7-2003

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Abstract

The literature cites numerous studies involving the analysis of movement patterns in anterior cruciate ligament deficient (ACLD) patients. Although several in vivo biomechanical studies have shown that ACLD patients develop protective mechanisms against degenerative diseases, it seems that these adaptations fail to protect the knee from future pathology. Some authors state that ACLD patients adapt to the injury by avoiding quadriceps contraction during gait when the knee is near full extension. However, others have found increased hamstrings and decreased gastrocnemius activity, which normally contribute to the stability of the knee. It seems that further in vivo biomechanical investigation is required to understand the mechanisms of pathological knee joint motions and develop rehabilitation programs, which would delay the progress of developing long-term degenerative diseases.

Keywords Anterior cruciate ligament · Compensatory · Mechanisms

Introduction

An estimated 80,000 human anterior cruciate ligament (ACL) tears occur annually in the United States [20]. Patients endure this type of knee injury from a wide variety of contact and noncontact activities. Ultimately the injured person must make a decision either to have the ligament surgically repaired or to rehabilitate the injured knee joint without repair to the ligament. In general, younger patients desiring to continue participating in high stress activities, for

example, competitive and recreational sports, choose the option of reconstructive ACL surgery. However, patients who tend not to participate in high impact activities can forgo the surgical procedure or are advised to use conventional physical therapy interventions to heal their injured knee [49].

Numerically, Noyes et al. [40] estimated that approximately one-third of patients who sustain an ACL injury compensate enough to pursue recreational activities, another one-third make compensations but must discontinue many activities, and one-third experience instability with conservative treatment and option for surgical intervention. Daniel et al. [16] observed chondral lesions in 23% of the ACL-deficient (ACLD) patients, while the risk of secondary meniscal tears was 20% over a 5-year follow-up period. As a result patients who decide not to have surgical repair run the risk of having knee degenerative changes. However, even though ACLD patients are at a greater risk for obtaining these long-term problems, a substantial portion of this population makes the necessary functional adaptations to compensate for the loss of the ACL [40]. An understanding of the mechanisms of these adaptations that occur for ACLD patients during daily living activities (i.e., gait) is extremely valuable for the development of better prevention and therapeutic strategies.

These adaptations can be evaluated with the use of in vivo biomechanical studies. In such studies the *kinematics*, *kinetics*, and *electromyography* of the knee joint are analyzed. The term kinematics is used to describe the three rotational motions of the tibia with respect to the femur (flexion-extension, adduction-abduction, internal-external rotation) or the three translational motions of the tibia with respect to the femur without reference to the forces or moments that cause the movements. Kinetics is used to describe the joint forces and moments associated with motion. Electromyography is used to describe the electrical activity of the muscles (i.e., gastrocnemius) that surround the knee joint. The present review summarizes and critically discusses the available literature on in vivo biomechanical studies regarding adaptations that occur with ACLD patients. We also provide recommendations for future research work that can practically assist orthopedists to provide a better quality treatment.

In vivo kinematics of the ACLD knee

The injury to the ACL disrupts the linkage system that connects the femur and the tibia resulting in abnormal anterior translation of the tibia during flexion and extension of the knee. Jonsson et al. [24] reported that the active extension of the ACLD knee is related to displacement of 1.9 mm more anteriorly than normal knees extending from 15° to 10° of flexion. Moreover, the absence of the ACL was not completely compensated for active knee extension past 30°. The movements tended to normalize as the knee was close to full extension probably due to increased joint surface contact forces, efficient muscle stabilization, and factors related to the anatomy of joint surfaces. However, these results on anterior-posterior tibial translation and knee stability are the outcome of static measures. Thus the question is whether (i.e., during gait) we have similar results dynamically.

Using video-based motion analysis we can address this problem. However, some other difficulties arise with this methodology. It is difficult to evaluate anterior-posterior tibial translation during gait with the use of reflective markers applied on the skin [4, 29, 30, 46]. The principal calculation difficulties are mainly attributed to the relative movement of the markers with

respect to the underlying skin. It can be certainly supported that this source of error represents one of the most important unsolved problems in in vivo kinematic analysis, because such perturbations are difficult to be removed with low-pass filtering since their frequency content is close to that of the motion. According to Cappozzo et al. [12] the motion of the marker with respect to the underlying bone due to skin movement ranges from a few millimeters up to 40 mm. Attaching the reflective markers on intracortical pins can eliminate this source of error. Thus, using reflective markers attached on intracortical pins fixed on tibia and femur it has been observed that the linear drawer motion of the tibia in the intact knee amounts to 3.6 mm posteriorly during stance. During the swing phase –14.3 mm of posterior motion has been observed [30]. Similar techniques of data acquisition with markers attached on intracortical pins have been reported in the literature, which have been applied in the ACLD knee during various activities such as landing on the ACLD limb [47].

Certainly the implantation of intracortical pins is a highly invasive procedure that may cause discomfort or pain to the patient and result in restriction of movements. In addition, we believe that the implantation of intracortical pins is a method that is limited by the sample size since an effective number of volunteers cannot be found. Lucchetti et al. [35] tried to approach the skin movement artifact ignoring the solution of pin fixation and developed a technique of skin movement artifact assessment and compensation in the estimation of knee-joint kinematics. The method assesses the skin movement error caused by joint movement on the surface marker cluster located on an adjacent body segment. Knowledge of the artifacts on an adjacent body segment permits for their compensation during estimation of the kinematics of the joint at the other end of the same body segment. The main disadvantage of this method is that requires the subject to perform an ad hoc movement that makes the calculation procedure more difficult. It seems that this technique is very accurate since the ranges of tibial rotation and translation are very close to the ranges derived from markers attached on intracortical pins. Further accuracy could probably be achieved as proposed by the authors if their procedure is extended to both segments adjacent to the target joint.

Alternatively Chèze et al. [13] developed a numerical solidification procedure to facilitate kinematic analyses based on video system data which allows an average reduction in kinematic errors of 20–25% when the maximum distance between markers is small (e.g., 15 cm). Similar research [27] has been conducted for the solution of the problem related to the effect of local tissue accelerometer vibration from surface measurements of vibration over the spine.

An additional solution to the problem of skin movement error has been proposed by Halvorsen et al. [21]. In most video-based motion analysis systems simulation of human gait requires linked rigid bodies as models of the whole extremities. The joints are represented either as hinge models or ball and socket models. Halvorsen et al. [21] introduced a different approach for estimating the axis of rotation and the center of rotation that can be considered a contribution in the field of motion analysis. Their method does not assume a rigid body motion but only that each marker rotates around the same fixed axis of rotation or center of rotation. In the knee joint where the skin movement artifacts are large the proposed method seems to be more accurate, since the method does not make the assumption on the rigidity of the body. However, to our knowledge no knee kinematic studies have been published in which the new method is applied.

On the other hand, the method of simulated gait has been applied in the ACLD knee to calculate the amount of tibial translation [34]. Simulated gait was accomplished via a two-dimensional anatomical knee model while the effect of various levels of hamstring muscle activation on restraining anterior tibial translation in the sagittal plane was studied. The model included the tibiofemoral and patellofemoral joints, four major ligaments, the medial capsule, and five muscle units surrounding the knee. The results showed that the rupture of the ACL could result in increased anterior tibial translation by 11.8 mm. A simulated peak external flexion moment during the early stance phase resulted in tibial translation of 1.4 mm posteriorly in the normal knee, while in the ACLD knee the tibia translated anteriorly by 10.4 mm. It is also interesting that a simulated hamstring force at 56% of its maximal isometric strength restored near normal anterior-posterior translation in the ACLD knee. These findings suggest that hamstring strengthening rehabilitation programs could possibly protect the knee from increased anterior-posterior tibial translation by increasing the amount of hamstrings contraction during gait.

In addition to the increased hamstring contraction, it was found [2] that there is another compensatory mechanism for avoiding increased anterior displacement of the tibia. This mechanism involves the quadriceps muscle group. Specifically, Andriacchi [2] reported that ACLD patients during level walking tend to avoid quadriceps contraction when the knee is near full extension. A quadriceps contraction when the knee is near peak extension causes strain on an uninjured ACL. Therefore in an ACLD patient a reduced quadriceps contraction would most likely reduce anterior displacement of the tibia relative to the femur. This could be considered as a compensation that protects the knee from excessive anterior drawer.

The exact mechanism by which avoidance of the quadriceps contraction reduces anterior tibial translation has been investigated by studying the effects of strain on the ACL cadaver knees with a transducer placed upon the ACL [7]. The strain on the ACL varied upon whether the knee flexion angle was changed passively or through the stimulation of quadriceps contraction. Simulated isometric quadriceps contraction significantly increased anterior-medial ACL strain above the normal resting level through the first 45° of knee flexion. The same quadriceps contractions produced lower strain on the ACL during 60° of flexion or greater. This reduction in strain was significant at 105° of flexion and at 120° of flexion. According to these findings, excessive anterior tibial translation during gait would be avoided if the patients were able either to avoid the quadriceps activation when the knee is near full extension or avoid the excessive activation of the quadriceps by walking with the knee more extended.

Although the above compensatory mechanisms have been proposed to occur with ACLD individuals, what also remains unclear is whether ACL reconstruction restores normal knee joint motion, or whether such compensations still occur. Several authors have investigated the effect of ACL reconstruction on restoring normal flexion extension kinematics, but contrary results are reported in the literature. During the 3–5 weeks after the ACL reconstruction the knee and hip joints are significantly more flexed particularly at heel contact and midstance [17, 18]. Nevertheless, 5 weeks after the ACL reconstruction the flexion extension kinematics usually recover to prereconstruction values [17, 18]. However, it must be pointed out that initially there is an increased flexion at the hip, knee, and ankle (10° more flexion at all joints), particularly during stance. Probably during the early postinjury or postoperative period no gait adaptation has developed so the increased knee flexion angles found result in higher external flexion moments. It

is possible that a stronger quadriceps contraction is necessary for the elimination of the increased external flexion moments, which in the ACLD knee may result in excessive anterior tibial translation.

This hypothesis seems correct since there is some evidence that in the long term ACLD patients may walk with increased knee extension angles. Wexler et al. [52] found that 7.5 years after injury ACLD patients walk with increased knee extension angles during the terminal stance. Walking with the knee in a more extended position results in lower demands that are placed on the quadriceps. Thus this finding can also be considered as an additional mechanism, which produces the quadriceps avoidance gait pattern in chronic ACLD knees as the nervous system adapts to the injury [9].

Although chronic ACLD patients (7 years postinjury) can probably compensate for the loss of ACL, it seems that 2 years after the injury the increased hamstring activity remains the main compensatory mechanism. According to the findings of Beard et al. [8] who examined the gait and muscle activity of ACLD patients approximately 2 years after their injury, subjects walked with significantly greater terminal knee flexion angle. Additionally, the ACLD patients had during stance a prolonged period of average hamstrings activity for the injured limb relative to the normal (40.1% vs. 24.4% of the stance phase). Quadriceps activity duration was similar in the ACLD and control groups. Because of a significant correlation between the prolonged hamstrings activity and knee flexion angle in the ACLD patients they suggested that the ACLD patients walked with hamstrings facilitation rather than displaying a quadriceps avoidance gait (Fig. 1).

Patel et al. [43, 44], who studied how the avoidance of the quadriceps contraction is developed, found two distinct mechanisms. The first was that 72% of the patients with a quadriceps avoidance gait walked with a significantly reduced midstance knee flexion angle. The average knee flexion angle for this group during midstance was 7.4°, while the control group had an average of 19.5°. This adaptation allowed the patient to reduce the demand placed upon the quadriceps during the stance phase of gait.

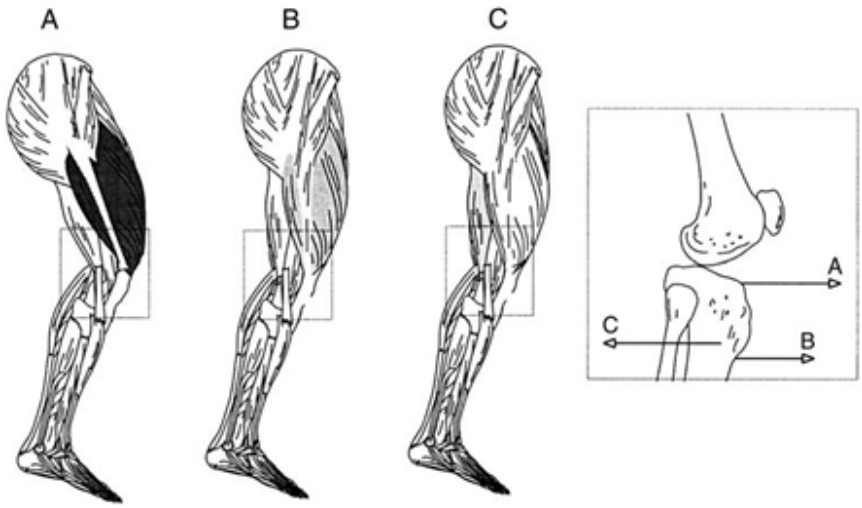

Fig. 1 Mechanisms of avoiding excessive anterior tibial translation during walking. ACLD patients place low demands on the quadriceps muscle due to either increased knee extension angle or diminished external flexion moments (*B*). Therefore the resultant force applied on the tibia by quadriceps contraction is of lower magnitude (*B*) than the magnitude of the force applied on the tibia when the knee is more flexed (*A*). Another protective mechanism is the prolonged hamstrings activity during the entire gait cycle (*C*), which contributes to the agonistic role of the ACL in avoiding excessive anterior tibial translation

Therefore the anterior pull on the tibia was reduced, and the knee was more stable. However, the second mechanism found in the remaining 28% of patients with quadriceps avoidance gait was an increased peak external hip flexion moment compared to all of the other subjects. The authors hypothesized that a forward trunk lean by these patients probably produced the increase in the hip flexion moments, thereby helping to decrease the strain placed upon the quadriceps during midstance.

However, less is known regarding the transverse and the frontal plane movements of the tibia with respect to the femur. This is probably due to the complexity and past technical limitations of a three dimensional analysis. Thus, although flexion extension knee kinematics have been extensively investigated during gait in ACLD and reconstructed patients, tibial adduction-abduction and internal external rotation have not received similar attention. Most in vivo research studies performed are focused on tibial rotation during active or passive extensions [22, 23, 24, 25, 26, 38, 45]. Karrholm et al. [26] recently showed that normal tibial rotation during active extension ranges from 9.9° of internal to 1.6° of external rotation. Lafortune et al. [30], who examined the tibiofemoral kinematics during gait, reported similar patterns but different magnitudes. Possible explanation to this difference in the ranges of rotation are the variable accuracies of the methods applied as well as the fact that the knee joint loading is different during gait and active extension. Moreover, normal knee kinematics are characterized by variability probably due to the variable degree of laxity of the ligaments along with the different knee joint anatomical configuration between the subjects.

Jonsson et al. [24] studied the knee kinematics of active extension in ACLD knees and found no significant difference in tibial rotation or adduction-abduction between injured and intact knees. It has been reported that when the tibia is displaced anteriorly, as during the pivot shift test, the internal rotary laxity increases [25, 39]. Because of a minimal anterior tibial displacement and probably of the rotational muscle stabilizing effect no abnormal tibial rotations were observed in this study. However, in 7 of the 13 ACLD patients the knees rotated more internally and in 5 the rotation was more external than on the unilateral side.

Except for knee testing during active extension ACLD knee kinematics has been evaluated during stress loading. Karrholm et al. [25] studied the three-dimensional movements of the knee in three conditions: passive knee flex- ion, anterior traction (150 N), and posterior traction (80 N). The injured knees had more internal medial rotation during 28–29° of knee flexion. During anterior traction the injured knee had more than 1.4° of flexion, less than 4.2° internal medial rotation, and less than 2.3° of adduction. The injured knees had 1.8° of external lateral rotation while the normal knees displayed 1.4° of internal medial rotation when a posterior traction was applied to the sub- jects' knees. Similar results have been reported by Czerniecki et al. [15], who studied tibiofemoral rotation during walking and running. Through the use of a triaxial electrogoniometer the researchers found that absolute stance phase rotation significantly increased when ambulations speed increased. However, there were no significant differences in absolute stance phase rotation between the subject groups at any condition. They concluded that ACL injury most likely has no effect on tibiofemoral rotation during forward locomotion and that the extent of tibial rotation is correlated significantly with the strength of the quadriceps and hamstrings.

More recent studies [6, 42] have shown that ACLD subjects tend to walk with the tibia more internally rotated than healthy and reconstructed patients. It is possible that an increased internal tibial rotation can produce higher strains in the secondary structures such as the menisci resulting in degenerative diseases in the long term. In these studies [6, 42] the patients were evaluated during the low stress activity of walking, while further work must be carried out in vivo during more stressful activities such as pivoting or stair climbing with or without bracing.

Ramsey et al. [47] have investigated the protective role of bracing during moderate to intense stress activity in a three-dimensional kinematic study, in which intracortical pin implanted markers were utilized to increase the accuracy of the kinematic measurements. Patients with ACLD knees jumped for maximal horizontal distance and landed onto their deficient limb to sufficiently stress the knee. Although only four patients were tested, no significant reductions in anterior tibial displacement were noted, and the intrasubject differences between unbraced and braced conditions were small. However, increased intersubject variability was found in amplitudes and positional changes at touchdown for the tibiofemoral kinematics.

Further investigation is required to validate the results of Ramsey et al. [47] since the functional braces are designed with the objective to protect the knee joint from pathological tibiofemoral kinematics and extensive linear tibial displacements. In addition, contrary conclusions are reported by Beynnon et al. [10] who observed that bracing not only significantly reduced ACL strain values for anterior directed loads applied to the tibia but also reduced strain values in response to internal-external torque applied about the long axis of the tibia. Since internal torque of the tibia is considered an ACL injury mechanism the role of braces is recognized to be protective, although it is diminished as the magnitude of applied anterior load and internal-external rotary torque is increased [47].

In conclusion, the recent technological advances and the above results show that in the future researchers should perform knee functional testing in all planes. Movements in the sagittal plane have been extensively investigated. Compensatory mechanisms reported in the literature which may contribute to the avoidance of excessive knee instability are (a) increased hamstrings activity, (b) quadriceps avoidance gait pattern, and (c) forward trunk lean. However, it remains unclear which movements in the transverse and frontal planes can be considered as compensatory or play a role in the development of knee joint degenerative diseases. In addition, longitudinal studies are needed to evaluate the effect of the gait analysis findings on long-term knee pathology (i.e., osteoarthitis, chondral and meniscus pathology). Investigation is also required on the role of bracing in protecting the knee during stressful activities of daily living such as sidestep cutting maneuvers. In vivo investigation [37] on the mechanisms of ACL injury in ACL intact individuals during the sidestep cutting maneuvers revealed that sidestepping induces significantly greater maximum knee joint rotations than running. Although these movements cannot alone elicit spontaneous noncontact ACL injury, the high intrasubject variability in cutting maneuvers as a result of an abnormal and potentially dangerous sidestep can lead the individual to display “atypical” knee joint biomechanics, which increase the risk of ACL injury.

Kinetics of the ACLD Knee

Kinetic data offer the researcher and clinician information regarding *cause*. Thus we can understand the nature of the kinematic differences or the *effect*. For example, joint moments are important for determining the muscle groups (i.e., flexors) responsible for possible kinematic changes and the type of the muscular contractions that occur as a result of pathology. Therefore it is not surprising that most gait studies conducted with ACLD individuals have incorporated a kinetic evaluation.

One of the most accepted gait adaptations in ACLD individuals that was first described kinetically is the so-called "quadriceps avoidance gait pattern" [9] mentioned above. According to Berchuck et al. [9], ACLD patients produced a significantly lower knee flexion moment during midstance. The value of this moment was 140% smaller than the corresponding one generated by healthy control subjects. The diminished knee flexion moment was explained as a reduction by the ACLD patients of the quadriceps contraction. The reduced quadriceps contraction can be related to the anterior directed force of the patellar ligament when the knee is near full extension. When the knee is flexed above 60° the direction of the force vector reverses and the quadriceps contraction contributes to the reduction in anterior tibial translation [7].

The research study group from the University of Vermont (Burlington, Vt., USA) has conducted extensive investigation regarding the ACL strain in vivo during various degrees of knee flexion [10]. Ligament strain values were measured with the differential variable reluctance transducer (5 mm long), which was arthroscopically attached to the intact ACL of patient volunteers. Their contribution is significant in understudying compensatory mechanisms of the ACLD knee since they highlighted the role of quadriceps and hamstring muscles in the kinetics of the knee in vivo. They recorded the ACL strains during isometric contraction of (a) quadriceps (b) hamstrings, and (c) simultaneous contraction of quadriceps and hamstrings at 15°, 30°, 60°, and 90° of knee flexion during active flexion-extension of the knee and squatting. According to their findings isometric contraction of the quadriceps produces significant increase in ACL strain at 15° and 30° of knee flexion, and no change at 60° and 90°. Furthermore, it seems that the increased quadriceps contraction increases the anterior tibial translation irrespective of the type of contraction (concentric vs. eccentric) [28]. In contrast, isometric contraction of the hamstrings is not related with significant change in ACL strain at all flexion angles and when combined with isometric contraction of the quadriceps produces significant ACL strain only at 15° of knee flexion. However, it cannot easily be concluded that the coactivation of quadriceps and hamstrings is a protective mechanism since Kvist et al. [28] reported that although an ACLD knee can limit the anterior tibial translation during concentric muscle contraction, the limitation depends on other mechanisms than hamstrings coactivation. Furthermore, the mean translation of the tibia with respect to the femur was 38% larger in the ACLD knees during eccentric contraction of the quadriceps.

In light of the above findings [7, 10] during the first 45° of knee flexion the anterior directed force applied on the tibia would probably produce increased anterior translation of the tibia with respect to the femur. During gait it seems that ACLD patients avoid excessive anterior tibial translation by reducing the quadriceps activation and the subsequent anterior directed force applied on the tibia since this aberrant motion may cause damage to the menisci and articular cartilage. Such damage may contribute to the early development of degenerative

changes at the knee [36, 40]. The incidence of this gait pattern ranges from 50% to 75% among ACLD patients [41]. There have been more studies that found results similar to Berchuck et al. [9] and support the theory of quadriceps avoidance gait [5, 49, 52]. Further, Wexler et al. [52] observed that with increasing time after the injury, changes in the sagittal plane knee moments are more pronounced probably because of neurological adaptations.

Indeed, it is more likely that gait adaptations are the result of reprogramming of the locomotor process such that the adaptations occur before excessive anterior displacement results [5]. The study of Wexler et al. [52] performed on 30 patients with ACLD knees demonstrated that the adaptations start to develop early and are more pronounced in the long term. All of the above findings could probably contribute to the establishment of such rehabilitation exercises that produce low or untrained ACL values. Currently it is clear that exercises dominated by the hamstrings muscle group are appropriate although the safe limits of ACL graft strain during healing are not defined. However, it can be concluded that excessive quadriceps activation can lead to damaging effects if used during the early rehabilitation period or performed at knee flexion angles less than 60°. On the other hand, it should be highlighted that is currently unknown whether the avoidance of quadriceps contraction during various activities can result in lower incidence of knee degenerative changes in the long term.

Some experiments have refuted that the compensatory mechanisms described as quadriceps avoidance gait occur in ACLD subjects during gait. No differences were demonstrated in the internal knee extension moment during mid- stance between the injured and healthy contralateral knee, while quadriceps electromyographic (EMG) activity was noted throughout most of the stance phase for the ACLD patients [48]. Furthermore, it has been reported that the observed increase in the internal knee flexion moment in the ACLD patients might be related to the increase in hamstrings activity rather than a decrease in quadriceps activity [8].

In conclusion, it is true that there is no agreement among researchers on what kinetic adaptations actually occur in ACLD patients. However, the most accepted functional adaptation described to date is the so-called “quadriceps avoidance gait pattern.” Current data indicate that this gait pattern still exists at 6 month after ACL reconstruction [18], but it seems that 9–12 months after the reconstruction it is no longer present [50]. However, during more demanding activities such as jog-and-cut maneuvers functional adaptations are still present in ACL reconstructed patients with no significant quadriceps and hamstrings strength deficit and minor meniscal loss or osteoarthritic deterioration [11]. It seems that the decreased ability of the patients to generate a normal peak external flexion moment is related to their microinstability or decreased proprioception [11]. Irrespective of the mechanisms of functional adaptations, all researchers agree that ACLD patients develop neurological adaptations, which tend to decrease the excessive anterior tibial translation.

In the future, researchers should address whether graft placement, angle at which tensioning of the graft is performed, and type of graft have any effect on the above mechanisms. These data will support the scientific sports medicine community with evidence regarding the suitable ACL reconstruction protocol that must be followed. Our review supports the proposition of Andersen and Amis [1] who concluded after reviewing studies on the tension in the

natural and reconstructed ACL that although extensive investigation has been conducted in vitro little work has been carried out in vivo.

Dynamic EMG anaylsis and ACL deficiency

EMG has been an important tool for examining the activity of specific knee muscles during gait. Various research groups have used this experimental technique to understand the functional adaptations that occur with ACLD patients and complement the above kinematic and kinetic evaluations. In general the literature has identified the altered muscle activation patterns that occur in ACLD patients.

Particularly it has been reported that there is an earlier onset of EMG on the lateral hamstrings and medial gastrocnemius muscles in the ACLD patients throughout the gait cycle and that these bursts tended to be longer when compared to healthy controls [31, 33]. In addition, ACLD patients have less activity in the gastrocnemius and quadriceps musculature, while they experience greater activity in the hamstrings during the swing to stance phase transition period [31, 33]. The vastus lateralis appears to be more active in ACLD patients during early swing, while the hamstrings were more active during the mid-stance and terminal stance phases of the gait cycle [31, 33]. These findings collectively indicated that the knee flexors tend to be active for a longer duration in the gait cycle for ACLD subjects. It is a common agreement among researchers that there is a synergist effect of the hamstrings to the ACL in preventing excessive anterior tibial translation [19, 49]. In addition it seems that the decreased gastrocnemius activity protects the knee from excessive anterior tibial translation since gastrocnemius activation during the 45° of knee flexion produces strain on the ACL [19]. Although, the quadriceps and hamstrings may be the main contributors of knee joint stability it can be theoretically supported that the decreased gastrocnemius activity is an additional compensatory mechanism.

However, what is not well understood is if the ACLD patients compensate enough during stressful activities. Although ACLD knees may present all of the above mechanisms of protection, it seems that after the influence of mild fatigue patients do not compensate with increased hamstring EMG activity. Van Lent et al. [32] performed EMG analysis of the vastus lateralis, vastus medialis, biceps femoris, and medial hamstrings while ambulating on a treadmill at 1.2 and 2.8 m/s after the employment of a 10-min duration mild fatigue protocol. The results showed that the mean EMG of the biceps femoris and medial hamstrings was significantly lower in the injured knee of the ACLD patients. There were no significant changes in EMG activity for the control group. Thus, while during walking the hamstrings group contributes to the stability of the knee joint, fatigue results in decreased hamstrings activity that can be associated with lower antagonistic effect on the mechanisms of excessive anterior tibial translation and higher incidence of knee instability. However, more studies need to be conducted to evaluate movement behavioral changes in ACLD individuals under stressful activities.

It would be useful to know whether the normal muscle activity can be restored either with rehabilitation alone or combined with ACL reconstruction. Ciccicoti et al. [14] studied the effect of rehabilitation on the EMG profiles and found that rehabilitation alone does not result in

restoration of normal EMG profiles. For this reason the authors reported that reconstruction is recommended for young and active patients.

Interesting observations were also made via intra-articular knee effusion that can affect the development of compensatory mechanisms of muscle activity. Torry et al. [51] have found that the knee effusion produced by injecting a saline solution into the knee joint results in decreased quadriceps activity and increased hamstrings activity. The results of this experiment suggest that knee joint capsular, distention, via knee joint effusion, are responsible for gait adaptations reported for knee injured individuals in previous investigations. Knee joint effusion and the subsequent capsular distention could be considered a causative factor promoting the acquisition on quadriceps avoidance gait patterns.

In conclusion, it seems that EMG studies focused on the adaptive mechanisms developed by the ACLD patients have shown that the hamstrings can provide a dynamic substitute for the ACL in avoiding excessive anterior tibial translation. Furthermore, the decreased activity on gastrocnemius muscle can be considered as a further protective mechanism against excessive anterior tibial translation since the gastrocnemius contracture during the first 45° of knee flexion produces significant strain on the ACL [19]. The findings of EMG studies have significant implications regarding the surgical interventions performed on the ACLD patients. For example, it has not been clear by in vivo studies that the use of hamstrings as a graft for ACL reconstruction does not impair the stabilizing role of the muscle.

Summary

In summary, several studies have determined that ACLD patients adapt to the lack of the ACL over a prolonged period of time. Some researchers suggest that ACLD patients walk using an altered gait pattern called as quadriceps avoidance pattern. Others deny that this phenomenon exists and maintain that ACLD subjects adapt their gait by other means such as increased hamstrings or decreased gastrocnemius activity. In spite of this disagreement over mechanisms by which these adaptations occur, it is likely that these adaptations happen as a result of repetitive experiences following the loss of the ACL [3]. Future studies should assess the neural mechanisms by which the adaptations developed, evaluate ACLD for possible adaptations in the transverse and frontal plane motions and identify rehabilitation programs that contribute to the accelerated development of protective mechanisms. Longitudinal studies are also needed to investigate possible correlation between the functional gait adaptations and the incidence of knee osteoarthritis as well as chondral and meniscus pathology.

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