Do Various Factors Affect the Frequency of Manipulation Under Anesthesia After Primary Total Knee Arthroplasty?

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Abstract

Background One of the most important goals of primary total knee arthroplasty (TKA) is to achieve a functional range of motion (ROM). However, up to 20% of patients fail to do so, which can impair activities of daily living.

Questions/purposes The purpose of this study was to evaluate the effect of various (1) demographic factors;

(2) comorbidities; and (3) knee-specific factors on the frequency of manipulation under anesthesia, which was used as an indicator of knee stiffness after a primary TKA. **Methods** We evaluated the registries of two high-volume centers and reviewed all 3182 TKAs that were performed between 2005 and 2011 to identify all patients who had undergone manipulation under anesthesia (MUA). A total of 156 knees in 133 patients underwent MUA after an index arthroplasty. These patients were compared in a one-to-four ratio with a group of patients with satisfactory ROM drawn from the same database who met prespecified criteria and who had not undergone MUA. Effects of various factors, including age, sex, body mass index, race, comorbidities, and the underlying cause of knee arthritis, were compared between these two cohorts using multivariable logistic regressions.

Results After controlling for various confounding, non-white race was associated with an increase (odds ratio [OR], 2.01; $p = 0.03$), and age ≥ 65 years (OR, 0.17; 95% confidence interval [CI], 0.04–0.74; $p = 0.0179$) was associated with a reduction in the incidence of MUA. In comorbidities, diabetes (OR, 1.72; 95% CI, 1.02–2.32; $p = 0.03$), high cholesterol levels (OR, 2.70; $p = 0.03$), and tobacco smoking (OR, 1.59; 95% CI, 1.03–2.47; $p = 0.03$) were associated with an increase in frequency of MUA. In knee-specific factors, preoperative knee ROM of less than 100° (OR, 0.80; $p < 0.0001$) and knee osteonecrosis ($p = 3.61$; 95% CI, 1.29–10.1; $p = 0.014$) were associated with increased frequency of MUA.

Conclusions We identified several demographic, medical, and knee-specific factors that were associated with poor

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postoperative ROM in our patients undergoing TKA. Patients who have multiple risk factors may benefit from preoperative counseling to set realistic ROM expectations. *Level of Evidence* Level III, prognostic study. See Guidelines for Authors for a complete description of levels of evidence.

Introduction

One of the most important goals of TKA is to achieve a functional ROM [4]. According to gait analyses and biomechanical studies, patients require at least 83° of flexion to ascend stairs, 90° to 100° to descend stairs, 93° to 105° to rise from a standard or short chair, and more than 115° to squat or kneel [3, 11, 13]. However, up to 20% of patients may develop knee stiffness and achieve knee flexion of less than 90° after their index arthroplasty [12, 17, 19, 28]. If left untreated, knee stiffness reduces the functional outcomes of patients including their daily living activities and satisfaction [25, 26].

There is no consensus on what factors might be associated with stiffness after TKA. Decreased preoperative ROM, younger age, genetic predisposition, diabetes mellitus, socioeconomic status, previous knee surgery, malpositioning of the prosthesis, inadequate resection, lack of patient compliance with rehabilitation as well as complex regional pain syndrome have all been proposed to influence this condition [1, 2, 5–7, 11, 13–16, 18–23, 29]. However, not all of these factors have been identified by all studies, and many of the reports on the topic have small sample sizes and have failed to account for various confounding variables using multivariable regression analyses. We therefore sought to more extensively evaluate the effect of various demographic factors, patient characteristics, and comorbidities in our practice on the incidence of manipulation under anesthesia (MUA), which was used as an indicator of knee stiffness after primary TKA. Specifically, we compared (1) demographic factors such as age, sex (men-to-women ratio), body mass index, and race; (2) comorbidities such as diabetes, cardiovascular disease, hypertension, hypercholesterolemia, multiple joint disorders, and tobacco smoking; and (3) knee-specific factors such as the underlying cause of knee arthritis (osteoarthritis or osteoarthritis), pre-TKA ROM, and the type of implanted prosthesis (cruciate-retaining versus posterior stabilizer [PS]) between patients who did and did not develop knee stiffness.

Materials and Methods

We reviewed our database of all patients who had undergone a primary TKA at two high-volume institutions by four surgeons between January 2005 and December 2011 to determine the number of patients who had received a MUA, which was used as an indicator of knee stiffness.

During the period in question, all patients who underwent an MUA had completed a course of rehabilitation before being offered MUA; patients who were not gaining or were losing ROM were offered MUA. In our institutions, all surgeons generally had a low threshold for performing early MUAs. All patients with < 110° ROM at 6 weeks post-TKA (range, 3–48 weeks) with no recent gains after physical therapy were offered MUA. Patients with certain religious, cultural, or personal reasons requiring higher ROM such as kneeling specifically requested higher flexion ROMs. Absolute contraindications for manipulation included ongoing infection, component malalignment, elevated joint line, or not being able to tolerate anesthesia. Lack of extension was not used as an indicator for MUA; however, all patients with greater than 10° of flexion contracture were excluded from this study. In 36 patients who met absolute or relative indications for MUA, the procedure was delayed 3 to 30 weeks because they initially declined their treating surgeon's recommendation to undergo the procedure. Also, 14 patients had continued to refuse the procedure until final followup. These patients were not included in the study or the comparison cohort in this report.

Of the total of 3182 TKAs, 156 knees in 133 patients who had undergone MUA were identified. We had records on 3146 of the 3182 at 3 months, which was the point in time by which 77% (121 of 156) of the MUAs had been performed. These patients were compared in a one-to-four ratio with group of patients with satisfactory ROM drawn from the same database who met prespecified criteria and who had not undergone MUA. In the comparison cohort, all patients had minimum knee flexion ROM of 115° at minimum 2-year followup; no patient had developed knee stiffness or had requested or was offered MUA. All available medical records, including admission history and physical examinations, preoperative studies, postoperative and discharge reports as well as office notes, were reviewed. Appropriate institutional review board approval for this study was obtained from each institution.

A standard medial parapatellar approach was performed for all TKAs at both institutions. All patients received cemented TKA prosthetic components (the majority had received either Triathlon® prostheses [n = 2464], Stryker Orthopedics, Mahwah, NJ, USA; or Scorpio® prostheses [n = 358], Stryker Orthopedics) prepared with the use of standard universal cutting blocks. Overall, 72% of the

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implants were cruciate-retaining (CR) and 26% were PS. During rehabilitation after the index arthroplasty, all patients were encouraged to fully weightbear in the immediate postoperative period. All patients underwent routine postoperative rehabilitation protocols, including 4 weeks of quadriceps muscle strengthening, gait, and ROM exercises.

All data were recorded using an Excel spreadsheet (Microsoft Corporation, Redmond, WA, USA). Statistical data analysis was performed in SAS Version 9.2 (SAS Institute Inc, Cary, NC, USA) using Fisher's exact test, paired two-tailed Student's t-test, and logistic regression models to compare differences in various demographics, patient characteristics, and comorbidities between patients who received MUA to patients who did not. A *p* value of < 0.05 was used as a threshold for significance.

Results

The only demographic findings we found that were associated with MUA were nonwhite race, which was associated with more MUAs, and age ≥ 65 years, which was associated with fewer MUAs (Table 1). After controlling for confounding variables, nonwhite race was associated with twofold higher odds ratio (95% confidence interval [CI], 1.06–3.84; *p* = 0.03), and age ≥ 65 years was associated with approximately sixfold lower odds ratio (95% CI, 0.04–0.74; *p* = 0.0179) of undergoing MUA. However, there was no increase in the frequency of MUA in women compared with men (odds ratio [OR], 1.04; 95% CI, 0.49–2.21; *p* = 0.91) and body mass index (BMI) of < 25 kg/m² compared with all other BMI subcohorts (*p* > 0.54).

The only comorbidities that were identified to be associated with higher frequency of MUA were diabetes, high cholesterol levels, and tobacco smoking. After controlling for confounding variables, diabetes was associated with 1.7 OR (95% CI, 1–2.3; *p* = 0.03), high cholesterol levels with 2.7 OR (95% CI, 1.10–6.61; *p* = 0.03), and tobacco smoking with 1.6 OR (95% CI, 1.03–2.47; *p* = 0.03) of higher frequency of MUA. However, the presence of any of the following comorbidities was not associated with a higher frequency of MUA: hypertension (OR, 1.16; 95% CI, 0.51–2.65; *p* = 0.72), cardiovascular disorders (OR, 2.18; 95% CI, 0.80–5.96; *p* = 0.12), gastrointestinal disorders (OR, 0.69; 95% CI, 0.29–1.65; *p* = 0.40), and hypothyroidism (OR, 1.65; 95% CI, 0.85–2.83; *p* = 0.071).

The only knee-specific factors that were associated with higher frequency of MUA were lower preoperative knee ROM and an underlying diagnosis of knee osteonecrosis. After controlling for confounding variables, preoperative knee ROM of greater than 100° was associated with

Table 1. Logistic regression analysis of various factors contributing to knee stiffness after TKA

Metrics	OR* (95% CI)	<i>p</i> value
Pre-TKA ROM		
ROM ≤ 100	Referent	0.0001
ROM > 100	0.80 (0.77–0.84)	
Age (years)		
< 45	Referent	
45–54	0.87 (0.21–3.54)	0.8435
55–64	0.33 (0.08–1.33)	0.1191
65–74	0.17 (0.04–0.74)	0.0179
≥ 75	0.06 (0.01–0.37)	0.0022
Sex		
Male	Referent	
Female	1.04 (0.49–2.21)	0.9142
Race		
White	Referent	
Nonwhite	2.01 (1.06–3.84)	0.0336
Body mass index (kg/m ²)		
< 25	Referent	
25–29	0.75 (0.25–2.25)	0.6073
30–34	1.13 (0.40–3.24)	0.8157
35–39	0.71 (0.24–2.12)	0.5401
≥ 40	0.71 (0.21–2.41)	0.5778
Prosthesis		
Cruciate-retaining	Referent	
Posterior-stabilized	0.89 (0.39–1.35)	0.4119
Diabetes	1.72 (1.02–2.31)	0.0311
Hypertension	1.16 (0.51–2.65)	0.7273
Cardiac disease	2.18 (0.80–5.96)	0.1293
Smoker	1.59 (1.03–2.47)	0.0350
Gastrointestinal disease	0.69 (0.29–1.65)	0.4035
Hypothyroidism	1.65 (0.95–2.83)	0.0715
High cholesterol	2.70 (1.10–6.61)	0.0298
Knee osteonecrosis	3.61 (1.29–10.13)	0.0144

* Models adjusted for preoperative ROM, age, sex, race, body mass index, prosthesis, diabetes, hypertension, cardiac disease, smoking, alcohol, gastrointestinal disease, high cholesterol, and hypothyroidism; OR = odds ratio; CI = confidence interval.

0.8-fold (95% CI, 0.77–0.84; *p* < 0.0001) lower OR and underlying diagnosis of knee osteonecrosis was associated with 3.6-fold (95% CI, 1.29–10.1; *p* = 0.014) higher OR of undergoing MUA. There was no difference between PS compared with CR prostheses (OR, 0.89; 95% CI, 0.24–1.35; *p* = 0.41).

Discussion

Knee stiffness after primary TKA is a debilitating condition for patients and, if left untreated, can negatively affect

patient satisfaction and functional outcomes including activities of daily living [1, 27]. This is because many daily activities, including descending stairs, rising from a chair, or tying shoelaces, routinely require more than 90° of knee flexion [13, 24]. Previous studies on this topic generally had small sample sizes and failed to account for various confounding variables using multivariable analyses. Thus, we attempted to evaluate the association of various demographic factors, patient comorbidities, and knee-specific factors on the frequency of MUA after TKA, which was used as a surrogate for knee stiffness after primary TKA. This study examined the contribution of various potential preoperative, intraoperative, and postoperative risk factors in an attempt to identify important sources of knee stiffness in our practice. After controlling for potential confounding variables, we found, that nonwhite race, age older than 65 years, diabetes, high cholesterol, tobacco smoking, knee osteonecrosis, and decreased pre-TKA ROM were associated with an increased frequency of undergoing MUA.

There were several limitations of this study. This was not a prospective study that could have potentially reduced sampling biases; however, we had attempted to reduce this by choosing the comparison group of patients from the same database, during the same time period, and same surgeons who had met prespecified criteria. MUA was used as an indicator to identify patients who had developed stiffness after TKA; thus, an unknown number of patients may have had poor motion but elected not to proceed with further surgery. Nonetheless, we generally adhered to the indications defined in this study, and we believe it was a generally good surrogate for knee stiffness during the period of study. Indications to undergo knee MUA may have been different for this study compared with other institutions. Evaluation of preoperative narcotic medication use was not performed in this study; however, we have previously reported on the effect of narcotic use in TKA. Intra- and postoperative contributing risk factors such as overstuffing of the patellofemoral joint, mechanical knee alignment, joint line elevation, patellar complications, or poor patient compliance with physical therapy were not evaluated in this study. Also, preoperative functional outcomes were not measured between the two cohorts; however, our previous reports have evaluated these metrics.

Previous studies have also attempted to evaluate the effects of various patient demographics on the frequency of MUAs. Springer et al. [26] evaluated patients undergoing 1557 TKA, of whom 521 required MUA resulting from knee stiffness. In their bivariate analyses, they reported that there were no differences with respect to sex ($p = 0.16$), BMI ($p = 0.58$), or femoral component design ($p = 0.52$) between patients who had received a MUA compared with those who had not. However, the MUA cohort was younger (60 versus 65 years; $p < 0.0001$), had a higher proportion of

nonwhite patients (13% versus 6%; $p < 0.0001$), had more patients with private insurance (63% versus 43.5%), and had fewer with Medicare coverage (29.6% versus 52%). In their multivariable regression model, nonwhite race and patients < 45 years had twofold higher odds of undergoing manipulation compared with whites and patients > 75 years. These outcomes are comparable with our findings that younger age and nonwhite race were associated with higher OR of MUAs. Although we do not know the exact genetic, biological variation, and biomechanics of stiffness between different age groups or among various races, higher demand for work and/or physical activity in younger patients and a lower threshold for it may partly explain the higher frequency of MUA in younger patients.

Concerning comorbidities, Robertson et al. [22] compared the outcomes of 367 TKAs in patients who had Type 2 diabetes mellitus with a matched nondiabetic control group. They reported that at 1-year ($p < 0.001$) and 5-year ($p < 0.001$) followup, patients who had diabetes had lower maximal flexion and total ROM compared with the nondiabetic control group. Their results were consistent with the findings from our study in that diabetics had an increased risk of undergoing MUA compared with nondiabetics. Our study also found more knee stiffness in patients who had a diagnosis of osteonecrosis compared with those who had osteoarthritis. In addition, the prevalence of hypothyroidism in our MUA cohort was noteworthy. To our knowledge, no previous study has identified these comorbidities as potential contributing factors to knee stiffness. Although we do not know the exact mechanism leading to stiffness with these diseases, hypothyroid disorders may cause musculoskeletal pain [8] and indirectly contribute to lower ROM in some patients.

Regarding knee-specific factors, lower preoperative knee ROM has been reported to be a contributing factor to postoperative knee flexion according to many previous studies, which is in agreement with our study [9, 10, 16, 19, 21]. In our study, a lower preoperative knee ROM was associated with a higher odds ratio of MUA (and indirectly stiffness). However, a recent report found that preoperative ROM was not a predictor of final ROM [23]. Russell et al. [23] evaluated 153 TKAs performed using a rotating-platform posterior cruciate-substituting design. Patients were divided into two groups (Group 1 $< 95^\circ$, Group 2 $> 95^\circ$) based on their preoperative ROM. At 12 months followup, they reported no differences in flexion ROM (mean 120° versus 123°) or Knee Society scores between the two groups. Potential reasons for these differences compared with our study may partly be the result of baseline variations in patient demographics, types of prostheses, rehabilitation protocols, surgeons' threshold to perform MUAs, and preoperative narcotic use differences between the two studies.

In summary, knee stiffness after TKA has a multifactorial etiology with several risk factors, including demographic variables, specific comorbidities, and certain diagnoses. Important findings included a higher frequency of MUAs with nonwhite race and a lower frequency of MUA with older age. In comorbidities, diabetes, high cholesterol levels, and tobacco smoking were associated with an increase in the frequency of MUA. In knee-specific factors, preoperative knee ROM of less than 100° and knee osteonecrosis were associated with increased frequency of MUA. We found no evidence that retention or sacrifice of the posterior cruciate ligament played a significant role and no differences between men and women after controlling for potential baseline confounding variables. Patients may benefit from preoperative counseling when they have multiple risk factors to set realistic ROM expectations. More prospective studies are necessary to better evaluate if specific patient demographics or comorbidities may affect the frequency and clinical outcomes of MUA.

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References

- Boldt JG, Munzinger UK, Zanetti M, Hodler J. Arthrofibrosis associated with total knee arthroplasty: gray-scale and power Doppler sonographic findings. *AJR Am J Roentgenol*. 2004;182:337–340.
- Boldt JG, Stiehl JB, Hodler J, Zanetti M, Munzinger U. Femoral component rotation and arthrofibrosis following mobile-bearing total knee arthroplasty. *Int Orthop*. 2006;30:420–425.
- Bong MR, Di Cesare PE. Stiffness after total knee arthroplasty. *J Am Acad Orthop Surg*. 2004;12:164–171.
- Carr AJ, Robertsson O, Graves S, Price AJ, Arden NK, Judge A, Beard DJ. Knee replacement. *Lancet*. 2012;379:1331–1340.
- Desai AS, Karmegam A, Dramis A, Board TN, Raut V. Manipulation for stiffness following total knee arthroplasty: when and how often to do it? *Eur J Orthop Surg Traumatol*. 2013 Dec 11 [Epub ahead of print].
- Freeman TA, Parvizi J, Della Valle CJ, Steinbeck MJ. Reactive oxygen and nitrogen species induce protein and DNA modifications driving arthrofibrosis following total knee arthroplasty. *Fibrogenesis Tissue Repair*. 2009;2:5.
- Freeman TA, Parvizi J, Della Valle CJ, Steinbeck MJ. Mast cells and hypoxia drive tissue metaplasia and heterotopic ossification in idiopathic arthrofibrosis after total knee arthroplasty. *Fibrogenesis Tissue Repair*. 2010;3:17.
- Gillan MM, Scofield RH, Harley JB. Hashimoto's thyroiditis presenting as bilateral knee arthropathy. *J Okla State Med Assoc*. 2002;95:323–325.
- Harvey IA, Barry K, Kirby SP, Johnson R, Elloy MA. Factors affecting the range of movement of total knee arthroplasty. *J Bone Joint Surg Br*. 1993;75:950–955.
- Issa K, Kapadia BH, Kester M, Khanuja HS, Delanois RE, Mont MA. Clinical, Objective, and functional outcomes of manipulation under anesthesia to treat knee stiffness following total knee arthroplasty. *J Arthroplasty*. 2014;29:548–552.
- Kettelkamp DB, Leaverton PE, Misol S. Gait characteristics of the rheumatoid knee. *Arch Surg*. 1972;104:30–34.
- Kim J, Nelson CL, Lotke PA. Stiffness after total knee arthroplasty. Prevalence of the complication and outcomes of revision. *J Bone Joint Surg Am*. 2004;86:1479–1484.
- Laubenthal KN, Smidt GL, Kettelkamp DB. A quantitative analysis of knee motion during activities of daily living. *Phys Ther*. 1972;52:34–43.
- Nelson CL, Kim J, Lotke PA. Stiffness after total knee arthroplasty. *J Bone Joint Surg Am*. 2005;87(Suppl 1):264–270.
- Pankaj A, Malhotra R, Bhan S. Femoral component malrotation and arthrofibrosis after total knee arthroplasty: cause and effect relationship? *Int Orthop*. 2007;31:423; author reply 425.
- Parsley BS, Engh GA, Dwyer KA. Preoperative flexion. Does it influence postoperative flexion after posterior-cruciate-retaining total knee arthroplasty? *Clin Orthop Relat Res*. 1992;275:204–210.
- Parvizi J, Nunley RM, Berend KR, Lombardi AV Jr, Ruh EL, Clohisy JC, Hamilton WG, Della Valle CJ, Barrack RL. High level of residual symptoms in young patients after total knee arthroplasty. *Clin Orthop Relat Res*. 2014;472:133–137.
- Parvizi J, Tarity TD, Steinbeck MJ, Politi RG, Joshi A, Purtill JJ, Sharkey PF. Management of stiffness following total knee arthroplasty. *J Bone Joint Surg Am*. 2006;88(Suppl 4):175–181.
- Pivec R, Issa K, Kester M, Harwin SF, Mont MA. Long-term outcomes of MUA for stiffness in primary TKA. *J Knee Surg*. 2013;26:405–410.
- Ries MD, Badalamente M. Arthrofibrosis after total knee arthroplasty. *Clin Orthop Relat Res*. 2000;380:177–183.
- Ritter MA, Stringer EA. Predictive range of motion after total knee replacement. *Clin Orthop Relat Res*. 1979;143:115–119.
- Robertson F, Geddes J, Ridley D, McLeod G, Cheng K. Patients with Type 2 diabetes mellitus have a worse functional outcome post knee arthroplasty: a matched cohort study. *Knee*. 2012;19:286–289.
- Russell RD, Huo MH, de Jong L, Jones RE. Preoperative flexion does not influence postoperative flexion after rotating-platform total knee arthroplasty. *Knee Surg Sports Traumatol Arthrosc*. 2013 Jan 12 [Epub ahead of print].
- Scuderi GR. The stiff total knee arthroplasty: causality and solution. *J Arthroplasty*. 2005;20:23–26.
- Seyler TM, Marker DR, Bhave A, Plate JF, Marulanda GA, Bonutti PM, Delanois RE, Mont MA. Functional problems and arthrofibrosis following total knee arthroplasty. *J Bone Joint Surg Am*. 2007;89(Suppl 3):59–69.
- Springer BD, Odum SM, Nagpal VS, Lombardi AV Jr, Berend KR, Kim RH, Dennis DA. Is socioeconomic status a risk factor for stiffness after total knee arthroplasty? A multicenter case-control study. *Orthop Clin North Am*. 2012;43:e1–7.
- Vince KG. The stiff total knee arthroplasty: causes and cures. *J Bone Joint Surg Br*. 2012;94:103–111.
- Zmistowski B, Restrepo C, Kahl LK, Parvizi J, Sharkey PF. Incidence and reasons for nonrevision reoperation after total knee arthroplasty. *Clin Orthop Relat Res*. 2011;469:138–145.
- Zywiell MG, Stroh DA, Lee SY, Bonutti PM, Mont MA. Chronic opioid use prior to total knee arthroplasty. *J Bone Joint Surg Am*. 2011;93:1988–1993.