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# Central Somatosensory Changes and Altered Muscle Synergies in Subjects with Anterior Cruciate Ligament Deficiency

Carol Courtney

Rose Marie Rine

*Marshall University*, [rinero@marshall.edu](mailto:rinero@marshall.edu)

Penny G. Kroll

*Marshall University*, [kroll@marshall.edu](mailto:kroll@marshall.edu)

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# Central somatosensory changes and altered muscle synergies in subjects with anterior cruciate ligament deficiency

## Abstract

To clarify the mechanisms of compensation in subjects with anterior cruciate ligament deficiency (ACL-D), we examined proprioception, quadriceps strength, somatosensory evoked potentials (SEPs) and muscle responses during gait in varied combinations of speed and incline. Seventeen subjects with ACL-D were grouped by functional level and report of giving way. Seven subjects without ACL-D served as a control sample for muscle response measures. ACL-D subjects with quadriceps weakness experienced giving way and could not resume sport activities. Those without weakness fell into one of two groups: (1) copers, who had full return to previous activity and no giving way despite proprioceptive loss and altered SEPs, and (2) adapters, who were unable to return to previous activity level and experienced giving way despite neither proprioceptive loss or altered SEPs. The unique muscle pattern in copers during inclined fast walking included larger and earlier hamstring activation. These results suggest that in individuals with ACL-D without a strength deficit, altered SEPs and altered neuromuscular patterns are the factors that enable resumption of pre-injury functional levels. Loss of proprioception may drive the central changes, which in turn drives the development of altered muscle patterns.

## 1. Introduction

Although, the role of somatosensory information in the control of gait is well documented [1], the mechanisms of compensation in individuals with peripheral somatosensory loss, such as those with complete tear of the anterior cruciate ligament (ACL-D), have not been clearly delineated. Some patients with ACL-D recover and return to sport (copers) [2–5] while others complain of instability and ‘giving way’ with basic activities (non-copers) [2–4]. Reports of the mechanisms responsible for recovery in copers, or the lack thereof in non-copers, have been conflicting [6–10]. Research has confirmed that the passive knee laxity in individuals with ACL-D is unrelated to the ability to return to sports [5]. It has been proposed that the lack of proprioception or central nervous system changes in response to proprioceptive loss may determine the different outcomes [11]. Inconclusive reports may be due to the divergent methodologies used and the lack of concurrent measurement of function, strength, proprioception and neuromuscular responses that is needed to answer this question.

### 1.1. Mechanisms of compensation

Proposed mechanisms for compensation following ACL-D include: (1) maintenance of proprioception at the knee as measured by threshold to detection of passive motion (TDPM) [8,10]; (2) activation of a capsular-hamstring reflex due to increased mechanical laxity at the joint [6,7] and (3) modulation of central programming [9,11]. Mechanoreceptors within the ACL contribute to the compilation of limb position information from numerous receptors [12,13] that enables the dynamic postural control needed for gait [1]. Reportedly, the giving way is related to higher thresholds on TDPM testing [8,10]. Johansson et al. [14] suggested that as a consequence of proprioceptive loss, the ensemble sensory feedback and existing motor program is disrupted, resulting in the ‘giving way’ reported. However, conclusions must be guarded. Weakness alone can result in giving way, and strength was not measured in these studies. Furthermore, to relate proprioceptive loss to alteration or lack of muscle response and consequent giving way requires measurement of neuro-muscular output in those with and without proprioceptive loss, which was not done in these studies.

Altered neuromuscular patterns have been reported in subjects with ACL-D during resisted knee extension and gait [6,15,16]. Investigators [6,16] concluded that poor functional outcome was due to peripherally mediated reflex stiffness at the knee, with changes in timing, not amplitude of muscle responses. However, neither proprioception nor strength was measured in any of these studies. Di Fabio et al. [9] postulated that the altered synergies may represent central reorganization.

Di Fabio et al. [9] reported earlier and less variable response of hamstrings in the affected ACL-D limb, as compared to the unimpaired limb during an external platform perturbation, and dominance of hamstring, as com-

pared to quadriceps, activity during unilateral stance on the ACL-D limb. These authors [9] concluded that the generalized increase in gain of hamstring response was due to increased input from the joint capsule of the involved limb, with secondary changes in central nervous system activity and the output generated. However, without concurrent measurement of proprioception and central changes, these conclusions may be overstated.

Somatosensory evoked potential (SEP) testing yields electrical recordings representative of activity in peripheral and central neural pathways in response to external stimuli. Using this test, investigators have demonstrated evoked potentials in response to stimulation of an intact ACL [17], and absence of the P27 potential in ACL-D subjects with proprioceptive loss [11]. Valeriani et al. [11] postulated that loss of the P27 potential represents central nervous system (CNS) reorganization. Although tempting to conclude that functional deficits in those with ACL-D are due to loss of proprioception and central changes, measures of function, leg strength, and neuromuscular activation patterns were not obtained.

To address the question of compensation, or the lack thereof, and to identify the mechanisms involved requires concurrent measurement of functional status, neuromuscular response patterns, proprioception and SEPs. The purpose of this study was to obtain these measures and subjective report of function in subjects with ACL-D, and to compare response measures from involved and un-involved limbs. Identification of the mechanisms enabling compensation and recovery may assist in the development of optimal interventions for individuals with ACL-D.

## 2. Methods

We compared proprioception and SEPs between groups of individuals with ACL-D and compared muscle synergies during gait between groups with ACL-D as well as to a normative sample.

### 2.1. Subjects

Seven males and ten females of 23–50 years of age (mean age 34.5, s.d. 8.6 years) with ACL-D who never had surgical repair agreed to participate. Seven 23- to 34-year-old individuals (six females and one male; mean age 27, s.d. 4.1 years) without ACL-D participated as a control sample for the treadmill walking test. Exclusion criteria for all subjects were: (1) history of any vestibular dysfunction; (2) uncorrected vision problems; (3) history of orthopedic lower extremity injury and (4) history of neurological disease or injury. Informed consent, as approved by the University of Miami Medical Sciences Review Committee, was obtained prior to testing.

ACL-D was confirmed by arthroscopy or MRI. All subjects were at least 2 months post-injury, had no swelling, pain, limiting gait deviation or limits in passive knee range of motion. Each subject completed a questionnaire regarding medical history and activity level and in the case of subjects with ACL-D, incidence of giving way and ability to return to cutting and pivoting sport activities. Based on reports of functional level and giving way, subjects were placed into one of two groups: (1) non-copers, who complained of numerous incidents of giving way during typical daily activities and difficulty with any sport activity, and (2) copers, who never experienced giving way and had a full return to sport activity, to include cutting and pivoting tasks.

### 2.2. Experimental procedures

#### 2.2.1. *Strength testing: maximum voluntary isometric contraction of the quadriceps*

The maximum voluntary isometric contraction (MVIC) force of the subjects' quadriceps femoris muscle was determined by using a burst-superimposition method on both lower extremities [18]. The subject was seated on an isokinetic dynamometer [19] with the ankle secured to the dynamometer force arm and the hip and knee placed at 90° (resting position). Self-adhesive stimulating electrodes were applied to the proximal vastus lateralis and the vastus medialis muscles. A supramaximal burst of electrical stimulation (100 pulses per second, 600 ms pulse duration, 10 pulse tetanic train) [20] was superimposed on a maximal voluntary contraction, with force recorded by the dynamometer. Three trials were completed on each limb to obtain the peak MVIC. Relative strength or quadriceps index (QI) of the involved leg was calculated from peak MVIC of each limb ( $QI = (\text{involved limb peak MVIC} / \text{uninvolved limb peak MVIC}) \times 100$ ). A QI less than 80 was indicative of quadriceps weakness.

#### 2.2.2. Somatosensory evoked potential testing

SEP testing [21] was performed with subjects lying on a plinth in a semi-darkened room. Surface stimulating electrodes were placed proximal to the knee in the popliteal fossa to stimulate the common peroneal nerve. Stimulus intensity was adjusted to just above motor threshold, with a 0.2 ms duration and frequency set at 1.5 Hz. Surface recording electrodes were placed over two scalp points, Cz and Fz using the international 10–20 system [11]. The electrode montage was as follows: (1) reference electrode was placed at the Fz position and the active electrode was placed at the Cz position, midway between the ears; (2) ground electrode was placed on the shoulder ipsilateral to the stimulated nerve. Impedance of the three electrodes was verified to be below 5 k. The average of 1000 trials was obtained for both limbs. Latencies and peak to peak amplitudes were calculated to identify the presence of the P27, with the normal limit being the mean + 3 standard deviations.

### 2.2.3. Knee proprioception testing

The protocol described by Barrack et al. [22] was used. Subjects were seated at a 60° incline to encourage relaxation, with the knee flexed to 40° and the ankle in the neutral position. They were blindfolded to prevent visual feedback. Air splints were placed on the lower leg and thigh and inflated to a pressure of 20 mmHg to minimize cutaneous sensation. Passive, slow (0.5 8/s) flexion or extension of the knee was accomplished by the use of a motor and pulleys attached to the air splint (Fig. 1). Subjects were instructed to indicate when they detected movement or change in position of the knee by pressing a switch placed in their hand, and to note the direction of movement. One trial was performed for each limb. The amount of linear movement of the pulley (X) was documented to calculate threshold to detection (Y, in degrees) using the following formula:

$$Y = \tan^{-1} \frac{X}{R}$$

where R = shank length (medial joint line of the knee to inferior aspect of the medial malleolus) [22]. Scores obtained on the affected knee were determined to be deficient if thresholds were greater than 45% of that of the unaffected knee.

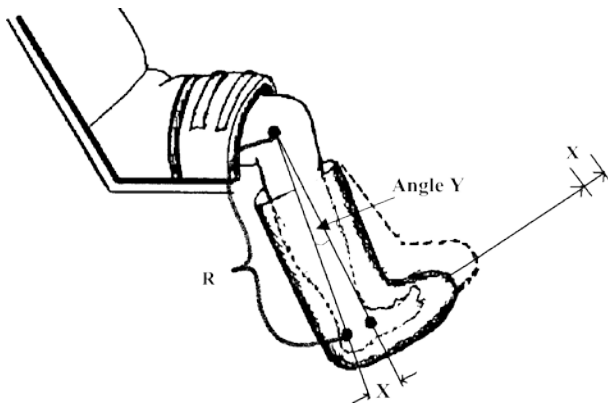


Fig. 1. Threshold to detection of passive motion (TDPM) apparatus. When subjects indicated detection of knee position change, movement of the pulley stopped. TDPM, angle Y in degrees, was calculated:  $Y = \tan^{-1}(X/R)$ , where R = shank length and X = linear movement of line on pulley.

### 2.2.4. Electromyographic recording during treadmill walking

To obtain measures of muscle group responses [23] Ag–AgCl surface electrodes were placed bilaterally on the rectus femoris, medial hamstrings, tibialis anterior and medial gastrocnemius during walking on a treadmill, at different combinations of incline and speed. Electrodes were placed

1.0 cm distal to the motor point of each muscle, 2.0 cm apart. Data were sampled at 1000 Hz, signals band passed between 20 and 500 Hz, amplified, rectified and filtered. Subjects walked at each of three different inclination/speed settings: (1) 2.5 km/h at 0 incline (slow level); (2) 4.0 km/h at 0 incline (fast level) and (3) 4.0 km/h at 25 incline (fast inclined), with sequence assigned in a random manner. A pressure switch at the heel of the shoe of the affected extremity marked heel strike onset (HS), enabling the calculation of muscle latencies (initB; in milliseconds (ms)) and amplitudes, as well as to delimit a stride for calculation of gait period (HS2 – HS1= period; in ms). Normalized latencies (MUSCLE<sub>lat</sub>) were calculated for each muscle group as a proportion of the period (MUSCLE<sub>lat</sub> = initB/period). Four strides were analyzed and relative latencies calculated: (1) gastrocnemius<sub>lat</sub>/tibialis anterior<sub>lat</sub> (GA<sub>lat</sub>/TA<sub>lat</sub>); (2) hamstrings<sub>lat</sub>/tibialis anterior<sub>lat</sub> (HAM<sub>lat</sub>/TA<sub>lat</sub>); (3) hamstrings<sub>lat</sub>/gastrocnemius<sub>lat</sub> (HAM<sub>lat</sub>/GA<sub>lat</sub>). Averages were used for analysis. Normalization of response amplitudes was performed by calculating the within subject ensemble average of four strides [24].

### 2.2.5. Analysis

Descriptive characteristics of ACL groups were obtained and analyzed. Mann–Whitney U-test was used to compare muscle responses attained by those with ACL-D to the normative group, as well as to compare the ACL-D groups, using SPSS for Windows, version 9.0. ANOVA and t-tests were used to compare proprioception test results between ACL-D groups and between limbs, respectively. Although significance level of 0.05 was established, a significance level of 0.10 was accepted to indicate a trend, due to small sample size.

## 3. Results

Of the 17 subjects with ACL-D, one refused SEP testing and one refused treadmill testing. All subjects in the study considered themselves at least minimally active (1-7 h of exercise/week). Time since injury ranged from 2 to 214 months (mean = 72, s.d. = 67.7 months), with equivalent distribution of less than one year and greater than two years between groups. Most ACL-D subjects had concomitant meniscal tears (12 medial and one lateral). Interestingly, based on reports of function and giving way, a third group, adapters, emerged. Individuals in this group reported that they had adapted their activities to avoid giving way. Adapters (n = 10; 59%) did not have a strength or proprioceptive deficit, reported giving way and a return to ‘guarded’ sport activity with no cutting or pivoting. Data is presented in Tables 1 and 2. Further analysis was based on three ACL-D groups.

### 3.1. Proprioception, somatosensory evoked potentials and strength

Interestingly, those with the higher level of function (copers) had a proprioceptive deficit and altered SEPs. Non-copers had deficits in proprioception but did not present with altered SEPs (Tables 1 and 2). Although TDPM of the unaffected limb did not differ between ACL-D groups, TDPM for the injured knee of non-copers and copers was larger than that of adapters. Only non-copers had quadriceps weakness. Summary data are presented in Table 2.

### 3.2. Treadmill walking

On visual inspection of the data, a distinct second peak of gastrocnemius activation at the end of the gait cycle (average normalized latency of 0.76, s.d. 0.13) was observed in 50% the ACL-D group. Because measures from the non-coper group were inconsistent, possibly due to the quadriceps weakness, EMG data from this group was omitted from further statistical analysis.

Characteristics of subjects with ACL-D by group

Subject no.	Age	Group	Months since injury	Giving way?	Cutting/pivoting	Weakness
1	29	Non-coper	94	Yes	No	Yes
2	38	Non-coper	7	Yes	No	Yes
3	36	Non-coper	214	Yes	No	Yes
4	25	Non-coper	45	Yes	No	Yes
Mean/mode	32		90 (90.01)	Numerous	No	Yes
5	22	Adapter	24	No <sup>a</sup>	No	No
6	23	Adapter	2	No	No	No
7	40	Adapter	96	No	No	No
8	45	Adapter	31	No <sup>a</sup>	No	No
9	41	Adapter	106	No <sup>a</sup>	No	No
10	23	Adapter	16	No	No	No
11	29	Adapter	142	No <sup>a</sup>	No	No
12	42	Adapter	7	No	No	No
13	31	Adapter	168	No	No	No
14	50	Adapter	2	No <sup>a</sup>	No	No
Mean/mode	36		59 (62.72)	No	No	No
15	32	Coper	140	No	Yes	No
16	44	Coper	60	No	Yes	No
17	36	Coper	8	No	Yes	No
Mean/mode	37		69 (66.49)	No	Yes	No

<sup>a</sup> Experienced giving way when injury occurred, but no longer.

Table 2  
Strength, proprioception and SEPs by ACL-D group

Group	QI <sup>a</sup>	TDPM <sup>b</sup>		SEP <sup>c</sup>	Amp <sup>d</sup>	Relative latency (slow level walking) <sup>e</sup>			Relative latency (fast inclined walking) <sup>e</sup>		
		ACL-D	Non-ACL-D			GA/TA	HA/TA	HA/GA	GA/TA	HA/TA	HA/GA
Adapters, n = 10	94	0.54 (0.11)	0.54 (0.12)	10 (-)	GA	0.26	1.30	5.84	0.25	1.45	11.64
Copers, n = 3	96	1.46 (0.43)	0.68 (0.24)	3 (+)	HA	0.43	1.35	3.20	0.35	1.00	2.84
Non-copers, n = 4	68	1.53 (0.61)	0.79 (0.27)	3 (-), 1 (+)	-	-	-	-	-	-	-

<sup>a</sup> Quadriceps index; value < 80 indicative of weakness.

<sup>b</sup> Threshold to detection of passive motion (in degrees); standard deviation in parentheses; values for affected and non-affected limb.

<sup>c</sup> Somatosensory evoked potential test; (+) indicates positive for loss of P27, (-) indicates it was normal.

<sup>d</sup> Amplitude; dominant muscle noted (GA = gastrocnemius; HA = hamstrings).

<sup>e</sup> Relative latencies of gastrocnemius/tibialis anterior (GA/TA), hamstrings/tibialis anterior (HA/TA), hamstrings/gastrocnemius (HA/GA).

During slow level walking, GA/TA ( $P = 0.014$ ) and HAM/GA relative latencies ( $P = 0.06$ ) differed between adapters and copers due to earlier gastrocnemius onset in the adapters. This trend was found during all walking conditions in adapters. In contrast, the normalized latency of gastrocnemius in copers resembled that of controls.

The trend of altered timing and difference between adapters and copers was evident during inclined walking. The HA/GA relative latency differed between these groups ( $P = 0.07$ ) due to earlier gastrocnemius onset in adapters and earlier hamstrings onset in copers. The earlier hamstring onset in copers also affected a difference in HA/TA relative latency between groups ( $P = 0.07$ ). These temporal differences are clearly evident in Fig. 2.

The amplitude of muscle activation differed between groups only during fast inclined walking. Specifically, copers demonstrated more hamstring activation and less gastrocnemius activation as compared to adapters or controls ( $P = 0.011$ ). Gastrocnemius activation of adapters was like controls. (Fig. 3).

#### 4. Discussion

Results presented here suggest that among individuals with ACL-D who do not have quadriceps weakness, altered neuromuscular patterns and SEPs are the factors that enable resumption of pre-injury functional levels. Furthermore,

our results suggest that loss of proprioception may drive changes in its central representation, which in turn facilitates the development of a more efficacious functional synergy. Interestingly, all subjects with altered SEPs had a proprioceptive deficit, but not all with a proprioceptive deficit had altered SEPs.

Within our subject population, despite similarities of age and time since injury, those functioning at the highest (copers) and lowest (non-copers) levels had a proprioceptive deficit, whereas those functioning at a moderate level (adapters) did not. Neither adapters nor non-copers had altered SEPs. However, based on reports that extensive training has a positive effect on TDPM [25,26], and non-copers had weakness of the quadriceps muscle, the interpretation of the TDPM scores in this group must be guarded. In contrast, copers had a proprioceptive loss and changes in synergy and SEPs. This, and the return to full sports activity in this group, suggests that loss of proprioception may drive these changes.

Copers, presented with loss of the P27 potential and neuromuscular patterns during treadmill walking, differed from controls. Hamstring activation was earlier and larger. The neuromuscular pattern of adapters also changed, but hamstring activation remained like that of controls, and gastrocnemius was activated earlier. Age cannot be a factor since most controls were within the age range of those with ACL-D. As previously suggested [27], the increased gastrocnemius activity in adapters may be an attempt, albeit insufficient, to stabilize the knee. The chronic status of our subjects would argue against the presence of a quadriceps avoidance pattern [28]. These results suggest that the change in SEPs is the critical element for the development of a more efficacious synergy to stabilize the knee (i.e. increased hamstring activity) in patients with chronic ACL-D.

The second major finding in our study is the identification of a third, heretofore not described group within the population of individuals with ACL-D: the adapters. In contrast to studies limited to copers and non-copers, the majority of our subjects with ACL-D did not experience ‘giving way’ during typical daily tasks, but had modified or adapted activities so that they did not experience problems. This lifestyle adaptation may explain, at least in part, the less than optimal return to sports activity in this group, despite similar time since injury and retention of proprioception.

Findings of concurrent proprioceptive loss, altered SEPs and altered synergies in those achieving the highest level of function (i.e. copers) suggest that central changes have occurred with consequent changes in motor

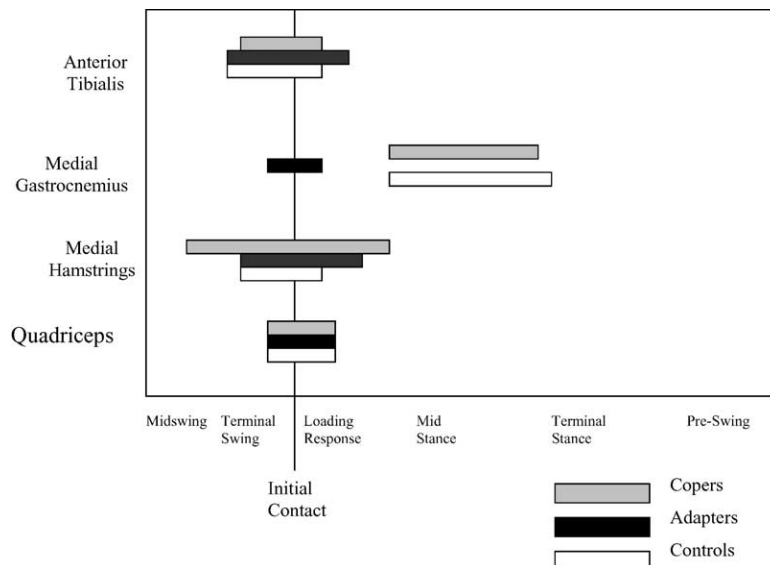


Fig. 2. Comparison of temporal muscle patterns. The earlier and longer hamstring activation in copers and earlier gastrocnemius activation in adapters are evident. Each bar represents the onset and offset of muscle activation within one period during fast-inclined walking.

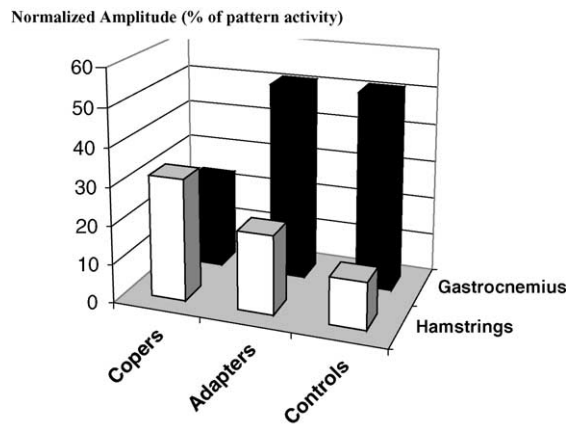


Fig. 3. Comparison of muscle group dominance. The plot of amplitudes, normalized to the within subject ensemble, reveals that hamstrings dominated muscle activity in copers, whereas gastrocnemius dominated the pattern in adapters and control subjects during fast-inclined walking.

output to successfully maintain joint stabilization. The lack of central change in the non-coper group may be a consequence of the quadriceps weakness, recurrent giving way and failure to return to activities that may facilitate compensatory changes. Similarly, the guarded activity of the adapters may have precluded central change or the selection of an efficacious synergy to stabilize the knee. The role of use or activity in the emergence of central change and selection of synergy is beyond the scope of this study, but should be pursued in future investigations.

While conclusions must be guarded due to small sample size, our results suggest that in humans, central somatosensory pathways may be modified by lesions to peripheral mechanoreceptors of the ACL. This change and continued use of the limb in situations that challenge knee stability may enable learning of an alternate synergy that minimizes instability and optimizes functional level. These results do not contradict the concept that a capsular hamstring reflex is the mechanism by which individuals with ACL-D stabilize the knee. However, the lack of a capsular reflex response in non-copers, and the identification of central changes presented here would argue against a spinal level mediated mechanism. Future studies should be completed with a larger sample of each of the ACL-D groups to provide support to the concepts presented here.

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