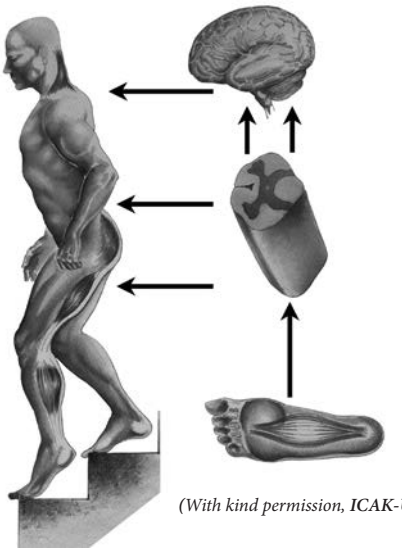

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CHAPTER FOUR

Foot and Ankle

04

The foot is often considered an odd-looking and unglamorous appendage with the sole purpose of bearing one's weight. It usually does not attract a physician's attention unless a patient complains of foot pain. In reality, foot dysfunction is often the cause of remote health problems that respond poorly or not at all to treatment until the dysfunction is corrected.

The American Podiatric Medical Association says that 85 percent of the US population will someday seek medical care for foot pain. **http://www.apma.org/**

The importance of evaluating all patients' feet is emphasized by a case (SC) treated when he first joined in practice with (DSW). "Peggy's" chief complaint was intractable left shoulder pain that had failed to respond to any physician's therapeutic efforts. Cortisone injections, manipulation of the shoulder and neck, immobilization of the shoulder, and various types of muscle relaxants and analgesics had all failed to bring anything but temporary relief. During consultation Peggy informed SC that she had been unable to sleep except for very brief periods for about two months. Examination of the shoulder with applied kinesiology techniques proved difficult. Range of motion was extremely limited, with almost no ability to abduct the shoulder. Direct testing of the shoulder muscles was impossible because of pain and limited motion. My therapeutic attempts consisted of local muscle treatment, various reflexes, acupuncture, spinal and pelvic corrections, cranial manipulations, and several forms of electrotherapy, all to no avail. At one time Peggy did indicate that she was able to get two hours of sleep a night as a result of these treatments. She stated that the only way she could obtain relief was to stand next to a wall and press her shoulder hard against the wall by leaning on it.

After numerous treatments with minimal positive results, Peggy became the type of patient a doctor hates to see come in the office. It got to the point that when I would see her name as the next patient the thought would surface, "Do I

have to go in and see her again?" Just before I went in with her I described to DSW the problems I had been having with Peggy. DSW suggested, "You ought to evaluate her feet." To make matters worse, as I walked into the treatment room it seemed empty with no patient on the examination table. Glancing around the room, I found Peggy leaning against a wall, pressing her shoulder hard against it.

After discussing this case with DSW and having already spent more time evaluating and treating Peggy's shoulder and spine than is usually allotted for a single treatment in our office, I asked Peggy to move her arm into abduction with minimal improvements and some pain. I then asked Peggy to remove her shoes and socks. I found the most gnarled set of feet I have ever seen, and immediately wondered how she could get them into shoes and still walk on them.

The results of examining Peggy's feet were absolutely amazing! After finding subluxations in the feet by challenge and making the corrections, the pain in the shoulder was reduced. After correcting her feet to the maximum her shoulder muscles could be tested without pain, and a large percentage of them tested normal. This enabled me to evaluate and correct the remaining shoulder dysfunction. Unfortunately, as soon as she walked she lost all corrections in her feet, and the shoulder pain returned. Again her feet were corrected and solidly taped for support.

The rest of Peggy's story is uneventful. The shoulder pain was gone after several more treatments, most of which were directed toward the feet. Apparently the reason she was able to obtain relief in her shoulder by leaning against a wall was that the position changed the structural relationship of her feet.

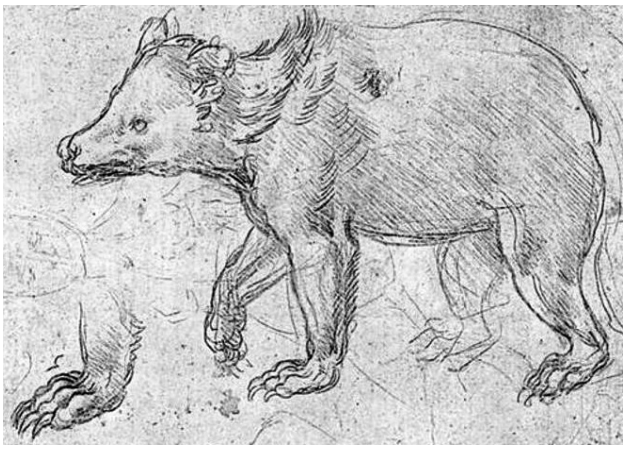
Since that time, a firm rule has been practiced in our office: No new patient is examined with their shoes and socks on. Some in chiropractic recognize the need to examine and correct the feet; **(Brantingham et al., 2009)** more often they are overlooked, even though there have been many efforts to encourage their routine examination. **(Ferrari, 2007; Dananberg, 2007; Keating, 2002; Greenawalt, 1980)** It is important to at least screen the foot as a possible contributing factor to remote health problems.

The type of problem not generally overlooked by the physician is foot symptoms in the form of pain, corns, calluses, or bunions, or an ankle injury. Most of these will be considered here; however, they are not the major problem areas. These conditions are generally looked into because they produce specific complaints for which the patient requests attention. The area of most concern is that of aberrant neurologic ramifications that the symptomatic or asymptomatic foot is capable of producing. In some types of primary foot dysfunction, the patient's complaint is remote from the foot, and the foot is asymptomatic. It is often the secondary conditions that receive attention, possibly for years, without any permanent help. Treatment may be directed to the shoulder, tension headache, low back pain, and on and on — and the basic underlying cause of the problem is not found until the patient possibly develops a symptomatic condition in the foot, or comes in contact with a doctor knowledgeable about the many ramifications of foot dysfunction.

Routine applied kinesiology evaluation of the foot cannot be overemphasized. Yes, the foot is often considered an unglamorous appendage, primarily for holding the body's weight. It seems unglamorous because it is often ugly, distorted with dysfunction; it may have obvious circulatory problems, corns, calluses, in-grown or broken nails. All these are evidence of foot dysfunction. A general rule is that a pretty foot is a healthy, properly functioning foot.

Leonardo da Vinci (1450-1519) regarded anatomic-artistic studies as not only having to do with muscles and the skeleton, but as being part of every other aspect of anatomy and physiology, as well as a polemic with the great anatomists of the past – from Hippocrates to Galen, Avicenna, and Mondino. For instance when Leonardo dissected a bear, he also focused his attention on the anatomy of the leg and foot which he drew.

Comparative anatomy reveals significant alteration from the hominoids to man for the conversion to bipedal stance. **(Lovejoy, 2007, 1988; Burke et al., 1982)** Bipedalism mostly occurred for hominoids because it was essential for species survival. We moved down from the trees because the climate cooled and there were no longer enough trees or sufficient food in the trees that remained to sustain our ancestors there. **(Gould, 2000)** The development of spinal curves and change in pelvic shape allow for a conservation of energy as man's trunk balances over the pelvis and legs. The spinal engine theory **(Gracovetsky, 1989)** proposes that the spine improves its efficacy of motion by using the spine and pelvis to propel the legs forward by capturing the ground reaction force to derotate the spinal segments with each step of the gait cycle. Converting the ground reaction force as potential energy in the viscoelastic tissues of the lower limb and spine into kinetic energy as the spine derotates makes the mechanics of the foot an essential factor in optimal locomotion. **(Dananberg, 2007)** In man, the gluteus maximus has become the largest of the gluteal muscles, while in apes it is much smaller; the gluteus medius in apes is larger than that in man. In apes, the ischium is long, giving the hamstring muscles leverage for great strength. In man the lever is short, which is consistent with the speed required of the hamstrings in walking and running.



Leonardo's anatomical study of the bear

In apes, the foot has grasping capability with divergence of the great toe. Man has lost the ability of the great toe to diverge, with the other four digital rays being

Leonardo — Bear foot anatomical study

aligned toward the great toe. The load lines of the foot in man project from the calcaneus through the 1st and 5th digital rays. This creates a tripod, comprising the heel, hallux, and small toe, with weight bearing primarily at the heel and across the entire width of the distal metatarsals. **(Cavanagh, 1987)** This is important in the bipedal stance of man. Relating to man's weight passing through a coronal plane to the firmly planted tripodal feet, Tobias **(Burke et al., 1982)** states, "His back and front are nearly evenly balanced on either side of this plane. Seen in this light, the upright posture is a precariously balanced state. If our body were merely a nerveless framework, with atonic muscles, it could be thrown off–balance by a push or a gust of wind." He goes on to state, "Long ago, Schopenhauer pondering man's individuality said that our walking is admittedly nothing but a constantly prevented falling." Bipedalism also made us much slower runners than when we were quadrupeds, and many of the big predators can run several times faster than we can. **(Johanson, 1981)**

The foot has many homologues to the hand, yet often when they are compared the foot comes out second–best because of the hand's ability to grip, its dexterity, and its apparent beauty in comparison to the foot. From an evolutionary point of view, several million years spent sitting in trees helped bipedalism emerge because it freed up the prehominoid hand, which allowed the gradual evolution of the opposable thumb. This led to a leap forward in hominoid survivability and versatility in every environment the hominoid lived. (**Napier, 1993)**

When the foot's functional architecture is understood however, its functionality should at least equal that of the hand and perhaps surpass it, especially in human evolution. Bordelon **(Bordelon, 1987)** cites a change in concept indicating the importance of man's foot being different from primates. He cites a *National Geographic* (November 1985) article stating that "…recently discovered fossils have revolutionized our concepts of the human past. Our earliest, most distinguished characteristics were not large brains, language or tool making, but the ability to habitually walk upright." With this ability man's hands were freed to allow for their development. Hand use may have then caused the need for greater brain development. **(Blechschmidt, 2004; Napier, 1993)**

The 26 bones of the foot and its soft tissue comprise a marvel of engineering design. With 200,000 nerve endings, 19 major muscles, 33 joint centers and 17 ligaments, it may be that 6 million years of evolution created the perfect foot. When one considers the stress of walking, running, and jumping that must be accommodated and dissipated by the foot, it's a wonder that anyone has healthy feet. The normal stresses the foot must adapt to are compounded by poor quality or improperly designed and fitted shoes.

The foot has gone through many changes to adapt to human bipedal upright posture. There are three basic ways the human foot differs from that of other primates. (1) In man the 1st ray is not markedly divergent; thus it is normally not useful as a grasping appendage, as is the hand. (2) The human foot has both longitudinal and transverse arches. (3) In certain functions the foot is a rigid structure with strong ligamentous support, yet it can become supple to adapt to the contour of the ground. These factors are true of the normal foot, which is best adapted to weight bearing. Many poorly functioning feet revert back to the simian or prehensile foot, with loss of the arches, hypermobility, and medial deviation and flexibility of the 1st ray.

British trained osteopath Hugh Milne remarks in his excellent book *The Heart of Listening: A Visionary Approach to Craniosacral Work*, that a former agent told him that the C.I.A. teaches its novice agents to watch a suspect's body language, especially their feet. **(Milne, 1995)** Milne notes that since watching the feet and toes of patients under duress, "it is amazing what tales a toe can tell you. The person says one thing, the toe says another. Watch, always watch."

The human foot normally develops its rigidity and lack of flexibility during normal use and development as a weight–bearing appendage. Its marvelous ability to adapt can be observed in those born without arms who use their feet from early childhood to grasp objects. Many have developed the ability to write and paint by holding a pencil or brush between the toes, to eat by similarly holding a spoon or fork between the toes, and even to drive automobiles by using the feet to steer and shift gears. **(BBC News, 2002)**

If the feet are so marvelous, why do so many people have poor foot function? There is a tendency to attribute foot dysfunction to congenital conditions, or failure of foot design to meet the requirements of weight bearing and the general pounding they receive on a daily basis. Stress to the feet has certainly increased with the recent popularity of running and jogging, which is now shifting to walking for exercise. From 1970 to '79 the "bible" of the running crowd, *Runner's World*, increased its circulation from 3,000 to 500,000. **(Clement & Taunton, 1981)** Running USA released a portion of the results from their 2009 *State of the Sport* series. **(Running USA, 2008)** In 2008 there was an

18% increase in the total running population (35,904,000 runners) compared to 2007, and a 15% increase in the estimated number of trail runners in 2008 (4,857,000). The mean age of road race finishers in 2008 was 36.3. If you ran at least 100 days in 2008, you were one of 14.9 million who did likewise. And despite the recession, running shoes (39.9 million pairs) and dollars spent on them ($2.3 billion) exceeded those of the previous year. Many people are unaware of foot dysfunction. It may be manifested as end–of–the–day fatigue. When activity increases, as in jogging, it may present a serious problem. **(Abshire, 2010; Marshall, 1978)**

General Examination and Body Language

One should recognize that much foot dysfunction is locally asymptomatic, or the patient fails to discuss the problem because "I have normal aching in my feet after being on them all day." In addition, women especially recognize that their foot problems result from improper shoes, but with style-conscious thinking they fail to discuss the matter with their physician. It is therefore necessary to be capable of reading the body language of foot dysfunction.

Body language may be recognized in the pattern of symptoms, by visual observation of the foot, by palpation, and/or by specialized tests. Dananberg, Maffetone and Cailliet **(Dananberg, 2007; Maffetone, 2003; Cailliet, 1997)** point out that there are three ways in which the foot functions: (1) in willed, free movements, (2) as a static support mechanism subject to gravitational forces and postural changes, and (3) as a dynamic mechanism for moving the body. Keeping these three functions in mind, we will direct our examination toward each aspect.

Willed, free movements are present when the foot is non-weight-bearing, such as on an examination table. The foot is not generally used as a freely movable appendage, yet analysis of the foot in manual muscle testing is usually done in this manner. It is necessary to consider the other two aspects — a static support mechanism and a dynamic mechanism for moving the body — since many foot dysfunctions are only observable under these circumstances. The physician who examines the foot and other weight-bearing mechanisms only when the patient is non-weight-bearing is destined to overlook much dysfunction.

With the thought in mind of non-weight bearing, static weight bearing, and dynamic weight bearing, body language of foot involvement will be described as (1) symptomatic, which is primarily the patient's complaint recorded during consultation, (2) the foot's appearance as observed by the examiner, and (3) shoe quality and shoe wear. The examination is divided into three sections: (1) non-weight bearing, which includes structural balance of the foot, palpatory findings, and applied kinesiology's shock absorber and sensorimotor challenge tests, (2) static weight bearing, which evaluates postural structural balance and strain (muscle tests will be used to examine for remote dysfunction with various foot positions), and (3) the dynamic examination, which takes walking, running, and treadmill activities into consideration.

Most foot dysfunction is functional, such as that discussed in this chapter. **(Abshire, 2010; McDowall, 2004; Maffetone, 2003)** As with any health problem, however, it is necessary to thoroughly consider all possibilities. Neuropathy, which may be due to diabetes, Hansen's disease, tabes dorsalis, yaws, syringomyelia, vascular occlusion of vasa nervorum, or direct trauma, can cause foot deformities simulating those of dysfunction. **(Staal et al., 1999)** Foot deformity may result in the loss of intrinsic musculature, causing clawing or hammering of the toes, cavus feet, and/or depressed metatarsal heads simulating functional problems. There may also be varus or valgus deformities as a result of extrinsic muscle imbalance. Breakdown of the foot can result from a Charcot joint. **(McCormack & Leith, 1998; Cailliet, 1997; Wagner, 1983)**

Symptomatic

It should be determined during consultation whether the patient's symptoms become worse with standing, running, and other weight-bearing activities, regardless of the chief complaint. Many low back conditions are not optimally treated because the fault is diagnosed primarily within the lumbar spine. It may be hyperlordosis leading to facet syndrome, but the question "Why is the hyperlordosis present?" must be asked and answered. As noted later, if the positive support reaction is not functioning properly, extensor muscles of the pelvis may be giving inadequate support, which allows anterior pelvic rotation and increases the lumbar lordosis. In this case, the primary condition is foot dysfunction even though the feet are asymptomatic. Treatment directed to the facet syndrome may well give relief, but if the basic underlying cause of the condition is not corrected, it will only be temporary. One of the documented reasons for facet syndromes and lumbar hyperlordosis is inhibition of the gluteus maximus muscles. **(Travell & Simons, 1992)** One of the advantages of the applied kinesiology approach to manual muscle testing is that the gluteus maximus muscles can be tested in both the prone, supine, and standing positions – particularly after applied kinesiology challenge procedures have been applied to the foot. Immediate strengthening or weakening of the gluteus maximus muscles after such a challenge provides immediate, non-invasive evidence that foot dysfunction is related to the gluteus maximus muscle inhibition and related low back and pelvic problems.

Additional questions should be asked to further categorize any increase of symptoms with weight bearing. Some patients develop symptoms on static standing, with no problem when walking and moving about; on the other hand, some have no problem from static weight bearing, but they experience significant symptoms when walking or running. Structural strain and disorganization may not develop until the gait mechanism is brought into play.

Knowledge of disturbance from the weight-bearing mechanism can be enhanced by determining if the patient feels better on arising in the morning, and the symptoms develop or increase with weight-bearing activity

AK Foot and Ankle Examination

Body language is observed by the health problem and history, e.g., worse when on feet for a long time, running, eases with sitting. Muscular imbalances observed standing and in supine position. Corns, calluses and fat pads easily observed during static examination. Hammer toes indicate weak intrinsic foot muscles.

Foot Pronation
Talus subluxation
Also:
Tarsal, metatarsal dysfunctions

Ankle support muscle impairments:
- Peroneus tertius
- Peroneus longus, brevis

Anterior and posterior tibial

Tarsal Tunnel Syndrome
Compare for weak flexor hallucis brevis and strong flexor hallucis longus. Correct foot pronation first.

Challenge calcaneus and adjust. Calcaneus will usually be in a posterior position.

Golgi tendon organ or muscle spindle cell treatment is frequently needed, especially for intrinsic muscles of the foot. Raw bone-concentrate and/or Folic Acid-B12 for myofascial trigger points if nutritional support if corrections do not hold.

Weight bearing that causes weakness indicates need for further evaluation and support. Evaluation of knees, pelvis, and other structures above may be needed.

Tape Support
Figure 8 Support
Anchor on leg. Tape extends under scaphoid to return onto leg in figure 8 pattern.
Calcaneus Anchor
First application around & behind calcaneus, followed by a support to scaphoid with 2 or 3 strips of tape.

Frequent cause of recurring cranial, sacral, pelvic, respiratory, shoulder and neck involvements.

Meridian involvement through area
Liver Kidney Stomach
Gallbladder Bladder Spleen

Neck, shoulder girdle, and shoulder dysfunction are frequently caused or perpetuated by foot pronation. These specific interactions dynamically assessed with AK MMT.

Bladder Involvement
Infection or irritation and chronic pelvic pain can cause reflex muscle weakness, perpetuating foot and ankle problems. These problems can cause recurrent or chronic bladder involvement: viscero-somatic interactions.

Circulatory disturbances of any nature can be complicated by foot/ankle dysfunction.

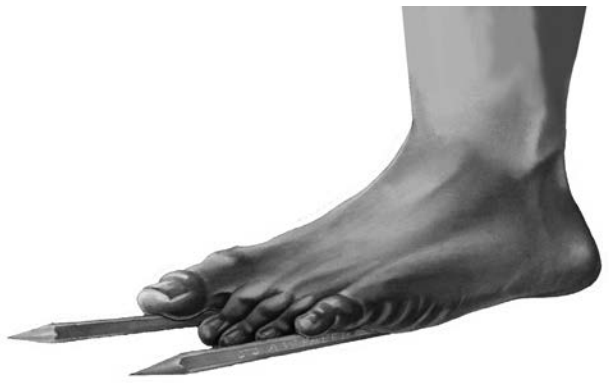
Calcaneal spurs from traction on plantar aponeurosis.

To further evaluate the holding capability of foot and ankle corrections, have the patient walk, jump, run, or repeatedly activate the involved muscles. Re-examine immediately to determine return of any factors which were corrected. If return, there is indication to test for:
- Gait Mechanism
- Microavulsions of muscles involved
- Shoes the patient wears
- Consider orthotic supports

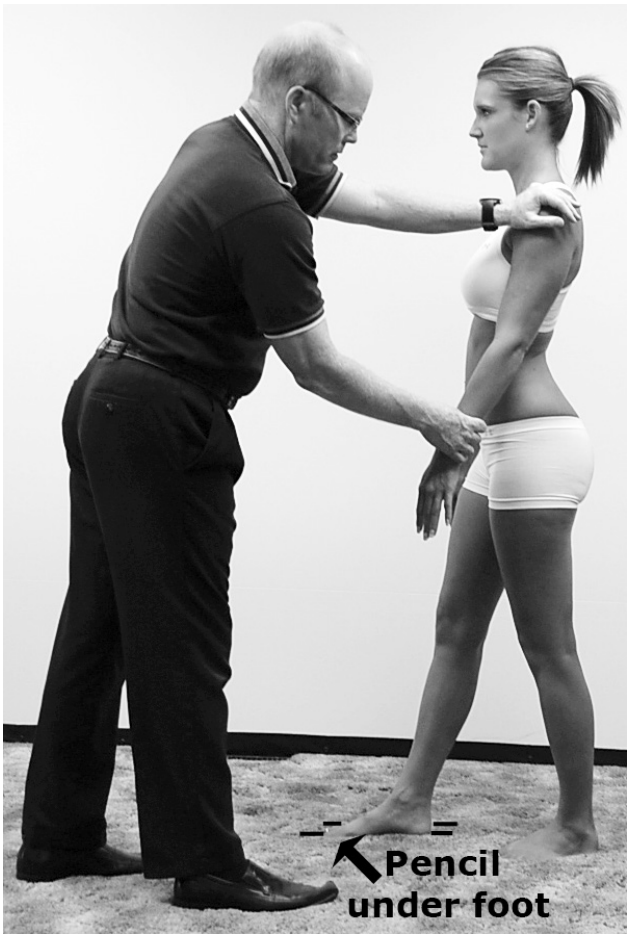
throughout the day. The weight-bearing mechanism is especially indicted if relief is obtained by sitting or lying down at the end of the workday.

Foot dysfunction is the cause of recurrent neurologic disorganization (switching) in many individuals. **(Walther, 1983)** The afferent supply from the foot is instrumental in much organization of body movement and postural regulation. Many studies have been done in other areas of the body showing that improper stimulation to proprioceptors disturbs the proper temporal pattern of muscle contraction, i.e., agonist and antagonist muscles contract at the same time, or there is contraction or inhibition not in keeping with the joint movement taking place at that time. **(Gardner-Morse & Stokes, 1998; Graven-Nielsen et al., 1997; Bard et al., 1992; Munro, 1972)** It is now well established that the feet are very important for proprioception as well as for posture and balance. Applied kinesiology clinical evidence certainly indicates this. Normal facilitation and inhibition of muscles are often demonstrated in applied kinesiology by testing the shoulder flexors and extensors with an individual in various gait positions. **(Walther 2000, 1983, 1981; Goodheart, 1982)** When an individual is in a simulated gait position with the right leg forward and the left trailing, the left shoulder extensors test weak by manual muscle tests although they were strong in a normal stance. This is expected in order for the arm to swing forward easily during that phase of gait. However, if one places pencils under the 1st and 5th metatarsals, causing an artificially produced dropped metatarsal arch, the normal facilitation and inhibition of the shoulder muscles with various gait positions become unpredictable. It is not known how they will test, but in nearly all cases the results are different from the predictable norm.

All patients who exhibit recurrent neurologic disorganization, known as "switching" in applied kinesiology, should be evaluated for foot and gait problems. Improper stimulation to the proprioceptors of the joints, skin, and muscles of the feet and legs can cause a bombardment of improper afferent impulses to the neuronal pools. This is especially true if there is a facilitated state at a spinal segment from a vertebral subluxation. **(Korr, 1976, 1975)** The facilitated spinal neurons are initiated by afferent impulses arising from nociceptors or visceral receptors and



Pencil under 1st & 5th metatarsals



are then transmitted to the dorsal horn of the spinal cord, where they synapse with interconnecting neurons. These stimuli are then transmitted to motor and sympathetic efferents, resulting in changes in the physical tissues, such as skeletal muscle, skin and blood vessels.

Areas of facilitation can be exacerbated by stressors of many types which place increased stress upon many aspects of the individual, primarily the triad of health in applied kinesiology – physical, chemical, and psychological. Korr's concept of facilitation fits directly into the applied kinesiology contextual model for understanding pain and dysfunction. It also provides a model for understanding the influence of a wide gamut of stressors (postural, nutritional, emotional, etc.) on the health of individuals.

General structural strain throughout the body gives indication to evaluate the weight-bearing mechanism. Rolf and Myers **(Myers, 2001; Rolf, 1977)** and many others have pointed out the continuity of myofascia throughout the body, and how distortion in one area can transmit strain to remote body parts through the fascia. When there is structural strain, such as that present when the positive support reaction is not functioning adequately, the most superior aspect of the fascia — at the crown of the head — is tense. Tenderness to digital pressure posterior to the bregma indicates probable fascia tension. Applied kinesiology clinical evidence indicates that the pull of the fascia around the skull can jam sutures. Especially important is the sagittal suture which, when jammed, may cause weak abdominal muscles. **(Walther, 2000, 1983)**

The weak abdominal muscles allow the pelvis to rotate anteriorly, compounding the increased AP spinal curves from poor function of the positive support mechanism. It is possible that the abdominal muscle weakness correlates with a sagittal suture cranial fault because the abdominal ptosis causes a generalized traction on the fascia, which ultimately results in tightening of the superficial and deep fascia of the epicranium. This could provide a vicious circle of cranial etiology that disturbs the function in such a manner that the cranial faults cause additional muscle weakness. Another observation is the important role the abdominal muscles play in pelvic support, **(Hodges & Richardson, 1996)** especially in category I pelvic faults. The category I pelvic fault is especially important in the cranial-sacral primary respiratory mechanism. Correcting the sutural fault will very likely be only temporary unless other structural corrections are made, because the tense fascia will cause the suture to jam again.

Recurrent cranial faults are body language to examine the weight-bearing and gait mechanisms. There are many reasons for this. Ferguson **(Ferguson, 1991)** suggests that the powerful muscles attaching directly to the cranial bones are capable of generating sufficient pressure or forces upon the skull to produce the flexibility and palpable motion at the cranial sutures which may exist for this purpose. Just as it is imprudent to diagnose the status of the pelvic joints without knowledge of the status of the dynamic muscles which attach to the bones of the pelvis (such as the gluteus maximus, hamstrings, piriformis, quadratus lumborum, or psoas), so must evaluation and treatment of cranial dysfunctions demand the evaluation of those muscles which attach to the skull. Page **(Page, 1952)** documents the continuity of the fascia from the abdominal area to the dura mater. The continuity of the fascia is emphasized by Wright and Brady **(Wright & Brady, 1958)** in their statement, "The deep cervical fascia is so continuous with that of the head that the two should be considered as one." In addition, the deep cervical fascia "…forms the pectoral fascia which in turn blends with the fascial covering of the rectus abdominis." Chaitow **(Chaitow, 2005)** observed: "Pick, **(1999)** in his landmark text on cranial sutures, inexplicably fails to mention the profound potential impact of muscular attachments that frequently overlie and traverse sutures." Chaitow counsels that treatment of dysfunctional muscles attaching to the cranial and facial sutures are essential prior to any attempt at treating the osseous structures themselves.

It is clinically obvious that many times when there is a "rigid" skull on applied kinesiology examination, it relates with postural strain often associated with foot dysfunction. One can often feel greater rigidity of the skull when challenging an individual in the standing weight-bearing position than when sitting, supine, or prone. Before lasting corrections can be obtained in the stomatognathic system, the structural strain from weight-bearing dysfunction must be corrected.

The applied kinesiology approach has shown that because of the connection in the fascial system, a change in any part of the body may create a disorder elsewhere. An anterior cruciate ligament injury can produce changes in the masseter, anterior temporalis, posterior cervicals, upper and lower trapezius and sternocleidomastoid muscles. **(Tecco et al., 2006)** Dvorak & Dvorak **(Dvorak & Dvorak, 1990)** injected a saline solution into the transverse process of C7 while using electromyography and observed muscle contractions in zones distal from the spinal myomere where the injection was made. An increase in active mouth opening and a decrease in myofascial trigger point sensitivity in the masseter muscle were observed in response to the stretch of the hamstring muscles, demonstrating a functional relationship between the masticatory and hamstring muscles. **(Fernández-de-las-Peñas et al., 2006)**

During normal walking, there is alternate facilitation and inhibition of the sternocleidomastoid and upper trapezius muscles to keep the head pointed forward as the shoulder girdle rotates with the gait; otherwise, one's head would rotate with the shoulder girdle. The organization of these muscles relates to the input from the foot and ankle proprioceptors. **(Dananberg, 2007)** Since the muscles insert into the skull, an improper temporal pattern can cause strain in the cranium and possibly create cranial faults. Tecco et al. **(Tecco et al., 2010)** have also shown there are detectable interrelationships between occlusion and locomotion. It is not uncommon in applied kinesiology practice to observe loss of cranial fault correction as soon as the patient walks or runs. The correction is usually maintained after a foot, gait, or some other modular problem is corrected.

As noted, shoulder motion harmony depends on proper inhibition and facilitation of the shoulder muscles, which are integrated with proper proprioception from the foot, leg, and pelvis during walking. **(Geyer & Herr, 2010)** All patients with shoulder problems should have screening tests of the foot mechanism early in the evaluation. With recurrent or resistant shoulder problems, a thorough evaluation of the feet, legs, and pelvis must be done.

Afferent supply from the feet is also instrumental in the organization of the sacrospinalis and other large muscles of the spine. **(Kavounoudias et al., 2001)** Disorganization of these muscles can cause spinal or pelvic subluxations to recur as soon as the patient walks, even though they were adequately corrected.

Proper muscle activity is important in supporting the ankle and arch during walking and running. Disorganization and possible muscle weakness in the foot and ankle are often indicated by frequent twisting of the ankle. Again, unfortunately, the patient will not necessarily volunteer this information because it is often considered a normal occurrence, or "I just have weak ankles, and nothing can be done about it."

During consultation the presence of symptoms may be volunteered by the patient, but more often they are observed or dug out by the physician. One must often ask leading questions that can be analyzed by the physician's knowledge of body integration.

The patient can often provide a clue about what is taking place by comparing the two extremities. Ask the patient if one extremity seems to feel different from the other when running or walking. Is there recurrent injury to only one side? Does the patient seem to favor one side in walking or running? Is there more difficulty turning in one direction than in another? Answers to these questions may lead an applied kinesiologist to think of the conditions that cause unilateral dysfunction, such as a lateral atlas subluxation or cranial faults; in addition, can modular factors or equilibrium proprioceptive function be at fault?

Appearance of the foot

It cannot be overemphasized that a patient should remove his shoes and socks for the initial examination. Most chronic foot involvements have characteristic body language that can be readily observed during examination. Foot problems that are not of a long-standing nature may not have revealing visual body language, but dysfunction can easily be determined by examination procedures that include palpation, range of motion, muscle testing, and applied kinesiology therapy localization and challenge. **(Logan, 1995)**

Goodheart has often said the body language does not lie. Clinical experience finds this to be true. When there is no apparent correlation between body language and clinical findings, it pays to persist in an effort to find a correlation. Take, for example, the situation described above where an individual gains relief after getting home and relaxing. If the weight-bearing and other factors are not found positive, persist with your evaluation by asking the patient about his working conditions. The type of physical activity may not be reproduced in examination for gait, etc. Situations have been discovered where an individual stands on one leg and pushes a lever with the other leg all day long. Simulating that activity in the office reveals a reactive muscle condition which, when corrected, eliminates the problem. In another case, it may be the work boots an individual wears. We have had people bringing in their work shoes and found them to be severely run over, with broken down counters and heel wear. Simply standing in the work boots reveals many positive findings using applied kinesiology. In some cases, correction of a low back problem is as simple as having the patient purchase a new pair of boots.

In general, a pretty foot is a normally functioning one. If a pretty foot is dysfunctioning, it probably has not done so for long. Dysfunction causes the foot to distort or appear strained, and ultimately become gnarled. **(Langer, 2007)**

Visual observation will reveal wear points on the skin. Corns and calluses on the tops and sides of the toes and foot usually indicate improper shoe fit. In the average individual, calluses on the sole of the foot usually indicates improper weight distribution of the foot, **(Maffetone, 2010, 2003; Langer, 2007)** probably relating with subluxations. They may also be due to improper footwear. Often there will be calluses under the distal heads of the metatarsals. They may be limited to only one metatarsal, usually the 2nd or 5th, or there may be calluses across all the metatarsal heads. The callus, of course, is nature's effort at building resistance to the abnormal weight distribution and is the natural response of skin to external pressure applied against the underlying bony surface. Neurovascular corns are very tender and painful and should be evaluated by a podiatric specialist. Distance runners may have normal calluses; they will not only be concentrated at abnormal points of wear or pressure.

There may be a subcutaneous fat pad along with abnormal calluses, indicating an additional body effort to protect the structure. In this case, the fat pad usually develops prior to a callus; thus an increased fat pad without a callus generally indicates the condition is not as chronic. In some chronic conditions there may be a fat pad without a callus, especially in individuals who lead a sedentary life with minimal weight-bearing activities.

What appears to be an increase in the fat pad may be localized edema from the tissue trauma of improper function. Also, the fat pad may move anteriorly, discussed with forefoot dysfunction.

When adequate correction is obtained, calluses that have been present for many years may slowly disappear. This amazes the patient because of the long-term necessity of having to have the calluses removed by abrasion or application of a keratolytic agent.

Observation of the skin often provides clues to circulation deficiency. The circulatory problem might be the patient's complaint during consultation, but more often it is found by the physician during examination. Poor circulation can be caused or aggravated by foot dysfunction, or it may be coincidentally present with a foot condition. Venostasis is observed as cyanosis of the nail beds. Further evaluation can be obtained if the doctor blanches the skin by digital pressure and records the time it takes for blood to return. **(Langer, 2007; Greenawalt, 1982)** Vascular tests such as the evaluation of pulses and for deep vein thrombosis have been described by Petty & Moore. **(Petty & Moore, 1998)** The entire foot and ankle, as well as the lower extremity, may show evidence of plethora. If the plethora is localized in the foot only, there may be compression of the posterior tibial vein in the tarsal tunnel.

The foot should be examined for temperature variances, edema, dependent cyanosis, and blanching of the elevated foot. An ischemic foot will blanch when elevated and flush when dependent. Skin will have a thin, elastic appearance and lack hair growth on the dorsum of the proximal toes. Arterial deficiency causes the foot to become cyanotic on dependency and blanch with elevation. **(Veves et al., 2002; Cailliet, 1997)**

Venous insufficiency is indicated by pitting edema, which is absent or decreased after a night's rest. Varicosities enhance the diagnosis, but edema must always create suspicion of a systemic condition. Good muscular function of the lower extremity enhances venous and lymphatic drainage. Contraction of neighboring muscles compresses lymph vessels, moving lymph in the directions determined by their valves. Manual or mechanical lymphatic reflex techniques may be effective ways to increase lymph removal from stagnant or swollen tissue. **(Chaitow & DeLany, 2008; Goodheart, 1998; Chaitow, 1987)** Arterial supply can be evaluated at the dorsalis pedis between the 1st and 2nd metatarsals, and at the posterior tibial artery behind the medial malleolus.

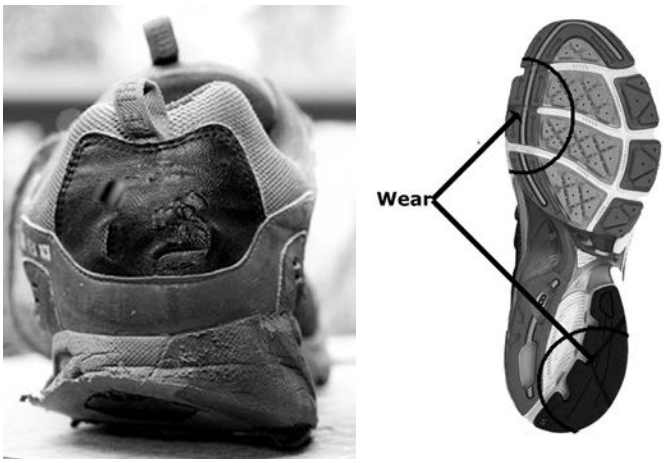
Weak, brittle toenails may indicate circulatory disturbance. Ingrown toenails may indicate short shoes or socks. Stretch socks are particularly problematic for some individuals. A dark lesion under the nail may be melanoma. **(Langer, 2007; Kalivas, 1983)**

Shoe wear

Evidence of disturbed function of the foot and ankle is often indicated by a patient's shoes. **(Abshire, 2010; Langer, 2007)** The heel should wear on the posterolateral aspect. When the wear deviates more laterally, pronation is indicated. Sometimes excessive sole wear underneath the metatarsal heads can be observed, but it is not a constant

finding with dropped metatarsals. The counter and vamp of the shoe should remain balanced over the heel and sole. Running over of the shoe in any direction indicates foot problems or poorly constructed shoes. Toe wear is indicative of weakness of the dorsiflexor muscles. Runners' shoe wear varies considerably from that of the average patient.

The patient should be questioned regarding his work,



Shoe wear with extended pronation.

dress, sport, and any other type of shoes worn. Often the shoes worn to the doctor's office are not those in which the patient spends most of his time. Questioning the patient about whether symptoms develop when certain types of shoes are worn may elicit no positive response at the time; however, it plants a seed for the patient's future evaluation and may later evoke a positive response. For example, correcting upper cervical subluxations and balancing the cervical musculature can effectively reduce a patient's headache for two to three days, only to have it return and need correcting again. It is not uncommon for the patient to report that the headache returned after he wore a pair of Western boots over the weekend. Evaluate the possible connection by having the patient bring the boots in question to the office. Correct the cervical subluxation and have the patient walk in the shoes usually worn during the week. If the subluxation does not return, have the patient walk in the boots. If the subluxations and muscular imbalance return, either the boots or the patient's feet are indicted as the cause of the recurrence; probably both are at fault.

Meridian system influenced by foot/ankle dysfunction

The meridian system is covered in the text ***Applied Kinesiology Essentials.*** However, it should be mentioned as part of the body language associated with foot and ankle problems. There are six meridians that flow through the ankle and foot. The normal flow of Chi (the energy of acupuncture meridians) may be interfered with by foot and/or ankle problems such as trauma or structural strain. Correction of a subluxation, fixation, or fascial or muscular strain may immediately improve energy flow in a meridian. When there is interference with a meridian's energy flow, it may cause a problem associated with that meridian, such as a gallbladder attack when the energy flow in the gallbladder meridian is reduced. If the patient has had no previous disposition toward gallbladder involvement and develops an attack within a day or two after ankle or foot trauma, the meridian system should be evaluated. Appropriate treatment to the gallbladder meridian at the location of injury may produce gratifying results within minutes. **(O'Connor & Bensky, 1981)**

There are occasions when a gallbladder attack is so severe that hospitalization is required if results are not obtained rather quickly. Returning the gallbladder meridian to normal by simply stimulating the meridian through the area of trauma at the ankle may alleviate the attack within minutes. If the physician does not have this specialized knowledge, the patient may have to be hospitalized and have gallbladder studies done; often the diagnosis will be a "sluggish gallbladder." Treatment usually consists of dietary recommendations and probably medication, which is credited for the improvement of the condition. On the other hand, the gallbladder dysfunction would probably have been self-limiting as the ankle healed and the meridian spontaneously returned to normal function.

Other meridians running through the ankle and foot are the stomach, bladder, spleen, liver, and kidney. When considering the foot and ankle for possible meridian involvement, remember that not only the meridians traversing the foot and ankle may be involved; the other six meridians can also be involved as a result of the mother-child effect, five-element law, or other pathways of energy exchange within the meridian system.

Costa and Araujo **(Costa & Araujo, 2008)** demonstrated one of the approaches applied kinesiology that has used for decades in evaluating the meridian system. They showed that stimulation of the sedation point for the Bladder meridian (acupuncture point stomach-36) induced decreased strength in the tibialis anterior muscle as measured by electromyography. According to applied kinesiology, the tibialis anterior muscle corresponds to the Bladder meridian.

Moncayo & Moncayo have written extensively about AK and the meridian system and have been able to demonstrate the usefulness of the working principles of AK in relation to Traditional Chinese Medicine. **(Moncayo & Moncayo, 2009)** Garten, Walther, Dale, Corneal and Dick, and Larson have demonstrated this relationship as well. **(Garten, 2002; Walther, 2000; Dale, 1993; Corneal & Dick, 1987; Larson, 1985)** (The AK approach to traditional Chinese medicine will be substantially updated in teh subsequent volume of this series, ***Applied Kinesiology Essentials.***)

Non-Weight Bearing Examination

Foot-ankle-leg balance.

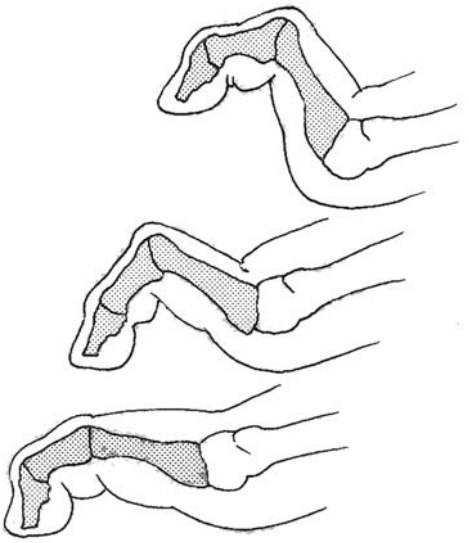
When the patient is prone or supine and the foot and ankle are hanging over the table end, the stabilized structure becomes the leg; the foot may deviate into structural distortion, which may not be observed when standing.

Abnormal flexion of the toes can be of three types. 1) "Mallet toe" is flexion deformity of the distal interphalangeal articulation. 2) "Hammertoe" is flexion deformity of the

proximal interphalangeal articulation with extension of the metatarsophalangeal articulation. 3) "Claw toe" is flexion deformity of both interphalangeal articulations with extension of the metatarsophalangeal articulation. **(Kwon et al., 2009)**

The most commonly observed deviation of the toes is claw toes. The claw-like appearance to the toes is often to the point that weight bearing on the toes is on the distal end of the distal phalanges. This position may be due to anomalous insertion of the extensor tendons, **(Kwon et al., 2009; Sgarlato et al., 1969)** for which surgical procedures are available. **(Kwon et al., 2009; Sgarlato, 1970)** Surgery in advanced cases may not even restrict marathon runners.

A more common cause of claw toes is relative weakness of the intrinsic compared to the extrinsic flexor muscles. **(Kwon et al., 2009; Ramamurti, 1979)** The extrinsic flexor muscles are the flexor digitorum longus and flexor hallucis longus. They insert into the distal phalanx and receive their nerve supply from the tibial nerve prior to the tarsal tunnel. The major intrinsic flexors are the flexor digitorum brevis and flexor hallucis brevis. The flexor

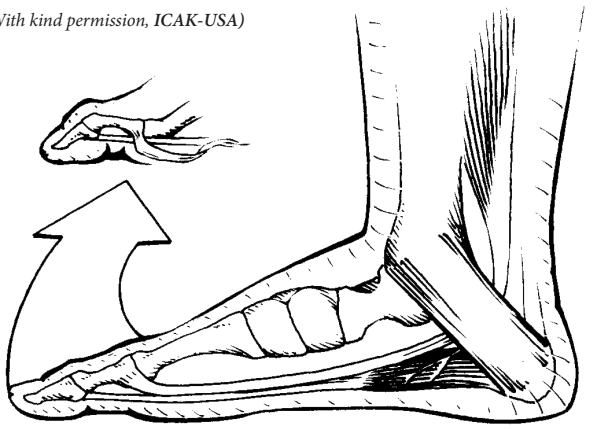


Toe deformities.

digitorum brevis inserts into the sides of the shaft of the intermediate phalanx, and the flexor hallucis brevis inserts into the sides of the base of the proximal phalanx of the hallux. Nerve supply to both muscles is the medial plantar nerve, a division of the tibial nerve at the tarsal tunnel. **(Gray's Anatomy, 2004; Hamilton, 1985)** Peripheral nerve entrapment at the tarsal tunnel may deprive the intrinsic muscles of normal nerve supply, causing the muscular imbalance that allows the interphalangeal articulation to rise, while the distal phalanx is pulled into flexion by the strong extrinsic muscles. Because of the imbalance between the longus and brevis muscles of the foot the distal toes curl inferior and posterior creating claw toes. Tarsal tunnel syndrome and foot pronation almost always precedes the phenomenon of claw toes.

With poor nerve supply to the intrinsic muscles, there is often plantar muscle atrophy. This is often mistaken by both the patient and the physician as a high arch. In reality,

*(With kind permission, **ICAK-USA**)*



Insertion of the muscles into the toes. (See Tarsal Tunnel Syndrome, Chapter 3)

the arch is usually broken down, receiving very poor support while walking and running. Generally, when there is extension-flexion deformity of the toes there is also an elevation of the extensor tendons, giving a strained look to the foot. This apparently fits the observation in applied kinesiology that when there is muscle weakness, antagonist muscles contract.

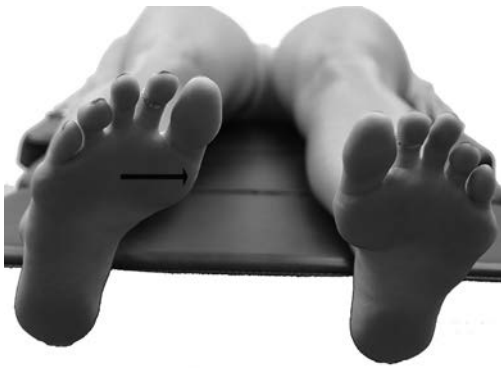
Because the abductor hallucis muscle also receives nerve supply from the medial plantar nerve, hallux valgus may develop along with the peripheral nerve entrapment at the tarsal tunnel. Other causes of hallux valgus are poorly fitting shoes or socks, and congenital variations of bone and of the insertion of the abductor hallucis.

Non-weight-bearing balance of the muscles that cross the ankle can be observed with the patient supine. The feet should hang over the end of the table far enough so that the edge of the table does not interfere with the foot position.

An imaginary line down the anterior ridge of the tibia should extend into the second toe. When it extends lateral to the second toe, the turgor of the lateral ankle stabilizing muscles — the peroneus tertius, longus, and brevis — is less than the medial ankle stabilizing muscles. This indicates either weakness of the peroneus group or hypertonicity of the tibialis anterior and posterior. This is more common than when the line projects medial to the second toe, indicating less turgor of the tibialis muscles. Again, it could be caused by hypertonicity of the peroneus group or weakness of the tibialis muscles.

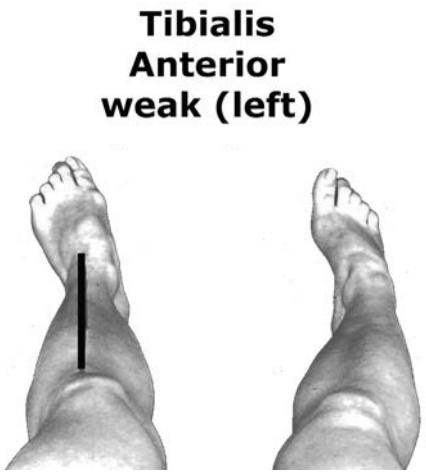
When observing lateral or medial deviation of the foot, care must be taken that it is properly related to the anterior tibial ridge. There should be neutral thigh rotation observed by the position of the patella. Some individuals have increased muscle or adipose tissue on the lateral aspect of the leg. In that instance, a line drawn down the center of the leg does not correspond with the tibial ridge.

Balance between the dorsiflexor and plantar flexor muscles can be observed by the amount of plantar flexion of the relaxed foot hanging over the end of the table. The peroneus tertius and tibialis anterior tendons

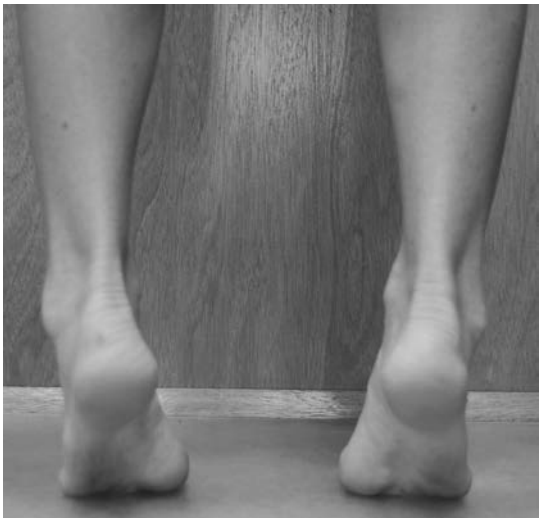


Peroneus longus and brevis weakness.

Tibialis posterior weakness.



Tibialis posterior weakness



Indicates strain of left posterior tibialis muscle

course anterior to the malleoli and are thus dorsiflexors. The peroneus longus and brevis and the tibialis posterior tendons course behind the malleoli and are plantar flexors; thus, if the relaxed foot is adducted with considerable plantar flexion, the peroneus group has less turgor than the tibialis group, and the peroneus tertius less turgor than the peroneus longus and brevis. This only takes into account the peroneus and tibialis groups. When considering dorsiflexion and plantar flexion, the length and strength of the soleus and gastrocnemius must be taken into account, as well as the flexor and extensor digitorum longus muscles. (Length of the gastrocnemius and soleus will be discussed further under "Hypermobile Flatfoot" and "Pronation".)

Palpation

A major tool in examination of the foot and ankle is palpation. Palpatory findings rapidly give the physician considerable knowledge. Observe for subluxated articulations, indications of congenital anomalies, muscles that are strained or hypertonic, and evidence of muscle atrophy. Palpatory findings may give evidence of pathologic or systemic conditions, such as arthritis, edema, circulatory disturbances, or neuropathy. Palpatory findings will be discussed with the conditions to which they relate.

Shock absorber test

The shock absorber test **(Zampagni et al., 2009; Walther 2000, 1981)** is unique to applied kinesiology. It appears to evaluate joint integrity to determine if the nervous system can rapidly recover from stimulation to joint proprioceptors applied as a quick, shocking force. Clinical evidence reveals that when the joint is functioning normally, there will be normal function of muscles associated with it (or remote from it) immediately after the shock is applied. When there is joint fixation or subluxation, a previously strong muscle will test weak for many seconds up to several minutes following the shock.

Muscles to test when applying the shock absorber test to the foot are those of the foot or leg involved with the joints being tested, and muscles of gait that have associated function with the foot, like the tensor fascia lata. **(Zampagni et al., 2009)** The psoas muscle is often used as an indicator muscle when doing the shock absorber test on the foot or ankle.

In addition to being a valuable screening test for the physician's examination, the shock absorber test can often be used to educate the patient about his condition. For example, if the patient is being examined for a shoulder condition and the examination is leading to the basic underlying cause being the foot and/or gait mechanisms, one can test shoulder muscles before and after the shock absorber test. If foot dysfunction disturbs normal activity of the shoulder muscles, it will be revealed in the shoulder muscle tests following the shock to the foot. One may find relatively normal tests of the shoulder muscles in conjunction with a shoulder problem; following the shock absorber test several muscles may test weak, and there may be pain in the shoulder upon testing the muscles when none was present in the clear. These findings demonstrate to the patient in a clear and dramatic way the relationship of foot dysfunction with the shoulder complaint. As noted before, the patient often has no complaint about his feet, yet the basic underlying cause of his chief complaint — in this case the shoulder — resides in foot dysfunction.

Because of the numerous articulations in the foot, the shock absorber test must be applied with many vectors to analyze most of the joints. Varying vectors of force into the distal metatarsal heads with glancing blows are usually effective in testing for dropped metatarsals; however, challenge — discussed later — is a better test. The transverse tarsal arch is shocked with vectors completely encircling it, that is, from the plantar surface to the lateral, dorsal, and medial surfaces, ending on the plantar surface. Sharp blows are applied continuously as the transverse tarsal bones are circled. Solid blows to the entire plantar surface of the foot test many of the articulations, including the ankle and perhaps even the knee

Shock Absorber Test

and hip. Medial, lateral, and posterior blows to the calcaneus further evaluate the ankle mortise, subtalar articulation, and the talar and calcaneal articulations with the midfoot.

The shock absorber test is designed to evaluate for joint dysfunction only; however, it is possible that shock to dysfunctioning neuromuscular spindle cells, Golgi tendon organs, or cutaneous proprioceptors may cause a muscle to test weak. It is also possible that joint dysfunction may not be discovered by this test because of the many articulations and vectors required to stimulate them.

Recent research from the Rizzoli Orthopedics Institute in Bologna specifically investigated the AK shock absorber test and showed that altered signals originating in the subtalar joint of the foot induce inhibition when the tensor fascia lata muscle is tested experimentally in soccer players with ankle injuries and muscle imbalances, eventually altering stability of the knee. **(Zampagni et al., 2009)**

The tensor fascia lata muscle, in particular, plays an important role in maintaining stability of the body and of the knee joint. If this muscle control should become impaired, functional disturbance of the lower extremity might occur and thus represent a risk factor for both the knee and ankle joints. The experimental team evaluated whether the shock absorber test would trigger differences in the force activation pattern of the tensor fascia lata muscle in athletes with ankle imbalance. In particular, the maximal isometric strength of the tensor fascia lata muscle was evaluated to determine whether soccer players who suffer from ankle imbalances experienced weakness in this muscle after the shock absorber test, comparing the unstable and stable ankles from groups of 15 and 14 male soccer players, respectively.

Using load cells to detect forces on the subject's ankle (electromyography), the experimenter applied manual percussion under the subtalar joint of all subjects. Before the shock absorber test was applied, the soccer players who experienced ankle imbalance showed no differences from the control (balanced ankle group) in the duration of tensor fascia lata muscle resistance to the force imposed by the operator during the manual muscle test. After the shock absorber test, however, the ankle imbalance group now displayed a unique and significant decrease in the duration of muscle resistance to the force applied by the operator. No such differences were seen in the uninjured side. **(Zampagni et al., 2009)**

This finding suggests that there is a change in the activation of the tensor fascia lata muscle in subjects with ankle imbalance, and that the AK shock absorber test could dependably diagnose this impairment. In practical terms, this observation suggested that ankle imbalance could be a risk factor for tensor fascia lata muscle weaknesses and ultimately instability of the knee.

It was suggested by these researchers that broader use of the shock absorber test might prevent further injury to the lower extremities exposed to external shocks, which could conceivably be experienced during everyday walking. When the shock absorber test is positive, a whole kinetic chain of events may occur including misalignment of the knees, anterior tilt of the pelvis, upper crossed syndrome, and forward head posture. **(Dananberg, 2007; Rothbart, 2006)**

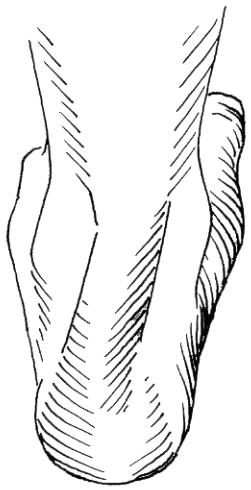
The shock absorber test is a good screening examination for joint dysfunction when put into proper perspective in the total examination. It should be remembered that it does not find most muscular and cutaneous receptor dysfunction; there may be joint dysfunction in a weight-bearing position that is not discovered in the non-weight-bearing shock absorber test.

Static Weight-Bearing Examination

Static postural examination of the foot includes visual inspection, palpation of the foot and remote structures, and remote muscle testing while standing with the foot in various positions. A weight-bearing examination should be done with the patient barefooted and also wearing shoes. Examination of the effect of various types of shoes and orthopedic support to the feet will be discussed later.

Helbing's sign

In the structurally sound ankle and foot, a line bisecting the calcaneus is in alignment with one bisecting the lower one-third of the leg when both feet are resting on the ground in bipedal stance. **(Evans, 2008)** Stated another way, a line bisecting the lower one-third of the leg should enter into the midline of the Achilles tendon and continue to project down through the midline of the calcaneus without deviation. If the line deviates laterally, it is called Helbing's sign. In the pronated foot, the line turns laterally as it extends to the midline of the calcaneus. There should be an approximately equal amount of medial and lateral malleolus viewed on each side of the Achilles tendon when observation is made directly posterior to the foot.

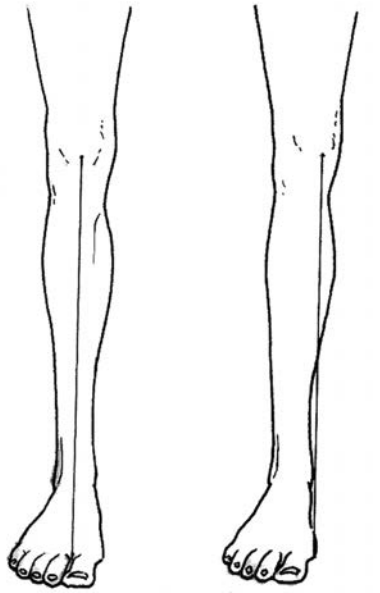


Helbing's sign.

Knee and leg position

When viewed from the anterior, the patella should be centered. With pronation the knee medially rotates. **(Dananberg, 2007; Prior, 1999; Rothbart & Estabrook, 1988)** A vertical line from the center of the patella should drop between the 1st and 2nd toes. **(Cailliet, 1997)** When pronation or internal tibial torsion or internal femoral torsion is present, the line from the patella will drop medial to the 1st and 2nd toes.

An individual with tibial or femoral torsion, as well as primary genu varus or valgus, is very likely to develop secondary involvement of the foot. Treatment, if applicable, should be directed to the primary cause and attention given to the foot dysfunction.



Line from center of the patella should drop between the 1st and 2nd toes.

General postural balance should be related to possible foot dysfunction. As previously noted, a normally functioning positive support reaction is important to facilitation of the extensor muscles for upright posture. Increased spinal curves in the sagittal plane may be due to improper proprioception from foot dysfunction.

Weight-bearing tests

The results of testing muscles remote from the foot will change when weight bearing is causing improper proprioceptive impulses. Muscles that tested strong in the prone or supine position may fail to perform adequately during manual muscle testing when in the static weight-bearing position. Delahunt et al have shown that patients with ankle instability **(Delahunt et al., 2006)** exhibit altered muscle activation and movement patterns in areas remote from the primary physiopathology in the foot. Higher motor system functions usually compensate for functional instability and motor weakness that may result from subluxations, soft-tissue injuries, proprioceptive disturbances and pain. Edgerton and colleagues **(1996)** proposed that decreased muscle recruitment (from an inhibited muscle) can result in increased recruitment from compensating motor neuron pools, possibly leading to further injury. In some cases, basically all muscles tested while weight bearing will fail, while in others only certain muscles do not initially lock on the manual muscle test. When muscle dysfunction is selective, it usually mirrors the patient's symptoms. For example, when foot dysfunction contributes to a shoulder problem, some or all of the shoulder muscles will fail to test normal during a weight-bearing manual muscle test.

Many factors can contribute to the change in muscle function from non-weight bearing to weight bearing. In addition to foot and leg problems, weight bearing in the spinal column can change the vertebral subluxation or fixation status. Various baroreceptors in the vessel systems of the body can be influenced, as well as receptors in the skin, abdominal cavity, equilibrium mechanism, and others. To help differentiate the change of muscle function due to static standing from other causes, have the patient stand on the lateral borders of his feet in the case of pronation. Usually this will cause the muscles that weakened when standing flatfooted to test normal. Standing on the lateral borders of the feet changes stimulation to the foot receptors and also to the receptors throughout the leg, knee, thigh, hip, and pelvis. **(Rothbart, 2006; Prior, 1999)**To more specifically localize the change to the foot, one can place pads under the navicular bone, metatarsals, or other appropriate area of the foot, and then re-test muscles for improvement.

Some muscles will develop or have increased tenderness when the patient with foot dysfunction stands. The origin, insertion, and belly of the muscles should be evaluated. Common muscles having increased tenderness with weight bearing are the upper trapezius, sternocleidomastoid, gluteus maximus, and tibialis posterior. **(Simons et al., 1999)** The tenderness can often be relieved by having the patient stand on the lateral borders of his feet for a few minutes, or stand on pads as previously mentioned. Temporary taping, discussed later, may also relieve strain. The physician can hold various bones of the foot in different positions, such as elevating the navicular bone while palpating the tibialis posterior or gluteus maximus for reduction of tenderness. A two-person examination team can do the same with muscles that are more distant from the foot, such as the sternocleidomastoid and upper trapezius.

The effect of pronation on circulation can be observed in a general manner with a finger pressure test. **(Greenawalt, 1982)** With the patient in a neutral standing position, observe for localized redness of the medial dorsal portion of the foot. Apply finger pressure to different areas, expressing blood from the area. Upon releasing the pressure, observe the length of time required for the color to return. Have the patient externally rotate the leg while the foot remains in place. This takes pressure off the pronated foot. Repeat the blanching test and observe for reduced time for normal color to return.

Many subtle factors regarding the patient's stance can be observed when he is not aware that a postural evaluation is being made. The patient may subconsciously move into a more comfortable position, or one that in all probability does not adversely stimulate the proprioceptors. This is often seen, especially in children who have foot pronation; they stand on the outer borders of their feet. The tests described previously explain why this is a more comfortable position. Many additional observations can be made by keeping in mind that the body subconsciously puts itself in a position for better function or relief.

In foot pronation there is excessive tension on the plantar fascia. To evaluate plantar fascia tension, have the patient stand with two blocks under each foot. One block is put under the calcaneus and the other under the metatarsal arch. Palpate the tension of the plantar muscles and their fascia when the patient is in neutral stance on the blocks. Then have the patient rotate the knees externally, changing the stance to stand on the lateral foot border. Again palpate the muscle and fascia. There should be little tension change if the ligaments and bones are supporting the arch as they should be in static weight bearing. **(Basmajian & Stecko, 1963)**

Dynamic Examination

The weight-bearing examination up to this point has dealt only with static weight bearing. As the examination progresses into walking and running, many other factors come into play, such as the equilibrium proprioceptors, gait mechanism, dural tension, body module organization (PRYT technique), reactive muscles, and others discussed thoroughly elsewhere. **(Leaf, 2010; Frost, 2002; Walther, 2000; Maffetone, 1999; Goodheart, 1998-1964)**

It serves here to put into perspective the addition of walking and running when specifically evaluating for weight-bearing influence on and by foot function. Occasionally, adverse influence of foot dysfunction is not recognized until after the patient walks. Also, there may not be loss of foot and ankle correction until the patient walks. It is important to have the patient walk in a figure-eight pattern or in a circle, first in one direction and then in the opposite one, because turning in either direction may stress the foot in a manner different from walking in a straight line.

The importance of this is illustrated by a patient being treated for phlebitis. Her major etiologic factor was foot dysfunction. On initial examination it was recognized that severe pronation and foot strain were present and contributing to leg strain and the phlebitis. The patient easily recognized the foot problem, but she was hesitant to believe that it might be contributing to the phlebitis. Foot corrections and attention to the supporting musculature were accomplished, but the patient lost the corrections immediately upon walking. Spinal Pelvic Stabilizers® from Foot Levelers, Inc., **(Foot Levelers, Inc.)** and adequate shoes were prescribed. The patient refused the Spinal Pelvic Stabilizers®, stating that eight to ten orthotics had been prescribed and fitted over the years and none helped her chronic foot strain. (She had spent well over a thousand dollars on them.) In light of this, it was necessary to provide tape support to the foot and ankle. She did agree to purchase and wear proper shoes.

The current episode of phlebitis had lasted approximately three months before my examination and had not responded to standard medical treatment. Her condition was so severe that she could walk no farther than across her living room. With correction of her foot problem and other chiropractic and AK treatment, the condition significantly improved in one week; within three weeks there was no evidence of phlebitis. During the three weeks, most of the structural corrections were maintained. The foot and ankle were re-taped as needed. At the end of the three weeks, an attempt was made to eliminate the tape support; within a week, the phlebitis began returning. She then agreed to be fitted for Spinal Pelvic Stabilizers® which, with proper shoes, allowed the corrections to be maintained.

Approximately nine months passed with no recurrence of phlebitis, and the patient was relieved of the chronic foot and leg pain she had previously experienced. Then she mentioned on a routine maintenance office visit that she was beginning to develop leg pain again. Examination revealed foot subluxations and numerous muscle weaknesses of the leg and foot. It was curious that the corrections, which had held so well, had been lost. She was questioned about wearing her Spinal Pelvic Stabilizers® on a regular basis; she stated that she did. Corrections were made, and she was asked to walk; the corrections were not lost. The patient was again scheduled for a routine one-month examination. Within two weeks she made an appointment because the leg pain was worsening. Examination revealed similar findings; corrections were made, and she was able to walk without losing the corrections. Again she was scheduled for re-examination in one month. In two weeks she again returned to the office with worsening of the leg pain. Examination revealed the same dysfunction; again correction was not lost after walking. She began to make appointments more frequently until she was being seen twice a week. Each time she came in with foot and leg dysfunction; each time it was corrected and the corrections were not lost upon walking. By this time two-and-a-half months had passed since the recurrence of symptoms, and frank phlebitis was now present. She was back to the point of not being able to walk farther than across her living room without stopping because of pain. Her days were again spent with the leg elevated and heat applied. She was ready to return to the previous doctor for medication, even though his therapy had failed.

Because of her discouragement, SCC spent more time talking with her on this occasion, completing the record and discussing with her who she could be referred to. When I finished she walked ahead of me to leave the treatment room; she made a sharp turn, and I observed a major foot roll within her shoe. Calling her back, I re-examined her; all the corrections just made had been lost. My previous test was for her to walk in a straight line, stop, turn around, and walk in a straight line. In other words, she was not walking and turning at the same time. Under those conditions no corrections were lost. When she turned while walking, her foot would roll and the corrections were lost.

I asked her when she started wearing the shoes she had on, and it turned out to be just prior to the time her symptoms began to return. The purchase of a new pair of shoes of the same type that I had originally prescribed allowed the corrections to be maintained, and her recovery from the phlebitis was uneventful. Her condition was followed for several years; there was no recurrence of the phlebitis, although she previously had been having several episodes per year.

Since that experience, it has been standard procedure to always test a patient walking in either a figure-eight pattern or around a circle, first in one direction and then in the other, so that both directions of turning are incorporated into the walking pattern. The figure eight-pattern is optimal.

Examination proceeds from dynamic evaluation of the foot to correlating it with the patient's gait. There are several AK methods that find functional conditions otherwise missed. Corrective treatment is usually easily applied and produces lasting results.

It must be remembered that whether you consider the foot as an architectural wonder or a pair of stumps to be ignored, it is an integral component of whole-body function with its influences being felt both locally and throughout the body. As this case demonstrated, the health and integrity of the foot should be a high priority in most therapeutic interventions.

Foot and Ankle Anatomy and Physiology

Divisions and Motions of the Feet

The foot is divided into three functional segments: the hindfoot, the midfoot, and the forefoot. The hindfoot is composed of the talus and calcaneus. The talus is the foot's only bony connection with the rest of the body. It is unique in that it has no direct muscular attachment. The heel of the foot is composed of the calcaneus. The talus and calcaneus articulate with the cuboid and navicular, which is the hindfoot's connection to the midfoot. This joint complex is called Chopart's joint. In addition to the cuboid and navicular, the midfoot is composed of the three cuneiform bones.

The mechanics between the talus, calcaneus, navicular, and cuboid bones allow the two important foot movements: inversion/eversion and pronation/supination. The flexibility of these movements allows the foot to accommodate to the shape of the substrate, and yet lock together to become a solid lever of great strength during certain phases of gait. **(Langer, 2007; Dananberg, 2007; De Wit, 2000; Hamilton & Ziemer, 1983)**

The forefoot is composed of 5 metatarsals and 14 phalanges, making a total of 26 bones in the foot. The articulations of the cuboid and cuneiform bones with the metatarsal bones are collectively called Lisfranc's joint. The anterior pillars of the longitudinal arches are composed of the forefoot, which is active in the propulsive stage of gait.

Classification of the forefoot

The forefoot is classified according to the relationship of the length of the 5 rays or toes and the relationship of the length of the metatarsals. These are the digit and metatarsal formulae, respectively. Viladot **(Viladot, 1982)** reports the percentage of individuals with each type.

Classifying the forefoot according to the length of the toes, there are three types: 1) big toe shorter than the 2nd toe, 22%, Greek foot; 2) big toe longer than the 2nd, 69%, Egyptian foot; and 3) big toe same length as 2nd toe, 9%, squared foot.

Metatarsals are classified according to three types. 1) The 1st metatarsal is equal in length to the 2nd, termed index plus–minus, 28%. 2) The 1st metatarsal is shorter than the 2nd, termed index minus, 56%. 3) The 1st metatarsal is longer than the 2nd, which is termed index plus, 16%.

The digit formula can be combined with the metatarsal formula. The Egyptian–type foot with index–minus metatarsals is the most frequent type.

The Greek-type foot with index plus-minus (short big toe and 1st metatarsal equal to 2nd) is ideal. **(Hoppenfeld, 1982)** It rarely has pathological alterations. The metatarsal index–minus predisposes an individual to hallux valgus, and the metatarsal index–plus favors hallux rigidus in men and sesamoiditis in women. The Greek-type foot with index–plus or index–plus–minus adapts best to modern footwear.

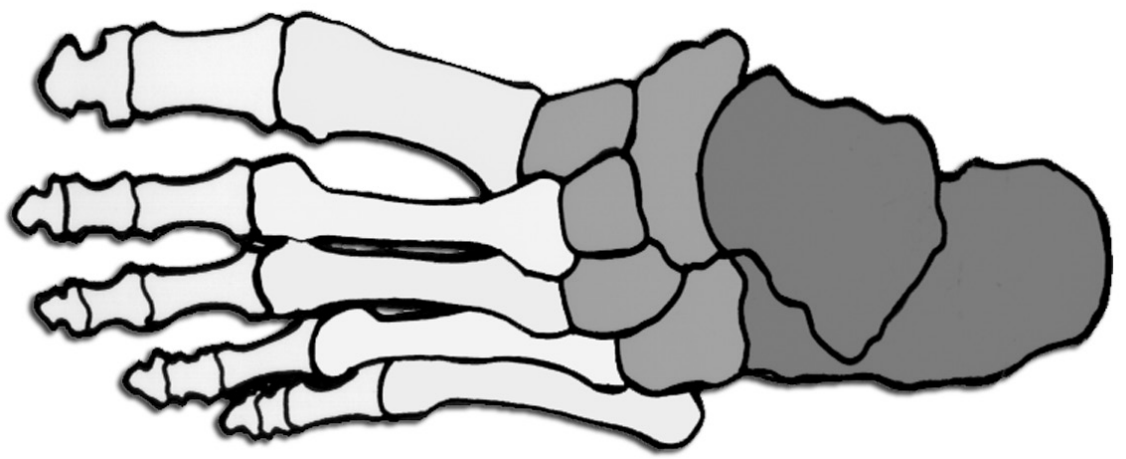
Motions, positions, and fixed structural positions of the foot

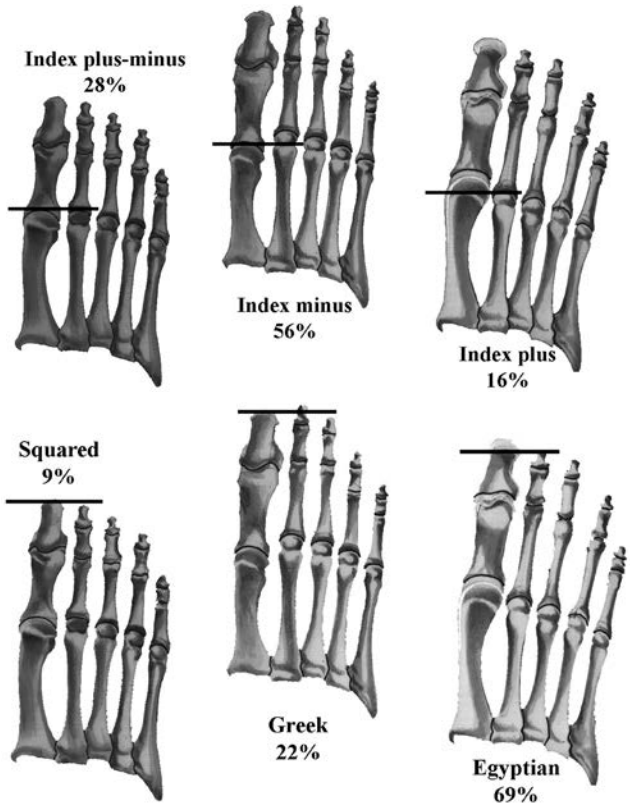
Terms used to describe motions, positions, and fixed structural positions vary somewhat among authors. **(Gray's Anatomy, 2004; Kleiger, 1956)** Levangie & Norkin **(Levangie & Norkin, 2001)** point out in reference to the terminology issue for foot motions, "Terms used in research and published literature should carefully be defined to impart the most and clearest information."

The description of terms given here is that generally used. In any event, the reader can correlate with information provided by other authors. **(Table 1)**

Adduction. Adduction, which takes place in the transverse plane, is movement of the forefoot toward the midline of the body. An adducted foot has the forefoot deviated toward the midline of the body. The term "adductus" refers to a fixed structural position of a part of the foot, as if it were adducted.

Abduction. Abduction is motion in the transverse plane in which the forefoot moves away from the midline of the body. An abducted foot occurs when the forefoot has moved away from the midline in the transverse plane. "Abductus" means a fixed structural position of the forefoot, as if it were abducted.





There is different use of the terms "abduction" and "adduction" when referring to active movement of a part, such as the hallux, than that presented for the forefoot. Abduction of the hallux, as with activity of the abductor hallucis, is movement away from the midline of the foot; adduction of the hallux, as accomplished by the adductor hallucis, is movement toward the midline of the foot. **(Gray's Anatomy, 2004; Turek, 1984)**

Inversion. Inversion is often used synonymously with supination and occurs in the frontal plane. It is motion of the foot so that the plantar aspect of the foot, or a portion of it, tilts to face toward the midline of the body. Inverted refers to the foot, or a portion of it, in the position of inversion. When the foot, or a portion of it, is fixed in an inverted position, it is termed "varus."

Eversion. Eversion is often used synonymously with pronation and is foot motion in the frontal plane in which the plantar surface of the foot faces away from the midline of the body. When the foot, or a portion of it, is in eversion, it is termed "everted." If the everted position of the foot, or a portion of it, is fixed, it is termed "valgus."

Dorsiflexion. When the foot, or a portion of it, moves in the sagittal plane so that the forefoot moves toward the tibia, it is termed "dorsiflexion." Note that all dorsiflexion does not take place at the ankle joint. A position of dorsiflexion is termed "dorsiflexed." A fixed limitation of dorsiflexion at the ankle joint is called "ankle equinus" or "talipes equinus." Less than 10° of ankle dorsiflexion is considered talipes equinus.

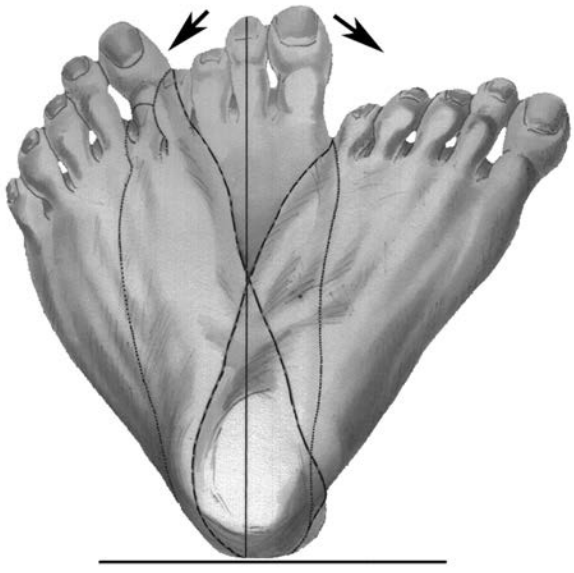
Plantar flexion. Movement of the foot, or a portion of it, in the sagittal plane so that the forefoot moves away from the tibia is termed "plantar flexion." Occasionally in the literature one will find the term "plantar extension," which is synonymous with plantar flexion. Plantar flexed means that the foot, or a portion of it, is in plantar flexion. A fixed limitation of plantar flexion of the foot at the ankle joint is called "ankle calcaneus" or "talipes calcaneus."

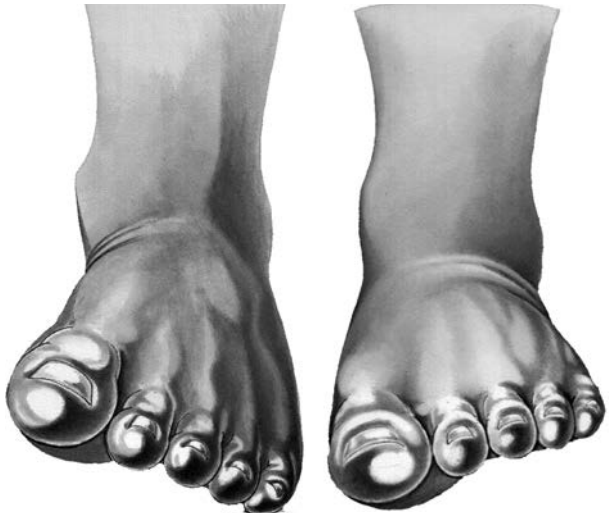
Pronation. Pronation is simultaneous abduction, eversion, and dorsiflexion. The term "pronated" refers to the position into which the foot moves with pronation.

Supination. Supination is simultaneous adduction, inversion, and plantar flexion movement of the foot. "Supinated" is the position of the foot as a result of supination.

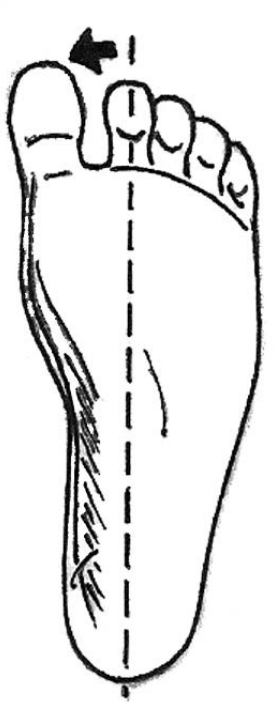
Foot Osteology

The foot consists of 26 bones that can be divided into the tarsus, metatarsus, and phalanges. Almost ¼ of the body's bones are in the feet. The tarsus consists of the bones making up the posterior half of the foot — the talus, calcaneus, cuboid, navicular, and 1st medial, 2nd





Inversion ***Eversion***



Hallux abduction

intermediate, and 3rd lateral cuneiform. The metatarsus consists of 5 bones, and there are 14 phalanges, 3 for each toe except the first, which has 2 phalanges.

A digital ray is a digit of the hand or foot consisting of the metacarpal or metatarsal and its corresponding phalanges. The entire unit or the great toe, then, is referred to as the 1st ray, 2nd toe the 2nd ray, and so on.

For convenience sake anatomists divide the foot into three main sections: the *hindfoot*, the *midfoot*, and *forefoot*.

Hindfoot

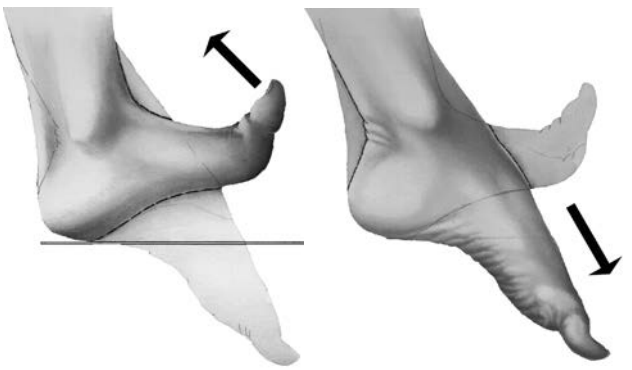
The hindfoot consists of only two bones, the calcaneus and talus. The calcaneus is the major contact of the posterior foot with the substrate, and the talus is the foot's only contact with the leg.

Calcaneus (os calcis). The calcaneus is the largest bone of the foot. It forms the heel of the foot and transmits the hindfoot's weight to the substrate. The posterior surface projects posteriorly beyond the bones of the leg to provide an effective lever for the insertion of the calf muscles. The superior surface has two or three facets that articulate with the talus. The posterior articular surface is the largest. Anteriorly there are one or two articular surfaces to reciprocate with the corresponding surfaces of the talus.

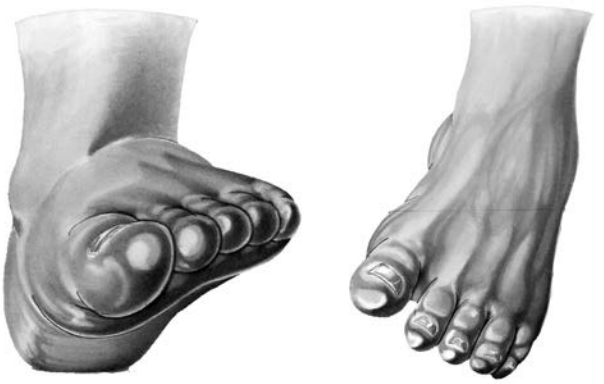
The sustentaculum tali arises at the anterior superior medial surface of the calcaneus. Its superior surface is the medial articular surface for the talus mentioned above. The inferior surface of the sustentaculum tali is grooved for the tendon of the flexor hallucis longus. The entire anterior surface of the calcaneus is a concavoconvex facet for articulation with the cuboid bone.

Talus (astragalus). The talus is the only articulation between the foot and leg. It is unique because it is the only bone in the body that does not have direct muscular attachment. The body of the talus is basically cuboid. The superior surface is a trochlear surface for articulation with the tibia. The medial surface articulates with the medial malleolus of the tibia, and the lateral surface articulates with the fibula. The medial and lateral surfaces form a wedge, with the widest portion at the anterior. The articulation of the body of the talus with the tibia and fibula is called the ankle mortise; it will be discussed later with the ankle joint. The ankle mortise is designed to handle high levels of force. Compressive forces transmitted across this joint during gait may reach five times the body weight, while tangential shear forces (the result of internal and external rotational forces associated with the body moving over the foot) may reach 80% of the body weight. **(Gray's Anatomy, 2004)** The fibula has little weight-bearing responsibility. Levangie & Norkin state "no more than 10% of the weight that comes through the femur is transmitted through the fibula." **(Levangie & Norkin, 2001)**

The inferior or distal surface of the body has two or three surfaces that articulate with the calcaneus, called the subtalar or talocalcaneal articulations. The larger surface is posterior and is concave to reciprocate with the convex surface of the calcaneus. These surfaces allow multiaxial movement between the two bones, including inversion and more limited



Dorsiflexion ***Plantar flexion***



Pronation ***Supination***

MOTION	POSITION	FIXED	COMBINATION
Abduction	Abducted	Abductus	
Eversion	Everted	Valgus	Pronation – Pronated
Dorsiflexion	Dorsiflexed		
Adduction	Adducted	Adductus	
Inversion	Inverted	Varus	Supination – Supinated
Plantar flexion	Plantar flexed		

Table 1
Summary of foot and ankle positions and motions

Superior view of left calcaneus

eversion, adduction and abduction. Combinations of these movements contribute to pronation and supination.

These motions of the foot are not limited to the subtalar articulation; they include movement at the transverse tarsal articulations as well.

The posterior articular surface is separated from the anterior one by a groove called the sulcus talus. The calcaneus has a similar groove called the sulcus calcanei. When the two are combined, the tarsal canal is formed between the two bones. The largest end of the canal is called the sinus tarsi, and it opens laterally. It can be palpated in front of the fibular malleolus when the foot is markedly inverted. From the sinus tarsi the canal angles slightly posteriorly and medially to open just behind the sustentaculum tali of the calcaneus.

Anterior to the sulcus talus is the anterior calcaneal articulation, which may be singular or double. The anterior facets are convex on the talus to reciprocate with the concave articular surfaces of the calcaneus. This is exactly opposite the posterior articulation of the talus and calcaneus.

The talocalcaneal articulations are also called the subtalar articulations. The major motion at the subtalar articulation is inversion and eversion. With the foot in free motion, the calcaneus moves on the talus, whereas when the foot is planted on the floor, the talus moves on the calcaneus.

Projecting anteriorly and medially from the body is the neck of the talus, which joins the head of the talus to the body. The head of the talus is a large oval surface for articulation with the navicular bone.

Midfoot

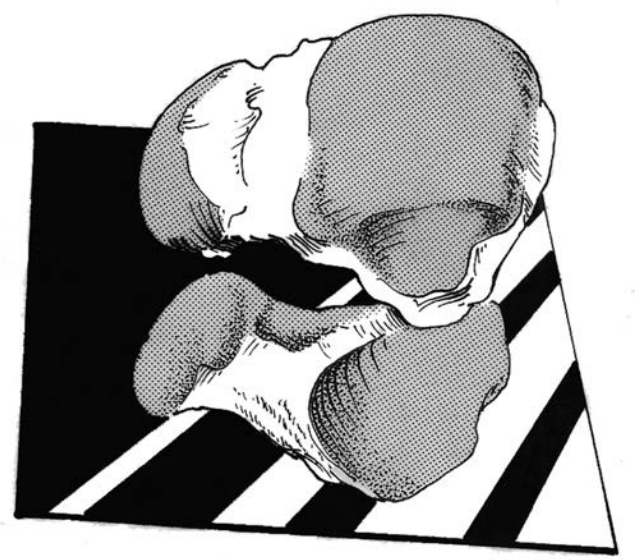
The midfoot consists of five tarsal bones — the navicular, cuboid, and three cuneiforms. These bones are wedge–shaped, being wider dorsally and narrower plantarly. When articulated they form longitudinal and transverse curves, with the concavities facing plantarly. The bases of all five metatarsal bones are also wedge–shaped, forming a medial–to–lateral arch. **(McDougall, 2009; Levangie & Norkin, 2001; Cailliet, 1997)** The hindfoot and midfoot are joined by the transverse tarsal articulation (Chopart's joint). The head of the talus fits into the posterior concave surface of the navicular bone. Inversion and eversion are allowed at this articulation; there is a significant gliding action because the articular surface of the head of the talus is larger than that of the navicular.

The rest of the transverse tarsal articulation is the calcaneocuboid joint. More stability is gained in the foot if the talonavicular and calcaneocuboid articulations are not parallel. This causes joint restriction for better foot stability. In pronated feet the articulations are usually parallel.

Navicular. The navicular bone (scaphoid) provides three facets for articulation with the cuneiform bones on its anterior surface. The lateral surface usually does not articulate with the cuboid; however, it sometimes does present an articulating surface. Its posterior concave surface articulates with the head of the talus. The navicular bone often requires manipulation. When it is subluxated with the talus, the deep articulation of the head of the talus into the navicular must be considered when designing a line of drive for correction. For this reason, and because of the varying articulating surfaces, careful challenge is necessary to find the optimal vector of force for effective adjustment of the bone.

Cuboid. The cuboid bone derives its name from its cuboidal shape. It is lateral to and longer than the navicular bone. The posterior surface is almost flat and articulates with the calcaneus. The anterior surface is divided into two facets for articulation with the 4th and 5th metatarsal bases. The proximal medial surface is adjacent to, but only occasionally articulates with, the navicular. The distal medial surface has a flat facet for articulation with the lateral 3rd cuneiform.

Cuneiform bones. The three cuneiform bones are called medial, intermediate, and lateral or 1st, 2nd, and 3rd, respectively. They are wedge–shaped, being wider dorsally and narrower plantarly. The 1st cuneiform is the largest. The



Left talus *(With kind permission, ICAK-USA)*

posterior surface articulates with the most medial and largest of the three facets on the anterior surface of the navicular. The inferior or plantar surface is wider than the wedge portion of the bone, forming the base of the wedge. The lateral surface is concave, articulating with the 2nd cuneiform.

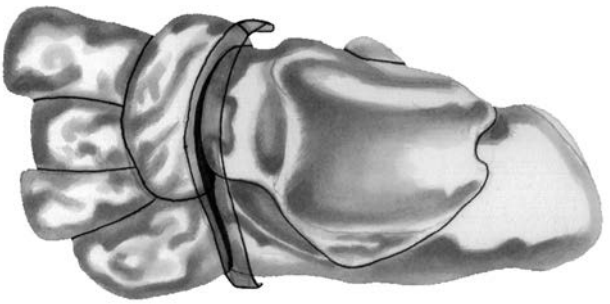
The 2nd cuneiform is the smallest of the three. Specifically it is shorter than the 1st and 3rd cuneiform bones, thus forming a groove for articulation of these three bones with the 2nd metatarsal base. The lateral surface is a smooth facet for articulation with the 3rd cuneiform bone.

The 3rd cuneiform is intermediate in size between the 1st and 2nd cuneiform bones. The posterior surface articulates with the lateral facet of the navicular surface. The proximal surface articulates with the 2nd cuneiform, and the distal one with the lateral surface of the base of the 2nd metatarsal. The lateral surface of the 3rd cuneiform also has two articular surfaces. The proximal one articulates with the cuboid, and the distal with the medial side of the base of the 4th metatarsal bone. The anterior articular surface of the 3rd cuneiform mates with the 3rd metatarsal bone.

Forefoot

Metatarsal bones. The metatarsal bones have common characteristics of a base, which is proximal and wedge–shaped to articulate with the tarsal bones. The base has facets on its sides to articulate with contiguous metatarsal bones. The body is the shaft, which gradually grows smaller as it progresses distally. The head is the distal portion extremity, which presents a convex surface to articulate with the proximal phalanx. The plantar surface of the head is grooved for the flexor tendons.

The 1st metatarsal is the thickest, and usually the shortest, of the metatarsal bones. Differing from the other metatarsals, the base as a rule has no articular facet on its sides. The proximal portion of the base of the 1st metatarsal is kidney–shaped to articulate with the 1st cuneiform. It has dorsal and plantar flexion and rotation, as well as a gliding action on the surface of the 1st cuneiform. The



Foot integrity is improved when the articulations of Chopart's joint are not in a straight line.

head is large and has two grooved facets on the plantar surface to articulate with sesamoid bones, which are incorporated into the tendons of the flexor hallucis brevis. They bear body weight and act as a fulcrum for the tendon. This arrangement is important for action of the medial longitudinal arch, which will be discussed later.

The 2nd metatarsal bone is usually the longest of the metatarsals. Its base has four articular surfaces. Proximally it articulates with the 2nd cuneiform. The medial surface of the base articulates with the 1st cuneiform. There are two lateral articular surfaces that articulate with the 3rd cuneiform and 3rd metatarsal bones.

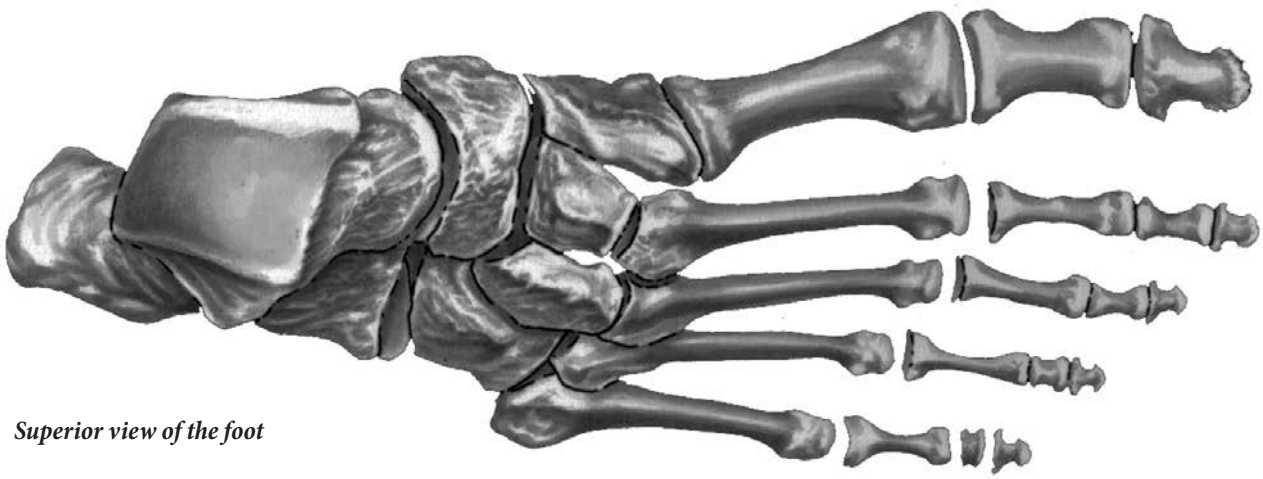
The 3rd metatarsal bone articulates proximally with the 3rd cuneiform. The medial and lateral surfaces of the base articulate with the 2nd and 4th metatarsals, respectively.

The 4th and 5th metatarsal bones are progressively smaller. The base of the 4th metatarsal articulates with the cuboid. The lateral and medial surfaces of the base articulate with the 3rd and 5th metatarsals, respectively. The 5th metatarsal base articulates with the cuboid, and medially with the 4th metatarsal. It has a tuberosity on its lateral side, into which the tendon of the peroneus brevis inserts. The peroneus tertius inserts into the medial part of the dorsal surface of the 5th metatarsal bone.

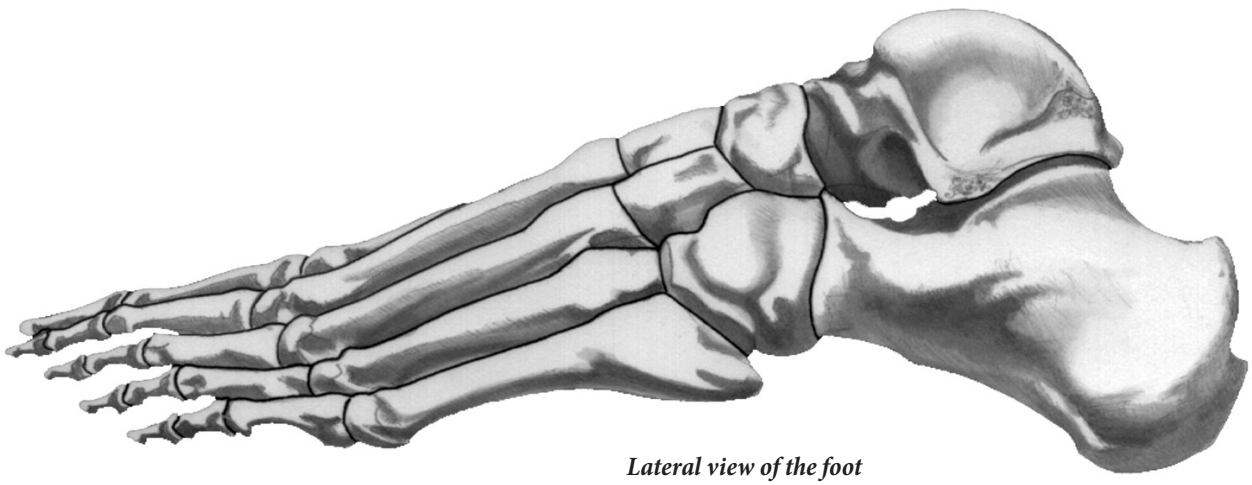
The wedge shape of the mid–tarsal bones, along with the wedge shape of the base of the metatarsal bones, forms longitudinal and transverse curves with the concavities facing plantarly, thus forming a medial–to–lateral arch frequently called the transverse arch. The proximal, medial, and lateral portions of the 3rd and 4th metatarsals, obliquely shaped, permit a progressive rotary motion of the 3rd on the 2nd, 4th on the 3rd, and 5th on the 4th. This motion increases the transverse arch and thus "cups" the sole of the foot.

Phalanges. There are three phalanges for each ray, with the exception of the first where there are two. The great toe has the flexor hallucis longus attached to the distal phalanx on the plantar surface, and on the dorsal surface the extensor hallucis longus. The proximal phalanx of the great toe has attachment on its dorsal surface for the extensor digitorum brevis. At the medial side of the base there is attachment for the abductor hallucis. The plantar surface of the proximal metatarsal has two attachments for the flexor hallucis brevis on the lateral and medial aspects. There is also attachment on the lateral plantar surface of the base for the adductor hallucis.

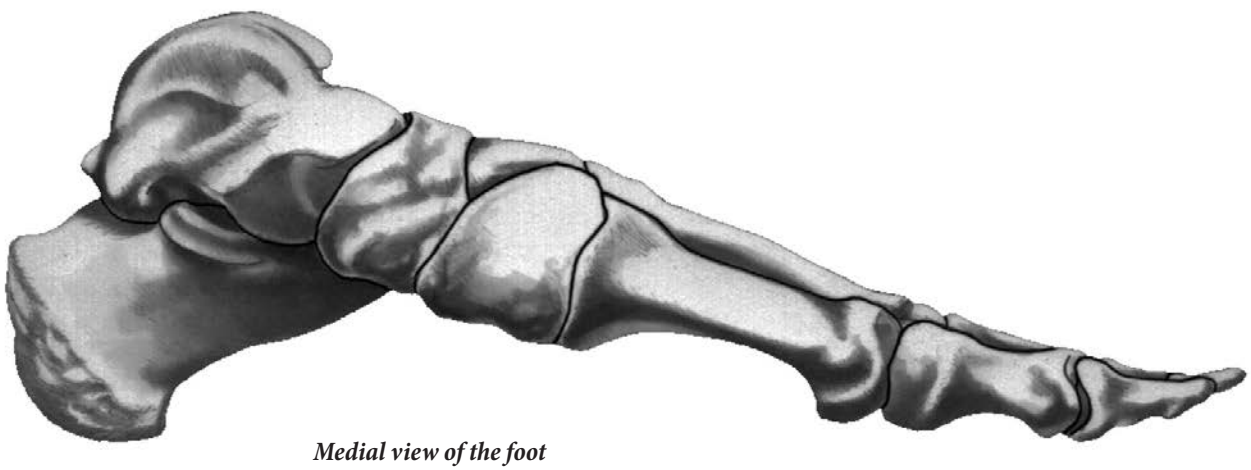
The proximal or 1st phalangeal row of the 2nd through 5th rays has proximally a base that is concave and a head that presents a trochlear surface for articulation with the



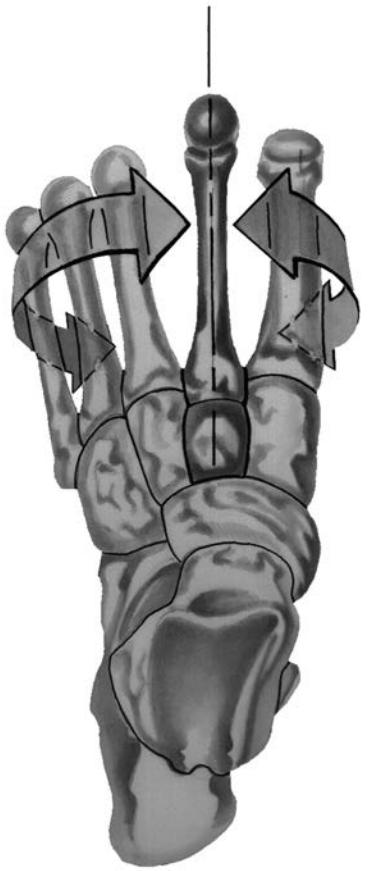
Superior view of the foot



Lateral view of the foot



Medial view of the foot



Rotation around 2nd metararsal

middle phalanx. The 1st tendon of the extensor digitorum brevis inserts into the dorsal surface of the 1st metatarsal bone; the 2nd through 4th tendons attach to the lateral sides of the extensor digitorum longus tendons.

The middle or second row of the 2nd through 5th toes has characteristics similar to the proximal row, but they are shorter and broader. The flexor digitorum brevis inserts on the plantar surface at the base, and the extensor digitorum brevis on the dorsal surface.

The distal row is called the ungual phalanges. Each presents a base to articulate with the trochlear surface of the head of the middle phalanx, and ends in a point. The distal phalanx of the 2nd through 5th toes has an attachment point on the plantar surface for the flexor digitorum longus, and on the dorsal surface for the extensor digitorum longus.

There are many additional, smaller intrinsic muscles of the metatarsals and phalanges that are extremely important in proprioceptive function of the foot and its relationship with the positive support mechanism.

Accessory bones. Accessory bones of the foot are supernumerary bones. They appear by age 10 and are fully formed by age 20. **(Espinosa et al., 2010; Jaffe & Laitman, 1982)** They follow fairly standard types and may cause significant problems or be unnoticed.

The accessory navicular bone can give the appearance of a flat foot. **(Hamilton, 1985)** There are three types of accessory navicular bones. **(Coskun et al., 2009; Sgarlato et al., 1969)** Type 1 is a sesamoid in the tibialis posterior tendon. Approximately 30% of accessory naviculars are this type. Type 2 accessory naviculars are united to the navicular by a cartilaginous synchondrosis measuring 1-3 mm. This is the type that creates the major problem of accessory naviculars. Type 3 is an accessory navicular united to the parent navicular by a bony ridge, producing a cornuate navicular.

Another common accessory bone is the ununited lateral tuberosity of the posterior aspect of the talus, which is called the os trigonum tarsi. It is present in 3-8% of the population. It sometimes remains attached to the talus as the trigonal process. It is rarely symptomatic, but it can cause symptoms with certain types of stress.

The foot and the ankle together possess some thirty-three joints. The bones and joints provide a solid foundation and leverage for the muscles to move the body. More than one hundred ligaments are part of the foot-ankle system and they stabilize and help the body sense the position and functional state of the bones of the foot during movement.

Foot Mechanics – The Close Examination

The American Medical Athletic Association reports that every year 37 to 50 percent of runners suffer injuries severe enough to reduce or stop their training or cause them to seek medical care. **(Wilk et al., 2009)** With about 36 million runners in the United States, this means that 14 to 18 million runners are getting hurt every year.

Abshire, Maffetone, and Robbins and Hanna **(Abshire, 2010; Maffetone, 2003; Robbins & Hanna, 1987)** view the foot with a different perspective than most. Rather than being a functional unit that adapts poorly to the stresses put upon it and needs arch supports and supportive shoes to prevent injury, they find the properly conditioned bare foot is more injury resistant. Their investigation originated from recognizing that barefoot runners in international competitions have minimal running–related injuries. They developed the hypothesis that the bare foot contacting uneven surfaces of the substrate is stimulated to react with the shock absorbing mechanism of the arches and muscular action of the foot. The feet of a shoe–wearing population are insulated from the uneven stimulating surface of the substrate, causing an impaired somatosensory feedback.

To test this hypothesis, Robbins and Hanna compared a shoe–wearing group — who shifted to a minimum of one hour per day of barefoot activity, and were encouraged to do their running and walking in this manner — with a control group who continued wearing shoes. In the barefoot group there was a significant ($p<0.05$) shortening of the median longitudinal arch, indicating an elevation of that structure. They attribute this to activation of the muscles by increased somatosensory feedback from foot contact with the substrate. The foot, along with the hand, has "…an extremely high density of neuroreceptors which respond to small discrete displacements, directionally applied force (shearing force), and low intensity repetitive force (vibration). These mechanoreceptors have a relatively low threshold. There is another group of receptors that respond to similar forces, but with such high thresholds that they only respond to mechanical stimuli intense enough to induce tissue damage

(nociceptors). These two groups of receptors are responsible for the perception of pressure and pain.

"The data suggest that, in our subjects, their normal footwear prior to the experiment did not produce the sensation necessary to induce protective adaptations inherent with barefoot weight–bearing activity. It is obvious that footwear with soft flexible polymer foam and a relatively inflexible rubber sole does not allow discrete skin deflections from irregular surfaces. Foam, in addition, has vibration–dampening properties. It has been elegantly demonstrated that there is diminished shearing force in the barefoot state. **(Pollard, 1983)** The shearing force which is applied by the plantar skin when barefoot is transferred to the shoe lacings and counter of the shoes when shod."

Robbins and Hanna **(Robbins & Hanna, 1987)** give the example of almost complete joint obliteration in Charcot joints because of damage resulting from lack of somatosensory feedback as a result of neurological damage of tertiary syphilis. "The runner, like the neuropathic syphilitic, damages his or her lower extremities due to lack of somatosensory feedback–mediated protective behavior."

"The arch support, which is present in all running footwear, would interfere with the downward deflection of the medial arch on loading. Furthermore, the use of orthotics, or structures that are fitted to the mould of the soft tissues of the foot, could cause similar difficulty. Such designs occur when an engineer looks at the foot as an inflexible lever that is delicate and thus requires packaging. Various myths persist about foot behavior due to poor understanding of its biology." **(Robbins & Hanna, 1987)** It is now accepted that proprioceptive awareness of movement in the ankle is heightened when the person is barefoot than when wearing shoes. **(Waddington & Adams, 2003)** Sensory information coming from proprioceptors in the muscles and joints and from the plantar surface of the foot is essential for postural control.

A growing body of research on "functional footwear" has occurred simultaneously with the development of new athletic shoes such as Vibram Fivefingers, Masai Barefoot Technology, FitFlop, Nike Free's, Vivo Barefoot and Newton among others. This research has shown that when shod and unshod populations are compared, unshod populations have a lower prevalence for many of the most common running injuries including ankle sprain, plantar fasciitis, iliotibial band syndrome, patellar pain, back pain, shin splints, and Achilles tendinopathy, **(Warburton, 2001)** and the "functional footwear" populations have better arch development **(Mauch et al., 2008; Rao & Joseph, 1992).**

Researchers have found impairments in muscle strength, endurance, and activation times in the hip abductors for subjects with ankle instability and injury. **(Zampagni et al., 2009; Beckman & Buchanan, 1995; Bullock-Saxton et al., 1994; Nicholas et al., 1976)** Functional limitations and muscle weaknesses are not uncommon for patients with a history of chronic ankle sprains, and up to 70% of individuals involved in basketball with an initial ankle sprain could suffer from injury recurrence. **(Yeung et al., 1994)** Two studies revealed that the gluteus maximus muscle was significantly delayed in activation in subjects following an ankle sprain injury compared to control groups. **(Bullock-Saxton, 1994; Bullock-Saxton et al., 1994)**

A major reason that the manual muscle test for the muscles of the foot and ankle should be added to the standard diagnostic methods used and taught in the manipulative professions, including podiatry, is that patients with foot and ankle disorders demonstrate joint instability, ligament strain, and muscle inhibition. **(Maffetone, 2010; Logan, 1995; Yeung et al., 1994)** Applied kinesiology MMT adds significant information to foot and ankle examination because it detects and specifically treats these muscle impairments that either cause or perpetuate extremity and whole body dysfunctions. **(Goodheart, 1998)**

The foot evolved some 270 million years ago whereas shoes have been on human feet for a few thousand years at most. **(Haines, 2000)** Foot dysfunction and its relation to total body function are better understood today, and effective methods of examination have been developed, within applied kinesiology and without **(Maffetone, 2010; Logan, 1995)** Bahler **(Bahler, 1986)** cites a quotation by Georg Hohmann, "…the old master of orthopaedics," from his book *Fuss und Bein*. "The human foot is one of nature's works of art and as such, it has not yet been fully recognized and explained. It will require a great deal of scientific investigation before this structure is fully understood." Partially answering this need is the use of computed tomography and magnetic imaging to diagnose pathology and trauma in the foot. **(Tourne et al., 2010; Sartoris & Resnick, 1987)** Far less attention has been given to functional aspects that influence performance. The relationship of the foot to remote body function is still understood by too few in general health care. For the most part doctors in every field are inadequately prepared in the anatomy and physiology of the foot; consequently, they tend to overlook it. This accentuates the basic fact that we cannot recognize what we do not know. Increased attention by surgeons has been directed to athletes' feet. Sammarco **(Sammarco, 1986)** wrote, "Unfortunately [foot and ankle injuries] were often relegated to the trainer or paraprofessional since such conditions and injuries were felt to compromise performance level only to a minor degree. As more information has become available, however, it is obvious that such injuries tend to affect the performance much more than expected."

Another notable recognition of the foot's importance to total body organization is by Dananberg **(Dananberg, 2007)** and Bordelon. **(Bordelon, 1987)** Dananberg notes that a specific condition of the foot (*functional hallux limitus*, discussed later), "because of its asymptomatic nature and remote location, has hidden itself as an etiological source of postural degeneration. Functional hallux limitus is a unifying concept in understanding the relationship between foot mechanics and postural form. Identifying and treating this can have a profound influence on the chronic lower back pain patient." Bordelon notes that "since the function of the foot is to set the stage for the entire gait pattern and since the pattern of gait and the function of the foot are what set the stage for any activity of bipedalism whether it be walking, running, or jumping and since the entire function of the ankle, hip, knee, and back depends upon the shape of the foot, its function, and its position, perhaps we should consider the shape and function of the foot in evaluating any abnormality of the lower extremity and back." In applied kinesiology we could add: Consider the function of the foot in any health problem.

Goodheart, Walther, Chaitow and DeLany, Liebenson, Steindler **(Goodheart, 1998-1964; Walther, 2000; Chaitow & DeLany, 2002; Liebenson, 2007; Steindler, 1955)** and many others have written extensively on the closed kinematic chain of the body. When the foot is in contact with the ground, the foot, leg, thigh, and pelvis make up a modified closed kinematic chain. Imbalance in any part of the chain will cause change in function of the remote portions of the chain; thus extended pronation puts torsion into the leg, thigh, and pelvis, which would not ordinarily be present. Because foot malfunctions lead to instability during gait, compensation patterns emerge that have body-wide implications. Simons et al **(Simons et al., 1999)** report that a cascade of myofascial conditions are likely to emerge in the patient with disturbances in foot structure and function, including pain in the low back, thigh, knee, and foot.

The manual muscle test as used in AK makes the diagnosis of these specific interactions between the joints and muscles of the foot and remote structures and muscles throughout the body far easier. The ***visual diagnosis*** of a specific joint or muscle impairment in the foot and simultaneously its relationship to a specific joint or muscle impairment in the hip, shoulder, neck or jaw is difficult. **(Lederman, 2010)** The different elements within the chain of events occurring during any particular movement that a patient exhibits in front of the examiner occur within a fraction of a second; far too rapidly to be accessed individually in the absence of laboratory tools. Therefore, what is actually observed by the examiner who depends upon visual diagnosis of these muscle-joint interactions is the grand total of how rapidly and smoothly a person's global posture can change between two activities – it is almost impossible to make a diagnosis of a specific muscle or joint dysfunction on this basis.

Visual diagnosis of specific joint-muscle interactions is very difficult.

The AK manual muscle test permits a specific challenge to a specific muscle or joint in the foot to be immediately followed by another specific test to a distant joint or muscle, thereby making evident to both the physician and the patient the dynamic interactions going on between two distant structures. Additionally if a specific dysfunction is suspected by visualization or palpation, then the AK sensorimotor challenge to this articulation or muscle can be tested in relationship to the remote muscle dysfunction.

Many think of the sacrum as the foundation of the spine, but as Gillet and Liekens **(Gillet & Liekens, 1981)** point out the ischia are the base when sitting and the feet when standing. We see that many in the chiropractic profession's history have emphasized the foot's role in spinal function. **(Keating, 2002; Greenawalt, 1980)** In 1954 Janse **(Janse, 1976)** recognized faulty body mechanics from serial distortions as he described the possible scenario resulting from foot pronation or "falling of the medial longitudinal arch." As the foot pronates the talus rotates, carrying the tibia into medial rotation that extends up to the femur, moving the greater trochanter anteriorly and laterally and stretching the piriformis by a windlass mechanism. Because of improper piriformis support, a subluxation of the sacrum may result in an anterior and inferior position. To compensate, the gluteus maximus muscle contracts to resist the forward and downward disposition of the pelvis. Secondary to the gluteus maximus contraction, the innominates become subluxated. As a result of the sacral anterior inferior subluxation, the 5th lumbar is "mobile" and, according to Lovett's Law, will gravitate and rotate toward that side; thus the beginning of structural scoliosis is established. This has been a synoptic review of a description originally presented by Janse in the May, 1954, issue of the *National Chiropractic Association Journal.* Other early descriptions of spinal problems resulting from the feet include hyperlordosis **(Harrison, 1964)** and sciatica. **(Goodheart, 1967)**

Lewin **(Lewin, 1959)** states, "Flat feet and a weak back are often found in the same child and adult." Patients will sometimes lose a vertebral subluxation correction as soon as they walk, often before they reach the reception area to pay for the office visit.

The importance of foot function on the spinal column is shown in a study (n=72) by Ceffa et al. **(Ceffa, 1982)** in Italy. The feet and spine were studied with thermography on individuals who complained of back pain and were under chiropractic care. In this study, 12 had flatfeet, 41 "hollow" feet, and 47 load alterations revealed by thermography. The study was instituted because of "…significant reoccurrence, after a successful manipulative treatment, of muscular

pains and defense contractures, in the same area or in other segments of the spine…." The patients were treated with orthotics and examined several times and at various time intervals. The imbalanced thermographic patterns in the lumbar and thoracic regions were balanced or greatly improved, as was the thermographic pattern of the feet. Comparison of the subjective and objective results of this study showed 21 patients pain-free with normalized thermographic pictures; 34 had been pain-free over one year, with significant improvement in the thermographic pictures; 14 had less frequent and less severe pain, with improved thermographic pictures; and 3 had no improvement, with no improvement of the thermographic pictures.

In 1954 Janse **(Janse, 1976)** summarized the body's structural integration and the mid-20th-century chiropractic conceptual approach by stating "…faulty body mechanics is usually a consequence of a serial distortion rather than a single local lesion. We mean thereby that the problem of postural and mechanical pathology is the result of distortions that may begin in the foot or feet, extend up into the leg or legs, then into the knee or knees. From there it may ascend into the hips and sacroiliacs, reflecting onto the vulnerable lumbosacral articulations and eventually the spine to the occiput."

Early in applied kinesiology, Goodheart **(Goodheart, 1967)** associated psoas muscle dysfunction and sciatic neuralgia with excessive foot pronation. He dramatically demonstrated the immediate effects of improving body function and reducing pain by eliminating adverse stimulation to the foot proprioceptors by simply having the patient stand on the lateral borders of his feet to take the strain off the medial longitudinal arch. This can easily be demonstrated when some muscles test weak when standing but are strong when the patient is supine or prone. The muscles will immediately test strong when the patient stands on the foot's lateral border, and weaken with usual weight bearing. Foot dysfunction is not the only reason muscles may become weak when standing, but it is the most frequent cause.

Monte Greenawalt, the founder of the orthotics company Foot Levelers that has been a staple within the chiropractic profession for more than five decades, quoted Goodheart's research on the feet regularly in his books and published research papers. **(Greenawalt, 1980)**

An individual who has had a hyperextension/hyperflexion cervical sprain/strain, or the so-called "whiplash" accident, may have their condition complicated by extended foot pronation. This, of course, is probably not part of the original traumatic condition; however, it can be a perpetuating factor of the cervical problem. This results from the role the foot proprioceptors play during walking on the facilitation and inhibition of the head rotating muscles, such as the sternocleidomastoid. **(Meyer et al., 2004)** Because of this disorganization, the insertion of these muscles at the mastoid processes may be exquisitely tender. Travell & Simons **(Travell & Simons, 1992)** have noted that myofascial trigger points from as far away as the soleus muscle can refer pain and muscular dysfunction into the temporomandibular region. Simply having the patient stand on the lateral borders of his feet for one or two minutes often reduces or eliminates the pain at the mastoid processes.

Because of the intricate connections in the neuromuscular system, a change in any part of the body can disturb function elsewhere in a distant part. An example of this would be that an anterior cruciate ligament injury has been shown to generate changes in the posterior cervical, upper and lower trapezius, sternocleidomastoid, anterior temporalis and masseter muscles. **(Tecco et al., 2006)** There is no easy way to visually assess the specific muscle impairments seen in the neck after a knee injury; but the use of the AK challenge test to the knee, followed immediately by specific AK MMT to the muscles of the neck, can make each of these specific cervical muscle impairments produced by an anterior cruciate ligament injury evident to the examiner. **(Sprieser, 2003; Duffy, 1999; Schmidhofer, 1997; Zatkin, 1990; Raffelock, 1987)**

Another area that might be influenced by foot dysfunction is the stomatognathic system. **(Cuccia, 2011)** Cranial faults can be created or perpetuated by disorganized function of the sternocleidomastoid muscles as they pull on the mastoid processes during gait, **(Walther, 1983)** or by the fascial pull that may develop with postural sagging. This is a primary cause of neurologic disorganization because of the stomatognathic system's close integration with the equilibrium proprioceptors. Hicks notes that talocalcaneonavicular restriction reduces our ability to stand on one foot. **(Hicks, 1953)** Richie **(Richie, 2001)** notes that balance and postural control of the ankle appear to be diminished after a lateral ankle sprain and this can be restored through treatment; this improvement in balance is mediated through central nervous system mechanisms. One might question what is major; does the inflexibility of the foot or the improper stimulation of the foot proprioceptors contribute more to the body's inability to maintain proper orientation in space?

Unilateral hyperpronation and the resultant "short leg" causing pelvic obliquity have been mentioned as factors in scoliosis development. **(Vleeming et al., 1997; Cailliet, 1997; Silverman, 1986; Botte, 1981; Janse, 1976)** Possibly a greater contribution to the problem is the foot–related neurologic disorganization found by applied kinesiology examination. A major component of almost every idiopathic scoliosis case is neurologic disorganization. **(Walther, 2000)**

Dananberg has shown that symptoms associated with foot dysfunction include low back pain, tibialis posterior dysfunction, and anterior knee pain. **(Dananberg, 2007)** Dananberg and others have also shown that functional hallux limitus **(*FHL*, discussed later)** to be a remote, often hidden source of postural degeneration and pain. Functional hallux limitus involves limitation in dorsiflexion of the 1st metatarsal-phalangeal joint during walking, despite normal function of this joint when non-weight bearing. Mattson, Ferrari, and Dananberg have each shown that improvements in foot function, mobility and strength resulted in marked improvements for patients with chronic nonspecific low back pain. **(Mattson, 2008; Ferrari, 2007; Dananberg & Guiliano, 1999)**

The work of Gracovetsky **(Gracovetsky, 1989)** (the spinal engine theory), developed in the mid-1980s (and published in 1989 in his book of the same name), proposes that the spine optimizes its efficiency of motion in the

gravitational field by using the spine to propel the legs forward by capturing the ground reaction force to decouple the spinal segments with each step of the gait cycle. Storing the ground reaction force as potential energy (similar to the Hicks windlass mechanism which stores elastic energy through the plantar fascia during the swing phase of gait) in the viscoelastic tissues of the lower extremity and spine, and then expressing that potential energy as kinetic energy as the spine propels forward. Disturbances then in the feet may alter the ground reaction force described by Gracovetsky and thereby impair the force transmission potentials of the spinal engine through the rest of the body.

In addition to foot dysfunction adversely affecting spinal function, the converse is applicable. Interplay between the spine and feet are demonstrated by Gillet and Liekens. **(Gillet & Liekens, 1981)** They examined the spine but made no corrections to it; only foot corrections were made. "In 86% of the cases, there were varying changes (small to important) in the spine…either immediately or slowly." In another portion of the study, the height of the arch was periodically measured while spinal corrections were administered. "In 37% of these cases there was definite change in the feet as the spinal fixations were eliminated."

General association may be made between different areas of the foot with levels of the spine. Gillet and Liekens **(Gillet & Liekens, 1981)** observed that the toes relate with the upper cervical vertebrae and the metatarsals to the rest of the cervical spine, while the joints between the tarsals and metatarsals correspond to the dorsal spine. Intrametatarsal fixations relate with fixations between the ribs and the spine, and between the ribs themselves. Fixations in the midfoot — that is, cuneiform–navicular and cuboid with the calcaneus — are associated with the lumbar region.

In an applied kinesiology practice, most foot correction is for chronic problems. When acute trauma is present, one must not only provide the proper type of treatment for the local injury, but also be aware of remote conditions that might develop. The effects of trauma to the foot may cause the afferent nerve supply to cause disorganization, adversely affecting remote areas as well as the problems that develop with disuse during rehabilitation. Nicholas and Marino **(Nicholas & Marino, 1987)** state, "*…the more distal the injury site, the greater the total weakness of the affected limb.* Thus, distal injuries produce more weakness to the entire limb than do proximal ones." When foot and ankle trauma causes continued problems, there is significant weakness of the hip adductors and abductors when compared to the uninvolved contralateral side. "Immobilization of the foot and ankle triggers a cascade of negative effects on skeletal muscle tissue, including muscle atrophy, adaptive shortening of muscle, and periarticular tissue (contracture), a decrease in muscle activity through a diminished motor discharge frequency, and/or recruitment and synchronization, a shift from a tonic to a more phasic pattern of firing, loss of myofibrillar and sarcomal protein, and diminished blood flow. It is well established that muscles atrophy when not used either because of immobilization or disuse."

Rothbart and Estabrook **(Rothbart & Estabrook, 1988)** found a high correlation between excessive pronation, static pelvic abnormalities, and chondromalacia patellae, with 96% of the patients (n=97) in their study showing excessive pronation and low back pain. Treatment was based on a combination of chiropractic and podiatric therapy with a 6-month follow-up. Analysis of the success in this tandem approach was promising.

Rothbart and Estabrook suggest a model that asymmetrical pronation patterns (one arch dropping more than the other) initiates a forward and downward rotation within the sacroiliac joint. Entrapment of the sciatic nerve then occurs between the piriformis muscle and sacrospinous ligament. They suggest that paresis is then observed clinically with weakness, numbness, and eventually paralysis of the affected limb. They also proposed a model for chondromalacia explaining the pathomechanical events associated with oblique tracking patellar syndrome. They suggest that excessive pronation is the causative factor directing asynchronous rotation between the shin and femur. This forces the patella out of its normal tracking groove, which in turn generates erosion between the inferior margin of the patella and femoral epicondyles. In patients with symptomatic and asymptomatic patellar pain, assessment for muscle strength impairments will be essential to restoring the normal "track" or "path" of the patella through any particular movement. Any aberrations in the recruitment or coordination of these sequential movements of the patella (generated by the muscles crossing the knee) will be signaled to the central nervous system. In patients with knee or patella pain, the CNS will seek to inhibit this inappropriate movement by weakness, stiffness or pain. **(Kapandji, 2010)** This reorganization of movement control is a protective strategy which serves to alleviate some of the stresses imposed on the damaged tissues of the knee.

When the feet are subjected to a foundational change, such as in heel height, muscular balance is changed by the nervous system. With the negative heel of the "earth shoe," activity in the erector spinae and soleus decreases, while it increases in the tibialis anterior, rectus femoris and biceps femoris muscles. **(Soderberg & Staves, 1977)** Barefoot running and running in "functional footwear" like Vibram Fivefinger shoes results in more of midfoot or forefoot strike, whereas running in traditional shoes results in more heel strike. **(Squadrone & Gallozzi, 2009)** In the first month of running with Vibram Fivefingers the author (SC) discovered more 2nd metatarsal head pain than when running in normal gym shoes. However as Squadrone & Gallozzi note, barefoot running creates a pressure loading spike around the 2nd metatarsal head; almost identical to running in Vibram Fivefingers. High heels increase the activity of the gastrocnemius and peroneus longus muscles during quiet standing. **(Csapo et al., 2010; Basmajian & Bentzon, 1954)** Normally there are only bursts of muscle activity seen on electromyography to maintain balance. This activity change indicates a failure to conserve energy, probably resulting in excessive fatigue over that which would ordinarily develop during quiet standing.

Applied kinesiology uses numerous reflexes that effectively improve muscle function when the reflex is active and therapeutically stimulated. It has long been observed that reflex stimulation results are not long–lasting if there is a primary factor causing the weakness. For example, if foot dysfunction is causing disorganization to the sternocleidomastoid muscles, as previously mentioned, they will usually test weak with manual muscle testing. The

neurolymphatic reflexes for the muscles will probably be active; stimulating the reflexes will usually improve the muscles' function, providing a strong test. The primary effect of foot dysfunction is evidenced by the muscles again testing weak immediately after the patient walks. The effect of muscle dysfunction causing active associated reflexes may also be seen in neurovascular reflexes and stress receptors becoming active secondary to muscle dysfunction from another cause. When the foot is properly corrected, the active secondary reflexes will usually become inactive spontaneously. Applied kinesiology techniques are seldom employed in isolation but as part of a process designed to restore maximal pain free movement of articulations, restoration of postural balance, systemic functionality and facilitation of the self-regulatory mechanisms of the body.

One reason the cause of a patient's musculoskeletal pain is so enigmatic is that an adequate examination to cover the most common causes of neuromusculoskeletal pain requires skills characteristic of as many as 5 disciplines. The clinician may be required to examine for muscle imbalance in the kinesiological sense, neurologic function, myofascial trigger points, somatization in the mind-body sense, and articular dysfunction. Such a complete examination is indicated for patients with chronic musculoskeletal pain who have seen many specialists without finding a satisfactory answer to the cause of their pain.

The examination for functional muscle imbalance is part of the applied kinesiologist's training. The examination detects weak muscles, inhibited muscles, compensatory movement patterns, antalgic movement patterns and muscles recruited in an abnormal sequence. **(Walther, 2000)** The manual muscle test and AK diagnostic tests help to identify which muscle or muscles are dysfunctional and where this trouble is coming from. The weakness of muscles in the distribution of the motor nerve must by distinguished from the dysfunctional patterns of weakness induced by micro-evulsions, enthesopathies, myofascial trigger points, fear of movement, acupuncture meridian problems, cranial-sacral problems and other strains related to functional muscle groups, regardless of innervation. An effective examination requires the development of adequate manual muscle testing skills so that specific muscles can be isolated during the testing and knowledge of every muscle's origin and insertion and myofascial attachments, anatomy and physiology. The details of this examination also vary from muscle to muscle and are not yet routinely taught in many chiropractic and medical training programs. With the development and expansion of applied kinesiology, more and more chiropractors, physical therapists, some physiatrists, osteopathic physicians, dentists, and medical practitioners of other clinical specialties have subsequently learned these important skills. **(ICAK, 2012)**

It appears that there are considerable neurologic ramifications to foot dysfunction that are not charted in the scientific literature. Science scoffs at foot reflexology, yet numerous books have been written on the subject and there are thousands who attest to its effectiveness. It is not our purpose to defend or question the effectiveness or mechanism of the method. It is of interest to observe some parallels that exist between the muscle-organ/gland associations of applied kinesiology and foot reflexology. The most common reflexes indicated in reflexology books for the sinuses are at the tips of the toes and the metatarsal heads. **(Kunz & Kunz, 2005; Berkson, 1977; Carter, 1969)**

Stimulating these points affects the sternocleidomastoid muscle test, which is the muscle applied kinesiology associates with the sinuses. If the muscles test weak, the stimulation will usually strengthen the muscle, at least temporarily. If the muscles test strong in the clear, the reflex stimulation will temporarily weaken the ipsilateral sternocleidomastoid muscle in the normally functioning individual. The ipsilateral sternocleidomastoid muscle is the one normally inhibited during the last portion of the stance phase of gait.

A lateral cuboid subluxation has been associated with tensor fascia lata weakness. The neurologic reason for the muscle inhibition is discussed later under foot reflexes and reactions, and has recently been investigated by Zampagni et al. **(Zampagni et al., 2009)** showing that the applied kinesiology shock absorber test to the foot produces specific and measurable inhibition of the tensor fascia lata muscle. For now it is interesting to observe another correlation between foot reflexology and applied kinesiology. The colon reflex is established in foot reflexology over the cuboid bone; applied kinesiology finds the colon to be associated with the tensor fascia lata muscle. The same type of normal and abnormal relationship for the sternocleidomastoid muscle, foot reflex, and sinuses is applicable for the colon.

There is also a relationship between the Gillet and Liekens' **(Gillet & Liekens, 1981)** spine–foot association, mentioned earlier, and foot reflexology.

Foot reflexology is an area needing thorough physiologic investigation. **(Figure next page)** How accurate foot reflexology is with regard to actual health problems is unknown. Foot and hand massage is useful as an effective nursing intervention in controlling postoperative pain, **(Degirmen et al., 2010)** and foot reflex stimulation is beneficial for insomnia. **(Gong et al., 2009)** It is observed that the reflexes will show positive therapy localization when associated with muscle weakness; their stimulation changes muscle function, at least temporarily. Usually there is some underlying foot dysfunction that needs to be corrected, or the positive reflex returns shortly after the patient walks. It may be that foot reflexologists are, by good luck, sometimes correcting foot subluxations, fixations, or muscle dysfunction and obtaining permanent corrections.

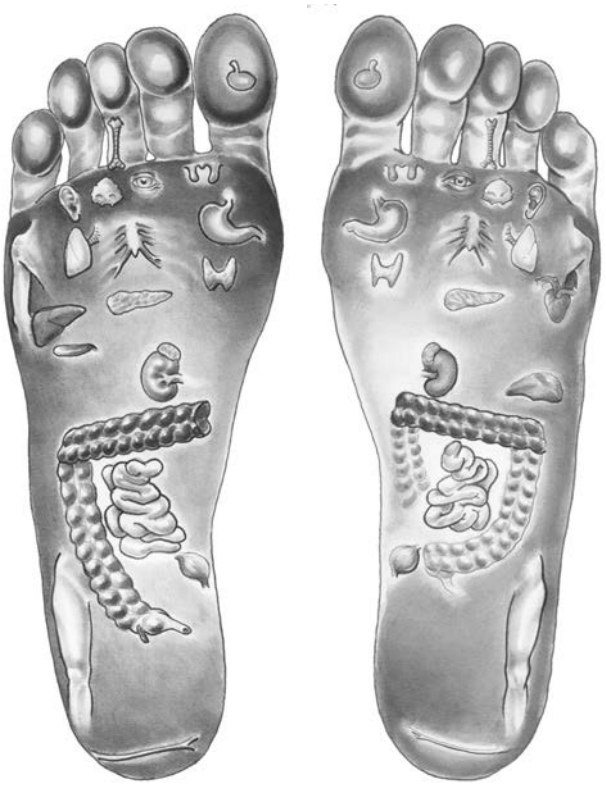
There are remote orthopedic conditions caused by foot dysfunction, many of which are generally recognized. Entrapment neuropathies at the tarsal tunnel can cause sciatic pain. Knee problems are commonly caused by extended foot pronation and other foot dysfunction. Reactive muscle weakness, caused by one of the foot muscles, can be responsible for knee, back, neck, or shoulder pain. **(Marshall et al., 2009; Prior, 1999; Rothbart & Estabrook, 1988)**

With today's knowledge we see that foot dysfunction can influence the total body more than just the spine and its nervous system relationship, as described by Janse **(Janse, 1976)** and many others. **(Goodheart, 1998; Walther 2000; Dananberg, 2007; Liebenson, 2007)** Some of the health problems caused by foot dysfunction are well described in the scientific literature; others, such as interfering with the energy flow over the meridian system or causing remote neurolymphatic reflex activity, are not. To put the possible ramifications into proper perspective requires a wide area of interest in the cause of health problems. When first studying

this integration, it can certainly be puzzling. Sometimes leading to the confusion is disorganization from improper stimulation to proprioceptors in the foot and leg as a result of extended pronation or some other dysfunction. This may send signals not in keeping with the body's needs; response to the afferent system causes confusion within the nervous system — a neurologic disorganization or, as has been known in applied kinesiology, "switching." In any event, the primary cause of many health problems that originate in the feet is overlooked by doctors, resulting in symptoms being treated rather than the primary cause.

Foot function and its effect on remote structure and function is an excellent example of the body's integration, and how the entire body must be evaluated in order to find the basic underlying cause for many health problems. The major way the foot influences other areas is by way of the nervous system. Proprioceptors in the foot and ankle send organizing impulses throughout the body. The role of these functional sensory receptors is to inform the central nervous system about ground reaction forces when the body stands and moves. When observing the different types of reflexes within the foot, it becomes apparent that orthopedic conditions in the form of stress and strain develop as a result of improper foot function. **(Cuccia, 2011; Abshire, 2010; Maffetone, 2003; Chaitow & DeLany, 2002; Hill, 1995; Robbins & Hanna, 1987)** Additionally the foot may be suffering an adaptive response to pathomechanical alterations in other parts of the body, including the stomatognathic and oculomotor systems. **(Berthoz, 2000)** Rothbart has demonstrated that foot pronation causes problems in the knees. **(Rothbart & Estabrook, 1988)** Rothbart **(Rothbart, 2008)** also demonstrates a fascinating correlation between foot pronation, innominate rotation and vertical facial dimensions, theorizing an ascending foot-pelvic-cranial model to explain these findings.

> "Abnormal foot pronation (inward, forward, and downward rotation) displaces the innominates anteriorly (forward) and downward, with the more anteriorly rotated innominate corresponding to the more pronated foot; 2) anterior rotation of the innominates draws the temporal bones into anterior (internal) rotation, with the more anteriorly rotated temporal bone being ipsilateral to the more anteriorly rotated innominate bone; 3) the more anteriorly rotated temporal bone is linked to an ipsilateral inferior cant of the sphenoid and superior cant of the maxilla, resulting in a relative loss of vertical facial dimensions; and 4) the relative loss of vertical facial dimensions is on the same side as the more pronated foot." **(Rothbart, 2008)**



Foot reflexes.

Because of the wide-ranging effects of foot dysfunction on the total body, it is important to consider its possibility early in an examination. It is generally estimated that at least 90% of our population has significant foot biomechanical variation from the ideal; **(American**

FOOT	GILLET & LIEKENS	FOOT RELEXOLOGY
Toes	Upper cervical	Cervicals
Metatarsals	Rest of cervicals	Cervicals
Tarsometatarsal	Thoracic spine	Thoracic spine
Intrametatarsal	Rib-spine fixation & fixation between ribs	
Midfoot fixation	Lumbar spine	Lumbar spine
Chopart's joint	Lumbar spine	Sacrum
Calcaneus		Coccyx & sacrum

Foot reflexes (based on empirical reports).

Podiatric Medical Association, 2010; Langer, 2007; Subotnick, 1991, 1975) and approximately 25,000 people sprain an ankle every day in the United States. A study in Spain found that less than 3% of those over sixty had "normal" feet. **(Langer, 2007)** The majority can be understood and improved by applied kinesiology examination and treatment. When there is dysfunction, especially when it adversely affects the nervous system, "Take out the foot factor first." **(Dananberg, 2007; Maffetone, 2003; Aronow & Solomone-Aronow, 1985)**

As with all other body structure, a physician must have a thorough knowledge of foot anatomy and function in order to properly examine and treat many problems. Sometimes it is necessary to use orthotic support to the feet, but as more knowledge is obtained in applied kinesiology, the frequency and length of use are getting shorter and overall functional correction is getting better. It is hoped the information presented here will arouse interest in the study of foot anatomy and physiology to better understand problems affecting the feet. Foot dysfunction can literally cause health problems throughout the body.

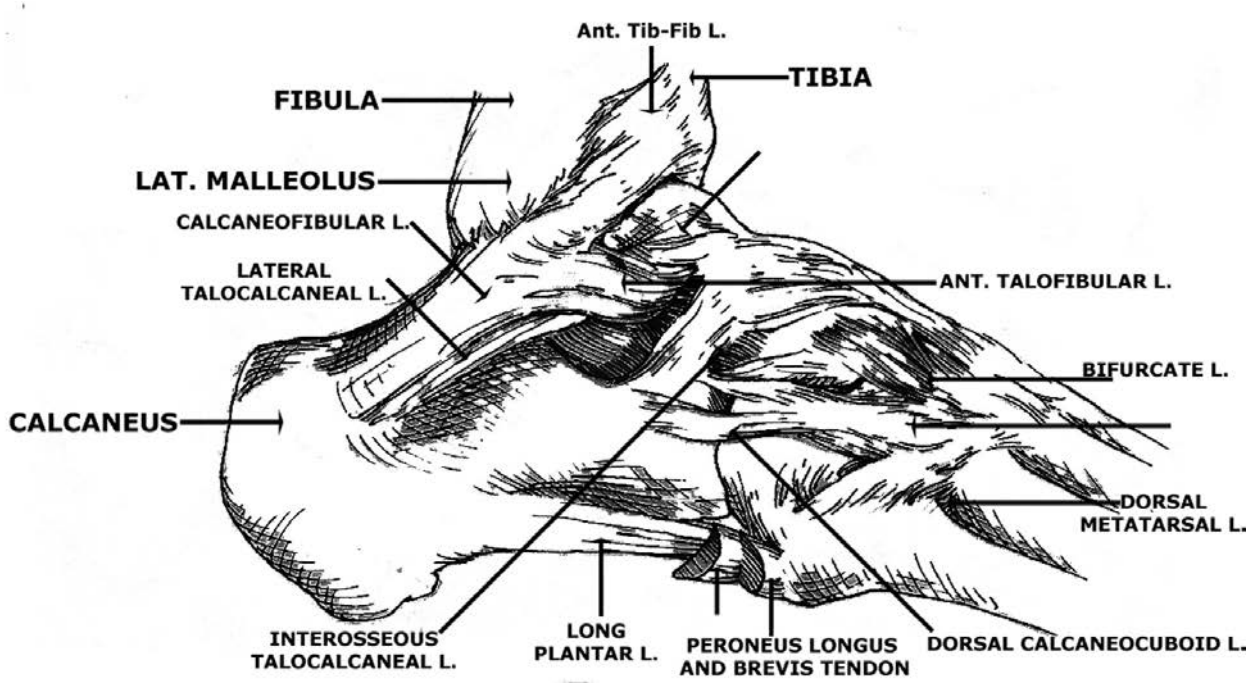
Ligaments of the Foot

Ligaments are far from merely being restrictive structures that are strategically placed to support and stabilize joints, while maintaining normal tracking during movement. In reality they are sensory organs that provide proprioceptive input to the CNS, as well as having vital reflexive influences on associated muscles, and thereby ligaments become major elements in the stabilization of joints.

It was suggested as far back as the turn of the century that ligamento-muscular reflexes exist from sensory receptors in ligaments to muscles that modify the load imposed on the ligament and joint. Goodheart first discussed the law of the ligaments in 1973. (**Goodheart, 1973)** Goodheart found that pressure applied to the ends of ligaments towards the belly of the ligament tightens it. The opposite force will elongate the ligament.

It has been shown that ligamento-muscular reflexes exist in most of the lower extremity joints. **(Solomonow et al., 1998; Freeman & Wyke, 1967)** The ligaments associated with each extremity are richly endowed with afferents that produce reflex activation of the many muscles associated with the extremity's movement. The muscles therefore are a major component in maintaining the stability of the extremity's ligaments, bursae and capsules. **(Solomonow et al., 2009, 1987)** Goodheart also suggested that when there is a chronically weak muscle there will usually be a ligament involvement (the ligament is stretched) that provides stability in the same direction as the muscle. Conversely, a stretched ligament will cause a weakness in a muscle that provides stability in the same direction. **(Solomonow et al., 1987)** The usefulness of the manual muscle test and therapy localization and challenge procedures makes the diagnosis of specific ligament involvement (and even the specific portion of the injured ligament) obvious. For foot and ankle dysfunctions, detection of ligament injuries through these ligamento-muscular reflexes can be specifically assessed with the MMT. **(Leaf, 2010; Lever, 2007; Sprieser, 2003; Kharrazian, 2001)**

Numerous ligaments tie the bones of the foot together. They are extensive and vary somewhat from individual to individual. Nomenclature for the ligaments varies from authority to authority. Emphasis here will be primarily on the massive ligaments of the arch.



Lateral foot and ankle ligaments

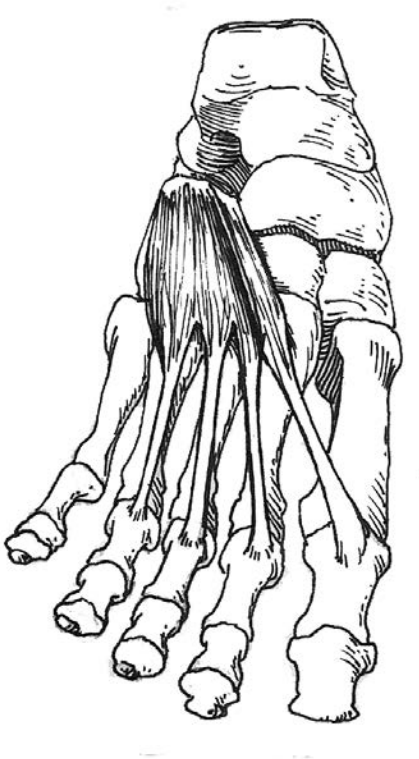
The posterior talocalcaneal articulation has an articular capsule and anterior, posterior, lateral, medial, and interosseous talocalcaneal ligaments. The lateral talocalcaneal ligament attaches, as its name infers, from the lateral process of the talus to the lateral surface of the calcaneus. The medial talocalcaneal ligament connects the medial tubercle of the talus with the posterior sustentaculum tali. It blends with the deltoid ligament, discussed later under the ankle joint. The anterior talocalcaneal ligament connects the anterior and lateral surfaces of the neck of the talus to the superior surface of the calcaneus. The posterior talocalcaneal ligament is short; it connects the lateral tubercle of the talus with the calcaneus.

The major connection between the talus and calcaneus is the interosseous talocalcaneal ligament. It lies somewhat within the sinus tarsi, binding the calcaneus and talus firmly together.

The talocalcaneonavicular articulation often requires adjustment in an applied kinesiology examination. The major portion of the articulation is the head of the talus into the concavity of the posterior surface of the navicular. Contribution to the articulation is made by the anterior articular surface of the calcaneus. The joint has an articular capsule that thickens posteriorly, joining with the interosseous ligament of the talocalcaneal articulation. The talonavicular ligament connects the two bones dorsally.

Five ligaments support the calcaneocuboid articulation: the articular capsule, the calcaneocuboid portion of the bifurcated ligament, the long plantar, and the dorsal and plantar calcaneocuboid ligaments. The bifurcated ligament attaches to the calcaneus on the anterior surface. It divides to attach to the navicular and cuboid bones. The long plantar ligament is the longest of the tarsal ligaments, attaching to the plantar surface of the calcaneus anterior to the tuberosity. Its deep fibers attach to the posterior portion of the plantar surface of the cuboid bone. The superficial fibers are longer than the deep fibers and attach to the bases of the 3rd, 4th, and 5th — and occasionally 2nd — metatarsal bones. The plantar calcaneocuboid ligament is deep to the long plantar ligament. It is short but very strong in its connection between the calcaneus and the cuboid bones. These ligaments are very important in maintaining the lateral longitudinal arch.

Support to the medial longitudinal arch is provided by the plantar calcaneonavicular ligament, sometimes called the "spring" ligament, although there is usually no actual articulation between the calcaneus and the navicular bones. The plantar calcaneonavicular ligament arises from the anterior margin of the sustentaculum tali of the calcaneus, connecting to the plantar surface of the navicular. It supports the head of the talus. Its medial border joins with the anterior part of the deltoid ligament of the ankle joint. In addition, support is given by the medial band of the bifurcated calcaneonavicular ligament.



Dorsal intrinsic muscles.

Plantar ligaments of the foot

The American and English versions of *Gray's*

Anatomy differ regarding the histology of the plantar calcaneonavicular ligament. The American version states, "This ligament contains a considerable amount of elastic fibers, so as to give elasticity to the arch and spring to the foot; hence it is sometimes called the 'spring' ligament." **(Gray's Anatomy, 1995 and 1973)** The English version says, "Despite its vernacular name there is no real evidence that the 'spring' ligament is peculiarly resilient." **(Gray's Anatomy, 1980)**

The cuneonavicular articulation consists of the three cuneiform bones and the navicular. There are dorsal and plantar ligaments. The plantar ligaments are supported by slips from the tibialis posterior tendon.

The cuboideonavicular articulation connects the two bones by dorsal, plantar, and interosseous ligaments. This articulation is usually a syndesmosis, but it may be replaced by a synovial joint.

The tarsometatarsal articulations are gliding joints. The 1st metatarsal articulates with the 1st cuneiform. The 2nd metatarsal is given added support by its base articulating with the 2nd cuneiform, and the medial and lateral facets of its base articulating with the 1st and 3rd cuneiform bones, respectively. This forms a groove into which the base of the 2nd metatarsal fits. The 3rd metatarsal articulates with the 3rd cuneiform, the 4th with the 3rd cuneiform and cuboid, and the 5th with the cuboid only.

Giving additional strength to these articulations are the dorsal, plantar, and interosseous ligaments. The plantar ligaments for the 1st and 2nd rays are strongest.

The metatarsophalangeal articulations have plantar and two collateral ligaments. The transverse metatarsal ligament runs across and connects the heads of all the metatarsal bones. The interphalangeal articulations also have plantar and collateral ligaments.

Muscles of the Foot and Lower Leg

The muscles acting on the foot can be divided into extrinsic and intrinsic systems. The extrinsic muscles, in addition to acting on the foot, control its relationship with the leg at the ankle.

Extrinsic muscle system.

The extrinsic muscle system of the lower leg and foot is contained in the five compartments of the leg: the anterior, lateral, and three posterior compartments.

In the anterior compartment are the dorsiflexor and toe extensor muscles: the tibialis anterior, extensor hallucis longus, and the extensor digitorum longus. These are innervated by the deep peroneal nerve.

The peroneal muscles — longus, brevis, and tertius — are in the lateral compartment. The peroneus longus and brevis are innervated by the superficial peroneal nerve, and the tertius by the deep peroneal nerve.

In the superficial posterior compartment are the gastrocnemius, soleus, and plantaris muscles. The deep posterior compartment contains two bipinnate muscles, the flexor digitorum longus and flexor hallucis longus. Finally, there is the tibialis posterior compartment, which contains only that muscle. **(Gray's Anatomy, 1995; Davey et al., 1984)**

Intrinsic muscle system.

There are two intrinsic muscles on the dorsum of the foot, the extensor digitorum brevis and the extensor hallucis brevis. The extensor digitorum brevis originates from the distal and lateral surfaces of the calcaneus, distal to the groove for the peroneus brevis. It inserts by three tendons into the proximal phalangeal bases at their lateral sides. The extensor hallucis brevis originates medial to the extensor digitorum brevis and inserts into the dorsal surface of the proximal phalanx base of the hallux. Sometimes this muscle is considered a division of the extensor digitorum brevis.

The intrinsic plantar muscles often need applied kinesiology origin and insertion, percussion, or neuromuscular spindle cell treatment to maintain corrections made by manipulation of the foot's articulations. **(Leaf, 2010; Cuthbert, 2002)** These muscles may also need treatment to correct dysfunction of the positive support reaction, discussed later.

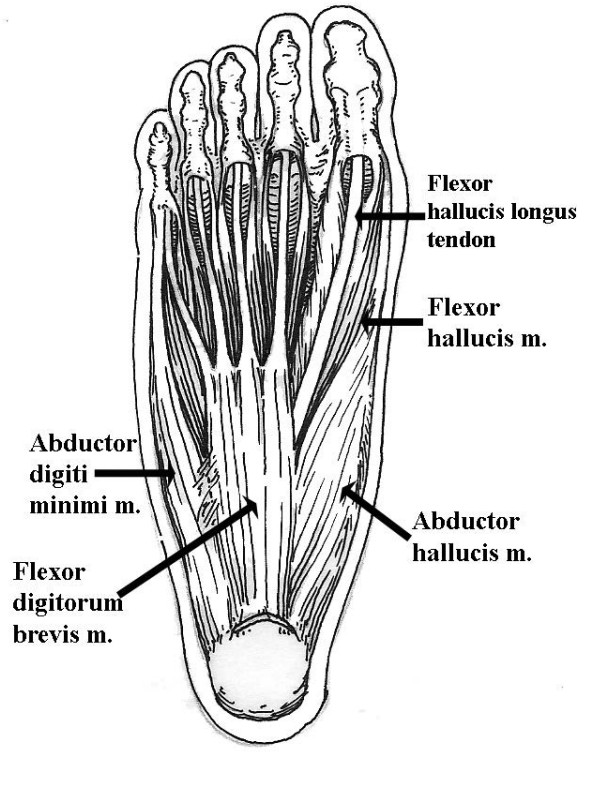
There are four layers of intrinsic plantar muscles — the superficial, middle, and two deep layers — all of which are deep to the plantar aponeurosis. Although many of these muscles cannot be easily tested by manual muscle testing, it is necessary to have their anatomy firmly in mind to effectively apply local treatment to them.

The superficial layer contains three muscles, all of which originate from the inferior aspect of the calcaneus. The abductor hallucis inserts with the medial tendon of the flexor hallucis brevis into the medial side of the base of the proximal phalanx of the great toe. The flexor digitorum brevis inserts into the middle phalanges of the 2nd through 5th toes. The abductor digiti minimi inserts into the lateral side of the 5th toe's proximal phalanx base.

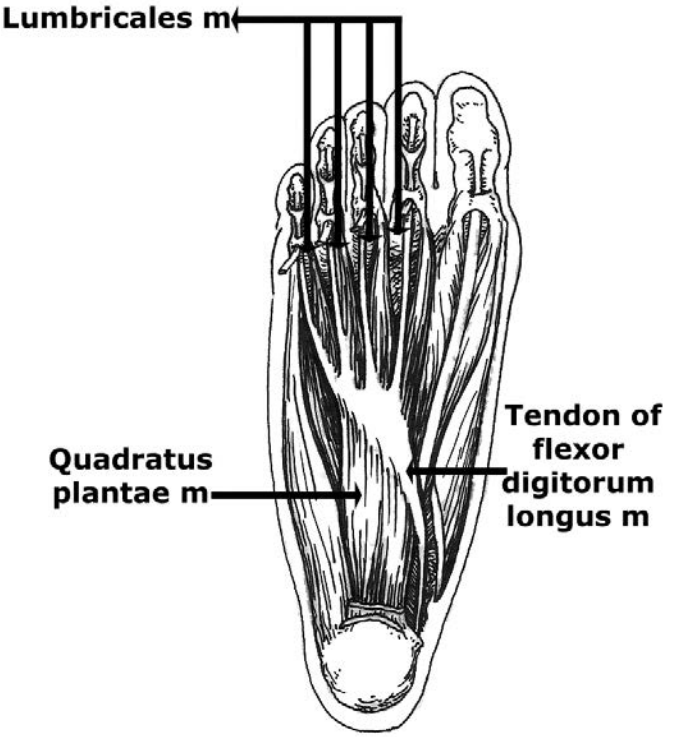
The middle layer contains the quadratus plantae and the lumbrical muscles. The quadratus plantae has four tendons that join with the tendons of the flexor digitorum longus to pass through the flexor digitorum brevis tendons and insert on the distal phalanx. The lumbrical muscles originate between the tendons of the flexor digitorum longus, with the exception of the first, which arises from the medial side of the 1st tendon of the flexor digitorum longus. The tendons of these muscles insert on the medial side of the proximal phalanx of the 2nd through 5th toes. Muscle imbalance and specifically weakness of these muscles contributes to "claw toes." **(Kwon et al., 2009; Hamilton, 1985)**

Briefly, the neurophysiology of tarsal tunnel syndrome exists because the brevis muscles of the proximal intrinsic foot muscles receive their nerve supply after the entrapment at the tarsal tunnel. The longus muscles which attach to the distal toes receive their nerve supply before entrapment at the tarsal tunnel. Because of the imbalance between the longus and brevis muscles of the foot the distal toes curl inferiorly and posteriorly thereby creating claw toes. Tarsal tunnel syndrome and foot pronation should be understood to precede the development of claw toes.

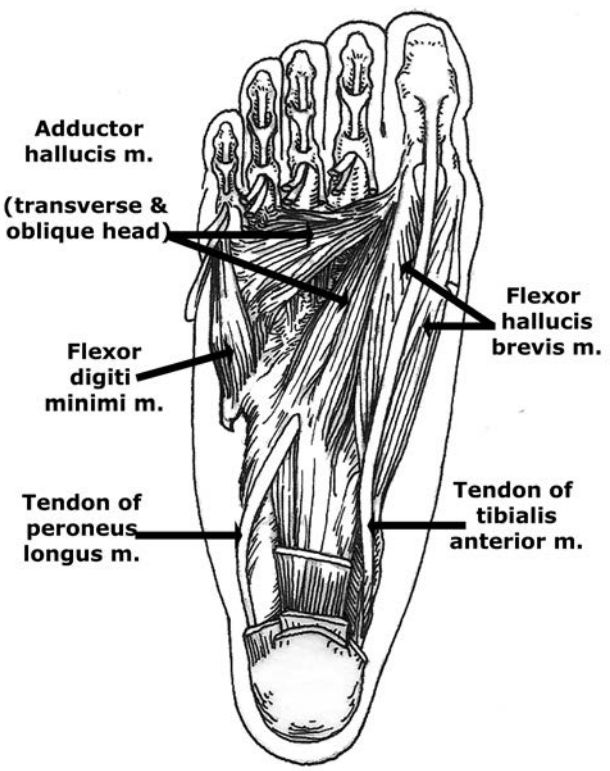
The deep layer consists of the flexor hallucis brevis, adductor hallucis, and the flexor digiti minimi brevis. The flexor hallucis brevis originates at the cuboid bone with prolongation of the tibialis posterior tendon. It inserts by two tendons into the medial and lateral sides of the hallux



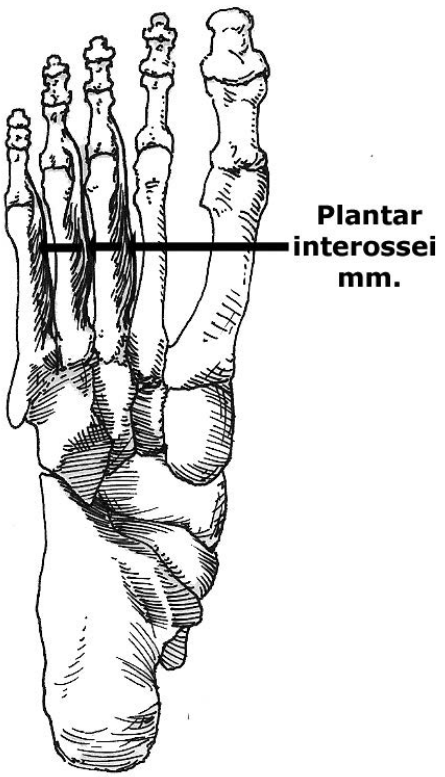
Superficial plantar muscles.



Middle plantar muscles.



Deep plantar muscles.



Deepest plantar muscles.

proximal phalanx. Each tendon usually contains a sesamoid bone. The oblique head of the adductor hallucis muscle arises from the bases of the 2nd, 3rd, and 4th metatarsals, and its transverse head from the joint capsules of the 3rd-5th metatarsophalangeal articulations. Its insertion is into the lateral base of the hallux proximal phalanx.

The fourth and deepest layer consists of the four dorsal and three plantar interosseous muscles. "In humans and gorillas, the second ray is the axis of the foot; the dorsal interosseous muscles abduct from that axis and the plantar interosseous muscles adduct to the axis. Thus, the first dorsal interosseous muscle is attached to the medial side of the second toe, and the second, third, and fourth dorsal interosseous muscles attach to the lateral side of their respective toes." **(Hamilton, 1985)** These muscles contract during walking to resist the forefoot's tendency to splay.

Travell and Simons **(Travell & Simons, 1992)** note that myofascial trigger points in the intrinsic plantar muscles are aggravated by the wearing of poorly fitting, tight, or badly designed shoes; ankle and foot injuries, articular dysfunction, conditions that allow the feet to become chilled and systemic conditions that affect the feet. In these cases, the muscle stretch reaction will be present and will guide the clinician toward the solution of this problem in the intrinsic muscles. **(Cuthbert, 2002)**

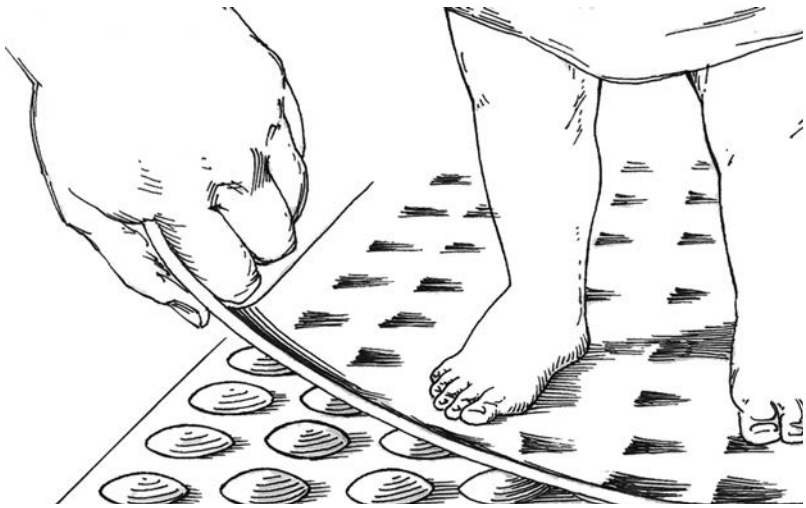
Arches of the Foot

Lake, **(Lake, 1937)** in discussing the development of the arch for adaptation to the upright posture, speaks of man needing support for his foot behind the axis of the ankle joint in order to keep the center line of gravity within the base. The calcaneus forms a lever for the large calf muscles to balance the weight in the foot. Lake goes on to state, "By Wolff's law, bone would be formed in the proper place and to the extent necessary to resist the stresses it encountered, and the os calcis therefore developed both backwards and downwards to resist the great additional stress thrown upon it in walking." Thus we see great differences in the human foot compared with that of lower primates.

The development and strength of man's arches are not limited to the evolutionary process. The stimulation of the foot from early walking to adult activity is important to the foot's strength and adaptability. Infants have no arch. It has been said that the arch is present in infants but is obscured by a fat pad, **(Morley, 1957)** but this has been shown by pedotopographic x-ray to be untrue. **(Gould, 1989)** The arch begins to develop with walking. With early walking, the arch develops faster in those wearing shoes with support to the medial longitudinal arch than in those without the support but still wearing shoes. At five years of age the arch develops at approximately the same rate with or without an arch support in shoes. **(Gould, 1989)** When the child's feet are subjected to uneven surfaces during early stages of walking, there is less chance of excessive pronation and weak feet developing. **(Lieberman et al., 2010; Rao & Joseph, 1992; Herzmark, 1947)**

The best long-term development is without shoes. A Nigerian study has shown that students wearing shoes most of the time are predisposed to poorly developed arches compared with those who seldom wear them. **(Mauch, 2008; Rao & Joseph, 1992)** This appears to be due to the shoe preventing direct contact with the ground, reducing foot action that would exercise the muscles and plantar fascia. **(Squadrone & Gallozzi, 2009; Didia, 1987)** The feet of people who do not wear shoes are more supple and appear almost flat in the relaxed state, but they become highly arched in action. **(Rasch & Burke, 1978)** Olympic champions and world record holders Abebe Bikila, Tegla Loroupe, and Zola Budd have shown that barefoot running is consistent with superb running performance.

Shoes restrict movement of the feet, reducing the activity and development of the plantar fascia and muscles. They also reduce stimulation to the sensitive mechanoreceptors. **(Richards et al., 2009; Robbins et al., 1989)** Shoes may reduce force to the metatarsals by half. **(Robbins et al., 1988; Collis & Jayson, 1972)** Stimulation to the mechanoreceptors is needed for optimal development of the nerve reflexes and reactions, discussed later. Proprioceptive



Herzmark constructed a playpen pad with an uneven surface and demonstrated better foot development in children.

stimulation coming from the unrestricted sole of the foot may facilitate afferent neural pathways as well as optimizing efferent muscle response and recruitment strategies. As in all sensory-motor complexes, the information going out is only as good as the information coming in. Shoe restriction produces greater demand on the bony architecture with the loss of dynamic activity.

The bony architecture of the human foot is designed for weight-bearing bones to lock together. The importance of proper bony architecture in arch maintenance is emphasized by the fact that a completely paralyzed foot, if once normal, maintains its arch, even when maximum weight is placed upon it. **(Harris & Beath, 1948)** The architectural design of the human arch is a wonder of nature that blends all of the elements of the foot – joints, muscles and ligaments – into a unified system that permits the foot to operate with the best mechanical advantage under the most varied conditions. Any functional or pathological disturbance in this architecture will interfere significantly with the function of the body as it stands as well as with running, walking, and the maintenance of erect posture.

There are three arches in the foot: the medial and lateral longitudinals, and the transverse. They have varying degrees of flexibility, and act as shock absorbers.

In general, an arch or curved structure can be supported in two ways: 1) by being tied together to prevent splaying, and 2) by having a keystone shape of the parts that make up the arch so that they wedge together to support weight from above. The foot uses both methods. **(Kapandji, 2010; Hamilton & Ziemer, 1983)** In addition, muscles have a critical role in arch support under certain conditions.

As study of the foot has progressed over the years, some controversy has developed regarding maintenance of the arches. As we look at the three types of tissue factors in the feet — bony architecture, muscles, ligaments and aponeurosis — we should recognize that there is no single factor responsible for the arch's maintenance. Any one of the three, when deficient, can cause a breakdown of the arches. The proponents of a single approach to the examination and treatment of the feet are right in some instances, but not in all.

The foot's arches have been compared to mechanical structures. This is helpful, but in the final analysis one must bring in the living, dynamic action of function controlled by an active nervous system. Static analysis does not take into consideration the adaptation necessary to meet the onslaught of forces the foot must deal with in daily living.

Only with a thorough combination of actions can the foot provide the shock absorption needed to deal with the daily forces to which it is subjected. The calcaneonavicular ligament has been referred to as the "spring" ligament, which is a misnomer. The concept of the arch having a springing, shock-absorbing effect is based on ligaments having elasticity. Two levers formed as an inverted "V," with a hinge at the apex and the other ends of the levers connected by a spring, would be a simple analogy to the arch of the foot being a spring shock absorbing mechanism. The foot would be a more complex inverted "V," with numerous springs attached to the various bones. **(Lapidus, 1943)**

There is considerable evidence that the arches of the feet are maintained in a static weight-bearing position primarily by ligaments and not muscles. (1) There is minimal activity of the muscles in a neutral standing position, as evidenced by electromyography, except for the control of sway for maintaining balance. **(Landry et al., 2010; Gray's Anatomy, 2004; Gray, 1969; Gresczyk, 1965; Mann & Inman, 1964; Basmajian & Stecko, 1963)** (2) An individual with complete paralysis of the foot and lower leg, such as from poliomyelitis, maintains the full height of the original arch, even though weight bearing is maintained by the use of braces. **(Harris & Beath, 1948)** (3) Nature is a conserver of energy. **(Basmajian, 1961)** If muscle action were necessary to maintain the arch, fatigue would rapidly result from a static stance. Compare a normal static stance with that of standing on the tiptoes for equal lengths of time. The foot is a combination of 1) arches, an example of which is the masonry arch, 2) truss structures like those used in bridges and roofs, and 3) a motor system provided by the muscles for movement. The line of pull for the intrinsic and extrinsic muscles of the foot are essentially in the long arch of the foot and perpendicular to the transverse tarsal joints; for this reason the muscles are important contributors to the muscular support of the arch during gait. **(Gray's Anatomy, 2004)**

A typical masonry arch can stand with great strength, as long as each end of the arch is firmly anchored to the ground. When the arch bears weight, the components wedge against each other, maintaining the arch. This construction is illustrated in the proximal metatarsal and transverse arches of the foot.

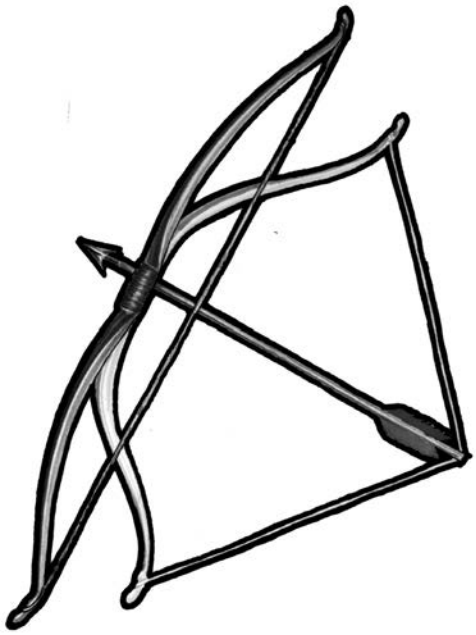
Whether standing, walking, or running, the foot is subjected to considerable stress. In mechanical considerations, stress is divided into tension, compression, and shear forces. **(Damm & Waugaman, 1948)** Tensile stress tends to separate particles of the material. Compression stress compacts or brings the particles closer together. Shear stress occurs when the particles tend to slide over one another at a given section, creating a cutting action. Bending stress is a combination of compression and tensile stress.

A simple beam is a combination of tensile and compression strength. When the beam is supported at its two ends and weight is placed on the center of the beam, the force to the lower portion of the beam is tension; from the point of weight to the two support points, it is compressive strength. The area shaded in the illustration produces negligible support to the weight.

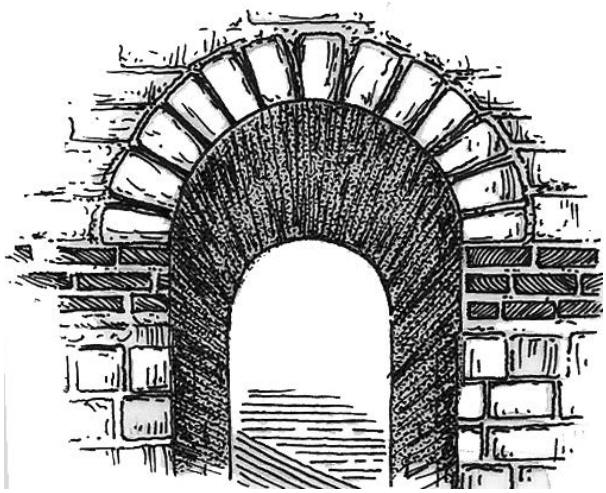
A truss is built of materials selected to provide maximum strength with minimum material bulk. To accomplish this, the upper members of the truss are made of materials selected to provide compressive strength, while the lower member is a cable or some other material that provides tensile strength. This provides an example of the medial longitudinal arch of the foot. The bones are excellent providers of compressive strength, while the ligaments provide the tensile strength. Muscles provide the dynamic action of foot function. Proper function and/or structure of all three are necessary to provide optimal support and locomotion to the body.

Bony architecture

The medial longitudinal arch is the highest of the arches. It is formed by the first three rays, the three cuneiforms, the navicular, the talus, and the calcaneus. The 1st metatarsal, its sesamoid bones, and the 2nd and 3rd metatarsals comprise the



The string of a bow represents tensile strength like the ligaments in the arches.



The stones in a Roman arch wedge together with compressive strength like some of the bones in the arches.

(With kind permission, ICAK-USA)

anterior pillar. The 1st ray contributes to the strong pillar function because of the 1st tarsometatarsal joint's limited range of motion. **(Wanivenhaus & Pretterklieber, 1989)** The tuberosity of the calcaneus is the posterior pillar as viewed from the medial side. The head of the talus is the keystone. **(Kapandji, 2010; Hamilton & Ziemer, 1983)** The arch is slanted in the forefoot, and more steeply arched in the hindfoot.

The 4th and 5th metatarsals make up the anterior pillars of the lateral longitudinal arch, while the tuberosity of the calcaneus comprises the posterior. The apex is the cuboid. The lateral longitudinal arch has minimal height in comparison with the medial. **(Hamilton & Ziermer, 1983)**

The transverse arch is rigid, very similar to a standard Roman arch. It is made up of the wedge-shaped navicular, cuneiforms, and cuboid. The wedge portion of the bones is narrower toward the plantar surface and wider dorsally. When articulated, the bones form longitudinal and transverse arches, with the concavities facing inferiorly. This arch is continued into the metatarsal bones, which are wedge-shaped at their bases.

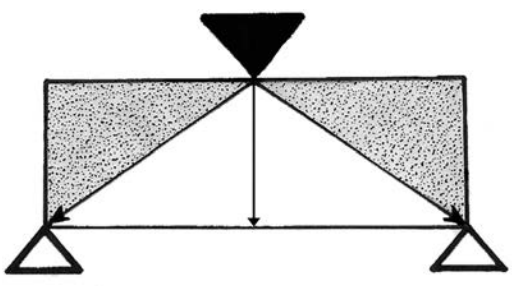
A basic cause of pronation and flatfoot can be a congenital anomaly of the bones, especially the talus and calcaneus. **(Vukasinović et al., 2009; Harris & Beath, 1948)** In this case, there will be excessive pronation, regardless of the efficiency of the other two aspects — ligaments and aponeurosis, and muscles. Types of bony congenital anomalies that cause pronation will be discussed later under that subject.

Ligaments and aponeurosis

Hicks **(Hicks 1951, 1953, 1954, 1955)** studied the dynamic function of the longitudinal arches of the foot by dissection, x-rays of movement at sequential steps of the dissection, and x-rays of the living foot. He concluded that in the normal foot the arch is maintained by the plantar aponeurosis, with no contribution from the intrinsic or extrinsic muscles. The operative phrase here is "normal foot." It might be added that there must be normal activity of the intrinsic and extrinsic muscles during walking, running, and jumping.

The mechanism that Hicks described is of significant interest in function. Extension of the toes tightens the plantar aponeurosis to elevate the arch. This can be easily observed when one stands with most of the weight on the heel. The toes are easily extended, and it can be observed that the arch rises. When weight is evenly distributed over the full length of the foot, causing compression of the arch, the extension of the toes is limited by tension on the aponeurosis; indeed, one feels tension develop in the plantar aponeurosis.

The influence of toe extension on the plantar aponeurosis results from the attachment of the aponeurosis to the proximal phalanges. As the toe extends, it pulls with it the plantar pad, which is an extension of the aponeurosis. The plantar pad, sliding around the metatarsal head, pulls on the plantar aponeurosis, tightening it as if it were a cable arrangement being pulled around the drum of a windlass. Maximum toe extension shortens the effective length of the aponeurosis by approximately 1 cm. This mechanism exists in all five toes, but it is most marked in the great toe. When

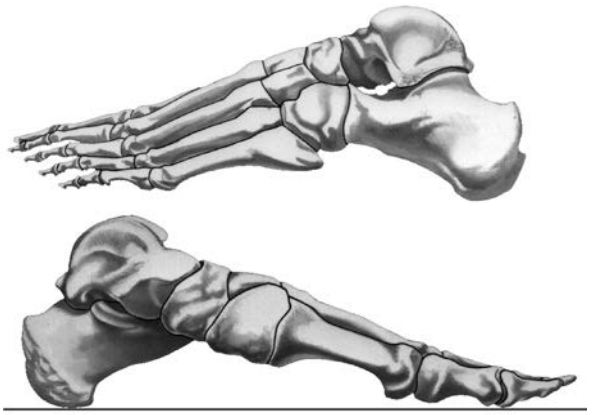


Locations of compressive and tensile strength in a beam.

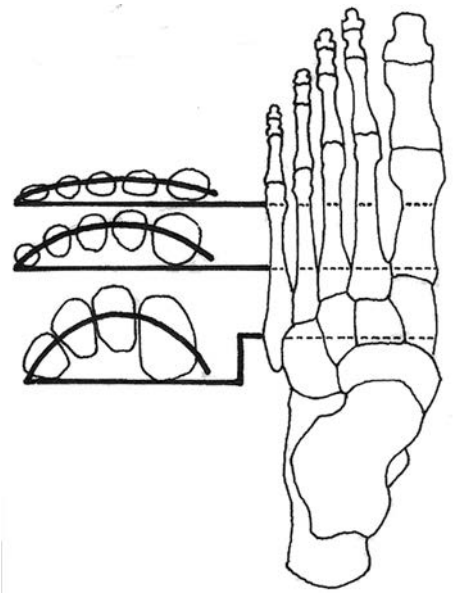
the hallux is amputated, its role in the windlass is lost; the forefoot weight-bearing shifts laterally throughout gait, and the height of the medial longitudinal arch diminishes. **(Mann et al., 1988)**

This mechanism is important in gait activity. The medial longitudinal arch, controlled by the windlass mechanism, rapidly shortens just before toe-off; it then lengthens at heelstrike, becoming the longest at about the time of toe contact. **(Levangie & Norkin, 2001; Kayano, 1987)** This occurs because during the last portion of the stance phase, toe extension pulls on the plantar aponeurosis, raising the height of the arch. This increases the range and speed of planter flexion over and above that which occurs in the ankle alone. It helps the foot become a solid lever and avoid yielding to increasing forces at the toe-raising phase. This causes the foot to plantar flex and thrust downward, with an additional "flick" on take-off.

In static stance, the mechanism works in reverse. Body weight flattens the arch, which unwinds the windlass mechanism and causes the toes to flex toward the ground. This gives a "gripping" action of the toes on the ground.



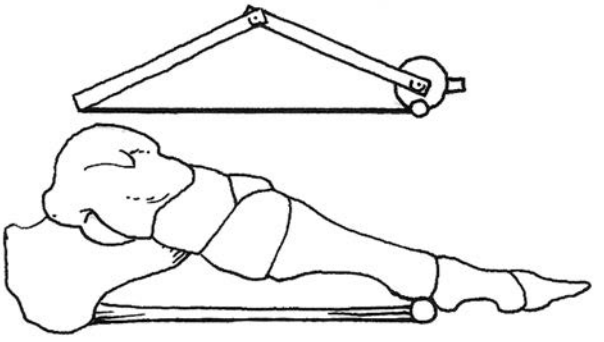
Medial longitudinal arch & Lateral longitudinal arch.



Traverse arch diminishes distally

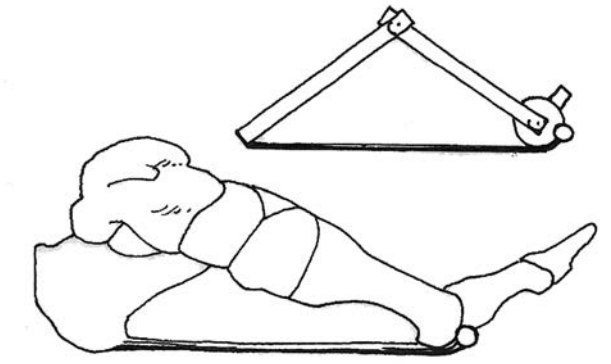
Muscle role in support of the arch

There are three ways in which the foot functions: (1) in directed free movements, (2) as a static support mechanism subject to gravitational forces and postural changes, and (3) as a dynamic mechanism for moving the body. **(Chaitow & DeLany, 2002; Houtz & Fischer, 1961)** The foot has limited function as a freely-moving appendage, yet analysis of the foot and manual muscle testing are usually done under this circumstance. Understanding the role of muscles in weight bearing and the dynamic activity of walking and running is important in accurately determining areas of dysfunction so that proper treatment can be applied.



Hicks mechanism without toe extention

(With kind permission, ICAK-USA)



With toe extension the plantar fascia tightens to raise the arch

From anatomical studies made prior to electromyography, it was concluded that several muscles have a role in arch maintenance in the normal *static* foot. **(Jones, 1941; Kaplan & Kaplan, 1935; Lake, 1937)** Electromyographic studies have modified this conclusion. **(MacConnaill & Basmajian, 1969; Basmajian & Bentzon, 1954; Basmajian & Stecko, 1963; Gray, 1969; Gresczyk, 1965; Mann & Inman, 1964; Smith, 1951)** In gait however both the longitudinally and transversely oriented muscles

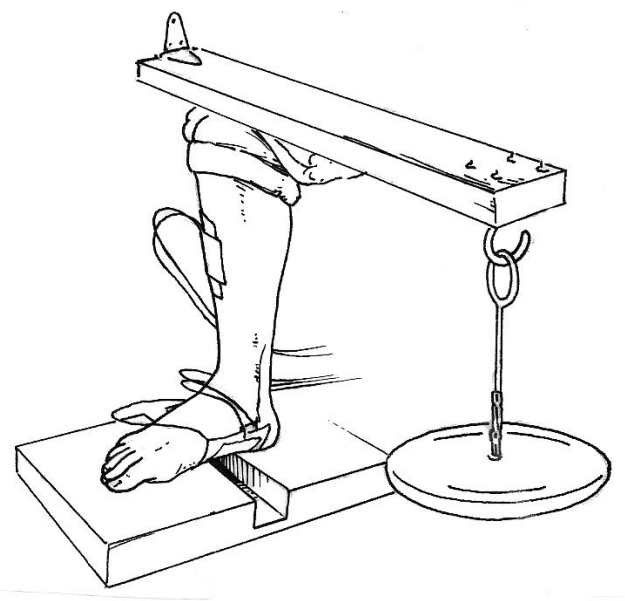
become active and contribute to support of the arches of the foot. **(Levangie & Norkin, 2001)** Muscles of interest are the tibialis anterior for evaluation of its ability to maintain elevation of the medial longitudinal arch; the tibialis posterior and peroneus longus to determine if they might provide sling support; and the flexor hallucis brevis, abductor hallucis, and flexor digitorum brevis, as all three are intrinsic to the foot and may provide longitudinal bowstrings. **(Thordarson D, et al., 1995)** It is also important to compare the muscles in both static and dynamic action for normal feet and pronated or flatfeet.

Basmajian and Stecko **(Basmajian & Stecko, 1963)** did a fine-wire EMG study of the static foot. Muscles studied were the tibialis anterior and posterior, peroneus longus, flexor hallucis longus, abductor hallucis, and flexor digitorum brevis. The subjects were seated, with the foot supported on an adjustable platform. Weight was applied on the flexed knee in the amount of 100, 200, and 400 pounds by a lever mechanism so arranged that no leg movement would take place during the experiment. One hundred pound weights were chosen to approximate or exceed the normal load on each foot in the upright bipedal stance, and 200 pounds to approximate or exceed the load on the arch in one-legged stance. The 400-pound load was the maximum that could be applied without extreme discomfort at the knee.

Recordings were made with no load, then progressively adding the loads noted previously. The foot platform was set to horizontal and 20° each of dorsiflexion, plantar flexion, inversion, and eversion. With no load there was no muscular activity recorded; with 100 pounds, there was negligible activity or, in a very small percentage of the population, minimal activity. Two hundred pounds caused the muscular activity to increase generally, but only slightly. With 400 pounds of weight, many muscles continued to show no activity. The tibialis anterior had the highest incidence of activity, and the tibialis posterior was the most active in general. Muscle activity varied considerably among the individuals in the various foot positions.

A contradictory EMG needle study by Suzuki **(Suzuki, 1956)** was done on normal subjects positioned in a manner similar to that mentioned earlier. With a load of 60 kg on the flexed knee, the muscles of the foot and leg revealed the following. Contracting against the load were the flexor hallucis longus, tibialis posterior, abductor hallucis, flexor digitorum longus, and the abductor digiti minimi. There was no action in the peroneus longus and brevis, tibialis anterior, and flexor digitorum brevis, which correlated with Basmajian and Stecko's study.

The preponderance of evidence is that muscle activity is not necessary to maintain the *static* arches in the normal foot. **(MacConnaill & Basmajian, 1969; Basmajian & Bentzon, 1954; Basmajian & Stecko, 1963; Gray, 1969; Gresczyk, 1965; Mann & Inman, 1964; Smith, 1951)** Bony architecture and ligaments support the normal static foot. These studies support Basmajian's thought: "Muscles [are] spared when ligaments suffice." **(Basmajian, 1961)** Man stands on his skeleton. Muscle is used only in short bursts to maintain sway control. **(Burke et al., 1982)** When flatfooted subjects are studied however, the findings are significantly different. These are the type of feet that walk into clinician's offices for evaluation and treatment.



Apparatus of Basmajian and Stecko (1963)

In Gray's study **(Gray, 1969)** of the static foot, subjects were evaluated by EMG while standing for two minutes, having been cautioned not to sway. In the normal group there was marked activity of the soleus and no activity of other muscles. Of the 27 subjects with flatfeet, there was marked activity of the muscles as follows: soleus, 26; tibialis anterior, 23; tibialis posterior, 23; and peroneus longus, 22. Gresczyk **(Gresczyk, 1965)** also found activity in muscles other than the soleus in a flatfooted group (n=27). In no instance was there activity of only the soleus in this group.

When an individual is standing and shifts his weight from one leg to the other, there is a short burst of activity of the posterior leg muscles, the peroneus longus and brevis, and the intrinsic muscles of the foot; there is no activity during the static stance. **(Levangie & Norkin, 2001; Smith, 1954)**

Mann and Inman **(Mann & Inman, 1964)** did an excellent fine-wire EMG study of the intrinsic muscles of the dynamic foot. The following muscles were studied: extensor digitorum brevis, abductor hallucis, flexor hallucis brevis, flexor digitorum brevis, abductor digiti minimi, and the dorsal interosseous muscle between the 3rd and 4th toes. In addition, the extrinsic gastrocnemius and tibialis anterior were considered.

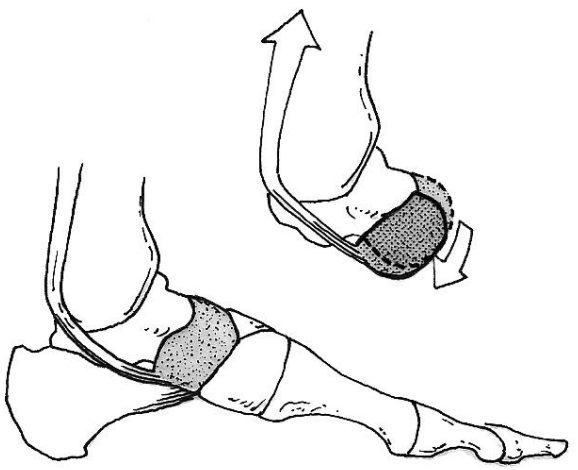
Some of the subjects had asymptomatic bilateral flatfoot, and their muscular patterns varied from subjects with normal feet during level walking. Normal and flatfooted subjects had comparable muscular activity going up or down stairs or slopes, and standing on their toes. When standing on the toes, all intrinsic muscles were active, along with the gastrocnemius. The tibialis anterior muscle was electrically silent. The intrinsic muscles were inactive during quiet standing, except for sporadic bursts.

During walking, there is electrical activity of the intrinsic muscles during the stance phase only. Individuals with normal feet do not have activity in the intrinsic muscles from heelstrike until 40% into the cycle. In flatfeet, the muscular activity begins 10% into the cycle. Likewise,

transverse tarsal stabilization begins at 40% into the cycle in the normal subject, and 10% into the cycle in flatfeet. This study shows that the intrinsic muscles of the foot act together as a functional unit. The pronated foot requires greater intrinsic muscle activity to stabilize the transverse tarsal and subtalar joints than does the normal foot.

A balance of muscle turgor is necessary to maintain balance in the arch. When in the weight-bearing position, the triceps surae is normally stretched beyond its resting length. When non-weight bearing, the resting length yields approximately 30° of plantar flexion. When standing, this causes additional tension on the Achilles tendon attachment to the calcaneus, maintaining the calcaneus position. When there is paralysis of the gastrocnemius and soleus, a progressive deformity of the foot occurs. The calcaneus is rotated into the vertical position to cause its posterior aspect to move inferiorly. The paralysis, if combined with a completely paralyzed foot, does not cause deformity. This gives evidence that there is balance between the intrinsic muscles of the feet and the triceps surae to maintain the normal arch. **(Gondin et al., 2004)**

All positions and actions of the feet must be considered in evaluating the muscles' role on arch maintenance. Hamilton, **(Hamilton, 1985)** in a discussion of the surgical anatomy of the foot and ankle, indicates that the tibialis posterior and anterior muscles are important in maintaining the longitudinal arches. Referring to the tibialis posterior, he states, "Clinically the importance of this muscle is seen when its tendon ruptures and a true fallen arch occurs with great rapidity and severity." The tibialis posterior plays an important role in moving the navicular bone slightly on the head of the talus. **(Kapandji, 2010)** By moving the navicular inferiorly and slightly posteriorly, the anterior pillar of the medial longitudinal arch is angled to increase the arch height during the latter portion of the gait's stance phase. In addition, the head of the talus has a different radius from medial-to-lateral as opposed to superior-to-inferior. "Thus as the talonavicular joint is loaded, increasing stability is brought about by the seating of the convex head of the talus into the concave navicular." **(Mann, 1982)**



Navicular bone movement with tibialis posterior contraction.

Even muscles remote from the foot and leg may play a role in extended pronation. In addition to supporting the posterior pelvis to decrease lumbar hyperlordosis, the gluteus maximus externally rotates the thigh. With external leg rotation, the arches of the feet rise. This can easily be demonstrated on yourself by contracting the gluteal muscles while standing. Feel the external leg rotation and elevation of your arches. **(Greenawalt, 1988)** As will be seen later, it is apparent that poor foot function can cause an increase in the spinal AP curves; conversely, poor remote muscle function and an increase of the spinal AP curves can cause excessive foot pronation.

Notwithstanding the EMG data indicating that muscles are spared if ligaments suffice, **(Basmajian, 1961)** clinical data suggests that a common form of flatfoot is caused by muscle failure resulting in stretched ligaments and bones in altered positions. When muscles fail to support the arch, the ligaments can be stretched and the medial longitudinal arch of the foot is lost. **(Langer, 2007)** In addition, when excessive pronation is present — from whatever cause — the muscles are abnormally active in an attempt to control the problem.

Strength and flexibility of the musculature spanning the foot is very important. The majority of muscles function eccentrically, not concentrically, during walking. For this reason their strength and flexibility are important. If muscles are weak they are unable to control function and movement; if they are inflexible they will not permit function and movement. Particularly with weakness of the posterior tibialis there will be reduced control of subtalar joint pronation. **(Zwipp et al., 2000)** Subtalar joint pronation may also be compensation for limited dorsiflexion at the ankle joint which encourages abnormal dorsiflexion at the subtalar joint or midtarsal joints **(Donatelli and Wooden, 1996; Bohannon et al., 1989).**

When examination and correction of foot problems are considered, we will see that there is much that the physician using applied kinesiology can do to correct excessive foot pronation when there are subluxations of the bones of the feet and muscle dysfunction. In some instances, especially those of congenital anomaly of the bones, support or surgery is necessary to effectively treat the foot. Understanding foot function enables one to choose the proper direction of treatment.

Foot weight bearing. Early descriptions indicated that static weight-bearing forces to the foot are those of a tripod. With modern measuring equipment, **(Thordarson et al., 1995; Hughes et al., 1987; Lord, 1981; Soma Staff, 1988)** this has been shown to be incorrect. Weight distribution is in the general form of a triangle, but the forces are not concentrated on the ends of a transverse arch at the metatarsal heads and the calcaneus. Considerable differences are described in the literature regarding the concentration of forces on the static weight-bearing foot. Some indicate that the 1st ray carries twice the weight of each of the others, giving a distribution of 2:1:1:1:1. **(Hamilton, 1985; Viladot, 1973; Manter, 1946)** Other studies **(Luger et al., 1999; Rodgers & Cavanagh, 1989; Cavanagh, 1987)** indicate that the 2nd ray has the highest force. This discrepancy probably relates to the muscle's and nervous system's role in weight distribution, as well as the structure of the arches. In any event, the transverse arch does not extend to the distal metatarsals; thus there is weight bearing across all the metatarsal heads.

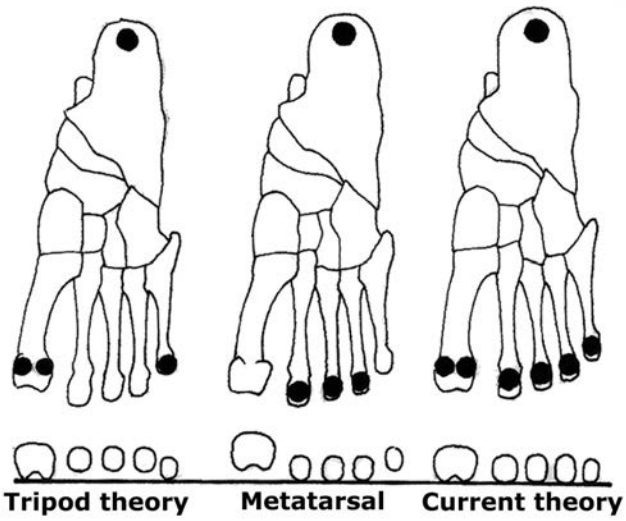
Each of the five rays contributes to weight bearing, but not equally. Lying side by side, they are tied together posteriorly and mechanically independent anteriorly, where the level of each ray is independently determined by its amount of flexion and not by any relationship between it and its neighbors. Any metatarsal head projecting below the general level takes a heavier load than the others. Each ray in the neutral weight-bearing foot bears weight by acting variously as a beam and as an arch (or truss). With toe extension there is increased tension on the plantar aponeurosis by the windlass mechanism, causing more arch support and less beam support. If the weight-bearing foot is put on a block where the toes can flex, it relaxes the tension on the plantar aponeurosis. The arch drops somewhat and there is more contribution to weight bearing by the beam function of the rays. **(Hicks, 1955)**

Although the metatarsals are the major weight bearers of the forefoot, it is the toes that "grip" the substrate. The metatarsals are incapable of making this adjusting contact because they lack any tendinous insertions. **(Abshire, 2010; Viladot, 1973)** The great toe also presses against the ground because it has only two phalanges. **(Cailliet, 1997)**

There is great conflict in the literature on weight balance into the foot. The bottom line is *in vivo* weight balance. Levangie and Norkin and Hamilton **(Levangie & Norkin, 2001; Hamilton, 1985)** describe the role of muscle activity on the distribution of forces to the foot. With muscle relaxation, weight loaded onto the knee is distributed 80% into the metatarsals and 20% into the calcaneus. Normally contraction of the triceps surae distributes the weight between the metatarsals and the calcaneus, with greater weight on the hindfoot. In his description the weight distribution into the metatarsals is 2:1:1:1:1, with double the weight being distributed to the 1st metatarsal. In a weight-loaded cadaver foot, weight is distributed 4:1:1:1:1. The 1st ray bears as much as the others combined, rather than half as much as in the normal living foot.

Supraspinal and local neurologic factors play a role in foot protection. Hennig and Cavanagh **(Hennig & Cavanagh, 1987)** studied muscular action of the foot during expected and unexpected falls onto a flatfoot. When a fall is unexpected, there is stronger force to the heel. During an expected fall, there is supination and flexion so the force goes more into the lateral forefoot and midfoot borders, providing protection for the heel. Stimulation to the foot mechanoreceptors must be received by the central nervous system for foot protection. In rheumatoid feet in which there is no neurologic deficit, there are low forces where active arthritis is present. **(Yavuz et al., 2010; Collis & Jayson, 1972)** In diabetic feet, peak pressure is higher at the point of ulcer formation, **(Veves et al., 2002; Rogers et al., 1987)** indicating poor neurologic feedback from diabetic neuropathy. The pressures of the insensitive foot are different and greater than normal when walking. **(Veves et al., 2002; Bauman & Brand, 1963)**

The forefoot support provided by the metatarsals is very irregular. **(Viladot, 1982)** There is consensus that the usual peak pressures during gait are rarely located underneath the 1st ray. **(Chaitow & DeLany, 2002; Levangie & Norkin, 2001; Cavanagh et al., 1987; Viladot, 1982)**



Foot weight bearing theories

Ankle Anatomy and Function

The articulation between the distal tibia and fibula and the talus is referred to as the ankle mortise or ankle joint (also called the tibiotalar or talotibiofibular or talocrural joint). A high level of integrity is required of this joint for normal foot and leg function. Gray's anatomy **(Gray's Anatomy, 1995)** points out that compressive forces of five times the body weight and tangential shear forces of 80% of the body weight are transmitted across this joint. Stress is put into this joint from excessive foot pronation or abnormal hip or leg rotation. The overall articulation between the three bones is a synovial ginglymus joint. Its structure and ligaments allow a single oblique axis of motion that provides dorsiflexion and plantar flexion of the foot.

The nerve supply to the joint comes from the deep peroneal and tibial nerves while the arterial supply comes from the malleolar rami of the anterior tibial and peroneal arteries.

The distal tibia and fibula make up the proximal portion of the joint. The union between the tibia and fibula is a syndesmosis (a fibrous joint in which relatively opposing surfaces are united by ligaments). Separation of the bones is prevented by the ligaments, primary of which is the crural tibiofibular interosseous ligament. It is continuous with the interosseous membrane, which extends most of the length of the tibia and fibula. This ligament's strength is so great that forces that tend to separate the bones will usually fracture the fibula proximal to the ligament before it will tear. Also supporting the inferior tibia and fibula articulation are the anterior and posterior tibiofibular ligaments and the interosseous membrane. The tibialis posterior muscle has an important role in the physiologic separation of the bones and maintenance of ankle joint integrity. (This will be discussed later.)

The distal aspect of the tibia projects medially to form the medial malleolus. The lateral surface of the medial

malleolus is an articular surface that extends about one-third of the way down the medial talar body. The distal aspect of the fibula comprises the lateral malleolus. Its medial articular surface covers the entire lateral articulating surface of the talar body. Together the bones form a U-shaped articular surface, which is congruent with the articulating surfaces of the talar body.

The ankle joint should be seen as two joints with integrated functional action: the ankle mortise and the subtalar joint. Inman **(Inman, 1976)** showed that during the gait cycle there is more visible medial rotation of the tibia than can be explained by movement solely at the talotibiofibular joint. Inman demonstrated that the increased tibial rotation described resulted from calcaneal eversion about the subtalar axis.

The superior articulating surface of the talus is called the trochlear surface. The trochlea is wider anteriorly than posteriorly, giving it a wedge shape. It is approximately 5 mm wider anteriorly. **(Kapandji, 2010)**

Several ligaments are important in stabilizing and limiting ankle motion. The medial ligament is also called the deltoid collateral ligament. It is a strong ligament that generally attaches proximally at the medial malleolus and spreads with superficial and deep fibers to the navicular anteriorly, the talus, and the calcaneus distally and posteriorly.

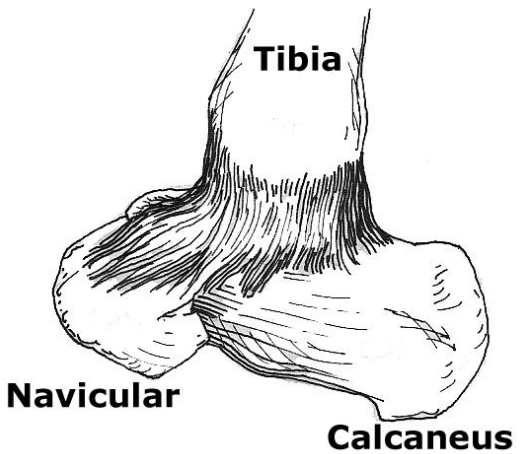
The lateral ligament is composed of the anterior talofibular ligament, posterior talofibular ligament, and the calcaneofibular ligament. The lateral ligament, as a whole, is weaker than the medial one; consequently, it is more prone to injury. Freeman reports that 40% of injuries to the lateral ligaments result in functional instability of the ankle. **(Freeman, 1965)** The medial ligament will often avulse the bone rather than tear with trauma.

An *in vitro* study was done by Stormont **(Stormont, 1985)** on the contribution of the ligaments and articular surfaces to the ankle's stability. Evaluation was done in both the loaded and unloaded states for internal and external rotation, inversion, and eversion. In internal rotation, the two primary restraints are the anterior talofibular ligament and the deltoid ligament. In external rotation, the primary restraint is the calcaneofibular ligament. In both internal and external rotation during loading, the articular surface accounts for 30% of the stabilization.

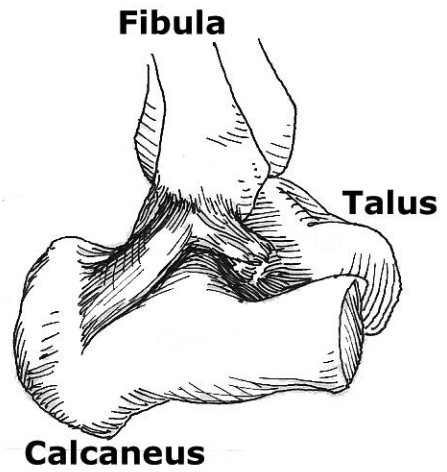
The calcaneofibular ligament is the primary restraint in inversion, and the anterior talofibular ligament is secondary. The deltoid ligament is the primary restraint in eversion. In both inversion and eversion during loading, ligaments do not contribute because 100% of the stabilization is provided by the articular surface. Ankle instability may occur during loading and unloading; it does not occur once the ankle is fully loaded because of the articular surface's resistance to inversion displacement. This helps one understand the importance of the shape of the bones in providing ankle strength to resist the application of forces that may traumatize the ankle. It must be recognized that this study **(Stormont, 1985)** was done *in vitro* by cutting the ligaments. One must consider the effect of muscles in both the loaded and unloaded condition.

Rotation of the leg into the foot, which is fixed on the substrate, may account for clinically symptomatic ankle instability. **(Stormont, 1985)** The ankle is more vulnerable to injury when sports shoes make firm contact with the playing surface. **(Abshire, 2010)**

For optimal function, the ankle mortise must be a congruent articulation throughout its range of motion. An adequate ankle range of motion is a necessary component for many activities such as running, ascending and descending stairs and normal gait **(Brukner and Khan, 2006; Donatelli, 1996).** Since the body of the talus is wedge-shaped, being wider anteriorly, there must be some accommodating mechanism in the tibia and fibula to maintain the articulation's integrity. This is accomplished by changing the distance between the medial and lateral malleoli.



Medial ankle ligaments.



Lateral ankle ligaments.

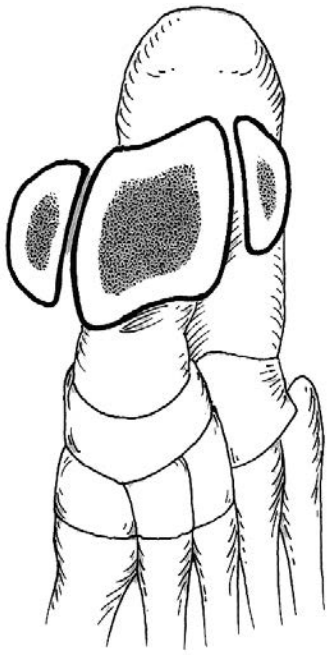
Grath **(Grath, 1960)** studied the widening of the ankle mortise from plantar flexion to dorsiflexion by implanting steel pins into the lateral and medial malleoli of living, locally-anesthetized volunteers. Measurement was accurately taken on the exposed ends of the pins during ankle motion. The mortise width is less on maximal plantar flexion and greater on maximal dorsiflexion. The increase

of mortise width may be maximal between plantar flexion and neutral, or neutral and dorsiflexion. It is rare that widening of the mortise fails to occur with dorsiflexion.

It is important that the mortise not widen with weight bearing. Measurement by the above method "… demonstrates that increases in mortise width often fail to occur on loading the ankle with the weight of the body, and that when they do occur they are so insignificant as to defy demonstration with the conventional millimeter gauge." **(Grath, 1960)**

Grath's study definitively documents the adaptation of the tibia and fibula to the wedge shape of the talus body. The adaptation of the mortise to position is of great importance when one examines the ankle for integrity. There is considerable difference in the amount of joint play, depending on whether motion is passive or active.

Jaskoviak **(Jaskoviak, 1983)** indicates that the ankle mortise is weakest during plantar flexion because the narrow posterior wedge shape of the talus causes the joint to loosen. He continues by stating that the mortise is strongest in dorsiflexion because the wide anterior



Talus positiong between the malleoli.

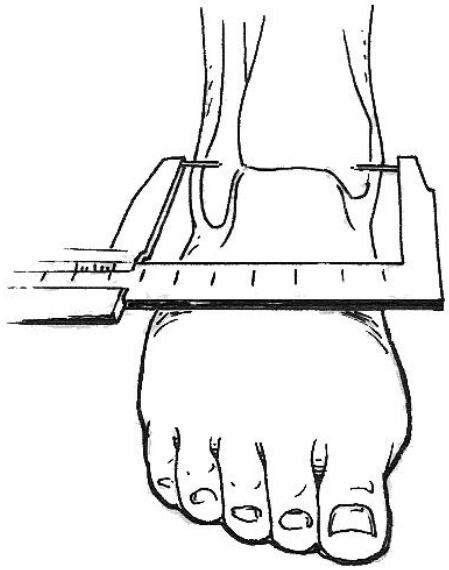
wedge of the talus tightens between the medial and lateral malleolus. This is applicable when one examines the ankle mortise by motion palpation when the ankle is non-weight-bearing and movement is passive. When ankle motion is active, especially when weight-bearing, there should be no difference in the articulation's integrity (joint play) in full plantar flexion or dorsiflexion.

Ankle Stability in Active Function

The usual and generally accepted description of human ankle mechanics is based on cadaver studies rather than how the ankle functions *in vivo*. The accepted description of ankle mechanics, based on cadaver studies, is summarized by Cailliet. **(Cailliet, 1997)** The body of the talus is wedge-shaped with the wider portion anterior. As the ankle dorsiflexes, this wider portion comes up between the two malleoli and wedges between them. Plantar flexion of the foot presents the posterior narrower portion of the talus between the malleoli and in this position permits some lateral motion of the talus within the mortise. This mobility creates instability of the joint and places an added burden on its supporting ligaments.

In vivo studies and additional functional consideration show that there is greater functional integrity of the ankle joint than is indicated in cadaver analysis. Weinert et al. **(Weinert et al., 1976)** have shown by high-speed photography of running, followed by cineroentgenography, that during weight bearing the fibula moves inferiorly on the talus to provide greater articular surface to the lateral aspect of the ankle mortise, providing greater stability. This inferior fibular movement is probably a result of the contraction of the peroneus longus and brevis, soleus, and tibialis posterior, all of which have origin or partial origin on the fibula and are active in plantar flexion.

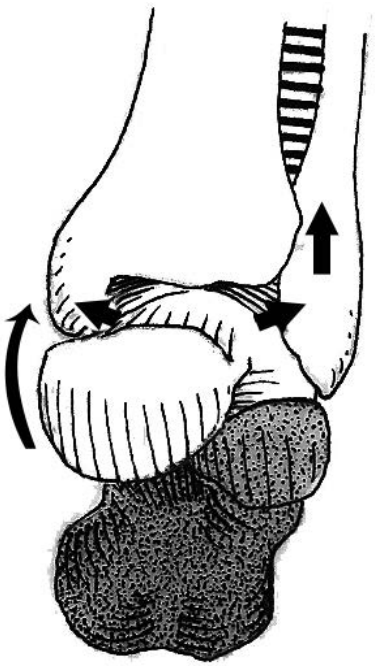
Further dynamic support to the ankle is provided by the arrangement of the tibialis posterior and its action during dorsiflexion and plantar flexion. **(Kapandji, 2010)** The origin of the tibialis posterior is from the lateral part of the posterior surfaces of the tibia and the upper two-thirds of the medial surface of the fibula. Contraction of the muscle pulls the tibia and fibula together because of the muscle's bipinnate arrangement. Since tibialis posterior action is plantar flexion with inversion, the ankle mortise narrows as the narrower portion of the talus body enters the mortise, especially when one is weight-bearing. Rising onto the toes requires strong action of the plantar flexor muscles. The tibialis posterior draws the tibia and fibula together, keeping the ankle mortise tight on the talar body. When the foot is dorsiflexed, the mortise must open to allow the wider portion of the talar body to enter. This occurs because the tibialis posterior is inhibited with dorsiflexion, which releases the approximation of the tibia and fibula.



With plantar flexion, the distance between the medial and lateral malleoli narrows.

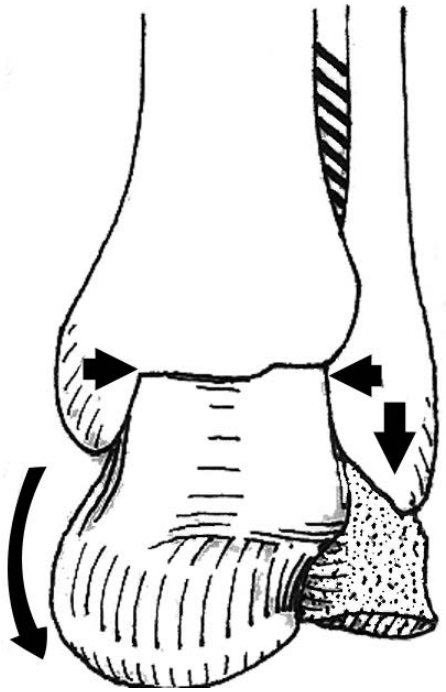
As the mortise widens to accept the wider portion of the talar body, the fibula moves slightly superiorly and finally rotates medially. **(Kapandji, 2010)** This changes the orientation of the interosseous ligament to horizontal and tightens it, limiting further dorsiflexion. **(Cailliet, 1997)**

The function of the tibialis posterior muscle in adjusting the tibia and fibula to the trochlear surface of the talar body may be augmented by a portion of the soleus. Michael and Holder **(Michael & Holder, 1985)** described an aspect of the soleus muscle that is not in standard anatomy texts. It is a separate and distinct bipennate portion at the anterior surface of the soleus, called the accessory fasciculus muscle. This was a consistent finding in 28 dissections by Michael and Holder. The soleus muscle has a U-shaped origin from the upper one-third of the fibula, the soleus line of the proximal tibia, and the posterior medial third of the tibia. With this origin, it is possible that the bipinnate section of the soleus acts to approximate the tibia and fibula in a manner similar to the tibialis posterior, as described by Kapandji. **(Kapandji, 2010)** This would provide additional strengthening of the ankle mortise as the wedge-shaped body of the talus moves with plantar flexion and dorsiflexion.



Dorsiflexion

The gastrocnemius and soleus have a greater role in ankle motion than is generally recognized in popular textbooks. The combination of these muscles with the Achilles tendon is called the triceps surae. The term should be quadriceps surae because the soleus functions as medial and lateral divisions. Campbell et al. **(Campbell et al., 1973)** did a fine-wire EMG study on the two heads of the gastrocnemius and the medial and lateral divisions of the soleus. The medial soleus stabilizes the leg on the foot and is a strong mover of the foot. The lateral soleus is not as powerful in these actions; however, it has an ongoing activity stabilizing the leg on the foot. When there is instability of the foot platform, activity of the lateral soleus increases.



Plantar flexion; fibula movement exaggerated.

The role of the soleus in stabilizing the leg on the foot requires understanding the soleus' contribution to the Achilles tendon. **(O'Brien, 2005)** Its tendon makes up the anterior half of the Achilles tendon, with the gastrocnemius contributing to the posterior half. As the Achilles tendon approaches the calcaneus, it rotates 90° so that the soleus portion inserts on the medial one-third of the calcaneus. The gastrocnemius portion inserts on the lateral two-thirds, causing the lateral portion of the soleus to be an inverter of the calcaneus. **(Michael & Holder, 1985)**

The two heads of the gastrocnemius are inactive until movement begins. **(Campbell et al., 1973)** The gastrocnemius is more sensitive to conditions of length, strength, and rate of contraction. The gastrocnemius restrains the tibia from rotating on the talus during gait as the bodyweight is shifted from the heel to the ball of the foot during the stance phase of the gait cycle. **(Travell & Simons, 1992)** The soleus has a more constant role. The gastrocnemius has its greatest activity when the ankle is plantar flexed, in large contractions, and in the rapid development of tension. The soleus is most active with the ankle in dorsiflexion and in minimal contractions, **(Herman & Bragin, 1967)** usually in controlling postural sway.

Ligament stretch reaction in AK: Preventing recurrence

Of interest is the reason an athlete will sprain an ankle with a turning activity that has been done thousands of times before with no trauma. A possible answer is failure of the soleus, gastrocnemius and tibialis posterior to act in the role of approximating the tibia and fibula during strong plantar flexion. Applied kinesiology

Bipennate fiber arrangement of the tibialis posterior muscle approximates the tibia and fibula during plantar flexion.

recognizes that these muscles are related with the adrenal glands. This is especially of interest in an athlete under the stress of the game but with adrenals incapable of meeting the body's needs. **(Wilson, 2002)** Roberts et al. **(Roberts et al., 1993)** report that overtrained male athletes had significantly increased cortisol levels. Goto et al. **(Goto et al., 2005)** have suggested that exercise-induced metabolic stress is associated with acute responses of growth hormone, epinephrine, and norepinephrine following resistance training. One sees this type of deficiency often in an applied kinesiology practice that focuses on the treatment of athletes. **(Maffetone, 1989)**

The ligament stretch reaction **(Walther, 2000)** is also related to stressed adrenal glands. This is a condition in which previously strong associated muscles test weak immediately after ligaments of the joint are stretched.

Schmitt **(Schmitt, 1977)** using applied kinesiology, designed a clinical study (n=16) to determine the reproducibility and apparent association of the ligament stretch reaction to the adrenal gland. The study consisted of stretching the ligaments of various articulations in the body, and then re-testing muscles associated with the articulation as well as general indicator muscles. The ligament stretch reaction was identified in three separate areas of the body to determine that the reaction was present generally rather than in any one area. After this was accomplished, various factors associated with the adrenal gland in applied kinesiology were evaluated by therapy localization. The reflexes examined were the neurolymphatic, neurovascular, meridian alarm point, and cranial stress receptors. A point -- such as the neurolymphatic -- was therapy localized, and the ligament stretch procedure was repeated, followed by the same muscle tests. If therapy localization to the adrenal reflex point abolished the ligament stretch reaction, there was probable adrenal involvement associated with the mechanical stretch to the ligament. After all reflexes were tested, the individual was asked to chew adrenal concentrate; its effect was evaluated by re-testing for ligament stretch reaction.

Following are the results of the nutritional administration and various reflexes tested in the study of sixteen individuals:

REFLEX POINT TL	ELIMINATED REACTION
Neurolymphatic reflex	16
Neurolymphatic reflex	6
Meridian alarm point (Circulation sex)	8
Cranial stress receptor	8
Adrenal concentrate	10

If therapy localization to the adrenal point listed abolished the positive ligament stretch reaction, it was listed as positive. Note that in 50% or more of the subjects, each reflex point or the nutrition cancelled the positive reaction, with the exception of the neurovascular reflex.

Treatment indicated by the positive tests was initiated. The ligament stretch reaction was removed with these approaches in fourteen of the sixteen patients. Two cases required specialized meridian therapy to abolish the positive ligament stretch reaction. "In all but one case, the symptoms of the patients were generally improved at the next office visit. In most cases, there was no recurrence of the ligament stretch/muscle weakness patterns. In the few where the pattern did recur, fewer reflex areas were found to be involved."

This association between ligament stretch and muscle weakness patterns has been presented in contemporary research by Solomonow; **(Solomonow et al., 2009, 1987)** additionally, the relevance of adrenal hormones (particularly their mineralocorticoid function) to ligament injury has been expanded in the applied kinesiology approach. **(Leaf, 2010; Maffetone, 1999)**

Durlacher **(Durlacher, 1977)** points out the importance of evaluating athletes for this reaction. A rapidly moving individual places strain on the ligaments, appearing to cause immediate weakening of the muscles supporting the articulation just when they are needed. Under these circumstances, weakening appears to be the same as that observed when the articulation is stretched and manual muscle testing is performed immediately afterward. It is possible that the weakened muscle could even be more significant in an athletic endeavor, because muscle demand occurs at the same time the ligament is being stretched.

Clinically it has been observed that patients susceptible to the ligament stretch reaction have exacerbations of symptoms when under considerable stress. Stress is cumulative, and can be classified as emotional, chemical, thermal or physical. The athlete has some -- and probably

all -- of these stress factors during competition. Clinical evidence shows that performance is superior, and injury less probable, when all factors known to influence the adrenal gland are functioning normally. This provides an opportunity for the stress of the endeavor to properly enhance performance and not be a possible cause of injury.

Recently Lever **(Lever, 2006)** evaluated 200 asymptomatic patients for the involvement of ligaments in many of the different joints of the foot. The research design consisted of spreading apart the ligament and then manual muscle testing 40 different muscles throughout the body to see how this inhibits or facilitates the remote muscles.

Twenty-one joints and ligaments were tested in these 200 patients and the specific correlations between these joints and ligaments and the muscles they affected were listed. Generally, the calcaneal ligaments were found to affect pelvic and lower limb muscles, while talar ligaments were more involved with neck, upper thoracic and shoulder muscles.

Lever suggests that "Because of the importance of foot proprioception and the foot's relationship to so many body problems from neurological disorganization to gait imbalances, fascial disturbances, and the inhibition of so many muscles when faulted, physical evaluation of patients should include more attention to the feet."

Sprieser **(Sprieser, 2002)** also reports an in-office clinical trial (n=50) where the ligament stretch reaction was present in every case that had confirmatory adrenal stress disorder. Each of the patients in this study showed a drop in systolic blood pressure from lying to sitting or sitting to standing, or a positive Ragland's sign. Nutritional support was needed in all cases to correct the ligament stretch reaction and included adrenal support with choline or adrenal tissue extract, and/or a low dosage of vitamin E from wheat germ oil or octacosanol.

Finally, Hansen **(Hansen, 1999)** reports on a case-series of 5 patients with a medically diagnosed mitral valve prolapse who were also found to demonstrate the ligament stretch reaction. On physical examination, those patients were found to be hypoadrenic. The patients were treated to stabilize the ligament stretch reaction. This included dietary measures such as eliminating stimulants like coffee, tea, cola and refined sugars. AK oral nutrient testing showed a need for adrenal gland nutritional support. Hansen suggests that a mineral imbalance due to depressed adrenal function may cause a systemic weakening of ligaments including those of the heart valves.

In this discussion Selye's observation about the adrenal gland's pervasive influence should be noted. "A general outline of the stress response will not only have to include brain and nerves, pituitary, adrenal, kidney, blood vessels, connective tissue, thyroid, liver, white blood cells and especially muscles, but will also have to indicate the manifold inter-relations between them." **(Selye, 1976)** The relationship of applied kinesiology to the adrenal glands and the endocrine system is one that creates success for the clinician where other manual modalities might fall short. Each of the endocrine organs has been given diagnostic tests, (muscle-organ-gland inter-relationships), therapeutic protocols, nutritional correlations, and treatment monitoring methods. The endocrine glands are of course controlled by the nervous system, and this is why chiropractic and the other manipulative professions have been helpful throughout their history for endocrine-related disorders. **(Masarsky & Masarsky, 2001)** From the diagnostic viewpoint the AK manual muscle test has significance because it makes possible the detection of a "dis-ease" process affecting the ligamentous system in advance of the emergence of symptoms. Whether the muscle inhibitions we find on AK MMT related to the articulations of the lower extremity are primary (as in a postural subluxation) or of secondary reflex origin (as in a ligamentous disturbance due to impaired adrenal gland function), we must recognize that this component in the musculoskeletal system's function is a contributing, exacerbating, and perpetuating influence, that must be specifically diagnosed and given effective treatment regardless of the primary etiology.

First, the muscle to be tested after the ligaments are stretched must be evaluated to determine that its functional quality during a manual muscle test is normal. If the muscle tests weak in the clear, it should be strengthened with the appropriate treatment. Its strength can then be compared after the ligament stretch has been done. If general testing of the body is to be done for the ligament stretch reaction, another general indicator muscle can be chosen.

The stretching procedure should be designed to limit the stretch to the ligaments of the articulation as much as possible as was done in the Lever study described previously. **(Lever, 2006)** This can be done by tractioning the articulation, or attempting to move it in a direction of which it is usually incapable, such as attempting to laterally bend the knee. When attempting to stretch the ligaments, care must be taken not to go through a range of motion which stretches the articulation at the end of the motion; this stretches the muscles as well as the ligaments. A positive test may therefore be for the AK muscle stretch reaction or the ligament stretch reaction.

Care must also be taken that the weakening observed in a muscle is not the result of challenging the articulation; this could be erroneously interpreted as ligament stretch reaction. When there is a ligament stretch reaction, it will be in any direction the ligaments are stretched. In challenging the articulation, one vector of force will weaken the muscle while an opposing vector will strengthen it.

Correction. The therapeutic effort is directed toward support of the adrenal gland. The appropriate reflexes and muscles described in previous applied kinesiology literature should be evaluated and corrected, if involved. **(Leaf, 2010; Garten, 2004; Frost, 2002; Gerz, 2001; Walther, 2000; Goodheart, 1976)** Nutritional support -- usually in the form of adrenal concentrate -- is frequently of value. **(Wilson, 2002; Goodheart, 1973)** Evaluation of stress and the entire endocrine system is important in treating the functional hypoadrenic.

As well as examining the ankle muscles for function with the various means described in this text, one should also evaluate the adrenal and its function, especially under stress.

Foot and Ankle Motion

The structurally sound foot has all metatarsal heads resting on the ground while maintaining good longitudinal and transverse metatarsal arches with a neutral calcaneus position. **(Langer, 2007; Lee et al., 2003; Cailliet, 1997)** A variety of motion is necessary between the hindfoot and

forefoot to meet the demands of adjustment during foot action. The foot's numerous articulations have excellent architecture that meets this demand. It is complex but paradoxically simple when each component is considered individually. The important factor is that each simple component must be functioning properly for ideal foot-ankle action.

Movements within the foot are rotations about axes, with the axis located at the articulation; thus the motions in the foot are ginglymus or hinge-type. This causes predetermined motion of each joint, as opposed to a ball-and-socket joint motion in which the plane of movement is determined by the direction of forces acting upon it. **(Levangie & Norkin, 2001; Root et al., 1966)** The predetermined plane of movement in foot articulations is even applicable in the ball-and-socket joint of the talonavicular articulation. In function this joint is half of two different joint complexes. With the talocalcaneal articulation, it forms the talocalcaneonavicular joint complex. The combination of the talocalcaneonavicular and the calcaneocuboid articulations forms the mid-tarsal joint complex, called Chopart's joint. **(Gray's Anatomy, 2004; Jaffe & Laitman, 1982; Elftman, 1960; Hicks, 1953)** The talonavicular hinge movement is different, depending on which of the joint complexes moves. If action takes place at both complexes, there is yet another axis of hinge movement. The calcaneocuboid articulation is slightly saddle-shaped, giving two axes of motion. **(Gray's Anatomy, 2004; Elftman, 1960)** The axis of its movement also depends on whether one or both joint complexes are moving.

The complex arrangement of ginglymus joints between the talus, calcaneus, navicular, and cuboid bones produces the two important movements of the foot: inversion-eversion and pronation-supination. These two movements allow the foot to accommodate to the shape of the substrate and to lock together or unlock the tarsal bones, either forming a stable platform or creating a resilient lever. **(Levangie & Norkin, 2001; Hertling & Kessler, 1996)**

It is easily observed that when the foot pronates, the leg rotates internally; when it supinates, the leg rotates externally. The interdependent relation of leg rotation and foot pronation can be observed by attempting to extend one's toes during weight bearing without allowing external leg rotation. This relates to Hicks' **(Wallden, 2010; Hicks 1954, 1953)** windlass mechanism of the plantar fascia; the toes can easily be extended with external rotation of the tibiotalar column because the fascia is released. With internal rotation of the column, the arch lowers and the fascia becomes taut; toe extension is limited. Balanced weight bearing on the forefoot and hindfoot must be considered in this activity. The toes will rise fully if the weight is shifted to the hindfoot, because there will be minimal weight on the arches and they will easily rise. If the toes rise easily without external leg rotation or shifting one's weight to the hindfoot, the plantar fascia is not providing optimal support and control of the longitudinal arches. This is probably due to midfoot breakdown with plantar fascia stretching. **(Morley et al., 2010)**

Putting further demand on proper foot motion is the internal and external rotation of the pelvis and leg during gait. The foot does not rotate significantly in relation to the substrate during gait. This motion is taken up in pronation and supination. The relationship of leg rotation with pronation and supination was dramatically demonstrated by Inman **(Inman et al., 1981)** by inserting pins in the tibia and midfoot so that the movement could be visualized.

Lundberg et al. **(Lundberg et al., 1989, part 1-3)** studied this motion by roentgen stereophotogrammetry. The subjects had at least three radiopaque markers put into each of the tibia, talus, calcaneus, navicular, medial cuneiform, and 1st metatarsal bones. They stood on a platform capable of tilting the foot in pronation and supination. X-rays were taken in 10° steps, from 20° of pronation to 20° of supination; the three-dimensional motion was calculated from the resulting x-rays. All bones contribute to pronation and supination. The greatest amount is at the talonavicular articulation, with less rotation between the calcaneus and talus. Rotation in the transverse plane at the talocrural joint did not exceed 2°, showing that no significant varus/valgus instability occurred at that location.

There must be structural integrity between the foot and leg that still adapts effectively to the needs for foot flexibility and rigidity. The joints allowing inversion-eversion and pronation-supination give this ability in a manner similar to a universal joint. The standard mechanical universal joint consists of two forks interconnected by a central piece of crossed arms. Each fork is attached to a shaft or similar device for potential rotation. The purpose of the universal joint is to transmit rotation in exact ratio from one rotating member to the other, but in different axes; in other words, it enables rotation to be transmitted around an angle. When the foot rotates in pronation and supination, motion is imparted to the tibiofibular column, but not in the exact ratio as with a standard universal joint. For every degree of foot supination, there is an average of 0.44° of external rotation of the tibia. This is accomplished by the distal fork of the universal joint changing configuration through its range of motion. This distal fork consists of the calcaneus, the cuboid, and the navicular. Together with a central piece, the talus, it constitutes a three-arm link system that explains the configurational change. **(Levangie & Norkin, 2001; Hertling & Kessler, 1996; Olerud & Rosendahl, 1987)**

Universal joint.

Joint action describing motion from the leg to the foot is sometimes described as an oblique hinge, which is an inadequate description. **(Engsberg & Andrews, 1987)** Rotation of the subtalar and transverse tarsal joints is interdependent. **(Levangie & Norkin, 2001; Wright et al., 1964; Manter, 1941)** Normal movement of all the joints is necessary for full range of motion. From a chiropractic and

manipulative point of view, fixation of one joint will disrupt normal activity of the other joints. It is important to have the considerable range of pronation and supination for the foot to adapt to body movements and the substrate without imparting great motion to the leg. What motion is normally imparted to the leg can easily be adapted to by rotation at the hip joint, under normal conditions. The mechanism may have greater demands placed upon it by congenital anomalies. For example, when there is a tibial varus or valgus deformity, the joint complex of the foot must compensate. **(Lee et al., 2003; Rothbart & Estabrook, 1988; Ting et al., 1987)** Another example of this interaction is when ankle range of motion is limited by inhibited musculature; many differing compensation patterns have been shown to occur, such as genu recurvatum, early knee flexion, early heel lift or excessive pronation at the subtalar joint. **(Prior, 1999)**

Since the foot must have flexibility or rigidity under different conditions, its action is complex; this makes clear comprehension of foot function difficult. Sarrafian's **(Levangie & Norkin, 2001; Sarrafian, 1987, 1983)** twisted plate model helps explain the mechanism under different circumstances. Here the forefoot is in the transverse plane of the plate, and the hindfoot in the sagittal plane. The joint architecture, ligaments, and aponeurosis are responsible for elevation of the medial longitudinal arch with certain motions and, conversely, arch depression with contrary motions. Additional twisting of the footplate causes the forefoot portion to further pronate in relation to the midfoot to stay horizontal with the substrate; the hindfoot supinates (varus) and the arch rises. In this process, the footplate shortens. If the footplate is untwisted, the forefoot must supinate to stay parallel with the substrate. The hindfoot pronates, the arch drops, and the foot lengthens.

As noted, with the foot in contact with the substrate, the medial longitudinal arch rises and lowers with external and internal leg rotation, respectively. This is due to the architecture of the joint angles and their ligamentous integrity. With external rotation the navicular and calcaneus supinate, and the distal forefoot pronates in relation to the rest of the foot to maintain its contact with the substrate. This causes elevation of the medial longitudinal arch and shortening of the foot, as indicated by the plate model. Conversely, with internal rotation of the tibiotalar column the navicular and calcaneus bones pronate, and the distal forefoot supinates to maintain contact with the substrate; otherwise, the lateral portion of the forefoot would lift from the substrate. The medial longitudinal arch lowers, and the foot elongates.

For optimal function, the components of the foot must be aligned so the axis of the talus body points to the 2nd/3rd toe interspace. This affects axial loading of the metatarsals. "If the axis of the talus points more medially, the foot is described as metatarsus abductus. If the axis of the talus points more laterally, the foot is described as metatarsus adductus." **(Hamilton & Ziemer, 1983)** Abnormal loading of the metatarsals can cause pain in the proximal or distal forefoot.

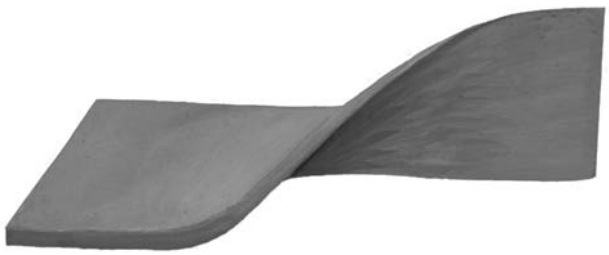
As a result of the oblique orientation of the subtalar axis of rotation, torsion of the tibia changes the forces in the foot. If external tibial rotation puts force into the foot, weight is shifted to the lateral four metatarsals. Similarly, internal tibial rotation causes increased weight on the 1st metatarsal. **(Morley et al., 2010; Jones, 1945)**

Normally the rotator muscles of the thigh are balanced when the foot is pointed approximately 10° outwardly. One can evaluate this muscular balance by pointing a foot directly forward and then standing on that leg only. Normally, when standing on the right foot, the body will rotate to the left approximately 10° to the neutral position of the hip rotator muscles.

There is a 2:1 ratio of inversion to eversion; thus if there is a normal 30° of subtalar joint motion, 20° would be inversion and 10° eversion. **(Brody, 1980; Subotnick, 1975)** In impairment rating, 30° of inversion and 20° of eversion are considered normal. **(American Medical Association, 2007)** This range is estimated rather than measured with a goniometer. Cailliet **(Cailliet, 1997)** recommends evaluating subtalar motion with ankle dorsiflexion to lock the mortise.

Plantar flexion and dorsiflexion.

The greatest motion in plantar flexion and dorsiflexion is at the talocrural articulation, with additional motion contributed by the joints of the longitudinal arches. This participation is in varying degrees. The distribution of motion between the joints of the arch in plantar flexion



The flat portion of the rectangular plate represents the forefoot, the twisted portion the arch, and the vertical portion the hindfoot.

If the plate's twist increases, the hindfoot must supinate the forefoot to remain horizontal. The arch rises and the the plate shortens.

If the plate's twist decreases, the hindfoot pronates for the forefoot to remain horizontal. The arch lowers and the plate lengthens.

varies greatly among subjects, with some subjects obtaining 40% of plantar flexion in these joints. In dorsiflexion, participation of the joints of the arch is small in normal subjects. **(Kitaoka et al., 1995; Lundberg et al., 1989)** Under certain circumstances there can be excessive dorsiflexion contributed by the longitudinal arches. This abnormal condition is often part of the extended pronation complex. **(Discussed later).**

Foot Reflexes and Reactions

There are numerous reactions that result from stimulation to the foot nerve receptors. The reactions discussed here are those presented in the general anatomical and physiological literature. One can wonder what future research will bring. With AK examination, correlations are observed that provide excellent opportunities for basic research. An example is Colum's observation of an apparent interaction between ankle dysfunction and carpal tunnel syndrome, an observation confirmed by Mondelli & Cioni. **(Mondelli & Cioni, 1998)** Colum observed in recurrent carpal tunnel syndrome that an interactive AK treatment to the contralateral ankle eliminated the recurrent nature of the carpal tunnel syndrome. **(Colum, 1983)** This interaction is probably on a ligament interlink basis.

The study of Lever discussed earlier confirms interactions between ligaments and muscles. **(Lever, 2006)** His study showed the relationships between the AK sensorimotor challenge to specific ligaments in the foot and muscular impairments resulting throughout the body.

The response to stimulation of foot nerve receptors is an important function to provide for protection. Stimulation of the nociceptors of the lower limb causes coordinated movements of withdrawal. **(Kugelberg et al., 1960)**

Afferent input from the sole of the foot affects postural awareness significantly. In fact sensory receptor information plays numerous roles in creating motor responses. **(Holm et al., 2002)** Cutaneous reflexes from the foot are important to posture and gait. **(Kavounoudias et al., 2001)** Disturbed sensory supply from cutaneous receptors in the feet alters the strength, timing, and velocity of muscle activation as well as altering gait. Lower limb sensory supply alone provides enough information to maintain upright stance and are critical in perceiving postural sway. In addition, movement discrimination in the ankle is better barefoot when compared to wearing shoes **(Waddington & Adams, 2003)**

There are specific reflex patterns for each skin area stimulated that make up an elaborate mechanism. The appropriate withdrawal movement is obtained by integration of flexion and extension reflexes. There is certainly nothing 'primitive' about such a highly purposeful reflex system. **(Holm et al., 2002; Kugelberg et al., 1960)** The highly selective reaction from stimulation to the ball of the foot evokes plantar flexion of the toes; stimulation a few millimeters away produces dorsiflexion. These responses are based on function and are not anatomical. They are responses of multisegmental reflexes where two skin points may be innervated by the same spinal segment and the same cutaneous nerve, yet the responses are the reverse of each other.

Positive support reaction

One tends to think of posture only in a living, dynamic sense. A dead body also has posture — the position gravity impresses on it with no counteracting forces. Sherrington **(Denny-Brown, 1979)** states, "Active posture largely encompasses the counteraction of those effects which gravitation, etc., produce in the dead body. Active postures may be described as those reactions in which the configuration of the body and of its parts is, in spite of forces tending to distract them, preserved by the activity of contractile tissues, these tissues then functioning statically." The tissues functioning statically refer to the muscle turgor, sometimes called tonus. In addition, one should consider the bones and ligaments of the skeleton. When the bones are properly formed and tied together with ligaments, there is strong compressive structure, as demonstrated by the foot's structural integrity. As we will see later, many of the muscles thought to be constantly active in maintaining balanced posture are inactive, as observed by electromyography. It is when there is postural imbalance that the "postural muscles" are active. In normal, balanced posture the body has an economy of muscle function, and balance is maintained with only short, slight twitches of muscle activity.

When the substrate angle changes under the foot, remote muscles contract to maintain balanced posture. When body sway is induced from above, the muscles contract in a different order to maintain posture. **(Fink et al., 2003; Nashner, 1977)** This indicates fixed patterns of muscle organization, with control coming from the foot and many other equilibrium proprioceptors. Excitability of alpha-motoneurons throughout the body is influenced by the supraspinal motor centers of the CNS, segmental spinal interneurons, rhythmic movement pattern generators, involuntary reflexes and sensory input from proprioceptors in muscle, articular structures, and skin. Once again, the diversity of sensorimotor activity going on in the patients being assessed by the clinician requires a system of evaluation that can encompass these various sources of neural activity quickly and reliably: the manual muscle test and the AK sensorimotor "challenge" and "therapy localization" procedures offer this kind of assessment technology.

The positive support reaction, first observed in decerebrate animals, is a basic reflex of posture. When pressure is applied to the plantar surface of the foot, the limb extends strongly enough to support the animal's body weight, leaving the animal standing in a rigid position. **(Hodgson et al., 1994; Langworthy, 1970)** The pathways involve complex circuits in the interneurons, similar to those responsible for the flexor and crossed extensor reflexes. The reaction was originally called the "magnet reaction" or "extensor thrust reaction"; now it is often referred to as the "positive support reaction." **(Guyton & Hall, 2005)** The term "magnet reaction" was used by

early experimenters because when pressure was applied to an animal's foot, it followed the examiner's withdrawing hand as if it were attached to a magnet. The term has no reference to electromagnetic activity within the body. Ruch **(Ruch, 1979)** acknowledges that the reaction is more readily demonstrated in a decerebrate preparation, but it is present in normal animals and in man. In the spinal animal the reflex is so sensitive that it can be elicited by simply touching the sole of the foot.

The positive support reaction does not come solely from the myotatic reflex since there is inadequate time for response to the muscle stretch. **(Vedula et al., 2010; Guyton & Hall, 2005)** It appears to come from a combination of stimulation to muscle, joint, and cutaneous mechanoreceptors in the foot. These combine to form a complex union of stimuli to provide facilitation and inhibition of muscles contributing to posture and gait. When the foot is weight-bearing, the force to the interphalangeal joints and stretching of the interosseous, adductor hallucis, and other muscles stimulate the joint receptors and muscle spindles to provide facilitation of the postural extensor muscles, which is the positive support reaction. On the other hand, if the phalanges and metatarsals are squeezed together or flexed instead of being spread apart, there is inhibition to the extensors and all joints of the extremity flex. **(Gowitzke & Milner, 1980; O'Connell & Gardner, 1972)** This takes place if the foot is functioning normally. With many types of foot dysfunction, the positive support reaction fails to function as noted, and apparent confusion develops in the nervous system. Stimulation to the receptors as stated will be used in the applied kinesiology examination of joint neurologic functions.

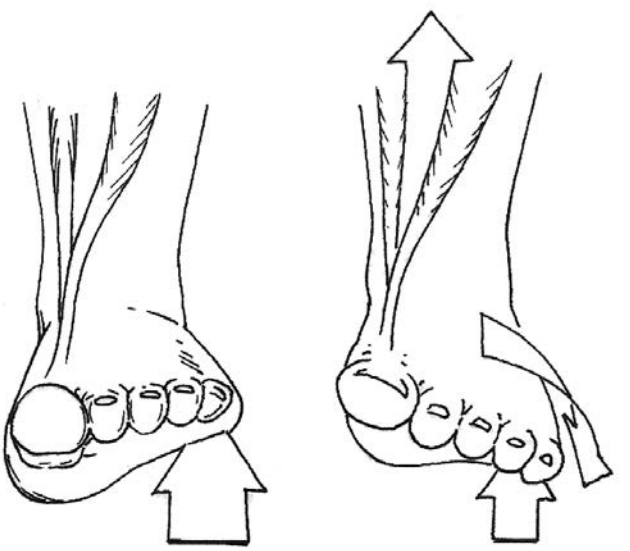
O'Connell **(O'Connell, 1971)** describes an experiment that has now been done in many physiology laboratories to demonstrate the positive support reaction. She used a swing arranged so that it could be raised and lowered various distances from the floor. The subject sitting in the seat of the swing was raised and lowered, and the seat was randomly tilted to dump the subject toward the floor. The individual landing on his feet maintained an upright posture. After repetitions of the procedure, the individual was blindfolded and the procedure repeated with enough swinging and random elevating and lowering to disorient the subject. Again, random spilling dumped the individual toward the floor. Correct posture was maintained when he landed on his feet, but with greater difficulty and more slowly than when not blindfolded. Finally, the individual's feet were immersed in iced water for a period of twenty minutes to produce local anesthesia, and the blindfolded procedure was repeated. In this instance, the individual was unable to maintain an erect posture and crumpled to the mat. The conclusion, of course, is that chilling of the feet interfered with proprioceptive communication to the body; consequently, there was no facilitation of the extensor muscles.

The positive support reaction, in addition to facilitating upright posture, provides nerve impulses to control lateral sway. This, along with the equilibrium proprioceptors, keeps sway to a minimum in so-called static stance. **(Guyton & Hall, 2005; Hellebrandt & Braun, 1939)**

Location of pressure on the sole of the foot determines the direction the foot turns. **(Guyton, 2005)** In a simple manner, this is observed in the magnet reaction described above. If the experimenter presses flat on the decerebrate animal's paw, there is straight extension. If pressure is applied on one side of the foot, the foot moves toward the source of stimulation in an effort to return balance to the foot position. **(Guyton, 2005)** The ability to maintain balance is enhanced by these reactions, particularly in the biped. A simplified example is seen when one sways to the left; there is adduction of the left femur and abduction of the right one, with eversion of the left foot and inversion of the right one. **(O'Connell, 1958)** With the sway there is stimulation to the muscle, joint, and cutaneous receptors to return the body from lateral sway to neutral. These impulses are interpreted to cause facilitation of the left foot inverter muscles, such as the tibialis anterior. At the right foot and ankle, there is facilitation of the peroneus group to cause eversion. At the pelvic and thigh levels, there is facilitation of the left gluteus medius and tensor fascia lata to cause left hip abduction. There is also facilitation of the right adductors to cause hip adduction. This muscular action brings the body back to neutral from its lateral sway. **(Gowitzke & Milner, 1980)**

Foot dysfunction has been statistically correlated with hip abductor and adductor muscle weakness, as measured by the Cybex II dynamometer. **(Gleim et al., 1978; Nicholas et al., 1976)** The term functional ankle instability is frequently used for recurrent ankle sprains, and functional ankle instability is associated with arthrogenic muscle weakness that occurs throughout the body, including the peroneal muscles of the ankle, **(Palmieri-Smith et al, 2009; Tropp, 1986)** the tensor fascia lata muscle of the hip -- after the AK shock absorber test was administered to the feet **(Zampagni et al., 2009)** -- and in many other muscles throughout the injured limb.

(With kind permission, ICAK-USA)

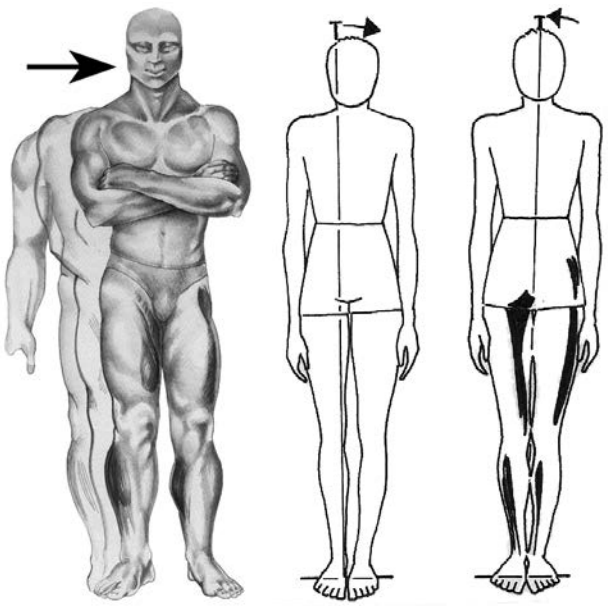


Stimulus ***Reaction pushes against the stimulus.***

The evidence now shows with greater clarity than ever before that inflammation or injury most frequently produces specifically identified inhibited muscles.

Controlled clinical studies have shown that dysfunction and pain specifically in the ankle, **(Nicholas & Marino, 1987)** knee, **(Slemenda et al., 1997; Stokes & Young, 1984)** lumbar spine, **(Hossain & Nokes, 2005; Hodges & Richardson, 1996)** temporomandibular joint, **(Zafar, 2000)** and cervical spine **(Cuthbert et al., 2011; Jull, 2000; Vernon et al., 1992)** will produce ***inhibited muscles***. These data indicate that the body's reaction to injury and pain is ***not increased*** muscular tension and stiffness; rather ***muscle inhibition is often more significant***. The use of a clinical tool like the manual muscle test is uniquely designed to detect this important neuromuscular impairment in patients with lower extremity dysfunction.

It appears that almost any type of dysfunction — including subluxations, fixations, extended pronation or intrinsic muscle problems — can cause poor or disorganized remote muscle function. **(Zampagni et al., 2009)** In the first decade of applied kinesiology, Goodheart **(Goodheart, 1973)** reported that Edward Doss, Sr., D.C., of Stuttgart, Arkansas, told him of his frequent observation that a lateral cuboid subluxation correlated with a tensor fascia lata muscle that tested weak. Correction of the subluxation returned normal function to the muscle, as observed by manual muscle testing. Goodheart concurred with the observation, and also found that adjusting the medial transverse arch (medial cuboid) often corrected adductor muscles that tested weak. This logically fits very well with the available information about the magnet or placing reaction. Lateral sway of the body that causes inversion and eversion of the foot to regain balance would stimulate the cutaneous and joint receptors. A medial or lateral subluxation of the transverse tarsal bones would be equivalent to body sway as far as the joint receptors of the cuboid would be able to discern; consequently, impulses would be transmitted to facilitate and inhibit muscles to return balance to the body. Unfortunately, when this neurologic activity is due to a subluxation, it is inappropriate to body needs and creates further imbalance of function.



Muscle reaction to bring lateral sway back to balance.

Examination of the positive support reaction

Dysfunction of the positive support reaction can develop as a result of improper stimulation to the muscle, joint, or cutaneous receptors. Typically, but not always, when muscle receptors of the feet are at fault, the dysfunction will be in the extensor muscles. Joint receptors often disturb muscles of ab- and adduction and muscles of gait; cutaneous receptors relate primarily with local muscles and muscles of gait. There are many muscles that must have organized function during gait. **(Landry et al., 2010; Craik & Oatis, 1995; Smidt, 1990)** These include head-turning ones such as the sternocleidomastoid and upper trapezius, shoulder and hip muscles, and muscles of the sacrospinalis. Some dysfunction must be examined for by combination muscle tests, such as in applied kinesiology gait testing. **(Walther, 2000)**

Stimulation of the nerve receptors responsible for the positive support reaction appears to relate with stretching the intrinsic plantar foot muscles. The muscles stretched by weight bearing include the flexor hallucis brevis, abductor hallucis, adductor hallucis, flexor digitorum brevis, lumbricals, and dorsal and plantar interossei. The muscles that are longitudinal to the foot — such as the flexor digitorum brevis and lumbricals — are stretched when the longitudinal arch is flattened; the dorsal interossei and adductor hallucis, especially the transverse head, are stretched when the metatarsal arch flattens and spreads.

In normal weight bearing, when the longitudinal arches and metatarsal arch flatten, there is stimulation to the mechanoreceptors that causes facilitation to the extensor muscles of the body. **(Guyton & Hall, 2005)** The easiest method for evaluating the positive support reaction is with the patient prone, making it easy to evaluate extensor muscles such as the hamstrings, gluteus maximus, deep neck extensors, and upper trapezius. First the muscles should be tested to determine that they are functioning normally. If not, make corrections with the usual applied kinesiology approaches, such as correcting vertebral subluxations and reflexes. When the intrinsic muscles of the foot are stretched to simulate weight bearing, the previously strong postural extensor muscles should remain strong or even test stronger, because these muscles are normally facilitated by stimulation to the receptors of the positive support reaction. Simulation of weight bearing is done by flattening the longitudinal arches and spreading and flattening the metatarsal arch. Immediately after the challenge, one or more of the extensor muscles should be tested. A positive indication of foot involvement is weakening of the extensor muscle(s). Both the gluteus maximus and the hamstring muscles are pelvic extensors, but it is usually best to test the gluteus maximus; the hamstring muscles are both pelvic extensors and knee flexors in their two-joint function.

Another type of challenge for the positive support reaction that is sometimes done in applied kinesiology is squeezing the metatarsal arch together. This frequently shows a weakening of the extensor muscles with positive support dysfunction, but it is not as accurate a challenge as the spreading mechanism. There is a normal action of facilitation of the limb flexors when the phalanges and the metatarsals are squeezed

together or flexed instead of being abducted. **(Gowitzke & Milner, 1980)** When there is facilitation of the flexors, there is normally inhibition of the extensors on the basis of reciprocal inhibition. When there is improper stimulation to the positive support reaction mechanoreceptors, the resulting effect of the muscles is unpredictable. Sometimes squeezing the metatarsal arch and phalanges together will cause weakening of the extensor muscles. This may be due to challenging foot subluxations. Forces applied to the normal foot do not cause weakening of the extensor muscles.

When there is weakening of the extensor muscles after challenging the intrinsic foot muscles, treatment is directed to the muscles of the foot. Treatment necessary is usually to the neuromuscular spindle cell, Golgi tendon organ, fascia (fascial release), percussion, trigger point pressure release, and occasionally use of the origin-insertion technique. **(Leaf, 2010; Cuthbert, 2002; Walther, 2000)** The muscle dysfunction is usually secondary to a structural foot problem such as pronation (with or without a short triceps surae), metatarsal subluxations, or other structural foot problems that will be discussed later.

Often the method of flattening the longitudinal arches and spreading the metatarsal arch, then testing the extensor muscles, will provide an adequate examination. In some instances, this simulated weight bearing does not reveal the problem. If body language indicates improper function of the positive support reaction but the prone tests are negative, test the patient in a standing position. Information revealed in standing, sitting, supine and prone positions with manual muscle testing assessments may be combined with other AK assessment measures to reveal distinct patterns of muscular adaptation and/or structural imbalances. When these patterns are assessed and combined with the patient's symptomatology, habits of use, and pain pattern a clearer picture emerges as to what is dysfunctional and how to support the patient.

The standing assessment of the positive support reaction, of course, puts more strain on the longitudinal arches than is usually done in the previously described procedure, and it may cause the extensor muscles to weaken. In addition, the proprioceptors react differently to stimulation, depending on the limb's position at the time of stimulation. Feedback and feed-forward mechanisms regulate motor control by correcting movement after sensory supply has been received. They use closed reflex loops of proprioception from mechanoreceptors and muscles across the joints of the foot. **(Aniss et al., 1992)** Sherrington points this out, stating that the action taken "…is in part determined by the posture already obtaining in the limb at the time of the application of the stimulus." **(Denny-Brown, 1979)** It is usually easier to test the extensor muscles weight bearing, with the patient leaning face forward on an upright hi-lo table, as in this position the positive support reaction is being tested by the patient's positioning.

Pronation

Many terms are often used synonymously to describe foot pronation, such as flatfoot, pes planus, fallen arches, flexible flatfoot, peroneal spastic flatfoot, rigid flatfoot, pes valgo planus, and on and on. **(Shibuya et al., 2010)** What is important is to understand the dynamics of foot function and what causes dysfunction. All too often symptoms in the feet are treated with no regard for the primary cause. Often this is due to the fact that the clinician does not possess a method for measurement or detection of primary causes. Once a foot dysfunction has been identified by virtue of a postural, palpation, orthopedic or other test, it is necessary to define precisely what type of dysfunction exists. The effect of this dysfunction upon other areas and symptom complexes is very important. The effect of this foot dysfunction upon attaching or remote muscle function is offered by the challenge and therapy localization procedures in applied kinesiology. The associated muscle weakness, easily determined by the manual muscle tests as outlined in this text, is then evaluated with the challenge procedure or therapy localization (the AK sensorimotor stimulus). Appropriate angular and pressure stimulation of the articulation produces immediate strengthening of inhibited muscles in remote body areas and symptom complexes due to the foot dysfunction(s).

Orthotics may be prescribed when muscle, joint, or neurologic corrections would more permanently and cost-effectively solve the problem. **(Page et al., 2010; Maffetone, 2003)** On the other hand, treatment by these methods may be applied when there is a congenital anomaly or some other condition requiring orthotics or surgery to correct the primary problem.

Furthermore, one must recognize the influence of the foot on remote body function and of remote body function on the foot. **(Marshall et al., 2009; Mattson, 2008; McVey et al., 2005)** In Lutter's **(Lutter, 1980)** sports medicine clinic, 76% of knee symptoms were related to pronation abnormalities. Structural strain, as in knee problems, is generally recognized; what is often missed is the disturbance to normal neurologic function that may cause dysfunction throughout the body. Researchers have found weakness and changes in muscle activation and velocity in the knee, hip, trunk, shoulders and neck in subjects with ankle instability **(McVey et al., 2005; Beckman & Buchanan, 1995; Bullock-Saxton, 1994)**

These findings point to the importance of manual muscle test examinations beyond the site of injury; the entire muscle system is vulnerable to aberrations in foot function. Applied kinesiology provides methods to find these types of remote dysfunctions and to specifically relate them to the foot for both the doctor and the patient using the sensorimotor challenge and therapy localization procedures.

Treating foot symptoms rather than correcting the cause may adversely affect another part of the body. It is only natural to think that a therapeutic approach that relieves foot symptoms is good for the body; this is not necessarily true. It is not unusual to find improperly fitted orthotics to be the cause of low back, shoulder, or other pain during examination of a new patient. Your credibility may be questioned when the patient says, "My feet have been better since I started wearing orthotics six months ago." Questioning the patient reveals that the back pain slowly developed about two weeks after wearing the new orthotics. Examination may find that the positive support reaction does not function properly when the orthotics are worn, causing the extensor muscles to test weak. Correcting

the back problem is accomplished by correcting the foot dysfunction and/or changing the orthotics. **(Sahar et al., 2007; Robbins & Hanna, 1987)**

When working with the feet, it is necessary to be capable of evaluating how remote areas of the body affect them, and also how any therapeutic approach applied to the feet affects the rest of the body. Proper therapeutics and correction enhance function in the rest of the body; improper therapeutics, even though providing relief in the feet, may cause remote disturbances that manifest new symptoms that the patient usually does not associate with foot treatment.

There are many therapeutic approaches to excessive foot pronation; all work to some degree. It is necessary to have a system that evaluates how treatment affects the foot, and also how the rest of the body reacts to the therapeutic approach. As will be seen, not only does applied kinesiology fill this need, it also enables the physician to determine how remote areas of the body may be causing foot dysfunction.

General use of the term "foot pronation" connotes abnormal position or function of the foot to many people. Actually, foot pronation during function is a necessary and important aspect of the gait cycle. Typically, reference to pronation with the patient standing alludes to excessive pronation or "flatfoot." When the term is used in the dynamic sense, it refers to "extended pronation," i.e., the foot comes out of excessive pronation too late in the gait cycle. In this text, when discussing abnormal conditions we tend to use the term "pronation" as a static pronated foot, "extended pronation" in reference to gait, and "excessive pronation" when relating to static and dynamic conditions.

Gait

Volumes have described the individual kinematics, movement patterns and mechanics of gait. **(Cailliet, 1997; Craik & Oatis, 1995; Smidt, 1990; Hoppenfeld, 1976)** The fundamental characteristic of human movement, walking, is a series of prevented catastrophes. Soderberg, Cailliet, Hoppenfeld, Slocum and James, and others **(Soderberg, 1997; Cailliet, 1997; Hoppenfeld, 1976; Slocum & James, 1968)** describe gait as a series of events consisting of the support and swing phases. There are three divisions of the support phase: 1) footstrike, 2) mid-support, and 3) take-off. A common term for the first phase of the support phase is "heelstrike," but there are many patterns of gait, especially in running, when the heel is not and should not be the first part of the foot to strike the ground. **(Abshire, 2010)**

Wallden **(Wallden, 2010)** has described the evolving literature on foot biomechanics indicating that running with a heel strike is not functional after all. In a review of the literature and mechanics on barefoot running and athletic performance, it may be that forefoot and midfoot strike is the natural, functional style of gait at footstrike. Heel striking can lead to many kinds of injuries.

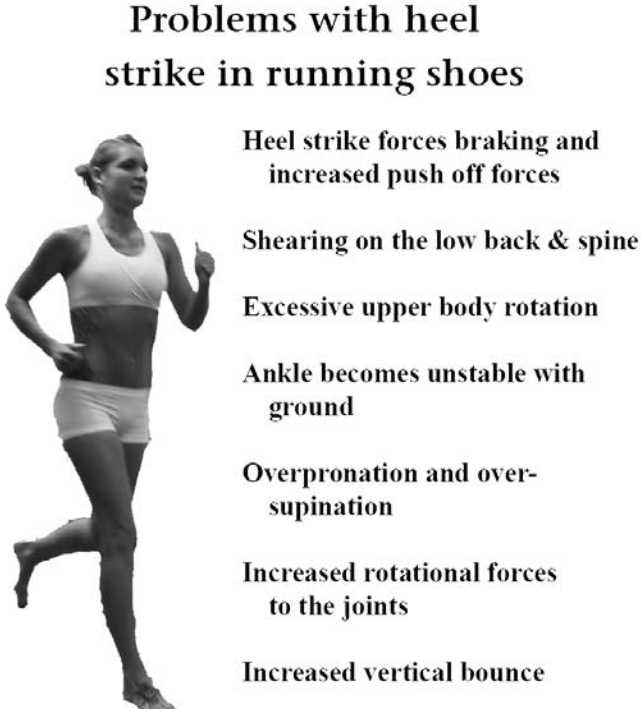
Heel striking during running forces the body to brake slightly thus requiring increased push off forces to maintain velocity. This produces shearing into the lower back and spine, and forces excessive upper body rotation. The ankle becomes unstable when adapting to the substrate. This produces overpronation and oversupination, increased rotational forces into the joints, and an increased vertical bounce with each foot strike. **(Abshire, 2010)** Generally speaking, modern running shoes are traditionally made with lifted heels. The high heel gets in the way of the foot landing parallel to the substrate.

The term "footstrike" is more inclusive for the first part of the support phase. **(Cavanagh, 1982)** Both footstrike and heelstrike will be used in this text; in general the two terms are synonymous primarily for shod feet, indicating the first part of the support or stance phase.

The swing phase of gait can be considered as forward recovery. It, too, is divided into three phases: 1) follow-through, which is immediately after toe-off, 2) forward swing, and 3) foot descent. **(Soderberg, 1997; Slocum & James, 1968)** Gait analysis should take into consideration the lower extremities, the pelvis, spine, head movement on the spine, and shoulder and arm motion. **(Walther, 2000)** Here we will consider only foot function in the stance phase of gait.

At heelstrike in the shod foot, the subtalar joint is supinated and the tibia is in external rotation. Rapid pronation begins as the foot comes into full contact with the substrate, with the subtalar joint moving into a neutral position and the tibia coming out of external rotation. Pronation acts as a shock absorber mechanism to the forces being put into the foot. Authors vary regarding which of the long muscles are active in supporting the medial longitudinal arch. The tibialis anterior and posterior and peroneus longus muscles may all support the medial longitudinal arch during pronation of normal gait, and especially in extended pronation. The joint configurations allow the foot to adapt to the underlying surface. The ligaments, joint constraints, and muscles combine to dissipate stress to the foot and body, similar to a flexible band. The tibia follows the internal rotation of the talus as pronation continues.

At 15-25% of the stance phase, the foot should begin to come out of pronation. At this time it starts to become a rigid



lever for the toe-off propulsive stage of gait. "The foot that is not re-converted into a rigid lever by 75% weight bearing is defined as having increased pronation." **(Lutter, 1980)** Seventy-five percent weight bearing is at approximately 25% of the stance phase. **(Ramig, 1977)** Up to this point the mid-tarsal joints should be unlocked to allow the foot to adapt to the terrain. At 25% of the gait, the foot should begin supinating to reach neutral at mid-stance (50-65% of stance), and then supinate to become the rigid lever for toe-off. The peroneus longus contributes by everting the foot and flexing the first metatarsal. **(Beardall, 1975)**

Pronation which lingers on beyond 15-25% of the stance phase of gait is abnormal and causes torques in the lower extremities which lead to an overuse syndrome. **(Subotnick, 1991, 1975)** As weight transfers to the metatarsal arch and the toes extend, the arch rises and the foot, as a rigid lever, obtains more support from the plantar aponeurosis, which is tightened by the windlass mechanism described by Hicks. **(Hicks, 1951, 1953, 1954, 1955)** Classically the windlass mechanism operates as the gait cycle moves from mid-stance to toe-off, when the toes move into hyperextension, ideally reaching 65 degrees of extension, and the plantar fascia is thereby drawn tight increasing the arch along the medial aspect of the foot creating a spring like mechanism to push the person forward as they toe-off.

The intrinsic plantar muscles are important in maintaining the arches in the dynamic foot. They must work harder in the foot that pronates excessively. **(Mann & Inman, 1964)** Unfortunately, entrapment of their nerve supply at the tarsal tunnel often accompanies extended pronation, causing atrophy of the intrinsic muscles so they cannot contribute to arch maintenance.

The medial longitudinal arch is important as a shock absorber. During the stance phase of gait it allows pronation from heelstrike to flatfoot. If pronation is extended, difficulties may arise in the midfoot, hindfoot, tibia, and knee from structural stress. **(Morley et al., 2010)**

The knee remaining too long in internal rotation is a good example of structural stress. Maximum knee extension during gait is present when the center of gravity is over the foot, causing strain if the tibia is still in internal rotation. Increased subtalar joint pronation has also been identified as a contributing factor in patellofemoral pain. **(Crossley et al., 2006)** In addition to structural stress, other remote problems may develop from neurologic disorganization due to improper stimulation of the foot mechanoreceptors during extended pronation. Foot pronation during gait, then, is not bad; it is necessary and useful as a shock absorber mechanism. It is when supination and transformation of the foot to a rigid lever are delayed that problems develop.

Although it is important to treat extended pronation, care must be taken not to overcontrol it with orthotics. Pronation in the amount of 4° provides the shock absorption necessary and accommodates the internal rotation of the leg. **(Subotnick, 1975)** Artificially reducing this normal pronation decreases the foot's ability to act as a shock absorber and limits its adaptability to the changing substrate surfaces. Overcorrection of pronation may cause foot or remote problems revealed by symptoms and by applied kinesiology examination.

Many factors are responsible for abnormal foot function. Authors have individually stressed improper bony architecture, poor ligament integrity, or muscle and fascia dysfunction as causes of extended pronation. All these factors come into play, either singly or in combination. If the initial cause is only one component, the other two will probably be secondarily involved if the condition is allowed to continue.

A flatfoot is classified as dynamic or static. The dynamic flatfoot has a normal arch when non-weight bearing and a flat one when weight bearing. This is observable by x-ray. The static flatfoot does not change appreciably from non-weight bearing to weight bearing. The abnormality is present on x-ray, whether weight bearing or not. A normal arch can be formed in the dynamic flatfoot by digitally molding the foot. In the static flatfoot, a normal arch cannot be formed by the examiner's efforts. **(Subotnick, 1991, 1975)**

Etiology

The etiology of flatfoot can be congenital maldevelopment. There are several gross congenital variations that are usually diagnosed and treated in infancy or early childhood. Those conditions — most often treated by orthopedic surgeons — are out of the scope of this text. Full descriptions of them are found elsewhere. **(Galois et al., 2002; Hamilton, 1985; LeNoir, 1982; Tachdjian, 1982)** Here we will deal with the less severe congenital deficiencies that can be managed with conservative treatment, and conditions that develop from misuse and trauma.

Family history provides a good indication of the possibility of congenital flatfeet in children that is not of the severe type. **(Harris, 2010; Cobey, 1958)** Family members with hypermobility and a tendency to an everted weight-bearing position of the entire foot are examples. As a child develops, flatfoot becomes more apparent. As he first begins to walk, the flatfoot becomes easily recognizable.

Congenital anomalies must be put into proper perspective as the etiology for foot dysfunction. They can be overemphasized as the cause of the patient's complaints. Dysfunction, when attributed to a congenital anomaly, may cause the physician to cease efforts to obtain functional correction and rely only on orthotics, shoe correction, and life-style change; much better function could be obtained if dysfunctioning joints, muscles, ligaments, fascia, and skin were examined and corrected. On the other hand, one may fail to observe a congenital anomaly and persist in trying to obtain correction by these methods when an orthotic, shoe correction, or surgery would be more appropriate.

Short triceps surae

The short triceps surae is often referred to as a short Achilles tendon, equinus foot, gastrocnemius equinus, or hypermobile flatfoot with short Achilles tendon. Emphasis is placed on the Achilles tendon when, in reality, it is more often the muscle bellies that are short. Ryerson **(Ryerson, 1948)** points this out in his objection to surgically lengthening the Achilles tendon, stating, "The tendo achillis is not short; the muscle bellies are short, but the tendo achillis is long enough." His treatment approach is directed

toward lengthening the muscle. Rarely is it necessary to surgically lengthen the Achilles tendon. **(Greenhagen et al., 2010; Logan, 1995; Lillich & Baxter, 1986)** Of additional concern are the associated complications of any surgical intervention. The risk of any operative procedure is estimated to be as high as 5% with infection and wound breakdown being the predominant concerns. **(Soma et al., 1995)** Pulmonary embolism and sensory deficits due to injury to the sural and other cutaneous nerves can also occur. **(Fierro & Sallis, 1995)**

The triceps surae is composed of the gastrocnemius, the soleus, and their shared tendon. Its length must be adequate to allow at least 10° of dorsiflexion at the ankle. **(Wright et al., 1964)** A short triceps surae was associated with flatfeet in early observations. Royal Whitman, **(Whitman, 1970)** in his classic 1888 presentation on flatfeet, observed "…that there seemed to be an abnormal resistance in the calf muscle…."

Limitation of dorsiflexion can be congenital or acquired. When the triceps surae is congenitally short, the deformity is called talipes equinus. Gastrocnemius equinus is a lack of 10° of dorsiflexion at the ankle with the knee flexed. Gastrocnemius-soleus equinus is a lack of 10° of dorsiflexion with the knee both extended and flexed. The flatfoot associated with short triceps surae is dynamic; that is, the arch appears normal when non-weight bearing and flat when weight bearing.

There are many methods in applied kinesiology that lengthen shortened or hypertonic muscles. Treatment, such as percussion, fascial release or stretch and spray, may be locally directed to the muscle or to the muscle proprioceptors. Remote treatment of many types increases the range of motion throughout the body. The patient should be examined for conditions such as dural tension and modular disorganization, and treated with PRYT, cloacal synchronization, and gait techniques. The initial AK treatment generally increases range of motion; stretching exercises, if needed, provide much better results when the AK procedures are done first.

If there is not 10° of ankle dorsiflexion, compensation must take place elsewhere during the mid-stance phase of gait. **(Craik, 1995; Nicholas & Marino, 1987; Subotnick, 1971)** Stress to the foot develops when the last amount of dorsiflexion needed in walking is obtained at the subtalar and mid-tarsal joints, rather than at the ankle mortise. **(Milgram, 1983)**

As body weight is forced into the midfoot, compensation takes place by pronation of the subtalar and mid-tarsal articulations. In order to compensate for the lack of ankle dorsiflexion, these joints must pronate more than normal, causing the ligaments to stretch and flexible flatfoot to develop. This breakdown of the midfoot is to accommodate the short triceps surae.

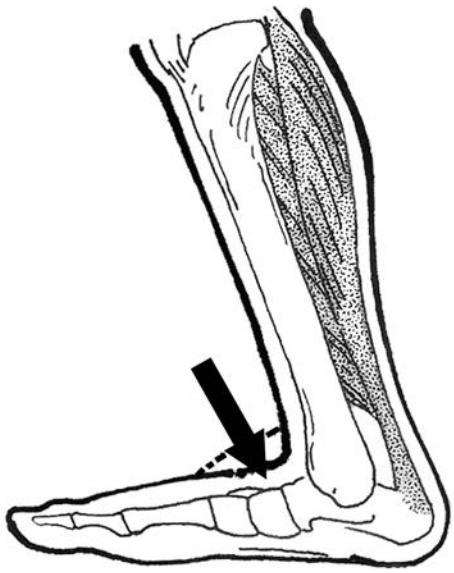
When compensation does not take place by midfoot breakdown, it may occur by early heel-off, which happens when the heel leaves the ground before 65-75% of the stance phase is completed. **(Hamilton, 1985; Subotnick, 1971)** The propulsive phase of gait is thus lengthened, which puts more stress on the ball of the foot than it is prepared to accept. As a result, metatarsalgia, intrinsic muscle fatigue, and calluses develop. **(Nicholas & Marino, 1987)** Leaf and Mennell state that metatarsal head pain may be the result of triceps surae shortening. **(Leaf, 2010; Mennell, 1969)**

Major stress is put on the plantar fascia when there is no midfoot breakdown. Myers and Waller **(Myers, 2001; Waller, 1982)** describes the plantar fascia as an extension of the triceps surae-Achilles tendon complex. "The gastrocsoleus muscle complex, Achilles tendon, and plantar fascia should be viewed as a single linkage system." According to this analysis, tension is created in the plantar fascia from the posterior as one rises on his toes or otherwise contracts the calf muscles, and from the windlass mechanism described by Hicks. **(Hicks, 1951, 1953, 1954, 1955)** The strain can be great enough to rupture the plantar fascia. **(Kim et al., 2010)**

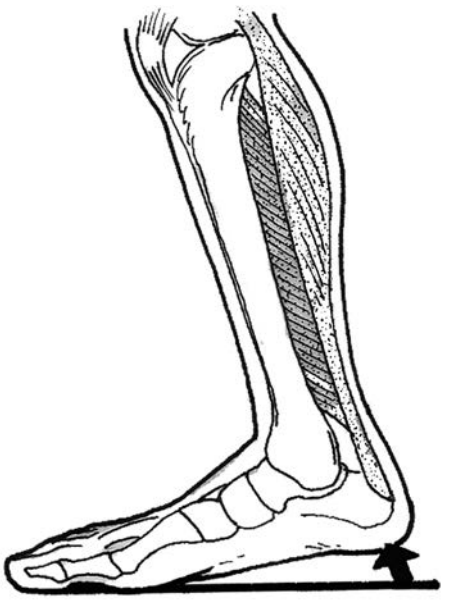
The newborn child should have 30° dorsiflexion at the ankle joint with the foot in a neutral position. Subotnick **(Subotnick, 1973)** believes that any child with less than 5° of dorsiflexion is a poor candidate for conservative treatment with neutral control by orthotics, and considers lengthening of the Achilles tendon by surgery. Lengthening when there is more than 5° might have a detrimental effect on the knee joint following the establishment of neutral control. Lengthening of the tendon, which provides for more than 13° to 15° dorsiflexion, appears to be excessive.

According to Subotnick, **(Subotnick, 1973)** when there is definite congenital shortness of the triceps surae a child will not tolerate neutral control by rigid orthotics. The foot's effort to pronate in orthotics will be painful, and the child will refuse to wear the device. If the child does wear the device, there may be adaptation to it by keeping the knee in a more flexed attitude throughout gait. Since the gastrocnemius crosses the knee, this takes some of the strain off the triceps surae, which may allow more ankle dorsiflexion.

During the Second World War, Harris and Beath **(Harris & Beath, 1948)** studied 3,619 military enlistees and described the condition found as "…hypermobile flatfoot with short tendo-Achillis." They attributed the flatfeet in the soldiers to congenital anomaly of the calcaneus and talus, where the calcaneus provides insufficient support to the head of the talus; thus, weight bearing from childhood on causes the talus to be pushed downward and inward, while the forepart of the foot twists upward and outward. Hypermobility develops in the midfoot from the process



Midfoot breakdown from short triceps surae.



Without extended pronation and midfoot breakdown, there will be early heel-off because of a short triceps surae.

of walking. They state that the short triceps surae is not primary but secondary, and "…probably develops because of the structure of the foot and the laxity of the tarsal joints deprive [the triceps surae] of tension stresses which normally in use would facilitate its elongation."

Flatfoot that results from congenital anomaly of the calcaneus and talus develops early in childhood, but it usually does not cause symptoms until later, possibly in the early teens.

Tarsal coalition

Tarsal coalition is absent or restricted movement between two or more tarsal bones. **(Schenkel et al., 2010; Zaw & Calder, 2010)** It is usually a congenital anomaly with a fibrous (syndesmosis), cartilaginous (synchondrosis), or bony (synostosis) union between the adjoining involved bones, and it may be bilateral in 80% of cases. **(Leonard, 1974)** The most common bridges are between the calcaneus and navicular, and the talus and calcaneus; they are less common between the talus and navicular bones. **(Murray & Jacobson, 1977)** Other conditions, such as trauma, arthritis, or tumor, can limit the motion in the hindfoot.

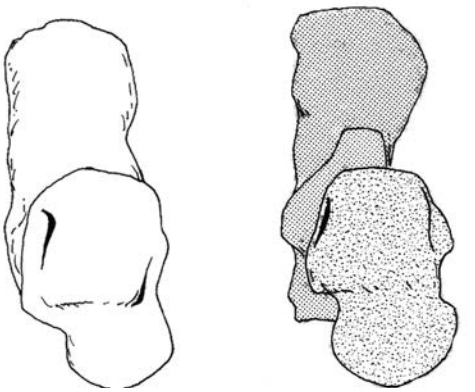
Tarsal coalition causes a lack of foot mobility, specifically in the subtalar joints. This may result in repeated ankle sprains and strain. Snyder et al. **(Snyder et al., 1981)** found retrospectively that 63% of patients who had ankle sprains had calcaneonavicular coalition. This is a much higher incidence than expected, indicating there may be a predisposition to ankle sprains from this condition.

The ankle joint may be a ball-and-socket type, in which the talus appears on x-ray as a convex dome shape on both the AP and lateral projections. Sixty-five percent of patients with a ball-and-socket ankle joint have associated tarsal coalitions. **(Morgan & Crawford, 1986)**

There is no general consensus regarding the appearance of the foot. There is limited motion that may be great enough to class the foot as rigid flatfoot; **(Zaw & Calder, 2010; Percy & Mann, 1988)** however, as few as 10% of those who have tarsal coalition may have markedly pronated feet. **(Elkus, 1986)** The more subtle cases of coalition may be recognized in an AK practice during the motion palpation phase of examining the tarsal joints.

Tarsal coalition may be asymptomatic or cause symptoms, apparently from the stress that it places on other articulations in the gait cycle. Symptoms resulting from the disorder are aggravated by activities that put additional stress into the feet.

The condition is often recognized in adolescent athletes, probably because of its progression toward ossification of cartilage and the increasing demands on the articulation from athletic endeavors. **(Jack, 1954)** However, it is not necessarily diagnosed at an earlier age in athletes. **(O'Neill & Micheli, 1989)** "Talonavicular coalition ossifies at approximately 2 to 5 years of age, the calcaneonavicular at 8 to 12 years, and the talocalcaneal in early adolescence." **(Percy & Mann, 1988)** The greater general mobility of the foot and ankle in early childhood accounts for the lack of symptoms until more rigidity develops and stress is applied in athletics.



Congenital anomaly of the calcaneus and talus fails to provide balanced suport.

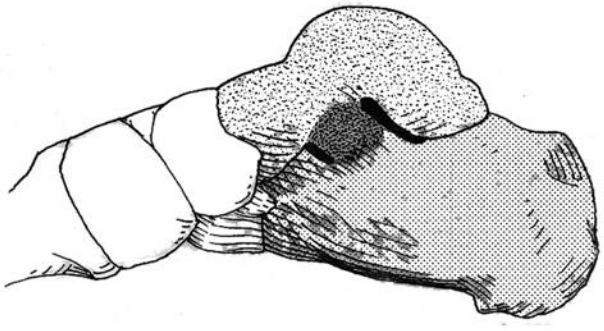
Morgan and Crawford **(Morgan & Crawford, 1986)** state, "The majority of patients with tarsal coalition in the general population require only conservative methods of treatment such as shoe modifications (wedges, inserts, counters) or foot orthotics." Many methods of non-operative treatment are aimed at reducing foot stress in tarsal coalition. Many are directed toward relaxing the peroneal muscles, and supporting the foot by use of pes planus shoes, molded inserts, ankle-foot orthoses, physical therapy, and plaster casts. **(Zaw & Calder, 2010; Elkus, 1986)** Most of these adjuncts are not needed with the applied kinesiology examination and treatment methods discussed in this chapter. Some may provide additional help in resistant cases.

The adolescent athlete with tarsal coalition is a more select patient. **(Schenkel et al., 2010)** The bones are ossifying at the time that requires much mobility of the foot and ankle complex for maximum performance. In the absence of favorable conservative treatment, a complete resection of the coalition may be necessary. **(Morgan & Crawford, 1986)** Arthrodesis may be the proper treatment for tarsal coalition when it is associated with advanced

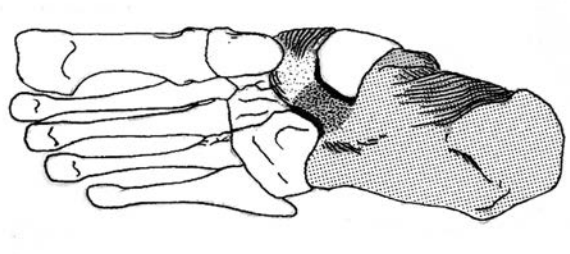
degenerative changes or deformity. **(Zaw & Calder, 2010; Peterson, 1989)**

Peroneal spastic flatfoot. Lowy as well as Harris and Beath **(Lowy, 1998; Harris & Beath, 1948)** draw attention to tarsal coalition and its association with peroneal spastic flatfoot. They indicate that the peroneal muscles are not really in spasm, but are secondarily shortened as a result of the foot dysfunction. Jack **(Jack, 1954)** disagrees with this, pointing out the relief obtained when the muscles are anesthetized. It appears that the peroneal muscles go into reflex spasm as a result of the abnormal stresses on the other tarsal articulations. **(Turek, 1984)**

There are three types of peroneal spastic and rigid flatfoot: 1) rigid flatfoot due to talocalcaneal bridge, 2) rigid flatfoot due to calcaneonavicular bar, and 3) other factors that can cause reflex spasm. It should be recognized that when arthritis is the cause for the third type, it would be more accurately designated as "arthritic flatfoot with peroneal spasm."



Calcaneonavicular coalition.



Talocalcaneal coalition.

Peroneal spastic flatfoot occurs much less than other types of flat-footedness. In Harris and Beath's study of 3,619 military enlistees, 2% had the condition. Symptoms are rarely present before twelve to fifteen years of age, when bone growth has been rapid and is more complete. It often develops as a result of another injury leaving residual problems. Continued walking when there is peroneal spasm will continue to break down the arch. If the condition has been present for a prolonged time, there may be actual shortening of the peroneal muscles. **(Lowy, 1998; Jack, 1954)**

Internal tibial torsion or malleolar torsion. Internal leg rotation, whether it is due to tibial or malleolar torsion or even femoral torsion, may be a cause of flatfoot. The foot is adducted into the position of pigeon-toeing. In order to walk with the foot pointed forward, it must excessively pronate. Unless there is severe leg rotation, excessive pronation is not great in this condition. If there is inherent weakness or laxity of the ligaments, or there are other factors that cause excessive pronation, internal leg rotation compounds the problem. In a study by Morley, **(Morley, 1957)** no correlation of genu valgus to foot pronation was found in the age range of one to eleven years.

Prehallux or accessory navicular

The accessory navicular appears between ten and twelve years of age and is the most common accessory bone of the foot; **(Leonard & Fortin, 2010; Turek, 1984)** it is present in 4-10% of the population. **(Logan, 1995; Hamilton, 1985)** (The terms "prehallux" and "accessory navicular" or "accessory scaphoid" are often used interchangeably in the literature.) There are three types of prehallux or accessory navicular bones. **(Sella et al., 1986)** Type 1 is a sesamoid in the tibialis posterior tendon. Approximately 30% of accessory naviculars are this type. Type 2 accessory naviculars are united to the navicular by a cartilaginous synchondrosis measuring 1-3 mm. This is the type that creates the major problem of accessory naviculars. Type 3 is an accessory navicular united to the parent navicular by a bony bridge, producing a cornuate navicular.

The accessory navicular can be responsible for flatfoot or, because of its bulk, give the appearance of flatfoot when the arches are well-formed. **(Leonard & Fortin, 2010; Hamilton, 1985)** In the latter case, x-ray investigation and the diagnostic approach for extended pronation (discussed later) provide the differential diagnosis. If a normally functioning foot with prehallux is treated with navicular pads or orthotics, it will be painful to the patient and probably cause remote neurologic dysfunction, as observed by applied kinesiology testing. Usually x-ray examination will demonstrate the existence of the prehallux/accessory navicular at about ten years of age.

The type 2 accessory navicular creates problems because of biomechanical forces acting on the synchondrosis. This frequently develops as a result of mild increase in recreational activities. **(Sella & Lawson, 1987)** Sella et al. **(Sella et al., 1986)** list three types of force that act simultaneously on the synchondrosis in varying degrees. Tension and shear are produced by action of the tibialis posterior to its tendon insertion at the accessory navicular. When foot pronation is present, it adds compression. This trauma causes a continual breakdown and repair of the synchondrosis, with neither prevailing. Since cartilage has a limited capacity for repair, a painful non-union will result.

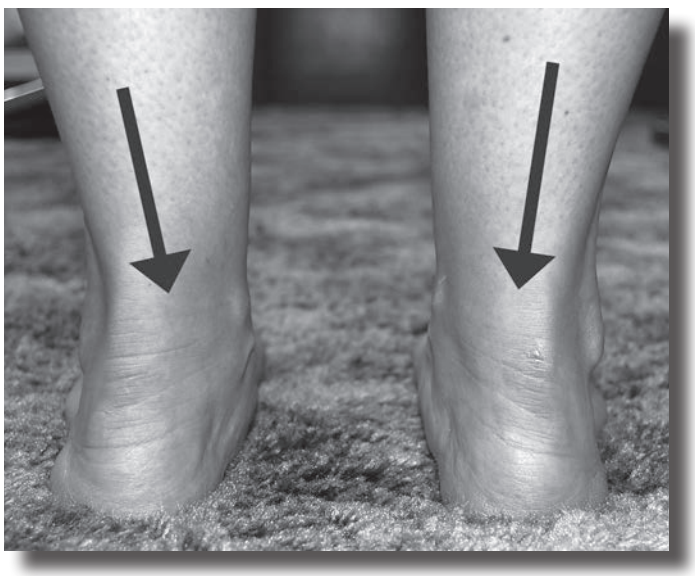
The tibialis posterior tendon may be transposed by continual deforming forces of walking. Prehallux develops, and the tendon ceases to be in a position under and medial to the navicular bone; it assumes a position medial and superior to the bone itself. **(Cobey, 1958)** Thus the tibialis posterior action does not move the navicular to increase the anterior strut angle for added medial longitudinal arch strength during the latter portion of the stance phase.

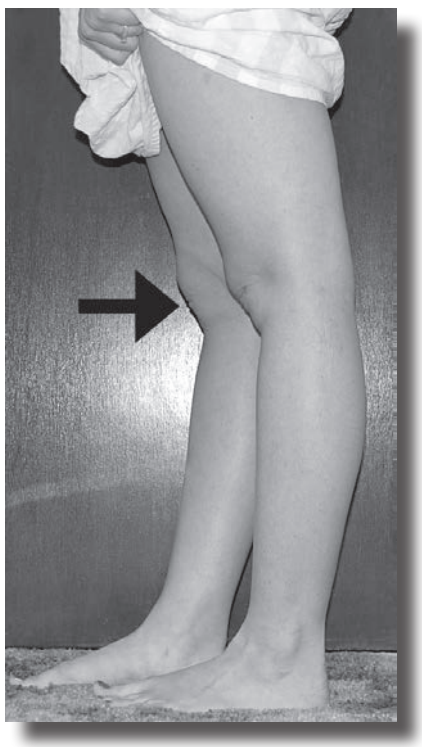
Other factors — such as osteochondritis, breakdown of the midfoot from extended pronation, and stress fractures — can cause pain in this area. A bone scan provides definitive diagnosis for determining the symptomatic or non-symptomatic accessory navicular. **(Sella et al., 1986)**

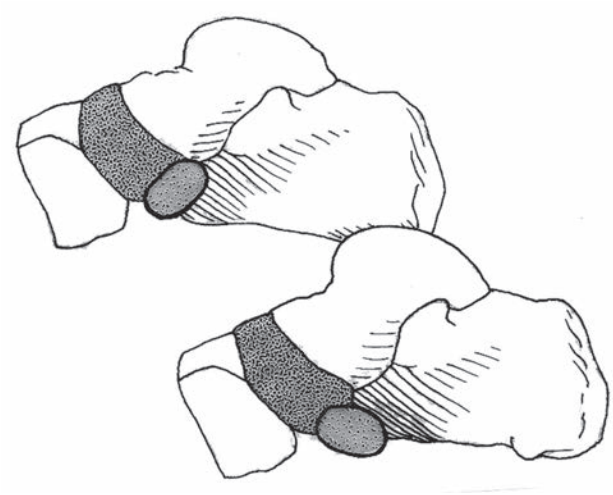
Hypermobility Syndrome

Sprains that cause tearing of ligaments supporting the arch cause breakdown and, ultimately, prolonged pronation. **(Duarte, 2004)** Systemic conditions, such as Ehlers-Danlos syndrome, cause ligamentous insufficiency. Of course, if there is a systemic condition it should be treated, if possible. Rather than simply accepting the flatfoot as a result of ligament laxity, examine for and correct any muscular or other dysfunction affecting the foot. The foot will probably require support with a suitable orthotic.

Hypermobility syndrome is recognized as a connective tissue variant, though it can relate to specific disease processes like Marfan's and Ehlers-Danlos syndromes. A possible explanation for recurrent joint injuries in hypermobile people may be the proprioceptive impairments that disturb muscle function observed in hypermobilie joints. **(Fatoye et al., 2009; Hall et al., 1995; Mallik et al., 1994)** In the treatment of patients with hypermobility syndrome, many of their most-common musculoskeletal complaints (including recurrent dislocations) are effectively treated with the muscle strengthening procedures used in applied kinesiology practice. Hudson et al **(Hudson et al., 1998)** suggest that physical conditioning and regular exercise are probably protective from the effects of hypermobility syndrome. Colloca and Polkinghorn suggest that chiropractic care may benefit some patients with connective tissue disorders, including Ehlers-Danlos syndrome. **(Colloca & Polkinghorn, 2003)**







Accesssory navicular.

The accessory navicular is usually asymptomatic, but it may be traumatized to cause pain. Injury usually occurs from repeated small injuries during recreational athletics. Conservative treatment is directed toward obtaining optimal foot and ankle function, as indicated in this and the next chapter; additionally, soft orthotics, rest, and physiotherapy may be of value. If these fail, surgical excision is necessary.

The Kidner surgical procedure **(Prichasuk & Sinphurmsukskul, 1995; Kidner, 1929)** is indicated when there is pain over a prominent accessory navicular bone, or enlarged medial protuberance over the navicular. This procedure removes the accessory navicular and/or re-shapes the navicular, and re-attaches the tibialis posterior tendon. Early in Kidner's use of this procedure, he observed a marked improvement of a flatfoot condition in a patient from whom an accessory navicular had been removed. During the operation the tendon of the tibialis posterior, which originally attached to the accessory navicular, was changed to attach to the navicular as it would under normal circumstances. Kidner attributed improvement in the flatfoot to the re-directed force of the tibialis posterior, giving a greater lifting effect on the arch. It has been observed that the transplanted tendon, even under general anesthetic, will hold the foot in a correct position. The importance of the tibialis posterior muscle for proper arch support has been noted throughout this volume, and dysfunction in this muscle in AK evaluation often produces many foot, ankle, and knee problems. Secondary to posterior tibialis inhibition there often results tightness of the soleus and gastrocnemius muscles, with resulting pain in the Achilles tendon. Without the manual muscle test, the wide variety of nonspecific symptoms resulting from tibialis posterior dysfunction makes the diagnosis of this disorder far more difficult.

Talipes calcaneovalgus

Talipes calcaneovalgus is a congenital, flexible flatfoot deformity with characteristic findings at birth. **(Paton & Choudry, 2009)** This is pronation of the heel combined with supination of the forefoot. It is congenital, and the suggested cause is intrauterine malposture.

Bresnahan as well as Ferciot, **(Bresnahan, 2000; Ferciot, 1972)** studied the balance of infants' feet in this condition and their propensity to develop flatfoot, found muscle imbalance to be the etiology. The combination muscle imbalance is an overactivity of the tibialis anterior and peroneus brevis, and weakness of the peroneus longus and tibialis posterior muscles. Ferciot states that 5% of all newborns have this condition.

The examination for functional muscle imbalance is part of the applied kinesiologist's training and adds much to the examination and treatment of children and young athletes in particular. **(Cuthbert & Rosner, 2010; Cuthbert & Barras, 2009; Blum & Cuthbert, 2009; Karpouzis et al., 2009; Cuthbert, 2008, 2007; Pauli, 2007; Goodheart, 2003; Maykel, 2003; Mathews et al., 1999; Froehle, 1996; Cammisa, 1994; Mathews & Thomas, 1993)** The examination detects inhibited muscles, compensatory movement patterns, temporal aberrations in muscle firing, and muscles recruited in an abnormal sequence. The mixture of weakness and tightness seen in the muscle imbalances involved in foot and ankle disorders alters body alignment and gait and changes the equilibrium points of the joints. There is a growing body of literature suggesting that athletic performance can be enhanced by chiropractic and other manual interventions. **(Hoskins & Pollard, 2010; Costa et al., 2009; Sandell et al., 2008; Shrier et al., 2006; Schwartzbauer et al., 1997; Lauro & Mouch, 1991)**

Talipes calcaneovalgus is indicated by excessive dorsiflexion of the foot at birth. Diagnostically, the foot cannot be brought down from the dorsiflexed position to below a right angle with the pressure of one finger. The dorsiflexed position is maintained by overactivity of the tibialis anterior muscle, which maintains the foot in supination along with the dorsiflexion. This position overstretches and overpowers activity of the peroneus longus muscle. The tibialis posterior muscle is also elongated.

At approximately one month, an active thrust reflex develops. The triceps surae muscles become active, being resisted by the short tibialis anterior muscle. Ferciot **(Ferciot, 1972)** observes that at four to six weeks of age the peroneus brevis muscle begins to be active, along with the strong triceps surae pulling the heel into marked eversion. At three to four months, if the baby is held in a position where the feet press on the floor, the lateral aspect of the foot does not touch the floor. A fixed supination of the forefoot with eversion of the heel develops.

Ferciot states that the condition is aggravated as babies begin to crawl and weight is borne on the medial aspect of the foot, further causing the forefoot bones to develop in a supinated position. He also states that belly sleeping tends to sustain and aggravate this deformity. Walking is usually delayed in these children until twelve to fourteen months of age, and it is marked by poor balance and a toeing-out position.

Subotnick **(Subotnick, 1973)** states that the calcaneovalgus foot responds well to conservative treatment if initiated early and carried out with persistence. Subotnick begins with manipulative reduction of the deformity. The foot is manipulated into plantar flexion and inversion 15 to 20 times, 3 times daily. If manipulations are not entirely

successful, he applies corrective casts to hold the feet in the position acquired by manipulative stretching exercises.

Applied kinesiology muscle balancing techniques should be added to the manipulations and should include both muscle lengthening and strengthening procedures, as discussed later and in other AK references. **(Walther, 2000, 1981)**

Subotnick states that 85-90% of these feet can be adequately corrected if treated in infancy. After eight months of age the prognosis is poor for conservative treatment.

Postural influence

Foot pronation is almost always associated with a postural fault. Either can be primary, with the excessive pronation causing the postural fault or vice versa. **(Prior, 1999)** As with all other conditions, optimal treatment is directed toward the primary fault.

A common postural fault is accentuation of the spinal AP curves, with an anteriorly rotated pelvis and hyperlordosis in the lumbar spine. This may result from failure of the foot's positive support reaction, previously discussed. If the gluteus maximus muscles, which support the posterior pelvis, are strong in the clear, one can easily challenge the positive support reaction by stretching the longitudinal arches and spreading the transverse arch; then re-test the gluteus maximus muscles. If they weaken, it is affirmative evidence that the positive support reaction is dysfunctioning. If the gluteus maximus muscles are bilaterally weak in the clear, an upper cervical fixation is indicated which, when corrected, will immediately cause the muscles to test strong. In the latter case, the postural distortion is probably primarily contributing to the extended foot pronation. In addition to stabilizing the posterior pelvis, the gluteus maximus muscles are external hip rotators. Their weakness allows internal hip and leg rotation, taking the forefoot into adduction. The body accommodates this with additional pronation for forefoot abduction. Although there is generally a primary connection between pronation and remote structural distortion, both conditions are usually present. If the primary condition is not corrected, the secondary problem will continue to recur. For example, if the patient has a facet syndrome from an anterior pelvic tilt and hyperlordosis of the lumbar spine, the primary complaint may be low back pain. If extended pronation and dysfunction of the positive support reaction are present, one will be unsuccessful in obtaining lasting improvement of the low back pain.

Lee **(Lee, 1997)** points out that the consequences of gluteus maximus muscle inhibition (in this example due to failure of the positive support reaction from a foot dysfunction) during gait.

> "The consequences to gait can be catastrophic when gluteus maximus is weak. The stride length shortens and the hamstrings are overused to compensate for the loss of hip extensor power. The hamstrings are not ideally situated to provide a force closure mechanism and, in time, the sacroiliac joint can become hypermobile. This is often seen in athletes with repetitive hamstring strains. The hamstrings remain overused and vulnerable to intramuscular tears."

Bullock-Saxton has also shown that the gluteus maximus muscle was significantly delayed in activation in both the injured and uninjured limbs in subjects following ankle sprain. **(Bullock-Saxton, 1994; Bullock-Saxton et al., 1994)**

The common interaction between foot dysfunction and postural distortion makes it difficult to determine the primary problem. Similarly, the relationship between pain and motor control and muscular inhibition is so intimate that pain and muscle strength may represent two dimensions of a common neural event. **(Melzack, 1999)**

Failure of the positive support reaction, with increased AP curves, may ultimately cause an upper cervical fixation, in turn causing greater weakness of the bilateral gluteus maximus muscles. The patient's primary complaint may be suboccipital headaches. When one examines the prone patient, there is no stress on the positive support mechanism in the feet; it may appear that the upper cervical fixation is the primary condition. When it is corrected, the bilateral gluteus maximus muscles test strong, but as soon as the patient walks on the feet, the correction will probably be lost; the entire complex will return to its original status if the feet are not corrected.

Goodheart **(Goodheart, 1967)** first described psoas muscle weakness as a result of foot pronation. This probably results from the excessive internal leg and thigh rotation that excessive pronation causes. This situation is particularly involved during walking and running. The iliopsoas produces its main activity during the swing phase, but there is a secondary peak of activity at mid-stance. **(Basmajian & DeLuca, 1985)** The latter occurs when the foot is coming out of pronation with external leg rotation. With extended pronation, it appears there is excessive stress on external hip-rotating muscles, such as the psoas. The psoas insertion on the lesser trochanter gives minimal leverage for external rotation, and it appears the psoas is working in a vain effort to correct the excessive internal leg and thigh rotation.

As with many conditions in the body, there seems to be reversible interaction between foot pronation and psoas muscle dysfunction. In some cases, psoas muscle dysfunction can cause loss of foot corrections, perpetuating foot pronation. There are applied kinesiology tests that determine and differentiate psoas muscle involvement; these are discussed later in the section on examination of muscle involvement.

Examination

The incidence of flat foot (pes planus) is 20% in adults, the majority of which are flexible. **(Chaitow & DeLany, 2002)** The previous discussions of general examination and body language of the foot provide the basis for foot examination. Here are more specific factors of the pronated foot and extended foot pronation during gait.

The first impression of excessive pronation comes from visual observation, as described in the section on body language of foot dysfunction. What may appear to be

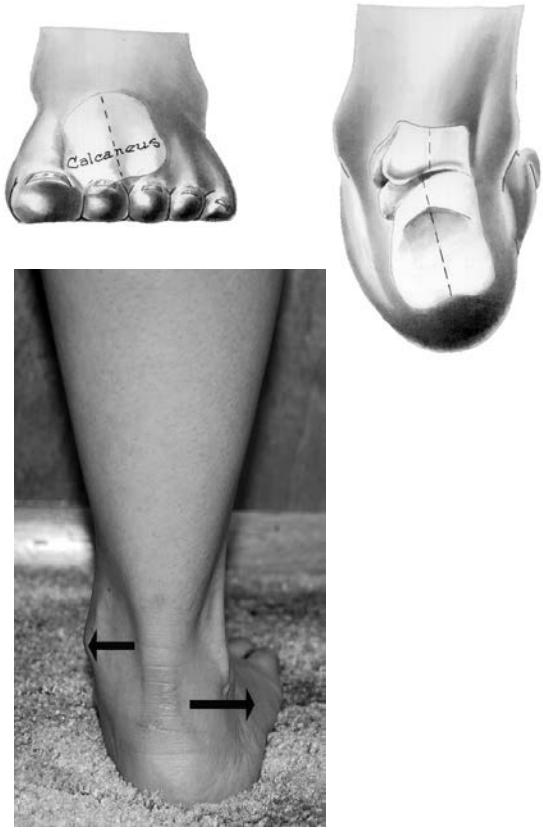
flat-footedness needs to be put into proper perspective. An infant's foot has some arch, but it appears flat because of the fat pad. **(Langer, 2007)** Full arch development comes with foot use. A study by Staheli et al **(Staheli, 1986)** showed that the medial longitudinal arch usually becomes evident during the first decade of life. Persistent flatfeet into the teenage years are still within normal range.

Flattening of the arch is not pathognomonic of extended pronation. An individual can have a high arch that is pronated, or the foot can be flat without extended pronation. **(Aronow & Solomone-Aronow, 1986)** To be considered physiologically normal flatfeet, there must be the abundant amount of subcutaneous fat and joint laxity, as opposed to the pathologic, rigid flatfoot that requires treatment. Staheli et al. **(Staheli et al., 1986)** state, "The common practice of 'treating' physiologic flatfeet with shoe modifications, orthotics, or surgery is unnecessary and inappropriate."

Observation of the calcaneal and other bone positions gives indication the foot has moved into pronation. Also, both shod and unshod gait analysis should be done to determine if pronation is extended, and whether walking causes detrimental effects to remote body function.

Static stance. In this discussion, static stance refers to standing still. It is acknowledged that there is no absolute static standing posture, because there are normally small balancing movements.

Observe for three basic factors in the static stance: 1) eversion of the calcaneus, 2) depression and strain of the medial longitudinal arch, and 3) medial bulging of the talonavicular articulation.



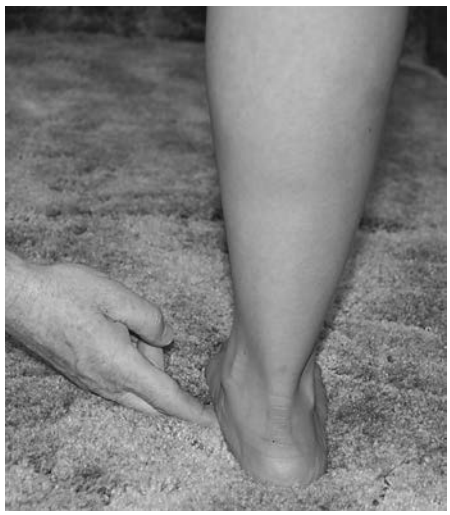
Helbing's sign and calcaneal eversion with Chopart's joint moving medially.

Looking at an individual from behind, one observes first for Helbing's sign, **(Dorland's Illustrated Medical Dictionary, 2007)** which is a medial bowing of the Achilles tendon, i.e., the convexity of the curve faces medially. This is most easily observed by visualizing an imaginary line down the center of the leg into the center of the Achilles tendon, continuing into the center of the calcaneus. When Helbing's sign is positive, there will be a break in the line as it deviates laterally into the calcaneus. Confirming Helbing's sign is an appearance of the calcaneus in eversion.

Usually with foot pronation there is depression of the medial longitudinal arch in static stance. As previously noted, a high arch can be pronated in static stance and have extended pronation. If so, there will be tension on the plantar fascia in the weight-bearing position. Normally the plantar fascia is at its greatest tension with metatarsophalangeal extension, which tightens it by the windlass mechanism. In the case of a high arch, the examiner can place his fingers under the medial longitudinal arch and palpate for tension. Have the patient externally rotate his leg to put weight on the lateral longitudinal arch. If there is considerable relaxation of the plantar fascia in this position, pronation is probably present. There will usually be excessive tenderness of the medial longitudinal arch ligaments and of the plantar fascia with digital pressure.

In pronation the talus adducts, moves anteriorly, and plantar flexes in its relationship to the calcaneus. **(Lau & Daniels, 1998; Ramig, 1977)** As the patient is viewed from behind, this creates an appearance of more foot medial to the leg than lateral. The entire appearance from behind is due to Chopart's joint moving medially, which causes a bulge of the talonavicular articulation and a loss of the straight line along the lateral aspect of the foot, with a break at the calcaneocuboid articulation. This causes the forefoot to appear abducted. **(Prior, 1999; Aronow & Solomone-Aronow, 1985)**

Dynamic evaluation. A patient should be evaluated for pronation in a relaxed, normal gait, both with and without shoes. There should be enough steps included in the walk for normal gait to develop. The limited space in most offices prohibits this unless a hallway is used. A treadmill has some value in evaluating for pronation; however, it must be



Palpation of medial longitudinal arch while standing

realized that the foot does not strike the moving treadmill in the same way it does solid substrate. After the patient walks on the treadmill for some time, the gait becomes more natural. Evaluating leg and foot motion is similar, whether the patient walks on a treadmill or the substrate.

Movement throughout the body should be symmetrical. Observe the temporal pattern of foot movement during the gait cycle. When the barefooted patient walks, one may observe tendons of the toe extensors rise to aid the tibialis anterior with foot dorsiflexion against a tight triceps surae. **(Cailliet, 1997; Craik & Oatis, 1995; Milgram, 1983, 1964)** This is not always present, but it is a sure sign of extended pronation. If the foot does not yield to a short triceps surae, the individual may walk with a slightly flexed knee. **(Dananberg, 2007; Subotnick, 1975)**

When there is hypermobility of the midfoot with a normal Achilles tendon and triceps surae, heel-off is delayed, causing the arch to maintain flatness for a longer time with excessive medial bulging at the talonavicular area. The flatfoot position may be maintained almost to the point of weight being transferred to the metatarsal heads. As the toes begin to extend, the windlass mechanism tightens the plantar aponeurosis to give the arch some stability, aiding the heel in rising. This is in contrast to the rigid lever the foot should become at 50-65% of the stance phase.

More broadly, with foot pronation the talus dips downward and flattens the medial longitudinal arch due to its tri-planar motion being disturbed. In extended pronation, there is increased force along the medial side of the foot near the time of toe-off. **(Prior, 1999; Mann, 1983)** This causes push-off to appear to be more from the medial side of the hallux. Hallux valgus or a callus on the medial plantar aspect of the hallux confirms chronicity of this condition.

In pronation, heelstrike is usually lateral. As full weight is borne by the foot, it appears to flop over onto the medial side. The lateral strike of the heel appears to follow the rule that for every action there is a reaction. The pronated foot, as it goes into the swing phase, inverts so that heelstrike is on the outer, slightly posterior, portion, with a quick snapping back into pronation as the stance phase begins. The lateral heelstrike is confirmed by excessive shoe wear in that area.

As the foot goes into extended pronation, there will be excessive internal patella rotation at the same time that the foot appears to flop onto the medial arch. Immediately after toe-off, one may see the patella quickly move laterally.

Palpation for pain

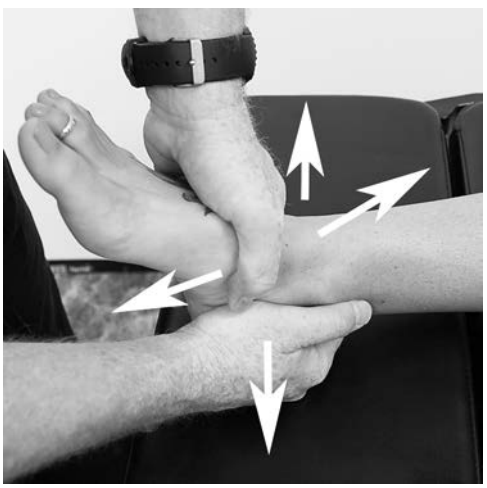
When pronation is extended during walking, there are specific areas in which the ligaments are constantly strained if the foot is not completely broken down. Exquisite tenderness of these areas on digital pressure is indicative of excessive pronation. Evaluation for this pain is usually done in combination with evaluating joint motion and challenging the joints.

If there are other signs of excessive pronation and there is no tenderness of the ligaments, they have already been strained and stretched to the point that they no longer maintain the arch. Thus, *painful ligaments are a good prognostic sign* for the possibility of regaining foot integrity without permanent use of orthotics to provide support where there is ligament insufficiency. **(Solomonow, 2009)**

With collapse of the medial longitudinal arch, the head of the talus will drop medially and inferiorly. It will be palpable between the medial malleolus and the navicular tubercle. There will probably be exquisite tenderness in that area. In the individual without foot pronation, the head of the talus is not usually palpable. Digitally press into the plantar area of the talonavicular and cuboideonavicular articulations. If the arch is in strain, these ligaments will be tender.

AK versus Palpation in Foot-Ankle Diagnosis

An interesting comparison of the ideas of Mennell in the examination of the foot with applied kinesiology methods can be made. **(Mennell, 1964)** Mennell stated that it was important to differentiate subtalar problems from those involving the talotibiofibular problems. He differentiated these by stabilizing various components of these complex joint surfaces and introducing movements into them. "If these movements are full, free and painless, there is obviously no pathological condition of this joint…Pain in the performance of this movement indicates [dysfunction] giving rise to pain at the subtalar joint." The applied kinesiology challenge procedure introduces similar forces into the patient's sensorimotor, articular, and ligamentous system of the foot and examines for changes in muscle function. This permits the examiner to bring Panjabi's stability model **(Panjabi, 1992)** into the examination, and does not leave the examiner dependent upon subjective pain and subjective palpation sensations for diagnosis of disturbances in the foot. In the AK approach, if the challenge produces weakness in a strong indicator muscle or strength in a weak indicator muscle, then evidence has been provided that confirms the patient's complaint of pain and muscular dysfunction (local or remote). Additionally, one of the consequences of the dysfunction found (muscle inhibition) can be reassessed after correction for improvements. The reliability of the manual muscle test is superior to those for both motion and static palpation. **(Cuthbert & Goodheart, 2007)**



The muscles are also in strain in their effort to counteract extended pronation. Microavulsions of the tibialis posterior muscle have been described. **(Holmes et al., 1990)** The major area of tenderness is at a muscle's insertion. **(Mense & Simons, 2001)** The tibialis posterior has its major insertion into the tuberosity of the navicular bone, and the tendon spreads out to insert into the plantar surface of the 1st cuneiform, bases of the 2nd, 3rd, and 4th metatarsals, and cuboid bones. It also has a slip that inserts onto the plantar surface of the sustentaculum tali. The tibialis anterior inserts into the medial and plantar surfaces of the 1st cuneiform bone and the base of the 1st metatarsal bone. Both of these muscles are more active when there is excessive pronation **(Gray's Anatomy, 2004; Gray, 1969; Gresczyk, 1965)** in an effort to control it. Usually there will also be tenderness at the origins and the muscle bellies.

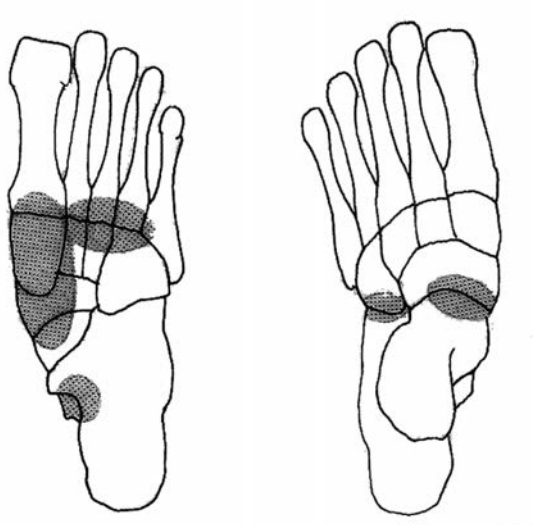
With the patient weight-bearing, evaluate pain on digital pressure at the insertions of the tibialis posterior and anterior. Have the patient externally rotate his leg to take his foot out of pronation. Some diminishment of pain on digital pressure is indicative of excessive pronation, because these muscles are active in the standing position when pronation is present; they are not in a normal foot. **(Gray's Anatomy, 2004; Gray, 1969; Gresczyk, 1965)**

In excessive pronation the talus moves medially, anteriorly, and plantar flexes in its relationship to the calcaneus. **(Prior, 1999; Ramig, 1977)** In doing so, the trochlear surface internally rotates, carrying with it the tibia and fibula. This causes a strain at the ankle mortise, manifested as tenderness of its ligaments. Digital pressure applied around the internal and external malleolar ligaments will usually elicit pain when there is excessive pronation.

Joint motion and challenge

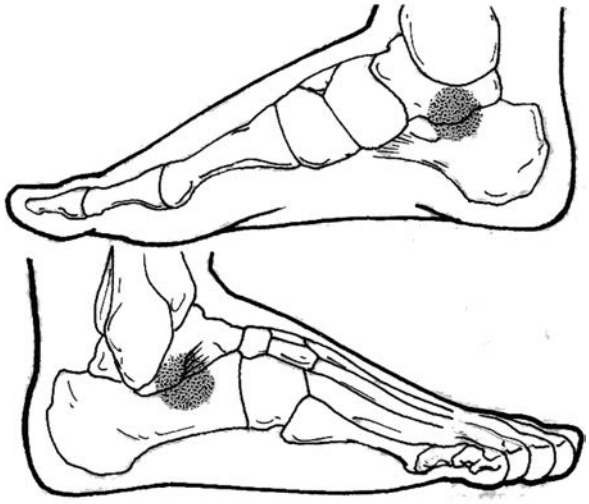
Treatment of excessive pronation includes joint manipulation, correction of muscle malfunction, and possibly structural support, physical therapy measures, and rehabilitation. The first order of importance is to correct subluxations, fixations, and muscle dysfunction. The order in which these corrections are made depends on the primary or secondary nature of the dysfunction, which is easy to determine with the applied kinesiology techniques of challenge and therapy localization. **(Walther, 2000)** Often examination techniques of muscle testing, joint motion palpation and challenge, skin challenge, therapy localization, and possibly even other AK techniques — such as respiration assist during evaluation — are used in combination. First the joints and then the muscles are considered, followed by the interaction of various structures and functions.

Evidence of foot subluxations or fixations is usually elicited first by the shock absorber technique, described earlier. Joint challenge is applied to evaluate limited or excessive motion of an articulation, and to determine if movement of the joint stimulates joint receptors to adversely affect muscle function, as determined by manual muscle testing. In many cases, the neurologic and motion aspects can be evaluated at the same time by joint challenge. In other cases, it is necessary to specifically stabilize a bone to determine excessive motion or fixation.



Palpatory pain locations with extended pronation.

Care must be taken to adequately stabilize the navicular bone when evaluating motion between the cuneiform bones and the navicular. **(Gillet & Liekens, 1981; Mennell, 1964)** Failure to do so may lead to evaluating motion at the talonavicular articulation, which is often hypermobile. This may cause one to miss immobility between the navicular bone and cuneiforms. The hypermobility of the talonavicular articulation results from foot hyperpronation when there is lack of dorsiflexion from short triceps surae muscles.



Pain to digital pressure around internal and external malleoli in extended pronation condition.

It is necessary to have motion between all articulations of the foot to allow for flexibility, springiness, and resilience of the foot to meet the substrate. The arch that is fixed, whether it is high or flat, is abnormal. **(Maffetone, 2003; Gillet & Liekens, 1981; Mennell, 1964)**

To examine motion of the subtalar articulation, the body of the talus can be locked between the tibia and fibula. This is accomplished by maximum dorsiflexion if the plantar flexor muscles are not too tight. The anterior

wide wedge of the talus body locks between the tibia and fibula as it spreads them with dorsiflexion. **(Cailliet, 1997)** In excessive pronation, especially that associated with a short triceps surae, there is excessive motion in the subtalar articulation. **(Logan, 1995; Harris & Beath, 1948)** This increased range of motion, along with increased motion at Chopart's joint, allows the heel to come into contact with the ground in spite of the short triceps surae.

Chopart's joint should be evaluated for excessive range of motion by holding the hindfoot solidly and moving the midfoot with the other hand contacting around the proximal metatarsals. As mentioned, excessive range of motion in any of these joints is often present with excessive pronation. Prognosis for correcting the foot without the use of support lessens with increased excessive joint motion.

Motion between the metatarsals should be evaluated at both the proximal and distal ends. Although there is no actual joint between the distal metatarsals, there is often restricted movement that must be mobilized. Both ends of the metatarsals can be evaluated for motion with a scissors-type action. At the proximal end, the physician contacts the bases of two adjacent metatarsal bones with his thumbs and forefingers and attempts to move one plantarly and the other dorsally. The heads are contacted at the distal end in a similar manner, with one metatarsal moved into flexion and the other into extension.

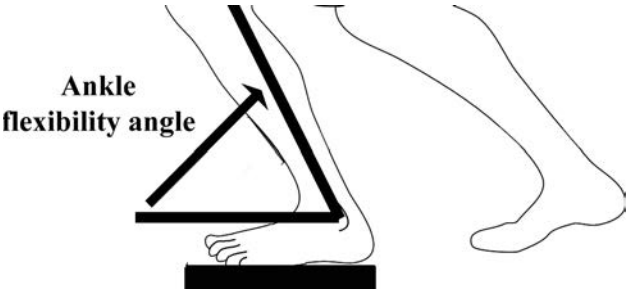
With excessive pronation, there is nearly always a subluxation of the talus in the ankle mortise. Applied kinesiology challenge indicates that a lateral-to-medial adjustment is often needed. Because the talus moves medially in pronation, this may seem incorrect. The more common lateral subluxation may be due to the body's effort for correction, or from the excessive supination during the swing phase of gait. In any event, best results are obtained when the talus is adjusted in the direction of challenge that strengthens associated weak muscles. When challenging the talus, contact should be immediately under the malleolus. This is especially important when challenging from lateral to medial because of the limited amount of talus extending below the malleolus.

Muscle evaluation

Evaluating the muscles is done with manual muscle testing and range of motion for the triceps surae. An equinus foot occurs when there is inability to reach 6°-8° of ankle dorsiflexion. **(Charles et al., 2010; Logan, 1995; Subotnick, 1975)** Runners tend to overdevelop the calf muscles compared to the anterior ones. Sometimes they are able to get by for a while, because pre-running stretching procedures increase dorsiflexion to the minimum 10°. There are many other situations responsible for the short triceps surae, e.g., high-heeled shoes, prolonged sitting, general poor conditioning, and many of the remote conditions treated by applied kinesiology.

Myofascial pain in the calf muscles has been documented to cause a biomechanical abnormality of gait, resulting in an excessive knee flexion angle during the stance phase of gait **(Wu et al., 2005).** Adequate ankle dorsiflexion (>10°) is also necessary in midstance so that the tibia can advance over the foot permitting forward body movement **(Norkin and White, 2003).** If this ankle ROM is restricted by inhibited or hypertonic musculature, compensation may occur in the form of genu recurvatum, early knee flexion, early heel lift or excessive pronation at the subtalar joint **(Prior, 1999).** These compensatory mechanisms put undue stresses on many structures superior to them throughout the leg and may lead to foot disorders such as plantar fasciitis, Achilles tendonitis and metatarsalgia **(Hill, 1995).** Increased subtalar joint pronation has also been identified as a "remote" contributing factor in patellofemoral pain **(Crossley et al., 2006).**

A short Achilles tendon may be present in children; when present, the range of motion should be increased even if the child has no symptoms. **(Chen & Greisberg, 2009; Wickstrom & Williams, 1970)** As some of the general flexibility usually present in children is lost, symptoms and increased pronation will probably develop. When there is a short triceps surae in children and adolescents, the arch can be as often elevated as depressed, and there is slight pes valgus. Clawing and a tendency toward hammertoes develop, often to the point that there is negligible activity of the toes in weight bearing. **(Kwon et al., 2009; Milgram, 1983)**



Measurement of loaded dorsiflexion.

When measuring dorsiflexion at the ankle, the measurement should be limited to motion at the ankle mortise. A minimum dorsiflexion of 10° at the ankle mortise is necessary for normal function; **(Charles et al., 2010; Subotnick, 1991, 1975, 1971)** in the infant there should be 30°. **(Subotnick, 1973)** There is usually increased laxity of ligaments in the subtalar and mid-tarsal articulations in the hypermobile flatfoot. In the normal foot, ligaments limit mid-foot dorsiflexion. **(Hamilton, 1985; Hicks, 1951; Manter, 1941)** Ligament breakdown allows increased range of motion that often conceals limited dorsiflexion at the ankle. The examiner should grasp the calcaneus and midfoot to stabilize the calcaneus, navicular, and cuboid bones against the talus before moving the foot into dorsiflexion. **(Harris & Beath, 1948)** Examination of ankle dorsiflexion should be done in subtalar inversion and in neutral. **(Prior, 1999; Milgram, 1983)** Limitation in either test is evidence of a short triceps surae.

When the patient's knee is maintained in extension, the soleus or gastrocnemius or both can limit dorsiflexion. When the knee is flexed, only the soleus can limit dorsiflexion. In the former case, stretching procedures are done with knee extension; in the latter they are done with knee flexion.

Evaluating range of dorsiflexion is a static test that

does not indicate how the muscle will elongate during an eccentric contraction. **(Prior, 1999)** There is no specific test to determine this. Cramping of these or other muscles at night, after physical activity, or during muscle testing is indication for further systemic evaluation, probably of calcium metabolism.

One is often amazed at the limited amount of motion at the ankle. The foot may fail to reach a right angle with the tibia by as much as 25°. In normal feet and ankles, dorsiflexion may reach 20° beyond the right angle. **(Charles et al., 2010; Logan, 1995; Harris & Beath, 1948)**

In impairment rating, 20° of dorsiflexion is considered normal, with a 4% impairment rating of the lower extremity when there is only 10° of dorsiflexion. **(American Medical Association, Guides to the Evaluation of Permanent Impairment, 2007)** It must be put into perspective that impairment measurement is of the goniometer base placed in alignment with the axis of the tibia; the degree of dorsiflexion is based on the goniometer arm placed parallel to the sole of the foot. This includes motion at the subtalar and midfoot joints, which gives a varying amount of additional dorsiflexion. When range of dorsiflexion is used to consider the length of the triceps surae, only motion at the ankle should be measured.

Although it is important to evaluate the range of motion at the ankle for assessing the triceps surae length, it is difficult to obtain an accurate measurement of the degree of foot dorsiflexion. The method recommended by Charles et al. **(Charles et al., 2010)** and Lindsjo et al. **(Lindsjo et al., 1985)** provide reproducible measurements of foot dorsiflexion and plantar flexion. This is necessary for assessment of the progress from stretching procedures, discussed later. Their method measures the range of motion in a weight-bearing position. An individual's foot is put on a 12"-18" (30-46 cm) high stool. He then leans forward to dorsiflex the ankle to the maximum amount while carrying most of the body weight on the side being evaluated. To obtain reproducibility of the measurement, no rotation is allowed in the lower leg; internal rotation increases the range of dorsiflexion by allowing increased pronation. A goniometer is used to measure the angle between the support line of the foot and the long axis of the leg. The amount of dorsiflexion measured by this method will be greater than that obtained when the movement is limited to the ankle, because foot breakdown is included in the former.

Plantar flexion is measured in a similar manner; however, the individual plantar flexes the foot to the maximum amount while maintaining as much body weight on the foot as possible. Again, the measurement is made between the plantar surface of the foot and the long axis of the leg. This method can be combined with x-ray to determine if the mid-arch is breaking down with triceps surae stretching procedures. (X-ray procedures are described next. Triceps surae stretching is described in the foot and ankle rehabilitation section.)

Psoas inihibition & foot pronation

Psoas muscle dysfunction can develop from foot pronation. **(Greenawalt, 1992)** In some cases, the muscle weakness can perpetuate extended pronation by causing loss of foot corrections. When psoas muscle weakness is secondary to excessive pronation, one will often find its neurolymphatic, neurovascular, and other reflexes active. When indicated, treatment to these reflexes will cause the psoas muscle to test normal; however, when the muscle weakness is due to pronation, there will nearly always be a return of muscle dysfunction as soon as the individual stands and walks in his customary manner.

To determine if foot pronation is indeed responsible for psoas dysfunction, one can correct factors influencing the muscle without any foot correction. This may include reflexes, spinal subluxations, the muscle itself, or any other associated factor. Have the patient walk in a figure-eight pattern. If foot pronation is the probable cause for the psoas dysfunction, it will again test weak. This is not pathognomonic; other factors may be involved.

Further evidence that pronation may be responsible for psoas dysfunction can be obtained by re-correcting the muscle, again not correcting the foot. Have the patient stand and walk on the outer borders of his feet to avoid excessive pronation. After walking in this manner, even with turning right and left as in the figure-eight walk, the psoas muscle frequently will not weaken if foot pronation is actually the cause of the muscle dysfunction. This seems to support the hypothesis that psoas dysfunction develops with pronation because of the muscle's inability to control excessive leg and thigh internal rotation.

Sometimes foot corrections are lost as soon as the patient walks or runs. Schmitt **(Schmitt, 1988)** describes a method to determine when psoas muscle dysfunction is perpetuating foot pronation. This can be done during the initial examination of the foot. It should always be done when foot corrections are lost after the patient walks. When a positive shock absorber test or challenge to the foot is positive, evaluate the test again while the patient is therapy localizing to a muscle-organ associated point, such as the kidney alarm point, neurolymphatic reflex, or neurovascular reflex. If this cancels the previously positive test, it indicates that psoas muscle weakness may be a contributing factor to the foot pronation, rather than the psoas dysfunction being secondary to pronation. A similar rationale to that given for psoas muscle weakness secondary to foot pronation is applicable in this situation. If the psoas fails in its role of bringing the leg out of internal rotation, the foot must accommodate by pronation. There will often be no return of foot dysfunction after correcting the psoas muscle. The psoas muscle is the one most often involved in this relation, but other external thigh muscles, such as the piriformis or gluteus maximus, may have the same relationship.

Important muscles stabilizing the arches during gait are the intrinsic muscles of the feet, which receive their nerve supply from branches of the posterior tibial nerve after it has traversed the tarsal tunnel. If there is peripheral nerve entrapment at the tunnel, there may be secondary atrophy of the intrinsic muscles contributing to foot problems. Removing the entrapment and rehabilitating the muscles are necessary if atrophy is present **(The tarsal tunnel syndrome was discussed earlier in *Peripheral Nerve Entrapment of Lower Extremity*).**

At this point in the examination, the tibialis and peroneal muscles should be evaluated with standard

manual muscle testing. With knowledge of joint mobility and function of the major muscles of the foot and ankle, one can begin the corrective procedures resulting from excessive pronation.

X-ray Evaluation

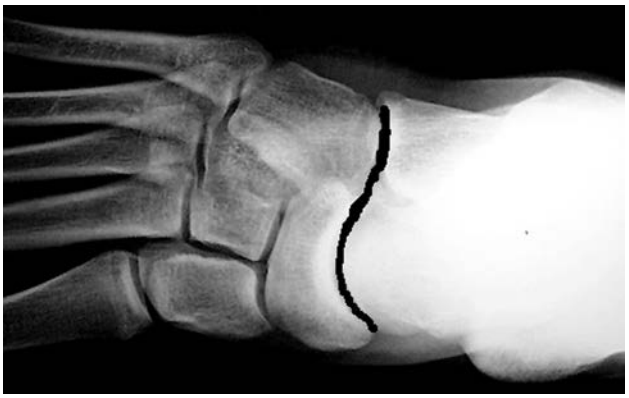
Postural lateral x-ray of the foot is taken in a specially constructed cassette holder, with full weight on the foot being examined. This is done by relaxing the contralateral knee (Hanch position). The central ray is parallel to the floor and aimed at the anterior superior corner of the cuboid. **(Yochum & Rowe, 2004; Logan, 1995; Aronow & Solomone-Aronow, 1986)**

A line along the talonavicular and calcaneocuboid articulations should represent a gentle reverse "S" curve, called the cyma line. It is seen on both the lateral and AP projections, and separates the forefoot from the hindfoot. **(Hlavac, 1967)** In a normal foot, the curve is unbroken and undistorted. In the lateral projection, it is shallower in a high arch than in a low one; in a pronated foot, the curve assumes a question-mark shape as a result of the forward slide of the talus in relation to the calcaneus during its triaxial motion.

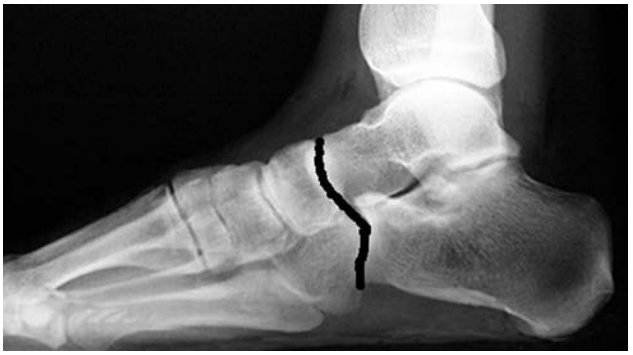
For an AP projection, the cassette is in the holder parallel with the weight-bearing patient's plantar surface. The central ray is aimed at the base of the 2nd metatarsal, with the tube angled at 10°-15° off vertical, depending on whether the arch height is high, medium, or low. This keeps the ray perpendicular to the metatarsal bone shafts.

Hypertrophy of the two medial metatarsals may be seen as compensation for long-standing extended pronation. This is more evident in the 2nd metatarsal, in which the cortex will be hypertrophied and its diameter increased by about half the normal.

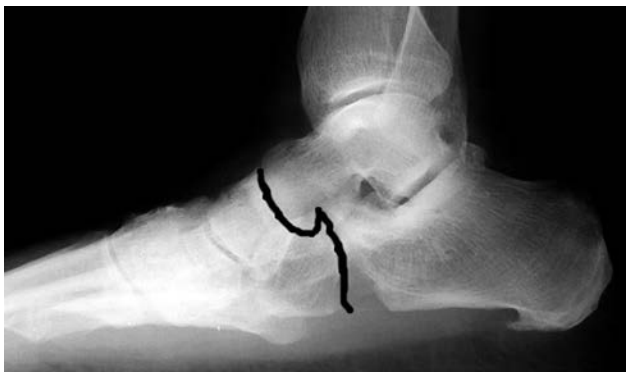
Tarsal coalition. Standard foot x-rays will not usually demonstrate a calcaneonavicular coalition. It is generally observed by the 45° medial oblique projection. The central ray is directed to the center of the everted midfoot. This is a valuable view for studying the tarsus and the tarsometatarsal articulations. **(Yochum & Rowe, 2004; Murray & Jacobson, 1977)** Caution is necessary in interpreting the 45° oblique view for the calcaneonavicular bar. Incorrect positioning may cause bony overlap and simulate a bony coalition. **(Yochum & Rowe, 2004; Elkus, 1986)**



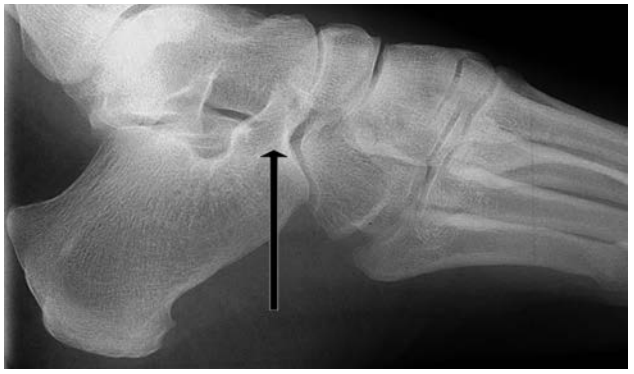
Normal Cyma line



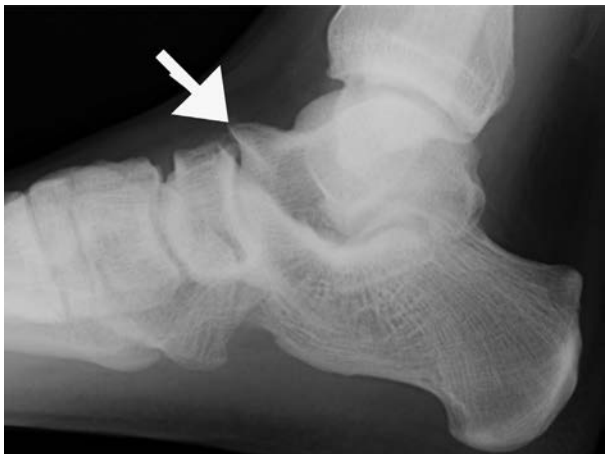
Lateral Normal Cyma line



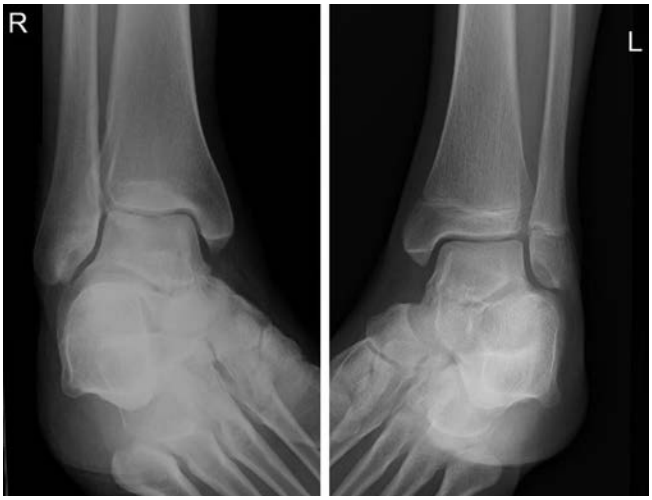
Lateral Broken Cyma line of pes planus



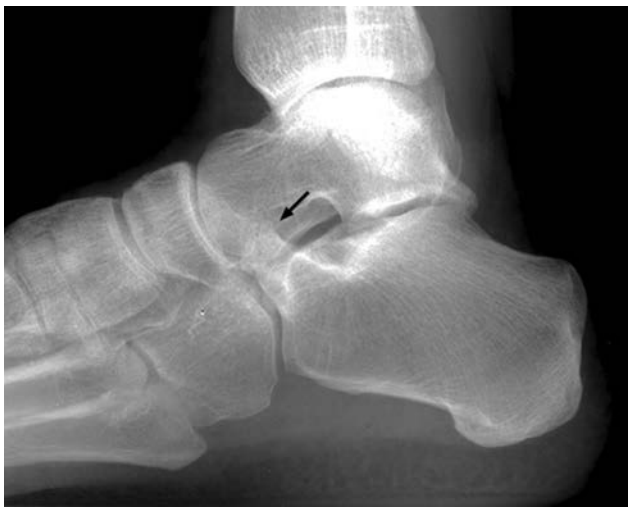
Calcaneonavicular coalition, ant-eater sign



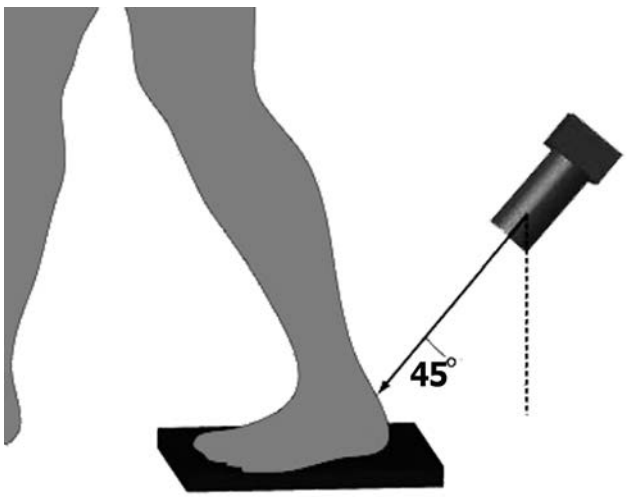
Talocalcaneal coalition



Medial oblique projection of foot



The arrow indicates the location of calcaneonavicular coalition when it is present.



Harris Beath X-ray position.

A view described by Harris and Beath **(Harris & Beath, 1948)** is used to visualize the talocalcaneal coalition. It is an axial view with the central ray angled at 45° and centered between the two malleoli. The properly angled central ray passes between the sustentaculum tali and neck of the talus, **(Murray & Jacobson, 1977)** clearly showing the joint space. When there is a talocalcaneal bridge, the joint is obliterated. This view, or slight variations of it (40°-50 if necessary), will usually reveal a bony coalition of the medial or posterior facet. X-rays of both feet are taken simultaneously to compare symmetry. The talocalcaneal joint angle can be quite variable. When adequate visualization of the joint facet is not obtained, lateral standing views are used to obtain the precise joint angle to determine the exact projection. **(Yochum & Rowe, 2004; Percy & Mann, 1988)**

Subtle changes must be sought in considering fibrous and cartilaginous unions. These changes include close proximity of the two bones, irregular and sclerotic articular surfaces, and hypoplasia of the talus head. **(Conway & Cowell, 1969)**

Plain x-rays are usually the first approach when investigating for tarsal coalition, with computerized tomography reserved for questionable cases. **(Yochum & Rowe, 2004; Elkus, 1986)** The latter can distinguish between bony, cartilaginous, and fibrous fusion. CT is the choice method for talocalcaneal coalition; it is not as useful in other types. **(Logan, 1995; Pineda et al., 1986)**

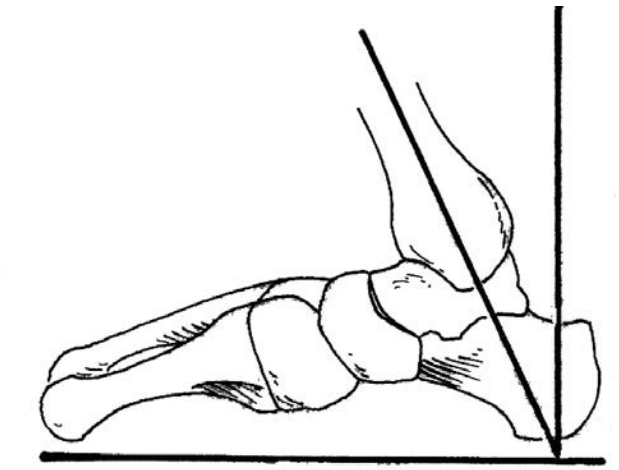
Tibiotalar motion. Ankle dorsiflexion and plantar flexion range of motion can be measured at the tibiotalar articulation on lateral x-rays. **(Weseley et al., 1969)** The transverse anatomic axis of motion can be established on the same x-ray. This is the amount of articular surface available for dorsiflexion and plantar flexion. Usually dorsiflexion is limited by a tight triceps surae; however, it can be limited by a bony abutment. Arthroplasty may allow 10°-15° of additional dorsiflexion. **(Lillich & Baxter, 1986)** Using this method, one can determine if limited dorsiflexion is due to bony encroachment or caused by soft tissue limitation, such as a short triceps surae.

The accuracy of this method is limited by the inherent problem of measuring movement of a three-dimensional structure with two-dimensional plain x-ray, and by potential placement and position errors of the patient's foot and leg. **(Yochum & Rowe, 2004)** Since the angles being measured are relatively large, the distortion inherent in the x-ray is acceptable and certainly gives more accurate information than any of the goniometer methods of measurement. Placement and position errors can be controlled by carefully following a protocol to enable comparative studies to be made. Placement of the patient refers to putting the patient's foot in a particular relation to the x-ray central ray to provide a base position from which to measure. This enables one to reproduce the starting point of the examination. Positioning refers to moving the patient into maximum dorsiflexion or plantar flexion without disturbing the base position of the foot, thus maintaining a static source from which to measure.

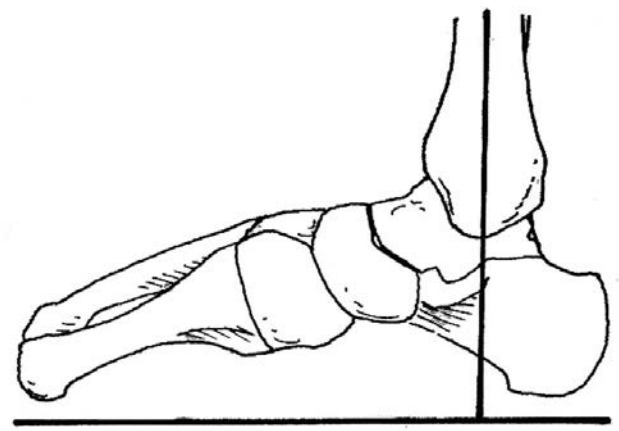
The patient's foot is placed on a cassette holder approximately 15" high. The surface on which the patient's foot rests must be visible on the resulting x-ray, as it will be the base from which measurements will be taken. In relation to a 10" x 12" film, place the foot so the metatarsal

heads will be close to the edge of the film, yet clearly visible. Align the foot so that a line from the center of the ankle mortise through the 2nd and 3rd toe interspace parallels the film, with minimal distance between the lateral malleolus and the film. Most of the body weight is put on the target foot by slightly bending the other knee and using that leg to provide stabilization. The central ray projects perpendicular to the center of the medial malleolus.

Positioning of the patient is in neutral, dorsiflexion, and plantar flexion. The neutral projection requires the patient to stand mainly on the foot being examined, with balanced posture. The dorsiflexion view can be done with the knee flexed or extended. In the flexed position, only the soleus is contributing to the triceps surae dorsiflexion limitation; with knee extension the gastrocnemius is also included. For the former view, the patient maximally flexes at his hip and knee and dorsiflexes at the ankle by crouching down. To include both muscles, the patient's stabilizing leg is put on a bench about the same height as the special cassette holder. It is moved forward as necessary with hip and knee flexion to maintain balance. Take care that the patient does not lift his heel from the bench or internally rotate his tibia. The latter is accomplished by increasing pronation, which is to be avoided.



Line drawing dorsiflexion



Line drawing extension

The extension view can be taken by having the patient move backward while keeping his foot static; this view is not usually taken.

The potential range of motion is determined from the potential articular surface and the anatomic axis of motion. The anatomic axis of motion is the meeting point of the anterior and posterior margins of the talus' lateral articular surfaces. The anterior and posterior limits of the trochlear articular surface are marked anteriorly at the junction of the neck and trochlea, and posteriorly at the depression adjacent to the posterior talar tubercle. A perpendicular line is drawn through the anatomic axis to measure the potential dorsiflexion and plantar flexion.

Tibiotalar dorsiflexion is determined by the angle of a line from the anterior tip of the tibial articular surface to the anatomic axis with the perpendicular anatomic axis line. In a similar manner, plantar flexion can be measured by projecting a line from the posterior tip of the tibial articular surface to the anatomic axis.

The combined tibiotalar and foot dorsiflexion range of motion is determined by the angle of a line projecting through the center of the tibial shaft and the center of its articular surface with the substrate. This amount is always greater than the tibiotalar motion alone. The difference indicates the amount of midfoot breakdown with extended pronation.

X-ray can be used to determine if the midfoot is breaking down with triceps surae stretching procedures. The foot can be x-rayed in a non-weight-bearing position, weight bearing, and then with full ankle dorsiflexion while weight bearing before starting the stretching program, and after performing it for a specified time.

Joint and Muscle Correction

Ideally, correction is directed toward primary factors. Primary factors can be found by applying numerous examination techniques at once or in succession to determine what eliminates dysfunction. **(Motyka & Yanuck, 1999; Schmitt & Yanuck, 1999)** For example, if the tibialis posterior tests weak and challenge to the talus or some other bone causes it to test strong, the subluxation is primary. Although the tibialis posterior could probably be strengthened by stimulating the neurolymphatic or neurovascular reflexes or with other types of treatment, it very likely would immediately lose its correction with walking or some other structural stress to the foot. If so, the primary cause of the tibialis posterior's weakness appears to be the result of improper stimulation to receptors by the subluxated joint. It might be due to the muscle vainly trying to stabilize the talus or some other bone in subluxation, and weakening because of its inability to do so. It is more likely that the neurologic model is correct. This, along with many other hypotheses, remains to be tested in basic research. Therapeutically the corrective sequence allowing corrections to be maintained after the articulation or muscle is stressed is the appropriate approach.

Muscles that control and support an articulation

should be evaluated to determine if they contribute to the development of the subluxation or fixation. Reversing the preceding scenario, one might find a subluxation of the talus by challenge that causes a previously normal muscle to test weak. An example is a lateral-to-medial challenge of the talus that causes a previously strong tensor fascia lata to weaken. Medial ankle support and integrity of the ankle mortise are largely the responsibility of the tibialis posterior. In addition to the associated ligaments, one can therapy localize to factors of the tibialis posterior such as the neurolymphatic reflex. While continuing the therapy localization, re-challenge the talus in the same manner as before and re-test the tensor fascia lata. If the challenge is no longer positive, the tibialis posterior is probably responsible for the subluxation. The same principle applies if the articular problem is a subluxation or a fixation. The talus in the ankle mortise rarely, if ever, fixates, but other areas can have either. In case of a fixation, the muscle(s) antagonistic to the weak one is probably hypertonic or shortened, which maintains the fixation consistent with Sherrington's Law of Reciprocal Innervation. **(Denny-Brown, 1979)**

When evaluating for articular fixations by motion palpation or applied kinesiology challenge, one of the bones must be adequately stabilized, especially when evaluating the cuneonavicular articulations. These are frequently in fixation, but in combination with a hypermobile talonavicular articulation. **(Chaitow & DeLany, 2002; Hammer, 1999; Gillet & Liekens, 1981)** Stabilize the navicular bone, then attempt motion between the 1st and 2nd cuneiform bones and the navicular bone.

Specific muscular dysfunction is found with several foot subluxations. A medial navicular subluxation often causes adductor muscle group weakness, while a lateral cuboid subluxation causes weakness of the tensor fascia lata and/or gluteus medius. These two subluxations appear to relate with improper nerve receptor stimulation, which correlates with lateral or medial sway of the standing extremity as explained under the magnet reaction. To test for tensor fascia lata or gluteus medius weakness resulting from a lateral cuboid, challenge the cuboid from lateral to medial. The tensor fascia lata and/or gluteus medius will immediately test strong with manual muscles testing if the association exists. Likewise, challenging the navicular bone from medial to lateral strengthens the adductor muscles. With different vectors of challenge, the associated muscle(s) performs to varying degrees during the manual muscle test. There will be one specific vector that provides optimal muscle function; this is the direction in which the bone must be adjusted.

These remarks are designed to help make sense of muscular, postural, orthopedic, and palpation findings and to offer an ideal method for confirmation. The changes that occur on MMT with specific challenges to the painful joints demonstrate for the patient and the doctor what is wrong and whether or not a HVLA manipulation has made a difference.

Once a dysfunction has been identified by virtue of a manual muscle, postural, palpation, orthopedic or other test, it is necessary to define precisely what type of dysfunction exists. The affect of this dysfunction upon attaching or remote muscle function is uniquely demonstrated for the doctor and patient by the challenge and therapy localization procedures in applied kinesiology. The associated muscle weakness, easily determined by the manual muscle test, is then investigated with a challenge procedure which seeks to find the articular change that corrects the muscle inhibition. Appropriate angular and pressure stimulation of the articulation usually produces immediate strengthening of inhibited muscles due to the dysfunction.

An interesting finding in some chronic musculoskeletal pathology is bilateral dysfunction in unilateral injury. Bullock-Saxton **(Bullock-Saxton et al., 1994)** found that subjects with chronic ankle sprain exhibit altered muscle activation patterns on both the injured and the uninjured sides. This supports the view that chronic pain is mediated by the CNS and suggests that clinicians remember to consider areas beyond the pain zone when addressing any chronic joint pain.

Articular adjusting. Subluxations and fixations are common in the foot and ankle. Unfortunately, this is also one of the most overlooked and unsuccessfully adjusted areas. Applied kinesiology challenge determines exactly how the correction should be made. With that information, plus an excellent knowledge of foot anatomy, manipulation should be relatively easy. Standard techniques of adjusting the feet can often be used to apply the optimal challenge vector for a successful correction. One advantage of applied kinesiology challenge is that it determines the individual characteristic of a subluxation. Sometimes it is necessary to modify or develop a different technique for adjusting the foot to properly apply the challenge vector for a particular patient.

It is a characteristic of applied kinesiology methods as used in clinical practice to move seamlessly from the gathering of information toward treatment. As a doctor searches for information through the manual muscle test, the appropriate challenge or therapy localization on the involved subluxation can turn "finding" into "fixing" in a moment. One treatment modality follows another as a rather "custom made" application is created that not only varies from patient to patient but should vary from one session to the next for a particular individual as their dynamic condition changes and improves. **(Motyka & Yanuck, 1999; Schmitt & Yanuck, 1999)**

In adjusting the feet the authors have consulted over a dozen books, DVDs, and some 50 articles on specific manipulative approaches to foot dysfunction. With applied kinesiology, you can modify the methods of manipulation to fit the particular needs of the patient. For instance in some standard lateral adjustments of the talus bone, the line of drive is nothing like that illustrated in standard textbooks for the manipulative maneuver. Applied kinesiology permits the discovery of the precise angle of manipulation determined by the sensorimotor challenge system of diagnosis. This information will change the contact points, the line of drive and the patient positioning significantly. The sensorimotor challenge system of diagnosis in AK allows us to be more specific in terms of the manipulative effort. In a bone as complex and multi-planar as the talus is for instance, challenge may show that only one or two of the articulations of the talus bone are suffering from subluxation or fixation. In this case, a broad general manipulation to the talus will not be helpful and may cause

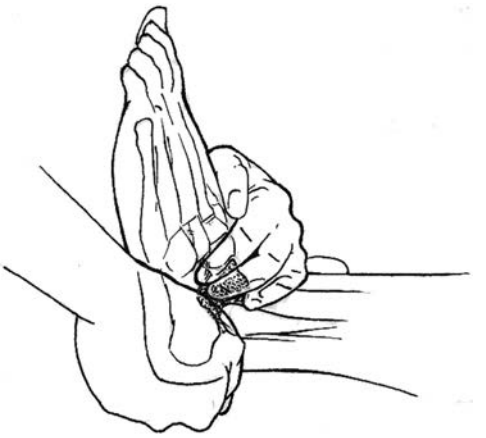
iatrogenic problems. In a case where a small portion of the talus is in a subluxated state with the adjoining bones, ligaments and muscles, a general manipulation that creates great crepitus may be inducing further trauma into the already injured joint. **(Ndetan et al., 2009)**

The most common factor causing corrective effort failure is not obtaining proper relaxation in the foot prior to the manipulative thrust. Only a small force is required for effective correction when proper relaxation has been obtained. Most chiropractors can relate to obtaining proper relaxation when adjusting the cervical spine. Sometimes the doctor can feel the patient signal the proper time by a sensation of "letting go" as relaxation is obtained. At other times it is necessary to distract the patient from the cervical area by having him place his hands on his abdomen, visual synkinesis just prior to the HVLA manipulation, or otherwise drawing attention away from the cervical spine.

Similar relaxation is required in the foot/ankle area prior to an adjustive thrust. Patients tend to hold the foot rigid while contact is being made prior to the adjustment. Sometimes it is adequate to simply say to the patient, "Let me have your foot; just make it loose." Other methods include having the patient move the foot against resistance and then letting it go, or distracting the patient by having him move an upper extremity. The latter method, however, is not as successful as when used to help relax the cervical spine. This is probably because of reciprocal inhibition when the arm and shoulder move toward the abdomen and the neck extensors - especially the upper trapezius - relax. The same principle of reciprocal inhibition can be used to aid manipulation of the foot. Ask the patient to move the foot in the direction opposite that of the manipulative effort just prior to applying the adjustive thrust. An example can be seen when the calcaneus is to be moved inferiorly and anteriorly, as in a typical tarsal tunnel syndrome. Have the patient dorsiflex his foot, then make the adjustive thrust as the patient relaxes to neutral; this causes reciprocal inhibition of the soleus and gastrocnemius.

It is necessary to recognize the importance of a correct vector of adjustment. Challenge provides information for the precise direction in which the correction should be made. Applying the technique to meet that need provides better results and makes adjusting easier. The only disadvantage is that sometimes the standard techniques do not precisely fit the pattern required by the patient's foot. Standard techniques can very often be adapted to fit the needs of the patient's joint. Manipulation of some of the smaller tarsal bones and their articulations is often best accomplished with the activator or Arthrostim instruments. **(Fuhr, 2008; IMPAC, 2012)** Sometimes it is necessary to develop an entirely new technique to meet the conditions presented.

It is not necessary to obtain an articular sound of separation when adjusting an articulation of the foot or ankle. Whether or not articular release is obtained, one should always re-evaluate the articulation with therapy localization and challenge to determine the success of the attempt. Continuing to attempt to obtain articular release when therapy localization and challenge are negative does not aid in the correction; the only result can be joint trauma.



There is limited talus contact area inferior to the malleolus for challenge and adjustment.

Contact for talus adjustment.

Described here are some standard techniques commonly used. Many manipulative techniques have been described in the literature. **(Bergmann & Peterson, 2010; Leaf, 2010; Greenman, 2003; Chaitow & DeLany, 2002; Hammer, 1999; Goodheart, 1998-1964; Logan, 1995; Hearon, 1981; Gillet & Liekens, 1981; Gertler, 1981, 1978; Stierwalt, 1976; DeJarnette, 1973)**

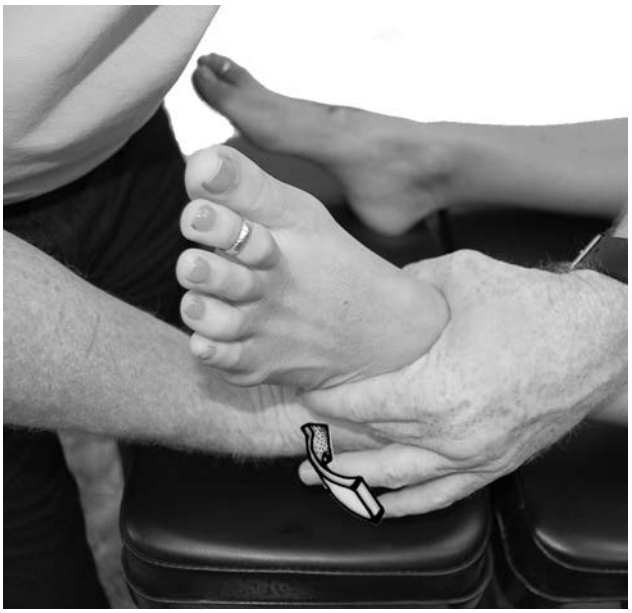
Lateral or medial talus. Most cases of excessive foot pronation require an adjustment of the talus in the ankle mortise. The direction of correction is usually lateral-to-medial, with a slight superior vector at the end of an extension maneuver. If the talus is subluxated medially, the correction is the reverse of that described here, which is for a left lateral talus.

With the patient supine, the physician cradles the calcaneus in his right fingers and palm. The thenar eminence wraps tightly under the lateral malleolus to contact the talus. It is emphasized that the contact must be as tight under the lateral malleolus as possible because of the limited lateral exposure of the talus. The physician's left hand contacts the dorsum of the foot. The little finger contacts the talus just distal to the tibia. Again, this contact should be as tight against the tibia as possible for the major

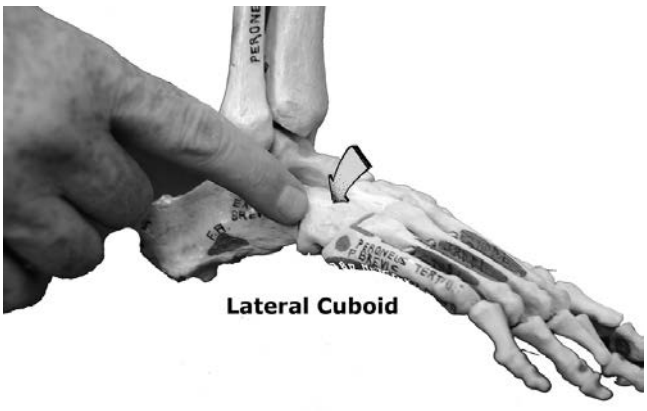
contact to be on the talus rather than distal to Chopart's joint. When proper patient relaxation has been obtained, the manipulation is performed in two quick maneuvers. First, both hands perform traction on the foot, with the left hand directing its force into the talus to open the ankle mortise. At the end of the traction thrust the physician's right hand moves the talus medially and slightly posteriorly, resulting in slight eversion of the foot.

Rotated cuboid. The cuboid often has a rotational component. It rotates about its longitudinal axis so that the plantar surface of the cuboid rotates laterally and the medial surface drops plantarward. Positive challenge that strengthens a weak associated muscle is a medial vector on the lateral plantar surface of the cuboid or a dorsal vector on the medial plantar surface. When challenging the cuboid, use the base of the 5th metatarsal as a landmark.

Correction of the rotated cuboid often requires adjustment in two steps. It is usually best to correct the lateral component of the plantar surface first. With the patient supine, the physician cradles the left calcaneus in his right palm. The left middle finger contacts the lateral dorsal surface of the cuboid. The index and ring fingers slightly overlap the middle finger to provide extra support. The rest of the physician's right hand wraps around the foot with the thenar eminence over the left middle finger, which covers the lateral dorsal aspect of the cuboid. While maintaining a firm contact on the cuboid with a firm wrist and hand, the physician adducts his right elbow to transmit the force through the wrist into the lateral superior aspect of the cuboid.



Adjustment of the lateral component of the rotated cuboid subluxation.



Contact for lateral cuboid correction

Sometimes the medial component of the rotated cuboid can best be adjusted by contacting the medial plantar surface of the cuboid with an activator or Arthrostim instrument. **(IMPAC, 2012; Fuhr, 2008)** This may be needed following the adjustment described above when there is still positive challenge on the medial plantar surface of the cuboid bone, and challenge to the lateral portion of the bone is negative. The adjustment is simple; all that is needed is proper alignment of the instrument in the optimal direction of challenge.

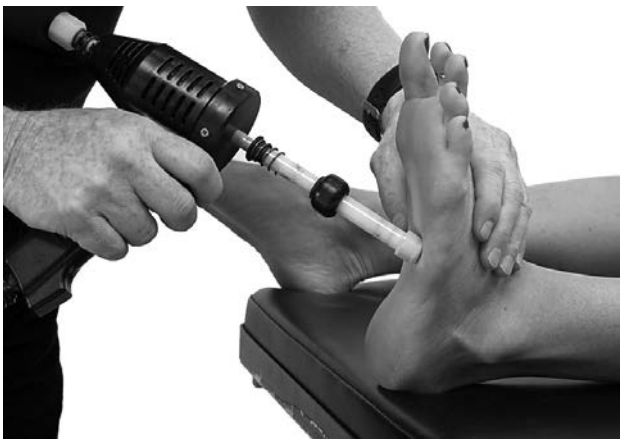
Lateral cuboid. The supine patient's left calcaneus is cradled in the physician's right hand. The physician's left thumb contacts over the lateral cuboid, and the thenar eminence of his right hand provides support over the thumb. The rest of the physician's left hand broadly contacts over the dorsal surface of the forefoot for general support.

The patient's leg is raised from the able with approximately 45° internal rotation. In doing so the doctor's arms flex. The corrective thrust is made by rapid contraction of the physician's triceps muscles, similar to a toggle recoil. With slight wrist action the thumb and thenar eminence move the cuboid in the direction of positive challenge.

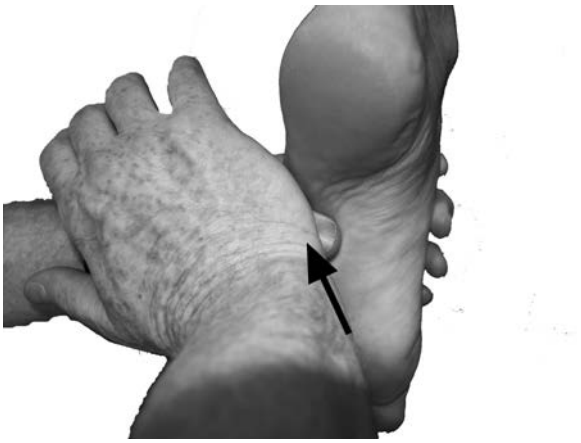
Inferior cuboid. The prone patient's left knee is flexed, and the physician holds the patient's dorsal foot in his left hand with his fingers wrapping around the plantar surface of the foot to support the metatarsals. The physician's left thumb contacts the inferior cuboid, and the pisiform of his right hand is placed over the thumb. The thrust is a toggle recoil-type in the direction of maximum challenge. The force is directed primarily to the cuboid bone, with support being provided only for the forefoot. Care should be taken to avoid direct torsion into the foot or ankle by twisting the patient's metatarsal bones or hindfoot.

Inferior navicular. The prone patient's left knee is flexed, and the physician cradles the dorsum of the foot in his left hand with his fingers wrapping around the dorsum of the foot. His index finger contacts the talus and his 4th and 5th fingers the cuneiform cuboid, and bases of the metatarsal bones. This leaves minimal contact on the dorsal aspect of the navicular bone by the middle finger. The left thumb contacts the navicular plantar surface, and the pisiform of the right hand is placed over the left thumb. A toggle recoil-type adjustment is made with a rapid triceps contraction. The dorsal contact by the physician's left hand supports and protects the talus and distal aspect of the midfoot.

Superior mid-tarsal bones. The talus, cuneiforms, and/or cuboid may have superior subluxations, i.e., toward the dorsal (anterior) surface of the foot. In some techniques these are adjusted by contacting the dorsum of the supine patient's foot and applying a traction thrust. In most cases it is better to avoid this technique because of the frequent hypermobility of the subtalar articulation and Chopart's joint.



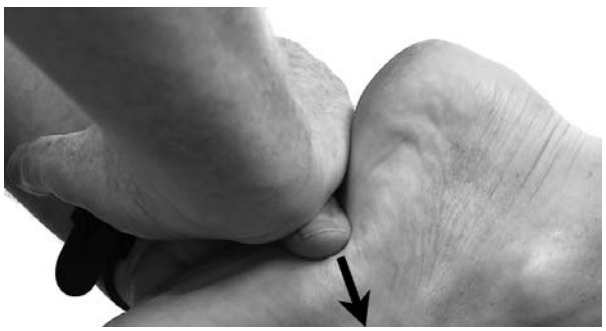
Adjustment of the medial component of the rotated cuboid subluxation.



Inferior cuboid adjustment.

An easy correction for superior mid-tarsal subluxations is to have the patient seated, leaning back. And supported by his hands. The hip and knee are flexed, and the foot is flat on the examination table. Contact the superior tarsal bone with a pisiform contact and use a quick toggle recoil-type thrust in the direction of positive challenge.

Another correction very often effective is cradling the patient's foot in any comfortable position and using an activator or Arthrostim instrument **(IMPAC, 2012; Fuhr, 2008)** to direct specific force in the direction of positive challenge.



Inferior navicular adjustment.

Inferior cuneiforms. A positive challenge to the 2nd or 3rd cuneiform is usually accompanied by a cuboid, navicular, or 1st cuneiform subluxation. Frequently when these corrections are made, the 2nd and 3rd cuneiform bones will no longer be subluxated. If there is a persistent inferior cuneiform subluxation, it is generally easily corrected by using an Arthrostim or Activator instrument **(IMPAC, 2012; Fuhr, 2008)** on the bone in the direction of positive challenge while the physician cradles the foot in any comfortable position.

Plantar Fascitis and Heel Spurs

Etiology and symptoms

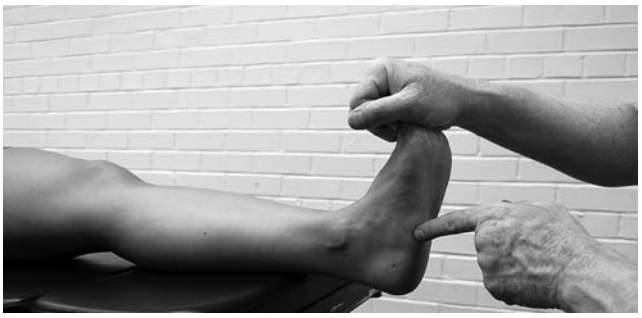
Painful heel is a relatively common condition and the most common cause of heel pain, affecting 10% of the population. **(Crawford, Thomson, 2003)** Plantar fasciitis was first attributed to tuberculosis by Wood in 1812, and since then has been called by many pseudonyms. Plantar fascial insertitis is another, as well as calcaneal enthesopathy, stone bruise, calcaneal periostitis, heel spur syndrome, jogger's heel, subcalcaneal bursitis, subcalcaneal pain syndrome, neuritis and calcaneodynia. **(DeMaio et al., 1993)** As will be seen, plantar fasciitis often presents as a combination of clinical realities rather than one discrete pathophysiological disturbance. For this reason plantar fasciitis should be considered as a syndrome rather than a single condition.

Painful heel is usually caused by plantar fasciitis, and the pain is intensified where the origin of the fascia attaches to the medial calcaneal tubercle.

There may also be diffuse tenderness up the medial or lateral sides of the calcaneus, and is typical of more severe inflammatory processes. Pain is more severe when first rising in the morning. Many patients have a problem putting weight on their feet, and the pain may be severe for the first 50 to 100 steps. Sometimes the pain is so great upon rising that it is impossible to walk; the patient may have to sit on the edge of his bed for 10-15 minutes, slowly putting more weight on his feet with rest periods in between. The pain will eventually decrease to the point that the patient can walk. It continues to gradually decrease with ordinary walking, only to again be increased the next morning.

This pain upon arising after rest may be due to the accumulation of inflammatory byproducts which are compressed into the nerve endings with weight bearing. Another theory is that the muscles and fascia involved in the production of plantar fasciitis are quiescent at night and non-weightbearing, so part of the heel pain occurs when the weight is placed upon the injured muscles and fascia. During periods of immobility, the plantar fascia relaxes and is relieved of its muscular activity and inflammation. The foot basically is re-injured each morning. **(Langer, 2007)**

Generally, plantar fasciitis has an insidious development. Most patients will tolerate the pain in the

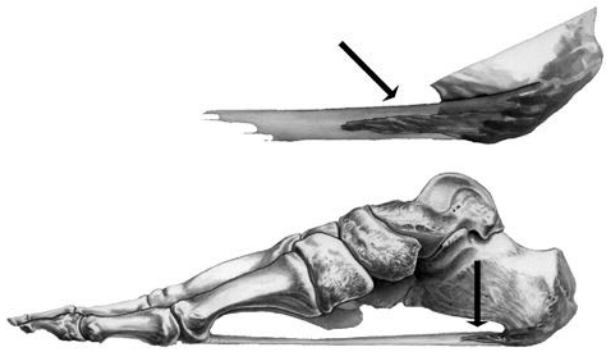


Position of heel spurs

earlier stages, expecting that it will improve. The condition typically intensifies to the point treatment is sought, with approximately 1 million physician visits per year for the condition. **(Riddle & Schappert, 2004)**

Both sedentary and active individuals can develop plantar fasciitis, but it is more common in runners. Lutter **(Lutter, 1997)** reports that 65% of plantar fasciitis sufferers are overweight, with unilateral involvement of the foot most common in 70% of the cases. Approximately 10% of all running athletes have the condition as well, which is about the same rate reporting it in the non-athletic population. **(Bartold, 2004)** In another sports medicine clinic, it is the fourth most common diagnosis. **(James et al., 1978)** The running and jumping in most athletic activities cause the condition to advance more rapidly. A runner's symptoms are further exacerbated by hill running and sprinting. If a runner tries to "run through" his pain during a workout it may improve, only to increase in severity at the end of the workout. To minimize symptoms, a patient tends to maintain his foot in supination from heelstrike to toe-off to avoid tension on the fascia. **(Bartold, 2004; Leach et al., 1986)** This disturbs the gait pattern, the strain of which may cause remote secondary problems.

Plantar fasciitis must be differentiated from generalized trauma to the soft tissue of the calcaneus, plantar bursitis, abductor hallucis myositis, and some types of arthritides. Plantar fasciitis has been associated with differing enthesopathies that occur with connective tissue diseases, particularly rheumatoid arthritis, psoriatic arthritis and ankylosing spondylitis. Men under 40 presenting with bilateral heel pain should be evaluated for Reiter's syndrome and ankylosing spondylitis. The foot is second only to the knee as the site of rheumatoid arthritis symptomatology. When pain is bilateral, the chance is greater that the condition has a systemic background. It must further be differentiated from peripheral nerve entrapment. Of particular interest is entrapment of the medial plantar nerve as it passes under the abductor hallucis muscle. It causes a burning heel pain, aching in the arch, and reduced sensation on the sole of the foot behind the great toe. It is believed to be caused by running with extended pronation, which puts tension on the nerve to stretch it against the tunnel whose superior surface is the navicular bone. It is at this point that maximum tenderness will be found on digital pressure. **(Bartold, 2004; Rask, 1978)**



Heel spur

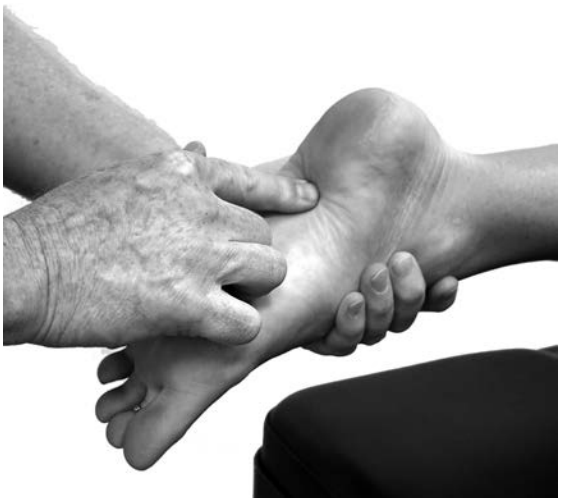
Painful heel may also be due to tarsal tunnel syndrome. For example, the mixed nerve to the abductor digiti minimi can be entrapped as it rises from the tibial nerve in the tarsal tunnel. Plantar fasciitis and calcaneal spur formation are associated with abductor digiti minimi atrophy on MRI of the foot. **(Chundru et al., 2008)** The nerve courses deep to the plantar aponeurosis near the muscle's origin at the calcaneus. **(Hamilton, 1985)** Many of the foots' vital neurovascular structures are in close proximity to the plantar aponeurosis. Both of these entrapment conditions are usually caused or perpetuated by excessive pronation. **(Cornwall & McPoil, 1999)** (Further discussion of peripheral nerve entrapment in the lower extremity is presented in **Chapter 3**.)

Conditions likely to increase the chance of plantar fasciitis are excessive pronation, cavus feet, leg length inequality, and obesity. **(Cornwall & McPoil, 1999)** When excessive pronation is a factor, it is usually related with a short triceps surae. This causes excessive strain on the plantar fascia, in turn producing chronic inflammation. Running and other athletic activities that stress the foot increase the chance of developing plantar fasciitis, especially in an older athlete. **(Langer, 2007; Bartold, 2004; Hill & Cutting, 1989; Warren & Jones, 1987; Brody, 1987; Krissoff & Ferris, 1979)**

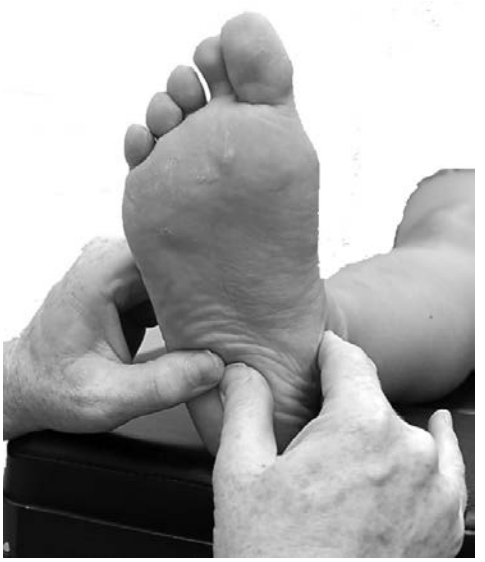
Although plantar fasciitis is common, rupture of the plantar fascia is not often seen. The propensity toward rupture of the plantar fascia may be accelerated by previous treatment of plantar fasciitis with steroid injections. **(Brinks et al., 2010; Leach et al., 1978)** When there is rupture, the patient may report having had a snapping feeling in his foot, along with sharp pain. There is severe localized swelling and acute tenderness. As the swelling diminishes, a palpable defect in the plantar fascia is observed. Conservative treatment is preferable if the plantar fascia ruptures, but occasionally surgical repair is needed. Rupture and partial or complete surgical sectioning of the plantar fascia, may lead to progressive pes planus with associated complications. **(Sharkey et al., 1998)**

Treatment to reduce pressure or shock to the heel is directed toward symptoms rather than the cause of the problem. Two examples of this are use of a heel pad with a depression and shock absorption material. In both cases temporary relief may be obtained, but the basic problem usually continues to get worse. Use of shock absorption material in heel pads or shoe construction may even ultimately increase the problem.

Wallden, Squadrone, Robbins and co-workers **(Wallden, 2010; Squadrone & Gallozzi, 2009; Robbins et al., 1987, 1988, 1989)** present strong evidence that shoes overprotect the foot, isolating it from actively contacting



Palpation for signs of heel spurs.



the substrate of natural turf. This diminishes the neurologic stimulation necessary to develop proper arch function. The observation that barefooted runners in international competition have fewer foot injuries stimulated research on the subject. Use of the recently developed shock absorption materials for athletic shoes decreases stimulation to the foot even more.

The need for manufactured shock-absorbing material under the foot can be questioned on the basis of the excellent absorption quality the natural heel pad appears to have. An *in vitro* **(Jorgensen & Bojsen-Moller, 1989)** study found the cadaver heel pad to have significantly greater shock-absorbing quality than 1.0 cm thick ethyl vinyl acetate (EVA) foam or Sorbothane (R) shock absorbers. It seems reasonable that the living heel pad tissue would perform even better. In those who have a painful heel syndrome, there is an increase in the natural heel pad thickness. **(Wearing et al., 2007; Amis et al., 1988)** However, a study by Tsai et al. **(Tsai et al., 2000)** investigated with ultrasound the heel fat pad in patients with plantar fasciitis. They concluded that the heel pad thickness was not altered in the control group compared to subjects with plantar fasciitis. It may be that other mechanical properties of the older foot and heel pad, for example the relative diminishment of shock absorbency (due to fixations or subluxations of the heel and foot), or to changes in the plantar aponeurosis connective tissue that occurs with aging, may all combine to increase the prevalence of plantar fasciitis with age.

The importance of shock absorption by the heel pad is questioned by Robbins et al., **(Robbins et al., 1989)** who point out that shock absorption of the heel pad diminishes when it "bottoms out" by tissue compression. They conclude "…that the heelpad does not impart significant shock absorption to the body during locomotion, and particularly during running." They propose that the role of the fat pad is protection of the calcaneus by selective tissue deformation. The plantar and palmar surfaces of the feet and hands are similar histologically. They have a pad of adipose tissue with a dense network of fibrous trabeculae providing protection to the bones. At the heel there is less penetration from uneven forces, probably due to tighter tethering of the fibrous trabeculae. The tissue is looser at the metatarsophalangeal articulations for joint movement. Robbins et al. **(Robbins et al., 1989)** suggest that the primary protection from concussive forces to which the foot is subjected comes from plantar sensory feedback. The key factor of the natural heel pad, as opposed to manufactured ones, is that it does not insulate the nerve endings from receiving normal stimulation from the substrate.

In the feet and hands there is a high density of mechanoreceptors and nociceptors, including Meissner's corpuscles that are found only in the digits of higher primates. There is greater sensitivity at the plantar surface of the metatarsophalangeal articulation than at the heel or distal toe. **(Squadrone & Gallozzi, 2009; Robbins et al., 1989)** Muscular reaction varies with stimulation to different areas of the foot. **(Lever, 2006; Nakajima et al., 2006; Kugelberg et al., 1960)** Stimulation to the sensitive metatarsophalangeal area, such as in the later portion of the stance phase, causes reflex contraction of the foot's intrinsic flexor muscles. **(Nakajima et al., 2006; Kugelberg et al., 1960)** They show maximum EMG activity from approximately the time of metatarsal strike until the metatarsals break contact with the substrate. **(Basmajian & De Luca, 1985; Sheffield et al., 1956)** This provides muscular support to the longitudinal arches, and aids the plantar fascia (Hicks' windlass mechanism) **(Hicks, 1954, 1955)** in returning the pronating foot to a solid lever by resupination. There is greater activity in the intrinsic muscles in extended pronation during gait than is apparent in the normal foot. **(Mann & Inman, 1964)** This appears to be the body's adaptation when the plantar fascia is not adequate to resupinate the foot by the windlass mechanism.

Stimulation to the hollow of the medial longitudinal arch causes toe extension. **(Nakajima et al., 2006)** This is an area that receives little or no stimulation when a person is barefooted; in the shod foot it is stimulated, especially when the shoe has arch supports. In the normal foot, arch support may be counterproductive because it interferes with the reflex activity of intrinsic muscle contraction provided by stimulation at the metatarsophalangeal area. **(Squadrone & Gallozzi, 2009)** This occurs when the foot needs the added support of the intrinsic muscles to stabilize the subtalar and mid-tarsal joints, making it a solid lever for

push-off. The evidence that sensory receptors and reflexes in the foot and lower leg are functionally linked with the lower back (gluteals, erector spinae muscles, among others) has been well-documented. **(Clair et al., 2009)**

In the normal foot, reflex control of muscles provides the complex adaptation the foot needs to meet the extreme stresses of daily activity. Failure or interference with these mechanisms often results in the painful foot or heel syndrome, and may be an etiological factor in recurrent low-back, shoulder and neck pain syndromes as well. **(Lephart & Fu, 2000)**

Heel spurs have little to do with the pain one experiences; they are simply the result of chronic plantar fasciitis. **(Merck Manual, 2011; Bartold, 2004)** There are no clear studies to show the association of heel spurs and plantar fasciitis. There is no relationship between the size of heel spur and the amount of trouble the patient experiences, **(Lewin, 1959)** nor in his ability to recover. **(Leach et al., 1986)** The heel spur develops in extended pronation because the windlass mechanism increases stress on the plantar aponeurosis. **(Bartold, 2004; Marshall, 1978)** What appears to be stress to the plantar aponeurosis and flexor brevis attachments to the calcaneus is evidenced by a high percentage of positive bone scans at that area. In one study, **(Williams, 1987)** 60% had a positive bone scan; those who did had more severe pain than those with a negative scan did. These authors describe the "saddle sign," which is a concavity in the calcaneus just posterior to a heel spur, if one is present. There is not usually an erosive quality to the saddle sign; rather, it appears to represent a healing of the bone.

Treatment

First it is necessary to differentially diagnose the cause of heel pain. There may be more than one condition present, but frequently the problem will be extended pronation. The function of the plantar fascia during gait is augmented by the dynamic actions of several other extrinsic muscles of the foot. Correcting or controlling extended pronation usually requires strengthening muscles that test weak, adjusting subluxations and fixations, and stretching the triceps surae. Lutter **(Lutter, 1983)** stresses strengthening the tibialis posterior muscle. This muscle is frequently found to be an etiological factor in painful heel syndromes in applied kinesiology practice. Tibialis posterior is particularly important in this regard, with the anatomic location and activity profile of the tibialis posterior muscle suggesting that it helps maintain the medial longitudinal arch during locomotion. The actions of flexor digitorum longus and flexor hallucis longus are also critical to arch stability and may assist the actions of the plantar aponeurosis in the later stages of the stance phase of gait.

The applicability of orthotics is an individual consideration. Orthotics may be needed when there is severe breakdown of the midfoot and stretching of the plantar fascia, at least on a temporary basis. In other cases, they may do more harm than good by stimulating the sensory nervous system in the hollow of the medial longitudinal arch, as previously explained.

Conservative treatment is effective for this condition. A report by Pfeffer **(Bartold, 2004)** to the *American Orthopaedic Foot and Ankle Society*, supports this. In this randomized and blinded clinical trial of 256 patients with isolated heel pain syndrome, 72% improved over the 8 week study period with treatment to the muscles (stretching) alone.

Lutter (1997) reports that 85% of patients with symptomatic plantar fasciitis will respond to conservative management, with surgery indicated for the remaining 15%. However this report concludes that plantar fasciitis is a degenerative, not inflammatory process, which contradicts the bulk of the literature and the pathology and imaging studies. Hambrick, **(Hambrick, 2001)** describing the applied kinesiology management of plantar fasciitis, shows how this kind of assessment for inflammatory as well as degenerative plantar fasciitis provides a more comprehensive method of management of this condition. Most researchers and clinicians agree that athletes and non-athletes with insertional plantar fascial pain can achieve good results without resorting to surgery. Toomey **(Toomey, 2009)** reports that 90% of patients will experience a full recovery with conservative medical management but that this may require 6 to 12 months of treatment and positive encouragement by a physician. Loomey et al **(Loomey et al., 2011)** employed Graston instrument soft-tissue treatment for 10 patients with plantar heel pain with 70% of the patients experiencing clinically meaningful improvements. Treatment was directed to the triceps surae, soleus, plantar fascia, and medial calcaneal tubercle. Areas of fibrous adhesions were detected and treated with the Graston instrument, followed by static stretches and finally ice was applied to the plantar surface of the foot for 15 to 20 minutes.

Relief of pain usually comes within the first week or two of appropriate, multi-modal applied kinesiology treatment. **(Wyatt, 2006; Wynne et al., 2006; McDowall, 2004; Hambrick, 2001)** With chronic conditions it takes time to obtain optimal correction. **(Martin et al., 1998)** Lutter **(Lutter, 1983)** states that plantar fasciitis takes longer to recover from than other running injuries to the foot, with an average length of 7.7 weeks away from running. Cycling or swimming can be substituted during the rehabilitation.

In our experience, treatment by surgery or injection is very rarely indicated. Steroid injections cause marked local osteoporosis **(Brinks et al., 2010; Amix et al., 1988)** and increase the chance of plantar aponeurosis rupture. **(Leach et al., 1978)** The effectiveness of proper conservative treatment is indicated by a report from orthopedic surgeons who state, "We have not found it necessary to operate on a calcaneal spur for the past 24 years and we have not injected the painful bursa over such spurs in 10 years." **(Wickstrom & Williams, 1970)** Although many types of treatment, from casting to drugs, are used for the painful heel, **(Bartold, 2004; Gill, 1987)** the conservative approach described here seems best.

Sometimes it is necessary to provide temporary symptomatic support. When early morning walking is intolerable, it can usually be improved by applying moist heat to the foot's plantar surface. **(Greenawalt, 1989)** If a heel pad is used, the portion under the pain should be relieved with a horseshoe opening rather than a hole. This transfers weight to the surrounding painless tissue and takes

pressure off the plantar fascia. **(Stuber & Kristmanson, 2006; Milgram, 1983)**

Sometimes elevation of the heel with heelpads or high-heeled shoes is recommended for painful heel. This increases the load on the posterior foot **(Katoh et al., 1983)** and is not recommended. When this method gives relief, it is due to relief of strain caused by a tight triceps surae. The proper treatment is to correct extended pronation, which will probably require stretching the triceps surae.

As the plantar fascia returns to normal, gradually increase the patient's activities and sports. Shoe evaluation with AK techniques is critically important. Using this method, one sees how often orthotics and certain types of supportive shoes create more problems than they help. Unless the patient's foot function is severely broken down, encourage him to increase foot function by training or doing some daily activities barefooted, preferably on natural turf. The glabrous epithelium is quite capable of hyperkeratinization to resist wear, **(Robbins et al., 1989)** but the activity should begin slowly for the adaptation to take place.

Plantar fasciitis due to a cavus foot is from failure of shock absorption because of the foot's rigidity. **(Badlissi et al., 2005)** The stress is then transferred to the plantar fascia. The therapeutic approach is to correct any fixations, begin stretching procedures to regain foot mobility, and stretch the tight fascia. Often the plantar muscles require application of the applied kinesiology fascial release and percussion techniques. **(Leaf, 2010)**

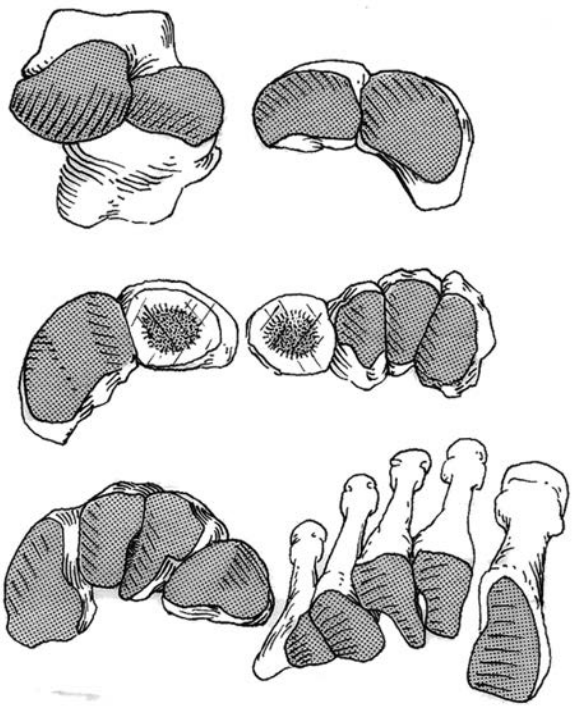
Conservative treatment for plantar fascia rupture is non-weight-bearing, with crutches, for as long as pain persists. Use local application of ice three times a day for fifteen minutes until acute symptoms subside, which usually takes eight to fourteen days. After pain subsides, use a small felt pad and taping to support the ruptured area, with the patient gradually increasing weight bearing over the next week. **(Sharkey et al., 1998; Leach et al., 1978)**

Forefoot in Extended Pronation

As previously indicated, the forefoot participates in pronation. It may be a factor in creating extended pronation, or stressed as the result of extended pronation. There are several types of forefoot problems, such as functional hallux limitus, hallux rigidus, metatarsalgia, stress fractures, and hallux valgus. In these or other dysfunctions of the forefoot, extended pronation should be considered since it causes or contributes to many of these conditions. Treatment to the localized forefoot problem will be ineffective or only partly successful if extended pronation is not corrected.

Transverse Arch in Extended Pronation

The transverse arch is important in stress dissipation. **(Abshire, 2010; Bartold, 2004)** Extended pronation, metatarsal head predominance, or improper footgear may result in pain from repeated microtrauma to the area. Both flexibility and strength of the metatarsal arch must be present.



The left illustrations are looking at the distal aspects of the articulations and the right look at the proximal aspect.

It helps to think of the transverse arch as being most of the length of the foot. **(Kapandji, 2010)** It begins with the calcaneus and talus articulating with the cuboid and navicular bones, and extends to include the three cuneiform and proximal metatarsal bones. In addition to the ligamentous support of the arch at this point, muscle action is important. The tibialis posterior tendon has slips that cross under the foot, with varying attachments to the cuboid, cuneiforms, and metatarsal heads. Kapandji **(Kapandji, 2010)** attributes maintenance of the cuboid-navicular transverse arch to action of the peroneus longus and the tibialis posterior. With medial longitudinal arch breakdown, there typically is weakness of the tibialis posterior. Excessive pronation worsens, and the scene is set for the transverse arch to break down as well.

Continued analysis of the transverse arch distally reveals that the wedge shape of the cuneiform and the cuboid bones provides Roman arch bony support for the transverse arch. At this point there is continued support from the peroneus longus and tibialis posterior muscles, which have intercrossing tendons. These tendons extend to the wedge-shaped base of the metatarsals. Kogler **(Kogler et al., 1996)** observes that although the foot manifests an arch-like appearance, it is not a true arch structurally and cannot maintain its arched shape solely as a result of its own architecture. Rather, the arch of the foot is primarily dependent upon adjacent soft tissues to maintain its arched position. As the transverse arch progresses distally into the metatarsals, it flattens with less support from ligaments and bony architecture. The transverse metatarsal ligament is a reinforcement of the deep plantar aponeurosis. **(Gray's Anatomy, 2004; Viladot, 1982)** It helps

keep the heads of the metatarsals together. The interosseous and adductor hallucis muscles hold the metatarsals together, with major contribution from the transverse head of the adductor hallucis.

A basic surgical principle, enunciated recently by the *American College of Foot and Ankle Surgeons*' heel pain committee, is that the dome of all the foot's arches should be maintained. **(Thomas et al., 2010)** This objective extends to conservative care. With loss of the arches, there are usually many subluxations and/or fixations of the articulations. Much can be accomplished in maintaining or rebuilding the arches by correcting fixations and subluxations, and improving muscular function with applied kinesiology techniques and rehabilitation exercises. **(Logan, 1995)**

There have been three theories about the forefoot's weight bearing and these are reviewed by Viladot. **(Viladot, 1982)** The tripod theory relates to primary weight bearing on the calcaneus and the 5th and 1st metatarsals. A second theory is that only the central metatarsals bear weight, based on the high incidence of callus formation under the 2nd, 3rd, and 4th metatarsals. These theories have been abandoned in favor of one that states all metatarsals bear weight. Using a split force plate measurement method, Morton **(Morton, 1935)** substantiated the theory. He also found that the 1st metatarsal carries twice as much weight as the others. He reasoned that since the 1st metatarsal has two sesamoid bones, each making contact, there are actually six contact points, each carrying equal weight. Using more sophisticated measuring devices, Cavanagh et al. **(Cavanagh et al., 1987)** substantiated that normally all metatarsals bear weight, but the maximum weight is born by the 2nd metatarsal head.

The metatarsals carry most of the weight of the forefoot, but balance and function are greatly dependent upon the action of the toes. The metatarsals make firm contact with the substrate because they do not have long, tendinous insertions. The toes, because of the grasping action of the flexor longus and brevis, give dynamic action to weight bearing of the foot. **(Squadrone & Gallozzi, 2009; Warburton, 2001; Viladot, 1973, 1982)**

The rays of the metatarsal arch vary in their flexibility. The 1st and 5th rays are most flexible and, because of the anatomical arrangement of Lisfranc's joint, the middle metatarsals — especially the 2nd and 3rd — are the most rigid. **(Viladot, 1982)** This can readily be observed in motion palpation of the normal foot.

The heads of all metatarsals are normally protected by a fat pad during their weight-bearing function. The 1st metatarsal, because of its role during the thrusting action of toe-off, has additional protection in the form of the sesamoid bones. These are embedded in the tendons of the flexor hallucis brevis muscle. Their ossification occurs between the ages of nine and eleven. The head of the 1st metatarsal is protected as it rolls on the sesamoid bones, while they have solid contact with the substrate as one stands on his toes or at the toe-off position of gait. A graphic picture of the sesamoid bones' role is painted by Viladot. **(Viladot, 1982)** He quotes the pictorial description of Hohmann, who describes the function of the sesamoid pad structure as acting like "a shoe that nature would have created for the support of the first metatarsals."

The lateral four metatarsals rarely have sesamoid bones, but their heads, like the 1st, have fat-pad protection. The fat pads are located between fibrous tissue attached to the proximal phalanges and the flexor digitorum longus tendon, to which they are attached by fibrous slips. The fat pads for the 1st and 5th metatarsals are generally separate from the pads for the 2nd-4th metatarsals, which may join to form one single pad.

With flexion of the proximal phalanx, as in toe-off, the fat pad is pulled forward by its fibrous attachment to the proximal phalanx. In normal function, this is advantageous because it keeps the fat pad under the metatarsal head as the toes flex during the stance phase of gait, thus providing protection. As described earlier, a clawing deformity of the toes develops with tarsal tunnel syndrome or any other condition that causes weakening of the short flexors while the long flexors maintain strength. In this case, the proximal phalanx hyperextension keeps the fat pad forward, causing loss of protection between the metatarsal head and the substrate. **(Hlavac & Schoenhaus, 1970)**

Types of Metatarsalgia

Pain under the forefoot is not necessarily metatarsalgia. Tarsal tunnel syndrome, Morton's neuroma, and Buerger's disease can cause pain under the forefoot, but they are distinct clinical entities in themselves and must be treated as such for a successful outcome. Sometimes symptomatic padding under the metatarsal bones will relieve the forefoot pain, but the condition will probably worsen until the proper treatment is given. **(Cailliet, 1997)**

In a study by Scranton **(Scranton, 1981, 1980)** of 98 cases of pain under the forefoot, 23 different diagnoses were made. He classified these as primary, secondary, and from other than pressure in the forefoot.

Primary metatarsalgia is a condition in which pain under the forefoot is secondary to static imbalance in the weight-bearing distribution across the metatarsophalangeal joint(s).

- Static disorders
- Iatrogenic (post surgical)
- Secondary to hallux valgus
- Hallux rigidus
- D.J. "Morton's foot"
- Congenital
- Long 1st ray
- Freiberg's disease

Secondary metatarsalgia manifests increased pressure under the metatarsal heads, secondary calluses, and pain. Although there is obviously abnormal pressure under the metatarsal head, both the metatarsophalangeal problem and the primary problem must be addressed.

- Rheumatoid arthritis
- Sesamoiditis
- Post-traumatic
- Neurogenic
- Stress fractures
- Gout
- Short ipsilateral leg

There is pain under the forefoot, but not from direct pressure in the region. Primary treatment is directed to the originating cause of the forefoot pain.

- Morton's neuroma
- Plantar fasciitis
- Causalgia
- Tarsal tunnel syndrome
- Tumors
- Intermittent claudication
- Buerger's disease
- Plantar verruca (wart)

Metatarsalgia should not be used as a diagnostic term because it only describes the symptomatic picture, not the cause of the condition. One must determine the cause of the problem before proper therapeutics can be applied. Often the cause is found in several types of dysfunction. As is applicable in nearly all body function, all possible relating factors must be evaluated or only partial results may be obtained.

Metatarsalgia is very common, with a much higher incidence seen in women. **(Latinovic et al., 2006)** In a study of over 1,000 feet with metatarsalgia, 88.5% of those involved were women and 11.5% were men. **(Viladot, 1982)** Many indict women's shoes as a major cause of the condition. The reasonableness of this accusation will be clearly seen with the investigation of the mechanics of a frequent cause of metatarsalgia.

As the cause of metatarsalgia is corrected, there may be other benefits to the patient. As with all foot dysfunction, nerve receptors can be improperly stimulated to cause remote dysfunction in the body. The foot dysfunction may change the individual's gait, such as greatly reducing the time and force of weight bearing in the stance phase. **(Aniss et al., 1992; Bard et al., 1992; Grundy et al., 1975)**

Pronation. Although extended pronation is not mentioned in the three categories listed previously, it is a common predisposing factor. **(Cailliet, 1997)** Metatarsalgia usually results from breakdown of the midfoot. There may seem to be limited evidence of a short triceps surae. **(Hamilton, 1955)** This is usually due to the dorsiflexion range of motion examination taking into account the movement of the midfoot when only ankle dorsiflexion should be measured. Adequate ankle dorsiflexion is of particular importance in the athlete. **(Whitting et al., 2010; Lillich & Baxter, 1986)**

In correcting extended pronation, one must take into account bony subluxations and fixations and examine the function of the peroneus longus, tibialis posterior, and the intrinsic muscles of the foot. Of course, in correcting extended pronation, potential proximal problems at the pelvis, hip, knee, and ankle must be evaluated and corrected, as well as modular and gait dysfunction.

Loss of transverse arch. Goldthwait **(Goldthwait, 1894)** considers loss of the transverse arch to be one cause of metatarsalgia. An imprint of the normal foot reveals what he calls the re-entering angle. This is the sharp indentation of the imprint posterior to the ball of the foot, which joins with the imprint made by the lateral aspect of the foot. When there is loss of the transverse arch with a normal lateral longitudinal arch, there is a bulge rather than the sharp re-entering angle. Quite often loss of the transverse arch is in combination with loss of the medial longitudinal arch, and no re-entering angle is observable.

The re-entering angle can be observed on imprints of the foot made on paper by various methods like "The Wet Test", or by observing the foot imprint while a patient stands on elevated glass, or by observing imprints made in impression material, such as that supplied by Foot Levelers, Inc., to fabricate Spinal Pelvic Stabilizers®. **(Foot Levelers, Inc.)**

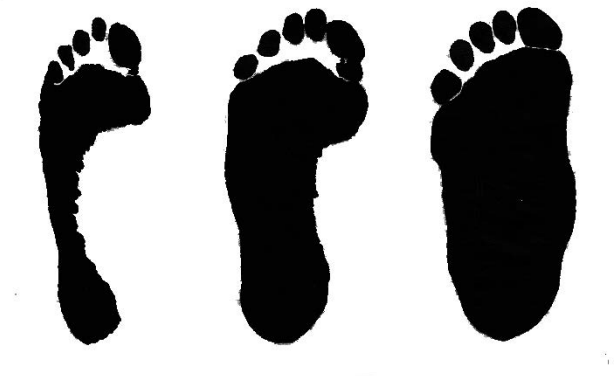
The wet test involves imprinting a wet footprint on a piece of paper to determine what kind of arch you have. **(Abshire, 2010)**

Fill a pan with water and submerge just the bottom of the foot. Quickly remove the foot and step onto a brown grocery sack, newspaper or dark-colored construction paper. Push the foot straight down and quickly remove it. Look at the shape left by the wet foot to identify the heel, midfoot, and forefoot and see if it fits one of the images.

You might see the other extreme on the paper with your wet footprint: a fairly smallish space for your forefoot that tapers back sharply to the lateral (outside) of the foot, so much that you might see the heel print as a separate imprint, just a round ball that is nearly or completely disconnected from the rest of the footprint. This means you have a high to very high arch and could be an under-pronator or supinator, but you could also be a fairly neutral runner if you have strong, developed feet.

If you see something that's somewhere in between the two prints just described, such as a wide forefoot print that tapers down gradually to the midfoot/arch region (but still leaves a wet spot that's an inch or wider) and then widens back to a round heel imprint, you likely have a medium arch. It's called a normal arch because it typically leads to normal pronation.

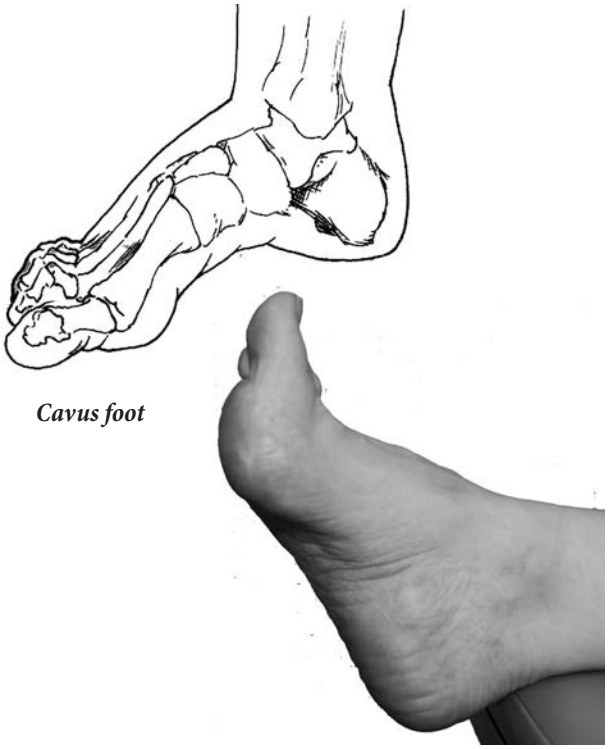
Footwear One of the most common causes of



Re-entering angle

Metatarsalgia from high-heeled shoes.

High heels and foot shape



Cavus foot

Cavus foot patient

metatarsalgia is women's high-heeled shoes. Whenever there is an increase in weight bearing at the metatarsal heads, there is the likelihood of metatarsalgia. **(Ko et al., 2009)** Three times the weight is carried on the metatarsal heads when high heels are worn as opposed to flat heels. **(Hong et al., 2005; Viladot, 1982)**

Pain at the metatarsal heads can be caused by tight shoes. **(Chaitow & DeLany, 2002; Mennell, 1969)** Women's shoes are further indicted here because of the pointed toes that fashion so often dictates. The foot is forced down into the wedge shape of the toe-box, cramping the metatarsal heads together. The pain is caused from chronic irritation of the bursa between the metatarsal heads.

Metatarsalgia may be due to improper weight bearing on the distal aspects of the metatarsal heads, from jamming the metatarsal heads together, or from both. Differential diagnosis can be obtained by palpating the plantar and distal surfaces of the metatarsal heads, taking care not to apply pressure between the metatarsal heads. Compare this with squeezing the metatarsal heads together. If pain is elicited on the plantar and/or distal aspects of the heads, the problem is from weight bearing. If no pain occurs there but does when the metatarsal heads are squeezed together, the problem is bursitis due to a tight toe-box. Pain on squeezing the metatarsal heads together is unique to bursitis. A common misconception is that pain from Morton's neuritis can be elicited by squeezing the metatarsal heads together. **(Hoppenfeld, 1982)**

Cavus foot. In the cavus foot, the anterior pillars of the longitudinal arches are too steep; consequently, there is an overload of weight bearing at the metatarsal heads. Typically, there is hyperextension of the metacarpophalangeal articulations and flexion of one or more interphalangeal articulations; however, all cases of pes cavus do not have this deformity. In this condition, there is poor tolerance to distance running. **(Fields et al., 2010; James et al., 1978)** In the severe cavus foot, the first stage of the stance phase is on the toes and metatarsal heads rather than on the heel. **(Fields et al., 2010)**

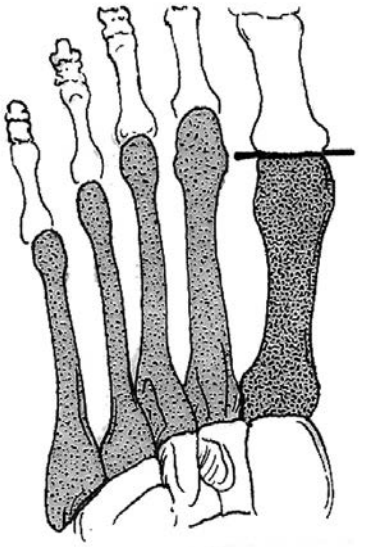
The cavus foot may be associated with severe neurologic disease, creating a need for a complete neurologic work-up. **(Hsu & Imbus, 1982)** It usually develops as a result of weakness of the triceps surae in neurologic conditions. The calcaneus moves into a cavus position because the intrinsic muscles are unopposed. **(Houtz & Walsh, 1959)** Shortening of the plantar fascia accentuating the cavus foot follows. **(Cailliet, 1997)** In the pathological cavus foot, surgery may be required for the individual to have a functional foot.

A milder form of cavus foot can develop from non-weight bearing over a prolonged period, such as in a protracted illness from which one finally recovers and can resume weight bearing. Mild pes cavus can be treated conservatively, and sometimes more progressive cases must be treated because they are poor surgical candidates.

Conservative treatment of the cavus foot requires regaining mobility of the articulations. Fixations and subluxations must be corrected. Foot and toe stretching exercises may be needed to stretch the ligaments and aponeurosis. Stretching the triceps surae and peroneal muscles is usually necessary. Vitamin B, large shoes, barefoot exercises, massage, diathermy, and hydrotherapy are all applicable therapeutic approaches. **(Maffetone, 2003; Viladot, 1982)**

Imbalance of weight distribution. The balance between the length of the metatarsals and toes may be a factor in metatarsalgia. As weight bearing moves through the forefoot with heel rise, it must move in a smooth arc around the metatarsal heads. **(Elftman, 1969)** Each ray must perform its function in weight bearing and ambulation. Failure to do so places additional strain on the other rays.

First ray insufficiency syndrome **(Viladot, 1982)** occurs when the 1st ray fails to carry its share of forefoot weight. This can be caused by a short 1st metatarsal, commonly called Morton's foot after Dudley Morton. **(Morton, 1935)** The condition may be confused with Morton's neuritis, which was first described by Thomas Morton. **(Morton, 1876)** The two conditions are totally independent, although each can cause metatarsalgia. When Dudley Morton's name is used to describe 1st ray insufficiency syndrome, it is called Morton's syndrome. When Thomas Morton's name



Morton's foot - short 1st metatarsal

is used for the nerve condition, which is usually between the 3rd and 4th toes, it is called Morton's neuritis, Morton's neuralgia, Morton's neuroma, or Morton's metatarsalgia, as well as several other names. In this text, the preferred term is plantar interdigital neuralgia. **(Plantar interdigital neuralgia is discussed in Chapter 3).**

In Morton's **(Morton, 1935)** description of the 1st ray insufficiency syndrome, the 1st ray was considered to carry twice the weight of the other individual rays. It has been demonstrated by more recent measuring devices that the 2nd ray carries more weight in both the Morton and non-Morton foot, but more in the former. **(Rodgers & Cavanagh, 1989)**

Metatarsalgia may be secondary to untreated hallux valgus **(Wang et al., 2009)** or the result of surgical treatment for the condition. Many types of operations for hallux valgus shorten the 1st metatarsal, creating an insufficiency syndrome like that of Dudley Morton's. Also described by Morton is a varus position of the 1st metatarsal of more than 15°, and posterior displacement of the sesamoids, both of which cause reduced weight bearing of the 1st ray.

The peroneus longus muscle, by its insertion on the plantar base of the 1st metatarsal, provides the strength of 1st ray flexion during the last portion of the stance phase. **(Beardall, 1975; Bartold, 2004; Lusskin, 1982)** Weakness of the peroneus longus leads to increased pressure on the 2nd and 3rd metatarsal heads, which may cause metatarsalgia. This weakness may also be responsible for inferior subluxation of the 2nd and 3rd metatarsal heads.

Normal adjustment of pressure on the forefoot is controlled in part by differential action of the toe flexors. **(Elftman, 1969)** Thus, when there is muscular imbalance of the intrinsic muscles, the dynamics of pressure on the substrate change; this may cause functional overload to certain portions of the foot. Accurate testing of the intrinsic and long toe flexors is limited. What often appears to be dysfunction of the intrinsic muscles can be observed by positive plantar therapy localization that does not associate with a subluxation. Treatment by neuromuscular spindle cell, Golgi tendon, or origin and insertion techniques often

Sesamoid and fat pad of hallux

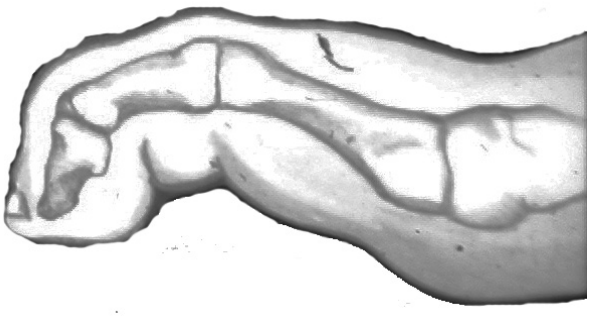
eliminates the positive therapy localization and appears to improve muscle function.

Sesamoiditis. The sesamoid bones of the hallux are covered with hyaline cartilage, providing an articular surface. **(Gray's Anatomy, 2004)** They increase the metatarsophalangeal flexion power by increasing the moment arm between flexion and extension to maintain a functional arc of motion. They also serve as shock absorbers by dispersing impact forces on the metatarsal head. The bones are concave in contact with the metatarsal head, and convex on the plantar surface. Between this convex surface and the skin is an adipose cushion of varying thickness. The mobility of the skin under the sesamoids helps reduce shear stress of the tissue itself and on the sesamoids during acceleration and deceleration. Between the plantar medial capsule of the 1st metatarsophalangeal joint and the medial sesamoid bone there is an inconsistent bursa. **(Richardson, 1987)**

Heavy activity such as running, jumping, and dancing on the balls of the feet can cause damage to the sesamoid bones. The resulting painful inflammation is called sesamoiditis. It heals slowly and has a tendency to recur. Sesamoiditis is typically found in the recreational long-distance runner, less frequently in other sports, and not usually in the sedentary person. The unusual factor is that generally there is no specific instance of trauma the athlete can describe to account for the beginning of his troubles. The complaint is that of increasing, poorly localized pain over several weeks. The condition tends to advance, and chondromalacia or roughening may result. Osteoarthritis may occur in these joints as a result of recurrent trauma. **(Cohen, 2009)**

Gentle palpation over the sesamoid area will generally localize the pain effectively. Swelling is present only in the more advanced chronic cases. Differential diagnosis is made by x-ray, and possibly a bone scan. Possible conditions are bursitis, sesamoiditis, osteochondritis, chondromalacia, degenerative arthritis, and fracture. Bursitis is indicated by swelling and redness. Other non-localized conditions, such as one of the rheumatoid variants, should be considered. A conservative trial is justified, except in displaced fractures. **(Cohen, 2009; Richardson, 1987)**

Treatment involves shifting the weight elsewhere by means of pads until the sesamoid heals. Padding, made from adhesive sponge rubber, is placed under the shaft of the 1st metatarsal for primary support to relieve the pressure on the sesamoid bones. The pad is extended under the shafts and heads of the 2nd and 3rd metatarsals to further reduce weight carrying of the hallux. **(Cohen, 2009; Bartold, 2004)** This type of J-shaped pad can be incorporated into an orthotic, and can even give relief in the case of sesamoid fracture. The support is not effective



Typical appearance of metatarsophalangeal hyperextension and interphalangeal flexion. Note that only the tips of the toes are visible.

in activities that require toe walking, such as dancing and skating. **(Axe & Ray, 1988)**

Non-surgical treatment is best. **(Cohen, 2009; Viladot, 1973)** Excising sesamoid bones is not as popular as it once was because of the unpredictable success rate. It may be needed in fracture cases, **(Axe & Ray, 1988)** such as acute fracture, stress fracture, or spontaneous osteonecrosis. **(Guebert & Thompson, 1987)**

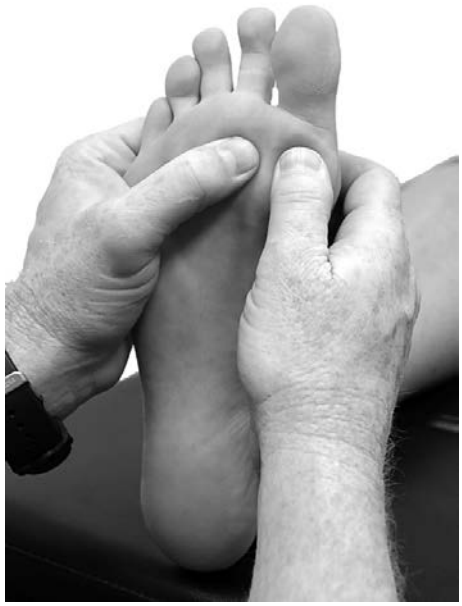
Metatarsalgia differential diagnosis. Metatarsalgia from improper weight distribution or function of the structures must be differentiated from interdigital nerve entrapment, functional hallux limitus, hallux rigidus, hallux valgus, Morton's foot, 1st metatarsal jam or turf toe, **(Maffetone, 1989)** intermetatarsophalangeal bursitis, various types of arthritis, and direct trauma to the area. These are some of the more common factors. The lists at the beginning of this section cover most of the conditions that can cause pain under the forefoot, but even the lists are not all-inclusive.

Consider factors peculiar to the patient, such as stress fractures in runners and Freiberg's disease in adolescents, which may require x-ray examination. A special view to determine standardized quantitative measurement of distal metatarsal head distance from substrate with weight-bearing has been described, **(Dreeben et al., 1987)** but its greatest value is in research.

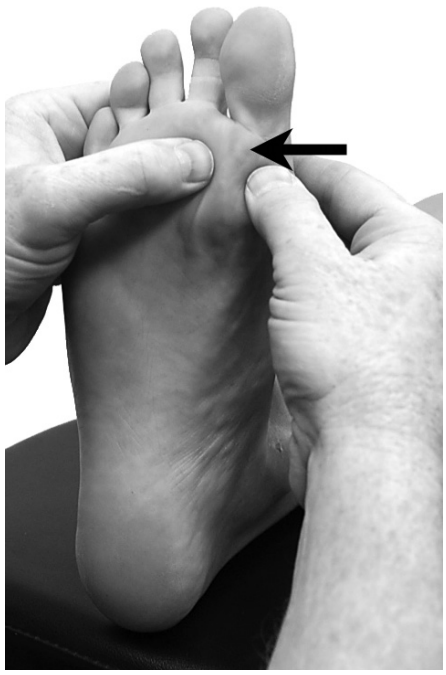
Diagnosis of local conditions is made by careful palpation of the plantar and distal aspects of the metatarsal heads for pain, and the metatarsals for position, flexibility, and joint play. Anterior displacement of the fat pad(s), as previously described, is a common cause of metatarsalgia. The metatarsal heads are tender to palpation. There is puffiness just anterior to the metatarsal heads. Because metatarsophalangeal hyperextension is usually the cause of the fat pad anterior displacement, one may see only the tips of the toes from the plantar view, with no visualization of the shaft of the phalanges.

Light palpation along the plantar surface of the metatarsal heads will reveal the inferiorly displaced bone. This is confirmed by either pressing the head inferiorly to weaken a strong associated muscle, or pressing it superiorly to strengthen a weak associated muscle.

These findings are correlated with the evaluation of the entire foot and remote influences on the foot, such as leg, knee, thigh, hip, and pelvic position and function. Applied kinesiology articular challenge and therapy localization, as well as the function of muscles, provide additional evaluation of the functional status of the total complex. In addition to evaluating the five factors of the IVF for the cause of muscle weakness, consider the possibility of peripheral nerve entrapment. Examples are: entrapment of the common peroneal nerve may be responsible for peroneus longus muscle weakness; tarsal tunnel entrapment may cause intrinsic muscle weakness. Both of these conditions are often associated with foot dysfunction. In the final analysis, all examination findings must correlate to arrive at the proper diagnosis for the cause of metatarsalgia.



Palpate inferior to the metatarsal heads for bony displacement and pain.



Palpate the distal aspect of the metatarsal head for pain, which indicates fat pad displacement.

Treatment

Since metatarsalgia is often only part of a total dysfunctioning complex, most of the conservative treatment is discussed elsewhere (pronation, proper shoes, and rehabilitation). The most severe cases of metatarsalgia are usually found in runners and others who similarly stress their feet. Conservative treatment will usually be effective even in these, with surgery only occasionally needed. **(Abshire, 2010; Maffetone, 2003; Lillich & Baxter, 1986)** Presented here are manipulative procedures for subluxations and fixations.

Sometimes, when challenging the transverse metatarsal arch to improve muscle function, one finds that re-forming the entire arch from the cuboid-navicular area all the way to the distal metatarsals best strengthens the associated muscles. In this case, there are probably many subluxations that must be corrected within the transverse arch and in the entire foot. One must progressively find and correct the subluxations and fixations to obtain maximum improvement of the associated weak muscles.

Positive challenge can be observed as weak associated muscles strengthen, or strong muscles weaken when a specific vector of force is applied to the articulation. It is best to find a muscle that is weak as a result of the subluxation and strengthen it with challenge. This determines the exact vector of force needed to correct the subluxation. When a previously strong muscle weakens, the opposite vector of force is required to make the correction. The problem with using this approach is that one cannot as specifically determine the precisely opposite vector of a positive challenge.

There are many descriptions of metatarsal manipulation and adjustment in the literature. **(Bergmann & Peterson, 2010; Leaf, 2010; Greenman, 2003; Chaitow & DeLany, 2002; Walther, 2000; Hearon, 1994; Viladot, 1982; Gertler, 1978, 1981; Gillet & Liekens, 1981; Maitland, 1977; Stierwalt, 1976; DeJarnette, 1973; Mennell, 1969, 1964; Strachan, 1954; Laylock, 1953)** Most are effective; each individual must find the procedures easiest for him to perform.

Because of the AK concept of the five factors of the IVF influencing each muscle, the determining factor regarding which treatment method to employ is reduced to the ones which practitioner has mastered and feels most confident. One technique may work as well as another so long as it is designed for the condition being addressed, muscular imbalance, and the principles of the treatment modality used are borne in mind.

Two types of manipulative procedures are necessary. One is to mobilize articulations that are fixed; the other is to correct subluxations. Motion should be available at each articulation. **(Petty & Moore, 1998; Gillet & Liekens, 1981)** Joint fixation is often found when challenging the various articulations for subluxations when the joint does not yield to the challenge. This is further evaluated by holding one bone and attempting to move its articulating partner. This type of palpation is described by Mennell **(Mennell, 1969)** Gillet, **(Gillet & Liekens, 1981)** Chaitow & DeLany, **(Chaitow & DeLany, 2002)** and many others.

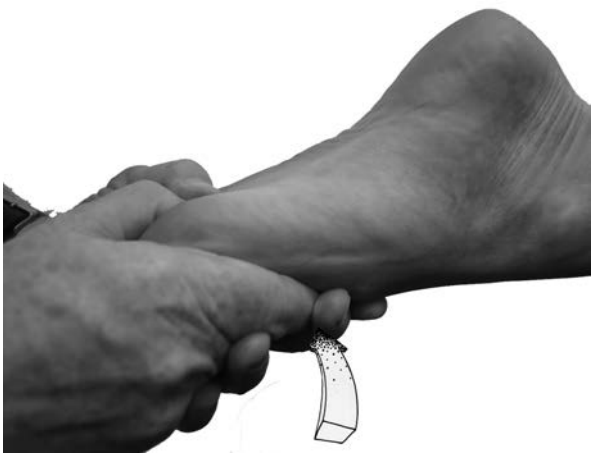
A fixation can be corrected by direct manipulation of the articulation, with the precise vector determined by challenge. Experienced clinicians will realize that many times the vector of misalignment is not the one commonly described in textbooks; the applied kinesiology challenge method will reveal the biomechanical individuality of patients with clarity. Another effective approach is to mobilize the joint by holding one bone and maneuvering the adjacent one in all directions.

The golf ball exercise, described under rehabilitation, is very effective in aiding foot mobilization. Restricted movement of the bones is often caused by tight shoes, which should be eliminated or the condition will recur.

One of the most common positive metatarsal challenges is a dorsal base with the head plantarly positioned. There may or may not be a subluxation of the metatarsophalangeal articulation. The two metatarsals most commonly subluxated are the 2nd and 4th. There will be a positive subluxation of one or both ends of the metatarsal. They will show a positive challenge independently; if both are present, they will show a positive simultaneous challenge.

Adjustment of the metatarsal whose proximal end has moved dorsally and distal end plantarly is accomplished in one maneuver. The physician's thumb is placed longitudinally over the plantar surface of the dropped metatarsal head. Depending upon the size of the patient's foot, the physician's 3rd or 4th finger is wrapped over the dorsal surface of the metatarsal base. The other thumb is placed crosswise over the plantar-contacting thumb, and

1st contact step - thumb contact on metatarsal head.



2nd contact step - finger contact on metatarsal base.

the fingers are wrapped over the finger contacting the dorsal base of the metatarsal. Solid, specific contact is maintained on the head, and specific contact is obtained on the base by emphasizing pressure on the contact finger directly over the base with the support finger. The patient's foot is slightly plantar-flexed so that when traction is applied, a separation is obtained at the base of the metatarsal. Continued traction moves the base plantarly, and there is a quick follow-through of the thumb to move the head of the metatarsal dorsally. This is a quick, continuous maneuver with three successive actions: 1) traction for separation of the base from the cuneiform(s), 2) plantar movement of the base, and 3) dorsal movement of the head. There will often be an audible release, but it is not necessary.

Support to the metatarsal bones is often done with padding, or it is built into an orthotic. Specialized x-ray has shown the effective lifting of the metatarsal heads by a pad placed just proximal to the heads. **(Dreeben et al., 1987)** Sometimes the pad is inadequately placed, thus creating new problems. For example, a pad to shift weight away from the sesamoid bones in sesamoiditis may provide adequate support under the 2nd and 3rd metatarsal bones and abruptly stop, causing the 4th metatarsal to drop inferiorly and resulting in a new subluxation. After placing new padding or obtaining a new orthotic, have the patient walk; then use applied kinesiology testing procedures to determine the support's effectiveness. No new muscle weakness should develop.

Freiberg's Infraction and Bone Fragmentation

Pain in the foot may be due to fragmentation of bone, generally of the metatarsals. The condition is usually found in the juvenile during the rapid growth period; in this case, it is considered an osteochondrosis called Freiberg's disease. It is a dorsal trabecular stress injury of the metatarsal head. Overuse and repetitive stress are initiating factors. **(Thordarson, 1996; Scartozzi et al., 1989; Trott, 1983)** Although frequently found in athletes, it is also common in those who do considerable marching, such as in drum and bugle corps and in the military. **(Trott, 1983)** Freiberg's disease occurs in the 2nd and 3rd metatarsals; it has rarely been found in the 4th. Young et al. **(Young et al., 1987)** describe a mechanism whereby the condition in adults may be due to a shearing compression-type recurrent injury at the interface between mineralized and non-mineralized articular cartilage (tidewater mark), rather than a true avascular necrosis.

Smillie **(Smillie, 1967)** described five stages of the disease, beginning with Stage I in which a fissure fracture develops in the ischemic epiphysis, to the final Stage V where there is total flattening, deformity, and arthritis. He found that the condition developed in structurally weak feet with short, varus, or hypermobile 1st metatarsals. **(Smillie, 1957)**

In the early stage, the only physical sign of Freiberg's disease may be pain on weight bearing and tenderness over the head of the metatarsal or over the metatarsophalangeal joint on digital pressure. **(Thordarson, 1996; Katcherian, 1994; Helal & Gibb, 1987)** Symptoms may subside, perhaps for many years, only to return with athletic activities, excessive walking, or with a girl wearing her first pair of high-heeled shoes. **(Maresca et al., 1996; Turek, 1984)**

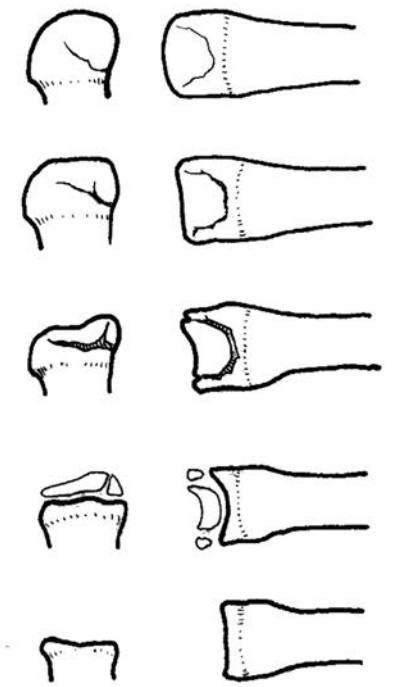
Plain x-rays may show no changes. A "hot spot" will be revealed over the affected head with a radionucleotide bone scan. A stress fracture of the metatarsal head is often mistaken for Freiberg's disease. **(Lutter, 1983)**

The condition may be present in a higher number of persons than those with symptoms in their feet. In routine x-rays of children attending Pottenger's **(Pottenger, 1945)** clinic, fragmentation of foot bones was found in a high percentage. Pottenger's procedure was to routinely take skull and foot x-rays to determine bone development in all the children he examined. This report is of the first 400 children studied between the ages of six weeks and eleven years. Most of the children were in the clinic because of health problems, with the following conditions diagnosed: asthma, 190; chronic bronchitis, 59; eczema, 45; allergic rhinitis, 11; gastric allergy, 9; rheumatic fever, 15; tuberculosis, 3; developmental problems, 39; severe malnutrition, 7; and "normal" children, 22. The study was divided into two groups. There were 221 subjects from six weeks to six years old, of which 182 had active fragmentation. Of the 179 in the second group, from six to eleven years, 103 had active bone fragmentation in their feet. The fragmentation was found in many areas of the foot, including the cuboid, navicular, cuneiforms, and metatarsals.

The interesting aspect of this study is that during the period of marked foot bone fragmentation, the children showed little or no outward evidence of extensive bone damage. There was no pain, limping, or other evidence of foot disturbance. Pottenger **(Pottenger, 1945)** attributed the bone fragmentation to repeated physiologic stress, as in early infant walking, and to "…severe metabolic upset such as an acute illness, or, of even greater importance, the recurrent insult of allergic manifestations."

Pottenger's study seems to indicate the need for general health care, including proper nutrition and correction of childhood illness. According to Trott, **(Trott, 1983)** proper foot support and cessation of the causative activity help resolve the situation in many instances. Surgery is usually required when the metatarsal head becomes involved, but it should not be removed. **(Katcherian, 1994; Drez, 1982)**

The usefulness of the applied kinesiology approach is that it focuses on factors which may be amenable to change and which make up the constellation of dysfunctions affecting the individual's total health picture. In applied kinesiology manual hands on diagnostic and treatment methods are used to assess and treat neuromusculoskeletal imbalances, and exercise and life style changes are given to help rehabilitate the biomechanical, biochemical, and psychosocial factors in a patient's total health picture. Simultaneously and integratively for the dysfunctions found, nutritional modifications are employed to address biochemical imbalances. Then psychological approaches are used to deal with psychosocial influences. Each of these modalities are employed in the care of most patients to address the totality of dysfunction that are found in most



Five stages of Frieberg's infraction.

patients, as was reported by Pottenger. It is necessary to address which ever of these (or additional) influences on musculoskeletal pain that can be identified in order to remove as many etiological and perpetuating factors as possible.

Pottenger, like Goodheart in the development of applied kinesiology methods, found that no tissue exists in isolation but it acts upon and is interwoven with many other structures. The body is inter-related from top to bottom, side to side and back to front, by the integrated muscular and nervous systems. When we work on a local area in the foot, we need to maintain a constant awareness that we are potentially influencing the whole body. Manual muscle testing evaluation can demonstrate this integrated functional relationship readily for both the doctor and the patient.

In the earlier stages, a surgical procedure developed by Smillie **(Smillie, 1967)** is effective in correcting the condition; for later use, a prosthesis of a small bi-stemmed universal joint spacer made of silicone elastomer has been used satisfactorily. **(Helal & Gibb, 1987)**

Although not as frequently, other areas of the foot can have bone fragmentation. There may be avascular necrosis of the talar head; **(Early, 2004; Thordarson, 1996; Katcherian, 1994; Schmidt & Romash, 1988)** this, along with osteochondritis dissecans of the midfoot, is difficult to diagnose. The condition is not typically demonstrable on plane x-rays. It is characterized by complaints of pain exacerbated by activity, followed by aching discomfort at rest that fails to respond to the usual applied kinesiology treatment. There may be loss of motion and swelling. A definitive diagnosis is made by radionucleotide bone scan. If the bone scan is normal, no further tests are needed. If the scan is positive, tomograms or a CT scan should be ordered. **(Early, 2004; Lehman & Gregg, 1986)** Treatment is usually conservative in the skeletally immature patient; surgery is performed in the skeletally mature.

Stress Fractures

Stress fractures tend to develop in untrained individuals who begin running or some other athletic programs too rapidly. Stress fractures can develop without athletic activity or increased training. McBryde **(McBryde, 1975)** reports that 95% of all stress fractures occurred in the lower extremity for athletes. New shoes, a change of running surface, or a new sport can cause stress fractures, especially of the metatarsals, upper third of the tibia and the fibula. **(Yochum & Rowe, 2004; Aspegren et al., 1989)** Unusual stress fractures can result from structural faults, such as hallux valgus causing stress fracture of the 1st toe proximal phalanx. **(Yokoe & Mannoji, 1986)** Activities that put unusual stress on the structure may result in stress fractures. The most common pathologic bone problem in dancers is the stress fracture. **(Goulart et al., 2008; Sammarco, 1986)**

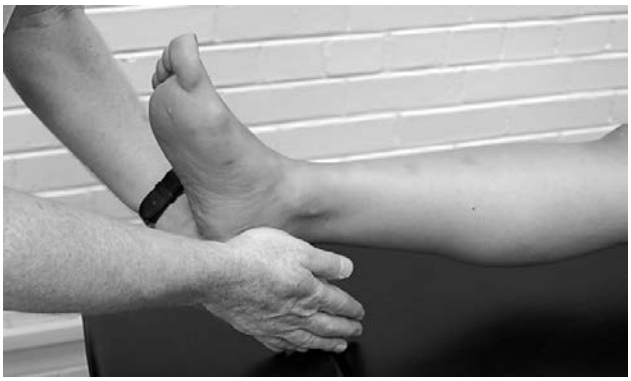
Military marching and other training procedures are examples of stress fractures that develop with rapidly increasing activity. Two-hundred ninety-five Israeli infantry recruits were evaluated for stress fractures that developed during basic training. **(Milgrom, 1985)** Thirty-one percent sustained stress fractures during basic training. Seventy-two percent of those who sustained stress fractures were followed for a minimum of one year to determine their continued status. Those who initially sustained stress fractures had more subjective complaints of bone pain during subsequent training. They were high risks for recurrent stress fractures, being over six times more susceptible than those who had not had stress fractures. This study reveals a markedly different recovery course in the individuals. The commonly recommended rest/recovery schedule must be adapted to the individual. Evaluation of recurrent stress fractures revealed that it was rare to have a stress fracture in a previously affected anatomical site.

Stress fractures are more common in the forefoot than in the mid- or hindfoot. A common area is in the center of the 2nd or 3rd metatarsal shaft, **(Lutter, 1983)** because the highest bending strain in shod distance running is in the 2nd and 3rd metatarsals. **(Gross & Bunch, 1989)** The frequency of occurrence is 2, 3, 4, 1, 5. **(McBryde, 1975)** Patients with low longitudinal arches are more likely to develop stress fractures in the forefoot. **(Simkin et al., 1989)**

The body adapts to stress when training is done properly. Evidence of the body preventing stress fractures is seen in hypertrophy of the 2nd metatarsal that develops in runners. **(Madjarevic et al., 2009)** This is the body's innate method of preventing the problem.

Generalized pain and unexplained doughy pitting edema over dorsal metatarsals indicate the possibility of a stress fracture. **(Merck Manual, 2011; Jahss, 1982)** X-ray may be negative for up to six weeks after initial onset of the condition; **(Yochum & Rowe, 2004; Jahn, 1985)** radionucleotide bone scan provides the definitive diagnosis.

Stress fracture of the 5th metatarsal is less common than in the other four. It may occur with no history of acute injury. **(De Lee et al., 1983)** When it does occur, the usual treatment is intramedullary screw fixation. **(Delee et al., 1983)** Three cases that refused surgery and were treated by casting are described. **(Acker & Drez, 1986)**



Heel squeeze test for possible calcaneal stress fracture.

They returned early to their regular athletic participation without recurrent symptoms or refracture.

It has been found that ballet dancers, female distance runners and gymnasts with irregular menstrual periods have calcium and other nutrient deficiencies in the diet that contribute to stress fractures due to loss of bone density. **(Lloyd & Triantafyllou, 1986)** Training in running shoes that are older than 6 months is also a risk factor for stress fractures. **(Gardner et al., 1988)** Once again it is important to note that in this text and its companion volume, substantial attention is given to musculoskeletal stress resulting from postural, emotional, chemical, and other influences. As will become clear in these discussions, there is a constant merging and mixing of such fundamental influences on the particular AK findings each patient presents. In making sense of the patient's problems, it is clinically essential to differentiate between these interacting environmental factors. The model which is used in applied kinesiology classifies negative influences into three categories:

- Structural
- Biochemical or nutritional
- Mental or psychosocial

Each of these influences can be assessed specifically (using the AK sensorimotor challenge and therapy localization procedures) in relationship to the muscular imbalances found using the manual muscle test.

Differential diagnosis of calcaneal stress fracture must be made from Achilles tendinitis and retrocalcaneal bursitis. More difficult to differentially diagnose is calcaneal stress fracture from plantar fasciitis. Pain from digital compression applied to the medial and lateral aspects of the calcaneus and positive x-ray findings at three weeks confirm calcaneal stress fracture. Treatment is not critical, and symptoms usually permit resumption of a full running schedule after three weeks. **(McBryde, 1975)**

Functional Hallux Limitus

Functional hallux limitus (FHL) was first described by Dananberg, a friend and patient of Goodheart. **(Goodheart, 1996; Dananberg, 1986)** FHL is the inability of the hallux to properly extend at the metatarsophalangeal articulation at the proper stage during the stance phase of the gait cycle, despite normal extension of this joint when non-weight bearing. Dananberg describes a new method of viewing gait efficiency, and presents a new entity not visible to even the most trained observer of gait. In evaluating disturbances in gait and their possible relationship to disturbances in the low back, Dananberg notes: **(Dananberg, 1997)**

> "When viewing X-rays of the patient, it is well known that a single view of the body is not acceptable. Generally, three views provide a far more accurate picture of a three-dimensional being. Viewing a patient walk is no different. Simply watching a subject walk back and forth in a hallway loses the entire sagittal plane view. Although most offices are not equipped for gait analysis, the use of the treadmill can be helpful in providing the multiple viewpoints necessary for accurate determination of cause and effect."

It is to Dananberg's credit that a podiatric blockage in the first toe is no longer seen only as a blockage of the joint or a podiatric problem alone, but as a neuromusculoskeletal dysfunction affecting the entire ambulant organism. Even a slight alteration in the mechanics of the foot can induce effects throughout the musculoskeletal system; the extreme sensitivity of the muscle spindles is responsible for this. **(Cramer & Darby, 2005; Mense & Simons, 2001)** The most common approach for diagnosing gait imbalances (for practical purposes) is based primarily on the visual observation of standing and gaiting posture. While this approach may sometimes be useful, it does mean that treatment based on this finding may be somewhat non-specific. Additionally, aside from subjective symptomatology, progress is difficult to gauge with such subjective approaches. Dananberg cautions that the visual diagnosis of muscular and gait problems is difficult. The different elements within the chain of events occurring with any particular movement that a patient undertakes before the examiner can occur within a fraction of a second, and too rapidly to be accessed separately in the absence of laboratory tools. Therefore, what is actually observed by the examiner is the grand total of how rapidly and smoothly a person can change between two activities – inaccurate, but for many clinicians, good enough. The AK manual muscle testing approach for dysfunction in the strength and movement of the great toe, and testing the muscles that move the great toe during the stance position of gait, as well the influence of functional hallux limitus upon remote muscle function throughout the body, is a great help in diagnosing this subtle but critical disorder.

Functional hallux limitus had gone unrecognized because there is complete range of motion at the metatarsophalangeal articulation when non-weight-bearing, as it is most often examined. The term "functional" differentiates this condition from one in which there is limited range of motion of the hallux at all times, such as in hallux rigidus.

It is obvious there is still some hallux extension in gait even with FHL, as noted by the upper shoe crease in this area. The failure of metatarsophalangeal joint extension is the timing. The restriction can be objectively observed with the Electrodynogram™ by the Langer Biomechanics Group, **(Langer Biomechanics Group)** which records foot forces with ground contact. Six sensors are applied to the

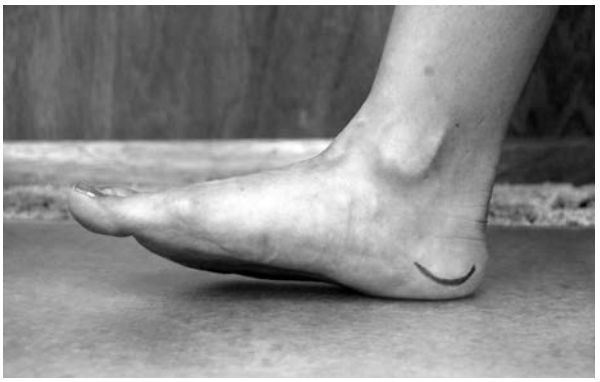
following areas: the medial heel; lateral heel; 1st, 2nd, and 5th metatarsal heads; and the interphalangeal articulation of the hallux. A seventh sensor can be placed at the will of the investigator. The Electrodynogram™ makes a computer recording of the temporal and force patterns of foot contact. The first metatarsophalangeal restriction varies in length and may be less than 100 milliseconds in duration and *invisible to the naked eye*. **(Dananberg, 1986)** Symptoms caused by FHL can be almost anywhere in the body, but they are rarely in the foot. Dananberg shows that many cases of acute or chronic low back pain are related to gait abnormalities that result from FHL. **(Dananberg, 2007)** To understand this it is necessary to consider the mechanics and dynamics of gait.

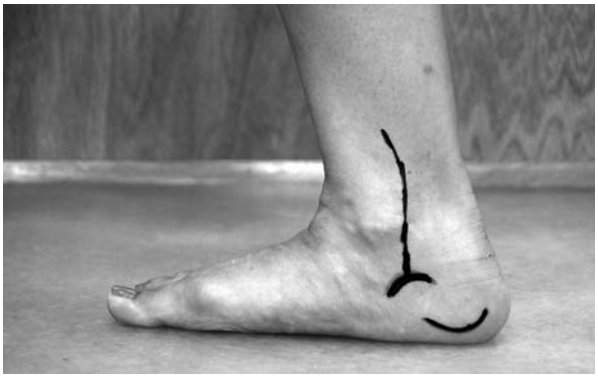
The gait mechanism is an excellent example of muscle action being conserved by the body when possible. It might seem that forward propulsion is primarily powered by muscle contraction, such as by the hip extensors of the stance leg; this is not the case. For example, the gluteus maximus is inactive except at the beginning and end of the stance phase. **(Dananberg, 1995; Basmajian & DeLuca, 1985)** Forward movement power is from the kinetic energy of the swing leg. This pull phase is sufficient to move the center of the body up and over the ipsilateral leg. **(Dananberg, 2007; Mann et al., 1986)** Gluteus maximus contraction at the end of the stance phase may be to control the swing phase energy.

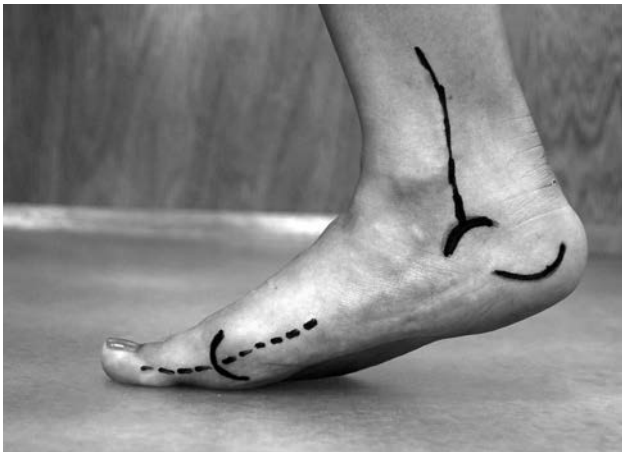
The gait cycle begins with a slight backward and then forward body movement as the swing limb flexes at the hip and knee. As the swing limb moves forward, movement through gait is powered by the kinetic energy of the swing leg. The forward motion of the limb moves the body's center of gravity forward, advancing it over the stance limb. The pendular kinetic energy of the stance leg is called the "pull force." The entire body weight pivots over the metatarsophalangeal articulation, which is fixed with the ground. The kinetic energy of the swing limb continues to advance the body's center of gravity past the ground contact. The stance leg produces rearward thrust on the ground, attempting to push it backward. Since the ground can't be moved, the pressure on the stance limb provides propulsion for forward motion without muscle action, except for stabilization. The greatest power input to the ground force for forward motion is at the point of weight bearing of the metatarsophalangeal pivot point. It is at this phase of stance limb motion that a momentary halt of hallux extension can disrupt the gait pattern. **(Dananberg, 1993)** There is maximum power to move the body forward at this point; limitation of hallux extension interrupts the proper sequential dissipation of energy, which must take alternate paths not in keeping with normal function. The relationship between FHL and more proximal postural, gait, and muscle problems have been discussed by Lewit, Chaitow & DeLany, and Liebenson. **(Liebenson, 2007; Chaitow & DeLany, 2002; Lewit, 1999)**

Chaitow & DeLany (**Chaitow & DeLany, 2002)** note:

> "This condition limits the rocker phase since 1st MTP joint dorsiflexion promotes plantar flexion. If plantarflexion fails to occur, there will be early knee joint flexion prior to the extension of that leg. The result of early knee flexion prevents the hip flexors from gaining mechanical advantage, thereby reducing the efficiency of the motion of







Upon heel strike, the round underside of the calcaneus serves as the pivot for gait movement. Once the flat foot is achieved, forward motion occurs at the ankle joint with dorsiflexion. Upon heel lift, ankle motion reverses to plantarflexion as the metararsophalangeal joint provides the balance for the forward plane motion.

> the swing limb. A further effect is that gluteals and quadratus lumborum on the contralateral side become overactive in order to pull the limb into its swing action. Overactivity of quadratus lumborum and/or the gluteals may encourage overactivity of piriformis. 'The reduced hip extension converts the stance limb into a dead weight for swing, which is exacerbated by hip flexor activity…resulting in ipsilateral rotation of the spine, stressing the intervertebral discs.'" **(Prior, 1999)**

The 1st metatarsophalangeal joint combines ginglymus and arthrodial type movement; that is, it has both hinge and glide functions. The hinge motion is approximately 15-20° of the total motion and the glide makes up approximately 50°, providing the total of 65-70° extension range of motion. With this amount of action, the heel can adequately rise to advance the leg while maintaining digital ground contact. The mechanisms that stabilize the foot come into play, and the plantar aponeurosis is shortened to raise and resupinate the arch and externally rotate the lower leg. This keeps the lower leg synchronous with the external thigh and pelvic rotation brought about by the swing limb pull force, and the energy is sequentially dissipated within the foot and leg. When full extension of the 1st metatarsophalangeal joint is limited and/or the temporal pattern is disturbed, the forces are directed elsewhere; this ultimately causes remote dysfunction and symptoms that are often not recognized as being caused by foot dysfunction.

The paradox of FHL is that the first MTP joint's sagittal plane pivotal motion is locked during all or portions of the single support phase of the gait cycle. This is true even though there is full range of motion of the MTP joint during the non-weight bearing examination. Compensatory movements for pain or dysfunction eventually become ingrained in the motor cortex, essentially reprogramming normal movement patterns. This aberrant gaiting, repeated at least 2,500 times per day (or 1 million strides per year), occurs over and over for the patient within the time span of approximately 600-750 milliseconds. **(Dananberg, 2007)** Pain will manifest most notably in those articulations moved by the inhibited muscles such as the medial and lateral knee, greater trochanter, sacroiliac, lumbosacral, lumbodorsal, cervicodorsal and upper cervical joints. The theory of the spinal engine **(Gracovetsky, 1989)** may be implicated in the FHL syndrome and lead to premature exhaustion of the spinal engine during gait. The alterations in the spinal engine with dysfunctional feet will presumably initiate pain, and pain forces the CNS to unload the overstressed articulations, i.e. produce inhibited muscles throughout the body during gait.

Foot Stabilization

Dananberg **(Dananberg, 1993)** describes three distinct mechanisms that permit the foot to support the applied stress during gait: (1) calcaneocuboid locking secondary to aponeurosis tightening, (2) the locking wedge and truss effect, and (3) the windlass effect. They rely little, if at all, on muscle function and are referred to as "autosupport."

Calcaneocuboid Locking.

Dananberg cites Bojsen-Møller's observation that the transition from the anthropoid to human foot to provide a rigid lever for propulsion and protection of the tissues from the extreme repetitive forces of walking and running. **(Bojsen-Møller, 1979)**

Relaxed, the ball of the foot is a soft and pliable pad. The plantar skin can be moved from side to side as well as proximally and distally. Rigidity of the tissue and bones comes with toe dorsiflexion. This is accomplished by the tissue arrangement divided into three transverse areas, each with a different mechanical function: (1) a series of transverse bands proximal to the metatarsal head in which the deep fibers of the plantar aponeurosis form 10 sagittal septa, eventually connecting to the proximal phalanges, (2) inferior to the head where vertical fibers form the joint capsules and the sides of the fibrous flexor sheaths to form a cushion below each metatarsal head with fat bodies, and (3) a distal area where the superficial fibers of the plantar aponeurosis insert into the skin. Metatarsophalangeal extension tenses the three areas, anchoring the skin firmly to the skeleton so that forces to the skin during push-off and breaking are transferred to the skeleton. **(Kapandji, 2010; Bojsen-Møller, 1979; Bojsen-Moller & Flagstad, 1976)**

The tissues of the ball of the foot are a complex network that provides plantar fascia tension to the calcaneocuboid articulation that becomes close-packed by pronation of the forefoot in relation to the hindfoot. It is congruency between the joint surfaces obtained in this position that provides strength. The calcaneus overhangs the cuboid dorsally, which stops the movement. The peroneus longus is a key to pronating the forefoot for high gear push-off and locking of the calcaneocuboid articulation. It assists in internal rotation of the crus, forcing the foot to use the transverse axes. **(Dananberg, 1993)**

Truss and Locking Wedge Effect.

In engineering a truss is a structure usually formed by a triangle or series of triangles. This is a stable arrangement because a triangle cannot be distorted by stress. **(*Encyclopedia Britannica*, 2011)** The triangles of the foot are made by the bones of the foot in combination with the plantar aponeurosis. The wedge effect is demonstrated by the stones in a Roman arch which, like some of the bones in the foot wedge, combine with compressive force to provide foot strength.

Windlass Effect.

The British research physician Hicks **(Hicks, 1951, 1953, 1954, 1955)** studied the dynamic function of the foot longitudinal arches by dissection, by x-rays of movement at sequential steps of the dissection, and by x-rays of the living foot. He concluded that the normal foot arch is maintained by the plantar aponeurosis, with no contribution from the intrinsic or extrinsic muscles. The operative words here are "normal foot." There must be normal activity of the intrinsic and extrinsic muscles during running and walking. The mechanism that Hicks describes elevates the arch by tightening the aponeurosis with extension of the toes. The influence of toe extension on the plantar aponeurosis results from the attachment of the aponeurosis to the proximal phalanges. As the toe extends it pulls with it the plantar pad, which is an extension of the aponeurosis. The plantar pad, sliding around the metatarsal head, pulls on the plantar aponeurosis and tightens it as if it were a cable arrangement being pulled around the drum of a windlass. Maximum toe extension shortens the effective length of the aponeurosis by approximately 1 cm. This mechanism exists in all five

toes, but it is most marked in the hallux. When the hallux is amputated, its role in the windlass is lost; the forefoot weight bearing shifts laterally throughout gait, and the height of the medial longitudinal arch diminishes. **(Mann et al., 1988)**

The mechanism Hicks describes can be observed in the normally functioning subject. With the person standing in a neutral position, the arch will rise and the tibia will externally rotate. Failure of this action may be due to abnormal alignment of the first ray. **(Rose, 1982)** As described later, there is failure of the hallux to extend properly in this test when FHL is present.

Sequence of Motion. Upon heel strike the subtalar articulation rotates into pronation for shock absorption and to accommodate internal leg rotation. As the heel unloads, weight is transferred through the foot with supination to accommodate external limb rotation. The plantar aponeurosis tightens, causing the calcaneus and cuboid to align in such a manner as to "lock" the foot and prevent arch collapse. The weight continues to move toward the toes and transfers to the second and first rays. Dananberg **(Dananberg, 1994)** calls the body weight flow an automatic natural arch support, noting the importance of its efficiency because forces can be two to three times body weight. As forward motion continues, the weight of the body moves over the metatarsophalangeal articulation. This extends and tenses the aponeurosis to bring the calcaneus closer to the toes, supporting the medial longitudinal arch by the windlass action. The windlass action also rotates the posterior part of the foot externally (supinates) to synchronize with the tibia external rotation. **(Hicks, 1954)** Strands of the aponeurosis attach to the skin of the ball of the foot, which tightens the tissue as the metatarsal head rotates within it to prevent tissue trauma.

As the body moves over the stance limb all of the forward movement is centered on the ball of the foot, with the most important weight bearing being carried on the firmly planted 1st metatarsophalangeal articulation. The metatarsals and midfoot become more vertical, and the body weight is carried more by compression of a column than by the arch.

The Effects of Functional Hallux Limitus

Failure of hallux extension when the body is being pulled over the 1st metatarsophalangeal articulation during gait causes the kinetic energy to be dissipated in compensations that usually stress the weakest links. In the foot and ankle there are five alterations in movement that may be combined or individually present to adapt to functional hallux limitus: (1) altered heel lift, (2) vertical toe-off, (3) inverted step, (4) abducted toe-off, and (5) adducted toe-off. **(Dananberg, 1993)**

Heel lift occurs as the body weight moves forward over the metatarsophalangeal articulation. If there is failure of hallux extension the adjacent midfoot joints become obliged to move, giving the appearance of excessive pronation. Over a prolonged period the bones will remodel according to Wolff's law, **(Wolff, 1986)** causing greater instability and extended pronation. Since the average person takes 5,000 steps per day, or 2,500 steps per foot, this subtle imbalance is repeated thousands of times per day. When the heel cannot lift because of hallux extension failure, the next form of compensation — vertical toe-off — develops. Direct lifting of the toe from the substrate eliminates the thrusting forward of normal forward motion often seen as the slow, shuffling gait of the elderly.

The last three variations due to functional hallux limitus are avoidance compensations. Normally weight is transferred through the foot from the heel along the lateral longitudinal arch, across the metatarsals to the hallux. In the presence of hallux limitus there is prolonged weight bearing along the lateral longitudinal arch, with failure to move over to the hallux because of its inability to properly extend. The aponeurosis is never tightened by Hicks' mechanism. With heel lift there is a rapid stretching of the aponeurosis that has not been properly tightened. This results in trauma to the aponeurosis attachment at the calcaneus that may cause inflammation and ultimately a heel spur with the consequent pain. Typically there will be excessive lateral wear on the forefoot of the shoe. In this case functional pathologies within the foot and elsewhere are not due to the ineffectiveness of the windlass mechanism, but the functional inability of the great toe to extend, producing another reason for plantar fasciitis and heel spurs to develop.

It must be emphasized that pain may be located almost anywhere in the body, and there may be no foot complaint. **(Dananberg, 2007)** Remote pain develops as a result of compensation that takes place to dissipate the kinetic force that is not properly transmitted through the foot and leg for forward propulsion. When the forces of gait are dissipated abnormally in the body, there is recurring remote strain with each step, creating thousands of microtraumas at the area each day. The body responds to the trauma by inflammatory reaction, swelling, and stiffness. The resulting chronic inflammation is the hallmark of degenerative joint disease of old age, especially when it is combined with systemic problems that cause failure of tissue regeneration. **(Hurley & Newman, 1993; Dananberg et al., 1990)**

The following table lists the normal motion that occurs during the second half of single support during gait and the compensations when functional hallux limitus is present. **(Dananberg, 1994)**

Symptoms

Functional hallux limitus has been overlooked for several reasons. Symptoms are rarely in the foot until well advanced; they are then attributed to the extended pronation, not to its original cause. In many cases gait may appear normal on visual observation, yet objective recording by the Electrodynogram™ clearly shows the disturbance. Finally, the hallux range of motion in the non-weight-bearing foot examination is completely normal.

Symptom possibilities are as follows: lower leg, knee, thigh, sciatic-type pain, lower back, neck, TMJ, **(Dananberg, 2007; 1994; 1988)** and chronic headache among others. Many other corrections may not remain intact as a result of this remote imbalance. Treatment to the areas of pain and dysfunction may help the symptoms; however, if the primary cause of gait dysfunction is not corrected, symptomatic relief is not long-lasting or may manifest in some other area of the body…a consistent

Joint	Normal Motion	Compensatory Motion
Mid-tarsal joint arch	Supination	Pronation
Ankle	Plantar flexion	Dorsiflexion
Knee	Extension	Flexion
Hip	Extension	Flexion
Lumbar spine	Lordosis	Lumbar flexion
Cervical spine	Lordosis	Cervical flexion

Compensatory motion that may take place during the second half of single support due to FHL.

finding in applied kinesiology, reiterated throughout the applied kinesiology literature. The ability to specifically test the muscles in a kinetic chain commonly involved in FHL allows the AK practitioner to explore the many ramifications of this condition with greater ease and specificity than before.

Visualizing the weakness or imbalance in the hamstrings, or psoas, or contralateral shoulder or cervical flexor muscles for example is quite challenging. **(Goodheart, 1996; Dananberg, 1986)** Visualizing whether the movement pattern anomalies resulting from the FHL are due to inhibition or over-facilitation of these muscles' antagonists is impossible without the MMT. Furthermore is there an associated joint subluxation or fixation specifically relating to these muscular imbalances related to the FHL in the body? The AK method of challenge and/or therapy localization can detect this related factor as well.

By broadening the examination process to include the entire kinetic chain resulting from FHL, the AK practitioner has a tool that permits the discovery of the many consequences of FHL that a local focus on the 1st metatarsophalangeal disorder would limit.

Examination

In the non-weight-bearing foot there should be full hallux range of motion. The condition is found with the patient weight bearing. With the patient standing in a neutral position, i.e., with the feet in the normal position of gait, the hallux is passively lifted into extension by the physician. Normally the medial arch will rise and the tibia will externally rotate. **(Kitaoka et al., 1995)** An easy way to test for this is to have the patient stand on a platform, such as a 1"-thick board, with the ball of the foot close to the edge so the toes hang over. The patient stabilizes himself by holding on to the physician's shoulder or something else and stands only on the foot being examined. The physician attempts to lift the hallux into extension; failure of passive extension when weight bearing is a positive test.

Weight bearing can be simulated with the patient supine. With one thumb push up on the ball of the foot directly under the 1st metatarsal head until some resistance is felt. With the other thumb, attempt to extend the hallux while maintaining the constant upward pressure on the 1st metatarsal head. Resistance of the hallux to move into extension indicates the standing test will probably be positive.

The extensor hallucis longus and brevis muscles will test weak in the presence of flexor hallux limitus. This weakness causes secondary contraction of the flexor hallucis longus and brevis muscles. The contracted muscle is secondary to the weak antagonist muscle, a familiar finding in applied kinesiology. Applied kinesiology treatment for FHL is directed to returning the weak extensor hallucis muscle(s) to normal.

Podiatry

There are several modes of treatment by podiatrists that specialize in gait analysis and the foot's relation to remote body function. Treatment may consist of orthotics, various pads and strapping, manipulation, and shoe modification. **(Dananberg et al., 1996)** Dananberg describes the Kinetic Wedge® orthotics **(Langer Biomechanics Group)** for correction of FHL. The Kinetic Wedge® is also built into athletic shoes. **(Dananberg, 1988)**

Dananberg and Guiliano **(Dananberg & Guiliano, 1999)** have demonstrated that 84% of patients with chronic lower back pain, who have been prescribed the orthotics described above, significantly improved. Another study of podiatric treatment of functional hallux limitus resulted in 46% of the patients reporting they were 75-100% better and 35% reporting 50-75% better, making a total of 77% to be at least 50% better. **(Dananberg et al., 1990)** The most interesting aspect of this study is that none of the patients complained of foot pain or discomfort. The primary symptoms were all remote from the feet.

Dananberg **(Dananberg, 2001)** also shows the effect of podiatric treatment of FHL upon hip extension range of motion. He retrospectively reviewed hip joint range of motion on 20 subjects walking before and after intervention with custom foot orthotics. He demonstrated a 50% increase in hip joint extension by the conclusion of single support phase during gaiting as a result of the functional changes brought about by the orthotics.

Applied Kinesiology and FHL

Examination and treatment of functional hallux limitus in applied kinesiology is directed to finding the cause and making lasting correction of extensor hallucis muscle weakness. Disturbances in muscle function due

to subluxation or fixations of the foot or ankle, myofascial trigger points, low back or pelvic joint dysfunctions producing muscle imbalances that alter the gait cycle are not sufficiently treated with the use of orthotics alone. Supporting the 1st MTP joint when it is not required (because its cause is elsewhere) is also treating the symptom not the cause. A major factor in all applied kinesiology corrections is that the weakness in the extensor hallucis muscle does not return after walking. Test the extensor hallucis with the ankle in dorsiflexion and the hallux in extension. Apply the testing force to flex the hallux only at the metatarsophalangeal articulation. The weakness may be uni- or bilateral. The extensor digitorum muscles are also tested and may or may not be weak. The weak muscles are treated with the five factors of the IVF involved with the weakness. All foot subluxations should be corrected before examining for FHL. Correcting foot subluxations and the muscles will not usually correct FHL; it is a separate entity. Any or most of the following may need attention for lasting correction.

Liver meridian. The liver meridian begins at the lateral nail point of the hallux. LV 3 is where the 1st and 2nd proximal metatarsal bones join, just lateral to the extensor hallucis tendon. Mann **(Mann et al., 1992)** places a high degree of importance on the LV 3 area. He discusses it as an area rather than an acupuncture point. Goodheart **(Goodheart, 1998)** noted that activity at LV 3 is often present in FHL. Often a weak extensor hallucis temporarily strengthens with therapy localization to the liver alarm point (LV 14); when it does, stimulation of LV 3 strengthens the muscle. Stimulation can be done by any standard method of acu-point stimulation, e.g., teishein, fingertip tapping, laser, needle, electrical, and others. When experience indicates that FHL is present but the extensor hallucis is not weak, it will often show a subclinical weakness with therapy localization to the liver alarm point. When the extensor hallucis is strengthened by stimulation of LV 3, the improved function will often be lost after walking if other corrections are not made.

Origin and insertion technique. Therapy localize the origin and insertion of the extensor hallucis; if it strengthens, apply the hard digital pressure used in that technique. Most often the involvement will be at the origin along the middle one-half of the medial aspect of the fibula.

Repeated muscle activity – patient induced. If the extensor muscles weaken after the patient actively extends the toes ten times, origin and insertion technique is needed along with nutritional support with water for additional hydration and wheat germ oil three times a day.

Muscle stretch reaction. If the extensor muscles weaken following stretching, apply fascial release, trigger point, or myofascial gelosis technique as indicated by examination. The most common need is for fascial release. Vitamin B_{12} may be necessary for lasting correction.

Maximum muscle contraction. If the extensor muscles weaken following maximum contraction, apply strain/counterstrain technique. Chaitow has presented Goodheart's techniques for strain/counterstrain approvingly in a number of his books. **(Chaitow, 2008, 2005, 2002, 1988)** The tender point will usually be at a more proximal area of the muscle. A collagen source of glycine may be needed for lasting correction.

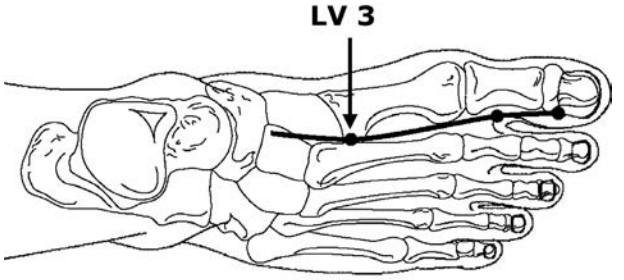
Rib pump technique. Determine the need for rib pump technique by therapy localizing in the 4th and 5th anterior rib area. Strain/counterstrain technique often needs to be applied in this condition to ribs 4 and 5, both anteriorly and posteriorly, for adequate rib pump activity.

Deep peroneal nerve. The deep peroneal nerve may be entrapped by distal tibia and fibula spreading, much like nerve entrapment at the carpal tunnel by the spreading of the distal radius and ulna. Test for extensor hallucis strengthening by having the patient approximate the proximal one-third of the tibia and fibula by holding them together. The approximation could also be done at the distal ends, but most patients cannot reach that far down. The distal tibia is adjusted toward the fibula with the patient side-lying and the physician using a high velocity thrust.

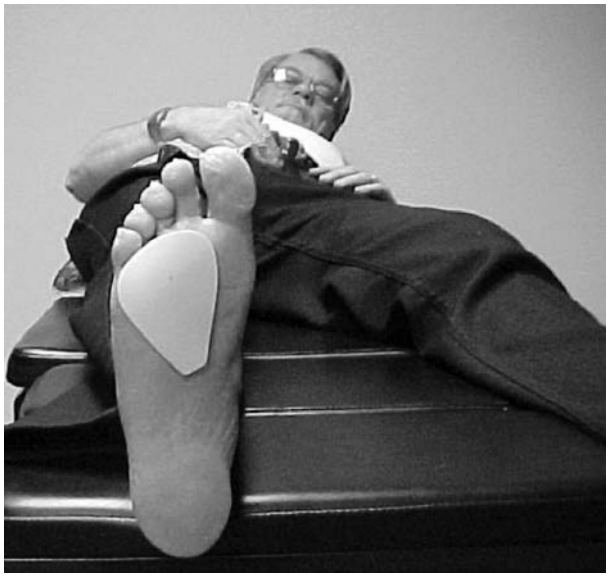
Support. Most often the patient will lose the corrections immediately upon walking. If the corrections are lost with walking, support is added to give the corrections an opportunity to stabilize.

The correction at the tibia and fibula is supported by taping with porous adhesive elastic tape, such as Elastikon by Johnson and Johnson. Use two layers, taking care not to apply too much tension to the elastic and impede circulation. The patient should wear the support over the distal tibia and fibula for one week. He can bathe or shower without taking the tape off because it dries rapidly and will not loosen. Patients are not usually allergic to this type of tape. If there is a problem with allergy, prevent the adhesive dermatitis by placing the first circular wrap with the sticky side out and then apply two more wraps over the first one in the usual manner.

Finally the podiatric approach involves a triangular adhesive felt or foam pad placed under the 2nd-5th metatarsal heads. The felt pad provides a more solid support than the foam pad, but the foam is tolerated better than felt regarding comfort and usually provides adequate support. The pads are available in 1/8" or 1/4" thickness. They are 2" wide at the point under the metatarsal heads. The lateral border of the pad extends along the 5th metatarsal as the pad narrows to about 1/2" width at the end pointing toward the calcaneus. Most patients can easily accommodate the pad in their regular shoes. The metatarsal foam pad survives bathing or showering better than the



Liver 3 *(With kind permission, ICAK-USA)*



Correct placement of metatarsal pad with FHL

felt one. Neither lasts as well as the adhesive tape wrap. It is advisable to give the patient several pads to take home with instructions for application. Caution the patient to not walk without the pad(s). If it is removed for bathing it should be replaced before walking. The pad(s) can be left in place when showering and then replaced before dressing. The triangular pads are available from podiatric suppliers.

The immediate prescription of an orthotics or metatarsal pad should not be the first line of therapy for FHL. The benefits of these devices is well documented, however the need for other therapies for causative factors in the FHL syndrome may be neglected.

Hallux Rigidus

Hallux rigidus is the most common degenerative arthritis of the foot affecting most commonly people between 30 to 60 years of age, but it is a common disorder in young athletes as well. It is a premature arthritis of the first metatarsophalangeal (MTP) articulation, and the disability resulting from hallux rigidus is greater than that seen with hallux valgus. **(Zammit et al., 2009)**

Hallux rigidus is commonly preceded by some form of physical trauma to the first MTP joint; the patient may or may not remember the incident. Manral **(Manral, 2004)** lists the causative factors: physical trauma is primary, followed by systemic conditions affecting the first MTP. Secondary etiological factors include pronation of the foot, improper shoes (with a narrow toe box), interposed sesamoid bones, age related arthritis, disorders of the proximal parts of the lower limb, gait abnormality due to any cause, obesity, and occupational ergonomic factors. Anatomically it involves the periosteum, joint capsule, synovium and subchondral bone. Occasionally the MTP joint of the smaller toes is affected as well; in that case it is called Freiberg's disease. **(Turek, 1984)** Besides the visual inspection of the first MTP joint (arthritic inflammation and swelling are not always present in FHL), an important diagnostic factor distinguishing hallux rigidus from FHL is that there is decreased range of motion of the first MTP joint weight-bearing ***as well as*** non-weight-bearing. In FHL, the hallux can still extend when passively examined or when the patient is non-weight-bearing, except during a very brief portion of stance phase of gait that may be only 100 milliseconds in duration. **(Dananberg, 2007)** In hallux rigidus, X-ray shows joint degeneration, dorsal osteophyte formation, and MTP joint narrowing. **(Zammit et al., 2009)** Passive extension of the great toe is also painful with swelling and tenderness present, and the toe held in slight flexion.

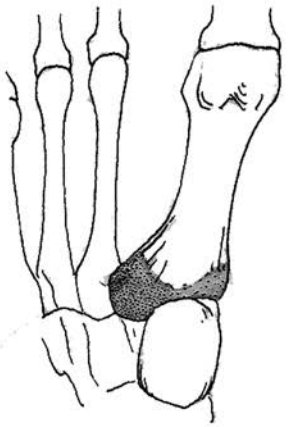
Because the great toe of the patient with hallux rigidus has limited dorsiflexion, push-off during gait can be painful. Cheilectomy (the chiseling away of bony irregularities on the lips of a joint cavity) is commonly performed in which not only the dorsal spur but also the dorsal third of the metatarsal head is removed. **(Mulier et al., 1999)** Mulier et al claim that this provides long-term pain relief in most patients. If this fails, arthrodesis (surgical fusion of the joint) is suggested. Surgeons suggest that mobilization of the toe should be initiated soon after surgery, emphasizing the functional importance of movement therapy in the treatment of disorders like this one. The widespread negative influences of FHL (see section above) also suggest that the musculoskeletal impairments resulting from hallux rigidus may also benefit from the functional improvements delivered for the first MTP, via conservative treatment or other means. **(Dananberg, 2007; Manral, 2004)** Conservative treatment is worth a clinical trial in the early stages of this disorder. **(Smith et al., 2000)** Smith et al. reviewed 24 cases with hallux rigidus, and showed that in 75% cases, the patients would "still choose not to have surgery" if they had to make the decision again.

Hallux Valgus

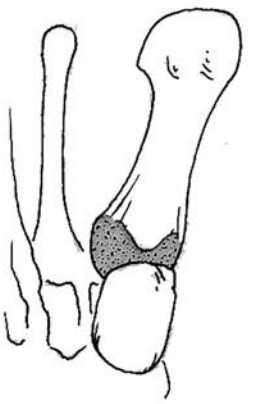
Hallux valgus is the lateral deviation of the great toe with secondary medial deviation of the 1st metatarsal. The condition modifies foot function so that the weight load of the forefoot shifts toward the lateral side of the foot. The 2nd and other metatarsals begin to take more of the load, and metatarsalgia develops. **(Nix et al., 2010; Mann, 1983)** The bursa located on the medial aspect of the first metatarsal head may become inflamed (usually due to rubbing on the shoe), resulting in the formation of a bunion.

Nix et al **(Nix et al., 2010)** conducted a meta-analysis of the literature involving a total of 78 papers reporting results of 76 surveys (total 496,957 participants). Prevalence estimates for hallux valgus were 23% in adults aged 18-65 years, and 35.7% in elderly people aged over 65 years. Prevalence increased with age and was higher in females (30%) compared to males (13%).

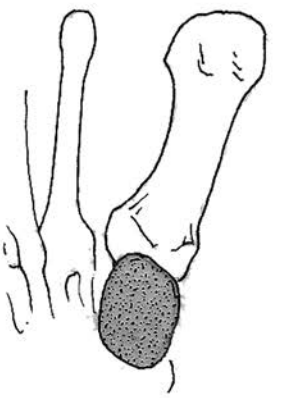
The terms "hallux valgus" and "bunion" are often used interchangeably in the literature. Here, reference to hallux valgus means the disrelation or subluxation of the articulation(s). Characteristic features are the bony overgrowth on the medial distal head of the 1st metatarsal, and the lateral deviation of the great toe at the metatarsophalangeal articulation (rarely the interphalangeal articulation). General agreement is that hallux valgus is present when there is 8-10° of lateral



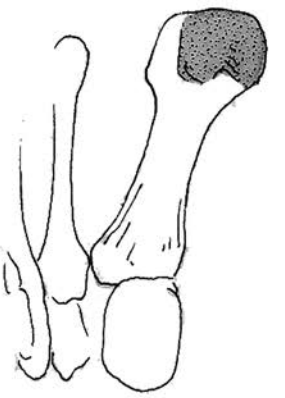
Enlarged 1st metatarsal base.



Angled 1st metarsal base.



Angled 1st cuneiform.



Enlarged 1st metatarsal head.

deviation at the metatarsophalangeal articulation. **(Chhaya et al., 2008; Lidge, 1976)**

The term "bunion" is not as specific. It probably is derived from the irregular French word "bunny," which means swelling. **(Webster's Third New International Dictionary of the English Language, 2002)** Langer, Maffetone, and Kelikian **(Langer, 2007; Maffetone, 2003; Kelikian, 1982)** refer to hallux valgus as a complex. The lateral deviation of the great toe is only occasionally an isolated entity. Often there are also deformities of the lesser toes, and the metatarsal bones are distorted from their normal positions.

Etiology. Throughout the literature on hallux valgus there is strong opinion that the condition's etiology relates to (1) congenital anomaly or function of the bones, (2) muscle anomaly or improper function, and (3) improper footwear. Although not mentioned as often, Nix and Mann **(Nix et al., 2010; Mann, 1983)** emphasize extended foot pronation as a cause of hallux valgus; this puts an increased force on the medial side of the foot near the time of toe-off. Goodheart, Bandy, Leaf, and Lee et al **(Leaf, 2006; Lee et al., 2003; Bandy, 1976; Goodheart, 1998-1964)** recognized these etiologies as applicable to applied kinesiology's therapeutic approach. Conditions such as rheumatoid arthritis must also be taken into consideration as an etiology of hallux valgus. Any condition that causes progressive loss of integrity of the capsular structure supporting the metatarsophalangeal joints, thus providing less lateral support by the lesser toes, must be systemically treated. There is debate as to whether the lateral deviation of the great toe or the medial deflection of the 1st metatarsal is the primary deformity in the hallux valgus complex. **(Mann, 1982)**

Although many authors present convincing evidence that their favorite etiology is the primary cause of hallux valgus, it is obvious that each one — and perhaps a combination — is primary in individual cases. It becomes necessary, then, to analyze each case to determine if surgery, conservative treatment, or palliative care is the optimal approach.

Congenital bone formation. X-ray examination can determine if there are congenital malformations that predispose an individual to hallux valgus. Malformation between the 1st cuneiform and 1st metatarsal can cause the metatarsal to angle medially, causing a secondary hallux valgus. There may be wedging of the 1st cuneiform, or obliquity of the base of the metatarsal so that the latter articulates in a varus position. **(Hammer, 1999; Turek, 1984)** There may also be a large lateral facet or exostosis at the base of the 1st metatarsal that holds it away from the 2nd, causing medial angulation. **(Mann, 1983)**

Footwear. The distortion of feet caused by restriction is classically evident in the no longer practiced tradition of binding Chinese women's feet. **(Chen, 1992)** Any footwear that places lateral pressure on the hallux can be a potential cause of hallux valgus. Two classifications are particularly indicted as causing hallux valgus. Children's shoes are often poorly fitted or not changed often enough during rapid growth periods. Hicks **(Hicks, 1965)** states, "We realize that the onset of hallux valgus and other foot deformities is slow and insidious. Mistakes made in shoe selection when young become painfully apparent only in middle age." Regarding extensive research on the hallux angle in children he states, "Our results show that distortion of the big toe starts at the age of 7 years."

The other major problem is the high-heeled, pointed-toe shoes worn by women. Not only do the pointed toes cause angulation of the hallux, the high heels cause gravity to force the foot into the narrow toe portion of the shoe. Unfortunately, shoes are often selected for style rather than function.

There is conflicting evidence indicating that hallux valgus is caused by wearing shoes. Supporting the shoe-wearing etiology is a 33% incidence of hallux valgus in a shoe-wearing Chinese population versus 1.9% in those who do not wear shoes. In addition, the deformity is nine times more prevalent in females than in males. **(Sim-Fook & Hodgson, 1958)**

Another study was made of most of the population of a small island centered in the south Atlantic Ocean. **(Shine, 1965)** It revealed significant difference between the shoe-wearing and unshod populations. In this study of 3,515 people, hallux valgus of 15 degrees or greater was present in fewer than 2% of those barefoot, and in 16% of the men and 48% of the women who had worn shoes for

more than 60 years. There was no significant correlation with age, social class, occupation, or exercise habits. The higher incidence of hallux valgus among women appears to be female-related rather than shoe-related, since both sexes in this population wear similar shoes.

Some consider the higher incidence in women a result of the shoes worn by females, **(Hong et al., 2005; Mann, 1983)** while others believe it is due to females having weaker ligamentous structure. Sports podiatrists have observed that women runners complain of a widening of the foot and a "knot" forming. Those who work with these athletes indicate that it is due to weaker ligaments in females. **(Nguyen et al., 2010; Hlavac, 1977)**

When the 1st metatarsal abducts in spread foot, there is propensity for secondary hallux valgus to develop. In a high-heeled shoe, the metatarsophalangeal articulation is extended throughout gait. The sesamoid bones are thus anteriorly displaced on the 1st metatarsal head at heelstrike. The ridge between the sesamoid bones ordinarily stabilizes them in their distal and proximal movements, but the height of the ridge as it progresses distally is less, and it fails to stabilize the bones adequately. This loss of bony guidance causes resistance to the 1st metatarsal abduction to depend entirely on the ligament of the medial sesamoid bone. At the moment of and immediately after heelstrike the tibialis anterior eccentrically contracts to prevent the forefoot from slapping on the ground. **(Winter & Scott, 1991)** This action is exerted on the 1st metatarsal, which is inadequately stabilized by the usual mechanism. The tibialis anterior in this way contributes to 1st metatarsal abduction in a spread foot. **(Haines, 1947)**

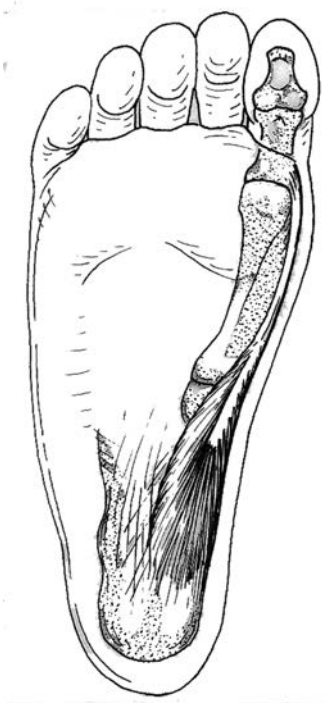
Although evidence points toward improper shoes as a contributing cause of hallux valgus, it certainly is not the only one. Kaplan **(Kaplan, 1955)** considers that shoes are not a major cause of hallux valgus. The condition was observed in World War I and II military personnel who came from a farm background in which shoes were infrequently worn. Some central African tribes and Australian aborigines who never wore shoes developed hallux valgus. This brings us to the primary role that muscles play in the condition.

Muscle role. Two considerations must be made regarding the role muscles play in hallux valgus. First is the anatomical arrangement of the muscles, a factor with which conservative treatment cannot cope. Second is the function of the muscles, as readily determined by applied kinesiology examination methods.

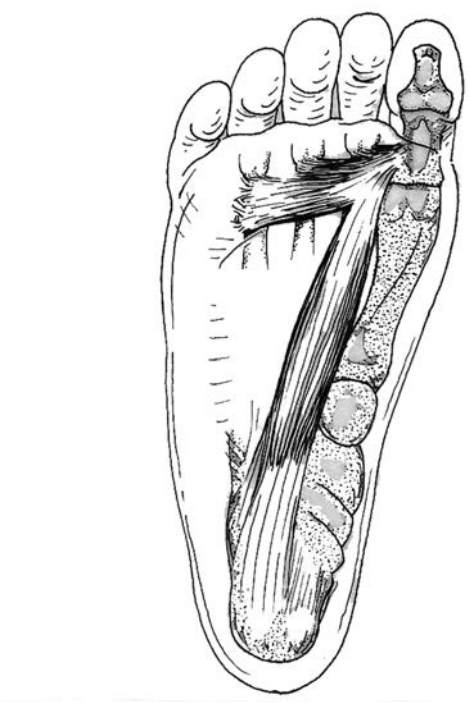
There is considerable congenital variation of the foot muscles. **(Bejjani & Jahss, 1986)** Extensive study has been done by dissection and electromyography of the muscles' action, both in normal and congenital variations. Possibly the reason for the variations is the bipedal stance unique to man. Thomson **(Thomson, 1960)** considers the abductor and adductor hallucis muscles as two of the most important primitive muscles not necessary to the human foot. Since the major motion of the great toe in the human is flexion and extension, he believes that "…these two muscles lose their functions or their insertions had drifted to aid in the normal plane of movement." Of specific interest to us is the action of the abductor hallucis.

Lovejoy **(Lovejoy, 2007)** offers a list of distinct lower limb morphological characteristics relating to the structural and functional anatomical adaptations in the lower leg and feet. These adaptations reflect bipedal gait in early hominids (including *Australopithecus*):

- High anterolateral lip of the lateral femoral condyle providing patellar retention with the fully extended knee
- Transverse distal tibial plafond
- Wedged talar dome
- Longitudinal and transverse pedal arches
- Permanently adducted first ray



Abductor hallucis.



Adductor hallucis.

- Vertical groove for the tibialis anterior
- Distinct dorsiflexion profile in metatarsophalangeal joints for all five rays
- Markedly expanded calcaneal tuber for energy dissipation at heel strike

Thomson **(Thomson, 1960)** discusses an unpublished study by Basmajian and Kerr that is mentioned only briefly in Basmajian and Deluca's text. **(Basmajian & DeLuca, 1985)** Twenty-two adult feet were dissected to investigate the insertion of the abductor hallucis muscle. "In only 5% of the specimens did the tendon lie on the medial border of the foot, and insert into the medial side of the base of the proximal phalanx as an obvious abductor." **(Thomson, 1960)** In 19% of the specimens, the abductor hallucis joined with the medial head of the flexor hallucis brevis, inserting into the base of the medial sesamoid to be an obvious flexor. Between these two extremes, in 19% of the cases the abductor tendon took a plantar position, passing over the medial portion of the sesamoid without attaching to it, finally attaching to the base of the proximal phalanx. In 25% of the cases, the lateral slip of tendon inserted into the medial sesamoid before its insertion on the phalanx. In another 25%, a common slip of the insertion was to the medial head of the short flexor into the sesamoid. In only 19% of the cases was the abductor hallucis anatomically located as a true abductor. In 83%, the adductor hallucis acted to flex the great toe at the metatarsophalangeal articulation. Another study **(Agawany & Meguid, 2010)** investigates the insertion of the abductor hallucis muscle and also shows the variations in the insertion of this muscle.

This makes the role of the abductor hallucis muscle in the prevention of hallux valgus questionable from what one would think after studying the standard anatomy texts, which clearly show the insertion into the medial 1st phalanx base.

Iida and Basmajian **(Iida & Basmajian, 1974)** studied the balance between the adductor and abductor muscles in "idiopathic" hallux valgus and normal feet with electromyography. In hallux valgus, the adductor hallucis muscle becomes markedly weak with time, but the abductor hallucis muscle loses its abduction function completely and works only as a flexor. Because of mechanical misalignment, the flexor hallucis brevis tendon stretches so that the muscle loses some of its function as a flexor. The stresses of weight bearing with time cause the muscle imbalance to become worse. In this study, they could not conclude whether hallux valgus is caused from muscular imbalance, or the muscular imbalance develops secondarily to the structural imbalance. In any event, one must not count on the abductor hallucis' participation in the correction of hallux valgus until one determines that it is actually an abductor.

The length and function of both heads of the adductor hallucis must be evaluated. Their shortness or hypertonicity can contribute to the deformity. It appears important to provide a therapeutic approach to return optimal balance to the intrinsic muscles.

Another congenital variation that may produce hallux valgus was demonstrated by Kaplan. **(Kaplan, 1955)** By dissection he found a consistent expansion of the tibialis posterior tendon into the flexor hallucis brevis and the oblique head of the adductor hallucis in those with hallux valgus. The same expansion of the tendon was not found in normal subjects. Pulling on the tendon caused an increase in the hallux valgus deformity. There was no influence on normal subjects' toes when the tendon was pulled.

Kaplan also demonstrated an increase in the hallux valgus deformity by stimulating with faradic current the tibialis posterior in living subjects. On the other hand, there was no visible movement of the big toe in normal subjects with the stimulation.

If spread foot and hallux valgus begin to develop, muscle action can be responsible for increasing the problem even when there was originally proper muscle attachment to the bones. **(Arinci et al., 2003)** Action of the flexor hallucis longus increases the valgus position of the hallux and the varus position of the 1st metatarsal when hallux valgus is present. **(Snidjers & Philippens, 1986)** For this reason, lateral pressures on the hallux during childhood — from shoes or socks that may start the process that will be continued by the muscle activity — should be avoided. This supports the finding of Hicks **(Hicks, 1965)** "… that if a woman can reach the age of 20 years with a big toe angle of less than 10, she is unlikely to develop bunions in old age."

An unusual cause of hallux valgus is, ironically, surgery to correct hallux varus, due to the change in insertion of the abductor hallucis. **(Thomson, 1960)**

Muscles may play a further role in hallux valgus by developing histologic abnormalities. This appears to develop from "…chronic ischemia caused by the elevated pressure occurring within the foot during gait." **(Hoffmeyer, 1988)** Fifty-three of 57 patients with hallux valgus had histologically abnormal biopsies of muscle taken at the time of surgery for correction of the deformity. There were myogenic and neurogenic alterations, as well as ultrastructural changes. Gait evaluation revealed that this group, in comparison with a control group, had 70% abnormal rollover pattern; in the control group, only 20% failed to have the usual pattern (heelstrike, 5th, and then 1st metatarsal head contact, ending with great toe push-off). Finally, the muscle activity, as indicated by surface EMG, was abnormal in the patient group when compared with the volunteer group. The difference in distribution of the electrical activity patterns was statistically significant ($p<0.0005$).

Histological abnormalities are common for injured muscles. Proprioceptive acuity depends upon intact mechanoreceptors and their peripheral to central pathways. **(Bard et al., 1992)** However in injuries the damage is to the proprioceptive apparatus and mechanoreceptors themselves. Later this may be accompanied by adaptive central re-organization throughout the motor cortex.

Pronation. Eversion of the foot at toe-off in extended pronation is an important contributor to hallux valgus. The examination and therapeutic approach for extended pronation, if present, should be routine in all cases of hallux valgus.

Examination and treatment. Many factors must be taken into account before deciding to attempt conservative management and treatment of hallux valgus. Limiting effective conservative care may be an advanced stage of degenerative development, congenital anomalies such as

wedging of the 1st cuneiform, functional hallux limitus, hallux rigidus, and malformation of the base of the 1st metatarsal. **(Chhaya et al., 2008; Maffetone, 2003; Cholmeley, 1958)** Occasionally conservative management is necessitated because the patient is a poor candidate for surgery, such as when there is a circulatory deficit. **(Veves et al., 2002; Kelikian, 1982)** If circulation is a problem, as in diabetes or patients on blood-thinning medications, the case becomes much more critical. One must carefully guard against pressure necrosis and infection. **(Veves et al., 2002; Aronow & Solomone-Aronow, 1986)** Further complicating the case is an insensitive foot. Finally, before deciding on conservative care, factors must be found that contribute to or cause the hallux valgus.

Although hallux valgus may appear to be a localized problem, total body function must be analyzed. Disturbance in gait function, which may relate with pelvic and spinal organization, knee problems, and especially excessive pronation, must be evaluated and corrected. The examination and treatment of these conditions are dealt with elsewhere in these texts.

Local examination of the foot for hallux valgus should begin with x-ray to evaluate for congenital anomalies and general bone condition. Even if the x-ray indicates referral for surgery, intrinsic muscle evaluation and correction are advisable.

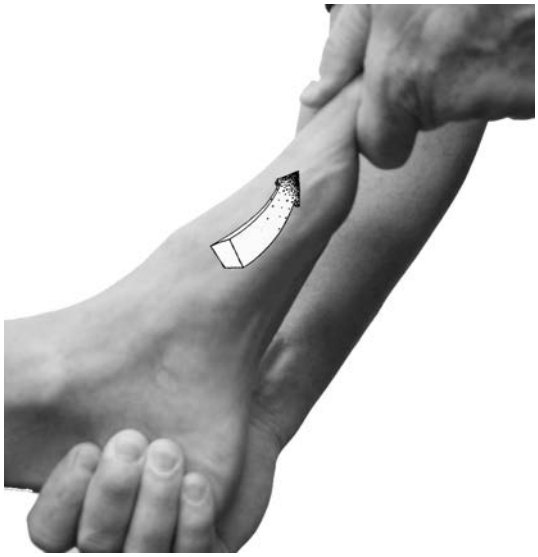
Determine abduction capability of the abductor hallucis by having the patient attempt to abduct the hallux. While the patient attempts the action, the physician should palpate the abductor hallucis muscle to determine if it is contracting. If there is simply flexion of the hallux with the muscle's contraction, one cannot count on correcting hallux valgus by improving the muscle's function.

Even in normal individuals, this is a difficult muscle to selectively contract because wearing shoes restricts the activity. In those with hallux valgus, the abductor hallucis is typically weak making isolated contraction even more difficult. The patient should learn this activity by abducting the hallux against his finger, spreading his toes, and sliding the forefoot toward the other foot while keeping the heel stationary. In addition, the hallux can be abducted passively to stretch the adductor hallucis. **(Chaitow & DeLany, 2002; Ramamurti, 1979)** With persistence, an individual will be able to make a small abduction movement if the tendon is inserted to perform the activity.

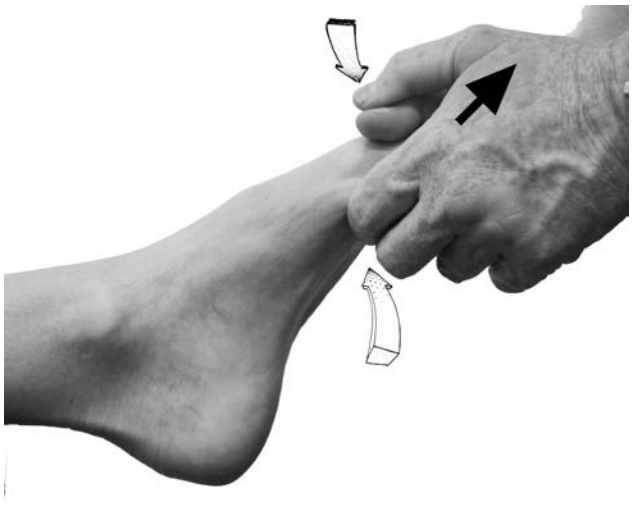
The distal tendons of the flexor hallucis brevis contain the sesamoid bones and insert into the medial and lateral sides of the proximal phalanx of the hallux. This muscle is often found weak on manual muscle testing. **(Leaf, 2003; Bandy, 1976)** To help align the sesamoids and supply some stability to the hallux, both the abductor hallucis and the flexor hallucis brevis should be strengthened with applied kinesiology techniques.

The intrinsic muscles of the foot often respond to origin and insertion technique. Treatment to the Golgi tendon organ or neuromuscular spindle cell may be necessary. The neurolymphatic reflexes are located bilaterally inferior to the symphysis pubis at the height of the obturator foramen. Posteriorly they are between the posterior superior iliac spine and L5 spinous process. The neurovascular reflexes are located bilaterally on the frontal bone eminences.

The adductor hallucis may be hypertonic. Both the oblique and transverse heads contribute to adduction of the hallux. Fascial release technique can be applied to help ensure normal function of these muscles. Manipulation of the 1st metatarsal and hallux, when needed, should be done after other foot corrections have been obtained.



First metatarsal and 1st cuneiform adjustment.



Separation between the 1st and 2nd toes and contact on the dorsal and plantar surface of the 1st toe proximal phalanx.

Severe arthritis or other joint disease contraindicates HVLA manipulation. **(Bergmann & Peterson, 2010; Chaitow & DeLany, 2002)** Mennell **(Mennell, 1969)** states that it is all right to manipulate a hallux valgus with early osteoarthritic change, but there will probably be failure if there is any dorsal lipping.

First metatarsal and 1st cuneiform adjustment.

The adjustment may be needed to correct a subluxation or fixation of the articulation. Applied kinesiology challenge will determine the necessary direction of correction. To adjust the articulation, the physician holds the supine patient's left calcaneus in his right hand, with the heel of his hand applying firm pressure around the distal calcaneus and cuboid for stabilization. His left hand grasps the 1st

metatarsal, with the fingers wrapping underneath and the thumb contacting the bone's dorsal base. Traction is applied with the thumb and fingers moving the metatarsal in the direction of optimal challenge. The base may need to be moved in any direction, but it usually requires a lateral thrust. Often the combination is separation of the base from the 1st cuneiform and lateral movement of the head. It is sometimes difficult to get a good contact on the metatarsal head because of the soreness of the individual's bunion. A towel for padding aids in avoiding pain, and also helps keep the doctor's hand from slipping on the extremity.

First metatarsophalangeal adjustment.

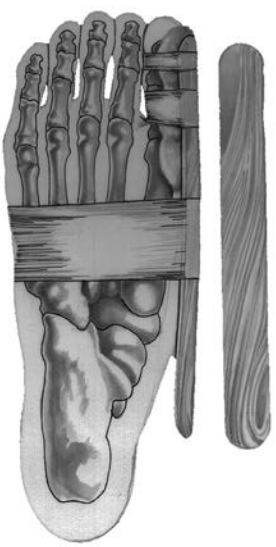
The metatarsophalangeal articulation of the hallux should be challenged and adjusted accordingly. Techniques typically emphasize reduction of the valgus position and traction. **(Hearon, 1994; Schafer, 1982; Bandy; 1976)** Traction of the phalanx is emphasized in the three techniques described by Gillet, **(Gillet & Liekens, 1981)** Bandy, **(Bandy, 1976)** and Schultz. **(Schultz, 1979)** One of these three techniques should meet the needs of the challenge.

Traction is the main factor in Gillet's technique. **(Gillet & Liekens, 1981)** It is applied to the phalanx to separate the articulation. It is necessary to obtain a good grip on the hallux, and the traction must be rapid and strong. There is rarely any pain. If the grasping fingers tend to slip, a better purchase can be obtained by wrapping the toe in a paper towel or facial tissue.

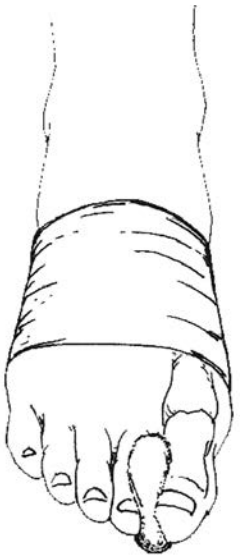
In Bandy's adjustment, **(Bandy, 1976)** the physician stands to the left of the supine patient for correction of the left hallux. Grasp the toe with the left hand and apply pressure in the direction of optimal challenge. The head of the 1st metatarsal is contacted with either the thumb or the softer web between the thumb and index finger. If the head of the 1st metatarsal is exquisitely tender, contact slightly proximal to it. The physician stabilizes the ankle and foot with his right thigh. With a quick traction of the toe, the 1st metatarsal is thrust laterally in a quick one-two motion.

Schultz **(Schultz, 1979)** recommends that care be exercised in the use of his adjustment for the hallux because the digit does not allow as much flexion as the other digits. Description is for the left foot. With the patient supine, the physician puts his right thumb between the 1st and 2nd toes so that the web between the patient's toes and that between the doctor's thumb and index finger approximate each other. The index finger of the doctor's left hand contacts under the hallux. Added support is accomplished by the right finger supporting the left finger, and the left thumb supporting the right. Apply axial traction to the toe to separate the metatarsophalangeal articulation. Without releasing the traction, quickly — by wrist action — bring the fingers up and the thumbs down. The doctor's thumb between the hallux and the 2nd toe brings the toe medially, which is usually the direction challenge indicates for correction. There should be minimal pain to the patient. Pain indicates that too much downward pressure is being applied with the thumbs. Remember, there is less flexion at this articulation.

In addition to the manipulation described above, it is often valuable to mobilize the hallux in all directions. For general stretching and mobilization of the foot, the patient will often relax better when lying supine, with his hip and knee flexed and his foot flat on the table. **(Maitland, 1977)**



Nighttime hallux valgus support.



Daytime hallux valgus support.

Support.

Following correction of the muscles and joints, support is added until maximum correction has been obtained. Ramamurti **(Ramamurti, 1979)** recommends that a night abduction splint be worn when passive abduction is limited to an angle short of the longitudinal axis of the foot.

A night splint can be simply made from a tongue depressor taped to the toe and foot; it is especially valuable in juvenile hallux valgus. **(Cailliet, 1997)** The tongue blade is applied to the medial foot, with cotton padding as needed. Daytime taping can be applied with spacers, which are available from suppliers or can be made by building pads out of 2" x 2" sponges or cotton. **(Krissoff & Ferris, 1979)** Acute pain from a bunion is due to acute bursitis, which should be appropriately treated. **(Mennell, 1969)** When bursitis is present, there is an inflammation of the lubricating membrane.

There are four classic signs of inflammation: pain, heat, redness, and swelling. The basic reason inflammatory processes develop is injury to the tissue. When the bursa is inflamed it cannot perform its primary lubricating function properly. This is particularly important because the bursa around the bunion is a point of high stress and wear.

Maffetone **(Maffetone, 2003)** has described the "biochemical foot factors" which should be considered and tested for using applied kinesiology methods in order to control inflammation in patients with foot pain and disorders. Schmitt and McCord, in their applied kinesiology approach called *Quintessential Applications*: *A(K) Clinical Protocol* **(Schmitt & McCord, 2010)** has provided excellent methods for evaluating and conservatively treating excessive inflammation.

In applied kinesiology the key factor when bursitis occurs is not removal of the symptoms of pain and limited function of the area; rather, it is finding the exact cause of the bursitis and eliminating it. Unfortunately, symptomatic treatment for bursitis in the form of painkillers and anti-inflammatory drugs is very common.

Bursitis very often parallels the different forms of

arthritis and, indeed, is caused by some of the same factors that cause arthritis. Protein and calcium metabolism are very important in the development of certain types of arthritis, as they are in the development of bursitis. Uric acid metabolism is important in gouty arthritis; it can also be the cause of bursitis.

A common cause of bursitis is excessive structural strain. The bursa becomes inflamed as a result of excessive wear in an area that already has excessive use, misuse or abuse. This excessive stress often occurs in the feet because of imbalanced muscular pull. If muscular balance around the forefoot is not present it must be regained or, regardless of the treatment to the bursa itself, the condition will remain and probably flare up again after the medication has worn off.

Bursitis may develop because the membranes comprising the bursa have an inadequate nutritional level; thus tissue is not as strong as it should be. It is very important for the bursal membranes to be healthy because, by the very nature of the membranes, they are subjected to significant stress. A lowered health level results in breakdown during periods of wear. Sometimes an individual will work extra hard in the garden, at sports, or at some other physical activity, and develop bursitis. The physical activity gets blamed for the bursitis; actually, the membranes — in a lowered state of health — were just waiting for extra stress to begin manifesting symptoms. If, upon examination, an inadequate protein level or some other factor causing lowered tissue health is found, it may be necessary to change an individual's diet, add nutritional supplements, or improve the digestive system so the body can correctly use the food ingested. It is very important to remove the cause of bursitis, because long-term bursitis can ultimately result in permanent damage.

Surgery. In 1973, Viladot **(Viladot, 1973)** reported that there were more than 100 operations proposed for the treatment of hallux valgus, with the majority concerned more with foot esthetics foot than with function. There are cases where it is appropriate to refer the patient for surgery; however, one must recognize that surgery is not always successful. Mann **(Mann, 1983)** states, "I believe that only on rare occasions should a hallux valgus deformity be corrected solely for cosmetic reasons. If the patient has a painless hallux valgus deformity, I believe the patient should keep the deformity because even after successful surgery there may be discomfort about the [metatarsophalangeal] joint. I do not believe that the treating orthopedist should ever be in a hurry to correct the hallux valgus deformity because of concern that the deformity will progress. Once the deformity becomes symptomatic, that is the time to contemplate surgery."

A recent Cochrane Systematic Review **(Ferrari et al., 2009)** showed that the numbers of participants who remained dissatisfied at follow up after surgical treatment for hallux valgus, even when the hallux valgus angle and pain had improved, were consistently high (25-33%).

Shoes

Improperly fitted shoes are a major cause of foot dysfunction; perhaps shoes of any kind limit optimal foot development. **(Abshire, 2010; Squadrone & Gallozzi, 2009; Maffetone, 2003; Ramamurti, 1979; Hoppenfeld, 1976)** Abshire, Squadrone & Gallozzi, Warburton, Robbins and others **(Abshire, 2010; Squadrone & Gallozzi, 2009; Warburton, 2001; Robbins et al., 1987, 1988, 1989)** have investigated why barefoot runners in international competition have fewer running injuries than those who have trained in "scientifically" designed running shoes. There is a strong case indicating that foot integrity is developed when adapting to the ground by natural foot contact. In cultures that do not wear shoes, less than 10% of the population will seek medical care for foot pain. **(Langer, 2007)** After studying barefoot populations in China and India, Schulman concluded that shoes were "the cause of most of the ailments of the human foot." Health care access is more limited in these countries, but is has been well-established that shoe-wearing populations are prone to chronic foot problems. In fact, according to the American Podiatric Medical Association, 85% of the U.S. population will seek medical care for foot pain at some point in their lives.

As has been indicated, afferent input from the sole of the foot affects postural awareness significantly. Cutaneous reflexes from the foot are important to posture and gait. **(Kavounoudias et al., 2001)** A shoe dampens the stimulation to foot nerve receptors; it smoothes out the substrate and restricts motion. As far back as 1932, Herzmark **(Herzmark, 1947)** found that providing a mat with uneven surfaces in a playpen helped develop strong arches and good foot function in young children. In 1947, Janse et al. **(Janse et al., 1947)** encouraged barefoot activity for foot rehabilitation in the chiropractic setting. Kerrigan et al **(Kerrigan et al., 2009)** concluded that there was far more impact to the ankle, knee, and hip joints in runners wearing traditional running shoes with elevated heels compared to those running barefoot.

Langer, **(Langer, 2007)** a podiatrist and clinical advisor to the American Running Association, notes that "Walking barefoot allows the feet to function naturally, forcing the muscles to expand and contract, and the joints to bend and stretch to absorb step impact. This, in turn, promotes muscle strength and healthy joint alignment. Conversely, wearing shoes can inhibit the natural function of the feet by providing artificial cushioning and support, constricting the feet into unnatural positions and limiting air flow around the skin and nails." Similar to the way that gloves interfere with the sense of touch in the hands, cushioning in shoes interferes with the sense of touch in the soles.

In the past decade, Masai Barefoot Technology shoes have become a hot item in the marketplace for athletic shoe wear. At the same time Nike, the largest producer of sports shoes, recognized that in order to improve running performance and to decrease running injuries, athletes should frequently train barefoot. (Nike was the originator of the market for "running shoes" with annual revenues of more than $10 billion). **(McDougall, 2009)**

Ramamurti **(Ramamurti, 1979)** calls attention to the effect shoes have on foot development.

> "During barefoot walking, the intrinsic toe flexors invariably participate during push-off. Try to walk barefooted without toe gripping, particularly through sand or over gravelly surfaces. Progress through sand will be tedious and, over a gravelly

surface, painful. Full employment of toe gripping facilitates both tasks. During shoe walking, on the other hand, these provocations to toe gripping are blocked. Hence, the use of shoes will, for most people, decrease the participation by the intrinsic muscles of the foot. However, it will not decrease participation by the long dorsiflexors of the ankles and toes as recovery and heel strike demand their participation, whether in or out of shoes. *As the intrinsic muscles weaken*, the strain on the plantar ligaments and aponeurosis increases, and the tendency of the long extensors and flexors to flex the (interphalangeal) joints while extending the (metatarsophalangeal) joints will be unopposed by the lumbricals and interossei. If, in addition, the heel of the shoe be elevated, the tendency toward talar slip will increase, and if the toe of the shoe be pointed, the adduction forces on the great toe will exceed abduction forces much of the time. Thus, as normal function is prevented, and abnormal postures are forced, painful distortions in the anatomy will begin to emerge."

Maffetone emphasizes this point: **(Maffetone, 2003)**

"An extreme example of this is wearing high-heeled shoes, especially those with very small pointed heels and a small toe box. In this case, all the body's weight is directed into the ground through a very small area – through the heel and the front of the foot. When we wear a flat shoe or are barefoot, the distribution of weight is over a larger area, although even a flat shoe can interfere with our weight bearing."

While society's desire for fashionable shoes drives the footwear industry toward profit, the shoe-wearer's desire for comfort, practicality and diversity of foot use also creates strong countervailing needs. The advent of the modern shoe industry and their new promotion of Barefoot Technologies is a sign that the shoe industry is becoming aware of the science of barefoot walking. An elegant and thorough review of the Barefoot Technologies available to consumers – and the scientific literature regarding life in shoes versus barefoot – has been given by Wallden in the superb *Journal of Bodywork and Movement Therapies*. **(Wallden, 2010)**

At this time there is no need to further discuss the advantages of barefoot function. The constraints of our society require wearing shoes for both protection and comfort, and most of the patients who have foot dysfunctions that present to clinicians for evaluation are wearing shoes during their daily life. However it should be remembered that a growing consensus among runners, fitness experts, podiatrists and researchers suggests that "barefoot therapy" is a critical part of foot rehabilitation. Restoring optimal muscle function in the feet is better achieved barefoot than with many other therapies. Spending ones free time at home, recreationally, and while driving barefoot is an excellent idea. **(Abshire, 2010; Maffetone, 2003)**

There are three prevalent problems with shoes: (1) they are too small for the needs of the feet, (2) they are not properly or soundly constructed, and (3) they are too often selected for style and fashion, rather than the functional features of protection and comfort. **(Abshire, 2010; Maffetone, 2003)**

A Chinese fortune cookie reads, "To forget your troubles, wear tight shoes." Notwithstanding this, Lewin **(Lewin, 1959)** colorfully points out, "There is no shoe too small for an ambitious female's foot." Furthermore, he says, "Fit your feet and not the other person's eye." Unfortunately, it is often difficult to have the proper shoes because the marketability is on style rather than function. **(Abshire, 2010; Robbins & Waked, 1997; Hicks, 1965)** Shoe companies have funded studies hoping to reveal the secrets of shoe comfort, without conclusive results. **(Miller et al., 2000)**

Chantelau & Gede **(Chantelau & Gede, 2002)** noted in their study of foot measurements in subjects 65 and older that two-thirds were wearing shoes that were too narrow. Frey et al. **(Frey et al., 1993)** found in their survey of women with shoe-related pain that most women experienced foot-related problems in their twenties; 88% wore shoes that were too small as measured by an orthopedic surgeon; 79% had not had their feet measured in the last 5 years when buying shoes; 59% wore uncomfortable shoes daily; 76% had foot deformities, with bunions and hammertoes the most common finding; and 80% of women reported significant foot pain while wearing shoes!

Robbins et al **(Robbins et al., 1997)** showed that over-cushioned shoes do not protect from step impact and can even increase impact when compared to firmer footwear. "Expensive athletic shoes are deceptively advertised to safeguard well through 'cushioning impact' yet account for 123% greater injury frequency than the cheapest ones." Maffetone notes "Shoes that are too cushioned can give the nervous system the improper perception that the impact is much less than it actually is, which can result in the inadequate or improper response by the foot (and rest of the body) to the actual impact. In the course of a walk, run, or all day moving about in your shoes, this could add up to be a significant cause of injury." **(Maffetone, 2003)** Nike introduced in 1979 (the company already owned 50% of the U.S. market) its first shoes with Air-Sole cushioning technology (sturdy, sealed pouches of pressurized gas set in the sole that compressed under impact and then sprang back). This new shoe-cushioning technology was an instant success and pushed Nike further into the lead among running shoe corporations and led other brands to develop softer foams, gel packets, and other gimmicks to keep up with Nike's fast-growing promotional and advertising advantage. **(Abshire, 2010)**

Maffetone and Stewart **(Maffetone, 2003; Stewart, 1945)** both provide interesting histories about shoe development. They began as simple sandals of various thicknesses and rigidity to protect the plantar surface from injury by the substrate. The upper portion of the foot needed protection from the cold in more adverse climates. This was originally supplied by loose animal fur and skins. It was not until much later that form-fitted and confining shoes were developed.

Shoes or boots with a true heel did not appear until the 15th century. It is said that Timur, in the battle of Seistan, developed a contracted heel cord as a result of injuries received in battle. After the injury he was known as Timur the Lame, or Tamerlane. By putting a heel under that foot, he

was able to walk without pain; to prevent a limp, he put a heel under the other side also. The uniqueness of the heels would be hidden if everyone in his court wore them. This became a historical method for covering royal defects. According to Stewart, it is probable "…that the heel is essentially a prosthesis for shortened heel cords, and that men and especially women are still paying unconscious tribute to the vanity of a wounded Tamerlane…." **(Stewart, 1945)**

The metal shank was introduced in the shoe to maintain the heel in position and give the foot support, which had been removed by the introduction of the heel. The use of a metal shank is not recorded prior to 1875. In 1875 Charles Goodyear, Jr., developed a new machine that made shoes using a new material called rubber, a substance invented by his father…and the modern shoe industry was born.

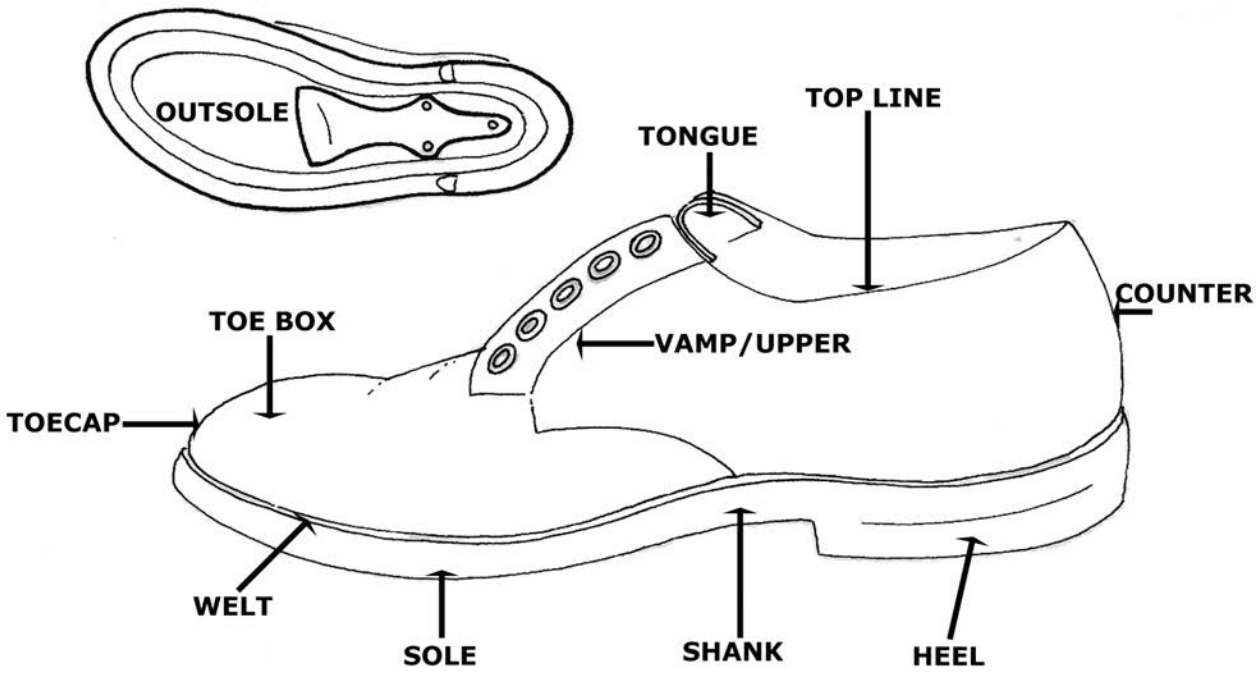
In his historical account of shoe development, Stewart **(Stewart, 1945)** made observations on the effect of shoes on our current status: "One should remember that the heel is essentially a prosthesis for a deformity. Too many writers assume that heels — any heels — are an essential part of a shoe; *few if any approach the heel analytically from the standpoint of physiology*. What is the effect of the heel on normal foot physiology?" In providing an analytical assessment of the heel, he states, "The toes as they are dorsiflexed tend to spread apart, so that the higher the heel the broader the toes should be, but in modern fashions the higher the heel the more pointed is the toe. Thus the strain is doubled.

"Thus it appears that heels from the physiological standpoint are of primary rather than tertiary importance, being the most destructive factor in foot physiology."

Muscle activity changes with varying heel height. For example, there is increased activity in the lateral gastrocnemius in women with 2-1/2″ high heels; this muscle imbalance causes other muscles to lose significant function. **(Butler, 1991; Basmajian & Bentzon, 1954)** Negative heels (earth shoes) decrease activity in the erector spinae and soleus, while the activity increases in the tibialis anterior, rectus femoris, and biceps femoris muscles. **(Soderberg & Staves, 1977)** There have been periodic efforts to popularize the earth shoe to help low back conditions. In the 1930s there was an attempt to popularize the heel-less shoe in England. **(Mennell, 1939)** Some have varied heel height to regulate the lumbar index. None of these attempts have become popular. Maykel, **(Maykel, 1984)** using applied kinesiology testing, found that there is a point of heel elevation in which general muscle weakness develops. He calls this the "calcaneal tolerance factor." Further testing by Maykel and Manning **(Maykel & Manning, 1987)** found a range of 1-1/2″ to 3-1/4″ in which the heel can be raised before general weakness is demonstrated on manual muscle testing.

Heel Helper ™

The current concept of a shoe varies from molded plastic slippers to the conventional shoe, consisting of its various parts. Whether a person can function adequately in molded slippers and sandals depends on the individual's



feet. Obviously this type of footwear is inadequate if foot support or orthotics is needed.

The shape of the conventional shoe is determined by its last, which is the form upon which the shoe is built. There are lasts for neutral, adducted, markedly adducted, and abducted feet, as well as for those with bunions. **(Enke et al., 2009; Richards et al., 2009; Milgram, 1964)**

The base of the shoe is the sole; giving it strength is the shank, which is necessary to provide strength from the ball of the foot to the heel. The shank is usually made of steel or some other solid substance. It is anchored at the heel and extends forward under the longitudinal arches. The shoe should bend at the metatarsophalangeal articulations; **(Langer, 2007; Turek, 1984)** this should not be restricted by the shank extending too far forward. In the absence of a strong shank, there is inadequate support under the longitudinal arches. Shank strength can easily be tested by pressing anterior to the heel to determine if the shoe yields. It is common to lose foot corrections as soon as a patient walks in this type of shoe; it can even be the original cause of the foot dysfunction. A weak shank is especially a problem if longitudinal arch support is provided by an orthotic. When the shank yields to the force of the supporting material of the flexible orthotic, there is no support.

A wedge shoe does not have the "bridge" from the ball of the foot to the heel. Support is provided by the shoe's continuous contact with the ground throughout its length. This provides flexibility without yielding to any supporting pressure.

The shoe portion surrounding the heel is the counter. It should be solidly constructed in all shoes, and is especially important when there is extended pronation. Some counters are made of soft, flexible leather; these do not provide proper heel stabilization. Counters made of solid leather, but having weak attachment to the sole, provide poor support and may become misaligned with use. **(Langer, 2007; McMahan, 1983)** Even new shoes may be improperly lasted so the counter is misaligned with the shoe. In either case, the counter will tend to put the calcaneus into inversion or eversion. **(Langer, 2007; Vixie, 1982)** When a person has good foot function the solidity is not as important, but in all cases the counter must be properly aligned.

The front part of the shoe is the vamp; it covers the forefoot and a portion of the midfoot. The anterior portion containing the toes and anterior metatarsals is called the toe box. The tongue extends from the vamp to cover some of the midfoot. The portion of the shoe that generally connects the counter and vamp is the quarter; it extends over the tongue and has eyelets for lacing. This is called a blucher shoe. It provides good support and is generally preferred over slip-on shoes.

Athletic shoes are designed for specific activities. **(McDougall, 2009; Maffetone, 2003)** Court shoes, including those for tennis, basketball, and racquetball, are designed to cushion against jumping, as well as medial and lateral stresses. Running shoes are developed with forward motion as the primary purpose. Heel and arch cushions are the main features of these shoes. In running shoes, pronation is of primary concern. Firm midsoles are considered important to limit extended pronation. **(Langer, 2007; McMahan, 1983)** Increasing lateral heel flare increases initial pronation. **(Abshire, 2010; Nigg & Morlock, 1987)** Since running styles, speed, and distance differ, it should be obvious that one shoe design will not be optimal for all. **(McDougall, 2009; Langer, 2007)**

The most important aspect in selecting shoes is that they aid natural function, not restrict it. Improved performance can usually be determined by a stopwatch or in an athlete's increased general ability. Applied kinesiology examination helps determine when shoes are creating foot dysfunction, or causing loss of structural corrections that were made by adjusting the articulations or treating muscles. What we have seen in patients who have already seen numerous clinicians previously for their foot problems (podiatrists, chiropractors, massage therapists and others), is that many foot subluxations and muscle inhibitions are still present throughout the foot and ankle. In order to help reform the architecture of the foot in this kind of recurrent foot pain patient, often times as many as 6 or 7 subluxations in the foot will be found using the applied kinesiology sensorimotor challenge, and after correction of these persisting factors will improve muscle function in the foot and ankle as well as throughout the body. **(Leaf, 2006; Lee et al., 2003; Goodheart, 1998-1964)**

The athlete can be educated regarding how his shoes affect his performance by applied kinesiology testing. Unfortunately, selection of athletic shoes may not be for optimal function and safety; psychological and financial rewards may be the ruling factors. **(Chaitow & DeLany, 2002; Ellfeldt, 1983)**

Sometimes shoe construction is based on erroneous assumptions. For example, the competitive cycling shoe typically has a rigid sole, with the midsole made of wood or plastic. The purpose has been to distribute the pedal force over a larger area of the foot to prevent ischemia and consequent paresthesia that develop in competitive cycling. In steady speed cycling, it has been demonstrated that the distribution of force is more even in the forefoot with running shoes than in the cycling shoe, thus negating the supposed advantage of the solid shoe. **(Sanderson & Cavanagh, 1987)**

Proper shoe fit. Regardless of how well a shoe is designed and made, all is lost if it does not fit properly. Shoes cause a high percentage of foot deformities, the most common of which is hallux valgus, discussed previously. In general, foot deformities increase when foot mobility is lost and decrease with increased foot mobility. There is greater mobility in those not wearing shoes than in those who do. **(Abshire, 2010; Wallden, 2010; Squadrone & Gallozzi, 2009; Sim-Fook & Hodgson, 1958)** Additionally, there is more foot dysfunction — such as subluxations, fixations, and muscle weakness — when there is loss of mobility. Loss of mobility is primarily due to wearing shoes that are too small. This is a common finding; rarely do people wear shoes that are too large. **(Chaitow & DeLany, 2002)**

A shoe store should have an adequate stock in all shoe styles so that a person can progressively evaluate larger shoes. Sometimes an improper fit occurs because the store doesn't have the right size. Both feet should be measured while standing. Asymmetry is common, but there is no significant correlation between limb dominance and foot breadth. **(Rothbart, 2006; Didia & Nyenwe, 1988)** The shoe fit should be for the larger foot. **(Mennell, 1939)**

Becoming friendly with the owner and salespeople of a quality shoe store is a valuable service to your patients.

This referral service is two-way. You will learn the types of shoes available, and the sales people will know the type of conditions you successfully treat for referring those who have foot problems. For some patients, it may be necessary to find a shoe store that still makes custom shoes that specifically fit a specific patient's anomalous foot. An Internet search for a local custom shoemaker may be the best method for offering custom shoes with precise measurements and service for these patients.

Langer **(Langer, 2007)** offers a shoe-store clerk's perspective:

> "I worked in shoe stores while a podiatry student and have seen first hand how difficult it can be to convince some women that they are wearing shoes that are too small for their feet. In my experience, many women have become so accustomed to tight, constricting shoes, that when they wear properly fitting shoes, they are convinced that the shoes feel 'sloppy'. Both as a student and as a practicing podiatrist, numerous women have insisted to me that their shoe width was double-A or triple-A when they clearly needed footwear two or three sizes wider. As a shoe clerk, I had to give them what they wanted, and they almost always wanted the shoes that were too small. As a podiatrist, I can now advise both women and men that their foot pain will only get worse if they insist on wearing poorly fitting shoes."

A shoe must fit the foot properly at the heel, across the metatarsals, and lengthwise. A study by Schwartz et al. **(Schwartz et al., 1935)** shows the need for a snug counter and solid last if extended foot pronation is to be controlled. The snug fit is around the medial and lateral aspects of the calcaneus, not just at the posterior of the heel. A loose or flexible counter allows the calcaneus to slip, especially when an individual makes sharp turns while walking. The importance of good fit in this portion of the shoe is exemplified by the case of phlebitis perpetuated by foot dysfunction, discussed in the **Dynamic Examinaton Section of this Chapter.**

Sometimes one cannot obtain proper fit of the counter around the calcaneus and still have the other aspects of the shoe fit correctly. This is especially true in those who have a wide forefoot and narrow hindfoot. Lack of snugness posteriorly can be improved by applying orthopedic felt to the tongue to force the foot back into the counter. **(Mennell, 1939)** Padding around the calcaneus to make a snug counter fit is usually not adequate.

Many people wear shoes that are too short. This may result from children's feet growing rapidly or adults thinking their feet never change in size, and that all shoes of the same size will fit their feet properly. Wearing shoes that are too small may be the result of vanity, even though probably no one would notice the change if the proper size were worn. Generally, shoes without laces — such as loafers and pumps — are fitted too short to avoid heel slippage. Patients may be accustomed to a poor fit and say that the shoe is comfortable. Frey et al found that the average woman wears shoes that are 1-1 ½ inch too narrow and a half size to a full size too short. **(Frey et al., 1993)** They may be unaware of the problems the shoe is causing because symptoms such as neck pain or headaches are remote from the foot.

Proper shoe fit in the forefoot is different from that of the heel counter, where a snug fit is needed. In the forefoot, adequate length and width are needed for proper foot function.

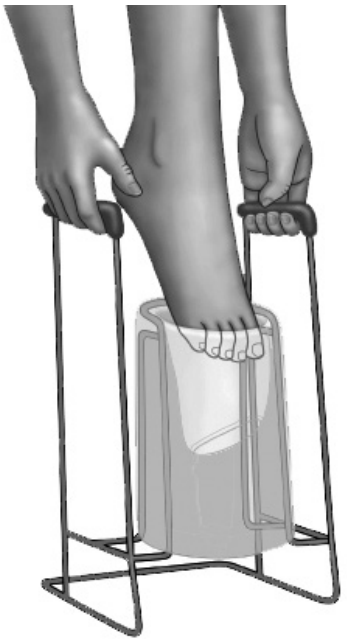
Shoes should always be fitted for the weight-bearing and functioning foot. This requires consideration of several foot positions. The greatest length of the shoe is required during the stance phase near toe-off; as the shoe flexes the counter stops posterior foot movement, causing the toes to slip forward on the surface of the sole. Mennell **(Mennell, 1939)** considers that 2/3″ over the standing foot length is necessary for this slipping action of the foot in the shoe.

An approximation of proper length can be obtained by standing in stocking feet on a length of cardboard 3/4″ wide. Mark the cardboard straight down from the heel and mark the end of the toe. Cut the cardboard to this length, and put it inside the shoe to be evaluated. When the end of the cardboard touches the front end of the shoe, there should be approximately 1/2″ to 3/4″ spaces between the end of the cardboard and the back of the shoe based on a round toe box. The distance from the ends of the toes to the end of a pointed shoe must be considered on an individual basis. Since children's feet grow sporadically and in very rapid spurts, this measurement should be done on them approximately every three or four weeks. **(Greenawalt, 1985)**

Leaf **(Leaf, 2006)** demonstrates how changes in foot size occurred in 180 people after applied kinesiology procedures were employed to the foot and ankle. First, the patient's foot was placed lightly on paper and the foot outlined. Second, with their foot bearing their body weight another tracing was drawn with another colored pencil. An increase of more than ¼ inch indicated the loss of intrinsic foot support. Only 15% of the participants in this study had a difference of less than ¼ inch weight bearing compared to non-weight bearing. Applied kinesiology testing and treatment procedures were applied to the muscles, joints, and skin in the ankles and feet of all patients. Skin imbalances were treated using Kinesio tape. **(Kinesio Tape)** Proprioceptive neuromuscular facilitation was applied to the involved ankle and foot muscles. Spinal subluxations from L4 to the sacrum were treated if present. The patient was then instructed to walk for 30 steps. A new piece of paper and tracing measurement of the foot was taken and compared to the original. In all the cases with more than ¼ inch difference non-weight bearing compared to weight bearing, after the applied kinesiology protocols were utilized, the second tracing showed markedly less difference in foot size than the original tracing.

Maffetone **(Maffetone 2003, 1989)** has observed a common condition in athletes that he calls the "short-shoe syndrome" or "first metatarsal jam". This results in a 1st metatarsal subluxation that develops insidiously. Because of the slow adaptation the foot is forced to make to the restriction, the athlete does not usually complain of pain at that location; he has other foot complaints. It is important to recognize the 1st metatarsal's problem and its cause, since change in 1st ray function may be the basic cause of gait change resulting in other foot problems or remote dysfunction.

Wearing shoes that are too short tends to jam the metatarsal back into the 1st cuneiform, subluxating the articulation. Indicating the problem may be 1st metatarsal swelling, blisters or calluses of the toe, or discoloration of



Sock-donner

the nail bed (the "black toenail"). Although the patient does not usually complain of pain in the 1st ray, it will generally be there when the joint is palpated and moved. The subluxation is confirmed by challenge. The vector of correction is usually the 1st metatarsal base in a plantar direction. It may have a more medial or distal vector. Since the cause of the problem is improper shoe fit, the patient's shoes must be evaluated as indicated later, and the patient educated regarding the cause of the problem. Maffetone recommends evaluating an athlete's training shoes, especially for the 1st toe position. It may be necessary to look at the shoe's insole, or to palpate the inside of the shoe to find the wear pattern from the 1st toe. This condition must be differentially diagnosed from functional hallux limitus as discussed earlier.

Before leaving the discussion on shoe length, we should consider short socks or stockings that can be just as problematic as too short a shoe. This foot restriction can cause bony deviation, fixations, subluxations, and ingrown toenails. Socks are an important part of footwear as they protect the skin from friction, keep the feet dry and thereby minimize bacteria and fungal problems. Many older patients do not wear socks because they are too difficult to get on. A device called a sock donner can make this task much easier. This tube-like apparatus stretches the sock and helps pull it up the leg with less strain on the hands or the low back.

The widest portion of the foot at the metatarsophalangeal articulations should match the widest portion of the shoe. This is called a ball-fit; correct fit at this point is crucial. **(Greenawalt, 1989; McMahan, 1983)** The 1st metatarsophalangeal articulation should rest at the widest part of the shoe. If it passes that point and is closer to the toe of the shoe, the shoe is too short. Seldom will you find a shoe too long. **(Greenawalt, 1985)** In addition, the shape of the shoe should match the forefoot. Some people need specially lasted shoes to match forefoot deviation. **(Enke et al., 2009; Wickstrom & Williams, 1970)**

The toe box should be large enough that toe movement is not restricted within the shoe. Width and height are important. The circumference around the metatarsal heads can increase up to 3/4" from non-weight-bearing to standing. The vamp of the shoe should not taper excessively toward the toes or toward the sides of the shoe, decreasing the forefoot space. **(Mennell, 1939)**

Observe whether the foot causes the vamp to spread over both edges of the sole; if it does, the shoe was too narrow for the foot when purchased. If the vamp rides over the sole on the lateral side, a Dutchman's wedge can help restore even sole wear. **(Greenawalt, 1985)** It is a smoothly cut, thin-layered wedge built into the shoe under the lateral forefoot.

It is common to wear shoes that are too small. Over an 18-month period in Maffetone's clinic, **(Maffetone, 1989)** 52% of the new athletic patients were found to be training in shoes too small, confirming a similar finding for patients 65 and older cited earlier. **(Chantelau & Gede, 2002)** Many people automatically buy the same shoe size, even though shoes vary from brand to brand regardless of the same indicated size. They even vary from the same manufacturer. A recent Internet search for "walking shoes" found 864 different models! In addition, people's feet change.

When buying shoes, it may be of value to measure the feet two or even three times during the day. Feet typically are larger at the end of the day, but there should not be more than a half-size difference. A larger difference requires additional examination to determine the cause of the edema fluctuation. Women's feet in pregnancy do not significantly increase in length or width, but they do significantly increase in volume that lasts for up to eight weeks postpartum. **(Wetz et al., 2006; Alvarez et al., 1987)** This indicates no change in ligamentous laxity; the complaint of tight shoes is due to edema.

Try on several pairs of shoes and spend enough time to make a thorough evaluation. You may not find the best shoe for you in the first store you visit. Most shoe stores carry only a few of the 864 different models available. Walk on a hard surface rather than on the soft carpet often found in shoe stores. Maffetone **(Maffetone, 2010, 1989)** recommends shoe fitting by "trying on the size you normally wear. Even if that feels fine, try on a half size larger. If that one feels the same (or even better), try on another half size larger. Continue trying on larger half sizes until you find the shoes that are obviously too large. Then go back to the previous half-size and usually that's the best one." Use comfort as the main criteria. Similarly, try various widths. The width is checked easily by placing the thumb and forefinger across the widest part of the forefoot and squeezing. If the foot is stretching the upper tightly, then the shoe is too narrow. "Don't let anyone say that you will have to 'break them in' before they feel good." Shoes should not be purchased unless they feel comfortable after walking in them sufficiently at the store.

Foot Support

The patient's foot and its supporting muscles should be thoroughly examined and treated before foot support is considered. With a comprehensive understanding of the foot and any remote dysfunction, the decision regarding

support can be made. Support is needed when corrections do not hold, or when immediate relief of pain is necessary. In either case, the need for support means that correction has not been made or is not permanent. The best approach is to obtain a lasting correction and a properly functioning foot without the need of any support. Obviously, this is not always possible; knowledge of padding, strapping, and orthotics is of value for temporary use or on a permanent basis. As greater knowledge of how to correct and rehabilitate the foot is gained, less and less support is needed.

As previously discussed in this chapter, foot dysfunction can cause the return of cranial faults, shoulder problems, spinal subluxations, and a myriad of other conditions as soon as the patient walks. The fact that remote conditions return with walking does not necessarily mean that the feet are the problem. Other factors such as gait and pelvic motion must be considered. If foot subluxations or other foot dysfunction returns with walking, it is a good indication that the problem is still in the feet.

A method to help determine if the feet are the cause of a condition returning immediately after walking is to provide tape support to the foot, or to have the patient walk on the outside edges of his feet to eliminate extended pronation. If the remote condition does not return under these conditions, further foot correction or support to the feet is probably needed.

Foot supports are prescribed to improve function or relieve symptoms. This may be done to transfer weight from painful areas, separate structures such as in Morton's neuroma, rehabilitate functional hallux limitus, reduce strain on muscles, give support under hammertoes, or support under forefoot varus. The use of pads and supports for any of these examples, as well as for many other reasons, may be necessary, but function is often overlooked. The literature dealing with treatment by padding tends to emphasize the painful foot rather than pain and function. **(Ball & Afheldt, 2002)**

Examination with applied kinesiology methods helps address the question of function. This discussion on pads and taping will deal primarily with the painful foot and providing temporary support. Possibly more important is the portion of the discussion on evaluating the effect of supports that the physician has applied to the foot and ankle. This two-phase discussion then deals with applying the physician's knowledge to the condition, and evaluating how the patient's body reacts to the analysis and the supportive corrections the physician makes.

Material for padding and taping. Foot support may be needed only temporarily, for a moderate time, or on a permanent basis. **(Refshauge et al., 2009)** A temporary support, provided early in treatment, gives the ability to evaluate the need for and proper placement of the material. A few supplies are necessary to be able to provide quick office support.

There are four basic approaches to support: 1) evaluating and correcting, if needed, the patient's shoe selection, as previously discussed; 2) directly applying support to the patient; 3) putting a pad or an orthotic in the shoe; and 4) modifying or changing the patient's shoes.

When support is applied directly to the patient, as in taping or by adhesive pads, the skin should be properly prepared. Any hair in the area should be removed. Small electric clippers used by barbers to trim sideburns and mustaches trim very close to the skin and do not irritate it as shaving does. Compound Tincture of Benzoin applied to the skin increases adhesion of the material to be applied and reduces the possibility of allergic reaction.

Adhesive-backed 1/4″ sponge rubber from 8″ stock rolls is an easy material to apply directly to the patient. The paper or plastic backing that protects the adhesive surface is left in place until the material is cut to the correct size, edges beveled, and ready for placement. Greater thickness can be obtained by layering the material. When this is necessary, the edges should be smoothly cut into thin layers so there are no lumps in the pad. With weight bearing, the layered pad quickly unites into a single mass. When the pad is properly placed, as indicated by testing described later, it is covered with adhesive material such as moleskin or tape. Moleskin is a smooth, adhesive-backed material that has many uses in padding and supporting the feet.

Adhesive support can be protected during bathing or showering by covering the foot with a plastic bag held in place with a rubber band around the ankle. After bathing, the exposed portion of the foot around the support can be washed separately.

Removal of adhesive pads and tape should be done from distal to proximal to work with the grain of the skin. This is particularly important over calluses. Adhesive solvent is available to make removal easy for both technician and patient. It is usually packaged in a can with a spout for control of application. As the adhesive material is removed, apply a few drops of solvent between the adhesive surface and skin. Continue to remove the support while adding more drops as necessary. After the support is removed, any residual adhesive on the patient can be removed with the solvent applied to a gauze sponge. Wash the skin with soap and water, and dry.

Medial longitudinal arch.

The usual reason for supporting the medial longitudinal arch is to control extended pronation. This can be accomplished with rigid or pliable orthotics. **(Kogler et al., 1996)** One study showed how pronation control was achieved using rigid orthotics in a group classed as pronators. **(Bates et al., 1979)** They had been successfully treated with orthotic appliances. Their function was evaluated with high-speed photography while running barefoot, in a regular shoe, and in a regular shoe plus an orthotic device. The period of pronation and the amount of maximum pronation were significantly reduced using the foot orthotic device. These results were compared with normal or asymptomatic runners. During running, the normal group had greater ankle dorsiflexion and less corresponding values for maximum pronation. This may indicate that a short triceps surae was contributing to the excessive pronation in the orthotic group.

Christensen **(Christensen, 1983)** used high-speed photography to evaluate the effect of Spinal Pelvic Stabilizers®, **(Foot Levelers, Inc.)** a flexible orthotic used for controlling extended pronation. He demonstrated that "…maximum pronation and percentage of support time were significantly decreased in the SPS condition when compared to the shoe condition."

There is a tendency to support the longitudinal arches

in the cavus foot. In most cases this should not be done because it takes away ligament stretching **(Wenger, 1989; Turek, 1984)** and often gives poor relief of symptoms. **(Dugan & D'Ambrosia, 1986)** It is justified only when applied kinesiology testing indicates need for support. If support is given, begin a stretching program and gradually reduce the support elevation as improved foot position is obtained.

When support to the medial longitudinal arch is needed, care must be taken not to err toward overcorrecting the arch. The individual with a short triceps surae should not have support that eliminates pronation, which is necessary to give flexibility to the foot that must adapt to the limited dorsiflexion. **(Wyndow et al., 2010; Warburton, 2001)** First the triceps surae must be lengthened with techniques indicated by AK examination, e.g. pincer palpation followed by percussion, neuromuscular spindle cell, Golgi tendon organ, fascial release, trigger point pressure release, occasionally the use of the origin-insertion technique, Graston Instrument, PRYT, dural tension, and others. **(Leaf, 2010; Cuthbert, 2002; Walther, 2000)** If there is still limited dorsiflexion, the stretching program described later should be taught to the patient.

Rubber pads of various types and sizes can be stocked; they provide good temporary support to the medial longitudinal arch while foot corrections are being obtained. The patient may be evaluated for need of a more permanent type of support, such as orthotics or shoe modification, at a later time. Temporary support for the medial longitudinal arch is put in the shoe under the navicular bone. These are called navicular or scaphoid pads; in the shoe industry, they are called "cookies." They are available in different heights, lengths, and widths. The cookie is temporarily placed in the patient's shoe, and the patient is tested to determine if the support is correct. If the cookie tends to slip while the patient walks, it can be held in place by a small piece of double stick tape. When the correct size cookie and its proper location are determined, solidly fasten it to the shoe with additional double-stick tape. It often helps to cover the cookie with moleskin, which extends about 1/2″ to 1″ over the edges of the cookie. Rather than gluing the cookie in place it is better to use double-stick tape and moleskin, because any residual adhesive can usually be easily removed with adhesive solvent when the cookie is no longer desired. The cookie does not tend to move in the shoe as much as metatarsal pads.

Versatility of AK diagnosis

Because of the versatility of manual muscle testing it is possible to evaluate patients in many different positions and states (including walking, sitting, supine, prone and while moving any number of parts of the nervous system) while the manual muscle test is being conducted. The AK manual muscle test system for testing patients while moving or in positions of physical stress adds to the ability of the doctor to diagnose the muscular dysfunctions that occur with everyday activities. In the typical AK clinical encounter patients are frequently put into positions of pain during the MMT examination. For instance, many patients suffer their foot, ankle or leg pain primarily when standing. The MMT of the muscles of the foot and ankle are frequently tested after walking in these cases. This is thought to also evaluate dysfunctions in the feet that may be producing weaknesses that are not present in the supine position of normal examination. The muscle weakness connected to these patients' foot and leg pain may only be found after walking or in the standing position. **(Zampagni et al., 2009; Nicholas & Marino, 1987)**

An example of applied kinesiology is offered by Gangemi **(Gangemi, 2011)** who presents an approach for treatment of tibialis posterior muscle dysfunction. Adding extra stress to the tibialis posterior muscle with a specific challenge before the manual muscle test, and then supporting any inhibition found with a simple taping procedure, will assist in the recovery of tibialis posterior dysfunction.

> "The tibialis posterior muscle is often a muscle that is harder to keep corrected (facilitated) after treatment…. Provided the physician has thoroughly evaluated and corrected the cause of the tibialis posterior problem, whether structural, chemical/nutritional, or emotional, and has verified that any and all footwear or supporting devices are not hindering its function, the patient is ready to provide a stress test to the tibialis posterior.
>
> "The physician will ask the patient to stand and put the majority of weight on the affected tibialis posterior leg. Next, the patient should be instructed to shift the majority of weight to the forefoot, lifting the heel slightly off the ground as if trying to push an object through the floor with the ball of the foot. Finally, the patient will be instructed to torque the lower leg by rotating the foot, ankle, and lower leg back and forth three to four times. This movement can be likened to doing the opposite motion of twisting a cork out of wine bottle, by pushing rather than pulling. After this procedure is done, the patient should immediately lay supine on the treatment table and the physician should re-test the tibialis posterior muscle. Re-inhibition to the muscle after the stress test proves that either the interosseous membrane and/or the deep fascia is unable to handle the overwhelming stress of all their functions and allow the muscle to heal. Therefore, a simple taping method is used to help the support the tibialis posterior and the surrounding tissues so the patient may recover much faster.
>
> "Once the tibialis posterior is stressed and the muscle once again shows inhibition, the patient is then asked to assist the physician by placing the hands on either side of the lower leg and compressing (squeezing) them together, with a moderate amount of force. The patient begins at the top of the lower leg at the head of the fibula and the physician tests the tibialis posterior muscle. If the muscle becomes facilitated, the physician notes this and stops. If the muscle does not facilitate, then the patient is instructed to move the hands down about one-inch and the physician retests the muscle. Each time the patient moves distally down the leg, the physician retests the tibialis posterior. There will be one place somewhere

between the head of the fibula and the medial and lateral malleoli, where the muscle will facilitate (become strong). In the example here **(photo 1)**, it is approximately half-way down the lower leg.

"Once the exact area of maximum facilitation is found, it is supported with athletic tape. The taping method used here is to simply wrap the tape one to two times around the lower leg, compressing the fibula and tibia together **(photo 2)**. The tape should be wrapped tightly enough to provide the needed support but not so tightly that it causes any skin irritation or vascular compromise.

photo 1

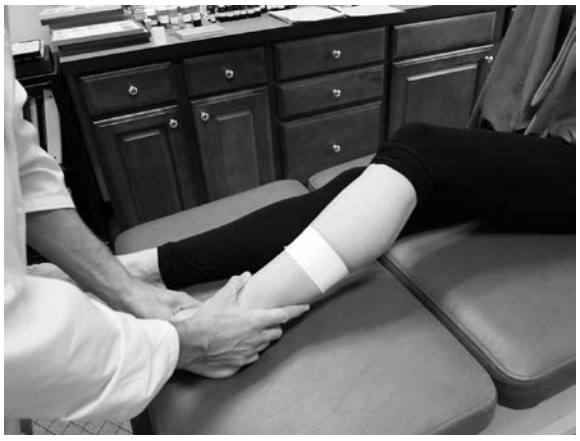


photo 2

"The patient should then re-perform the same torque test that previously weakened. This will ensure the tape is properly supporting the tibialis posterior, deep fascia, and interosseous membrane so the patient will not "walk back into the problem." If the tape does not provide support, it is either too loose or on the wrong location of the leg.

"The tape ideally should stay on the rest of the day until the patient is off his or her feet and into bed for the night. It can then be removed. If there is some reoccurrence of pain or return of the problem the next morning, the tape can be reapplied and worn as long as it continues to help. Sometimes two to three days of tape support are needed. In some cases, there is a different spot which needs to be taped. This can only be verified by a return visit to the office to see exactly what area now needs tape application. The author has seen this in cases where the patient has placed significant torque stress on the lower leg…

"Additionally, fascial release technique can often be beneficial to certain tibialis posterior problems. This can be verified by stretching the tibialis posterior and testing for a reoccurrence of the inhibition. **(Walther, 2000)** Fascial release treatment should then be performed and can also be done on the area where patient compression strengthens prior to applying the tape, as previously described." **(Gangemi, 2011)**

Metatarsal support. Metatarsal support is commonly used and offers considerable relief for many patients, particularly for the functional hallux limitus (FHL) described earlier. When used correctly, it can help hold corrections obtained by adjusting subluxations or fixations. Improperly used, it can cause subluxations and pain.

The stock metatarsal pad is pear-shaped, supporting the medial metatarsals. It may be a separate pad or incorporated into off-the-shelf "arch supports." Patients often self-prescribe this type of support and purchase it at a drugstore. It provides support best for the 3rd metatarsal, and supplies some support to the 2nd and 4th metatarsals. When the 1st metatarsal is involved, the stock-type pad provides no support. If specific support is required for the 4th, 2nd, or 1st metatarsal(s), it must be prescribed or custom-made by the physician as a temporary support. It may be best to use the temporary approach over several weeks to determine need for a more permanent appliance. A trial period of support provides information about the width, height, and exact placement prior to having custom supports made.

Metatarsal pads are used to help maintain metatarsal subluxation correction and to control pain. When painful walking is the problem, subluxation of one or more metatarsals — or perhaps a metatarsal fixation — is almost always present.

The thickness of padding can be estimated by determining the amount of motion between the metatarsals. Using scissors-type motion palpation, determine the flexion-extension motion between each two metatarsals. More padding is needed and tolerated under metatarsals that have considerable motion, and less under those that are restricted. **(Nordsiden et al., 2010; Jahss, 1982)**

The first consideration is to pad for recurrent subluxations. The subluxated metatarsal head can usually be easily palpated as displaced plantarly. Palpation may reveal exquisite tenderness, which will help delineate the offending metatarsals. Padding is put under the distal metatarsal shaft and does not extend under the metatarsal head. Outline the area indicated by palpation and tenderness with a marking pencil. The pad is formed with the adhesive backing in place and smoothly cut into thin layers to match the outlined area. Peal off the backing and apply directly to the prepared skin, using the markings as a guide. Cover

the pad with moleskin or non-compressive adhesive tape (Dermicel). If you choose to have the tape encircle the foot, it should be applied loosely to avoid impeding circulation with normal forefoot spreading during weight bearing. Covering only the plantar and lateral aspects of the foot is usually adequate.

Donner **(Donner, 1985)** recommends putting the pad in the patient's shoe. To accurately locate the position for a metatarsal pad, put a small dot of water-soluble paint on the offending metatarsal head(s). Have the patient plantar flex his toes while putting his foot into his shoe. This may be difficult, but it protects the paint until the patient's foot is properly placed. The patient now stands comfortably with his toes in neutral position. Remove the shoe and glue a single or multiple metatarsal pads just posterior to the dot to contact the distal metatarsal shaft. A hot glue gun or other fast-drying glue should be used.

Occasionally it is necessary to take pressure off most of the metatarsal heads. This is done with a J-shaped pad made to support the distal shafts and transfer weight to the 1st metatarsal, which is the strongest and most capable of providing support to the weight of the body. **(Kang et al., 2006; Milgram, 1983, 1964)** The long portion of the J is applied transversely across the foot, with the short portion extending to just behind the metatarsal heads that need support. Several variations of this type of right-angle pad can be used to selectively supply support where needed.

Many types of health problems originate from a mechanism in the body that has an integrated function about which the majority of all doctors is unaware. The reasons the function of the foot is not better recognized as a cause of health problems is the difficulty in determining when function is improper. Applied kinesiology has taken a major step forward in the understanding of health problems which relate to the functional gait and foot mechanism; AK provides an effective method of evaluating and treating this system.

Orthotics

Generally orthotics are needed only in chronic conditions that are uncorrectable, or sometimes for a short term while correction is being accomplished. **(Bartold, 2004)** Usually orthotics are not needed or desirable for young children. It has been shown that in flexible flatfoot, there is no statistically significant improvement in three years of wearing corrective shoes or inserts in young children. Wenger et al **(Wenger et al., 1987)** suggest that in most cases orthotics do not change osseous relationships and are ineffective. They suggest that arch supports may make the patient's symptoms worse if the shortened triceps surae is not corrected.

Off-the-shelf orthotics for adults rarely provides proper support. The height, length, and width of the medial longitudinal arch vary so much between people that the chance of obtaining the proper support is very slim. If the patient also needs metatarsal support, there is increased chance the support will be incorrect and may even do harm.

When support is needed on a long-term basis, it is usually best to provide the patient with custom orthotics. **(Prior, 1999)** Wise use of this kind of foot support can turn a patient's poor response into a successful outcome. Although foot support can be the difference between success and failure, its application should always be based on physiologic need. Improper application may be more than unnecessary; harmful effects may result.

An example of the improper use of orthotics is a case of low back pain in a 7-month pregnant dentist treated by SCC. This patient was an active, healthy 30-year-old who continued her running exercise during her pregnancy, as she had during a previous one. Her back pain developed over a 2-month period prior to examination. At the time of examination, the back pain was severe enough that she could stand only for approximately a half hour before having to lie down. She had to quit work, whereas in her previous pregnancy she worked until delivery. Initial examination was relatively unremarkable. The only dysfunction found was a 2nd lumbar subluxation and a pelvic Category I, which were adjusted; she was re-scheduled for follow-up in three days. At that time she said she had been completely free of pain throughout the rest of the day of the initial examination; the pain returned while she was cooking breakfast the next morning. Again I found a 2nd lumbar subluxation. This time I spent more time examining and correcting the intrinsic muscles of the spine at that location. The vertebra was again adjusted, and she was scheduled for follow-up in two days. She came in and again remarked that she'd had no more pain the remainder of the treatment day, but it came back while cooking breakfast the following morning. I questioned her about her activities from the time of rising until cooking breakfast when the pain developed. She said she did her daily run shortly after getting up. She experienced no pain during the run, came home and showered; the pain developed while cooking breakfast. I told her it would be necessary for me to test her gait pattern because the subluxation might be recurring during her run. She said she felt there was no problem with her running; in fact, at a recent running clinic her gait had been analyzed and declared to be nearly perfect, except for a little "flick" of her foot just after toe-off. She further stated this had been corrected with orthotics, which she now wore while running. I was astonished to hear that she used orthotics while running, because during the initial routine foot examination always done on every new patient, I found no problem whatsoever with her feet. Her feet were unusually pretty, showing no stress, calluses, or other body language indicating foot dysfunction. This time I examined her using the applied kinesiology gait testing technique and found no problem. Again I corrected the 2nd lumbar subluxation and told her to come in the next day. She was not to run before the office visit, and I wanted her to bring her running shoes and orthotics. When she came in, she had cooked breakfast and spent half the morning with no back pain developing. Upon examination there was no subluxation in the lumbar spine. I had her put on her running shoes with the orthotics and go across the street to run in the park. When she returned, examination disclosed the presence of the 2nd lumbar subluxation. Upon questioning I learned that her backache had started slowly, approximately one week after the running clinic. I again corrected the 2nd lumbar subluxation and she ran in her running shoes without the orthotics; no subluxation developed. I had her discontinue the orthotic use, and she needed no more adjustments. She is occasionally re-evaluated for other health issues; she has had no more back pain and continues her running program

successfully.

The orthotics prescribed in the above case were rigid. In a popular article entitled *The Great Orthotics Debate* Gill **(Gill, 1985)** highlights the controversy and emphasizes the cost, which ranged from $200 to $300. He summarizes by writing, "If you decide to invest in a pair of orthotics, don't expect any miracles. They're not cure-alls, merely aids."

Langer **(Langer, 2007)** concurs: "An insole or orthotics is only as supportive as the shoe in which it is placed. Shoes that do not have room for an insole or orthotics are not capable of helping a painful foot, and unsupportive shoes will not magically become supportive by adding an insole or orthotics."

Our experience indicates that rigid orthotics create problems more often than the flexible orthotic. It appears there are three reasons for this. 1) They are made to conform to what the physician's education indicates the foot balance should be. 2) They are unforgiving, i.e., they do not work with the foot but rather attempt to control it. 3) They are made from a cast of the non-weight-bearing foot.

The non-weight-bearing plaster cast does not represent the foot in its functional position. Prior to forming the cast, the physician puts the patient's foot in what appears to him the correct position. The orthotic is fabricated from the information the cast provides. Rigid orthotics may provide hindfoot or forefoot posting. A post is a wedge in the orthotic to control valgus or varus distortions. Michaud **(Michaud, 1997, 1987)** points out that rigid orthotics designed to post for rearfoot or forefoot deformities must be cast properly, otherwise iatrogenic problems might result. With flexible orthotics, there is less chance of iatrogenic problems because they flex with the foot. Lillich and Baxter **(Lillich & Baxter, 1986)** characterize the ideal orthotic as lightweight, dynamic, and durable. Langer and Subotnick **(Langer, 2007; Subotnick, 1991, 1975)** use rigid, semi-rigid, and flexible orthotics, but they recommend the flexible type for certain types of competition.

Spinal Pelvic Stabilizers® **(Foot Levelers, Inc.)** are flexible orthotics widely prescribed in the chiropractic profession. We have found these very satisfactory when orthotic supports are needed. They provide the advantage of flexibility and most often provide proper support for the patient, confirmed by applied kinesiology testing. The orthotic is fabricated from measurements obtained from a weight-bearing cast. As in measuring for shoe fit, the weight-bearing factor is important. People's feet change in different ways from non-weight-bearing to weight bearing. The measurements are best made in the manner in which the feet function, and feet function as weight-bearing structures. Another apparent advantage of these orthotics is the lateral arch support, which is rarely present in rigid supports.

Most patients who need orthotics have limited motion in the forefoot. This is usually from wearing too-tight shoes. The orthotics will start to mobilize the forefoot, which often causes discomfort. Evaluate the forefoot motion when a patient is molded for orthotics; if it is limited, teach him to do the golf ball exercise, described later. This will help avoid most of the discomfort when he starts wearing the orthotics.

Another cause of discomfort when first wearing new orthotics is limited range of motion in areas remote from the foot. **(Dananberg, 2007; Prior, 1999; Aronow, Solomone-Aronow, 1986)** The body works as an integrated whole. Emphasis has been made on how foot dysfunction can adversely affect remote areas on a neurologic basis. Likewise, symptoms may develop in remote areas of the body that have limited range of motion and cannot accept increased activity created by improved foot function. Examining for the muscle stretch reaction and the need for strain/counterstrain technique will usually demonstrate the type of treatment needed. In addition, the patient may need to do some generalized stretching techniques. **(Looney et al., 2011; Page et al., 2010; Dananberg, 2007; Prior, 1999)** Athletes often overdevelop certain areas relating to their sport. This should give clues indicating where to examine for limited range of motion or antagonist muscle weakness.

Custom-made flexible orthotics, such as Spinal Pelvic Stabilizers®, may occasionally need to be modified. This can be accomplished by adding folded pieces of adhesive tape to the underside of the orthotic. For example, if there is a recurring inferior distal 4th metatarsal subluxation, fold three or four layers of tape into a pad about 1/2″ wide by 1″ long; with another piece of tape, attach it to the underside of the orthotic. Have the patient use the orthotic for a while, then challenge to determine if the subluxation returns. The temporary pad can be readjusted until the proper amount and placement are found. It is usually best to have the patient use the orthotic for several weeks before sending it in for modification, because the feet will change as you continue to treat the dysfunction. Often it will not be necessary to send the orthotic in for modification, because the tape pad adequately provides the necessary support. If modification is necessary, contact Foot Levelers, Inc., with a description of the procedure you have done and send the orthotic to them for the modification. **(Foot Levelers, Inc.)**

When it is necessary to prescribe an orthotic, it does not mean the patient is destined to wear them permanently. **(Gangemi, 2011; Maffetone, 2003; Michaud, 1997)** Greenawalt **(Greenawalt, 1987)** describes a study in which weight-bearing x-rays were used to evaluate femur head height, sacrovertebral angle, and lumbosacral disc angle before and after wearing Spinal Pelvic Stabilizers® without receiving any other treatment. The postural x-rays were taken with the subject wearing no shoes, and each parameter measured improved. The balancing of femur head height may have been a combination of improvement in the arch and/or a decrease in pelvic rotation.

As previously discussed, there is no muscle activity supporting the normal arch in a static stance position. It is in the abnormal foot with excessive pronation that muscle activity develops. Franettovich et al. and Suzuki **(Franettovich et al., 2008; Suzuki, 1956)** demonstrate electromyographic activity in the peroneus longus in conditions of flatfoot. When support is put under the arch, the electrical activity of the peroneus longus ceases. Suzuki observed, "In the patient whom symptoms of flat foot have been alleviated by applying an arch-supporting instrument for a long period, action currents from the (peroneus longus) were not observed while standing without use of the instrument." Possibly the improvement in function developed from the body repairing the ligaments and plantar fascia as they were relieved from stress while wearing the orthotic. If foot corrections are made and an effective rehabilitation program is followed, one can often improve

foot function enough that support is no longer needed.

A very important factor in evaluating the effectiveness of orthotics is that the patient's shoes be adequate. This has been previously discussed, but it needs to be highlighted here because this is the reason that properly made flexible orthotics are often ineffective. The counter should be solid and fit relatively tightly around the calcaneus. The shoe should be wide enough in the forefoot, with a large toe box, and be a ball fit. Finally — and very important when using longitudinal arch support — the shoe should have a solid shank. When the shank collapses, support provided by the orthotic is lost.

An ancient Indian book of fables and wisdom tales called the *Pancha Tantra* says that for a foot in a shoe, the whole world seems paved with leather.

Foot Rehabilitation

Triceps surae stretch.

The triceps surae should allow 10° of dorsiflexion at the ankle. Dorsiflexion measurement should always be done passively. When the movement is patient-assisted, it is always greater. **(Bohannon et al., 1989)** One must also limit motion to ankle joint movement only. The examiner should grasp the calcaneus and midfoot to stabilize the calcaneus, navicular, and cuboid bones against the talus before moving the foot into dorsiflexion. It is in this action that there should be a minimum of 10° dorsiflexion. Remember that in excessive pronation, there is increased laxity of the subtalar and mid-tarsal articulations. If the entire foot is brought into dorsiflexion by applying pressure at the metatarsals, it will appear that there is much greater dorsiflexion than is being allowed at the ankle mortise. When motion measurement is limited to the ankle, one is often amazed at how little there is. The foot may fail to reach a right angle with the tibia by as much as 25°. In normal feet and ankles, dorsiflexion may reach 20° beyond the right angle.

When the triceps surae is short, a surgical procedure to lengthen the Achilles tendon is sometimes done. At one time this was the most common procedure performed by orthopedic surgeons. **(White, 1943)** Ryerson **(Ryerson, 1948)** points out, "The tendo-Achilles is not short. The muscle bellies are short." He recommends stretching the muscle to lengthen it.

If there is less than 10° dorsiflexion at the ankle mortise, the triceps surae must be lengthened. **(Logan, 1995)** First obtain as much increase as possible in range of motion by using applied kinesiology techniques directed to the muscular and remote neurologic factors. On a local basis percussion, stretch and spray, trigger point pressure release and fascial release, and treatment to the muscle and cutaneous receptors will often increase the muscle's length. Folic acid helps relieve the pain associated with the tender points in strain-counterstrain involvements, and vitamin B-12 relieves the pain of Travell's myofascial trigger points. There are several techniques in applied kinesiology — such as PRYT, cloacal synchronization, and dural tension relief — that increase range of motion throughout the body. **(Leaf, 2010; Walther, 2000)**

It may be necessary to apply stretching procedures to the triceps surae. The major problem with stretching the triceps surae is that the foot must be used as a lever to apply the stretch. This tends to compound the problem of stretching the ligaments of the midfoot and breaking it down further.

The muscles can best be stretched by a contraction, relaxation, and stretch technique, which has been shown more effective than a ballistic stretch. **(Simons, 2002; Wallin, 1985)** In a ballistic stretch the muscle is rapidly stretched, activating the intrafusal muscle spindles to reflexly cause a protective muscle contraction. The proper stretching procedure is done with the patient facing a wall with his feet approximately three feet from it; his legs are in internal rotation so the feet are in a "pigeon-toed" position, **(Logan, 1995; Travell & Simons, 1992)** and weight is directed to the lateral borders of the feet. This helps lock the midfoot to reduce stretch on its ligaments. The foot is further locked by contracting the intrinsic plantar muscles. **(Janse, 1947)** The patient should be taught to isolate these muscles as much as possible in order to flex the metatarsophalangeal articulations and not the interphalangeal articulations. The latter are flexed with the long flexors, which usually do not need strengthening, especially when claw toes are present. The hands are placed on the wall and the patient leans toward it, stretching the gastrocnemius and soleus almost to their limit. In this position, an isometric contraction of the triceps surae is held for seven seconds. The muscles are then relaxed for two to five seconds, followed by moving the hips forward, keeping the knees extended, and flexing the arms to place maximum stretch on the gastrocnemius and soleus for seven to eight seconds. If the major stretch is desired at the soleus, the knees are allowed to bend slightly. The cycle of contract-stretch is repeated five times on a daily basis. After thirty days of stretching, one can expect approximately 10° increase in dorsiflexion. When the desired range of motion is obtained, it can be maintained by a stretch series once a week. **(Wallin et al., 1985)** It is important to emphasize that MTrPs in the soleus muscle specifically relate to a restriction of dorsiflexion of the ankle **(Grieve et al., 2011; Travell and Simons, 1992).** Additionally, metatarsal head pain may be the result of a shortened triceps surae. **(Mennell, 1969)**

Toft et al. **(Toft et al., 1989)** evaluated the contract-stretch method of triceps surae stretching by measuring passive tension on the muscles. This may be an improved method of evaluating for increased length of the triceps surae over measuring range of motion, since it appears to be more objective. It is reproducible and unaffected by the subjects' desire to demonstrate progress from their flexibility program. Range of motion can also be influenced by a raised muscle pain threshold. The method of contract-stretch evaluation began with eight seconds of maximum contraction of the plantar flexors, followed by two seconds of relaxation, and — finally — eight seconds of slow stretch with the knee flexed. Straight knee stretching was not evaluated. The stretching procedure had a short-term effect lasting for at least 90 minutes after stretching, and a long-term effect lasting for three weeks after twice daily stretching. This method of stretching muscle is effective, regardless of whether the person has considerable motion restriction or good flexibility. Stretching may remove all the symptoms caused by the shortness. Logan notes that it is not uncommon for symptoms as far away as in the mid-thoracic spine to improve after stretching of the triceps

Triceps Surae Stretch

Your doctor has determined that it is necessary for you to lengthen the calf muscles in your lower leg. The purpose for the stretching exercise is to increase flexibility in your ankles. Increased flexibility will permit better function of your feet. You will receive the greatest benefits if you follow these directions exactly as given.

Position:

1. Remove your shoes and socks.
2. Stand 2 ½ to 3 feet from a wall.
3. Place your hands on the wall.
4. Point your toes inward (pigeon-toe).
5. Place your weight on the outer edges of your feet.

Step 1

1. Contract the muscles on the bottom of your feet by curling your toes as if you were trying to pick up the carpet with your toes.

Step 2

1. Contract your calf muscles by pushing down on the floor with your forefoot as if you were going to stand up on your toes, but do not let your heels come off the floor. This is an isometric contraction of your calf muscles, which means "muscle contraction without movement."
2. Hold this contraction for eight seconds.
3. Relax for a second, but continue leaning against the wall to be ready for the next step.

Step 3

1. Lean forward toward the wall, keeping your back and pelvis straight until you feel significant stretching of your calf muscles and behind your knees.
2. Hold this stretch for eight seconds.
3. Push back from the wall and rest for five seconds.
4. Do not bounce to increase the stretching action. Research has shown the procedure described will give the best and quickest lengthening of your muscles.
5. Repeat steps 1-3 five times.
   Repeat the series ___ times daily.

The average person will gain 10° of increased ankle movement (dorsiflexion) in one month by doing these procedures, if done properly. Once adequate range of motion has been gained, you can maintain it with one series of stretching exercises per week.

In the clinic we offer patients the instructions above to specifically achieve the triceps surae lengthening.

surae. **(Logan, 1995)**

The passive tension method of evaluating the contract-stretch procedure provides information about the muscles throughout the joint's range of motion, i.e., the amount of tension on the muscle halfway through the motion can be compared. Since tension was reduced throughout range of motion, it can be seen that the muscle was affected at the lengths used during walking and running.

Travell & Simons and Janse et al. **(Travell & Simons, 1992; Janse, 1947)** describe a seated method for stretching the triceps surae and exercising the intrinsic plantar muscles. This is done by active muscle contraction to obtain maximum ankle dorsiflexion and toe flexion. This position is held with maximum contraction for about ten seconds and then relaxed. It is repeated 10-20 times with each foot. Each contraction can have a slightly varying amount of forefoot adduction or abduction. This method enables one to exercise while doing some other activity, such as working at a desk. Additionally, it exercises the dorsiflexor muscles, which are underdeveloped in many runners.

The soleus can be stretched by having the patient stand with his feet slightly toed-in and close together. The patient then assumes a squat position without raising his heels from the floor. As the buttocks approximate the calf of the legs, a strong pull in the Achilles tendon should be felt. Hold the squatting position 10-15 seconds and repeat six times. **(Greenawalt, 1988)** This procedure does not stretch the gastrocnemius because it is shortened with knee flexion.

Foot rehabilitation with yoga

The classic yoga asana downward facing dog achieves this kind of triceps surae lengthening with elegance and safety. A growing body of western scientific evidence indicates that yoga practice is associated with improvements in overall physical fitness, **(Cowen & Adams, 2005)** along with increases in muscular strength, endurance, and flexibility. **(Tran et al., 2001)** – each of these outcomes are consistent with the AK approach.

This is probably the most widely taught forward bending posture in hatha yoga. In its ideal form the downward dog asana takes on the shape of an upside-down V, with only the hands and feet touching the floor. The hips are flexed sharply, the ankles are flexed 45 degrees, and the lumbar lordosis is kept intact. The pose enables the gastrocnemius and soleus muscles, as well as the ankle joints, to increase their flexibility, counteracting lifelong habits for functioning within limited lengths and ranges of motion.

Besides the downward facing dog, the Standing Lunge is one of the best practices for correcting an incapacity for flexion at the ankle.

The experience of lengthening the entire posterior myofascial channels of the body is impressively felt in the forward bend. Entering and exiting the forward bend from the hips permits the experience of lengthening the entire posterior body that envelops you from head to toe. Each inhalation releases the stretch slightly, and each exhalation lowers the head further down increasing the myofascial lengthening.

Logan **(Logan, 1995)** reports on another effective way

Downward Facing Dog
Anusara yoga instructor MG Ballantyne
(with kind permission)

to stretch the triceps surae is by using a slant board.

> "At home the patient may stand on the board 10 to 15 times per day. Each time the patient should spend only 1 to 2 minutes, attempting to stand up as straight as possible. This allows a gradual elongation of the muscles. Usually, 10 to 14 days of home stretching on the slant board will restore full range of motion in most patients."

When the stretching program results in poor dorsiflexion increase, one should suspect formation of osteophytes that can develop on the anterior aspect of the talar neck. A similar lesion can develop on the tibialis anterior. (Hontas et al., 1986) These lesions are more apt to develop in athletes whose major role is running and jumping. Along with the decreased dorsiflexion, there is usually

Standing Lunge

Forward bend

tenderness to digital pressure at the anterior ankle. Lateral x-ray of the ankle provides the definitive diagnosis. The joint itself is usually free of degenerative changes. "This condition can easily be treated by surgical excision of the offending spur. There is usually not any problem with returning to previous activity levels." **(Hontas et al., 1986)**

Thigh and spinal muscles. When one is rehabilitating from foot injury or dysfunction, the muscles of the extremities and spine must be considered. Disuse, abuse, misuse, postural change, or trauma may cause muscle shortening when there is distal dysfunction. **(Nicholas & Marino, 1987)** Altered feedback from sensory receptors in the feet and ankle alters gait, strength, and patterns of muscle activation throughout the body above. **(Nurse & Nigg, 2001; Freeman & Wyke, 1967)** Patients with supinated or pronated feet exhibit poor postural control compared to people with neutral feet. **(Tsai et al., 2006)** Any shortening or contraction – usually producing the tightness-weakness pattern on manual muscle testing described by Janda **(Janda, 1993)** -- must be eliminated during rehabilitation. When returning to activity, tight muscles may improperly transfer the load onto the weight-bearing foot; this can lead to sprains, strains, or stress fractures, especially in athletes. Tight quadriceps may lead to increased patellofemoral joint compression and resist free patella excursion distally. **(Sprieser, 2003; Duffy, 1999; Zatkin, 1990)** Tight iliotibial bands may cause patellar and/or trochanteric pain; they can lead to iliotibial band tendinitis, especially in distance runners. **(Leaf, 2003)** Chinn, Nicholas, Schafer and co-workers **(Chinn & Hertel, 2010; Nicholas & Marino, 1987; Schafer, 1986)** routinely use a number of tests for flexibility: the Thomas test for hip flexors, Ober test for iliotibial band, Ely test for quadriceps, and the sit-and-reach for lumbodorsal fascia and hamstrings.

Plantar muscle exercise. The plantar muscles are often atrophied in foot dysfunction, especially when tarsal tunnel syndrome causes poor nerve control of the muscles. Loss of strength in the flexor hallucis brevis muscle may lead to an unstable first ray. Consequently, the movement of the foot into pronation and the effectiveness of the windless mechanism of Hicks **(Hicks, 1951)** can be compromised if the first ray becomes unstable and lifts during load bearing. This is particularly evident in cases of functional hallux limitus as discussed previously.

Explain to the patient the difference between the intrinsic and extrinsic flexor muscles, how flexion of the base of his toes is done by the muscles that need exercising, and how flexion of the distal joints is done by the muscles that do not need exercising.

After correcting the tarsal tunnel syndrome **(discussed in Chapter 3)** and other foot conditions, teach the patient to contract the intrinsic foot muscles by flexing at the metatarsophalangeal articulations with minimal or no flexing of the interphalangeal articulations. For most patients this will be difficult at first, but with a little practice it can be easily accomplished.

There are two exercises to strengthen the plantar muscle when the patient can easily control them. Picking up marbles from the floor by flexing the toes will exercise the plantar muscles and help develop mobility of the forefoot. Have the patient pick up a marble from a pile, rotate the foot to its limit, and put the marble down. Turn the foot back as far as it will go in the direction of the original pile, and pick up another marble to put in the second pile. With practice the patient will be able to pick up a marble with any toe, gaining great dexterity in his feet. Make certain the patient avoids using only the distal interphalangeal joints of his toes; rather, he should use the entire toe, with emphasis on bending at the metatarsophalangeal articulation.

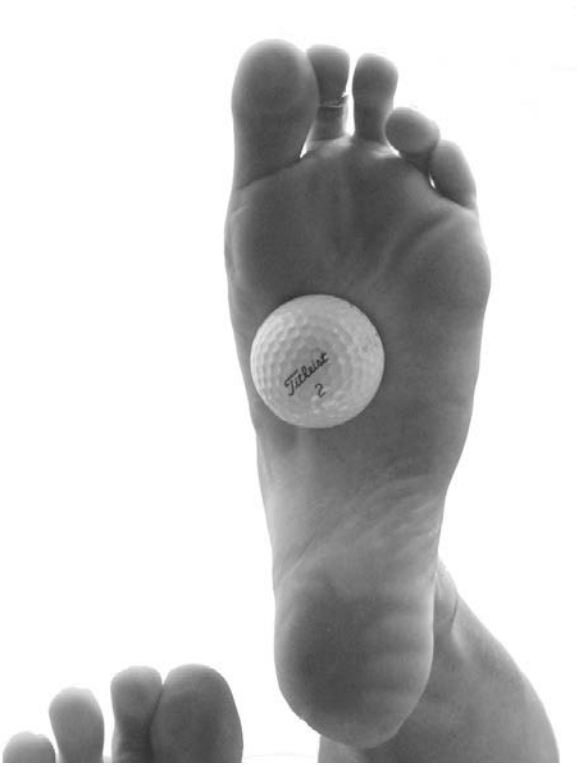
Another exercise that requires muscle control to

flex at the metatarsophalangeal joint is towel gathering. **(Gangemi, 2011; Crawford & Thomson, 2003; Maffetone, 2003)** Put a hand towel on a hard surface floor and have your seated patient put his foot on one end of the towel. The towel is gathered beneath his foot with toe action. Again, it is necessary for the toe action to be primarily at the metatarsophalangeal articulations. Encourage the patient to relax his calf muscles and concentrate on contracting the muscles in the bottom of his foot.

Toe-rising exercises can also strengthen the intrinsic muscles, but they have the disadvantage in some cases of also exercising the plantar-flexing muscles. This may be a problem when the triceps surae needs stretching, as in some runners who have overdeveloped the calf muscles in comparison to the dorsiflexor muscles. **(Logan, 1995)** Toe rising can be done by simply putting the forefoot on a raised area, such as a stair step, and raising up on the toes. The patient drops his heel down as far as possible, and again rises up on his toes. This exercise is contraindicated if there is anterior displacement of the metatarsal fat pad, and in some other cases of metatarsalgia.

Obtaining foot flexibility with a golf ball. Normally the foot is very flexible and has no painful areas. The foot that has been functioning abnormally for a prolonged period will have many tight and painful areas. The best way to regain motion in these areas is to work the foot on a golf ball. The extremely painful areas are the ones needing the most work.

Have your patient roll her foot back and forth over a golf ball, then from side to side. The uncomfortable areas give indication of where to concentrate the pressure. As the patient's foot loosens and gains mobility, have her apply more pressure on the golf ball until she reaches the point where she can stand and put considerable pressure on the ball. If the ball tends to be lost from under the foot and scoots across the floor, it can be put inside a Mason jar ring for containment.



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