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Neuromuscular Consequences of Anterior Cruciate Ligament Injury

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The neuromuscular consequences of anterior cruciate ligament (ACL) injury are important considerations because these deficits play a crucial role in patient's recovery following ACL injury or reconstruction. The purpose of this article is to review and synthesize the known neuromuscular consequences of ACL injury and reconstruction. Specifically, changes in somatosensation, muscle activation, muscle strength, atrophy, balance, biomechanics, and patient-oriented outcomes are discussed. Understanding neuromuscular consequences aids in the construction of optimized rehabilitation strategies.

SOMATOSENSATION

The ACL and the knee joint capsule are composed of mechanoreceptors, such as free nerve endings, Ruffini endings, Golgi tendon organs, and Pacinian corpuscles, which provide information pertaining to joint position to the central nervous system for communication with the muscle [1,2]. Evidence for the physiologic connection between the ACL and the sensory cortex has been confirmed using detection of somatosensory evoked potentials following electrical stimulation of the ACL [3]. There is some controversy within the

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literature regarding the existence of altered proprioception or somatosensory deficits in ACL-deficient (ACL-D) and ACL-reconstructed (ACL-R) patients. This controversy may be because of the wide variety of methods used to evaluate somatosensory deficits in these populations.

Active joint repositioning has been used to assess proprioceptive deficits, and generally consists of passively moving a joint to a target point in a specific range of motion and then instructing the participant to actively reposition the joint to that target position. The ability to actively reposition the knee following ACL injury has been reported to be diminished in the involved leg compared with the uninvolved leg [2–6]. Deficits in active repositioning have been reported to persist in ACL-R patients [5]. Other authors [7–10] have reported no differences in the ability to reposition injured and uninjured knee joints in ACL-D patients.

Rasmussen and Jensen [5] reported that there were significantly greater errors in ACL-D and ACL-R patients when starting at a flexed position and extending the knee compared with starting at an extended position and actively flexing the knee. Inaccuracies during active extension joint repositioning may cause increased anterior translation, which may cause inaccuracies compared with during flexion repositioning of the knee. Another study [9] performing active joint repositioning going from full extension to a flexed position has reported no difference between involved and uninvolved ACL-D, ACL-R, and control knees. Four of the five studies [2,3,5,6] reporting a decreased ability to actively reposition the injured knee were moving from a flexed position to an extended; only one study [4] reporting a decrement was moving from an extended position to a flexed position. Of the studies that reported no difference in the ability to actively reposition the injured knee, one study [7] had subjects moving from a flexed position to an extended position and four studies [5,8–10] had subjects moving from an extended position to a flexed position. Authors [8] suggested that it is possible that muscle receptors dominate afferent signaling of position during joint repositioning and compensate for altered signals from joint proprioceptors. Furthermore, it has been reported that the larger the deficit in joint position sense the worse the performance is in vertical jump measures ($r = -0.389$, $P < .05$) and one-leg hop measures ($r = -0.444$, $P < .05$) in ACL-D patients, suggesting that deficits in active joint repositioning may affect function [6].

Although actively repositioning a joint provides some information about the somatosensory system, the test is confounded by the motor component. Deficits or compensations from the central nervous system or motor neurons may not depict pure somatosensory function, but rather the function of the nervous system following contributions from sensory, central, and motor neuron deficits and compensation. Instructing the subject to passively detect the repositioned target point may be a purer measurement of proprioception. A decreased ability to passively detect joint position has been reported in ACL-D [5,11] and ACL-R patents [4,5,10] in the injured limb compared with the uninjured limb. Fischer-Rasmussen and Jensen [5] reported that ACL-D

patients had a significant .21° difference between injured and involved legs, ACL-R patients had a significant .17° between legs, and there was no difference found between legs in healthy controls. The latency in which afferent signals are processed has been studied by passively moving the knee, instructing participants to identify when they first feel movement. The ability to detect passive motion of the knee has been reported to be significantly diminished in the involved leg of ACL-D patients [12–14] compared with the uninvolved leg. Friden and colleagues [14] reported a deficit in detection for movements into extension and flexion.

Somatosensory function of the lower extremity following ACL injury has been studied by electrically stimulating nerves in the lower extremity and examining impulses, termed somatosensory evoked potentials (SEP), recorded in specific zones of the sensory cortex [12,13,15]. SEPs have been reported to be altered following common peroneal nerve stimulation in patients who had damaged ACLs [15]. It has been reported that ACL-D patients displaying proprioception impairments also had altered SEPs, yet all patents who had altered SEPs did not have altered proprioception [12]. This finding may be explained by the hypothesis that altered proprioception of ACL-D knees is a chronic pathology that becomes more apparent over time [8].

MUSCLE ACTIVATION

Neuromuscular reorganization around the ACL-D or ACL-R knee may be the underlying contributing factor for other more conventionally recognized clinical impairments, such as strength loss, atrophy, and altered function. Although some researchers have examined the activation of the popliteus [16] or tibialis anterior [17] following ACL injury, most of the literature to date has focused on neuromuscular alterations in the quadriceps and hamstring muscle group. For the most part there has been a consensus among authors that a decrease in volition activation or motor unit firing exists in the quadriceps of patients who have ACL injuries [18–26].

Muscle inhibition attributable to knee joint pathology was first described by de Andrade and colleagues [27], who concluded that deformation of joint mechanoreceptors in injured knee joints relayed altered afferent information to the central nervous system, which they believed was caused by inhibition of the motor neurons of the surrounding quadriceps musculature. This phenomenon is now termed arthrogenic muscle inhibition [28] and is characterized by a reflexive decrease in motor neuron pool excitability [29], modulated by pre- and postsynaptic mechanisms [30,31], that inhibits the ability to activate the surrounding uninjured musculature following joint injury. Researchers [28,29,32] have suggested that mechanoreceptors, such as Ruffini fibers, Pacinian corpuscles, and Golgi-like endings, in the knee joint capsule or ligamentous structures of the joint are stimulated because of mechanical deformation caused by structure damage or distention of the capsule, which sends altered information to the spinal cord. Once the afferent information reaches the spinal cord it can be modulated presynaptically by GABA interneurons or

postsynaptically by Renshaw cells, which are situated on motoneuron collateral fibers [30,31].

Other authors [33,34] have studied the electromyographic delay in the extensor mechanism following ACL reconstruction to determine how mechanisms other than reflexive ones previously explained might contribute to neuromuscular changes. Unfortunately there is controversy about whether an electromyographic delay in the extensor mechanism exists following ACL reconstruction. The absence of a mechanical delay has been reported and suggests that the efferent component of the neural system is not affected following ACL-R [33], whereas other authors [34] report an increased delay in an extensor response following a bone patellar bone autograft, which may be explained by increased stiffness of the extensor mechanism of alterations in the excitation coupling system.

Past researchers [35–40] have hypothesized that injury to the ACL would increase the activation of the thigh muscles to improve joint congruency and decrease shear forces at the knee joint. The ability to generate torque from the hamstrings has been hypothesized to be imperative in decreasing anterior translation of the tibia in ACL-R patients [41]. It has been hypothesized that the mechanoreceptors within the ACL and other knee ligaments transmit afferent information that may be processed as a reflex with the purpose of contracting musculature to decrease forces at the knee [42–44]. It has been reported that electrically stimulating the ACL with a train of two stimuli produced activity in the hamstring at rest [45] and inhibited the knee extensors and flexors during their respective contractions [46]. This finding provides evidence that an ACL reflex exists, and can have both an excitatory and an inhibitory component. In a study with limited subjects (three), increased activation of the hamstrings in response to a posterior perturbation has been reported in ACL-D patients, whereas healthy patients used the quadriceps to stabilize [47]. This finding provides evidence that the hamstrings are used to co-contract to respond to a perturbation.

Tsuda and colleagues [48] reported that this hamstring reflex arc was reestablished in subjects ranging from 37 to 80 months post bone patella bone autograft ACL reconstruction, suggesting that mechanoreceptor may reinnervate the grafted ACL allowing for more normalized afferent function. Reflex activity has been reported to be decreased after the ACL is anesthetized suggesting the ACL provides key information to the central nervous system about joint position sense [45]. Because of the small amount of activity that is produced in the hamstring following stimulation of the ACL, however, it has been hypothesized that this ligamentous structure is not solely responsible for sending afferent information about joint position [45]. Biedirt [49] reported that no hamstring reflex was elicited after tugging on the ACL, yet a reflex was found following a Lachman test suggesting joint receptors in structures other than the ACL are influential in producing a hamstring reflex.

The presence of altered neuromuscular control in the lower extremity has been evaluated using electromyogram (EMG) in ACL-D [16,50–55] and

ACL-R patients [19,23,41,52,56]. Some of these EMG studies have evaluated the neuromuscular alterations of the lower extremity in dynamic activities and supported the hypothesis that the hamstrings increase in activity while quadriceps activation is inhibited during landing from a jump in ACL-D patients [19].

Others reported no changes in the quadriceps but a decrease in the activation of the gastrocnemius [52]. Limbird and colleagues [57] reported that the quadriceps and gastrocnemius were inhibited while hamstrings were activated during gait. Further studies [58] have also reported that hamstring activity increases before landing, suggesting that the neuromuscular system may alter activation strategies using a feed- forward mechanism. Neuromuscular control has been reported to be altered in ACL-D patients during closed-chain activities, suggesting that altered neuromuscular control is needed to adequately perform a closed-chain task [59]. Altered neuromuscular control of the quadriceps has been termed quadriceps dyskinesia, which is an encompassing term that describes not only unwanted inhibition of the quadriceps but also inability to shut the quadriceps off during open-chain knee flexion tasks in which quadriceps tone was not needed [60].

A study by Boerboom and colleagues [51] evaluated hamstring activity in three separate groups, including copers, noncopers and healthy controls. There was no difference in hamstring activity during the stance phase of gait between copers and healthy controls, yet noncoping ACL- D patients had significantly more hamstring activity and knee flexion. Houck and colleagues [53] added that copers, noncopers, and controls used distinct activation patterns of the medial and lateral hamstrings and the vastus lateralis during unanticipated change of direction tasks during walking, which may be a possible explanation for why some ACL-D patients can cope with the injury and others cannot. Aalbersber [61] reported that ACL-D patients did not differ in the amount of quadriceps-hamstring co-contraction strategies compared with normal subjects when a shear force was applied to the knee. Ostering [56] reported less hamstring coactivation in maximal knee extension, which they attributed to an afferent denervation of the ACL following injury or reconstruction.

Central Mechanisms

Friemert and colleagues [45] concluded that the nature of the long latency (65 to 95 milliseconds) of the hamstring reflex that followed the double stimulation of the ACL suggests that the reflex is polysynaptic thus allowing central mechanisms to modulate muscle response. Other studies evaluating the motor cortex have suggested that altered function exists in ACL-injured individuals. Baumeister and colleagues [62] reported differences in cortical excitability measured by EEG during a repositioning of the ACL-R knee compared with control subjects. Other measures, such as the resting motor threshold of the motor cortex, have also been reported to be altered in the cortical hemisphere corresponding to the ACL-D knee compared with the uninvolved knee [63]. Reports of bilateral quadriceps inhibition in cases of unilateral ACL injury suggests that a crossover effect exists that most likely is caused by central nervous system mechanisms [20,26].

Gamma Motor Neuron Dysfunction

Some authors [64,65] hypothesize that although decreased sensory information caused by damaged ACL mechanoreceptors may not have a direct impact on alpha motor neuron function, alterations in afferent signals from joint receptors directly affect the gamma motor neuron system. The gamma motor system adjusts the shortening of the intrafusal fibers of the muscle spindles, regulating sensitivity, thus affecting the ability to produce a muscle contraction. Deficits in the gamma loop system of ACL-R [64,66,67] and ACL-D patients [65,68] have been reported following repetitive stimulation of the patellar tendon. Control subjects show marked decreases in quadriceps maximal voluntary contractions and EMG activity attributable to neurotransmitter depletion, heightened Ia threshold, or other presynaptic inhibitory mechanisms following repetitive vibratory stimulation. Maximal voluntary quadriceps contractions and EMG activity of ACL-injured patients remains relatively unaffected by repetitive vibratory stimulation, which suggests decreased activity in the gamma loop system. Interestingly, gamma loop dysfunction has been reported bilaterally in patients who had unilateral ACL injury, providing evidence that central nervous system mechanisms, which may be interneuronal or supraspinal in nature, may influence neuromuscular control of the entire organism following unilateral ACL injury [68]. This bilateral quadriceps gamma loop dysfunction has been reported early following reconstruction of the ACL, yet seems to resolve after approximately 18 months in the uninjured side, whereas deficits seem to persist in the injured leg [66].

Median Frequency

A decrease in median frequency has been reported in the quadriceps of the ACL-D limb [23,25,69] and when compared with healthy controls [23]. Authors [25,69] suggested that this was caused by an atrophy of type II muscle fibers.

MUSCLE STRENGTH

Quadriceps isokinetic strength deficits have been reported following ACL injury and seem to persist for patients following rehabilitation and in those who do not engage in structured rehabilitation (Table 1). Knee extension strength deficits have been reported between 6 months and 15 years postinjury in ACL-D patients who have not undergone reconstructive surgery [25,55,70–72]. Torque deficits for knee extension have been reported to vary between 10% and 38% of the torque generated in the uninjured leg [70,71,73]. When compared with matched healthy controls, quadriceps torque values have been reported bilaterally, leading some researchers [74] to suggest that torque deficit percentages relative to the uninjured leg may underestimate the true strength deficits in the injured leg following ACL injury. There is some evidence that quadriceps torque deficits in ACL-D patients decrease with time [70], indicating that there may be some ability to regain bilateral symmetry in knee extension force capabilities. Researchers [70] hypothesize that decreased

Table 1
Concentric isokinetic torque information following anterior cruciate ligament reconstruction

Author	Population	Average chronicity	Graft type	Velocity (°/s)	Quadriceps deficits (%)	Hamstring deficits (%)
Ageberg et al [95]	36 males, 20 females	15 y	—	90	5	No deficit
Anderson et al [77]	39 males, 18 females	6 mo, 1 y	22 PT, 23 HT	60	6 mo = 25, 1 y = 20	6 mo = 16, 1 y = 7
Blyth et al [96]	15 males, 15 females	2–8 y	9 PT, 21 HT	60, 180, 360	9, 6, 4	+1, +1, +6
Bryant et al 2008 [78]	9 males, 4 females	6–9 mo	PT	180	30	Not reported
Carter and Edinger [145]	106 patients	6 mo	38 PT, 68 HT	18, 300	No deficit	No deficit
De Jong et al [79]	191 patents	6 mo, 9 mo, 1 y	HT	60, 180	6 mo (36, 25), 9 mo (25, 18), 1 y (19, 16)	No deficit
Grossman et al [80]	22 males, 7 females	~16 y	22 PT, 3 HT, 3 Gortex	180, 240	12, 18	12, 12
Hiemstra et al [74]	9 males, 7 females	>1 y	8 PT, 16 HT	50–250	25	17
Hiemstra et al [81]	12 subjects	<1 y	HT	20–250	24.8	26.8
Jarvela et al [82]	65 males, 21 females	5–9 y	PT	60, 18, 240	10.3, 4.5, 5.2	0, 0, 2.9
Keays et al [83]	22 males, 9 females	33 mo	HT	60, 100	7.3, 7.8	10, 9.9
Kobayashi et al [84]	11 males, 25 females	6 mo, 1 y, 2 y	PT	60, 180	6 mo = 37, 311 y = 27, 182 y = 11, 9	6 mo = 10, 101 y = no deficit 2 y = no deficit
Makihara et al [97]	3 males, 13 females	26 mo	HT	60	Not reported	6%
Konishi et al [85]	39 males, 31 females	<1 y	HT	60, 180	9, 8	Not reported
Lee et al [86]	58 males, 9 females	6 mo, 1 y, 2 y	Quadriceps tendon	60, 180	6 mo = 36, 26 1 y = 18, 18 2 y = 18, 11	Not reported
Mattacola et al [87]	11 males, 9 females	1.5 y	PT	120, 240	16, 9	6, 5
Moisala et al [88]	39 males, 9 females	5 y, 9 mo	16 PT, 32 HT	60, 180	PT (10, 5) HT (7, 2)	PT (1, +1) HT (3, 0)
Nakamura et al [89]	36 males, 40 females	2 y	HT	60, 180	15, 11	8, 13
Nyland et al [90]	7 males, 11 females	2 y	Tibialis anterior	60	11	+7
Østeràs et al [91]	90 subjects	6 mo	PT	60, 240	28.7, 21	3.1, 1
Segawa et al [92]	34 males, 28 females	1 y	HT	60	7	2
Seto et al [93]	19 males, 6 females	~10 y	Not reported	120, 240	33, 41	15, 16

Abbreviations: HT, hamstring tendon; PT, patellar tendon.

ability to produce quadriceps torque in ACL-D patients exists to decrease anterior shear forces at the knee. Others [40] have suggested that the hamstring muscles alter their function to assist in stabilizing the knee joint in the presence of an ACL ligamentous insufficiency.

Although knee flexion torques have also been reported to be diminish in ACL-D patients [70], the affect of ACL injury on the hamstring muscle group does not seem to be as devastating as reported in the quadriceps. Knee flexion torque deficits in ACL-D patients have been reported between 2% and 15% of the uninjured knee [70,71]. It has been hypothesized that the hamstrings play an important role in stabilizing the knee following ACL injury, and it has been suggested that hamstring strength may be an important factor in determining ACL-D patient function level [75]. The hamstring muscle group plays an important role during athletics and in activities of daily living, eccentrically contracting allowing for controlled deceleration and proper force attenuation. Hamstring torque deficits have been reported to practically double when assessed eccentrically (15%) compared with concentrically (8%), which may be because of altered muscle recruitment patterns that could increase the risk for subsequent injury [71].

Deficits in quadriceps strength following ACL-R have been reported at various speeds and years post-reconstruction [23,74,76–94]. Although the largest quadriceps strength deficits are reported in the first 6 to 12 months following surgery [77,84,86], deficits of between 5% and 18% of the uninvolved limb have been reported between 5 and 15 years following ACL reconstruction and extensive rehabilitation [80,88,93,95]. These quadriceps strength deficits following ACL- R are reported to be to some extent bilateral when compared with healthy matched controls. Quadriceps avoidance gait patterns and decreased ability to absorb shock during stance have been suggested to be possible risk factors to chronic joint pathologies following ACL injury.

Data regarding the effect of hamstring strength following ACL-R are not as conclusive as those concerning quadriceps strength. Some authors have reported increased hamstring deficits compared with the quadriceps following ALC-R [81,83], whereas others identify the quadriceps as having deficits that far exceed those of the hamstrings [77,82,88,91,96,97]. The controversy surrounding the amount of hamstring weakness following ACL-R may be related to graft used in the reconstruction. There has been some evidence emerging that hamstring weakness may be more associated with semitendinosus or gracilis grafts compared with bone patella bone grafts [74,88,98]. A recent study by Nyland and colleagues [90] used tibialis anterior tendon grafts, which may be the best representation of pure arthrogenic muscle inhibition following ACL-R because strength results were not confounded by tendon damage at the donor site. Nyland and colleagues [90] reported an 11% decrease in quadriceps strength and a 7% increase in hamstring strength, which may indicate altered neuromuscular control strategies present in an ACL reconstructed knee.

Although many studies [74,77,81,89,90,92] have reported strength deficits 1 to 2 years following reconstruction, little research [80,95] has determined long-term strength outcomes with modern reconstructive procedures.

ATROPHY

Muscular atrophy in the thigh muscles and legs of ACL-R and ACL-D patients is concerning because of the potential effects on the force-producing capabilities of the atrophied muscles. Quadriceps atrophy has been documented for ACL-D [72] and ACL-R [99,100] patients. The vastus medialis of ACL-D patients demonstrates decreased glycolytic activity and a shift toward more oxidative metabolism, a possible sign of active compensation for knee instability [101]. Noncopers demonstrate significantly greater quadriceps atrophy than copers [102]. Further, harvesting the semitendinosus tendon for ACL-R results in atrophy and shortening of the semitendinosus [103–105].

Adaptations in other muscles to compensate for atrophy and lost force-producing capabilities are also concerns. For example, noncopers have larger tibialis anterior muscles in the injured leg compared with the uninjured leg, possibly because of altered gait patterns in noncopers [17].

Atrophy can be prevented or become less apparent with eccentric exercise, particularly in the quadriceps and gluteus maximus muscles [106], with protein supplementation [107], or electrical stimulation [108]. Interestingly, vascular occlusion may also diminish postoperative disuse atrophy, possibly because of hormonal secretions triggered by the vascular occlusion [109].

BALANCE

The ability to maintain one's posture has been closely linked to proprioception and neuromuscular control strategies. Postural control or balance measurements have been assessed in ACL-D patients and ACL-R patients with various evaluation techniques. There is a consensus within the literature that no difference in balance exists during double-leg stance among ACL-D and ACL-R patients compared with healthy controls. Lysholm and colleagues [110] reported a significant deficit in postural control during single-leg stance with both eyes open and closed in unilateral ACL-D patients compared with healthy controls. Lysholm and colleagues [110] reported that differences were not present between injured and uninjured legs, suggesting that a bilateral deficit compared with controls was present in this group of ACL-D patients.

There is evidence that suggests a decrease in postural control measurements during closed-eye trials compared with eyes-open trials and single-leg compared with double-leg stance trials for ACL-injured patients and healthy controls [111]. In contrast to Lysholm and colleagues [110] other authors have reported no evidence of deficits in static measures of postural control among ACL-D [111,112] and ACL-R patients [87,113,114] when compared with healthy controls. Tecco and colleagues [112] reported that although no difference was found in center of pressure movement between healthy and ACL-D patients during static measures, a difference in location of center of pressure relative

to the foot was found between the patient and healthy groups. The ACL-D patients were reported to encompass more anterior and medially positioned center of pressure before the commencement of static balance trials compared with the healthy controls [112]. Although an altered positioning of the center of pressure did not affect static balance trials it may be suboptimal positioning for maintaining posture following a perturbation.

There is more of a consensus among authors that the ability to maintain balance following a perturbation differs between healthy subjects and ACL-D and ACL-R patients [110,113,114]. Lysholm and colleagues [110] reported that reaction time to a perturbation was longer in ACL- D patients compared with the healthy subjects and that the injured leg had a longer reaction time compared with the uninjured leg on the healthy subjects. Henriksson and colleagues [114] also reported differences in sagittal plane ground reaction forces between ACL-R patients and healthy subjects following a perturbation, yet no differences were reported in the frontal plane. It has been stated that balance measures following a perturbation may be a better indicator of function compared with static measures because they better represent demands placed on the neuromuscular systems during functional activities [111]. Impaired balance has been hypothesized to be caused by decreased or altered mechanoreceptor information regarding joint position [113] from the injured knee, possibly resulting in modified neuromuscular control while attempting to maintain balance.

BIOMECHANICS

Following ACL injury and reconstruction changes in lower extremity kinematics, kinetics, and temporal variables have been shown to occur. Biomechanical changes in gait (walking, jogging, running), stair ambulation, and jumping have been researched extensively, but have inconsistent findings. This inconsistency may be attributed to methodologic differences between studies and the use of heterogeneous populations. Individuals who are ACL-D can be categorized into two groups, based on clinical criteria, as copers and noncopers [115,116]. Most ACL-deficient individuals fall into the classification of noncoper and experience knee instability after injury, which requires surgical reconstruction [75]. Conversely, copers are ACL-D individuals who use compensatory stabilization strategies and do not experience episodes of "giving way." These individuals have movement strategies that resemble individuals who do not have lower extremity pathology.

Biomechanical compensatory strategies are believed to be task dependent with more difficult tasks accentuating the adaptation [115,117]. Although tasks such as walking, jogging, running, stair ambulation, and jumping have similarity there are also distinct differences in muscle activation, kinematics, and kinetics. Performance on these tasks also depends on time elapsed since injury and surgical reconstruction [118,119]. Surgical reconstruction and rehabilitation have been shown to influence biomechanical adaptations and restore movement patterns that are similar to uninjured subjects [19,52,118].

Gait

Walking

There is discrepancy regarding temporal-spatial parameters of gait during walking. Earlier studies indicated that individuals who are ACL-D walk with symmetric gait pattern and with changes occurring in both the involved and uninvolved limbs [120]. Based on this finding it was suggested that the uninvolved limb not be used as a valid comparison of normal gait biomechanics [120]. More recent studies have indicated that the uninvolved limb may not have compensatory changes to the same degree as the involved limb. Step length and walking base have been shown to be smaller for the ACL-D limb compared with the uninvolved limb when comparing within the same subjects [118,119]. Based on this finding it was suggested that the uninvolved limb not be used as a valid comparison of normal gait biomechanics [120]. When comparing between healthy individuals, copers, and noncopers, step length, cadence, swing time, and stance time have been shown to be more similar [115,121]. Following surgical intervention and 4 months of rehabilitation, step length and walking base values are not significantly different from healthy individuals [118,119].

Walking electromyography. ACL-D individuals tend to stabilize the knee by using a co-contraction of the quadriceps and hamstring muscles [121,122]. Higher hamstring activity is present in the involved limb from initial contact to midstance [121,122]. Noncopers also have an earlier onset of medial gastrocnemius and a longer total duration when compared with copers and healthy controls [117]. Controversy exists regarding the presence of "quadriceps avoidance" [120,123] or decreased quadriceps activity during gait [115,118,124]. At initial contact decreased quadriceps activity may be present, but at midstance quadriceps activity between limbs is similar [122]. Decreased quadriceps activity at initial contact is coupled with higher soleus activation on the involved side [117], which may act as a secondary knee extensor by directing the tibia posteriorly when the foot is in contact with the ground [125]. At midstance the magnitude of soleus activity was lower compared with the uninvolved limb, whereas quadriceps activity was similar between limbs [122]. The compensation of the soleus is not likely needed at midstance. Eight months following ACL-R normal muscle EMG patterns of the lower extremity have been shown to return [118].

Walking kinematics. Noncopers have less knee flexion at initial contact compared with copers [115,117,121] and healthy controls [117]. Joint angles for the hip and ankle are similar for the three groups [115,121]. When comparing injured to uninjured limbs the knee flexion angle at initial contact is similar, but the involved knee has more flexion at midstance [122]. Copers use greater knee flexion during walking than noncopers and healthy controls [121]. Following surgical intervention and 4 months of rehabilitation, knee flexion angles are not similar to those of healthy individuals [118].

Walking kinetics. During the loading phase of walking individuals who have ACL-D knees demonstrate decreased knee moments that resist knee flexion [117,120] and have lower peak ground reaction force [115,117]. This pattern continues through the midstance of gait on the involved limb [122] and is believed to reduce the stress placed through the knee joint and decrease anterior tibia translation [120,126]. Load from decreased knee moments at initial contact coexists with increased contribution of hip moments [120,122]. At midstance there is a shift from greater hip moments to increased ankle moments [122]. Noncopers are believed to demonstrate the greatest decrease in knee moments [117,121] and increased hip joint moments when compared with copers and healthy controls [117].

Jogging/running

In general, jogging/running tends to exaggerate gait abnormalities compared with walking [19,52,115,117]. Compared with healthy subjects individuals who are ACL-D (copers and noncopers) have decreased jogging speed and stride length [117]. Jogging speed is slightly greater for copers when compared with noncopers, but is not significantly different [115]. Following surgical reconstruction and rehabilitation individuals tend to begin to have jogging and running biomechanics that resemble those of healthy individuals [19,52].

Jogging/running electromyography. Individuals who are ACL-D have higher hamstring EMG activity without a decrease in quadriceps EMG activity compared with individuals who have ACL reconstructions or healthy knees [19,117]. Noncopers have higher hamstring EMG activity than copers [117]. The magnitude of the differences between groups tends to be diminished during a more difficult task, such as jogging compared with walking [117].

Jogging/running kinematics. When examining a heterogeneous ACL-deficient population the amount of knee flexion during jogging was the same as healthy subjects [120]. Further examination while classifying individuals as noncopers indicated they typically limit knee joint flexion at initial contact and during the stance phase of jogging [115,117]. Copers have knee joint angles that are symmetric between sides during jogging [115]. Kinematics at the ankle do not differ between copers and noncopers [115].

Jogging/running kinetics. Noncopers have decreased knee moments at peak knee flexion on the involved limb [115,117] and decreased vertical ground reaction force during jogging compared with copers [115]. ACL-D individuals demonstrate decreased peak knee flexion moment at midstance compared with healthy subjects [120]. The hip moments increased and ankle moments remained the same and were comparable to findings during walking [117]. Loading patterns were symmetric during jogging for copers [115,120], but differed between sides for noncopers [115].

Stair Climbing

There is little difference in range of motion and forces through the lower extremity when going up and down stairs when comparing healthy subjects

and ACL-D subjects [120]. When examining noncopers, they use less knee flexion in the involved limb when ascending stairs compared with copers [115]. Noncopers also have decreased peak vertical ground reaction force compared with copers [115]. Both groups flex their involved knee less during stair descent [115].

ACL-R individuals demonstrated decreased knee extension moment during a lateral step-up task compared with healthy subjects [127]. Summated extensor moments (hip + knee + ankle) were equal to the contralateral limb or comparable to healthy values [127]. The relative contribution of each individual segment may be varied in the presence of pathology, but the sum is likely to be the same [127]. This finding indicates that although the knee extensor moment is decreased in ACL-R individuals, there is a relative increase in hip and ankle extensor moments [127].

Vertical Jump

Compared with healthy individuals, ACL-R individuals demonstrated decreased knee extension moments during vertical jump takeoff and landing [127]. Subjects also demonstrated decreased summated extensor moments (hip + knee + ankle) during vertical jump landing [127]. Summated extensors moments were not significantly different between groups for vertical jump takeoff, but were significantly different for the landing portion of the vertical jump. This finding would indicate that although the summated extensors moments were equal, the extensor moments of the hip and ankle were increased to compensate for the decreased knee extensor moment to preserve function of the lower extremity [127]. This observation was similar to findings during the step-up task. Forces during landing place the most stress on the system and that is why this task demonstrates the greatest differences [127].

FUNCTION

Instruments commonly used to assess subjective outcomes in people who have knee injury are numerous. The International Knee Documentation Committee (IKDC) subjective evaluation form was developed and validated as a "knee specific" outcomes instrument that was designed to "detect improvement or deterioration in symptoms, function and sports activity in persons with knee injury" [128–131]. Although this instrument was designed as an outcomes instrument for general knee injuries, it has been used extensively in clinical research in ACL-D and ACL-R populations. The most widely accepted scoring convention for the IKDC subjective knee evaluation form includes a normalized sum of response scored [128–131], wherein a score of 100 indicates the patient perceives no limits to function. Greater perceived limitations to function are indicated by reduced score. In retrospective study designs, a score greater than 70 can be interpreted as a successful subjective outcome; however, the scale is most effective as it is responsive to changes in perceived function over time. Other common outcomes instruments that have been used to track outcomes in knee-injured populations include the Lysholm knee scale [132], the

knee disorders subjective history [133], and the Cincinnati knee score [134]. Because of the high prevalence of osteoarthritis in the ACL-injured and -reconstructed population, the Western Ontario and McMaster Universities Osteoarthritis index (WOMAC) [135] may be used in mid- and long-term outcomes studies in this population. Because ACL reconstructions are most common in young and active populations, self-reported activity rating instruments, such as the Tegner activity scale [132], are commonly used in ACL outcomes research. Finally, an extension of the WOMAC was created to evaluate short- and long-term subjective outcomes, including symptoms and function in young, physically active patients who had knee injury and osteoarthritis [136].

Recent clinical studies reporting only subjective outcomes are rare; however, subjective instrument use is ubiquitous in orthopaedic outcomes research and is typically presented descriptively or as comparisons between treatment groups or over time. Several recent studies have reported excellent subjective outcomes in ACL-R [137–139] and ACL-D [66,140] patients in the short term (2 years) [141] and mid term (5–15 years) [66,137,140]. Although ACL-R and ACL-D individuals seem to report similar levels of postinjury outcomes, people who have ACL-D knees may be achieving optimal outcomes by modifying or reducing their activity levels [66]. Although it is certainly possible for the ACL-D knee to participate in a preinjury level of activity or sport, meniscus or cartilage injury may be likely [142]. There does not seem to be a gender bias in subjective outcomes following ACL-R [143,144]; however, females may exhibit slightly greater knee laxity during clinical examination [143]. Overall, it is possible to achieve excellent outcomes and maintain a healthy and active lifestyle following ACL injury or reconstruction and there does not seem to be a difference between various graft choices or between males and females. Achieving optimal perceived outcomes and patient satisfaction remains paramount in the continuum of care for the injured athlete. Heightened risk for long-term injury and knee joint degeneration, which certainly reduces subjective outcomes, may involve neuromuscular factors that go unnoticed in the short and mid term.

SUMMARY

ACL injury and surgical reconstruction have been shown to alter lower extremity kinematics, kinetics, and temporal variables during gait. The compensatory strategy is believed to be task dependent with more difficult tasks accentuating adaptations [115,117]. Biomechanical adaptations are influenced by the time elapsed since injury [118,119] and can return to a pattern similar to uninjured subjects following surgical reconstruction and rehabilitation [19,52,118].

ACL injury seems to affect lower extremity performance during functional activities and gait. Alterations in strength may be attributable to dramatic changes in muscle activation strategies of the lower extremity, particularly the inhibition of the quadriceps and activation of the hamstring muscle groups. Although altered motor patterns may be a protective mechanism following

injury, evidence suggests they persist long after ACL-R, suggesting that neuromuscular function needs to be addressed during rehabilitation.

Functional outcomes following ACL-R are generally excellent; however, persistent somatosensory and neuromuscular deficits and possible biomechanical aberrations may help explain why ACL-injured people are likely to experience early-onset knee joint osteoarthritis.

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