

## ANTERIOR CRUCIATE LIGAMENT INJURIES IN SPORT: ARE THEY PREVENTABLE?

Julie R Steele  
University of Wollongong, Wollongong, NSW, Australia

Anterior cruciate ligament (ACL) rupture is one of the most disabling knee injuries an athlete can sustain. Consequently, strategies by which this injury can be prevented are urgently required. Based on observations of the compensatory adaptations displayed by functional ACL deficient patients it is recommended that preventative strategies for non-contact ACL injuries should focus on reprogramming the locomotor processes used by athletes to control knee motion at foot-ground contact in landing. However, further research is warranted to determine if healthy athletes can be efficiently trained to use the same muscle recruitment strategies developed by functional ACL deficient patients to protect their knees and how effective these strategies are in minimising the incidence of non-contact ACL injuries.

KEY WORDS: anterior cruciate ligament, landing, injury prevention.

ANTERIOR CRUCIATE LIGAMENT INJURIES IN SPORT - THE DILEMMA: Rupture of the anterior cruciate ligament (ACL) is one of the most disabling knee injuries an athlete can sustain, especially the younger athlete (McCarroll et al., 1995). As early as 1939 the disabling nature of ACL ruptures was noted by Bristow who wrote:

*A completely ruptured crucial is a serious disability, the results of operative repair are poor, and it is unlikely that the knee will be stable enough to stand up to hard manual work, or strenuous games (p. 465).*

Although treatment regimes for an ACL rupture have altered radically since 1939, the injury remains the *nightmare* of both coaches and athletes. Unresolved ACL rupture has been associated with a distinct syndrome that affects all other components and total performance of the knee (Noyes and McGinniss, 1985). This syndrome is manifested by a predictable natural history typified by episodes of anterior and rotary instability, quadriceps atrophy, degeneration of the articular surfaces, meniscal damage, osteoarthritis, osteophyte formation, recurrent pain, and other symptoms of progressive knee dysfunction and deterioration. Prognosis for the ACL deficient knee therefore appears poor, particularly in young athletes who wish to continue participating in competitive sport (Hawkins et al., 1986). Consequently, strategies by which ACL ruptures can be prevented are urgently required. However, before effective prevention strategies can be developed we need to understand the mechanisms of ACL injuries in sport.

### HOW DO ACL INJURIES OCCUR IN SPORT?

**Non-contact** Mechanisms of ACL Injuries: Mechanisms of ACL injuries in sport can be classified into two main categories: (a) *contact injuries* caused when an external force is applied to the knee causing ACL injury; and (b) *non-contact injuries* caused when an indirect force is applied to the knee (Zarins and Nemeth, 1983). It has been estimated that 66 to 78% of ACL injuries occur via a non-contact mechanism (Baker, 1990; Noyes et al., 1983). Whereas contact injuries have mainly been attributed to chance, non-contact injuries are more related to characteristics of the individual suffering the injury, such as the degree of muscular weakness (Bender et al., 1964) or muscular co-ordination, and the movement pattern performed at the time of injury. Non-contact ACL injuries are therefore more feasible to be prevented.

**Deceleration and Tibial Rotation:** Numerous methods have been proposed to explain the manner by which the ACL can rupture in sport. Abrupt deceleration is one of the most frequent non-contact mechanisms of isolated ACL injury, particularly in field and court sports that involve running, sudden changes of direction, quick stops, twisting, and jumping movements (Daniel et al., 1994; Noyes et al., 1983). These injuries usually involve a combination of deceleration and internal tibial rotation with minimal knee flexion (Fetto and Marshall, 1980). Combined deceleration and rotation of the landing limb places anterior shear and rotary stress

upon the knee which, if excessive, can rupture the ACL (Cailliet, 1984). Such an injury can occur in activities where the athlete 'plants' their foot in an attempt to decelerate before suddenly changing direction as they quickly pivot or cut away from the fixed (planted) foot (Fantom, 1991).

**Deceleration Without Tibial Rotation:** Injury to the ACL in sport can also occur in non-contact deceleration situations that do not involve tibial rotation. For example, injury can occur when the athlete attempts to decelerate rapidly from open running with the foot fixed (Feagin et al., 1980) or lands suddenly from a jump or leap on one leg near extension and attempts to rapidly halt their forward progression (Baker, 1990; Fantom, 1991). Noyes et al. (1983) reported 22% of a sample of 103 chronic ACL deficient patients injured the ligament on landing from a jump.

#### WHY IS THE ACL SO VULNERABLE TO INJURY WHEN LANDING?

**Quadriceps Contraction and ACL Injury:** During initial deceleration during landing, the resistive ground reaction forces applied to the foot abruptly halts forward momentum of the body. This force is combined with a tractive force applied to the tibial tuberosity via the quadriceps-patellar tendon-retinacula mechanism (White, 1975). Quadriceps contraction results in compression forces of the articular surfaces at the centre of contact whilst simultaneously pulling the tibia forward (Cailliet, 1984). The angle between the patellar ligament and the tibia is greatest at knee angles between 20° of flexion and full extension (Nisell, 1985). Therefore, quadriceps contraction contributes significantly to shear forces acting across the knee in terminal extension, tending to displace the tibia anteriorly and loading the ACL (McNair and Marshall, 1994). Therefore, in abrupt landings in which the knee is only partially flexed, the ACL is particularly vulnerable to injury.

**Hamstring Contraction: An ACL Synergist:** By attaching posteriorly to the upper aspect of the tibia and fibula, hamstring contractions impart an increasingly posteriorly directed force on the proximal tibia as the knee flexes. Therefore, the hamstring muscles act synergistically with the ACL to resist abnormal anterior tibial translation (ATT) relative to the femur (Cross, 1996), negating increased ACL strain caused by quadriceps contractions (Beynon et al., 1995) and decreasing quadriceps induced ATT (Woo et al., 1992). For this reason, general recommendations for preventing ACL rupture or for rehabilitation of the ACL deficient knee following knee surgery have emphasised development of hamstring muscle strength to stabilise the tibia posteriorly relative to the femur (Collins, 1980). However, because their angle of insertion on the leg is acute, the hamstring muscles are ineffective in generating a posterior drawer force to reduce ATT at the extremes of knee extension (Shelburne and Pandy, 1997). Instead, the hamstring muscles act at full knee extension more to compress the tibia and femur during a quadriceps-hamstring muscle co-contraction (Yack et al., 1994). Furthermore, although hamstring co-contraction appears important for functional stability in the normal or ACL deficient knee, strengthening exercises in isolation probably are not sufficient (Lass et al., 1991). Maximising peak torque output via strengthening exercise alone would be important for facilitating knee stabilisation if functional activities such as landing demanded that the thigh muscles act maximally (McNair et al., 1991). However, isolated strengthening exercises do not increase the degree of muscle reaction (Lutz et al., 1991) and are not sufficient as a measure of success in neuromuscular rehabilitation after ACL injury (Zatterstrom et al., 1994). More important than developing strength in isolation is developing hamstring muscle coordination to ensure optimal recruitment of the muscles so that they can be used to reduce stress on the ACL imposed by the quadriceps muscles. Therefore, it has been advocated that rehabilitation programs for the ACL injured knee should include elements of muscle-coordination and neuromuscular-proprioceptive training of the knee rather than strengthening alone to account for deficits due to disruption of the ACL-mechanoreceptor reflex arc (Lass et al., 1991).

But could healthy athletes prevent non-contact ACL ruptures via appropriate hamstring muscle training? Solomonow et al. (1987) claimed that good muscle tone could prevent subluxation of the tibia relative to the femur during submaximal slow loading conditions. However, large external loading forces beyond the sustaining capabilities of the muscles, or forces applied at rapid loading rates may not allow the hamstring muscles to be activated fast enough or with

sufficient opposition to prevent subluxation. Furthermore, reflexive functions are associated with a finite time delay. Therefore, rapid loading of the knee does not allow the reflexes existing in the knee, its ligaments, capsule, and muscles to respond fast enough, irrespective of whether the ACL is intact or deficient, to prevent subluxation (Solomonow et al., 1987). However, some ACL deficient athletes develop functional adaptations during locomotor activities to enable them to perform dynamic tasks involving rapid loading of the knee without experiencing 'giving-way'\* episodes. What do these athletes do to protect their knees during such dynamic tasks and what can we learn from this that could be used to prevent ACL ruptures occurring in the healthy knee?

**WHAT CAN WE LEARN FROM FUNCTIONAL ACL DEFICIENT PATIENTS?** Although many ACL deficient athletes display the distinct syndrome described previously, others have minimal impairment and no functional limitations following complete ACL rupture, even when they are involved in very strenuous activities, and after receiving only conservative treatment (Daniel et al., 1994; Wojtys and Huston, 1994). How one subset of ACL deficient athletes (referred to as 'functional patients') can overcome considerable passive joint laxity to maintain a high level of function, whereas other ACL deficient athletes have difficulty in **compensating** for even minor increases in laxity remains unclear (Beard et al., 1996; McNair et al., 1989). It is thought the functional ACL deficient athletes develop compensatory adaptations in response to their injury; adaptations that are presumably produced by subconscious protective mechanisms acting to avoid excessive ATT that could occur in the absence of the ACL (Andriacchi, 1990; Berchuck et al., 1988). It has been suggested that these adaptations are developed as a consequence of the ACL deficient athlete's experiences after injury rather than as a result of stimuli occurring during an activity. That is, instantaneous adaptations associated with muscle contraction could not occur in sufficient time to produce the adaptations in response to a stimulus that occurred at any point in time during a locomotor process. Therefore, the adaptations are thought to involve 'reprogramming' of the locomotor process such that compensation occurs before excessive anterior displacement of the tibia (Berchuck et al., 1988). The rhythmical and symmetric nature of the adaptations and the manner in which they were transferred to the contralateral extremity in gait supported this reprogramming hypothesis (Andriacchi, 1990).

Investigating movement patterns and muscular **coordination** of these functional chronic ACL deficient athletes during physiological conditions that would predispose less functional ACL deficient athletes to giving-way episodes can provide valuable information about their compensatory mechanisms following ACL rupture (Beard et al., 1996) and, in turn, insights which may be used in rehabilitating the acutely ACL injured knee. Could this information also be used to develop strategies for preventing non-contact ACL injuries in otherwise healthy athletes?

**ACL Deficiency and Landing – An Investigation** (Steele and Brown, in press): To gain insight into possible protective strategies, the influence of chronic ACL deficiency on muscle activation patterns displayed during a task known to excessively stress the ACL, relative to timing of the tibiofemoral shear forces generated during the task, was examined. Eleven unilateral, functional, chronic, isolated ACL deficient patients and 11 control subjects matched to the patients for age, gender, anthropometry, activity level, and sports experience, and with no history of knee joint disease or trauma, participated in the study.

The subjects performed an abrupt landing task in which they accelerated to receive a chest level pass, landed on the test limb in single-limb stance, and stabilised their position without raising the landing foot. After adequate familiarisation, the procedures were repeated five times on both the subjects' right and left lower limbs. Each subject's deceleration motion in the plane of progression was filmed using a high-speed camera (200 Hz) and positioned to minimise perspective errors. Orientation of the foot, leg, and thigh segments of the landing

---

\* Giving-way is a clinical term used to define when the knee translates out of its ligamentous and muscular boundaries so that the articular surfaces partially or totally disengage and the patient loses control of single-leg stance (Cross, 1996).

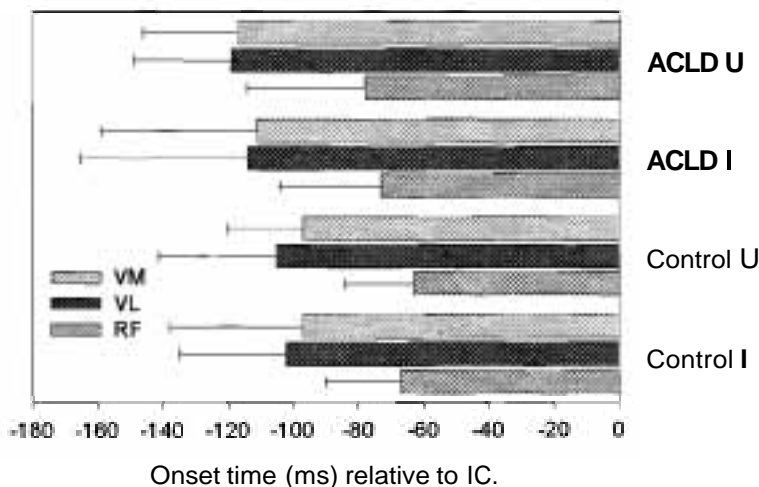
limb were determined from the manually digitised, low-pass filtered data using a least square fitting of two foot, four leg, and three thigh markers. The centre of rotation of the knee was identified at the point at which the lines representing the proximal leg and the distal thigh bisected. Two-dimensional kinematic variables in the plane of progression, required for later calculation of the knee joint moments, were then analysed from initial foot-ground contact (IC) until generation of the peak resultant ground reaction force.

The three orthogonal components and point of application of the ground reaction forces were sampled during the deceleration task using a Kistler force platform. Joint reaction forces and planar net moments of force for the knee joint in the sagittal plane were then calculated using Newtonian equations of motion and an inverse dynamics approach (Winter, 1990). The tibiofemoral joint shear forces were calculated from the net joint reaction forces and the patellar tendon force occasioned by the net moments and inertial forces predicted to be acting about the knee. Biomechanical data reported by Nisell (1985) were used to model knee joint musculoskeletal geometry in calculating the shear forces.

After standard subject preparation, electromyographic (EMG) activity of six lower limb muscles (rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM), biceps femoris (BF), semimembranosus (SM) and gastrocnemius) were sampled during the deceleration task using bipolar pellet electrodes and a Noraxon Telemetry EMG system. The film, force and EMG data were time-synchronised during each trial by marking the film with an ultrabright current-limited light emitting diode system. To quantify temporal characteristics of the muscle bursts, the EMG data were zero-offset, full-wave rectified and then smoothed to create linear envelopes, which were then screened, using a threshold detector. Muscle burst onset and offset were deemed to have occurred when 14 samples passed through a threshold of 7% of the maximum amplitude of the muscle burst of interest. The tibiofemoral shear force and EMG data were analysed using a two-way ANOVA design with one between factor (subject group) and one within factor (test limb). For all analyses, the lower limbs of the ACL deficient and control subjects were matched for limb dominance.

The Results: No significant main effects or any significant interactions on muscle burst duration were found. However, consistent with previous literature, there was a tendency for the ACL deficient subjects to display longer muscle burst durations than the controls for all six muscles analysed. Prolonged muscle activation is thought to facilitate stability in the ACL deficient knee by increasing tibiofemoral joint compression forces. It is suggested that some ACL deficient subjects employed this strategy in the present study but that prolonged muscle activity was not a consistent functional adaptation.

Both subject groups activated their quadriceps muscles approximately 60 to 100 ms before IC (see Figure 1). Accounting for electromechanical delay, quadriceps activation appears to have been preprogrammed before landing so that onset of force generation occurred close to foot-ground impact. Peak quadriceps activity, however, occurred after IC and was thought to effect a knee extension moment that was evident after this point in time to prevent the stance limb from 'collapsing' under body weight. However, there were no significant main effects or any significant interactions on quadriceps synchronisation. Therefore, altered quadriceps

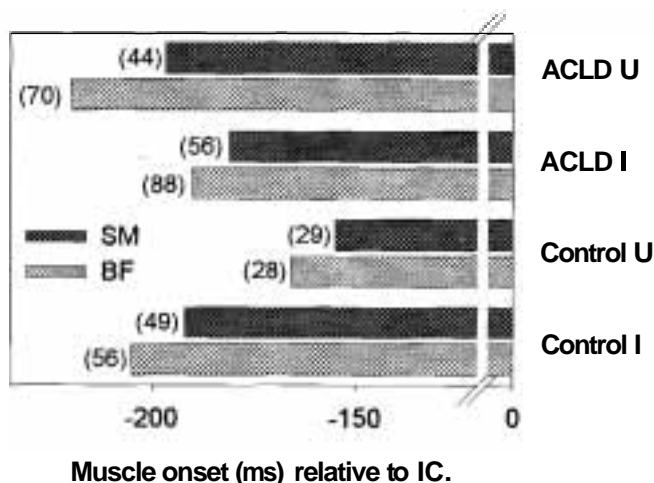


**Figure 1 - Mean (SD) values for the timing of the onset of quadriceps muscle activity relative to IC (0 ms) for the uninjured (U) and injured (I) limb of ACL deficient and control subjects. The negative values indicate muscle onsets occurred before IC.**

activation noted previously for ACL deficient subjects in activities such as treadmill walking and stair climbing were not evident in the present study.

Hamstring onset and peak hamstring activity both occurred before quadriceps onset or peak quadriceps activity. It has been suggested that this pattern of hamstring-quadriceps muscle sequencing provides optimal ACL protection by maximising efficacy of the hamstrings to produce a posterior tibial drawer. This posterior drawer force may be somewhat limited at IC due to the extended knee posture displayed by both subject groups at this time where mean knee flexion angles approximated 8°. However, hamstring co-contraction at IC would increase joint stability via increasing compression of the tibial and femoral surfaces.

A significant subject group x test limb interaction for both SM onset was found when the data were normalised for muscle burst duration and for BF onset when the data were normalised for time to the peak ground reaction force. Whereas control subjects displayed earlier SM and BF activation relative to IC on their 'injured'\* lower limb compared to their uninjured lower limb, ACL deficient subjects displayed the reverse trend. That is, the ACL deficient subjects displayed a delay in the onset of SM and BF relative to IC for their injured limb compared to their uninjured limb (see Figure 2). Later onset times of the hamstrings relative to IC for the ACL deficient subjects' injured lower limbs appear to contradict much of the previous literature in which earlier recruitment of the hamstring muscles has been associated with ACL deficient patients (Lass et al., 1991).



**Figure 2 - Mean (SD) values for the timing of the onset of hamstring muscle activity relative to IC (0 ms) for the uninjured (U) and injured (I) limb of ACL deficient control subjects. The negative values indicate muscle onsets occurred before IC.**

Peak hamstring activity for both subject groups occurred 30 to 60 ms before IC. Accounting for electromechanical delay (Zhou et al., 1995), peak hamstring force generation coincided approximately with generation of the peak tibiofemoral shear forces which occurred about 30 ms post IC. It is suggested that the delay in hamstring activation displayed by the ACL deficient subjects for their injured limbs was employed to ensure that peak hamstring activity on the injured limb was more synchronous with IC and with the high tibiofemoral shear forces than for the contralateral limb. However, without direct evidence of the exact electromechanical delay between hamstring activation and muscle force generation during deceleration to support this hypothesis, further investigation is warranted.

Alterations in hamstring activity displayed during the deceleration task were not evident bilaterally, thereby, conflicting with the reprogramming hypothesis (Andriacchi, 1990). It is possible that the novel nature of the task performed by the subjects precluded them from having sufficient experience with the task demands to have generalised these adaptations to the contralateral limb. Furthermore, high within subject group variability for the muscle activation patterns displayed during deceleration implied that individual strategies were being used by both control and ACL subjects to decelerate, despite the constrained nature of the task.

---

**Although the control subjects had no knee injuries, the limbs that were matched for dominance to the ACL deficient patients' injured limbs were referred to as the control subjects' injured limb.**

It was concluded that delayed hamstring activation was an adaptation developed by these functional ACL deficient subjects to enable peak hamstring activity to better coincide with the high tibiofemoral shear forces generated during the deceleration task. As landing occurred with the knee near full extension, the more synchronous activation of the hamstrings with the peak tibiofemoral shear forces was thought to assist in stabilising the knee mainly via increasing tibiofemoral joint compression and, to a lesser extent, posterior tibial drawer when the knee would be most vulnerable to anterior subluxation.

Implications for the Healthy Athlete: So what are the implications of this study of ACL deficient patients for athletes with intact knees? It is evident that the strategies used by the functional ACL deficient patients to protect their knees against giving-way episodes were learned. For example, both the hamstring and quadriceps muscle groups were recruited before IC. This supported the notion of a learned motor program being used by subjects to engage the lower limb to assist in withstanding the high landing forces. Can healthy athletes be trained in learning these same protective muscle recruitment strategies and, if so, will they effectively protect their knees from non-contact ACL rupture episodes? Further research is warranted to address these questions.

**MUSCULAR CONTROL OF LANDING: IS IT TRAINABLE?** Although several studies have investigated muscle activation patterns during the landing phase of dual-limb drop jumps (eg. Irvine et al., 1993; McNitt-Gray, 1993; Welch et al., 1993), only limited research has examined neuromuscular control during single-limb abrupt deceleration tasks that have been implicated as a mechanism of non-contact ACL rupture. As muscles are dynamic stabilisers of the knee, investigating muscular control during landing may provide insight into methods by which subjects maintain joint integrity and reduce knee joint loading during a task known to stress the knee. White (1975) claimed the voluntary and reflex actions of muscles about the knee had a profound effect on resultant reaction forces intrinsic to the knee, particularly during acts involving sudden deceleration. However, Lees (1981) claimed the important components of impact landings occurred in a time which was shorter than human reaction time and was too rapid to enable reflexive actions of the neuromuscular system to operate to control segmental interactions.

Voluntary control of the leg and foot muscles during unexpected falls is not present for at least 200 ms after fall initiation (Greenwood and Hopkins, 1976). For this reason, it is assumed the sequence of muscular commands issued to cope with the impact of unexpected landings is not influenced by consciously mediated feedback but rather under the control of a 'motor program' (Lees, 1981; Mizrahi and Susak, 1982). By way of contrast, in landing after falls that were expected, precise timing of muscle activity before impacting the ground has been shown to occur (Greenwood and Hopkins, 1976). Melvill Jones and Watt (1971a,b) claimed that muscular control of landing from a single intended step or jump to the ground was based upon a preprogrammed neural message which was formulated in and dispatched from the central nervous system well before the moment of landing. This was completed before a functionally useful stretch reflex due to the landing could become effective in decelerating the body mass. Melvill Jones and Watt (1971b) found that gastrocnemius activity associated with landing began, on average, 141 ms before contact with the ground and ended 131 ms after contact. Aune et al. (1995) found that during a landing task in downhill skiing the knee flexors were recruited before initial ski-ground contact and 60 ms earlier than the knee extensors. Peak knee flexor and knee extensor activity occurred simultaneously at ski-ground contact. The correct timing and sequence of muscle contractions was thought to be learned as a motor program through previous experience (Melvill Jones and Watt, 1971b).

In analysing the landing phase of heel-toe running, Bobbert et al. (1992) noted extremely rapid changes in the net muscle moments about the knee during the initial 25 ms of the landing phase of running. The authors hypothesised these rapid changes were caused by spring-like behaviour of pre-activated muscles (coactivation of the knee flexor and knee extensor muscles) which served as an essential element in preparation for landing. This hypothesis

implied that at landing, there was no opportunity for athletes to control body segment rotations during initial deceleration other than by selecting a certain segmental alignment and muscular activation levels before foot-ground contact (Bobbert et al., 1992). Furthermore, absence of coactivation of the knee extensor and knee flexor muscles before landing could lead to loss of passive control of body motion during the landing phase (Bobbert et al., 1992). Other studies (Mann and Hagy, 1980) have also shown existence of a strong coactivation of quadriceps and hamstring muscles before touch-down and during the first part of the support phase in running. Therefore, muscle activity about the knee during anticipated abrupt deceleration tasks appears to influence knee joint forces during landing via preprogrammed neural messages rather than the voluntary and reflex actions of synergistic and antagonistic muscles about the knee as proposed by White (1975). Mizrahi and Susak (1982) claimed the preprogrammed non-reflex muscle actions, which operated during the early phase of impact in landing, were an important mechanism in peak force attenuation. Furthermore, active musculature appears to play a greater role in controlling motion of the lower extremities during landing as the velocity of impact increases. Relatively greater demands are placed on the knee extensor muscles to generate large peak moments at higher landing velocities than on the ankle plantar flexors and hip extensor muscles (McNitt-Gray, 1993). Lees (1981) claimed that alterations to the structure of the motor program, such as would be required in an attempt to reduce force levels during the impact absorption phase of a landing, can only be produced as a result of an appropriate training program. It is therefore apparent that alterations to the structure of the motor program may also be feasible as a means to train athletes to protect their knees against non-contact ACL injuries.

**TO RECONSTRUCT OR NOT TO RECONSTRUCT: CAN WE PREDICT WHICH ACL DEFICIENT ATHLETE REQUIRES SURGERY?** Those unfortunate athletes who have not been able to prevent sustaining an ACL rupture face another dilemma: to reconstruct or not reconstruct their ACL following injury? Despite a proliferation of new tests and diagnostic aids, management of ACL injuries remains an enigma. Within the abundant literature related to ACL injuries is an array of contradictory treatment schemes, surgical procedures, and rehabilitation programs, many of which are justified on transient clinical impressions rather than scientific fact. Suggested management of the patient following isolated ACL rupture ranges from conservative non-operative rehabilitative regimens to early reconstructive surgery (Fleming *et al.*, 1993; Hawkins *et al.*, 1986). However, controversy persists as to which proposed treatment regime is most appropriate for a given individual (Kannus and Jarvinen, 1987). It is not within the scope of this paper to examine the dilemma of clinical practices pertaining to ACL injuries. The conflicting views related to diagnosis, natural history, and treatment of ACL injuries are reviewed by Johnson (1982, 1983), Noyes and McGinniss (1985), and Wroble and Brand (1990). Williams and Bach (1996) present a review of operative and non-operative rehabilitative options for the ACL deficient knee.

As the totally ruptured ACL does not spontaneously heal due to its poor blood supply, surgical reconstruction of the ligament is usually recommended for young and active patients to regain sufficient knee stability to continue participating in sport (McCarroll et al., 1995; Williams and Bach, 1996). McNair et al. (1989) noted that ACL deficient patients experienced considerable functional disability, particularly during sports that involved pivoting, sudden stops, and jumping activities. Nisonson and Goldberg (1991) cautioned that without surgical reconstruction following ACL rupture there was at least a 60% chance that an athlete participating in sports that involved jumping or pivoting would have another episode of the knee giving-way. Multiple giving-way episodes would lead to further ligamentous and meniscal damage and ultimately to traumatic arthritis (Nisonson and Goldberg, 1991). However, regaining preinjury performance levels and sufficient knee stability to perform athletic manoeuvres after ACL reconstruction can be difficult, despite lengthy rehabilitation protocols (Moore and Wade, 1989). Furthermore, no surgical procedure has yet proven to be consistently successful (Friden et al., 1991). Although reconstructive techniques are continuously being refined, duplication of the functional structure of the ACL is still elusive to the orthopaedic surgeon (MacDonald et al., 1996). Treatment of ACL deficient patients is further complicated by difficulty in trying to accurately predicting those

athletes who will be functionally impaired by ACL rupture and those athletes who will have minimal symptoms (Andriacchi, 1993). If a relationship between knee function during clinical assessment of knee laxity and functional performance during a dynamic task could be established this might enable objective identification of acutely injured athletes who may be suited to conservative treatment or those requiring early surgical intervention. Although the effects of ACL rupture on knee function during locomotor tasks have been studied, no comprehensive research was located which examined whether knee function during knee laxity assessment could be associated with the use of compensatory adaptations by functional ACL deficient subjects to perform dynamic tasks. Therefore, Steele et al. (1998) conducted a study to identify the relationship between knee function during arthrometric knee laxity assessment and knee function during a dynamic movement known to stress the ACL, abrupt landing.

Active and passive anterior knee laxity were assessed using a Dynamic Cruciate Tester (DCT) arthrometer for 11 functional chronic unilateral ACL deficient patients and 11 uninjured controls matched to the patients for age, gender, sports experience and anthropometry. Muscle activation patterns for two quadriceps muscles (RF and VL) and two hamstring muscles (BF and SM) were recorded in synchrony with ATT data during knee laxity assessment. Ground reaction force data and EMG data for the same lower limb muscles were then sampled (1000 Hz) as the subjects landed in single-limb stance on a force platform after receiving a ball thrown at chest level and decelerating abruptly. Each subject's deceleration motion in the plane of progression was also filmed using high-speed cinematography (200 Hz). Temporal characteristics of each muscle burst relative to IC and were then analysed for each trial following the procedures described previously. Two-dimensional kinematic variables characterising lower limb alignment and motion during landing, and the tibiofemoral joint shear and compressive forces were then calculated. The dependent variables were analysed using Pearson product moment correlations to determine the relationship between variables characterising knee function during knee laxity assessment (ATT, BF and SM muscle intensity, and VL and RF duration) and knee function during deceleration (characterised by selected kinetic, kinematic, and neuromuscular variables).

Thirty-five correlations between variables characterising knee laxity and deceleration were significant ( $p \leq 0.05$ ) across the pooled subject group results. However, the correlations were low ( $r = 0.299 - 0.483$ ) such that most of the variance within the variables characterising knee function during deceleration could not be explained by their relationship to the variables characterising knee function during arthrometric knee laxity assessment and, in turn, were not considered useful for predicting performance. A major objection to static clinical tests designed to assess knee laxity is that they are performed under non-physiological circumstances where the applied loads are far less than those generated in functional activities (Gauffin and Tropp, 1992). Furthermore, there are several important factors in maintaining knee stability including muscular coactivation and tibiofemoral compression loading that are generally absent when performing passive static tests. Although knee laxity assessment in this study was extended relative to previous research to include an active component to the knee laxity assessment protocol, inherent differences between the demands of a multilimb dynamic deceleration task and knee laxity assessed supinely in a closed environment were still too great to enable the clinical test to be useful for predicting dynamic performance. In the non-weight bearing supine position required during knee laxity assessment, externally applied forces or moments of force in the unloaded knee are internally resisted by the passive restraints of the ligaments and joint capsule. However, during the weight bearing deceleration task, axial loading of the knee greatly enhances joint stability. Furthermore, muscular contractions during active knee laxity assessment were limited to isometric or concentric contractions. In contrast, the dynamic functional adaptations used by ACL deficient subjects during a task such as abrupt deceleration involves the musculature functioning isometrically and eccentrically, as well as concentrically (Lephart et al., 1992). It was concluded that predicting knee function of ACL deficient subjects during an open dynamic deceleration task using a clinical test such as the DCT was not feasible. Trying to accurately predict those athletes who will be functionally impaired by



ACL rupture and those athletes who will have minimal symptoms still appears elusive. This further highlights the need to ensure ACL injuries are prevented from occurring in the first place.

**FUTURE DIRECTIONS:** Due to the prevalence of ACL injury and the potential negative consequences associated with it, ACL injury has received more attention and engendered more controversial discussion than any other orthopaedic injury (Kannus and Järvinen, 1990). In 1935 Milch wrote:

*In spite of much that has been written about the functions of the crucial ligaments, there is still far from unanimity of opinion on the subject (p. 805).*

This lack of unanimity of opinion still exists 64 years later. As the economic, emotional and social costs of ACL injuries are still excessive, research into strategies to prevent ACL rupture is still warranted to remove this nightmare of the coach and athlete. Unanswered questions that require immediate investigation include:

1. Can healthy athletes train muscle recruitment patterns to protect the ACL from non-contact injury mechanisms in sport? If so, what are the optimal training methods?
2. What factors inherent in sport may interfere with protective muscle recruitment patterns that would then predispose athletes to greater risk of ACL injury and how can the negative effects of these factors be minimised?
3. Does the wearing of prophylactic knee braces influence learned muscle recruitment patterns and, in turn, the risk of ACL injury?

Answering such questions via systematic investigation will enable us to better understand how effectively we can prevent or minimise non-contact ACL injuries from occurring in sport in the future.

#### REFERENCES:

- Andriacchi, T.P. (1990). Dynamics of pathological motion: Applied to the anterior cruciate deficient knee. *Journal of Biomechanics*, **23**(Suppl. 1), 99-105.
- Andriacchi, T.P. (1993). Functional analysis of pre and post-knee surgery: Total knee arthroplasty and ACL reconstruction. *Journal of Biomechanical Engineering*, **115**, 575-581.
- Aune, A.K., Schaff, P., & Nordsletten, L. (1995). Contraction of knee flexors and extensors in skiing related to the backward fall mechanism of injury to the anterior cruciate ligament. *Scandinavian Journal of Medicine and Science in Sports*, **5**, 165-169.
- Baker, B.E. (1990). Prevention of ligament injuries to the knee. In K.B. Pandolf, & J.O. Holloszy (Eds.), *Exercise and sport sciences reviews* (pp. 291-305). Baltimore: Williams & Wilkins.
- Beard, D.J., Soundarapandian, R.S., O'Connor, J.J., & Dodd, C.A.F. (1996). Gait and electromyographic analysis of anterior cruciate ligament deficient subjects. *Gait & Posture*, **4**, 83-88.
- Bender, J.A., Pierson, J.K., Kaplan, H.M., & Johnson, A.J. (1964). Factors affecting the occurrence of knee injuries. *Journal of the Association for Physical and Mental Rehabilitation*, **18**, 130-134.
- Berchuck, M., Andriacchi, T.P., Bach, B.R., & Reider, B. (1988). Functional adaptation in ACL-deficient patients. *Transactions of the 34th Annual Orthopedic Research Society Meeting*, **13**, 84.
- Beynon, B.D., Fleming, B.C., Johnson, R.J., Nichols, C.E., Renström, P.A., & Pope, M.H. (1995). Anterior cruciate ligament strain behavior during rehabilitation exercises in *vivo*. *American Journal of Sports Medicine*, **23**, 24-34.
- Bobbert, M.F., Yeadon, M.R., & Nigg, B.M. (1992). Mechanical analysis of the landing phase in heel-toe running. *Journal of Biomechanics*, **25**, 223-234.
- Cailliet, R. (1984). *Knee pain and disability* (2nd ed.). Philadelphia: F.A. Davis Company.
- Collins, H.R. (1980). Anterolateral rotatory instability of the knee. In *American Academy of Orthopaedic Surgeons Symposium on the Athlete's Knee: Surgical repair and reconstruction* (pp. 184-194). St. Louis: The C.V. Mosby Company.

- Cross, M. (1996). Clinical terminology for describing knee instability. *Sports Medicine and Arthroscopy Review*, 4, 313-318.
- Daniel, D.M., Stone, M.L., Dobson, B.E., Fithian, D.C., Rossman, D.J., & Kaufman, K.R. (1994). Fate of the ACL-injured patient. A prospective outcome study. *American Journal of Sports Medicine*, 22, 632-644.
- Fanton, G. (1991). Anterior cruciate ligament injuries: Understand the step-by-step process of treating one of the most common injuries in athletics. *Coaching Volleyball*, June/July, 18-21.
- Feagin, J.A., Curl, W.W., & Markey, K.L. (1980). Anterior cruciate ligament loss: Complications and late results. In *American Academy of Orthopaedic Surgeons Symposium on the Athlete's Knee: Surgical repair and reconstruction* (pp. 173-177). St.Louis: The C.V. Mosby Company.
- Fetto, J.F., & Marshall, J.L. (1980). The natural history and diagnosis of anterior cruciate ligament insufficiency. *Clinical Orthopaedics and Related Research*, 147, 29-38.
- Fleming, B., Beynon, B., Johnson, R.J., McLeod, W.D., & Pope, M.H. (1993). Isometric versus tension measurements. A comparison for the reconstruction of the anterior cruciate ligament. *American Journal of Sports Medicine*, 21, 82-88.
- Fridén et al., (1991).
- Gauffin, H., & Tropp, H. (1992). Altered movement and muscular-activation patterns during the one-legged jump in patients with an old anterior cruciate ligament rupture. *American Journal of Sports Medicine*, 20, 182-192.
- Greenwood, R., & Hopkins, A. (1976). Muscular responses during sudden falls in man. *Journal of Physiology*, 254, 507-518.
- Hawkins, R.J., Misamore, G.W., & Merritt, T.R. (1986). Followup of the acute nonoperated isolated anterior cruciate ligament tear. *American Journal of Sports Medicine*, 14, 205-210.
- Irvine, D.M.E., McNitt-Gray, J.L., Munkasy, B.C., & Welch, M.D. (1993). Muscle activity and kinetics during landings with reduced vertical reaction forces. *Journal of Biomechanics*, 26, 359.
- Johnson, R.J. (1982). The anterior cruciate: A dilemma in sports medicine. *International Journal of Sports Medicine*, 3, 71-79.
- Johnson, R.J. (1983). The anterior cruciate ligament problem. *Clinical Orthopaedics and Related Research*, 172, 14-18.
- Kannus, P., & Jarvinen, M. (1987). Conservatively treated tears of the anterior cruciate ligament: Long-term results. *Journal of Bone and Joint Surgery*, 69-A, 1007-1011.
- Kannus, P., & Jarvinen, M. (1990). Nonoperative treatment of acute knee ligament injuries. A review with special reference to indications and methods. *Sports Medicine*, 9, 244-260.
- Lass, P., Kaalund, S., leFevre, S., Arendt-Nielsen, L., Sinkjær, T., & Simonsen, O. (1991). Muscle coordination following rupture of the anterior cruciate ligament. *Electromyographic studies of 14 patients. Acta Orthopaedica Scandinavica*, 62, 9-14.
- Lees, A. (1981). Methods of impact absorption when landing from a jump. *Engineering in Medicine*, 10, 207-211.
- Lephart, S.M., Perrin, D.H., Fu, F.H., Gieck, J.H., McCue, F.C., Irrgang, J.J. (1992). Relationship between selected physical characteristics and functional capacity in the anterior cruciate ligament-insufficient athlete. *Journal of Orthopaedic and Sports Physical Therapy*, 16, 174-181.
- Lutz, G.E., Stuart, M.J., & Sim, F.H. (1991). Rehabilitative techniques for athletes after reconstruction of the anterior cruciate ligament. *Physiotherapy in Sport*, 13, 9-13.
- MacDonald, P.B., Hedden, D., Pacin, O., & Sutherland, K. (1996). Proprioception in anterior cruciate ligament-deficient and reconstructed knees. *American Journal of Sports Medicine*, 24, 774-778.
- Mann, R.A., & Hagy, J. (1980). Biomechanics of walking, running, and sprinting. *American Journal of Sports Medicine*, 8, 345-350.
- McCarroll, J.R., Shelbourne, K.D., & Patel, D.V. (1995). Anterior cruciate ligament injuries in young athletes. Recommendations for treatment and rehabilitation. *Sports Medicine*, 20, 117-127.

- McNair, P.J., & Marshall, R.N. (1994). Landing characteristics in subjects with normal and anterior cruciate ligament deficient knee joints. *Archives of Physical Medicine and Rehabilitation*, 75, 584-589.
- McNair, P.J., Marshall, R.N., & Matheson, J.A. (1989). The gait of subjects with anterior cruciate ligament deficiency. In R.J. Gregor, R.F. Zernicke, & W.C. Whiting (Eds.), *Proceedings of the XIIIth International Congress of Biomechanics* (abstract no. 184). Los Angeles, California: University of California, Los Angeles.
- McNair, P.J., Wood, G.A., Marshall, R.N., & Strauss, G. (1991). Response of hamstring muscles to a perturbation in people with chronic anterior cruciate ligament deficiency. *Scandinavian Journal of Medicine and Science in Sports*, 1, 215-220.
- McNitt-Gray, J.L. (1993). Kinetics of the lower extremities during drop landings from three heights. *Journal of Biomechanics*, 26, 1037-1046.
- Melvill Jones, G., & Watt, D.G.D. (1971a). Observations on the control of stepping and hopping movements in man. *Journal of Physiology*, 219, 709-727.
- Melvill Jones, G., & Watt, D.G.D. (1971b). Muscular control of landing from unexpected falls in man. *Journal of Physiology*, 219, 729-737.
- Milch, H. (1935). Injuries to the crucial ligaments. *Archives of Surgery*, 30, 805-819.
- Mizrahi, J., & Susak, Z. (1982). Analysis of parameters affecting impact force attenuation during landing in human vertical free fall. *Engineering in Medicine*, 11, 141-147.
- Moore, J.R., & Wade, G. (1989). Prevention of anterior cruciate ligament injuries. *National Strength & Conditioning Association Journal*, 11, 35-40.
- Nisell, R. (1985). Mechanics of the knee: A study of joint and muscle load with clinical applications. *Acta Orthopaedica Scandinavica*, 56(Suppl. 216), 1-42.
- Nisonson, B., & Goldberg, B. (1991). Anterior cruciate ligament injuries. Conservative vs surgical treatment. *Physician and Sportsmedicine*, 19, 82-89.
- Noyes, F.R., & McGinniss, G.H. (1985). Controversy about treatment of the knee with anterior cruciate laxity. *Clinical Orthopaedics and Related Research*, 198, 61-76.
- Noyes, F.R., Mooar, P.K., Matthews, D.S., & Butler, D.L. (1983). The symptomatic anterior cruciate-deficient knee. *Journal of Bone and Joint Surgery*, 65-A, 154-162.
- Shelburne, K.B., & Pandy, M.G. (1997). A musculoskeletal model of the knee for evaluating ligament forces during isometric contractions. *Journal of Biomechanics*, 30, 163-176.
- Solomonow, M., Baratta, R., Zhou, B.H., Shoji, H., Bose, W., Beck, C., & D'Ambrosia, R. (1987). The synergistic action of the anterior cruciate ligament and thigh muscles in maintaining joint stability. *American Journal of Sports Medicine*, 15, 207-213.
- Steele, J.R. & Brown, J.M.M. (in press). Effects of chronic anterior cruciate ligament deficiency on muscle activation patterns during an abrupt deceleration task. *Clinical Biomechanics*
- Steele, J.R. (1986). *A kinetic and kinematic analysis of netball landing techniques: Implications for injury prevention and shoe design*. Technical Report I, Part A. A Report to the Australian Sports Commission and the All Australia Netball Association. Canberra: National Sports Research Centre, Australian Sports Commission.
- Steele, J.R., Brown, J.M.M. & Milburn, P.D. (1998). Relationship between knee function of chronic ACLD patients during arthrometric knee laxity assessment and dynamic deceleration. In P. Hume, R. Marshall, P. Hunter, S. Stanley, I. Anderson, & P. McNair (Eds.), *Book of Abstracts of the 2<sup>nd</sup> Australian and New Zealand Society of Biomechanics* (pp 46-47). Auckland, New Zealand: The University of Auckland, New Zealand.
- Walla, D.J., Albright, J.P., McAuley, E., Martin, R.K., Eldridge, V., & El-Khoury, G. (1985). Hamstring control and the unstable anterior cruciate ligament-deficient knee. *American Journal of Sports Medicine*, 13, 34-39.
- Welch, M.D., McNitt-Gray, J.L., Irvine, D.M.E., & Barbieri, C. (1993). Adjustments in muscle activation patterns during landings of low and high peak vertical forces. *Journal of Biomechanics*, 26 320.
- White, J.E. (1975). Helical instability of the knee: A concept and surgical reconstruction. *Journal of Sports Medicine*, 2, 329-342.

- Williams, J.S., & Bach, B.R. (1996). Operative and nonoperative rehabilitation of the ACL-Injured knee. *Sports Medicine and Arthroscopy Review*, **4**, 69-82.
- Winter, D.A. (1990). *Biomechanics and motor control of human movement* (2nd ed.). New York: John Wiley & Sons, Inc.
- Wojtys, E., & Huston, L.J. (1994). Neuromuscular performance in normal and anterior cruciate ligament-deficient lower extremities. *American Journal of Sports Medicine*, **22**, 89-104.
- Woo, S.L-Y., Livesay G.A., & Engle C. (1992). Biomechanics of the human anterior cruciate ligament. Muscle stabilization and ACL reconstruction. *Orthopaedic Review*, **21**, 935-941.
- Wroble, R.R., & Brand, R.A. (1990). Paradoxes in the history of the anterior cruciate ligament. *Clinical Orthopaedics and Related Research*, **259**, 183-191.
- Yack, H.J., Washco, L.A., & Whieldon, T. (1994). Compressive forces as a limiting factor of ATT in the ACL-deficient knee. *Clinical Journal of Sports Medicine*, **4**, 233-239.
- Zarins, B., & Nemeth, V.A. (1983). Acute knee injuries in athletes. *Clinics in Sports Medicine*, **2**, 149-166.
- Zatterstrom, R., Friden, T., Lindstrand, A., & Moritz, U. (1994). The effect of physiotherapy on standing balance in chronic anterior cruciate ligament insufficiency. *American Journal of Sports Medicine*, **22**, 531-536.
- Zhou, S., Lawson, D.L., Morrison, W.E., & Fairweather, I. (1995). Electromechanical delay of knee extensors: The normal range and the effects of age and gender. *Journal of Human Movement Studies*, **28**, 127-146.