Effect of Orthoses on Changes in Neuromuscular Control and Aerobic Cost of a 1-h Run

LUKE A. KELLY, OLIVIER GIRARD, and SEBASTIEN RACINAIS
ASPETAR, Qatar Orthopaedic and Sports Medicine Hospital, Doha, QATAR

ABSTRACT

KELLY, L. A., O. GIRARD, and S. RACINAIS. Effect of Orthoses on Changes in Neuromuscular Control and Aerobic Cost of a 1-h Run. Med. Sci. Sports Exerc., Vol. 43, No. 12, pp. 2335–2343, 2011. Purpose: The study’s purpose was to determine the effect of foot orthoses on neuromuscular control and the aerobic cost of running. Methods: Twelve recreational athletes ran for 1 h on a treadmill at a constant velocity (i.e., 10% higher than their first ventilatory threshold) with and without custom-molded foot orthoses, in a counterbalanced order. Surface EMG activity of five lower limb muscles, together with oxygen consumption and HR, was recorded at 8-min intervals, starting after 2 min, during the run. A series of neuromuscular tests including voluntary and electrically evoked contractions of the ankle plantar flexors was performed before and after running. Results: Peroneus longus root mean square amplitude decreased with time, independently of the condition (−18.9%, P < 0.01). Lower root mean square signal amplitude for vastus medialis (−13.3%, P < 0.02) and gastrocnemius medialis (−10.7%, P < 0.05), combined with increased peroneus longus burst duration (+14.7%, P < 0.05), occurred when running with orthoses. There was no main effect of the condition for oxygen consumption (P > 0.05), whereas HR was significantly lowered while wearing foot orthoses (−3%, P < 0.02). Maximal strength capacity (−9%, P < 0.01), normalized EMG activity (−17%, P < 0.001), and peak twitch torque (−14%, P < 0.01) declined from before to after exercise, independently of the condition. Smaller fatigue-induced decrements in the rate of torque development within the first 200 ms (−6% vs −33%, P < 0.01) were reported after running with foot orthoses. Conclusions: Wearing foot orthoses alters neuromuscular control during a submaximal 1-h treadmill run and partly protects from the resulting fatigue-induced reductions in rapid force development characteristics of the plantar flexors. However, these changes may be too small to alter the aerobic cost of running. Key Words: FOOT ORTHOSES, NEUROMUSCULAR FATIGUE, TREADMILL RUNNING, MUSCLE ACTIVITY, COST OF LOCOMOTION

Neuromuscular fatigue has been defined as a temporary reduction in force-producing capability (24), originating from both neural (reduced drive to the muscle) and muscular (reduced contractility) factors (33,35). Pre- to postrun muscle assessments using voluntary and electrically evoked contractions are widely used to gain knowledge about neural and muscular adjustments that occur after running exercises (33,35).

As fatigue progressively develops during sustained submaximal runs, significant alterations in neuromuscular function including increased joint motion (8,9) and loading (10,40), combined with reduced leg stiffness (32) and/or increased vertical impact forces (10), have been reported. There is emerging evidence that these manifestations of fatigue may relate to an increased risk of lower limb overuse injury (3,9,11,40,41).

Foot orthoses are widely used as part of treatment and prevention of lower limb injury (15). It is suggested that wearing foot orthoses leads to reduced loading in the lower limb via alterations to neuromechanical function (23,27). Previous studies examining the effect of foot orthoses on muscle activation patterns have been conducted during walking and running (25,26,28), in a nonfatigued state. However, given that sustained running leads to significant alterations in lower limb neuromechanics (described in the second paragraph) (8,10,41), it is important from an injury prevention and management perspective to understand whether interventions such as foot orthoses can alter muscle responses to fatigue.

Saunders et al. (36) stated that reduced muscle activation and increased joint stiffness improve running economy because of less energy expenditure and better utilization of stored elastic energy. Interestingly, it has been reported that foot orthoses increase joint stiffness (20) and reduce muscle activation (26) during running. Nigg’s (29) “impact tuning” theory suggests that leg muscles will be tuned in response to forces acting on the foot during the previous stride.
According to this theory, a more efficient running pattern will lead to a reduction in muscle activity and a reduced energy cost. In light of Nigg’s (29) theory, it is plausible to suggest that alterations in mechanical loading while running with foot orthoses (23) could subsequently reduce the demand imposed on lower limb muscles, leading to a lowered aerobic cost of running. However, current evidence for this theory is minimal and inconclusive (1). To our knowledge, no studies have linked the aerobic cost of running and EMG activity of the lower limb muscles, simultaneously concerning a foot orthoses intervention, in the context of fatigue.

The aim of this study was to determine whether wearing foot orthoses can reduce lower limb muscle activation during sustained submaximal running, i.e., limiting the already described alterations in neuromuscular and cardiovascular functions that occur when runners experience fatigue. We hypothesized that wearing foot orthoses would lead to reduced muscle recruitment and aerobic cost during the run, consequently lessening the magnitude of postexercise neural and muscular adjustments.

METHODS
Participants
Power calculations were conducted using variability (SD) of average rectified EMG, recorded during submaximal running in our own pilot studies. Assuming an SD of 10 and a difference of 9–10 μV, the required sample size was $9 - 12 (\alpha = 0.05$ and $\beta = 0.2)$. Therefore, 12 male recreational athletes (age $= 31.2 \pm 3.8$ yr, mass $= 76 \pm 3.9$ kg, height $= 180.8 \pm 4.4$ cm) with no history of injury in the preceding 6 months volunteered to participate in this study. Participants were the same running shoes throughout the duration of the entire experiment. None of the participants currently used foot orthoses. All subjects were informed of the study requirements, benefits, and risks before giving written informed consent. All procedures conformed to the standards set by the Declaration of Helsinki, and the protocol was approved by the scientific research ethics committee of ASPETAR, Qatar Orthopaedic and Sports Medicine Hospital.

Experimental Procedures

The subjects visited our laboratory on three occasions, with at least 7 d apart. After a familiarization session, they participated in two experimental sessions consisting of running with (intervention) and without (control) foot orthoses, in a counterbalanced order, preceded and followed by neuromuscular tests.

Familiarization session. The subjects were first introduced to and familiarized with the experimental procedures. They performed maximal voluntary contractions (MVC) of the plantar flexors until they felt accustomed to the equipment; the coefficient of variation in three successive trials was <5%. This session was also used to accustom the subjects to the electrical stimulation of the tibial nerve and determine the individual optimal stimulation intensity. A passive isometric recruitment curve was drawn to carefully search stimulus intensity for the maximal soleus (SO) M-wave response. Briefly, the current was progressively increased by 10-mA increments until there was no further increase in both peak twitch torque and concomitant M-wave amplitudes ($M_{\text{max}}$). This intensity was further increased by 50% (i.e., supramaximal) and subsequently maintained for the entire experiment. After a 15-min period, during which they were allowed to drink without restriction, the subjects were then asked to perform an incremental test on a motorized treadmill (h/p/cosmos gmbh, Nussdorf, Germany) to determine their first ventilatory threshold (33). The test consisted of an initial 2-min workload of 8 km h$^{-1}$ followed by increases of 0.5 km h$^{-1}$ every minute (1% incline) until exhaustion.

Experimental sessions. Each session lasted for 150 min and was conducted as follows: i) preparation of the subject’s leg (i.e., shaving, abrasion, and cleaning); ii) placement of the EMG electrodes, marking electrode sites and securing both the electrodes and cables with a cohesive bandage; iii) neuromuscular pretests (see following text); iv) preparation of the subject for running (i.e., placement of the mask for expired gas collection); v) 1 h of running exercise; and vi) neuromuscular posttests performed immediately after the end of the fatiguing exercise bout. The two 1-h runs were performed on the same treadmill as in the familiarization session, at a constant velocity corresponding to 10% higher than the first ventilatory threshold determined during the preliminary session. The treadmill was inclined at 1%. During the two runs, EMG, HR, and oxygen consumption were recorded continuously during the period of 1 h. Participants ran in their own footwear. Efforts were made to avoid caffeine intake and to ensure that no strenuous exercise was performed in the 24 h that preceded testing. All tests were performed at the same time of the day. Ambient temperature was $\sim$23°C.

Foot Orthoses

During the familiarization session, prefabricated foot orthoses (Formthotics; Foot Science International, Christchurch, New Zealand) were heat molded to the participants’ feet inside their own running shoes (the same pair was used for all trials). This procedure was conducted in accordance with manufacturer guidelines and previous research (19). Briefly, orthoses were matched and trimmed to the size of an existing innersole then placed in a shoe after removing the existing innersole. They were heated inside the shoe for 3 min using a previously described heating apparatus (19). After the heating period, the participant put the shoes on and tied the laces. Standing with feet shoulder width apart and weight evenly distributed between feet, the participant was then asked to bend knees as far as possible keeping heels on...
the ground and knees over second toes. This position was held for 1 min while foot orthoses cooled, providing customization to the plantar arch contour in each subject. Foot orthoses were removed from the shoes and given to the participant 7 d before the corresponding trial. An acclimatization period of 7 d was used because this is what is used commonly in clinical practice (19). During this time, the patient is advised to wear the orthoses for as long as possible to allow the feet and body to adjust to any biomechanical changes induced by the devices.

**Measurements**

**EMG.** Surface EMG activity of the vastus medialis (VM), gastrocnemius medialis (GM), peroneus longus (PL), SO, and tibialis anterior (TA) muscles was recorded using integral dry reusable electrodes with an interelectrode distance of 20 mm (Biometrics SX230, Gwent, United Kingdom). The reference electrode was placed on the right wrist. Low impedance was obtained after careful preparation of the skin (i.e., abrading the skin with emery paper and cleaning with alcohol). Electrodes were positioned and secured as recommended by the Surface ElectroMyoGraphy for the Noninvasive Assessment of Muscles (SENIAM) organization (16). All electrode sites were marked with a permanent marker pen to allow the investigators to determine whether electrodes had moved during the run, also allowing consistent electrode positioning for all sessions. Subjects were required to maintain pen markings between laboratory visits. In addition, all electrodes and cables were secured with an elastic cohesive bandage to reduce any movement of electrodes during the run or artifact in the signal. EMG signals were preamplified and filtered (bandwidth = 20–450 Hz, gain = 1000) and recorded with a sampling frequency of 2500 Hz using Biometrics hardware (Biometrics DataLOG). Clinical testing using specific tasks to isolate recruitment for each muscle was conducted as recommended by SENIAM (16). These procedures were undertaken to ensure signal quality and to reduce the risk of crosstalk. Good to excellent between-session reliability has been reported for lower limb surface EMG during running (14). The recommendations of SENIAM (16) were implemented to maximize reliability.

**Cardiorespiratory responses.** Values of oxygen uptake \((\dot{V}O_2)\) were determined by breath by breath during both the incremental test and the 1-h run (COSMED B2, Rome, Italy). Gas analyzers were calibrated before each test with a gas mixture of known composition \((O_2 = 16.0\%, O_2 and CO_2 = 5.0\%)\). An O_2 analyzer with a polarographic electrode and a CO_2 analyzer with an infrared electrode sampled expired gases at the mouth. The facemask that had a low dead space \((70 \text{ mL})\) was equipped with a low-resistance bidirectional digital turbine \((28 \text{ mm in diameter})\). This turbine was calibrated before each test with a 3-L syringe (Hans Rudolph, Inc., Dallas, TX). Furthermore, HR was continuously \((5\text{-s intervals})\) recorded via a wireless Polar monitoring system (Polar Electro Oy, Kempele, Finland). Participants were asked to report their RPE on a scale of 6–20 (2), at 8-min intervals, commencing after 2 min.

**Torque production.** Isometric ankle plantarflexion torque of the right foot was recorded using the footplate of a dynamometer (Biodex, Shirley, NY). Subjects were seated with the trunk inclined 30° with respect to the horizontal and the knee joint flexed at 120° \((180° = \text{full extension})\). The axis of rotation of the dynamometer was aligned with the anatomical ankle flexion–extension axis. The dynamometer was connected to the EMG acquisition system, allowing for synchronization of torque and EMG data.

**Neuromuscular Tests**

The tibial nerve was stimulated using a cathode electrode placed in the popliteal cavity with a constant compression supplied by a strap. The anode \((5 \times 10 \text{ cm})\) was positioned beneath the patella. Electrical stimulations \((400 \text{ V}, \text{rectangular pulse of } 0.2 \text{ ms})\) were delivered by a high-voltage stimulator (Digitimer DS7AH; Digitimer, Hertfordshire, United Kingdom). Stimulation intensity was set according to results obtained during the familiarization session. Each testing session began by evoking at rest five maximal \((M_{\text{max}})\) stimuli, each interspaced by 5 s. Afterward, subjects were instructed to perform three brief \((4\text{-to } 5\text{-s duration})\) maximal isometric voluntary contractions \((MVC)\) of the plantar flexors with at least 1 min between trials. Subjects were encouraged to increase plantarflexion torque from a fully relaxed state as fast (and hard) as possible. They were strongly encouraged to perform maximally. The preexercise test session was preceded by a standardized warm-up consisting of 10 plantarflexions \((4\text{-s duration}, \text{interspaced with } 6\text{-s rest periods})\); contraction intensities were self-adjusted by the subjects to be progressively increased up to MVC level during the last three trials. These methods were used to maximize reliability of our recordings, in line with a previous research (17).

**Data Analysis**

**Running EMG.** EMG data from 10 consecutive strides were extracted at eight separate time intervals, separated by 8 min \((i.e., \text{after } 2, 10, 18, 26, 34, 42, 50, \text{ and } 58 \text{ min})\) and used for subsequent analysis. Muscle burst onset and offset were identified (LabVIEW; National Instruments, Austin, TX) when the root mean square \((\text{RMS})\) signal amplitude exceeded a threshold, defined as the average rectified activity during the control run (rest and activation periods combined; Fig. 1). The burst onset threshold remained constant for both conditions, for all time intervals. In addition, manual inspection of all data was conducted to ensure accuracy and consistency of burst identification. Burst duration and average RMS amplitude \((\text{within the muscle burst})\) were then calculated.

**Respiratory response.** During both runs, \(\dot{V}O_2\) was recorded and averaged over 8 periods of 1 min every 8 min.
Running EMG. PL RMS ($F_{1,11} = 5.16, P < 0.01, ES = 0.319$) (Fig. 3) and TA burst duration ($F_{3,29.2} = 3.61, P < 0.022, ES = 0.286$) (Fig. 4) decreased with time, independently of the condition. VM RMS ($F_{1,11} = 7.92, P < 0.02, ES = 0.49, -13.3\%$) and GM RMS ($F_{1,5} = 6.72, P < 0.05, ES = 0.573, -10.7\%$) were significantly lower in the intervention compared with the control session. In addition, longer PL burst durations ($F_{1,11} = 5.12, P < 0.05, ES = 0.318, 14.7\%$) occurred in the intervention trial. No significant interactions between time and condition were evident for any variables.

Responses to exercise. HR ($F_{1,23.5} = 198.51, P < 0.01, ES = 0.947$), VO$_2$ ($F_{1,2.13.1} = 21.12, P < 0.01, ES = 0.658$), and RPE ($F_{3,1.28.3} = 132.62, P < 0.01, ES = 0.936$) increased with time during the run (Fig. 5). HR values were significantly lower in the intervention trial ($F_{1,11} = 8.77, P < 0.013, ES = 0.444, Fig. 5$). There was no significant effect of condition for VO$_2$ or RPE (all $P > 0.05$) or interactions between time and condition.

Neuromuscular testing. Significant main effects of exercise were observed for MVC torque ($F_{1,11} = 16.7, P < 0.01, ES = 0.6$), normalized EMG activity ($F_{1,11} = 17.74, P < 0.001, ES = 0.62$), peak twitch torque ($F_{1,11} = 11.62, P < 0.01, ES = 0.5$), and resting M-wave ($F_{1,11} = 5.95, P < 0.03, ES = 0.35$) (Table 1). However, there was no significant difference between conditions or interaction between condition and exercise for the same variables (all $P > 0.05$). There were significant main effects of exercise ($F_{1,11} = 20.65, P < 0.001, ES = 0.652$) and condition ($F_{1,11} = 54.49, P < 0.001, ES = 0.85$) and a significant interaction ($F_{1,11} = 15.69, P < 0.01, ES = 0.59$) between these two factors for RFD$_{200}$ (Table 1).

DISCUSSION

This study examined the effect of foot orthoses on lower limb neuromuscular control (muscle recruitment) and oxygen uptake during running, as well as the nature of end-exercise neuromuscular adjustments. Our results indicate that running

![Figure 1](image-url)  
**Figure 1**—EMG data for the GM in a representative individual. Burst onset and offset were defined using predetermined thresholds for each muscle burst onset threshold (A)—corresponding to the average rectified signal amplitude, during the entire run (rest and activation periods combined) in the control condition. The burst onset threshold remained constant for all time intervals in both conditions. Burst onset (B) was defined as when the RMS signal amplitude exceeded the onset threshold. Burst offset (C) was defined when the signal dropped below the average RMS threshold.

(i.e., same time intervals with EMG), commencing after the second minute of exercise.

**Neuromuscular tests.** The peak-to-peak amplitude of SO M-waves was determined. The peak twitch torque was determined from the mechanical response of the evoked potential. For voluntary contractions, SO RMS values were calculated during a 1-s period of the plateau. Raw SO RMS values were normalized to the corresponding superimposed M-wave. Furthermore, rate of torque development (RFD$_{200}$) was derived as the average slope of the torque–time curve (torque/time) at 200 ms relative to the onset of contraction and normalized to the maximal torque value (38) (Fig. 2). The mean over three or five trials (voluntary and evoked contractions, respectively) was used for subsequent analysis.

**Statistical Analysis**

Sphericity (homogeneity of covariance) was verified by the Mauchly test. When the assumption of sphericity was not met, the significance of $F$-ratios was adjusted according to the Greenhouse–Geisser procedure. EMG and responses to exercise data collected during the 1-h run were analyzed using a two-way (condition (orthoses) × time (eight time intervals)) ANOVA with repeated measures. Pairwise comparisons were used for post hoc analysis. Neuromuscular variables were compared by a two-way (condition (orthoses) × exercise (before vs after examination)) ANOVA with repeated measures. Effect size (ES) was calculated using partial $\eta^2$ to determine the magnitude and the clinical relevance of the significant findings. Level of significance was set at $\alpha = 0.05$. All analyses were conducted with SPSS version 18 (SPSS, Inc., Chicago, IL).

![Figure 2](image-url)  
**Figure 2**—Torque–time curve for rapid maximal isometric plantarflexions in a representative individual measured before (dotted line) and after (full line) the 1-h run. The vertical line denotes torque levels at 200 ms.
1 h on a treadmill induces several neuromuscular perturbations, irrespective of foot orthoses use. This is evidenced by 1) altered muscle recruitment strategies (RMS and burst duration) in TA and PL during the run and 2) postexercise neural and peripheral adjustments in the ankle plantar flexors (decrease in maximal force). We hypothesized that running with foot orthoses would reduce the level of muscle recruitment (because of altered mechanical loading) and in turn reduce aerobic cost and the extent of postexercise neural and muscular adjustments. Our data showed that running with foot orthoses altered neuromuscular control of running (as evidenced by modified recruitment strategies in VM, GM, and PL) and partly protected against postexercise deterioration of rapid force development characteristics in the ankle plantar flexors. However, these alterations were probably too small in magnitude to affect the aerobic cost of running.

![FIGURE 3](image1.png)  
**FIGURE 3**—RMS signal amplitude (μV) for VM, GM, PL, SO, and TA. EMG signals were recorded at 8-min intervals during a 1-h submaximal treadmill run, with (full line) and without (dotted line) foot orthoses. *Significant difference between conditions, as displayed for VM and GM (both P < 0.05). $Significant effect of time for PL (decline in first 18 min of running, P < 0.05).

![FIGURE 4](image2.png)  
**FIGURE 4**—Burst duration (ms) for VM, GM, PL, SO, and TA muscles. Data were recorded at 8-min intervals during a 1-h submaximal treadmill run, with (full line) and without (dotted line) foot orthoses. *Significant difference between conditions in PL (P < 0.05). $Effect of time in TA (decline in first 18 min of running, P < 0.05).
An important finding of this study is the lower RMS activity for VM and GM muscles, coupled with an increase in burst duration for PL during treadmill running while wearing foot orthoses. In light of Nigg’s (29) impact tuning theory, we suggest that a reduction of RMS in VM and GM while running with foot orthoses may be a neuromuscular adaptation to reduced demand for these muscles to control kinetics of the knee and rear foot (22). Our finding of increased PL duration is similar to that of Murley et al. (26), who also reported an increase in PL activity during walking with the use of foot orthoses in nonfatigued subjects. Longer burst duration in this muscle may be a strategy to counteract any lateral instability due to increased ankle inversion moments with the use of foot orthoses (23). In addition, Murley et al. (26) have suggested that this change may also help stabilization of the first metatarsal in late midstance and propulsion because this is a secondary function of PL. Interestingly, no interaction effects were reported between condition and time for any of the muscles. This may be explained by the lack of fatigue-induced modifications in recruitment of VM and GM because no interactions are likely to occur if there is no global effect of fatigue. This study is the first to describe changes in muscle activity in VM and GM initially and throughout the duration of a sustained submaximal run. Our results suggest that neuromuscular adaptations to foot orthoses are resistant to lower limb fatigue induced by a sustained bout of running.

It may seem somewhat contradictory that reduced muscle recruitment (as observed in this study) can be described as both beneficial (as a result of foot orthoses use) and potentially injurious (as a result of fatigue [41]). However, these findings need to be contextualized. Foot orthoses may reduce joint loading (23) and therefore may lead to reduced muscle recruitment because of less mechanical demand (29). On the other hand, in the context of fatigue, reduced muscle recruitment may occur because of reduced neural drive to the muscle or the inability of the muscle to contract (33–35). In this instance, the demands on the muscle have not been reduced; it is merely that the muscle simply cannot sustain the recruitment level (24). This may then lead to the increases in joint motion and loading that have been reported under fatiguing conditions (8–10,40,41).

In this study, we report that running with foot orthoses had no significant effect on \(\dot{V}O_2\) or RPE, despite altered neuromuscular control (as evidenced in the preceding paragraph). Our results are similar to those of Burkett et al. (3), who reported no difference in running economy with foot orthoses compared with shoes, after 7 min of running. However, the same study also reported no change in lower limb kinematics. Thus, it is possible that the biomechanical effect of their orthoses was too small to induce modifications in oxygen consumption. In addition, the orthoses used in the study of Burkett et al. (3) varied greatly in age (duration of prior use), materials, and prescription type, possibly confounding the results because of interindividual differences in biomechanical response to the intervention. Interestingly, in this study, we found a reduction of HR during the entire duration of the run (3%) with the use of foot orthoses. Lower HR values in the trial with foot orthoses may indicate that use of these devices may reduce the cardiorespiratory load imposed on the runner. However,
this result is in divergence with our findings in VO₂, where we report no difference between conditions. Sproule’s (37) finding that running economy is significantly impaired by running 1 h at 80% but not at 70% of maximum VO₂ may partly explain why alterations in neuromuscular control and HR did not relate to changes in VO₂ consumption. Thus, the relatively low running intensity endured by participants in our study (10% above ventilatory threshold) may not have been high enough to impair running economy. Thus, foot orthoses were unable to protect against fatigue-related declines in VO₂. In addition, intrindividual (test/retest) variability of VO₂ measures during running can be as high as 3% (136) and may also explain why we have reported a reduction in HR with foot orthoses use (3%) but no difference in VO₂.

Previous studies have shown that plantar flexor muscles are subjected to neural adjustments after fatiguing runs (33–35). Irrespectively of the condition, the ~9% reduction in PF MVC torque under fatigue is similar to existing literature for sustained submaximal running (7,33,35). In our experiment, normalized EMG values decreased during brief maximal contractions, supporting a potential neural origin in the reduced voluntary strength. At the peripheral level, the fall in resting M-wave amplitude suggests that a substantial part of the loss in force-generating capacity could be ascribed to a loss of excitability at the neuromuscular junction or along the muscle membrane (13). Moreover, the reduced peak twitch torque after the run, reported also after constant runs of the same nature (10,33,34), is an indirect sign of excitation–contraction coupling failure. Whatever the neural and muscular mechanisms contributing to alterations in strength capacity may be, it is important to note that none of the previously mentioned parameters were affected by the use of foot orthoses.

For the first time, we observed a ~17% decline in plantar flexors’ RFD₂₀₀ (200 ms) from before to after a 1-h treadmill run. Similar rapid muscle force-generating capacity changes were observed in knee extensors of fatigued football (~9% [38]) and handball (~16% to 21% [39]) players after simulated match play. Interestingly, the magnitude of exercise-induced reductions in rapid force development character-

| TABLE 1. Neuromuscular data obtained before and after the 1-h run performed with and without foot orthoses (N = 12, mean ± SD). |
|-----------------------------|-----------------------------|-----------------------------|-----------------------------|-----------------------------|
|                             | Without Orthoses | With Orthoses | Without Orthoses | With Orthoses | ANOVA Main Effects |
|-----------------------------|-----------------------------|-----------------------------|-----------------------------|-----------------------------|
| At rest | | | | |
| Peak twitch torque (Nm) | 16.4 ± 5.6 | 17.2 ± 5.2 | 14.9 ± 5.8 | 13.6 ± 4.5 | * |
| M-wave amplitude (mV)    | 4.4 ± 1.1 | 4.6 ± 1.0 | 4.0 ± 1.2 | 4.0 ± 1.2 | * |
| During contraction | | | | |
| MVC torque (Nm)           | 131.7 ± 29.1 | 133.2 ± 33.8 | 120.7 ± 25.3 | 119.0 ± 34.0 | * |
| RFD₂₀₀ (%max)            | 62.3 ± 9.1 | 69.6 ± 12.2 | 42 ± 7 | 64 ± 12 | * |
| Voluntary activation (%)  | 93.4 ± 11.8 | 92.0 ± 9.1 | 90.6 ± 11.9 | 94.0 ± 6.7 | * |
| Normalized RMS (au)       | 0.061 ± 0.027 | 0.063 ± 0.036 | 0.051 ± 0.019 | 0.043 ± 0.018 | * |
| M-wave amplitude (mV)     | 4.1 ± 1.3 | 4.1 ± 1.4 | 4.1 ± 1.2 | 4.2 ± 1.1 | * |

* Significant (P < 0.05) main effects of exercise.
** Significant (P < 0.05) main effects of condition.
*** Significant (P < 0.05) interaction between exercise and condition.

MVC torque, maximal isometric voluntary contraction torque; M-wave, muscle compound action potential; RFD₂₀₀, maximal rate of torque development obtained at 0–200 ms.

**Limitations.** The participants in this study displayed several characteristics of neuromuscular fatigue, as evidenced by reductions in maximal force production, maximal rate of force development, reduced M-wave, and voluntary EMG. However, the magnitude of fatigue may not have been sufficient enough to cause changes in muscle recruitment at a submaximal (running) level in GM or VM. This finding is similar to that of Finni et al. (10) who reported alterations in muscle recruitment at maximal but not at submaximal levels after a 10-km submaximal run. For the current study, the submaximal running speeds (10% above first ventilatory threshold) were chosen to best replicate the level of fatigue that may occur in recreational runners (8,9). Further studies may benefit from examining the accumulative effect of running during multiple days because this may also increase...
CONCLUSIONS

Wearing foot orthoses alters neuromuscular control during a submaximal 1-h treadmill run and partly protects from the resulting fatigue-induced reductions in rapid force development characteristics of the planar flexors. However, these changes may be too small to reduce the aerobic cost of running. Use of foot orthoses may be warranted for runners when reduced loading in VM or GM is deemed beneficial. In addition, the use of these devices may have some beneficial effects in reducing ankle plantar flexor muscle fatigue.

This work was not funded by the National Institutes of Health, Wellcome Trust, Howard Hughes Medical Institute, or any others. The authors have no financial interest in any of the products used in this study, nor do they have any other conflict of interest to report. The authors thank Ivana Matic for her assistance in data collection and analysis and Mohammed Aziz Farooq for his statistical advice. In addition, they thank Foot Science International for donation of foot orthoses used in this study.

The results of the present study do not constitute endorsement by the American College of Sports Medicine.

REFERENCES


