THE PHYSIOLOGY OF MECHANORECEPTORS IN THE ANTERIOR CRUCIATE LIGAMENT

AN EXPERIMENTAL STUDY IN DECEREBRATE-SPINALISED ANIMALS

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The physiological role of mechanoreceptors in the anterior cruciate ligament (ACL) was studied in unanaesthetised decerebrate-spinalised cats and dogs. Tonic activity in the quadriceps and the hamstring increased in response to physiological loading of the ACL. Evoked potentials in the posterior articular nerve (PAN) were elicited by electrical stimulation of the surface of the ligament. ACL loading also induced significant discharges from the PAN. The results suggest that ACL loading has an excitatory effect on the thigh muscles through a multimotor neurone output, and that the PAN is one of the afferent routes from the mechanoreceptors of the ACL. The ACL-muscle reflex may therefore play a physiological role in maintaining knee kinematics.

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Over the past 30 years there have been many experimental studies of the physiological significance of joint receptors (Gardner 1950; Palmer 1958; Ekholm, Eklund and Skoglund 1960; Stener and Petersen 1962; Newton 1982; Rossi 1983; Ferrell, Gandevia and McCloskey 1987). At present there is much clinical interest in the anterior cruciate ligament (ACL), both in its surgical reconstruction (Noyes, Barber and Mangine 1990) and in methods for preventing its rupture (Miyatsu et al 1987).

Histological studies (Kennedy, Weinberg and Wilson 1974; Kennedy, Alexander and Hayes 1982; Schultz et al 1984; Schutte et al 1987) have shown the existence of mechanoreceptors and free nerve endings in the ACL, and altered muscle co-ordination has been demonstrated in ACL-deficient patients (Kålund et al 1990). Barrack, Skinner and Buckley (1989) reported that ACL-deficient

knees had impaired proprioception and suggested that stabilising reflexes originating in ACL receptors may be altered. Miyatsu, Iwahara and Yamashita (1989) recorded the afferent potential of ACL receptors elicited by local electrical stimulation on the surface of the ligament. Solomonow et al (1987) showed the existence of the ACL-hamstring reflex arc using cats anaesthetised with chloralose, but Pope, Cole and Brand (1990) using the same anaesthetic could detect no reflex response of the knee muscles to anterior tibial displacement. It seems therefore that the physiological properties of the mechanoreceptors of the ACL have not yet been determined. These contradictory results may have been due to differences in the method of anaesthesia, in the experimental preparation or in the ACL loading technique. Our aim was to measure, in real time, the fine changes of tone in the muscles around the knee in response to ACL loading. We sought to eliminate the effects of anaesthesia, which might diminish the motor neurone excitability, by using decerebrate-spinalised animals.

MATERIALS AND METHODS

General preparation. The experiments were performed on eight dogs weighing 5 to 10 kg and on three cats weighing 2.0 to 3.5 kg. Under oxygen-halothane anaesthesia, we performed tracheal intubation and ligation of both carotid arteries. The cranium was opened and the cortex overlying the midbrain and thalamus was removed by suction ablation. A precollicular transection was then performed using suction, and the trough formed was packed with gelfoam. In addition to the decerebration, spinal-cord transection was carried out at mid-thoracic level.

Skin incisions were then made from the mid-thigh to the mid-calf to expose the muscles and tendons around the knee. The insertions or origins of the muscles crossing the joint were all detached except for the popliteus muscle. The popliteal neurovascular network and the posterior articular nerve (PAN) were carefully dissected using a magnifying glass. The quadriceps and hamstring tendons were identified and detached from the tibia. The anterior capsule of the knee was partially incised to confirm the normal appearance of the ACL which was

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then detached from the tibia in continuity with a block of bone, $10 \times 10 \times 10$ mm. The pelvis, femur and tibia of the experimental animal were firmly fixed to a frame by bone pins and a system of clamps to prevent even minimal movements of the hindlimb.

Muscle motor unit activity. The halothane anaesthetic was discontinued after completion of the surgical procedures and several hours elapsed before any motor unit recordings were taken. The quadriceps and hamstring tendons were connected to a controlled loading system and were preloaded by 5 to 20 N to induce their tonic contraction. The EMG of these muscles was recorded using a pair of fine wire electrodes placed in the middle portion of each muscle.

recorded on magnetic tape using a DISA Neuromatic 2000 (Dantec Medical A/S, Skovlunde, Denmark) and an MR-30 cassette DATA Recorder (Teac Corporation, Tokyo, Japan). The ECG and body temperature were monitored continuously throughout the experiment to maintain the physiological condition of the animals within normal limits.

Afferent recordings from the posterior articular nerve. In two dogs and two cats the afferent potentials of the ACL mechanoreceptors were recorded from the PAN. The nerve was exposed for 2 to 3 cm under a magnifying glass (Ferrell et al 1987) and laid on platinum bipolar electrodes. First, the surface of the ACL was stimulated by electrical current with a 3 mm bipolar stimulator. The

Fig. 1

Diagram to show the procedure for applying axial loading to the anterior cruciate ligament (ACL) by a bone block detached from the tibia. Bone pins connected to a frame immobilised the preparation. PAN = posterior articular nerve, Q = quadriceps, H = hamstring. Thick arrows indicate the load direction of the ACL and each muscle.

The bone block from the tibia, with the attached ACL, was connected to an Instron machine which applied a graded tensile force. The knee was fixed in 90° to 120° of flexion. A schematic diagram of the experimental procedure is shown in Figure 1.

There were two steps in each experiment. The first, before loading the ACL, was to acquire a steady-state EMG discharge in the quadriceps or the hamstring by applying, to each muscle, loads ranging from 5 to 20 N. The second was to measure the change from this steady-state EMG discharge when a load of 10 to 60 N was applied to the ACL. The EMG changes produced were evaluated by filtered (60 Hz to 3 kHz) and rectified signals

amplitude of the stimulation was 5 to 20 mA and the frequency 1 Hz. The ACL was then gradually loaded and the afferent potentials from the PAN were recorded.

RESULTS

Muscle motor unit activity elicited by ACL loading. An axial load of 5 to 20 N applied to the patellar tendon typically produced a tonic steady-state quadriceps discharge (Qss) at a tendon force between 10 and 20 N (Fig. 2).

The quadriceps EMG discharge changed from the steady-state Qss in response to ACL loads of 10 to 30 N,

the amplitude of this response corresponding to the magnitude of the ACL load. Decrease in the EMG discharge after the release of the ACL load was also reproducible (Fig. 3). This response was shown in 10 of the 11 animals. The whole sequence was repeated at least twice in each animal and was found to be reproducible.

The hamstring muscle behaved similarly. Figure 4 shows a typical pattern of change from steady-state hamstring discharge (Hss) in response to ACL loading. Hss increased in response to ACL loads of up to 30 N but loads of more than 30 N produced no further changes (Fig. 4).

Fig. 2

A typical tonic quadriceps EMG discharge in a decerebrate-spinalised animal with no load applied to the ACL. An axial load of 5 to 20 N, gradually delivered to the quadriceps through the patellar tendon, typically produced a tonic steady-state quadriceps discharge (Qss) at a tendon force of 10 to 20 N.

The average maximum integrated EMG discharge induced by ACL loading was 2.1 ± 1.1 times the steady-state discharge in the quadriceps, and 2.2 ± 0.9 times that in the hamstring. The average ACL load which elicited the maximum EMG discharge was 26 ± 11 N for the quadriceps and 30 ± 13 N for the hamstring.

Afferent readings from the posterior articular nerve. The evoked potentials in the PAN induced by electrical stimulation of the proximal part of the ACL surface are shown in Figure 5. The calculated nerve conduction velocity was 20 to 30 m/sec. Stimulation of the distal part of the ACL did not evoke PAN afferent potentials, nor did stimulation of the tissue surrounding the ACL.

Loading the ACL with 10% to 50% of the body-weight also evoked afferent activity in the PAN (Fig. 6).

DISCUSSION

This study suggests that the muscles which stabilise the knee can be controlled by the load on the ACL. The activity of the quadriceps and hamstring both increased when load was applied to the ACL.

The average ACL load which induced the maximum EMG discharge was less than the animals' body-weight

and this suggests that the levels of thigh muscle tone could be altered by loads within the physiological range (Krauspe, Schmidt and Schaible 1992).

Solomonow et al (1987) reported that the ACL-muscle reflex arc in cats had a high load threshold but in our experiments the loading which caused motor unit discharges was well below that required to rupture the ACL. In a previous experiment, we found the average maximum tensile strength of the ACL in five adult dogs to be 700 ± 70 N. The load threshold to activate the ligament-muscle reflex was therefore 1% to 10% of the maximum tensile strength of the ACL. Perhaps the

Fig. 3

A typical change from steady-state quadriceps EMG (Qss) in response to ACL loads of 10 to 30 N. The amplitude of the EMG response depended on the magnitude of the ACL load. The decrease in the EMG discharge after the release of the ACL load was also reproducible. The black arrowheads indicate the times of the ACL load applications.

hamstring reflex elicited by Solomonow et al was initiated by receptors in the capsule or tendons and not from the ACL.

Decerebrate animals have been used in many experiments to study motor neurone output (Ekholm et al 1960; Baxendale, Ferrell and Wood 1988), because in such a preparation it is easier to detect dynamic change in motor unit discharges generated from joint and muscle afferents. In our experiment, because all the muscles and tendons across the knee were detached, and because no movements could occur with the limb in the frame, no other tendon or muscle afferents could be activated. Since the ACL load was applied through a small block of tibial bone, not only were the surface and the substance of the ACL left intact but no other soft tissue (capsule, tendon) was stimulated.

The ACL has several types of mechanoreceptor (Kennedy et al 1974; Schultz et al 1984; Morisawa and Yamamoto 1989). Some adapt rapidly and some slowly, and therefore the latency of the motor units responding to ACL stimulation is variable. The recruitment level of these motor units was not constant (Figs 3 and 4), possibly because the excitatory effects were generated from a complicated neural pathway (Hongo, Jankowska and

Fig. 4

A typical pattern of change from steady-state hamstring EMG (Hss) induced by 20 N loading in this muscle in response to ACL loads. There was an EMG response to ACL loads up to 30 N, but loads of more than 30 N did not produce further changes. At these higher loads, however, the level of EMG activity was still greater than that before ACL loading.

Fig. 5

Tracing of the evoked potentials from the posterior articular nerve induced by electrical stimulation (intensity 10.2 mA; frequency 1 Hz) of the proximal part of the ACL surface. Upward deflection indicates negativity of the active recording electrode. The latency of this potential was about 1.2 msec, measured from the time of stimulation to the onset of the first spike (between the two arrow heads).

Fig. 6

The integrated afferent activity of the posterior articular nerve (PAN) evoked by mechanical loading of the ACL from 20 to 60 N, in a dog weighing 12 kg. The increase in the activity corresponded to the magnitude of the ACL load and the activity decreased rapidly after release of the ACL load. The upward deflection indicates a negative electric potential.

Lundberg 1969; Fedina and Hultborn 1972; Goslow, Reinking and Stuart 1973; Lundberg, Malmgren and Schomburg 1978; Johansson, Sjölander and Sojka 1986).

The delay between ACL loading and muscle response varied between animals and between experiments on the same animal. The maximum level of muscle tone in response to ACL load also varied between animals. These results suggest that the receptor afferents from the ACL do not act on alpha motor neurones. The same conclusion was reached by Johansson, Sjölander and Sojka (1990).

The ACL-deficient knee is a serious problem in clinical practice. Our results suggest that the role of the ligament in controlling muscle tone in the human limb deserves to be studied in more detail (Brand 1989; Berchuck et al 1990).

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