PROPRIOCEPTION IN THE KNEE AND REFLEX HAMSTRING CONTRACTION LATENCY

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There are various methods of measuring proprioception at the knee. Beard et al (1993) have described a delay in reflex hamstring contraction in anterior cruciate deficient knees.

We have repeated their experiment and were unable to detect any significant difference in reflex hamstring contraction between the injured and uninjured legs. We discuss possible neurophysiological and biomechanical causes for the conflicting results and conclude that this method may not be a valid measure of proprioception.

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Injury to the anterior cruciate ligament (ACL) is a major cause of sporting disability: the incidence is 1 per 1000 of the population (Hirschman, Daniel and Miyasaka 1990). The biomechanics of the ligament and the best form of treatment have been studied and operations have been designed to provide anteroposterior and anterolateral stability. Until recently, physiotherapy aimed to increase general muscle strength, but in the last five years there has been more emphasis on the strength and control of the hamstrings (Beard and Fergusson 1992). It has also become apparent that the ACL is more than just a mechanical constraint, and that it has a role in proprioception of the knee. Awareness of the position, movement and pressure in a joint is both a conscious and unconscious process and is served by various receptors (Williams and Warwick 1980). Since Sherrington (1900) first suggested that muscle spindles provide ‘muscle sense’ there has been much discussion of the relative role of joint receptors and muscle spindles. It is believed that the latter help to determine the position of the joint and to detect movement, but that joint receptors may also detect movement. The origin of static position sense is less clear (Ferrell and Craske 1992).

The ACL contains all the receptors likely to be involved in proprioception (Halata and Haus 1989) and these may provide an ACL-hamstring protective reflex (Gruber, Wolter and Lierse 1986; Solomonow et al 1987). It has been suggested that the ACL afferents may regulate the stiffness of the muscles around the knee (Johansson, Sjölander and Sojka 1991) and that these receptors may also signal movement, especially extension and internal and external rotation (Krauspe, Schmidt and Schaible 1992).

Conscious proprioception has been measured (Barrack, Skinner and Buckley 1989; Barrett 1991; Corrigan, Cashman and Brady 1992), but the results are conflicting and fail to take into account that ACL-deficient knees may also have suffered changes in the capsule and the muscles. These measurements are therefore rather non-specific.

McNair, Wood and Marshall (1992) have measured muscle stiffness and found a correlation between hamstring stiffness and the functional stability of the knee, but they showed no difference between injured and normal knees. Beard et al (1993) recently reported a delay in the reflex hamstring contraction in ACL-deficient knees after an impulse producing anterior movement of the tibia relative to the femur which put the ACL under tension in the normal limb. We have tried to reproduce their results.

PATIENTS AND METHODS

We investigated 11 patients (10 men and 1 woman) of an average age of 27 years (23 to 33) and an average weight of 79 kg (63 to 95). All had had ACL injuries diagnosed by arthroscopy eight months to ten years previously, except one who was examined within three weeks of arthroscopy. Each patient was assessed by a modified Cincinnati functional scoring system and clinical and instrumented measures of laxity, using a KT1000 arthrometer. EMG surface electrodes were placed on the medial and lateral hamstrings, vastus medialis and vastus lateralis. They were connected to an MIE telemetry system and data were collected by a Biodata Microlink system and stored on a computer. Tibial movement was recorded from an accelerometer attached to the tibial tuberosity.

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RESULTS

Of the 11 patients examined, seven showed no significant difference in reflex hamstring contraction latency (RHCL) between injured and normal legs. Only two showed a significant delay in reaction time in the injured leg (18 ms, 5.5 ms), while one showed a significant delay in the uninjured leg (29.5 ms), and one had conflicting results for the medial and lateral hamstrings separately.

The mean values for RHCL combined for both weight-bearing and non-weight-bearing tests (Table I) were 31.7 ms ± 7.6 ms for the injured knee and 41.4 ± 19.35 ms for the uninjured knee. The mean difference was 9.7 ms which was significantly longer in the uninjured leg than in the injured leg. Detailed study showed that 12 of the RHCL values were greater than 50 ms and that nine of these 12 were in the uninjured leg. If these slow reaction times were ignored then the mean value for the injured knee was 30.4 ± 4.2 ms and for the uninjured leg was 32.1 ± 5.0 ms. The difference of 1.7 ms was statistically insignificant and the RHCL was essentially the same in both legs.

**Weight-bearing v non-weight-bearing.** The mean RHCL for the injured leg when weight-bearing was 31.4 ± 6.0 ms and when non-weight-bearing 32.0 ± 9.2 ms. For the uninjured leg weight-bearing, the RHCL was 42.9 ± 6.4 ms and non-weight-bearing 40.0 ± 22.1 ms. If the values for RHCL > 50 ms are again excluded there is no statistical difference between the two standing conditions. For non-weight-bearing there was a difference between the RHCL for the two sides, the injured leg reacting faster than the uninjured leg (p < 0.1), but for weight-bearing there was no statistical difference in the RHCL.

**Tibial movement.** The mean anterior movement of the tibia was greater in injured legs, both weight-bearing and non-weight-bearing. In two cases, however, the injured leg moved less than the uninjured leg (Table II).

**EMG.** The results from the EMG electrodes on the hamstrings were consistent throughout the tests. EMG signals from the quadriceps were inconsistent because of the way in which the experiment was set up and gave no useful information.

DISCUSSION

Our results were very different from those reported by Beard et al (1993): we found no statistical difference in the RHCL between ACL-deficient and normal knees. If all the results were pooled then the RHCL was longer in the uninjured leg, but we found that some uninjured legs had very slow reaction times (> 50 ms). This finding was not consistent and values of less than 50 ms were also recorded in the same patients. We therefore discarded all values greater than 50 ms and recalculated the means. There was then no statistical difference between injured and uninjured legs and our results were very similar to
those reported by Beard et al (1993) for normal individuals, with a mean difference between the sides of 1.54 ± 3.74 ms.

Beard et al (1993) reported significantly slower RHCLs in recently injured ACL-deficient knees than in the contralateral knees, or normal control knees. They considered that the reflex response of muscles depended onafferent signals from joint receptors, and on the level of preresponse muscle stiffness, and suggested that the slowing of reflex muscle activity could be due to loss of proprioceptive input from the cruciate ligament or other receptors in the joint. There are, however, other interpretations of the test.

During the experiment, the patella and the thigh rest against rigid supports and the femur is therefore relatively immobile. The piston applies a force of about 140 N to the posterior aspect of the proximal tibia and the knee is flexed at 30° so that the collateral ligaments are lax. It can therefore be postulated that the ACL is being stretched. The hamstrings, however, are also stretched by this movement. Any ACL-hamstring reflex would be expected to disappear after ACL rupture, and absence of the reflex rather than delay would therefore be expected; the delayed response is due to a secondary capsular reflex. If an ACL-hamstring reflex is not being observed what is the explanation for what is happening in this experiment?

Normal muscles show two responses; the spinal monosynaptic reflex, which is tested clinically by the knee jerk, and a later stretch reflex which is transcortical (Marsden, Merton and Morton 1973) and is delayed by about 30 to 40 ms. The normal knee-jerk reflex occurs about 25 ms after the patellar tendon is tapped. There is then a silent period of about 20 ms before the normal activity returns (Bahniuk et al 1973). A second response cannot therefore take place during these 20 ms. We have been unable to find values for the hamstring reflex in the literature, but we have measured it at about 18 ms. A muscle reflex is a graded response, but there is a threshold below which there is no response. Although the magnitude of the reaction may vary after this the speed will not vary because the conduction velocity of the nerve fibres changes only in some rare disease states.

It seems possible that Beard et al (1993) were recording a hamstring tendon reflex in the uninjured leg and a stretch reflex in the injured leg. This would explain the delay in hamstring reaction in the injured knees, tested three weeks after arthroscopy.

Our experiments were identical to those of Beard et al (1993) except that we used 140 N force rather than 100 N, and they tested patients three weeks after arthroscopy. In the single patient whom we tested soon after arthroscopy there was more movement in the uninjured knee. This was surprising but could be explained by the presence of an effusion in the injured knee. In this patient, one value for the RHCL was 90 ms and several tests elicited no response. Other patients showed much greater consistency of results.

We found that forces of less than 100 N produced no contractions in the hamstrings and we therefore used 140 N to provide more forward acceleration of the tibia. As a result it seems possible that the force applied to the hamstrings by Beard et al may not have been sufficient to elicit a monosynaptic reflex in injured, post-arthroscopic knees, but sufficient to produce a transcortical stretch reflex. This would explain the difference which they found.

An argument against this hypothesis is that the initial contraction in the normal knee was delayed for much longer than the standard spinal reflex, but in favour is the fact that the mean difference between the two sides was of a similar order of magnitude as that between the spinal and stretch reflexes (40 ms). It seems probable that the hamstring stretch is delayed until the tibia has been displaced fully, some time after the initial movement. Maximum acceleration was up to 10 ms after initiation; if this period is subtracted from the RHCL measurements, the results approach those for the spinal reflex.

In our experiments most of the unduly prolonged RHCL values (> 50 ms) were seen in the uninjured leg in which forward movement of the tibia was less than on the injured side. This probably reduced the force applied to the hamstring tendons on the intact side, perhaps below the threshold for the tendon reflex, resulting only in the slower stretch reflex.
Conclusions. We have been unable to reproduce the results of Beard et al (1993) and have discussed possible reasons for this. At present, reflex hamstring latency does not seem to be a valid method for measuring proprioception: there are too many unknown factors in the experiment.

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REFERENCES


