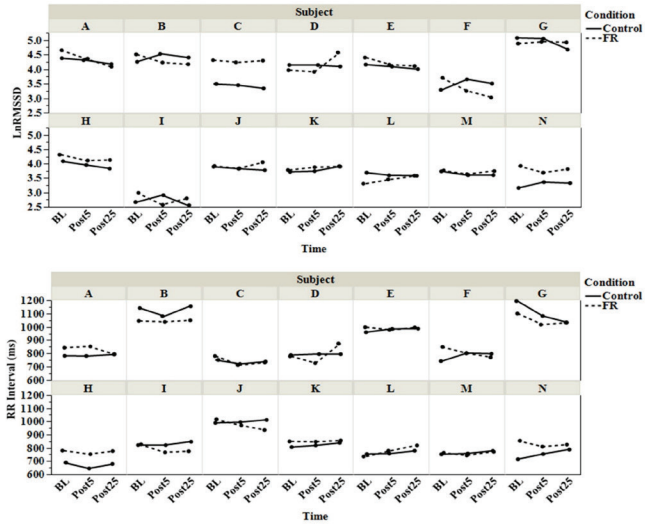

FIGURE 1. Individual RR interval and natural logarithm of the root mean square of successive differences (LnRMSSD) responses to foam rolling (FR) vs. control.

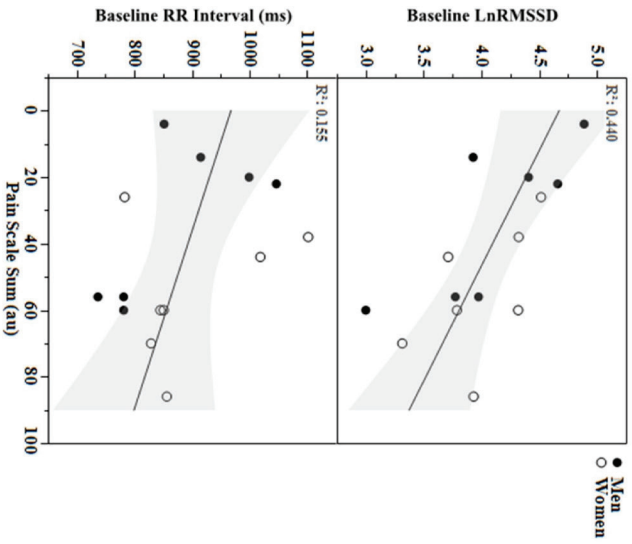

FIGURE 2. Association between pain scale sum and baseline RR interval and natural logarithm of the root mean square of successive RR interval differences (LnRMSSD).

assessed by plasma nitric oxide concentrations, reduced arterial stiffness indexed by pulse-wave velocity) following a foam rolling intervention relative to control (n = 10 healthy young adults, $p < .05$).[30] Although lacking a control condition, similar findings have recently been reported that showed reductions in central blood pressure indices and arterial stiffness, predominantly at the 30 min time point following foam rolling ($p < .05$).[31] However, in agreement with the current findings, no changes in resting heart rate (i.e., RR interval) were observed ($p > .05$).[31] Also in support of the current findings, foam rolling was recently demonstrated to have no appreciable influence on indices of autonomic function (i.e., RMSSD and pulse wave velocity) following a demanding high-intensity sprint session.[19] Collectively, foam rolling seems to favorably augment hemodynamic parameters under resting conditions,[18,30,31] but its impact on centrally-mediated cardiac control remains equivocal.

Compared to therapeutic massage, the more active nature of foam rolling exercise inherently necessitates a transient withdrawal of parasympathetic modulation that may be anticipated to acutely depress HRV. An increased oxygen demand as a result of both dynamic and isometric skeletal muscle activation to support one's body mass whilst foam rolling, stimulates an increase in respiration and heart rate. However, given the low intensity and short duration of our foam rolling intervention, in combination with the recreationally active nature of our participants, complete recovery of HRV would be anticipated by our 30 min time point (i.e., T3).[32] Nevertheless, passive relaxation during therapeutic massage may explain observations of acute reductions in HR and improvements in HRV.[12,33] Other notable differences that may amplify parasympathetic modulation from massage include the use of aromatic oils,[34] and potential massage-induced increases in oxytocin and reductions in adrenocorticotropic hormone and cortisol,[35,36] that do not occur with foam rolling.[37] In addition, manual techniques by trained therapists may be more effective at targeting trigger points, which has been shown to increase vagal HRV parameters.[20] Further identification of the mechanism(s) underlying the therapeutic differences between passive massage and self-myofascial release using a foam roller may help to guide future interventions seeking to increase the efficacy of foam rolling as a strategy to mitigate autonomic stress.

To aid in the discovery of mechanisms responsible for HRV responses to foam rolling, we were the first to query participant discomfort via pain scale summation. The null association between this metric and relative changes in HRV at either postintervention time point implies that the lack of observed cardiac parasympathetic benefit was not a product of excess nociceptor stimulation. Contrasting with our finding,

changes in perceived pain via visual analog scale in response to manual trigger point relief of the neck was associated ($n = 21$, $r^2 = 0.272$, $p < .05$) with high frequency spectral power (a vagal-related HRV index).[20] It was hypothesized that overuse of muscles builds an excess of acetylcholine at the motor endplate, causing a sustained contractile state in muscle fibers (i.e., knots), which leads to local ischemia- or hypoxia-induced pain.[20] Alleviation of pain through trigger point relief is thought to be a result of reduced sympathetic activity, which may increase peripheral blood flow, promote removal of noxious byproducts, and blunt excess discharge of acetylcholine.[20] An alternative rationale for our lack of an association may be explained by pain being more commonly associated with indices of sympathetic baroreflex activity more so than vagal-related HRV.[21] Additionally, effects of pain on cardiovascular and autonomic parameters may only be transient[21] and, thus, uncaptured after 5 min of seated rest post-foam rolling in the current protocol.

Intriguingly, baseline LnRMSSD was significantly associated with the magnitude of pain sensation among the current sample. The association between chronic pain and reduced vagal-related HRV is well established.[38] However, few studies have investigated the association between basal HRV and pain responses.[21] It's been shown that a derivative of high-frequency spectral power predicted perceived pain intensity (0–10 scale) among conscious burn patients undergoing scheduled wound treatment.[39] Contrastingly, greater low-frequency spectral power (reflective of baroreflex activation) was associated with lower ratings of unpleasantness, but not pain intensity, during thermal hand pain (4°C cold plate exposure),[40] and high-frequency power showed no predictive ability for any of the pain-related outcomes.[40] Though HRV was not assessed, a previous investigation reported that vagal nerve stimulation via an implantable device suppressed pain scale ratings among 10 epileptic patients undergoing experimentally induced pain.[41] Interpreted together, these findings allude to a potential roll for targeting the parasympathetic nervous system for pain management.

Strengths of this study include our randomized, crossover design, as well as the inclusion of both male and female participants with a recent history of foam rolling. Unlike previous research in the field, our participants performed the foam rolling exercise at a self-selected pace, which may enhance external validity. However, interindividual differences in foam rolling speed are acknowledged as a limitation of the present investigation. Furthermore, whether similar HRV responses would be observed among individuals without foam rolling experience is unclear. Though foam rolling sessions were consistently monitored by the lead investigator, compressive forces on the foam roller could be somewhat self-regulated by participants. Thus, subtle modifications to body positioning could have alleviated foam roller-induced discomfort, effecting perceived pain ratings. Further investigation is therefore warranted to support the current findings, particularly in regard to HRV and pain sensitivity. Finally, the study was underpowered for detecting smaller effects than those which were targeted, which is another limitation.

The major finding of the present investigation was that a total body foam rolling session failed to influence vagally-mediated HRV or resting mean RR interval in healthy, recreationally active young adults. The lack of association between self-reported pain and changes in HRV or RR interval following foam rolling exercise suggests that pain-related factors were not likely responsible for interindividual difference in cardiac autonomic responses. However, our novel finding of an association between basal HRV and perceived pain in response to foam rolling suggests that parasympathetic activity may influence pain sensitivity.

ACKNOWLEDGMENTS

We thank the participants for volunteering their time to partake in the study. The study was conducted without funding.

CONFLICT OF INTEREST NOTIFICATION

The authors declare there are no conflicts of interest.

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REFERENCES

1. Ulrich-Lai YM, Herman JP. Neural regulation of endocrine and autonomic stress responses. *Nature Rev Neurosci*. 2009;10(6):397–409.
2. Stanley J, Peake JM, Buchheit M. Cardiac parasympathetic reactivation following exercise: implications for training prescription. *Sports Med*. 2013;43(12):1259–1277.
3. Kim HG, Cheon EJ, Bai DS, Lee YH, Koo BH. Stress and heart rate variability: a meta-analysis and review of the literature. *Psychiatry Investig*. 2018;15(3):235–245.
4. Vreijling SR, Troudart Y, Brosschot JF. Reduced heart rate variability in patients with medically unexplained physical symptoms: a meta-analysis of HF-HRV and RMSSD. *Psychosom Med*. 2021;83(1): 2–15.
5. Dekker JM, Crow RS, Folsom AR, Hannan PJ, Liao D, Swenne CA, et al. Low heart rate variability in a 2-minute rhythm strip predicts risk of coronary heart disease and mortality from several causes: the ARIC Study. *Circulation*. 2000;102(11):1239–1244.
6. Zulfiqar U, Jurivich DA, Gao W, Singer DH. Relation of high heart rate variability to healthy longevity. *Am J Cardiol*. 2010;105(8):1181–1185.
7. Kiviniemi AM, Hautala AJ, Kinnunen H, Nissila J, Virtanen P, Karjalainen J, et al. Daily exercise prescription on the basis of HR variability among men and women. *Med Sci Sports Exerc*. 2010;42(7):1355–1363.
8. Flatt A, Allen J, Bragg A, Keith C, Earley R, Esco M. Heart rate variability in college football players throughout preseason camp in the heat. *Int J Sports Med*. 2020;41(09):589–595.
9. Tulppo MP, Kiviniemi AM, Junttila MJ, Huikuri HV. Home monitoring of heart rate as a predictor of imminent cardiovascular events. *Front Physiol*. 2019;10.
10. Laborde S, Mosley E, Mertgen A. A unifying conceptual framework of factors associated to cardiac vagal control. *Heliyon*. 2018;4(12):e01002.
11. Buttagat V, Eungpinichpong W, Chatchawan U, Arayawichanon P. Therapeutic effects of traditional Thai massage on pain, muscle tension and anxiety in patients with scapulocostal syndrome: a randomized single-blinded pilot study. *J Bodywk Mov Ther*. 2012;16(1):57-63.
12. Buttagat V, Eungpinichpong W, Chatchawan U, Kharmwan S. The immediate effects of traditional Thai massage on heart rate variability and stress-related parameters in patients with back pain associated with myofascial trigger points. *J Bodywk Mov Ther*. 2011;15(1):15–23.
13. Delaney JP, Leong KS, Watkins A, Brodie D. The short-term effects of myofascial trigger point massage therapy on cardiac autonomic tone in healthy subjects. *J Adv Nurs*. 2002;37(4):364–371.
14. Arroyo-Morales M, Olea N, Martinez M, Moreno-Lorenzo C, Díaz-Rodríguez L, Hidalgo-Lozano A. Effects of myofascial release after high-intensity exercise: a randomized clinical trial. *J Manip Physiol Ther*. 2008;31(3):217–223.
15. Beardsley C, Škarabot J. Effects of self-myofascial release: a systematic review. *J Bodywk Mov Ther*. 2015;19(4):747–758.
16. Markovic G. Acute effects of instrument assisted soft tissue mobilization vs. foam rolling on knee and hip range of motion in soccer players. *J Bodywk Mov Ther*. 2015;19(4):690–696.
17. Pearcey GE, Bradbury-Squires DJ, Kawamoto JE, Drinkwater EJ, Behm DG, Button DC. Foam rolling for delayed-onset muscle soreness and recovery of dynamic performance measures. *J Athl Training*. 2015;50(1):5–13.
18. Lastova K, Nordvall M, Walters-Edwards M, Allnutt A, Wong A. Cardiac autonomic and blood pressure responses to an acute foam rolling session. *J Strength Cond Res*. 2018;32(10):2825–2830.
19. D'Amico A, Gillis J, McCarthy K, Leftin J, Molloy M, Heim H, et al. Foam rolling and indices of autonomic recovery following exercise-induced muscle damage. *Int J Sports Phys Ther*. 2020;15(3):429–440.
20. Morikawa Y, Takamoto K, Nishimaru H, Taguchi T, Urakawa S, Sakai S, et al. Compression at myofascial trigger point on chronic neck pain provides pain relief through the prefrontal cortex and autonomic nervous system: a pilot study. *Front Neurosci*. 2017;11:186.
21. Koenig J, Jarczok MN, Ellis RJ, Hillecke TK, Thayer JF. Heart rate variability and experimentally induced pain in healthy adults: a systematic review. *Eur J Pain*. 2014;18(3):301–314.
22. Javaloyes A, Sarabia JM, Lamberts RP, Moya-Ramon M. Training prescription guided by heart rate variability in cycling. *Int J Sports Physiol Perform*. 2019;14(1):23–32.
23. Huikuri HV, Kessler KM, Terracall E, Castellanos A, Linnaluoto MK, Myerburg RJ. Reproducibility and circadian rhythm of heart rate variability in healthy subjects. *Am J Cardiol*. 1990;65(5):391–393.
24. Garra G, Singer AJ, Taira BR, Chohan J, Cardoz H, Chisena E, et al. Validation of the Wong-Baker FACES Pain Rating Scale in pediatric emergency department patients. *Acad Emerg Med*. 2010;17(1):50–54.
25. Vandecasteele K, De Cooman T, Gu Y, Cleeren E, Claes K, Paesschen WV, et al. Automated epileptic seizure detection based on wearable ECG and PPG in a hospital environment. *Sensors*. 2017;17(10):2338.
26. Penttilä J, Helminen A, Jartti T, Kuusela T, Huikuri HV, Tulppo MP, et al. Time domain, geometrical and frequency domain analysis of cardiac vagal outflow: effects of various respiratory patterns. *Clin Physiol*. 2001;21(3):365–376.
27. Hedges LV. Distribution theory for Glass's estimator of effect size and related estimators. *J Edu Stat*. 1981;6(2):107–128.

28. Hopkins W, Marshall S, Batterham A, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc*. 2009;41(1):3.
29. Weerapong P, Hume PA, Kolt GS. The mechanisms of massage and effects on performance, muscle recovery and injury prevention. *Sports Med*. 2005;35(3):235–256.
30. Okamoto T, Masuhara M, Ikuta K. Acute effects of self-myofascial release using a foam roller on arterial function. *J Strength Cond Res*. 2014;28(1):69–73.
31. Ketelhut S, Möhle M, Hottenrott K. Acute effects of self-myofascial release using a foam roller on arterial stiffness in healthy young adults. *Artery Res*. 2020;26(4):219–222.
32. Michael S, Graham KS, Davis GMO. Cardiac autonomic responses during exercise and post-exercise recovery using heart rate variability and systolic time intervals—a review. *Front Physiol*. 2017;8:301.
33. Lindgren L, Rundgren S, Winsö O, Lehtipalo S, Wiklund U, Karlsson M, et al. Physiological responses to touch massage in healthy volunteers. *Auton Neurosci*. 2010;158(1-2):105-110.
34. Seifert G, Kanitz J-L, Rihs C, Krause I, Witt K, Voss A. Rhythmical massage improves autonomic nervous system function: a single-blind randomised controlled trial. *J Integrat Med*. 2018;16(3):172–177.
35. Field T, Hernandez-Reif M, Diego M, Schanberg S, Kuhn C. Cortisol decreases and serotonin and dopamine increase following massage therapy. *Int J Neurosci*. 2005;115(10):1397–1413.
36. Morhenn V, Beavin LE, Zak PJ. Massage increases oxytocin and reduces adrenocorticotropin hormone in humans. *Altern Ther Health Med*. 2012;18(6):11–18.
37. Kim K, Park S, Goo B-O, Choi S-C. Effect of self-myofascial release on reduction of physical stress: a pilot study. *J Phys Ther Sci*. 2014;26(11):1779–1781.
38. Tracy LM, Ioannou L, Baker KS, Gibson SJ, Georgiou-Karistianis N, Giummarra MJ. Meta-analytic evidence for decreased heart rate variability in chronic pain implicating parasympathetic nervous system dysregulation. *Pain*. 2016;157(1):7–29.
39. Papaioannou V, Chouvarda I, Gaertner E, Benyamina M, Ferry A, Maurel V, et al. Heart rate variability and cardiac baroreflex inhibition-derived index predicts pain perception in burn patients. *Burns*. 2016;42(7):1445–1454.
40. Appelhans BM, Luecken LJ. Heart rate variability and pain: associations of two interrelated homeostatic processes. *Biolog Psychol*. 2008;77(2):174–182.
41. Kirchner A, Birklein F, Stefan H, Handwerker H. Left vagus nerve stimulation suppresses experimentally induced pain. *Neurology*. 2000;55(8):1167–1171.

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