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A literature review of autograft and allograft anterior cruciate ligament reconstruction

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Abstract Knee anterior cruciate ligament reconstructive surgery has significantly evolved and now includes the option of using an allograft. This has resulted in numerous studies evaluating the advantages and disadvantages of allografts. The purpose of this literature review is to evaluate this research and present important findings to allow the selection of the most appropriate graft source when considering allograft versus autograft reconstruction.

Keywords Orthopaedic surgery \cdot Ligament grafts \cdot Knee · Autogenous graft · Allogenic graft

Introduction

The anterior cruciate ligament (ACL) is an important stabilising structure of the knee, preventing anterior translation of the tibia in relation to the femur [\[58](#page-12-0)]. Rupture of the ACL is one of the most common sports injuries in active young people [[14,](#page-11-0) [101\]](#page-13-0), with an estimated 250,000 new ACL ruptures in the United States each year [\[75](#page-13-0)]. Rupture of the ACL impairs the stability of the knee, resulting in difficulty with athletic performance, increased risk of subsequent meniscal injury, and increased risk of early

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degenerative disorders [[23,](#page-11-0) [110](#page-14-0), [111\]](#page-14-0). A torn ACL cannot heal with conservative management and the outcome of repair alone is inferior to the results after reconstruction [\[23](#page-11-0), [30](#page-12-0), [64](#page-13-0)]. Consequently, ACL reconstruction (ACLR) has become the standard of care with over 100,000 ACLR procedures performed annually in, for example, the United States [[38\]](#page-12-0).

Given such a high incidence of ACL injury it is not surprising that there has been a large number of papers concerning the ACL—there have been well over 2000 scientific articles on the ACL published in the last 25 years [\[33](#page-12-0)]. Indeed, very few subjects in contemporary orthopaedic surgery have evoked as much controversy, thought and opinion as that of how to optimally reconstruct the ACL [[33\]](#page-12-0).

Reconstruction using autogenous tendonous tissue has emerged as the most popular method for reconstruction and has produced good clinical results [[1,](#page-11-0) [17](#page-11-0), [27](#page-12-0), [50](#page-12-0), [53](#page-12-0), [54,](#page-12-0) [82,](#page-13-0) [83](#page-13-0), [112\]](#page-14-0). However, a desire to avoid the sacrifice of autogenous tissue and to minimise surgical trauma and postoperative morbidity has prompted the consideration of alternative graft sources [[101,](#page-13-0) [106\]](#page-14-0). One such alternative is allogenic tissue—tissue from a cadaver. The use of allografts has risen tremendously over the past decade [\[88](#page-13-0)]. However, allograft ACLR carries its own problems, and surgeons are therefore faced with a dilemma when deciding which type of graft to use. The following review will debate the use of allograft versus autograft for ACLR. The review shall

- 1. Discuss autograft ACLR including the results of experimental and clinical trials.
- 2. Discuss allograft ACLR including the advantages and disadvantages, methods of graft preparation and the results of experimental and clinical trials.
- 3. Critically analyse the results of clinical trials comparing the outcome of allograft versus autograft ACLR.
- 4. Identify gaps in the literature where further research is required.

Autograft ACLR

In 1917, Hey-Groves [[41\]](#page-12-0) presented the first published report of a procedure to reconstruct the ACL, using a tethered fascia lata graft. Almost 90 years later, autogenous grafts remain the most popular method for ACLR [\[10](#page-11-0)]. The most common sources of autograft are the patella tendon (PT) and hamstring tendons (combined semitendinosus and gracilis tendons—STG). Other possibilities include the iliotibial band (ITB) and quadriceps tendon [[40\]](#page-12-0).

Noyes et al. [[81\]](#page-13-0) demonstrated the strength of patellar tendon autograft to be 159–168% of native ACL and this led to a surge in the popularity of bone-patellar tendonbone (BPTB) autograft surgery. The development of improved fixation techniques and the publication of numerous clinical studies solidified the role of this method in our armamentarium. Consequently, BPTB autograft has evolved to be, for many, the 'gold standard' [\[21](#page-11-0), [36,](#page-12-0) [39,](#page-12-0) [50,](#page-12-0) [112\]](#page-14-0) and it is the most common surgical method for treating ACL deficient knees [[39,](#page-12-0) [57,](#page-12-0) [85](#page-13-0)]. Advantages of the autogenous BPTB include: (1) it has the highest tensile strength of the available tendons around the knee; (2) it permits bone-to-bone union at the insertion sites thus producing greater and earlier fixation strength; (3) its use does not sacrifice a significant stabiliser of the knee [[25,](#page-11-0) [36,](#page-12-0) [61\]](#page-12-0).

Clinical studies have shown good long-term results using BPTB autografts [[1,](#page-11-0) [17](#page-11-0), [27,](#page-12-0) [50,](#page-12-0) [53](#page-12-0), [54,](#page-12-0) [82](#page-13-0), [83,](#page-13-0) [112\]](#page-14-0). Despite this success, BPTB autograft ACLR is also associated with troublesome donor-site morbidity, including tenderness, anterior knee pain, disturbance in anterior knee sensitivity, and the inability to kneel in approximately 40–60% of patients [[57](#page-12-0)]. Additional complications associated with BPTB autograft include quadriceps weakness, arthrofibrosis, patellar tendonitis or rupture, patellar fracture, and patella infera syndrome [\[2](#page-11-0), [13,](#page-11-0) [71,](#page-13-0) [72](#page-13-0), [94](#page-13-0), [113\]](#page-14-0). These complications are discussed in detail below.

Complications of patellar tendon autograft ACLR—studies without control groups

Bonamo et al. [[13\]](#page-11-0) reported two cases of rupture of the remaining patellar tendon at three-and-a-half months and eight months after use of the central third of this tendon for ACLR. Two cases of much later patellar tendon ruptures

after removal of its central third for ACLR were described by Marumoto et al. [\[71](#page-13-0)]. These occurred more than 3 and 6 years, respectively, after ACLR. The authors postulated that the remaining tendon may have been devascularised during graft procurement, thus, a slow, silent avascular degeneration might have ensued, predisposing otherwise healthy young patients to rupture their patellar tendon.

McCarroll [[72\]](#page-13-0) reported a transverse patellar fracture during a golf swing, 6 months after ACLR using a patellar tendon. The author suggested that this was possibly a stress fracture due to the decreased vascularity of the patella following surgery.

While complications, such as patellar fracture and patellar rupture, are rare, anterior knee pain and patellofemoral joint (PFJ) problems seem to be the most frequent complications following patellar harvest [\[59](#page-12-0)]. The incidence of PFJ problems after BPTB ACLR has been reported in the literature to vary between 1.5 and 58% [\[2](#page-11-0)].

Sachs et al. [[94\]](#page-13-0) reported that the most prevalent complications following ACLR were quadriceps weakness, flexion contracture, and PFJ pain and that there was an intimate relationship between these three factors. However, this study is now somewhat dated as the patients had a 3-week postoperative period in which the knee was immobilised in a cast at 30° of flexion. Presently, more aggressive rehabilitation protocols emphasising early range of motion and strengthening undermine interpretation of studies performed before such postoperative management [\[96](#page-13-0)]. Advances in surgery since this study was published further undermine its results. Also, there was no control group and thus the findings may have been due to factors other than the patellar tendon harvest. Several other studies have reported significant donor site morbidity following ACLR with a BPTB autograft [\[2](#page-11-0), [92\]](#page-13-0).

Kleipool et al. [[59\]](#page-12-0) studied 33 patients who had undergone ACLR with a BPTB autograft. They found that the position of the patella was lower after ACLR compared to preoperative values. They also found that 18 out of the 33 patients complained about anterior knee pain after surgery. Change in patellar tendon length could be implicated as contributing toward anterior knee pain by altering the area of PFJ contact and pressure. Van Eijden et al. [[108\]](#page-14-0), using a mathematical model, found that shortening the patellar tendon increased proximal tendofemoral compression and anterior translation force on the tibia near full extension, and meant that a greater muscle force was needed to generate the same extensor moment toward terminal extension.

Other studies have reported patellar tendon shortening following ACLR with a BPTB autograft [\[16](#page-11-0), [26](#page-12-0), [82](#page-13-0)]. O'Brien et al. [\[76](#page-13-0)] reported that the remaining tendon shortened by an average of 20% in more than half their patients and that this shortening was associated with anterior knee pain. Burks et al. [\[16](#page-11-0)] also reported corroborating evidence of patellar tendon shortening in a canine model. An average of 10% shortening was measured at 6 months after harvesting the patellar tendon in 25 canines, despite there having been no intraarticular surgery and no immobilisation. They also reported a significant increase in cross-sectional area of the operated tendon compared to controls, with poor organisation of collagen across the entire tendon. The failure load of the operated tendon was 60% of controls at 6 months.

In contrast to the above findings, Shaffer et al. [\[96](#page-13-0)] reported that no patients demonstrated a statistically significant change in their patellar tendon length following ACLR using a BPTB graft. They concluded that BPTB autograft ACLR does not result in patellar tendon shortening when combined with an early aggressive rehabilitation programme. Indeed, it could be argued that the studies which demonstrated patellar tendon length change [\[16](#page-11-0), [82](#page-13-0)] have several limitations and need to be interpreted with care. O'Brien et al. [\[82](#page-13-0)] suggested that patellar tendon length change was related to patellar pain, but they also acknowledged that 'symptoms are probably multi-factorial'. It is possible that one of these factors was the 6 weeks cast immobilisation that the patients experienced postoperatively. Thus, the interpretation and relevance of this study is questionable as current rehabilitation protocols tend to favour a more aggressive approach emphasising early range of motion and strengthening. Criticisms can also be made of the canine study by Burks et al. [\[16](#page-11-0)]. For example, the contralateral limb was used as a control, introducing potential error in assuming equal right and left hindleg patellar tendon lengths. In addition, there was no specific rehabilitation protocol, only unrestricted weightbearing, which differs from the very structured protocol used in human subjects. Finally, perhaps the detected shortening was a consequence not so much of the harvest itself but of the surgical trauma to the limb. Other control groups with different types of surgery would provide useful additions to this important study [\[96](#page-13-0)]. In addition, D'Agata et al. [\[25](#page-11-0)] reported, by using pressure-sensitive film and isometric quadriceps forces, that harvesting the central third of the patellar tendon made no difference to the PFJ contact area and pressure in five cadaver knees. This implies that anterior knee pain is unlikely to be caused by a change in PFJ contact area and pressure secondary to patellar tendon shortening due to patellar tendon harvest. However, this study was conducted immediately after graft harvest and therefore it does not account for the possibility of shortening over time due to excessive scar tissue formation, as reported by Burks et al. [[16\]](#page-11-0).

In a more recent prospective study, Muellner et al. [[76\]](#page-13-0) investigated patellar height changes following ACL surgery in 114 patients at a mean follow-up of 22 months. Fifty-two patients (group A) were treated by multiple suture repair, 27 patients (group B) underwent acute ACLR and 35 patients (group C) underwent ACLR greater than 6 weeks after injury with a BPTB autograft. Patellar vertical height ratios (patellar tendon length/patella length) were evaluated preoperatively, 6 months postoperatively and at follow-up. They reported that the change in patellar height was the same in all groups. A significant shortening of the patellar tendon occurred in 30% of patients and the shortening process was finished at 6 months. Anterior knee pain was present in 27% of patients and occurred significantly more after patellar tendon grafting but did not correlate significantly with change in patellar height. The postoperative protocol included continuous passive motion immediately after surgery, therefore any change in patellar tendon length is not easily attributed to immobilisation. However, there was inconsistency, as surgery was performed by five different surgeons. Also, a ligament augmentation device was used on each occasion. It would have been useful to have included a control group, without the ligament augmentation device, to determine any possible effect it may have had.

This study has several points worthy of further discussion. First, it appears that it is not only knee surgery involving patellar tendon harvest that can cause patellar tendon shortening, as demonstrated by the shortening experienced in group A and the occurrence of patella baja after tibial nailing for fractures. Second, anterior knee pain occurred significantly more in groups B and C and the pain was chiefly localised at the patellar apex, where the bone block was removed. Also, 15% of patients in group A suffered anterior knee pain, thus it cannot be explained solely by donor site morbidity. Third, because there was no correlation between patellar tendon shortening and anterior knee pain, one could argue that the shortening is just a coincidence of ACLR. However, without the aggressive rehabilitation employed in this study, shortening may have been more pronounced thus creating more potential to cause anterior knee pain. The authors concluded that it is plausible that shrinkage of the patellar tendon could influence the PFJ and alter the alignment and pressure distribution. However, further studies are required to determine whether anterior knee pain after ACLR is secondary to changes in PFJ alignment.

Finally, Jarvela et al. [\[52](#page-12-0)] reported a high incidence of patellar tendon shortening following autograft BPTB ACLR at an average follow-up of 7 years. Moreover, the amount of shortening was correlated with the severity of PFJ osteoarthritis. However, this study involved immobilisation of the knee in 35° of flexion for 2 weeks after surgery.

In the literature, several imaging studies show that the patellar tendon at the donor site does not normalise in the short- or mid-term after harvesting its central third.

Numerous MRI studies have shown that the thickness of the patellar tendon increases, at least up to 2 years postoperatively [[11,](#page-11-0) [12,](#page-11-0) [24,](#page-11-0) [67,](#page-13-0) [73,](#page-13-0) [78\]](#page-13-0). Svensson et al. [[107\]](#page-14-0) demonstrated that the patellar tendon had not normalised 6 years after harvesting its central third. Jarvela et al. [[51\]](#page-12-0) reported damage to the morphology of the patellar tendon up to 10 years after ACLR with a BPTB autograft. Thirtyone patients were studied and they reported intratendonous calcification in 9 patients, hypoechoic lesions in 20 patients and peritendonous changes in 1 patient. Only three patients had no changes in the harvested patellar tendon, but no abnormalities were visible in any of the contralateral patellar tendons. All the harvested patellar tendons were significantly thicker than the contralateral patellar tendons. Therefore, the authors suggest that the harvested patellar tendon should not be considered as a graft option if revision ACLR is required [[51\]](#page-12-0).

When a standard anterior incision technique is used to harvest the central third of the patellar tendon, the infrapatellar branch of the saphenous nerve running close to the tibial tubercle is in danger [[55\]](#page-12-0). The ability to kneel after ACLR has been shown to correlate with the loss of, or disturbance to, anterior knee sensitivity [\[57](#page-12-0)]. Kartus et al. [\[55](#page-12-0), [56\]](#page-12-0) presented a different harvesting technique to reduce the risk of injury to the infrapatellar nerve. This 'subcutaneous two-incision' graft harvesting technique was shown in clinical trials to produce less loss of sensitivity and less knee-walking discomfort [[55\]](#page-12-0).

Complications of patellar tendon autograft ACLR—studies with control groups

The actual morbidity caused by harvest of the patellar tendon autograft is debatable, as the majority of studies reporting donor site morbidity have no control group. It is therefore not clear if results are due to patellar tendon donor site morbidity or the inherent complications of ACL surgery. To distinguish between these two possibilities, Rubinstein et al. [\[93](#page-13-0)] studied 20 patients who had their patellar tendon graft taken from their contralateral knee. Quadriceps strength in the donor leg averaged 69% of preoperative values at 6 weeks and returned to 93% at 1 year and 95% at 2 years. All patients regained full range of movement in the donor knee by 3 weeks and at followup there was no patella baja or PFJ pain. The authors believe that arthrofibrosis and patellar length change may be mistakenly attributed to the patellar tendon autograft when instead they may be more closely related to postoperative rehabilitation. They concluded that morbidity of an isolated patellar tendon autograft appears to be of short duration and largely reversible. However, their postoperative rehabilitation regime was very aggressive including full weightbearing from day one. This approach is not possible following ipsilateral harvest as the graft needs to be protected. Therefore donor site morbidity following ipsilateral patellar tendon harvest may be more significant due to its combined effect with the inevitable restrictions in mobilisation. Thus, the results of Rubinstein et al. [\[93](#page-13-0)] should not be generalised to ACLR with ipsilateral patellar tendon harvest.

Hamstring autograft

Hamstring tendon or STG autograft has been advocated as a means to lower the incidence of donor site complications. Although there is still the possibility of anterior knee pain with this type of graft, the incidence of this is much lower than following BPTB harvest. There is also a smaller scar following STG harvest. The disadvantage of STG grafts is the longer period required for soft tissue-to-bone fixation and the concern that the initial tensile strength of the graft may not be sufficient to achieve postoperative stability. This originates from the findings of Noyes et al. [\[81](#page-13-0)], who demonstrated that the semitendinosus and gracilis tendons had only 70 and 49% of the strength of the native ACL, respectively. Although harvest site morbidity is believed to be less with STG autografts, studies have shown potential rehabilitative and long-term effects from weakening knee stabilisers and hip extensors [[28,](#page-12-0) [42](#page-12-0), [70\]](#page-13-0). There is also the possibility of Saphenous nerve neuroma and neuralgia following STG harvest [[10](#page-11-0)].

A recent resurgence in the popularity of hamstring tendons has been fuelled by techniques allowing the hamstring graft to be doubled or even quadrupled, thus improving the initial tensile strength and increasing the cross-sectional diameter of the graft. In addition, new hamstring fixation techniques have evolved to match and even exceed the initial pullout strength of the BPTB graft fixed with interference screws [[8\]](#page-11-0).

Several non-prospective studies have looked specifically at the effect of STG and BPTB autograft on muscle strength. Carter et al. [\[19](#page-11-0)] compared quadriceps and hamstring isokinetic results 6 months postoperatively in patients having BPTB or STG autograft ACLR. Although quadriceps strength was less in the BPTB group and hamstring strength was less in the STG group, the differences were not statistically significant. Heimstra et al. [\[42](#page-12-0)] investigated knee strength in more detail by assessing different speeds and types of contraction at different knee angles at a minimum follow-up of 1 year. They reported deficits localised to specific contraction types and ranges of movement between ACL and control groups that were dependent upon autograft donor site. The BPTB group showed greater deficits in knee extension strength and the STG group showed greater deficits in knee flexion strength.

Within the last two decades, several controlled studies have been performed specifically comparing BPTB and STG autograft for ACLR. Because different techniques, fixation devices, assessment tools and follow-up periods were used, it is necessary to evaluate each study for the quality of its results [\[99](#page-13-0)].

In a prospective randomised trial, Marder et al. [[70\]](#page-13-0) evaluated 80 patients with chronic knee laxity due to a torn ACL. Forty patients underwent ACLR with an autogenous BPTB graft and 40 patients underwent ACLR with an autogenous doubled STG graft. Seventy-two of 80 patients were evaluated at a mean follow-up of 29 months post-op. Subjectively, 13 patients (8 BPTB, 5 STG) complained of slight pain with activity (but the pain did not impair their ability to perform daily activities). Four patients (3 BPTB, 1 STG) complained of moderate pain with activity (which did inhibit their ability to perform daily activities). However, this difference was not statistically significant. There was also no significant difference between groups with respect to subjective score according to the Zairns-Rowe subjective rating score. Objectively, mean KT-1000 scores (anterior tibial displacement side-to-side difference at 20 lbs of load) were 1.6 ± 1.4 mm for the BPTB group and 1.9 ± 1.3 mm for the STG group. Thirty-two of 37 BPTB patients (86%) and 26 of 35 STG patients (74%) had an injured-to-uninjured knee difference of 2 mm or less. These differences in knee laxity were not statistically significant. One patient with a BPTB graft had a 15° loss of full extension and three patients (3 BPTB, 1 STG) had a $5^{\circ}-10^{\circ}$ loss of full extension as compared with the uninvolved knee. Again, these differences were not found to be statistically significant. Marder and colleagues did find a statistically significant weakness in peak hamstring torque at 60 \degree /s in the STG group ($P < 0.025$). The authors failed to describe in any detail their methods for measuring muscle strength. In addition, only 40 patients (50%) were tested for muscle strength and they did not mention how these patients were chosen. Furthermore, they did not mention who conducted the testing, nor if they were blinded to the graft type. Care should also be taken when interpreting this study as patients had chronic laxity, although the time between injury and surgery was not given.

In another prospective randomised trial, Aglietti et al. [\[3](#page-11-0)] compared 30 patients who had undergone ACLR using a four-strand STG autograft with 30 patients who had undergone ACLR using a BPTB autograft. After 28 months of follow-up return to sport participation was more frequent in the BPTB group compared to the STG group (80 vs. 43%, $P < 0.01$). A minor extension loss (<3°) was more frequent in the BPTB group (47 vs. 3%, $P < 0.001$). Recurrent giving way was present in only one knee, from the STG group, but this difference was not statistically significant. A KT-2000 side-to-side difference in anterior displacement >5 mm at 30 lbs of load was present in 13% of BPTB patients and 20% of STG patients. Patellofemoral crepitation developed in 17 and 3% of the two groups, respectively. These differences were not statistically significant. As with the previous study by Marder et al. [[70\]](#page-13-0), Aglietti et al. [\[3](#page-11-0)] looked at patients with chronic knee laxity with an interval between injury and surgery of at least 6 months.

O'Neill [\[83](#page-13-0)] evaluated 127 patients following ACLR after a mean duration of follow-up of 42 months. In the prospective study, patients were randomly assigned into one of three groups. Group I included 42 patients who had a two-incision reconstruction using an autogenous STG graft. Group II included 40 patients who had a two-incision reconstruction using an autogenous BPTB graft. Group III included 45 patients who had a single-incision reconstruction (endoscopic technique) using an autogenous BPTB graft. The significant findings were that patients in group II returned to a greater level of athletic activity $(P < 0.02)$ and that a higher percentage of patients in this group had a side-to-side difference of 3 mm or less on testing with the KT-2000 than in the other two groups $(P < 0.08)$. However, the over-all outcome of all three groups did not differ significantly according to the IKDC scale. Muscle strength deficit between groups was not significantly different and the authors concluded that there were no clear advantages to any of the three techniques. It is worth noting that the three aforementioned studies [[3,](#page-11-0) [70,](#page-13-0) [83](#page-13-0)] involved different fixation methods for the two types of graft.

In a prospective sequential trial, Corry et al. [[23\]](#page-11-0) compared autogenous STG and BPTB ACLR in 167 patients 2 years after surgery. Importantly, interference screw fixation was used for both BPTB and STG grafts. Results showed no significant differences between groups in terms of ligament stability, range of motion and general symptoms. Kneeling pain after reconstruction with the STG graft was significantly less common than with the BPTB graft (6 vs. 31%). They also reported less thigh atrophy in the first year in the STG group, suggesting early quadriceps recovery, but the difference was not statistically significant at 2 years. Knee laxity measured with the KT-1000 was found to be slightly higher in the female patients in the STG group compared to the BPTB group. Care should be taken when interpreting this study due to the potential bias resulting from its sequential design.

More recently, Ejerhed et al. [[29\]](#page-12-0) reported a prospective randomised study comparing BPTB and STG autografts, both with interference screw fixation. After a 2 year follow-up the study found no statistically significant difference between the groups in terms of Lysholm score, Tegner

activity level, KT-1000 measurements, single-leg hop test, IKDC classification results and anterior knee pain. Patients in the STG group had a significantly better ability to walk on their knees.

In a similar study, Shaieb et al. [\[97](#page-13-0)] reported a prospective randomised study comparing BPTB and STG autografts, both with interference screw fixation. Seventy patients were followed up at 2 years. Patients in the BPTB group experienced significantly more PFJ pain and less range of movement.

Two meta-analyses have been completed to summarise the numerous studies that have compared BPTB and STG autograft over the last two decades. In the most recent meta-analysis, Freedman et al. [[34\]](#page-12-0) pooled data from 34 studies including 1,976 subjects. The study found significantly lower rates of graft failure, less laxity and higher patient satisfaction in the BPTB group. However, there was a higher incidence of anterior knee pain in the BPTB group.

In a meta-analysis performed 2 years earlier, Yunes et al. [\[114](#page-14-0)] pooled together data from four studies, including 411 subjects. The authors reported less laxity in the BPTB group and a higher rate of return to pre-injury level of activity compared to the STG group.

After reviewing this evidence it is clear that the controversial debate regarding patellar versus hamstring autograft is still unresolved. Although some of the bestdesigned prospective randomised studies show no difference between the two grafts, it can be argued that the subject size is too small and the follow-up is too short. Meta-analysis alleviates the problem of a small sample size yet it is a design which has problems of its own, notably the differences between the grouped studies. Although patients who must kneel a lot may wish to consider harvest site morbidity, there have yet to be data that disprove the BPTB autograft as the gold standard autograft in ACLR [[99\]](#page-13-0).

Allograft ACLR

Although BPTB autograft has been considered the 'gold standard' for ACLR there are many potential advantages to the use of allograft tissue, the most notable being elimination of donor site morbidity. Further advantages include a shorter operation time with smaller incisions and consequently a cosmetically better outcome [[18\]](#page-11-0). Also, there is no size limitation with an allograft and it may be more appropriate for revision surgery, for multiple ligamentous injury or in the presence of patellar baja. The incidence of postoperative arthrofibrosis is also lower [\[88](#page-13-0)]. There are, however, risks associated with the use of allograft tissue, most notably the prospect of disease transmission, both viral and bacterial. There is also the possibility of a host immune response against the donor tissue, delayed incorporation [\[47](#page-12-0)] and bone tunnel enlargement [\[31](#page-12-0)]. Other concerns include the alteration of mechanical properties secondary to sterilisation and preparation of the graft, increased postoperative traumatic rupture rate, long-term results and graft cost. As a result of these drawbacks, many authors have condemned the use of allografts for ACLR. Table [1](#page-6-0) summarises these advantages and disadvantages.

Allograft sources

To date, the most frequently used allograft materials for ACLR include patellar ligament, Achilles tendon and fascia lata [[99\]](#page-13-0). Other potential allograft sources have been biomechanically tested and produced favourable results [\[84](#page-13-0)]. Pearsall et al. [\[84](#page-13-0)] found that the average failure loads for doubled tibialis anterior, tibialis posterior, and peroneus longus were 3,412, 3,391, and 2,483 N, respectively. These results compared well to historical data, equalling or exceeding nearly all currently described ACL graft sources.

Lawhorn et al. [[63\]](#page-13-0) advocate the use of soft tissue allografts such as tibialis anterior tendon or doubled STG rather than allografts with bone plugs such as BPTB and Achilles tendon. This is mainly due to the slow incorporation of the allograft bone plug, the greater cross sectional area available with the soft tissue allograft and the fact that soft tissue grafts are more readily available since each donor provides six soft tissue grafts but only four bone plug grafts.

Cost comparison

To the best of our knowledge, comparison of the economic cost associated with allograft and autograft ACLR has only been addressed by one study. Cole et al. [\[22](#page-11-0)] studied 122 patients undergoing ACLR. They reported that the average hospital charge for the 37 patients who received a fresh frozen Achilles tendon allograft was \$1,072 cheaper compared to the 86 patients who received a BPTB autograft $(P < 0.001)$. Autograft costs were significantly higher for the surgery centre $(P < 0.001)$, day hospital $(P < 0.001)$, anesthesia ($P < 0.001$), radiology ($P < 0.001$), and pharmacy. These categories represent costs associated with operating room time, post operative observation/admission, anaesthesiologists' charges, charges for fluoroscopy, and anaesthetics/analgesics, respectively. It would therefore appear that the extra cost of the allograft tissue itself is offset by the savings made in other areas of hospital care. Consequently, instead of being a disadvantage, the cost of allograft ACLR appears to be less than that of an autograft and must therefore be considered an advantage. However,

Table 1 Summary of advantages and disadvantages of allograft anterior cruciate ligament reconstruction (from Prokopis and Schepsis [[80](#page-13-0)])

Advantages	Disadvantages
1. No donor site morbidity	1. Possibility of disease transmission
2. Cosmetically better outcome	2. Host immune response
3. No size limitation	3. Delayed incorporation
4. Shorter operation time	4. Local bone resorption
5. Decreased incidence of postoperative knee stiffness	5. Cost

care must be taken when generalising these results as they represent only one surgeon in one country.

Preparation of allograft tissue

There are three ways by which ACL grafts are prepared. The most common is deep fresh-freezing. Acceptance or rejection of allograft tissue is mainly determined by class II major histocompatibility proteins on the donor cells. Research indicates that such deep freezing reduces graft antigenicity by destroying these cells [\[6](#page-11-0)]. This method has no effect on the strength of the graft [\[46](#page-12-0), [77\]](#page-13-0) and allows storage up to 6 months.

The second method, freeze-drying, allows for storage up to 2 years. This procedure also kills the cells carrying major histocompatibility proteins thus reducing graft antigenicity [[35\]](#page-12-0). Freeze-drying has no effect on graft strength if it is reconstituted for 24 h [\[88](#page-13-0)]. The third method is cryopreservation, which has no known advantage over fresh-freezing but incurs a considerably higher cost.

Since freezing alone does not kill the HIV virus the graft can either be harvested sterilely or harvested non-sterilely making secondary sterilisation mandatory. The two main methods of secondary sterilisation are ethylene oxide and gamma irradiation. Ethylene oxide is an industrial fumigant that has been widely used to sterilise medical equipment. Anterior cruciate ligament allografts sterilised with ethylene oxide have shown poor results [[48,](#page-12-0) [49,](#page-12-0) [90](#page-13-0), [105](#page-14-0)]. Roberts et al. [\[90](#page-13-0)] showed a 53% failure rate in patients with ethylene oxide sterilised allografts with 25% having persistent effusions. There was cyst formation around the tunnels in 36% of patients, while 22% had complete graft dissolution discovered at revision surgery. Allografts treated with ethylene oxide cannot be recommended for ACLR [\[88](#page-13-0)]. Gamma irradiation studies have indicated that there is a dose-dependent effect of irradiation on biomechanical properties causing a loss of graft strength [\[32\]](#page-12-0). The optimal dose of irradiation remains unclear since low doses do not completely eliminate the risk of viral transmission but high doses have a deleterious effect on mechanical properties of the graft. Doses as low as 4 Mrads [[89\]](#page-13-0) and 2.5 Mrads [[68\]](#page-13-0) have shown deleterious effects on allograft strength. It is therefore recommended by many authors to use sterile harvesting and thus avoid the need for secondary sterilisation though this is no longer advocated by tissue banks [\[88](#page-13-0)].

Disease transmission with allografts

The main concern here is in regards to viral pathogens such as HIV. Buck et al. [\[15](#page-11-0)] conducted the classic study on this issue. They estimated that the risk of procuring bone from a donor who is infected with HIV and has been thoroughly screened is one in 1,667,000. If screening techniques were not followed, the risk theoretically increases to one in 161. It must be noted that this study was done before antigen testing was available. For comparison, the risk of HIV transmission in a unit of blood is one in 200,000–800,000 [\[7](#page-11-0)]. As tests have become more sensitive, the risk has further reduced but there is still a 'window' period after infection with HIV when infected donors may not be detected. There have also recently been cases of polymicrobial infections (septic arthritis) following allograft ACLR [\[5](#page-11-0)]. We clearly do not know all there is to know about allograft material and the potential for disease transmission. It is incumbent on us to ensure that diseases (including those we do not already know about) are not transmitted with the allograft material [\[9](#page-11-0)].

Experimental and clinical trials of allografts

Allografts have been used successfully for ACLR in animal studies [[6,](#page-11-0) [102](#page-14-0)] and clinical results of allograft reconstruction have also been encouraging [\[4](#page-11-0), [43](#page-12-0), [44](#page-12-0), [65,](#page-13-0) [74,](#page-13-0) [79,](#page-13-0) [80](#page-13-0), [103,](#page-14-0) [110](#page-14-0)]. Noyes et al. [\[79](#page-13-0)] reported an excellent or good result in 89% of 47 allograft ACLRs at an average follow-up of 40 months. The allogenic tissue was either a BPTB or fascia lata graft. In a continuation of the same study, with a 5–7 year follow-up, Noyes et al. [\[80](#page-13-0)] reported no significant difference in knee anterior laxity, PFJ crepitus, pain, jumping score or the overall knee rating over the time period studied. Satisfactory results have also been reported following allograft ACLR in patients with chronic ACL deficiency using BPTB allografts [[44\]](#page-12-0), in selected skeletally immature athletes using Achilles tendon or fascia lata allografts [[4,](#page-11-0) [37](#page-12-0)] and in patients over 40 years of age using Achilles tendon or fascia lata allografts [\[60](#page-12-0)].

The biologic phases of incorporation of allografts have been studied in both animal and human biopsies [\[45](#page-12-0)]. Some

studies [[47,](#page-12-0) [77,](#page-13-0) [69\]](#page-13-0) have suggested suboptimal healing of allografts compared with autografts. In a study of 46 goats, Jackson et al. [[47\]](#page-12-0) reported that allografts had a slower rate of biologic incorporation, a prolonged inflammatory response, and a greater decrease in implantation structural properties compared with autogenous ACLR at 6 months. Other studies [[86,](#page-13-0) [91,](#page-13-0) [100,](#page-13-0) [101](#page-13-0)] have demonstrated that allografts undergo remodelling similar to autografts. Shino et al. [[101\]](#page-13-0) concluded that the allograft was viable by 6 months, and at 12 months the graft resembled the control ACL with a single layer of synovial lining cells. From 18 to 55 months the grafts were stable with well-arranged collagen bundles exhibiting a good crimp pattern, spindle shaped nuclei and normal cellularity. The general conclusion is that, like autografts, allografts become revascularised and viable after implantation but their rates of incorporation and remodelling are slightly slower [\[33](#page-12-0)].

Suboptimal healing of allogenic tissue may also be suggested by greater bone tunnel enlargement observed after allograft ACLR in humans [\[31](#page-12-0), [66\]](#page-13-0). Fahey et al. [[31\]](#page-12-0) demonstrated that, 1 year after ACLR, the average tunnel enlargement was 1.2 mm for allograft BPTB patients compared with 0.26 mm for autograft BPTB patients $(P < 0.05)$. The reason for this finding remains unknown but it may be related to host immunogenicity. There was no difference in clinical outcome between groups.

More recently, tissue engineering has been explored to develop cell-seeded patellar tendon allografts. In this approach, intrinsic cells are removed from the graft to reduce antigenicity and then the graft is seeded with extrinsic cells to improve ligamentization. Cartmell et al. [\[20](#page-11-0)] reported that this method removed 70–90% of the intrinsic patellar cells without changing the mechanical properties of the graft. The authors concluded that they had created viable tissue-engineered grafts, which could potentially be used for ACLR.

Clinical trials comparing allograft and autograft ACLR

There are 11 published clinical studies evaluating allograft and autograft use in ACLR and these are summarised in Table [2](#page-8-0). The findings vary greatly and these discrepancies are due in large part to the variety of tissues used and the tremendous variation in surgical and postoperative protocols. Most clinical studies comparing autograft to allograft show little difference in long-term outcome [[39,](#page-12-0) [62,](#page-12-0) [64,](#page-13-0) [85,](#page-13-0) [87,](#page-13-0) [95,](#page-13-0) [98,](#page-13-0) [104](#page-14-0)]. However, some studies have reported an increased postoperative traumatic rupture rate in allograft groups [[21,](#page-11-0) [106](#page-14-0)] and other authors have recommended against allografts due to a reported deterioration in stability over time [\[109](#page-14-0)]. Of special note is the fact that all the comparative studies that have described the graft source have used BPTB tissue and so these findings may not be applicable to comparisons between STG allo- and autografts.

The ideal study would randomly assign patients to receive either an autograft or allograft. Random assignment would minimise naturally occurring differences between groups and improve the ability to implicate surgical treatment type to differences found between groups. However, a true prospective, randomised clinical trial of autograft versus allograft is difficult because patients are part of the decision to use or not use allograft tissue. Since none of the following studies [\[21](#page-11-0), [39,](#page-12-0) [62](#page-12-0), [64,](#page-13-0) [85](#page-13-0), [87,](#page-13-0) [95](#page-13-0), [98,](#page-13-0) [104](#page-14-0), [106,](#page-14-0) [109](#page-14-0)) were prospective randomised trials, the ability to state with certainty that the observed results were solely due to graft type is compromised. Also, none of these studies used a blinded assessment. In addition, there are four types of bias resulting from the design of investigations of ACLR as described by Frank [\[33](#page-12-0)]. First, although the system developed by the International Knee Documentation Committee (IKDC) is beginning to be used more routinely, several different scoring systems are still being used to report the outcome of ACLR. This problem, known as detection bias, confounds the ability of the reader to interpret results. The pooling of patients who have fundamentally different prognoses, which is known as susceptibility bias, is another source of confusion. Also, performance bias results from pooling the results of different surgeons and rehabilitation protocols, and transfer bias results from losing patients to follow-up.

In general, use of allograft tissue appears to be much more favourable than unfavourable [[88\]](#page-13-0). Early studies have shown good clinical results up to 3 years after allograft reconstruction. Shino et al. [[104\]](#page-14-0) evaluated 92 subjects who had undergone unilateral ACLR 18–36 months previously. Group one consisted of 47 patients who received a fresh-frozen allograft (type of allograft was not specified) and group two consisted of 45 patients who received a BPTB autograft. The anterior laxity side-to-side difference was statistically less for the allograft patients. Also, the knee extensor torque at 60° s⁻¹ was significantly better in the allograft group. The authors concluded that the allograft procedure was advantageous over the BPTB autograft in terms of better restoration of anterior stability and quadriceps muscle strength. Limitations of this study include the fact that only patients who were rated as successes after ACLR were included. Three patients (one allograft, two autografts) were excluded due to flexion contractures and three more patients were excluded due to traumatic rupture of the graft, but the authors failed to mention which type of graft these patients had received. By including results taken at 18 months, this study does not examine the long-term outcome of ACLR, which may differ from the short-term results. Perhaps the most

Author	\boldsymbol{N}	Follow-up	Main findings
Lephart et al. $[64]$	33	12,24/12	No significant differences in strength/function.
Saddemi et al. [95]	50	24, 52, 104/ 52	No significant differences in perioperative morbidity. Two persistent effusions in the allograft group, two flexion contractures in the autograft group.
Shino et al. $[104]$	92	$18 - 36/12$	Better anterior stability and recovery of quadriceps strength at $60^{\circ}/s$ in the allograft group.
Harner et al. [39]	90	45/12	No significant differences except loss of terminal extension in the autograft group (not clinically significant).
Stringham et al. [106]	78	34/12	No significant difference in subjective results or effusions, range of movement, atrophy, or tenderness. Trend toward better stability in the autograft group and better quadriceps strength in the allograft group but this was not statistically significant. Four traumatic ruptures in the allograft group.
Shelton [98]	60	3,6,12,24/12	No significant differences in objective outcome measures.
Victor $[109]$	73	6,12,24/12	Greater anterior translation in autograft group at 6 and $12/12$ but at $24/12$ the allograft group showed more anterior translation. Greater quadriceps strength in the allograft group at 6 and $12/12$ but greater in the autograft group at $24/12$. Re-rupture in three allografts.
Peterson [85]	60	3,6,12,24,63/12	Equivalent patient satisfaction and objective results. Greater loss of extension in the autograft group but not clinically significant.
Chang $[21]$	79	$33 - 40/12$	No difference in subjective scores, anteroposterior stability, crepitus or patello femoral pain. More allografts had flexion deficits and there were three cases of traumatic rupture in the allograft group.
Kustos et al. $[62]$	79	38/12	No significant difference in Lysholm, Tegner and IKDC scores. Two traumatic ruptures in the allograft group, one in the autograft group.
Poehling et al. $[87]$	159	Pre-op; 1 and $6/52$; 3 and $6/12$ and annually for 5 years	Similar long term results in both groups. Less pain and better function in allograft group during first year.

Table 2 Clinical trials, in chronological order, comparing allograft and autograft anterior cruciate ligament reconstruction

significant limitation of this study is the fact that groups were not considered comparable in terms of age and activity level since older, less active people tended to prefer the theoretically safer autogenous ACLR. This could be one reason for the better quadriceps muscle strength in the allograft group, therefore the muscle strength data should be cautiously interpreted. The allograft group also included more acute cases and less meniscectomies than the autograft group. Finally, the postoperative protocol involved 5–19 days of cast immobilisation. Consequently, the findings of this study cannot be generalised to contemporary patients who participate in more aggressive rehabilitation.

Lephart et al. [[64\]](#page-13-0) retrospectively compared quadriceps strength and functional capacity in 33 male athletes, 12–24 months after BPTB allograft or autograft ACLR. The authors reported no significant difference between the groups in either of these parameters. Muscle strength was measured using an isokinetic dynamometer, and functional capacity was evaluated based on the results of three specially designed functional performance tests and the hop test. They concluded that harvesting the central third of the patellar tendon does not diminish quadriceps strength or functional capacity in highly active patients who have intensive rehabilitation. This study used a more aggressive

postoperative protocol, which is more in line with current practice. However, the number of patients was small and they were not representative of the wider population as they were highly active, competitive sportsmen. Their youth, combined with their athletic goals, may have lead to a stronger commitment to regain strength and functional capacity. The study was retrospective and the authors did not mention how the sample was selected or the characteristics of each group, including what type of sport they played. It may have been that one group was faster than the other which would have affected the functional performance since all the functional scores were calculated by time. Additionally, surgery was performed by different surgeons.

Saddemi et al. [\[95](#page-13-0)] found no significant difference between autograft or allograft BPTB reconstruction with regard to perioperative morbidity at a minimum follow-up of 2 years. This was measured in terms of hospital stay, swelling, thigh atrophy, laxity, strength, endurance, range of motion, PFJ symptoms, and complications. There was one traumatic rupture in each group. Two patients in the autograft group had flexion contractures but there was no statistically significant difference in prevalence of flexion contracture between the groups. Two allograft patients demonstrated persistent effusions, which were statistically significant $(P < 0.05)$. This study is limited by its retrospective design

and small sample $(n = 50)$. Additionally, there is no subjective or functional evaluation and there is no description of group characteristics such as activity level.

A more recent retrospective study by Harner et al. [[39\]](#page-12-0) concurred with the earlier positive findings concerning allograft ACLR. Sixty-four patients who received nonirradiated allografts were compared to 26 patients who received autografts at 3–5 years after surgery. Detailed symptoms, activity-level, functional outcome, physical examination, and instrumented knee laxity were recorded. No statistically significant differences were found between groups except a higher incidence of loss of terminal extension in the autograft group. This range of motion difference was small [mean loss of extension (allograft vs. autograft): active 1° vs. 3° , passive 1° vs. 4°] and not considered clinically significant. The overall conclusion was that there were no significant clinical differences in outcome between allografts and autografts in ACLR 3– 5 years after surgery. This study had a detailed report of methodology and a long-term follow-up. It also looked at functional and subjective outcome and used the internationally recognised IKDC form. The rehabilitation programme was less aggressive than commonly used today, therefore caution must be used when generalising these results. However, it was a retrospective design, and there was inconsistency in the study as surgery was performed by two surgeons and the postoperative knee brace was locked for some individuals and restricted to varying degrees in others. Also, there was a lack of follow-up of all patients (244 patients underwent the surgery and an attempt was made to contact them all—a total of 88 patients returned for follow-up), and the groups were not identical as 81% of the allograft group had acute injuries compared to only 4% of the autograft group. The authors did not describe what type of autograft or allograft was used.

Shelton et al. [[98\]](#page-13-0) demonstrated good clinical results in groups of 30 BPTB allografts and 30 BPTB autografts, up to 2 years after surgery in a prospective non-randomised trial. No statistically significant differences between groups were shown for swelling, pain, knee anterior laxity, pivot shift test, range of movement, or PFJ pain and crepitation when evaluated at 3, 6, 12 and 24 months postoperatively. The methodology of this study was not reported in much detail and the authors did not explain how they selected their sample. The groups were well matched for most characteristics but there were 24 acute injuries in the autograft group but only 15 in the allograft group. This study did not look at functional outcome or use any validated questionnaires to evaluate subjective scores such as pain. Instead they simply asked the patient to define their pain as one of the following: (1) no pain, (2) minimal and occasional, (3) moderate and daily, or (4) severe and affecting activities of daily living.

However, not all trials have shown allograft reconstruction in a favourable light. A retrospective study by Stringham et al. [[106\]](#page-14-0) compared 47 BPTB autografts to 31 BPTB allografts at an average of 34 months follow-up. They reported no significant differences between groups in subjective results or in joint effusions, knee tenderness, range of motion, patellofemoral scores, laxity, knee muscle strength or quadriceps atrophy. However, two trends of potential significance were identified. Ten percent more autograft patients achieved good to excellent restoration of knee anterior laxity, and concentric peak extension torque results at 60 s^{-1} was found to favour allografts. Statistical power analysis revealed that these trends would not show a significant difference unless more than 1,000 patients were examined. Traumatic ruptures were sustained by four allograft patients at an average of 11 months postoperatively compared with no ruptures in the autograft group. The authors concluded that autograft tissue remained their first choice due to the increased rupture rate in the allograft cohort. This study had well-reported methodology and thoroughly evaluated the outcome of ACLR by assessing functional capacity with the hop test, muscle strength with an isokinetic dynamometer and subjective results using the Lysholm and Tegner knee-rating scales, as well as physical examination findings. However, bias may have resulted from differential follow-up since 29% of autografts and 34% of allografts were lost to follow-up. Although the groups were well matched in demographic details, activity level was not recorded and because the study was not randomised, this or any other variable may have affected the results. For example, it is possible that the allograft group may have been more active, thus predisposing them to re-rupture. In addition, two different surgeons performed surgery and the postoperative rehabilitation was inconsistent.

A prospective study by Victor et al. [[109\]](#page-14-0) of 73 ACLRs using a BPTB autograft or allograft found no statistically significant differences between groups in thigh muscle strength, knee anterior laxity, functional scores, hop test performance, knee swelling or thigh atrophy. However, allograft knees showed slightly greater quadriceps strength and reduced anterior knee laxity at 6 and 12 months. By 24 months this had reversed with a trend towards greater strength and less laxity in the autograft group. KT–1000 evaluation showed a non-significant trend of increasing laxity with time in the allograft group. There were also three occurrences of re-rupture in the allograft group compared to none in the autograft group. They concluded that allografts are not recommended as stability deteriorates with time and, by 2 years, quadriceps strength returns to normal following autograft ACLR.

Two of the more recent clinical trials have found slightly conflicting results. Peterson et al. [[85\]](#page-13-0) conducted a prospective non-randomised trial comparing 30 BPTB allografts and 30 BPTB autografts. Reconstruction was performed by a single surgeon and was followed by early aggressive rehabilitation. Follow-up was long-term, at an average of 63 months. Patients were assessed subjectively with the Lysholm and Tegner scores and objectively including assessment of swelling, pain, range of movement, crepitus and laxity using a KT-1000. They reported no statistically significant difference in any of these parameters other than a greater loss of extension in the autograft group $(2^{\circ}, \text{range } 2^{\circ} - 5^{\circ})$ compared to the allograft group $(1^{\circ}, \text{ range } 0^{\circ}-10^{\circ})$ at the 5-year follow-up. This difference was not considered clinically significant. There was no late stretching of the allografts since those knees that were stable at 2 years remained stable at 5 years. There was one rupture in each group and there were more incision site complaints in the autograft group. The authors concluded that the use of allografts is an acceptable choice for ACL reconstruction. The authors did not explain how the sample of 60 patients was chosen from the 119 ACLRs. There were no exclusion criteria given and the groups were not exactly matched as there were only nine chronic injuries in the autograft group compared to 16 in the allograft group. There is also a possibility of examiner bias since the operative surgeon conducted the objective assessment.

Chang et al. [[21\]](#page-11-0) conducted a retrospective review comparing 46 BPTB allografts and 33 BPTB autografts at a minimum follow-up of 2 years. The grafts were augmented with an ITB tenodesis, and a single surgeon performed surgery. They reported no statistically significant differences between groups in any of the subjective scores or in Lachman and pivot shift tests, knee anterior laxity, crepitus, atrophy or effusion. The allograft group showed a non-significant higher incidence of retropatellar pain and a significantly higher incidence of flexion deficit (although this was only 5°). Three allograft patients had traumatic ruptures compared to none in the autograft group. The authors concluded that results for allograft ACLR were comparable but not as good as the results for autograft ACLR. They believe that autograft BPTB should remain the 'gold standard' but allograft remains a reasonable alternative. However, these results must be interpreted with caution, as the groups were not identical. The allograft group was older, predominantly men and less adherent to rehabilitation. They also had greater preoperative laxity and a higher rate of chondromalacia of the medial tibial plateau. These factors may be partly responsible for the observed higher incidence of flexion deficit and retropatellar pain. The authors did not assess strength or functional capacity and the results cannot be generalised to reconstructions not involving an ITB tenodesis.

In 2004, Kustos et al. [\[62](#page-12-0)] compared BPTB autografts and allografts in a retrospective, non-randomised trial.

They reviewed 79 patients who had ACLR for chronic ACL deficiency. At an average of 38 months follow-up, subjective and functional results were collected by an independent examiner using the Lysholm knee scoring scale, the Tegner activity score, and the IKDC knee ligament evaluation form. The two groups did not differ significantly in terms of subjective or functional results. Two revision ACLRs were needed in the allograft group and one was needed in the autograft group due to traumatic rupture of the ligament. There were no cases of bacterial infections or viral transmissions. Apart from the obvious weakness of this study due to its retrospective, non-randomised design, the authors did not describe the characteristics of each group other than age and gender. They failed to mention if patients had associated injuries or other surgical procedures, if they had exclusion criteria, or how many different surgeons performed the operations. They did not control activity level in any way from 1-week post operatively until follow up, other than advising them not to return to previous levels of activity until the end of the first year post operatively. They did not objectively measure knee laxity with an arthrometer or use any functional tests such as a single leg hop test.

Finally, in the most recent comparative trial, Poehling et al. [\[87](#page-13-0)] conducted a prospective study comparing 41 freeze dried Achilles tendon allografts (without bone block) and 118 BPTB autografts. Patients were evaluated preoperatively and postoperatively at 1–2 weeks, 6 weeks, 3 months, 6 months, and then annually for 5 years. Objective outcome measures included KT-1000 measurements, range of motion, thigh atrophy, and IKDC score. Subjectively, patients completed five questionnaires documenting functional status, pain, and health-related quality of life. Autograft patients reported significantly more pain than the allograft group at 1 week ($P = 0.0006$), 6 weeks $(P = 0.0007)$, and 3 months $(P = 0.0270)$. Fewer activity limitations were reported by allograft patients than autograft patients at 6 weeks $(P = 0.0501)$, 3 months $(P = 0.0431)$, and 6 months $(P = 0.0014)$. Physical functioning was significantly better in the allograft group at 1 week ($P = 0.0016$), 3 months ($P = 0.0494$) and 1 year after surgery ($P = 0.0409$). Knee range of motion was abnormal in fewer allograft patients than autograft patients at 2 years. There was no difference in the overall IKDC evaluation between the groups except at 2 year follow-up $(P = 0.0374)$ when more patients were categorised as ''normal'' and fewer as ''severely abnormal'' in the allograft group. There was no difference in knee laxity on KT-1000 arthrometer testing at all time points post-operatively. Laxity measurements decreased over time in both groups. This was a very thorough study which looked at many different outcome measures. The authors described their methods and exclusion criteria in detail. The main

weakness was that randomisation was not used. In addition, three different surgeons performed the procedures, different graft fixation methods were used, examiners were not blinded and the autograft group had a lower mean age $(P = 0.058)$. Also, attrition of the original patient sample after 2 years, particularly in the autograft group, may have caused some bias.

Conclusions and future research

Despite tremendous success with the use of autogenous tissue for ACLR, reasonable alternatives have been pursued in an attempt to limit the inherent complications of autograft harvest. Theoretically, the use of allograft tissue could match the stability achieved with autogenous tissue and limit donor site morbidity. The literature suggests that an allograft may be a reasonable alternative to an autograft and provides a viable alternative to 'robbing Peter to pay Paul' [9]. It may become increasingly difficult to maintain that 'no harm' comes from using a perfectly normal structure to repair an abnormal one. However, the potential for disease transmission, delayed incorporation, immune reactions, sterilisation and graft preparation problems, bone tunnel enlargement, increased postoperative traumatic rupture rate, graft cost, long-term results, and a paucity of comparison studies have tempered the enthusiasm about using allografts for ACLR [21].

Comparison trials of allograft and autograft have largely shown little difference in outcome between the two but they are limited by the fact that they have not been prospectively randomised. Large, well-controlled, prospective studies reporting the long-term (>5 years) results of ACLR are still needed to define the optimal surgical treatment of the ACL deficient knee [\[36](#page-12-0)].

References

- 1. Aglietti P, Buzzi R, D'Andria S, Zaccherotti G (1992) Longterm study of anterior cruciate ligament reconstruction for chronic instability using the central one-third patellar tendon and a lateral extraarticular tenodesis. Am J Sports Med 20(1):38–45
- 2. Aglietti P, Buzzi R, D'Andria S, Zaccherotti G (1993) Patellofemoral problems after intraarticular anterior cruciate ligament reconstruction. Clin Orthop 288:195–204
- 3. Aglietti P, Buzzi R, Zaccherotti G, De Biase P (1994) Patellar tendon versus doubled semitendinosus and gracilis tendons for anterior cruciate ligament reconstruction. Am J Sports Med 22(2):211–217
- 4. Andrews M, Noyes FR, Barber-Westin SD (1994) Anterior cruciate ligament allograft reconstruction in the skeletally immature athlete. Am J Sports Med 22(1):48–54
- 5. Anonymous (2001) Septic arthritis following anterior cruciate ligament reconstruction using tendon allografts—Florida and Louisiana. MMWR Morb Mortal Wkly Rep 50(48):1081–1083
- 6. Arnoczky SP, Warren RF, Ashlock M (1986) Replacement of the anterior cruciate ligament using a patellar tendon allograft. J Bone Joint Surg Am 68A(3):376–385
- 7. Asselmeier MA, Caspari RB, Bottenfield S (1993) A review of allograft processing and sterilization techniques and their role in transmission of the human immunodeficiency virus. Am J Sports Med 21(2):170–175
- 8. Avery FL (2004) The sports medicine centre ACL graft options [online]. Available: http://www.orthoassociates.com/ ACL_grafts.htm [accessed 20.7.2004]
- 9. Barber FA, McGuire DA, Johnson DH (2003) Should allografts be used for routine anterior cruciate ligament reconstructions? Arthroscopy 19(4):421–425
- 10. Bartlett RJ, Clatworthy MG, Nguyen TNV (2001) Graft selection in reconstruction of the anterior cruciate ligament. J Bone Joint Surg Br 83B:625–634
- 11. Berg EE (1992) Intrinsic healing of a patellar tendon donor site defect after anterior cruciate ligament reconstruction. Clin Orthop 278:160–163
- 12. Bernicker JP, Haddad JL, Lintner DM et al (1998) Patellar tendon defect during the first year after anterior cruciate ligament reconstruction: appearance on serial magnetic resonance imaging. Arthroscopy 14(8):804–809
- 13. Bonamo JJ, Krinick RM, Sporn AA (1984) Rupture of the patellar ligament after use of its central third for anterior cruciate reconstruction. J Bone Joint Surg Am 66A(8):1294–1297
- 14. Boni DM, Herriott GE (2002) Hamstring tendon graft for anterior cruciate ligament reconstruction. AORN J 76(4):610–627
- 15. Buck BE, Malinin TI, Brown MD (1989) Bone transplantation and human immunodeficiency virus. An estimate of risk of acquired immunodeficiency syndrome (AIDS). Clin Orthop 240:129–135
- 16. Burks RT, Haut RC, Lancaster RL (1990) Biomechanical and histological observations of the dog patellar tendon after removal of its central one-third. Am J Sports Med 18(2):146–153
- 17. Buss DD, Warren RF, Wickiewicz TL et al (1993) Arthroscopically assisted reconstruction of the anterior cruciate ligament with use of autogenous patellar-ligament grafts. J Bone Joint Surg Am 75A(9):1346–1355
- 18. Caborn DNM, Selby JB (2002) Allograft anterior tibialis tendon with bioabsorbable interference screw fixation in anterior cruciate ligament reconstruction. Arthroscopy 18(1):102–105
- 19. Carter TR, Edinger S (1999) Isokinetic evaluation of anterior cruciate ligament reconstruction: hamstring versus patellar tendon. Arthroscopy 15(2):169–172
- 20. Cartmell JS, Dunn MG (2004) Development of cell-seeded patellar tendon allografts for anterior cruciate ligament reconstruction. Tissue Eng 10(7/8):1065–1075
- 21. Chang SKY, Egami DK, Shaieb MD et al (2003) Anterior cruciate ligament reconstruction: allograft versus autograft. Arthroscopy 19(5):453–462
- 22. Cole DW, Ginn TA, Chen GJ, Smith BP, Curl WW, Martin DF, Poehling GG (2005) Cost comparison of anterior cruciate ligament reconstruction: autograft versus allograft. Arthroscopy 21(7):786–790
- 23. Corry IS, Webb JM, Clingeleffer AJ, Pinczewski LA (1999) Arthroscopic reconstruction of the anterior cruciate ligament. Am J Sports Med 27(3):444–454
- 24. Coupens SD, Yates CK, Sheldon C, Ward C (1992) Magnetic resonance imaging evaluation of the patellar tendon after use of its central one-third for anterior cruciate ligament reconstruction. Am J Sports Med 20(3):332–335
- 25. D'Agata SD, Pearsall AW, Reider B, Draganich LF (1993) An in vitro analysis of patellofemoral contact areas and pressures following procurement of the central one-third patellar tendon. Am J Sports Med 21(2):212–219
- 26. Dandy DJ, Desai SS (1994) Patellar tendon length after anterior cruciate ligament reconstruction. J Bone Joint Surg Br 76B(2):198–199
- 27. Deehan DJ, Salmon LJ, Webb VJ et al (2000) Endoscopic reconstruction of the anterior cruciate ligament with an ipsilateral patellar tendon autograft. J Bone Joint Surg Br 82B(7):984– 991
- 28. Devita P, Hortobagyi T, Barrier J et al (1997) Gait adaptations before and after anterior cruciate ligament reconstruction surgery. Med Sci Sports Exerc 29(7):853–859
- 29. Ejerhed L, Kartus J, Sernert N, Köhler K, Karlsson J (2003) Patellar tendon or semitendinosus tendon autografts for anterior cruciate ligament reconstruction? A prospective randomized study with a two-year follow-up. Am J Sports Med 31:19–25
- 30. Engebretsen L, Benum P, Fasting O et al (1990) A prospective, randomized study of three surgical techniques for treatment of acute ruptures of the anterior cruciate ligament. Am J Sports Med 18(6):585–590
- 31. Fahey M, Indelicato PA (1994) Bone tunnel enlargement after anterior cruciate ligament replacement. Am J Sports Med 22(3):410–414
- 32. Fideler BM, Vangsness CT, Lu B et al (1995) Gamma irradiation: effects on biomechanical properties of human bone-patellar tendon-bone allografts. Am J Sports Med 23(5):643–647
- 33. Frank CB, Jackson DW (1997) The science of reconstruction of the anterior cruciate ligament—current concepts review. J Bone Joint Surg Am 79A:1556–1576
- 34. Freedman KB, D'Amato MJ, Nedeff DD, Kaz A, Bach BR Jr (2003) Arthroscopic anterior cruciate ligament reconstruction: a metaanalysis comparing patellar tendon and hamstring tendon autografts. Am J Sports Med 31(1):2–11
- 35. Friedlaender GE, Strong DM, Sell KW (1976) Studies on the antigenicity of bone. Freeze-dried and deep-frozen bone allografts in rabbits. J Bone Joint Surg Am 58A(6):854–857
- 36. Fu FH, Schulte KR (1996) Anterior cruciate ligament surgery 1996. State of the Art? Clin Orthop 325:19–24
- 37. Fuchs R, Wheatley W, Uribe JW et al (2002) Intra-articular anterior cruciate ligament reconstruction using patellar tendon allograft in the skeletally immature patient. Arthroscopy 18(8):824–828
- 38. Glickson J (2004) American Academy of Orthopaedic Surgeons. Academy News. The 2004 annual meeting edition of the AAOS Bulletin [online]. Available: http://www.aaos.org/wordhtml/ 2004news/c10-4.htm [accessed 20.7.2004]
- 39. Harner CD, Olson E, Irrgang JJ et al (1996) Allograft versus autograft anterior cruciate ligament reconstruction: 3- to 5-year outcome. Clin Orthop 324:134–144
- 40. Haut Donahue TL, Howell SM, Hull ML, Gregersen C (2002) A biomechanical evaluation of anterior and posterior tibialis tendons as suitable single-loop anterior cruciate ligament grafts. Arthroscopy 18(6):589–597
- 41. Hey-Groves EW (1917) Operation for the repair of crucial ligaments. Lancet 2:674–675
- 42. Hiemstra LA, Webber S, MacDonald PB, Kriellaars DJ (2000) Knee strength deficits after hamstring tendon and patellar tendon anterior cruciate ligament reconstruction. Med Sci Sports Exerc 32:1472–1479
- 43. Indelicato PA, Bittar ES, Prevot TJ et al (1990) Clinical comparison of freeze-dried and fresh frozen patellar tendon allografts for anterior cruciate ligament reconstruction of the knee. Am J Sports Med 18(4):335–342
- 44. Indelicato PA, Linton RC, Huegel M (1992) The results of freshfrozen patellar tendon allografts for chronic anterior cruciate ligament deficiency of the knee. Am J Sports Med 20(2):118–121
- 45. Jackson DW, Corsetti J, Simon TM (1996) Biologic incorporation of allograft anterior cruciate ligament replacements. Clin Orthop 324:126–133
- 46. Jackson DW, Grood ES, Cohn BT et al (1991) The effects of in situ freezing on the anterior cruciate ligament. An experimental study in goats. J Bone Joint Surg Am 73A(2):201–213
- 47. Jackson DW, Grood ES, Goldstein JD et al (1993) A comparison of patellar tendon autograft and allograft used for anterior cruciate ligament reconstruction in the goat model. Am J Sports Med 21(2):176–185
- 48. Jackson DW, Grood ES, Wilcox P et al (1988) The effects of processing techniques on the mechanical properties of boneanterior cruciate ligament-bone allografts. An experimental study in goats. Am J Sports Med 16(2):101–105
- 49. Jackson DW, Windler GE, Simon TM (1990) Intraarticular reaction associated with the use of freeze-dried, ethylene oxidesterlized bone-patella tendon-bone allografts in the reconstruction of the anterior cruciate ligament. Am J Sports Med 18(1):1–9
- 50. Jarvela T, Nyyssonen M, Kannus P et al (1999) Bone-patellar tendon-bone reconstruction of the anterior cruciate ligament. Int Orthop 23:227–231
- 51. Jarvela T, Paakkala T, Kannus P et al (2004) Ultrasonographic and power doppler evaluation of the patellar tendon ten years after harvesting its central third for reconstruction of the anterior cruciate ligament. Am J Sports Med 32(1):39–45
- 52. Jarvela T, Paakkala T, Kannus P, Jarvinen M (2001) The incidence of patellofemoral osteoarthritis and associated findings 7 years after anterior cruciate ligament reconstruction with a bone-patellar tendon-bone autograft. Am J Sports Med 29(1):18–24
- 53. Johnson RJ, Eriksson E, Haggmark T, Pope HM (1984) Five- to ten-year follow-up evaluation after reconstruction of the anterior cruciate ligament. Clin Orthop 183:122–140
- 54. Jomha NM, Pinczewski LA, Clingeleffer A, Otto DD (1999) Arthroscopic reconstruction of the anterior cruciate ligament with patellar-tendon autograft and interference screw fixation. J Bone Joint Surg Br 81B(5):775–779
- 55. Kartus J, Ejerhed L, Eriksson BI, Karlsson J (1999) The localization of the infrapatellar nerves in the anterior knee region with special emphasis on central third patellar tendon harvest: a dissection study on cadaver and amputated specimens. Arthroscopy 15(6):577–586
- 56. Kartus J, Ejerhed L, Sernet N et al (2000) Comparison of traditional and subcutaneous patellar tendon harvest. Am J Sports Med 28(3):328–335
- 57. Kartus J, Movin T, Karlsson J (2001) Donor-site morbidity and anterior knee problems after anterior cruciate ligament reconstruction using autografts. Arthroscopy 17(9):971–980
- 58. Katz JW, Fingeroth RJ (1996) The diagnostic accuracy of ruptures of the anterior cruciate ligament comparing the Lachman test, the anterior drawer sign, and the pivot shift test in acute and chronic knee injuries. Am J Sports Med 14(1):88–91
- 59. Kleipool AE, van Loon T, Marti RK (1994) Pain after use of the central third of the patellar tendon for cruciate ligament reconstruction. Acta Orthop Scand 65(1):62–66
- 60. Kuechle DK, Pearson SE, Beach WR et al (2002) Allograft anterior cruciate ligament reconstruction in patients over 40 years of age. Arthroscopy 18(8):845–853
- 61. Kurosaka M, Yoshiya S, Andrish JT (1987) A biomechanical comparison of different surgical techniques of graft fixation in anterior cruciate ligament reconstruction. Am J Sports Med 15(3):225–229
- 62. Kustos T, Balint L, Than P, Bardos T (2004) Comparative study of autograft or allograft in primary anterior cruciate reconstruction. Int Orthop 28:290–293
- 63. Lawhorn KW, Howell SM (2003) Scientific justification and technique for anterior cruciate ligament reconstruction using autogenous and allogenic soft-tissue grafts. Orthop Clin N Am 34:19–30
- 64. Lephart SM, Kocher MS, Harner CD, Fu FH (1993) Quadriceps strength and functional capacity after anterior cruciate ligament reconstruction. Patellar tendon autograft versus allograft. Am J Sports Med 21(5):738–743
- 65. Levitt RL, Malinin T, Posada A, Michalow A (1994) Reconstruction of anterior cruciate ligaments with bone-patellar tendon-bone and achilles tendon allografts. Clin Orthop 303:67–78
- 66. Linn RM, Fischer DA, Smith JP et al (1993) Achilles tendon allograft reconstruction of the anterior cruciate ligament-deficient knee. Am J Sports Med 21(6):825–831
- 67. Liu SH, Hang DW, Gentili A, Finerman GAM (1996) MRI and morphology of the insertion of the patellar tendon after graft harvesting. J Bone Joint Surg Br 78B(5):823–825
- 68. Mae T, Shino K, Maeda A et al (2003) Effect of gamma irradiation on remodeling process of tendon allograft. Clin Orthop 414:305–314
- 69. Malinin TI, Levitt RL, Bashore C et al (2002) A study of retrieved allografts used to replace anterior cruciate ligaments. Arthroscopy 18(2):163–170
- 70. Marder RA, Raskind JR, Carroll M (1991) Prospective evaluation of arthroscopically assisted anterior cruciate ligament reconstruction. Am J Sports Med 19(5):478–484
- 71. Marumoto JM, Mitsunaga MM, Richardson AB et al (1996) Late patellar tendon ruptures after removal of the central third for anterior cruciate ligament reconstruction. Am J Sports Med 24(5):698–701
- 72. McCarroll JR (1983) Fracture of the patella during a golf swing following reconstruction of the anterior cruciate ligament. A case report. Am J Sports Med 11(1):26–27
- 73. Meisterling RC, Wadsworth T, Ardill R et al (1993) Morphologic changes in the human patellar tendon after bone-tendonbone anterior cruciate ligament reconstruction. Clin Orthop 289:208–212
- 74. Meyers JF, Caspari RB, Cash JD, Manning JB (1992) Arthroscopic evaluation of allograft anterior cruciate ligament reconstruction. Arthroscopy 8(2):157–161
- 75. Miller AC (2004) A review of open and closed kinetic chain exercises following ACL reconstruction [online]. Available: http://www.brianmac.demon.co.uk/kneeinj.htm [accessed 20.7.2004]
- 76. Muellner T, Kaltenbrunner W, Nikolic A et al (1998) Shortening of the patellar tendon after anterior cruciate ligament reconstruction. Arthroscopy 14(6):592–596
- 77. Nikolaou PK, Seaber AV, Glisson RR et al (1986) Anterior cruciate ligament allograft transplantation. Long-term function, histology, revascularization, and operative technique. Am J Sports Med 14(5):348–360
- 78. Nixon RG, SeGall GK, Sax SL et al (1995) Reconstitution of the patellar tendon donor site after graft harvest. Clin Orthop 317:162–171
- 79. Noyes FR, Barber SD, Mangine RE (1990) Bone-patellar ligament-bone and fascia lata allografts for reconstruction of the anterior cruciate ligament. J Bone Joint Surg Am 72A(8):1125– 1135
- 80. Noyes FR, Barber-Westin SD (1996) Reconstruction of the anterior cruciate ligament with human allograft. Comparison of early and later results. J Bone Joint Surg Am 78A(4):524–537
- 81. Noyes FR, Butler DL, Grood ES et al (1984) Biomechanical analysis of human ligament grafts used in knee-ligament repairs and reconstructions. J Bone Joint Surg Am 66A:344–352
- 82. O'Brien SJ, Warren RF, Pavlov H et al (1991) Reconstruction of the chronically insufficient anterior cruciate ligament with the

central third of the patellar ligament. J Bone Joint Surg Am 73A(2):278–286

- 83. O'Neill DB (1996) Arthroscopically assisted reconstruction of the anterior cruciate ligament. A prospective randomized analysis of three techniques. J Bone Joint Surg Am 78A(6):803– 813
- 84. Pearsall AW, Hollis JM, Russell GV, Scheer Z (2003) A biomechanical comparison of three lower extremity tendons for ligamentous reconstruction about the knee. Arthroscopy 19(10):1091–1096
- 85. Peterson RK, Shelton WR, Bomboy AL (2001) Allograft versus autograft patellar tendon anterior cruciate ligament reconstruction: a 5-year follow-up. Arthroscopy 17(1):9-13
- 86. Pinczewski LA, Clingeleffer AJ, Otto DD et al (1997) Integration of hamstring tendon graft with bone in reconstruction of the anterior cruciate ligament. Arthroscopy 13(5):641–643
- 87. Poehling GG, Curl WW, Lee CA, Ginn TA, Rushing JT, Naughton MJ, Holden MB, Martin DF, Smith BP (2005) Analysis of outcomes of anterior cruciate ligament repair with 5-Year follow-up: allograft versus autograft. Arthroscopy 21(7):774–785
- 88. Prokopis PM, Schepsis AA (1999) Allograft use in ACL reconstruction. Knee 6:75–85
- 89. Rasmussen TJ, Feder SM, Butler DL, Noyes FR (1994) The effects of 4 Mrad of γ irradiation on the initial mechanical properties of bone-patellar tendon-bone grafts. Arthroscopy 10(2):188–197
- 90. Roberts TS, Drez D, McCarthy W, Paine R (1991) Anterior cruciate ligament reconstruction using freeze-dried, ethylene oxide-sterlized, bone-patellar tendon-bone allografts. Two year results in thirty-six patients. Am J Sports Med 19(1):35–41
- 91. Rodeo SA, Arnoczky SP, Torzilli PA et al (1993) Tendonhealing in a bone tunnel. J Bone Joint Surg Am 75A(12):1795– 1803
- 92. Rosenberg TD, Franklin JL, Baldwin GN, Nelson KA (1992) Extensor mechanism function after patellar tendon graft harvest for anterior cruciate ligament reconstruction. Am J Sports Med 20(5):519–525
- 93. Rubinstein RA, Shelbourne KD, VanMeter CD et al (1994) Isolated autogenous bone-patellar tendon-bone graft site morbidity. Am J Sports Med 22(3):324–327
- 94. Sachs RA, Daniel DM, Stone ML, Garfein RF (1989) Patellofemoral problems after anterior cruciate ligament reconstruction. Am J Sports Med 17(6):760–765
- 95. Saddemi SR, Frogameni AD, Fenton PJ et al (1993) Comparison of perioperative morbidity of anterior cruciate ligament autografts versus allografts. Arthroscopy 9(5):519–524
- 96. Shaffer BS, Tibone JE (1993) Patellar tendon length change after anterior cruciate ligament reconstruction using the midthird patellar tendon. Am J Sports Med 21(3):449–453
- 97. Shaieb MD, Kan DM, Chang SK, Marumoto JM, Richardson AB (2002) A prospective randomized comparison of patellar tendon versus semitendinosus and gracilis tendon autografts for anterior cruciate ligament reconstruction. Am J Sports Med 30:214–220
- 98. Shelton WR, Papendick L, Dukes AD (1997) Autograft versus allograft anterior cruciate ligament reconstruction. Arthroscopy 13(4):446–449
- 99. Sherman OH, Banffy MB (2004) Anterior cruciate ligament reconstruction: which graft is best? Arthroscopy 20(9):974–80
- 100. Shino K, Inoue M, Horibe S et al (1988) Maturation of allograft tendons transplanted into the knee. J Bone Joint Surg Br 70B(4):556–560
- 101. Shino K, Inoue M, Horibe S et al (1991) Surface blood flow and histology of human anterior cruciate ligament allografts. Arthroscopy 7(2):171–176
- 102. Shino K, Kawasaki T, Hirose H et al (1984) Replacement of the anterior cruciate ligament by an allogeneic tendon graft. An experimental study in the dog. J Bone Joint Surg Br 66B(5):672–681
- 103. Shino K, Kimura T, Hirose H et al (1986) Reconstruction of the anterior cruciate ligament by allogeneic tendon graft: an operation for chronic ligamentous insufficiency. J Bone Joint Surg Br 68B(5):739–746
- 104. Shino K, Nakata K, Horibe S et al (1993) Quantitive evaluation after arthroscopic anterior cruciate ligament reconstruction. Allograft versus autograft. Am J Sports Med 21(4):609–616
- 105. Sterling JC, Meyers MC, Calvo RD (1995) Allograft failure in cruciate ligament reconstruction. Am J Sports Med 23(2):173–178
- 106. Stringham DR, Pelmas CJ, Burks RT et al (1996) Comparison of anterior cruciate ligament reconstructions using patellar tendon autograft or allograft. Arthroscopy 12(4):414–421
- 107. Svensson M, Kartus J, Ejerhed L et al (2004) Does the patellar tendon normalize after harvesting its central third? Am J Sports Med 32(1):34–38
- 108. Van Eijden TMGJ, Kouwenhoven E, Weijs WA (1987) Mechanics of the patellar articulation. Effects of patellar liga-

ment length studied with a mathematical model. Acta Orthop Scand 58:560–566

- 109. Victor J, Bellemans J, Witvrouw E et al (1997) Graft selection in anterior cruciate ligament reconstruction—prospective analysis of patellar tendon autografts compared with allografts. Int Orthop 21:93–97
- 110. Wainer RA, Clarke TJ, Poehling GG (1988) Arthroscopic reconstruction of the anterior cruciate ligament using allograft tendon. Arthroscopy 4(3):199–205
- 111. Webb J (2001) Hamstrings and the anterior cruciate ligament deficient knee. The Knee 8:65–67
- 112. Webb JM, Corry IS, Clingeleffer AJ, Pinczewski LA (1998) Endoscopic reconstruction for isolated anterior cruciate ligament rupture. J Bone Joint Surg Br 80B(2):288–293
- 113. Yasuda K, Ohkoshi Y, Tanabe Y, Kaneda K (1992) Quantitative evaluation of knee instability and muscle strength after anterior cruciate ligament reconstruction using patellar and quadriceps tendon. Am J Sports Med 20(4):471–475
- 114. Yunes M, Richmond JC, Engels EA, Pinczewski LA (2001) Patellar versus hamstring tendons in anterior cruciate ligament reconstruction: a metaanalysis. Arthroscopy 17(3):248–257