Acute Orthotic Intervention Does Not Affect Muscular Response Times and Activation Patterns at the Knee

By: Holly M. Rose, Sandra J. Shultz, Brent L. Arnold, Bruce M. Gansneder, David H. Perrin

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Abstract:

Objective: To evaluate the short-term effect of a semirigid foot orthotic device on response times and activation patterns of knee musculature in individuals with hyperpronation after a lower extremity perturbation in a single-leg, weight-bearing stance.

Design and Setting: We used a lower extremity perturbation device designed to produce a forward and either internal or external rotation of the trunk and femur on the weight-bearing tibia to evoke a reflex response. Subjects were tested both with and without orthotic devices.

Subjects: Seventeen (13 male, 4 female) volunteers (age, 20.6 ± 1.8 years; height, 181.0 ± 8.1 cm; weight, 87.4 ± 19.5 kg; navicular drop, 12.1 ± 1.8 mm) with a navicular drop greater than 10 mm volunteered for this study.

Measurements: Long latency reflex times were recorded via surface electromyography for the medial and lateral hamstrings, gastrocnemius, and quadriceps muscles.

Results: A dependent-sample *t* test revealed a significant decrease in navicular drop with orthotic intervention ($P < .0001$). With that confirmed, separate repeated-measures analyses of variance with 2 within factors (orthotic condition and muscle) revealed no significant difference in muscle response time between orthotic and nonorthotic conditions for either internal or external rotation perturbation. Although we found a main effect for muscle for both internal ($P < .0001$) and external ($P < .0001$) rotation, indicating a preferred muscle activation order, this activation order did not differ between orthotic and nonorthotic conditions (internal rotation $P = .674$, external rotation $P = .829$).

Conclusions: Our findings suggest that a short-term application of a semirigid orthotic device does not alter muscle response times or activation patterns of the muscles that stabilize the knee. Further research is needed to determine whether changes in activation patterns may occur over time since mechanical adaptations occur with long-term wear.

Keywords: hyperpronation | subtalar pronation | electromyography | long latency reflex

Article:

Subtalar joint pronation has been implicated as a predisposing factor in a variety of lower leg injuries[1–7] and, in particular, noncontact anterior cruciate ligament injuries.[2,7] Although researchers have found significantly greater navicular drop values in anterior cruciate ligament–injured subjects versus nonmatched controls,[2,7,8]investigators to date have failed to explore the mechanisms by which this static postural fault may influence dynamic joint control and injury. However, because the anterior cruciate ligament injury mechanism typically involves some combination of valgus, extension, or rotary moments,[9] alignment faults that add to valgus or rotary stress at the knee during functional activity may increase the risk of anterior cruciate ligament injury from these mechanisms.[8]

Research indicates that hyperpronation significantly alters knee joint rotation, laxity, and biomechanical function.[1–3,8,10–12] When hyperpronation occurs, the talus plantar flexes and adducts on the calcaneus,[13]causing the tibia to follow the talus medially, resulting in a compensatory increase in internal tibial rotation.[2,3,5,7,10] This increase in internal tibial rotation is thought to increase joint laxity and create a preloading, rotary stress to the knee joint during weight-bearing activities, particularly when the pelvis is rotating externally.[1–4,14] These changes in laxity and biomechanical function may adversely influence proprioceptive orientation or feedback from the hip and knee, thus altering neuromuscular reflex behaviors and joint stabilization.[8,11] Further, the mechanical efficiency and relative contribution of a muscle to knee joint stabilization are likely affected if the muscle's orientation or length-tension relationship is sufficiently altered.[15,16] Others suggest that compensatory muscle activity, both proximal[17] and distal[17,18] to the knee, may occur to control for decreased foot stability and increased internal tibial rotation with excessive pronation, leading to muscular fatigue and overuse. Given this theoretic framework, neuromuscular function and control of knee stability may be substantially affected in athletes with lower extremity malalignments, suggesting an increased risk of musculoskeletal stress and injury.

Efficient neuromuscular control is essential for dynamic joint stiffening and knee joint protection.[19–27]Forces imposed on the knee joint during sport activity are often greater than the capacity of the ligament, requiring active muscle forces to adequately stabilize the joint. Hence, muscles serve as the primary active stabilizers of the knee during functional loading conditions, protecting against ligament injury.[23,26]Neuromuscular factors that contribute to functional knee stability include preparatory muscle stiffness,[28–30]reflexive muscular activation (reflex latency and electromechanical delay),[16,22,31–34] muscle coactivity,[19,21,23–25,35] and order of muscle recruitment.[35–37] Any factor that delays or inhibits one or more of these neuromuscular factors will likely compromise neuromuscular control of knee stability and, thus, the maximal load the knee can withstand.

Few researchers to date have evaluated reactive neuromuscular response and recruitment patterns at the knee joint in a functional environment.[35,38–40] However, factors such as muscle preactivity, weight-bearing status, and trunk position can significantly influence the timing, extent, and relative contribution of a particular muscle to knee joint stabilization.[21,25,41] Using a functional, weight-bearing, lower extremity perturbation model, Shultz et al[42] confirmed that neuromuscular activation patterns are, in fact, quite different from those previously reported using non–weight-bearing, partial weight-bearing, or uniplanar perturbation models. Moreover, evaluating response characteristics in a functional, weight-bearing stance provides an avenue by which to investigate

the influence that lower extremity limb malalignments or their corrections may have on joint stress and reactive neuromuscular control of knee stability.

Traditionally, clinicians have sought to reduce lower extremity stress and to correct lower extremity malalignments, particularly those associated with hyperpronation, with the use of orthotic devices.[43] Many researchers have investigated the short-term[3,44–48] and long-term[1,43,49] effectiveness of an orthotic foot device in modifying pronation and lower leg alignment and mechanics. In the short term, orthotic devices have been reported to maintain the hyperpronated foot in a more neutral position,[3,46] decrease the amount of transverse tibial rotation,[3,50] and decrease the standing quadriceps angle (Q angle).[47] However, these findings are not without controversy, since other investigators have shown little or no change in pronation with orthotic devices.[48,51]

Whether changes in lower leg alignment and mechanics with orthotic intervention are sufficient to influence muscular timing or activation patterns, and thus neuromuscular control of knee stability, has received little attention to date. Only a few studies have investigated the influence of orthotic devices on muscle activity,[17,18,52] with only one study[17] assessing muscle activity both proximal and distal to the knee joint. Further, the focus of these studies has been solely on the level of activation, and no measures of reactive muscular timing characteristics were assessed. Tomaro and Burdett[18] appear to be the first to have studied the effects of orthotic devices on muscle activity. Specifically, they evaluated the effect of a semirigid orthotic device on electromyographic activity of the tibialis anterior, peroneus longus, and gastrocnemius muscles during treadmill walking. Although they found no difference in the average electromyographic activity of these muscles between conditions, they noted a significantly longer duration of tibialis anterior activity after heel strike with orthotic wear. However, their data were limited to muscles of the lower leg, and no inferences can be made to muscle activation patterns around the knee.

Although not specifically using an orthotic device, Hung and Gross[52] evaluated the effect of foot wedging on electromyographic activity of the vastus medialis obliquus and vastus lateralis muscles. In healthy subjects with normal foot alignments, they found no difference in normalized muscle activity of the vastus lateralis, vastus medialis obliquus, or vastus medialis obliquus:vastus lateralis ratio among flat surface, 10° medial wedge, and 10° lateral wedge foot positions. The authors conceded that these results were limited and could not be generalized to subjects with pronated or supinated feet. Further, their findings were limited to quadriceps muscle activity, and no conclusions can be drawn as to whether the change in foot position influences reactive muscular timing or whether similar changes would be noted in the hamstrings or gastrocnemius muscles.

Nawoczenski and Ludewig[17] performed the only study we found that measured changes in muscle activity with orthotic devices in muscles both proximal and distal to the knee joint. They measured electromyographic amplitude in the tibialis anterior, medial gastrocnemius, vastus medialis, vastus lateralis, and biceps femoris muscles in recreational runners, with and without orthotic devices, during the first 50% of stance phase. Biceps femoris activity decreased and tibialis anterior activity increased with orthotic devices. Based on their previous work evaluating 3-dimensional kinematics in the same subjects,[50] the authors attributed the decrease in biceps

femoris activity to a reduced need to control internal tibial rotation when orthotic devices were worn.[17] Whether similar changes would be noted in the medial hamstrings is unknown.

We found no published studies that directly evaluated the influence of orthotic intervention on protective neuromuscular response characteristics at the knee with sudden joint loading. Whether orthotic intervention in hyperpronating individuals has any impact on reactive neuromuscular responses around the knee may be important to our understanding of factors that influence neuromuscular control of knee joint stability and injury prevention. Given the role of the hamstrings in stabilizing the tibia and protecting the anterior cruciate ligament from mechanical strain, factors that influence the timing and adequacy of their response may have significant clinical implications for injury control. Considering the location of insertion of the medial hamstrings (MH) and lateral hamstrings (LH) on the proximal tibia, substantial changes in muscle response time and recruitment order may occur with reduction of compensatory internal rotation that results from orthotic control of excessive pronation. Therefore, our purpose was to evaluate the short-term effect of a semirigid foot orthotic device on muscle response times and recruitment order of the MH, LH, medial gastrocnemius (MG), lateral gastrocnemius (LG), medial quadriceps (MQ), and lateral quadriceps (LQ) muscles in hyperpronating individuals after a sudden, lower extremity perturbation in a single-leg, weight-bearing stance.

METHODS

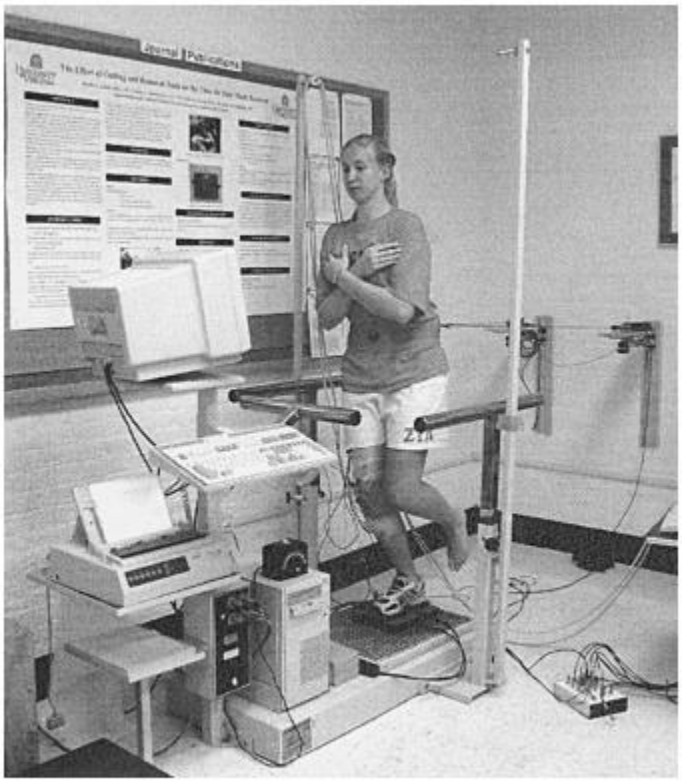
Subjects

Seventeen (13 male, 4 female) physically active volunteers (age, 20.6 ± 1.8 years; height, 181.0 ± 8.1 cm; weight, 87.4 ± 19.5 kg; navicular drop, 12.1 ± 1.8 mm; standing Q angle, 11.5 ± 5.5°) met the following inclusion criteria: (1) hyperpronation of the subtalar joint greater than 10 mm, as measured by navicular drop[53]; (2) no history of significant injury to the knee, ankle, or foot of the dominant leg within the last 6 months that may have affected neuromuscular responses at the knee; (3) no orthotic device use during the 6 months before the study; (4) no history of ligamentous surgery to the dominant extremity; (5) no grossly excessive knee valgus angulation (as screened by standing Q angle); and (6) otherwise healthy with no complaints of pain that would prevent them from being able to perform a single-leg squat. All subjects read and signed an informed consent form, approved by an institutional review board for the protection of human subjects, before participating in the study. The board also approved the study.

Instrumentation

To invoke the perturbation and reflexive response, we used a custom-designed, lower extremity perturbation device (LEPD, University of Virginia, Charlottesville, VA) to produce a forward and either internal rotation (IR) or external rotation (ER) moment at the knee with the subject in a single-leg, weight-bearing stance (Figure). The design, reliability, and validity of this device have been previously reported.[42] To record muscle activity, we placed bipolar Ag-AgCl surface electrodes measuring 10 mm in diameter, with a center-to-center distance of 2.5 cm, over the muscle bellies of the MQ, LQ, MH, LH, MG, and LG muscles. An 8-channel Noraxon Myosystem 2000 Surface Electromyogram (Noraxon, Scottsdale, AZ) measured the timing of muscular activation for each muscle in the dominant leg after perturbation. The specifications for

the surface electromyography unit included an amplification of 1 mV/V, a frequency bandwidth of 16 to 500 Hz, common mode rejection ratio of 114 dB, input resistance of 20 MΩ to 1 GΩ, and a sampling rate of 1000 Hz. We interfaced the electromyography and perturbation devices with Data Pac 2000 Lab Application Software (Run Technologies, Laguna Hills, CA) to acquire, store, and analyze the data. At the time of trigger release, a voltage signal was sent from the LEPD to the computer software to mark the time of stimulus and begin data recording. We recorded muscle activity from 100 milliseconds before to 500 milliseconds after the perturbation stimulus.



Lower extremity perturbation device. Testing was performed with the shoe only (no insole) and with the shoe plus orthotic device in place.

To standardize the subject's knee angle before perturbation, we attached an electrogoniometer (Penny and Giles, Santa Monica, CA) to the lateral aspect of the knee joint to consistently reproduce a knee-flexion angle between 30° and 35°. Trunk position relative to the knee was standardized across all subjects and trials with the Chattecx Balance System (Chattanooga Group, Inc, Hixson, TN) force platform by consistently placing the subject's center of pressure over the midfoot. Using the visual training target of the Chattecx, we positioned each subject's foot on the force platform so that when he or she assumed the test position, the center of pressure was located at the intersection of the horizontal and vertical midlines of the target.[42]

Procedures

We asked each subject to report to the laboratory on 2 separate occasions.

First Visit. During the first session, we recorded physical characteristics of age, height, weight, navicular drop, and standing Q angle and obtained an injury history. We measured navicular drop using a modification of the Brody method[53] by assessing the difference in navicular height between standing subtalar joint neutral and standing relaxed with the subject barefoot. A single investigator (H.M.R.) took all measurements, and we averaged 3 measurements to determine if subjects met the inclusion criteria. Test-retest intratester reliability of this measurement method was found to be excellent (intraclass correlation coefficient [3, k], 0.92; SEM, 0.85 mm).

We then took a foot mold for fitting of a custom, full-length, semirigid sport orthotic device according to the manufacturer's guidelines (Foot Management, Pittsville, MD). We obtained foot molds of each subject while seated, with the knee and hip flexed to 90°, the talus aligned directly under the knee, and the second ray of the foot in line with the tibia. Foot molds were taken with the foot positioned in subtalar neutral, and the orthotic device was fashioned by creating a negative impression of the mold. No additional posting was performed.

Second Visit. During the second session, we performed all perturbation trials for both orthotic and nonorthotic conditions. The subject wore the same, self-selected athletic shoe for both conditions. For the orthotic condition, the orthotic was placed in the participant's shoe with the insole removed. For the nonorthotic condition, the participant wore only the shoe with no insole. To determine the degree of correction the orthotic device provided each subject, we measured navicular drop as before, but with the subject standing barefoot on the orthotic device. We performed all testing on the dominant leg (ie, the leg used to kick a ball).

We prepared the skin and placed electrodes in a parallel arrangement over the midline of the muscle bellies of the MH, LH, MG, LG, MQ, and LQ muscles. We verified all electrode placements using manual muscle testing and checked for cross-talk from adjacent muscle groups. We secured an electrogoniometer to the lateral joint line and aligned the arms from the greater trochanter to the lateral femoral condyle and from the head of the fibula to the lateral malleolus. We then secured the electrodes and the electrogoniometer with an elastic bandage to prevent cable tensioning and reduce movement artifact.

We then positioned the subject in the LEPD according to the protocol reported by Shultz et al.[42] Subjects received a thorough explanation of testing procedures and were given at least 3 practice trials to become comfortable and acquainted with the task. With the subject standing on the dominant leg and in the testing position, either the left or right cable was released from the wall at random time intervals between 1 and 10 seconds, causing IR or ER of the trunk and femur on the weight-bearing tibia. Subjects were instructed to look straight forward and, on cable release, to attempt to hold their single-leg balance. For both orthotic and nonorthotic conditions (counterbalanced to control for order effect), subjects performed 5 trials each of IR and ER perturbations. The direction of rotational perturbation was randomized to minimize anticipatory responses. Subjects were given a 30-second rest period between trials and were instructed to shift their weight to the nontest leg to avoid fatigue.

Statistical Analysis

For each orthotic (orthotic or nonorthotic) and perturbation (IR or ER) condition, we used the average of the first 3 acceptable trials for data analysis. An acceptable trial was defined by the following criteria: (1) long latency reflex identified within 150 milliseconds after cable release; (2) baseline muscle activity sufficiently quiet and stable to ensure an acceptable signal-to-noise ratio; (3) readable signal obtained from all 6 muscle sites; and (4) signal free of movement artifact to allow clear interpretation of the signal.[42] We determined muscle response time as the time delay from the onset of the perturbation to the onset of the long latency reflex. We used a 2-SD threshold from baseline activity (recorded for 100 milliseconds before perturbation) to define the onset of muscle activity for the hamstrings and gastrocnemius and a 1-SD threshold for the quadriceps to improve onset sensitivity due to its greater baseline activity level.[42] To determine whether muscular response times and activation order differed significantly between orthotic and nonorthotic conditions for either IR or ER perturbation, we used 2 separate, repeated-measures analyses of variance with 2 within variables (orthotic condition at 2 levels [orthotic, nonorthotic] and muscle at 6 levels [MG, LG, MH, LH, MQ, LQ]). We performed all analyses using the SPSS Statistical Software Package, version 9.0 (SPSS Inc, Chicago, IL). The α value was set a priori at $P < .05$.

RESULTS

To confirm that a treatment effect occurred, we compared navicular drop values for each subject with and without the orthotic device in place. Mean navicular drop values changed from 12.18 ± 1.8 mm to 8.4 ± 1.7 mm. A dependent *t* test confirmed that the change in navicular drop was significant ($t = 7.07$, $P < .0001$).

The muscle response times for IR and ER perturbation are listed in Tables 1 and and 2, respectively. We found no significant differences in the overall muscle response times between orthotic and nonorthotic conditions for either IR (72.4 versus 75.3 milliseconds) ($F_{1,16} = 2.004$, $P = .176$, power = 0.265) or ER (74.1 versus 73.4 milliseconds) ($F_{1,16} = 0.154$, $P = .700$, power = 0.066). We found a significant main effect for muscle for both IR and ER ($P < .0001$), indicating a preferential muscle activation order. For ER, pairwise comparisons with Bonferroni correction revealed no difference in response times for the MG (59.4), LG (59.4), and MH (67.5), which fired first, followed by the LH (76.8) after a significant delay, then by the MQ (87.2) and LQ (92.4) after another delay. Activation order was similar for IR but without a significant delay between the firing of the LH and the LQ and MQ (ie, MG = LG = MH < LH = MQ = LQ). However, when we evaluated the orthotic-by-muscle interaction, this activation order did not differ between orthotic and nonorthotic conditions for either IR ($F_{5,80} = 0.426$, $P = .829$, power = 0.157) or ER ($F_{5,80} = 0.635$, $P = .674$, power = 0.220).

Table 1. Muscle Reaction Times (in Milliseconds) for Internal Rotation Perturbation

Group	Medial Gastrocnemius	Lateral Gastrocnemius	Medial Hamstrings	Lateral Hamstrings	Medial Quadriceps	Lateral Quadriceps
Orthotic	55.3 ± 9.0	55.4 ± 10.2	67.5 ± 13.3	81.6 ± 28.4	86.8 ± 16.9	88.0 ± 20.3
Nonorthotic	59.6 ± 7.4	55.9 ± 9.1	67.9 ± 12.7	84.8 ± 21.5	88.0 ± 21.9	95.7 ± 21.0
Total	57.4 ± 8.4	55.7 ± 9.5	67.7 ± 12.8	83.2 ± 24.8	87.4 ± 19.2	91.9 ± 20.7

Table 2. Muscle Reaction Times (in Milliseconds) for External Rotation Perturbation

Group	Medial Gastrocnemius	Lateral Gastrocnemius	Medial Hamstrings	Lateral Hamstrings	Medial Quadriceps	Lateral Quadriceps
Orthotic	59.2 ± 9.7	59.6 ± 11.1	68.8 ± 10.2	75.9 ± 17.8	86.6 ± 21.1	94.5 ± 23.4
Nonorthotic	59.7 ± 7.8	59.1 ± 11.0	66.2 ± 12.8	77.7 ± 17.6	87.7 ± 14.9	89.9 ± 15.2
Total	59.4 ± 8.7	59.4 ± 10.9	67.5 ± 11.5	76.8 ± 17.5	87.2 ± 18.0	92.2 ± 19.6

DISCUSSION

Given the fact that previous studies have demonstrated significant changes in lower leg kinematics[44,45,47,54]and muscle activity[17,18] with short-term (less than 4 weeks' accommodation) orthotic control of hyperpronation, we hypothesized that protective reflexes would similarly be influenced by orthotic intervention. However, our primary finding indicates that a short-term application of a semirigid orthotic device does not significantly affect muscle response times or activation order of the muscles that stabilize the knee after a functional, lower extremity perturbation.

Neuromuscular control of knee stability is provided through both intentional (preparatory) and reactive (reflexive) responses that are mediated by proprioceptive feed-forward and feedback mechanisms, respectively.[22,55,56] Muscle coactivity and recruitment patterns also contribute to knee stability.[19,21,23,35,37]Thus, factors that alter proprioceptive feedback or neuromuscular function under sudden loading conditions may influence neuromuscular control of knee stability and the maximal load the joint can withstand. Such factors may include joint configuration, forces created by muscles crossing the joints, and forces affected by weight-bearing status and joint position.[11] In fact, changes in limb position (ie, knee-flexion and hip-flexion angles) have been found to significantly alter muscle coactivity patterns and joint stability.[15,21,25,41]

Furthermore, limited evidence suggests that distal pathologic conditions may affect reflex activation patterns when the lower extremity is in a weight-bearing environment. Beckman and Buchanan[57] found changes in gluteus medius onset latency after an inversion ankle perturbation in subjects with chronic ankle sprains. Although we recognize that hyperpronation represents a very different condition than a chronic ankle sprain (ie, structural alignment versus neurologic deficit), significant changes in rotational and valgus stresses at the knee joint that result from lower leg malalignments (or their correction) may similarly alter protective neuromuscular activation patterns, and, thus, a muscle's contribution to knee stability. In the absence of any significant findings, we considered potential explanations for our results.

Statistical Power

We thought it was important to first explore whether our lack of significant findings was due to insufficient statistical power (ie, insufficient number of subjects evaluated) necessary to gain

meaningful results. As we noted, power values were quite low, ranging from 0.066 to 0.265. However, the question remains of whether this low power was due to a low number of subjects or low effect size (ie, magnitude of difference between means). We suspected that it was due to a low effect size and confirmed this by calculating the effect size for each muscle between orthotic and nonorthotic conditions (Table 3). With the exception of the MG (0.58) and LQ (0.37) for IR perturbation, effect sizes ranged from 0.02 to 0.20, which by convention indicate very small differences between the 2 conditions.[58,59] Hence, even if we were to add substantially more subjects to achieve statistically significant differences between conditions, the actual difference would not be of clinical importance.

Table 3. Effect Sizes for Each Muscle Between Orthotic and Nonorthotic Conditions

Muscle	Internal Rotation	External Rotation
Lateral quadriceps	0.37	0.07
Medial quadriceps	0.05	0.30
Lateral hamstrings	0.15	0.20
Medial hamstrings	0.02	0.10
Lateral gastrocnemius	0.05	0.09
Medial gastrocnemius	0.58	0.05

Treatment Effect

Although researchers have demonstrated changes in transverse tibial rotation when comparing orthotic with barefoot conditions,[3] we found no changes in reflex response times with the application of an orthotic device. Although we did not directly measure changes in tibial rotation with orthotic wear, it may be that the changes were not of sufficient magnitude to cause a change in muscle activation patterns, as suggested by our effect size. Unfortunately, we did not simultaneously measure changes in joint kinematics to be able to make this direct comparison. However, we did note only a modest change in navicular drop values with the orthotic intervention in some of our subjects (mean, 3.8 ± 2.2 mm; range, 1–8 mm) and believe it is reasonable to suspect that this minimal change likely did not result in substantial changes in transverse tibial rotation or lower extremity alignment. Hence, changes in joint stresses, proprioceptive feedback, or muscle length-tension relationships may not have been sufficient in these subjects to alter muscle response times to a lower extremity perturbation.

Although some investigators have demonstrated significant changes in subtalar position,[3,46] transverse tibial rotation[3,50] and standing Q angle[47] with short-term orthotic application, others have observed insignificant changes in pronation with the use of orthotic devices. Comparing barefoot, shoe, and shoe-with-orthotic conditions, Rodgers and Leveau[48] found no significant differences in maximum angular displacement in pronation, support time in pronation, or angular velocity of pronation. Similarly, Brown et al[51] observed no significant differences in maximum pronation, maximum pronation velocity, or calcaneal eversion between shoe and shoe-with-orthotic conditions. Although it is somewhat difficult to adequately compare these studies because of the variety of methodologic differences, variables studied, and type of orthotic device used, it is apparent that controversy exists as to how effective orthotic devices may be in altering subtalar position and lower extremity alignment.

To further explore the relationship between degree of correction and changes in muscle activation patterns, we performed a follow-up analysis by once again comparing the effect sizes between orthotic and nonorthotic conditions, but this time we only used the 5 subjects who showed a 5-mm or greater change in navicular drop with orthotic application (Table 4). In this selected sample, the effect sizes were substantially larger than those in our original sample (Table 3), particularly for the MQ and LQ (IR and ER), MG (IR and ER), and MH (IR). Although we realize this subgroup represents a very small sample and substantially more subjects would be needed to yield statistically significant findings, the increased magnitude of these effect sizes indicates that the degree of correction may influence muscle response times and, in particular, activation order. Further investigations should explore the magnitude of correction that is required to effect a change both kinematically and neuromuscularly.

Table 4. Effect Sizes Between Orthotic and Nonorthotic Conditions for Those With a Greater Than 5-mm Change in Navicular Drop

Muscle	Internal Rotation	External Rotation
Lateral quadriceps	0.51	0.39
Medial quadriceps	0.35	0.41
Lateral hamstrings	0.02	0.04
Medial hamstrings	0.88	0.03
Lateral gastrocnemius	0.34	0.07
Medial gastrocnemius	1.13	0.91

Short-Term Versus Long-Term Adaptations

Our findings are limited to short-term changes in neuromuscular response characteristics with orthotic intervention, since measures were taken immediately on application of the orthotic device. With continued wear, long-term adaptations in lower extremity mechanics may occur, resulting in alterations in activation patterns of the knee-stabilizing musculature over time. Because knee kinematics are improved with return to a more neutral foot alignment, proprioceptive feedback from the muscles and tendons surrounding the knee may change as joint loads are altered and the optimal length-tension relationship of the muscles is restored.[60–62] Because these changes may not occur immediately, neuromuscular adaptations may be manifested at some delay. Although the short-term versus long-term adaptive effects of orthotic devices on neuromuscular control have yet to be studied, others have recognized the significant contribution of muscle spindle receptors in modifying neuromuscular control strategies when joint kinematics are altered with anterior cruciate ligament injury and restored with reconstruction.[60–62] We recommend that further research explore changes in neuromuscular control over time with continued orthotic wear.

Interaction of Orthotic Device and Shoe

A potential confounding variable in this study is the interactive influence of the shoe with the orthotic device. Subjects were allowed to wear their own athletic shoes during testing, and we made no attempt to control shoe type (ie, court versus running shoe). Considering that subjects served as their own controls, we did not believe that this would be a significant confounding factor. We also believed this was a clinically relevant approach, since orthotic devices are

commonly placed in the shoes the athlete or patient provides the clinician. However, Nawoczenski and Ludewig[17] noted that the magnitude of change in muscle activity between orthotic and nonorthotic conditions was different between the 2 shoe types in one subject. Further studies exploring issues of shoe–orthotic device interaction are needed. In the interim, future investigators should consider using a standardized shoe to control for this potentially confounding variable when trying to elucidate the relationship among orthotic wear, lower extremity alignment, and neuromuscular response characteristics.

Influence of Other Lower Extremity Malalignments

Hruska[11] and Loudon et al[8] contended that the most important contributing influence on knee stability is postural control of lower extremity alignment. With an anterior pelvic tilt, femoral rotation, hip flexion, genu valgus, genu recurvatum, subtalar eversion, and forefoot or rearfoot pronation increase. Therefore, instability of the pelvis and associated lower extremity positional changes may place the athlete in a hyperextended and internally rotated position during dynamic activity, accentuating subtalar joint pronation. It becomes apparent then that a combination of postural faults may affect knee joint mechanics, soft tissue strain, and proprioceptive input.

Furthermore, multiple lower extremity factors can be manifested as a hyperpronated foot posture that may be more or less sensitive to orthotic correction of subtalar joint alignment. Hyperpronation can be the result of an anatomic disorder, such as forefoot varus, forefoot valgus, rearfoot varus deformities, or a plantar-flexed first ray of the foot.[5] Other contributing factors may include the state of alignment of the legs, such as genu valgus,[63] and developmental conditions, such as internal femoral torsion, limb length discrepancy, or short hamstrings or iliopsoas muscle.[5] We did not explore the causative factor for the subject's hyperpronation or exhaustively evaluate lower extremity posture for other potential malalignments. However, we did measure and document standing Q angle (11.5° ± 5.5°) in an effort to control for excessive knee valgus angulation. Thus, depending on the cause of the hyperpronated posture and the presence of other lower extremity or postural malalignments, an orthotic intervention alone may have a limited effect on factors that influence neuromuscular control of knee stability.

Hyperpronation continues to be implicated as a predisposing risk factor in anterior cruciate ligament injury. Evaluating neuromuscular response characteristics in a functional, weight-bearing stance provides a research model by which to investigate the influence of distal lower extremity malalignments on joint stress and reactive neuromuscular control of proximal joints. Our primary goal was to evaluate the short-term effect of an orthotic device on the muscular activation patterns at the knee in subjects with excessive subtalar joint pronation. Our findings suggest that a short-term application of a custom, semirigid orthotic device did not produce a change in reactive neuromuscular control at the knee. However, these findings are limited to this research model, and other weight-bearing testing methods (ie, landing or cutting characteristics) may yield different results. Further, whether adaptations may occur over time with improved lower extremity mechanics and effect a change in neuromuscular control cannot be determined from this study, and additional research is needed to explore long-term adaptations in neuromuscular control with continued orthotic wear. Other considerations for future studies include determining the degree of correction required to effect a change in muscular activity, controlling for shoe type, simultaneously quantifying both kinematics and electromyographic

changes using a variety of functional weight-bearing testing models, and comparing muscular activation patterns between subjects with and without selected lower extremity malalignments. Understanding how hyperpronation and other lower extremity malalignments influence protective neuromuscular responses and dynamic control of knee stability under sudden loading conditions may make a significant contribution to our understanding of anterior cruciate ligament injury risk and prevention.

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