Ankle Inversion Injury and Hypermobility: Effect on Hip and Ankle Muscle Electromyography Onset Latency

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ABSTRACT. Beckman SM, Buchanan TS. Ankle inversion injury and hypermobility: effect on hip and ankle muscle electromyography onset latency. Arch Phys Med Rehabil 1995;76:1138-43.

Objective: Changes in reflexes associated with chronically sprained ankles were examined by measuring the reflex response latency of hip and ankle muscles during instantaneous ankle/foot inversion.

Design: Randomized trials.

Setting: All studies were performed in the Research Department laboratories at a major rehabilitation center in a large metropolitan area.

Patients and Other Participants: Twenty subjects were assigned to 2 groups (normal and hypermobile) based on goniometry testing. Subjects were recruited from hospital and University staff and had a mean age of 31 ± 5 years.

Outcome Measures: Subjects stood on a platform constructed such that either foot/ankle could be instantaneously inverted. Latency was measured by EMG surface electrodes placed over the right and left gluteus medius and peroneal muscles. Two-factor analysis of variance was calculated to determine significant muscle onset latency differences ($p < .01$) between groups.

Results: Significant EMG latency differences were found in comparing right gluteus medius of the hypermobile group (127.35 ± 6.02msec) with the normal group (150.49 ± 6.49msec) during right ankle perturbation, and the left gluteus medius of the hypermobile group (120.71 ± 6.16msec) with the normal group (136.24 ± 5.88msec) during left ankle perturbation.

Conclusions: These data suggest that there is decreased latency of hip muscle activation after ankle inversion in the hypermobile population. In treating ankle instability, clinicians must decide to address the altered hip muscle recruitment pattern or accept this recruitment pattern as an injury-adaptive strategy and thus accept unknown long-term consequences of premature muscle activation (ie, possible articular predisposition to degenerative changes, altered joint reaction forces, and muscle imbalances).

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THE ANKLE COMPLEX is the most frequently injured joint both in athletics and in daily life. The ankle sprain is the most frequent sports injury.[1] Ten percent to 25% of injuries in athletics involve the ankle and 85% of these injuries involve sprain of the lateral ligaments.[2-4] Up to 30% of the population who sustain injury to the lateral ligaments of the ankle complain of residual dysfunction 9 months after the injury.[5] As many as 10% to 30% of people with acute lateral ligament injuries develop chronic mechanical instability.[6]

Common injuries such as the ankle sprain may be perceived as noncomplicated, local events; consequential dysfunction either at the site of injury or beyond the site of injury is often not considered.

After inversion ankle sprain, there are obvious changes in the anatomic structure often characterized by local hemorrhage and edema. Studies, however, also have noted changes not evident to casual observation.

Lemont[7] discussed the occurrence of perineural fibrosis of the superficial peroneal nerve in a patient with an inversion ankle sprain. Nitz and colleagues[8] studied subjects with grade II and III ankle sprains to determine the distribution of nerve injury, if any, in these populations. Electromyography (EMG) performed 2 weeks after injury showed that 17% of subjects with grade II sprains had mild peroneal nerve injury and 10% had mild tibial nerve injury. Eighty-six percent of the subjects with a grade III sprain injured their peroneal nerve, whereas 83% injured their posterior tibial nerve. The authors concluded that a high percentage of patients with a grade III ankle sprain sustain a significant nerve injury and their rehabilitation time is prolonged.

Konradsen and Ravn[9] compared individuals with no history of ankle inversion injury with subjects who had functional ankle instability. "Functional instability" was used to describe the condition of recurrent ankle sprain or sensation of joint weakness. Electromyographic activity of the peroneal muscles was recorded during sudden ankle inversion with the subject standing. The functionally unstable subjects displayed an increased peroneal reaction time.

The effects of ankle injury are not restricted to the ankle itself. Changes in postural sway and hip abductor muscle strength after inversion ankle sprain suggest that the consequences of ankle injury are manifest at structures proximal to the ankle in addition to local structures.

Cornwall and Murrell[10] studied single-limb sway of individuals with a history of unilateral inversion ankle sprain and found that their postural sway amplitude was significantly greater than in the noninjured control group. These changes were noted up to two years after injury. The authors suggest that these changes may be attributed to ankle joint laxity or to deafferentation of the nerves that innervate the joint capsule and ligaments.

In a similar study, Konradsen and Ravn[11] cited postural reactions to inversion ankle perturbation, including a peripheral reflex action (the peronei contraction) and a centrally elicited pattern consisting of flexion of the hip, knee, and ankle relieving the vertical pressure on the ankle and producing ankle eversion. They reported no defect in how the functionally unstable subjects centrally processed the afferent information.

Nicholas and colleagues[12] investigated torque-generating capacity of lower limb muscles after inversion ankle sprain and found diminished values when testing ipsilateral hip abduction. This weakness noted with isokinetic testing suggests that inversion ankle injury may encompass more than an isolated, singular joint pathology.

These previous studies suggest functional change of more proximal structures after inversion ankle injury. There are, however, no studies of hip muscle reflex latency as a result of either ankle injury or ankle hypermobility.

The purpose of this study was to investigate latency of hip muscular response (ipsilateral and contralateral gluteus medius) as measured by EMG during instantaneous ankle inversion.

METHODS

Experiments were performed on 10 subjects with symmetrical right and left ankle/foot mobility (normal) and 10 subjects with a minimum of 10° of excessive right ankle/foot inversion and a history of at least one inversion ankle sprain in the last 2 years (hypermobile). Each group had 5 men and 5 women. The mean age of the normal group subjects was 29.8 ± 3.6 years; the mean age in the hypermobile group was 32.3 ± 5.7 years. One subject had a history of slight hearing loss; no other neurological or vestibular deficits were reported by the 20 subjects. Nineteen subjects were right upper and lower extremity dominant; one person in the normal group was left upper and lower extremity dominant.

Before testing, both groups were not actively involved in a rehabilitation program and were not under medical care. By self report, the normal group subjects had no history of ankle inversion sprain and no history of lower extremity pathology, including fracture, sprain, or arthritis. Ankle passive and active range of inversion, eversion, plantarflexion, and dorsiflexion motion was painless. No subject had observable ankle edema. Palpation over the calcaneofibular and anterior talofibular ligaments was painless. Passive ankle inversion range of motion was determined by goniometry. A maximum of 3 degrees difference between the right and left ankles was allowed in the normal group. There was no other significant lower extremity pathology (defined as an injury requiring medical attention) in either group, and no known neurological deficits or vestibular deficits. Subjects were questioned about their dominant side. The dominant upper extremity was defined as the extremity with which the subject writes. The dominant lower extremity was defined as the extremity with which the subject would normally kick a ball. Subjects were between 24 and 41 years of age to minimize reflex latency variance due to age.

Measurement

Passive range of ankle/foot inversion motion was measured by goniometry with the subject sitting.[13] Latency was measured by EMG surface electrodes placed on the skin over the right and left gluteus medius and peronei muscles. Two recording surface electrodes were placed 2cm apart longitudinally over the mid–gluteus medius muscle belly, located at the midpoint between the greater trochanter and the iliac crest. Two recording surface electrodes were placed longitudinally over the mid–peronei muscle belly located just proximal to the midfibular level. Manual muscle resistance was applied before testing to ensure there was no "cross talk" between the studied muscles. A grounded electrode was placed over the ulnar styloid process.

A standardized list of instructions was read to each subject before testing. The subjects stood barefooted on a stationary platform constructed such that either foot/ankle could be spontaneously inverted independent of the opposite foot/ankle (fig 1). Subjects were not told in advance which foot/ankle would be tested, and were not told when the perturbation would occur. The perturbations were administered on variable intervals, each greater than 1sec to prevent neuromuscular habituation.

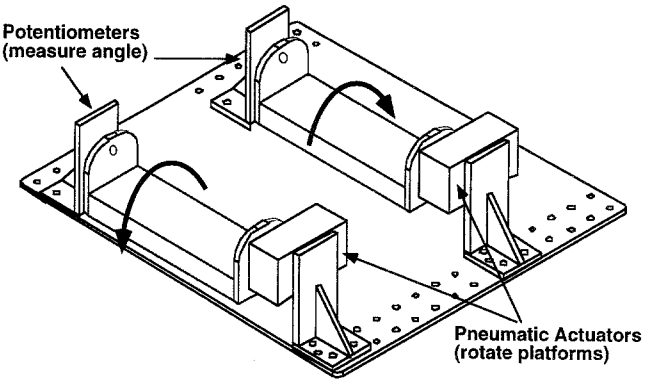

Fig 1. Device used to apply the perturbations. Subjects stand with each foot on a rotatable platform. Pneumatic actuators on the foot platforms allow them to be rapidly rotated to provide a 30° inversion motion. The actuators are triggered via a transistor-transistor logic (TTL) pulse from a computer. Ankle position was determined from potentiometers.

The ankle was inverted 30° at 700°/sec about an axis located near the subtalar joint and midtarsal joint axes.[14] Ankle/foot inversion occurred by computer activation of an actuator driven by a pneumatic impulse resulting in rapid displacement of the support surface. Twenty trials were randomly performed to each lower extremity.

Instrumentation

The 2 electrodes from each muscle were connected to a differential preamplifier/filter with a gain of 1,000 and two pole high-pass and low-pass filters with cutoff frequencies of 30 and 10,000Hz, respectively. The signal was then further conditioned using a Frequency Devices amplifier/filter model 9064,[a] which amplified with a gain between 8 and 30 and lowpass filtered (8-pole Butterworth) at a cutoff frequency of 250Hz. The EMG signals, along with the ankle angle signals as measured from potentiometers on the device, were then sent to a Macintosh computer[b] via an A/D converter, where the signals were sampled at a rate of 1,000Hz and stored on disk. The EMGs were rectified off-line.

The EMG latency was algorithmically determined as described in Kim and associates.[15] This method begins by finding the time of peak EMG activity. Then the burst onset is identified by moving backwards in time and stopping when 3 consecutive data points within 3 standard deviations from the baseline activity are found. (Baseline activity is taken from the EMG signal in the 100msec prior to movement.) The use of 3 standard deviations and 3 data points is sufficient to identify the true reflex onset and not just random activity. It is also less subjective than visual inspection.

RESULTS

Two factor analysis of variance (ANOVA) (factor 1: subject; factor 2: trial) was calculated to determine if the right and left gluteus medius mean onset latencies of the pathological group were significantly different from the right and left gluteus medius mean onset latencies of the normal group during right and

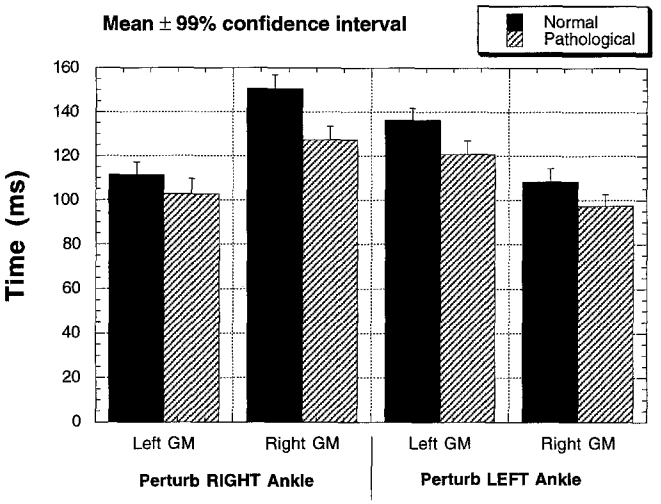

Fig 2. Comparison of EMG onset latencies for the gluteus medius muscles from the normal and pathological groups. The right ankles of the pathological groups were hypermobile. Means for all subjects and 99% confidence intervals are shown. All differences shown were statistically significant ($p < .01$).

left side testing. These statistical tests found that the pathological group mean values were significantly less than the normal group mean values ($p < .01$). Mean latencies of the pathological group muscles including the right gluteus medius during right side inversion perturbation, the left gluteus medius during right side inversion perturbation, the right gluteus medius during left inversion perturbation, and the left gluteus medius during left inversion perturbation were less than respective mean latencies of the normal group (fig 2).

In summary, subjects with right ankle hypermobility generally showed shorter gluteus medius EMG onsets after perturbations than unimpaired subjects. This was most pronounced for the gluteus medius ipsilateral to the perturbed hypermobile ankle (ie, the right gluteus medius after right perturbations).

Investigation to ascertain whether there is a recruitment pattern of ipsilateral and contralateral gluteus medius muscles during ankle/foot inversion perturbation was performed. Two factor ANOVA was used to determine if there was a significant difference between means of the ipsilateral and contralateral gluteus medius muscle onset latencies in both the normal and hypermobile groups. In both the normal and pathological groups, contralateral gluteus medius mean onset latency was significantly less ($p < .01$) than the ipsilateral gluteus medius mean onset latency when the right and left sides were tested (fig 3).

To clarify the mechanical events that occurred during testing, pelvic displacement curves resulting from ankle inversion were attained for one subject using an Optrotrak motion analysis system.[c] Inspection of these curves found that the pelvis on the ipsilateral and contralateral side displaced in a superior direction during unilateral testing. The pelvis on the side of perturbation elevated by 15 to 20mm and the opposite side remained within 4mm of the original position. This suggests that a greater amount of stretch is introduced to the gluteus medius on the side of perturbation (fig 4).

The latencies of the right (injury-side) peroneal muscles were also examined. A two factor ANOVA was used to determine if the mean onset latency of the pathological group was significantly greater than the mean onset latency of the normal group. The mean peroneal muscle onset latencies of the pathological and normal group were not significantly different. Consequently, peroneal latencies are similar when comparing right and left ankle perturbation, regardless of whether testing a normal or pathological subject (fig 5). However, like the gluteal muscles, the onset latencies of the peroneal muscles were significantly shorter after perturbations of the contralateral side ($p < .01$). Additionally, the peroneus muscles demonstrated considerably shorter latencies than gluteal muscles.

DISCUSSION

Hip and ankle muscle latencies of activation were investigated in response to ankle/foot inversion perturbation. Subjects with symmetrical ankle/foot inversion range of motion and subjects with right ankle/foot inversion hypermobility were tested. The results indicate that persons with unilateral ankle inversion hypermobility recruit hip muscles prematurely in response to right or left ankle inversion when compared with persons with symmetrical ankle mobility.

The results also showed a hip muscle recruitment pattern in response to ankle inversion, as the contralateral gluteus medius muscle activates first, followed by the ipsilateral gluteus medius muscle. There was no evidence that ankle hypermobility causes delays in peroneal muscle activation.

The gluteal activation results are discussed from the perspective of three models: (1) an articular neurology model, (2) a hip strategy model, and (3) a central set model.

Articular Neurology Model

Because greater amounts of motion are available at the hypermobile joint complex, activation thresholds of stretch-sensitive periarticular facilitory nerve receptors may never be met due to increased joint laxity. If receptor activation is delayed because of the ankle hypermobility, polysynaptic reflexes originating from the articular receptors and destined for hip muscles would also be delayed.

However, mean gluteus medius onset latencies of the pathological group were less than the normal group. Decreased activation latencies of the right gluteus medius during right ankle inversion and the left gluteus medius during left ankle inversion

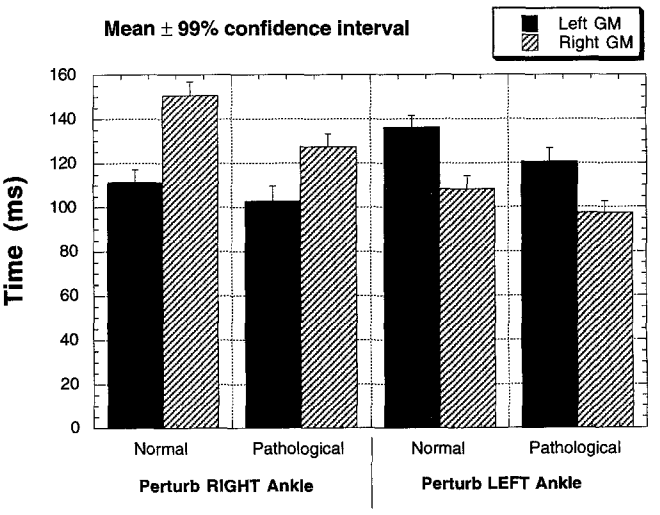

Fig 3. Comparison of EMG onset latencies for the right and left gluteus medius muscles from both the normal and pathological groups. The right ankles of the pathological groups were hypermobile. Means for all subjects and 99% confidence intervals are shown. All differences shown were statistically significant ($p < .01$).

were observed. Consequently, the hypothesis representing the articular neurology model was not supported.

An additional explanation of observed results also involves the function of the joint mechanoreceptors. In this case, however, the joint receptors may play an inhibitory role. In the uninjured ankle these receptors may cause an inhibitory delay in the ipsilateral gluteus medius muscle in response to stretch reflex originating from ankle muscles. This would allow extra time for the contralateral gluteus medius to stabilize posture before the ipsilateral gluteus medius activated. Hypermobile ankles may lack this delay circuitry (or it may be much weaker) because of injury, and the absence of this inhibition may allow reflexes from ankle structures to activate ipsilateral hip muscles prematurely.

Hip Strategy Model

Lemont[7] and Nitz[8] have detailed ankle peripheral nerve deficits after ankle inversion injury. Cornwall and Murrell[10] have documented increased postural sway in subjects with a history of unilateral inversion ankle sprain and concluded that these changes were attributed to deafferentation of the nerves that innervate the joint capsules and ligaments.

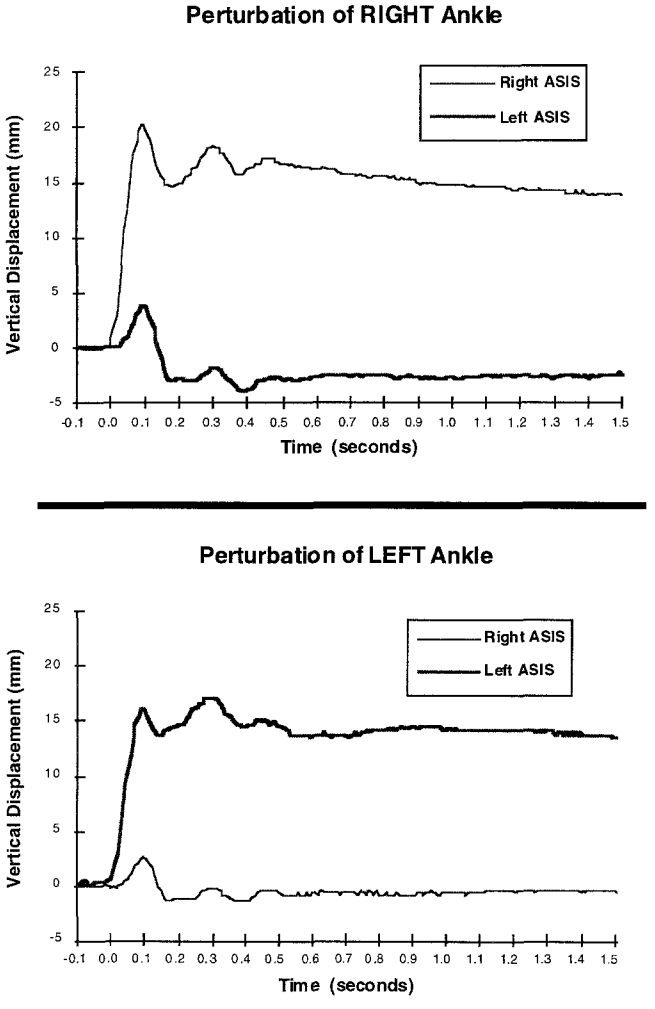

Fig 4. Pelvic displacement curves during unilateral ankle testing. Light-emitting diodes (LEDs) were placed over the left and right anterior superior iliac spine (ASIS) and their vertical position was tracked with an Optrotrak motion analysis system during an experiment. Left and right ankle perturbations began at time 0.

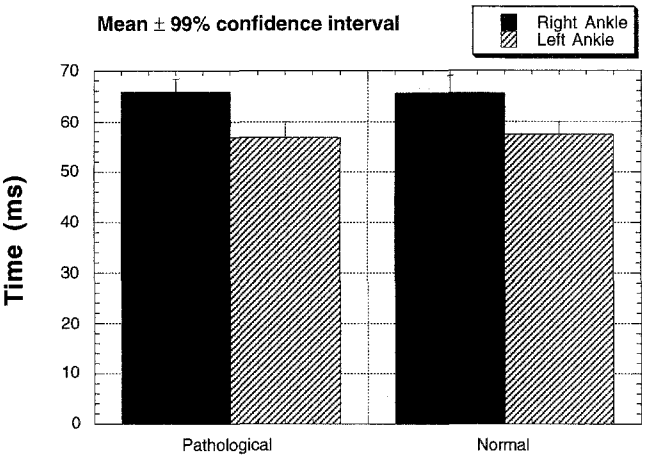

Fig 5. Comparison of EMG onset latencies for the right peroneal muscles from the normal and pathological groups. The right ankles of the pathological groups were hypermobile. Means for all subjects and 99% confidence intervals are shown. No statistically significant difference was observed between the normal and pathological groups, although differences in onset after right and left perturbations were significant ($p < .01$).

The pathological group was not tested for these impairments. Subjects with a history of chronic ankle sprain and hypermobility, however, are likely to have variable amounts of somatosensory loss at the ankle because of peripheral nerve injury and joint deafferentation.

Horak and colleagues[16] concluded that ankle somatosensory loss resulted in an increased hip strategy for postural correction. They noted that subjects demonstrated decreased hip muscle activation latencies when the ankle was perturbed using a horizontal platform displacement.

The conclusions of Horak are pertinent to this research as well. The decreased latency of hip muscle activation observed with the pathological group may represent the subject's attempt to recruit a hip strategy (hip movement altering the center of mass) to compensate for the partially deafferented ankle complex. The ankle may provide inadequate or altered sensory information regarding the perturbation itself or the bodies' position in space resulting from the perturbation. As a result, the nervous system may perceive that the ankle musculature is inadequate in compensating for the perturbation. This altered sensory information originating from the ankle complex may facilitate compensatory reactions at more proximal sites that are different from compensatory reactions resulting from normal sensory information.

Central Set Model

It is not clear whether the observed muscle EMG responses are influenced by supraspinal centers. Evidence indicates that a supraspinal-generated response is too slow to be the origin of postural reactions observed in this study.[17]

Through prior learning experiences (repeated ankle inversion injury) the central nervous system perceives the ankle as inadequate to compensate for high velocity ankle inversion. Through a feedforward mechanism, the central nervous system may increase sensitivity to this particular balance disturbance by providing enhanced gamma motor neuron activity to the appropriate hip musculature. As a result, select hip muscles are activated prematurely to compensate for an inadequate ankle response.

The models presented attempt to describe the possible events

leading to premature ipsilateral gluteus medius activation in the hypermobile group. Despite the early activation of this muscle, the normal contralateral/ipsilateral sequencing pattern of gluteus medius activation, established by this research, was preserved in the hypermobile group. The contralateral gluteus medius activates first, followed by the ipsilateral gluteus medius. Bilateral synergist muscle activation patterns have been documented in response to various means of ankle perturbation,[18] but hip muscle synergy patterns in response to ankle inversion balance disruption have not been previously reported.

Pelvic Displacement in Response to Ankle Inversion Experimental Testing

When examining pelvic displacement curves during unilateral ankle inversion, the pelvis on the ipsilateral and contralateral side was found to be displaced in a superior direction. The pelvis on the side of perturbation elevated more than the contralateral side. This indicates that a greater amount of stretch is introduced to the gluteus medius on the side of perturbation. Greater amplitude stretch should result in faster muscle activation of the ipsilateral gluteus medius.

The contralateral gluteus medius EMG, however, activated first when compared with the ipsilateral gluteus medius EMG despite greater pelvic displacement on the ipsilateral side. If greater amplitude stretch results in faster muscle activation, these results suggest that stretch reflex originating at the hip muscles does not mediate the muscular response of the contralateral gluteus medius. Rather, this response may represent a polysynaptic spinal reflex originating from the ankle mechanoreceptors or a supraspinal-influenced response as previously mentioned.

Peroneal Latency as a Function of Ankle Mobility

When comparing the data of the hypermobile right ankle group and the data of the normal group, no significant differences were found. This finding conflicts with data presented by Konradsen and Ravn,[9] who concluded that functional ankle instability resulted in increased peroneal reaction time in a similar population of subjects.

Differences in experimental protocol could explain conflicting findings. In the previously mentioned study, a trapdoor-type perturbation was used instead of the pneumatically driven perturbation used in our study. Konradsen and Ravn performed their statistical analysis by comparing the shortest reaction time of three trials performed on each lower extremity. Because there was latency variability between trials, the average of 20 trials was used in our research.

CONCLUSION

This study examined hip muscle recruitment latencies during ankle inversion. The EMG response in subjects with symmetrical and asymmetrical ankle/foot mobility was investigated. Hip muscle activation latency changes observed in the pathological group suggest there are changes in postural-compensatory strategies. Factors mediating altered muscle activation times remain unclear.

Some possible sources of the hip muscle response may be eliminated. Because the gluteal muscles contralateral to the perturbation are the first to be activated, we do not believe that the reflex timing is dominated by stretch reflex. Furthermore, the response is too fast to be generated from a supraspinal signal.

These findings lead to the conclusion that the gluteus medius response is activated via a polysynaptic reflex generated by ankle mechanoreceptors. However, when the ankle has been repetitively sprained to the point of causing joint hypermobility, a learned but subconscious lowering of hip muscle activation threshold ensues to respond to ankle inversion. This protective, steady-state threshold-lowering most likely is controlled by spinal-activated gamma motor neurons.

Clinicians whose patients have ankle dysfunction remain unsure whether to treat the ankle complex or the hip complex and whether to treat with the patient in a weight-bearing or non–weight-bearing position. Some clinicians may question whether intervention should occur at all, because the motor system may self-compensate for the ankle dysfunction.

The conclusions of Gauffin and Tropp[19] regarding movement patterns of the anterior cruciate ligament deficient knee population may be applicable when considering the clinical aspects of this ankle research. Finding altered knee positions when this group landed from a one-legged jump, these authors concluded that the subjects had established adaptive strategies to compensate for the ligamentous insufficiency. Consequently, Gauffin and Tropp advised against attempts to reteach normal landing knee positions.

In treating ankle instability, clinicians must decide to treat the altered hip muscle recruitment pattern as demonstrated by the hypermobile group or accept this recruitment pattern as an injury-adaptive strategy. The clinician who decides to accept the muscle recruitment pattern as an injury-adaptive strategy must also accept unknown long-term consequences of premature muscle activation (ie, possible articular predisposition to degenerative changes, altered joint reaction forces, and muscle imbalances).

To avoid the unknown consequences of premature muscle activation, it is more reasonable for the clinician to address the muscle recruitment strategy demonstrated by the hypermobile group. Therapeutic techniques should be employed that require ankle stability and hip muscle activation. These techniques include closed kinetic chain exercises that require concurrent isometric hip and ankle activation, progressing to exercises that require active ankle stability followed by reciprocal gluteus medius activation. These therapeutic measures combining ankle and hip muscle activation may prevent and/or correct abnormal muscle recruitment patterns while simultaneously enhancing ankle stability.

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Suppliers

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b. Macintosh; Apple Computer, 20525 Mariani Avenue, Cupertino, CA 95014.
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