Quantitative and Temporal Differential Recovery of Articular and Muscular Limitations of Knee Joint Contractures; Results in a Rat Model.

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Running title: Reversibility of Articular Contracture
Abstract

Joint contractures alter the mechanical properties of articular and muscular structures. Reversibility of a contracture depends on the restoration of the elasticity of both structures. We determined the differential contribution of articular and muscular structures to knee flexion contractures during spontaneous recovery. 250 rats divided in 24 groups had 1 knee joint surgically fixed in flexion for 6 different durations, from 1 to 32 weeks, creating joint contractures of various severities. After the fixation was removed, the animals were left to spontaneously recover for 1 to 48 weeks. After the recovery periods, animals were killed and we measured the knee extension before and after division of the transarticular posterior muscles using a motorized arthrometer. No articular limitation had developed in contracture of recent onset (≤2 weeks of fixation; \( P > .05 \)); muscular limitations were responsible for the majority of the contracture (34±8° and 38±6°, respectively; both \( P < .05 \)). Recovery for 1 and 8 weeks reversed the muscular limitation of contractures of recent onset (1 and 2 weeks of fixation; respectively). Long-lasting contractures (≥4 weeks of fixation) presented articular limitations, irreversible in all 12 durations of recovery compared to controls (all 12 \( P < .05 \)). Knee flexion contractures of recent onset were primarily due to muscular structures, and they were reversible during spontaneous recovery. Long-lasting contractures were primarily due to articular structures and were irreversible. Comprehensive temporal and quantitative data on the differential reversibility of mechanically significant alterations in articular and muscular structures represent novel evidence on which to base clinical practice.

Keywords: contracture; knee; rehabilitation; joint capsule; muscle; immobilization
Normal joints kept immobile lose their passive range of motion, a condition called a joint contracture. Joint contractures are a major health issue as a consequence of bed rest, paralysis, joint arthroplasty, burn, and numerous other acute and chronic health conditions. (6,9,12,21,43)

Thirty-nine (39%) of patients in intensive care for a minimum of 2 weeks developed on average 3.6 joint contractures at the shoulders, hips, knees, and/or ankles.(9) Joint contractures chronically limit a person’s activities of daily living, cause pain, and complicate hygienic care. (10) Specifically, knee flexion contractures increase energy expenditure, cause unequal leg length and limping, may mandate the use of a walking aid, and even confine patients to a wheelchair or to bed. (12,43)

The lack of knee range of motion can be due to alterations in articular and muscular structures (1,2,5,8,15,19,20,22,24-26,29,33,35,36,38,40,42,45-48,50-54,57-62). In a previous investigation, when knee joints with a contracture were left to spontaneously recover for 2 weeks or more, full reversal was never observed (Trudel G. unpublished). A partial and modest gain of 20° of knee extension took place after 2, 4, and 8 weeks of recovery. Longer durations of spontaneous recovery failed to add any knee extension.

That investigation was not designed to distinguish the contribution of articular and of muscular structures limiting knee extension. Depending on the etiology of the joint contracture, various structures may limit joint motion: skin, fascia, muscle, ligaments, capsule, menisci, cartilage, and/or bone (10). Dividing the skin and muscle allow deriving their contribution to the joint contracture. After myotomy, the remaining restriction in knee extension can potentially be attributed to the following articular structures: ligaments, capsule, menisci, cartilage and bone.
In this paper, we report the respective contributions and the potential for reversibility of articular and muscular structures during the recovery period of joint contractures.

The recovery period corresponds to the time patients are treated to recover range of motion (ROM) from joint contractures. Throughout recovery, knowledge of the tissues’ contributions to the loss of knee extension is crucial to guide management. Tailored preventive or therapeutic interventions can target the affected tissues, optimizing clinical outcome and use of resources.

In the current study, our objectives were to: 1) measure the mechanical contribution of articular and muscular structures to the loss of knee extension in recent-onset and long-lasting flexion contractures and 2) measure the reversibility of each structural alteration after 1 to 48 weeks of spontaneous recovery. Our hypotheses were that: 1) spontaneous recovery will not reverse the articular limitation in long-lasting knee joint contractures and 2) spontaneous recovery will reverse muscular limitation in recent-onset knee contractures.

**MATERIALS AND METHODS**

This project was submitted for review and approved by the institutional animal care committee. A previous manuscript on material from this experiment described recovery of the joint contractures with all periarticular joint structures preserved (Trudel G. unpublished).

Knee joint contractures of various severities were induced in a rat model in order to study the effect of spontaneous recovery. 250 male Sprague-Dawley rats had 1 knee surgically fixed for 1
of 6 durations; 1, 2, 4, 8, 16, or 32 weeks. The internal fixation procedure has been described previously and induces knee flexion contractures of various severities (49). Briefly, under general anesthesia, 2 separate 1-cm incisions were made to attach a Delrin plate to the proximal femur and distal tibia with screws to fix the knee at a 45° angle. The plate followed an internal but extraarticular submuscular course, preserving the knee joint integrity. We alternated the side of surgery. Postoperatively, rats were allowed unrestricted movement in their cages. After completion of the assigned fixation period, the plate and any covering fibrous tissue were removed. Animals with surgical failures or requiring euthanasia were replaced.

For each duration of fixation, 4 groups of rats were left to spontaneously recover (Tables 1 and 2). For the groups internally fixed for 1, 2, 4, and 8 weeks, groups were harvested immediately after the plate was removed (baseline) and at 1, 2, and 4 times the internal fixation duration. For the groups internally fixed for 16 and 32 weeks, groups were harvested immediately after plate removal, at a time equal to half the fixation duration and at 1 and 2 times the fixation duration, except for the group fixed for 32 weeks, which was harvested after 48 weeks of recovery. Groups are defined as "week-week" where the first is the duration of fixation and the second is the duration of recovery.

At the predetermined endpoint of spontaneous recovery, the rats were killed by carbon dioxide inhalation and the angle of knee extension was measured. The experimental leg mounted on a previously described operator-independent motorized arthrometer (Trudel G. unpublished). Knee extension was defined as the position between knee flexion fixation (45°) and full knee extension (180°). The femur was clamped into a metal groove with the lateral condyle positioned over the
centre of rotation of the arthrometer. The leg rested on a movable arm that pushed the leg into
extension at 0.69 rad/sec. The speed gradually slowed to reach a torque of 12.5 N-cm and then
stopped, at which point a camera took a picture. This torque allowed reaching full extension
without creating hyperextension injury in control rat knees. The grooved clamp and the movable
arm moving horizontally ensured that exact lateral views were captured.

Then, all posterior knee transarticular muscles were divided. The measure of knee extension was
repeated. The contralateral knee was tested in the same manner and constituted controls.

Knee extension angles were measured on calibrated camera images by the same person blinded
to the experimental condition. A line was drawn from the middle of the femur clamp to the
lateral condyle and constituted the first side of the angle. A second line drawn from the lateral
condyle to the lateral malleolus constituted the second side of the angle.

The initial measure of knee flexion contracture included both arthrogenic and myogenic
contractures. The angle of extension after division of transarticular muscles allowed deriving 2
sets of data: 1) the angle of extension reached after myotomy indicates the articular limitation,
and 2) the gain in knee extension reflects muscular restriction (57,58). We defined articular
limitation to joint contractures as the angle of control minus experimental after transarticular
muscles had been divided [(c+d)−(a+b); Figure 1]; a direct measure of articular structures
limiting knee extension. We defined muscular limitation to joint contractures as the gain of
extension from myotomy in the experimental minus control knees [(b−d); Figure1]. Finally, we
calculated the articular and muscular limitations as a proportion of the total contracture (Figure 1) (35, 51).

Statistical Analysis
All data was analyzed with SPSS version 20 (IBM, Armonk, New York). We assumed a smaller angle of knee extension in the experimental group and used a paired, 1-tailed t-test to compare experimental and control knees after myotomy. The effect of increasing durations of recovery on knee extension after myotomy was compared using a univariate analysis. Finally, we compared the gain in knee extension from myotomy between experimental and control using 2-tailed t-tests. P values of ≤.05 were considered statistically significant.

RESULTS
Thirteen rats required local wound care, of which 12 received antibiotics; all 13 were treated and included. At endpoint, data for 12 animals were not analyzed for persistent fibrous adhesions, leg fracture during testing, extension angle over 195° or images not recorded (Table 1). Both knees of 238 rats were analyzed.

Articular Limitation
Immediately after fixation removal, myotomy restored knee extension to control levels in recent-onset contractures (fixation for 1 and 2 weeks), indicating that no significant articular limitation persisted after the removal of the fixation device (Figure 2, Table 1). At baseline, just after fixation removal, myotomy did not restore knee extension to control levels in long-lasting
contractures; fixation for 4, 8, 16, and 32 weeks progressively reduced the angle of extension, indicating significant articular limitations (all 4 \( P < .001 \); Figure 2, Table 1). Spontaneous recovery for 4 and 8 weeks allowed contractured knees to achieve angles of extension after myotomy comparable to controls in recent-onset contractures (fixation for 1 and 2 weeks, respectively; Figure 2). At the other 16 durations of recovery, a significant articular limitation to knee extension persisted (all 16 \( P < .05 \); Figure 2). Spontaneous recovery permitted partial improvements of the articular limitation in groups 4-8 and 16-8 (Figure 2 and Table 1).

Muscular Limitation

At baseline, division of transarticular muscles caused large gains in knee extension in contractures of 1, 2 and 4 week-onset compared to control knees (46±7°, 51±6°, and 38±4°, vs. 13±4°, 12±4° and 16±3°, \( P = .007, .000 \) and \( .002 \) respectively; Figure 2), indicating a significant muscular limitation contributing to the flexion contracture (Figure 3). At baseline, myotomy in long-lasting contractures (fixation of 8, 16, and 32 weeks) did not lead to significant gain in extension (Figure 3, Table 2). Spontaneous recovery for 1 week after 1-week-onset contracture reversed the muscular limitation (\( P > .05 \); Figure 3). Recovery for 2 and 4 weeks following a 2-week-onset contracture did not reverse the muscular limitation compared to control (\( P = .042 \) and \( .047 \) respectively; Figure 3) but 8 weeks of recovery did. Interestingly, myotomy performed in long-lasting contractures produced less gain in knee extension compared to control knees for groups 8-8, 16-16, 32-16 and 32-48 (all \( P < .05 \); Figure 3).

Proportion of Knee Contracture
The contribution of articular limitation to the original knee joint contracture at baseline increased from recent-onset to long-lasting contractures (from 18% after 1 week internal fixation to 100% after 32 weeks, *P* < .001; Figure 4, Table 1). Spontaneous recovery identified the calculated articular contribution to the joint contracture as predominant (Figure 4, Table 1).

**DISCUSSION**

The articular and muscular limitations to knee range of motion during spontaneous recovery of contractures have not been systematically characterized. We collected quantitative and temporal knee extension data to assess both the articular and muscular limitations. The articular contribution was measured directly by sectioning posterior muscles while the muscular contribution was calculated from data measured on knee joints including both articular and muscular structures. Results evaluated the extent of contracture reversibility—or lack thereof—in the rat immobilization-induced knee flexion contracture model.

Baseline assessment of contracture severity immediately after fixation showed that knees with recent-onset contractures (1 or 2 weeks of fixation) had no significant articular limitation. However, knees with long-lasting contractures developed articular limitation intensifying with the duration of fixation. In contrast, muscular limitation arose rapidly in response to immobilization and limited the knee extension of recent-onset contractures (1 and 2 weeks of fixation).
After characterizing the contractures without recovery, we measured the temporal evolution of the articular and muscular limitations to knee extension of rat knees left to spontaneously recover.

**Articular limitation**

Recovery for extended durations did not reverse the articular limitation in knee extension angle of long-lasting contractures (4 or more weeks of fixation). This incomplete mechanical reversibility of the posterior articular alterations confirmed our first hypothesis.

Of all articular structures, which may be responsible for the irreversible articular limitation?

Bone and cartilage undergo microscopic alterations but have not been reported to limit range of joint motion (26,47). Intra- and extra-articular ligaments from immobilized knees demonstrated weaker biomechanical characteristics in a rabbit model (36,46). Yasuda and Hayashi (61) reviewed 37 studies on joint disuse and concluded that “stress deprivation rapidly reduces the mechanical properties of the tendon and ligament tissues”. Based on these studies, knee ligaments do not constitute candidate structures to explain the intensifying articular limitation with increasing severity of the joint contracture found in our study.

In the rat model used in the current study, the posterior capsule adopts a folded configuration when the knee joint is flexed. Upon extension of the knee the posterior capsule unfolds to a full stretch (Figure 5). Opposing synovial intima folds glide on each other possibly owing to constant movement, contact inhibition and synovial fluid (50). Internal fixation in flexion alters this homeostasis: opposing synovial folds become adherent, which decreases the length of the
posterior capsule synovial intima (50). Long-lasting folding of the posterior capsule show histological evidence of adhesions between folds that become organized and fused (1,2,50). In the rat knee flexion contracture model, posterior synovial intima length had decreased from a normal value of 8.6mm to 1.4mm after 16 weeks of immobilization, a sizeable shortening of 84% (48) (Figure 5). The synovial layer also has fewer proliferating synoviocytes and produces less synovial fluid (25,44,48). In addition, the capsule undergoes subsynovial changes characterized by disordered alignment of collagen fibers, increased type I collagen and advanced glycation end-products (29).

This structural reorganization of the synovial and subsynovial tissues forming the posterior capsule resists elongation after myotomy and can explain the articular limitation to knee extension in long-lasting contractures. Adhered and fused capsular folds reducing capsule length and rearranged subsynovial collagenic matrix cannot spontaneously re-establish a linear, foldable, and extensible posterior knee capsule; attempts at stretching such a capsule may end in a tear (Figure 5). In this study the irreversibility of articular limitation may be attributable to a) the large extent of the articular structural alterations and b) the poor potential of these structures for reversibility.

Muscular limitation

In a purely myogenic contracture, dividing the transarticular muscles restores full range of knee motion in the absence of arthrogenic limitation (35,51,57,58). This was the case in the current study where spontaneous recovery reversed the muscular limitation of recent-onset contractures confirming our second hypothesis. In long-lasting contractures, the potential for reversibility of
posterior knee muscles was blunted by articular limitations. Interestingly, recovery of
countreces caused by 8 or more weeks of internal fixation created situations in which posterior
myotomy resulted in less gain of knee extension than control knees (shown as negative values in
Table 2 and Figure 3). This finding was unexpected and, to our knowledge, is reported here for
the first time. This demonstrated that spontaneous recovery reversed the muscular limitation to
an angle nearer to the fixed articular limitation angle than in controls.

The potential of muscular structures to adaptively shorten or lengthen has been precisely
documented (5,15,20,33,42,53,54,57,59,60). Loss of sarcomeres characterizes muscles
immobilized in a shortened position after 4, 7, and 14 days (20,52,54,59), and was accompanied
by reduced protein synthesis and downregulated pathways of energy metabolism, mitochondrial
function, and cell cycle regulation (8,40). Muscle adaptive shortening happened longitudinally,
as opposed to the folding of the posterior capsule. In addition to the depolymerization of thick
and thin myofilaments, changes in muscle connective tissues contribute to myogenic
contractures. Järvinen (22) showed increased endo- and perimysial connective tissue with
disturbed crimp angle while Okita (38) demonstrated increased circumferential rearrangement of
endomysium fibrils with immobilization. Muscle connective tissue increases were less in a
shortened compared to a lengthened position (24,45) and with slow compared to a fast
lengthening (57) as in the current study. Lastly, atrophy of muscles around the immobilized knee
can participate in the myogenic component of joint contractures. However, atrophy
characterized by decrease in protein synthesis, increase in protein degradation resulting in a
decreased protein content, smaller fibre diameter, force production, fatigue resistance and muscle
volume was shown to decrease the muscle passive extensibility. (4,13,14,23,56) This
mechanical effect of muscle atrophy is opposite to the measures we obtained in this model of knee joint contractures. In the current experiment, the increased muscular limitation may be explained by a rapid loss of sarcomeres from adaptive shortening of knee flexor muscles in the posterior thigh and leg and by muscle connective tissue alterations.

Reversibility of adaptively shortened muscles can proceed with addition of sarcomeres, via the elastic properties of connective tissue through stretching and active muscle contraction (5,54,57). Quantitatively, Grieve (17) measured shortening of the human gastrocnemius muscle of 10% with the knee flexed 150° from full extension. Visser (55) measured shortening of human gastrocnemius and biceps femoris at 7% and 4%, respectively between extension and the knee flexed 100° from full extension (Figure 5). During bone distraction in humans, muscles lengthen 20% to 30% during limb lengthening (28,31). The high plasticity of muscle to elongation is reflected in a case report where the tibia was elongated by 22 cm in 371 days (30). The reversibility of the muscular limitation in recent-onset contractures may be attributable to a) the small extent of the muscular structural alterations and b) their favorable potential for reversibility.

Quantitative and temporal data in the current study correlate with the pathophysiology of articular and muscular structures. Spontaneous recovery can reverse early-onset knee flexion contractures if posterior knee muscles regain up to 10% of their original length longitudinally through adaptive lengthening in combination with posterior capsule unfolding not mechanically limiting knee extension; myotomy leading to complete knee extension show that the capsule was not involved in the contracture. In contrast, spontaneous recovery cannot reverse long-lasting
knee flexion contractures if structural alterations in the posterior capsule mechanically prevent regaining 84% of its original length and prevent exploiting the muscle lengthening potential; myotomy barely improving knee extension show that adhesions between the capsule folds may have blocked contracture reversibility.

Clinical Relevance
Articular limitations herein temporally and quantitatively characterized may explain the high prevalence of irreversible chronic joint contractures identified in the literature (2,62). The current study design corresponds to the clinical practice where most knee joint contractures presenting to clinicians have formed over some duration of immobility and may have undergone some duration of spontaneous recovery. The posterior knee capsule had been identified as the primary cause of extension loss in posttraumatic and postsurgical contractures (7). The lack of reversibility has led many surgeons to perform posterior or posteromedial knee capsulotomy with positive results in contractures secondary to hemophilia, ligament repair, fracture, or infection (7,18,27,32,34,39,41). However many avoid this procedure because of the high risk of neurovascular damage. Chen (7) summarized the procedure: “Posterior capsular release, although technically demanding, is effective for treating flexion contractures secondary to scarring and contracture of the posterior capsule.” Importantly, dividing the posterior capsule without the need to lengthen muscles suggested spontaneous reversibility of the muscular limitation (18,27,32,39,41). Our finding that the articular structures, including the capsule, but not the muscular structures prevent reversibility of extension in long-lasting knee flexion contractures supports the clinical recommendation for posterior capsulotomy.
Limitations

The rat habitual knee flexion position may be more resistant to flexion contracture; we used the contralateral knees also in habitual flexion to isolate the change in knee range of motion and attribute the contracture to the intervention. Mechanical testing carried out before myotomy may have weakened articular structures, underestimating the articular limitation. Oki et al. (37) directly assessed muscular limitation in immobilized rat ankles by dividing articular structures first. They found muscular limitations superior or equal to articular limitations after 1, 2, and 4 weeks of immobilization, which is comparable to our results. We found no data on the direct measurement of muscular limitation after long periods of knee fixation or on their evolution over time after spontaneous recovery.

Our findings may not be generalized to all diarthrodial joints. Immobilized rat shoulders demonstrated similar structural changes of capsular folding, adhesion, decreased synovial length and lack of improvement with myotomy (25). Clinically, shoulder capsulotomy increased ROM without intervention on shoulder muscles (3,11,16).

Future research can look at parsing the role of 1) loss of sarcomere in length, loss of myofibrils in parallel (atrophy) and connective tissue accumulation in the myogenic contracture; and 2) capsular folding, intimal length, synoviocytes, collagen fibres in the arthrogenic contracture.

CONCLUSION

Knee flexion contractures of recent onset were primarily due to muscular structures, and they
were reversible during spontaneous recovery. Long-lasting contractures were primarily due to
articular structures and were irreversible. Posterior capsule structural alterations are the candidate
culprit given their unlikely return to anatomical integrity and function. Comprehensive temporal
and quantitative data on the differential reversibility of mechanically significant alterations in
articular and muscular structures represent novel evidence on which to base clinical practice.

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**Figure legends**

*Figure 1.* Illustration of the measures and calculations used to generate data on articular and muscular limitations to knee extension in the tables and figures of this manuscript. Angle of knee extension reached at torque = 12.5 N-cm.

*Figure 2.* Angles of knee extension of rat knee joint fixed in flexion and spontaneously recovering for various durations and of controls with articular and muscular structures present and with muscular structures divided. All data were rounded half to even, which explains differences of ±1° between figures, text and tables. *P < .05* knee extension after myotomy compared to control, †*P < .05* knee extension after myotomy compared to previous time point.

*Figure 3.* In recent-onset contractures, myotomy produced larger gains of knee extension in experimental compared to controls (muscular limitation). Recovery of long-lasting contractures produced the reverse effect, lesser gains of knee extension in experimental compared to control joints, possibly explained by the muscle lengthening from a shortened position to an angle nearer to the articular limitation angle compared to controls. *P < .05* gain in knee extension statistically smaller than control for groups 8-8 [p=.047], 16-16 [p=.043], 32-16 [p=.028], and 32-48 [p=.031].

*Figure 4.* Proportion of the total contracture differentially contributed by articular and muscular limitations after various durations of internal fixation and spontaneous recovery.
Figure 5. Illustration of the differential effect of recovery on articular structures (e.g., posterior capsule) and muscular structures (i.e., posterior knee muscles) in a flexed human knee joint. A. With knee extended, posterior muscles and articular structures are stretched to their full lengths. B. With knee flexed, posterior muscles are loosened and shortened longitudinally by approximately 4-10%. In the rat, the posterior knee capsule is loosened by 84% by approximating the posterior femoral and tibial ends. C. Contractures of recent onset allow posterior capsule to reestablish as linear, foldable, and extensible; long-lasting contractures cause fusion of the folds and collagenic structural rearrangement, mechanically preventing distraction between posterior tibia and posterior femur. Arrows indicate synovial intima lengths in knee flexion. Modified with permission from Anatomy images, copyright Primal Pictures Ltd, https://www.primalpictures.com.
TABLE 1
Articular contribution to knee flexion contracture

<table>
<thead>
<tr>
<th>Immobilization (weeks)</th>
<th>No Recovery</th>
<th>Recovery Duration 1</th>
<th>Recovery Duration 2</th>
<th>Recovery Duration 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>(%)</td>
<td>Proportion of the Contracture (%)</td>
<td>n</td>
<td>(%)</td>
</tr>
<tr>
<td>1</td>
<td>6</td>
<td>7/40</td>
<td>18</td>
<td>7</td>
</tr>
<tr>
<td>2</td>
<td>9</td>
<td>6/44</td>
<td>14</td>
<td>10</td>
</tr>
<tr>
<td>4</td>
<td>9</td>
<td>28/50</td>
<td>56</td>
<td>11</td>
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<tr>
<td>8</td>
<td>11</td>
<td>53/59</td>
<td>90</td>
<td>10</td>
</tr>
<tr>
<td>16</td>
<td>11</td>
<td>68/70</td>
<td>97</td>
<td>10</td>
</tr>
<tr>
<td>32</td>
<td>10</td>
<td>71/70</td>
<td>100</td>
<td>11</td>
</tr>
</tbody>
</table>

Articular limitation larger than the total contracture indicated that myotomy produced less gain of extension in experimental compared to control (Figures 1 and 2). Data above 100% were expressed as 100%.
TABLE 2
Muscular contribution to knee flexion contracture

<table>
<thead>
<tr>
<th>Immobilization (weeks)</th>
<th>No Recovery</th>
<th>Recovery Duration 1</th>
<th>Recovery Duration 2</th>
<th>Recovery Duration 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>(°)</td>
<td>Proportion of the contracture (%)</td>
<td>n</td>
</tr>
<tr>
<td>1</td>
<td>6</td>
<td>34/40</td>
<td>85</td>
<td>7</td>
</tr>
<tr>
<td>2</td>
<td>9</td>
<td>38/44</td>
<td>86</td>
<td>10</td>
</tr>
<tr>
<td>4</td>
<td>9</td>
<td>22/50</td>
<td>44</td>
<td>11</td>
</tr>
<tr>
<td>8</td>
<td>11</td>
<td>6/59</td>
<td>10</td>
<td>10</td>
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<td>16</td>
<td>11</td>
<td>2/70</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>32</td>
<td>10</td>
<td>−1/70</td>
<td>0</td>
<td>11</td>
</tr>
</tbody>
</table>

Muscular limitation below 0° indicated that myotomy produced less gain of extension in experimental than in control knees (Figures 1-3). Data below 0% were expressed as 0%.
Angle of knee extension (°)

Articular limitation
Gain from myotomy

Experimental before myotomy: a
Control before myotomy: c
Experimental after myotomy: a+b; Fig. 2
Control after myotomy: c+d; Fig. 2
Articular limitation to knee extension: (c+d)-(a+b); Fig. 2
Muscular limitation, gain in knee extension from myotomy compared to control: b-d; Fig. 3
Articular limitation, % of the contracture: (c+d)-(a+b)/(c-a)*100; Table 1
Muscular limitation, % of the contracture: (b-d)/(c-a)*100; Table 2