Abstract. [Purpose] There are no reliable evidences that the weakening of intrinsic foot muscles causes the decrease of the medial longitudinal arch (MLA) height. The purpose of this study was to confirm whether the fatigue of intrinsic foot muscles decrease the MLA height during standing and gait using 3D motion analysis system. [Subjects and Methods] Twenty healthy male subjects participated in this study. Foot kinematics was measured using an Oxford Foot Model before and after fatigue-inducing exercises of the abductor hallucis and flexor hallucis brevis muscles. [Results] Following fatigue-inducing exercise, in both standing and gait, the MLA height did not decrease but slightly increased. In addition, the reduction of a rear foot eversion angle was noted. [Conclusion] Fatigue of the abductor hallucis and flexor hallucis brevis muscles did not cause a change associated with collapsing of the MLA during both standing and gait. This suggested that the MLA support force from these muscles would be compensated by other MLA support structures, such as extrinsic foot muscles.

Key words: Intrinsic foot muscle, Fatigue, Foot kinematics

INTRODUCTION

The loss of the medial longitudinal arch (MLA) height has been described as a significant etiologic factor of several lower extremity injuries. Recently, several authors have reported that the intrinsic foot muscles have the capacity to support the MLA. These findings indicated that the intrinsic foot muscles provide some MLA support force as a part of the MLA support structures. However, because these muscles have a relatively small cross sectional area (CSA) and there are stronger MLA support structures, such as the extrinsic foot muscles, the idea that the weakening of the intrinsic foot muscles causes the decrease of the MLA height is doubtful. In individuals who may suffer overuse injuries related to a collapsing of the MLA such as plantar fasciitis and medial tibial stress syndrome, there is a possibility that the weakening of the intrinsic foot muscles is compensated by other MLA support structures. In fact, it was reported that subjects with pes planus had a smaller CSA of the intrinsic foot muscles and a larger CSA of the extrinsic foot inversion muscles. This is also important for some overuse injuries, because compensation is related to excessive stress.
There are a few studies with insufficient evidences of the weakening of the intrinsic foot muscles causing a decrease in the MLA height\(^6\)\(^\text{-}^7\). However, in these studies a navicular drop test (ND) was used to measure the change in the MLA height. The ND was performed by manually using a caliper, and the standard error of the ND was reported to be 1.68–2.57 mm\(^8\), \(^9\). So there is some doubt that the ND was an appropriate method to measure a 2–3 mm change in the MLA height in an experimental design that did not allow for a blinding of the examiner to the weakening status of the intrinsic foot muscles. Furthermore, although injuries related to the loss of the MLA height are attributed to overload during dynamic activities, there is no evidence that the weakening of the intrinsic foot muscles causes the decrease in the MLA height during dynamic activities, such as gait and running.

The Oxford Foot Model (OFM) is a 3D multi-segment foot model. The repeatability of the OFM has been reported to be good to excellent\(^10\). And the between-trial standard deviations calculated for the angle of the rear foot relative to the tibia and of the forefoot relative to the rear foot have been reported to be less than ± 0.7°\(^11\). Therefore, the OFM would be a more accurate and objective method than the ND and would enable measurement during dynamic activities.

The purpose of this study was to use an OFM to examine if the weakening of intrinsic foot muscles cause a decrease in the MLA height during standing and gait. Our hypothesis is that the weakening of the intrinsic foot muscles does not cause a decrease in the MLA height for both standing and gait conditions.

**SUBJECTS AND METHODS**

The weakening of the intrinsic foot muscles was simulated by fatigue. The abductor hallucis muscle (AH) which has the largest CSA of the intrinsic foot muscles\(^4\) was the main target to be fatigued and we chose repetition of flexion of the 1st MP joint as the method for fatiguing AH\(^7\). As a matter of course, this method included flexor hallucis brevis muscle (FHB) activity. Thus, in this study, we analyzed the change of foot kinematics during standing and gait that results from AH and FHB fatigue.

Twenty healthy male subjects (age: 22.0 ± 3.3 years; height: 171.7 ± 3.6 cm; mass: 64.4 ± 9.2 kg) participated in this study. The exclusion criteria included a history of a lower extremity injury up to six months prior to participation and excessive pronation determined by the ND. Excessive pronation was defined as a ND of >10 mm, similar to previous studies\(^8\), \(^9\). This study was approved by the Ethics Committee of the Prefectural University of Hiroshima (Approval Number: 15MH027), and written informed consent was obtained from all subjects.

Motion capture was conducted using a VICON system (Oxford Metrics, UK). The VICON system, comprised of 12 MX-T20S cameras, running at 100 Hz; 6 force plates running at 1,000 Hz (2 Kistler and 4 AMTI), was also used to identify the stance phase. Prior to the gait and standing analyses, the 29 reflective markers (9.5 mm and 14 mm) were placed on the pelvis and both lower extremities in accordance with the Plug-in-Gait lower model and OFM specifications\(^10\), \(^11\). The specific markers for the OFM were attached right lower extremity and these markers were 9.5 mm diameter. Then the calibration was started with the subjects in an anatomical neutral position. After that, medial malleolus, distal 1st metatarsal and posterior calcaneus proximal markers were removed, according to the protocol. Subsequently a static standing trial was conducted with 26 markers. The subjects were required to stand in a normal, relaxed upright position with a shoulder-width stance and each foot placed on an individual force plate. After approximately equal weight bearing was confirmed, recording for 5 seconds was conducted. Immediately after that the dynamic gait trials were conducted. The subjects were asked to walk on the 10 m walkway at their preferred normal speed and 5 gait trials were recorded. Upon completion of the fatigue-inducing exercises, these trials were immediately repeated. After static standing and gait trials in the pre-fatigue condition were completed, the markers attached to the right shank and foot were removed to perform the fatigue-inducing exercise. Because the places where these markers were attached were marked with a permanent marker beforehand, realignment of these markers to conduct post-fatigue trials could be done quickly and accurately.

The AH and FHB were fatigued using a self-fabricated pulley system (Fig. 1). The fatigue was confirmed using a dynamometer made of a strain gauge and surface EMG (TeleMyo G2 EMG instrument; Noraxon USA Inc., Scottsdale, AZ, USA). In this study, similar to previous studies\(^7\), \(^13\), AH and FHB fatigue is defined as at least a 50% reduction in force output of these muscles and a minimum of a 10% drop in median frequency (MedF) of AH. Force output of AH and FHB was represented by the 1st MP joint flexion torque. First, the baseline force output of AH and FHB and MedF of AH were determined. Subjects were placed in the long sitting position while the right ankle was fixed in the maximum plantar flexion position by a plaster cast. The dynamometer was placed under the 1st interphalangeal (IP) joint (Fig. 1). Two EMG electrodes were placed on the muscle belly of the AH parallel to the muscle fiber orientation\(^7\), and a ground electrode was placed on the tibia. The plaster cast had a window under the medial malleolus to enable installation of EMG electrodes (Fig. 1). The subjects were instructed to perform a maximum voluntary isometric 1st MP joint flexion against the dynamometer without flexing the 1st IP joint. At that time, the 1st MP joint flexion torque and MedF of AH were recorded simultaneously. This was repeated 3 times and there was a 60-second period between each of the 3 trials. The largest flexion torque and MedF of the 3 trials were used as the baseline. Then the dynamometer was removed, and the subjects performed the fatigue-inducing exercise. The fatigue-inducing exercise was performed in the same position as the baseline measurement, except the subjects had a leather belt attached to their 1st IP joint, which was used as a part of a self-fabricated pulley system (Fig. 1). The subjects performed a set of 75 isometric contractions of AH and FHB against a 1.4 kg weight, flexing the 1st MP joint without
flexing the 1st IP joint, starting with the great toe parallel to the wall and then having the pad of the great toe touch the wall. The subjects performed these contractions at a rate of 40 repetitions per minute as paced by a metronome. Following a set of 75 contractions, a 1st MP joint flexion torque and a MedF of AH was again recorded. This was repeated until a reduction in the 1st MP joint flexion torque of at least 50% was observed or the subjects were unable to follow a specific range of motion and rate. A final measurement of the 1st MP joint flexion torque was taken immediately following the post-fatigue gait and standing analyses. Because calculating the MedF of AH during a fatigue-inducing exercise was difficult, this was performed after all the experimental measurements were finished.

In the standing trials, rear foot eversion (frontal plane) angle relative to the tibia and forefoot dorsiflexion (sagittal plane), eversion (frontal plane) and abduction (transverse plane) angle relative to the rear foot associated with the decrease of the MLA height were selected for statistical analysis. Additionally, the MLA height defined as the normal distance of the plane of the forefoot from the proximal 1st metatarsal marker by the OFM was also selected. Each parameter of the intervals, excluding the first and last second of the 5-second recording, was averaged. In the gait trials, the gait velocity, the time of right stance phase and the parameters that were identical with that of the standing trials were selected for statistical analysis. In each foot kinematic parameters, the maximum and minimum values during stance phase were selected. Each parameter was averaged. To evaluate differences between pre- and post-fatigue conditions for all parameters, paired sample t-tests were conducted. Differences were considered significant at the $p<0.05$ level. All statistical tests were conducted using the SPSS 20.0 for Windows.

**RESULTS**

In this study, 4 subjects were excluded from the statistical analysis, because they had not achieved a drop in the MedF of AH of at least 10%. The rest 16 subjects performed $4.0 \pm 1.0$ sets of the fatigue-inducing exercise. Immediately following the post-fatigue standing and gait analyses, their 1st MP joint flexion torque had recovered to $63.7 \pm 11.1\%$ of baseline.

Table 1 shows the result of the standing analysis. Following the fatigue-inducing exercises, the rear foot eversion angle significantly decreased, and the MLA height defined by the OFM significantly increased.

Table 2 shows the result of the gait analysis. There were no statistically significant differences in the gait velocity and the time of right stance phase between pre- and post-fatigue conditions. Following the fatigue-inducing exercises, similar to the standing analyses, both the maximum and minimum rear foot eversion angles during the stance phase significantly decreased, and maximum MLA height defined by the OFM during the stance phase significantly increased. Typical forefoot and rear foot motions and MLA height variations in the stance phase were shown in Fig. 2.

**DISCUSSION**

The purpose of this study was to confirm whether the weakening of the AH and FHB caused a decrease in the MLA height during standing and gait. In this study, the fatigue-inducing exercise for the AH and FHB decreased the rear foot eversion angle in both standing and gait, which is generally associated with an increase in the MLA height. Moreover the MLA height
defined by the OFM also showed an increase. Although these changes were small, it was confirmed that the weakening of these muscles did not cause the decrease of the MLA height not only during gait but also during standing. Probably, the MLA support force from these muscles could be compensated by other MLA support structures, such as the extrinsic foot inversion muscles, which was also suggested by the significant decrease of the rear foot eversion angle after the fatigue-inducing exercise. Previously, Pohl et al.13) reported no substantial changes in foot kinematics following the fatigue-inducing exercise of the tibialis posterior, although the tibialis posterior was regarded as a main MLA support structure14, 15). They also concluded that a reduced force output of the tibialis posterior might be compensated for by other muscles. Because such compensation may bring excessive stress to the limited structures, this is useful information for some lower extremity overuse injuries related to a collapsing of the MLA, such as in a medial tibial stress syndrome. Therefore, future studies using EMG are needed to confirm the compensatory strategies employed by extrinsic foot muscles.

This study had several limitations. First, though our results of the standing analysis disagreed with previous studies 6, 7), there was a difference in the method of MLA height measurement. However, the OFM is a more accurate and objective method than the ND used in previous studies. Moreover, in disagreement with previous studies, we believe that following our fatigue-inducing exercise the navicular tuberosity landmark for the ND elevated, because the rear foot eversion angle decreased after the fatigue-inducing exercise, which is associated with the elevation of the navicular tuberosity. Therefore, we consider that our results to be more reliable.

### Table 2. Group mean ± SD gait parameter and foot kinematics variables during gait

| Variables                  | Pre       | Post      |
|----------------------------|-----------|-----------|
| Gait velocity (m/s)        | 1.3 ± 0.1 | 1.3 ± 0.07|
| Time of stance phase (s)   | 0.6 ± 0.03| 0.6 ± 0.03|
| MLA height (mm)            |           |           |
| max                        | 13.6 ± 2.4| 14.6 ± 2.7|
| min                        | 7.1 ± 2.4 | 7.7 ± 2.8 |
| Forefoot angle (*)         |           |           |
| DF max                     | 6.9 ± 3.2 | 6.6 ± 3.5 |
| min                        | −2.6 ± 3.6| −2.9 ± 3.7|
| EV max                     | 2.8 ± 3.9 | 3.8 ± 5.0 |
| min                        | −4.0 ± 5.0| −3.7 ± 6.1|
| AB max                     | 10.0 ± 4.9| 9.2 ± 3.5 |
| min                        | 3.2 ± 4.0 | 2.3 ± 3.2 |
| Rearfoot angle (*)         |           |           |
| EV max                     | −0.9 ± 4.4| −2.3 ± 5.2|
| min                        | −10.7 ± 5.0| −12.6 ± 6.1|

DF: dorsiflexion; EV: eversion; AB: abduction

*p<0.01

**Fig. 2.** Typical forefoot and rearfoot motions and MLA height variation in the stance phase

The solid line circle shows maximum value point and the dashed line circle shows minimum value point.

HC: heel contact; TO: toe off
Second, we did not measure the EMG activity of the flexor hallucis longus, one of the extrinsic foot muscles. Therefore, we cannot be completely sure that this muscle was not active during the fatigue-inducing exercise. In this study, besides using the method of Headlee et al.7 that required flexion of the MP joints without flexion of the IP joints to reduce the extrinsic foot muscles activity, we fixed the subject’s ankle in a maximum plantar flexion position using a plaster cast. In this position, the extrinsic foot flexors would be in a shortened position and, therefore, less able to generate a force.16, 17 Furthermore, the activity of the extrinsic foot muscles as fixators would also be decreased. Therefore, we believe that our fatigue-inducing exercise is the most effective method for minimizing activity of the extrinsic foot muscles.

Next, it could be possible that the fatigue-inducing exercise caused pain or muscle shortening, which could have affected the results of this study. Prior to this study, we performed a pilot study18 that examined the influence of the fatigue protocol used in the present study on plantar pressure distribution during gait. In this pilot study, we confirmed that the increase of plantar pressure on the lateral regions of the foot associated with an avoidance response from pain of the AH and FHB did not occur. In addition, we confirmed that a decrease in the plantar pressure on the region of the great toe would not effect muscle shortening of the AH and FHB.

Finally, because lower extremity overuse injuries related to a collapsing MLA are common in athletes who perform high intensity activities, e.g. running19, 20, future studies should be performed during activities with higher intensity.

In conclusion, the fatigue of the AH and FHB did not cause changes in foot kinematics associated with a decrease in the MLA height, not only during gait but also during standing. This suggests that other MLA support structures may have compensated for the weakening of these muscles.

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