The Role of Passive Plantar Flexion in Floating Toes Following Weil Osteotomy

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Floating toes are a common complication following Weil osteotomy. The toes are passively plantarflexed via the windlass mechanism, which may play a role in floating toe. Five cadaver lower limb specimens were loaded on a custom frame and 3 different interventions were tested, including control group, Weil osteotomy group, and Weil osteotomy plus plantar plate-shortening group. The extensor tendon to the second toe was loaded with 20 Newtons of tension during the trials, and non–weight-bearing and simulated weight-bearing radiographs were taken to measure the metatarsophalangeal joint extension angle. The extension angle was: 8.00° ± 1.41° in the Weil osteotomy group, and 6.60° ± 1.15° in the Weil osteotomy plus plantar plate–shortening group. Comparison of the amount of passive plantarflexion between the groups revealed statistically significant changes between the control and Weil osteotomy groups (P = .0001), and the Weil osteotomy compared with the Weil osteotomy plus plantar plate–shortening (P < .0001); whereas no statistically significant difference was observed between the control and Weil osteotomy plus plantar plate–shortening groups (P = .0893). These results support the idea that the toes undergo passive plantar flexion due to the windlass mechanism, which is dampened by the Weil osteotomy. Dampening of the windlass mechanism may be responsible for floating toe following a Weil osteotomy. Level of Clinical Evidence: 5 (The Journal of Foot & Ankle Surgery 47(6):520–526, 2008)

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The plantar plate and collateral ligaments are commonly considered the primary stabilizers of the lesser metatarso-phalangeal (MTP) joints (1–4). Although the plantar plate has been statistically shown to significantly restrain dorsal dislocation of the second MTP joint (2, 3), it's role as a dynamic stabilizer of the toe is not commonly addressed. Because plantar plate rupture may lead to an extension deformity of the MTP joint as seen in claw, hammer, and floating toes (5–9), it is hypothesized that sectioning the plate will have a similar effect.

The distal attachment of the plantar plate is onto the proximal phalanx and most of its proximal attachment is to a slip of plantar fascia forming a plantar fascia/plate complex. There is only a tenuous connection to the metatarsal in the form of a synovial fold (5, 6), suggesting that the plantar plate cannot act as a ligament and thus there must be an alternative method by which the MTP joint is stabilized.

Given the anatomic connection between the toes and the plantar plate, it is the belief of the authors that the plantar fascia plays a considerable role in the function of the toes. Weil osteotomies have demonstrated effectiveness in treating lesser metatarsal overload by off-loading the lesser MTP joints (10–15). There is, however, a consistently high prevalence of floating toe following Weil osteotomy, and published reports describe the range to be 20% to 68% (10–13). It should be noted that the Weil osteotomy, even when resulting in the development of a floating toe, consistently relieves metatarsalgia and other digital and metatarsal symptoms (10–13). In fact, the Weil osteotomy is considered by many to be a proven procedure that is effective in relieving pain, has fewer complications than a Helal osteotomy, and rarely results in a metatarsal nonunion (12, 14–16). Interestingly, the development of a transfer lesion following this procedure is less common than is floating toe (10, 11, 13, 15, 16).

Some theories on the etiology of floating toe following a Weil osteotomy concentrate on excessive dorsiflexion of the MTP joint (16). However, the authors, as well as others (17), consider floating toe to result from a lack of plantarflexion with weight bearing, rather than excessive dorsiflexion. Hicks (18) proposed that the toes are actively plantarflexed in late midstance by the flexors and passively by the windlass mechanism. McGlamry (17) stated, “... floating toe is caused by a failure of the flexor mechanism. This usually involves a slip of the plantar fascia that fails to load with weight bearing.” It is generally understood that tension...
in the plantar fascia is critical to the function of the plantar fascia (windlass mechanism). As such, shortening the metatarsal could reduce tension in the plantar fascia and possibly cause a dampening of the windlass mechanism. Laxity in the plantar fascia, or dampening of the windlass mechanism, may be responsible for the development of a floating toe following Weil osteotomy.

The purpose of this study was to determine if the passive plantarflexory mechanism of the toe was dampened by a Weil osteotomy. It was further hypothesized that if the loss of tension in the plantar fascia were observed to be responsible for the development of a floating toe, then restoration of tension, by means of shortening the plantar plate, would result in a measurable degree of passive MTP joint plantarflexion.

Materials and Methods

Specimen Acquisition and Preparation

Five fresh-frozen cadaveric lower limb specimens, ranging from 62 to 73 years of age at the time of death, were obtained from the Department of Biological Services at the University of Washington. All of the specimens had intact foot and ankle joints and were without visible deformity. The specimens were preserved by wet towel wrappings and deep frozen to ~20°C. Before testing, each specimen was completely thawed to room temperature, and dissection of the second ray was carried down to the MTP joint, with care being paid to preserve the long extensor tendon. The metatarsal was exposed at a level approximately 1 cm proximal to the MTP joint, and the collateral ligaments of the MTP joint were released to gain access to the metatarsal head. The base of the proximal phalanx, just distal to the MTP joint, was also exposed. A plantar intermetatarsal incision was then made lateral to the second metatarsal, and dissection was carried down to the level of the long flexor sheath. The sheath was incised in a linear fashion to allow retraction of the tendons and gain full exposure to the plantar plate. Proximally, the Achilles and extensor digitorum longus tendons were isolated and a Dacron (DuPont, Wilmington, DE) cord was used to trap them in order to apply tensile loads, allowing manipulation at the ankle and second toe by means of a pneumatic actuator.

Marker Placement

Radiopaque markers were placed in the proximal phalanx and metatarsal head, using a 0.062-inch Kirschner wire (K-wire) to drill a canal in the metatarsal approximately 15 mm proximal to the MTP joint, and in the proximal phalanx approximately 5 mm distal to the joint. Care was taken to orient the canals perpendicular to the long axis of each bone, and the plantar cortex was preserved at each site. A 0.054-inch K-wire was then manually inserted into each canal and cut flush to the dorsal cortex, thereby leaving a 3- to 5-mm linear marker for radiographic identification of MTP joint movement.

Loading Frame

All of the specimens were mounted and tested on a custom loading frame designed by BioConcepts, Inc., Seattle, Washington, and fabricated by Advanced Biomedical, Inc., Oakland, California. The frame was designed so that an axial load could be applied through a polycarbonate rod to the tibia and fibula via a central actuator that allowed for near physiologic loading. Smaller actuators surrounded the central actuator, and these were used to apply tensile loads through the extrinsic tendons. The testing platform was covered with a nonskid surface to prevent specimen slippage during mechanical loading.

Testing Trials

A total of 3 trials were run for each of the 5 specimens, including a control, a Weil osteotomy, and a Weil osteotomy combined with shortening of the plantar plate. In the control group, non–weight-bearing and weight-bearing lateral radiographs were taken for each trial. The non–weight-bearing radiographs were taken with the foot at 90° to the leg, under no axial load and without tension in the Achilles tendon, while 20 Newtons of tension was placed on the long extensor tendon in order to reorient the foot in the same alignment as in the weight-bearing views. Weight-bearing radiographs were taken with loads of 400 Newtons placed axially down the tibia and 250 Newtons on the Achilles tendon. A Weil osteotomy was then performed on the second metatarsal, approximately 1 mm from the dorsal edge of the cartilage. Care was taken to ensure that the saw blade was parallel to the weight-bearing surface of the foot. The capital fragment was then translated proximally for a distance of 5 mm, after which the fragment was fixated with two 0.054-inch threaded K-wires. The dorsal overhang, caused by the proximal translation of the capital fragment, was removed. Non–weight-bearing and weight-bearing lateral radiographs were then taken in the fashion previously described for the control model.

Following testing of the Weil osteotomy model, a #15 scalpel blade was used to remove a 3-mm wedge of tissue from the plantar plate. The proximal and distal margins of the plantar plate were then reapproximated using 2-0 nylon sutures, thereby shortening it. In a fashion similar to that described for the control and Weil osteotomy models, the plantar plate–shortening model was then tested and the lateral radiographs were obtained.
Metatarsophalangeal Joint Extension Angle

The lateral MTP joint extension angle was measured for all trials in the control, Weil osteotomy, and plantar plate–shortening models. The extension angle was defined by the resultant angle created by the intersection of the sagittal plane longitudinal axes of the second metatarsal and proximal phalanx (Figure 1). The angles were measured and recorded by independent investigators blind to the intervention.

Statistical Plan

The results of the trials were statistically analyzed and described in terms of means and standard deviations. Two-tailed, paired Student t tests were used to compare the non–weight-bearing to simulated weight-bearing second MTP joint angles for the control, Weil osteotomy, and Weil osteotomy plus plantar plate–shortening groups. Two-tailed, unpaired Student t tests were used to compare the change in the radiographic second MTP joint angle between the intervention groups.

Results

The MTP joint extension angle in the control group averaged 11.20° ± 3.42° of plantarflexion upon simulated weight bearing (Table 1). Passive plantarflexion was observed when axial load was applied to the tibia and tension was placed on the Achilles tendon without tension being applied to the long flexor tendon. Passive plantarflexion was eliminated after execution of the 5-mm shortening Weil osteotomy and the MTP joint extension angle averaged 0.40° ± 0.89° of plantarflexion upon simulated weight bearing in this group (Table 2). Passive plantarflexion was partially reestablished after shortening the plantar plate by 3 mm, and the MTP joint extension angle averaged 8.00° ± 1.41° of plantarflexion upon simulated weight bearing in this group (Table 3). Tables 1 to 3 show that, in the control group, the change between the extension angle between the non–weight-bearing and weight-bearing conditions was statistically significant (P = .0019); in the Weil osteotomy group, the change was not statistically significant (P = .3739); and in the Weil osteotomy plus plantar plate–shortening group, the change was statistically significant (P = .0002).

The results of the comparison of the change in the radiographic second MTP joint angle, by intervention group, are depicted in Table 4. There was a statistically significant difference between the control and Weil osteotomy groups in regard to the amount of passive plantarflexion observed.
with simulated weight bearing (P = .0001). There was also a statistically significant difference in passive plantarflexion between the Weil osteotomy group and the Weil osteotomy plus plantar plate–shortening group (P < .0001). However, there was no statistically significant difference in passive plantarflexion between the control group and the Weil osteotomy plus plantar plate–shortening group (P = .0893).

Discussion

The current study supports the theory that toes possess a passive plantarflexory mechanism, which is engaged upon weight bearing (Figure 2). Simulated weight bearing in the control group caused an average of 11.2° ± 3.42° of plantarflexion at the second MTP joint when 20 Newtons of force was applied to the extensor tendon (Table 4). This reduction of the MTP joint extension angle was observed on consecutive lateral radiographs in both non–weight bearing and simulated weight-bearing views, and we believe that the observed plantarflexion occurred as a result of axial load on the tibia and tension on the Achilles tendon without tension on the muscles of the toes. It should also be noted that passive plantarflexion of the toes upon weight bearing is not a novel observation. According to Sarafian (19), as early as 1910, Fick recognized that the plantar fascia flexed the toes. Furthermore, Hicks (18) noted that the windlass mechanism also worked in reverse, in that flattening the arch upon weight bearing tenses the plantar fascia and plantarflexes the toes. Sheck (20), in a landmark article on the etiology of

### TABLE 3 Passive plantarflexion angular change (°) with simulated weight bearing following Weil osteotomy with plantar plate shortening

<table>
<thead>
<tr>
<th>Trial</th>
<th>Weil Osteotomy + Plantar Plate Shortening Group (n = 5)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Non–Weight Bearing (°)</td>
<td>Weight Bearing (°)</td>
</tr>
<tr>
<td>1</td>
<td>43</td>
<td>34</td>
</tr>
<tr>
<td>2</td>
<td>34</td>
<td>25</td>
</tr>
<tr>
<td>3</td>
<td>41</td>
<td>32</td>
</tr>
<tr>
<td>4</td>
<td>40</td>
<td>34</td>
</tr>
<tr>
<td>5</td>
<td>47</td>
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</tr>
<tr>
<td>Mean average</td>
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<td>33</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>4.74</td>
<td>5.39</td>
</tr>
</tbody>
</table>

*P value* .0002

*Two-tailed, paired Student’s t-test

### TABLE 4 Comparison of the change in the radiographic second metatarsophalangeal joint angle (°), by intervention group

<table>
<thead>
<tr>
<th>Trial</th>
<th>Radiographic Second MTPJ Angle (°) by Intervention Group</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control Group</td>
<td>Weil Group</td>
</tr>
<tr>
<td>1</td>
<td>12</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>16</td>
<td>2</td>
</tr>
<tr>
<td>5</td>
<td>12</td>
<td>0</td>
</tr>
<tr>
<td>Mean average</td>
<td>11.2</td>
<td>0.40</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>3.42</td>
<td>0.89</td>
</tr>
</tbody>
</table>

*P value* .0001†

†Comparison of control group to Weil osteotomy group.
‡Comparison of control group to Weil osteotomy plus plantar plate-shortening group.
§Comparison of Weil osteotomy group to Weil osteotomy plus plantar plate–shortening group.

**Abbreviations:** MTPJ, metatarsophalangeal joint; Weil, Weil osteotomy; Weil + PPS, Weil osteotomy combined with plantar plate shortening.

**FIGURE 2** (A) This photograph demonstrates a non–weight-bearing foot. Note the position of the toes relative to the platform. The toes are positioned off of a ledge to accentuate the effect, as described by Stainsby (8). (B) This image demonstrates a weight-bearing foot. Note the passive plantarflexion of the toes at the metatarsophalangeal joints.
hammertoes, recognized the importance of the passive plantarflexory mechanism and emphasized the dynamic nature of the plantar joint capsule (plantar plate) and plantar fascia in regard to stability of the MTP joint.

The plantarflexory force caused by the plantar fascia is not trivial. Hamel et al (21) performed a cadaver study to isolate the relative force placed on the toes by the active muscular component and the passive component contributed by the plantar fascia. The study included pedobarographic testing to determine the relative pressure of the toes on the ground. When isolating the plantar fascia component, those investigators avoided load on the muscle and, when isolating the pull of the muscle, they sectioned the plantar fascia proximally to nullify its effect. Hamel found that the passive force provided by the plantar fascia was greater than the active force provided by the muscular component, however the observed difference was not statistically significant (21). Although the plantarflexory forces applied by the 2 components were not statistically significantly different, Hamel noted that when the plantar fascia was sectioned and the muscles were tensioned, the toes became hammered at the MTP joints. This led to the conclusion that the plantar fascia not only plantarflexed the toes but also stabilized the MTP joint, thereby enabling the muscles of the toes to function properly (21).

The current study demonstrated that the passive plantarflexory mechanism of the toes was compromised following Weil osteotomy. The passive plantarflexion observed upon simulated weight bearing in the control group was eliminated following the 5-mm shortening Weil osteotomy, and there was a statistically significant difference between the amount of passive plantarflexion in the control group and that observed in the Weil osteotomy group ($P < .0001$) (Table 4). Moreover, in this group, loading the tibia and the Achilles tendon did not plantarflex the toe against resistance, unlike that observed in the control model. There was no change in the MTP joint extension angle in the control group and following the Weil osteotomy with plantar plate shortening (Tables 1 and 3), and the average extension angles were $39.80^\circ \pm 5.26^\circ$ and $41.00^\circ \pm 4.74^\circ$, respectively. This indicated that shortening the plantar plate did not limit dorsiflexion of the MTP joint. It is the belief of the investigators that laxity in the plantar fascia/plate complex following shortening of the metatarsal is responsible for the loss of passive plantarflexion of the toe (Figure 3).

Further evidence that the fascia was responsible for the observed plantarflexion upon simulated weight bearing was obtained when it was noted that shortening the plantar plate led to partial restoration of passive plantarflexion upon simulated weight bearing. The MTP joint measured an average of $8.00^\circ \pm 1.41^\circ$ of plantarflexion upon simulated weight bearing following the plantar plate shortening (Table 3 and 4). There was a statistically significant difference between the Weil osteotomy and the Weil osteotomy plus plantar plate–shortening groups ($P < .0001$) in observed weight-bearing plantarflexion (Table 4). However, no statistically significant difference was observed between the control group and Weil osteotomy plus plantar plate–shortening groups ($P = .0893$) (Table 4). Furthermore, passive plantarflexion of the toe consistently occurred without tension on the muscle to the toes in all of the trials.

Shortening the plantar plate did not restrict dorsiflexion of the MTP joint, although it did reestablish tension between the plantar fascia and the proximal phalanx, causing passive plantarflexion of the toe. Removing a 3-mm wedge from the plantar plate eliminated some of the laxity in the plantar fascia–plantar plate complex created by the Weil osteotomy, whereas removal of the full 5 mm potentially could have reestablished all of the

**FIGURE 3** This illustration demonstrates passive plantarflexion of the metatarsophalangeal joint. (A) Non–weight-bearing view of lesser metatarsophalangeal joint. (B) Weight-bearing view, note the plantarflexory position of the proximal phalanx in response to a traction force on the plantar fascia. (C) After a Weil osteotomy, the tension in the plantar fascia is reduced secondary to shortening of the metatarsal segment and the fascia fails to plantarflex the proximal phalanx.
tension in the plantar fascia; removing such a wedge would not, in the authors’ opinion, represent common surgical practice. When repairing torn plantar plates the authors occasionally remove wedges of frayed tissue, but the wedges do not usually exceed 3 mm.

It was not the aim of this study to advocate plantar plate shortening for the treatment of a floating toe. The plantar plate was, however, chosen as the site best suited to surgically eliminate laxity along the plantar fascia/plate complex for 2 reasons. The first was that the plantar plate was simply a convenient anatomical location, and the robust nature of the tissues readily allowed for suture placement. Preliminary trials attempted to place sutures through fascia proximally, but its transverse fibers did not provide adequate hold. The second reason was to emphasize the dynamic role of the plantar plate in stabilizing the MTP joint.

Traditionally, the plantar plate is thought to be most responsible for the stability of the lesser MTP joints (1–4). The stabilizing effect of the plantar plate on the MTP joint has been experimentally studied (2, 3). Bhatia et al (2) investigated the restraints of the second MTP joints and concluded that the plantar plate, along with the collateral ligaments, were the strongest restraints to dislocation. Ford et al (3), in a similar study, compared the stabilizing effect of a flexor digitorum longus transfer to primary repair of the plantar plate. They concluded that primary repair of the plantar plate was a viable alternative to flexor tendon transfer for stabilizing the second MTP joint (3). Both studies measured the force required to dorsally dislocate the MTP joint statically but did not consider a dynamic situation. As stated earlier, the plantar plate does not have a substantial attachment to the metatarsal (Figure 4) and, as such, cannot behave like a ligament across the MTP joint (5, 6). It is the belief of the authors that the role of the plantar plate in stabilizing the MTP joint is the result of passive plantarflexion upon weight bearing, rather than functioning as a robust ligament. In the current investigation, all of the soft tissue dissection required to expose the metatarsal head dorsally, as well as that required to expose the plantar plate, was done before any measurement trials. This ensured that the differences observed with simulated weight bearing and the passive plantarflexory mechanism could not be attributed to any other soft tissue structures, including the collateral ligaments of the MTP joint.

Other theories related to the development of a floating toe following Weil osteotomy concentrate on excessive dorsiflexion as opposed to the current theory, which views the problem as lack of passive plantarflexion. The deformity in floating toe is centered at the MTP joint and it has long been thought that the absence of plantarflexion and stability at the MTP joint will allow the long flexors to hammer the toes (22). One of the latest theories on floating toe following a Weil osteotomy focuses on the position of the lumbricals relative to the joint axis (16). Inadvertent plantarflexion of the capital fragment in a Weil osteotomy is thought to shift the mechanical axis of the lumbricals, thereby changing their action from plantarflexory to dorsiflexory (16). However, this theory may overemphasize the role of the lumbricals, since these muscles have a small diameter and are consequently weak. Interestingly, Hamel et al (21) further demonstrated that the passive plantarflexory mechanism alone prevented the flexors from extending the MTP joints and, when the passive plantarflexory mechanism was eliminated, the extrinsic flexors caused hammertoes. Lack of the passive plantarflexory mechanism may account for MTP joint extension in ruptured or attenuated plantar plates, and this explains the association of hammer, claw, and floating toes with plantar plate ruptures (7–9). In such cases, the axes of the lumbricals are not changed, yet there is extension of the MTP joint.
It should also be noted that the proposed mechanism, in which shortening the metatarsal creates laxity in the plantar fascia, is not specific to the Weil osteotomy. Any shortening metatarsal osteotomy can theoretically cause laxity in the plantar fascia, and thus has the potential to cause a floating toe. Although other shortening osteotomies may not have as many reported cases of floating toe as the Weil osteotomy, this observation may simply be indicative of the popularity of the Weil procedure.

An apparent contradiction to the current theory is the lack of correlation between the amount of shortening in the metatarsal and floating toe (10, 11). One explanation for this finding is that the degree of arch height loss in individual feet differs greatly. The windlass mechanism requires tension in the plantar fascia, and this depends on the degree of arch height loss in the individual foot. It is logical to assume that the greater the amount of metatarsal shortening, the greater will be the amount of arch flattening required to compensate for the laxity created in the plantar fascia. Still further, individual feet may be able to compensate to a greater or lesser degree than others, in this regard.

There are several shortcomings of this study. First, conclusions gleaned from the cadaveric models may not translate into the clinical realm. Additionally, the amount of force placed on the toes by the passive mechanism was not measured directly, but it was measured indirectly by plantarflexing the toes against resistance and measuring the resultant change in the sagittal plane MTP joint extension angle. Furthermore, the Weil osteotomy displayed an average of less than 1° of change in the MTP extension angle from non-weight bearing to weight bearing (Table 2). As such, the authors cannot say with certainty that there was a complete lack of passive force following the osteotomy, only that the force was insufficient to overcome the 20 Newtons of force placed on the extensor tendon. Still further, attention was focused on the MTP joint and we did not actually compare the position of the toe to the ground. It is possible to have an extended MTP joint with a toe that purchases the substrate. The authors also did not test the assumption that any shortening of the metatarsal would dampen the windlass mechanism. Finally, the study could have been conducted using other osteotomies, although the primary interest of the authors was to investigate the influence of the Weil osteotomy.

In conclusion, this study supports the idea that the toes are passively plantarflexed upon weight bearing, and this is most likely caused by the plantar windlass mechanism. The passive plantarflexory effect is dampened following execution of a Weil osteotomy, and it is partially reestablished upon shortening of the plantar plate. Dampening of the windlass mechanism likely plays a role in the development of a floating toe deformity following a Weil osteotomy, and may be prevented by surgically shortening the plantar plate.

References