Evaluation and Retraining of the Intrinsic Foot Muscles for Pain Syndromes Related to Abnormal Control of Pronation

Bahram Jam, Mphty (Manip), BScPT, FCAMT

Abstract: *Little clinical research exists on the contribution of the intrinsic foot muscles (IFM) to gait or on the specific clinical evaluation or retraining of these muscles. The purpose of this clinical paper is to review the potential functions of the IFM and their role in maintaining and dynamically controlling the medial longitudinal arch. Clinically applicable methods of evaluation and retraining of these muscles for the effective management of various foot and ankle pain syndromes are discussed.*

Key Words: *intrinsic foot muscles, medial longitudinal arch, pronation, exercises*

Introduction:

The medial longitudinal arch (MLA) has been described as a critical structure of the foot that contributes to shock absorption and the attenuation of forces transmitted to the body during gait (Donatelli 1996). Many structures may contribute to varying degrees to support the MLA including the plantar fascia (Fuller 2000), ligaments such as the plantar calcaneo-navicular ligament (Borton & Saxby 1997), extrinsic foot muscles such as the tibialis posterior muscle (Soballe et al 1988) and the intrinsic foot muscles (IFM) (Fiolkowski et al 2003). Although there are many papers describing the role of the plantar fascia, ligaments and the extrinsic muscles of the foot in supporting the MLA, little clinical research exists on the contribution of the IFM in the maintenance and the dynamic control of the MLA. Moreover, there is little written on the topic of specific clinical evaluation and retraining of the IFM. The purpose of this clinical paper is to review the potential functions of this group of muscles and how dysfunction of the IFM may potentially be a contributing factor to various foot and ankle conditions. Clinically applicable methods of evaluation and retraining of the IFM will also be reviewed. This is to provide clinicians with a potentially effective management option for various pain syndromes related to abnormal control of pronation and reduced dynamic control of the MLA.

The IFM may be divided and grouped into four layers. The first layer consists of the abductor hallucis (ABH), flexor digitorum brevis (FDB), and the abductor digiti minimi (ABDM). The second layer includes the quadratus plantae (QP) and the lumbricals (LUM). The third layer includes the adductor hallucis transverse (ADHT), adductor hallucis oblique (ADHO), flexor hallucis brevis (FHB) and flexor digiti minimi brevis (FDMB). The forth layer includes the interossei (INT) muscles (Kura et al 1997). The IFM are diagrammatically represented in Figure 1. Of all the IFM, the abductor hallucis and the adductor hallucis have the greatest physiological cross-sectional area (Kura et al 1997), which supports the hypothesis that these are the most dominant IFM.

Several clinically common overuse injuries and syndromes have been linked to pes planus and excessive pronation, including plantar fasciitis, Achilles tendonitis, hallux valgus, tibialis posterior and tibialis anterior overuse syndromes (Hintermann & Nigg 1998, Van Boerum & Sangeorzan 2003, Kaufman et al 1999). It is the authors' hypothesis that the primary biomechanical cause of some foot and ankle syndromes is not 'excessive pronation', but rather a 'lack of pronation control'. The IFM may have a functional role for stabilizing the foot during single-limb balance (Travell & Simons 1992). They are also active during gait and function similar to elastic springs by supporting the MLA and maintaining the concavity of the foot (Moore 1985). It is therefore suggested that effective neuromuscular control of the IFM is essential in order to stabilize the tarsal and metatarsal bones and modulate the rate of pronation. This 'fine tune' control is not only required for static control of the MLA, but is likely essential for the dynamic control of the MLA from the heel-strike to the toe-off phase of the gait cycle. Considering the complex movements occurring at the inter-tarsal, tarso-metatarsal and the metatarso-phalageal joints during the gait cycle, the IFM must be able to consistently activate the right amount and at the exact right time during the stance phase of gait in order to be able to attenuate the forces placed on the articular and soft tissues throughout the lower extremity.
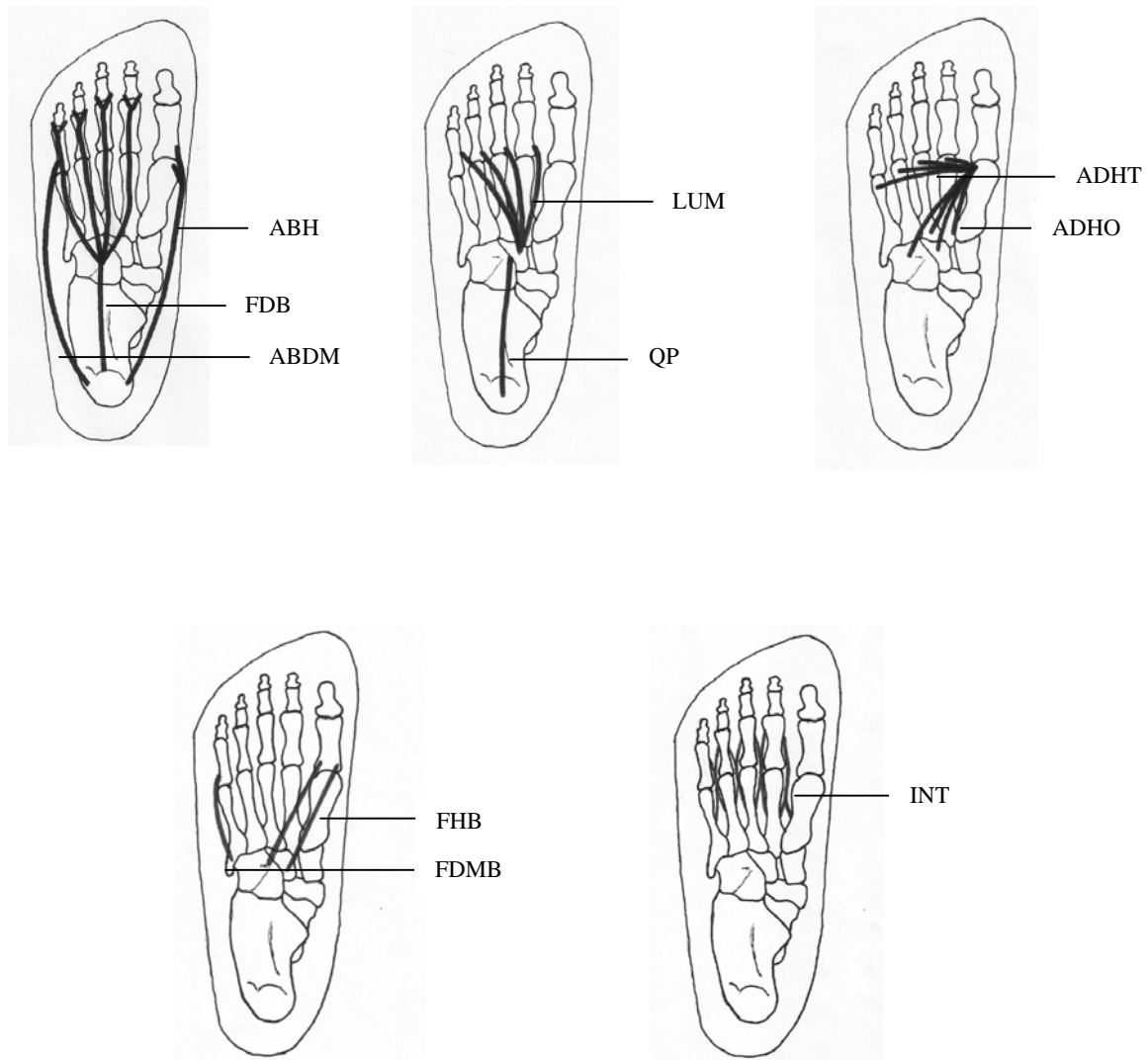
Figure 1. A diagrammatic representation of the intrinsic muscles of the foot

Panjabi (1992) brought forward a revolutionary concept for spinal stability and proposed that the spinal stabilizing system consists of three sub-systems, the passive, active and neural control. It has been proposed that if the passive system is impaired for any reason (e.g. post macro or micro-trauma), maximizing the contribution of the active and the neural control systems may enhance stability and reduce related pain (Panjabi 1992, Hodges 1999, Richardson et al 1999). Although this clinical concept was originally proposed for the spine, it may be transferable to any joint in the body including the foot and ankle complex.

There have been a number of studies that have demonstrated a compromised neural control, reduced endurance and often the atrophy of specific tonic stabilizing muscles following trauma, pain and/or inflammation. The insufficiency and neural control inhibition of these muscles have been associated with pain and dysfunction at the lumbar spine (Hides et al 1996), thoracic spine (Lee 2003) cervical spine (Jull et al 1999), shoulder (Magarey & Jones 2002), hip (Sims et al 2002) and at the knee (Stevens et al 2003). It has also been demonstrated that if the stabilizing segmental muscles are inhibited following pain or trauma, their recovery is not automatic and that specific motor control retraining may be necessary for functional recovery of these muscles (Hides et al 1996). Unfortunately, the compromised strength, tonic control and retraining of the IFM related to foot and ankle pain have not been described or established in any published papers to date.

Myofascial Pain Related to the Intrinsic Foot Muscles:

It has also been proposed that insufficient strength and endurance of the IFM may lead to overloading these muscles following sudden increases in walking, running and jumping activities which may result in the development of related myofascial trigger points (Travell & Simons 1992). Interestingly, many of the signs and symptoms of plantar fasciitis are also characteristic of specific IFM myofascial pain syndromes, which means that in some cases, the IFM may be the primary source of heel and foot pain (Travell & Simons 1992). It is possible that the effectiveness of supportive shoes or orthotics may sometimes be simply attributed to the fact that they help support the MLA, and therefore help partially unload overused IFM, hence reducing the myofascial pain. The effectiveness of massage, local pressure and passive stretching of the "plantar fascia" may be simply due to inhibition of the over-active IFM.

Since it is hypothesized that the IFM are more functionally active in individuals with pes planus (Grey et al 1968), it may help explain why these individuals are at greater risk of developing myofascial pain syndromes of the IFM. The risks of IFM overuse increases in individuals with pes planus especially if they participate in activities that involve prolong walking, running and jumping in non-supportive footwear.

Management of patients with IFM myofascial pain includes, rest by reducing the load on the muscles by temporarily avoiding prolong walking, running and jumping. They must also be educated on avoiding the use of tight fitting shoes especially at the forefoot. This is to prevent restriction of toe movement; shoe constriction may overload the IFM and activate the associated trigger points (Travel & Simons 1992). Another effective management includes the prescription of supportive shoes or the temporary use of orthotics to help unload the over-loaded IFM. Some patients with myofascial pain from the superficial or the deep IFM may have tried orthotics, but have usually quickly removed them because of intolerably greater pain from the increased pressure on the trigger points of the muscles (Travel & Simons 1992). In this situation, loser-fitting shoes with semi-rigid orthotics may be more effective and tolerable as they can help control pronation, yet not rigidly limit it. However a long-term solution to myofascial pain due to overused IFM may be to retrain and increase their endurance in order to allow them to tolerate the stresses placed on them during walking, running and jumping.

Evaluating pes planus and pronation using the Navicular Drop Test:

Although pes planus and hyper-pronation have been linked to many overuse pain syndromes, objective measurement and identification of this condition can be challenging. One relatively simple method that has been proposed is the navicular drop (ND) test (Brody 1982). This test is considered to be a composite measure of excessive pronation (Menz 1998) since it has been theorized that the amount of subtalar joint motion can be indirectly detected by measuring the amount of motion at the navicular bone (Picciano et al 1993). The ND test is performed by calculating the difference between the height of the navicular from the floor when the subtalar joint is positioned in neutral and the height of the navicular from the floor when in relaxed stance in a full weight bearing position (Brody 1982).

Figure 2: Subtalar joint neutral passive positioning

Firstly, the navicular tubercle is marked with a felt tip pen. Then in the seated position, the foot is passively placed into subtalar joint neutral (Fig. 2). A simpler option of approximating and achieving subtalar joint neutral is by performing active extension of the toes without raising the meta-tarsal heads off the floor (Fig. 3). The toes are then slowly dropped back down without "losing" the MLA. The height of the navicular tubercle from the floor is then measured with a standard ruler (Fig. 4) and this number is referred to as the navicular height in neutral (NHN).

Figure 3: Active extension of the toes

The second measurement is taken in relaxed and natural standing. Once again, the height of the navicular tubercle from the floor is measured and this number is referred to as the navicular height in standing (NHS). The navicular drop (ND) is calculated by subtracting NHN from NHS. (NHN-NHS = ND)

Figure 4: The Navicular Drop Test

The ND can be calculated for both feet and the presence of an asymmetry between the symptomatic and asymptomatic foot should be noted. If the ND

value is greater than 15mm (Brody 1982) on the injured / symptomatic side, it may be presumed that the IFM need to be addressed for the rehabilitation and that the patient may benefit from appropriate foot wear or orthotics to help control the excessive ND on the symptomatic lower extremity.

There are four challenges to the ND test with respect to its clinical application as an evaluation test. The first challenge is that the ND test has been shown to yield poor to moderate intratester reliability and poor intertester reliability when performed by inexperienced testers (Picciano et al 1993). They therefore suggest that clinicians should practice this measurement technique and take into account their potential measurement error.

The second challenge with the ND test is the lack of agreement in the literature for the normal and abnormal amount of navicular drop that is to occur during this test. In the original paper by Brody et al (1982), based on clinical experience, an ND of 10mm was believed to be normal and an ND of 15mm or greater was regarded as abnormal. Another study on individuals with and without lower extremity injuries concluded that an ND of 6-9mm was considered to be within the normal range and that an ND of greater than 10mm was considered abnormal (Loudon et al 1996).

The third challenge with the ND test is that it only measures the amount of navicular fall in static standing. This measurement is not necessarily associated with the control of navicular motion during gait or functional activities. It is perhaps for this reason many individuals with an "abnormal" ND test can be asymptomatic.

As previously mentioned, there are many active and passive structures that to varying degrees contribute to the MLA, therefore the fourth challenge is that the ND test can not discriminate the deficient structure(s) responsible for the drop of the MLA. It can be hypothesized that one of the causes of excessive ND may be insufficient tonic control of the IFM.

Even with all the challenges stated above, the ND test may still be clinically valuable as its primary goal is to simply help select individuals who may benefit from IFM retraining, taping and/or supportive footwear. The ND test is not intended for use as an accurate outcome measure to monitor progress, since a change in the ND is not anticipated following any conservative intervention.

The ND test may perhaps be made more clinically applicable if simple visual estimation is used instead of the ruler method. If plain visual observation and palpation demonstrate a significant navicular drop (estimating greater than 1-1.5cm), then the test may be considered positive and further evaluation of the IFM may be warranted.

IFM Functional Role:

It has been suggested that the basic primary function of the IFM is to permit flexibility for shock absorption and balance, and to provide rigidity and stability for propulsion (Travell & Simons 1992). A recent study (Fiolkowski et al 2003) performed the ND test on asymptomatic individuals before and after a local anaesthetic block of the tibial nerve at the level of the medial malleolus. The purpose of the tibial nerve block was to de-activate many of the IFM including the abductor hallucis muscle. Interestingly, a small (3mm) but significant ($P<0.05$) increase in ND was demonstrated following the tibial nerve block. The results of this study support the hypothesis that the IFM have a small yet significant role in maintaining the MLA.

It is important to appreciate that the results of this study may be deceptive as the relatively small 3mm drop was a mean value; most likely some individuals demonstrated greater than the 3mm drop while others demonstrated less than the 3mm drop in the navicular height. The contribution and the need for IFM activity may be unique for all individuals depending on the relative support from the other structures responsible for maintaining the MLA. Compared to an individual with pes cavus, an individual with pes planus may require relatively more support from the IFM during static standing and during gait. This hypothesis is to some extent supported by another study that used electromyography (EMG) and cinematography. They showed that the abductor hallucis and the flexor digitorum brevis muscles were generally very active and contributed significantly to support the MLA in "flatfooted" persons, however in individuals with "normal" feet, the activity and the contribution of the IFM was significantly less (Gray et al 1968). They also proposed that compared to those with "normal" feet, in individuals with "flat feet" the IFM may be recruited significantly more during the gait cycle to compensate for lax ligaments and general foot hypermobility. A different EMG study, (Duranti et al 1985) analyzed the activity of the abductor hallucis in individuals with "flat feet" and they also demonstrated significant abductor hallucis activity on the weight bearing foot during single leg standing.

IFM Evaluation:

Step #1: Stand in front of a wall, with the feet shoulder width apart and knees slightly flexed. The fingertips may be lightly placed on the wall.

Step #2: In order to achieve subtalar joint neutral, gently supinate the feet by lifting all the toes off the floor (Fig. 3), then slowly drop the toes down again but maintain the MLA. This most often results in a rise of the MLA and the navicular bone, due to the windlass effect via the plantar fascia. If this procedure proves difficult, then simply increase the height of the MLA, by actively attempting to approximate the head of the first metatarsal towards the heel, without flexing the toes (Fig. 5).

Figure 5a: IFM activation with incorrect patterning

Figure 5b: IFM activation with correct patterning

Step #3: While maintaining the MLA, stand on a single leg, as steady as possible. The fingertips should remain lightly on the wall for balance and fall prevention (Fig. 6).

Step #4: Count to 30 seconds and observe for the steadiness of the navicular height and for any compensatory extrinsic foot muscle activity.

Step #5: Repeat the process on the other lower extremity.

The therapist may evaluate over-activity of the extrinsic foot muscles by occasionally attempting to passively

Figure 6: IFM evaluation & retraining position

lift the toes off the floor. This is to ensure minimal resistance from the extrinsic/long toe flexors.

The IFM control may be subjectively classified as satisfactory, fair or poor. The IFM are evaluated as 'satisfactory' if steadiness of the neutral navicular height without over-activity of the extrinsic foot muscles is consistently observed during the entire length of the 30-second test. The IFM are evaluated as 'fair' if unsteadiness of the neutral navicular height and/or over-activity of the extrinsic foot muscles are inconsistently observed during the 30-second test. Finally, the IFM are evaluated as 'poor', if the patient is unable to maintain the neutral navicular height at all and/or over-activity of the extrinsic foot muscles are consistently observed during most of the 30-second test. The results of this test may be considered an outcome measure and should be documented separately for each foot; since with specific retraining, improvement in the control and endurance of the IFM is anticipated.

Clearly the IFM evaluation is somewhat similar to a single leg standing balance test. It is therefore important to be aware that there are several factors other than the IFM that may influence the outcome of this test including altered joint proprioception, various neurological conditions and vestibular dysfunctions. Basically, this IFM test evaluates the ability to maintain neutral navicular height without inappropriate compensatory patterns, during supported single leg standing.

Inappropriate Compensatory Patterns:

A common exercise prescribed by physical therapists for retraining the IFM is the "towel toe curls" (Fig. 7). Toe curl exercises primarily require the use of the flexor digitorum longus, flexor hallucis longus, tibialis anterior, and extensor digitorum longus muscles.

Figure 7: Towel toe curl exercise

Strengthening these extrinsic muscles by prescribing towel toe curl exercises may be of potential benefit for individuals presenting with general foot and ankle weakness, such as those post fractures or surgery. However, performing phasic towel toe curl exercises is unlikely to improve the tonic control of the IFM. This exercise may be comparable to performing phasic "sit ups" to improve lumbar stability and dynamic control.

One of the primary principles behind IFM retraining involves focusing on their tonic activation in isolation

of the extrinsic foot muscles. This principal in muscular control retraining has been adopted from studies related to the cervical and the lumbar spine. Segmental muscular control retraining in isolation of the superficial muscles has been repeatedly demonstrated to be clinically effective for the management of specific spinal conditions (Jull et al 2002, O'Sullivan 1997). It is hypothesized that the IFM may be comparable to the function of the segmental stabilizers of the spine and therefore their retraining in isolation of the extrinsic / superficial muscles may be clinically effective if applied to appropriate foot and ankle conditions.

The abductor hallucis attaches distally to the medial side or often to the plantar aspect of the proximal phalanx of the great toe (Clemente 1985). The abductor hallucis more often functions as a flexor than an abductor of the big toe (Basmajian et al 1985). Isolated activation of the abductor hallucis should ideally produce abduction and/or flexion of the proximal 1st phalanx. Therefore flexion of the distal 1st phalanx during the retraining exercises would indicate an incorrect compensatory activation of the extrinsic flexor hallucis longus muscle.

The flexor digitorum brevis attaches distally to both sides of the middle phalanx of the lesser toes (Clemente 1985). Therefore flexion of the distal phalanx of the toes during the retraining exercises would indicate an incorrect compensatory activation of the extrinsic flexor digitorum longus muscle.

The lumbricals in the foot flex the proximal phalanges (Clemente 1985). Therefore flexion of the distal phalanges of the lesser toes during the retraining exercises would indicate an incorrect compensatory activation of the extrinsic flexor digitorum longus muscle.

In summary, if flexion of the distal phalanges is performed (i.e. curling of the toes) during the IFM retraining exercises, it indicates inappropriate and excessive contraction of the extrinsic muscles such as the flexor digitorum longus and flexor hallucis longus (Fig. 5a). The patient must therefore be instructed to avoid this compensatory activation of these extrinsic muscles when performing the IFM exercises (Fig. 5b).

IFM Retraining:

In the author's clinical experience, IFM control can positively change from poor to fair and from fair to good in most individuals within a 2-6 week period. The rate of improvement is potentially dependent on many factors including the severity of the foot deformity, pain intensity and the patients' comprehension and compliance to the recommended exercise program. In order to maximize compliance to any therapeutic exercise program, the exercise should ideally be easy to understand, easy to perform in most settings and require no special equipment. The following suggested exercise fits these criteria.

Step #1: Stand in front of a wall, with the feet shoulder width apart and knees slightly flexed. The fingertips may be lightly placed on the wall.

Step #2: In order to increase the height of the MLA, gently supinate and actively attempt to approximate the head of the first metatarsal towards the heel, without flexing the toes (Fig. 5). Often the gluteal muscles may also need to be activated to facilitate femoral and tibial lateral rotation, which may assist in this active supination.

Step #3: While actively maintaining the MLA, stand on a single leg. The knee on the weight-bearing lower extremity should ideally be flexed 10-20° to help the contraction of the quadriceps muscle and the potential facilitation of the IFM. The fingertips should remain lightly on the wall for balance and fall prevention (Fig. 6).

Step #4: Hold the position for a count of 10 seconds and attempt to maintain the MLA as steady as possible during the entire time without any compensatory extrinsic foot muscle activity. Following the 10-seconds, slowly and with eccentric control allow the foot to pronate and the MLA to lower to a relaxed state.

Step #5: Following 1-2 seconds of rest, actively re-supinate and repeat steps 3-4.

Perform up to five repetitions of this concentric, isometric and slow eccentric IFM retraining exercise, several times per day. The exercise may be performed in either single leg standing or if it is too difficult, in double leg standing.

The therapist may evaluate over-activity of the extrinsic foot muscles by observing for excessive toe flexion and by occasionally attempting to passively lift the toes off the floor. This is to ensure minimal resistance from the extrinsic/long toe flexors.

The IFM retraining exercise may be progressed by gradually lifting the fingers off the wall and performing them in unsupported single leg standing with eyes open and eventually with eyes shut.

The patient should ideally be instructed to perform active supination throughout the day in order to facilitate the IFM during activities of daily living (e.g. when standing in a line up or when washing dishes). Therapeutic taping may also temporarily provide proprioceptive feedback for maintaining the MLA, which may also assist the patient in remembering to comply with the exercise program (Jam & Varamini 2004, Hadley et al 1999).

Conclusion:

Pronation is a natural and essential component of gait. It is the authors' opinion that the long-term management of some individuals with excessive pronation may not be to limit pronation (e.g. by using rigid orthotics), but to restore motor control of the IFM during pronation. Many individuals with pes planus or reduced MLA remain asymptomatic although they participate in many running and jumping activities. This may be related to the fact that these individuals with pes planus have adequate IFM control and endurance that help compensate for their insufficient passive structures. If however both the passive and the active systems are insufficient, as may be the case for an individual with pes planus and underlying poor IFM control, the development of foot and ankle pain syndromes may be inevitable.

It is important to appreciate that the proposed IFM evaluation is a non-specific clinical test and that there are no studies to support its validity or reliability. Evaluation of the IFM could be made more objective with the use of surface or needle EMG, however this is not feasible or practical in an outpatient clinical setting.

Many clinically common foot and ankle syndromes such as plantar fasciitis, Achilles tendonitis, shin splints and tibialis posterior tendonitis have been generally attributed to excessive pronation producing repeated traction forces on specific soft-tissue structures resulting in micro-tears, inflammation and eventual pain. In summary, the IFM may play a significant role in modulating pronation and minimizing repeated micro-trauma to related soft-tissues. However, more scientific studies are required to support or negate this clinical hypothesis. It is proposed that rehabilitation of many lower extremity pain syndromes should include the evaluation and when required, the appropriate retraining of these traditionally ignored, yet functionally essential muscles.

Although this paper has focused on muscular control localized to the foot complex, in clinical practice, a thorough evaluation of the entire lower quadrant including the pelvis and the hip is essential in order to identify other biomechanical and muscular patterning dysfunctions potentially contributing to abnormal control of pronation.

Appendix: Summary of the traditional versus a new proposed concept regarding the IFM

Traditional concept	New proposed concept
Excessive pronation is the primary biomechanical cause of many foot and ankle disorders	Lack of "control" into pronation is the primary biomechanical cause of some foot and ankle disorders
Rigid foot orthotics are clinically essential and should be the primary management option for many foot and ankle pain syndromes related to abnormal pronation	Prior to prescribing orthotics, retraining of the intrinsic foot muscles may also be clinically valuable for the management for some foot and ankle pain syndromes related to abnormal pronation
Rigid orthotics are required to maintain the subtalar neutral position and to stop / limit pronation in many foot and ankle pain syndromes related to abnormal pronation	Semi-rigid orthotics may also be effective for maintaining the subtalar neutral position and controlling pronation in some foot and ankle pain syndromes related to abnormal pronation
The purpose of retraining of the intrinsic foot muscles is to change the static foot posture and the height of the medial longitudinal arch	The purpose of retraining of the intrinsic foot muscles is neither to change the static foot posture nor the height of the medial longitudinal arch, but to help dynamically control the medial longitudinal arch following heel strike
Intrinsic foot muscles can be retrained using phasic towel toe curl exercises	Intrinsic foot muscles may be effectively retrained using specific, tonic exercises in isolation of the superficial muscles

References:

1. Basmajian RM, Deluca CJ: Muscles Alive, Ed.5. Williams & Wilkins, Baltimore, 1985 (pp.353-4)

2. Borton DC, Saxby TS. 1997 Tear of the plantar calcaneonavicular (spring) ligament causing flatfoot. A case report. J Bone Joint Surg Br. Jul;79(4):641-3

3. Brody DM. 1982 Techniques in the evaluation and treatment of the injured runner. Orthop Clin North Am. Jul;13(3):541-58

4. Clemente CD: Gray's Anatomy of the Human Body, American Ed. 30. Lea & Febiger, Philadelphia, 1985 (pp. 587-590)

5. Donatelli R. The Biomechanics of the Foot and Ankle, FA Davis, Philadelphia 1996

6. Duranti R, Galletti R, Pantaleo T. 1985 Electromyographic observation in patients with foot syndromes. Am J Phys Med 64:295-304

7. Fiolkowski P, Brunt D, Bishop M, Woo R, Horodyski M. 2003 Intrinsic pedal musculature support of the medial longitudinal arch: an electromyography study. J Foot & Ankle Surg 42(6): 327-333

8. Hadley A, Griffiths S, Griffiths L, Vicenzino B. 1999 Antipronation taping and temporary orthoses. Effects on tibial rotation position after exercise. J Am Podiatr Med Assoc. Mar;89(3):118-23

9. Fuller EA. 2000 The windlass mechanism of the foot. A mechanical model to explain pathology. J Am Podiatr Med Assoc. Jan;90(1):35-46

10. Gray EG, Basmajian JV. 1968 Electromyography and cinematography of leg and foot ("normal" and flat) during walking. Anat Rec 161: 1-16

11. Hides J, Richardson C, Jull G 1996 Multifidus muscle recovery is not automatic following resolution of acute first episode low back pain. Spine 21:2763-2769

12. Hintermann B, Nigg BM. 1998 Pronation in runners. Implications for injuries. Sports Med. Sep;26(3):169-76

13. Hodges P. 1999 Is there a role for transversus abdominis in lumbo-pelvic stability? *Manual Therapy* 4(2):74-86

14. Jam B, Varamini A. Therapeutic Taping for Peripheral and Spinal Syndromes. APTEI. Thornhill, ON   2004 www.aptei.com

15. Jull G, Trott P, Potter H, Zito G, Niere K, Shirley D, Emberson J, Marschner I, Richardson C. 2002 A randomized controlled trial of exercise and manipulative therapy for cervicogenic headache. Spine. Sep 1;27(17):1835-43

16. Jull G, Barrett C, Magee R, Ho P. 1999Further clinical clarification of the muscle dysfunction in cervical headache. Cephalalgia. Apr;19(3):179-85

17. Kaufman KR, Brodine SK, Shaffer RA, Johnson CW, Cullison TR. 1999 The effect of foot structure and range of motion on musculoskeletal overuse injuries. Am J Sports Med. Sep-Oct;27(5):585-93

18. Kura H, Luo Z, Kitaoka An K. 1997 Quantitative analysis of the intrinsic muscles of the foot. The anatomical Record 249:143-151

19. Lee D. The Thorax: An Integrated Approach. 2003 2nd Edn Diane G. Lee Physiotherapist Corporation. White Rock, BC www.dianelee.ca

20. Loudon JK, Jenkins W, Loudon KL. 1996 The relationship between static posture and ACL injury in female athletes. J Orthop Sports Phys Ther. Aug;24(2):91-7

21. Magarey ME, Jones MA. Dynamic evaluation and early management of altered motor control around the shoulder complex. Man Ther. 2003 Nov;8(4):195-206

22. Mann R, Inman VT. 1964 Phasic activity of intrinsic muscles of the foot. J Bone Joint Surg (46):469-481

23. Menz HB. 1998 Alternative techniques for the clinical assessment of foot pronation. J Am Podiatr Med Assoc. Mar;88(3):119-29

24. Moore KL. Clinically Oriented Anatomy. Willians & Wilkins 1985 p. 500

25. Nack JD, Phillips RD. 1990 Shock absorption. Clin Podiatr Med Surg. Apr;7(2):391-7

26. O'Sullivan et al 1997 Evaluation of specific stabilizing exercise in the treatment of low back pain with radiologic diagnosis of spondylolysis or spondylolisthesis. *Spine* (22): 2959-2967

27. Panjabi 1992a The stabilizing system of the spine. Part 1.Function, dysfunction, adaptation and enhancement. Journal of Spinal Disorders (5):383-389

28. Panjabi 1992b The stabilizing system of the spine. Part II. Neutral zone and stability hypothesis. Journal of Spinal Disorders (5):390-397

29. Picciano AM, Rowlands MS, Worrell T. 1993 Reliability of open and closed kinetic chain subtalar joint neutral positions and navicular drop test. J Orthop Sports Phys Ther. Oct;18(4):553-8

30. Richardson C, Jull G, Hodges P, Hides J. Therapeutic Exercise for Spinal Segmental Stabilization in Low Back Pain, Scientific Basis and Clinical Approach. Churchill Livingstone 1999

31. Sims KJ, Richardson CA, Brauer SG. Investigation of hip abductor activation in subjects with clinical unilateral hip osteoarthritis. Ann Rheum Dis. 2002 Aug;61(8):687-92

32. Soballe K, Kjaersgaard-Andersen P. 1988 Ruptured tibialis posterior tendon in a closed ankle fracture. Clin Orthop. Jun;(231):140-3

33. Stevens JE, Mizner RL, Snyder-Mackler L. Quadriceps strength and volitional activation before and after total knee arthroplasty for osteoarthritis. J Orthop Res. 2003 Sep;21(5):775-9

34. Travell JG, Simons DG. Myofascial Pain and Dysfunction: The Trigger Point Manual for the Lower Extremities. Wlliams & Wilkins, Baltimore, MD 1992

35. Van Boerum DH, Sangeorzan BJ. 2003 Biomechanics and pathophysiology of flat foot. Foot Ankle Clin. Sep;8(3):419-30