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FOOT & ANKLE INTERNATIONAL


# Role of the Peroneal Tendons in the Production of the Deformed Foot with Posterior Tibial Tendon Deficiency

Mark S. Mizel, M.D.,* H. Thomas Temple, M.D.,† Pierce E. Scranton, Jr., M.D.,‡ Richard E. Gellman, M.D.,§ Paul J. Hecht, M.D.,‖ Greg A. Horton, M.D.,¶ Leland C. McCluskey, M.D.,♦ and Kathleen A. McHale, M.D.♠
*Boston, Massachusetts, Charlottesville, Virginia, Seattle, Washington, Charlotte, North Carolina, Philadelphia, Pennsylvania, Kansas City, Kansas, Columbus, Georgia, and Washington, D.C.*

## ABSTRACT

**Ten patients were identified with traumatic, complete common peroneal nerve palsy, with no previous foot or ankle surgery or trauma distal to the knee, who had undergone anterior transfer of the posterior tibial tendon to the midfoot. Six of these patients had a transfer to the midfoot and four had a Bridle procedure with tenodesis of half of the posterior tibial tendon to the peroneus longus tendon. Average follow-up was 74.9 months (range, 18–351 months). All patients' feet were compared assessing residual muscle strength, the longitudinal arch, and motion at the ankle, subtalar, and Chopart's joint. Weightbearing lateral X-rays and Harris mat studies were done on both feet. In no case was any valgus hindfoot deformity associated with posterior tibial tendon rupture found. It seems that the pathologic condition associated with a posterior tibial tendon deficient foot will not manifest itself if peroneus brevis function is absent.**

## INTRODUCTION

The pes valgus deformity associated with posterior tibialis tendon incompetence or rupture has been well described.[8,9,13] After tendon rupture, the deformity is progressive with varying degrees of hindfoot valgus, midfoot collapse, and forefoot pronation ("too many toes" sign)[8] as common physical manifestations. Early surgical correction of the painful physical manifestations of the progressive deformity have generally addressed the medial side of the foot. Musculotendinous reconstruction and/or augmentation, ligamentous imbrication, arthrodeses, or hindfoot osteotomies have all been described as reconstructive alternatives.[1,5,6,8,9,11,13,17]

The primary evertor of the foot and ankle is the peroneus brevis.[2] This muscle opposes the primary invertor of the foot, which is the posterior tibialis. The balance between these muscles can be disrupted with rupture of the posterior tibialis tendon. This has been previously noted by Mann and Thompson.[13] The role of the peroneus brevis in producing pes valgus deformity, however, has not been thoroughly investigated.

The authors decided to evaluate arch structure and foot function in patients with no functioning peroneus brevis and no functional posterior tibial tendon. The authors evaluated patients that had sustained a complete common peroneal nerve injury and that had had a subsequent posterior tibialis tendon transfer to provide dorsiflexion function to the ankle. The evaluation of these patients' feet after sufficient follow-up should provide insight into the posterior tibial tendon-deficient foot and whether the peroneal tendons have a role in the creation of progressive pes valgus.

## MATERIALS AND METHODS

Ten patients were identified who had sustained a common peroneal nerve palsy and had also undergone an anterior transfer of the posterior tibial tendon as treatment for loss of active ankle dorsiflexion. These patients had a posterior tibial tendon transfer, with rerouting of the posterior tibial tendon to the anterior foot, at an average of 16.9 months (range, 7–41 months) after nerve injury. Four patients had a Bridle procedure,[15,18,19] which is tenodesis of half of the posterior tibial tendon to the peroneus longus tendon. None of the patients had any other foot or ankle surgical procedures before the transfer. Patients who sustained traumatic injury distal to the knee or

* Boston University, Boston, Massachusetts. To whom requests for reprints should be addressed at Boston Medical Center, 818 Harrison Avenue-Dowling 2 N, Boston, MA 02118.
† University of Virginia, Charlottesville, Virginia.
‡ Seattle, Washington.
§ Miller Orthopaedic Clinic, Charlotte, North Carolina.
‖ Allegheny University, Philadelphia, Pennsylvania.
¶ University of Kansas, Kansas City, Kansas.
♦ The Hughston Clinic, Columbus, Georgia.
♠ Walter Reed Army Medical Center, Washington, D.C.

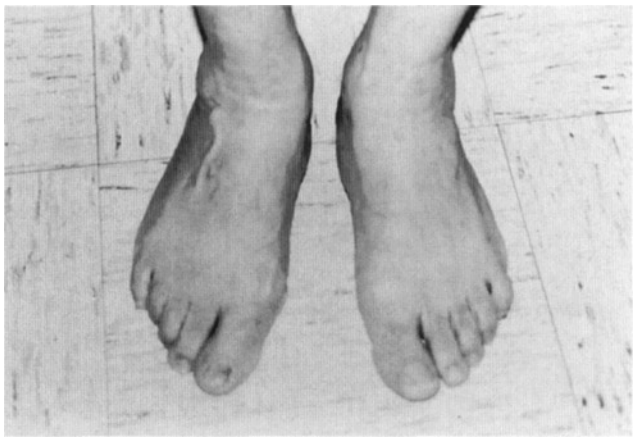

**Fig. 1.** Feet of a 34-year-old man who sustained a knee dislocation and common peroneal palsy. The patient is 114 months status post anterior transfer of the posterior tibial tendon. Both feet, with the affected right foot on the left side of the picture.

had leg, foot, or ankle surgery before the transfer were not included in the study.

Each patient had a bilateral foot and ankle physical examination, evaluating the appearance of the arch as well as motion of the ankle, subtalar, and Chopart's joint. Standing lateral roentgenograms and Harris mat studies of both feet were performed. The visual inspection of each involved foot focused on the presence or absence of hindfoot valgus or midfoot collapse. Other factors analyzed included: (1) whether or not subsequent foot or ankle surgery had been performed, (2) whether there was difficulty with ambulation, and (3) whether or not these patients required the use of ambulatory braces or orthoses.

## RESULTS

There were six men and four women. Seven patients sustained common peroneal palsy as a result of a knee injury, including four from a dislocated knee. One resulted from a dislocated hip, one from a malposition during a surgical procedure, and one from a suture used in a lateral meniscal repair of the knee. Nine patients (9 of 10) had electromyography to confirm a complete common peroneal nerve palsy. There were six right and four left lower extremities, and the average age of the patients at injury was 30.6 years (range, 16–51 years) and at follow-up was 38.0 years (range, 20–55 years). All patients were seen and evaluated by an orthopaedic surgeon at an average of 74.9 months (range, 18–351 months) after the tendon transfer.

Clinical inspection of these patients' feet demonstrated the dorsal view (Fig. 1); the forefoot was abducted in one patient. No other patient had any abduction noted.

Physical examination also showed that ankle range of motion of the affected limb was 21° (range, 0°– 35°) less than the contralateral limb. Subtalar joint motion was decreased in three (30%) of the affected feet, as compared with the contralateral foot, and seven (70%) had an equal range of motion bilaterally. Transverse tarsal joint motion was equal in all affected feet, as compared with the contralateral feet. All patients were able to stand on their toes, although no inversion was seen in the affected hindfoot.

Manual muscle testing about the ankle revealed no eversion strength in the six feet with implantation of the tendon into the midfoot. In the Bridle transfer patients, however, eversion strength was strong enough to oppose gravity. Inversion strength in all feet tested demonstrated inversion strength that was sufficient to oppose gravity. Dorsiflexion strength in all affected limbs was stronger than gravity but weaker than the normal contralateral foot, and plantarflexion strength of all the affected ankles was normal.

Weightbearing lateral roentgenograms of the feet revealed no loss of longitudinal arch, on careful visual inspection (Fig. 2). Measurement of the talar-metatarsal angle, compared with the contralateral foot, demonstrated a difference of 2.7° (range, 0°–10°). One lateral x-ray taken 18 months after transfer showed some widening of the plantar first-metatarsal first-cuneiform joint. Harris mat studies (Fig. 3) demonstrated no loss of the longitudinal arch in any of the involved feet when compared with the normal foot.

No patients required custom-made shoe wear. All were able to ambulate without difficulty on level surfaces and stairs. One patient required an orthotic device for metatarsalgia, and one patient required bracing with prolonged ambulation. No patient required shoe wear modifications.

## DISCUSSION

In an attempt to understand the role of the peroneal tendons in the creation of the classic hindfoot valgus and midfoot pes planus deformity, the authors chose to examine patients with no functioning posterior tibialis or peroneus brevis muscles. The model for this were those patients who had an existing common peroneal nerve palsy and also had no posterior tibialis tendon function as a result of anterior transfer of the tendon. In these patients with an absent inverting tibialis posterior and absent peroneal musculature function, the classic deformity of a posterior tibialis deficient valgus hindfoot was not present.

Posterior tibialis tendon transfer has been performed by orthopaedic surgeons for more than 60 years.[7,14,22] Mayer reported,[14] in 1937, that Putti had

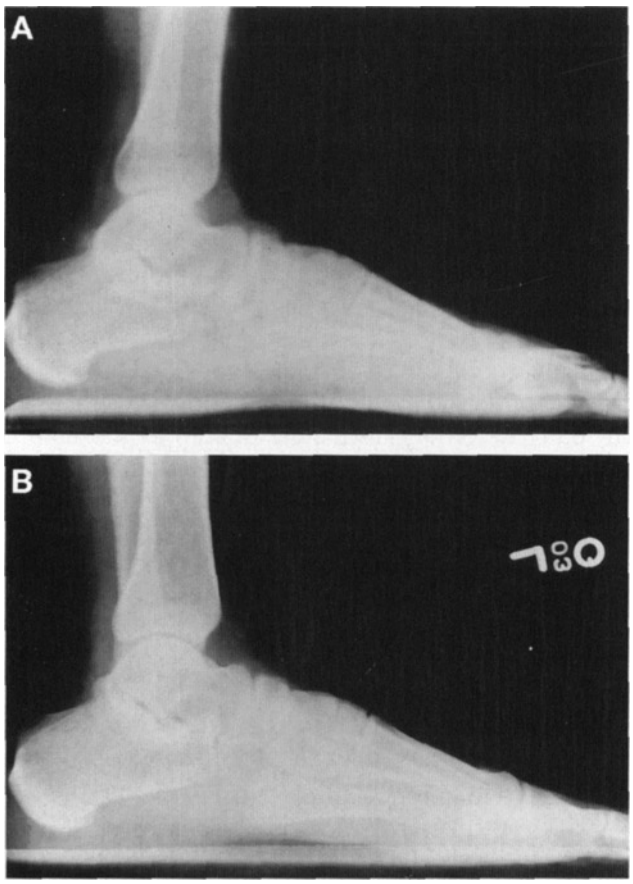


**Fig. 2.** Feet of a 34-year-old man who sustained a knee dislocation and common peroneal palsy. The patient is 114 months status post anterior transfer of the posterior tibial tendon. *A*, Weightbearing lateral radiograph of the affected right foot. *B*, Weightbearing lateral radiograph of the unaffected left foot.

transplanted the tibialis posterior through the interosseous membrane. However, there have been no reports of postoperative mid- and hindfoot collapse in patients who had previously sustained an injury that left the peroneus brevis muscle incompetent. These patients have become, as a result of surgery, posterior tibial-deficient without the sequelae that would be expected with the relatively more common attritional rupture of the posterior tibial tendon.

The observation that a patient with an attritional posterior tibial tendon rupture develops hindfoot valgus and midfoot collapse[5,10,13,17] and that the patient with a common peroneal nerve palsy and a posterior tibial tendon deficient foot resulting from an anterior transfer of the posterior tibial tendon does not is an important observation. The difference between patients in these two categories is intact peroneal function. This deformity most likely results from unopposed action of the peroneus brevis. Mann and Thompson[13] believed that unopposed peroneus brevis action led to hindfoot valgus and midfoot collapse because the unopposed tendon gradually resulted in elongation of the plantar ligamentous structures supporting the arch, resulting in plantarflexion of the head of the talus, pain in the plantar medial foot, midfoot collapse, and a valgus hindfoot deformity. The group of patients evaluated in this study with no peroneus brevis function did not develop the deformity, which confirms their hypothesis. In essence, there is a dynamic balance of medial and lateral musculotendinous units in a "normal" foot. In essence, there is a dynamic

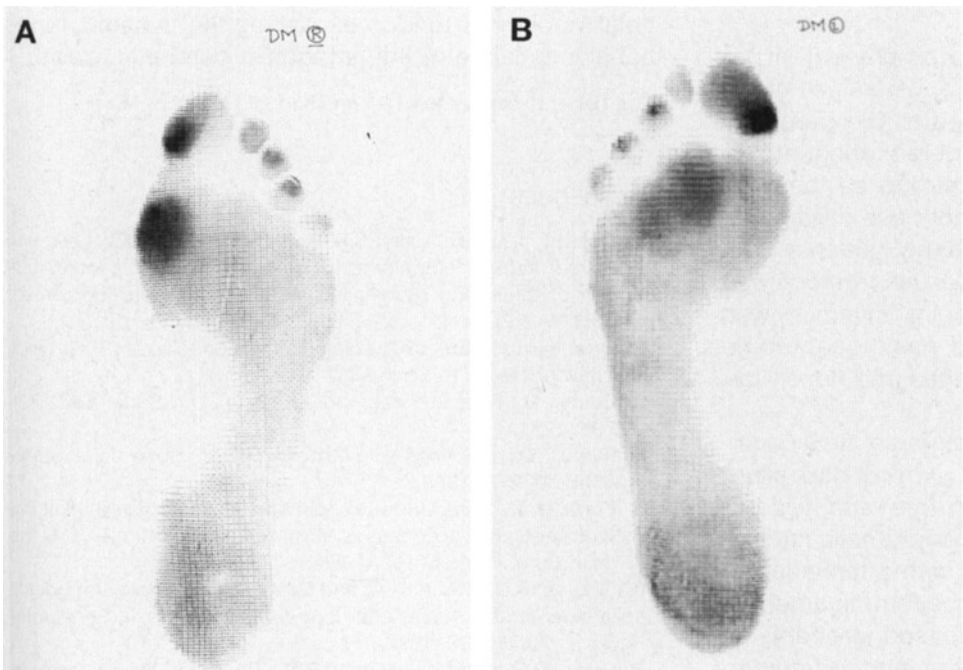


**Fig. 3.** Feet of a 34-year-old man who sustained a knee dislocation and common peroneal palsy. The patient is 114 months status post anterior transfer of the posterior tibial tendon. *A*, Harris mat footprint of the affected right foot. *B*, Harris mat footprint of the unaffected left foot.

balance of medial and lateral musculotendinous units in a "normal" foot. In these patients, with the loss of the peroneus brevis function (as a result of the peroneal nerve palsy) and the loss of the posterior tibialis function (as a result of the tendon transfer), there is a new acquired balance.

There is a structural static nature of this medial and lateral balance.[3,4] With deformities such as clubfoot, there is an inadequate medial column relative to the length of the lateral column. A principle of treatment of talipes equinovarus should be to restore the medial column.[3] It was thought that a long lateral column was associated with a varus deformity, and that a short lateral column was associated with a hindfoot valgus deformity.[4]

Importance of medial and lateral dynamic balance is seen when one examines the progressive loss of peroneal function with Charcot-Marie-Tooth disease, where cavus deformity and secondary forefoot equinus result from weakness of the tibialis anterior and peroneus brevis tendons.[12] A concomitant deformity will occur with the unopposed action of the peroneus longus, which will result in a forefoot valgus deformity, with the hindfoot varus deformity occurring as a result of unopposed action of the posterior tibialis as well as the intrinsic musculature.

A question appropriate for this study is whether the length of patient follow-up is adequate. The time from the recognition of a posterior tibial tendon deficient foot until collapse into a planovalgus deformity was noted to be 4 months.[21] It seems that the minimum follow-up of this study of 18 months is adequate to have observed if these patients are to develop a deformity.

Albeit weak, eversion strength was present in patients who had the Bridle procedure, where half of the posterior tibial tendon is tenodesed to the peroneus longus. This strength was considered adequate to resist gravity but, clearly, not as strong as the contralateral side.These patients did not have collapse of the medial longitudinal arch, and no patients with transfer of the tibialis posterior to the forefoot had eversion of the foot. A small amount of inversion was found in all of the affected feet and was thought to be caused by the flexor digitorum longus and flexor hallucis tendons.

Widening of the plantar first-metatarsal first-cuneiform joint was seen in one foot, which had slight abduction. This patient was pain-free and without need for bracing. As an isolated asymptomatic finding, this was thought to be consistent with attenuation of the plantar first-metatarsal first-cuneiform ligament.[16]

Ankle range of motion was decreased, probably as a result of the tenodesis effect of the tendon transfer. The decreased range of subtalar joint motion in three patients may have been caused by the diminished ankle motion, as noted by Mann,[11] or possibly as a result of previously unrecognized trauma or subsequent postoperative immobilization.

Replacement of the posterior tibial tendon has mainly been accomplished in a functional repair that has concentrated on the use of the flexor digitorum longus tendon (FDL). The mean anatomical cross-section of the FDL is approximately one-third of the tibialis posterior and is somewhat less than that of the peroneus brevis.[20] Because the FDL lacks the strength of the tibialis posterior, coupled with the fact that early deformity frequently occurs at the time of diagnosis of the tendon rupture, it seems that attention should be paid to increasing the strength of the medial replacement.

Weakening or totally releasing the peroneus brevis could also be considered as a possible benefit to this group of patients, before the elongation of the plantar structures. The complete lack of peroneus brevis function in the cohort of patients studied does not seem to have significantly negatively impacted their ability to ambulate. Release of the dynamic evertor may be a reasonable and more effective approach to this common, potentially debilitating cause of hindfoot and midfoot deformity in adults if it is performed early enough, before the development of ligamentous attenuation and structural collapse. The authors believe that further laboratory or clinical studies could be performed to better evaluate these possibilities. The future treatment of the posterior tibial tendon-deficient foot might address both the loss of function of the tibialis posterior tendon as well as the dynamic functional imbalance of the antagonist peroneus brevis.

This paper is dedicated to the memory of Harold H. Mizel.

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Orthopaedic Surgery, University Hospital, S-22185 Lund, Sweden, Tel: +46-46-171-500, Fax: +46-46-130-732.

**June 8–11, 1996**

**56th Annual Meeting of Scientific Sessions of the American Diabetes Association**. The deadline for abstracts was January 5, 1996. Original data may be accepted for exhibition in abstract, poster, and/or oral presentations. Abstract applications and meeting information may be obtained by contacting: Jill Thompson, American Diabetes Association, 1600 Duke St., Alexandria, VA 22314, Tel: 1-800-232-3472, ext. 212, Fax 703-683-1839.

**June 13–15, 1996**

**The Foot and Ankle in Elite Athletic Competition**, Atlanta, GA. Chairmen: Pierce Scranton, M.D., and Donald Baxter, M.D. For more information, contact the American Orthopaedic Foot and Ankle Society, 1-800-235-4855.

**June 26–29, 1996**

**Summer Meeting, American Foot and Ankle Society**, Hilton Head, SC.

**July 5–6, 1996**

**21st Annual Meeting of the Japanese Society for Surgery of the Foot**, Tokyo, Japan. For more information, contact Haruyasu Yamamoto, M.D., Department of Orthopaedics, Tokyo Medical and Dental University, 1-5-45, Yushima, Bunkyo-ku, Tokyo 113, Japan, Tel: 3-5803-5272, Fax: 3-5803-0142.

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**April 24–27, 1997**

**Third Congress of the European Federation of National Associations of Orthopaedics and Traumatology (EFORT)**, Barcelona, Spain. Organizer: Prof. A. Navarro, Hospital Vail d'Hebron, Orthopaedics Surgery and Traumatology Department. Organizing Secretary: Grupo Geyseco, Muntaner 77, Barcelona 08011, Spain, Tel: 34 3 453 92 89, Fax: 34 3 453 24 94.

## ERRATA

In the Roger A. Mann Award article "Juvenile Hallux Valgus: Etiology and Treatment" by M. J. Coughlin (**16:**682-697, 1995, November issue), the artist has misrepresented the hallux valgus angle in Figure 1. The hallux valgus angle is the acute angle formed by the longitudinal axes of the first metatarsal and of the proximal phalanx. The artist is representing the hallux valgus angle as the solid obtuse angle of 140, as measured.

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In the Author Index of December 1995 issue (**16:**819-824, 1995), the second entry under D. R. Bohay's name is incorrect. It should read:

**Bohay, D. R.**
Manoli, A., II: Foot Fellow's Review: Subtalar Joint Dislocations, 16:803

Sanders, Marcus, and Swanson did not contribute to this article.

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In the article "Foot Function in Diabetic Patients after Partial Amputation" by Garbalosa et al. (**17:**43-48, 1996, January issue), co-author James H. Campbell's academic degrees and honors were listed incorrectly. The correct listing is: MBAPO.HDip.PO.

We deeply regret these errors and any inconvenience they may have caused.