About the Authors

Dr. Frank Noyes is an internationally recognized orthopaedic surgeon and researcher who has specialized in the treatment of knee injuries and disorders for nearly 4 decades. He is the founder and chairman of the Cincinnati SportsMedicine and Orthopaedic Center and its nonprofit research foundation. Dr. Noyes completed his orthopaedic training at the University of Michigan Medical Center. He then received a 4-year clinical and research appointment as an orthopaedic surgeon in the United States Air Force, was commissioned as a Lieutenant Colonel, and began his landmark research into knee ligament injuries, the effects of immobilization, biomechanics of ligaments, prevention of ACL injuries in the female athlete, the diagnosis of many knee injuries and problems, and the results of treatment for a variety of knee disorders. Along with Dr. Edward Grood, Dr. Noyes established one of the first biomechanics laboratories in the United States at the University of Cincinnati College of Engineering. The laboratory was subsequently named in his honor as the Noyes Tissue Engineering and Biomechanics Laboratory.

Dr. Noyes has won every conceivable award for his clinical and laboratory research from societies such as the American Academy of Orthopaedic Surgeons, the American Orthopaedic Society of Sports Medicine, the Orthopaedic Research and Education Foundation, as well as the University of Cincinnati. He was inducted into the American Orthopaedic Society for Sports Medicine’s Hall of Fame in 2008. Dr. Noyes has been selected by his peers as one of the Best Doctors in America every year since 1992.

Dr. Noyes has published over 260 clinical and scientific research studies and textbook chapters. These publications detailed surgical techniques and clinical outcomes on many different types of knee injuries and disorders. He edited a textbook entitled, “Noyes’ Knee Disorders: Surgery, Rehabilitation, Clinical Outcomes” which was written for orthopaedic surgeons, physical therapists, and other sports medicine health care professionals. Dr. Noyes is also a co-editor of “ACL Injuries in the Female Athlete. Causes, Impacts, and Conditioning Programs”, a textbook written for sports medicine health care professionals, coaches, and trainers involved with female athletes.

Sue Barber-Westin has directed clinical research studies for Dr. Noyes’ research Foundation for nearly 3 decades. In the mid 1980’s, she authored one of the first studies that measured problems during single-leg hopping tests in patients with ACL injuries, “Quantitative Assessment of Functional Limitations in Normal and Anterior Cruciate Ligament-Deficient Knees.” She has co-authored 140 articles in medical journals and textbooks, focusing on the clinical outcome of various knee operative procedures, the methods used to determine the results of clinical investigations, differences in neuromuscular indices between male and female athletes, effects of neuromuscular training in female athletes, and prevention of ACL injuries in female athletes. Sue is the associate editor of “Knee Disorders: Surgery, Rehabilitation, Clinical Outcomes” and is the co-editor for “ACL Injuries in the Female Athlete. Causes, Impacts, and Conditioning Programs”. Sue has personally undergone 4 knee operations and played competitive junior and collegiate tennis.

In 2004, Sue and Dr. Noyes were members of the clinical research team that won the Clinical Research Award from the Orthopaedic Research and Education Foundation. They are frequently invited to speak at national and international conferences and review articles for orthopaedic and sports medicine journals.
INTRODUCTION AND DIAGNOSIS

It is well appreciated that patients with chronic insufficiency of the lateral and posterolateral structures of the knee may develop a gait abnormality that is characterized by excessive knee hyperextension during the stance phase (initial contact or heel strike, loading response, midstance, and toe-off) of the gait cycle.

The primary lateral and posterolateral structures of the knee joint are the fibular collateral ligament (FCL) and popliteus muscle-tendon-ligament unit (PMTL), including the popliteofibular ligament (PFL) and posterolateral capsule (PLC). These structures function together to resist lateral tibiofemoral compartment opening, posterior subluxation of the lateral tibial plateau with tibial rotation, knee hyperextension, and varus recurvatum. Posterolateral injuries are frequently accompanied by a rupture to the anterior cruciate ligament (ACL) and, in some cases, a rupture to the posterior cruciate ligament (PCL). In addition, many knees with insufficiency to the posterolateral structures also have varus osseous malalignment.

The gait abnormality described in this chapter is easily identifiable in the clinic if the examiner devotes a small amount of time to observation during the initial patient presentation. An abnormal knee hyperextension gait pattern involves increased extension (>0°) in the sagittal plane and, frequently, associated varus malalignment in the coronal plane (varus recurvatum). The varus recurvatum position of the knee will be markedly worse if there is associated osseous tibiofemoral varus malalignment. The triple varus knee refers to varus alignment caused by three factors: tibiofemoral varus osseous malalignment, increased lateral tibiofemoral compartment separation due to marked insufficiency of the FCL and PMTL, and varus recurvatum in extension. An abnormal increase in hyperextension usually indicates damage to not only the posterolateral structures but the ACL as well.

Some knees with uninjured but physiologically slack posterolateral structures demonstrate a passive varus recurvatum and excessive knee hyperextension. After a knee injury or muscle atrophy of any cause, the patient may demonstrate a hyperextension gait pattern due to muscle weakness. Other patients with symptomatic patellofemoral arthritis develop a hyperextension gait pattern to avoid knee flexion that loads the patellofemoral joint and causes anterior knee pain. These patients are the most difficult to manage, because the knee arthritis symptoms require treatment to relieve the painful state in addition to correcting quadriceps weakness and the knee hyperextension gait pattern.

In the authors’ experience, patients with knee hyperextension gait problems present with varying amounts of altered gait mechanics, symptoms, and functional limitations. Some may demonstrate a markedly abnormal gait that is severely disabling and limits ambulation, requiring crutch or cane support. Others may have a less noticeable alteration, with the abnormal knee hyperextension occurring only after excessive walking (or other weight-bearing activities) and muscle fatigue. The degree of the gait abnormality during the stance phase depends on the magnitude of associated ligamentous deficiencies, quadriceps muscle atrophy, and symptomatic patellofemoral arthritis.
Subjective complaints of knee instability of either partial or full giving-way during routine daily activities often accompany the knee hyperextension gait abnormality. Pain is frequently located in the medial tibiofemoral compartment, which is caused by increased compressive forces owing to the varus malalignment. Pain in the posterolateral tissues also occurs from increased soft tissue tensile forces. In addition to the pain and instability caused by this gait pattern, there is an increased risk of failure of posterolateral reconstructions if the gait abnormality is not corrected before surgery. This is due to the excessively high tensile forces from high knee extension and adduction moments that are expected be resumed during weight-bearing activities after surgery. In addition, patients with associated ACL or PCL deficiency may have an increased risk of failure of cruciate ligament reconstructions if the hyperextension pattern is not corrected before surgery. Therefore, gait retraining and avoidance of the gait hyperextension pattern are paramount for both resolution of patient symptoms and reduction of the risk of failure of soft tissue ligament reconstructive procedures.

Many investigators have described a quadriceps-avoidance abnormal gait pattern in patients with ACL ruptures. An investigation at the authors’ center documented diminished quadriceps activity and enhanced hamstring muscle activity in one half of 32 ACL-deficient varus-angulated knees. Whereas the increased hamstring muscle force could be assumed to be beneficial because it provides a protective mechanism in decreasing anterior tibial translation, the increased muscle force creates high axial compressive forces and, therefore, increases medial and lateral joint compartment (calculated) loads. These loads could be deleterious to the joint over the long term, especially in knees with associated varus osseous malalignment.

A hyperextension gait abnormality pattern is also found in patients who have suffered a stroke or traumatic brain injury, cerebral palsy, and poliomyelitis. In these instances, the disorder may occur owing to many factors including quadriceps weakness, ankle plantar flexion spasticity, heel cord contracture, and gastrocnemius-soleus weakness. The concern is that the abnormal knee hyperextension may cause stretching of the posterior ligamentous and capsular structures from the increased external extensor torque that is placed across the knee during stance. It is not the purpose of this chapter to discuss the treatment of this problem in patients with neurologic pathology. Rather, this chapter focuses on the treatment of this gait disorder in patients with chronic insufficiency of the posterolateral structures, with or without cruciate ligament ruptures or varus malalignment.

ABNORMAL KNEE HYPEREXTENSION PATTERNS

The normal pattern of knee motion that occurs during the gait cycle is shown below.
A detailed description of normal human gait mechanics is beyond the scope of this chapter and has been presented by many authors.\textsuperscript{3, 4, 21, 24, 25} The loss of the normal knee flexion and extension patterns throughout the stance phase noted in patients with hyperextension gait abnormalities has important functional implications. During the loading response, normal amounts of knee flexion are required for the knee joint to absorb shock. A limb that is instead hyperextended transfers body weight directly from the femur to the tibia, resulting in abnormally high compressive forces. The usual muscle energy absorption and cushioning effect a flexed knee provides is lost.\textsuperscript{22, 23} The thrusting hyperextension motion at the knee is associated with an abnormally high adduction moment, which tends to increase medial tibiofemoral compartment compressive forces and lateral distraction forces. The increased compressive forces manifest as pain in the medial tibiofemoral compartment and posterolateral soft tissues.

The authors have reported two distinct knee hyperextension gait patterns. In pattern I, the abnormal hyperextension occurs during two periods of the stance phase, heel-strike and terminal extension, with knee flexion noted during the loading response. Exceedingly high knee extension moments are present, along with an abnormal reversal of hip extension and ankle dorsiflexion.
**Pattern II** is characterized by a prolonged knee hyperextension pattern from heel-strike throughout midstance. In these patients, the knee flexion moment is markedly below normal, with its effects incurred primarily at the knee with only a slight delay in ankle dorsiflexion.

The authors' clinical observations on the various types of abnormal knee motions and thrusts in the sagittal, coronal, and transverse rotational planes that occur during the stance phase of gait are summarized in the table on the next page. Any abnormality in tibiofemoral alignment (varus-valgus osseous malalignment or anterior-posterior tibial slope) may affect knee-thrusting motions in the sagittal or coronal plane. The most common of these is a varus-thrusting gait abnormality, caused by a varus tibiofemoral osseous malalignment. The thrusting motion occurs with the knee near full extension immediately after heel-strike, during the loading response. There may be an associated external or internal rotational subluxation with the varus thrust, depending on the presence of associated deficiency to the posterolateral structures. A less common abnormality, a valgus-thrusting gait, is usually associated with valgus lower limb malalignment. This disorder is typically accompanied by external rotation of the tibia. Knees with these osseous malalignment and associated knee ligament deficiency require surgical correction of the malalignment by osteotomy prior to ligament reconstructive procedures.
### Table 1 Abnormal Knee Motion Patterns and Subluxations During Stance Phase

<table>
<thead>
<tr>
<th>Stance Phase Event</th>
<th>Abnormal Knee Position</th>
<th>Motion Limit Increased</th>
<th>Phase of Stance</th>
<th>Possible Additional Subluxations</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperextension motion (thrust)</td>
<td>Hyperextension (pattern I)</td>
<td>Extension</td>
<td>Initial contact, terminal extension phases</td>
<td>Varus-external tibial rotation (recurvatum)</td>
<td>Sudden knee hyperextension-flexion-hyperextension even during stance. Markedly abnormal gait, retraining difficult.</td>
</tr>
<tr>
<td>Varus thrust</td>
<td>Tibial adduction (lateral joint opening)</td>
<td>Adduction</td>
<td>Initial contact and loading response phase</td>
<td>Tibiofemoral compartment rotational subluxation*</td>
<td>Usually occurs at loading response. Gait retraining not possible. External tibial rotation may decrease thrusting, internal rotation increases thrusting. Increased external tibial rotation (lateral-posterolateral injury, medial-posteromedial injury, or both). Increased internal tibial rotation is rare, usually ACL plus lateral ligament injury.</td>
</tr>
<tr>
<td>Valgus thrust</td>
<td>Tibial abduction (medial joint opening)</td>
<td>Abduction</td>
<td>Initial contact and loading response phase</td>
<td>Tibiofemoral compartment rotational subluxation*</td>
<td>Increased external tibial rotation (medial-posteromedial ligament injury), with anterior subluxation of medial plateau. Increased external tibial rotation with lateral-posterolateral injury with posterior subluxation of the medial plateau.</td>
</tr>
</tbody>
</table>

*Rotational subluxations may also occur in the absence of a varus or valgus thrust.

GAIT RETRAINING PROGRAM FOR ABNORMAL KNEE HYPEREXTENSION

A gait retraining program has been successfully used at the authors’ center for knee hyperextension abnormalities since the mid-1980s. The program requires two to four initial clinical sessions (held preferable every week) with an experienced physical therapist to instruct the patient on the abnormal gait mechanics that occur and the adaptations required to restore a normal gait pattern.

Table 2  Gait Retraining Program for Abnormal Knee Stance Hyperextension

<table>
<thead>
<tr>
<th>Anatomic Part</th>
<th>Retraining Program</th>
</tr>
</thead>
</table>
| Trunk-upper body    | 1. Maintain erect position, avoid forward loading position, which shifts body weight anteriorly to knee joint during stance phase.  
|                     | 2. Avoid excessive medial-lateral sway during stance phase, which induces varus-valgus moments about the knee and hip.               |
| Hip                 | 1. Avoid excessive hip flexion during stance phase, which encourages knee hyperextension and fatigues hip extensors.  
|                     | 2. For valgus lower limb alignment, avoid excessive internal femoral rotation. Encourage external femoral rotation and walking on lateral foot border. Avoid knock-knee position (important for valgus thrusts).  
|                     | 3. For varus lower limb alignment, avoid external femoral rotation. Encourage internal femoral rotation, knock-knee position. |
| Knee                | 1. Avoid any knee hyperextension throughout stance phase by always maintaining a knee flexion position.  
|                     | 2. Practice knee flexion-extension control walking in a slow manner; often begin with crutches. Initially use an excessive knee flexion position.  
|                     | 3. Gradually resume a more normal walking speed, after flexion-extension control and a more normal gait pattern are resumed.  
|                     | 4. Look for increase in patellofemoral pain.  
|                     | 5. Look for varus or valgus thrust with knee flexion position.  
|                     | 6. Look for external or internal rotational tibiofemoral subluxations when flexion position resumed. |
| Ankle               | 1. Avoid excessive plantar flexion. Maintain dorsiflexion using soleus muscle to induce early heel rise (rocker action) to encourage forward tibial progression and knee flexion.  
|                     | 2. Initially use excessive dorsiflexion and walking aids (elevated heel) to increase early heel-off in stance phase. |
| Foot                | 1. Encourage push-off against forefoot and toes along with early heel-off in stance phase to assist knee flexion during stance phase.  
|                     | 2. With associated varus alignment, encourage toe-out position. For valgus alignment, encourage toe-in position. |

The patient is instructed to practice at home for at least 2 to 4 hours daily. In addition, the patient undergoes muscle strengthening and neuromuscular coordination training as part of the comprehensive rehabilitation process. In order to have a successful outcome, the patient must be compliant with the time commitment and constant motivation required of this program. A family member is also taught the same instructions so that they can observe and assist with the patient’s retraining at home. It is also helpful to video the patient’s abnormal and corrected gait to aid the education process. The patient may ultimately require 8 to 12 therapy sessions over 6 to 12 weeks to return to a normal asymptomatic gait pattern.

The initial focus of the retraining process is placed on the hyperextension of the knee in order for the patient to understand that this is the primary abnormality that requires modification. The patient is instructed to maintain 5° of flexion with each step. This requires walking in a very slow and deliberate manner. A visual aid to provide to the patient is the mental image of a woman walking in high heels, which produces 5° to 8° of knee flexion. In addition, a 1- to 2-inch elevated heel may be used to help maintain flexion throughout stance phase. The clinician should be aware that problems may occur during gait retraining when the patient practices the flexed knee stance. While the flexed knee gait is advantageous in contributing to quadriceps strengthening, it may aggravate preexisting patellofemoral pain, which must be treated promptly.

The second step of the training process involves educating the patient on the abnormal ankle and foot motions that occur concurrently with knee hyperextension. The patient practices elevating the heel and pushing off with the forefoot and toes in the midstance phase to avoid knee hyperextension. The patient must limit excessive ankle plantar flexion and assume early ankle dorsiflexion to maintain forward progression of the Tibia and flexion of the knee joint. It is helpful to have the patient notice and feel the pressure against the forefoot during the end of stance phase. With the first and second steps, the patient is taught to say “knee-foot” with each stance cycle as a reminder of the normal gait pattern.

When the patient is not practicing the “knee-foot” adaptation, he or she will revert to the abnormal knee hyperextension pattern. However, within 4 to 6 weeks, it is surprising to note that the gait pattern often returns to normal and the patient is no longer required to perform a conscious reminder of the gait adaptation. In short, the gait pattern becomes routine.
The third step analyzes the hip and body trunk position. The fourth and final step of the retraining program determines whether an abnormal lower limb alignment (varus or valgus thrust) or an external or internal rotational knee subluxation occurs during stance. This is important because a primary cause of a patient’s abnormal knee hyperextension may be instability of the knee with flexion, so that coronal plane or rotational transverse plane subluxations occur. In these patients, a functional knee brace and additional gait retraining may be required.

In some cases, a heel wedge may be used to place the knee in slight flexion in order to minimize the midstance hyperextension that can occur with poor quadriceps control. A significant varus or valgus malalignment may require use of an unloading brace or a lateral or medial heel wedge.

There are primarily two different types of unloading braces: single-hinged and bilateral-hinged. Single-hinged braces are able to create a pulling mechanism through the affected compartment, thereby creating the unloading (e.g., when the brace has a medial hinge, the medial compartment is unloaded). With a double-upright hinged system, the brace will typically have a pushing mechanism of unloading (e.g., when attempting to unload the medial compartment, the lateral hinge mechanism will be adjusted to push through the lateral to the medial joint). In custom unloading braces, a fixed amount of unloading can also be built into the brace. The potential added advantage of double-upright unloading braces is the ability to compensate for cruciate and/or collateral ligament instability. If a brace or heel wedge is used, the patient is reminded to actively practice the gait-retraining techniques and not to solely use the brace/wedge as a passive limit to hyperextension. These devices also assist in decreasing the medial or lateral pain complaints that can accompany either joint instability or arthritis.

Voluntary muscle control is critical to help minimize hyperextension gait mechanisms related to diminished quadriceps function. Normal gait requires adequate push-off from the gastrocnemius-soleus complex, sufficient quadriceps contraction in midstance, hip and knee flexion during swing, and an upright posture. Defects in any of these mechanisms will permit gait alterations, and, when completed with sufficient frequency, become the default gait pattern for the patient. Gait is a learned activity and eventually becomes habitual. Therefore, gait retraining should include exercises to target the specific muscles during gait, pre-gait activities to methodically force the patient to think about using these muscles functionally, and then the act to practice the new gait pattern so it eventually becomes the learned default pattern to avoid the hyperextension gait mechanism.

In some knees with varus malalignment, there is an associated internal tibial torsion and toe-in gait. The patient may voluntarily decrease the varus thrust by purposely walking with a toe-out gait; however, when the patient is not practicing, the toe-in gait resumes. The same situation applies to a valgus knee alignment with a pronated toe-out gait. The gait retraining, therefore, is primarily to address knee hyperextension in which most patients will achieve a beneficial response to the program.

The following sections represent examples of how exercises may be coupled with other activities to assist with gait retraining. An important component of gait retraining is to target exercises for the specific muscles required during the gait cycle. For the gastrocnemius-soleus complex, the patient is instructed on the toe-raise exercise. This exercise can be progressed to move from using double stance to eccentric/negative repetitions (up with two legs/down with a single leg) and eventually to single-ankle plantar flexion lifting. Other options are to move from the floor to the edge of a step or to add additional weight for increasing the workload. The exercise can also be combined with traditional step-up exercises.
Quadriceps control is critical for normal gait and represents the muscle group that demands the most attention. Common exercises which have been shown to be effective in helping with quadriceps reeducation include the straight leg plus raise, performed with the patient in the seated position. The quadriceps is contracted, the leg is lifted approximately 6 inches off the table or chair, held in the lifted position for 15 seconds, and then lowered and relaxed for 45 seconds. This 1-minute cycle is maintained and progressed in ratios (1:3, 1:2, and eventually, 1:1), and then progressed from 5 to 10 repetitions, with additional ankle weight being added to increase the difficulty of the exercise.

Isometric wall-sits are an excellent method of facilitating quadriceps activation. The desired angle of knee flexion is typically between 45° and 70°. Patients with patellofemoral joint arthritis may need to maintain the angle of flexion between 30° and 45° in order to decrease patellofemoral pain. Patients who do not have joint damage or pain may perform this exercise in deeper degrees of knee flexion to allow for an increase in quadriceps muscle fiber recruitment. Having the patient keep weight pressures through the heels will also decrease the potential for patellar pain complaints. This exercise is similar to a stationary leg press and is performed as two repetitions, four times a day to a maximum fatigue, experiencing a quadriceps burning sensation. The patient tries each exercise and attempts to reach a 3-minute-total duration for each wall-sit repetition.

Active isometric hip adduction during the wall-sit by compressing a ball between the knees may assist vastus medialis oblique recruitment. In addition to obtaining adequate quadriceps control, it is important to improve hip abduction control. A heavy elastic exercise band may be placed proximal to the knee (avoiding patellar pressure) and “clamshells” are performed in either the hook-sitting (shown below)
...or the side-lying position. Three to five sets of 10 repetitions are performed. Adjustments to either knee flexion or hip flexion angles can make the exercise easier or more difficult depending on what is necessary to challenge the gluteal musculature.

The single-leg squat test may be performed to detect poor hip strength and trunk control.

Normal hip abductor and external rotation strength and control of the lower limb are required to prevent the internal hip rotation-valgus lower limb position that is often observed in patients with chronic patellofemoral symptoms.

Balance and proprioception are important components of a gait retraining program. The patient must be able to maintain single-stance control, as well as move the opposite leg through swing and be ready for the transition into the next stride. Standard progression of balance activities is typically sufficient to allow for return of a normal gait. A sample of these activities include weight-shifting, tandem balance, single-leg balance (stable and unstable), rocker board (front-to-back and side-to-side), and Biodex Stabilometer (Biodex Medical Systems, Shirley, NY) for postural stability (random control and maze control). Focus of balance control is part of both the clinical treatment and home exercise programs.

In order to create a functional gait-retraining program, a blending of the individual exercise programs must progress to include specific gait activities. Patients must overcome a variety of contributing factors including apprehension, muscle weakness, lack of range of motion, compensation, and pain. These factors must be resolved in order for the patient to restore a normal gait cycle. Each of the following factors contribute to a symmetric gait pattern: adequate push-off, midstance quadriceps control, hip and knee flexion, and maintenance of an upright posture.
Two common gait activities can be used quite successfully to assist in both the early time period after injury or surgery and in later time periods. Initially, an activity known as cup-walking is used in order to encourage the patient to break down the actual gait cycle into more manageable tasks. Cups are set up in two staggered rows with symmetric stride lengths. The patient is asked to walk or march over the cups, emphasizing each of the earlier four mentioned tasks. This activity allows the clinician to observe the gait cycle to determine where emphasis needs to be placed. Common compensatory mechanisms that patients exhibit include circumduction of the lower extremity, inadequate push-off, midstance hyperextension, lack of coordinated hip and knee flexion, positive Trendelenburg sign, and forward flexed trunk. The clinician must be able to identify the deficiency in order to teach the patient and her or his support system how to observe the deficiency and to perform the corrective strategy. The normal automatic nature of gait now becomes a “thinking” activity for the patient. The patient is asked to exaggerate the gait cycle by stepping over the cups. This methodical approach allows the patient to apply a part of the gait cycle to the entire, normal gait pattern. Gait retraining activities such as this must be repeated thousands of cycles over time in order to create a “new default” gait pattern.

As time progresses from the injury or surgery, another gait activity may be used that requires the patient to wear a heavy elastic band around the distal thighs (~2 finger widths above the patella) and then produce a marching pattern to provide a resisted-gait cycle. With a central line on the floor, the patient, while marching, must maintain right foot-strike on the right side of the line and left foot-strike on the left side of the line. This movement into swing ensures that adequate hip flexion and hip abduction musculature is used during the gait cycle. In addition, the stance limb requires adequate push-off of the gastrocnemius as well as a voluntary quadriceps contraction to prevent a hyperextension episode. This resisted-gait activity can be completed in forward, backward, and lateral directions. Each direction allows the clinician to emphasize what muscle groups are used to allow for adequate voluntary muscle control. Again, these activities require voluntary thought processes to focus on recruiting the quadriceps to avoid the hyperextension mechanism from occurring.
In the authors' experience, after approximately two to four sessions, the patient will understand the abnormal mechanics and recognize when the hyperextension patterns occur. As already discussed, mental reminders, such as “knee bent, toe push-off”, are helpful in this stage. After approximately 4 to 6 weeks of training, the patient should convert to a more normal gait pattern. However, it may require 2 to 3 months to complete the training process to the point where a normal gait pattern becomes routine on a subconscious level and the patient does not resume the hyperextension gait pattern when walking quickly.

**CLINICAL INVESTIGATION**

*Methods and Materials*

The authors' gait-retraining program was studied in 5 patients with symptomatic knee hyperextension and deficiency of the posterolateral structures. Computerized gait analysis testing was conducted before and after the retraining program using a two-camera video-based optoelectronic digitizer for measuring motion and a multicomponent force plate for measuring ground reaction force. The force plate was camouflaged under a 10-m walkway. Patients were asked to walk at three speeds (normal, fast, slow) and measurements were obtained over these ranges to enable comparison of similar walking speeds. Data from complete cycles of stance and swing phase were obtained.

Kinematic data in the sagittal plane and kinetic data in the sagittal, coronal, and transverse planes of the hip, knee, and ankle were evaluated. Peak values during stance phase were recorded for each patient. Using a previously described mathematical model, joint reaction loads, lateral soft tissue forces, and muscle forces were calculated. The patient data were compared with those obtained from a control group of 11 subjects matched for age and walking speed. All moments, which were expressed as external moments, were normalized to body weight and height.

*Analysis of Gait Mechanics before Retraining*

Statistically significant differences were found between the pre-trained patients and the control subjects in the mean values for knee hyperextension during heel-strike and terminal extension, with the patients demonstrating a range of 5.4° to 18.4° less flexion \( P < .05 \). The patients had a significantly higher mean knee midstance extension moment (127%; \( P < .01 \)), knee adduction moment (28%; \( P < .05 \)), and calculated medial tibiofemoral compartment loads (43%; \( P < .001 \)) than the controls. The two knee hyperextension patterns previously discussed were identified; 3 patients demonstrated pattern I and 2 patients, pattern II.
Table 3: Measured and Calculated Peak Values for Control and Involved Limbs

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control Group</th>
<th>Study Group Pretraining</th>
<th>Study Group Post-training</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knee motion (°)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heel-strike</td>
<td>1.3 ± 1.6</td>
<td>-5.6 ± 2.8&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.2 ± 2.6&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Peak loading response</td>
<td>14.9 ± 5.0</td>
<td>6.2 ± 10.9&lt;sup&gt;d&lt;/sup&gt;</td>
<td>16.4 ± 4.2</td>
</tr>
<tr>
<td>Terminal extension</td>
<td>6.6 ± 4.0</td>
<td>-7.3 ± 4.4&lt;sup&gt;b&lt;/sup&gt;</td>
<td>11.1 ± 5.8&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Toe-off</td>
<td>35.0 ± 7.0</td>
<td>36.1 ± 5.3</td>
<td>41.8 ± 5.9&lt;sup&gt;e&lt;/sup&gt;</td>
</tr>
<tr>
<td>Ankle plantar flexion motion (°)</td>
<td>11.4 ± 6.0</td>
<td>16.1 ± 8.5</td>
<td>9.4 ± 1.5&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Hip moments (% BW x Ht)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abduction</td>
<td>1.2 ± 0.9</td>
<td>1.1 ± 0.4</td>
<td>0.7 ± 0.4&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Adduction</td>
<td>5.1 ± 0.9</td>
<td>6.0 ± 1.0</td>
<td>4.9 ± 1.4&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Knee moments (% BW x Ht)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heel strike extension</td>
<td>2.4 ± 0.5</td>
<td>2.6 ± 0.7</td>
<td>2.0 ± 0.5&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Peak midstance extension</td>
<td>1.5 ± 1.0</td>
<td>3.4 ± 2.0&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.7 ± 0.7&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Adduction</td>
<td>3.6 ± 0.5</td>
<td>4.6 ± 1.4&lt;sup&gt;d&lt;/sup&gt;</td>
<td>3.6 ± 0.7</td>
</tr>
<tr>
<td>Ankle dorsiflexion moment (% BW x Ht)</td>
<td>9.0 ± 0.5</td>
<td>9.1 ± 0.9</td>
<td>8.4 ± 0.9</td>
</tr>
<tr>
<td>Predicted forces (BW)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexor muscle group</td>
<td>1.5 ± 0.3</td>
<td>2.2 ± 0.7&lt;sup&gt;d&lt;/sup&gt;</td>
<td>1.3 ± 0.4&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Extensor muscle group</td>
<td>1.0 ± 0.4</td>
<td>1.1 ± 0.5</td>
<td>0.9 ± 0.4</td>
</tr>
<tr>
<td>Medial tibiofemoral load</td>
<td>2.1 ± 0.2</td>
<td>3.0 ± 0.4&lt;sup&gt;b&lt;/sup&gt;</td>
<td>2.1 ± 0.5&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<sup>b</sup>P < .01 for pretraining versus control.
<sup>c</sup>P < .01 for posttraining versus pretraining.
<sup>d</sup>P < .05 for pretraining versus control.
<sup>e</sup>P < .05 for posttraining versus pretraining.

**BW**, body weight; **Ht**, body height.

**Analysis of Gait Mechanics after Retraining**

After the program of gait retraining, the patients demonstrated a statistically significant increase in the degrees of knee flexion during heel-strike, terminal extension, and toe-off (mean, 10°). At terminal extension, an average increase of 18° of flexion was measured compared with the pretraining value (**P** < .01). There were significant decreases in the knee extension moment at heel-strike (23%) and terminal extension (80%). The mean adduction moment decreased to a normal value, and a significant reduction (30%) occurred in the predicted medial tibiofemoral load (**P** < .05).

There was a 36% decrease in the hip abduction moment at heel-strike (**P** < .01), and an 18% decrease in hip adduction moment during midstance after gait retraining (**P** < .01). The ankle plantar flexion motion decreased by 7° (**P** < .01), which was associated with an 8% decrease in the ankle dorsiflexion moment.

Four of the 5 patients successfully resolved or markedly reduced hyperextension at the knee and abnormal motion patterns at the hip and ankle. These individuals converted the knee flexion-extension moment to a normal biphasic pattern. One patient failed to complete the program and demonstrated continued gait abnormalities.
Effect of Retraining on Patient Symptoms
The patient symptoms before and after gait retraining were evaluated with the symptom rating scale of the Cincinnati Knee Rating System. A statistically significant improvement occurred in the pain scale (0-10 points) from a mean of 1.6 ± 0.9 points before training to 4.8 ± 2.3 points after training ($P < .05$), and in the partial giving-way scale from a mean of 2.4 ± 0.9 points before training to 5.2 ± 2.3 points after training ($P < .05$). Four of the 5 patients improved from experiencing moderate pain and instability with daily activities before retraining, to walking several hours per day without appreciable symptoms. Because of this improvement, 2 patients did not require ligament reconstructive surgery after retraining.

SUMMARY
In patients with chronic deficiency of the posterolateral structures, the use of gait analysis techniques and retraining can successfully change the kinetics and kinematics of the hip, knee, and ankle to more normal levels. The abnormal knee hyperextension can be significantly reduced, along with adduction and extension moments about the knee and medial tibiofemoral compartment loads. Hip abduction and adduction moments may be restored to normal levels. In addition, ankle plantar flexion and its counterbalancing dorsiflexion moment can be decreased. Gait retraining is indicated in knees with a hyperextension gait abnormality prior to any ligament reconstructive procedure and may, in some cases, eliminate the requirement for such soft tissue operations. If the gait abnormality is not corrected, cruciate and posterolateral ligament reconstructions may fail from the high tensile forces placed on the grafts postoperatively as the patient resumes an abnormal knee hyperextension gait pattern. These same principles apply to other individuals with knee hyperextension gait patterns such as those that occur with quadriceps muscle weakness from a variety of knee injuries and symptomatic patellofemoral arthritis.
References