

Growth influences knee laxity after anterior tibial spine fracture

A study on rabbits

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ABSTRACT – An anterior tibial spine (ATS) fracture, with or without displacement, was created in the right knee of 8-week and 22–23-week-old rabbits. After 4 weeks of cast immobilization and 8 weeks of free cage activities, the animals were killed.

A method to measure anterior knee laxity in rabbits was developed using a material testing machine. The laxity of the right knee was compared to the unoperated left knee in each rabbit. The difference was found to have increased in the adult rabbits after the healing of a displaced ATS fracture (median 0.5 mm, range 0.3–0.9), but not in the young rabbits (median 0.2 mm, range 0.0–0.4). The ultimate load of the anterior cruciate ligament (ACL) was reduced after healing of the ATS fracture.

The results support the theory that further growth may compensate for the functional elongation of the ACL caused by healing of a displaced ATS fracture. The study also indicates that an ATS fracture may affect the mechanical properties of the ACL.

Several clinical studies have found residual knee laxity in a substantial number of cases after fracture of the anterior tibial spine (ATS) in children, with or without subjective impairment of knee function (Clanton et al. 1979, Grönkvist et al. 1984, Bachelin and Bugmann 1988, Baxter and Wiley 1988, Willis et al. 1993, McLennan 1995). The reasons for the variations in outcome are not completely understood. It has been suggested that in young children the laxity of the anterior cruciate ligament (ACL), due to healing of an elevated

ATS fragment and/or elongation of the ligament at the time of injury, may disappear with further growth (Grönkvist et al. 1984, DeLee 1994).

We addressed this question in a study on rabbits. We developed a method to measure anterior knee laxity, and the influence of growth on laxity after healing of a displaced ATS fracture.

Animals and methods

Surgical procedure

An ATS fracture was created in the right knee of New Zealand White rabbits. The left knee remained intact and served as a control. The following surgical procedure was used.

After premedication with atropine sulfate, the animals were anesthetized with intravenous sodium pentobarbital and diazepam. Prilocaine, 5 mg/mL, was injected in the operation area. The joint capsule was opened with a medial parapatellar incision and the patella was retracted laterally. A 1.0 mm hole was drilled through the cortical bone in the center of the tibial insertion of the ACL. Sagittal cuts in the cortical bone were made with a chisel on both sides of the tibial insertion of the ACL. The ATS area was raised with a horizontal epiphyseal osteotomy beginning in the proximal part of the tuberosity. The aim was to resemble an ATS fracture Meyers-McKeever (1959) type III with a completely avulsed fragment, and to avoid injuries to the ACL itself. The anterior attachments of the menisci on the fragment were left untouched.



Figure 1. The right knee from one of the animals in group IV, 12 weeks after operation.

In 2 of the 4 experimental groups, the fragment was reduced to its original position. An 0.8 mm AO/ASIF-wire with the upper end bent into a loop, was inserted through the drill hole. Non-resorbable 5–0 monofilament osteosutures through the loop of the wire, the fragment and the underlying bone completed the fixation of the fragment. In the 2 remaining experimental groups, the ATS fragment was secured with the same procedure, but in an elevated position with the aid of a 1 mm thick bone spacer taken from the anterior edge of the fragment (Figures 1 and 2). The joint capsule was closed with absorbable 4–0 sutures, and the skin with non-resorbable 5–0 sutures.

The animals received daily intramuscular injections of sulfadoxine 40 mg/kg bodyweight and trimethoprim 8 mg/kg bodyweight for 3 days post-operatively. The limb was immobilized in a cast for 4 weeks at 90 degrees of flexion in the knee and 90 degrees of flexion in the ankle. The cast was changed two or three times to avoid growth disturbances in the young rabbits. In the adult rabbits, the cast was changed when it wore out or was too large due to atrophy of the thigh muscles.

The immobilization was followed by free cage activities for 8 weeks. The rabbits were then killed, i.e., after bone growth stopped (Khermush et al. 1972). The study was approved by the local ethics committee for animal experiments.

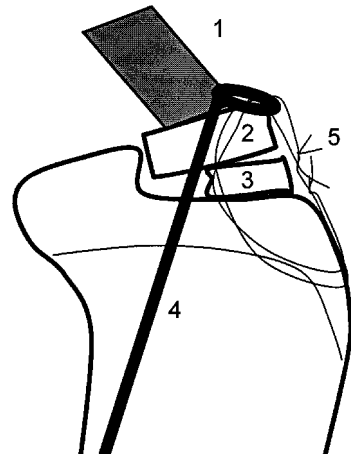


Figure 2. Osteosynthesis of a raised ATS fragment with a bone spacer. 1 ACL, 2 ATS fragment, 3 bone spacer, 4 AO wire, and 5 osteosutures.

Experimental groups

The study consisted of 4 experimental groups including 21 rabbits. Operation and treatment are described in "Surgical procedure":

Group I (n 6) consisted of 8-week-old rabbits. The ATS fragment was reduced.

Group II (n 5) consisted of 8-week-old rabbits. The ATS fragment was secured in a raised position (1 mm displacement).

Group III (n 5) consisted of 22–23-week-old rabbits, i.e., rabbits that have passed the period of femoral growth (Khermush et al. 1972). The ATS fragment was reduced.

Group IV (n 5) consisted of 22–23-week-old rabbits. The ATS fragment was secured in a raised position (1 mm displacement).

Complications

At first, 40 rabbits were included, but 19 were excluded due to postoperative complications or technical failures, leaving 21 in the study (Table 1).

Radiographic examination

Both hind limbs in each rabbit were saved. All soft tissues were removed, except the knee joint capsule. The joint was wrapped in saline-moistened gauze bandages. Radiographs were taken in the lateral and frontal projections. Specimens with growth disturbances or other skeletal abnormali-

Table 1. Distribution of complications in the 19 excluded rabbits

Complication	No.
Cast complications	8
Postoperative dislocation of the patella (effusion and arthritis in 2/2 cases)	2
Growth disturbance due to physeal injury at operation	2
No fracture healing, loose fragment (effusion and arthritis)	1
ACL partly detached from the fragment (effusion and arthritis in 4/5 cases)	5
Technical error at laxity measurement	1

ties were excluded.

Gross examination

The knee joint capsule and both menisci were removed, but the posterior cruciate ligament was kept intact. Increased amounts of synovial fluid, changes in the articular cartilage, or other pathological findings were recorded.

Measurement of laxity and ultimate load

The measurements were made in a material testing machine connected to a computer for registration of load and deformation (precision ± 0.004 N, ± 0.01 mm). The tests were performed in the Department of Solid Mechanics at the Royal Institute of Technology, Stockholm. A force was applied in the direction of the long axis of the femur and the ACL. To obtain this force in the sagittal plane, the knee joint was placed at 45 degrees of flexion (Woo et al. 1987).

The end-points for the laxity measurements were defined. The tibial and femoral joint surfaces were compressed at a deformation rate of 1 mm/minute, until a load of 3 N was reached. The load was kept constant for 2 minutes, after which no further creep was detected. The position of the tibia in relation to the femur was regarded as the posterior end-point. The femur-ACL-tibia complex was then distracted, again at a deformation rate of 1 mm/minute, until a tension load of 3 N was reached. The load was kept constant for a further 2 minutes, after which no further creep was found. The position of the tibia in relation to the femur was regarded as the anterior end-point. The test sequence was based on pilot studies. After 2 cycles, the curves were superimposed (Viidik 1966).

Load N

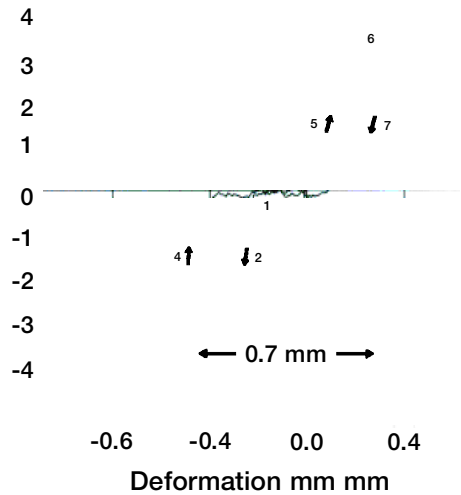


Figure 3. Load-deformation curve. Measurement of laxity (0.7 mm). Position 1 represents starting-point, followed by compression of 3 N (2), creeping for 2 minutes (3), unloading (4), traction of 3 N (5), creeping for 2 minutes (6), and finally, unloading (7).

The laxity was measured as the distance in mm between the two end-points on the second load cycle (Figure 3).

Pilot studies showed that a standardized positioning of the tibia in relation to the femur with respect to angulation, translation and rotation, is paramount for reliable laxity measurements. To ensure a standardized positioning and mounting of the specimen in the testing machine, we developed the following procedure. The soft tissues around the knee joint together with the menisci were removed to permit inspection of the joint surfaces. The position was set in a jig with the tibial and femoral articular surfaces in contact in both the lateral and medial compartments. A contoured 1.6 mm AO/ASIF wire across the joint was glued to the anterior surfaces of the femoral and tibial shafts to secure the position (Figure 4).

These shafts were inserted into brass cylinders already mounted on the testing device (Figure 5). The cylinders were filled with thermogluue. Cryo spray was used on the outside of the cylinders to protect the specimen from the heat of the thermogluue. The fixation was complete after 10–15 minutes. The PCL and the AO/ASIF wire were cut, and the previously described laxity tests were done.

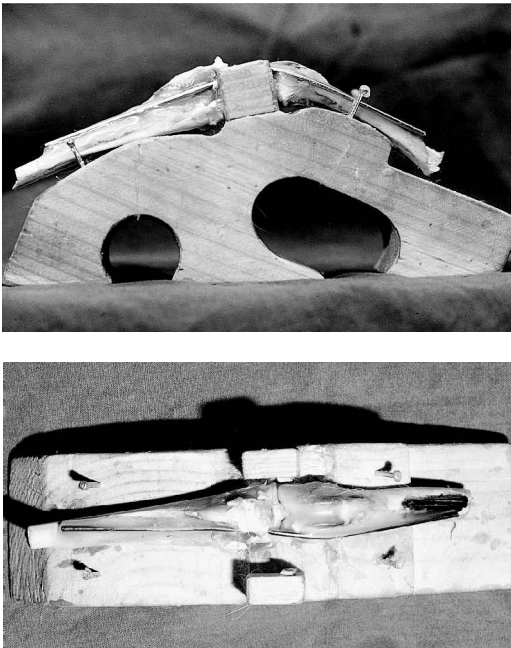


Figure 4. Specimen in the jig, prepared for the mechanical tests.

Changes in water content alter the mechanical properties of ligaments. To minimize such changes, the ACL was continuously moistened with the rabbit's plasma (Tkaczuk 1968). During transportation from preparation to test site, the specimen was wrapped in saline-moistened gauze bandages (Viidik 1968).

Approximately 1 hour passed between the testing of the right and left knees of each rabbit. In a

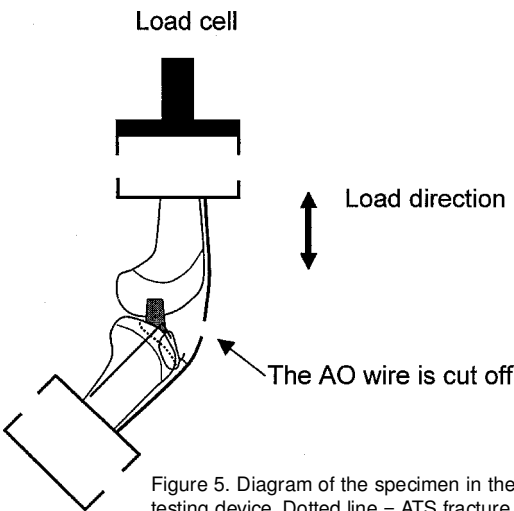


Figure 5. Diagram of the specimen in the testing device. Dotted line = ATS fracture.

pilot study, 2 knees were tested, as described above, and then retested after 1 hour. No differences were seen in the load-deformation curves (Viidik 1966, 1968). After completing the laxity measurements, each specimen was loaded in traction at the same deformation rate of 1 mm/minute until failure. Ultimate load and failure mode (rupture in the substance of the ACL, rupture of the insertions of the ACL or avulsion fracture) were registered.

We did 2 pilot studies to evaluate the described method for laxity assessment.

1. The laxity in both knees was measured in 5 consecutive, unoperated 22-week-old rabbits. The largest side-to-side difference in knee laxity was 0.1 mm (Table 2).

2. The right hind limb was saved from 5 22-week-old rabbits to judge the effect on knee laxity of a raised ATS fragment.

All soft tissue and the menisci were removed, except the PCL. A 1.1 mm hole was drilled through the center of the tibial insertion of the ACL and into the tibial metaphysis. The ATS fracture was created as previously described, and a 1.5 mm AO/ASIF screw was driven into the drill channel. The fragment was fixed in a raised position (1 mm displacement).

The laxity test was performed. The fragment was then reduced by tightening the screw, and the test was repeated. In 2 cases, the laxity was also measured half-way through reduction, i.e., at 0.5 mm of displacement. The laxity was proportional to the displacement of the ATS fragment (Table 3).

Statistics

For statistical analysis, we used the Mann-Whit-

Table 2. Results of laxity tests in mm of the right knee, left knee, and the difference between the knees in absolute values in 5 consecutive, unoperated 22-week-old rabbits

Rabbit	Right knee	Left knee	Difference, absolute value
A	0.2	0.2	0.0
B	0.5	0.5	0.0
C	0.7	0.7	0.0
D	0.3	0.4	0.1
E	0.4	0.5	0.1

Table 3. Results of laxity tests in mm of the right knee in 5 22-week-old rabbits with displacement of the ATS fragment of 1, 1/2, and 0 mm

Rabbit	Fracture displacement, mm		
	1	1/2	0
A	1.5	1.0	0.6
B	1.0	0.6	0.5
C	1.2	Not performed	0.6
D	1.0	Not performed	0.6
E	1.0	Not performed	0.5

ney U-test and two-way analysis of variance (ANOVA). $P < 0.05$ was considered statistically significant.

Results

Two-way analysis of variance (ANOVA) of the whole material showed that age affected laxity ($p = 0.04$) (Figure 6).

There was no statistically significant difference in knee laxity between group I (8-week-old rabbits with a reduced fragment) and group II (8-week-old rabbits with a raised fragment) ($p = 0.2$). There was a difference between group II (8-week-old rabbits with a raised fragment) and group IV (22–23 weeks old rabbits with an elevated fragment) ($p = 0.02$), and between group III (22–23-week-old rabbits with a reduced fragment) and group IV (22–23-week-old rabbits with a raised fragment) ($p = 0.01$) (Table 4).

The ultimate load to failure was reduced in the operated knee in all groups. Failure mode was rupture in the substance of the ACL in 12 of 20 cases. 4 specimens had a rupture in the tibial insertion of the ACL, and 3 had an ATS fracture. Femoral physeolysis was seen in 1 case (Table 5).

Rupture in the substance of the ACL in the unoperated left knee was seen only once. There was no rupture in the tibial insertion of the ACL. 2 specimens had an ATS fracture in the left knee. Femoral physeolysis, tibial shaft fracture, or failure in the femoral attachment to the testing machine occurred in the remaining cases (Table 5).

Moderate effusion and synovial irritation were often present in the operated right knee, but there

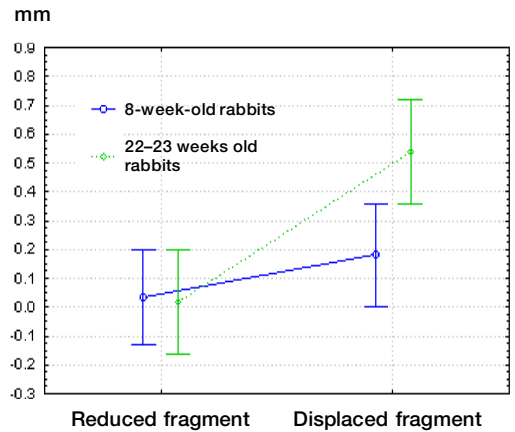


Figure 6. Two-way ANOVA of the whole material (n = 21). mm = Difference in laxity between right and left knees. Mean and 95% confidence interval.

Table 4. Median value and range of difference in laxity (mm) between right and left knees in the 4 experiment groups

Experiment groups	Median laxity	Range
8 wks old, fragment reduced (I)	0.0	-0.3–0.3
8 wks old, fragment displaced (II)	0.2	0.0–0.4
22–23 wks old, fragment reduced (III)	0.0	-0.1–0.3
22–23 wks old, fragment displaced (IV)	0.5	0.3–0.9

was no evident degenerative arthritis in the femorotibial compartments.

Discussion

In childhood, avulsion fractures are commoner than ligament or tendon ruptures (Ogden 1990). The anterior tibial spine fracture, a typical example of this, is the childhood equivalent of rupture in the substance of the ACL in adults.

Clinical studies of ATS fractures in skeletally immature patients have disclosed many cases with residual anterior knee laxity (Clanton et al. 1979, Grönkvist et al. 1984, Bachelin and Bugmann 1988, Baxter and Wiley 1988, Willis et al. 1993, McLennan 1995). The question arises whether growth of the child will reduce the laxity (Grönkvist et al. 1984, DeLee 1994). A possible mechanism for such a reduction could be that the

Table 5. Ultimate load and failure mode in groups I–IV, 21 rabbits

Group	Ultimate load		Failure mode ^a	
	Op	Control	Op	Control
I	TF ^b	147	TF ^b	FP
I	146	156	FP	FP
I	111	136	ST	FP
I	139	245	TI	FP
I	118	276	TI	FP
I	106	172	ST	FP
II	144	164	ST	FP
II	95	177	ST	FP
II	122	178	ST	FP
II	92	160	TI	FP
II	106	201	ST	FP
I+II median	115	172		
I+II range	(92–146)	(136–276)		
III	151	354	ST	ST
III	173	356	ST	AF
III	235	447	ST	AF
III	268	248	AF	TF ^c
III	287	392	ST	TF ^c
IV	241	300	AF	TF ^c
IV	157	282	TI	TF ^d
IV	216	380	AF	TF ^c
IV	88	388	ST	TF ^d
IV	88	198	ST	TF ^c
III+IV median	195	355		
III+IV range	(88–287)	(198–447)		

Op – right knee, operated on with an ATS fracture.

Control – left knee, unoperated.

^a FP – traumatic femoral physeolysis, Salter-Harris type 1, in the distal femoral physis.

^a ST – rupture in the substance of the ACL

^a TI – rupture in the tibial insertion of the ACL

^a AF – ATS fracture

^b TF – technical failure during testing of ultimate load.

^c TF – failure of the femoral attachment to the material testing machine during testing of ultimate load.

^d TF – tibial shaft fracture

ACL proper grows only in response to tensile forces, which will be reduced if the ligament is slack. When growth enlarges the skeletal parts of the knee joint, the tensile forces will be gradually restored and growth of the ACL will resume. If such a mechanism exists, the laxity in a knee with a raised ATS fragment will diminish in a growing individual, but not in an adult.

The aim of our study was to ascertain whether skeletal growth could compensate for the functional elongation of the ACL caused by a raised ATS fracture. To our knowledge, the question has

not been addressed in other experimental studies, although knee laxity measurements in rabbits have evaluated ACL reconstruction with autografts (Ballock et al. 1989, Muneta et al. 1993) and Dacron grafts (Engle et al. 1994). In these studies, the anteroposterior knee laxity was measured with forces of ± 5 –10 N applied perpendicular to the long axis of the tibia. Knee flexion angles varied between 30, 45 and 90 degrees. The menisci, PCL and periarticular connective tissue were kept intact.

The application of forces perpendicular to the tibia is convenient since it corresponds to the clinical examination of patients, but the differences in laxity between various groups, and between animals in the same group, were considerably larger in these studies, compared to the precision required in our study. The amount of displacement compatible with healing of an ATS fracture is limited. A displacement of 1 mm may seem insignificant, but it is considerable in an 8-week-old rabbit. Consequently, the method we used to measure laxity needed a precision better than 1 mm.

We could not measure changes in laxity caused by an ATS fracture displacement of 1 mm, using forces perpendicular to the tibia and with the PCL as the posterior end-point. We had to control simultaneously the compression forces between the joint surfaces. The pilot studies showed that, to attain the desired level of precision, laxity had to be measured along the long axis of the ACL, using the compression between the joint surfaces as the posterior end-point.

The pilot studies further disclosed that a precise positioning of the specimen in the testing machine was essential in the laxity measurements. The use of the jig, the initial preservation of the PCL, the temporary AO/ASIF wire across the joint, together with the removal of the menisci and the periarticular connective tissue, were necessary to achieve a controlled mounting in the testing machine.

Our findings support the theory that further skeletal growth reduces knee laxity after a functional elongation of the femur-ACL-tibia complex, caused by a raised ATS fragment. In a previous long-term follow-up of 61 children with ATS fractures including 20 cases of Meyers-McKeever type III, we found no correlation between age at

injury (i.e., remaining growth) and residual knee laxity (Janarv et al. 1995). Several explanations may account for the absence of this correlation. Some fibers of the ACL may be in contact with an intact part of the tibia. When performing arthroscopy in children with type III fractures, we have seen cases where the fragment does not include all posterior ACL fibers. In clinical work, there may be simultaneous elongation or partial ruptures of the ACL (Noyes et al. 1974), which we avoided in our experimental model. Arthroscopy and MRI were usually not done in the clinical study. Hence, we have no data about extension of injuries to the ACL and the tibial attachment.

A possible explanation of increased laxity after an ATS fracture could be poor fracture healing. To exclude this, the ultimate load tests were included. The use of a low deformation rate and traction in the long axis of the ACL were of help, as they have been reported to yield an increased number of avulsion fractures (Noyes et al. 1974, Woo et al. 1987). In all but 3 instances, no refracture occurred. When it occurred, the ultimate load was considerably larger (≥ 216 N) than the 3 N used for laxity measurements. We conclude that poor fracture healing was not responsible for the increased laxity found in adult animals after healing of the ATS fracture.

In contrast, many ruptures were seen in the substance of the ACL, with an obviously reduced ultimate load to failure, in the operated versus the control knees. This pattern was also seen in the groups with a reduced fragment. Our findings indicate that an ATS fracture may cause changes in the mechanical properties of the ACL. Several explanations may also account for these changes. Rigid immobilization with various types of external fixation or extensive casts for 8–12 weeks is known to affect the ACL. Changes at the cellular level and reduced cross-sectional area of the ACL in rabbits (Newton et al. 1995), as well as reduced ultimate load and stiffness in dogs (Klein et al. 1982), rats (Larsen et al. 1987), and monkeys (Noyes 1977) have been reported. Recovery of the ACL after immobilization was described in 2 of the studies (Noyes 1977, Larsen et al. 1987).

In our model, we used non-rigid immobilization with a cast not including the hip and a relatively short period of immobilization followed by a

longer period of free cage activities. Kanda et al. (1998) studied the effect of a similar immobilization for 2–8 weeks on the ACL in rabbits. The permeability of the synovial fluid and the histology (light and transmission electron microscopy) were unchanged in the ACL after 4 weeks of immobilization. Signs of degeneration were detected after 6 and 8 weeks, but were restored after 4 weeks of remobilization.

Damage to the blood supply of the ACL caused by the operation is another possible explanation. Robinson et al. (1992) devascularized the ACL in NZW rabbits by synovial stripping without damaging the ligament fibers. The operation caused a gradual worsening of the mechanical properties of the ligament as shown by reduced ultimate tensile strength. The frequently observed inflammatory reaction in the operated knees may have contributed to the reduced ultimate load to failure and the increased number of ruptures in the substance of the ACL. Similar changes in the properties of the femur-ACL-tibia complex were reported by Goldberg et al. (1982) in rabbits after induction of an immune synovitis in the knee joint.

Our findings indicate that residual anterior knee laxity after healing of an ATS fracture, in a preadolescent child, may diminish during growth. However, at present we do not think that this capacity for laxity reduction should lead to a more conservative attitude in treatment of acute ATS fractures in children.

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