

NONOPERATIVE TREATMENT OF POSTERIOR TIBIAL TENDONITIS

A PROSPECTIVE STUDY : USING THE ARIZONA AFO™ PATENTS: 6,155,997 AND 6,443,919

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Abstract:

Surgical treatment for Posterior Tibial Tendon Dysfunction (PTTD) has been well described in the literature. In contrast, few articles exist concerning the nonoperative management of PTTD. We hypothesize that Stages I and II of PTTD may be effectively treated nonoperatively using the Arizona Ankle Brace. In a prospective longitudinal analysis, patients with PTTD were treated with a lace-up molded leather and polypropylene AFO (Arizona). PTTD was classified based on the system of Johnson.¹⁸

All patients were evaluated pre-and post-treatment with the AFO using three clinical scoring systems: 1) the AOFAS hindfoot scoring system, 2) the Foot Function Index (FFI), and 3) the SF-36 questionnaire. Twenty-one patients (eighteen women and three men) with PTTD (six patients had bilateral involvement) were treated with the Arizona AFO. Six patients had Stage I disease (7 ankles), 12 had Stage II (15 ankles), and 5 had Stage III PTTD (5 ankles). The mean follow-up was 12.0 months (range, 3-19 months), and the average age was 57.3 years (range, 34-81 years). After brace treatment, AOFAS hindfoot scores increased from 37.7 to 76.0 ($p < 0.001$). Pre and Post treatment FFI results were: activity 58.6 vs 85.2 ($p < 0.05$), pain 34.0 vs 70.7 ($p < 0.0005$), and function 28.3 vs 67.7 ($p < 0.0005$). SF-36 data revealed a significant improvement in all measured categories ($p < 0.05$) except for a perception of change in health. Seventeen patients were still using the orthosis at the last follow-up evaluation. Two patients (40%) with Stage 3 PTTD discontinued brace use due to lack of pain relief, one patient with Stage II disease stopped use because of resolution of symptomatology, and one with Stage I disease discontinued use prophylactically because of newly discovered, localized vascular disease in the limb.

The efficacy of nonoperative treatment using the UCBL orthosis for PTTD has been reported as 67%;⁴ in contrast, our prospective study showed an improvement of 18/20 (90%). Patients with PTTD can be successfully treated nonsurgically with the Arizona to ameliorate the symptoms of the disorder and possibly obviate the need for any surgical intervention. Surgery may be reserved for those who fail to obtain relief from the brace. Significant pain relief and increases in daily function were demonstrated in patients with Stage I and II PTTD using the Arizona Brace.

Posterior tibial tendon dysfunction (PTTD) is a well-defined clinical entity that involves insufficiency, attenuation and/or rupture of the posterior tibial tendon. Although the disease has been recognized for decades, the nonoperative approach was generally believed to be futile.^{10,27} Therefore, historically the literature has focused on the surgical treatment rather than the nonoperative approach.^{2,6,9,11,14-19,21,22,24,27,31,33-37,40,42} As a result, to date there are very few studies concerning the conservative, nonsurgical approach to PTTD.^{8,15,26,30,41}

The exact underlying cause of PTTD is unknown; however, there are well-defined factors that predispose to developing this disease. These have been characterized as extrinsic and intrinsic factors. Extrinsic factors that have been shown to have an association with PTT dysfunction and rupture include injection of steroids into the tendon or the sheath. Jahss had 3 out of 10 patients in his study who suffered a spontaneous rupture of PTT after receiving local steroid injections.¹⁵

Holmes and Mann have also reported that either local injection or oral intake of steroids increases the risk of PTT rupture.¹³ Finally, traumatic injuries such as closed ankle fractures have also rarely been associated with PTT rupture.²⁸

Despite these data, however, intrinsic factors are thought to play a more dominant role in PTT dysfunction rather than extrinsic factors. Several differing opinions exist regarding the most important of these. Frey et al showed a relative avascular zone just distal to the medial malleolus in cadaver dissections that is comparable to the exact area of PTT degeneration and rupture in vivo.⁷ Myerson et al noted that in a series of 76 patients, 47 had a seronegative inflammatory disorder such as Reiter's syndrome, ankylosing spondylitis, or psoriasis.³¹ Most likely, a combination of these intrinsic factors predisposes to the development of PTT dysfunction rather than either one alone.

Initially, most patients in the literature had been previously undiagnosed with PTT dysfunction. Mann and Thompson noted 15 out of 17 patients were undiagnosed at the time of presentation.²⁷ The average time to diagnosis was 43 months (1 to 12 years). This interval should decrease as awareness of this disease entity increases among physicians. Most patients describe a vague aching about the medial aspect of the ankle, and the typical patient is a female over the age of 40.^{9,17,27,29} During the history-taking session, questions should be directed toward symptoms of a seronegative arthropathy since, as mentioned earlier, more than half of patients with this disease have either psoriasis, Reiter's syndrome, or ankylosing spondylitis.

The purpose of this prospective, longitudinal study is to evaluate the effectiveness of the Arizona AFO brace in the non-operative treatment of Posterior Tibial Tendon Dysfunction based upon three clinical outcome scoring systems: the AOFAS hindfoot score, the Foot Function Index, and the SF-36.^{3,20,39}

MATERIALS AND METHODS

Twenty-one patients (eighteen women and three men) with PTTD (6 patients had bilateral disease) were evaluated over a 2-year period using a prospective design. After careful examination and weight-bearing radiographs, a diagnosis of PTTD was given. The stage of PTTD was assigned based upon the system by Johnson.¹⁸ This data was used to later evaluate if the severity of disease predicted clinical improvement with the use of the brace. Patients were informed about the current recommendations for PTTD, including the operative vs nonsurgical approaches and all patients attempted at least a trial of bracing prior to considering any surgery. After full informed consent, patients were asked to fill out two questionnaires.

The first questionnaire is the Foot Function Index (FFI)³, which evaluates by analog visual scale the patient's own perception of function, pain, and disability prior to wearing the brace. Within each of the three categories, there are several questions pertaining to the particular topic; these are then averaged and a percentage score is obtained for function, pain, and disability.

The second questionnaire is the SF-36 questionnaire.³⁹ Based on multiple-choice responses, this evaluates the patient's overall perception of his/her health and function as well as pain level.

Finally, all patients were examined by a physician prior to dispensation of the brace and scored according to the American Orthopaedic Foot and Ankle Society (AOFAS) hindfoot score system.²⁰ A maximum score is 100 and points are deducted for use of assistive devices, pain, difficulty walking distances or over uneven terrain, and finally for decreasing motion in the hindfoot and midfoot joints.

Prior to brace use and on subsequent follow-up, clinical evaluation of patients was done using the three clinical rating systems: the American Orthopaedic Foot and Ankle Society (AOFAS) Hindfoot Score, the Foot Function Index (FFI) and the SF-36. The clinical outcome of the patient was determined at the point just prior to beginning brace use and then again after a minimum of 3

months of use. Hours per day of brace use and the use of non-steroidal anti-inflammatory drugs (NSAIDs) before and after the use of the brace was documented as well.

Table 1

Patient	Age	Sex	Stage	Hrs/day use	Follow-up(mo)	Comments:
1	73	F	3	6	15	
2	71	F	2	8	16	stopped use: relief of pain
3	81	F	3	12	5	stopped use: no relief of pain; required surgery
4	50	F	1	8	16	
5	73	F	2	10	17	
6	45	F	1	8	8	
7	52	M	2	10	14	
8	55	F	1	3	6	stopped use; vascular disease
9	45	F	3	12	3	
10	42	M	1	12	17	
11	53	M	2	14	14	bilateral disease
12	60	F	2	12	16	
13	51	F	2	14	19	bilateral disease
14	38	F	3	3	7	stopped use: unable to reach foot to fasten brace

15	77	F	1	10	13	bilateral disease
16	67	F	2	2	11	
17	55	F	2(R),2(L)			bilateral disease
18	45	F	3(R),2(L)	12	6	bilateral disease
19	34	F	1(R),2(L)			bilateral disease
20	48	F	2	14	8	bilateral disease
21	68	F	2	7	3	bilateral disease

AOFAS hindfoot scores were statistically examined using the Wilcoxon Signed Rank Test on a Macintosh PowerPC computer using ANOVA statistical software (Abacus Concepts, Berkeley, California). The FFI scores were examined using the same computer and software but the Paired Means Comparison were used for these data. And finally, the SF-36 scores were examined by an outside source using Wilcoxon Signed Rank statistics.

RESULTS

Twenty one patients (eighteen women and three men) were diagnosed with PTTD by history and the presence of the classic triad of valgus hindfoot, abducted forefoot with “too many toes” sign, and the inability to do a painless single heel rise (see Table 1). Radiographs were taken to ensure that there was no other concomitant pathology and to evaluate the ankle and subtalar joints for arthritis. Advanced radiographic studies such as MRI were not routinely obtained in these patients since the diagnosis of PTTD is based upon clinical criteria.

Following diagnosis, disease classification was according to the criteria of Johnson (see Fig 1). In our study, 6 patients had Stage I disease (7 ankles), 12 had Stage II disease (15 ankles), and 5 had Stage III PTT dysfunction (5 ankles). Follow-up averaged 12.0 months (range 3-19 months). Brace use averaged 10.0 hours/day (range 0-14) on follow-up. The average patient age was 57.3 years (range 34-81 years).

On follow-up, of the twenty-one patients, four patients (patients #2, #3, #8, and #14) stopped use of the brace for various reasons. Patient #2 had resolution of her pain and stopped brace use completely after 12 months; she did not require any NSAIDs after that point. Furthermore, she has had no further difficulties other than mild fatigue at the end of the day.

Patient #3 had continuing pain even in the brace and was dissatisfied with her results. She has subsequently undergone surgery for her PTTD by a physician in another part of the country. The patient was unavailable for follow-up questioning as to the type of surgery, the result after surgery, or residual symptoms.

Patient #8 had severe peripheral vascular disease that had declared itself after fitting for the brace; she began to develop pain in the calf, which was found to be vascular claudication. Her vascular

surgeon advised her that because she was not a candidate for a peripheral arterial bypass, she should discontinue the Arizona. She discontinued use of the brace before any follow-up data could be obtained; for this reason she was excluded from the study.

Finally, patient #14 discontinued use of her brace because, in her own words, she was unable to reach it to fasten the brace once it was on her foot. The patient was very obese and had difficulty in reaching her feet. Although she did subjectively have pain relief with the orthosis, she elected to discontinue use on her own. She was excluded from the study as well.

AOFAS hindfoot scores increased from 37.7 before use of the brace to 76.0 after brace use ($p < 0.001$). FFI scores were significant in all categories and were as follows: activity 58.6 vs 85.2 ($p < 0.05$), pain 34.0 vs 70.7 ($p < 0.0005$), and function 28.3 vs 67.7 ($p < 0.005$). All patients reported at least a moderate improvement in pain and function except for the two who had Stage II disease.

SF-36 questionnaires analyze patient perception in 9 areas: Physical Function, Social Function, Physical Role Function, Emotional Role Function, Mental Health, Fatigue/Energy, Pain, Health Perception, and Change in Health Perception. Of the nine measured categories, there was statistical significance to all measured areas ($p < 0.05$) except for Change in Health Perception ($p = 0.2168$).

Data on pain medication use was available for 15 patients. Eight patients completely stopped using pain medication after bracing (two had been using narcotics), and 3 have continued using NSAIDs, although now at lower doses since using the brace. Four patients had not used any medication at any time before, during, or after brace treatment.

DISCUSSION

PTTD is best understood by studying the normal anatomy and function of the PTT and then applying these concepts to the clinical disease process.^{23,25} The PTT courses behind the medial malleolus, posterior to the axis of the ankle, and medial to the axis of the subtalar joint to insert into the plantar and medial aspect of the navicular, the plantar aspect of the three cuneiforms, and the bases of the second, third, and fourth metatarsals. Because of its location and strength, the PTT is a powerful plantarflexor as well as an inverter of the foot.⁵

In normal gait, the PTT is crucial for inversion of the hindfoot so that the transverse tarsal joints can become divergent and "locked."^{1,23,25} This serves to create a rigid lever arm for efficient plantarflexion of the metatarsal heads by the gastrocnemius-soleus complex.

In patients with PTTD, the PTT becomes incompetent and the gastrocnemius complex begins to act on the talonavicular joint in plantarflexion instead of the metatarsal heads. Additionally, the peroneus brevis acts unopposed, causing eversion and abduction of the hindfoot, further exacerbating the deformity. Because of these changes, the static medial constraints of the longitudinal arch become attenuated with time.²⁷ Stretching of the deltoid ligament, talonavicular capsule, and spring ligament in this manner results in medial subluxation/plantarflexion of the talus, valgus alignment of the calcaneus, and increased abduction of the forefoot. Furthermore, as the heel assumes more of a valgus alignment, the Achilles tendon moves laterally and begins to act as an everter of the hindfoot. In this shortened position, an Achilles contracture develops. Over time, chronic changes occur; the initially flexible deformity becomes fixed, and secondary arthrosis of the subtalar and tibiotalar joints can develop.

Little has been written concerning the non-operative treatment of PTTD.^{4,8,15,26,30,41} Williams in 1963 described a "chronic nonspecific tendovaginitis of the tibialis posterior."⁴¹ that he treated with various conservative measures such as restriction of activities, arch supports, foot baths, plaster of Paris, calipers with T-strap, and injection of steroids. Although most patients improved, 12 (23.1%) failed to improve despite several months of conservative treatment. In his study, Williams failed to

give details of the protocol used and an analysis of patients using an outcome or functional scoring system was not available.

In 1982 Jahss reported a series of 10 relatively sedentary patients more than 52 years of age with the diagnosis of spontaneous rupture of the PTT. The first five patients were treated conservatively because Jahss' consensus was that the surgical technique available at that time was less than optimal. His regimen consisted of orthopaedic oxfords with long medial counters, low rubber scaphoids, medial heel wedge, and nonsteroidal anti-inflammatory drugs (NSAIDs).¹⁵ No relief was obtained, with the patients' status remaining static or becoming slightly worse. As a result, although Jahss believed all patients initially should be treated with a conservative course (length unspecified), he thought it was advisable to explore and repair all suspected tears of PTT, except in older patients who are asymptomatic and relatively sedentary.

Frey and Shereff recommended a conservative treatment of rest, ice, NSAIDs, and a medial-posted orthotic to decrease the extent of pronation during the weightbearing phase of ambulation or gait for patients with an acute PTT dysfunction.⁸ For the young athlete with severe tenosynovitis, recommendations include immobilization in a non-weight-bearing, short-leg cast with the foot in inversion for several weeks.

In regard to chronic tenosynovitis, Frey and Shereff recommended a protocol of rest, NSAIDs, and shoe modification, including medial sole and heel wedge with longitudinal arch support.⁸ The treatment concept is to relieve the PT tendon's stress by limiting the extent of excursion.

Mann believed the goal of conservative management of patients with ruptured PTT or symptomatic flatfoot is to establish some form of support for the medial longitudinal arch and valgus deformity of the calcaneus.²⁶ He recommended the use of an orthosis with a small correction and progressive build-up as the patient becomes more tolerant of the pressure beneath the longitudinal arch. Use of a University of California Berkley Laboratory (UCBL) orthosis was proposed for its ability to decrease eversion of the calcaneus while establishing some support beneath the arch.

Chao et al demonstrated that by using a UCBL brace, a 67% clinical improvement could be obtained in patients with PTTD. Patients with Stage III disease were treated with a MAFO. Because of the nonuniformity of treatments for Stage III as compared to the others, a meaningful comparison becomes difficult in this study between different disease stages.⁴

Recently, Myerson described his protocol for the treatment of patients with PTT dysfunction.³⁰ For the patients who present with acute tenosynovitis of PTT, an initial protocol consisting of rest, administration of appropriate NSAIDs, and immobilization was recommended. Corticosteroid use was not recommended because of the association of tendon attenuation and rupture with local injections.¹⁵ Myerson recommended immobilization with a rigid below-the-knee cast or removable boot for a period of 6 to 8 weeks. The patient is allowed to ambulate with the cast or removable boot during this period of immobilization.

After an adequate trial a decision is reached concerning further treatment. For patients who demonstrate significant improvement with immobilization, a molded heel and sole shoe wedge, hinged ankle foot orthosis (AFO), or orthotic arch support is used to invert the hindfoot. If these treatments result in only mild or moderate improvement, an additional period of immobilization (casting or use of removable boot) is recommended. Otherwise, a surgical procedure is contemplated. For patients with advanced disease, a rigid AFO is recommended to immobilize the involved and affected articulations.

In our study, we followed the treatment algorithm outlined by Myerson; however we used the Arizona ankle brace in place of all other orthotics, AFOs, or shoe inserts/modifications in the protocol. We

classified the disease according to the criteria set forth by Myerson, and we used NSAIDs in all patients to control the inflammatory phase of the disease process. Furthermore, the inclusion of patients with all stages of the disease helps to evaluate treatment efficacy based on disease severity.

The Arizona AFO is designed to hold the hindfoot in a more neutral position and out of the valgus position that it acquires in this disease process. It accomplishes this by three-point fixation much similar to that of a well-molded cast. Because it relies on passively correcting the deformity and thus resting the Posterior Tibial Tendon, those patients with Stage III disease can be expected to achieve less of a favorable response to treatment.

The Arizona AFO brace showed an overall statistically significant improvement in 18 out of 20 patients (90%). This is a marked improvement over prior studies using medial posted orthotics such as the UCBL or an AFO. In those studies, as mentioned earlier, the results have been 67% to 77%.⁴

All patients with Stage I or II disease showed pain relief referable to the brace and demonstrated an improvement in all 3 clinical measurement instruments used in this study (the AOFAS, FFI and SF-36).

Three out of 5 (60%) of patients with Stage III disease had relief of symptoms referable to the brace. Although the data is too small to draw a meaningful conclusion, the results are similar to other results in the literature for the nonoperative treatment of this stage of PTTD. Because the Arizona is more comfortable than a rigid AFO, it can be a useful adjunct in treating those patients who can not tolerate a plastic AFO.

Nonoperative treatment of PTTD can be successful with the Arizona AFO brace, and treatment success can be increased by treating the disease in its early stages. This, in turn requires a high index of suspicion on the part of the Orthopaedist and accurate diagnosis. A well-fitted custom lace-up molded leather and polypropylene orthosis can alleviate the pain and loss of function associated with PTTD and safely obviates the need for surgery in the majority of patients with a one year follow-up.

SF-36 data have been used in the past to evaluate patient perception of treatment outcome, perception of health, social function, emotional function and overall satisfaction. In today's climate of patient satisfaction-directed health care, a nonoperative treatment that yields pain relief and a perception of increased function may prove more valuable than similar pain relief obtained from surgery.

Future studies directed toward following these conservatively treated patients and evaluating their disease process by clinical exams and radiographs is essential to determine if disease progression and arthrosis occur despite symptomatic relief with the brace. Furthermore, an age- and disease stage-matched control group that undergoes surgery will be necessary to definitively evaluate if Arizona brace treatment is at all superior to surgery. Clearly, further research is needed on this challenging problem. At this time, the Arizona can be used as a very important and effective adjunct to the initial conservative treatment of patients with this difficult disease.

BIBLIOGRAPHY

1. *Ambagtsheer JBT: The function of muscle of lower leg in relation to movement of the tarsus: An experimental study in human subjects. Acta Orthop Scand Suppl 172:1-196, 1978.*
2. *Anderson AF, Fowler SB: Anterior calcaneal osteotomy for symptomatic juvenile pes planus. Foot and Ankle 4:274-283, 1984*
3. *Budiman-Mak et al: Foot Function Index. Clinical Epidemiology 44:561-570, 1981*
4. *Chao W, Lee TH, Hecht PJ, et al: Conservative management of posterior tibia tendon rupture.*

Orthopaedic Transactions 18:1030, 1994-1995

5. Duchenne GB: In Kaplan EB (ed): *Physiology of Motion*. Philadelphia, WB Saunders, 1959
6. Evans D: Calcaneo-valgus deformity. *J Bone Joint Br* 57:270-278, 1975
7. Frey C, Shereff M, Greenidge N: Vascularity of the posterior tibial tendon. *J Bone Joint Surg Am* 72:884-888, 1990
8. Frey CC, Shereff MJ: Tendon injuries about the ankle in athletes. *Clin Sports Med* 7:103-118, 1988
9. Funk DA, Cass JR, Johnson KA: Acquired adult flat foot secondary to posterior tibial tendon pathology. *J Bone Joint Surg Am* 68:95-102, 1986
10. Goldner JL, Keats PK, Bassett FH, III et al: Progressive talipes equinovagum due to trauma or degeneration of the posterior tibial tendon and medial plantar ligaments. *Orthop Clin North Am* 5:39-51, 1974
11. Graves SC, Mann RA, Graves KO: Triple arthrodesis in older adults: Results after long term follow-up. *J Bone Joint Surg Am* 75:355-362, 1993
12. Hinterman B, Gachter A: The first metatarsal rise sign: A simple sensitive sign of tibialis posterior tendon dysfunction. *Foot Ankle Int* 17:236-241, 1996
13. Holmes GB Jr, Mann RA: Possible epidemiological factors associated with rupture of posterior tibial tendon. *Foot and Ankle* 13:70-79, 1992
14. Horton GA, Olney BW: Triple arthrodesis with lateral column lengthening for treatment of severe planovalgus deformity. *Foot Ankle Int* 16:395-400, 1995
15. Jahss MH: Spontaneous rupture of the tibialis posterior tendon: Clinical findings, tenographic studies, and a new technique of repair. *Foot and Ankle* 3:158-166, 1982
16. Johnson KA: Tibialis posterior tendon release: Substitution. In Johnson KA (ed): *Master Techniques in Orthopaedic Surgery*. New York, Raven Press, 1994, pp 271-284
17. Johnson KA: Tibialis posterior tendon rupture. *Clin Orthop* 177:140-147, 1983
18. Johnson KA, Strom DE: Tibialis posterior tendon dysfunction. *Clin Orthop* 239:196-206, 1989
19. Johnson WL, Lester EL: Transposition of the posterior tibial tendon. *Clin Orthop* 245:223-227, 1989
20. Kitaoka HB, Alexander IJ, et al: Clinical Rating Systems for the Ankle-Hindfoot, Midfoot, Hallux, and lesser toes. *Foot and Ankle Int* 15(7):349-353, 1994
21. Koutsogiannis E: Treatment of mobile flat foot by displacement osteotomy of the calcaneus. *J Bone Joint Surg Br* 53:96-100, 1971
22. Lahr DD, Shereff MJ: Reconstruction for posterior tibia tendon insufficiency. Read at the Annual Summer Meeting of American Orthopaedic Foot Ankle Society. Vail, Colorado, July 1995
23. Mann RA: Biomechanical approach to the treatment of foot problems. *Foot and Ankle* 2:205-212, 1982
24. Mann RA: Acquired flatfoot in adults. *Clin Orthop* 181:46-51, 1983
25. Mann RA: Biomechanics of the foot. *Inst Course Lect* 31:167-180, 1982
26. Mann RA: Rupture of the tibialis posterior tendon. *Inst Course Lect* 33:302-309, 1984
27. Mann RA, Thompson FM: Rupture of the posterior tibial tendon causing flat foot: Surgical treatment. *J Bone Joint Surg Am* 67:556-561, 1985
28. Monto RR, Moorman CT III, Mallon WJ, et al: Rupture of posterior tibial tendon associated with closed ankle fracture. *Foot and Ankle* 11:400-403, 1991
29. Myerson M: Posterior tibia tendon insufficiency. In Myerson M (ed): *Current Therapy in Foot and Ankle Surgery*. St. Louis, Mosby-year Book, 1993, pp 123-135.
30. Myerson MS: Adult acquired flatfoot deformity: Treatment of dysfunction of the posterior tibial tendon. *J Bone Joint Surg Am* 78:780-792, 1996
31. Myerson M, Solomon G, Shereff M: Posterior tibia tendon dysfunction: Its association with seronegative inflammatory disease. *Foot and Ankle* 9:219-225, 1989.
32. Myerson MS, Corrigan J, Thompson F, et al: Tendon transfer combined with calcaneal osteotomy for the treatment of posterior tendon insufficiency: A radiological investigation. *Foot Ankle Int* 16:712-718, 1995.
33. Phillips GE: A review of elongation of os calcis for flat foot. *J Bone Joint Surg Br* 65:15-18, 1983.

34. Sammarco GJ: *Technique of triple arthrodesis in treatment of symptomatic pes planus.* *Orthopedics* 11:1607-1610, 1988.
 35. Sangeorzan BJ, Mosca V, Hansen ST Jr: *Effect of calcaneal lengthening on relationships among the hindfoot, midfoot, and forefoot.* *Foot and Ankle* 14:136-141, 1993
 36. Sangeorzan BJ, Smith D, Veith R, et al: *Triple arthrodesis using internal fixation in the treatment of adult foot disorders.* *Clin Orthop* 294:299-307, 1993.
 37. Saxby T, Myerson M: *Calcaneus osteotomy.* In Myerson M (ed): *Current Therapy in Foot and Ankle Surgery.* St. Louis Mosby-Year Book, 1993, pp 159-162
 38. Shereff MJ: *Clinical evaluation of the foot and ankle.* In Sammarco GJ (ed): *Foot and Ankle Manual.* Philadelphia, Lea & Febiger, 1993, pp 42-53
 39. Ware JE et al: *The MOS 36-Item Short-Form Health Survey (SF-36): Med. Care* 30 473-481, 1992
 40. Welton EA, Rose GK: *Posterior tibial tendon pathology: The foot at risk and its treatment by os calcis osteotomy.* *Foot* 3/4:168-174, 1993
 41. Williams R: *Chronic non-specific tendovaginitis of the tibialis posterior.* *J Bone Joint Surg Br* 45:542-545, 1963
 42. Vogler HW: *Triple arthrodesis as a salvage for end stage flatfoot.* *Clin Podiatr Med Surg* 6:591-604, 1989
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