Review Article

Flexion Instability After Total Knee Arthroplasty

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Abstract

Flexion instability after total knee arthroplasty (TKA) is caused by an increased flexion gap compared with extension gap. Patients present with recurrent effusions, subjective instability (especially going downstairs), quadriceps weakness, and diffuse periretinacular pain. Manual testing for laxity in flexion is commonly done to confirm a diagnosis, although testing positions and laxity grades are inconsistent. Nonsurgical treatment includes quadriceps strengthening and bracing treatment. The mainstays to surgical management of femoral instability involve increasing the posterior condylar offset, decreasing the tibial slope, raising the joint line in combination with a thicker polyethylene insert, and ensuring appropriate rotation of implants. Patient outcomes after revision TKA for flexion instability show the least amount of improvement when compared with revisions for other TKA failure etiologies. Future work is needed to unify reproducible diagnostic criteria. Advancements in biomechanical analysis with motion detection, isokinetic quadriceps strength testing, and computational modeling are needed to advance the collective understanding of this underappreciated failure mechanism.

Instability is one of the four most common failure mechanisms in contemporary total knee arthroplasty (TKA), accounting for 11% to 26% of failures.[1-3] Flexion instability can present with other forms of instability (ie, coronal or global instability), but as an isolated condition, it can be difficult to accurately diagnose and manage.[4,5] We aim at reviewing the available literature on this topic and highlighting the efforts needed to unify diagnostic criteria, including physical examination findings, radiographic parameters, and biomechanical testing.

Definition and Causes

Fundamentally, flexion instability is the result of a flexion space that is larger or more lax than the extension gap.[6] When the knee is bent to 90°, the resultant loss of articular congruity from the lax flexion space diminishes the compressive load on the knee and increases the force needed to achieve joint stability.[7] This imbalance places undue stress onto the surrounding supporting structures of the knee (ie, quadriceps, extensor mechanism, hamstrings, and collateral ligaments), leading to symptoms of instability during activities of weight bearing when the knee is flexed. Flexion instability is caused by an inability to balance the flexion and extension space at the time of index arthroplasty or from gradual laxity of the posterior capsule or posterior cruciate ligament (PCL) in cruciate-retaining (CR) designed implants[8,9] (Table 1). Flexion instability may also occur with posterior-stabilized (PS) knee designs.[10] Gap symmetry and soft-tissue balancing remain indispensable to prevent

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Table 1

Sagittal Balancing Errors and Those Leading to Flexion Instability (Bold)

Extension Space	Flexion Space		
	Tight	Balanced	Loose
Tight	Not enough tibial resection	Not enough distal femur resection Tight posterior capsule	**Undersized femoral implant** **Not enough distal femur resection**
Balanced	Not enough posterior femur resection PCL too tight	Well-balanced knee	**Too much posterior femoral condylar resection** **Increased tibial slope resection**
Loose	Oversized femoral implant	Too much distal femur resection	Too much tibial resection

PCL = posterior cruciate ligament

excessive anterior translation without cam-post impingement or dislocation. Technical factors that can lead to flexion instability include too little distal femoral resection in a preexisting flexion contracture (Figure 1), overly aggressive posterior condylar resection with undersized femoral implants (Figure 2), excessive posterior slope on the tibia (Figure 3), or over-release of the PCL in the CR knee.[6,11]

Mid-flexion Instability

Although flexion instability is described as laxity at 90° of flexion, mid-flexion instability is thought to be a different entity that is linked to rotational instability between 30° and 90° of flexion.[12] The cause of such rotational instability is debated but is thought to have a relation to the isometry of the collateral ligaments throughout the arc of motion. Mid-flexion instability has yet to be reproducibly described in clinical practice but is thought to present with subtle instability and pain when going from fully extended to early flexion with full muscle activation, such as ascending stairs.[13] Causes of mid-flexion instability are not agreed upon, with postulations ranging from altered ligament tension during motion from raising the joint line,[14,15] anterior positioning of the femoral prosthesis,[16] or multiradius femoral implant designs.[17,18] Currently, discerning the differences between true mid-flexion and flexion instability is difficult.

Diagnosis

Clinical Presentation and Symptoms

Patients who present with flexion instability usually have a constellation of complaints. They characteristically report a sense of distrust with their knee and that it wants to shift or slide, classically when rising from a seated position or navigating stairs.[8] Often, the knee never felt well since the index arthroplasty. Some patients who received a CR TKA may have excellent early flexion but soon develop feelings of instability as the anterior restraints (ie, quadriceps and extensor mechanism) fatigue and chronic anterior knee pain develops.[9] This feeling of instability when the knee is loaded in flexion activities is what most patients notice. However, flexion contractures can sometimes develop in cases of flexion instability. The purported mechanism for this counterintuitive phenomenon is that the quadriceps becomes overworked trying to provide sagittal support, leading to a weakened extensor mechanism. When combined with a tight posterior capsule (from a

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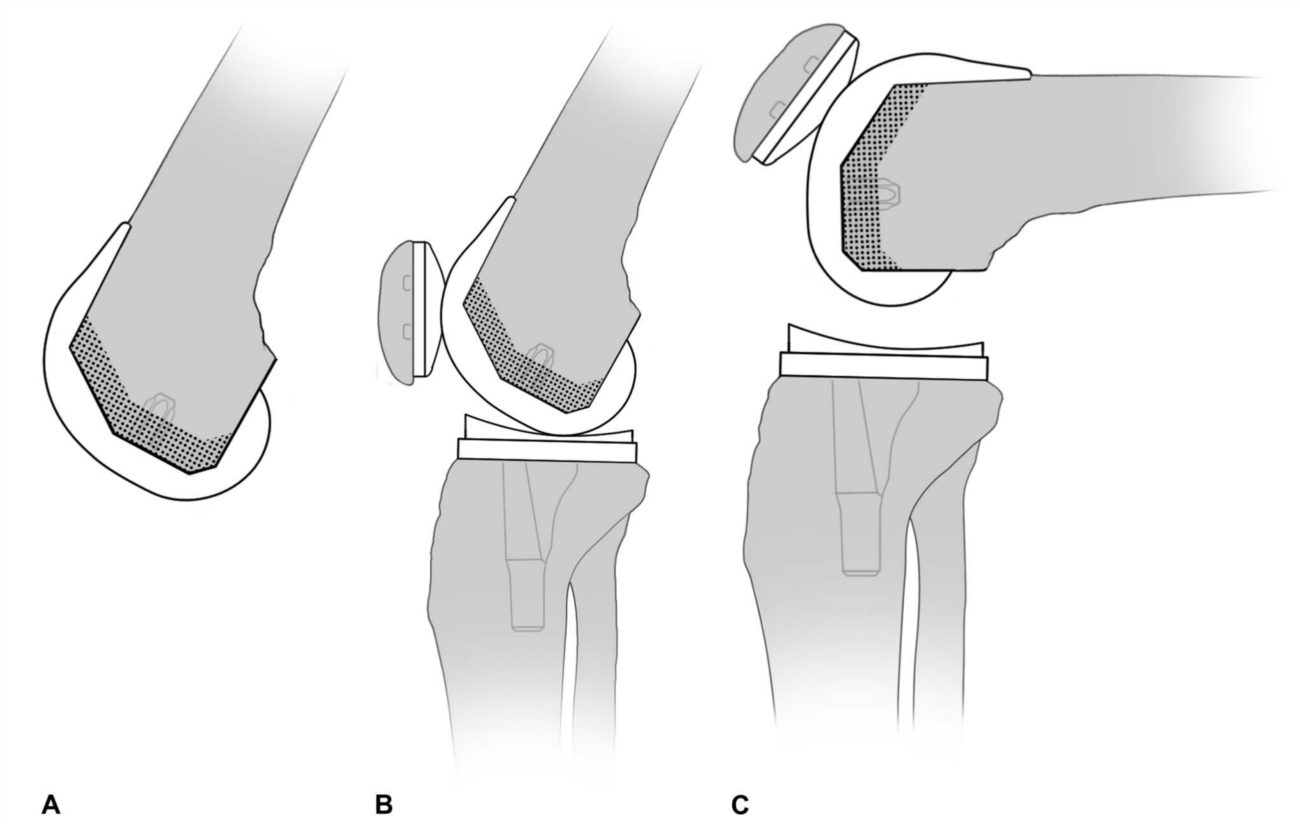
Figure 1



In patients with preexisting flexion contracture, it is possible to create an instance of flexion instability when the surgeon does not resect enough distal femur (**A**). The shaded area signifies the bone that should have been cut to properly balance the gaps. This scenario with under-resection of the distal femur translates to a knee remaining tight in extension (**B**), necessitating that the surgeon use a thin polyethylene insert, thereby increasing the flexion space (**C**) and creating laxity in flexion.

preoperative flexion contracture), the knee can assume a flexed resting position in this unique setting.[19] A high percentage of patients also report diffuse periretinacular tenderness and recurrent low-grade effusions.[20]

Physical Examination

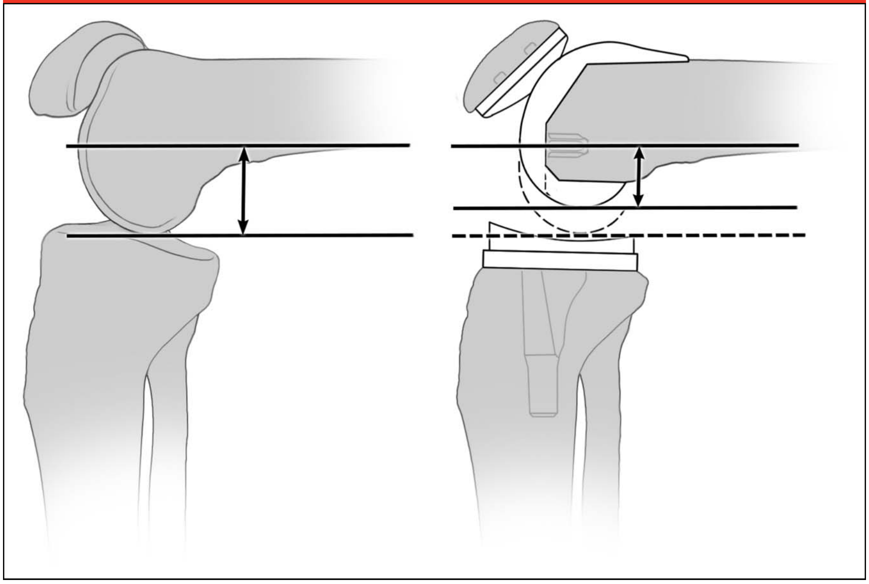
Although flexion instability is often thought of a diagnosis of exclusion, it remains a clinical diagnosis. Although many authors have reported excessive anterior-posterior translation in 90° of flexion as a mainstay of diagnosing flexion instability, no consensus exists quantifying the amount of motion that is pathologic. Abdel et al[21] described tibial translation as mild for motion of <5 mm, moderate if between 5 mm and 1 cm, and marked if >1 cm of anterior-posterior motion was present to diagnosis flexion instability. Tibial translation is measured in a similar manner to an anterior drawer test for anterior cruciate ligament (ACL) rupture in native knees, where an abrupt maneuver when the leg is fully relaxed provides the qualitative sense of instability. However, no correlation studies have confirmed the reproducibility of these measurements. Furthermore, it can be difficult to fully assess flexion instability when patients present with chronic pain and guarding. Stability testing should also be in mid-flexion, and extension should be done as well.

Others have described testing translation under anesthesia (Video, Supplemental Digital Content 1, http://links.lww.com/JAAOS/A306), but specific steps and thresholds defining instability are ill defined.[20,22] Pagnano et al described a posterior sag sign in which the tibia translates posteriorly when the knee is flexed to 90° and the heel is supported on the table to relax the quadriceps.[8,10] Again, no cutoffs for pathologic translation have been quantified for this test. Vince[6] has also described an examination maneuver to diagnose instability in which the patient is seated at the end of the examination table with the knee bent over the edge and the quadriceps is relaxed. If flexion instability is present, the larger flexion space will cause the tibia to descend and bring the polyethylene out of contact with the posterior condyles. When the patient is asked to actively extend, the physician will note the tibia pull up to articulate with the femur upon initiation of quadriceps contraction, and only after this contact is reestablished, will the tibia extend.

Additional examination findings include recurrent aseptic effusions. Hernandez et al[23] showed that more than 60% of patients with a diagnosis of flexion instability had a serosanguinous aspiration with less than 500 nucleated cells. Because of increased stresses placed on secondary musculotendinous stabilizers of the knee in cases of true flexion instability, a physician can also elicit pain by palpating the pes anserine and hamstring tendons. The discovery of bursal swelling and irritation overlying the pes tendon insertion along the medial tibia is not uncommon.[8]

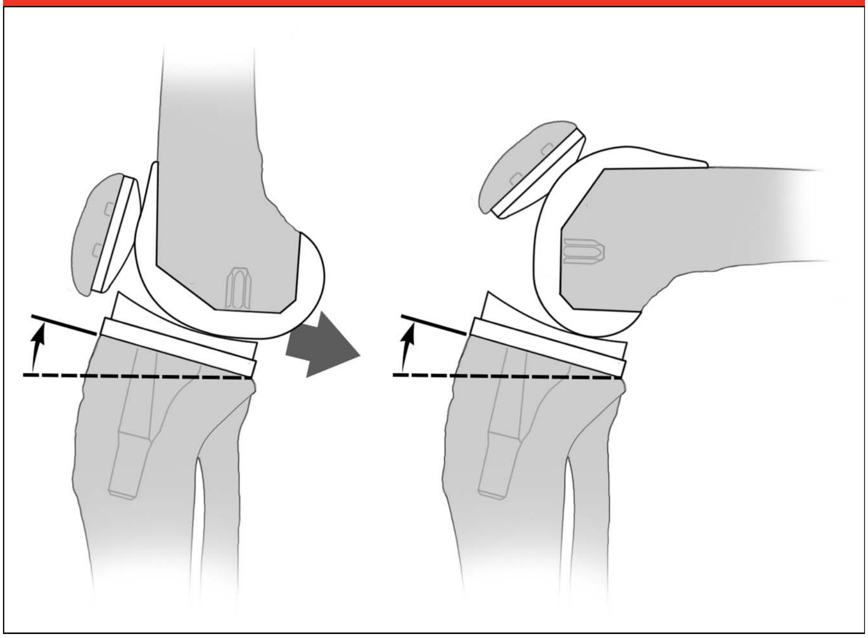
Physicians should also consider the possibility of concomitant multiligament imbalance because flexion instability does not always present in isolation. Yoshihara et al[24] described a subset of patients with flexion instability who also had greater than 4° of medial laxity and 7° of lateral laxity in flexion as more likely to be symptomatic than those without mediolateral laxity in flexion. Those with multiplanar instability in flexion must have femoral rotation scrutinized and should receive a varus-valgus constrained prosthesis to avoid recurring symptomatic laxity. Last, the patient's gait should be observed, with attention placed on coronal and anterior-posterior motion and overall stance to see how a patient may be compensating for a contracture of laxity in a given plane.[25]

Figure 2



An overly aggressive posterior condylar resection can lead to flexion instability. These bone cuts often lead to undersizing of the femoral implant, thereby not recreating the appropriate posterior femoral offset, as indicated by the dashed line.

Figure 3

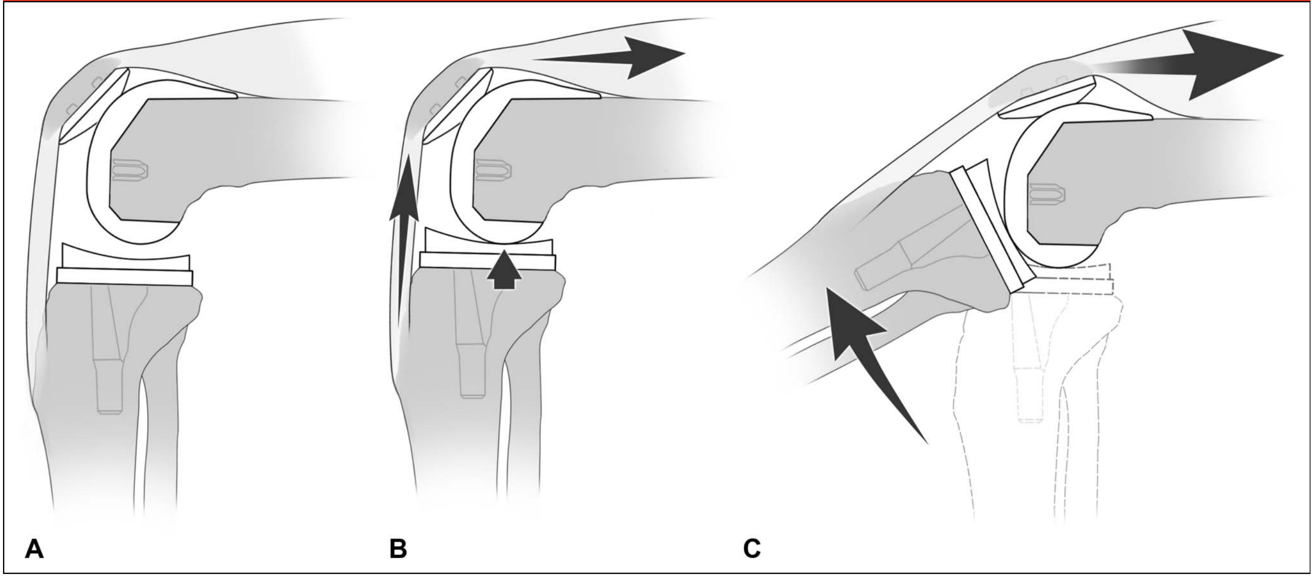


By cutting too much posterior slope into the tibia, the gap posteriorly increases disproportionately to the extension gap. This scenario allows the knee to roll posteriorly on the tibia in flexion, thereby translating the tibia anteriorly, leading to flexion instability.

Radiographs and Workup

The diagnosis of flexion instability is made only after a thorough workup for other common failure mechanisms. Serum inflammatory markers should be evaluated, and an aspiration should be done as directed by the Clinical Practice Guidelines set forth by the American Academy of Orthopaedic Surgeons.[26] Previous surgical

Figure 4



When a patient with flexion instability rests with their knee at a 90° flexed position without their foot contacting the floor, the femoral implant does not contact the polyethylene (**A**). To initiate knee extension, the quadriceps and extensor mechanism must first contract to pull the tibia up to contact the femur (**B**) before any extension can occur. The quadriceps must generate additional increased force to subsequently extend the knee once femoral-polyethylene contact is established (**C**).

reports and implant stickers should be requested from the index arthroplasty when a revision surgery is being considered.

As with any evaluation, a complete weight-bearing radiograph series, including AP, lateral, and sunrise views, should be obtained to scrutinize fixation, alignment, placement, and sizing of implants.[27] Full-length AP and lateral hip-knee-ankle radiographs should be evaluated for overall limb alignment potential joint line distalization. When a high suspicion for flexion instability exists, one should closely inspect the lateral radiograph to calculate the slope of the tibial tray and the posterior femoral condylar offset.[21] The size of the polyethylene insert and other radiographic clues, such as patella baja, may suggest a postoperative alteration in joint kinematics. The surgeon should also request preoperative radiographs as a comparison to obtain a better sense of femoral implant positioning in relation to the epicondylar axis.[21] A lateral of the contralateral knee, if not replaced, may also assist. Advanced scintigraphic or three-dimensional imaging is not necessary to confirm flexion instability but could be useful in cases of concomitant loosening or combined instability patterns in which implant malrotation is involved.[6] Although stress radiographs in 90° of flexion could prove useful, no literature exists to guide its current use or how to interpret such findings when planning a surgical correction.

Current Management

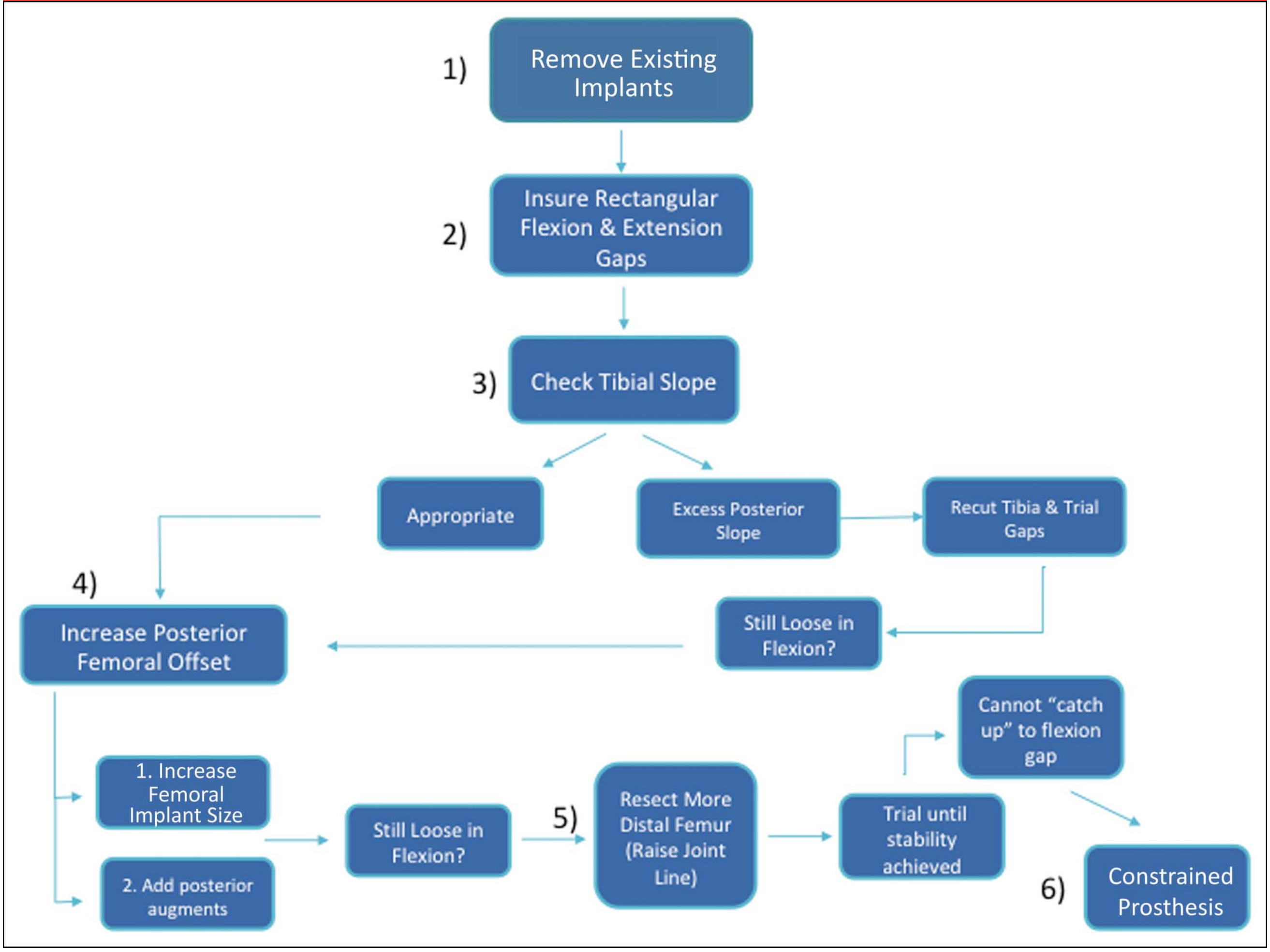
Nonsurgical: Therapy

A paucity of literature exists regarding the role of nonsurgical management for flexion instability. Some authors have mentioned a limited role for nonsurgical measures because the condition is viewed strictly as mechanical.[28] However, we are not aware of any specific recommendations or protocols in terms of bracing treatment, therapy, or pain control. Most patients with flexion instability can likely benefit from quadriceps strengthening because quadriceps weakness is related to the overworked extensor mechanism needed to initiate motion (Figure 4). A stronger quadriceps may help improve instability in flexion, and isometric quadriceps testing can provide a quantitative strength measurement to guide rehabilitation. A knee brace may also help with subjective feelings of instability. Further research is necessary to determine the utility of quadriceps-strengthening programs on subsequent laxity measures and their role in improving patient-reported outcomes and possible avoidance of revision TKA.

Surgical: Revision Total Knee Arthroplasty

For patients who have failed a trial of nonsurgical management, revision surgery should be considered to address the underlying cause of flexion-extension gap mismatch. In cases of previous well-functioning CR knees that have developed late

Figure 5



Flow diagram highlighting surgical steps during revision knee surgery to address flexion instability.

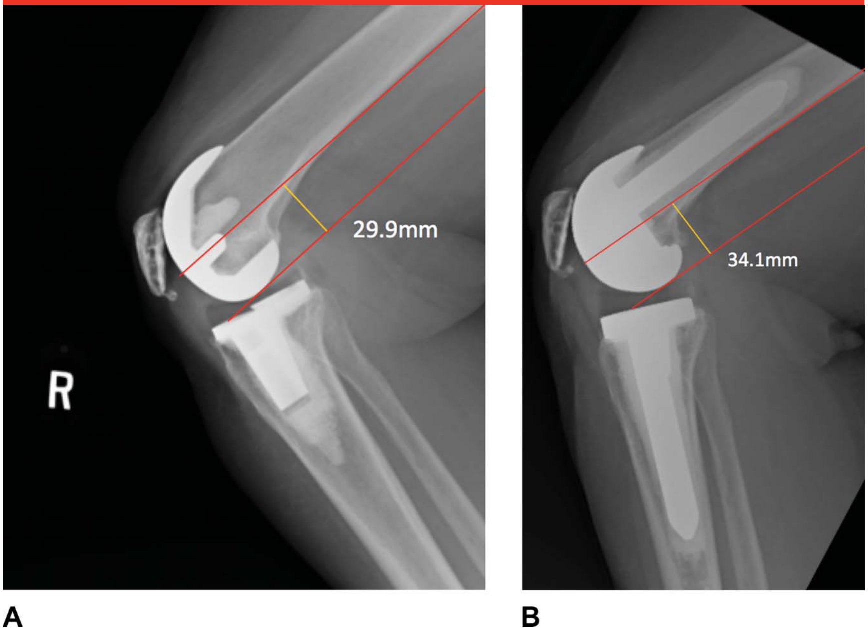
instability due to PCL incompetence, the knee should be revised to a more constrained implant. Depending on manufacturer specifications, it may be possible to keep a well-fixed tibial tray and only revise the femoral implant in this setting.[8] It is imperative, though, that the surgeon evaluate the flexion and extension spaces intraoperatively because it is possible that excessive tibial slope may necessitate removal of a tibial tray to appropriately balance the spaces and prevent post-cam impingement in extension.

Abdel et al[21] provided a generalized intraoperative sequence to guide equalization of the flexion and extension gaps. The suggested order of correction is to first normalize the tibial slope, followed by adjusting implant axis of rotation, correcting coronal imbalances, increasing femoral AP dimension, and raising the joint line. The surgeon should recheck the gaps at each step up the ladder and stop when gap symmetry and equalization is achieved. Vince[6] proposed a similar sequence for reestablishing equal flexion and extension spaces: (1) create a stable tibial platform, as this affects both extension and flexion gaps; (2) flexion gap balancing with a larger and appropriately rotated femoral implant; and (3) extension gap matching by adjusting the proximal-distal placement of the femoral prosthesis. We have included a flow diagram of our recommended intraoperative steps to address flexion instability (Figure 5).

Upsize Polyethylene

Treating flexion instability by only increasing the bearing size must be approached with caution. This practice is inherently limited because it equally adds to the extension and flexion space. Schwab et al[10] described this practice in their early experience for treating flexion instability. In 2 of their 10 patients, they managed the instability using a thicker PS polyethylene in combination with extensive posterior capsule

Figure 6



Lateral radiographs demonstrating flexion instability as a result of over-resection of the posterior femoral condyle (**A**) that was surgically corrected by increasing the posterior femoral offset by 4 mm using a revision femoral implant and posterior augment (**B**).

release. Although both patients had reported pain relief, one had a flexion contracture that never improved. The overall outcomes of isolated polyethylene exchange in revision knee arthroplasty are modest at best, with reports of a 50% failure rate within three years.[29] Because of the risk of not achieving full extension and adequately balancing the gaps, the use of isolated bearing upsizing is discouraged for isolated flexion instability.

Larger Femoral Implant

A mainstay of revision surgery for flexion instability should be to increase the posterior condylar offset by using a femoral implant with a larger AP dimension.[6,21] If the contralateral knee has not been replaced, the lateral radiograph can be used as a comparison when planning femoral revision to anticipate femoral implant size.[11] Femoral rotation should be checked with the knee in flexion using the tibial cut surface and native transepicondylar as references. Trial blocks or a tensioning device should be used to then set the rotation of the femur along an axis that parallels the tibial surface.[21] Upsizing the femur may lead to a gap between the posterior condyle and the implant, which should be managed with metal augmentation (Figure 6). Trialing with the larger implants is important to determine that any adjustments in femoral positioning and rotation allow for appropriate patellar tracking. The surgeon should aim for <5 mm of anterior tibial translation when the patella is reduced and the knee is at 90° of flexion.[21]

Joint Line Elevation

If the flexion and extension spaces cannot be adequately balanced by the already mentioned measures, the next step is to increase the extension space by resecting more distal femur.[10,21] Abdel et reported needing to raise the joint line >5 mm in 56% of the cases in their series, but that the vast majority required both increased posterior femoral offset and distal femoral resection (52/60 revisions).[21] The need to resect more distal femur is often encountered when the PCL becomes incompetent and the flexion space grows unequally. Chronic attrition of the PCL may place strain on the medial collateral ligament, resulting in a combined instability pattern. Revision in this situation requires a more constrained prosthesis.[20]

Constraint

When a proper sequence for balancing is followed, a surgeon may be able to rely on a cruciate-substituting bearing for a first-time revision for flexion instability.[21] The PS design provides a cam-post mechanism that limits tibial translation at varying degrees of flexion as the post engages. This post also confers an element of axial stability to prevent frank dislocation, deemed jump distance because of its dimensions.[30] However, often flexion instability is accompanied by medial-lateral instability. In situations when coronal laxity is encountered after removal of existing implants, it is often necessary to turn to varus-valgus constraint to confer additional stability.[5] Vince et al[9] thought that many cases of flexion instability had an implant of collateral insufficiency that was masked in full extension because of the tight posterior capsule. A condylar linked hinge prosthesis should be considered in cases in which notable femoral bone loss affects the epicondyles and compromises functionality of the collaterals or when the flexion space is so large that equalization to the extension space is impossible.

Outcomes

Although patients tend to make gains in their pain and functional scales after revision for flexion instability, the aggregate improvement is not as predictable compared with revisions for other etiologies.[31] Abdel et al[21] cited notable improvements in the mean Knee Society Score (KSS) and KSS functional scale scores in their series of 60 revisions for flexion instability at a mean 3.6 years of follow-up. Kannan et al[20] reported that 75% (27/36) of patients had improvements in function with an average 27.3-point gain in the overall KSS (34.5 to 61.8) and a 20.8-point improvement in KSS function (39.5 to 60.8) at a mean of 32 months and a minimum of 1 year. All patients in this cohort had a femoral revision, 8 of whom received a PS bearing, whereas 29 others required a varus-valgus constrained insert because of persistent laxity in flexion. They were unable to show an association with preoperative factors such as age, sex, BMI, and original bearing type (CR versus PS) and improvement in outcome scores. In addition, no association was found in terms of level of subjective improvement compared with the amount of radiological correction of the posterior condylar offset or tibial slope.

When comparing revisions for flexion instability (n = 35) with those done for infection or aseptic loosening, Grayson et al[31] found that the overall drop in median KSS expectation scores was notably worse in flexion instability patients (six points versus three points each, respectively; $P = 0.02$). These nuanced findings suggest that flexion instability patients experienced more disappointment with their results one year after revision surgery. This assumption was confirmed by an analysis of expectations, which showed that 40% to 60% of flexion instability patients were somewhat or a lot worse in terms of meeting their expectations for pain relief, ability to perform activities of daily living, and participation in recreational activities. In their comparative study regarding the outcomes of revisions for flexion instability versus those done for aseptic loosening and infection, Rajgopal et al[22] noted that patients with flexion instability often had markedly higher prerevision KSS and Western Ontario and McMaster Universities Osteoarthritis Index scores, which may explain the smaller increments for improvement, although they reached similar scores at 2-year follow-up. Last, Luttjeboer et al[32] reported that patients who received a condylar hinge compared with a varus-valgus constrained insert at revision had markedly worse KSS scores in all domains but similar patient satisfaction scores at 2-year follow-up. The 19 patients who received a PS insert, however, had an 80% complication rate with recurrent instability being the main culprit.

Future Directions

Physical Examination Grading System

The current benchmark for diagnosis is the tibial translation test in which the examiner subjectively grades instability as <5 mm, 5 to 10 mm, or >10 mm[21] with a consistent position of the knee, namely, 90° of flexion with the quadriceps relaxed and the foot free (open chain). Different practitioners have not tested these measurements in a blinded and repeated manner on the same patients to determine inter- and intra-observer bias. Similarly, measurements of mid-flexion instability and medial-lateral instability are currently not quantitative. These also may be affected by pain and muscle inhibition. The amount of inherent stability of a knee system that leads to good clinical results may also vary by prosthesis design. Anterior-posterior translation of 5 to 10 mm is normal for some patients, as suggested by the improved subjective outcomes at 2 years in those who had CR TKAs.[33] The ability to obtain reproducible measurements will remain paramount to appropriately diagnosing and treating patients with painful knee arthroplasties.

Stress Radiography and Laxity Testing

Although ACL and PCL laxity can be routinely measured after ligament reconstruction, this has not been adopted after knee arthroplasty. Stress radiography may allow us to unite flexion instability calculations. After TKA, Seon et al[34] have described a protocol in which a Telos stress device (Austin & Associated) is used to apply a consistent anterior force of 150 N to the tibia with the knee in 90° of flexion. They obtained lateral radiographs of the knee before and during force application, with measurements of displacement determined as the horizontal distance between a vertical line from the posterior aspect of the tibial tray and a vertical line from the posterior aspect of the femoral condyle. When reviewing 46 CR TKAs of a single manufacturer (Aesculap e-motion) placed with navigation, the same authors reported an average sagittal translation of 7.1 mm (SD, 4.1 mm). Of interest, they found that improved postoperative range of motion was correlated with anterior-posterior translation >5 mm, similar to that of earlier studies.[35] Their study was unable to determine an upper limit of anterior-posterior motion before impairment in joint function and stability.

Future efforts should focus on comparing patients with symptoms of flexion instability to sagittal displacement on stress measurements. In addition to Telos, other arthrometers have been studied in the biomechanics literature. The KT-1000 is a device used to measure the sum of the anterior and posterior tibial translations with the knee in 20° to 30° of flexion with 97 N force constantly applied.[33] The GNRB arthrometer is a device that gradually increases load from zero to 250 N over the tibial tubercle with the knee in 20° of flexion and digitally measures the posterior displacement from the tubercle to the anterior femur.[36] The Rolimeter measures anterior tibial translation when the physician applies a maximum Lachman force to the tibia with the knee in 20° of flexion. A theoretic benefit of such devices is that they do not expose the patient to undue radiation to compute displacement because radiography is not required. However, previous protocols for the use of these devices often called for relaxation of the limb to eliminate patient guarding, which detracts from their ease of use and generalizability for patients with painful knee arthroplasties. To date, the KT-1000, GNRB, and Rolimeter devices have not been tested specifically for flexion instability knee arthroplasty and have predominantly been used to measure knee laxity in patients with cruciate ligament injuries.

Motion-tracking sensors and wearable accelerometers have also gained popularity and could provide details regarding functional instability during certain movements and help establish thresholds for patient perceptions of laxity.[37]

Biomechanical Testing and Computational Modeling

Knee stability comprises three general mechanisms that have markedly interplay: articular congruity, ligamentous constraint, and muscle activation.[38] Because of varying implant designs and differing baseline laxity grades and muscle strength among individuals, knee stability after TKA may actually be a patient-specific occurrence. Recent research has shown that quadriceps and hamstrings can co-contract to generate added compressive forces across the knee joint in the first year after TKA that may improve joint stability.[39] Therefore, future research needs to quantify differences in quadriceps and hamstring co-contraction and determine differences in knees with and without flexion instability. Combining efforts with computational modeling to create simulations that incorporate ligamentous structure and balancing, implant design, implant positioning, and coordinated muscle contractions and strength measures will allow for an improved understanding of the relative contributions each of these domains has in the normal and physiologically unstable TKA.[7] These efforts can further be advanced with kinematic gait and motion analysis studies to elucidate differences in loading patterns and step length between those with and without flexion instability after knee arthroplasty.[40]

Summary

The experience of flexion instability after TKA is likely a patient-specific phenomenon that depends on the interaction of a multitude of implant, surgical, and host factors. Patients often present with recurrent effusions, subjective instability quadriceps weakness, and diffuse knee pain. The mainstay of nonsurgical treatment involves isometric quadriceps strengthening. Revision surgery is frequently needed to increase femoral implant offset to tighten the flexion space to achieve balanced flexion and extension gaps. This process usually entails a full revision TKA because increasing the polyethylene bearing size alone leads to persistent gap imbalance. Patient outcomes after revision TKA for flexion instability show the least amount of improvement when compared with revisions for other TKA failure etiologies.

To better care for patients with flexion instability, unified diagnostic criteria must be established, which consider the weighted contributions of the amount of sagittal laxity, radiographic positioning and alignment of implants, and the input of the surrounding muscles as secondary stabilizers. This phenomenon will allow future studies to determine whether any subset of flexion unstable patients can improve with nonsurgical measures and lead to comparisons of surgical strategies to enhance patient outcomes.

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