Current Concepts Review: Revision Anterior Cruciate Ligament Reconstruction

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Failed anterior cruciate ligament (ACL) reconstruction presents a difficult clinical challenge. Successful revision ACL reconstruction depends on identifying the causes of failure and correcting technical or diagnostic errors. Failed ACL reconstruction may be either traumatic or atraumatic. Atraumatic failures may be attributable to technical errors, diagnostic errors, or failure of graft incorporation. Published outcomes of revision ACL reconstruction have been worse than for primary ACL reconstruction. The preoperative evaluation, surgical techniques, and clinical outcomes of revision ACL reconstruction are reviewed.

Keywords: anterior cruciate ligament; ACL; revision; failure

Injuries to the ACL are common in the athletic population. Anterior cruciate ligament reconstruction has been successful in restoring knee stability and function. Injuries to secondary restraints, ligamentous and capsular structures, articular cartilage, and meniscus affect the overall success or failure of ACL reconstructions. Surgical technique, postoperative rehabilitation, and patient expectations also play important roles in outcome.[26]

Outcome measures used in the literature include subjective measures of pain and satisfaction as well as patient-reported instability episodes and return to preinjury level of activity. A poor outcome may result from surgical complications, recurrent instability, or meniscal or articular cartilage injury.[26,54,60,63] Several knee-specific health evaluation tools have also been used such as the Lysholm and International Knee Documentation Committee (IKDC). Objective measures include loss of motion, functional strength measurements, Lachman test and pivot-shift examination, and excessive anterior translation by arthrometric testing. Although the pivot-shift test has been shown to correlate with subjective symptoms and function,[42] other objective measures including the Lachman test and instrumented laxity do not correlate with subjective outcomes.[66]

Although there is a dearth of literature regarding revision ACL reconstruction (RACLR), the outcomes of RACLR have been reported primarily in case series (level IV studies) and appear to be inferior to the outcomes of primary ACL reconstruction (PACLR).[52,65,78,79] It is critical to understand the causes of failure to adequately address the challenges of revision surgery. Our purpose is to review the patient presentation, modes of failure, surgical management, and outcomes of RACLR.

COMPLICATIONS OF ACL RECONSTRUCTION

Anterior cruciate ligament reconstruction can sometimes be associated with postoperative complications. These include loss of motion, recurrent pain or arthritis, and extensor mechanism dysfunction.[26,38] These complications may occur concurrently and may be difficult to clearly distinguish.

Loss of motion is the most common complication after ACL reconstruction, occurring in 11% to 35% of ACL reconstructions.[27,64,73] Loss of motion may be caused by prolonged immobilization, intercondylar notch scarring, capsulitis with ligament scarring, cyclops lesion, nonanatomical graft placement, infection, or reflex sympathetic dystrophy, or the cause may be idiopathic.[12] Both knee flexion and extension may be limited after ACL reconstruction. It is thought that the loss of passive extension is more detrimental for the high-performance athlete than loss of flexion because the resultant bent-knee gait abnormality inhibits running and alters the normal gait pattern. In addition, the bent-knee gait causes increased loads in the patellofemoral articulation and significant anterior knee pain.[64]

Arthrofibrosis, or scarring and stiffness in the knee, appears in both acute and chronically ACL-deficient knees after reconstructions.[73] Postoperative stiffness may also complicate ACL reconstructions that are performed in the acute injury phase before preoperative range of motion and normal gait patterns can be restored.[69] If adequate motion is not achieved within a reasonable period of time, arthroscopic lysis of adhesions and manipulation under anesthesia should be considered to restore joint motion. It is unclear, however, if proper treatment of postoperative arthrofibrosis completely restores outcomes to the level of those knees with normal motion.[27,73]

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Extensor mechanism dysfunction infrequently complicates ACL reconstruction, particularly if proper postoperative rehabilitation is not performed. Inadequate rehabilitation may lead to quadriceps inhibition, loss of patellar mobility, and loss of knee motion. If left untreated, patellar entrapment may progress to infrapatellar contracture syndrome or pathologic hyperplasia of the anterior soft tissues of the knee. Early recognition and treatment of infrapatellar contracture syndrome are necessary to avoid permanent joint contracture and patella baja.[57]

Recurrent instability in the early (less than 6 months) postoperative period typically results from poor surgical technique, failure of graft incorporation, premature return to deceleration and cutting sports, or overly aggressive rehabilitation, which may cause plastic deformation of the ACL graft. Late instability (more than 1 year postoperatively) after resumption of preinjury levels of competition is usually attributable to either a single major trauma or repetitive trauma to the ACL graft.[26,38]

Historically, synthetic grafts such as Dacron (Stryker Corp, Kalamazoo, Mich) and GORE-TEX (W L Gore & Associates Inc, Flagstaff, Ariz) have been fraught with complications including recurrent pain, mechanical failure, infection, tunnel osteolysis, and massive effusions.[23,58,81] Up to 56% of ACL reconstructions with GORE-TEX grafts resulted in a fair or poor outcome.[58] In addition, graft augmentation devices have been associated with stress shielding and subsequent weakening of the graft tissue and delayed graft incorporation.[2,49] Regardless of the type of graft used, it appears that a functional reconstruction depends on biological incorporation of the ACL graft material.

FAILED ACL RECONSTRUCTION: TRAUMATIC

Posttraumatic instability in the early postoperative period may be attributable to trauma to the ACL graft before complete graft incorporation.[37] Failure at the graft fixation site may occur if the graft is traumatized before biological graft incorporation.[44] Premature return to athletics before complete restoration of neuromuscular control may leave the knee less capable of responding to stress and more prone to recurrent injury.[26]

Instability may occur in the late postoperative period as a result of a traumatic force similar in magnitude to that required for a primary ACL tear.[12] Late failure caused by recurrent trauma occurs in 5% to 10% of patients who have returned to their preinjury level of activity.[37] Similar to the initial ACL rupture, late failures typically occur through the midsubstance of the graft. A systematic review of 9 randomized control trials comparing autograft bone-tendon-bone versus hamstring grafts demonstrated an overall failure rate of 3.6%.[70]

FAILED ACL RECONSTRUCTION: ATRAUMATIC

Anterior cruciate ligament reconstruction may fail for a variety of reasons other than recurrent trauma. Frequently, a combination of factors contributes to a poor result. The causes of failure have been categorized into technical errors, failure of graft incorporation, and recurrent trauma (Figure 1).[38]

Technical Errors

Error in surgical technique is the most common cause of ACL graft failure.[36,38,39,65,78,79] Damage to the graft during harvest or during fixation may result in graft weakness and failure.[26] Nonanatomic graft placement, graft impingement on the intercondylar roof, improper graft tensioning and inadequate graft fixation, and failure to address concurrent ligamentous injury may result in a poor outcome.[39]

Nonanatomic femoral and tibial tunnel placement is an important cause of failure of ACL reconstruction.[29] Poor tunnel placement leads to excessive changes in graft length throughout the range of motion, leading to plastic deformation of the graft and consequent graft loosening. The ideal placement of the femoral tunnel is as far posterior in the notch as possible without violation of the posterior cortical wall.[26] Because the femoral attachment of the ACL is close to the axis of rotation of the knee at the posterior-lateral femoral ridge, small changes in the ACL attachment may have a significant effect on knee biomechanics.[12,22] The most common error in femoral tunnel placement is anterior tunnel placement, particularly in the all-endoscopic technique where it may be difficult to visualize the over-the-top position (Figures 2 and 3).[26] Anterior femoral tunnel placement leads to excessive tension on the graft in flexion, resulting in restriction of knee flexion, tension on the graft fixation site, and eventual stretching of the graft (Figure 4). Although it does not appear to be as harmful to the ACL graft, posterior tunnel placement results in excessive graft tension with knee extension and slight looseness in flexion.[12] Femoral tunnel placement too close to the central axis of the femur results in adequate anterior restraint but poor rotational restraint.[61,80]

The ACL footprint has been shown to be anterior to the tibial spine on the medial half of the tibial eminence, with no portion on the lateral half.[22] Many surgeons place the tibial tunnel in the posteromedial portion of the ACL footprint.[26,31,33,34,63,75] The tunnel should be parallel and posterior to the Blumensaat line on the full extension radiograph.[26,30,31,33,34] Placement of the tibial tunnel less than 23 mm from the anterior edge of the tibia consistently produces graft impingement and flexion contracture.[31,33,34] In addition, it has been shown that anteromedial placement of the tibial tunnel causes both flexion and extension contractures.[63] Adequate posterior tibial tunnel positioning is particularly important to avoid impingement in knees with significant recurvatum or vertical intercondylar roofs.[35] Extreme posterior tibial tunnel placement can, however, result in excessive laxity in flexion and impingement on the posterior cruciate ligament. Medial or lateral positioning of the tunnel may also damage the articular surfaces of the medial and lateral tibial plateaus and may impinge on the lateral aspect of the intercondylar roof.[31-33,47,78] In addition, vertical graft positioning may provide anteroposterior stability but not rotational stability.[1]

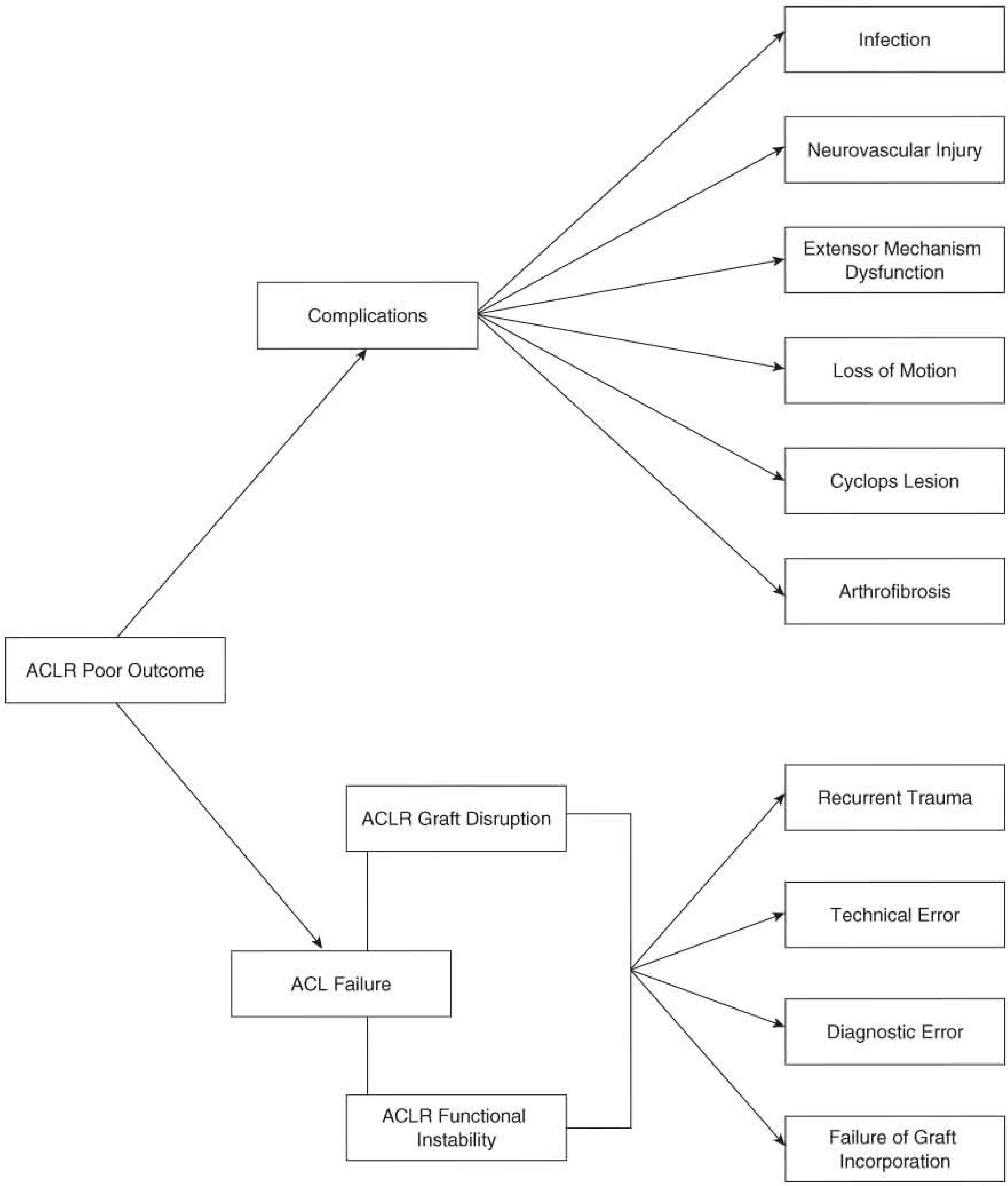

Figure 1. Poor outcomes of ACL reconstruction (ACLR).

Graft impingement on the intercondylar roof may be caused by improper tunnel placement, inadequate notchplasty, and oversized graft material.[35,75] Abrasion on the lateral femoral condyle or intercondylar roof can result in chronic synovitis, gradual ligament attrition, and eventual failure.[31,33,47,71,75] Cyclops lesions may present as continued pain and a block to full extension in response to graft impingement.[35]

During the early postoperative period, the graft fixation sites have a lower load to failure than the graft itself. Graft fixation must be secure enough to hold the graft in place during the process of biological incorporation.[44] Interference screws have been shown to be stronger than staples, suture fixation around a post, or soft tissue washer with screw fixation.[44,72] Interference screw fixation strength appears to be equal using the all-endoscopic technique compared with the rear-entry, outside-in technique of femoral fixation.[8] Use of interference screws, however, may be complicated by improper sizing of the bone plugs, osteopenic bone, divergent or convergent screw placement relative to the bone plug, and transection of the graft.[8,14,44,72]

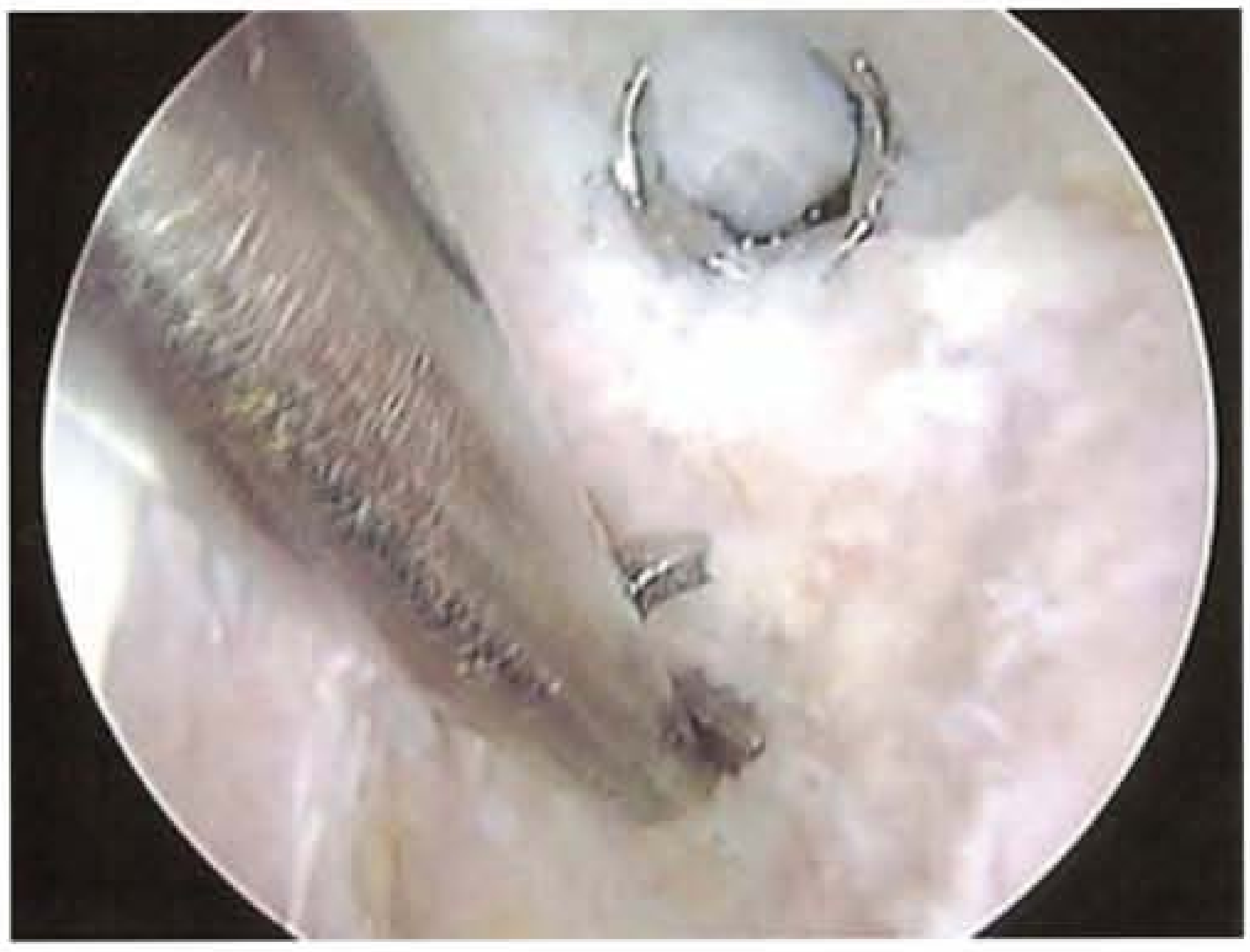

Figure 2. Anteriorly placed interference screw at revision surgery, indicating excessively anterior femoral tunnel placement.

Figure 3. Excessively anterior femoral tunnel at revision surgery after interference screw removal.

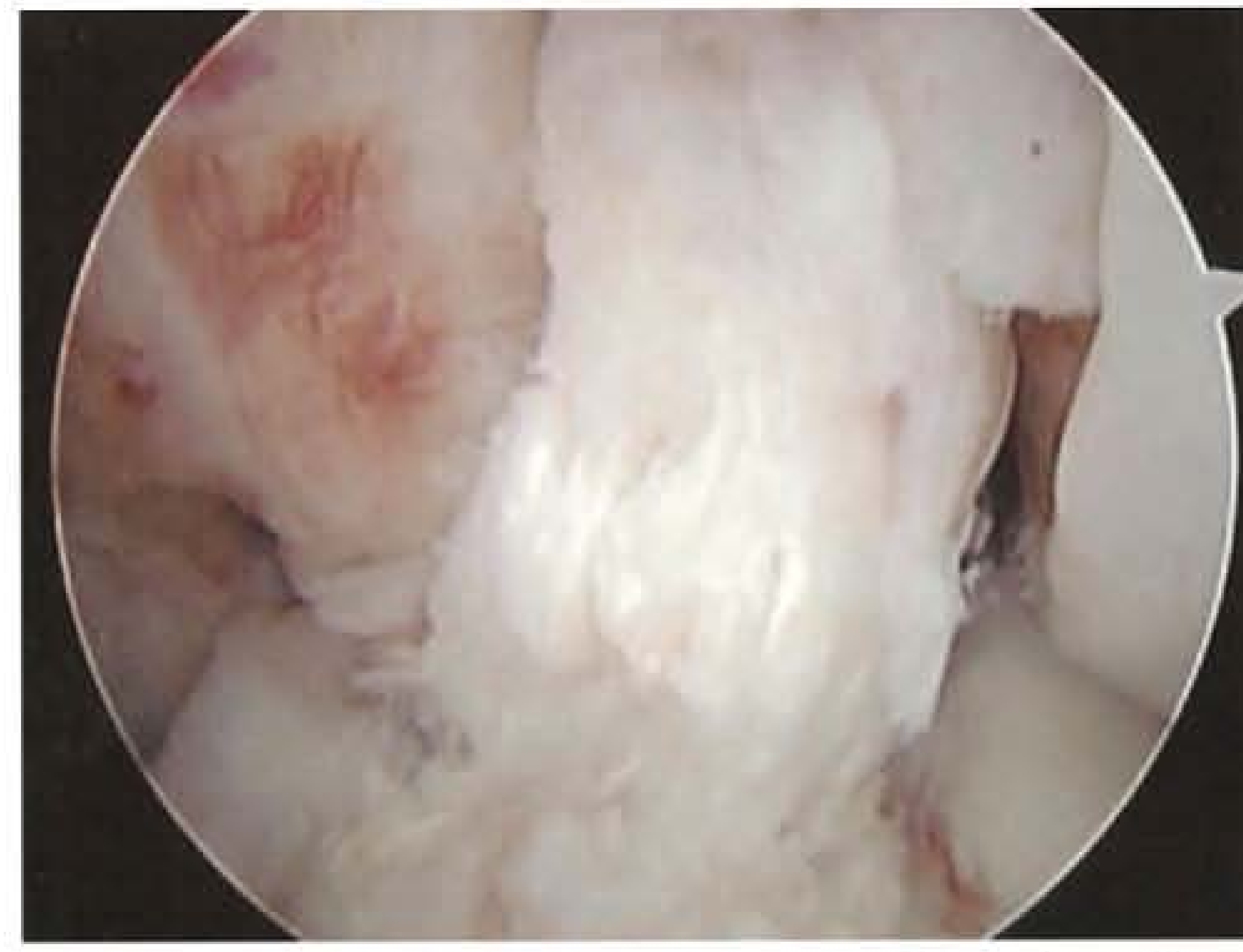

Figure 4. Excessively lax, stretched-out ACL graft attributable to anteriorly placed femoral tunnel, seen here at revision surgery.

The optimal graft tension is still unclear and may depend on the type of graft tissue.[9] In the dog model, overtensioning of the graft can lead to decreased motion, delayed revascularization, myxoid degeneration, and graft failure.[83] Changing the flexion angle from 0° to 30° at the time of tensioning appears to play a greater role in increasing graft force than the actual tension applied on the graft during fixation.[10,21] The dynamic role of the anteromedial and posterolateral bundles in stability at different flexion angles is unclear. Recent studies have shown the anteromedial bundle had a constant tension from full extension to 90° of flexion, whereas the posterolateral bundle decreased in tension with greater degrees of flexion.[45]

Diagnostic Errors

Anterior cruciate ligament injuries frequently occur concurrently with other capsular and ligamentous injuries in the knee. Failure to recognize and treat injuries to secondary and tertiary restraints can cause increased loads on the ACL reconstruction. Posterolateral instability is the most commonly unrecognized concurrent deficiency and is seen in 10% to 15% of chronically ACL-deficient knees.[20] The medial collateral ligament, posterior horn of the medial meniscus, and posterior capsule provide secondary stability in the ACL-deficient knee and must also be carefully assessed for injury.[26] Careful examination under anesthesia includes a complete assessment of varus, valgus, and rotational stability to recognize all associated deficiencies.

Preexisting conditions of the knee may also play an important role in the success of ACL reconstruction. Previous partial or complete meniscectomy and significant arthritis may be addressed with concurrent osteotomy or meniscal transplantation.[50,56] Varus knee alignment with lateral thrust can lead to chronic repetitive stretching of the ACL graft and may be treated with valgus tibial osteotomy at the time of ACL reconstruction.[50]

Failure of Graft Incorporation

The process of graft incorporation follows a predictable sequence for both autograft and allograft tissue. The graft undergoes a process of necrosis, followed by revascularization, cellular repopulation, collagen deposition, and finally matrix remodeling.[26] Inadequate graft vascularity caused by graft overtensioning, postoperative immobilization, infection, and immunologic reactions may delay or prevent graft incorporation.[2,48,72] Surgical factors such as roof impingement and excessive graft tensioning may also play a role in decreased vascularity and delayed graft incorporation. It is believed that incorporation of allograft tissue may be slower than that of autograft tissue.[49] The process of graft remodeling as well as healing of the bone plugs may also be delayed in allograft tissue.

Tunnel osteolysis may be seen with allografts as well as autografts.[59] The exact cause of osteolysis is probably multifactorial and has not been fully elucidated. In the case of allografts, it has been postulated that an immune response to the allograft tissue may cause osteolysis.[7] Other possible causes of tunnel osteolysis are graft micromotion inside the tunnel and stress shielding proximal to the fixation site.[59] The resultant bony deficiency may create a difficult technical problem during RACLR that must be dealt with appropriately.[6,40]

Differences between the rate of incorporation of the intra-articular portion of ACL autograft soft-tissue grafts (hamstring tendon) and bone-tendon-bone grafts (patellar tendon) have not been determined. It has been shown in animal models that fixation within the bone tunnels is delayed in soft tissue compared with bone-tendon-bone grafts.[62] There does not appear to be a clinically significant difference in PACLR outcome between autograft bone–patellar tendon–bone compared with hamstring tendon grafts. The aforementioned systematic review on PACLR showed no reproducible, clinically relevant difference in objective or subjective outcome measures except for more kneeling pain with bone-tendon-bone grafts.[70]

PREOPERATIVE EVALUATION

Careful patient evaluation is critical to the successful treatment of the failed ACL reconstruction. Patient activity level and symptom duration should be assessed. Subjective complaints may include pain, swelling, giving way, locking, noise, stiffness, or a limp.[43] Complaints of knee pain should be clearly distinguished from feelings of instability. In addition, a thorough medical and surgical history should include all previous graft sources, meniscal and articular cartilage injuries and treatment, and any other operative interventions on the knee. All past operative records should be carefully reviewed for surgically relevant details including intra-articular injuries and treatment, types of fixation, placement of grafts, and types of grafts. Special attention should be given to the "absorbable interference screws," which, although radiolucent, may in reality require removal even several years postoperatively.

Physical examination includes an assessment of knee effusion, range of motion, and other ligamentous deficiencies. Gait should be monitored because ACL-deficient knees may exhibit increased internal rotation during the initial swing phase.[19,76] Objective tests of ACL competency include the anterior drawer and pivot-shift tests. The pivot-shift test has been shown to be a very reliable measure for ACL insufficiency.[46] Furthermore, a positive pivot-shift test result is one of the few objective indications that is significantly associated with patient-oriented outcomes.[43] Surgical scars should also be carefully assessed and taken into consideration during the planning of operative approaches.

Radiographs should be used to determine the presence and location of hardware. Radiographs may reveal improper tunnel placement, tunnel osteolysis, and the presence of hardware. Magnetic resonance imaging is a useful adjunct to the radiological evaluation and has been shown to reliably assess the integrity of the reconstructed ACL.[60]

SURGICAL TECHNIQUES

Surgical technique and graft selection should be individualized; factors such as age, activity level, and previous surgery influence surgical decision making (Figure 5). The literature contains several detailed narrative reviews regarding surgical technique[1,3,4,16,77]; however, there is very little evidence based on clinical outcome studies to guide surgical decision making. Graft selection and fixation choice depend on the type of graft and fixation used at index as well as the index surgical technique and reason for failure. There are several techniques described for RACLR; however, the 2-incision[71] and endoscopic techniques are the most commonly reported (Table 1).

Options for graft fixation include ignoring previous fixation devices, removing them, and removing and reusing them. When tunnel placement and graft fixation can be achieved without interference from previous fixation, the hardware may be retained, which can avoid potential complications associated with hardware removal. Removing previous aperture fixation, particularly on the femoral side, can be difficult, may require fluoroscopic assistance, and can compromise surrounding bone and lead to tunnel expansion. Previous operative notes and current radiographs are essential for preoperative planning; however, if these data are unobtainable, a complete set of interference screwdrivers or universal screw removal sets are useful.

Previous surgical technique can influence the approach to RACLR, especially when index tunnel placement was appropriate. If index tunnel placement was appropriate and interference screws were used for fixation, then hardware removal will likely be necessary if the surgical technique used at index is used for RACLR, at least on the femoral side. Redirecting the tibial tunnel trajectory may avoid hardware removal on the tibial side. Altering the revision surgical approach from that used at index may avoid hardware removal on the femoral side. For example, if an endoscopic technique was used at index, a 2-incision technique at revision may avoid index hardware, and vice versa. The trajectory and location of the femoral tunnel may be changed by drilling via the anteromedial portal or by using the rear-entry technique, and these techniques should be available to surgeons performing RACLR.

The surgeon must be prepared for a myriad of clinical situations. Revision surgery may present unforeseen challenges such as bone voids and malpositioned hardware. The ability to draw from a wide range of surgical methods is critical for a successful outcome. The accurate placement of tibial and femoral tunnels is of the utmost importance and should not be compromised.

CLINICAL OUTCOMES

There are 2 key outcomes of interest with regard to graft failure and RACLR: risk factors for failure and prognosis after RACLR. Technical errors are believed to be the most common reason for failure, and although this may be true, there is little direct evidence to support this claim. Several studies have found that reinjury more often leads to revision surgery (Table 1). Most of the studies to date are procedure oriented, describing the outcome of RACLR after a particular technique or type of graft. There are no prospective studies with a control group comparing PACLR with RACLR.

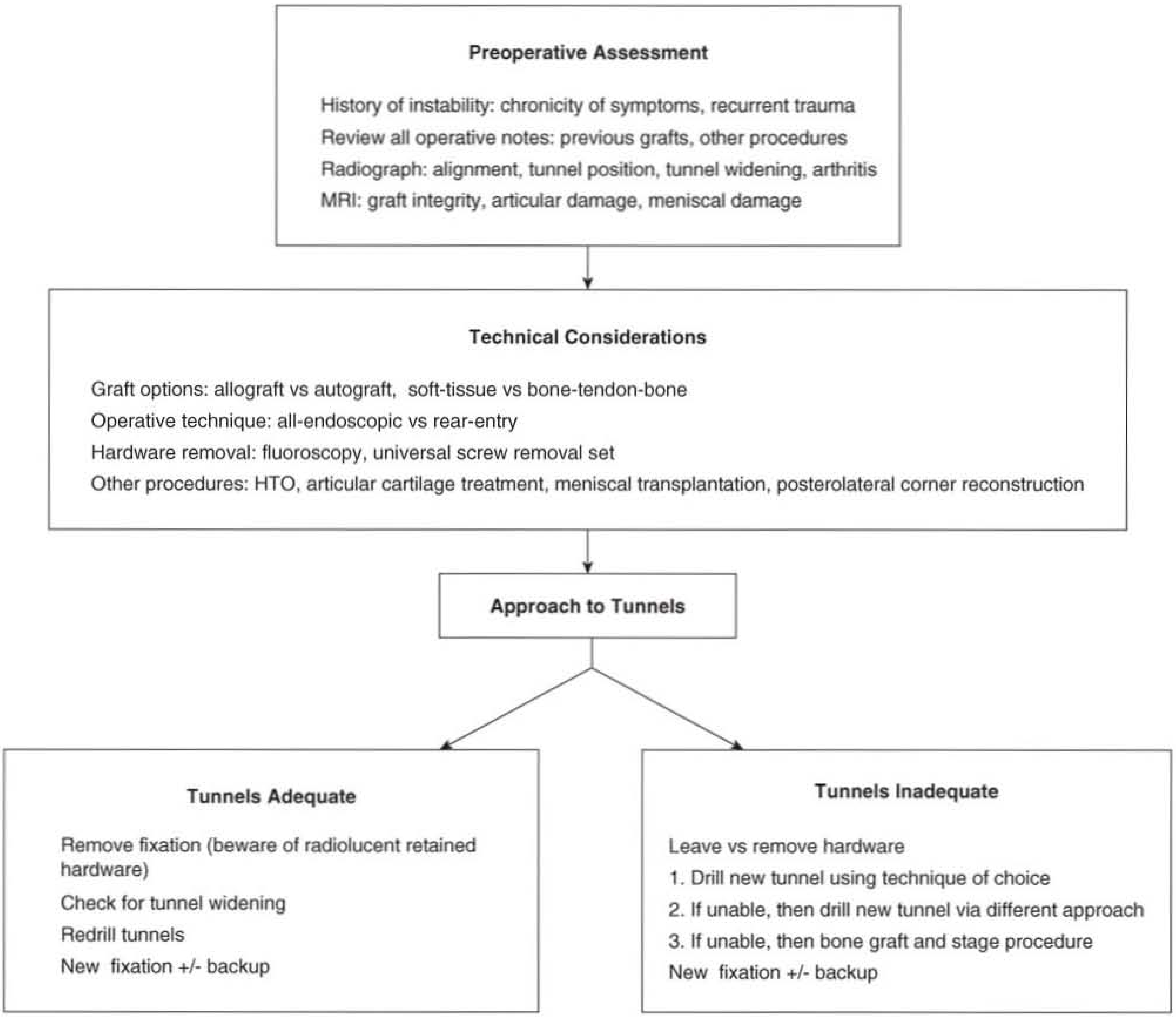

Figure 5. Approach to reconstruction of the failed ACL. MRI, magnetic resonance imaging; HTO, high tibial osteotomy.

Studies comparing different grafts either are nonconsecutive, retrospective case series or are subject to selection bias because graft allocation has been by patient and/or surgeon selection. Furthermore, studies that include allograft tissue are inconsistent with regard to reporting specific tissue banks and sterile preparation. Despite these weaknesses, some authors have reported statistically significant differences in KT-1000 arthrometer testing between allografts and autografts used for RACLR. Grossman et al[24] found a mean side-to-side difference after allograft RACLR of 3.2 mm, compared with 1.3 mm after autograft RACLR ($P < .05$); the IKDC, Lysholm, and Tegner scores were similar between the 2 groups. Uribe et al[78] found a mean side-to-side difference after allograft RACLR of 3.3 mm, compared with 2.2 mm after autograft RACLR ($P < .02$), but there were no significant differences between groups in the Lysholm or Tegner scores. The only other RACLR study that included both allografts and autografts did not formally stratify results by graft type but reported that of the 7 "failures" (>5 mm anteroposterior laxity on KT-1000 arthrometer testing), 4 were autografts and 3 were allografts.[74] Although a side-to-side difference of 1 to 2 mm may be statistically significant, it may not be clinically significant because all 3 of these studies concluded similarly that objective laxity measures do not correlate with subjective results.

A review of the available English literature was performed to summarize the available results of RACLR. Studies that reported results of RACLR were reviewed, and these results are summarized in Tables 1, 2, and 3. Studies with small sample size and insufficient methodological detail are not summarized, including several reports of the results of RACLR within narrative review articles.[11,12,16,82] In general, there is very little evidence, and no high-level evidence, regarding risk factors for graft failure or prognosis following RACLR. To date, study populations are very heterogeneous with regard to index surgical technique; index

TABLE 1
Summary of Revision ACL Reconstruction (RACLR) Studies[a]

Study	F/U (%)	Mean Years F/U (Range)	Mean/Median Age (Range)	Study Design	Graft Source[b]	Technique	Months to RACLR (Range)	Cause of Failure
O'Neill[54]	96	7.5 (2-13)	33 (13-57)	Case series	23 IL hamstring 25 IL BTB	34/48 endoscopic 14/48 2-incision	60 (7-276)	48/48 sports injury
Fox et al[17]	84	4.8 (2-12)	28 (16-57)	Case series	Nonirradiated BTB allograft	24/32 endoscopic 8/32 2-incision	50 (9-101)	—
Shelbourne and O'Shea[68]	94/57[c]	3.5 (2-9)	24.7 (16-46)	Case series	CL BTB	All mini-arthrotomy ACL reconstruction	46 (6-128)	22/54 technical, 28/54 sports injury, 1/54 MC trauma, 3/54 unknown
Colosimo et al[13]	87	3.3 (2-5.4)	27.2 (17-39)	Case series	Reharvest BTB	12/15 endoscopic 3/15 2-incision	83 (20-276)	5/13 reinjury, 8/13 gradual onset
Johnson et al[39]	100	2.3 (2-3)	25 (16-44)	Case series	25-kGy irradiated allograft: 13 BTB, 12 Achilles	All endoscopic	30 (6-175)	13/25 technical, 5/25 incorporation, 7/25 trauma
Noyes et al[53]	98	3.5 (1.9-6.5)	25 (13-45)	Cohort	BTB FF allograft (40 grafts: 25 000-Gy gamma irradiated), 32 LAD augmentation	All endoscopic	63 (6-279)	56/66 sports injury
Noyes and Barber-Westin[52]	96	2.7 (2-6.2)	27 (14-48)	Cohort	39 IL BTB, 5 CL BTB, 11 reharvest BTB	All endoscopic	80 (6-218)	47/54 sports injury
Taggart et al[74]	77	3.4 (1.2-5.3)	30 (22-55)	Case series	6 autograft hemi, 7 autograft BTB, 7 allograft BTB	All endoscopic	49 (19-30)	15/20 technical, 3/20 traumatic, 2/20 incorporation
Fules et al[18]	100	4.2 (1-8)	38 (24-53)	Case series	All autograft: 26 hemi, 2 quad, 1 BTB	All 2-incision	—	24/29 failed prosthetic grafts
Kartus et al[41]	100	A: 2.2 (1.7-2.8), B: 2.0 (1.9-2.2)	A: 27 (23-33), B: 27(24-33)	Case series	A: 12 reharvest BTB, B: 12 CL BTB	All endoscopic	A: 57 (15-132); B: 54 (20-108)	A: 10 technical, 2 trauma B: 8 technical, 4 trauma
Uribe et al[78]	84	2.5 (1.7-6.6)	23 (16-43)	Case series	19 allograft BTB, 2 IL hemi, 16 CL BTB, 17 IL BTB	76% endoscopic, 19% 2-incision, 5% over top	16 (<1-30)	61% technical, 22% trauma, 17% incorporation
Harilainen et al[25]	40	2 (—)	31 (—)	Case series	14 reharvest BTB, 7 CL BTB, 9 hemi	—	33.6 (—)[d]	23/30 technical, 7/30 trauma

(continued)

TABLE 1 (continued)

Study	F/U (%)	Mean Years F/U (Range)	Mean/Median Age (Range)	Study Design	Graft Source[b]	Technique	Months to RACLR (Range)	Cause of Failure
Wirth and Kohn[79]	100	8 (2-18)	26 (14-41)	Case series	57 IL BTB, 30 quad	Open and arthroscopic	—	Repeated trauma in a "minority"
Grossman et al[24]	83	5.6 (3-9)	30.2 (—)	Case series	22 allograft BTB, 6 CL BTB, 1 allograft Achilles	26/29 endoscopic, 3/29 2-incision	56 (2-192)	14/29 traumatic, 10/29 technical, 5/29 incorporation

[a]F/U, follow-up; IL, ipsilateral; BTB, central third; CL, contralateral; FF, fresh frozen; LAD, ligament augmentation device; MC, medial compartment; hemi, half.
[b]Unless reharvest is mentioned, all autografts are primary harvests.
[c]Subjective/objective follow-ups, respectively.
[d]Time from injury to operation (presumably, the original injury).

TABLE 2
Results of Subjective and Composite Grading Systems, Reported as Mean, Range (SD)[a]

Study	IKDC	KOOS	General QOL	Lysholm	Tegner	Modified Cincinnati[b]	Modified Noyes
O'Neill[54]	—	—	—	—	—	—	—
Fox et al[17]	71, 23-97 (22)	Pain: 84, 36-100 (18) Sx: 77, 25-100 (21) ADL: 91, 50-100 (14)	SF-12 mental: 55, 27-66 (8) SF-12 physical: 48, 20-59 (11)	75, 30-100 (22)	6.3, 0-10 (2.6)	7.2, 2-10 (2.2)	—
Shelbourne and O'Shea[68]	—	—	—	—	—	—	89.7, 65-100 (12.2)
Colosimo et al[13]	—	—	SF-36[c]	77.6, 61-98 (10.8)	5.8, 3-9 (1.4)	—	—
Johnson et al[39]	—	—	—	—	—	68, — (26.5)	—
Noyes et al[53]	—	—	—	—	—	77, — (13)	—
Noyes and Barber-Westin[52]	—	—	—	—	—	87, 62-100 (11)	—
Taggart et al[74]	—	—	—	85, 46-100 (—)	4.8, 2-7 (—)	—	—
Fules et al[18]	—	—	—	87.2, — (12.5)	4.45, — (1.6)	—	—
Kartus et al[41]	—	—	—	A: 62, 25-89 (—) B: 84, 55-95 (—) ($P = .002$)	A: 5, 1-7 (—) B: 5, 2-7 (—) ($P = .3$)	—	—
Uribe et al[78]	—	—	—	83, 59-95 (—)	5.5, 2-10 (—)	—	—
Wirth and Kohn[79]	—	—	—	68, — (12)	—	—	—
Harilainen et al[25]	—	—	—	89.5, — (—)	6, — (—)	—	—
Grossman et al[24]	84.8, 0-100 (—)			86.6, 0-100 (—)	5.2, 3-7 (—)		

[a]IKDC, International Knee Documentation Committee; KOOS, Knee Osteoarthritis and Outcome Survey; QOL, quality of life; Sx, symptoms; ADL, activities of daily living; SF, short form.
[b]Only the overall rating is included in the table.
[c]Reported as "within the average range compared with the normal population data for all but one patient."

graft choice; revision technique; staged procedures; concomitant ligament injuries; co-procedures such as articular cartilage restoration, meniscal transplant, osteotomy, and repair/reconstruction of other ligaments; and postoperative rehabilitation. To our knowledge, there is no published study with a concurrent control group of primary ACL reconstruction participants; instead historical controls have been used for comparison, which are themselves heterogeneous.

Despite the lack of a concurrent control group, several authors have concluded that RACLR portends a worse outcome compared with PACLR using historical controls. For instance, O'Neill[54] stated, "This study has confirmed that

TABLE 3
Objective Results[a]

Study	KT Side-to-Side Difference	Radiograph	IKDC[b]	Function of Graft[c]	Reoperation
O'Neill[54]	≤3 mm, 35/48 (73%) >3 to ≤5 mm, 10/48 (21%) >5 mm, 3/48 (6%)	18/48 (38%) "progressive changes"	A: 20/48 (42%) B: 20/48 (42%) C: 6/48 (12%) D: 2/48 (4%)	—	5/48 (10%)
Fox et al[17]	≤3 mm (84%) >3 to ≤5 mm, 3/32 (9%) >5 mm, 2/32 (6%)		—	—	None for HWR/ arthrofibrosis
Shelbourne and O'Shea[68]	≤3 mm, 26/31 (84%) >3 to ≤5 mm, 4/31 (13%) >5 mm, 1/31 (3%)	—	—	—	—
Colosimo et al[13]	≤3 mm, 12/13 (92%) >3 to ≤5 mm, 1/13 (8%)	—	—	—	—
Johnson et al[39]	≤3 mm, 5/25 (20%) >3 to ≤5 mm, 11/25 (44%) >5 mm, 9/25 (36%)	—	A/B: 3/25 (12%) C: 13/25 (52%) D: 9/25 (36%)	—	—
Noyes et al[53]	<3 mm, 6/16 (38%) 3-5.5 mm, 6/16 (38%) >5.5 mm, 2/16 (13%)[d]	—	—	Functional, 30/57 (53%) Partially functional, 12/57 (21%) Failed, 25/75 (33%)[e]	44/66 (67%)
Noyes and Barber-Westin[52]	—	—	—	Functional, 33/55 (60%) Partially functional, 9/55 (16%) Failed, 13/55 (24%)	9/55 (16.4%)
Taggart et al[74]	≤3 mm, 8/20 (40%) >3 to ≤5 mm, 5/20 (25%) >5 mm, 7/20 (35%)	—	—	—	—
Fules et al[18]	≤3 mm, 25/29 (86%) >3 to ≤5 mm, 3/29 (10%) >5 mm, 1/29 (4%)	Fairbank grade: 0: 5/29 I: 12/29 II: 8/29 III: 4/29	A: 0/29 B: 22/29 (76%) C: 5/29 (17%) D: 2/29 (7%)	—	—
Kartus et al[41]	—	—	A: B: 3/12 (25%), C: 7/12 (58%), D: 2/12 (17%) B: B: 7/12 (58%), C: 4/12 (33%), D: 1/12 (8%)	—	—
Uribe et al[78]	mean = 2.8 mm (autografts = 2.2 mm, allografts = 3.3 mm, $P = .02$)	10/54 (19%) "progression"	—	6% "possibly nonfunctional"	—
Wirth and Kohn[79]	—	Fairbank grade: 0: 31/87 I: 48/87 II: 6/87 III-IV: 2/87	—	—	—

(continued)

TABLE 3 (continued)

Study	KT Side-to-Side Difference	Radiograph	IKDC[b]	Function of Graft[c]	Reoperation
Harilainen et al[25]	Mean = 2.2 mm[f]	—	—	—	—
Grossman et al[24]	Mean = 2.9 mm (autografts = 1.3 mm, allografts = 3.2 mm, $P < .05$)	5/29 PFJ[g] 14/29 MC 2/29 LC	A: 17/29 (59%) B: 8/29 (27%) C: 4/29 (14%)		

[a]IKDC, International Knee Documentation Committee; HWR, hardware; PFJ, patellofemoral joint; MC, medial compartment; LC, lateral compartment.
[b]A, normal; B, nearly normal; C, abnormal; D, severely abnormal.
[c]Functional, KT-1000 arthrometer side-to-side difference <3 mm and no pivot shift; partially functional, KT-1000 arthrometer side-to-side difference between 3 and 5.5 mm with negative pivot shift result; failed, KT-1000 arthrometer side-to-side difference >6 mm or positive pivot shift result. Denominator is number of knees.
[d]Data limited to isolated revision ACL reconstruction with central third (BTB) allograft and no ligament augmentation device (LAD) augmentation or other major procedure; 2 missing.
[e]Includes 10 failures that occurred before 2-year follow-up interval; hence different denominator.
[f]Side-to-side difference measured with CA 4000 computerized analyzer (OSI, Hayward, Calif).
[g]Mild, moderate, and severe combined.

the results of RACLR surgery are less favorable than the results of primary ACLR." This is apparently based on a comparison of his previously published results after PACLR, where 92% of patients were found to be IKDC normal or nearly normal,[54] compared with 84% after RACLR.[55] In a nonconsecutive case series, Uribe et al[78] reported that return to preinjury activity was "significantly inferior to the authors' unpublished primary ACL results." Bach[4] found that 87% of RACLR subjects were either mostly or completely satisfied, compared with his previous study of PACLR that found 93% of subjects to be mostly or completely satisfied.[5] In 2001, Noyes and Barber-Westin[52] compared the graft failure rate after RACLR of 24% with their previously reported failure rate after PACLR of 7%.[51] Considering the 1-year follow-up of 30 RACLRs, Harilainen and Sandelin[25] concluded that "results of revision operations are not as good as those of well executed primary reconstructions" based on 28 age- and sex-matched historical controls. Although the authors reported mean Lysholm scores at 1 year of 83.5 and 93 for revisions and primaries, respectively ($P = .0156$), at 2-year follow-up, there was no statistically significant difference in the mean Lysholm scores between the 2 groups (89.5 and 99.0 respectively; P not significant).[25] Standing out against these conclusions, Shelbourne et al[68] reported similar modified Noyes subjective scores between a case series of RACLR subjects published in 2002 (mean score of 90) and the previous results of PACLR published in 1997 (mean score of 92).[67]

CONCLUSIONS

Studies to date are limited by their weak design, small numbers, heterogeneous populations, and lack of concurrent control groups. Because RACLR is relatively uncommon, single institution studies result in small sample sizes, which makes it difficult to control for confounding bias in heterogeneous populations. Many of these studies either used techniques at the time of revision that are outdated or revised subjects who had index procedures such as prosthetic grafts that are used less frequently today. Hence, the relevance of these studies to modern RACLR is uncertain. A prospective cohort study should be performed to identify risk factors for graft failure and compare the results of RACLR with PACLR while controlling for important confounders like co-procedures, articular cartilage, and meniscus status.

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