THE RELATIONSHIP BETWEEN FUNCTIONAL CAPACITY, MUSCLE SIZE AND STRENGTH IN CHRONIC ACL DEFICIENT AND ACL RECONSTRUCTED INDIVIDUALS.

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ABSTRACT: ***Aim:*** *The aim of this study was to assess the relationship between the quadriceps muscle atrophy and strength deficits caused by chronic ACL deficiency, and to ascertain whether these deficits were rectified in subjects who had undergone ACL reconstruction.*

Methods: *Thirteen ACL deficient subjects (ACLD) and eight subjects who had undergone ACL reconstruction (ACLR) participated in the study. Functional capacity, lean thigh volume (LTV), and isokinetic peak torque of the quadriceps and hamstrings muscles were assessed.*

Results: *The ACLD group had a significantly lower score for episodes of giving way compared to the ACLR group (7.4 ± 3.8 vs. 18.0 ± 3.7; $p < 0.01$) and a lower score for inability to perform jumping/twisting activities (1.8 ± 0.8 vs. 3.6 ± 1.1; $p < 0.01$; ACLD vs. ACLR) suggesting decreased functional capacity. However, there was no significant difference between the ACLD group and ACLR group for LTV differences (416.0 ± 276.5 vs. 238.3 ± 224.4 cc) and quadriceps eccentric peak torque differences (38.1 ± 13.7 vs. 23.7 ± 18.3 Nm) between involved and uninvolved limbs. The relationship between LTV and quadriceps isokinetic peak torque was $r = 0.59$ ($p < 0.05$) for the ACLD group and $r = 0.50$ (NS) for the ACLR group.*

Conclusion: *Quadriceps strength deficits are present in ACL deficient subjects, particularly during eccentric contractions. ACL reconstruction improved subjective function and reduced the episodes of giving way, but did not prevent eccentric quadriceps muscle weakness. A low correlation exists between reported function and LTV and eccentric peak torque activity, and between LTV and eccentric peak torque in both ACLD and ACLR groups. These findings suggest that factors other than muscle atrophy are responsible for the functional changes described in ACL deficient and ACL reconstructed groups.*

KEYWORDS: ISOKINETIC; ECCENTRIC; CONCENTRIC; TORQUE; LEAN THIGH VOLUME.

INTRODUCTION

The intact anterior cruciate ligament (ACL) is one of the primary stabilizers of the knee joint, and prevents excessive anterior tibial translation. In ACL deficiency, functional activity is compromised (Noyes *et al* 1983), and subjects report increased episodes of giving way and instability around the knee joint of the affected limb (Noyes *et al* 1983).

With ACL deficiency, the surrounding knee musculature, particularly the hamstrings muscles, act as secondary stabilizers of the knee joint to prevent anterior tibial translation and episodes of instability and giving way (Solomonow *et al* 1987). However, quadriceps muscle weakness is present in ACL deficient subjects (Eastlack *et al* 1999), and it has previously been suggested that this weakness may be caused by altered neuromuscular recruitment activity as a protective mechanism (Solomonow *et al* 1987; Valeriani *et al* 1996), to prevent the anterior tibial translation force which is part of the natural action of quadriceps muscle activity. Paradoxically, studies have shown that ACL deficient subjects who perform post-injury rehabilitation programs show improved quadriceps muscle strength capacity and improved muscle functional capacity (Seto *et al* 1988).

ACL reconstruction, particularly with the bone-patellar tendon-bone autograft, has been shown to improve functional capacity (Cameron et al 1995). However, the management of the ACL reconstructed limb is still controversial, as patients who have undergone ACL reconstruction demonstrate significant weakness of the quadriceps muscle (Delitto *et al* 1988; Natri *et al* 1996), despite reporting less frequent episodes of instability and giving way.

This quadriceps weakness after ACL reconstruction may be a residual consequence of the surgical procedure on the

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patellar-femoral joint (PFJ) (Shelbourne *et al* 1984), caused either by graft harvesting (Breitfuss *et al* 1996) or by tourniquet-induced quadriceps ischaemia (Gersoff *et al* 1989). While this hypothesis has merit, it is not clear why the ACL deficient subjects also have significant quadriceps muscle strength deficits, as they have no possibility of intra-operative knee joint morbidity. It is also not clear whether the reported improved general functional capacity in ACL reconstructed subjects can be related to changes in muscle strength or size deficits, given that significant strength deficits have been previously reported, despite the improved general functional capacity (Delitto *et al* 1988).

Accordingly, the aim of this study was to i) assess the extent of muscle changes caused by chronic ACL deficiency, and to determine whether these changes were rectified in subjects who had undergone ACL reconstruction; and ii) to assess whether the muscle changes in ACL deficient and reconstructed subjects were similarly or differently related to functional capacity.

METHODS

Patients

The study was approved by the Ethics and Research Committee of the University of Cape Town and all subjects signed an informed consent prior to the start of the trial. The ACL deficient (ACLD) subjects used in the trial were randomly selected from volunteers who satisfied the following inclusion criteria: i) the ACL rupture occurred between one and fifteen years previous to the trial; ii) the ACL rupture was diagnosed by an orthopedic surgeon and managed conservatively; iii) the normal contralateral knee joint had no previous injury; and iv) subjects had no other medical problems. An additional inclusion criteria for the ACL reconstructed (ACLR) group was that the subject's ACL was reconstructed surgically at least one year or more previously. In both ACL groups, the uninjured limb served as an internal control. Because the subjects' uninjured limb served as an internal control, age and gender were not regarded as exclusionary criteria. Thirteen ACLD subjects and eight ACLR subjects satisfied the inclusion criteria and participated in the trial.

In the ACLR group, six subjects' ACL were repaired using bone-patellar tendon-bone grafts, one using a semitendinosus graft, and one using a primary repair. Three ACLD subjects injured their menisci during the same episode causing the ACL injury. These meniscal injuries were all repaired immediately after the injury occurred, although the ACL was managed conservatively. All subjects in both ACLD and ACLR groups underwent home-based rehabilitation programs in the acute phase after both ACL injury and reconstruction.

Injury Score

A detailed history was recorded from each subject describing the episode which led to the ACL rupture and the post-injury symptomatology, using the Cincinnati functional rating scale (Noyes *et al* 1984). The ACLD and ACLR subjects completed an additional physical activity assessment score, rating level of activity from 0 - 5 before and after their ACL injury.

Magnetic resonance imaging (MRI) scanning

Each ACL deficient subject underwent a MRI scan (Esaote Biomedica Artoscan, Genoa, Italy) of the damaged limb to verify complete ACL deficiency. A scout scan was performed to assess whether the knee was correctly centered. If the knee was correctly centered, a set of sagittal and transverse T1 weighted 2D scans were performed. For the sagittal section, 16 sections were recorded from the medial side of the knee joint. The sections were 5 mm thick, with a gap of 0.5 mm between each section. If the ACL was not visualized, or if movement artifact occurred, the scan was repeated.

Anthropometry

Each subject's height and mass was recorded, and their body fat was assessed using the sum of the skinfold measurements of the right triceps, biceps, subscapular, supra-iliac skinfold sites (Durnin and Womersley 1974). In addition, the anterior mid-thigh skinfold measurement, the sub-gluteal, mid-thigh and above-knee circumferences were recorded in both limbs to calculate the lean thigh volume (LTV) of the involved and uninvolved limb. This technique for estimating LTV assumes the upper limb to have the shape of a truncated cone, which was adapted from Katch and Katch (1974). The technique has been validated against LTV assessed by MRI (Knapik *et al* 1996).

Isokinetic testing of skeletal muscle function

All subjects were tested on a Kin-Com isokinetic dynamometer (Chattanooga Group Inc., USA). Subjects were tested in a sitting position with a 100° angle of hip flexion. The hips, thighs and upper body of all subjects were firmly strapped to the seat of the dynamometer. The axis of rotation of the dynamometer arm was visually aligned with the lateral femoral condyle, and the lower leg was attached to the dynamometer at a level slightly above the lateral malleolus. The knee extensors and flexors were tested both concentrically and eccentrically at a testing speed of $60°s^{-1}$. The range of motion was between 7° of flexion and 83° of flexion, with the reference point being full extension. Tis *et al* (1993) showed that maximal torque is not altered by variations in the range of motion during isokinetic activity testing. The subjects were warmed up using submaximal concentric and eccentric contractions for five repetitions before testing commenced. The subjects performed three maximal trials for each test. The subjects were verbally encouraged to exert maximal effort during each test. The highest peak torque achieved during these three tests was used for subsequent analysis. Both involved and uninvolved limb were tested in all subjects. As the uninvolved limb served as an internal control, gravity correction factors were not applied to the data.

Statistics

All data are expressed as the difference between injured and uninjured limbs in the ACLD and ACLR groups. All data are expressed as mean ± standard deviation. A paired T test was used to compare data of the differences between uninvolved and involved limbs of ACLD and ACLR groups. An independent T test was used to compare differences in strength and size data of the involved

limbs of ACLD and ACLR groups. Statistical significance was accepted when $p < 0.05$. A Wilcoxon matched pairs test was used to analyze the non-parametric functional scale data. Pearson's product moment correlation and Spearman's rank order correlation were used to determine relationships between parametric and non-parametric variables respectively.

Table 1. Descriptive data of the subjects in the ACL deficient (ACLD) (n = 13) and the ACL reconstructed (ACLR) (n = 8) groups.

	ACLD	ACLR
Age (years)	37.8 ± 11.5	35.9 ± 6.1
Stature (cm)	173 ± 8	174 ± 7
Body mass (kg)	74.9 ± 14.3	79.1 ± 13.0
Body fat (%)	22.3 ± 7.2	24.1 ± 5.2

All values are mean ± SD

Table 2. The time period (years) since ACL injury occurred in the ACL deficient (ACLR) and the ACL reconstructed (ACLR) groups.

	ACLD	ACLR
1-5 years	n = 3	n = 4
6-10 years	n = 4	n = 3
11-15 years	n = 6	n = 1

Table 3. Physical activity levels of the ACL deficient (ACLD) (n = 13) and ACL reconstructed (ACLR) (n = 8) groups pre- and post-injury.

		Activity Level				
		0	1	2	3	4
ACLD	Pre-injury	0	0	0	0	13
	Post-injury	0	1	9	3	0*
ACLR	Pre-injury	0	0	0	0	8
	Post-injury	0	0	1	7	0*

All data are n subjects in each category: (0 - no activity; 1 - sedentary activity; 2 - light activity; 3 - moderate activity; 4 - strenuous activity)

* - $p < 0.05$ ACLD Pre-injury vs. Post-injury
ACLR Pre-injury vs. Post-injury

Table 4. Cincinnati functional score rating for the affected limb of ACL deficient (ACLD) (n = 13) and ACL reconstructed (ACLR) (n = 8) groups.

	MAX	ACLD	ACLR
Pain	20	11.7 ± 4.0	15.5 ± 2.6*
Swelling	10	7.9 ± 2.3	8.3 ± 2.3
Giving way	20	7.4 ± 3.8	18.0 ± 3.7**
Overall activity	20	12.3 ± 2.9	16.0 ± 3.0*
Walking	10	8.7 ± 1.9	9.0 ± 1.5
Stair climbing	10	8.6 ± 1.8	9.5 ± 0.9
Running	5	3.3 ± 1.1	4.3 ± 0.7*
Jumping/twisting	5	1.8 ± 0.8	3.6 ± 1.1**
Total	100	62.0 ± 10.0	83.6 ± 7.2**

All values are mean ± SD.

* - $p < 0.05$ Pain ACLD vs. ACLR
Running ACLD vs. ACLR

** - $p < 0.01$ Giving way ACLD vs. ACLR
Jumping/Twisting ACLD vs. ACLR
Total ACLD vs. ACLR

RESULTS

There were no significant differences between age, stature, mass or estimated body fat between ADLD and ACLR groups (Table 1). All subjects had injured their ACL or had undergone surgical reconstruction of their injured ACL between one and 15 years previously. The length of time since the injury occurred is described in Table 2.

Table 3 describes the level of physical activity in ACLD and ACLR groups prior to and after their ACL injury. Although all subjects in both groups decreased their level of physical activity after suffering the ACL injury, there were all able to continue with recreational activities of daily living.

Table 4 describes the functional capacities of the two groups as rated by the Cincinnati functional rating scale. A significantly higher number of episodes of giving way ($p < 0.01$) (Figure 1), increased pain symptoms ($p < 0.05$), decreased overall subjective activity ($p < 0.05$), decreased ability to perform jumping/twisting activities ($p < 0.01$), decreased running ability ($p < 0.05$), and lower total score ($p < 0.01$) were reported by the ACLD compared to ACLR group.

The LTV of the involved limb was significantly lower in the involved limb compared to the uninvolved limb in both ACLD (3542 ± 905 vs. 3126 ± 693 cc; involved vs. uninvolved limb; $p < 0.01$) and ACLR (3909 ± 841 vs. 3670 ± 849 cc; involved vs. uninvolved limb; $p < 0.05$) groups. The difference between involved and uninvolved limb was greater in the ACLD compared to ACLR group (416 ± 276 vs. 238 ± 439 cc; NS), although this difference was not significant (Figure 2).

The quadriceps of the involved limb was significantly weaker than the uninvolved limb in both ACLD and ACLR groups (Table 5) during both concentric and eccentric exercise. This difference in strength between involved and uninvolved limb was greater during eccentric than concentric activity in both ACLD (38.1 ± 13.7 vs. 16.6 ± 19.1 Nm; eccentric vs. concentric) and ACLR (23.7 ± 18.3 vs. 17.5 ± 12.4 Nm; eccentric vs. concentric) groups (Figure 3). The difference between maximal quadriceps concentric and eccentric

Table 5. Isokinetic strength data for the differences between involved and uninvolved limbs quadriceps and hamstrings muscles of the ACL deficient (ACLD) (n = 13) and ACL reconstructed (ACLR groups) (n = 8).

	ACLD			ACLR		
	Norm	Dam	Diff	Norm	Dam	Diff
Quadcon	155.0 ± 51.6*	125.3 ± 44.3	16.6 ± 19.1	174.7 ± 61.2**	143.4 ± 53.0	17.5 ± 12.4
Quadecc	207.0 ± 63.9**	129.2 ± 50.8	38.1 ± 13.7	220.3 ± 102.1*	158.9 ± 62.2	23.7 ± 18.3
Hamcon	92.6 ± 28.0	86.3 ± 22.1	4.5 ± 12.3	93.0 ± 26.4	92.0 ± 36.6	2.6 ± 16.5
HamEcc	104.1 ± 27.3*	94.3 ± 28.3	9.8 ± 10.8	97.4 ± 33.3	92.3 ± 41.4	7.1 ± 18.1

All values are mean ± SD.

* - $p < 0.05$	Quadcon	ACLD Involved vs. ACLD Uninvolved
	Quadecc	ACLR Involved vs. ACLR Uninvolved
	Hamecc	ACLD Involved vs. ACLD Uninvolved
** - $p < 0.01$	Quadcon	ACLR Involved vs. ACLR Uninvolved
	Quadecc	ACLD Involved vs. ACLD Uninvolved

strength in the uninvolved limb was ~25% for the ACLD group and ~21% for the ACLR group. In contrast, in the involved limb, the difference was ~3% for the ACLD group and ~10% for the ACLR group.

There were no significant differences in hamstrings concentric strength between the uninvolved and the involved limb in both ACLD and ACLR groups, and in the hamstrings eccentric strength of the ACLR group. However, the eccentric strength of the hamstring muscles of the involved limb in the ACLD group was significantly weaker than that of the uninvolved limb ($p < 0.05$) (Table 5).

There were no significant differences in peak torque angle for the quadriceps or hamstrings muscles of the involved or uninvolved limb in either the ACLD or ACLR groups (Table 6).

The relationship between LTV and quadriceps isokinetic peak torque was $r = 0.59$ ($p < 0.05$) for the ACLD group and $r = 0.50$ for the ACLR group. The relationship between Cincinnati rating score and LTV was $r = 0.44$ for the ACLD group and $r = -0.56$ for the ACLR group. The relationship between the Cincinnati rating score and quadriceps eccentric peak torque was $r = 0.43$ for the ACLD group and $r = -0.37$ for the ACLR group.

Table 6. Peak torque angle data for the quadriceps and hamstrings musculature of the ACL deficient (ACLD) (n=13) and ACL reconstructed (ACLR) (n = 8) groups.

	ACLD		ACLR	
	Norm	Dam	Norm	Dam
Quadcon	61.0 ± 8.6	62.0 ± 5.5	60.3 ± 10.2	65.1 ± 7.0
Quadecc	67.8 ± 8.1	60.2 ± 12.7	63.4 ± 10.9	63.7 ± 5.1
Hamcon	26.2 ± 11.1	30.6 ± 18.8	24.9 ± 16.5	24.6 ± 10.5
HamEcc	19.8 ± 12.3	24.7 ± 14.1	24.1 ± 19.5	29.4 ± 12.2

All values are mean ± SD.

DISCUSSION

In this study, there were no significant differences between the impairment in quadriceps isokinetic muscle function in the ACL deficient and the ACL reconstructed groups even though the ACLR group had better general function in their damaged limb compared to the ACLD group. Others have reported similar strength deficits after ACL reconstruction (Delitto *et al* 1988). Hence ACL reconstruction improves the individual's functional ability but not quadriceps isokinetic strength in this population group. It has been suggested that intra-operative insult, particularly to the patellar-femoral joint (PFJ) may be the cause of the quadriceps strength deficits present after ACL reconstruction (Amendola and Fowler 1992). However, this study indicates that the isokinetic strength deficits in the ACL reconstructed group may be intrinsic to the loss of the ACL itself, and may not be related to the intra-operative procedures, as there were no significant differences in the eccentric strength deficits present in the two chronic ACL deficient groups. The possibility exists also that in the ACL reconstructed individuals, the muscle strength deficits were the result of a combination of the loss of the ACL itself, and intra-operative PFJ complications occurring concurrently. However, if this were so one would expect greater strength deficits in the ALCR compared to ACLD group. But, in this study the strength deficits of the involved limb were less in the ACLR group.

Several studies have shown that there is a strong relationship between LTV and both quadriceps and hamstring isokinetic concentric and eccentric peak torque (Castro *et al* 1995) in uninjured individuals. In contrast a weaker relationship was found between LTV and eccentric peak torque in both ACLD ($r = 0.59$) and ACLR ($r = 0.50$) groups in this study. This suggests that factors other than the loss of muscle mass may have contributed to the loss of eccentric peak torque. As the majority of subjects were injured five years or more previously, and the minimum duration since injury was one year, it may be assumed that these muscle changes are permanent. It is not clear why this poor correlation between LTV and peak torque occurs in both ACLD and ACLR groups. This finding may be caused by i) submaximal recruitment of the knee extensors during testing; ii) inability of the subjects to maximally recruit their knee extensors due to alteration in the muscle contractile apparatus; iii) inability of the subjects to maximally recruit their knee extensors during eccentric testing due

Figure 1. Cincinnati functional score for reported episodes of giving way and jumping/twisting activities in the involved limb of the ACL deficient (ACLD) and ACL reconstructed (ACLR) groups.

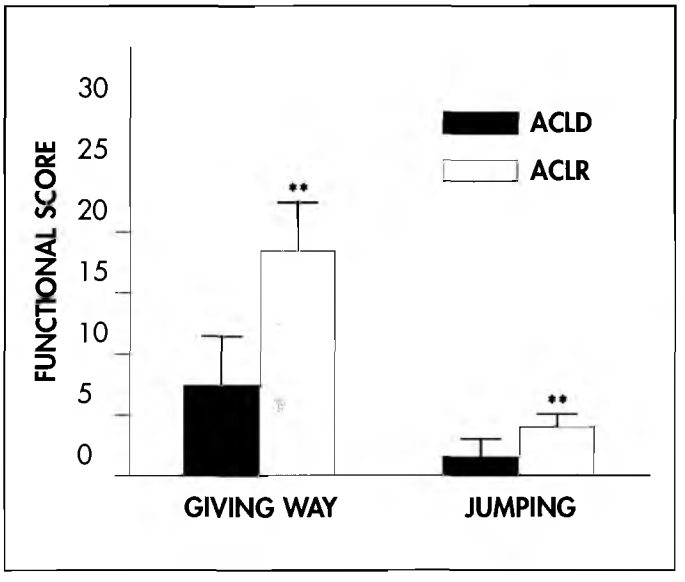


Figure 2. Lean thigh volume (LTV) (cc) differences between the uninvolved and involved limb of the ACL deficient (ACLD) and ACL reconstructed (ACLR) groups.

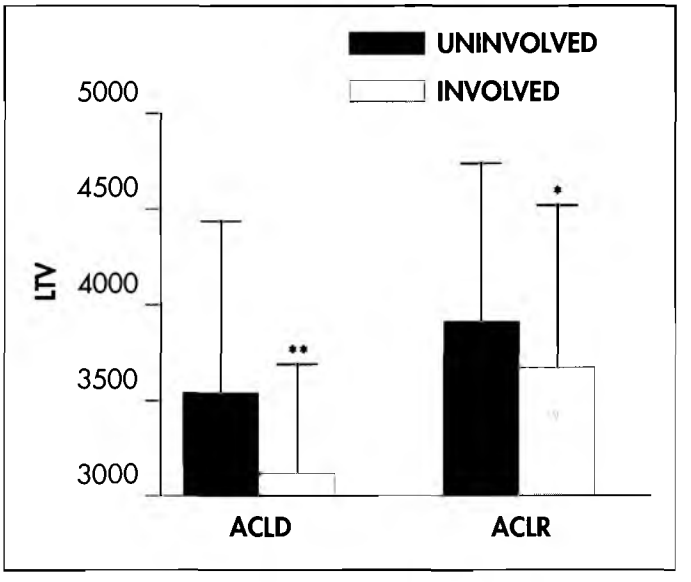
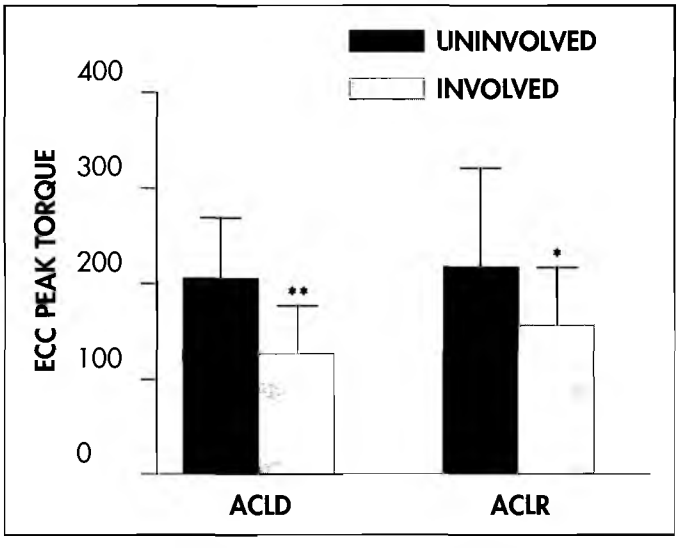


Figure 3. Quadriceps eccentric peak torque (NM) differences between the uninvolved and involved limb of the ACL deficient (ACLD) and ACL reconstructed (ACLR) groups.



to changes in efferent neural activity (Valeriani *et al* 1996) or altered proprioceptive input from the knee joint after ACL injury and reconstruction (Gleeson *et al* 1999).

If alterations to the muscle apparatus caused the strength deficits, one would except muscle to show signs of atrophy of the tissue itself. However, although studies have show that in the acute phase after ACL injury there is morphological muscle atrophy (St Clair Gibson 1997), studies of muscle morphology in subjects with chronic ACL deficiency or reconstruction with large strength deficits have failed to show morphological muscle atrophy (Lorentzon *et al* 1989). However, one can not exclude the possibility that changes to the tendon or elastic components of the quadriceps muscle caused the decreased strength output.

Several researchers have postulated that strength deficits after ACL rupture may be caused by alteration in afferent proprioceptive input inhibiting quadriceps strength output (Solomonow *et al* 1987; Valeriani *et al* 1996). This hypothesis may explain our findings, as although in the ACL reconstructed group, the ACL has been mechanically replaced by other tissues, no studies have shown that proprioceptors present in the native ACL and destroyed by ACL disruption are precisely maintained in the replacement graft tissue, and thus proprioceptive input would not be improved by ACL reconstruction. Also, studies have shown that after ACL rupture or reconstruction, subjects score significantly lower for proprioceptive testing in the involved limb (Gleeson *et al* 1999). Other studies have suggested that there might be reprogramming of the efferent CNS command to the muscle surrounding the knee joint after ACL disruption, either as a protective or a compensatory mechanism (Valeriani et al 1996).

A significant number of ACLD subjects scored poorly in their ability to perform activities such as running, jumping, walking up stairs, and in particular the ability to rotate on the injured knee. A large proportion indicated that the unexpected "giving way" or collapse of the affected limb was a major problem. A high proportion or athletes do not return to their previous levels of sports participation after ACL injury, either due to poor knee function (Solomonow *et al* 1987) or fear of further injury (Bjordal *et al* 1997). The poor relationship between the Cincinnati functional rating scores and both LTV ($r = 0.44$) and isokinetic peak torque deficits ($r = 0.43$) in the ACLD group indicates that LTV and peak torque deficits do not predict the Cincinnati score. The finding that the ACLR subjects had significantly less episodes of instability and giving way than ACLD subjects, despite not significantly different LTV and peak torque deficits, similarly indicates that the functional ability of the subject is not strongly related to the underlying muscle pathology.

It is unlikely that the changes in LTV and quadriceps eccentric peak torque in both ACL groups can be attributed solely to disuse atrophy as the uninjured limb of each subject was

used as an internal control. Although the differences between involved and uninvolved limb may have been caused by the subjects favouring the uninvolved limb after the injury, this difference would still be a direct result of the ACL injury itself. Indeed, studies have shown that permanent gait changes occur in both limbs either after chronic ACL deficiency or ACL reconstruction (DeVita *et al* 1997; St Clair Gibson 1997). However, if this was the reason for these findings, one would expect quadriceps and hamstring muscles to be similarly affected, which was not the case in this study.

It must be noted that the subjects recruited by this trial responded to an advertisement, and may have represented a group of subjects with more abnormalities than those of other studies of ACL deficient or reconstructed individuals. Eastlack *et al* (1999) described subjects who returned to previous activity as "copers", those that avoid causing episodes of instability in the involved limb by decreasing their activity levels as "adapters", and those that are unable to return to their pre-injury level of activity because of repeated episodes of giving way as "non-copers". They found that acute or chronic "non-copers" had greater strength deficits in the involved limb than "copers", but did not elaborate on why the different subjects had different strength deficits. On the basis of these descriptions, our subjects would be described generally as "non-copers", both because of their poor functional capacity and large strength deficits. Therefore, the results of our study should be taken to represent only this "non-coper" group.

The reason for the large strength deficits may be related to level of rehabilitation of the subjects after ACL rupture or reconstruction. All the subjects in this trial had no formal rehabilitation, instead using home-based self-driven rehabilitation protocols. Other studies have reported lesser strength deficits than that found in our study (Shelbourne and Gray 1984). Therefore, the changes in our study may have been caused by poor post-injury rehabilitation. However, the fact that the length of time since injury was five years or longer in most subjects weakens this hypothesis, as most subjects had resumed physical activity or performed routine activities of daily living.

As discussed previously, quadriceps isokinetic peak torque strength measurements in the ACLR group and the ACLR groups were significantly lower than in the involved limb. These strength deficits were present to a greater degree during eccentric compared to concentric activity. There are two possible reasons for these findings. Firstly, there may be a greater relative decrease in strength caused by the fact that eccentric torque output is greater than concentric torque output, thus would be more affected by whatever process is causing the decreased quadriceps force output. Secondly, the eccentric deficits may be related to the hypothesis that eccentric activity has different neural control mechanisms to concentric muscle activity (Enoka 1996). Therefore eccentric activity may altered by different efferent instructions to those of concentric activity, if neural activity changes are responsible for the strength deficits in the ACL deficient and ACL reconstructed subjects.

In contrast, the hamstring musculature of all groups did not exhibit significant differences in either concentric or eccentric isokinetic peak torque except in the ACLD group during eccentric activity. This finding is expected, as studies have shown the hamstrings muscles are natural synergists of the ACL, with increased activity in ACL deficient and reconstructed subjects to prevent anterior tibial translation, perhaps as a reflex action (Solomonow *et al* 1987).

The majority of the ACLR subjects in the trial underwent bone-patellar tendon-bone ACL reconstruction, with the remainder undergoing semitendinosus reconstruction or primary repair. These data are similar to that described in demographic studies (St Clair Gibson *et al* 1998) of the management of ACL injuries. Although the ACLR group had diverse surgical procedures, the strength of the study was that that subjects were randomly recruited, which makes the results generally applicable.

In conclusion this study shows that chronic ACL deficiency results in muscle size and strength deficits in the quadriceps muscle of the injured limb. ACL reconstruction, despite improving limb stability, does not rectify either the quadriceps strength or muscle deficit completely in this population group. A poor correlation exists between reported function and LTV and eccentric peak torque activity, and between LTV and eccentric peak torque in both ACLD and ACLR groups. These findings suggest that factors other than muscle atrophy are responsible for the changes in the involved limb of the ACL deficient and ACL reconstructed groups.

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