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Clinical and neurophysiological abnormalities before and after reconstruction of the anterior cruciate ligament of the knee

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Objectives – We aimed to study knee proprioception and somatosensory evoked potentials (SEPs) to stimulation of the common peroneal nerve (CPN) in 7 patients with lesion of the anterior cruciate ligament (ACL) before and after ACL reconstruction. *Materials and methods* – We recorded the spinal N14 and scalp P27 potentials in 5 patients, while in the remaining 2 patients we calculated scalp SEP maps by 20 electrodes. The knee proprioception was tested by comparing the sensitivity to movement of both the knees. *Results* – Before surgery, all patients showed decreased knee position sense and lack of the cortical P27 potential on the side of the ACL lesion. Arthroscopic reconstruction of the ligament improved neither the knee proprioception nor the somatosensory central conduction. *Conclusion* – We suggest that the loss of the knee mechanoreceptors can be followed by modifications of the central nervous system, which are not compensated by other nervous structures.

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Key words: proprioception; central nervous system; somatosensory evoked potentials; anterior cruciate ligament

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Patients with tears of the ACL often experience knee instability. This feeling increases when the knee of the patient fails under rotational stress conditions and the anterior profile of the tibia subluxates forwards. Earlier studies (1) focused on the loss of dynamic protective reflexes due to ACL failure (for instance, hamstring contraction and quadriceps relaxation near the extremes of the knee extension). Proprioceptive receptors, which are normally included in the ACL (2), are the first element of the protective reflex arc that rules the contraction of the tight muscles (3, 4). It has been more recently demonstrated that the position sense of an ACL damaged knee can be reduced (5, 6), so confirming the opinion that the ACL plays a primary role among all the sources of proprioceptive information from the knee. Both the sensory loss and impairment of the stabilizing reflexes following the ACL lesion can be explained by disruption of the ACL mechanoreceptors.

Although the techniques which have been developed to substitute the ACL function are very different from each other, a number of studies agree about the poor correlation between surgical results and patients' satisfaction (7, 8). While some patients with unsatisfactory surgical results are able to return to sport activity, others without remaining anterior knee laxity complain of "giving away" episodes (6). Since the ACL mechanical function can be restored by surgery, it is probable that some unbrilliant postoperative outputs are due to the permanent loss of the knee position sense following the ACL damage (6).

In 19 patients with ACL lesion, we had previously recorded SEPs to CPN stimulation at the popliteal fossa, above the site at which any articular branches would have joined the main trunk (9). Since it has been suggested that proximal inputs are only involved in the building of cortical SEPs to CPN stimulation (10–12), the recording of

cortical CPN SEPs allows to study the functional properties of the sensory afferents coming from the knee. In the past study (9), we had demonstrated not only that in our ACL patients proprioceptive deficits are correlated to CPN SEP abnormalities, but also that the functional impairment involves the sopralemniscal tract of the somatosensory pathways. Moreover, SEPs after both posterior tibial nerve stimulation at the ankle and tibial nerve stimulation at the knee were normal in our patients. We had concluded that the ACL lesion can be followed by both neuroreceptive loss and changes of the central nervous system (CNS), demonstrated by CPN SEP abnormalities.

In the present study, we recorded CPN SEPs and tested the knee position sense after surgery in the patients, who had showed neurophysiological abnormalities in our first report. Our aim is to investigate if the loss of proprioceptive information and the CNS modifications following ACL damage have to be considered as permanent or, instead, afferent inputs coming from other articular structures, including those used to substitute the ACL, may compensate either the sensory deficit or the CNS neurophysiological abnormalities.

Materials and methods

We studied 7 patients with ACL lesion, confirmed by arthroscopy excluding also any other intraarticular lesion. Sural nerve sensory conduction study, tibial and peroneal nerve motor conduction studies and concentric needle EMG examination in lower limb muscles did not show abnormalities. Blood tests were also performed to exclude the existence of other pathological conditions, such as diabetes mellitus or vitamin B_{12} deficiency. Our patients showed neither gait impairment nor pain and temperature hypesthesia; joint and touch sensation, tested by common clinical methods, was preserved in all patients. They underwent arthroscopic reconstruction of the ACL harvesting the medial third of the patellar tendon. While in all our 7 patients a clinical anterior laxity was demonstrated by anterior drawer test, Lachman test and pivot shift test before surgery, the postoperative control did not show any clinical sign of knee laxity. SEP and clinical studies were carried out in all patients at least 2 years after surgery.

In order to test the knee position sense, we used the apparatus already described in detail by Barrack et al. (5). Briefly, subjects were seated and custom-made Jobst air splints were placed above and below the knee joint and inflated to a pressure of 20 mmHg to minimize cutaneous sensation interference. Leg extremities were connected by wires and pulleys to a slow speed motor.

Table 1. Normal values of CPN SEPs and knee proprioception

	Mean ± SD	Limit of normal values (mean +3 SD)
CPN SEPs		
N14 latency (ms)	13.9 ± 0.96	16.8
P20 latency (ms)	19.6 ± 1.86	25.2
P27 latency (ms)	26.1 ± 2.3	33
P27–N14 int. latency (ms)	12 ± 2.4	19.2
Knee proprioception		
Knee position sense, Interside difference (degrees)	0.12 ± 0.08	0.36

The starting position was 40°, with legs suspended passively, and a flexion movement between 30° and 40° was performed. Both sides were examined independently. Subjects pressed a button when they felt position changes of the knee. The linear movement of the wire was then calculated and converted to angular deflection. The test was repeated 5 times on each side and the average value was considered as the result. The normal value of the interside (right vs left) difference of the angular deflection, which has been calculated from 15 healthy subjects, is shown in Table 1.

For SEP recording, patients lay on a couch in a warm and semidarkened room. CPN stimulations (0.3 ms duration, 5 Hz) were delivered by skin electrodes at the popliteal fossa; stimulus intensity was adjusted slightly above the motor threshold. Samples with excessive interference were automatically edited out of the average. Two averages of 2048 trials each were obtained and printed out by the computer on a desk-jet printer. In 5 patients, SEP recording was performed by using a filter bandpass ranging from 30 to 3000 Hz (–3 dB at cut off point, 6 dB per octave). Responses were averaged with a bin width of 196 μs on a total analysis time of 100 ms. The recording electrodes (impedance below 5 kohm) were placed over the spinal process of the 12th dorsal vertebra (T12) and at the scalp points Cz, Fz, P3, and P4 (10–20 system). In order to record spinal N14 potential, we connected grid 1 of the amplifier to the T12 electrode and grid 2 to an electrode placed over the anterior abdomen (Abd). This montage permits selective recording of the activity generated by the transverse dipolar source located in the lumbosacral spinal cord (13); moreover, this technique can cancel noise from the ECG activity that is picked up by both T12 and anterior electrodes (14). In order to assess conduction in peripheral nerve fibres we measured the peaking latency of the spinal potential. We referred scalp electrodes to linked ears to record cortical as well as far-field potentials (15). We calculated the latency of cortical P27 response at Cz and at the parietal

site ipsilateral to the stimulated nerve, where these potentials are recorded with the highest amplitude (15); we also considered the interpeak latency between the cortical and spinal responses to assess conduction in the central somatosensory pathways. Finally, we evaluated the latency of the positive P20 which is recorded at all scalp leads and immediately before the P27 at Cz. This scalp far-field is assumed to be generated in the brainstem tract of the lemniscal pathways (15). In the other two patients, disk recording electrodes (impedance below 5 kΩ) were placed at 20 locations of the 10–20 system (excluding Fpz). We referred scalp electrodes to linked ears. The analysis time was 64 ms with a bin width of 250 μs. The amplifier bandpass was 3–3000 Hz. In order to ensure baseline stabilization, SEPs were digitally filtered off-line by means of a digital filter with a bandpass of 19–1900 Hz. Maps showing the distribution of the responses over the scalp were obtained by linear interpolation from the 4 nearest electrodes. Normative data issued from 24 healthy subjects are shown in Table 1.

Results

Clinical findings

As shown in Table 2, before surgery all our 7 patients showed decrement of the knee position sense on the same side as the ACL lesion. After surgery, a proprioception impairment was still present in all patients and angular values of the position sense did not significantly differ from those which had been found before the ACL reconstruction (paired t-test $P=0.282$).

SEP findings

Before surgery, all our patients showed the same SEP pattern to CPN stimulation. The lemniscal P20 potential was recorded with normal latency on both sides, while the cortical P27 response was absent on the damaged ACL side. The topographic study (Fig. 1), which was performed in 2 patients (nos 6 and 7), showed that a positive potential in the 27 ms range of latency was recognizable neither at Cz nor in the other 19 scalp traces. Conversely, the P27 was recorded with normal latency after stimulation of the CPN contralateral to the ACL lesion. The 5 patients (nos 1–5), in whom the T12-abdomen recording technique was employed, showed a normal spinal N14 response on both sides. The central somatosensory conduction time, which was calculated by the P27-N14 latency difference, was within normal limits on the undamaged ACL side. After surgery, SEP results were unmodified in all our patients. In particular, no identifiable P27 was recorded on the same side as the ACL reconstruction (Fig. 2). SEP findings in our patients are shown in Table 3.

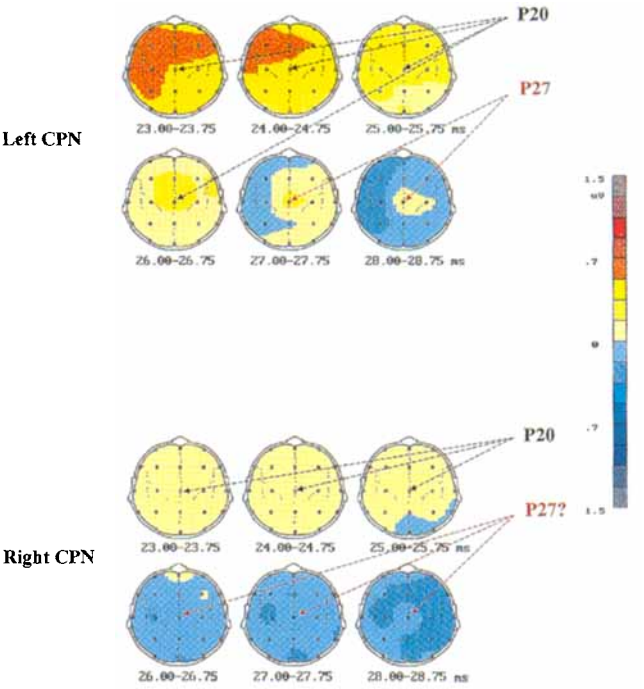

Fig. 1. CPN SEP findings before surgery in patient no. 6 with right ACL lesion. Scalp SEP topographies in the P20–P27 range of latency to stimulation of both left (upper) and right (lower) CPNs are shown. Scalp potential positivity is coded in red, and negativity in blue shades. While a clear positive potential, following the widespread P20, is evident on the vertex to left CPN stimulation, the right CPN stimulation shows a P20, which is not followed by any other positive response.

Table 2. Clinical findings

	Knee proprioception, interside difference (degrees)	
Patients	Before surgery	After surgery
1	0.6	0.7
2	0.63	0.65
3	0.8	0.7
4	0.75	0.8
5	0.85	0.83
6	0.55	0.6
7	0.46	0.6

Discussion

Before surgery, in all our patients the cortical P27 response was not identifiable on the same side as the ACL lesion. This result cannot be due to the great intersubject variability of the P27 scalp distribution, as demonstrated by the fact that 2

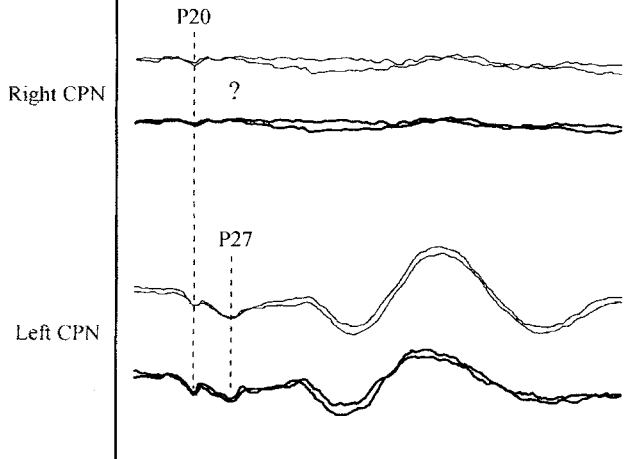

Fig. 2. CPN SEPs in patient no. 3 with right ACL lesion. Cz traces before (thin) and after (thick) surgery are shown. The P20 far-field potential is identifiable on both sides before as well as after the ACL reconstruction. While a clear P27 is evoked by the left CPN stimulation, the P27 lack, which is evident in the traces recorded before surgery, persists also after the ACL reconstruction.

patients, who underwent topographic study, showed no P27 response in Cz or in any other scalp traces. Moreover, the finding of a normal P20 far-field, which is probably generated in the brainstem (15), demonstrates that in our patients the central somatosensory dysfunction is located in the sopralemniscal tract of the somatosensory system. We suggest that the P27 absence is probably related to the loss of proprioceptive inputs from the ACL-deficient knee. Since the CPN stimulation at the popliteal fossa obviously activated fibres not only from the knee articular structures, but also from more distal sites, it might be inconceivable that a dysfunction of such a small amount of fibres from the knee entails the lack of cortical potentials which should be evoked also by many other afferents. However, it is possible that proximal leg afferents are mostly involved in the building of cortical CPN SEPs. Indeed, in healthy subjects the proximal CPN stimulation at the knee evokes cortical responses which are paradoxically lower in amplitude than those recorded to the distal stimulation of the peroneal nerve (PN) at the ankle (9, 10, 12). Interference between muscle and cutaneous afferent inputs at the central level (16) has been claimed to explain why distal afferent inputs contribute little to the cortical CPN SEPs (12). Moreover, in ACL deficient patients showing CPN P27 absence we had found that the PN stimulation at the ankle, which activates distal leg afferents, allowed to obtain normal cortical SEPs (9).

Our patients showing P27 absence with still preserved P20 probably underwent a CNS reorganization above the medial lemniscus, thus suggesting that CNS changes can occur also in minor lesions of proprioceptive afferent inputs. Experimental studies have demonstrated different kinds of CNS modification following the loss of peripheral inputs (17–19). On the basis of what has been found in animals, the lack of P27 in our patients may be explained by: 1) modifications in the response properties of the cortical or thalamocortical neurones, such as increased response threshold or latency; 2) progressive occupation of the CPN cortical representation by afferent inputs of the nearest areas; 3) reorganization of the spinal maps due to decrement of peripheral CPN inputs. However, the last mechanism can be excluded by

Table 3. CPN SEP findings

		N14 latency (ms)		P20 latency (ms)		P27 latency (ms)		P27-N14 int. (ms)	
Patients	Side	Before	After	Before	After	Before	After	Before	After
1	R	12.8	12.2	18.4	17.8	25.6	25.4	12.8	13.2
	L*	12.8	12.4	18.8	18.4	Absent	Absent	–	–
2	R*	15.5	15.5	19.8	19.8	Absent	Absent	–	–
	L	15.1	15	19.8	19.8	27.5	27.5	12.4	12.5
3	R*	12.7	13	16.8	17	Absent	Absent	–	–
	L	12.6	12.6	16.7	16.7	24	24.2	11.4	11.6
4	R	13.2	13.2	18.4	18.6	25.2	25.2	12	12
	L*	13.6	13.6	18	18	Absent	Absent	–	–
5	R*	14.8	14.6	19.6	19.6	Absent	Absent	–	–
	L	14	14.2	20	19.8	27.6	27.8	13.6	13.6
6	R*	–	–	20	20	Absent	Absent	–	–
	L	–	–	19.5	19.6	27.8	27.4	–	–
7	R	–	–	22.5	22.6	27.3	27.3	–	–
	L*	–	–	22.5	22.2	Absent	Absent	–	–

*Side of ACL lesion

the finding of a normal spinal N14 response in our patients.

In the postoperative recordings, the persistent P27 absence suggested that the CNS modifications following the ACL mechanoreceptors loss are not influenced by the reconstruction of the ligament, which did not allow for regeneration of nervous tissue. Moreover, although the knee mechanical function was completely restored after surgery, all our patients showed a persistent deficit of the knee position sense and were not able to return to previous sport activities. Therefore the loss of ACL proprioceptive inputs has to be considered as permanent. On the other hand, it is well known from the clinical practice that most patients, who undergo the ACL reconstruction, refer a subjective satisfaction and can return to the previous quality of life. Even if further studies on a larger series of patients are needed for definitive conclusions, we can suppose that the ACL deficient patients who do not develop a proprioceptive deficit are likely to have a better postoperative outcome. Previous studies agree that a decreased knee position sense does not always follow the ACL lesion (5, 6). It is possible that in some cases inputs from other knee and muscular proprioceptors can compensate the loss of ACL inputs. Since all our patients showing SEP abnormalities had also a proprioceptive impairment, the patients who are able to compensate for the loss of ACL inputs probably develop neither impairment of the knee position sense nor central somatosensory abnormalities. An improvement of the joint position sense after ACL reconstruction was reported by Co et al. (20) who hypothesized a compensatory function of muscular structures, such as the quadriceps, to explain their findings. We cannot exclude that in patients who develop proprioceptive deficit without CNS modifications compensative phenomena may occur. However, our results suggest that the functional recovery after ACL reconstruction is probably worse when the sensory deficit is coupled with central CPN SEP abnormalities.

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