THE MUSCLES IN CHRONIC LIGAMENTOUS INSTABILITY OF THE KNEE : AN EXPERIMENTAL STUDY

F. PELLISÉ, A. NAVARRO-QUILIS

Several authors have formulated the idea that insufficiency of the anterior cruciate ligament (ACL) might lead to a biological adaptation, consisting of atrophy of the quadriceps and other extensors (antagonists of the ACL) and relative preservation of the hamstrings and other flexors (agonists of the ACL).

We studied the modifications in hindlimb muscles that spontaneously occurred in 36 rabbits with surgically-induced chronic ligamentous instability of the right knee. The unoperated left hindlimb served as a control. Three months after surgery the rabbits were sacrificed and the muscles dissected for macroscopic and microscopic evaluation. In the anterior cruciate ligament-deficient limbs the extensors (quadriceps, glutei and extensor digitorum longus) showed significantly greater atrophy than the other muscles ($p < 0.05$). Six hundred twenty-two fibers from atrophic muscles, 373 from hypertrophic muscles and 879 from control muscles were evaluated by microscopy. The atrophic muscles showed a significantly lower percentage of type I fibers (responsible for maintenance of posture) and a reduction in the diameter of both type I and II (responsible for rapid movement) fibers with respect to the controls ($p < 0.05$). The hypertrophic muscles showed a significantly greater proportion of type I fibers and an increase in the diameter of type II fibers with respect to the controls ($p < 0.05$). Our results support the idea that there is spontaneous adaptation of the muscles to compensate for ligamentous instability.

Keywords : knee ; anterior cruciate ligament ; instabilities ; muscle.

Mots-clés : genou ; laxité ligamentaire chronique ; compensation musculaire ; genou.

INTRODUCTION

Documented evidence of spontaneous compensation for chronic ligamentous instability was reported almost 15 years ago (17), but there is still discussion regarding this matter. In a later work, Gerber *et al.* observed selective atrophy of the quadriceps in anterior cruciate ligament-deficient limbs by means of computed tomography (9). Similar findings, associated in some cases with spontaneous hypertrophy of the hamstrings, have been reported by other authors (12, 13). This combination of events is postulated to be dynamic compensation to reduce the anterior tibial translation, caused by the quadriceps, and to offset the compromised anterior cruciate ligament (2, 23). Whereas several authors believe that the structure and function of muscles around unstable joints is modified to enhance joint stability (1, 2, 4, 23), others do not (15).

In daily clinical practice an analysis of the true natural history of ligamentous injuries and a comparison of different patterns of ligamentous instability is difficult. We therefore designed this experimental study to determine whether the modifications spontaneously produced in the muscles around an unstable joint can be interpreted

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as a biological adaptation of the active joint stabilizers to compensate for the loss of joint stability.

MATERIAL AND METHODS

We studied 36 New Zealand white rabbits (31 females and 5 males), weighing between 3000 and 5200 grams (mean weight 3953 g). Six types of ligamentous knee instability were produced. The animals were divided into six groups according to the ligament that was surgically sectioned : MCL group (medial collateral ligament), LCL group (lateral collateral ligament), ACL group (anterior cruciate ligament), PCL group (posterior cruciate ligament), MCL + ACL group (medial collateral ligament and anterior cruciate ligament) and ACL + PCL group (both cruciate ligaments). In all animals the lesion was created in the knee of the right hindlimb ; the left hindlimb was used as the control limb. A medial or lateral incision was made over the ligament to section the collateral ligaments, and an anteromedial arthrotomy was used to divide the cruciate ligaments. The operated knees were not immobilized.

a. **Macroscopic evaluation**

Three months after surgery the animals were sacrificed and the muscles of both hindlimbs were evaluated. Fourteen muscles (tensor fasciae latae, gluteus medius, gluteus profundus and accessorius, vastus medialis, vastus lateralis, rectus femoris, vastus lateralis accessorius, gracilis, coccygeofemoralis, biceps femoris, semitendinosus, cruralis, extensor digitorum longus and gastrocnemius) (3) from each hindlimb were dissected and weighed (mg) on an accurate scale (Mettler AE 100).

We calculated the percentage of change (PC) of each right limb muscle with respect to the left control muscle as follows :

$$PC = (\text{(Weight Left - Weight Right) / Weight Left}) \times 100$$

Positive PC's indicate a relative loss of weight (atrophy) of the right muscle with respect to the left control muscle, and negative PC's indicate a relative weight increase (hypertrophy) of the right muscle with respect to the left control muscle. The muscles were evaluated individually and in groups. Four muscle groups were studied :

1. *extensor muscles* : quadriceps (rectus femoris, cruralis, vastus lateralis, vastus lateralis accessorius and vastus medialis), glutei (gluteus medius and gluteus profundus-accessorius) and extensor digitorum longus ;
2. *flexor muscles* : hamstrings (biceps femoris, coccygeofemoralis, gracilis and semitendinosus), gastrocnemius surale and tensor fasciae latae ;
3. *lateral muscles* (vastus lateralis, vastus lateralis accessorius, biceps femoris and semitendinosus) ;
4. *medial muscles* (vastus medialis, gracilis and semitendinosus).

The PC of each muscle was studied according to the ligamentous injury, and in each injury the PC's of the muscles and muscle groups were compared.

b. **Microscopic evaluation**

The muscles showing the greatest percentage of change were studied with plain microscopy. Eight pairs of muscles, consisting of one muscle from the operated limb and the same muscle from the control limb, were evaluated microscopically : five pairs of vastus lateralis accessorius, one pair of tensor fasciae latae, one pair of gracilis and one pair of biceps femoris. Samples for the histological examination were obtained immediately after sacrificing the animals, frozen in isopentane cooled by liquid nitrogen to -160°C, and stored at -80°C. Unfixed sections of the muscle samples were stained for myofibrillar adenosine triphosphatase at pH 9.4 and at pH 4.3, allowing for fiber-typing. Using enlarged microphotographs of representative histologic fields, we evaluated the number of fibers per field, the percentage of type I (maintenance of posture) and II (rapid movement) fibers and the mean diameter of each type of fiber by means of the "lesser fiber diameter" method (6). Finally, we evaluated any differences between the muscles of the right hindlimb and the same muscles of the control limb.

Statistical analysis was done with programs 1d, 2d, 3d and 7d of the BMDP software package (5). We used the ANOVA analysis of variance with the Bonferroni adjustment to compare the PC of a single muscle or muscle group in the different ligamentous lesions and to compare the PC of different muscles or muscle groups in a single ligamentous lesion. The nonparametric Mann-Whitney U test was used for one-to-one comparison between the different muscles and muscle groups. To compare the percentage of fiber types between the right and the left hindlimb muscles, we used the Chi-square test. A p value of less than 0.05 was considered to be statistically significant in the comparisons.

RESULTS

We dissected 1008 muscles (28 per animal) and discarded 66 (33 pairs) owing to problems resulting from the dissection. A total of 942 muscles were evaluated, giving an average of 26 muscles per rabbit.

a. Macroscopic evaluation

As to the flexion or extension function of the muscles, statistical analysis showed no significant variations in the PC's of the flexor muscles (hamstrings, tensor fasciae latae and gastrocnemius) in the different injuries. However, (table I) the extensor muscles (quadriceps, glutei and extensor digitorum longus) showed significantly greater atrophy in the ACL-deficient limbs (ACL, MCL + ACL and ACL + PCL groups) when compared to the limbs with isolated MCL, LCL or PCL injury.

Table I. — Percentage of change (PC) of the quadriceps, glutei and extensor digitorum longus (E.D.L.) according to the ligamentous injury, either excluding or including the ACL

	Quadriceps (PC)	*Glutei (PC)*	*E.D.L. (PC)*
ACL saved	1.126	1.052	-1.061
ACL divided	12.804	11.480	9.275
p	< 0.001	< 0.001	< 0.01

As to the type of injury, there were no significant differences in the muscles or muscle groups in the limbs with isolated MCL, LCL or PCL injury, in other words without ACL deficiency. However, (table II) in the ACL-deficient limbs (ACL, MCL + ACL and ACL + PCL groups) there was significantly greater atrophy of the extensor muscles with respect to the flexor muscles.

Table II. — Percentage of change (PC) of the muscular groups in ACL-deficient limbs

	Quadriceps (PC)	*Hamstrings (PC)*	*p*
ACL	13.070	3.213	< 0.001
ACL + MCL	13.612	2.889	< 0.001
ACL + PCL	11.730	5.987	< 0.001

b. Microscopic evaluation

Fiber type

In the microscopic study, 622 fibers from *atrophic* muscles and 522 fibers from the corresponding control muscles were evaluated. The percentage of type I fibers (maintenance of posture) in the atrophic muscles (8%) was significantly lower than in the control muscles (12%) (table III).

Table III. — Proportions of type I and type II fibers in the atrophic muscles

	Type I	**Type II**	Total
R. muscle	50 **(8%)**	572 **(92%)**	622
L. control	66 **(12%)**	456 **(88%)**	522
Total	116	1028	1144

p = 0.010
R. muscle = right muscle ;
L. control = corresponding left control muscle.

Three-hundred seventy-three fibers from *hypertrophic* muscles and 357 fibers from the corresponding control muscles were studied. The percentage of type I fibers in the hypertrophic muscles (7.5%) was significantly higher than in the control muscles (4%) (table IV).

Table IV. — Proportions of type I and type II fibers in the hypertrophic muscles

	Type I	**Type II**	Total
R. muscle	28 **(7.5%)**	345 **(92.5%)**	373
L.control	14 **(4%)**	343 **(96%)**	357
Total	42	688	730

p = 0.037
R. muscle = right muscle ; L. control = corresponding left control muscle.

Fiber diameter

The diameter of the type II fibers (rapid movement) was significantly smaller than that of the control muscles in three of the five atrophic muscles assessed ; no significant differences were observed in the two remaining muscles (table V).

Table V. — Diameter of type II fibres in the atrophic muscles assessed

Muscle	PC	R. muscle	L. control	p
V. Lat. Acc.	6.363	63.6μ +/-13.3 (n = 157)	72.0μ +/-11.1 (n = 98)	< 0.001
V. Lat. Acc.	3.725	67.6μ +/-18.7 (n = 84)	75.7μ +/-16.4 (n = 90)	0.002
V. Lat. Acc.	11.844	74.9μ +/-21.2 (n = 107)	82.6μ +/-21.7 (n = 87)	0.013
V. Lat. Acc.	3.821	74.5μ +/-23.5 (n = 99)	76.8μ +/-21.1 (n = 85)	NS
Biceps	2.680	66.5μ +/-17.1 (n = 120)	68.4μ +/-19.0 (n = 79)	NS

PC = percentage of change ; R. muscle = right muscle ; L. control = corresponding left control muscle ; NS = not significant.

In two of the three hypertrophic muscles, the type II fiber diameter was significantly greater than in the control muscles, and in one there were no significant differences (table VI).

Table VI. — Diameter of type II fibers in the hypertrophic muscles assessed

Muscle	PC	R. muscle	L. control	p
T.F. Latae	-0.428	72.6μ +/-20.3 (n = 119)	60.8μ +/-18.4 (n = 132)	< 0.001
Gracilis	-3.164	71.3μ +/-14.3 (n = 109)	63.5μ +/-18.0 (n = 142)	< 0.001
V. Lat. Acc.	-4.502	81.9μ +/-17.3 (n = 84)	84.9μ +/-17.6 (n = 90)	NS

PC = percentage of change ; R. muscle = right muscle ; L. control = corresponding left control muscle ; NS = not significant.

When the type I fibers from the five atrophic muscles were studied together, their average diameter (56.8 μ ± 10.3) was significantly smaller ($p < 0.001$) than that of the control muscles (67.2 μ ± 13.8). The average diameter of the type I fibers in the three hypertrophic muscles (74.5 μ ± 17.1), although larger, did not differ significantly from the controls (73.8 μ ± 14.4).

DISCUSSION

a. Macroscopic evaluation

The evaluation at three months after isolated section of a collateral ligament did not show any significant changes in the muscles surrounding the operated joint. The capacity of the collateral ligament for spontaneous healing three months after its section has been documented by several authors in animal models and humans (7, 20, 24). It is probable that in our model, healing of the transected ligament made muscle compensation unnecessary.

The quadriceps, glutei and extensor digitorum longus, all extensor muscles, showed significantly greater atrophy in the ACL-deficient limbs (ACL, MCL + ACL and ACL + PCL groups) when compared to the limbs with other ligamentous injuries. One could attribute the wasting of the quadriceps in the ACL-deficient limbs to the greater susceptibility of this muscle to undergo atrophy, or consider it to be a consequence of the surgical approach used (15). However, a look at the evolution of the quadriceps in the limbs with isolated PCL injury rules out these two possibilities. The results show that not every capsuloligamentous injury of the knee is associated with atrophy of the quadriceps ; and since the PCL injury was made using the same surgical approach as the ACL injury, the method cannot be responsible for the atrophy. The muscles showing greater atrophy in the ACL-deficient limbs were antagonists of the ACL (quadriceps), or muscles that participate in the extension of the knee and of the limb (extensor digitorum longus and glutei). The antagonistic action of the quadriceps with respect to the ACL has been demonstrated in digitigrade mammals and in humans (22). Experimental and clinical studies have shown that quadriceps contraction induces the greatest strain in the ACL, and therefore the greatest anterior tibial shearing force, between full extension and about 25° to 40° of knee flexion (1, 4, 11). In the rabbit, the 35° retroversion of the tibial plateau favors the anterior shearing of the tibia and increases the dislocating action of its powerful knee extensor muscle (10). When bearing weight,

the rabbit hindlimb acts like a closed kinetic chain (14) ; thus, knee extension is accompanied by extension in the hip. It has been suggested that a subconscious protective mechanism may produce muscular adaptations, avoiding the contraction of selected muscles and reprogramming the locomotor process, to avoid pathological joint displacement (1, 4, 23). The atrophy of the quadriceps, glutei and extensor digitorum longus (with insertion in the lateral femoral condyle) (3) observed in the ACL-deficient limbs (groups ACL, ACL + MCL and ACL + PCL) could be a consequence of a new locomotor process. To avoid joint giving-way, the activity of muscles that favor or set off joint failure is inhibited.

As in other digitigrade mammals with retroversion of the tibial plateau (10), the biomechanical importance of the PCL in the rabbit may be much less than that of the ACL (18). The retroversion of the tibial plateau and the powerful extensor muscles decrease the posterior shearing of the tibia, and therefore, the strain on the posterior cruciate ligament. PCL injury in the rabbit would not result in great knee instability, and this could explain why the muscles of the PCL-deficient limbs in our model did not develop compensatory modifications.

b. **Microscopic evaluation**

Mammalian skeletal muscles have a remarkable capacity for accommodating changes in demand, and acquire the physiological and biochemical characteristics which appear better suited to the new functional requirements (19). Type I fibers are closely linked to the maintenance of muscle tone (16). Prolonged tonic stimulation increases the muscle type I fiber content (19). The percentage of type I fibers, increased in the hypertrophic muscles we studied and decreased in the atrophic as compared to the controls, could be the result of increased and decreased tonic stimulation, respectively, and could be understood as an adaptation of these muscles to the new biomechanical situation of the limb. Maintained stimulation causes an increase in the diameter of muscle fibers while inactivity reduces it (6). The muscles that suffered significant macroscopic atrophy showed fiber atrophy that affected both types of fiber equally. The hypertrophic muscles showed an increase in the diameter of type II fibers but not a strong enough difference in the type I fibers. Some authors have suggested that type II fibers hypertrophy more easily than type I fibers (8, 21).

The histological data obtained in the present study reflect the macroscopic data. The atrophic muscles (positive PC) showed a lower percentage of type I fibers and a decrease in the diameter of both types of fiber, indicating inactivity and loss of muscle tone, while the hypertrophic muscles (negative PC) showed a higher percentage of type I fibers and an increase in the diameter of the type II fibers, evidencing greater stimulation and maintained muscle tone.

In the rabbit model used in this study, the evolution of the muscles of hindlimbs with chronic ligamentous knee instability was not uniform and varied according to the type of ligamentous injury incurred. The modifications in muscles we observed correspond to qualitative and quantitative changes in both the type I and type II fibers. In our opinion, these changes can be viewed as a biological mechanism to compensate for the instability of the joint and interpreted as a spontaneous adaptation of the dynamic stabilizers to the new joint biomechanics.

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SAMENVATTING

F. PELLISE, A. NAVARRO-QUILIS. Experimentele studie van de spierveranderingen bij de chronische ligamentaire instabiliteit van de knie.

Verschillende auteurs hebben het idee geformuleerd dat insufficiëntie van de voorste kruisband kan leiden tot biologische aantastingen, bestaande uit atrofie van de quadriceps en andere extensoren (antagonisten van de voorste kruisband) en relatief bewaren van de hamstrings en andere flexoren (agonisten van de voorste kruisband).

De spontaan optredende veranderingen in de musculatuur van de achterpoot van 36 konijnen met chirurgisch geïduceerde chronische ligamentaire instabiliteit van de rechterknie werd bestudeerd.

De niet-geopereerde linkerzijde werd als controle gebruikt. 3 maand postoperatoir werden de proefdieren gedood en de dieren gedisseceerd voor macroscopische en microscopische evaluatie.

Bij de voorste kruisbanddeficiënte ledematen waren de extensoren (quadriceps, glutei en extensor digitorum longus) meer geatrofieerd dan de andere spieren ($p < 0,005$). 622 spiervezels van de geatrofieerde spieren, 373 van de hypertrofische spieren en 879 van de controlespieren werden microscopisch nagekeken. De atrofische spieren tonen een significant lager percentage van type I fibers (voor bewaren van de stand) en een vermindering van de diameter van type I en II (verantwoordelijk voor snelle bewegingen). Dit alles in vergelijking met de controlezijde ($p < 0,005$). De hyprtrofische spieren toonden een significant grotere proportie van type I fibers en een toename in diameter van de type II fibers, in vergelijking met de controles

($P < 0,005$). Onze resultaten bevestigen het idee dat er een spontane aanpassing is van de spieren ter compensatie van ligamentaire instabiliteit.

RÉSUMÉ

F. PELLISÉ, A. NAVARRO-QUILIS. Étude expérimentale des modifications de la musculature dans l'instabilité ligamentaire chronique du genou.

Plusieurs auteurs ont avancé l'hypothèse que l'insuffisance du ligament croisé antérieur (LCA) pourrait conduire à une adaptation biologique consistant en une atrophie du quadriceps et des autres extenseurs (antagonistes du LCA) avec une préservation relative des muscles de la patte d'oie et des autres fléchisseurs (agonistes du LCA).
Les auteurs ont étudié les modifications qui se sont produites spontanément au niveau de la musculature du membre postérieur chez 36 lapins, après production d'une instabilité ligamentaire chronique du genou droit par section du LCA. Le membre postérieur gauche non opéré a servi de témoin. Trois mois après l'opération, les lapins ont été sacrifiés et leurs muscles ont fait l'objet d'une étude macroscopique et microscopique. Au niveau des membres présentant une déficience du LCA, les extenseurs (quadriceps, fessiers et long extenseur commun) présentaient une atrophie significativement plus importante que les autres muscles ($p < 0,05$).
Une étude microscopique a été réalisée sur 622 fibres provenant de muscles atrophiés, 373 provenant de muscles hypertrophiés et 879 provenant de muscles témoins. Les muscles atrophiés montraient un pourcentage significativement moins élevé de fibres de type I (responsables du maintien de la posture) et une réduction de diamètre des fibres de type I et II (responsables du mouvement rapide) par rapport aux témoins ($p < 0,05$). Les muscles hypertrophiés montraient une proportion significativement plus élevée de fibres de type I et une augmentation du diamètre des fibres de type II par rapport aux témoins ($p < 0,05$). Ces résultats semblent conforter l'hypothèse selon laquelle il se produit une adaptation spontanée des muscles pour compenser l' instabilité ligamentaire.