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Human Kinetics
ORIGINAL RESEARCH REPORT

Reduced Radial Displacement of the Gastrocnemius Medialis Muscle After Electrically Elicited Fatigue

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Context: Assessments of skeletal-muscle functional capacity often necessitate maximal contractile effort, which exacerbates muscle fatigue or injury. Tensiomyography (TMG) has been investigated as a means to assess muscle contractile function after fatigue; however, observations have not been contextualized by concurrent physiological measures. ***Objective:*** To measure peripheral-fatigue-induced alterations in mechanical and contractile properties of the plantar-flexor muscles through noninvasive TMG concurrently with maximal voluntary contraction (MVC) and passive muscle tension (PMT) to validate TMG as a gauge of peripheral fatigue. ***Design:*** Pre- and posttest intervention with control. ***Setting:*** University laboratory. ***Participants:*** 21 healthy male volunteers. ***Interventions:*** Subjects' plantar flexors were tested for TMG parameters, along with MVC and PMT, before and after either a 5-min rest period (control) or a 5-min electrical-stimulation intervention (fatigue). ***Main Outcome Measures:*** Temporal (contraction velocity) and spatial (radial displacement) contractile parameters of the gastrocnemius medialis were recorded through TMG. MVC was measured as an indicator of muscle fatigue, and PMT was measured to assess muscle stiffness. ***Results:*** Radial displacement demonstrated a fatigue-associated reduction (3.3 ± 1.2 vs 4.0 ± 1.4 mm, $P = .031$), while contraction velocity remained unaltered. In addition, MVC significantly declined by 122.6 ± 104 N ($P < .001$) after stimulation (fatigue). PMT was significantly increased after fatigue (139.8 ± 54.3 vs 111.3 ± 44.6 N, $P = .007$). ***Conclusion:*** TMG successfully detected fatigue, evident from reduced MVC, by displaying impaired muscle displacement accompanied by elevated PMT. TMG could be useful in establishing skeletal-muscle fatigue status without exacerbating the functional decrement of the muscle.

Keywords: muscle contractile properties, maximal voluntary contraction, TMG, passive muscle tension, peripheral fatigue

Muscle fatigue is characterized by a decrease in the external force or torque-generating capacity[1] and/or by impairment in peak power output.[2] The manifestation and magnitude of this reduced function depends on multiple factors including the muscle-contraction mode,[1] the nature of the fatigue protocol,[3] and the source of the fatigue.[4] Fatigue-related alterations of skeletal muscle can be observed, among other factors, by changes in its contractile and mechanical properties.

Since fatigue is a condition that affects both athletic performance and clinical mobility, the need for a valid monitor of muscle response is important to enable optimal management of athletes and patients. In situations of muscle fatigue, or indeed musculoskeletal injury, it is impractical to assess muscle function through a measure that makes use of voluntary efforts (ie, maximal voluntary contraction [MVC]), due to centrally mediated inhibition.[5] Furthermore, the potential for aggravation of any damage to the musculoskeletal unit cannot be ruled out. Having been developed over the last 15 years, tensiomyography (TMG) is a portable and noninvasive means of measuring muscle response through combined use of submaximal (below voluntary maximal activation) electrical stimulus and a digital displacement sensor,[6–8] similar to that used in mechanomyography.[9] TMG records spatial and temporal parameters of the radial displacement of the muscle belly in response to electrical stimuli[10] and is reliable within[11] and between days.[12] Furthermore, it has demonstrated good long-term stability after fatigue[13] and has displayed significant interclass correlation coefficient with decline and recovery of MVC after exercise-induced muscle damage.[14] In particular, muscle displacement (Dm) and contraction time (Tc) have shown greatest stability.[12]

TMG has successfully detected fatigue-associated changes after ultraendurance triathlon[15] and resistance exercise.[16] However, those studies report inconsistent results in the fatigue-induced alteration of the TMG parameters, perhaps due to the vast differences in the fatigue protocols administered and the different muscles measured. Furthermore, previous studies have failed to relate the TMG alterations to any valid functional measure such as MVC or passive muscle tension (PMT), which leaves the physiological interpretation of the TMG data open to question. Therefore, to effectively provide

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meaningful validation of TMG measurement of local fatigue it is important to overcome this limitation. In practical terms, submaximal TMG could offer an attractive measure for sport and medical practitioners in their assessment of muscle response and status after fatigue-based activity without necessitating voluntary contractile effort.

Accordingly, the aim of the present investigation was to evaluate peripheral fatigue-induced alterations in mechanical and contractile properties of the gastrocnemius muscle, as measured by TMG. MVC and PMT were measured before and after intervention to quantify the extent of muscle fatigue and allow us to better interpret changes in TMG response; to our knowledge this has not been previously reported. We hypothesized that a reduction in size and velocity of muscle displacement would indicate muscle fatigue in line with impairments in muscle function (decreased MVC) and elevated muscle stiffness (increased PMT). The findings of this study could help to establish TMG as a noninvasive alternative to quantify muscle fatigue.

Methods

Participants

Twenty-one healthy men with a mean ± SD age, height, and mass of 21.3 ± 3.4 years, 182.0 ± 6.1cm, and 79.5 ± 10.0 kg, respectively, volunteered and gave their written informed consent to participate in this study. All participants were recreationally active and free from injury. Women were excluded from the study to maintain cohort homogeneity. The study was performed in accordance with the principles outlined in the Declaration of Helsinki and was approved by the local research ethics committee.

Design

Mechanical and contractile properties of the right gastrocnemius medialis (GM) were monitored using TMG (BMC Ltd, Ljubljana, Slovenia). The GM is one of the propulsive muscles, fundamental to different types of human locomotion, and is located superficially, making it clearly measurable by TMG. Participants were also tested for PMT and MVC of the right plantar flexors. Testing was carried out on 2 occasions, 1 week apart, as illustrated in Figure 1. Measurements were taken at a number of time points before and after either the control or the fatigue intervention, according to the following order: TMG and PMT (measurement 1, M1); warm-up, TMG, and PMT + MVC (M2), either control or fatigue intervention in random order; TMG and PMT + MVC (M3). Both TMG and PMT measurements were recorded 3 minutes after the warm-up and after the control or fatigue intervention, to limit the effects of postactivation potentiation in the GM muscle.[17] Participants reported to the laboratory on the morning of each experimental trial in a fasted and rested state. Twenty-four-hour dietary intake records were completed on the day preceding each trial, and participants were instructed to replicate their dietary intake before each visit.

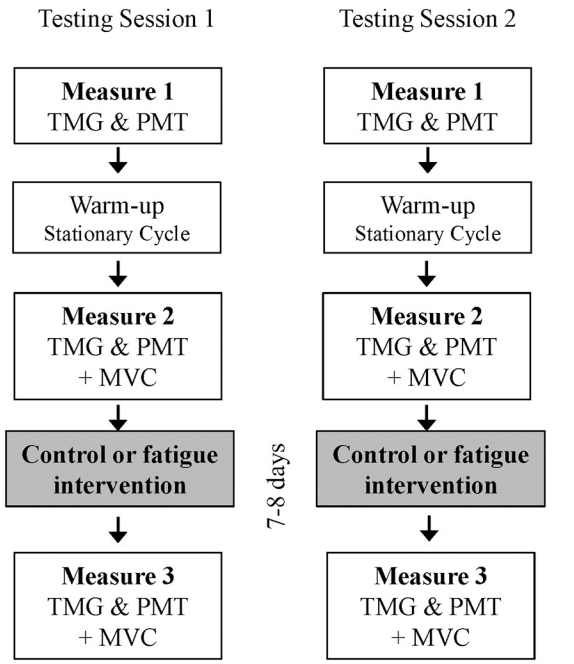

Figure 1 — Schematic representation of the research design. Abbreviations: TMG, tensiomyography; PMT, passive muscle tension; MVC, maximal voluntary contraction.

Warm-Up

Participants warmed up by cycling at a low intensity (75 W) on an electromagnetically braked cycle ergometer (Lode Ergometer, Netherlands) for 5 minutes at a cadence between 80 and 90 rpm.

TMG Protocol

TMG measurements were performed exactly as described by Ditroilo et al.[13] Briefly, participants lay in a prone position on a padded bench. A foam pad, placed slightly proximal to the ankle joint, supported a knee-flexion angle of around 5°. The digital displacement transducer (TMG–BMC Ltd, Ljubljana, Slovenia) was then positioned perpendicular to the muscle belly of the right GM with an initial pressure of 1.5×10^{-2} N/mm^2, controlled by consistently retracting the spring-loaded transducer probe to 50% of its length. This measuring position was selected by first manually palpating the GM to locate the thickest part of the muscle, and then later, if needed, the position was slightly adjusted to obtain the highest mechanical response with the least amount of coactivation when externally stimulated; coactivation was typically identified by a second peak in the TMG response curve. Once the appropriate position was obtained, it was marked with a permanent marker pen to ensure uniformity when the sensor was repositioned for subsequent measurements. The center point of each of the 2 stimulating electrodes (5 cm^2) (Axelgaard, USA) was located approximately halfway from the position of the sensor (~5 cm) to the

start of the respective GM proximal distal tendons. After each measurement these electrodes were left in place and unplugged to avoid any possible changes in muscle response via alterations in surface-electrode distance.[10] A single 1-millisecond-wide stimulation pulse was delivered, which applied initial current amplitude of 20 mA. This amplitude was progressively increased by 10-mA increments until maximal response was obtained, that is, when no further displacement of the muscle belly could be produced as identified by a plateau in the twitch-response curves. To minimize the effects of fatigue and potentiation, rest periods of 10 seconds were allowed between stimulation pulses. Typical maximal responses were observed at an amplitude between 40 and 70 mA, and only the output data for that particular stimulation intensity were used for analysis. Figure 2 shows a typical TMG displacement–time curve before and after the administration of the fatigue protocol. Output parameters were extracted and analyzed from each maximal twitch response[10]: Dm, the extent of maximal radial deformation (mm) of the muscle belly during contraction, and Vc, the rate (mm/s) of contraction between 10% and 90% of maximal displacement. Raw data were extracted from the TMG software, and Vc was calculated according to the formula: Vc = Dm80/Tc, where Tc = contraction time between 10% and 90% of peak radial displacement of the muscle belly and Dm80 = the radial displacement occurring during the time period of Tc.[18] Tc has been widely reported in previous studies,[10,15,16] as the temporal change from 10% to 90% of muscle Dm, providing a value relative to the spatial characteristics of each muscle. However, when assessing intramuscular alterations prefatigue and postfatigue, the significance of calculating Tc in this manner should be questioned. Indeed, in the absence of signal latency, it is possible that a decrease in Dm could associate with a decrease in Tc when calculated as just described. Apparent decreases in Tc, suggesting a faster twitch response, could be reported simply as a result of reduced overall Dm. We therefore proposed that assessment of Vc could provide greater insight when monitoring the fatigue status of a muscle.

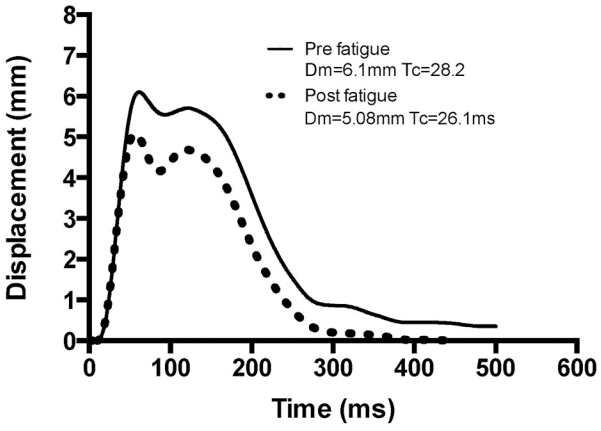

Figure 2 — Typical displacement–time curve of the tensiomyographic signal before and after the administration of the fatigue protocol. Abbreviations: Dm, muscle displacement; Tc, contraction time.

MVC Protocol

Plantar-flexor isometric MVC was performed on an isokinetic dynamometer (Kin-Com, Chattanooga Group Inc, USA). The participant had his right foot fastened securely into the plantar-flexion attachment, which was also held in place using 2 securely fastened shoulder straps and a lap belt. A 90° ankle angle to the tibia was ensured for each subject (Figure 3). After 2 submaximal warm-up sets, participants performed a 5-second MVC of the right plantar flexors. Three trials of the MVC were completed with 60 seconds recovery between attempts. Participants were verbally motivated to ensure the greatest possible effort for the duration of all attempts.

PMT Protocol

Measurements of PMT of the right plantar flexors were made on the same isokinetic dynamometer, with a setup identical to the MVC protocol (Figure 3). Participants were instructed to completely relax once in position, and the mean passive force of the ankle flexed at 90° was recorded during a period of 15 seconds as a measure of PMT in the plantar flexors in a static position.[19] A single measure was taken to determine PMT, as subsequent stretching of the ankle joint would cause an accumulative stretch effect. An intrasession reliability, as measured by the intraclass correlation coefficient, of ≥.80 has been previously reported for this type of measurement.[20]

Fatigue Protocol

The fatigue intervention used in the current investigation differs from previous studies in this area[15,16] in a number of key ways. First, fatigue was induced locally with a low-frequency stimulation that would necessitate a prolonged recovery, compared with

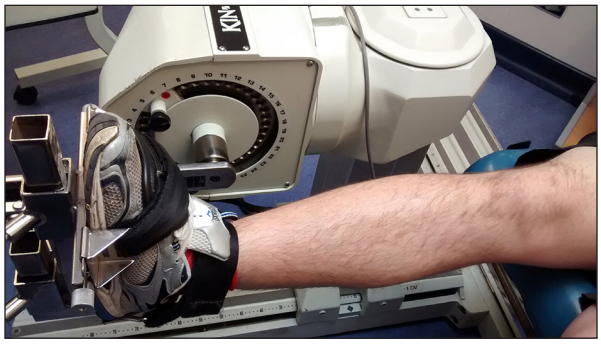

Figure 3 — Isokinetic dynamometer setup for assessment of passive muscle tension and maximal voluntary contraction. Ankle flexed at 90° relative to the tibia.

higher-frequency fatigue.[21] Second, as motor-unit discharge rarely exceeds 30 Hz during voluntary contraction,[21] low-frequency stimulus can be considered a more functionally relevant intervention. Finally, as TMG is a passive and peripheral measurement it will minimize confounding variables such as the variability of central control factors. While remaining secured in the same position as for PMT, the participants received the fatigue intervention, which consisted of a 5-minute electrical stimulation of the right GM, to evoke fatigue. The stimulation protocol involved a train of 15 electrical pulses (1 every 100 ms) with a 1-second gap before the start of each subsequent train. The protocol lasted 5 minutes and participants were asked to endure the maximum current they could to ensure fatigue (~110 mA). The control intervention consisted of the same positioning but receiving no stimulation for a period of 5 minutes to account for the effect of time. Also in the same position, with the ankle placed at 90°, isometric MVC of the plantar flexors was measured, before and after both intervention and control, to assess whether fatigue occurred.

Statistical Analysis

All data are presented as mean ± SD. After testing for assumption of normality of the dependent variables and log-transforming where necessary (ie, when not normally distributed), a 3 (measurements: before warm-up, M1; after warm-up, M2; after intervention, M3) × 2 (condition: control and fatigue intervention) ANOVA with repeated measures on both factors was used to detect differences in PMT and TMG parameters as a result of the fatigue or control protocol. Where a significant *F*-value was found, a Tukey post hoc test was used to identify where any significant difference occurred. Paired *t*-test was conducted to compare the prefatigue-to-postfatigue MVC difference between the control and fatigue interventions. Effect size (ES) was also calculated using eta-squared (η^2) and interpreted as small (0.01), moderate (0.06), or large (0.14).[22] The percentage differences between control and fatigue interventions were also calculated and interpreted based on the minimum detectable change as reported in a previous reliability study.[13] An alpha level of $P < .05$ was considered statistically significant. Statistical analysis was performed using Statistica version 10 (Statsoft Ltd, Bedford, UK).

Results

TMG Parameters

Dm demonstrated a fatigue-associated alteration. A significant main effect for condition ($F = 7.2$, $P = .002$, $\eta^2 = .27$) was documented for Dm, along with a post hoc difference at M3, demonstrating that the fatigue condition was significantly lower than the control condition (3.3 ± 1.2 vs 4.0 ± 1.4 mm, $P = .031$; Figure 4), with a difference of 17.7%. No significant difference was found for any of the factors or their interaction for Vc, which exhibited 121.8 ± 43.2 versus 124.7 ± 45.5 mm/s at M1, 121.3 ± 45.7 versus 124.9 ± 44.7 mm/s at M2, and 131.3 ± 44.6 versus 139.8 ± 50.6 mm/s at M3.

MVC and PMT

Plantar-flexor isometric MVC exhibited a significant interaction condition × measurement interaction ($F = 12.4$, $P = .001$, $\eta^2 = .91$), with post hoc analysis showing a significant decline after the fatigue intervention (–122.6 ± 104 N; $P < .001$) but not after control (–25.7 ± 71.3 N, $P = .115$). The PMT exhibited a significant condition × measurement interaction ($F = 5.9$, $P = .005$, $\eta^2 = .23$). The post hoc analysis revealed at M3 that fatigue caused significantly more tension than control (139.8 ± 54.3 vs 111.3 ± 44.6 N, $P = .007$; Figure 5), with a difference of 20.4%.

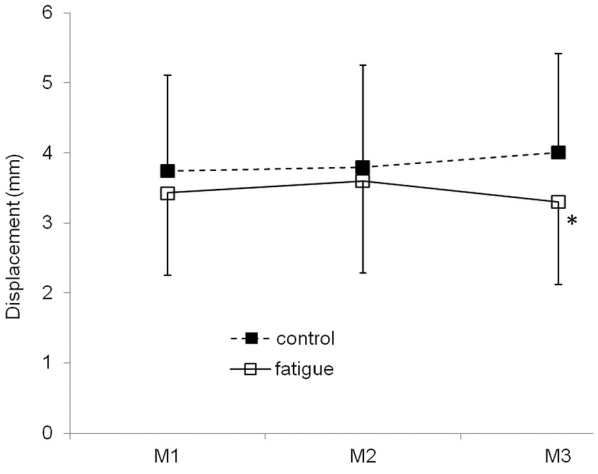

Figure 4 — Muscle displacement as assessed by tensiomyography at the 3 measurement points, mean ± SD. *Significantly different from control at M3, $P < .05$.

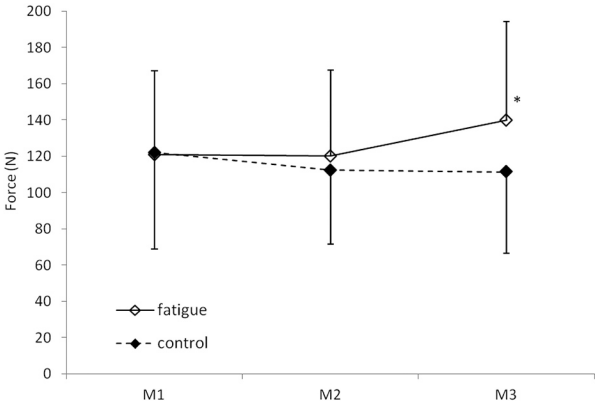

Figure 5 — Passive muscle tension as assessed on the isokinetic dynamometer at the 3 measurement points, mean ± SD. *Significantly different from control at M3, $P < .01$.

Discussion

This study was designed to evaluate the validity of TMG, as a submaximal assessment method to detect local muscle fatigue, against functional physiological measures. Fatigue of the GM was achieved, as evidenced by the significant decline in peak force (MVC), which was absent after the control condition. This alteration in functional capacity of the muscle was associated with a significant decline in TMG Dm, similar to previous studies after dynamic fatigue.[16,23] In addition, plantar-flexor PMT increased after the fatigue intervention, suggesting that the GM skeletal muscle–tendon unit became stiffer. Despite these alterations, muscle twitch Vc appeared to remain unaffected by fatigue.

When considering the physiological effects of fatigue, there are a number of important variables to examine. We have previously demonstrated that during fatigued voluntary contractions, muscle-fiber-conduction velocity declines due to a reduction in extracellular pH.[24] It is likely that this occurs due to a pH-driven alteration of the Na^+ and K^+ gradient across the sarcolemma[25] and impairs action potential propagation. Therefore, during TMG measurement the electrical stimulus applied to the surface of the fatigued muscle should result in a slowing down of the action potentials propagated to reduce $Ca2^+$ release and subsequent excitation–contraction (E-C) coupling. Low-frequency fatigue, as characterized by a disproportionate reduction in force at lower stimulation frequencies, has been associated with E-C uncoupling.[26] It has been suggested that E-C uncoupling is attributable to, among other factors, impaired $Ca2^+$ transport via ryanodine-receptor channels in the triadic compartment.[27] Furthermore, other contributing factors will be from increased Pi, which can push the cross-bridge into a low force-generating status[28] and may also cause actin and myosin to detach.[29] These altered characteristics of muscle function will inevitably impair its force-generation capacity, as shown by the significant decline in MVC.

It has been reported previously that a stiffer muscle, as we have evidenced here by the rise in PMT (Figure 5), will be associated with a reduced TMG Dm measurement.[8] In contrast to the current findings, Garcia-Manso et al[15] showed an increase in biceps femoris TMG Dm associated with fatigue after an ironman triathlon. The precise reasons for this disparity are unclear; however, Morin et al[30] showed a small decline in whole-leg stiffness during a running task after a 24-hour marathon. Those authors postulated that central fatigue would have been apparent, which would have been linked to altered peripheral feedback from muscle afferents triggered from cytokines. This, we suggest, may be why an increase in TMG Dm was observed after an ironman triathlon when a decline has been reported with other types of fatigue from far shorter contractile/exercise durations. Other studies have also demonstrated alterations in Dm alongside muscle architectural changes. First, Pisot et al,[8] showed that after 35 days of bed rest, TMG Dm increased alongside the reduction in muscle thickness, which the authors suggested would have contributed to reduced muscle stiffness. Second, we previously demonstrated[31] that altering the length of the muscle will determine the magnitude of TMG parameters, such that longer muscle length, as achieved through altered joint angle, results in reduced Dm. Third, although not relating the decline in TMG Dm to muscle-stiffness changes, other studies[16,23] have also demonstrated a decline in TMG Dm after fatigue, suggesting that this is an important parameter when assessing the muscle status in this regard.

In the current study we observed decreases in TMG Dm without significant alterations in Vc. Given previously described reductions in action-potential propagation and muscle-fiber-conduction velocity associated with fatigue,[24] it may have been expected that TMG Vc would be observed to decline postfatigue in concurrence with Dm. It is plausible that the lack of significant alteration in Vc is due to the high degree of interindividual variability associated with the measurement. Indeed, changes between measurements (M1, M2, and M3) ranged from about –25% to +25% between participants. The comparably low amplitude of the electrical stimulation used to elicit the peak TMG response may perhaps render these data difficult to compare with existing conduction-velocity findings. As such, it may be inappropriate to consider alterations in the speed–time component of the TMG response when assessing muscle fatigue, with the focus instead being placed on spatial alterations (Dm), which we have shown here to be indicative of increased muscle stiffness.

As with any type of physiological measurement there will be a degree of variability. We have previously accounted for this variability with TMG measured under different muscle conditions[13] and shown Dm to be well within acceptable limits. Analogous to this is establishing minimal detectable change so practitioners and researchers can be confident that the given magnitude of observed change after any intervention is real and physiologically significant. We have demonstrated in this study that the fatigue-altered Dm parameter (17.7%) clearly exceeds the minimal-detectable-change thresholds of 15.1%.[13] Furthermore, the effect size for the data presented in this study, as described by Cohen,[22] is large, suggesting that this particular TMG measure is sufficiently sensitive to adequately detect local muscle fatigue. Nonetheless, a number of limitations must be considered. Current findings can only be applied to a healthy, young, male population. It remains to be seen whether TMG measurements are sufficiently sensitive to detect fatigue-associated changes in alternative cohorts. In addition, the GM was selected for investigation as its anatomical position facilitates measurement using TMG. Muscles that are not located superficially, but may still be of interest, are not measureable using the methods described herein.

Conclusion

This is the first study to demonstrate that TMG was effective in detecting local muscle fatigue in the GM. We propose that this response was directly related to increased stiffness of the muscle from impaired contractile capacity.

It should be emphasized that, when assessing local muscle fatigue, Dm of the muscle is a valid measure; however, it remains to be seen whether TMG has the sensitivity to detect any changes in Vc in a different context. The current findings have important implications for researchers and practitioners seeking to establish fatigue status of skeletal muscle, with implications for prevention of overtraining injuries in sports-related activities. Given the noninvasive and submaximal nature of this type of measurement, TMG can be used to determine local muscle fatigue in patients who may be unable to exert the maximal effort required for voluntary muscle-function assessments. In addition, TMG measurements are exempt from the bias of volitional effort and motivation, facilitating the incorporation of the procedure into existing programs.[32] Furthermore, TMG could be used regularly as a monitoring tool without fear of detriment to muscle function.

Acknowledgments

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References

1. Garrandes F, Colson SS, Pensini M, Seynnes O, Legros P. Neuromuscular fatigue profile in endurance-trained and power-trained athletes. *Med Sci Sports Exerc*. 2007;39(1):149–158. PubMed doi:10.1249/01.mss.0000240322.00782.c9
2. Ditroilo M, Watsford M, Fernandez-Pena E, D'Amen G, Lucertini F, De Vito G. Effects of fatigue on muscle stiffness and intermittent sprinting during cycling. *Med Sci Sports Exerc*. 2011;43(5):837–845. PubMed doi:10.1249/MSS.0b013e3182012261
3. Babault N, Desbrosses K, Fabre M-S, Michaut A, Pousson M. Neuromuscular fatigue development during maximal concentric and isometric knee extensions. *J Appl Physiol*. 2006;100(3):780–785. PubMed doi:10.1152/japplphysiol.00737.2005
4. Berchicci M, Menotti F, Macaluso A, Di Russo F. The neurophysiology of central and peripheral fatigue during sub-maximal lower limb isometric contractions. *Front Hum Neurosci*. 2013;7:135. PubMed
5. Graven-Nielsen T, Lund H, Arendt-Nielsen L, Danneskiold-Samsøe B, Bliddal H. Inhibition of maximal voluntary contraction force by experimental muscle pain: a centrally mediated mechanism. *Muscle Nerve*. 2002;26(5):708–712. PubMed doi:10.1002/mus.10225
6. Dahmane R, Valencic V, Knez N, Erzen I. Evaluation of the ability to make noninvasive estimation of muscle contractile properties on the basis of the muscle belly response. *Med Biol Eng Comput*. 2001;39:51–55. PubMed doi:10.1007/BF02345266
7. Dahmane R, Djordjevic S, Simunic B, Valencic V. Spatial fiber type distribution in normal human muscle: histochemical and tensiomyographical evaluation. *J Biomech*. 2005;38:2451–2459. PubMed doi:10.1016/j.jbiomech.2004.10.020
8. Pisot R, Narici MV, Simunic B, et al. Whole muscle contractile parameters and thickness loss during 35-day bed rest. *Eur J Appl Physiol*. 2008;104(2):409–414. PubMed doi:10.1007/s00421-008-0698-6
9. Orizio C. Surface mechanomyogram. In: Merletti R, Parker P (Eds.). *Electromyography: Physiology, Engineering, and Noninvasive Applications* (pp. 305–322). Hoboken, NJ: John Wiley & Sons; 2004. doi:10.1002/0471678384.ch11
10. Tous-Fajardo J, Moras G, Rodriguez-Jimenez S, Usach R, Doutres DM, Maffiuletti NA. Inter-rater reliability of muscle contractile property measurements using non-invasive tensiomyography. *J Electromyogr Kinesiol*. 2010;20(4):761–766. PubMed doi:10.1016/j.jelekin.2010.02.008
11. Krizaj D, Simunic B, Zagar T. Short-term repeatability of parameters extracted from radial displacement of muscle belly. *J Electromyogr Kinesiol*. 2008;18(4):645–651. PubMed doi:10.1016/j.jelekin.2007.01.008
12. Simunic B. Between-day reliability of a method for non-invasive estimation of muscle composition. *J Electromyogr Kinesiol*. 2012;22(4):527–530. PubMed doi:10.1016/j.jelekin.2012.04.003
13. Ditroilo M, Smith IJ, Fairweather MM, Hunter AM. Long-term stability of tensiomyography measured under different muscle conditions. *J Electromyogr Kinesiol*. 2013;23(3):558–563. PubMed doi:10.1016/j.jelekin.2013.01.014
14. Hunter AM, Galloway SDR, Smith IJ, et al. Assessment of eccentric exercise-induced muscle damage of the elbow flexors by tensiomyography. *J Electromyogr Kinesiol*. 2012;22(3):334–341. PubMed doi:10.1016/j.jelekin.2012.01.009
15. García-Manso JM, Rodriguez-Ruiz D, Rodriguez-Matoso D, de Saa Y, Sarmiento S, Quiroga M. Assessment of muscle fatigue after an ultra-endurance triathlon using tensiomyography (TMG). *J Sports Sci*. 2011;29(6):619–625. PubMed doi:10.1080/02640414.2010.548822
16. García-Manso JM, Rodriguez-Matoso D, Sarmiento S, et al. Effect of high-load and high-volume resistance exercise on the tensiomyographic twitch response of biceps brachii. *J Electromyogr Kinesiol*. 2012;22(4):612–619. PubMed doi:10.1016/j.jelekin.2012.01.005
17. Pääsuke M, Saapar L, Ereline J, Gapeyeva H, Requena B, Oopik V. Postactivation potentiation of knee extensor muscles in power- and endurance-trained, and untrained women. *Eur J Appl Physiol*. 2007;101(5):577–585. PubMed doi:10.1007/s00421-007-0532-6
18. Valencic V, Knez N. Measuring of skeletal muscles' dynamic properties. *Artif Organs*. 1997;21(3):240–242. PubMed doi:10.1111/j.1525-1594.1997.tb04658.x
19. Fowles JR, Sale DG, MacDougall JD. Reduced strength after passive stretch of the human plantarflexors. *J Appl Physiol*. 2000;89(3):1179–1188. PubMed
20. Araújo VL, Carvalhais VOC, Souza TR, Ocarino JM, Gonçalves GGP, Fonseca ST. Validity and reliability of clinical tests for assessing passive ankle stiffness. *Rev Bras Fisioter*. 2011;15(2):166–173. PubMed
21. Allen DG, Lamb GD, Westerblad H. Skeletal muscle fatigue: cellular mechanisms. *Physiol Rev*. 2008;88(1):287–332. PubMed doi:10.1152/physrev.00015.2007
22. Cohen J. *Statistical Power Analysis for the Behavioural Sciences*. 2nd ed. Hillsdale, NJ: Lawrence Erlbaum; 1988.
23. Carrasco L, Sanudo B, de Hoyo M, Pradas F, Da Silva ME. Effectiveness of low-frequency vibration recovery method on blood lactate removal, muscle contractile properties and on time to exhaustion during cycling at VO_{2max} power

output. *Eur J Appl Physiol*. 2011;111(9):2271–2279. PubMed doi:10.1007/s00421-011-1848-9
24. Hunter AM, De Vito G, Bolger C, Mullany H, Galloway SDR. The effect of induced alkalosis and submaximal cycling on neuromuscular response during sustained isometric contraction. *J Sports Sci*. 2009;27(12):1261–1269. PubMed doi:10.1080/02640410903165077
25. Brody LR, Pollock MT, Roy SH, De Luca CJ, Celli B. pH-Induced effects on median frequency and conduction velocity of the myoelectric signal. *J Appl Physiol*. 1991;71(5):1878–1885. PubMed
26. Hill CA, Thompson MW, Ruell PA, Thom JM, White MJ. Sarcoplasmic reticulum function and muscle contractile character following fatiguing exercise in humans. *J Physiol*. 2001;531:871–878. PubMed doi:10.1111/j.1469-7793.2001.0871h.x
27. Balog EM. Excitation–contraction coupling and minor triadic proteins in low-frequency fatigue. *Exerc Sport Sci Rev*. 2010;38(3):135–142. PubMed doi:10.1097/JES.0b013e3181e3734d
28. Nocella M, Colombini B, Benelli G, Cecchi G, Bagni MA, Bruton J. Force decline during fatigue is due to both a decrease in the force per individual cross-bridge and the number of cross-bridges. *J Physiol*. 2011;589(Pt 13):3371–3381. PubMed doi:10.1113/jphysiol.2011.209874
29. Takagi Y, Shuman H, Goldman YE. Coupling between phosphate release and force generation in muscle actomyosin. *Philos Trans R Soc Lond B Biol Sci*. 2004;359(1452):1913–1920. PubMed doi:10.1098/rstb.2004.1561
30. Morin JB, Tomazin K, Edouard P, Millet GY. Changes in running mechanics and spring-mass behaviour induced by a mountain ultra-marathon race. *J Biomech*. 2011;44(6):1104–1107. PubMed doi:10.1016/j.jbiomech.2011.01.028
31. Ditroilo M, Hunter AM, Haslam S, De Vito G. The effectiveness of two novel techniques in establishing the mechanical and contractile responses of biceps femoris. *Physiol Meas*. 2011;32(8):1315–1326. PubMed doi:10.1088/0967-3334/32/8/020
32. Rey E, Lago-Peñas C, Lago-Ballesteros J. Tensiomyography of selected lower-limb muscles in professional soccer players. *J Electromyogr Kinesiol*. 2012;22(6):866–872. PubMed doi:10.1016/j.jelekin.2012.06.003