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# Patellofemoral Crepitus after Total Knee Arthroplasty: Etiology and Preventive Measures

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Patellofemoral crepitus and clunk syndrome are infrequent, yet troublesome complications of total knee arthroplasty with a reported incidence of 0%–18%. They are primarily associated with implantation of posterior cruciate substituting designs. These entities are the result of peripatellar fibrosynovial hyperplasia at the junction of the superior pole of the patella and the distal quadriceps tendon which becomes entrapped within the superior aspect of the intercondylar box of the femoral component during knee flexion. When the knee extends, a crepitant sensation occurs as the fibrosynovial tissue exits the intercondylar box. Numerous etiologies have been proposed such as femoral component designs with a high intercondylar box ratio, previous knee surgery, reduced patellar tendon length, thinner patellar components, reduced patella-patellar component composite thickness, and smaller femoral components. Preventative measures include choice of femoral components with a reduced intercondylar box ratio, use of thicker patellar components, avoidance of over-resection of the patella, and debridement of the fibrosynovial tissue at the time of knee arthroplasty. Most patients with crepitus are unaware of the problem or have minimal symptoms so that no treatment is required. If significant disability is incurred, symptoms can be eliminated in a high percentage of patients with arthroscopic debridement of the fibrosynovial hyperplasia.

**Keywords:** *Patellar crepitus, Total knee arthroplasty, Complication*

The excellent long term clinical results of total knee arthroplasty (TKA) utilizing a posterior stabilized (PS) prosthesis have been well documented.[1,2] However, despite these results, patellofemoral complications remain a common source of pain and dysfunction.[3,4] In 1982, Insall et al.[5] described a case of anterior knee pain associated with a peripatellar fibrous nodule that resolved following operative resection. In this report, two additional cases were proposed to have similar pathology. Conservative management was rendered. In 1989, *patellar clunk syndrome* was first described by Hozack et al.[6] in a cases series of patients treated with the Insall-Burstein I (IB-I) prosthesis (Zimmer, Warsaw, IN, USA), a first generation PS component. While cases of patellar clunk syndrome have been reported with the use of both cruciate retaining (CR) and patellofemoral replacement (PF) prostheses,[7,8] this complication has primarily occurred with use of PS TKAs with a reported incidence of 0%–18%.[3,5,9-22]

While patella clunk syndrome and patellofemoral crepitus are separate entities, they share a common pathophysiology with widely varying clinical presentations. The symptoms range from a painless subtle crepitation to a painful, palpable and audible clunk. These entities are the result of peripatellar fibrosynovial hyperplasia at the junction of the superior pole of the patella and the distal quadriceps tendon (Fig. 1). In the case of patellar clunk, a discrete fibrosynovial nodule becomes entrapped within the intercondylar box of the PS femoral component during knee flexion. Subsequently, when the knee is extended

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to within 30–45 degrees of full extension, the nodule dislodges resulting in an audible and often painful clunk (Fig. 2). In the case of patellofemoral crepitus, the fibrosynovial hyperplasia does not develop into a discrete nodule and no audible clunk is present. Conversely, these patients complain of anterior knee pain and a continued grinding sensation with loading of the knee in a 30–60 degree range. In either case, symptoms typically develop within 2 months to 2 years following TKA.[5,6,10-13,15,17-19,23] The natural history of patellofemoral crepitus and patellar clunk syndrome reveal that up to 50% of patients improve with conservative treatment and the majority are unaware the condition exists.[3,12,23] However, in both syndromes, the symptoms can be disabling warranting surgical intervention with either an arthroscopic or open debridement.[24-30] Nevertheless, prevention is preferred.

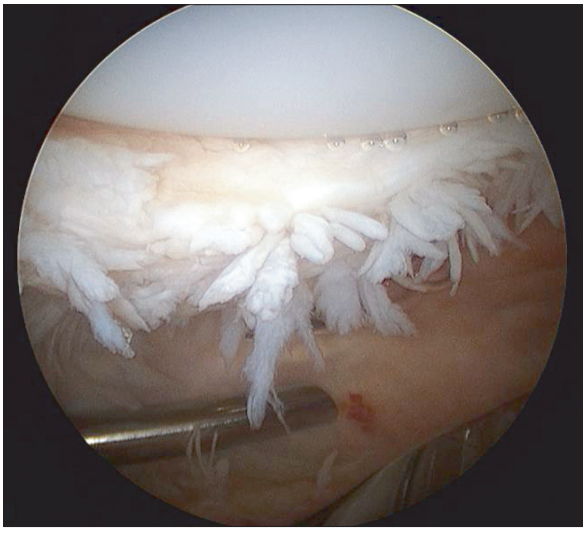

**Fig. 1.** Arthroscopic view of fibro-synovial hyperplasia at the junction of the superior pole of the patella and the distal quadriceps tendon in a patient with symptomatic patellar crepitus.

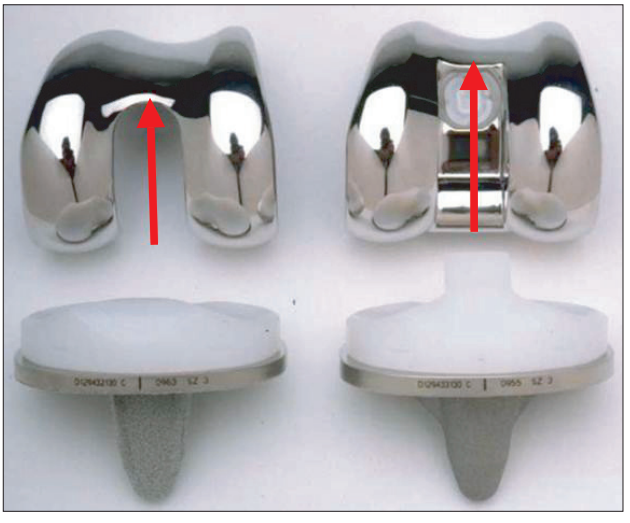

**Fig. 3.** Photograph of a cruciate retaining (left) and posterior stabilized (right) femoral components, demonstrating a higher transition height from the trochlear groove to the intercondylar box in the posterior stabilized femoral component.

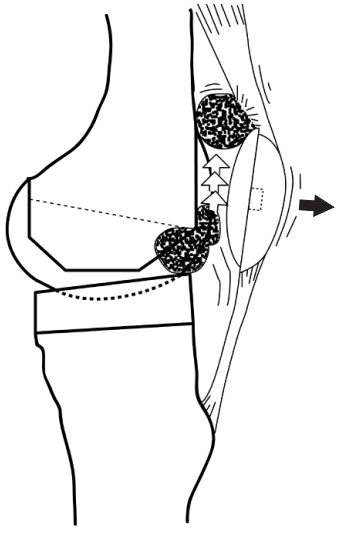

**Fig. 2.** Schematic drawing of the pathophysiology of patellar clunk syndrome. A discrete fibrosynovial nodule becomes entrapped within the intercondylar box of the posterior stabilized femoral component during flexion and is subsequently released when the knee is extended to within 30–45 degrees of full extension.

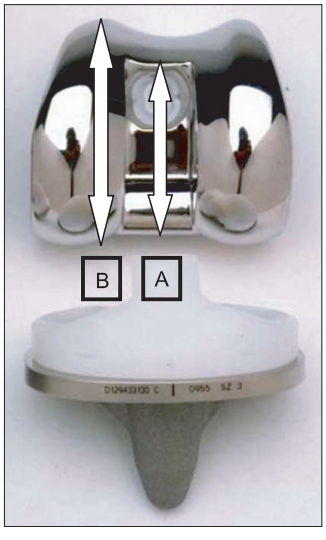


**Fig. 4.** Photograph of a posterior stabilized femoral component demonstrating the intercondylar box ratio, defined as the height of the intercondylar box divided by the anterior-posterior height of the femoral component.

In this chapter, we review the etiological factors of patellar crepitus and clunk syndrome and the preventative measures to avoid its development.

## HISTORICAL ETIOLOGIES

While the etiology of patellofemoral crepitus and clunk syndrome are multifactorial, the design and position of the femoral component are key factors in its development.[5,9-14,17-22] First generation PS designs included a high transition zone from the trochlear groove to the intercondylar box with an abrupt transition to the distal femoral articular geometry (Fig. 3).[10] It was theorized that this design feature led to the apposition of the distal quadriceps tendon to the anterior edge of the intercondylar box, resulting in soft tissue irritation, fibrosynovial hyperplasia and subsequent crepitus or clunk. Fukunaga et al.[11] further characterized this transition zone by defining the intercondylar box ratio, as the intercondylar box height versus the anterior-posterior height of the femoral component (Fig. 4). Second and third generation PS femoral components have typically been designed with a decreased intercondylar box ratio with a resultant decrease in the incidence of patellar clunk and crepitus (Table 1). Notably, those prostheses with an intercondylar box ratio of less than 0.7 were found to have no patellar clunk.[11] Femoral components with a high intercondylar box ratio allow contact of the distal quadriceps tendon earlier in flexion than components with a lower ratio. The authors concluded that this design feature may be responsible for the high incidence of patellofemoral crepitus and clunk with these prostheses.

The medial-lateral geometry of the intercondylar box has also been implicated as a causative factor. Pollock et al.[17] evaluated the incidence of soft tissue entrapment within the intercondylar box of three different PS TKA designs (Fig. 5). The incidence was 3.8% with the AMK

**Table 1.** Intercondylar Box Ratio and the Incidence of Patellar Clunk Syndrome with Various Femoral Component Designs

| Prosthesis | Intercondylar box ratios[6] | Incidence of patellar clunk (%) |
|---|---|---|
| Insall-Burstein II (Zimmer, Warsaw, IN, USA) | 0.71 to 0.72 | 7.5[16] |
| | | 10.2[13] |
| AMK PS (DePuy, Warsaw, IN, USA) | 0.84 to 0.85 | 3.3[8] |
| | | 18.3[13] |
| NexGen LPS (Zimmer, Warsaw, IN, USA) | 0.56 to 0.59 | 0[16] |
| | | 0[5] |
| Advance PS (Wright Medical, Arlington, TN, USA) | 0.62 | 0[17] |
| PFC Sigma PS (DePuy, Warsaw, IN, USA) | 0.85 to 0.87 | 13.3[6] |

PS: posterior stabilized.

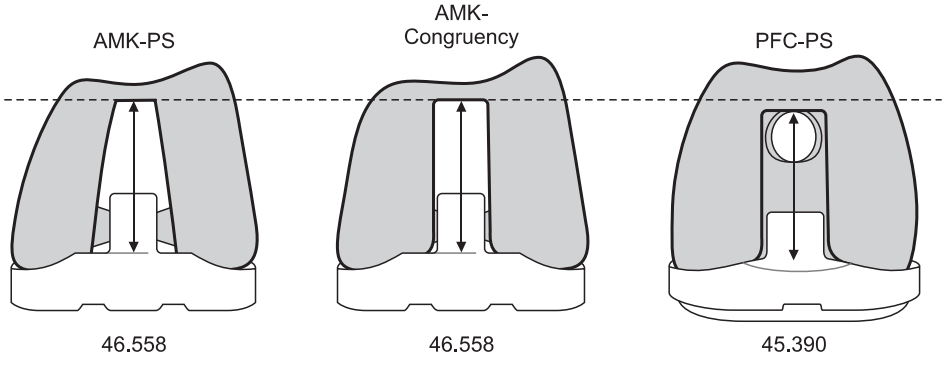


**Fig. 5.** Three posterior stabilized (PS) femoral components with different intercondylar box geometries. The design with the greatest intercondylar box height and narrowest intercondylar box width demonstrated the highest incidence of synovial entrapment (AMK Congruency; Depuy, Warsaw, IN, USA). Reprint from Pollock et al.[17] with permission from The Journal of Bone and Joint Surgery, Inc.

PS (DePuy, Warsaw, IN, USA), 13.5% for the AMK Congruency (DePuy), and 0% for the PFC Sigma PS (DePuy). The authors concluded that both the height and the width of the intercondylar box were design features leading to synovial entrapment.

Many other aspects of the femoral component design have been reported as etiologic factors for patellofemoral crepitus and clunk. Lonner et al.[13] compared the incidence of patellar clunk syndrome in patients who received two different generation PS TKA designs, the Insall-Burstein II (IB-II, Zimmer) and the NexGen LPS (NG-LPS, Zimmer). The incidence was 4% in the IB-II group and 0% in the NG-LPS group. The modifications made to the NG-LPS femoral component were a raised lateral flange, deepened trochlear groove, and a lengthened trochlear groove resulting in a more posteriorly positioned intercondylar box and a reduced intercondylar box ratio. The authors reported that these design features allow the patella to be engaged in the trochlear groove for a greater arc of motion, thus reducing the potential for frictional quadriceps tendon irritation and the subsequent development of patellar crepitus or clunk. In a similar study, Maloney et al.[19] found an increased incidence of patellar clunk in patients receiving the IB-II prosthesis (4%) compared to the Advance PS prosthesis (0%). The authors cited the posteriorly extended trochlear groove of the Advance PS as the differentiating design feature that led to the elimination of patellar clunk syndrome.

Aside from the femoral component design features, many authors suggest that patellofemoral crepitus and clunk occur are a result of intraoperative technical errors.[4,6,9,14,16,23] The incidence of patellofemoral complications in patients with PS TKAs has long been attributed to

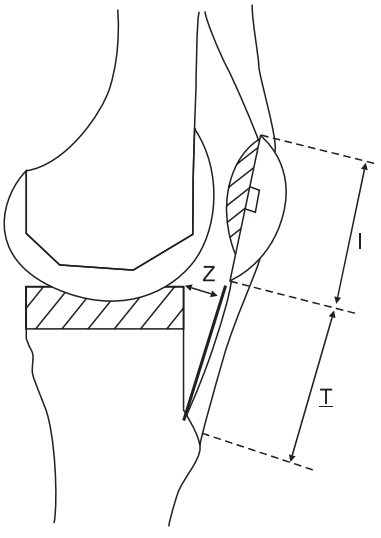


**Fig. 6.** Diagram demonstrating the Insall-Salvati ratio (T/I) and the perpendicular distance from the upper anterior corner of the tibial tray to the patellar tendon ([Z]: measuring the anterior posterior relationship of the tibial tray relative to the extensor mechanism). Reprint from Yau et al.[16] with permission from Elsevier.

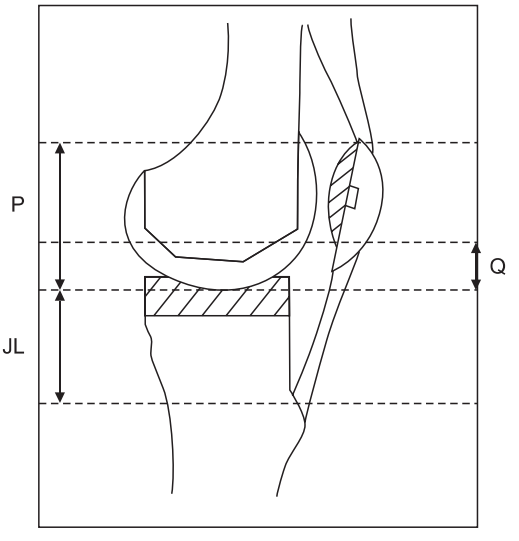


**Fig. 7.** Diagram demonstrating the joint line (JL), patellar button height (Q), and position of the proximal pole of the patella with reference to distal end of the femoral prosthesis (P). Reprint from Yau et al.[16] with permission from Elsevier.

**Table 2.** Radiographic Parameters of the Extensor Mechanism Affecting the Incidence of Patellar Clunk Syndrome

| Radiographic parameter | Patellar clunk syndrome group | Control group | *p*-value |
|---|---|---|---|
| Insall-Salvati ratio | 0.84 ± 0.12 | 0.97 ± 0.20 | < 0.001 |
| Relative position of tibial tray (mm) | 0.74 ± 2.33 | 9.23 ± 2.79 | 0.005 |
| Patellar component height (mm) | 12.09 ± 5.70 | 15.49 ± 6.59 | 0.008 |
| Position of the proximal pole of the patella (mm) | 47.04 ± 6.36 | 49.83 ± 7.12 | 0.045 |
| Level of joint line with reference to the tibial tuberosity (mm) | 24.84 ± 4.28 | 24.96 ± 4.12 | 0.904 |

patellar baja and anterior placement of the tibial tray.[4] Recently, Yau et al.[16] conducted a retrospective radiographic review of the extensor mechanism to identify possible causes of patellar clunk syndrome. In a consecutive series of 236 primary TKAs, 27 cases (11.4%) with patellar clunk syndrome were identified. The patients without patellar clunk served as a control group. Postoperative true lateral radiographs were utilized to evaluate the Insall-Salvati ratio, the relative anterior-posterior position of the tibial tray (Fig. 6), the height of the joint line, the patellar button height and the position of the proximal pole of the patella relative to the distal part of the femoral component (Fig. 7). A lower Insall-Salvati ratio, lower patellar component height, lower position of the proximal pole of the patella, and anteriorization of the tibial tray were all associated with a higher incidence of patellar clunk syndrome (Table 2). These data demonstrate that postoperative patella baja and anterior placement of the tibial tray is significantly related to the development of patellar clunk syndrome. In this study, there was no statistical difference in the elevation of the joint line between the two groups, which may suggest that the relative patellar baja is secondary to either inferior placement of the patellar component or an excessive distal femoral cut. In either case, the altered patellofemoral kinematics may be in part responsible for the development of patellar clunk syndrome.

Patellar crepitus and clunk syndrome have also been attributed to abnormal patellar tracking secondary to inadequate soft-tissue balance or tibial component malrotation.[4] In a second part of their study, Yau et al.[16] evaluated the congruency of the patellar component with the trochlear groove in patients who underwent bilateral TKAs but developed unilateral patellar clunk syndrome. They found an average patellar tilt of 9.59° ± 3.73° in patient with patellar clunk and (5.95° ± 3.04°) in patients without patellar clunk ($p$ = 0.019). Fukunaga et al.[11] similarly found an association between the incidence of patellar clunk and patellar mal-tracking. In their series, the mean patellar tilt was 6.00° ± 3.4° and 4.08° ± 2.9° in patients with and without patellar clunk respectively ($p$ = 0.020). The accompanying logistic regression analysis demonstrated that for every 1° increase in the postoperative patellar tilt, there is an increase the incidence of patellar clunk by 1.27.

Position of the patellar component has also been implicated as a cause of patellar clunk syndrome.[6] In their original case series, Hozack et al.[6] described one case where proximal overhang of the patellar button beyond the superior border of the patella led to a catching of the component on the anterior portion of the intercondylar box. Trimming of the overhanging portion of the button allowed complete resolution of symptoms. Subsequent studies have not supported this etiology. However, the pathophysiology may be applicable to occurrence of patellar clunk in TKA without patellar resurfacing. Shoji and Shimozaki[21] reported 11 cases of patellar clunk syndrome noted intraoperatively during PS TKA without patellar resurfacing. In this study, the increased medial to lateral width and intercondylar box ratio of the prosthesis (AGC; Biomet, Warsaw, IN, USA) allowed the superior pole of the patella to catch within the intercondylar box in a similar manner as the fibrosynovial nodule proposed by other authors. Shaving the superior pole of the patella eliminated the clunk in seven knees.

While many authors have identified component design and positioning as risk factors for the development of patellar crepitus, few have implicated the association of increased postoperative knee flexion.[15,18] Schroer et al.[15] retrospectively reviewed 747 consecutive TKAs in three contiguous cohorts for the occurrence of patellar clunk syndrome. In the first group, performed with the use of a standard median parapatellar arthrotomy and the Ascent PS (Biomet) femoral component, the incidence of patellar clunk was 0.5%. In the second group utilizing a mini-subvastus (MIS) approach, the same femoral component, and a resultant increase in the mean postoperative flexion, (113° vs. 124°) the incidence of patellar clunk syndrome was 6%. In the third group, they maintained the MIS surgical approach, transitioned to using a third generation femoral component (Vanguard PS, Biomet) with a resultant decrease in the incidence of patellar clunk to 0.4% despite the fact higher levels of flexion were obtained (125°). The preponderance of patellar clunk in the second group was attributed to the combination of increased mean postoperative flexion while using a second generation femoral component. These authors hypothesize that increased knee flexion positions the distal quadriceps tendon further distally and posteriorly on the femoral component, thus increasing the likelihood of soft tissue impingement in the intercondylar box. Third generation femoral components have a more posteriorly positioned intercondylar box and a more gradual transition to the distal femoral geometry, thus decreasing soft tissue impingement despite allowing higher levels of postoperative knee flexion. Similarly, patients with stiffness following TKA with a second generation femoral component rarely develop patellar crepitus since their flexion is not sufficient to allow the fibrosynovial tissue to become entrapped in the intercondylar space.[18]

**Table 3.** Patellar Crepitus Variables Analyzed

| Patient clinical variable* | Radiographic variable* | Surgical variable† |
|---|---|---|
| Gender | Preoperative alignment | Previous knee surgeries |
| Age | Postoperative alignment | Tibial component size |
| Height | Preoperative patellar tendon length | Femoral component size |
| Body weight | Postoperative patellar tendon length | Polyethylene thickness |
| Body mass index | Preoperative Insall-Salvati ratio | Patellar component size |
| Preoperative knee motion | Postoperative Insall-Salvati ratio | Patellar component shape |
| Postoperative knee motion | Femoral component flexion | Lateral release required |
| | Posterior femoral offset | Polyethylene bearing type |
| | Posterior tibial slope | |
| | Posterior tibial offset | |
| | Anterior tibial offset | |
| | Preoperative patellar thickness | |
| | Postoperative patellar thickness | |
| | Composite patellar thickness | |
| | Patellar tilt | |
| | Joint line | |

*Patient clinical and radiographic variables were treated as continuous variables. †Surgical variables were treated as categorical variables.

## CONTROL-MATCHED EVALUATION OF PAINFUL PATELLAR CREPITUS

Since its original description by Hozack et al.[6] in 1989, numerous studies have added to the long list of causes of patellar crepitus and clunk. However, this multifactorial nature makes it challenging for surgeons to identify with confidence areas of intervention to prevent its development as well as identify preoperatively patients who may be at high risk. Dennis et al.[23] conducted a large retrospective, multicenter case-control study to identify the onset of patellar crepitus, as well as clinical, radiographic, and surgical variables that increase the risk of patients developing painful, symptomatic patellar crepitus following PS TKA (Table 3).

The surgical databases of two institutions were searched to identify all patients who required surgical intervention for symptomatic patellar crepitus or clunk after PS TKA. Between 2002 and 2008 over 4,000 PS TKAs from the same implant system (PFC Sigma PS TKA [non-HP design]; Depuy) were identified, of which 60 met inclusion criteria. Sixteen (26.7%) had patellar clunk, whereas the remaining 44 (73.3%) developed patellar crepitus. Study patients were matched for age (± 3 years), gender, and body mass index (± 3 points) with a control patient group with well-functioning TKAs who did not experience patellar crepitus or clunk. A univariate analysis of the clinical, radiographic, and surgical parameters (Table 3) was completed to identify variables for a multivariate logistic regression model. Additionally, variables previously reported in the literature with clinical or surgical plausibility were also included in the regression model.

The average time of onset of the symptoms in the patellar crepitus group was 10.9 months (4 to 27 months). The univariate analysis identified 6 factors to statistically increase the risk of patellar clunk or crepitus: a reduced preoperative (54.54 mm vs. 57.90 mm; $p = 0.0089$) or postoperative (54.56 mm vs. 58.10 mm; $p = 0.0089$) patellar tendon length, a thinner postoperative composite patellar component thickness (22.33 mm vs. 23.24 mm; $p = 0.0219$), a reduced patellar component size ($p = 0.011$), an increased posterior femoral condylar offset (1.27 mm versus 0.17 mm; $p = 0.023$), and a history of pervious knee surgery ($p = 0.011$) (Table 4). The best fit multivariate model showed increased risk of developing patellar crepitus in patients with previous knee surgery ($p = 0.0009$),

**Table 4.** Factors Associated with an Increased Incidence of Patellar Crepitus (Univariate Analysis)

| Parameter | Patellar crepitus | Control | *p*-value |
|---|---|---|---|
| Preoperative patellar tendon length (mm) | 54.5 ± 6.6 | 58.0 ± 7.4 | 0.009 |
| Postoperative patellar tendon length (mm) | 54.6 ± 6.7 | 58.1 ± 7.8 | 0.009 |
| Posterior femoral offset (mm) | 1.3 ± 2.7 | 0.2 ± 2.3 | 0.023 |
| Composite patellar thickness (mm) | 22.3 ± 2.1 | 23.2 ± 2.4 | 0.022 |
| 32 mm patellar component size, no. (%) | 14 (23) | 4 (7) | 0.011 |
| Previous surgeries, no. (%) | 21 (35) | 13 (22) | 0.011 |

**Table 5.** Multivariate Logistic Regression Model for the Development of Patellar Crepitus

| Parameter | | Odds ratio | 95% confidence interval | *p*-value |
|---|---|---|---|---|
| Previous surgeries | 1 | 6.2 | 5.9–6.4 | 0.001 |
| | > 1 | 18.2 | 18.0–18.3 | < 0.001 |
| Femoral component (size < 3) | | 4.6 | 4.4–4.7 | 0.025 |
| Polyethylene bearing thickness (> 12.5 mm) | | 12.4 | 12.2–12.6 | 0.002 |
| Preoperative patellar tendon length | | 0.9 | 0.8–1.0 | 0.010 |
| Femoral component flexion | | 1.2 | 1.0–1.5 | 0.028 |
| Increase in posterior femoral condylar offset | | 1.5 | 1.2–1.9 | < 0.001 |

smaller femoral component size (size < 3; $p = 0.0253$), thicker polyethylene bearings (> 12.5 mm; $p = 0.0015$), reduced preoperative patellar tendon length ($p = 0.0098$), increased femoral component flexion ($p = 0.0282$), and an increase in the postoperative posterior femoral condylar offset ($p < 0.001$) (Table 5).

The results of this study further support many of the historically reported etiologies and risk factors for patellar crepitus and clunk. Numerous studies have implicated first and second generation femoral component designs in patellar crepitus,[5,9-22] especially those with an intercondylar box ratio of greater than 0.7,[11] like the PFC Sigma PS TKA (0.85–0.87) utilized in this study.

A decreased postoperative patellar tendon length and Insall-Salvati ratio have been cited by other authors as an etiologies for patellar crepitus.[4,16] The above data from the univariate analysis confirm that a decreased patellar height and shorter patellar tendon length both preoperatively and postoperatively are associated with the development of patellar crepitus. Moreover, the multivariate analysis demonstrated that a relative shortening of the patellar tendon via the use of thicker polyethylene inserts was significant risk factor. The authors suggest that these conditions increase the contact forces between the distal quadriceps tendon and the intercondylar box resulting in tendon irritation, development of fibrosynovial tissue proliferation, and eventual patellar crepitus or clunk. A reduced composite patella-patellar component thickness and use of a smaller (thinner) patellar component decrease the offset of the distal quadriceps tendon from the superior aspect of the intercondylar box, thereby increasing the risk of frictional irritation of the posterior aspect of the distal quadriceps tendon, fibrosynovial tissue development, and subsequent patellar crepitus.

Figgie et al.[4] found a correlation of anterior placement of the femoral component and the use of an undersized femoral component with patellofemoral complications following PS TKA. The above data do not support anterior placement of the femoral component as a risk factor. However, the multivariate analysis demonstrated a strong correlation between both the use of a small femoral component as well as a flexed posture of the femoral component with the development of patellar crepitus. They suggest the latter may irritate the quadriceps tendon as it

traverses the proximal aspect of the anterior flange of the femoral component.

No prior study has made the correlation of increased posterior femoral condylar offset and the development of patellar crepitus. The authors speculate that increasing the posterior condylar offset may result in a relative anterior shift of the intercondylar box, thus predisposing to quadriceps tendon irritation. The authors speculate the increased risk of patellar crepitus in subjects with prior knee surgery is secondary to development of increased intra-articular fibrosis from earlier knee operative procedures. This arthrofibrosis likely reduces soft tissue compliance, possibly reducing patellar height and increasing tension of quadriceps tendon as it traverses the intercondylar box of the femoral component.

The authors emphasize that this data cannot dispute other causes of postoperative patellar crepitus previously reported in the literature that were not found to be statistically significant given that the patient volume may not be adequately powered for each variable. Nevertheless, their data both supports many of the previous investigations as well as adding many new potential etiologies and risk factors for the development of patellar crepitus. In summary, the central pathophysiological hypothesis is that these factors in a multifactorial fashion result in increased contact forces and subsequent irritation of the distal quadriceps tendon as it traverses the superior aspect of the intercondylar box of a PS femoral component.

## CONTROL-MATCHED COMPUTATIONAL EVALUATION OF TENDO-FEMORAL CONTACT

To further evaluate the risk factors reported in the above discussed analysis, Hoops et al.[31] performed a control matched computational evaluation of tendo-femoral contact in patients implanted with a PS TKA. Their objective was to determine the influence of variations in patellar tendon length, flexion-extension alignment of the femoral component, position of the joint line, and patellar button size on the tendo-femoral contact and tendon-to-intercondylar notch spacing during a simulated deep knee bend activity.

The computational model was adapted from an *in vitro* experiment where a cadaveric knee was implanted with a PS TKA (PFC Sigma; DePuy) and subsequently mounted on the Kansas Knee Simulator (KKS). The KKS then facilitated a simulated deep knee bend activity. Magnetic resonance imaging of the implanted knee allowed for digitization of the implant surfaces and composite three-dimensional modeling. The clinical parameters were then varied by two standard deviations in both directions from the neutral implanted alignment and applied to the computational model. The potential for crepitation was then measured as a function of tendo-femoral contact during the simulated deep knee bend cycle to 120°. The cumulative tendo-femoral contact area was defined by any contact within 2 mm of the intercondylar notch. Subsequently this model was modified to match the parameters of 3 pairs of patients from the clinical case control study comparing patients with and without patellar crepitus.[23] The tendo-femoral contact areas, and the minimum tendon-to-notch distance at 120° of flexion was measured in these modeled patients.

Analysis of the model with variations in the clinical parameters demonstrated an increased cumulative contact area within 2 mm of the intercondylar notch with decreasing patellar tendon length, an extended femoral component posture, an elevated joint line, and use of a smaller patellar component (Fig. 8). Conversely, increasing the patellar tendon length, flexing the femoral component, lowering the joint line and using the largest possible patellar component decreased the cumulative tendo-femoral contact area. Of these parameters, variations in the patellar tendon length had the largest effect on the cumulative tendo-femoral contact area which is supported by the findings of previous clinical studies.[4,16,23]

Analysis of the model with patient matched parameters demonstrated significantly different contact areas. Crepitus patient models demonstrated increased tendo-femoral contact near the intercondylar notch, especially along its anterior border, which was not seen in the control

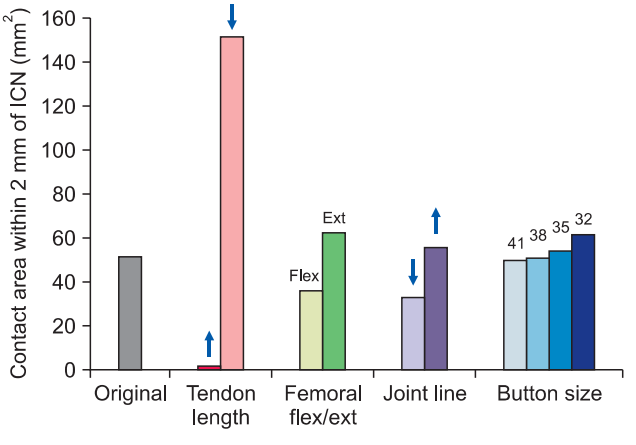


**Fig. 8.** Composite contract area within 2 mm of intercondylar notch (up to 120°) with original ligament and changes due to variation in patellar tendon length (alta and baja), flexion-extension alignment of the femoral component, join line and patellar button size. Reprint from Hoops et al.[31] with permission from John Wiley & Sons, Inc.

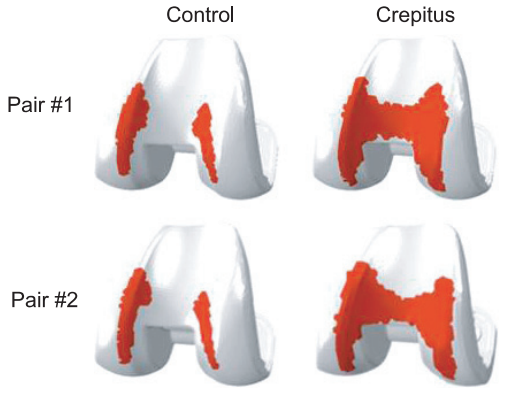


**Fig. 9.** Cumulative tendo-femoral contact patches in matched-control pairs of patients with and without patellofemoral crepitus.

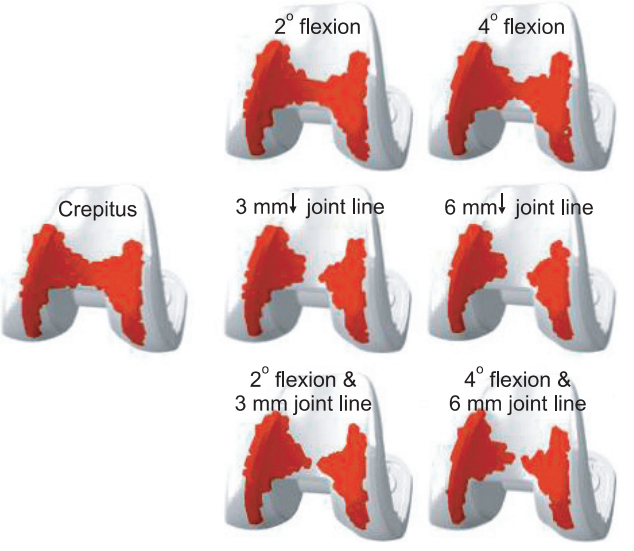


**Fig. 10.** Cumulative tendo-femoral contact patches in a patient with patellofemoral crepitus with potential changes in femoral flexion and joint line.

group (Fig. 9). Additionally, the tendon-to-notch distances were significantly less in the crepitus patient models in comparison to their matched control counterparts who had not developed patellar crepitus. Lastly, the alterations of the crepitus model patient parameters to test alternate surgical alignments demonstrated that flexing the femoral component and decreasing the joint line dramatically decreased the cumulative contact area near the intercondylar box (Fig. 10).

Notably, analysis of the control matched patient models demonstrated marked differences in contact areas and locations. Crepitus patient models showed increased contact of the distal quadriceps tendon on the anterior edge of the intercondylar box, while control patient models showed none. This gives supporting evidence to the central pathophysiological hypothesis that increased tendo-femoral contact is a contributing factor for the development of fibrosynovial tissue hypertrophy and subsequent patellar crepitus or clunk.

## PREVENTATIVE MEASURES

While many authors have demonstrated that painful patellar crepitus or clunk can effectively be treated with either an open or arthroscopic debridement,[24-30] prevention is preferred. The authors now counsel patients with previous knee surgery or with a decreased preoperative patellar tendon length that they are at increased risk for the development of patellar crepitus.

Selection of PS femoral components with a lower intercondylar box ratio (trochlear groove extended more posteriorly and distally) has been shown to lessen patellar crepitus and clunk.[11] It is wise to avoid use of an undersized femoral component positioned in a flexed posture.[4,23] Excessive femoral component flexion can be prevented by placing the entry site for intramedullary distal femoral cutting guide jig more anteriorly. Due to the anterior bow of the femur, a more posterior entry site may lead the surgeon to cut the distal femur in excessive flexion.

As previously stated, a preoperative diagnosis of patella baja may portend postoperative patella baja, in which case risk counseling is appropriate. Surgeons can avoid creating patella baja by avoiding excessive distal femoral resection. If an excessive amount of distal femur is removed, an increased tibial bearing thickness is required to adequately tension the extension gap, which raises the joint line and creates patella baja.

Yau et al.[16] demonstrated a higher chance of developing patellar crepitus with anterior placement of the tibial tray although this surgical variance is not supported by the authors' investigation.[23] Nevertheless, neutral or posterior placement of the tibial tray may lessen the development numerous patellar complications including crepitus and clunk.

Normal patellae contact the trochlear groove around 20° of flexion, and as knee flexion increases the contract stresses increase and shift superiorly. Therefore, the patellar component should be placed as superior as possible to avoid unresurfaced bone contact with the femoral component, but not extend superiorly beyond the superior border of the patella which can result in quadriceps tendon irritation and the subsequent development of patellar crepitus or clunk. Additionally, creation of a thinner

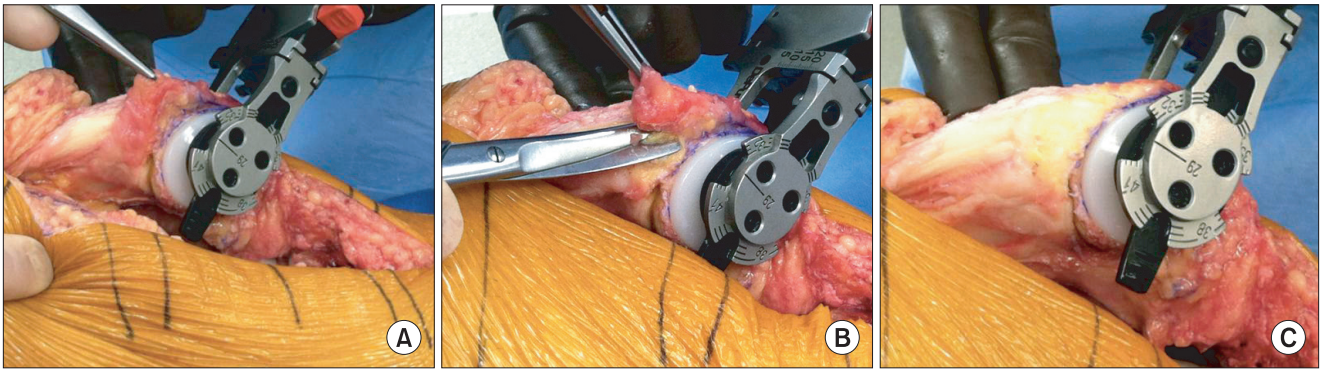


**Fig. 11.** Intraoperative photographs of the posterior aspect of the distal quadriceps tendon, demonstating synovial proliferation at the border of the superior pole of the patella and distal quadriceps tendon (A), and its removal (B, C).

composite thickness and use of a smaller (thinner) patellar component should be avoided which reduces the offset of the quadriceps tendon from the top of the trochlear groove.[23,31] Thus, surgeons should be careful to avoid overresecting the patella and should use the largest possible patellar component that fits the native bone stock. Meftah et al.,[3] further demonstrated a reduction in peripatellar crepitation from 40% to 21% by avoiding overstuffing the patellofemoral joint and resecting bone that is not covered by the patellar component. The senior author now routinely resects any uncovered lateral patellar facet to avoid postoperative anterior knee pain and patellar crepitus.

Lastly, the quadriceps tendon often has a proliferation of fibrosynovial tissue at the border of the superior pole of the patella and distal quadriceps tendon at the time of primary TKA. It is this tissue that has been implicated in the pathophysiology of patellar crepitus and clunk. The senior author excises this tissue routinely to avoid the development of patellofemoral crepitus and clunk (Fig. 11).

The excellent clinical results of patients with PS TKA are well documented. However, patellar crepitus and clunk syndrome remain a problem in a minority of patients. The cause of patellar crepitus is multifactorial with a myriad of proposed etiologies. Through acknowledgement of these etiological variables, use of modern femoral components with extended trochlear groove geometry, and avoidance of intraoperative errors, patellar crepitus and patellar clunk syndrome can be effectively minimized.

## CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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