Anterior cruciate ligament reconstruction using autologous double hamstrings: a comparison of standard *versus* minimal debridement techniques using MRI to assess revascularisation

A RANDOMISED PROSPECTIVE STUDY WITH A ONE-YEAR FOLLOW-UP

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Animal studies have shown that implanted anterior cruciate ligament (ACL) grafts initially undergo a process of revascularisation prior to remodelling, ultimately increasing mechanical strength. We investigated whether minimal debridement of the intercondylar notch and the residual stump of the ruptured ACL leads to earlier revascularisation in ACL reconstruction in humans. We undertook a randomised controlled clinical trial in which 49 patients underwent ACL reconstruction using autologous four-strand hamstring tendon grafts. Randomised by the use of sealed envelopes, 25 patients had a conventional clearance of the intercondylar notch and 24 had a minimal debridement method. Three patients were excluded from the study. All patients underwent MR scanning post-operatively at 2, 6 and 12 months, together with clinical assessment using a KT-1000 arthrometer and International Knee Documentation Committee (IKDC) evaluation. All observations were made by investigators blinded to the surgical technique. Signal intensity was measured in 4 mm diameter regions of interest along the ACL graft and the mid-substance of the posterior cruciate ligament.

Our results indicate that minimal debridement leads to earlier revascularisation within the mid-substance of the ACL graft at two months (paired *t*-test, $p = 0.002$). There was a significant reduction of mid-substance signal six months after the minimal debridement technique (paired *t*-test, $p = 0.00007$). No statistically significant differences were found in tunnel placement, incidence of Cyclops lesions, blood loss, IKDC scores, range of movement or Lachman test between the two groups.

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The success of anterior cruciate ligament (ACL) reconstruction depends on revascularisation of the graft,¹⁻⁶ with rehabilitation protocols dependent on the stage of that revascularisation. The literature suggests that autograft is strong initially, weakens during revascularisation between six and 16 weeks, and gradually regains strength after six months.^{7,8}

The process of revascularisation has been assessed by histological studies alone or combined with conventional MRI, or contrast-enhanced MRI using gadolinium-diethylenetriamine pentacetic acid (Gd-DTPA), on both autologous hamstring and patellar tendon grafts.^{7,9-17} Most histological studies have involved animals. Serial histological specimens at various intervals following grafting all showed revascularisation at three months

post-operatively.^{1,3-5} There have only been a few human studies assessing revascularisation using MRI. Non-contrast MRI studies of hamstring grafts^{7,12} and Gd-DTPA-enhanced studies of patellar tendon grafts,² have shown increased signal at three months. Other MRI studies with contrast have failed to show enhancement of the reconstruction, leading to the suggestion that graft viability may be caused by synovial diffusion alone.^{15,18}

There have been few studies correlating the signal intensity of the graft on MRI with histological or biomechanical parameters during early and late remodelling. Weiler et al⁸ assessed grafts in sheep with follow-up at various intervals over two years. At each time interval, grafts were assessed for vascularisation and mechanical strength, comparing histology and MRI findings using conventional

and Gd-DTPA-enhanced scans. They found that at three months, the graft was fully revascularised, and at six months it resembled a native ACL. This was confirmed both histologically and with increased signal intensity on MRI. In general, correlations for contrast-enhanced measurements were stronger than for non-contrast MRI. There was also a negative linear correlation between increased signal and reduced tensile strength and load to failure suggesting that the better the revascularisation process, the stronger the graft becomes.

The anatomical structures that mediate revascularisation of the graft include the infrapatellar fat pad, the ligamentum mucosa, the remnant of the ACL stump and the posterior synovial tissues as described by Arnoczky et al.¹ Anterior cruciate ligament reconstruction can be performed with minimal debridement of the intercondylar notch, which attempts to preserve the whole of the ligamentum mucosa, as much of the posterior synovial tissues, the fat pad, and the stump of the ruptured ACL, as possible. Analysis of the process of revascularisation has previously been based only on animal studies. If the process is accelerated it is not certain whether this would be matched by an increase in the strength of the graft or whether the strength would also return more quickly. There have been no studies investigating the factors that affect the speed of revascularisation in ACL reconstruction in humans.

We questioned whether minimal debridement of the notch and the remnant of the stump of the ACL could affect the speed of revascularisation. We therefore undertook a prospective randomised study to evaluate the fate of an autologous double-hamstring ACL graft using normal and minimal debridement surgical techniques, by measuring signal intensity using non-contrast MRI to assess revascularisation. We correlated the MRI results with our clinical findings at the one-year follow-up.

Patients and Methods

All patients included in the study gave their consent to participate. The patients were undergoing primary ACL reconstruction, all were skeletally mature and had injured their knee at least three weeks prior to the reconstruction. The patients were randomised into two groups by sealed envelopes in the operating theatre, depending on the operative technique used. All operations were performed by the same surgeon (PA). There were 25 patients in the normal debridement group (13 men and 12 women) with a mean age of 35.5 years (21 to 50) and 24 patients (14 male and 10 female) in the minimal debridement group with a mean age of 30.5 years (15 to 59). The mean time to surgery after injury was 10.4 weeks (6 to 52) in normal group, and 8.2 weeks (5 to 47) in the minimal debridement group. In the normal group, 13 patients (52%) had a partial meniscectomy or meniscal repair performed for associated injuries, in contrast to 17 patients (71%) in the minimal debridement group. In three patients in each group a chondroplasty was required. In total, three patients were excluded

from the study. One patient in the normal group was excluded because of graft re-rupture at five months, requiring re-operation, and hence did not receive subsequent MR scanning at the allocated intervals. In the minimal debridement group, two patients were excluded at six months, one because of a prominent tibial screw projecting into the intercondylar notch, and one because of graft impingement, both causing high artefactual signal intensity in the graft on MR scans. These two patients were also not available for the 12-month scan. We elected to use non-contrast MRI because of financial limitations and patient compliance, which we considered to be important to encourage adequate follow-up.

We recorded the intra-operative findings and the post-operative blood loss via a suction drain placed near the tibial tunnel. All patients attended for clinical review at two weeks, and at 2, 6 and 12 months post-operatively. Clinically we assessed swelling, range of movement, stability and complications. At the one-year review a clinical evaluation of the function and stability of the knees was performed by an author (SG), who was blinded to the treatment group. Measurements of passive extension and flexion using a goniometer, and stability using the KT-1000 arthrometer (MEDmetric corporation, San Diego, California) and the Lachman test were performed. Displacement was recorded in millimetres during a manual maximal translation. Stability was expressed as the difference in translation in millimetres between the reconstructed and the normal knees. Patient satisfaction and functional outcome were determined from the response to the International Knee Documentation Committee (IKDC) scoring system,¹⁹ including the one-legged hop test. The hop index is defined as the percentage of distance jumped on the reconstructed knee divided by the distance jumped on the normal knee.

Operative procedure. An arthroscopically assisted, double-loop semi-tendinosus and gracilis hamstring autologous ACL reconstruction was performed on each knee. The tendons were harvested through a 2 cm transverse incision. The two grafts were double-looped and secured with sutures at either end, using an endobutton (Smith and Nephew, Perth, Australia) at the proximal end. The combined cross-section of the four strands was measured by passing the graft through hollow cylindrical sizers. An Acuflex transtibial jig (Smith and Nephew) was used for tunnel placement. The tibial landmark for graft placement was just posterior to the site of the remnant of the ACL. The femoral tunnel was drilled just anterior to the posterior cortex in the intercondylar notch at either the 11 o'clock or the one o'clock position for the right and left knees, respectively, leaving a 1 mm to 2 mm posterior wall. The graft was secured in the femoral tunnel with an endobutton (Smith and Nephew) and in the tibial tunnel with an interference screw (Depuy Mitek, Perth, Australia). Graft impingement was assessed arthroscopically in full extension. A suction drain was placed near the tibial tunnel prior to skin closure.

Fig. 1

View through a separate lateral portal and second arthroscope in a right knee, showing the use of the operative arthroscope as a retractor (ACL, anterior cruciate ligament).

Fig. 2

Arthroscopic view in a right knee showing the minimal debridement technique in relation to the femoral notch (ACL, anterior cruciate ligament; PCL, posterior cruciate ligament; LFC, lateral femoral condyle).

In minimal debridement surgery the infrapatellar fat pad, the ligamentum mucosa, the remnant of the ACL and as much notch synovium as possible, were left untouched. This was done through the transtibial drilling technique and restricting the debridement 'window' to 1 cm² for placement of the femoral tunnel (Figs 1 and 2).

Post-operatively, all patients were placed in a splint in extension for two weeks and allowed to bear weight as tolerated. Early range of movement exercises were commenced in bed on the first post-operative day. All patients received one subcutaneous dose of 40 mg clexane six hours post-operatively. Drains were removed at ten o'clock each morning after surgery, and the post-operative blood loss was recorded. Standard swelling measures (elevation, compression and ice) and analgesic protocols were implemented; all patients had regular panadeine forte, two tablets four times a day, plus 50 mg to 100 mg tramadol two-hourly as required, and 50 mg diclofenac eight-hourly as required. All patients had early patellar mobilisation (manually-assisted vertical and horizontal patella movement in bed for six weeks, and at two months were allowed to progress to gentle exercise bicycle and swimming exercises).

MRI protocols. All patients (24 in the normal group and 22 in the minimal debridement group) underwent MR scanning at 2, 6 and 12 months post-operatively and were assessed clinically within two weeks of their scans. All were available for follow-up scans, with the exception of those who were excluded from the study. All scans were performed on the same Sigma GE 1.5T MRI scanner with excite platform (General Electric, Milwaukee, Wisconsin) in a dedicated knee coil. Imaging was confined to 3 mm thick slices with a 0.5 mm gap between each slice. Three types of sequencing were used: (A) sagittal and coronal proton density fast spin echo (FSE) sequences (repetition

time (TR) = 3000 ms, echo time (TE) = 30 ms, echo train length = 8, 2 number of excitations (NEX), 320/224 matrix); (B) sagittal and coronal T2 fat-suppressed FSE sequences (TR = 4000 ms, TE = 85 ms, echo train length = 12, 2 NEX, 256/192 matrix); (C) an additional sagittal proton density FSE sequence was performed through the intercondylar notch with the same parameters as in (A), but 2 mm thick slices were obtained, on which the study measurements were obtained.

MRI was used to measure the intensity of the signal, damage to the posterior cruciate ligament (PCL), the incidence of Cyclops lesions²⁰ and to assess impingement. The signal intensity of the graft was quantified using the signal/noise quotient (SNQ) formula:¹⁷

$$\text{SNQ} = \text{signal (ACL graft)} / \text{signal (background)}$$

Circular 4 mm diameter regions of interest were evaluated near the femoral tunnel, in the mid-substance of the graft, near the tibial tunnel, and within the tibial tunnel. The PCL signal was measured with the region of interest being placed in its mid-substance. Background measurements were calculated from the mean value of two measurements of a region of interest placed approximately 2 mm anterior to the patellar tendon ligament (Fig. 3).

Tunnel placement was evaluated using sagittal scans for the tibial tunnel and both coronal and sagittal scans for the femoral tunnel. On sagittal views, the position of the tibial tunnel was measured in millimetres posterior to Blumensaat's line²¹ drawn through the roof of the intercondylar notch with the knee in extension. The femoral tunnel placement was measured in millimetres anterior to the intersection of Blumensaat's line and the posterior femoral cortex (Fig. 4). On coronal views, the femoral tunnel position was determined in relation to ideal positioning on the

Fig. 3

Oblique sagittal MR scan in an operated knee showing positions of the femoral insertion (FI), mid-substance (MS), tibial insertion (TI), tibial tunnel (TT), mid-substance posterior cruciate ligament (PCL-MS) and background (BG) regions of interest for signal intensity measurement. The image also shows the double-bundle appearance of an implanted graft with increased signal intensity (arrows).

Fig. 4

Oblique sagittal MRI of an operated knee, showing the relationship between the anterior cruciate ligament graft and Blumensaat's line (BL).

Table I. Mean signal/noise quotient readings at different time intervals for each region of interest

Region of interest*	2 mths		6 mths		12 mths	
	Normal	Minimal debridement	Normal	Minimal debridement	Normal	Minimal debridement
Near femoral insertion	2.71 (0.79 to 6.99)	3.05 (0.58 to 4.65)	3.75 (1.22 to 8.31)	2.69 (1.32 to 6.21)	2.45 (1.1 to 4.58)	2.17 (1.01 to 4.34)
Mid-substance ACL	3.07 (0.79 to 7.25)	4.82 (1.55 to 11.2)	4.72 (0.87 to 8.02)	2.45 (1.06 to 4.22)	3.49 (0.86 to 11.28)	2.53 (0.97 to 4.03)
Near tibial insertion	3.53 (0.96 to 7.33)	4.76 (2.2 to 10.82)	4.61 (0.86 to 8.55)	3.63 (0.98 to 9.71)	3.48 (1.0 to 7.37)	3.09 (0.8 to 9.65)
Within tibial tunnel	4.56 (0.96 to 7.46)	7.12 (2.6 to 15.41)	4.79 (0.8 to 12.02)	4.66 (1.2 to 10.47)	3.64 (1.58 to 8.42)	3.35 (1.6 to 8.23)
Mid-substance PCL	4.1 (0.42 to 9.39)	6.13 (2.54 to 13.08)	7 (1.1 to 10.37)	3.3 (0.64 to 8.64)	3.47 (0.98 to 8.42)	2.22 (0.63 to 5.23)

* ACL, anterior cruciate ligament; PCL, posterior cruciate ligament

clock face of 11 o'clock and one o'clock for the right and left knees, respectively. A radiologist (WB) read each MR scan without knowledge of the technique used or the clinical outcome of each patient.

Data analysis. Statistical comparison of vascularity between the two groups was undertaken by using analysis of variance (ANOVA) for ordinal data, and an unpaired Student's *t*-test was used for interval data. A *p*-value ≤ 0.05 was considered statistically significant.

Results

MRI findings. All femoral tunnels were ideally positioned on the coronal scans in both groups, except for two left knees in the minimal debridement group. These were both biased towards the 12:30 position. Sagittal tunnel place-

ment was a mean of 1.26 mm (0 to 6) anterior to the intersection of Blumensaat's line and the posterior femoral cortex in the minimal debridement group, compared with a mean of 1.29 mm (0 to 4) in the normal group. Tibial tunnel placement was a mean of 4.3 mm (0 to 11) behind the intersection of Blumensaat's line and the tibial cortex in the minimal debridement group, compared with a mean of 3.4 mm (0 to 7) in the normal group.

Signal intensity was measured between the two groups and in various regions of interest (Table I). An interesting finding was that the signal/noise quotient values were consistently higher in the lowest part of the graft in the tibial tunnel (mean signal/noise quotient normal group, 4.56 (0.96 to 7.46); minimal debridement group, 7.12 (2.6 to 15.41), compared with near the femoral insertion (mean

Fig. 5

Graph showing mean signal intensity measurements using the signal/noise quotient formula, for the mid-substance of the anterior cruciate ligament graft. Error bars indicate the standard deviation.

Fig. 7

Graph showing signal intensity measurements using the signal/noise quotient formula, for the mid-substance of the native posterior cruciate ligament. Error bars indicate the standard deviation.

Fig. 6

An implanted anterior cruciate ligament graft showing high signal intensity (black arrow) at two months on sagittal MR scan, compared with the low signal (white arrow) in the posterior cruciate ligament.

signal/noise quotient normal group, 2.71 (0.79 to 6.99); and in the minimal debridement group, 3.05 (0.58 to 4.65)). However, when looking at the mid-substance of the ACL graft, there were significant differences between the two groups at both two and six months (paired *t*-test, $p = 0.002$ and $p = 0.00007$, respectively) but not at one year (paired *t*-test, $p = 0.12$) (Fig. 5). The minimal debridement

group had a higher signal intensity at two months (Fig. 6), which then diminished and remained lower than in the normal group at six and 12 months.

The mid-substance PCL signal intensity showed significant differences at two, six, and 12 months, (paired *t*-test, $p = 0.04$, $p = 0.03$ and $p = 0.02$), respectively. This followed a similar trend to the mid-substance of the ACL graft (Fig. 7).

The MR scans detected Cyclops lesions in nine normal (37.5%) and 13 minimal debridement patients (59%), with no statistically significant difference between the two groups (chi-squared test, $p = 0.30$).

Clinical data. The mean blood loss was 175 ml (50 to 400) in the normal group compared with 118 ml (30 to 180) in the minimal debridement group (paired *t*-test $p = 0.06$).

A clinical assessment of knee stability and function was obtained in 46 of 49 patients at one year. The range of movement and Lachman data at 2, 6 and 12 months are presented in Table II. The minimal debridement group achieved slightly more flexion at two and six months but without any discernible difference at 12 months.

An anterior translation -1 to +2 mm was observed in 18 patients (75%) in the normal group and 20 patients (91%) in the minimal debridement group in the operated knee compared with the normal knee, determined by the manual maximum translation test using the KT-1000 arthrometer. The remaining six patients (25%) in the normal and two in the minimal debridement groups had less than 3 mm side-to-side difference in anterior translation. Two patients in the normal group had loss of extension of 3° and 5° respectively at one year and two patients in the minimal debridement group had loss of extension of 3° and 5° at one year. All five patients had Cyclops lesions shown on MRI. Using the IKDC evaluation, patients rated their knees very functional, with an approximate score of 90.2% ± 13 in the normal and of 92.6% ± 11.4

Table II. Clinical data at 2, 6 and 12 months showing mean range of movement in degrees using goniometer readings, and mean manual anterior translation using the Lachman test

Mean values	2 mths		6 mths		12 mths	
	Normal	Minimal debridement	Normal	Minimal debridement	Normal	Minimal debridement
Extension (°)	1.4 (0 to 5)	0.8 (0 to 5)	0.6 (0 to 5)	0.7 (0 to 5)	0.3 (0 to 5)	0.5 (0 to 5)
Flexion (°)	113 (90 to 135)	120 (85 to 130)	130.7 (120 to 145)	135.5 (120 to 140)	138.5 (120 to 150)	137.6 (120 to 145)
Anterior translation (mm)	3.1 (2 to 7)	3.5 (2 to 6)	2.8 (2 to 6)	3.2 (2 to 5)	2.75 (2 to 5)	3.2 (2 to 5)

Table III. The percentage hop index (the percentage of distance jumped on the reconstructed knee divided by the distance jumped on the normal knee) at one year between the two groups

Hop index (%)	Normal group (24 patients)	Minimal debridement group (22 patients)
50 to 75	1	1
76 to 89	1	1
> 90	22	20

in the minimal debridement group. The ability to do the one-legged hop test on the reconstructed knee was restored in the majority of patients, with a hop index of greater than 90% (Table III).

Discussion

To our knowledge this is the first prospective study of ACL reconstruction in humans assessing graft revascularisation after either normal standard preparation or a minimal debridement of the synovium of the notch, the residual stump of the ACL and anterior fat pad.

We were able to measure graft revascularisation quantitatively using standard MR scans by measuring signal intensity, and by using the signal/noise quotient formula. The minimal debridement technique appears to accelerate revascularisation, as indicated by increased signal in the mid-substance of the graft at two months. This might lead to an early return to maximum strength of the graft. Our data are in agreement with other studies that have demonstrated that the graft undergoes a process of revascularisation.^{6-8,12,13,22}

There were several weaknesses in our study which could have been improved by using contrast-enhanced MR scans, two independent radiologists interpreting the scans, a second-look arthroscopy combined with histological analysis, and longer follow-up. Signal intensity combined with histology and Gd-DTPA might have yielded better correlation, as in other studies.¹⁶ However, Gd-DTPA would have made the study more costly, time-consuming for the radiologist and more invasive and we were concerned that patient compliance might have been compromised. Histological specimens of grafts in correlation with MRI would have been preferable, but in human studies would have been difficult to justify ethically. MRI cannot delineate the mechanism of graft remodelling in the same way that histological examination has been used to evaluate ACL grafts in animals.

Although minimal debridement surgery is more technically demanding, our tunnel position was similar in both groups. The incomplete debridement of the notch and the remnant of the ACL stump did not lead to an increased incidence of Cyclops lesions, which remained similar in both groups with no statistically significant difference. We identified a number of Cyclops lesions in both groups when the knee was fully extended (seven of nine patients with Cyclops lesions in the normal group, compared with ten of 13 patients with Cyclops lesions in the minimal debridement group). The remaining five patients with Cyclops lesions all lacked full extension, but given our overall findings it may be reasonable to question whether the Cyclops lesion is the primary cause of loss of full extension.

It has been reported that high signal intensity in the distal portion of an ACL graft is caused by roof impingement of the graft,^{7,12,15,18} or that the ACL remnant is a contributing factor.¹² We noted that the portion of the graft within the tibial tunnel consistently had increased signal compared with all other regions of interest in both study groups.

There were significant differences in PCL signal intensity at all time intervals, and the pattern was similar to the mid-substance of the ACL graft. Possibly the PCL becomes surrounded by vascularised peri-ligamentous tissue, as found in previous studies,^{5,8,18} or maybe the initial high readings were caused by minor trauma to the PCL, which may be a consequence of the transtibial drilling technique, with poorer direct vision of the PCL in the minimal debridement technique.

In conclusion, from our data we have inferred that minimal debridement leads to earlier revascularisation at two months, but we have no evidence that this accelerates the recovery of strength in the graft.

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References

1. **Arnoczky SP, Tarvin GB, Marshall JL.** Anterior cruciate ligament replacement using patellar tendon: an evaluation of graft revascularisation in the dog. *J Bone Joint Surg [Am]* 1982;64-A:217-24.
2. **Chiroff RT.** Experimental replacement of the anterior cruciate ligament: a histological and microradiograph study. *J Bone Joint Surg [Am]* 1975;57-A:1124-7.
3. **Amiel D, Kleiner JB, Roux RD, Harwood FL, Akeson WH.** The phenomenon of 'ligamentization': anterior cruciate ligament reconstruction with autologous patellar tendon. *J Orthop Res* 1986;4:162-72.
4. **Drez DJ, Delee J, Holden J, et al.** Anterior cruciate ligament reconstruction using bone-patellar-tendon-bone allografts: a biological and biochemical evaluation in goats. *Am J Sports Med* 1991;19:256-63.
5. **Scranton PE, Lanzer WL, Ferguson MS, Kirkman TR, Pfister DS.** Mechanisms of anterior cruciate ligament neovascularisation and ligamentization. *Arthroscopy* 1998;14:702-16.
6. **Deehan DJ, Cawston TE.** The biology of integration of the anterior cruciate ligament. *J Bone Joint Surg [Br]* 2005;87-B:889-95.
7. **Howell SM, Clark JA, Blasier RD.** Serial magnetic resonance imaging of hamstring anterior cruciate ligament autografts during the first year of implantation: a preliminary study. *Am J Sports Med* 1991;19:42-7.
8. **Weiler A, Peters G, Maurer J, et al.** Biomechanical properties and vascularity of an anterior cruciate ligament graft can be predicted by contrast-enhanced magnetic resonance imaging: a two-year study in sheep. *Am J Sports Med* 2001;29:751-61.
9. **Gallimore GW Jr, Harms SE.** Knee injuries: high resolution MR imaging. *Radiology* 1988;160:457-61.
10. **Mandelbaum BR, Finerman GAM, Reicher MA, et al.** Magnetic resonance imaging as a tool for evaluation of traumatic knee injuries: anatomical and pathoanatomical correlations. *Am J Sports Med* 1986;14:361-70.
11. **Reicher MA, Hartzman S, Bassett LW, et al.** MR imaging of the knee. Part I: traumatic disorders. *Radiology* 1981;162:547-51.
12. **Murakami Y, Sumen Y, Ochi M, et al.** MR evaluation of human anterior cruciate ligament autograft on oblique axial imaging. *J Comput Assisted Tomogr* 1998;22:270-5.
13. **Vogl TJ, Schmidt J, Lubrich J, et al.** Reconstructed anterior cruciate ligaments using patellar tendon ligament grafts: diagnostic value of contrast enhanced MRI in a 2 year follow-up regimen. *Eur Radiol* 2001;11:1450-6.
14. **Maywood RM, Murphy BJ, Uribe JW, Hechtman KS.** Evaluation of arthroscopic anterior cruciate ligament reconstruction using magnetic resonance imaging. *Am J Sports Med* 1993;21:523-7.
15. **Howell SM, Clarke JA, Farley TE.** Serial magnetic resonance study assessing the effects of impingement on the MR image of the patellar tendon graft. *Arthroscopy* 1992;8:350-8.
16. **Rak KM, Gillogly SD, Schaefer RA, et al.** Anterior cruciate ligament reconstruction: evaluation with MR imaging. *Radiology* 1991;178:553-6.
17. **Stöckle U, Hoffman R, Schwedtko J, et al.** Value of MRI in assessment of cruciate ligament replacement. *Unfallchirurg* 1997;100:212-18 (in German).
18. **Howell SM, Knox KE, Farley TE, Taylor MA.** Revascularization of a human anterior cruciate ligament graft during the first two years of implantation. *Am J Sports Med* 1995;23:42-9.
19. **Irrgang JJ, Ho H, Harner CD, Fu FH.** Use of the International Knee Documentation Committee guidelines to assess outcome following anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc* 1988;6:107-14.
20. **Jackson DW, Schaefer RK.** Cyclops syndrome: loss of extension following intra-articular anterior cruciate ligament reconstruction. *Arthroscopy* 1990;6:171-8.
21. **Jacobsen K, Bertheussen K, Gjerloff CC.** Characteristics of the line of Blumensaat: an experimental analysis. *Acta Orthop Scand* 1974;45:764-71.
22. **Corsetti J, Jackson D.** Failure of cruciate ligament reconstruction: the biologic basis. *Clin Orthop* 1996;325:42-9.