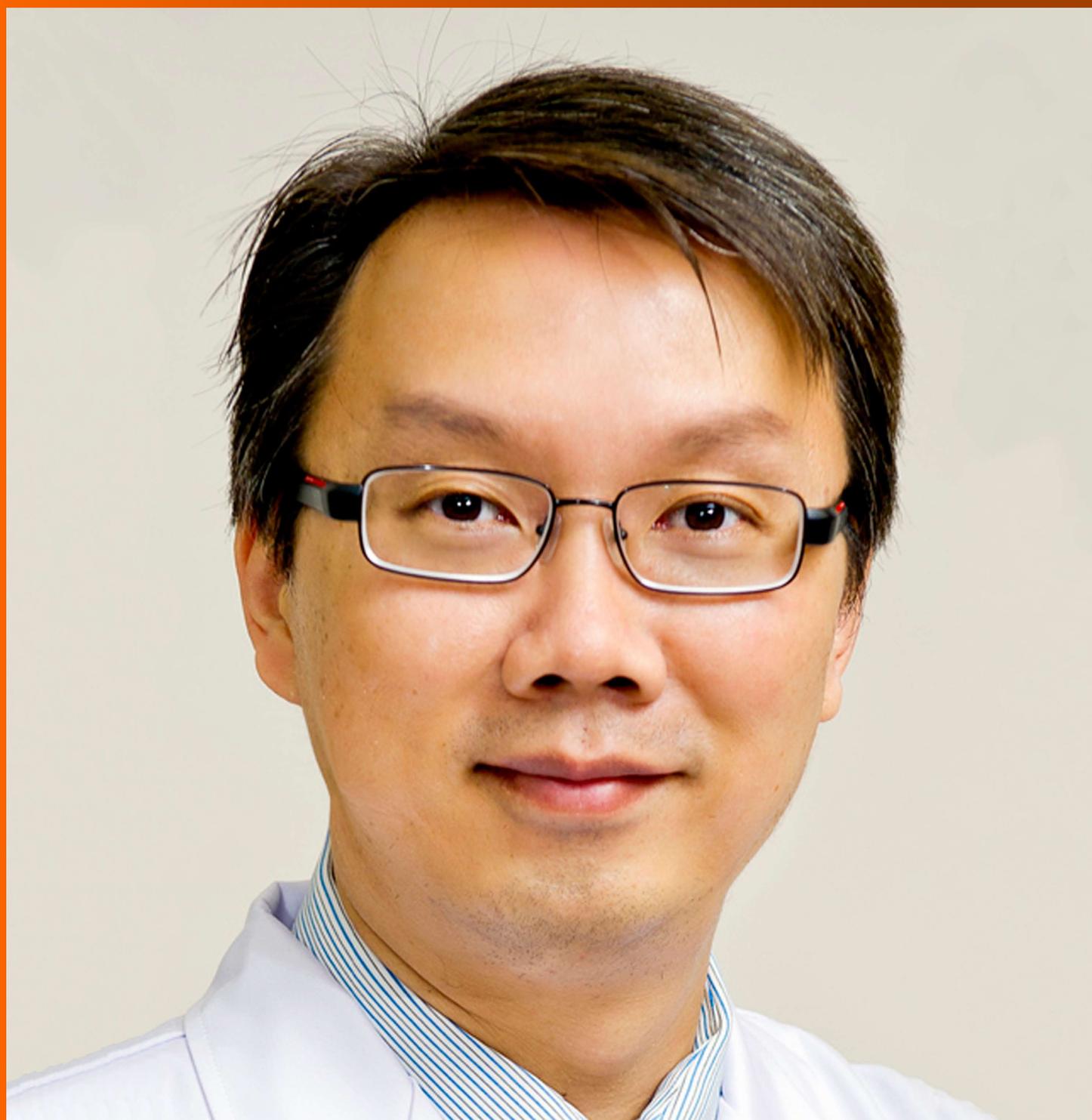
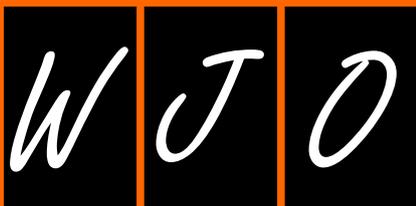


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Why total knees fail-A modern perspective review

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arthroplasty (TKA) failures included aseptic loosening, instability and malalignment. As polyethylene production improved, modes of failure from polyethylene wear and subsequent osteolysis became less prevalent. Newer longitudinal studies report that infection has become the primary acute cause of failure with loosening and instability remaining as the overall greatest reasons for revision. Clinical database and worldwide national registries confirm these reports. With an increasing amount of TKA operations performed in the United States, and with focus on value-based healthcare, it is imperative to understand why total knees fail.

Key words: Total knee arthroplasty failure mechanism; Total knee arthroplasty failure mode; Revision total knee arthroplasty; Periprosthetic joint infection; Aseptic loosening total knee; Total knee arthroplasty instability

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Core tip: With increasing number of revision total knee arthroplasty (TKAs) being performed, tighter control on healthcare costs and value based care may occur. Surgeons are tasked with the responsibility to avoid risk factors for revision TKA. Newer longitudinal studies report that infection has become the primary acute cause of failure with loosening and instability remaining as the overall greatest reasons for revision. The surgeon must be aware of the risk factors and preventative measures for these failure modes, including preoperative management, surgical techniques and enhanced materials.

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Abstract

Historically, the most common mechanism of total knee

INTRODUCTION

With more total knee arthroplasty (TKA) operations

being performed and patients' lifespan increasing, there is an ever-growing number with this operation in the United States. Based upon 2010 data, an estimated 4.7 million individuals (3.0 million females, 1.7 million males) are living in the United States with a total knee^[1]. Additionally as the average age for TKA is becoming younger and living longer, the total number of revisions performed increases. By 2020, it is estimated 1.3 million TKAs will be performed along with 127000 revisions^[2]. Also, with focus on value-based healthcare, and government bundled payment initiatives, it is imperative to understand why total knees fail^[3].

HISTORICAL PERSPECTIVE

In 1982, Rand *et al*^[4] reported 227 knees undergoing revision in the Mayo Clinic registry data from 1970 to 1980. Within those revisions, average time from initial arthroplasty to failure was 2.7 years with loosening (34.9%) being the major cause for TKA failure. Instability and malalignment were second and third at 16.7% and 14.8%, respectively. Component malposition, periprosthetic fracture and patellofemoral complications were all around 5%. Periprosthetic joint infections were reported to be exceedingly rare at 0.2% (but this will become the leading cause of revision). The dominance of loosening was likely because of high use of the older hinge prosthesis designs such as the Guepar that resulted in increased interface stresses and loosening.

In 1988, Moreland described the etiology of total knee failures, and loosening and instability were still the leading causes with secondary reasons being infection, extensor mechanism disruption, arthrofibrosis, periprosthetic fracture and complex regional pain syndrome^[5]. Loose implants were caused by inappropriate bony resection, poor ligamentous balancing, cement technique, patient factors such as high activity level, implant constraint level, and osteolysis. Instability had the characteristics of varus/valgus malalignment, imbalance of the flexion-extension gap, anteroposterior laxity and patellofemoral subluxation/dislocation. Moreland concluded a majority of failure mechanisms is under the surgeon's technical control.

In 2001, Fehring *et al*^[6] reported early (< 5 year) failure mechanisms between 1986 and 1999. Their most common identified etiology for failure in 279 knees was now infection at 38%. Aseptic loosening of cemented implants had plummeted to 3% with lack of cementless TKA ingrowth at 13%, and polyethylene wear/osteolysis which causes loosening at 7%. Instability was still high at 26% with patellofemoral failures (usually instability) being 8%. Five percent had miscellaneous problems such as arthrofibrosis, malalignment, or periprosthetic fracture. They also concluded some of these causes could be improved by surgical technique and perioperative care. They proposed that infection prevention could be reduced by addressing wound healing problems (such as albumin > 3.5 g/L, preoperative total lymphocyte count (TLC) > 1500 cells/mm³, and transferrin level > 200),

reducing traffic in the operating room (OR), appropriate sterile technique and managing the operating room air environment. Their emphasis was that early failures could be controlled to improve implant longevity.

Sharkey *et al*^[7] won the 2002 Knee Society award paper for their review of TKA failures. They categorized their 212 TKA failures into early (< 2 years) and late (> 2 years). Early failures most commonly were infection in 25% of knees, instability (21%), arthrofibrosis (17%) and loosening (16%). Late failure groups were similar in numerical order to the overall cohort, reporting polyethylene wear (44%), loosening (34%) and instability (22%) as the major 3 causes. Overall, the most common failure mechanisms in decreasing order were estimated at 27% polyethylene wear, 25% component loosening, 21% instability and 17% infection. These authors also concluded that attention to surgical technique and postoperative care by the surgeon was very important but because multiple failure mechanisms were often seen in one case, some of the failure mechanisms could be addressed by design and material improvement.

In 2006, Mulhall *et al*^[8] reported on overall, early (< 2 years) and late (> 2 years) TKA failure mechanisms in 318 patients. Overall the majority of revisions were after 2 years and thus mechanical issues were primarily the cause. Early revisions (31% of patients) were primarily due to infection (25%), with ultimate outcomes worse than the outcomes of knees with revision for aseptic loosening. Revisions after 2 years (69% of their patients), were mechanical with instability (29%), polyethylene wear (25%) and component loosening (41%). They also concluded that patient factors such as diabetic control, and technical factors such implant design could be modifiable between early and late failures to improve patient outcomes.

The summary of these longitudinal studies is that infection has become the primary acute cause of failure with loosening and instability remaining as the overall greatest reasons for revision (Table 1). The researchers' conclusions are that most failures can be avoided by improvements in technique and design.

SHIFT TO NEWER POLYETHYLENE MANUFACTURING PROCESSES

Design improvement has impacted failure mechanisms. As polyethylene production improved, modes of failure from polyethylene wear and subsequent osteolysis became less prevalent. Hossain *et al*^[9] studied revisions of 349 knees between 1999 and 2008. Infection had become the most common reason for revision overall, both in early (< 2 years) and in late (> 2 years) failures. Aseptic loosening was second most common, followed by polyethylene wear.

Hossain *et al*^[9] and Schroer *et al*^[10] performed multi-center analysis of etiology for 844 revision TKAs between 2010 and 2011. They found aseptic loosening (31.2%), instability (18.7%) and infection (16.2%) as the 3 major overall causes with early failures continuing to

Table 1 Clinical studies by failure mechanism (%)

Ref.	Knees	Loosening	Infection	Instability	Malalignment	Poly/Lysis	Other
Rand <i>et al</i> ^[4]	227	34.9	0.2	16.7	14.8	-	5
Moreland <i>et al</i> ^[5]		MC		2 nd MC			
Fehring <i>et al</i> ^[6]	279	3%	38	26	5	7	5
Sharkey <i>et al</i> ^[7]	212	17/34	25/7.8	21/22	12/12	12/44	
Mulhall <i>et al</i> ^[8]	318	41	25/7	29	9	6/25	
Hossain <i>et al</i> ^[9]	349	3/12	12/21	4/3	4/3	1/12	
Schroer <i>et al</i> ^[10] and Lombardi <i>et al</i> ^[11]	844	19/31	23/16	25/19	8/7	1/10	2/1
Sharkey <i>et al</i> ^[12]	781	22/40	38/28	12/8	3/2	2/4	
Delanois <i>et al</i> ^[14]	337597	20.3	20.4	7.5		2.6	12
Kasahara <i>et al</i> ^[19]	147	40	24	9		9	18
Koh <i>et al</i> ^[20]	634	33	38	7	1	15	8

Overall percentages listed above may be approximates. Percentages may not be mutually exclusive^[7,9-11]. Sharkey *et al*^[7] table: First number is early (< 2 year) failures, second number is late failures; Hossain *et al*^[9]: First number is early (< 2 year) failures, second is late failures; Schroer and Lombardi *et al*^[10-11]: First number is early (< 2 year) failures, second is overall failures.

be infection (23%) and instability (25%). Secondary causes included polyethylene wear (10%), arthrofibrosis (6.9%) and malalignment (6.6%) with stiff knees being predominantly an early cause for revision among these. Polyethylene wear represented less than 1% of revisions performed under 5 years, but remained common in revision failures greater than 15 years in knees with older polyethylene which confirmed benefit of newer polyethylene improvements. Aseptic loosening was the only failure mechanism consistent in all time intervals^[11].

Sharkey *et al*^[12] provided a 10-year update on their experience of performing 781 revisions of 10,003 total procedures (7.8% revision rate). They too saw a dramatic decrease from 25% to 3.5% in the rate of polyethylene wear as the cause of revision. Early (< 2 years) failures were 37.6% of all failures with infection most common, and more than half (51.4%) of the 62.4% late (> 2 years) revisions were aseptic loosening.

CLINICAL DATA VS LARGE DATA

While most published results of total knee revisions came from single-center or regional multi-center data, larger and more diverse cohorts have become possible with the advent of nationwide databases. In the United States, the Nationwide Inpatient Sample (NIS) database, developed in 1988 and revamped in 2012, provides a random sampling of approximately 20% of all United States hospital discharges and encompasses smaller community hospitals as well as larger urban academic centers. Using this registry, Bozic *et al*^[13] reported on 60355 TKA revision procedures performed between 2005 and 2006 across the United States. They found the most common cause of revision knee arthroplasty was infection at 25.2%, implant loosening at 16.1%, and implant failure or breakage at 9.7%. While noting the limitations of large administrative data, they reported their findings were similar to other studies that found infection to be the greatest contributing factor to at least early failure mechanisms.

Delanois *et al*^[14] provided an updated look at the revision rate in the United States using the same NIS database from 2009 through 2013 and reaffirmed that the two leading causes of revision TKA were infection and aseptic loosening at 20.4% and 20.3%, respectively. Both NIS-based papers reported on higher revision rates in the South with upwards of one-third of all revision performed in southern states. Although demographic data was provided, with well over 70% of all revision occurring in Caucasians, no analysis was performed to identify regional differences in failure mechanisms. All-component revision was the most common operation with a total healthcare cost averaging more than \$75000.

WORLD EXPERIENCES

The use of nationwide registries began in 1975 with the Swedish Knee Arthroplasty Register (SKAR) through the efforts of Goran Bauer^[15]. Since then, other countries have followed their example, including Finland (1980), Norway (1987), Denmark (1995), South Korea (1989), New Zealand (1998), England and Wales (2003), and Japan (2010) (Table 2).

The initial success of the registries prompted the creation of the Nordic Arthroplasty Register Association, a compilation of arthroplasty databases from Sweden, Denmark, and Norway who shared similar demographics, healthcare and socioeconomic systems, and were in close proximity to each other. Subsequently Niimaki in 2015 combined five worldwide registries: Australia, New Zealand, Norway, Sweden, and England and Wales^[16]. The leading indication for revision in each country was aseptic loosening (range 22.8%-29.7%). Pain was the second leading indication for revision in Norway and New Zealand (27.4% and 22.0% respectively), while infection was the second most common cause in the three remaining countries (20.6%-21.7%). However, Niimaki identifies inconsistencies in the categorization of failure mechanisms amongst the registries that clouds the ability to interpret the results. For example, pain and malalignment are not categories in the Swedish

Table 2 Large data and registry data by total knee arthroplasty failure mechanism

Ref.	Knees	Loosening, %	Infection, %	Instability, %	Poly/Lysis, %	Other, %
Bozic <i>et al</i> ^[13]	60435	16	25	7	8	
Delanois <i>et al</i> ^[14]	337597	20.3	20.4	7.5	2.6	12
Sadoghi <i>et al</i> ^[24]	36307	30	15	6	8	
Australia 2003-2012 ^[25]	31698	30	22	6	2	
England/Wales 2011-2012 ^[26]	5135	35	23	14	20	
New Zealand 1999-2011 ^[27]	4603	37	24	7	n/a	
Norway 1994-2009 ^[28]	3445	24	13	10	5	
Sweden 2001-2010 ^[29]	3375	26	23	13	5	

Overall percentages are listed above.

registry whereas polyethylene wear is not an option in the United Kingdom registry. He therefore suggests that standardizing the registries can help in compiling data to draw more compelling conclusions. Nevertheless, the data consistently supports aseptic loosening as the most common indication for revision TKA at similar rates to those found in the United States. Siqueira *et al*^[17] reviewed TKA failure modes outside the United States by combining both large clinical studies and national joint registry results. They concluded 1994-2012 national databases and reported aseptic loosening as the most common reason for failure, with infection being second.. Clinical studies reported by large tertiary referral centers also reported aseptic loosening as being the most common overall reason for revision, while early failures were due to infection. The data from Europe, although not consistently reported, confirms that the emphasis on failure needs to be focused on infection early and overall on aseptic loosening.

The performance of TKA is quickly rising in Asia, where over half the world's population resides, and it is especially prevalent in women, who have an 8-fold increased rate of primary TKAs compared to men^[18]. Kasahara *et al*^[19] recently reported on a multicenter experience of five arthroplasty referral centers in Japan with 140 TKA revision from 2006-2011. Overall revision rate was 3.3% with aseptic loosening as the leading cause at 40% followed by infection at 24%. Koh *et al*^[20] from South Korea, published a retrospective review of 634 revisions at 19 centers from 2008-2012, representing an estimated 10% of all procedures performed in the country. Overall revision rate was 3.0% with infection (38%) as the leading cause followed by aseptic loosening (33%) and wear (13%). Similar to other reports, they separated failures as early (< 2 years) versus late (> 2 years); infection dominated as the leading cause of early failure (77%) but it was only 23% of all late failures with aseptic loosening (44%) the most common as it is throughout the world. Wear was only an indication for revision in the late failure group and comprised 18%. With limited long-term registry data, it remains unclear whether the failure patterns of knee replacement differ between the Western and Eastern Hemispheres, but it seems that Asia is more similar to the United States with infection the early

cause while Aseptic loosening dominates all time periods in Europe.

CURRENT CHALLENGES

As total knee arthroplasty increases in demand and prevalence, the number of revision total knee operations increase as well. Kurtz *et al*^[3] predicted the number of revision TKAs performed in the United States by 2030 would be greater than 250000 operations. Hamilton *et al*^[21] reviewed risk factors for revision TKA which includes obesity, young age and comorbid conditions as the most common in both the United States as well as other countries.

Altogether, patients across the world with total knee arthroplasties face similar challenges today. Aseptic loosening/instability and infection are the primary causes of failure. Countries with higher rates of unicompartmental or bicompartmental arthroplasties increasingly cite pain as an indication for revision, though that remains highly dependent on the patient, the surgeon, and the reporting mechanism. As surgical implants continue to evolve, surgical techniques to achieve long-term fixation and careful attention to infection prevention remain the most challenging obstacles to achieve excellent long-term outcomes.

As the understanding of how total knees fail, orthopedic research has focused on improving the technology and surgical technique as well as in depth study of infection. There is a large volume of research dedicated towards lowering infection risk factors such as patient optimization, efficient surgery, maintaining ideal intraoperative conditions, and decreasing postoperative complications^[22]. Surgical technique has focused on understanding patient anatomy, and personalizing leg alignment and component position. Bellemans *et al*^[23] evaluated anatomic and mechanical axis in 250 asymptomatic adults. They reported that 32% of males and 17% of females had a natural mechanical axis of 3 degrees varus or greater. A common etiology of instability is malrotation of the femoral component relative to the tibia. Meticulous attention to surgical technique is critical as instrumentation is unable to adjust for this rotation. Personalization of a patient's normal anatomy and ligament balancing may be helpful to lower revision rates

and patient satisfaction.

CONCLUSION

With increasing number of revision TKAs being performed, tighter control on healthcare costs and value based care may occur. Surgeons are tasked with the responsibility to avoid risk factors for revision TKA. Newer longitudinal studies report that infection has become the primary acute cause of failure with loosening and instability remaining as the overall greatest reasons for revision. Knowledge of total knee arthroplasty failure mechanisms allows the arthroplasty surgeon to be aware of individual risk factors, and to strategize management for each patient to optimize their care.

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Basic Study

Snapping elbow-A guide to diagnosis and treatment

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Abstract**AIM**

To develop practical guidelines for diagnosis and treatment of the painful snapping elbow syndrome (SE).

METHODS

Clinical studies were searched in the databases PubMed and Scopus for the phrases "SE", "snapping triceps", "snapping ulnar nerve" and "snapping annular ligament". A total of 36 relevant studies were identified. From these we extracted information about number of patients, diagnostic methods, patho-anatomical findings, treatments and outcomes. Practical guidelines for diagnosis and treatment of SE were developed based on analysis of the data. We present two illustrative patient cases-one with intra-articular pathology and one with extra-articular pathology.

RESULTS

Snapping is audible, palpable and often visible. It has a lateral (intra-articular) or medial (extra-articular) pathology. Snapping over the medial humeral epicondyle is caused by dislocation of the ulnar nerve or a part of the triceps tendon, and is demonstrated by dynamic ultrasonography. Treatment is by open surgery. Lateral snapping over the radial head has an intra-articular pathology: A synovial plica, a torn annular ligament or a meniscus-like remnant from the foetal elbow. Pathology can be visualized by conventional arthrography, magnetic resonance (MR) arthrography, high resolution magnetic resonance imaging (MRI) and arthroscopy, while conventional MRI and radiographs often turn out normal. Treatment is by arthroscopic or eventual open resection. Early surgical intervention is recommended as

the snapping can damage the ulnar nerve (medial) or the intra-articular cartilage (lateral). If medial snapping only occurs during repeated or loaded extension/flexion of the elbow (in sports or work) it may be treated by reduction of these activities. Differential diagnoses are loose bodies (which can be visualized by radiographs) and postero-lateral instability (demonstrates by clinical examination). An algorithm for diagnosis and treatment is suggested.

CONCLUSION

The primary step is establishment of laterality. From this follows relevant diagnostic measures and treatment as defined in this guideline.

Key words: Elbow; Arthroscopy; Surgery; Diagnosis; Ultrasonography; Snapping

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Core tip: Elbow snapping is medial or lateral. Medial snapping is caused by dislocation of the ulnar nerve or a part of the triceps tendon, demonstrated clinically and by dynamic ultrasonography. Treatment is transposition of the nerve and/or resection of the snapping tendon. Lateral snapping is intra-articular by a synovial plica, a torn annular ligament or a meniscus-like remnant from the foetal elbow, demonstrated by arthrography, magnetic resonance arthrography, high resolution magnetic resonance imaging or arthroscopy. Treatment is arthroscopic resection. Early surgical intervention is recommended to reduce tissue damage. Medial snapping promoted by repeated, loaded activities might be treated by activity reduction.

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INTRODUCTION

Snapping elbow (SE) is a rare condition, which can be confused with more common pathologies like an intra-articular free body, lateral epicondylitis or medial epicondylitis. Symptoms from SE occur during dynamic activities. Therefore, standard radiographs and magnetic resonance imaging (MRI) are often normal, leaving risk that the condition remains undiagnosed.

It is useful to distinguish between lateral and medial snapping, as pathology, diagnostic strategy and treatment are different in the two situations.

We suggest a practical guideline for diagnosis and treatment of SE, based on our own experience with three elbows in two patients and a review of the literature.

MATERIALS AND METHODS

Literature was searched in Pubmed with four phrases: "SE" (resulting in 85 hits), "snapping triceps" (39 hits), "snapping ulnar nerve" (31 hits) and "snapping annular ligament" (9 hits). A similar search was performed in Scopus, and two additional, relevant articles were identified. We excluded papers that had no information about diagnostic strategy or treatment as well as articles in other languages than English or Scandinavian.

One article was not available online or from the author.

From the 36 remaining articles (Figure 1) information about number of patients, diagnostic method, patho-anatomical findings, treatments and outcomes was extracted.

We treated two cases with principally different reasons for snapping.

Case reports 1

A 16-year-old boy with painful locking of the right elbow during 2-3 years. No trauma in history. Standard MR scanning and radiographs were normal. A sore clicking was found at the lateral epicondyle with 80°-90° of elbow flexion, and it was most painful with simultaneous pronation. He had similar but milder symptoms on the left side. Both elbows were stable and with normal range of motion.

Dynamic ultrasound raised suspicion of a tight, lateral part of the triceps tendon as the cause of snapping. However, extraarticular injection of 1 cc Carbocain plus 1 cc of Depomedrol® in this area did not relieve the symptoms.

At arthroscopy an inter-positioned lunar plica antero-laterally (video 1) was resected, and a tight chord of capsule in relation to the plica was loosened.

Pain and snapping disappeared after the operation. A similar condition was treated by arthroscopy in the left elbow 6 wk later. At 3-years follow-up he felt a slight tenderness in the right elbow from time to time, but it was different from his preoperative pain and he had no snapping. The left elbow was symptom free.

Case reports 2

A 16-year-old boy with painful medial elbow clicking bilaterally for over a year, in particular during heavy resistance training of the triceps muscle. The snapping was mildly painful, but he had post-exercise pain that forced him to reduce the training load. Popping of the triceps muscle over the medial epicondyle at 110° of flexion was suspected by clinical examination (video 2). There were no neurological symptoms and a negative Tinel's test in relation to the ulnar nerve. Dynamic sonography visualized the popping tissue (video 3), but it was not possible to distinguish if this originated from the ulnar nerve or a part of the triceps tendon. The elbow appeared otherwise normal.

Open surgery showed that snapping was caused by

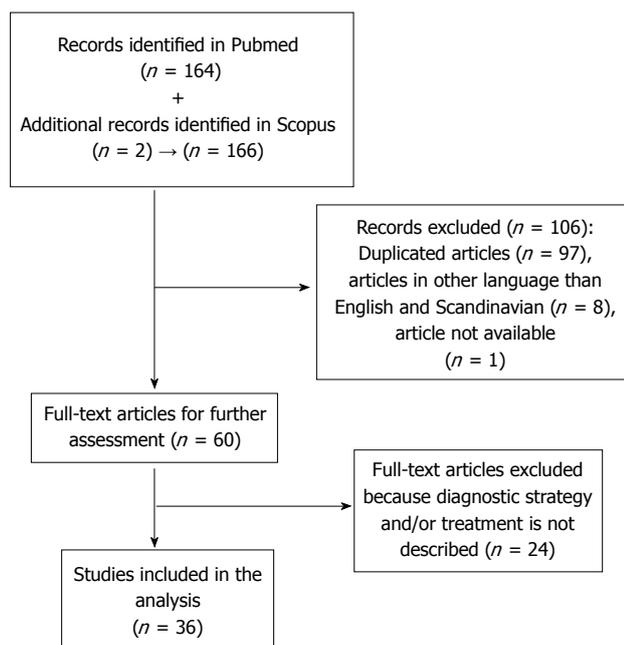


Figure 1 From the 36 remaining articles information about number of patients, diagnostic method, patho-anatomical findings, treatments and outcomes was extracted.

dislocation of the ulnar nerve (video 4). The sulcus was therefore deepened 3 mm, the nerve was repositioned, and a part of the medial triceps tendon was transpositioned to form a cover over the sulcus. This resolved the snapping condition, and the patient was able to exercise normally between two and five months postoperatively. Then a feeling of instability of the nerve during heavy resistance training occurred, but it was pain free. One year after the operation he stopped regular training and had no symptoms. This was still the case at 3-years follow-up. Both patients declared, that they were satisfied with the treatment, and with their present experience they would agree to be operated for SE again, if necessary.

RESULTS

The identified clinical reports of SE are summarized in the tables. They are divided into lateral, intra-articular cases (Table 1) and medial, extra-articular cases (Table 2) of SE. There are 105 cases of lateral snapping with a mean age of 39.7 years (range 11-66) and 42 cases of medial snapping with a mean age of 28.7 years (range 9-65). In some patients SE was presumably triggered by athletic performance (throwing and tennis typical for lateral snapping, and weightlifting, decathlon and dumbbell exercises for medial snapping). However, the majority of cases could not be connected to sports.

This review does not allow to make an estimate of the incidence of SE, but based on the reported cases, the different pathologies can be ranked with intraarticular plica as the most common, followed by medially snapping triceps (rare), snapping ulnar nerve (rare), snapping annular ligament (very rare)

and extremely rare pathologies: snapping medial antebrachial cutaneous nerve, intraarticular meniscus and laterally snapping triceps.

Intra-articular, lateral snapping

Intra-articular snapping is caused by annular ligament pathology, lateral meniscal remnants or hypertrophic synovial plicas^[1-12]. Clicking due to dynamic impingement of these soft tissues is difficult to demonstrate with static MRI.

Three reports describe snapping caused by anomalous meniscal remnants^[5,7]. Standard MRI did not reveal the pathology, but it was demonstrated by MRI- and radiographic arthrography, respectively.

In one report a plica was demonstrated by radiographic arthrography and MRI as a protruding shadow^[11]. Contrast MRI was used to diagnose three cases of a snapping plica^[10]. In one case conventional MRI raised suspicion of a small, pathological structure, which was diagnosed as a plica by high-resolution MRI^[13].

By conventional MRI it was in most cases not possible to visualize lateral SE pathology, and diagnoses were established with MR-arthrography, high resolution MRI or radiographic arthrography. With arthroscopy the intra-articular snapping pathology can be visualized and treated in the same procedure by resection of the snapping tissue.

Arthroscopic resection of plicas or meniscus-like tissues was standard treatment, except in three cases of open resection. Reported results were good with either method (Table 1), as elbow function normalized in the majority of cases.

One patient who had postero-lateral rotatory elbow instability did not improve, but the snapping might have been caused by instability and not by intra-articular pathology^[2].

Extra-articular, medial snapping

Subluxation of either the ulnar nerve or a medial part of the triceps tendon, or of both can cause extra-articular snapping of the elbow^[15-26]. In many cases it can be recognized by thorough physical examination. The ulnar nerve snaps in the interval 70-90 degrees of flexion, and the triceps around 115 degrees. These snaps are usually visible and audible^[27]. By dynamic ultrasound and dynamic MRI^[28] the snapping tissue can be visualized, but interpretation of the anatomical structures is not always conclusive, as described in our case 2^[20,27,28]. Dislocation of the snapping structure is not visible on standard MRI with the elbow extended, but with the elbow flexed dislocation can often be demonstrated^[29].

A snapping triceps can be treated by either resection^[19,22-23,25,30-34] or by suture of the snapping part to the main tendon^[18,25]. Medial epicondylectomy was used in one case^[19]. Ulnar nerve dislocation was treated by anterior transposition^[16,26,35] or fixation of the nerve in the deepened cubital tunnel^[35].

Table 1 Literature reports on intra-articular snapping elbow

Report and year	No. of patients	Snapping Pathology	Diagnostic procedure	Treatment	Results
Akagi <i>et al</i> ^[1] , 1998	One	Snapping plica	Arthrogram, arthroscopy	Open arthrotomy	Complete recovery
Antuna <i>et al</i> ^[2] , 2001	Fourteen	Snapping plica	MRI, arthroscopic examination	Arthroscopic resection	Ten complete recoveries, two partial recoveries, two failures
Aoki <i>et al</i> ^[3] , 2003	Two	Snapping annular ligament	Arthroscopic examination	One had arthroscopic resection	One complete recovery after resection
Chai <i>et al</i> ^[4] , 2004	One	Snapping annular ligament	Ultrasonography	Arthroscopic resection	Relief of snapping
Fukase <i>et al</i> ^[13] , 2005	One	Snapping plica	Special MRI ¹	Open resection	Complete recovery
Huang <i>et al</i> ^[5] , 2005	One	Snapping meniscus	MR-arthrography	Arthroscopic resection	Complete recovery
Huang <i>et al</i> ^[6] , 2005	One	Snapping annular ligament	MRI, arthroscopic examination	Arthroscopic resection	Relief of snapping, less pain
Kang <i>et al</i> ^[7] , 2010	Two	Snapping meniscus	MRI, arthrography	Arthroscopic resection	Complete recovery
Maruyama <i>et al</i> ^[8] , 2010	One	Snapping annular ligament	Arthroscopy	Arthroscopic release of the ligament	Complete recovery
Brahe Pedersen <i>et al</i> ^[9] , 2017	Sixtyfour	Hypertrophic synovial plica	Clinical examination and ultrasound	Arthroscopic resection	Significant improvement in Oxford Elbow score after 3 and 22 mo
Shinohara <i>et al</i> ^[14] , 2010	One	Tight fibrous structure	MRI, arthroscopy	Arthroscopic resection	Complete recovery
Steinert <i>et al</i> ^[10] , 2008	Three	Hypertrophic synovial plica	Contrast MRI (in two), arthroscopy	Arthroscopic resection	Complete recovery
Tateishi <i>et al</i> ^[11] , 2005	One	Snapping plica	MRI, arthrogram	Open resection	Complete recovery

¹Special MRI was high-resolution magnetic resonance imaging of the elbow using microscopy coils. MRI: Magnetic resonance imaging; MR: Magnetic resonance.

One case series reported snapping of the medial antebrachial cutaneous nerve^[35] that is medial to the medial humeral condyle (and not posterior as the ulnar nerve). There were no neurological symptoms reported from this nerve, and snapping was observed intra-operatively. In three of the four patients there was also snapping of triceps and the ulnar nerve. In retrospective review of the preoperative ultrasonography investigations, snapping of the medial antebrachial cutaneous nerve could be identified. In three patients the medial antebrachial cutaneous nerve was transpositioned and in one it was decompressed, while triceps- and ulnar nerve dislocation was also treated in three.

The most common symptoms were pain and painful snapping, and neurological symptoms from the ulnar nerve were rarely reported.

DISCUSSION

It is useful to divide snapping of the elbow joint into lateral and medial snapping, as these are caused by different pathological conditions and can easily be distinguished clinically (Figure 2).

Generally, intra-articular snapping pathology cannot be demonstrated by conventional MRI, and MR-arthrography, high resolution MRI or radiographic arthrography is necessary^[1,6,9,13].

In a series with 14 patients treated for lateral, intra-articular plicas snapping over the radial head by arthroscopic resection^[2], ten patients were completely relieved of their symptoms, two still experienced mild pain and one was asymptomatic for 4 years, but then

experienced recurrence of symptoms. In one patient treatment failed, but he was subsequently diagnosed with postero-lateral instability as the possible cause of the snapping phenomenon. In the largest series reported^[9] with 64 patients, there was a significant and clinically relevant increase in Oxford Elbow Score three and 22 mo after operation. Therefore, surgical treatment of the lateral SE is successful in the majority of cases, and it should preferably be performed arthroscopically, as this permits optimal visualization of the joint and minimize morbidity. Non-surgical treatment has not been described for lateral SE.

Dynamic ultrasonography is the best to visualize medial snapping, but it can be difficult to identify which anatomic structure that is snapping (as described in case 2)^[20,25,27]. Fabrizio *et al*^[36] reported a variation of the triceps brachii with a thin fourth muscular head inserting on the medial part of the olecranon as a cause of medial snapping. An accessory snapping triceps tendon can clinically be confused with snapping of the ulnar nerve, as the two structures are closely located at the medial epicondyle. Watts described this diagnostic pitfall in a report of three cases^[34], of which two primary had transposition of the ulnar nerve and one a fixation of the nerve in the ulnar sulcus. In all three cases snapping persisted, and the cause was identified as a discrete accessory tendon originating from the triceps. The authors concluded from their series that a subluxing ulnar nerve does not snap, and that medial snapping is always caused by anterior sliding of a strip of the triceps tendon during elbow flexion.

Snapping of a medial part of the triceps muscle is recognized as a reason for continuous snapping after

Table 2 Reports of extra-articular snapping elbow

Report and year	No. of cases	Snapping pathology	Diagnostic procedure	Treatment	Results
Anand <i>et al</i> ^[16] , 2012	One	Snapping ulnar nerve	Open surgery	Transposition of nerve	Complete recovery, returned to elite sport
Cesmebasi <i>et al</i> ^[35] , 2015	Four	Snapping medial antebrachial cutaneous nerve (4), triceps (3) and ulnar nerve (3)	Open surgery/sonography	Open decompression, stabilization or transposition of nerves, resection of tendon	Improvement (mild persistent symptoms to complete recovery)
Chuang <i>et al</i> ^[17] , 2016	One	Snapping triceps and subluxing ulnar nerve	Dynamic sonography	NSAIDs and reduction in repetitive elbow flexion activities at work	Partial improvement
Dreyfuss <i>et al</i> ^[18] , 1978	Two	Snapping triceps	Open reinsertion of the snapping part of the triceps	Reinsertion of the snapping tendon	1 complete recovery, 1 partial
Haws <i>et al</i> ^[30] , 1995	One	Snapping triceps	Open exploration	Open resection of tendon	Complete recovery
Hayashi <i>et al</i> ^[19] , 1984	One	Snapping triceps	Physical examination, radiographs, open surgery	Medial epicondylectomy and division of tendon	Complete recovery
Jacobson <i>et al</i> ^[20] , 2001	Three	Snapping ulnar nerve, snapping triceps	Dynamic sonography	Open "surgical treatment for each abnormality"	Not reported
Lasecki <i>et al</i> ^[21] , 2014	One	Snapping triceps and ulnar nerve	MRI and dynamic sonography	Not reported	Not reported
Minami <i>et al</i> ^[31] , 1999	One	Snapping triceps	Not reported	Open tendon resection	Complete recovery
Reis <i>et al</i> ^[22] , 1980	One	Snapping triceps	Open surgery	Open tendon resection	Complete recovery
Rolfesen ^[23] , 1970	One	Snapping triceps	Open surgery	Open tendon resection	Complete recovery
Spinner <i>et al</i> ^[32] , 2001	One	Snapping triceps	Physical examination	Open tendon Excision	Complete recovery
Spinner <i>et al</i> ^[24] , 1999	Two	Snapping triceps	Physical examination, MRI	Reduction in weight lifting	Symptoms only appeared during weight lifting
Spinner <i>et al</i> ^[33] , 1999	One	Snapping triceps lateral	Palpation and MRI	Open tendon resection	Complete recovery
Spinner <i>et al</i> ^[25] , 2000	Fifteen	Snapping triceps after operation for snapping ulnar nerve	Physical examination, MRI	Lateral transposition or excision of tendon (nine patients), six refused surgery	Complete relief of snapping in surgically treated patients. No relief in non-operated
Watts <i>et al</i> ^[34] , 2009	Three	Snapping triceps	Open exploration	Excision (two), tendon division (one)	Complete recovery
Xarchas <i>et al</i> ^[26] , 2007	Three	Snapping ulnar nerve	Palpation	Anterior nerve transposition (two), NSAIDs and change of job as waitress (one)	Complete recovery after surgery. Not reported for non-surgery

NSAIDs: Non-steroidal anti-inflammatory drugs; MRI: Magnetic resonance imaging.

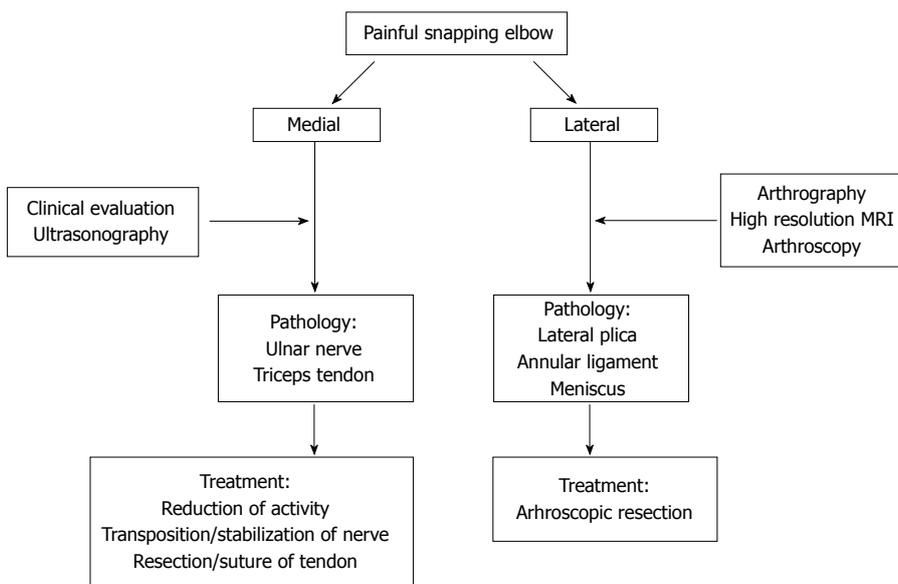


Figure 2 Snapping of the elbow joint into lateral and medial snapping caused by different pathological conditions and can easily be distinguished clinically.

ulnar nerve transposition^[22-23,25,30-32,34]. It is unclear in these cases if the snapping triceps was unrecognised during ulnar nerve surgery or if it was a complication to dissection of the tendon during release of the ulnar nerve. However, we and others present cases of medial snapping, treated successfully by ulnar nerve surgery^[20,26]. Therefore, the triceps should always be inspected in flexion and extension of the elbow during surgery for ulnar nerve snapping.

It is unknown to which extent the medial antebrachial cutaneous nerve is involved in medial snapping, as there is only one case report of this nerve as snapping structure^[35]. It is challenging to decide by ultrasonography which anatomical structure that causes medial snapping^[35] and the final diagnosis is established during operation. All snapping pathologies should be addressed.

Medial snapping in persons with repeated or loaded activities involving elbow flexion and extension during work (e.g., a waitress, a postman) or sports (weightlifting, body building) can be treated by reduction of these activities^[17,24,26]. In other cases, surgical intervention should preferably be early, as intra-articular pathologies can lead to damage of the cartilage, and snapping of the ulnar nerve can lead to neuropathy^[12,25].

Intra-articular loose bodies and postero-lateral elbow instability can cause locking, which may be interpreted as lateral snapping^[2,12]. Radiographs and CT are useful to identify intra-articular loose bodies, while postero-lateral instability is a clinical diagnosis. Also, lateral snapping is sometimes treated as epicondylitis, which is not a snapping condition.

In conclusion, SE is clinically divided into intra-articular (lateral) and extra-articular (medial) cases, based on the location of snapping. Intra-articular pathology is best visualized with high-resolution MRI, MR arthrography or radiographic arthrography. Arthroscopic or open resection of the pathological tissue is successful in most cases. Extra-articular pathology is best diagnosed by dynamic ultrasonography and during surgery. Solitary snapping of the ulnar nerve is extremely rare, and a triceps associated snapping tendon should always be suspected. Treatment is by open surgery.

ARTICLE HIGHLIGHTS

Research background

Patients with snapping elbow (SE) are seen by orthopaedic surgeons, rheumatologists and physical therapists, but the diagnosis is rare.

Research motivation

Most health care workers have no clinical experience with SE, as it is a rare condition. Therefore, there is a risk of misdiagnosis and delay of relevant treatment. Snapping can be visible, audible and palpable, but usual diagnostic measures can fail to demonstrate pathology.

Research objectives

From a literature search combined with our own clinical experience we wanted to analyse what is known about SE, its diagnosis and its treatment. The main purpose was to present a guideline to identify the patho-anatomical cause of

SE, its general binary categorization and the best treatment of each pathology.

Research methods

Literature was searched in PubMed and Scopus and key points in diagnosis and treatment were identified. Two typical cases are described.

Research results

Our review indicates that SE should be clinically divided into lateral and medial, and that diagnosis and treatment is a logic consequence of this. Lateral, intra-articular pathology is best diagnosed with high-resolution MRI, MR-arthrography or radiographic arthrography. Surgical intervention is the treatment of choice and successful in the majority of the cases. Medial, extra-articular pathology is best diagnosed by dynamic ultrasonography and during surgery. It is most commonly caused by subluxation of a medial part of the triceps tendon or the ulnar nerve. Treatment is by open surgery, except in patients with repeated, loaded activities during flexion and extension (at work or during sports), in which case symptoms may resolve by reduction of this activity.

Research conclusions

This guideline suggests a standardized approach to diagnosis and treatment of patients with SE. As early surgical intervention is recommended because the snapping can damage nerve (medial) or cartilage (lateral), this guideline is a tool for better patient care.

Research perspectives

There are no randomized studies on treatment of SE, but the largest series of 64 cases is on lateral SE, meaning that randomized controlled studies could be performed regarding treatment of this pathology. The other pathologies are too rare. There are probably many undiagnosed cases, and studies on incidence would describe the magnitude of this health problem.

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