Patellofemoral Crepitation and Clunk Complicating Posterior-Stabilized Total Knee Arthroplasty

Brian P. Gladnick, MD, and Alejandro González Della Valle, MD

Abstract

Patellofemoral crepitation and clunk (PCC) is an important potential complication of total knee arthroplasty. Numerous factors, including implant design, range of motion, and certain radiographic parameters, may contribute to the development of PCC. Although the diagnosis is primarily clinical, imaging modalities may be helpful in cases of diagnostic uncertainty. Arthroscopic débridement is the preferred method of treating PCC in patients whose symptoms require intervention. A full understanding of the diagnosis, management, and prevention of PCC is essential for orthopedic surgeons who perform total knee arthroplasties.

Patellofemoral crepitation and clunk (PCC) is an infrequent complication of posterior-stabilized total knee arthroplasty (TKA).[1,2] The inciting factor is the formation of proliferative, synovial-like fibrous tissue at the superior pole of the resurfaced patella. This tissue can grow to become a voluminous suprapatellar nodule that engages the intercondylar notch of the femoral component as the knee moves into deep flexion (**Figure 1**). Subsequently, as the knee is actively extended, the fibrous nodule disengages from the notch, causing symptoms that range from minor crepitation to a painful, palpable, and sometimes audible clunk.[2] Thus, PCC is best thought of as a wide spectrum of symptoms emanating from the same disease process. At one end of the spectrum, the symptoms are limited to minor but discomforting crepitation during knee extension; at the other extreme, the symptoms may present as painful, disabling clunking. However, the entire range of symptoms is related to the same pathologic development of a fibrous suprapatellar nodule and therefore, for the purposes of this review article, is considered together as PCC.[1,2]

PCC incidence ranges from 0% to 25%.[3-6] Various factors, the most recognized being the femoral component design, have been implicated in the development of symptoms. Some authors have reported reduced PCC incidence after design changes in the femoral component.[4,6-14] In addition, factors such as knee range of motion (ROM)[8,13,14] and certain radiographic TKA parameters have been associated with increased likelihood of developing PCC.[5,12,14]

The range of treatment options includes observation (benign neglect), nonoperative management centered on activity modification and pain control, and surgical resection of the suprapatellar lesion.[15-17]

In this review article, we discuss the factors commonly accepted as contributing to the development of PCC, describe the clinical presentation and diagnosis of PCC, and review the efficacy of treatment options. A full understanding of the diagnosis, management, and prevention of PCC is essential for orthopedic surgeons who perform TKAs.

Etiology

The Role of Prosthetic Design

The incidence of PCC as reported in the literature varies widely. Much of this variation has been attributed to the prosthetic design. Several investigators have postulated that femoral components with an increased intercondylar box ratio (ratio of intercondylar box size to anteroposterior component size), a shorter trochlea, and a sharp transition into the intercondylar notch have a higher incidence of PCC.[7,8,10,12]

The Insall-Burstein II prosthesis (IB-II; Zimmer, Warsaw, Indiana), one of the early "historic" TKA implants, can be used to describe the main design features that contribute to the development of PCC.[4,8,15,18] The IB-II femoral component had a shallow trochlear groove with no lateral buildup, which is thought to result in suboptimal patellar tracking. In addition, this component had a wide intercondylar box that extended throughout most of its anterior-posterior dimension (**Figure 2**). It is postulated that this design feature facilitated the development of PCC, as the suprapatellar nodule would engage easily into the capacious intercondylar box.[4,9]

Modifying the geometry of the femoral box may alter the incidence of PCC symptoms after TKA. Ranawat and colleagues[19,20] retrospectively evaluated PCC symptoms in 2 series of patients undergoing TKA: 1 with the IB-II prosthesis and 1 with the PFC Modular Knee (DePuy Orthopaedics, Warsaw,

Authors' Disclosure Statement: The authors report no actual or potential conflict of interest in relation to this article.

Indiana). The incidence of painful crepitation was 21% in the IB-II series and 3% in the PFC series. The authors suggested that this significant reduction in PCC symptoms was related to design improvements in the trochlear groove of the PFC femoral component.

Ip and colleagues[9] retrospectively compared 3 consecutive series of TKAs: 1 using the IB-II (n = 80), 1 using the Anatomic Modular Knee (AMK, DePuy; n = 60), and 1 using the Low Contact Stress rotating platform (LCS, DePuy; n = 106). PCC incidence was lower with the AMK (3.3%) and the LCS (0.0%) than with the IB-II (8.8%); the latter difference was significant ($P < .01$). The authors attributed this decreased incidence to design improvements in the femoral component. Specifically, the AMK femoral component has a narrower intercondylar notch, and the LCS lacks a discrete intercondylar notch. These modifications likely prevented fibrous tissue at the superior patellar pole from engaging the notch, thereby reducing the incidence of PCC.

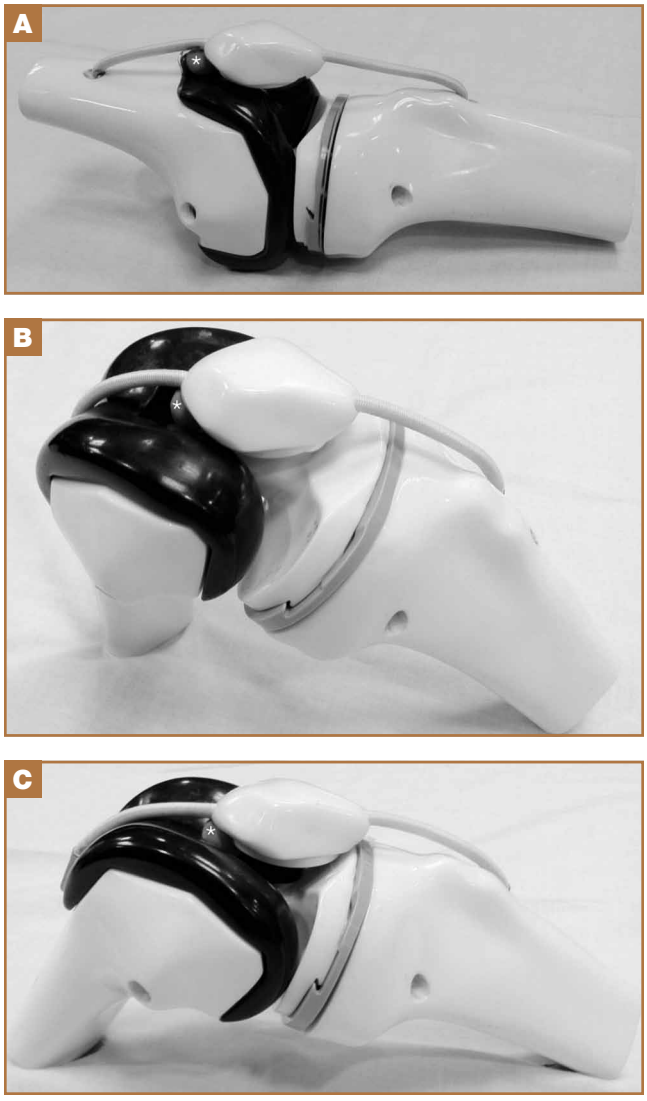
Anderson and colleagues[6] retrospectively compared PCC incidence with the Axiom PSK (n = 20) and the Advance Medial-Pivot (AMP; n = 20), both from Wright Medical Technology (Arlington, Tennessee). The Axiom is a traditional cam-and-post cruciate-substituting prosthesis with a shallow trochlear groove and a traditional open box femoral design. The AMP, which has a recessed trochlear groove, eliminates the large femoral box ("closed box," or no-box femoral design). Moreover, in the AMP, the trochlear groove is extended inferiorly, oriented in a slightly lateral direction, and raised on the lateral side—all design modifications intended to improve patellar tracking. Incidence of PCC symptoms was 25% with the Axiom and 0% with the AMP ($P = .0079$). The authors stated that the elimination of the intercondylar box and the improvements made to the patellofemoral geometry of the AMP ensure that the patella never leaves the trochlea of the femur until the knee is in deep flexion, thereby preventing fibrous tissue at the superior patellar pole from impinging on the femoral component.

On the basis of these studies, femoral components may be classified into 3 major types, involving the characteristics of the intercondylar box. First, original or historic designs (eg, IB, IB-II) have femoral components with an "open box."[2,5,7,12,15,16,21,22] These designs typically have wide, capacious intercondylar boxes that are more anteriorly positioned (**Figure 2**). Second, modern, "modified" components have intercondylar box design changes intended to limit the ability of the suprapatellar fibrous tissue to impinge on the femoral component.[4,8,10,11,13,14,18,23-25] These designs have narrower intercondylar boxes positioned more posteriorly (**Figure 3**). Third, closed-box designs or no-box femoral components[3,6,9,26] are similar to the cruciate-retaining femoral components in that they lack the discrete intercondylar box found in more traditional posterior-stabilized TKAs. Rather than using a post-and-cam mechanism, these designs often rely on other means (eg, enhanced congruity with a deep-dish polyethylene tray) to control anterior-posterior stability (**Figure 4**). The **Table** summarizes the literature on PCC incidence after TKA performed with different femoral component designs. Historic open-box designs (type I) have the highest PCC rates, modified intercondylar box designs (type II) have lower PCC rates, and the closed-box designs (type III) essentially eliminate the complication (**Figure 5**).

The Role of Range of Motion

Multiple authors have suggested that increased knee ROM after TKA predisposes to the development of PCC.[8,13,14,27] This makes intuitive sense, as knee flexion must necessarily reach a certain threshold angle at which the patella has traveled sufficiently far down the trochlear groove to allow fibrous tissue at the superior pole to engage the intercondylar notch.

Figure 1. (A) In full extension, suprapatellar nodule (*) is well clear of intercondylar box and does not cause mechanical symptoms. (B) When knee moves into high flexion, nodule engages intercondylar box. (C) When knee is subsequently moved back into extension, nodule impinges on intercondylar box, eliciting symptoms of patellar crepitation and clunk.



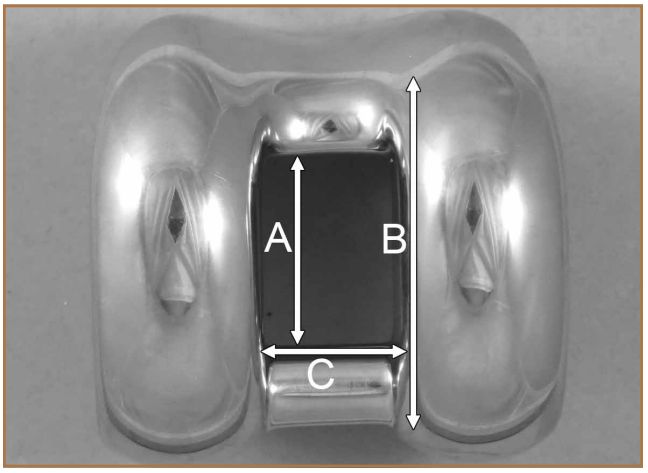


Figure 2. Femoral component of Insall-Burstein II prosthesis (Zimmer, Warsaw, Indiana). Intercondylar box extends far anteriorly (A) and is wide (C). Intercondylar box ratio (A:B) is relatively large.

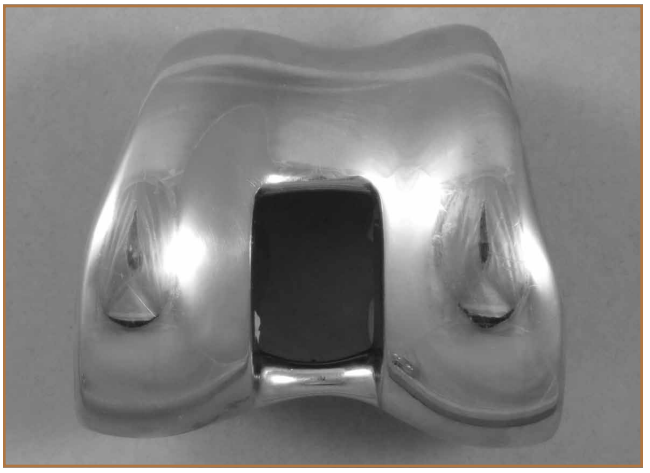


Figure 3. Modified femoral box of Genesis II prosthesis (Smith & Nephew, Memphis, Tennessee) is narrower and more posteriorly situated than more historical designs (eg, Insall-Burstein II).

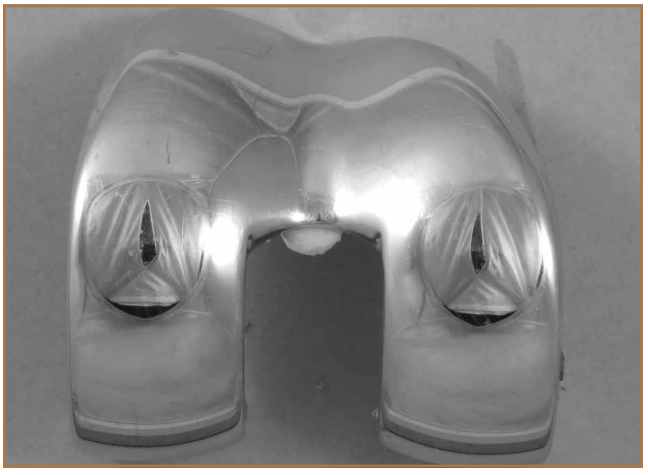


Figure 4. Femoral component of Low Contact Stress rotating platform (DePuy Orthopaedics, Warsaw, Indiana). Like cruciate-retaining designs, component lacks discrete intercondylar box.

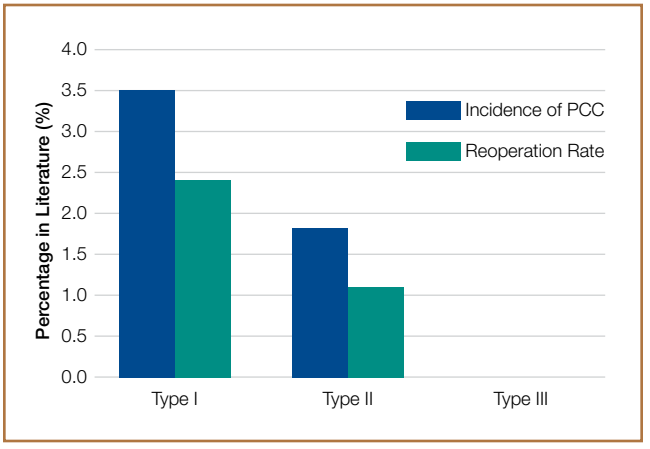


Figure 5. Incidence of patellofemoral crepitation and clunk (PCC) and rates of resulting reoperation in 3 types of femoral component designs: type I, historic open-box design; type II, modified intercondylar box design; type III, closed-box design.

Frye and colleagues[14] conducted a retrospective comparative study using the PFC Sigma prosthesis (Depuy). They studied the incidence of PCC before and after design modifications were made in the femoral component (trochlear groove deepening, smoother transition into intercondylar notch). These design changes appeared to eliminate PCC. In addition, the authors noted that average flexion angle was higher in patients who reported PCC symptoms (114°) than in patients who did not develop PCC (107°) ($P < .001$). The data suggested an association of ROM, prosthetic design, and development of PCC.

Schroer and colleagues[13] also retrospectively demonstrated that PCC incidence is associated with higher ROM after TKA. Of 275 patients undergoing TKA with the Ascent PS prosthesis (Biomet, Warsaw, Indiana), 17 (6%) developed PCC. At 24-week follow-up, average flexion angle was higher in these patients with PCC (131°) than in the patients who did not develop PCC (124°) ($P < .001$).

We recently retrospectively evaluated 570 TKAs that used the Genesis II TKA prosthesis (Smith & Nephew, Memphis, Tennessee).[27] Unadjusted logistic regression analysis revealed that, with each degree increase in flexion angle, the likelihood of developing PCC increased by 4.2% (OR, 1.042; 95% CI, 1.004-1.081). Our most recent extended analysis of 648 patients revealed that patients with a flexion angle above 120° had a 12% rate of this complication (**Figure 6**). This evidence supports the idea that increased ROM after TKA increases the risk of developing PCC.

The Role of Radiographic Parameters

It has been suggested that some postoperative TKA radiographic parameters have an association with likelihood of developing PCC. Yau and colleagues[5] retrospectively reviewed

Table. **Analysis of Reported Rates of Patellofemoral Crepitation and Clunk in the Arthroplasty Literature**

Author	Year	Knees, N	Prosthesis[a]	Overall PCC		Reoperation		Box Design Type[b]
				n	%	n	%	
Beight et al[2]	1994	1484	IB	20	1.3%	14	0.9%	I
Aglietti et al[21]	1996	51	IB	3	5.9%	1	2.0%	I
Shoji & Shimozaki[3]	1996	372	Y/S II	0	0.0%	0	0.0%	III[c]
		275	AGC	0	0.0%	0	0.0%	III[c]
Larson & Lachiewicz[22]	1999	118	IB-II	0	0.0%	0	0.0%	I
Lucas et al[15]	1999	900	IB-II	32	3.5%	32	3.5%	I
Ip et al[4]	2002	80	IB-II	6	7.5%	5	6.3%	I
		50	NexGen	0	0.0%	0	0.0%	II
Anderson et al[6]	2002	20	Axiom PSK	5[d]	25%	1	5.0%	I
		20	AdvanceMP	0	0.0%	0	0.0%	III
Pollock et al[7]	2002	212	AMK-PS	NR	NR	8	3.8%	I
		141	AMK-C	NR	NR	19	13.5%	I
		106	PFC Sigma	NR	NR	0	0.0%	I
Maloney et al[8]	2003	179	IB-II	7	3.9%	5	2.8%	I
		210	AdvancePS	0	0.0%	0	0.0%	II
Ip et al[23]	2003	60	NexGen	0	0.0%	0	0.0%	II
Yau et al[5]	2003	124	AMK-PS	17	13.7%	NR	NR	I
		112	IB	10	8.9%	NR	NR	I
Ip et al[9]	2004	80	IB-II	7	8.8%	0	0.0%	I
		60	AMK-PS	2	3.3%	1	1.7%	II
		106	LCS	0	0.0%	0	0.0%	III
Clarke et al[10]	2006	238	NexGen	0	0.0%	0	0.0%	II
Kolisek & Barnes[24]	2006	103	Scorpio PS	1	1.0%	NR	NR	II
Lonner et al[11]	2007	150	IB	6	4.0%	6	4.0%	I
		150	NexGen	0	0.0%	0	0.0%	II
Koh et al[16]	2008	1020	PFC Sigma	14	1.4%	14	1.4%	I
Anderson et al[18]	2008	300	IB-II	12	4.0%	NR	NR	I
		300	Optetrak	1	0.3%	NR	NR	II
Schroer et al[13]	2009	498	Ascent PS	18	3.6%	NR	NR	I
		250	Vanguard	1	0.4%	NR	NR	II
Fukunaga et al[12]	2009	113	Sigma RP	15	13.3%	5	4.4%	I
Dajani et al[17]	2010	546	NR[e]	NR	NR	25	4.6%	NR
Zmistowski et al[31]	2011	10,188	NR	NR	NR	53	0.5%	NR
Frye et al[14]	2012	108	PFC Sigma	13	12.0%	4	3.7%	I
		136	PFC Sigma[f]	0	0.0%	0	0.0%	II
Nam et al[26]	2012	24	Sigma RPF	2	8.3%	1	4.2%	I
		26	LCS	0	0.0%	0	0.0%	III
Choi et al[25]	2013	424	NexGen F	NR	NR	6	1.4%	II
		135	Scorpio PS	NR	NR	0	0.0%	II
		113	Sigma RPF	NR	NR	11	9.7%	I
		96	Genesis II	NR	NR	1	1.0%	II
		58	e.motion	NR	NR	0	0.0%	II
Present study	—	570	Genesis II	34	6.0%	6	1.0%	II

Abbreviations: PCC, patellofemoral crepitation and clunk; NR, none reported.

[a]IB indicates Insall-Burstein prosthesis (Zimmer); Y/S II, Y/S II total condylar prosthesis (Biomet); AGC, AGC total condylar prosthesis (Biomet); IB-II, Insall-Burstein II prosthesis (Zimmer); NexGen, NexGen Legacy PS (Zimmer); Axiom PSK, Axiom PSK (Wright Medical); AdvanceMP, Advance Medial-Pivot total knee arthroplasty (Wright Medical); AMK-PS, Anatomic Modular Knee–Posterior Stabilized (DePuy); AMK-C, Anatomic Modular Knee–Congruency (DePuy); PFC Sigma, Press Fit Condylar Sigma–Posterior Stabilized (DePuy); AdvancePS, Advance Posterior Stabilized total knee arthroplasty (Wright Medical); LCS, Low Contact Stress rotating platform (DePuy); Scorpio PS, Scorpio posterior-stabilized knee system (Stryker); Optetrak, Optetrak (Exactech); Ascent PS, Ascent posterior-stabilized total knee arthroplasty (Biomet); Vanguard, Vanguard posterior-stabilized total knee arthroplasty (Biomet); Sigma RP, PFC Sigma Rotating Platform (Depuy); Sigma RPF, PFC Sigma Rotating Platform–Flex Knee System (Depuy); NexGen F, NexGen Legacy PS–Flex Knee System (Zimmer); Genesis II, Genesis II total knee arthroplasty (Smith & Nephew); e.motion, e.motion Total Knee System (B Braun Aesculap).

[b]I indicates historical "open box" design; II, modified modern "open box" design; III, "closed box" or no box design.

[c]Cruciate-retaining design.

[d]One patient had both box impingement and crepitus reported in same knee.

[e]Study used at least 3 different manufacturers but did not specify individual designs.

[f]Design change in femoral component of system.

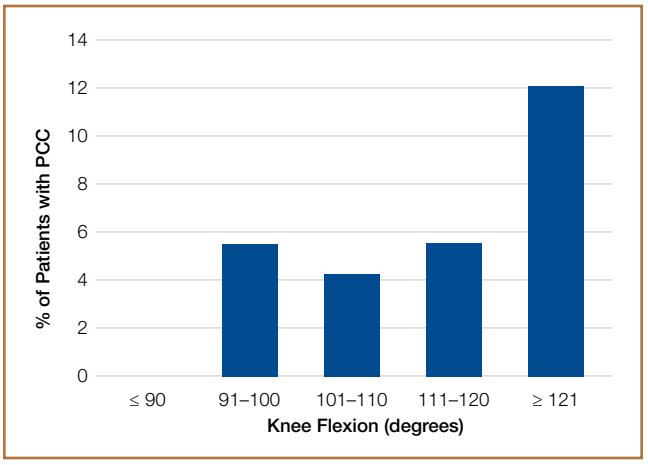


Figure 6. Association of patellofemoral crepitation and clunk (PCC) with knee flexion angle.

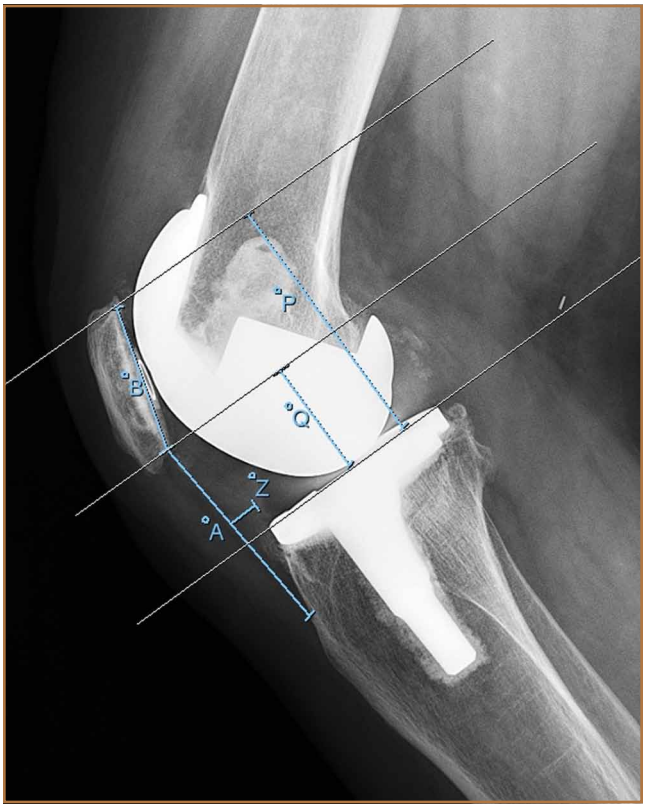


Figure 7. Common measurements associated with patellofemoral crepitation and clunk on lateral knee radiographs: Insall-Salvati ratio (A:B), anterior-posterior placement of tibial tray (Z), patellar button height (Q), and distance from proximal patella to distal femur (P).

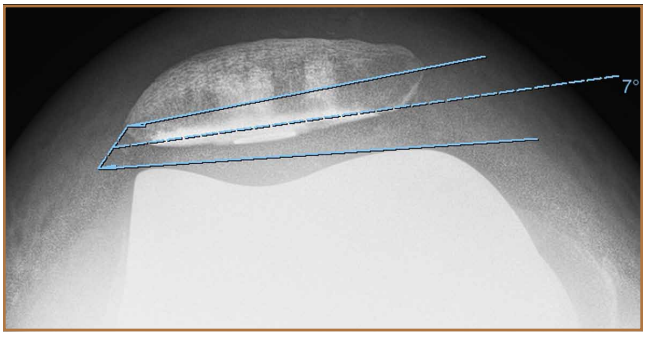


Figure 8. Lateral patellar tilt measured on skyline patellar radiographs.

radiographs of 27 TKAs with PCC symptoms and compared them with radiographs of controls without PCC. The groups differed significantly with regard to multiple radiographic parameters. For example, the position of the tibial component on lateral radiographs (**Figure 7**) was an average of 1.49 mm more anterior in the knees of patients with PCC than in the knees of patients without PCC ($P = .005$). This finding is consistent with an association reported by Figgie and colleagues[28]: between crepitation and catching symptoms at terminal extension and anterior placement of the tibial tray.

Three other radiographic parameters studied by Yau and colleagues[5] indicate a possible association between patellar height and likelihood of developing PCC (**Figure 7**). First, the Insall-Salvati ratio was lower in knees of patients with PCC symptoms (0.84) than in knees of patients without PCC (0.97) ($P < .001$). Second, the distance from the proximal pole of the patella to the distal aspect of the femur was shorter in knees with PCC (47.04 mm) than in knees without PCC (49.83 mm) ($P = .045$). Third, patellar button height (measured from distal aspect of femur to inferior edge of patellar button) was less in knees with PCC (12.09 mm) than in knees without PCC (15.49 mm) ($P = .008$). These data indicate that a low-lying patella may predispose to developing PCC, and the authors suggested this may be secondary to excessive cutting of the distal femur, leading to relative distal migration of the patella.

Yau and colleagues[5] also found group differences ($P = .019$) in lateral patellar tilt (PCC, 9.59°; non-PCC, 5.95°) (**Figure 8**). Similar findings were reported by Frye and colleagues.[14] Support for these data were provided by a logistic regression analysis performed by Fukunaga and colleagues,[12] who found that a 1° increase in postoperative patellar tilt was associated with a 1.27-fold increased incidence of PCC (OR, 1.27; 95% CI, 1.03-1.56). This finding suggests that optimization of patellar tracking could minimize development of postoperative PCC.

There is some debate as to whether TKA radiographic parameters are predictive of PCC symptoms. In our recent retrospective study, [27] we found no association between PCC incidence and 10 radiographic parameters. On radiographs of patients with PCC and radiographs of matched controls without PCC, we measured preoperative and postoperative Insall-Salvati ratio, preoperative and postoperative joint-line position, preoperative and postoperative knee mechanical alignment, flexion of femoral component, posterior tibial slope, anterior-posterior position of tibial tray on lateral radiographs, and patellar indices (tilt, thickness, and lateral displacement). No associations were found between PCC incidence and any of

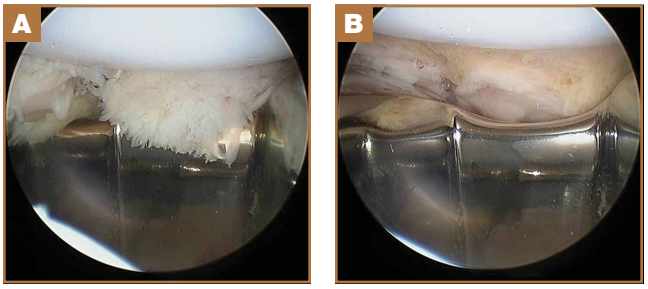


Figure 9. Arthroscopic débridement for patellofemoral crepitation and clunk. (A) Hypertrophic fibrous nodule is visualized at superior pole of patella. (B) After arthroscopic shaving and cautery, nodule has been removed.

the radiographic parameters analyzed. Thus, controversy still exists as to whether commonly used radiographic parameters may reliably predict symptoms of PCC.

Diagnosis and Clinical Evaluation

Clinical Findings

The diagnosis of PCC is usually based on patient history and physical examination. Presentation ranges from a few months to several years after surgery.[27] Patients complain of patellofemoral pain arising in an otherwise normally functioning TKA—including a sense of catching, locking, or crepitation of the knee during attempts to rise from a chair or actively extend the knee from a flexed position. On examination, patients usually demonstrate a stable knee with normal ROM and no obvious subluxation of the patella. There is no associated joint effusion or warmth, and the offending suprapatellar nodule is not palpable. With the patient in the supine decubitus position, the hip at 90° of flexion, and the knee in full gravity-assisted flexion, the clunk or crepitation and its accompanying symptoms can be elicited by asking the patient to actively extend the knee. As the knee approaches 30° to 45° from terminal extension, a sudden, painful "jump" or "pop" is produced as the suprapatellar nodule disengages from the intercondylar notch. This clunking is easily palpable and may even be audible from across the room.[1,2] It is important to emphasize that passive knee extension that negates the quadriceps function does not reveal the clunk.

Imaging

Although the PCC diagnosis is based on the pathognomonic clinical findings just mentioned, radiographs should be obtained to rule out prosthetic loosening, fractures, polyethylene wear, and other potential sources of knee pain. Cineradiography or dynamic fluoroscopy can be used to demonstrate the abnormal patellar motion that is appreciated on physical examination, but these imaging systems do not visualize the fibrotic suprapatellar nodule.[1] The lesion may be detectable on Doppler sonography, but this modality is not routinely used.[29]

In a recent retrospective study at our institution, Heyse and colleagues[30] found that PCC can be reliably seen on magnetic resonance imaging (MRI). Using an artifact-reducing modified MRI protocol, the authors successfully imaged a fibrotic suprapatellar nodule in 9 of 12 patients presenting with symptoms suggestive of patellar clunk. Although MRI is not required for diagnosis, MRI confirmation of the presence of a hypertrophic suprapatellar lesion may be useful in cases in which the diagnosis is questionable.

Treatment

Observation

Conservative treatment is a reasonable first-line option for patients who present with PCC symptoms. Ip and colleagues[9] reported the natural history of 8 PCC patients who refused operative intervention. At 4-year follow-up, half the patients had symptomatic improvement; the other half continued to have disabling patellofemoral symptoms. Potential nonoperative interventions include quadriceps strengthening, oral anti-inflammatory medications, and injections.[1,2] Activity modification, including avoiding activities that involve active extension of the knee from a high flexion position, can also be considered.[9] Patients should realize that these modalities are designed to manage PCC symptoms but are not necessarily curative.

Débridement and Synovectomy

Arthroscopic débridement of the fibrous suprapatellar nodule is the most commonly accepted definitive treatment for PCC.[15-17] A shaver and a radiofrequency ablation terminal are used to thoroughly débride the lesion while achieving rigorous hemostasis (**Figure 9**). The arthroscopic examination of the nodule and the general diagnostic arthroscopy can be performed through a lateral parapatellar portal. For instrumentation, we prefer using a lateral suprapatellar portal and, if necessary, a medial parapatellar portal. The arthroscope and the instruments should be alternated between the portals to promote visualization and to ensure complete débridement. At the end of the procedure, the patellofemoral articulation should be inspected, with the knee flexed and extended, to ensure there is no residual synovial impingement.[27] To simulate active quadriceps function under anesthesia, the surgeon should extend the knee while forcing the superior pole of the patella down into the femoral box. This gesture should be performed in the operating room, with the patient under anesthesia, before and after resection of the lesion. Rates of PCC recurrence after arthroscopic débridement range from 0% to 27%, and PCC may present as late as 48 months after the original débridement.[2,7,15-17]

Dajani and colleagues[17] used arthroscopic débridement to treat 15 patients with clinically reproducible patellar clunk and noted statistically significant improvement in Knee Society Score (KSS) Knee ($P < .0001$) and Function ($P = .017$) subscores. All patients reported postoperative improvement in symptoms and stated they would have the surgery again. Two patients (13%) required a second, open débridement for recurrent mechanical symptoms. Choi and colleagues[25] reported PCC symptoms in 18 patients from a series of 826 TKAs using 5 different implant systems. Of these 18 patients, 16 underwent arthroscopic resection of the fibrotic nodule. The other 2 patients did not have the patella resurfaced at the time

of their original TKAs; open synovectomy and patellar resurfacing were performed. All 18 patients subsequently showed improvement in KSS scores, Hospital for Special Surgery Knee scores, and Western Ontario and McMaster Universities Arthritis Index scores (all Ps < .001). No recurrences were found over an average follow-up of 29 months. Overall, the current data support arthroscopic débridement as an initial treatment for PCC. However, for patients who did not have patellar resurfacing at the time of original TKA, or whose PCC symptoms have recurred despite arthroscopic débridement, open synovectomy and débridement of the nodule should be considered.

Summary and Conclusions

PCC is an important and modifiable complication of posterior-stabilized TKA. Incidence of PCC is primarily related to the geometry of the femoral component, particularly of the intercondylar box, though ROM and certain radiographic parameters may also affect the ability of the suprapatellar nodule to impinge on the femoral component. PCC is classically diagnosed from its clinical presentation, but MRI can be used if the diagnosis is elusive. Arthroscopic débridement is the current definitive treatment for PCC. However there is a role for open synovectomy in the case of recurrence or for the rare patient who presents with PCC and an unresurfaced patella.

Dr. Gladnick is Resident, and Dr. González Della Valle is Associate Professor of Orthopaedic Surgery, Department of Orthopaedic Surgery, Hospital for Special Surgery, New York, New York.

Acknowledgment: This study was partially funded by the generous donation of Glenn Bergenfield, Muriel Bayroff, and the Sidney Milton and Leoma Simon Foundation.

Address correspondence to: Brian P. Gladnick, MD, Department of Orthopaedic Surgery, Hospital for Special Surgery, 535 E 70th St, New York, NY 10021 (tel, 212-774-7124; fax, 212-774-7505; e-mail, gladnickb@hss.edu).

Am J Orthop. 2014;43(10):458-464.

References

1. Hozack WJ, Rothman RH, Booth RE Jr, Balderston RA. The patellar clunk syndrome. A complication of posterior stabilized total knee arthroplasty. *Clin Orthop*. 1989;(241):203-208.
2. Beight JL, Yao B, Hozack WJ, Hearn SL, Booth RE Jr. The patellar "clunk" syndrome after posterior stabilized total knee arthroplasty. *Clin Orthop*. 1994;(299):139-142.
3. Shoji H, Shimozaki E. Patellar clunk syndrome in total knee arthroplasty without patellar resurfacing. *J Arthroplasty*. 1996;11(2):198-201.
4. Ip D, Wu WC, Tsang WL. Comparison of two total knee prostheses on the incidence of patella clunk syndrome. *Int Orthop*. 2002;26(1):48-51.
5. Yau WP, Wong JW, Chiu KY, Ng TP, Tang WM. Patellar clunk syndrome after posterior stabilized total knee arthroplasty. *J Arthroplasty*. 2003;18(8):1023-1028.
6. Anderson MJ, Becker DL, Kieckbusch T. Patellofemoral complications after posterior-stabilized total knee arthroplasty: a comparison of 2 different implant designs. *J Arthroplasty*. 2002;17(4):422-426.
7. Pollock DC, Ammeen DJ, Engh GA. Synovial entrapment: a complication of posterior stabilized total knee arthroplasty. *J Bone Joint Surg Am*. 2002;84(12):2174-2178.
8. Maloney WJ, Schmidt R, Sculco TP. Femoral component design and patellar clunk syndrome. *Clin Orthop*. 2003;(410):199-202.
9. Ip D, Ko PS, Lee OB, Wu WC, Lam JJ. Natural history and pathogenesis of the patella clunk syndrome. *Arch Orthop Trauma Surg*. 2004;124(9):597-602.
10. Clarke HD, Fuchs R, Scuderi GR, Mills EL, Scott WN, Insall JN. The influence of femoral component design in the elimination of patellar clunk in posterior-stabilized total knee arthroplasty. *J Arthroplasty*. 2006;21(2):167-171.
11. Lonner JH, Jasko JG, Bezwada HP, Nazarian DG, Booth RE Jr. Incidence of patellar clunk with a modern posterior-stabilized knee design. *Am J Orthop*. 2007;36(10):550-553.
12. Fukunaga K, Kobayashi A, Minoda Y, Iwaki H, Hashimoto Y, Takaoka K. The incidence of the patellar clunk syndrome in a recently designed mobile-bearing posteriorly stabilised total knee replacement. *J Bone Joint Surg Br*. 2009;91(4):463-468.
13. Schroer WC, Diesfeld PJ, Reedy ME, LeMarr A. Association of increased knee flexion and patella clunk syndrome after mini-subvastus total knee arthroplasty. *J Arthroplasty*. 2009;24(2):281-287.
14. Frye BM, Floyd MW, Pham DC, Feldman JJ, Hamlin BR. Effect of femoral component design on patellofemoral crepitance and patella clunk syndrome after posterior-stabilized total knee arthroplasty. *J Arthroplasty*. 2012;27(6):1166-1170.
15. Lucas TS, DeLuca PF, Nazarian DG, Bartolozzi AR, Booth RE Jr. Arthroscopic treatment of patellar clunk. *Clin Orthop*. 1999;(367):226-229.
16. Koh YG, Kim SJ, Chun YM, Kim YC, Park YS. Arthroscopic treatment of patellofemoral soft tissue impingement after posterior stabilized total knee arthroplasty. *Knee*. 2008;15(1):36-39.
17. Dajani KA, Stuart MJ, Dahm DL, Levy BA. Arthroscopic treatment of patellar clunk and synovial hyperplasia after total knee arthroplasty. *J Arthroplasty*. 2010;25(1):97-103.
18. Anderson JA, Baldini A, Sculco TP. Patellofemoral function after total knee arthroplasty: a comparison of 2 posterior-stabilized designs. *J Knee Surg*. 2008;21(2):91-96.
19. Ranawat AS, Ranawat CS, Slamin JE, Dennis DA. Patellar crepitation in the P.F.C. sigma total knee system. *Orthopedics*. 2006;29(9 suppl):S68-S70.
20. Ranawat CS, Luessenhop CP, Rodriguez JA. The press-fit condylar modular total knee system. Four-to-six-year results with a posterior-cruciate-substituting design. *J Bone Joint Surg Am*. 1997;79(3):342-348.
21. Aglietti P, Buzzi R, Giron F, Zaccherotti G. The Insall-Burstein posterior stabilized total knee replacement in the valgus knee. *Am J Knee Surg*. 1996;9(1):8-12.
22. Larson CM, Lachiewicz PF. Patellofemoral complications with the Insall-Burstein II posterior-stabilized total knee arthroplasty. *J Arthroplasty*. 1999;14(3):288-292.
23. Ip D, Wu WC, Tsang WL. Early results of posterior-stabilised NexGen Legacy total knee arthroplasty. *J Orthop Surg*. 2003;11(1):38-42.
24. Kolisek FR, Barnes CL. Scorpio posterior-stabilized knee system: 5-year clinical and functional results. *J Arthroplasty*. 2006;21(8):1187-1192.
25. Choi WC, Ryu KJ, Lee S, Seong SC, Lee MC. Painful patellar clunk or crepitation of contemporary knee prostheses. *Clin Orthop*. 2013;471(5):1512-1522.
26. Nam SW, Lee YS, Kwak JH, Kim NK, Lee BK. A comparison of the clinical and radiographic results of press fit condylar rotating-platform high-flexion and Low Contact Stress mobile bearing prosthesis in total knee arthroplasty: short term results. *Knee Surg Relat Res*. 2012;24(1):7-13.
27. Peralta-Molero JV, Gladnick BP, Lee Y, Lyman S, Della Valle AG. Patellofemoral crepitation and clunk following modern, fixed bearing total knee arthroplasty. *J Arthroplasty*. 2014;29(3):535-540.
28. Figgie HE 3rd, Goldberg VM, Heiple KG, Moller HS 3rd, Gordon NH. The influence of tibial-patellofemoral location on function of the knee in patients with the posterior stabilized condylar knee prosthesis. *J Bone Joint Surg Am*. 1986;68(7):1035-1040.
29. Okamoto T, Futani H, Atsui K, Fukunishi S, Koezuka A, Maruo S. Sonographic appearance of fibrous nodules in patellar clunk syndrome: a case report. *J Orthop Sci*. 2002;7(5):590-593.
30. Heyse TJ, Chong le R, Davis J, Haas SB, Figgie MP, Potter HG. MRI diagnosis of patellar clunk syndrome following total knee arthroplasty. *HSS J*. 2012;8(2):92-95.
31. Zmistowski B, Restrepo C, Kahl LK, Parvizi J, Sharkey PF. Incidence and reasons for nonrevision reoperation after total knee arthroplasty. *Clin Orthop*. 2011; 469(1):138-145.

This paper will be judged for the Resident Writer's Award.