



Contents lists available at ScienceDirect

The Journal of Arthroplasty

journal homepage: www.arthroplastyjournal.org

Why Are Total Knees Failing Today? Etiology of Total Knee Revision in 2010 and 2011

William C. Schroer, MD^a, Keith R. Berend, MD^b, Adolph V. Lombardi, MD^b, C. Lowry Barnes, MD^c, Michael P. Bolognesi, MD^d, Michael E. Berend, MD^e, Merrill A. Ritter, MD^e, Ryan M. Nunley, MD^f

^a St. Louis Joint Replacement Institute, SSM DePaul Health Center, St. Louis, Missouri^b Joint Implant Surgeons, Inc., New Albany, Ohio^c Arkansas Specialty Orthopaedics, Little Rock, Arkansas^d Center for Hip and Knee Surgery, Mooresville, Indiana^e Duke University Medical Center, Department of Orthopaedic Surgery, Hospital South, Durham, North Carolina^f Washington University Medical Center, Barnes Jewish Hospital, St. Louis, Missouri

ARTICLE INFO

Article history:

Received 18 August 2012

Accepted 28 April 2013

Keywords:

total knee revision

failure

aseptic loosening

instability

ABSTRACT

Revision knee data from six joint arthroplasty centers were compiled for 2010 and 2011 to determine mechanism of failure and time to failure. Aseptic loosening was the predominant mechanism of failure (31.2%), followed by instability (18.7%), infection (16.2%), polyethylene wear (10.0%), arthrofibrosis (6.9%), and malalignment (6.6%). Mean time to failure was 5.9 years (range 10 days to 31 years). 35.3% of all revisions occurred less than 2 years after the index arthroplasty, 60.2% in the first 5 years. In contrast to previous reports, polyethylene wear is not a leading failure mechanism and rarely presents before 15 years. Implant performance is not a predominant factor of knee failure. Early failure mechanisms are primarily surgeon-dependent.

© 2013 Elsevier Inc. All rights reserved.

Understanding why total knee arthroplasties fail today is important for improving outcomes and directing efforts to minimize the high medical and socioeconomic costs associated with knee arthroplasty failure. The number of primary total knee arthroplasty (TKA) performed in the United States has increased dramatically over the past 10 years to more than 615,000 in 2008 [1]. Projections estimate that demand will continue to grow to more than 3 million annually by 2030. [2] The volume of revision TKA increased to more than 75,000 during the same time period and has been projected to further increase by 600% by 2030 [2]. Whereas it is expected that increased primary knee volume would increase revision volume, the cause and rate of failure cannot be ignored.

Outcome studies can initiate surgeon and hospital efforts to improve surgical technique and clinical care guidelines, and can motivate industry to improve instrumentation, implant design, or materials. However, most studies on TKA failure describe single-surgeon, single-implant, and/or single-institution results that report a small percentage of failed knee arthroplasties [3–5]. Yet, population studies continue to demonstrate a much higher failure rate [6,7].

The current study attempts to report why TKA is failing today, similar to previous studies by Fehring et al and Sharkey et al who

looked at failure mechanisms for revision TKA performed between 1986 and 2000 [8,9]. These studies demonstrated that a majority of failures occurred in the first few years, with a disproportionate amount for infection and implant-associated failure mechanisms. Since these studies were published, efforts have been made to improve implant performance and instruct surgeons towards best practice total knee techniques. Unlike previous studies, this paper compiles results from a multi-center evaluation of revision TKA cases during 2010 and 2011. The purpose of this study is to report a detailed analysis of the failure mechanism and the time to failure to determine whether the failure mechanism of primary TKA has changed over the past 10–15 years.

Patients and Methods

A retrospective review of failed primary total knee arthroplasties that presented for revision surgery at six different orthopedic institutions (two major university academic centers, two non-university academic centers, and two community total joint centers) between January 1, 2010 and December 31, 2011 yielded 844 knees. Demographic data included patient gender, age, weight, height, and BMI at the time of revision. The primary mechanism of failure was determined by the operating surgeon with information collected during preoperative consultation, radiographic evaluation, intraoperative analysis, and laboratory results if necessary (Table). All institutions used a standardized spreadsheet to record data and categorize mechanism of failure. Only primary revision surgery was tabulated. Isolated polyethylene exchange for possible infection was not considered a revision

Approval from the Institutional Review Board at each institution was obtained by the authors to conduct this study.

The Conflict of Interest statement associated with this article can be found at <http://dx.doi.org/10.1016/j.arth.2013.04.056>.

Reprint requests: William C. Schroer, MD, St. Louis Joint Replacement Institute, SSM DePaul Health Center, 12266 DePaul Drive, Suite 220, St. Louis, MO 63044.

surgery to avoid counting a single septic failure multiple times. Time to failure was determined from patient health history forms, and classified into four time intervals (Table).

Results

Mean age was 65.0 years and BMI was 33.8 for patients undergoing revision. Men comprised 37% (313) of the knee failures and women 63% (531). Mean time to revision was 5.9 years (range 10 days to 31 years). More than one-third (35.3%) of knee revisions occurred in the first two years, 24.9% from 2 to 5 years, 29.5% from 5 to 15 years, and 10.3% after 15 years (Table).

Aseptic loosening was the predominant mechanism of failure, followed by instability, infection, polyethylene wear, arthrofibrosis, and malalignment (Table). These six mechanisms of failure represented 89.7% of all failures.

Discussion

Understanding TKA failure mechanisms is crucial for reducing the revision rate and its associated costs in this country. Initial reports of TKA outcomes were single-implant/single-surgeon studies that generally described the excellent results of high-volume, expert surgeons [3–5]. However, this type of study cannot compare outcomes of different implant designs, is less likely to be reported when results are negative [10], and demonstrates that not everyone is an expert high-volume surgeon. Whereas single-surgeon reports generally document low failure rates, overall TKA revision rates are much higher across the US [7]. National registries, in which all primary and revision cases are recorded, best determine TKA failure rates and can compare a large number of different implants, but they are poor at differentiating failure mechanism. Recently implemented ICD-9CM diagnosis and procedure codes have allowed analysis of 60,000 TKA revisions using administrative claims data from the Nationwide Inpatient Sample (NIS) database [11]. But while this study reported failure mechanisms, uncertainties exist regarding both compliance and accuracy of diagnosis and procedural coding. Additionally, administrative claims data analysis cannot determine failure rate or etiology of failure over time. The current multi-center study offers a more accurate evaluation of failure mechanism and its relation to time. A limitation of this study, in which a significant number of the index procedures were referrals, is the absence of a known baseline population from which all the knee failures derive. This prevents a failure rate calculation. In addition, while this multi-center study analyzes more cases than previous studies, the number of failures of any specific implant type was not large enough to determine specific implant’s contribution towards knee failure. Finally, surgeons were asked to determine the primary failure mechanism on each of their own revisions. Different surgeons may have used different determinations of the primary failure mechanisms for a similar case.

A common conclusion to all reports on primary total knee arthroplasty failure is that a unique and significant subset of revisions occurs early. Two studies presented in 2001 described knee failure in the 1990s. In a series of 440 knees reported by Fehring et al and revised over a 14-year period, 63% failed less than 5 years after the index arthroplasty [8]. Sharkey et al reported that 55.6% of 212 knees revised over a 3-year period failed less than two years after primary surgery, with mean time to failure 3.7 years (range 8 days to 28 years) [9]. In the current study, 35.3% of all revisions were performed less than two years from the index knee arthroplasty, 60% in the first five years. The mean time to failure was 5.9 years (range 10 days to 31 years). Of note, while the range of time to failure is similar to previous studies, the mean is 60% longer than reported a decade earlier by Sharkey et al [9].

The most common failure mechanisms in this study were aseptic loosening, instability, infection, polyethylene wear, arthrofibrosis, and malalignment, which combined represent 90% of the primary knee failures. However, failure mechanisms vary over time and this study’s ability to differentiate revisions across time allows analysis of each failure mechanism. Instability (25.2%) and infection (22.8%) were the most common failure mechanisms in the group revised under two years, but were rare after 15 years (Table). Arthrofibrosis similarly represented 10% of all revisions less than five years, but was uncommon after five years. Conversely, polyethylene wear represented less than 1% of revisions under five years, but was the leading failure mechanism after 15 years (48.3%). Aseptic loosening was the only failure mechanism that was consistent across time, representing more than 19% of failures in each time interval.

In contrast to the Sharkey et al paper in which polyethylene wear was the leading cause of TKA failure [9], polyethylene wear in the current study represented only 10% of all revisions. This drop reflects changes in both implant design and polyethylene manufacturing during the past 15 years that have led to decreased polyethylene wear. Implant design changes have improved articulations to avoid point-on-point contact areas, improved locking mechanisms to ensure secure capture, and produced highly polished surfaces to minimize polyethylene wear [12,13]. Even more significant was the realization that polyethylene sterilized by gamma irradiation in air caused oxidative degradation and increased polyethylene wear [14]. The majority of knee failures for polyethylene wear reported by Sharkey et al were done to replace damaged polyethylene components [9]. Polyethylene sterilization changes, such as sterilization in an oxygen-free environment, have significantly improved polyethylene wear properties [15]. Today, only revisions performed on knees more than 15 years postoperatively are likely to have polyethylene more susceptible to oxidation, leading to increased wear and clinical failure. Current knee components have improved designs and the wear properties of polyethylene have been improved, resulting in low knee failure due strictly to implant performance.

Table
Mechanism of Failure Over Time.

			<2 Years		2–5 Years		5–15 Years		>15 Years	
All Patients	844	100.0%	298	35.3%	210	24.9%	249	29.5%	87	10.3%
Aseptic Loosening	263	31.2%	56	18.8%	82	39.0%	99	39.8%	26	29.9%
Instability	158	18.7%	75	25.2%	39	18.6%	40	16.1%	4	4.6%
Infection	137	16.2%	68	22.8%	35	16.7%	29	11.6%	5	5.7%
Poly Wear	84	10.0%	3	1.0%	1	0.5%	38	15.3%	42	48.3%
Arthrofibrosis	59	7.0%	38	12.8%	15	7.1%	5	2.0%	1	1.1%
Malalignment	56	6.6%	24	8.1%	16	7.6%	15	6.0%	1	1.1%
Isolated Patella Revision	35	4.1%	15	5.0%	9	4.3%	8	3.2%	3	3.4%
Periprosthetic Fracture	27	3.2%	7	2.3%	5	2.4%	12	4.8%	3	3.4%
Other	13	1.5%	7	2.3%	4	1.9%	1	0.4%	1	1.1%
Extensor Mechanism	10	1.2%	5	1.7%	4	1.9%	1	0.4%	0	0.0%
AVN patella	2	0.2%	0	0.0%	0	0.0%	1	0.4%	1	1.1%

The predominant failure mechanisms in this study surround the surgical procedure. Infection, instability, malalignment, arthrofibrosis, and aseptic loosening are, at least in part, under the surgeon's control. In this study, infection accounted for 22.8% of revisions less than two years from the index procedure. Similar infection rates (25.2%) were seen in an assessment of the NIS database [11]. Infections that occur early are most likely the result of the operating room environment, intraoperative contamination, or immediate postoperative wound complications [16]. Risk factors often are divided into host and surgical factors. The surgeon has limited control over the host factors, but may affect them with careful patient selection, education, and aggressive medical optimization in an effort to decrease infection. Patients who are malnourished, morbidly obese, tobacco abusers, or have associated high-risk medical comorbidities such as renal failure and diabetes mellitus, are at increased risk of developing wound-healing problems [17]. Perioperative factors, such as appropriate antibiotic administration, minimizing operating room traffic, avoiding soft-tissue trauma, and careful hemostasis, all have contributed to decreased TKA infection rate [16,18]. Fehring et al reported infection as the leading cause in 38% of 279 revisions occurring less than two years from the index procedure. They concluded that prevention of early infection requires attention to perioperative detail [8]. While difficult to completely eliminate, infection rates can be reduced if the surgeon addresses host factors and mandates an emphasis on quality control and standardization of perioperative care.

In the current study instability was the leading cause of knee failure at less than 2 years, and the second most common failure mechanism from 2 to 15 years. Primary instability occurs intraoperatively when ligaments are balanced poorly, leading to varus–valgus instability or failure to properly balance the flexion–extension gaps [19]. This preventable mechanism of early failure occurred in 25.2% of knee revisions less than two years after their index procedure, which is similar to previous reports in the literature [8,19,20]. Progressive instability occurs in knees with initial well-functioning implants due to ligament loosening [19,20]. Progressive instability appears to be multifactorial, with possible causes including malalignment, excessive activity, and injury. [21] Repetitive injury from excessive stress on the collateral ligaments secondary to obesity also may lead to progressive instability [19].

Multiple patient-related conditions have been associated with increased stiffness after TKA. Diabetes, rheumatoid arthritis, ankylosing spondylitis, as well as restricted preoperative motion have been associated with arthrofibrosis [22]. In this study, revisions performed for knees with limited range of motion occurred in 12.8% of revisions in the first two years after the index procedure. Similar to other failure mechanisms that predominantly occur early, arthrofibrosis has several etiologies related to poor surgical technique. Poor ligament balance, component malposition, and oversized components all have been associated with restricted motion [23]. An unrecognized tight posterior cruciate ligament may lead to tight flexion, extension, or both [24,25]. Arthrofibrosis rarely was the cause of late revision with only 10% being performed after 5 years. An initially successful knee that develops arthrofibrosis over time likely has an underlying alternative primary mechanism of failure such as aseptic loosening, infection, or polyethylene wear, which all lead to increasing synovitis, mechanical pain, and secondary restriction of motion.

Implant malalignment has received more attention than any other aseptic mechanism of loosening over the past 15 years. Initial reports have demonstrated the increased risk of knee failure with a mechanical alignment error of more than three degrees, which is magnified in patients with higher BMI [26]. This has been questioned by a recent study suggesting that slightly malaligned knees do just as well [27]. However, the overall failure rate in this small cohort of patients was higher than the alignment-based aseptic loosening rates in the study by Ritter et al [26], suggesting a multifactorial pathophysiology of alignment-based failure in TKA.

Alternative surgical techniques have been offered over the past 15 years in an attempt to improve implant alignment and, as a result, TKA outcomes. Computer-assisted orthopedic surgery (CAOS) and patient-specific cutting guides have been developed as alternative surgical alignment techniques. While both techniques have demonstrated some improvement in implant alignment, both add significant cost to the overall episode of care and have not yet demonstrated a significant cost–benefit argument [28,29]. Minimally invasive surgical techniques, developed over the past decade in an effort to minimize soft-tissue damage and improve knee recovery, have been associated with an increased rate of malalignment resulting from the adoption of these MIS techniques [30]. None of these surgical techniques, whether potentially positively or negatively affecting alignment, have been adopted in large enough numbers to impact overall TKA failure rate. In this study, malalignment represented less than 7% of all revisions.

Aseptic loosening was the most common failure mechanism in this study (31.2%). Unlike other etiologies that occurred either early or late, aseptic loosening occurred frequently throughout follow-up. Of all the failure mechanisms, this is the least understood, and certainly is the “catch-all” diagnosis of failed knees in which an alternative diagnosis could not be made. This category represents an extended list of subcategories of failure mechanisms. Fehring et al reported 13% of early failures were failures of cementless fixation, pointing out that the etiologies of aseptic loosening of cementless implants will vary from cemented implants [8]. In addition, variations in surgical technique among surgeons may contribute to aseptic loosening. Higher loosening has been demonstrated in cemented knees when the tibial stem is left uncemented [31]. Variations among implants within a single product line and among different manufacturers may lead to different potential failure mechanisms. Tibial trays with short stems have been associated with an increased rate of aseptic loosening [32]. Implant specific failure reports have shown that specific implants have had a high early rate of aseptic loosening [33]. In this study, no attempt was made to subcategorize aseptic loosening. Moving forward, continued efforts need to be made to better define and address aseptic loosening. The authors would suggest that the term ‘aseptic loosening’ be used when the implant was initially well fixed and subsequently loosened. These patients were generally well satisfied with their knee initially. This would contrast with ‘failure of fixation’ for implants that were never secure in which patients were never satisfied with their knee.

In summary, the majority of knee failures still occur early; 35.3% within 2 years and 60.2% within 5 years of the index procedure. The mean time to failure was 5.9 years (range 10 days to 31 years). Aseptic loosening was the predominant failure mechanism, but is poorly defined and least understood. Infection, instability, arthrofibrosis, and malalignment were found in the current study to predominantly occur early. Multiple factors under the surgeon's control led to these early failure mechanisms. In contrast to previous reports, which likely are biased by differences in implant designs and polyethylene quality, wear was not a primary mechanism of failure and rarely presented prior to 15 years. Implant performance was not a predominant factor of knee failure. Improving surgeon performance through training, instrumentation, and technique development may reduce early revisions.

References

1. Losina E, Thornhill TS, Rome BN, et al. The dramatic increase in total knee replacement utilization rates in the United States cannot be fully explained by growth in population size and the obesity epidemic. *J Bone Joint Surg Am* 2012;94:201.
2. Kurtz SM, Ong K, Lau E, et al. Projections of primary and revision hip and knee arthroplasty in the United States from 2005 to 2030. *J Bone Joint Surg Am* 2007;89:780.
3. Font-Rodriguez DE, Scuderi GR, Insall JN. Survivorship of cemented total knee arthroplasty. *Clin Orthop Relat Res* 1997;345:79.

4. Keating EM, Meding JB, Faris PM, et al. Long-term follow-up of nonmodular knee replacements. *Clin Orthop Relat Res* 2002;404:34.
5. Vessely MB, Whaley AL, Harmsen WS, et al. The Chitranjan Ranawat Award: long-term survivorship and failure modes of 1000 cemented condylar knee arthroplasties. *Clin Orthop Relat Res* 2006;452:28.
6. Australian Orthopaedic Association's National Joint Registry, 2010.
7. Bourne RB, Maloney WJ, Wright JG. An AOA critical issue. The outcome of the outcomes movement. *J Bone Joint Surg Am* 2004;86:633.
8. Fehring TK, Odum S, Griffin WL, et al. Early failures in total knee arthroplasty. *Clin Orthop Relat Res* 2001;392:315.
9. Sharkey PF, Hozack WJ, Rothman RH, et al. Insall Award Paper. Why are total knee arthroplasties failing today? *Clin Orthop Relat Res* 2002;404:7.
10. Lynch JR, Cunningham MR, Warme WJ, et al. Commercially funded and United States-based research is more likely to be published; good-quality studies with negative outcomes are not. *J Bone Joint Surg Am* 2007;89:1010.
11. Bozic KJ, Kurtz SM, Lau E, et al. The epidemiology of revision total knee arthroplasty in the United States. *Clin Orthop Relat Res* 2010;468:45.
12. Azzam MG, Roy ME, Whiteside LA. Second generation locking mechanisms and ethylene oxide sterilization reduce tibial insert backside damage in total knee arthroplasty. *J Arthroplasty* 2011;26(4):523.
13. Galvin AL, Kang L, Udofia I, et al. Effect of conformity and contact stress on wear in fixed-bearing total knee prostheses. *J Biomech* 2009;42:1898.
14. Collier JP, Sperling DK, Currier JH, et al. Impact of gamma sterilization on clinical performance of polyethylene in the knee. *J Arthroplasty* 1996;11:377.
15. Griffin WL, Fehring TK, Pomeroy DL, et al. Sterilization and wear-related failure in first- and second-generation press-fit condylar total knee arthroplasty. *Clin Orthop Relat Res* 2007;464:16.
16. Panahi P, Stroh M, Casper DS, et al. Operating room traffic is a major concern during total joint arthroplasty. *Clin Orthop Relat Res* 2012;470:2690.
17. Malinzak RA, Ritter MA, Berend ME, et al. Morbidly obese, diabetic, younger, and unilateral joint arthroplasty patients have elevated total joint arthroplasty infection rates. *J Arthroplasty* 2009;24(6 Suppl):84.
18. Prokuski L. Prophylactic antibiotics in orthopaedic surgery. *J Am Acad Orthop Surg* 2008;16:283.
19. Vince KG, Abdeen A, Sugimori T. The unstable total knee arthroplasty: causes and cures. *J Arthroplasty* 2006;21(4 Suppl 1):44.
20. Yercan HS, Ait Si Selmi T, et al. Tibiofemoral instability in primary total knee replacement: a review, part 1: basic principles and classification. *Knee* 2005;12:257.
21. Leopold SS, McStay C, Klafeta K, et al. Primary repair of intraoperative disruption of the medial collateral ligament during total knee arthroplasty. *J Bone Joint Surg Am* 2001;83:86.
22. Jordan L, Kligman M, Sculco TP. Total knee arthroplasty in patients with poliomyelitis. *J Arthroplasty* 2007;22:543.
23. Scuderi GR. The stiff total knee arthroplasty: casualty and solution. *J Arthroplasty* 2005;4(Suppl 1):23.
24. Laskin R. Total knee replacement with posterior cruciate ligament retention in patients with a fixed varus deformity. *Clin Orthop Relat Res* 1996;331:29.
25. Lombardi AV, Mallory TH, Fada RA, et al. An algorithm for the posterior cruciate ligament in knee arthroplasty. *Clin Orthop Relat Res* 2001;392:75.
26. Ritter MA, Davis KE, Meding JB, et al. The effect of alignment and BMI on failure of total knee replacement. *J Bone Joint Surg Am* 2011;93:1588.
27. Parratte S, Pagnano MW, Trousdale RT, et al. Effect of postoperative mechanical axis alignment on the 15-year survival of modern, cemented total knee replacements. *J Bone Joint Surg* 2010;92:2143.
28. Novak EJ, Silverstein MD, Bozic KJ. The cost-effectiveness of computer-assisted navigation in total knee arthroplasty. *J Bone Joint Surg Am* 2007;89:2389.
29. Nunley RM, Ellison BS, Ruh EL, et al. Are patient-specific cutting blocks cost-effective for total knee arthroplasty? *Clin Orthop Relat Res* 2012;470:889.
30. Barrack RL, Barnes CL, Burnett RSJ, et al. Minimal incision surgery as a risk factor for early failure of total knee arthroplasty. *J Arthroplasty* 2009;24:489.
31. Bert JM, McShane M. Is it necessary to cement the tibial stem in cemented total knee arthroplasty? *Clin Orthop Relat Res* 1998;356:73.
32. Foran JRH, Whited BW, Sporer SM. Early aseptic loosening with a precoated low-profile tibial component. A case series. *J Arthroplasty* 2011;26:1445.
33. Arsoy D, Pagnano MW, Lewallen DG, et al. Aseptic tibial debonding as a cause of early failure in a modern total knee arthroplasty design. *Clin Orthop Relat Res* 2013;471:94.