Knee ligaments and proprioception

Sir–I enjoyed reading the guest editorial by Gillquist, entitled "Knee ligaments and proprioception", *Acta Orthop Scand* 1996; 67 (6): 533-535. I agree with the general line of reasoning in the article, although I would like to comment on a few points.

Review of the extensive literature on proprioception of ligaments has raised several issues in which repeated studies report findings contradicting the original study on a particular subject. This is mentioned in the editorial regarding the work of Barrack et al. (1989) and Barrett (1991) on proprioception tests after acl rupture, but I missed several comparable instances. For example, regarding "reflex hamstring contraction latency" (rhcl), the work of Beard et al. (1993) is said to have shown an increase in rhcl after acl rupture. However, Jennings (1994) was not able to confirm such an increase. Wojtys and Huston (1994), in a comparable experimental set-up, arrived at yet other values, with limited differences between affected and contralateral legs.

Interestingly, in a prospective series of 16 patients, Beard et al. reported an extreme variation of rhcl preoperatively (0–81 ms). Postoperatively, no clear difference was present between affected and contralateral legs. This led Beard et al. to state that "Change in proprioception is an effect of mechanical modification of the joint on a reflex of muscle spindle origin rather than other types of neurological conditioning" (Beard et al. 1996). This would seem to be an abandonment of their previous hypotheses on hamstring contraction latency, based on an "acl-muscle reflex" (Beard et al. 1993).

Regarding the work of Solomonow et al. (1987) it is important to note that validation of their EMG findings in animal experiments is still debated. Work by Pope et al. (1990) and by Cole et al. (1995) contradicts Solomonow's findings in animal experiments. Raunest et al. (1996), on the other hand, reported findings comparable to those of Solomonow's, but, like Solomonow, he applied considerable force (200–500 N in a sheep model) to demonstrate an effect. As the work of Cole et al. has shown, meticulous care must be taken in this type of experiment, with fixation of the limb to prevent stimulation of structures other than the specific ligament.

Regarding histological studies, several authors (DeAvilla et al. 1989, Koch et al. 1995, McLain 1995) have drawn attention to the difficulties of "classic" histology in identifying neuroreceptors in ligaments, and even more so in quantifying them. Certainly, immunohistochemical techniques (Gómez-Barrena 1990, Madey et al. 1993, Rivard et al. 1993) provide far better sensitivity for identification of neurostructures in the acl, although identification of the type of receptor is often difficult using these techniques.

Unwillingly, Gómez-Barrena et al., in their remarkable article in *Acta Orthop Scand* 1996; 67 (6): 545-552, compound histological confusion by remarking that their mean of 26 receptors per acl parallels the number found by Haus and Halata (1990), using classic histology. However, the 26 receptors of Haus and Halata comprise all the receptors found in a total of 21 acl's, and not a mean per acl.

Regarding the effects of ligament receptors on muscle stiffness, it should be noted that studies reporting such an effect in humans are lacking. The fusimotor system is a complex system with input from many sources. Johansson et al. (1989a, b) performed experiments in cats showing an influence of skin afferents and movement stimuli of the contralateral leg on the gamma system. Therefore, experiments with stimulation of ligament afferents in a more physiologic context may be difficult to devise, and even more difficult to interpret. Hitherto, it has been unclear whether any resulting changes in muscle stiffness will be large enough to cause a significant change in joint stiffness.

In conclusion, I agree with Gillquist that the acl has a sensory innervation, the number of receptors being rather small. As I hope to have indicated, many of the important issues regarding these acl receptors have not been settled unequivocally which arouses more interest in this field of research.

Tom Hogervorst

Dept. of Orthopaedic Surgery, OLVG Hospital, PO Box 95500, 1090 HM Amsterdam, The Netherlands. Fax +31 20 5993998. e-mail tomhog@pi.net

Sir—I agree completely with Dr. Hogervorst on the complexity of research on proprioception. While it is impossible to cover all possible contradictions in a short editorial, I agree that it is easy to find inconsistencies when different articles are compared. This is

because widely different techniques, like conventional histology, immunohistochemistry or physiologic experiments are used to address the problem. As compared to many other body systems (blood coagulation, endocrinology, etc), proprioception is incompletely understood. The research field is very incomplete. Anatomical detail is one thing and physiologic function another. I think that in coming decades picture-generating sciences (like histology of various kinds) will give way to physiologic experiments that may reveal more about the organization and functioning of this important system. Recent progress concerning the value of skin receptors for joint position sense is encouraging (Edin and Johansson 1995).

Jan Gillquist

Department of Sports Medicine, Faculty of Health Sciences, Linköping University, S-581 85 Linköping, Sweden
Tel +46 13-224176. Fax -224434

Sir—As stated by Dr. Hogervorst, propioception studies remain highly controversial. Among all the interesting issues raised by Dr. Hogervorst, we shall discuss quantification further.

Most researchers agree that quantification would provide a more precise definition of the propioceptive role of a certain structure, such as the anterior cruciate ligament. Difficulties in directly measuring the total number of ligament endings have been outlined by Dr. Hogervorst. In our study (Gómez-Barrena et al. 1996), we focused instead on indirect measurements.

We obtained the total number of spinal ganglia neurons receiving information from the ligament where we injected the tracer. This indirect quantification of ligament propioception relates to well-functioning neurons (to achieve the retrograde axonal transport from the periphery onto the neuronal body) related to this ligament. This technique is of value for comparing with other structures (in our paper, the patellar tendon). This relative quantification allowed us to discuss the relative importance of anterior cruciate ligament propioception.

Several reasons account for the discrepancy between a relatively small number of histologically-identified endings per ligament and the number of labeled neurons related to that particular ligament. Free nerve ending-uptake of the tracer may raise the number of labeled neurons considerably. Transsynaptic transport might lead the tracer to other related cells, although no convincing evidence has been provided of this kind of transport. Exploring transneuronal transport of the tracer would probably generate further data on the relationship of these labeled neurons, although this was not considered in our study. Other sources of overestimation and underestimation have been thoroughly discussed in our paper.

Our observation about the work of Haus and Halata (1990) relates to the scarcity of data concerning variability, rather than to the total number of endings. The parallelism mentioned is only a coincidence. In their publication, these authors do not provide enough data to clarify the number of endings per ligament. Unless the whole ligament is sectioned, the total number of endings cannot be obtained. However, this is particularly difficult in human ligaments due to the size and variety of methods used. The homogeneous distribution of the tracer was checked in our study and can be explained by its great diffusion in a small ligament. Of course, care must be taken in the controls.

We are aware that our study raises another controversy, but we do not focus on the number of ligament neural endings but that of related neurons. We feel that this number provides a more functional idea of how much propioception relates to a particular structure. Its main disadvantage: it is available only for animal experiments.

Enrique Gómez-Barrena

Servicio de Traumatología y Cirugía Ortopédica, Hospital Real y Provincial "Ntra.Sra.de Gracia", Ramón Cajal 60, Zaragoza 50004, Spain. Tel +34 972 44-0022. Fax -5869

Barrack R L, Skinner H B, Buckley S L. Proprioception in the anterior cruciate-deficient knee. Am J Sports Med 1989; 17: 1-6.

Barrett D S. Proprioception and function after anterior cruciate reconstruction. J Bone Joint Surg (Br) 1991; 73: 833-7.

Beard D J, Kyberd P J, Fergusson C M, Dodd C A. Proprioception after rupture of the anterior cruciate ligament. J Bone Joint Surg (Br) 1993; 75: 311-5.

Beard D J, Dodd C A, Simpson A H. The effect of reconstruction on proprioception in the acl-deficient knee. Trans Orthop Res Soc 1996; 21: 786.

Cole K J, Brand R A, Daley B J. The sensitivity of joint afferents to knee translation. Iowa Orthop J. 1995; 15: 168-73.

De Avila G A, O'Connor B L, Visco D M, Sisk T D. The mechanoreceptor innervation of the human fibular collateral ligament. J Anat 1989; 162: 1-7.

Edin B, Johansson N. Skin strain patters provide kinaesthetic information to the human central nervous system. J Physiol 1995; 487: 243-51.

Gillquist J. Knee ligaments and proprioception. Guest editorial.Acta Orthop Scand 1996; 67 (6): 533-5.

Gómez-Barrena E, Martinez-Morreno E, Munurea L. Segmental sensory innervation of the anterior cruciate ligament and the patellar tendon of the cat's knee. Acta Orthop Scand 1996; 67 (6): 545-52.

Haus J, Halata Z. Innervation of the anterior cruciate ligament. Int Orthop (SICOT) 1990; 14: 293-6.

Jennings A G, Seedhom B B. Proprioception in the knee and reflex hamstring contraction latency. J Bone Joint Surg (Br) 1994; 76: 491-4.

Johansson H, Sjöllander P, Sojka P, Wadell I. Effects of natural and electrical stimulation of skin afferents on the fusimotor system of triceps surae muscle. Neurosci Res 1989a; 6: 537.

Johansson H, Sjöllander P, Sojka P, Wadell I. Different fusimotor reflexes from the ipsi- and contralateral hind limbs of the cat assessed in the same primary muscle spindle afferents. J. Physiol (Paris) 1989b; 83: 281.

Koch B, Kurriger R, Brand R A. Characterisation of the neurosensory elements of the feline cranial ligament. J Anat 1995; 187: 353-359.

Madey S M, Wolff A J, Brand R A, Cole K J. Characterization of ACL neural endings with wheat germ agglutinin—Horse radish peroxidase. Trans Orthop Res Soc 1993; 18: 332.

McLain R F. Correspondence. J Bone Joint Surg (Br) 1995; 77: 982.

Pope D F, Cole K J, Brand R A. Physiologic loading of the acl does not activate quadriceps or hamstrings in the anesthetized cat. Am J Sports Med 1990; 18: 595-9.

Raunest J, Sager M, Burgener E. Proprioceptive mechanisms in the cruciate ligaments: an electromyographic study on reflex activity in the thigh muscles. J Trauma 1996; 41: 488-93.

Rivard C H, Yahia L H, Rhalmi S, Newman N. Immunohistochemical demonstration of sensory nerve fibers and endings in human acl. Trans Orthop Res Soc 1993; 18: 61.

Solomonow M, Baratta R, Zhou B H. The synergistic action of anterior cruciate ligament and thigh muscles in maintaining joint stability. Am J Sports Med 1987; 15: 207.13.

Wojtys E M, Huston L J. Neuromuscular performance in normal and acl-deficient lower extremities. Am J Sports Med 1994; 22: 89-104.