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Anterior cruciate ligament reconstruction using iliotibial tract: Histological and mechanical studies in rabbits

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Abstract: A histological and biomechanical study of anterior cruciate ligament (ACL) reconstruction using the iliotibial tract (ITT) was carried out in rabbits. Sixty-five young adult male rabbits were used for the experiments. In the histological study, 3 weeks after the substitution, specimens, showed acellular changes, and cellular repopulation was noted from the peripheral area at 6 weeks. At 24 weeks, the original spindle-shaped fibroblasts of the substitute had changed to round cells. The ligament bone junction was formed by 48 weeks and its structure was similar to that of a normal junction. Transmission electron microscopic examination showed collagen fibril distribution in each test group. The 12-week specimens had fibrils of small diameter only, but some 24-week specimens showed a biphasic appearance, like the ACL, which has both large and small fibrils. On mechanical testing, the maximal load was decreased until 18 weeks, and then recovered to 50% of that of the normal ACL at 48 weeks, this value being approximately the same as that of the ITT used in the reconstruction.

Key words: anterior cruciate ligament reconstruction, experimental study, iliotibial tract, histological maturation, biomechanical property

Introduction

Knee instability due to anterior cruciate ligament (ACL) insufficiency causes disability in both sporting and daily activities, because of the poor healing capacity of the ligament.[1,19,27] Anterior cruciate reconstructions have been performed to stabilize the symptomatic knees since 1917, when Hey Groves[10] used a free graft of proximal fascia lata to reconstruct the ACL. Since that time, various autogenous tissues, such as the patellar tendon (PT),[5] the iliotibial tract (ITT),[14,19,22,27] and the hamstring tendon have been used as substitutes for the ACL.[21] Recently, other types of substitutes, such as allografts,[7,25] xenografts, and artificial ligaments,[15,24] have been employed. However, the ITT autograft is still one of the most widely used tissues for ACL reconstruction. Since 1981, ACL reconstruction has been performed using pedicle strips of the ITT, fixed to the tibia and femur by the bone-tunnel fixation technique.[18,19] Clinical results have been satisfying compared to those in which other substitutes were used. Basic research into ACL reconstruction using ITT autografts has not been as extensive as research into the PT autograft. The maturation process of rolled ITT and ITT in the bony tunnels is still unclear. The purpose of our experimental study was to examine the biological maturation process of the ITT autograft in ACL reconstruction. We examined the histological maturation and biomechanical properties of the autograft in rabbits, particularly noting the behavior of substitutes at the interface of the bony insertion.

Materials and methods

Sixty-five young adult male Japanese white rabbits (6–8 months old and weighing 2.8–3.2 kg) were used for the experiments. ACL reconstruction, using the ITT (defined as the tissue extending from the distal aspect of the quadriceps muscle, covering the patella and inserting on the tibial tubercle) was performed on the right knee; the left knee served as a control. For the histological study, test groups, of 8 rabbits each, were sacrificed at 3, 6, 12, 24, and 48 weeks after the operation (total no., 40 rabbits). For the mechanical test, test groups, of 5

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rabbits each, were sacrificed at 6, 12, 18, 24, and 48 weeks (total no., 25 rabbits).

Surgical procedure

The technique we used was a modification of the over-the-top procedure.[14,16,18] The rabbits were anesthetized with an intravenous injection of pentobarbital (1.0 mg/kg). The right knee was sterilized with povidone-iodine solution and placed on the left side. A lateral parapatellar incision was made, extending from the proximal third of the thigh to the medial crest of the tibial tuberosity. After the arthrostomy was performed, the ACL was excised from its tibial and femoral insertions. Two drill holes (3.0 mm in diameter) were made. The femoral hole was drilled from just above the lateral epicondyle to inside the top of the lateral femoral condyle. The tibial drill hole was made from the lateral side of the tibial tuberosity to the center of the ACL insertion of the tibia (Fig. 1).

The ITT was stripped from its proximal side, 60 mm in length from the tibial tuberosity and 15 mm in width at the middle part, which corresponded to the intra-articular portion. It was sutured into a 2.5–2.8 mm strand, using absorbable thread and it was then passed through the femoral drill hole, the knee joint, and the tibial drill holes. After we confirmed the isometricity of the ligament through knee extension of 30 degrees and flexion of 120 degrees, we fixed the ligament to the tibia with the knee flexed at 60 degrees. The ends of the transplants were sutured to the periosteum of the medial crest of the tibia with nonabsorbable thread. The proximal side was also sutured to just above the bone tunnel at the lateral femoral condyle. After reconstruction, the rabbits were kept in cages without braces.

Histological study

Animals were sacrificed by an intravenous injection of pentobarbital sodium at 3 weeks (n=8), 6 weeks (n=8), 12 weeks (n=8), 24 weeks (n=8), and 48 weeks (n=8) for histological studies. At sacrifice, nonligamentous and nontendinous materials were removed by careful scraping and sharp dissection.

Light microscopic study. Specimens were fixed in buffered formalin for several days, after which they were decalcified in 3% hydrochloric acid and embedded in paraffin. Longitudinal slices, approximately 5- to 10-μm-thick, in the sagittal plane sections were stained by standard hematoxylin and eosin procedure for examination by microscopy. Photomicrographs of representative sections were taken at identical magnification (×40–×100) for further assessment of cellular details and matrix composition.

Electron microscopic study. The middle substance of the ligaments from three specimens in each group was prepared for transmission electron microscopic examination and immunofluorescent staining. Specimens were prefixed with 2% glutar–aldehyde for 24 h, fixed with osmium tetraoxide, and embedded in Epon. We inspected lead citrate and uranyl acetate-stained thin cross sections in an electron microscope with a 25-μ objective aperture at 80 KV.

The fibril diameters were measured and counted on five randomly sampled electron microphotographs (magnification ×15000) for each group[9]. A 15-cm square in each microphotograph was examined and a minimum of 1500 fibrils was measured for each group. The data were compared on a histogram showing the frequency of fibrils in nine diameter groups.

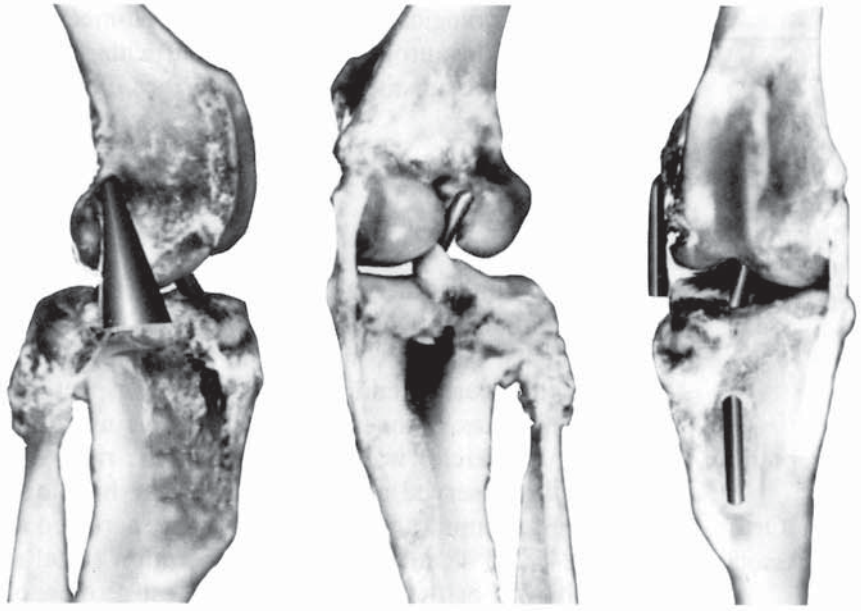

Fig. 1. Surgical procedures. The anterior cruciate ligament (*ACL*) is reconstructed with the iliotibial tract (*ITT*). The rolled ITT was passed through the femoral drill hole, the knee joint, and the tibial drill hole (substitute and reconstruction route are shown)

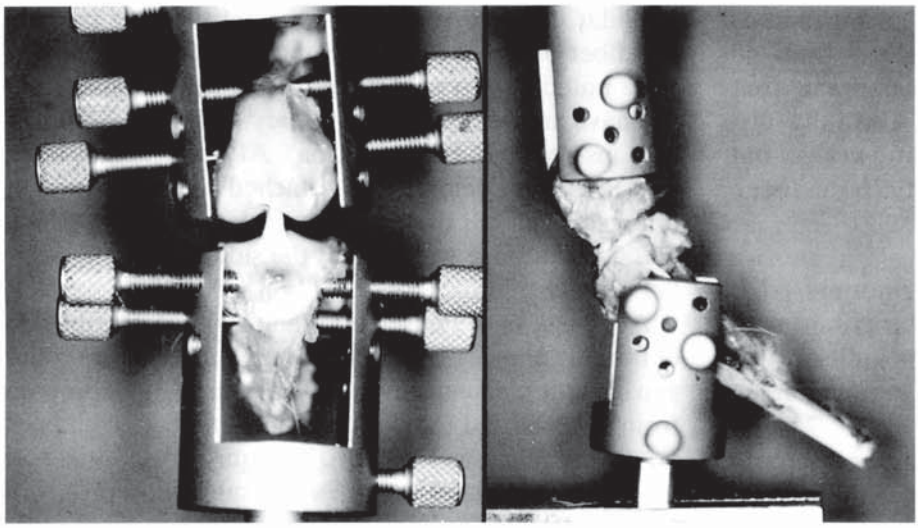

Fig. 2 Mechanical testing device. A femur-ACL-tibia complex (*FATC*) was mounted in a 45-degree knee flexion position

Immunohistochemical study. Serial sections of frozen specimens (5- to 10-μm thick) were cut on a cryostat and washed three times with phosphate-buffered saline (PBS). They were first incubated in a box at 22°C for 1 h with antibodies against type I and type III collagen (Southern Biotechnology Associates, Inc., AL, USA). Next, they were incubated with fluorescein isothiocyanate (FITC)-conjugated anti-goat IgG (Chemicon International, Inc., CA, USA) for 30 min. Both incubations were terminated by rinsing the sections three times with PBS. The specimens were mounted on glycerin jelly. Fluorescent photomicrographs were taken using a Nicon microscope equipped with a camera unit (Nicon Co., Tokyo, Japan) Normal rabbit serum, PBS, and FITC conjugates alone were each tested with control staining, performed in a manner similar to that for the antibodies.

Biomechanical study

For the biomechanical study of the reconstructed ligaments, we used femur-ACL-tibia complexes (FATCs). They were examined 6, 12, 18, 24, and 48 weeks after surgery. A total of 25 FATCs with normal controls from left knees were examined at each time point. After sacrifice, the FATCs were preserved at −60°C in a freezer. They were thawed at room temperature (approximately 20°C) for one night before the mechanical tests were performed. Loosening of the substitute was defined as antero-posterior instability, measured as a joint displacement length with 10 N anterior-posterior drawer manually with knee flexion of 90° before scraping nonligamentous tissues.

We used a special mounting device (Tanaka Medical Instrument Manufacturing Co., Tokyo, Japan) for mechanical stress to hold the specimens in a 45-degree knee flexion position to minimize the effect of knee flexion (Fig. 2). The tibial side of the device was designed to fix the load axis and the femoral components were designed to allow ±5 degree laxity. Both components could be rotated freely.

The crosshead speed (traction speed) as set at 5 mm/s (displacement rate of approximately 100% of the initial length of the specimens per sec.) and FATCs were loaded along the ACL axis.[4] The load-deformation curve was recorded with a mechanical testing machine, the Autograph DCS 2000 (Shimadzu Co., Kyoto, Japan). Mechanical properties, i.e., elongation, stiffness, and maximal load at failure were calculated. Stiffness was defined as a slope between 0 and 10 N on the load-deformation curve.

Statistical analysis

Statistical analyses of quantitative data were followed by Student's *t*-test, with significance being set at the level of $P < 0.05$. The data for each group were compared to examine significant differences.

Results

Histological study

One week after surgery, the rabbits were able to walk; after 3 weeks, they walked normally and jumped. Some rabbits limped 6 weeks postoperatively, but were able to jump and run later on. Two of the 65 rabbits died. The others were sacrificed according to the preprogrammed schedules. No infection was observed.

Control ACL structures in the left knee were white, glistening, and firm. A clockwise twist was noted in the left knee. At 6 weeks, the ACLs demonstrated moderate hypertrophy, being enlarged to approximately twice

their original size. A dull white and frayed appearance indicated necrosis of the tissue. Specimens from 48 weeks were covered with synovial membrane that was thicker at the tibial attachment. Carefully dissected ACLs were dull white, as at 6 weeks, but they were composed of firm and elastic hard tissue (Fig. 3).

Light microscopic study. Histologically, normal ACL showed round fibroblasts and fine fibrillar crimps with peritendineum (Fig. 4a). ITT showed spindle-shaped fibroblasts, coarse fibrillar crimp, and peritendineum (Fig. 4b).

Three weeks after surgery, the substitutes showed acellular changes, such as avascular necrosis. Fibrinoid degeneration was noted in some specimens. The arrangement of fibers with connecting tissue was almost the same as that of the ITT, except that 3-week substitutes had no spindle-shaped fibroblasts (Fig. 5a). In the bone tunnels, the interface between the substitute and bone was filled with coarse connective tissue. There were spindle-shaped fibroblasts and small round cells (Fig. 5b).

At 6 weeks, the peritendineum areas showed marked cellular proliferation which spilled out into the collagenous matrix, but the interior of the substitute was still acellular. In the connective tissue between the original ITT fibers, there were small round cells. These findings were noted in two specimens; the other specimens did not have an acellular area but they had spindle-shaped cells inside the bundles (Fig. 6a). At the ligament-bone interface, the ligament was attached to the bone with fibrocartilage, but the border was clearly visible. Calcified fibrocartilage was seen at the side of the bone tunnel (Fig. 6b).

At 12 weeks, we observed homogeneous distribution of fibroblasts and longitudinal arrangement of the collagen bundles. Connective tissues were first observed from 6 weeks and the original collagen fibers could not be distinguished at 12 weeks. The quantity of cells had increased, compared to the 6 week substitutes (Fig. 7a). The borders of the bone-ligament interface were complicated and indistinguishable from the former ligaments. In addition to the fibrocartilage cells, we found osteoblasts (Fig. 7b).

At 24 weeks, the fibroblasts in the graft had changed markedly, to a round shape from their original spindle-shape. The quantity of cells had decreased compared to the 12-week specimens. The arrangement of the fiber revealed fine strands and homogeneity (Fig. 8a). At the ligament-bone junction, the substitute had changed to fibrocartilage on the peripheral margins of the tunnels,

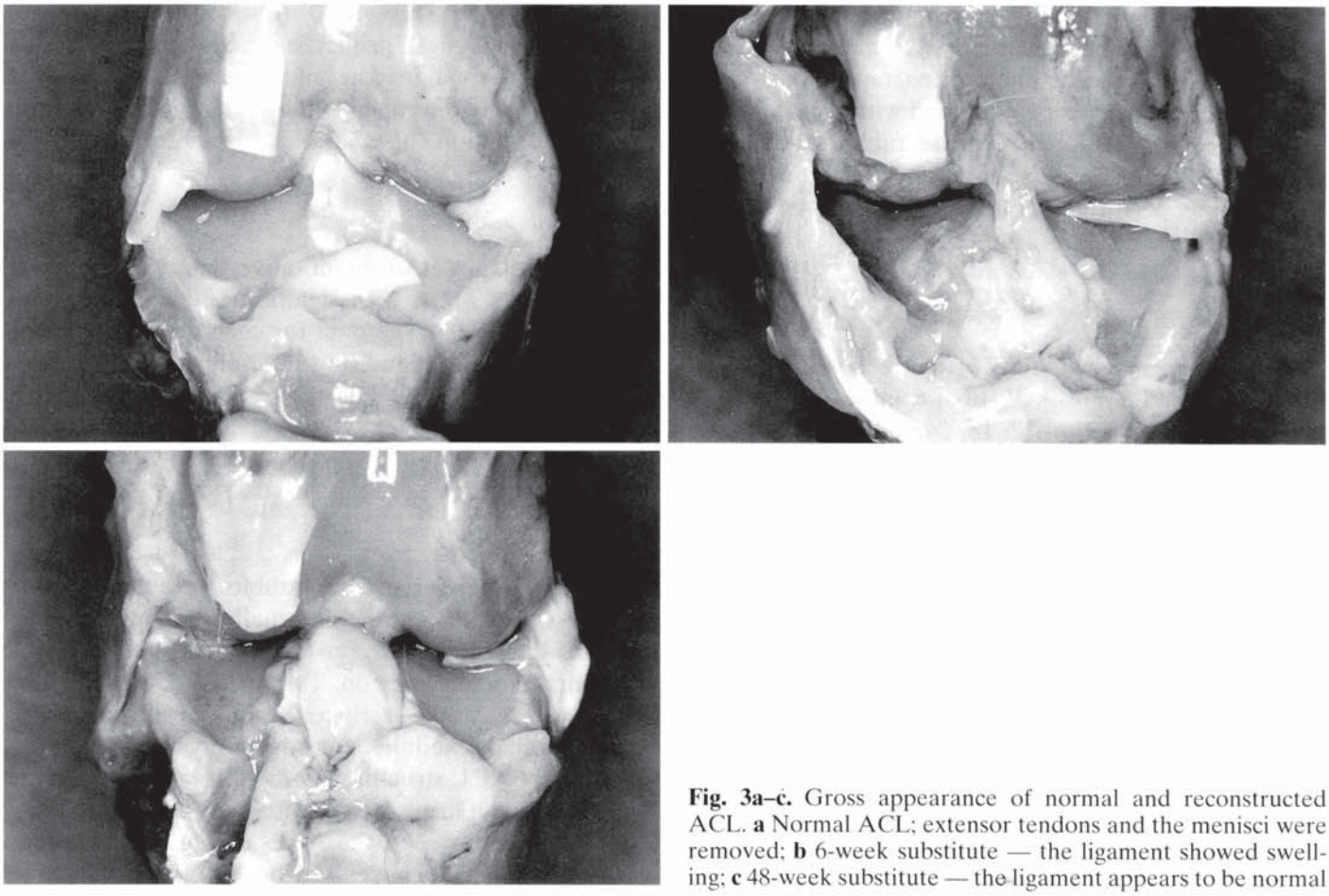

Fig. 3a–c. Gross appearance of normal and reconstructed ACL. **a** Normal ACL; extensor tendons and the menisci were removed; **b** 6-week substitute — the ligament showed swelling; **c** 48-week substitute — the ligament appears to be normal

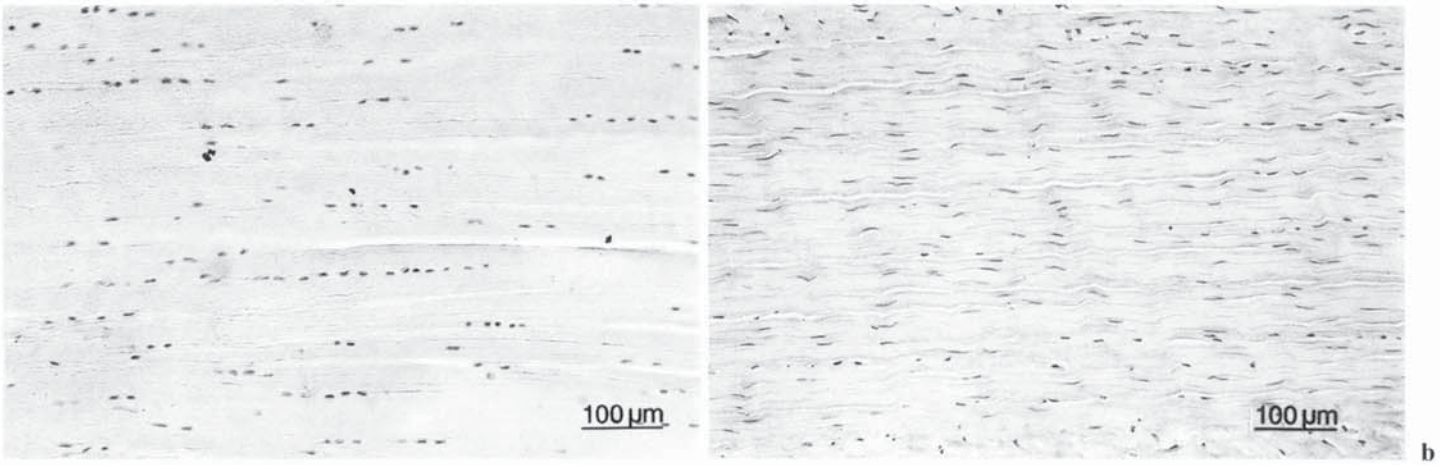

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Fig. 4a,b. Photomicrographs of normal ACL and iliotibial tract. **a** ACL; ACL cell is ovoid, with a halo that resembles those of chondroblasts. H&E, ×100. **b** Iliotibial tract; spindle-shaped fibroblasts are seen. H&E, ×100

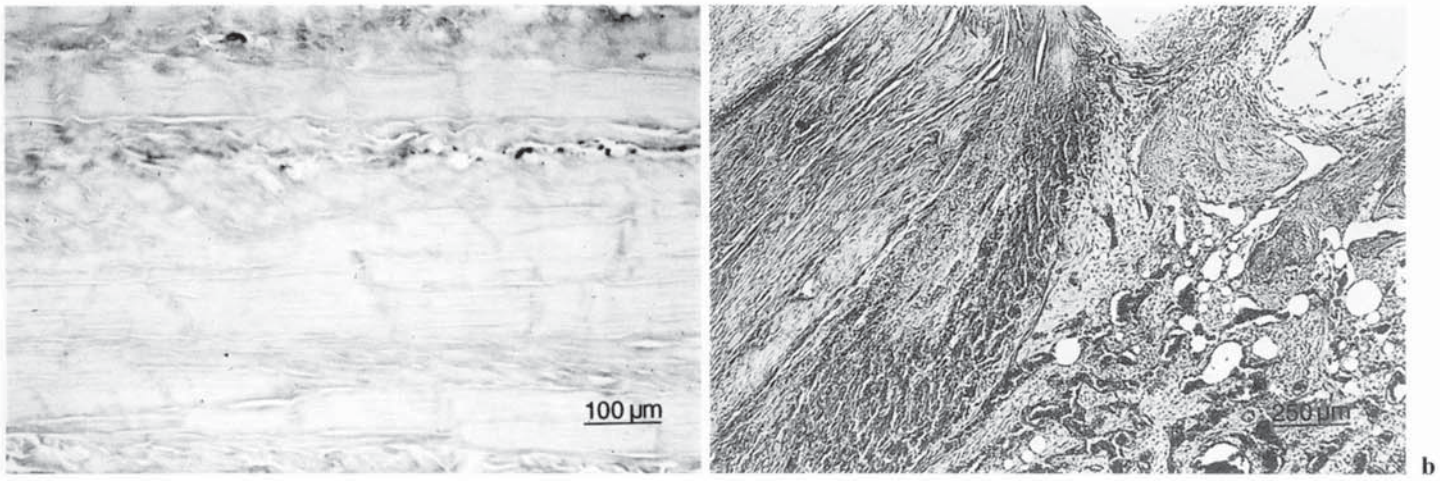

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Fig. 5a,b. Photomicrographs of 3-week substitutes. **a** Longitudinal section of the mid-substance; no cells are seen in the middle portion (avascular necrosis). H&E, ×100. **b** Longitudinal section of the tibial ligament-bone junction. Small round cells are seen in the rough soft tissue. H&E, ×40

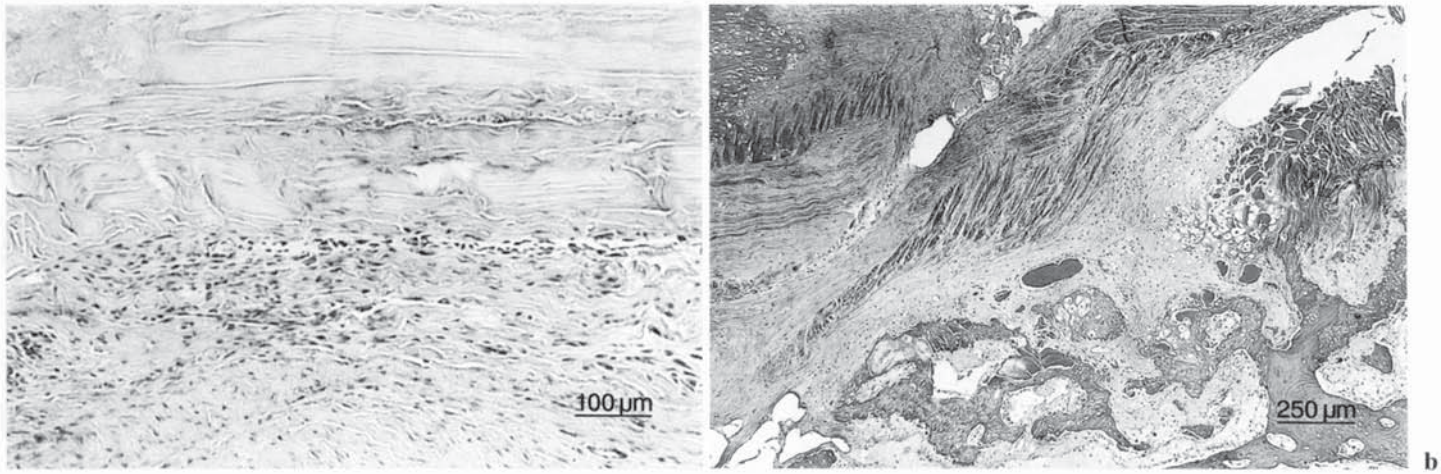

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Fig. 6a,b. Photomicrographs of 6-week substitutes. **a** Longitudinal section of the mid-substance. Repopulation of fibroblasts is beginning from the peripheral part. H&E, ×100. **b** Longitudinal section of the tibial ligament-bone junction; fibrocartilage cells are noted at the ligament-bone junction. H&E, ×40

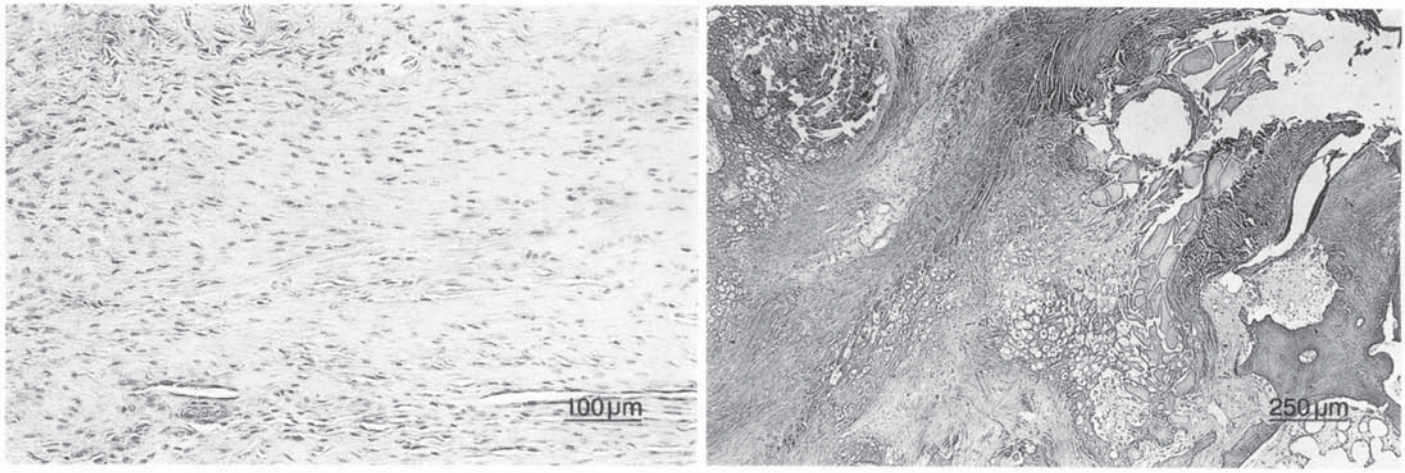

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Fig. 7a,b. Photomicrographs of 12-week substitutes. **a** Longitudinal section of the mid-substance. The fibroblast population is increasing. H&E, ×100. **b** Longitudinal section of the tibial ligament-bone junction; ossification of the chondrofibrocartilage is noted. H&E, ×40

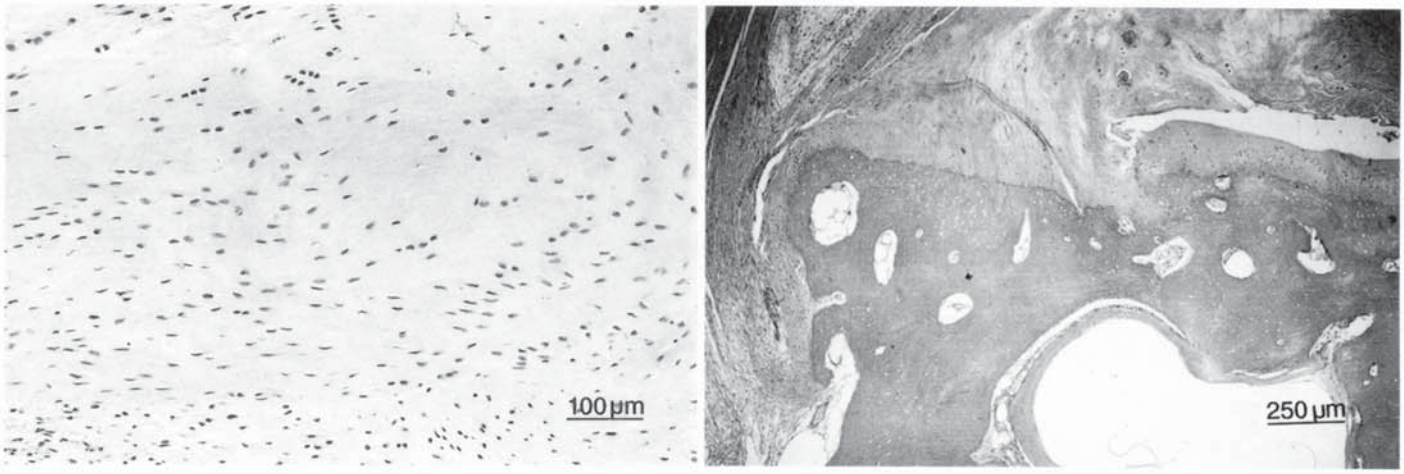

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Fig. 8a,b. Photomicrographs of 24-week substitutes. **a** Longitudinal section of the mid-substance. Cells are aligned with the fibers. H&E, ×100. **b** Longitudinal section of the tibial ligament-bone junction. The autografts are ossifying from the peripheral margin. H&E, ×40

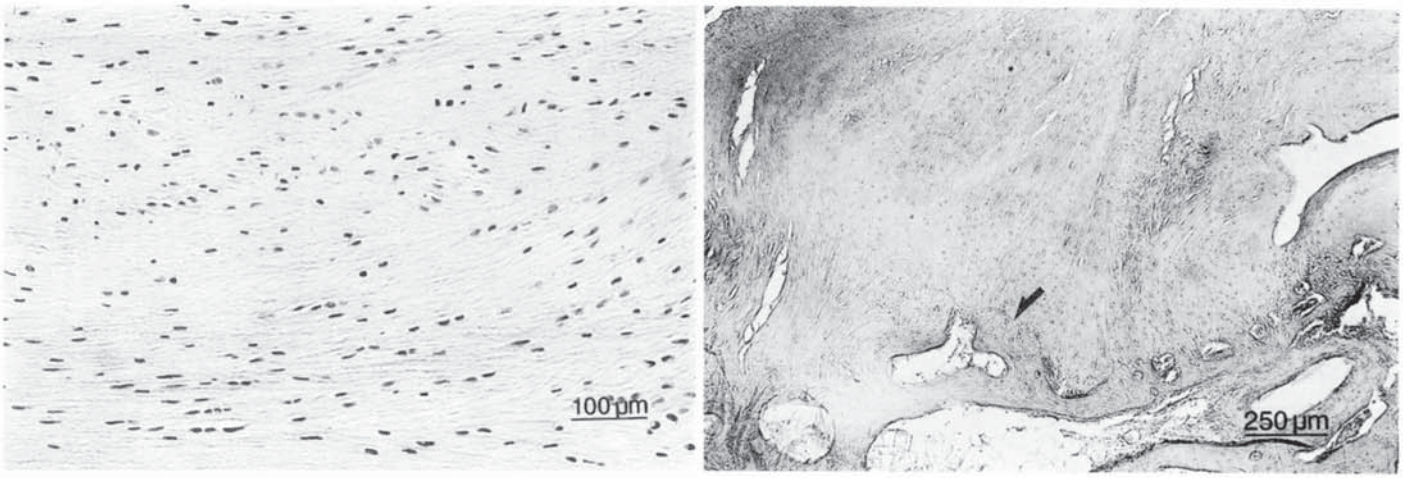

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Fig. 9a,b. Photomicrograph of 48-week substitutes. **a** Longitudinal section of the mid-substance. The reconstructed ACL appears to be normal; Cell quantities are similar to those in normal ACL. H&E, ×100. **b** Longitudinal section of the tibial ligament-bone junction. Basal bone is formed under the reconstructed ligament. A tide line is visible, as in the normal ACL (*arrow*). H&E, ×40

this being particularly notable from the posterior side. The basal bone of the ligament interface was also ossifying from the peripheral margin (Fig. 8b).

In 48-week substitutes, ovoid cells, similar to the native ACL cells, were noted. These ovoid cells were surrounded by a so-called amorphous matrix (Fig. 9a). The basal bone of the interface was completely formed in these substitutes. A columnar arrangement of cells and a tide mark were visible, as in the normal ACL (Fig. 9b). Enlargement of bone tunnels was found in two cases. Substitutes were not seen continuously in the bone tunnels.

Electron microscopic study. Transmission electron microscopic examination showed that the normal ACL had a biphasic appearance in regard to the diameter of the fibrils, the thinner ones being about 150 nm in diameter. However, the normal ITT showed mainly large fibrils with a diameter of about 150 nm.

Twelve weeks after reconstruction, most collagen fibrils were significantly smaller in diameter than those of the native ITT. Larger diameter fibrils appeared at 24 weeks; variation in fibril diameter was similar to that of the normal ACL (Fig. 10).

The diameter of the ACL was 107.3 ± 38.9 mm (mean ± S.D.), that of the ITT, 136.0 ± 29.2 mm, that of the 12-week autograft, 86.5 ± 23.8 mm, and that of the 24-week autograft, 114.3 ± 42.9 mm. There was no significant difference between the diameter of the ACL and that of the 24-week specimens (Fig. 11).

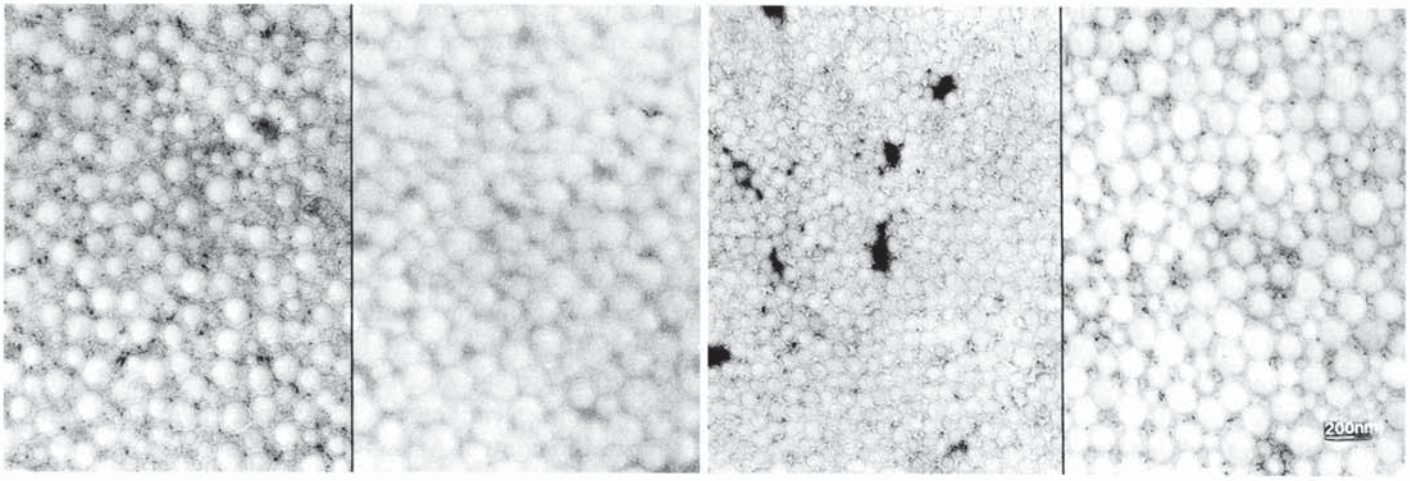

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Fig. 10a,b. Transmission electron photomicrographs of transverse sections at approximately ×15000 magnification. Fibrils of ACL, ITT, and autografts (12-weeks, 24-weeks) demonstrate variation of fibril diameter in subpopulations within a single cross section. **a** ACL (*left*) and ITT (*right*). **b** Twelve weeks (*W*; *left*) and 24-weeks (*W*; *right*)

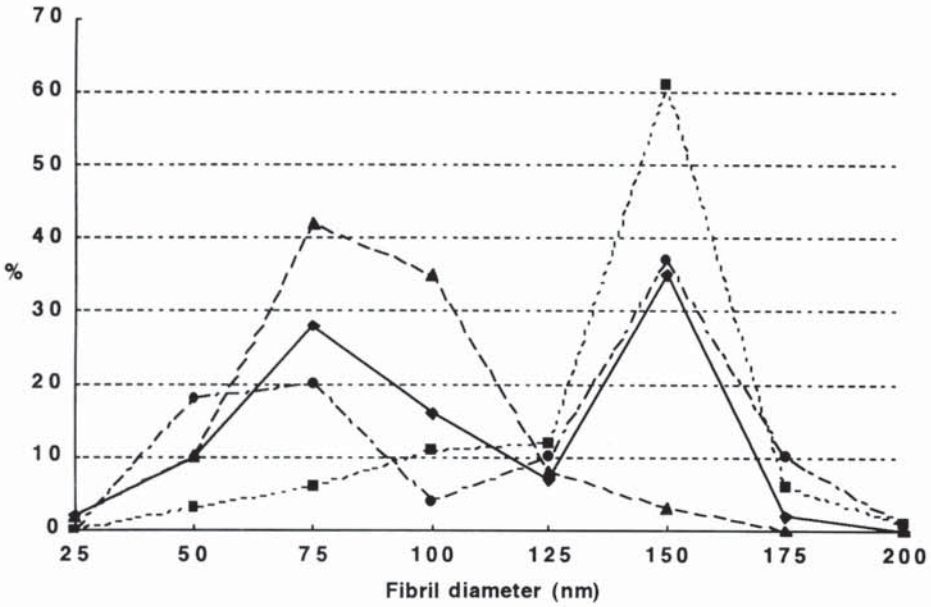

Fig. 11. Histogram of fibril diameter distribution in ACL (*diamonds*), ITT (*squares*) and autografts (12 weeks, *triangles*; 24 weeks, *dots*). Note significant skew of ACL distribution. A total of approximately 7500 fibrils was measured for each test group

Immunohistochemical study. On immunofluorescent staining, type I collagen was distributed evenly in the fibrillar collagenous network of primary bundles. Type III collagen was distributed mainly around the peritendineum areas rather than in the fibers. The fibroblasts in normal ACL and ITT were stained by anti-collagen type I. Type I collagen antibody stained newly formed oval shaped fibroblastic cells and fibers, this staining being more apparent in 24-week and 48-week specimens than in 12-week specimens.

Type III collagen, which appears in premature tissues, was observed in the area adjacent to the peritendineum of the ITT. Three-week specimens had bundles of type III collagen, which corresponded to the primary peritendineum of the ITT. The staining became more intense after 12 weeks, indicating that the collagenous tissue matured in this period. Type III collagen, which was found in the peritendineum areas of 3-week specimens, was not seen in 6-week specimens. The bundles had disappeared within this 3-week period (Fig. 12).

Biomechanical study

The lengths of the control ACL and the rolled ITT were 8.4 ± 1.4 and 10.2 ± 1.1 mm, respectively and their diameters were 2.6 and 2.7 mm, respectively. The diameter at the middle portion of the ACL was 2.6 ± 0.2 mm approximately, and it became wide and flat distally. The autografts were elongated postoperatively, the maximum length at 6 weeks being 12.0 mm ± 1.5 mm. A clockwise twist was found in the right ACL, but the reconstructed graft had a cone-shaped appearance without a twist. The graft diameter was maximum at 6 weeks, most of the grafts being narrower at 24 and 48 weeks.

The antero-posterior instability of the ACL was less than 2 mm, and in the mechanical test with knee flexion at forty-five degrees, four of five knees broke at the middle portion. One knee exhibited an avulsion fracture on the tibial side; the data for this knee were excluded from the control data. Maximal load for ACL failure at the middle portion was 269.7 ± 38.7 N and stiffness was 156.0 ± 7.0 N/mm. The maximal load for ITT 20 mm in width and 10 mm in length was 135.7 ± 22.7 N and stiffness was 136.3 ± 8.9 N/mm.

Positive antero-posterior instability of more than 2 mm was noted in three of the 6-week specimens, and in two of the 12-week specimens, although this occurred only in the early postoperative period. In the later postoperative period, only one specimen out of five in each group had antero-posterior instability at 24 and at 48 weeks.

At failure, three knees from the 6-week specimens and one knee from the 12-week specimens were pulled out from the tibial attachment. The rest were ruptured in the mid-portion of the ligament.

Elongation length at failure was between 1.9 and 3.3 mm. Although the difference was not significant, there was a smaller elongation length at failure in the later postoperative period (Fig. 13a).

Stiffness was lowest in 12-week specimens. Stiffness in 24-week specimens was 152.0 ± 11.7 N/mm, similar to that of the control ACL and significantly stronger than the 12-week specimens (Fig. 13b).

The maximal strength of the substitute at the reconstruction was 148.6 ± 32.7 N, that is, 55% of the control ACL strength. It was only 88.7 ± 17.7 N at 12 weeks, i.e.,

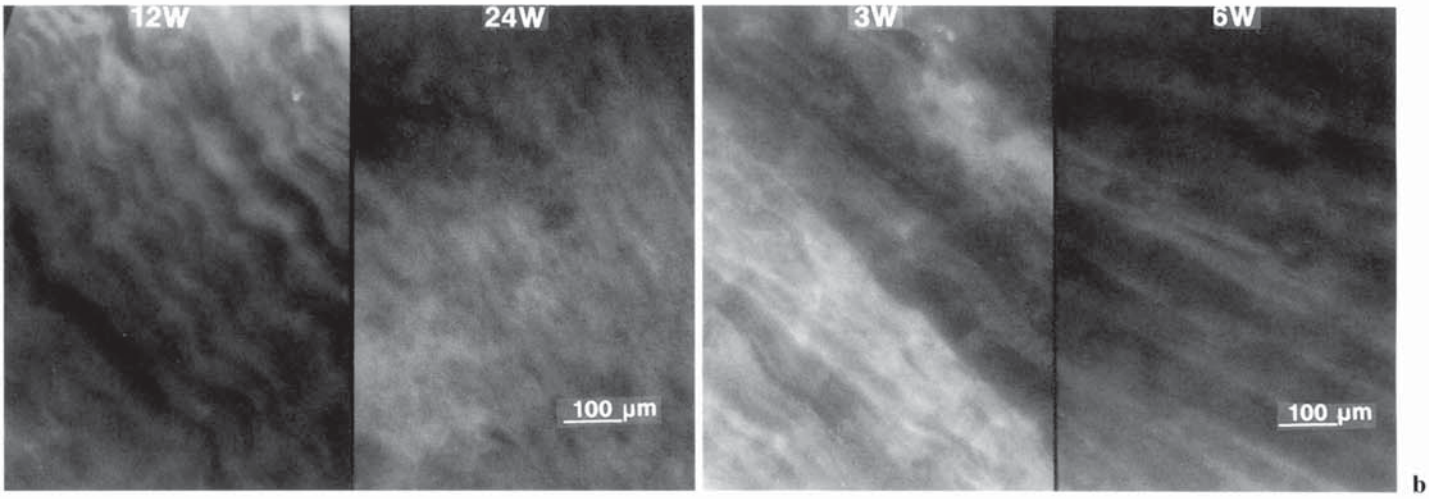

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Fig. 12a,b. Immunofluorescent photomicrographs of reconstructed ligament. **a** Localization of type I collagen (*12 W, 24 W*); type I collagen is distributed evenly in the fibrillar collagenous network of primary bundles. **b** Localization of type III collagen (*3 W, 6 W*); 3-week specimens had bundles of type III collagen that corresponded to the primary peritendineum of the ITT

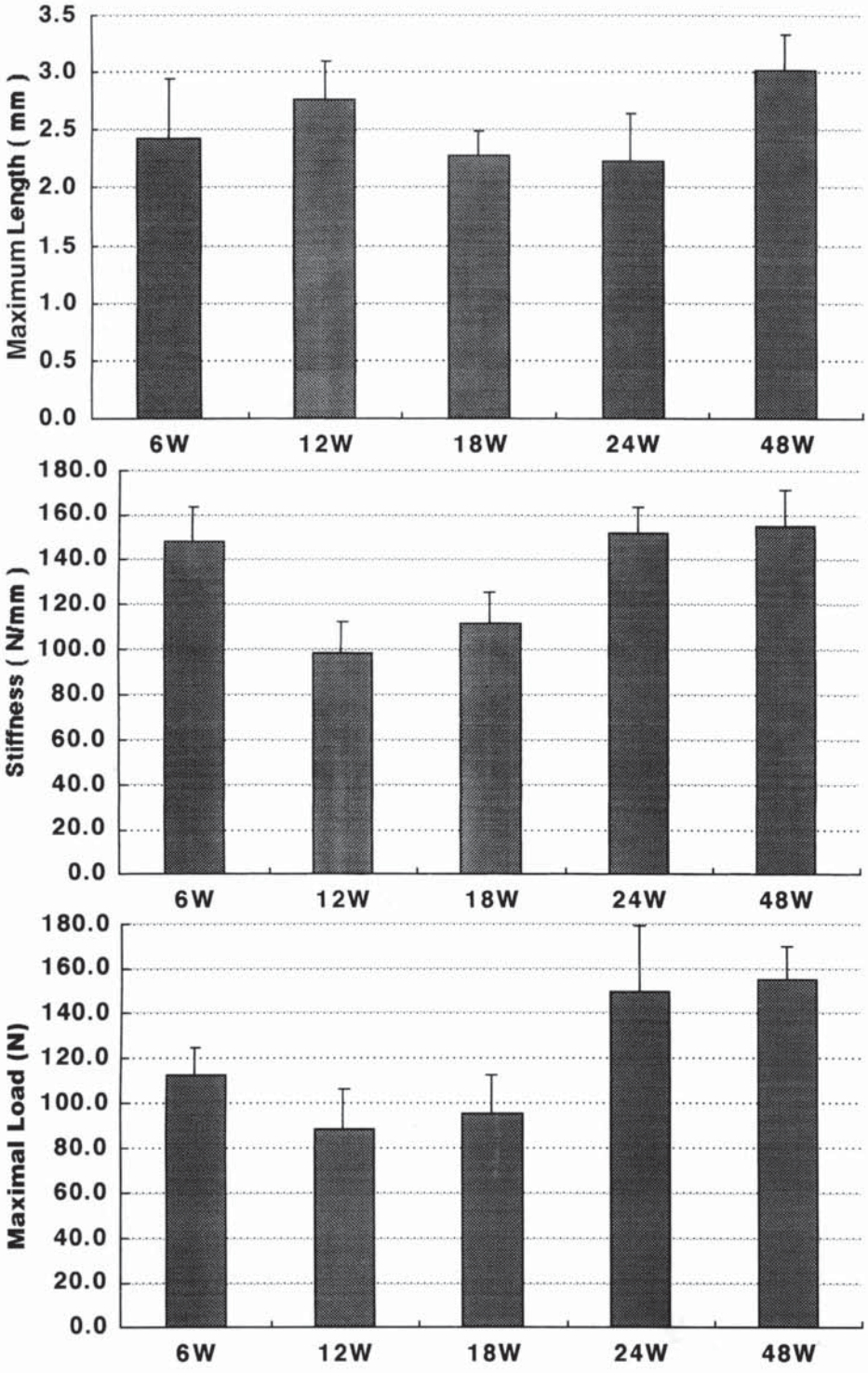

Fig. 13a–c. Histograms of mechanical properties of ITT autograft. **a** Elongation to maximum length; no significant differences were seen between groups at various postoperative times. **b** Ligament stiffness; 24-week specimens were significantly stronger than the 12-week specimens. **c** Maximal load; the maximal load of 24-week and 48-week specimens was significantly higher than that of 12-week and 18-week specimens. *Error bars* represent ±1 SD

26% of the normal ACL strength or 49% of the initial strength. The maximal load of 24-week and 48-week specimens was significantly greater than that of 12-week and 18-week specimens. The 48-week specimens had recovered to 155.4 ± 14.2 N, i.e., 55% of the normal ACL strength or 95% of the strength of the substitute used at the reconstruction (Fig. 13c).

Discussion

Histological maturation

Amiel et al.[1] have shown that the PT autograft undergoes dramatic changes in structure, effectively transforming itself into a ligamentous tissue. These histological changes can be divided into five stages: (i) 3 weeks post-surgery, evidence of avascular necrosis is

noted along with cell death, hypocellularity, and collagen fragmentation. (ii) The proliferation of mesenchymal cells is noted 6 weeks post-surgery. (iii) Twelve weeks post-surgery, there is cell proliferation. (iv) At 24 weeks, the graft appears less proliferative. (v) At 48 weeks, the autograft has the same histological appearance as normal ligament. In the many experimental studies of ACL reconstruction, histological maturation of the autograft is generally similar, regardless of the type of substitute used.[1,2,5,16–18,20,28] Our present study also indicates that substitutes underwent acellular changes and cell repopulation after 3 weeks. In 6-week specimens, we found that cellular repopulation was increasing from the peripheral margin of the substitute toward the inside. This suggests that the substitute is fed by the peripheral area in which synovial membranes exist. The finding that a free graft placed into the knee joint has the ability to produce collagen after 1 year postoperatively indicates that graft nutrition is provided by the synovial membrane.[8] Kleiner et al.[12] examined the origin of replacement cells for ACL autografts after 3 weeks and reported that the cells were of external origin. Compared with other studies,[1–5,21,25] our findings showed that the maturation of the ITT autograft was a little slower than that of the PT autograft. This may have been because the ITT we employed was wider and was used in a roll, so that the surface peritendinous tissue inhibited the rapid maturation that occurs in the PT autograft. Our immunohistological study showed that type III collagen was present in the peritendinous area until 6 weeks postoperatively.

Maturation of interface

The ligament-bone junction of the ACL contains many layers (ligament, fibrocartilage, calcified fibrocartilage, and bony layer).[6] This structure protects the ACL from intolerable tensile stress. It is advantageous to use the patellar tendon with a bone ligament junction for ACL reconstruction if this structure is preserved. It has been shown that the use of the patellar tendon in ACL reconstruction with bone leads to earlier and stronger completion of fixation than the use of the tendon without bone.[13] The same findings also apply when the ITT is used. Rens et al.[28] placed the bone plug into the bony tunnel and found that the ligament-bone junction appeared after 12 weeks postoperatively. If the ITT is used without a bone plug, the formation of the ligament-bone junction is not clear. There are conflicting reports as to whether the basal bony layer is formed and whether side-to-side attachments are made with the bony layer.[11,23,26,28] Experimental studies of the transplantation of tendons into the bony tunnel at the extra-articular space show a continuity of ligament and bone collagenous fiber within 4–6 weeks.[23] Imai[11] reported that, using fascia lata as a substitute for ACL, ligaments attached side-by-side to bone tunnels in the non-fixation groups, and ligament-bone junctions were noted in the 4-week postoperative fixation group. In our study, we did not employ postoperative fixation, and a ligament-bone junction like that of the normal ACL was noted in the 24-week specimens. We made 3.0-mm drill holes and the 2.5- to 2.7-mm substitute was passed through them. There was no tissue between the substitute and the bone tunnel. Postoperatively, this space was sometimes filled with blood, and, 12 weeks after surgery, the infiltration of spindle-shaped fibroblasts and inflammatory small round cells was noted. The substitute was fixed to the bone with fibrocartilarge 12 weeks after surgery. Ossification of the interface was gradually completed by 48 weeks. Finally, the basal bone was formed; this suggests that the use of a wider substitute requires a longer duration for the interface to mature. In contrast to the wide substitute, a few reconstructed ligament looked poor and peripheral bone defects were noted. This suggests that the space between the substitute and bone tunnel needs to be minimized and filled with tissue.

Biomechanics

Biomechanically, none of the ligaments had pulled out from their bone tunnels after 12 weeks, suggesting that the strength of the ligament-bone interface was greater than that of the midsubstances. The finding that a wider substitute delays histological maturation at the ligament-bone junction is not an obstacle to producing strong ligaments. Despite the many experiments that have been performed, there have been no reports that the autograft acquired the same strength as the normal ACL. The mechanical strength of reconstructed ligaments is affected by various factors, such as the quantity the substitute, their type,[21] the surgical procedure, and the postoperative rehabilitation program.[20] In this study, we made wider ligaments than those reported previously, so that the initial mechanical properties were the same as those of the control. However, the substitutes eventually became narrower, perhaps because of wear at the notch between the condyles, or because of a decrease in the capacity to synthesize collagen.

Clinically, a strong substitute is needed for the ACL. Noyes et al.[21] reported that one-third of the patellar tendon is 1.7 times stronger than the ACL in humans. An ITT band 4 cm wide has 60% of the strength of the ACL and a 6-cm-wide band has the same strength as the ACL. These findings are similar in rabbits. In our study, the strength of the transplanted ligaments at 48 weeks was almost the same as their initial strength. Theoreti-

cally, we could have made a ligament equivalent in strength to the ACL by using a wider ITT band, but there would have been many problems, such as wear, delayed maturation, and complications of harvesting substitutes.

Conclusions

1. The substitute underwent avascular necrosis 3 weeks postoperatively and cellular repopulation, originating from the external margin, was noted within 6 weeks.
2. In the ACL reconstruction in which the ITT autograft was employed without a bony plug, the ligament-bone junction was formed by 12 weeks and a tideline was noted at 48 weeks.
3. Biomechanically, the maximal load of the reconstructed ligament reached 55% of the strength of the normal ACL 48 weeks postoperatively, and it exhibited 95% of the strength of the initial ITT.

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