

Diploma thesis

**Influence of BMI on clinical outcome after
implantation of ACS[®] knee arthroplasty with metal-
backed patella resurfacing**

submitted by

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Graz, 30.06.2023

Declaration of Academic Integrity

I hereby confirm that the present diploma thesis is the result of my own independent scholarly work. I also confirm that in all cases, where material from the work of others (in books, articles, essays, dissertations, and on the internet) is acknowledged, quotations and paraphrases are clearly indicated. No material other than that cited in the reference list has been used. I have read and understood the Medical University's regulations and procedures concerning plagiarism.

Graz, 30.06.2023

Stefan Perner m.p.

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Glossary and Abbreviations

ACL	anterior cruciate ligament
ACS®	Advanced Coated System®
AP	anteroposterior
BMI	body mass index
CI	confidence interval
Co	cobalt
CoCrMo	cobalt-chromium-molybdenum
Cr	chromium
CR	cruciate retaining
FB	fixed bearing
IGF	insulin-like growth factor
KSS	Knee Society Score
KTEP	Knie-Totalendoprothese
LCL	lateral collateral ligament
lig.	ligamentum
MB	mobile bearing
MCL	medial collateral ligament
MCS-12	SF-12 Mental Component Summary
NSAIDs	nonsteroidal anti-inflammatory drugs
OA	osteoarthritis
OR	odds ratio
PCL	posterior cruciate ligament
PCS-12	SF-12 Physical Component Summary
PS	posterior stabilizing
PVD	physical vapor deposition

ROM	range of motion
SD	standard deviation
SF-12	12-item Short Form Survey
TiN	titanium nitride
TKA	total knee arthroplasty
VAS	Visual Analogue Scale
WHO	World Health Organization
WOMAC	Western Ontario and McMaster Universities Osteoarthritis Index

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Zusammenfassung in Deutsch

Hintergrund

Aufgrund der zunehmenden Adipositas steigt die Inzidenz der operationspflichtigen Gonarthrosen weltweit an. In der Literatur herrscht eine Kontroverse in Bezug auf das klinische Ergebnis von KTEPs bei adipösen Patientinnen und Patienten. Im Zuge dieser retrospektiven Studie soll das klinische Ergebnis bei Patientinnen und Patienten nach Erhalt einer ACS® KTEP in Bezug auf den Body-Mass-Index (BMI) bewertet werden.

Methoden

Es wurde ein bereits bestehender Datensatz von Patientinnen und Patienten verwendet, die im Landeskrankenhaus Bad Radkersburg eine primäre KTEP mit einem ACS® Implantat mit metal-backed Patella-Resurfacing erhalten haben.

Zur Bewertung des klinischen Ergebnisses nach Erhalt der KTEP wurden die folgenden Scores verwendet: Tegner, WOMAC, KSS-Knie, KSS-Funktion, SF-12/PCS, SF-12/MCS, ROM, VAS. Außerdem wurde berücksichtigt, ob eine Revision durchgeführt werden musste. Das mittlere Follow-up betrug 10,1 Jahre. Die Patientinnen und Patienten wurden in 3 Gruppen eingeteilt: nicht adipös (BMI unter 30 kg/m²), adipös (BMI von 30 bis 39,99 kg/m²) sowie morbid adipös (BMI über 40 kg/m²).

Ergebnisse

Von den 388 Implantaten bei 356 eingeschlossenen Patienten waren 49,7 % (n = 193) nicht adipös, 44,9 % (n = 174) adipös und 5,4 % (n = 21) morbid adipös. Sowohl bei den Scores als auch bei der Revisionsrate zeigte sich kein signifikanter Unterschied zwischen den Gruppen. Im Vergleich zu ähnlichen Kollektiven in der Literatur zeigte sich eine vergleichbare Revisionsrate nach 10 Jahren.

Schlussfolgerung

Somit konnte in dieser Studie anhand verschiedener etablierter Knie-Scores in Abhängigkeit vom BMI gezeigt werden, dass es keinen signifikanten Unterschied hinsichtlich der Kniefunktion, der Schmerzen und der Revisionsrate nach Erhalt einer primären KTEP mit einem ACS® Implantat mit metal-backed Patella-Resurfacing gibt. Aus diesem Grund kann, aus Sicht dieser Studie, die Einführung einer BMI-Grenze für die KTEP-Implantation nicht unterstützt werden. Es sollte jedoch beachtet werden, dass trotz der hohen Zahl der eingeschlossenen Patientinnen und Patienten die Größe der Gruppen relativ heterogen ist mit nur wenigen Fallbeispielen in der Gruppe der morbid adipösen Patientinnen und Patienten. Dadurch könnten die Studienergebnisse in der Gruppe der morbid adipösen Patientinnen und Patienten aufgrund der geringeren Zahlen weniger valide sein.

Abstract in English

Background

The rising number of obese patients is associated with an increase in patients receiving total knee arthroplasty (TKA). However, the debate about the clinical outcome of TKA in obese patients remains controversial since studies conducted in this field have arrived at different conclusions. This retrospective study aims to evaluate the clinical outcome of TKA patients in relation to their BMI.

Methods

A pre-existing dataset of patients who received primary TKA with an ACS® implant with metal-backed patella resurfacing at Bad Radkersburg State Hospital was used.

In order to evaluate the clinical outcome following TKA, a variety of scores (Tegner, WOMAC, KSS-knee, KSS-function, PCS-12, MCS-12, ROM, VAS) was applied. Additionally, potential revisions were documented. The mean follow-up was 10.1 years. We divided patients into 3 groups (non-obese [BMI less than 30 kg/m²], obese [BMI of 30 to 39.9 kg/m²], and morbidly obese [BMI higher than 40 kg/m²]).

Results

49.7 % (n = 193) of 388 implants in 356 included patients were non-obese, whereas 44.9 % (n = 174) were obese and 5.4 % (n = 21) were morbidly obese. There was no significant difference between the groups regarding both the scores and the revision rate.

Conclusion

In this study, no significant difference regarding knee function, pain, and revision rate after primary TKA with an ACS[®] implant with metal-backed patella resurfacing was shown by means of various well-established knee scores in relation to the BMI. Therefore, a BMI limit for performing TKA cannot be supported, as it has no impact on the result. However, it should be noted that despite the high number of (included) patients, the group sizes are heterogeneous with a rather low number of patients in the morbidly obese group, making the comparison to the morbidly obese group potentially less valid.

1 INTRODUCTION

1.1 The Knee Joint

1.1.1 Basics

The knee joint (articulatio genus), a modified hinge joint, is considered the largest joint in the human body (1). It consists of two partial joints: the femorotibial joint and the femoropatellar joint, both surrounded by a common capsule (2). Between the femoral condyles and the tibia, there are two fibrocartilaginous menisci, one on each side, which adjust their shape during joint movements, compensating incongruences of the articular surfaces (1,3). Flexion is composed of rolling and sliding movements and rotation is possible in the flexed state (1). In order to provide more stability, the knee joint is reinforced by ligaments and the surrounding muscles (4).

Due to the anatomy of the knee joint, there are several potential reasons for the frequency of knee injuries and degenerative changes. It connects the longest lever arms of the skeleton and thereby has an extremely low soft tissue coverage. Furthermore, the articulating joint bodies are not very congruent, which explains the great susceptibility of the knee joint to chronic degenerative diseases. (5)

1.1.2 Joint Bodies

Femur, tibia, and patella form the knee joint composed of two partial joints: the femoropatellar joint, which includes the articular surface of the patella and the facies patellaris of the femur and the femorotibial joint, which consists of the medial and lateral condyles of the femur and tibia. (5)

1.1.2.1 Distal End of the Femur

The distal end of the femur, the largest tubular bone of the body, is formed by the condyles (1). The cylindrical femoral condyles are spirally curved, with the curvature increasing from front to back resulting in less contact area between the femoral and tibial condyles when the knee joint is flexed. This leads to a reduction in the congruence between the joint bodies during flexion. (5)

The intercondylar fossa, which contains the cruciate ligaments, separates the femoral condyles dorsally. Cranial to the intercondylar fossa, the surface of the facies poplitea is located. The shallow gliding groove for the patella (facies patellaris) lies ventrally between the femoral condyles. Usually, the lateral femoral condyle protrudes further than the medial one resulting in a slightly larger gliding area. (5)

Dorsally and caudally, the cartilaginous covering of the femoral condyles is limited to the contact surfaces with the tibial condyles. Ventrally, the articular cartilages of the condyles are connected, forming the gliding surface for the patella. Laterally, approximately in the middle of the condyles, there are bone protuberances (medial and lateral epicondyle) that serve as attachment points for muscles and ligaments. (5)

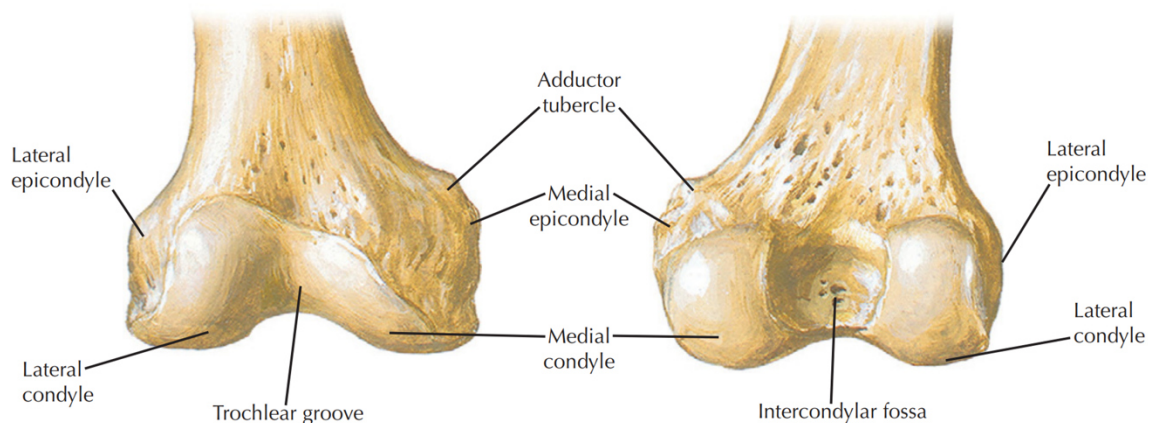


Figure 1. Distal end of right femur, anterior (left) and posterior (right) view; Source: (6)

1.1.2.2 Patella

The patella is the largest sesamoid bone of the body, incorporated into the tendon of the m. quadriceps femoris. It is triangle-shaped. The tip points distal, called the apex, and the proximal rounded end is called the base. Posteriorly, two cartilaginous facets, which articulate with the facies patellaris between the femoral condyles, are located. (5)

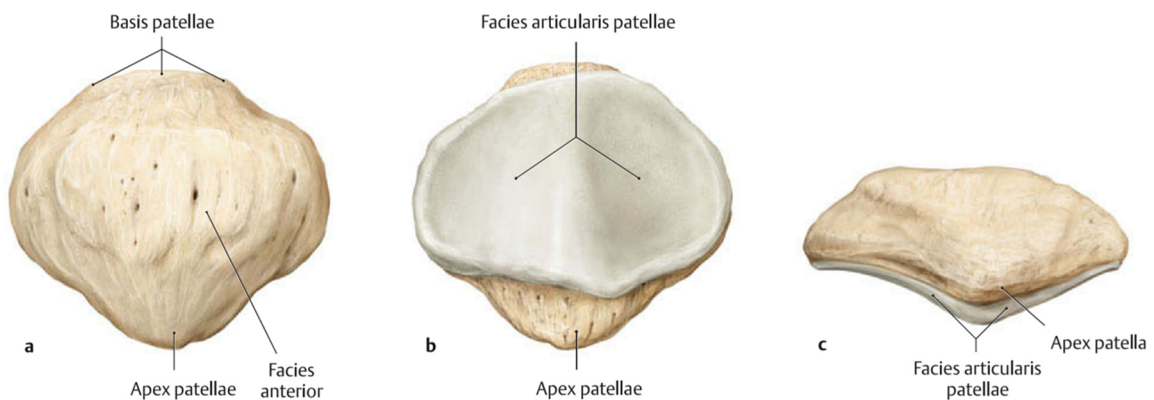


Figure 2. Right patellar, ventral (a), dorsal (b), and distal (c) view; Source: (5)

1.1.2.3 Proximal End of the Tibia

The articular surfaces of the tibial plateau are separated and adapt to the femoral condyles and the underside of the meniscus, resulting in a smaller rounded convex surface laterally and a larger oval concave surface medially. (7)

In the middle of the tibial plateau, the eminentia intercondylaris is located, which separates the posterior and anterior intercondylar areas. The cruciate ligaments and menisci are attached in this area between the articular surfaces. (5)

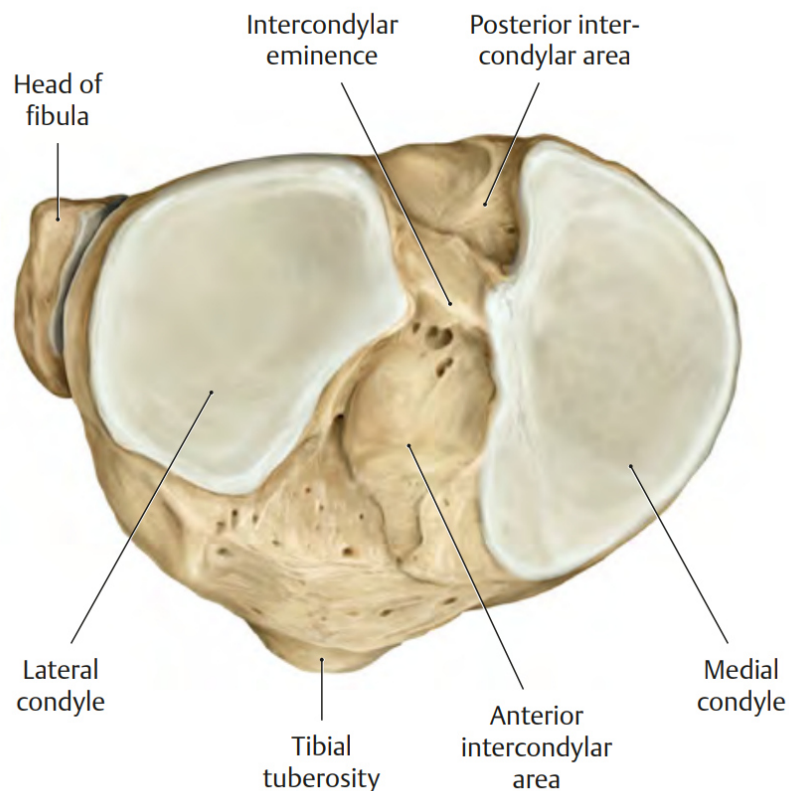


Figure 3. Right tibia, proximal view; Source: (8)

1.1.2.4 Articular Cartilage

The articular surfaces of the bone are covered with articular (hyaline) cartilage, which allows sliding between them due to its extremely low coefficient of friction. In adults, healthy articular cartilage has a white, slightly translucent, smooth, and shiny surface. It is not innervated and vascularized, which is why nutrition is provided by diffusion from the synovial fluid and the subchondral bone. Due to its flexibility and deformability, it acts as a shock absorber. (7)

With an average of 2-3 mm at the femoral and tibial condyles and 3 mm at the back of the patella, the knee joint has the thickest articular cartilage of all the joints. Extreme values of up to 7 mm can be measured, resulting in a wide radiological joint gap. This extremely thick articular cartilage somewhat mitigates the incongruity of the articular surfaces by deforming. (5)

1.1.3 Menisci

The two crescent-shaped menisci (one lateral and one medial) are located between the articular surfaces of the femoral condyles and the tibial plateau and serve to compensate for the lack of congruence between them. As a result, the pressure of the femoral condyles is distributed more evenly over the tibial plateau. In addition, the joint is stabilized and a shock-absorbing effect is achieved. (5,9)

The menisci have a wedge-shaped cross-section and consist of fibrous cartilage that is not covered by the synovial membrane. Peripherally, they are fused with the joint capsule. Their ends (anterior and posterior horn) are attached to the bone of the intercondylar area. In addition, the anterior horns of both menisci are connected by the transverse ligament. (5)

By deforming and shifting under axial pressure, the menisci adapt to different loading situations. During extension, both menisci glide ventrally, and in the course of flexion, they glide dorsally, resulting in a glide distance of approximately 12 mm for the lateral and approximately 6 mm for the medial meniscus. (9)

The menisci are supplied via the synovial fluid of the joint cavity and via blood vessels of the bony attachments and the joint capsule. (5)

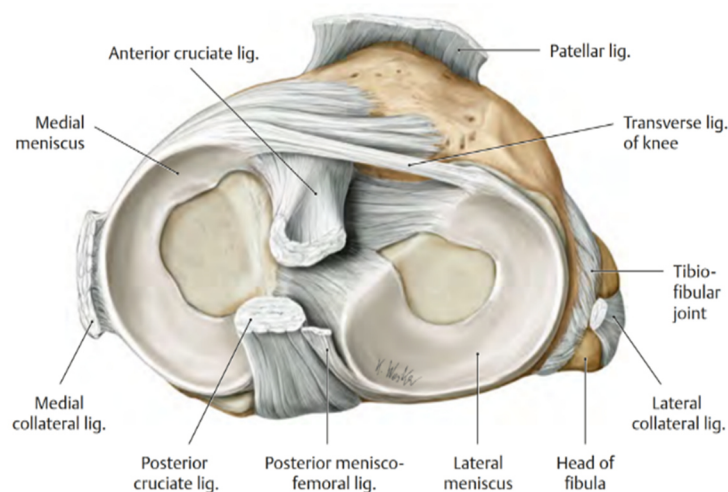


Figure 4. Right tibial plateau with menisci and ligaments, proximal view; Source: (8)

1.1.4 Joint Capsule

Of all the joints in the body, the knee has the most spacious and complicated joint cavity formed by the joint capsule, consisting of the synovial and fibrous membranes which are separated from each other by grease deposits on the front- and backside. (1,5)

The synovial membrane is attached to the edges of the articular surfaces and the superior and inferior outer edges of the menisci. It forms two main pouches, the subpopliteal recess, and the suprapatellar bursa, which allow low-friction movement of the tendons. There are other bursae located around the knee, which normally do not communicate with the joint cavity. The two cruciate ligaments are excluded from the joint cavity but are enclosed by the fibrous membrane. (3)

The fibrous membrane surrounds the articular cavity and the intercondylar region and is partially formed and reinforced by the tendons of the surrounding muscles. (3)

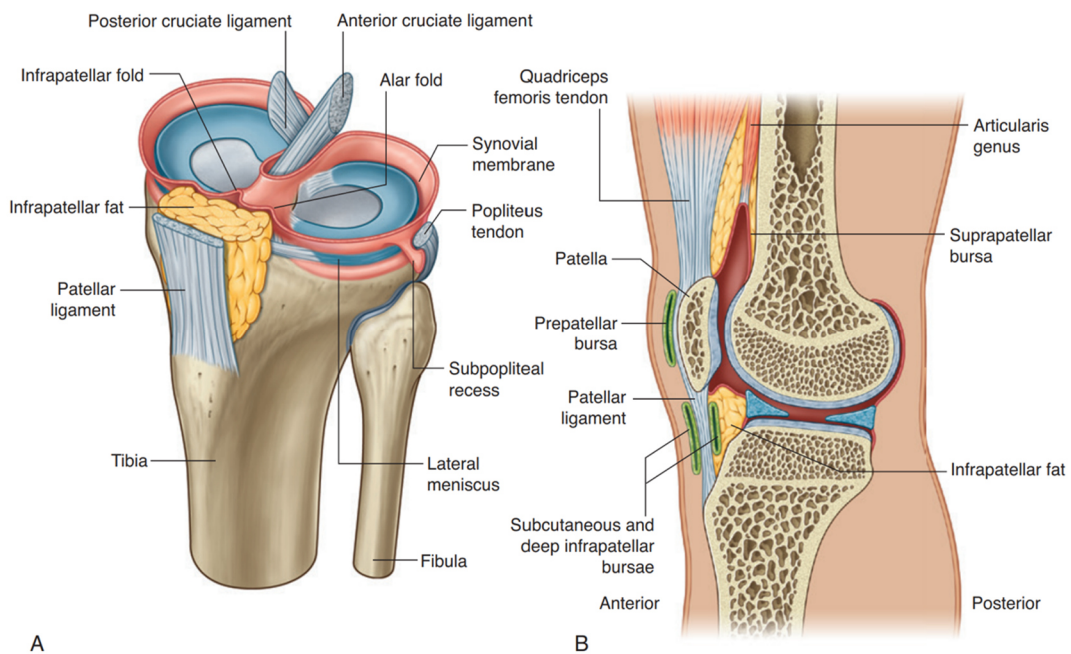


Figure 5. Joint capsule of the knee joint, superolateral view (A) and paramedial sagittal cut (B); Source: (3)

1.1.5 Ligaments

The knee has a complex ligamentous apparatus (5). The most important ligaments associated with the knee joint are the patellar ligament, the collateral ligaments, and the cruciate ligaments (3).

Ventrally, the lig. patellae, the final stretch of the quadriceps tendon between the patella and the tibial tuberosity, is located. Like the tendon of the quadriceps femoris muscle proximal to the patella and the patella itself, it is integrated into the joint capsule in order to reinforce it. The retinaculum patellae mediale and laterale consist of some tendon fibers of the m. quadriceps femoris that pass both sides of the patella and attach to the medial and lateral tibial condyle, respectively. (5)

The knee joint, like all hinge joints, is reinforced by collateral ligaments one on each side, serving as guide ligaments for flexion and extension. The medial collateral ligament (MCL) consists of two parts, reinforces the joint capsule, and prevents the medial opening of the knee joint. The lateral collateral ligament (LCL) is separated from the joint capsule and prevents lateral opening of the knee joint. (3,5)

The anterior and posterior cruciate ligaments (ACL and PCL) are located in the center of the knee intracapsular between the synovial and fibrous membrane and interconnect the adjacent ends of the femur and the tibia. Their main function is to prevent displacement of the tibia and femur in the sagittal plane. (3,5)

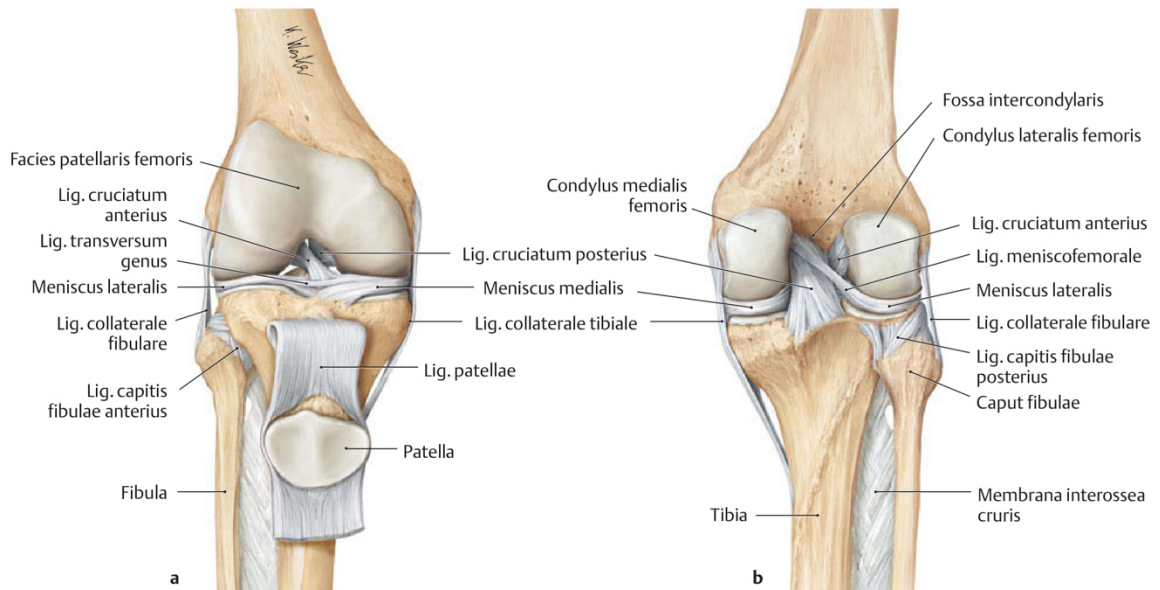


Figure 6. Ligaments of the right knee joint, ventral (a) and dorsal (b) view; Source: (5)

1.1.6 Functional Anatomy

The knee joint can be flexed and extended around an approximately transverse axis (1). In the flexed position, rotation around the lower leg axis can be performed (1). Since apart from the femoropatellar joint there is no bony guidance, it is mainly stabilized by the ligaments and surrounding muscles (4).

Flexion and extension are practically unrestricted due to the fact that the ligaments on the ventral side of the knee joint (lig. and retinacula patellae) are tendons of the passively extensible quadriceps muscle. However, the full extent cannot be actively used due to the insufficiency of the ischiocrural flexors. (5)

In extension, both collateral ligaments and the anterior portion of the anterior cruciate ligament are taut. Due to the tension of the anterior cruciate ligament and the shape of the medial condyle of the femur, the inevitable final rotation of about 5° occurs during the last 10° of extension before reaching the end position (1). This results in a locked position of the knee when standing, reducing the amount of muscle work required (3).

When the knee is flexed, both cruciate ligaments are tense, while the medial collateral tibial ligament is largely, and the lateral collateral ligament is completely relaxed. In this position rotation can be performed under the guidance of the cruciate ligaments. Less internal rotation than external rotation is possible, because during internal rotation the cruciate ligaments wrap around each other. (1)

The full range of motion (ROM) of the knee is shown in table 1.

Table 1. Range of motion of the knee joint; Source: (10)

Range of motion of the knee		
Flexion	140-160°	[active < passive]
Extension	0-10°	[active < passive]
Internal rotation	10°	[90° flexion]
External rotation	20-40°	[90° flexion]

1.2 Knee Osteoarthritis

1.2.1 Definition

Osteoarthritis (OA) is the most common disease of the musculoskeletal system and leads to loss of function and impaired quality of life. (11)

It is a degenerative joint disease characterized by degeneration of the cartilage with secondary bone lesions and shrinkage of the joint capsule due to inflammation. The spine is most commonly affected followed by the knee, hip, and shoulder joint. Morphological changes can also occur without corresponding clinical symptoms. (12)

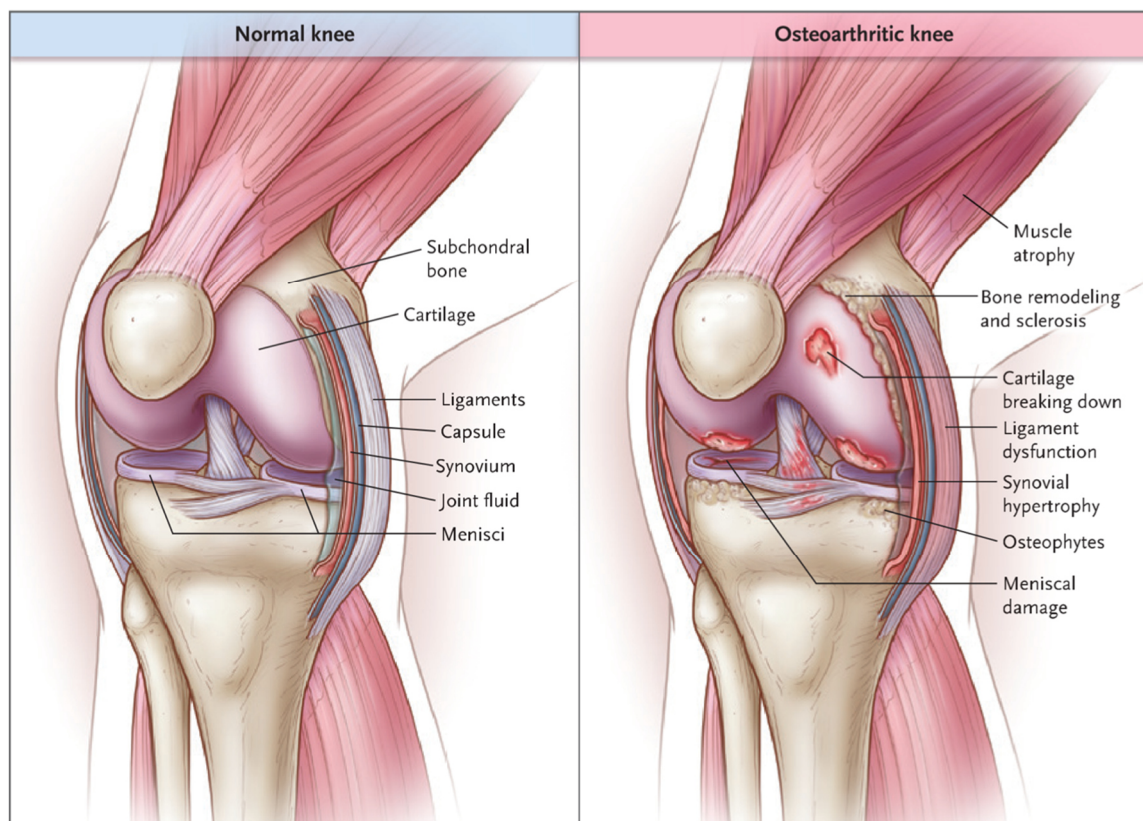


Figure 7. Difference between a normal knee (left) and a knee with osteoarthritis (right); Source: (13)

1.2.2 Epidemiology

The estimated worldwide prevalence of knee osteoarthritis in individuals aged 15 years and above is 16 % (95 % CI, 14.3-17.8 %) and 22.9 % (95 % CI, 19.8-26.1 %) in persons aged 40 years and older. In persons 20 years of age and older, the incidence is 203 per 10.000 person-years (95 % CI, 106-331). Prevalence and incidence increase with age, prevalence peaks at advanced age, incidence peaks at the age of 70 to 79 years. In terms of prevalence, women are 1.69 (95 % CI, 1.59-1.80, $p < 0.00$) times more likely to be affected than men, and in terms of incidence, 1.39 (95 % CI, 1.24-1.56, $p < 0.00$) times. (14)

1.2.3 Risk Factors

There are several systemic and local risk factors for the development of knee osteoarthritis, as shown in table 2.

Table 2. Systemic and local risk factors for knee osteoarthritis; Source: (15)

Systemic factors	Local factors
Age	Overweight and obesity
Gender	Occupation
Genetics	Trauma
Bone density	Exercise
	Mechanical environment of the joint

1.2.3.1 Systemic Factors

Gender

Whereas men seem to have a higher prevalence and incidence under the age of 50, the overall prevalence and incidence tends to be higher in women who are older than 50 years. At the age of 80 and older, the difference between male and female usually becomes less pronounced. This may be caused by estrogen withdrawal during menopause. (16)

Age

Increasing age is a significant risk factor for the occurrence of knee OA. (17)

The aging process is thought to lead to increased joint slackening, decreased joint proprioception, cartilage calcification, and decreased chondrocyte function, which leads to a tendency to osteoarthritis. (16)

Genetics

According to relevant literature, osteoarthritis has a significant polygenetic component of about 50 %, which has been demonstrated in twin studies. Genetic factors account for about 65 % of knee osteoarthritis. (18)

Bone Density

An inverse relationship between osteoarthritis and osteoporosis was observed, which may be explained by a higher load on weight-bearing articular cartilage due to increased subchondral bone density. (16)

1.2.3.2 Local Factors

Overweight and Obesity

Overweight (BMI 25-30 kg/m²) and obesity (BMI > 30 kg/m²) seem to be a significant risk factor for the development of knee OA. With an odds ratio (OR) of 2.66, obesity has a slightly greater impact on the occurrence of knee OA than in overweight patients (OR of 1.98). (17)

Studies have shown that obesity is the strongest modifiable risk factor (16).

Occupation

The risk of contracting osteoarthritis may increase in occupations with repetitive activities that overload the joints and strain the muscles that protect the joints in these joints. Knee osteoarthritis seems to be particularly common in jobs that involve kneeling, squatting, and heavy lifting. (15)

Trauma

According to relevant literature, tears of the cruciate ligament, collateral ligament, and meniscus, joint fracture and state after meniscectomy, increase the risk of knee osteoarthritis. (16)

Exercise

People who engage in more than four hours of heavy physical activity per day may have an increased risk of developing knee osteoarthritis. However, moderate levels of physical activity are unlikely to significantly increase the risk. (18)

Mechanical Environment of the Joint

There are variations in the mechanical environment of the joint that have a detrimental effect on the load distribution, such as knee laxity, proprioception, and knee alignment, which may promote the development and/or progression of knee osteoarthritis. (15)

1.2.4 Etiology

A distinction is made between primary (idiopathic) and secondary knee osteoarthritis. (19)

The cause of primary knee osteoarthritis, which is the most common form of knee osteoarthritis (20), is unknown, however, a multifactorial event is suspected of triggering it (19).

Causes of secondary knee osteoarthritis include mechanical intraarticular and extraarticular factors, inflammatory diseases, coagulation disorders, metabolic factors, collagenoses, neurologic diseases, and drug-induced factors, as shown in table 3. (19)

Table 3. Etiology of secondary knee osteoarthritis; Source: (19)

Secondary knee osteoarthritis	
Mechanical extraarticular factors	
<ul style="list-style-type: none"> • Axis deviation • Ligamentous instability • Muscular imbalance 	<ul style="list-style-type: none"> • Bone dysplasia • Microtrauma and overuse due to job, sports and overweight
Mechanical intraarticular factors	
<ul style="list-style-type: none"> • Meniscal lesions • Total meniscectomy • Osteochondritis dissecans 	<ul style="list-style-type: none"> • Ahlbaeck's disease • Posttraumatic changes (intra-articular fractures)
Inflammatory diseases	
<ul style="list-style-type: none"> • Rheumatoid arthritis 	<ul style="list-style-type: none"> • State after septic arthritis
Coagulation disorders	
<ul style="list-style-type: none"> • Hemophilia 	
Metabolic factors	
<ul style="list-style-type: none"> • Hyperuricemia • Chondrocalcinosis 	<ul style="list-style-type: none"> • Hemochromatosis • Ochronosis
Collagenoses	
<ul style="list-style-type: none"> • Marfan syndrome 	<ul style="list-style-type: none"> • Ehlers-Danlos syndrome
Neurological diseases	
<ul style="list-style-type: none"> • Neurogenic osteoarthropathy 	
Drug-induced	
<ul style="list-style-type: none"> • After chemotherapy (osteonecrosis) 	

1.2.5 Pathophysiology

There is a dynamic balance between cartilage formation and degradation, which is regulated by anabolic and catabolic influences. Anabolic influences include insulin-like growth factors such as IGF I and II, while catabolic influences include interleukin-1, tumor necrosis factor alpha, and proteinases. The harmful influences caused by osteoarthritis can thus be eliminated or compensated for to a certain extent by stimulating the metabolic activity of the chondrocytes. However, if the damage can no longer be compensated for, the cartilage degrades, and osteoarthritis develops. The exact causes of cartilage degeneration are not yet fully understood, but mechanical and enzymatic factors are thought to impair chondrocyte function and damage the matrix. (21)

1.2.6 Diagnosis

When diagnosing osteoarthritis of the knee, anamnesis is essential. In most cases, symptoms such as pain when starting a movement, after getting up, which subsides and reappears after prolonged exertion, stiffness (usually in the morning), and swelling (usually in the evening), are not acute but develop gradually. Later, nocturnal pain, constant pain at rest and on exertion, decreased ambulation, and flexion contracture with atrophy of the vastus medialis muscle also occur. (22)

During clinical examination, attention should be paid to axial misalignments, acute inflammatory signs, and a painful joint space under pressure. In addition, the ROM should be measured to demonstrate limited flexion capacity. (22)

Conventional x-rays in two planes of the knee joint are sufficient for radiological diagnosis, showing the typical 4 signs of osteoarthritis (narrowing of the joint space, subchondral sclerosis, osteophytes, and formation of boulder cysts). (22)

According to Kellgren and Lawrence, a person with a grade ≥ 2 is considered to have radiographic disease, which is the most common radiological classification, shown in table 4 and figure 8. (11)

Table 4. Radiographic classification for OA by Kellgren and Lawrence; Source: (23)

Grade	Criteria
0	No radiographic signs of osteoarthritis
1	Minimal osteophytes (uncertain findings), regular joint space width
2	Definite osteophytes, regular joint space width
3	Osteophytes, moderate reduction of the joint space width
4	Advanced narrowing of the joint space with sclerosis of the subchondral bone

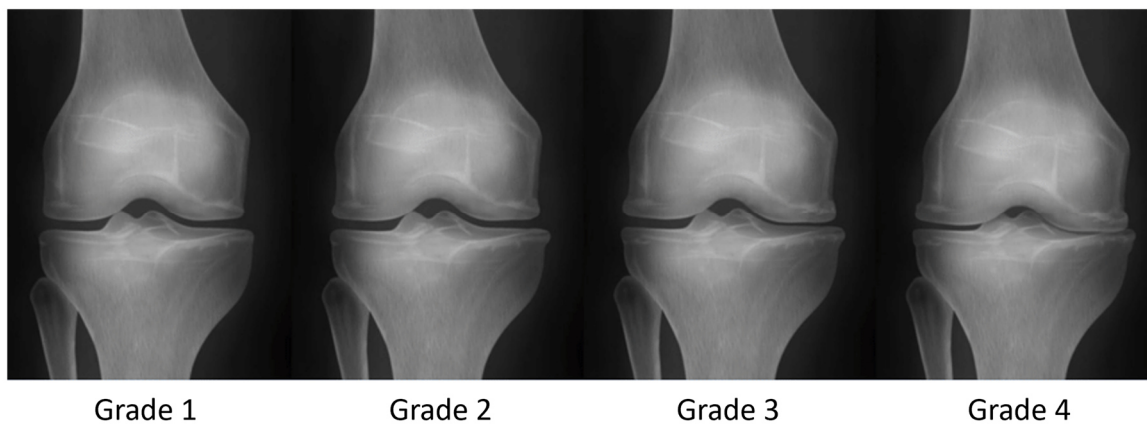


Figure 8. X-rays of knees with OA grade 1 (left) to 4 (right) according to Kellgren and Lawrence; Source: (24)

1.2.7 Treatment

In recent years, thinking has shifted from primary drug therapy to non-drug therapy in knee osteoarthritis. This is because primary drug therapy has shown limited benefit and there is evidence that non-pharmacological approaches are more likely to provide long-term symptom relief and delay or prevent functional loss. (13)

Non-pharmacological therapy focuses on exercise, diet, self-efficacy and self-management programs, and pain-coping skills training. Other non-pharmacological interventions such as massage therapy, manual therapy, and wedge insoles are not recommended in the guidelines due to insufficient benefit and data quality. (13)

As concerns pharmacological therapy, topical and oral non-steroidal anti-inflammatory drugs (NSAIDs) are the medication of choice. Topical NSAIDs should be preferred due to fewer side effects but may be less practical for a larger number of affected joints. Acetaminophen may be considered for short-term use for patients who cannot take NSAIDs. Duloxetine is effective for unselected patients and may be particularly useful for patients with widespread pain or depression. Intraarticular glucocorticoid injections are beneficial for short-term pain relief and usually last for a few weeks. They are useful as adjunctive therapy, but not recommended on a regular basis, due to greater cartilage loss. (13)

Regarding surgical treatment, arthroscopic partial meniscectomy has often been performed on patients with knee osteoarthritis with meniscal damage but is not recommended for the majority of patients. A meta-analysis showed that compared with non-surgical treatments, there are only small benefits of uncertain clinical significance and no significant improvement in knee function. (13,25)

In cases of advanced osteoarthritis, when pain cannot be controlled with other measures, knee replacement should be considered to provide significant pain relief, improved function, and high patient satisfaction. (13,26)

1.3 Total Knee Arthroplasty

1.3.1 Background

In over 90 % of cases, primary osteoarthritis is the main reason for the necessity of knee replacement. (27,28)

TKA was first developed at the Hospital for Special Surgery in the early 1970s and has evolved over the past 50 years. In recent years, TKA has undergone unprecedented development, ranging from traditional resurfacing prostheses to constrained prostheses and meniscal bearing prostheses. (29)

1.3.2 Indications

The minimum requirements that justify TKA are clinically relevant persistent symptoms that can be attributed to local pathology of the articular surfaces and knee alignment by clinical signs (30). Moreover, conservative therapy should have failed to be successful (31).

Typical indications are disability, pain and functional limitations due to osteoarthritis, rheumatoid arthritis, or any type of arthritic deformity in the knee. (32)

1.3.3 Contraindications

Absolute contraindications to TKA include active or latent (less than 1 year) knee sepsis, the presence of active infection elsewhere in the body, dysfunction of the extensor mechanism of the knee, and medically unstable patients. (33)

Relative contraindications include a neuropathic joint, poor condition of the overlying skin, lack of compliance due to a severe psychiatric disorder, alcohol or drug abuse, inadequate bone stock for reconstruction, low patient motivation or unrealistic expectations, and peripheral vascular disease. (33)

Currently, there is a controversial debate on whether obesity should be considered a (relative) contraindication, as patients with a body mass index (BMI) above 30 kg/m² seem to be at increased risk of contracting infections and medical complications compared to non-obese patients. This risk could further increase in patients with a BMI higher than 40 kg/m², as they face a statistically significant increase in superficial infections, deep infections, operative time, deep vein thrombosis, length of stay, renal insufficiency, reoperation, and wound dehiscence. (34)

1.3.4 Implants

1.3.4.1 Implant Designs

There are four main types of modern implant designs used for TKA: cruciate retaining (CR), posterior stabilizing (PS), constrained non-hinged, and constrained hinged implants. (34,35)

CR and PS TKA are widely used, however, so far there has been controversy in the relevant literature as to which method is better, as both have advantages and disadvantages and provide similar results. (36)

Cruciate Retaining (CR)

For cruciate ligament retaining TKA prosthesis, the PCL must be intact to ensure stability in flexion. Therefore, it cannot be used for patients with PCL insufficiency or patients with inflammatory arthritic diseases because of the increased risk of early PCL weakening. (35)

Some potential benefits of CR prosthetic designs include bone preservation, more natural kinematics of the knee, improved proprioception, rolling back of the femur onto the tibia during flexion, and improved stabilization of the prosthesis, where the PCL prevents anterior translation of the femur on the tibia. (37)

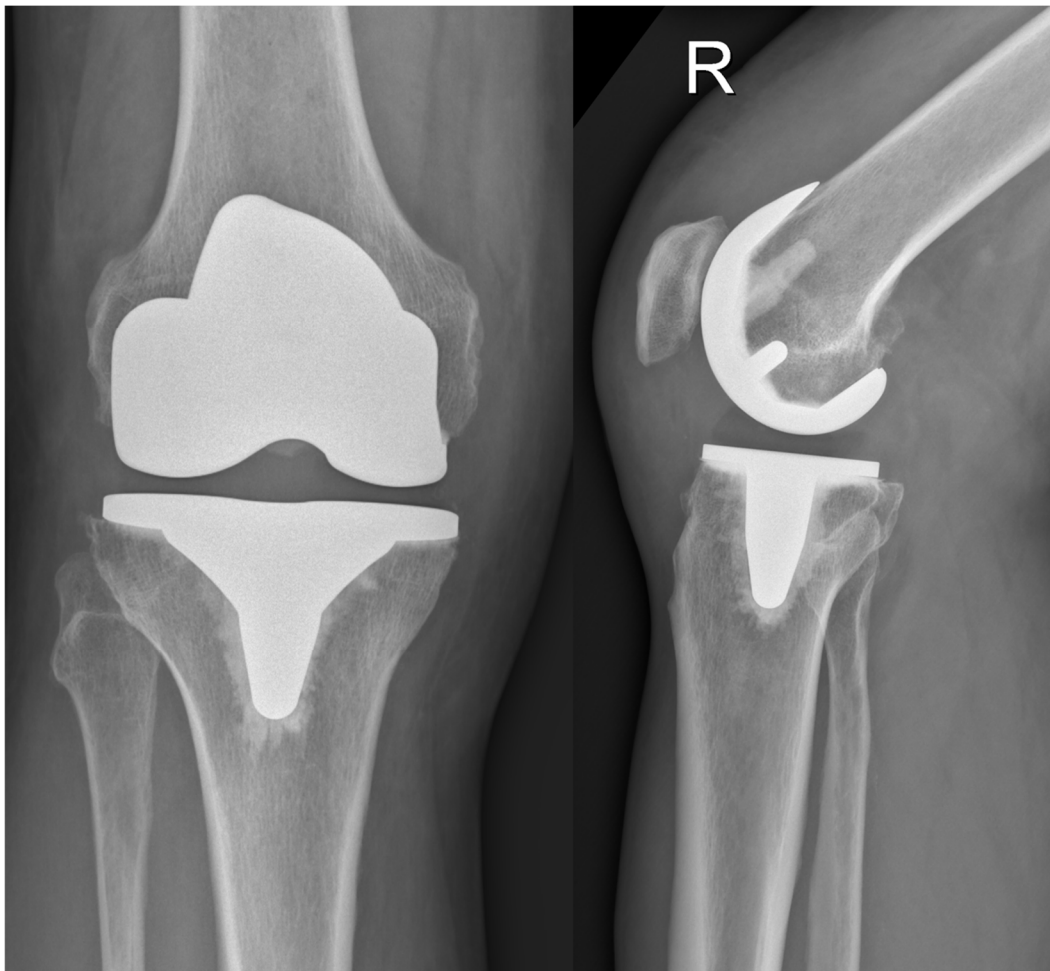


Figure 9. AP (left) and lateral (right) x-ray of the knee with implanted Attune™ CR knee system; Source: (38)

Posterior Stabilized (PS)

This system can be used for patients with absent or insufficient PCL, as it must be removed anyway. Due to a cam in the femoral component that engages the tibial polyethylene post during knee flexion, the PS TKA design is slightly more constrained than the CR design. (35)

Possible advantages of PS TKA are a less technically demanding surgery, a more stable component interface, and a greater range of motion. (37)

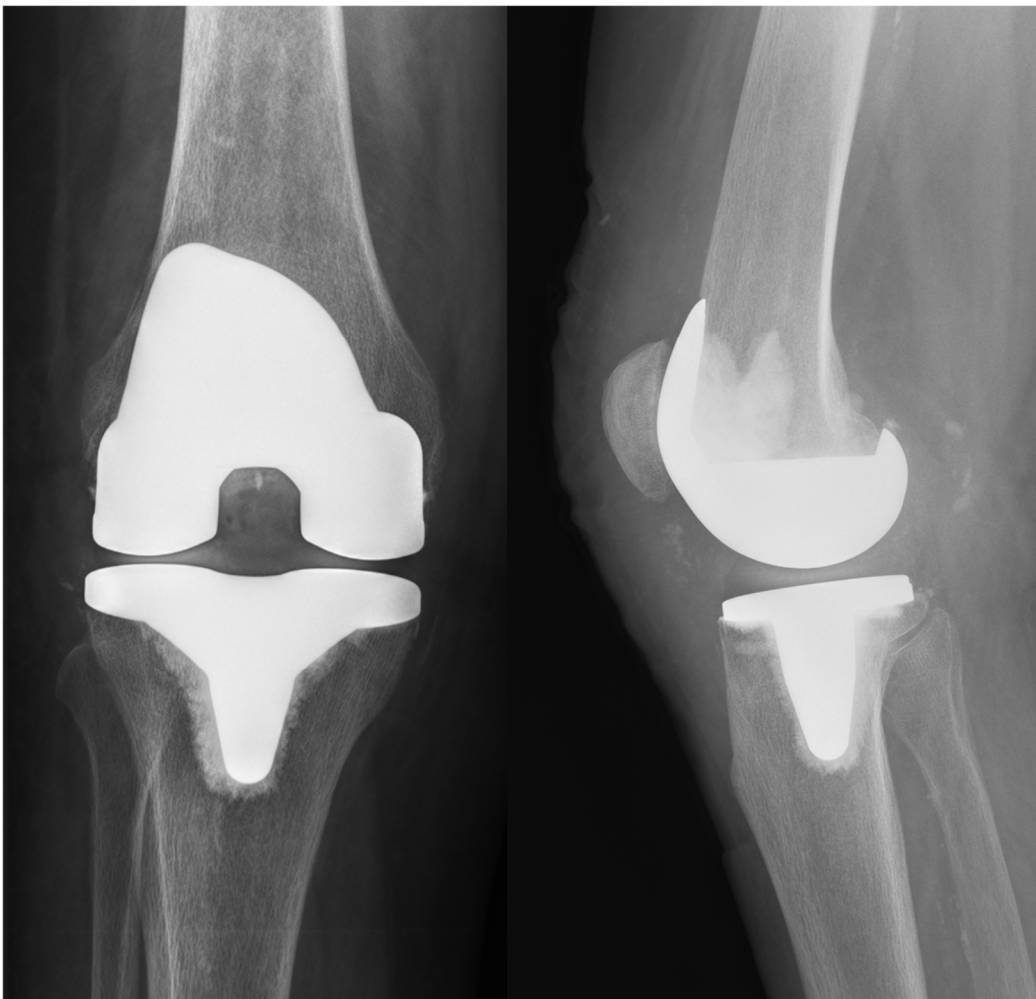


Figure 10. AP (left) and lateral (right) x-ray of the knee with implanted Attune™ PS knee system; Source: (38)

Constrained Non-hinged Design

The constrained non-hinged design is based on the posterior stabilized design and provides more stability and constraint in both the varus-valgus and internal-external rotational planes through the use of a larger tibial post and a deeper femoral box (35,39). This type of implant is used in cases of inadequate collateral ligaments, laxity of the flexion gap, and moderate bone loss in the setting of neuropathic arthropathy (35).

The disadvantage of this implant design is the increased risk of early aseptic loosening due to the increased loading between the components and the need for greater femoral bone resection to accommodate the components. (35)

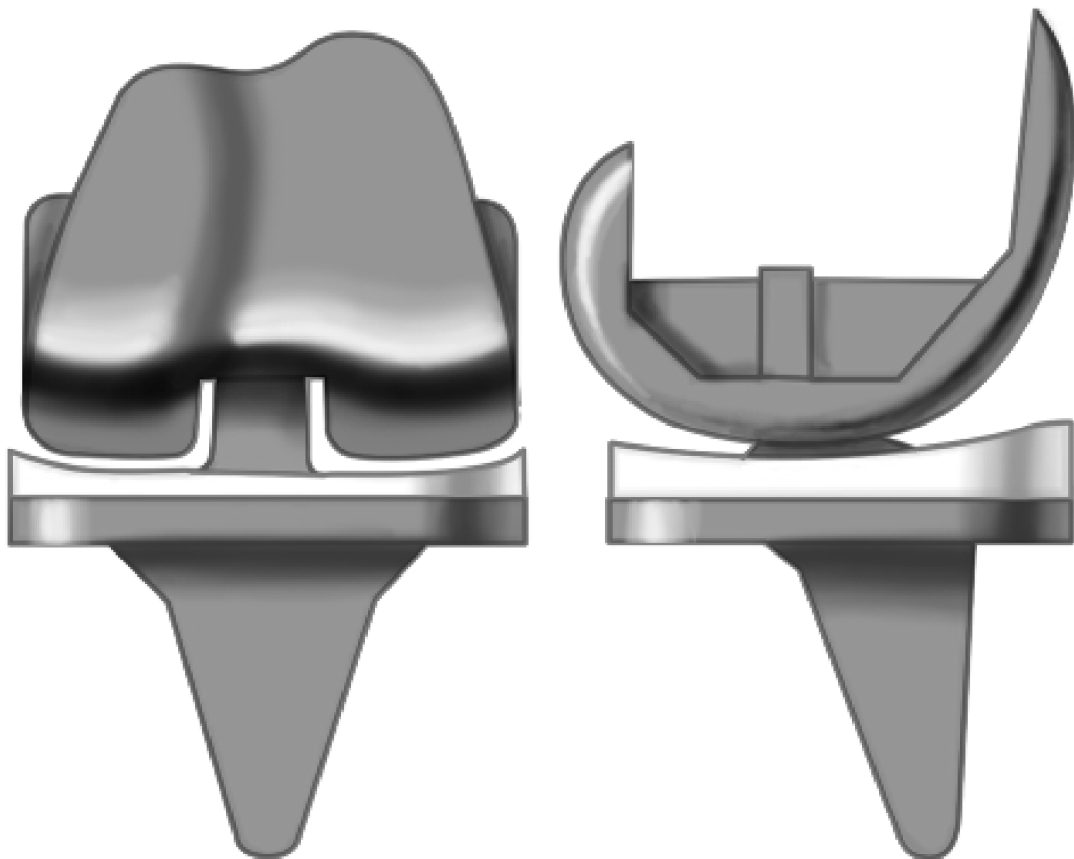


Figure 11. Constrained non-hinged implant design for TKA; Source: (40)

Constrained Hinged Design

In the constrained hinged design, the femoral and tibial components are connected by an axis, allowing the tibial bearing to rotate around the tibial platform. This should mitigate the risk of aseptic loosening at the expense of a higher degree of prosthetic constraint. (35)

This type of implant is rarely indicated in primary knee arthroplasty. It is mainly used in revision arthroplasty, where knee joints with extensive bone defects involving the collateral ligaments or insufficiency of the extension mechanism can be treated. (39)

Other indications include patients with global ligament defects, resections in the setting of tumors, and massive bone loss in the setting of a neuropathic joint. (35)

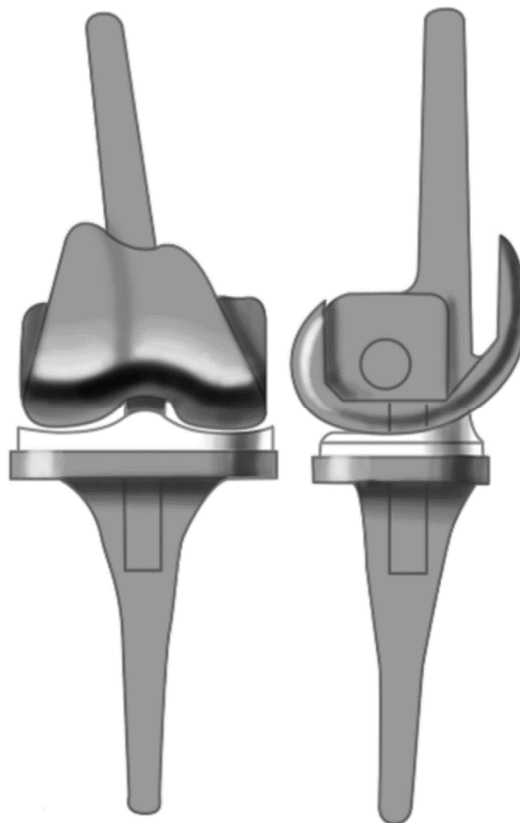


Figure 12. Constrained hinged implant design for TKA; Source: (40)

1.3.4.2 Mobile vs Fixed Bearing

TKA with a conventional fixed bearing (FB) insert has clinically proved effective over time, but due to the increasing number of younger patients with higher functional requirements, the mobile bearing (MB) inserts have been developed in order to achieve higher survival and better clinical outcome. (41)

MBs are intended to provide better clinical outcomes and a greater range of motion through a more physiological movement. Furthermore, the congruent design reduces point loading on the joint partners, which should result in less wear and longer service life. (42)

However, in contrast to the theoretical advantages of MB inserts, clinical studies have not confirmed that there is an advantage over the FB design. (41)

Disadvantages of the MB design include the risk of secondary wear between the MB and tibial plateau, the risk of inlay dislocation, and the higher implant cost. (42)

1.3.4.3 Advanced Coated System[®] TKA Implant from Implantcast

Implantcast GmbH (Buxtehude, Germany) provides a variety of TKA systems made of cobalt-chromium-molybdenum (CoCrMo) alloy for primary (fixed or mobile bearing) and revision surgery with a special 5.5 µm ceramic coating of titanium nitride (TiN) called Advanced Coated System[®] (ACS[®]) applied by physical vapor deposition (PVD). (43)

The TiN coating is used to modify the surface of cobalt-chromium (CoCr) alloy implants with the properties of TiN, but does not affect the material properties of the substrate or the biomechanical functionality (44). It offers high hardness, low coefficient of friction and resistance to adhesive wear (45). Compared to CoCrMo surfaces, it provides lower surface free energy in contact angle measurements, shows less adhesion to polyethylene in a shear test and the catalytic effect on the degeneration behavior of polyethylene is inert in in-vitro tests (46). Furthermore, the release of cobalt (Co) and chromium (Cr) ions due to corrosion is reduced by the TiN coating (47). This is important as studies have described delayed-type

hypersensitivity reactions to implant metals in form of localized or generalized eczema, wound or fracture healing disorders and implant loosening associated with allergy to Co or Cr (48).

A possible limitation is that due to the different elastic behavior of the hard TiN coating on the relatively soft substrate, the coating may flake off under high punctual pressure (thin ice effect). (49)

Regarding the clinical outcome, van Hove et al. (50) showed that TiN coated CoCrMo knee implants offer no advantage over conventional CoCrMo knee implants.



Figure 13. ACS® MB Prosthesis by Implantcast GmbH;Source: (51)

1.3.5 Surgical Technique

In primary total knee arthroplasty, clinical outcomes are influenced by surgical technique, regardless of whether a posterior cruciate ligament preserving or a posterior cruciate ligament replacing design is implanted. The goal of this procedure is to restore the normal mechanical axis with a stable and well-fixed prosthesis through bone resection and soft tissue balancing. (52)

The alignment of the femoral component should be 5-10° of valgus angulation in the coronal plane and 0-10° of flexion in the sagittal plane. For the resection of the tibia, an angle of $90 \pm 2^\circ$ to the longitudinal axis of the tibia in the coronal plane should be used. The posterior slope in the sagittal plane is determined by the prosthesis design, but the recreation of the natural posterior slope of the tibia seems preferable. (52)

There are three common approaches for primary TKA: the medial parapatellar, midvastus, and subvastus approach. The medial parapatellar approach is often used, allowing high-quality closure of the tissue at the end of the procedure by proximal dissection through a medial cuff of the quadriceps tendon. (35)

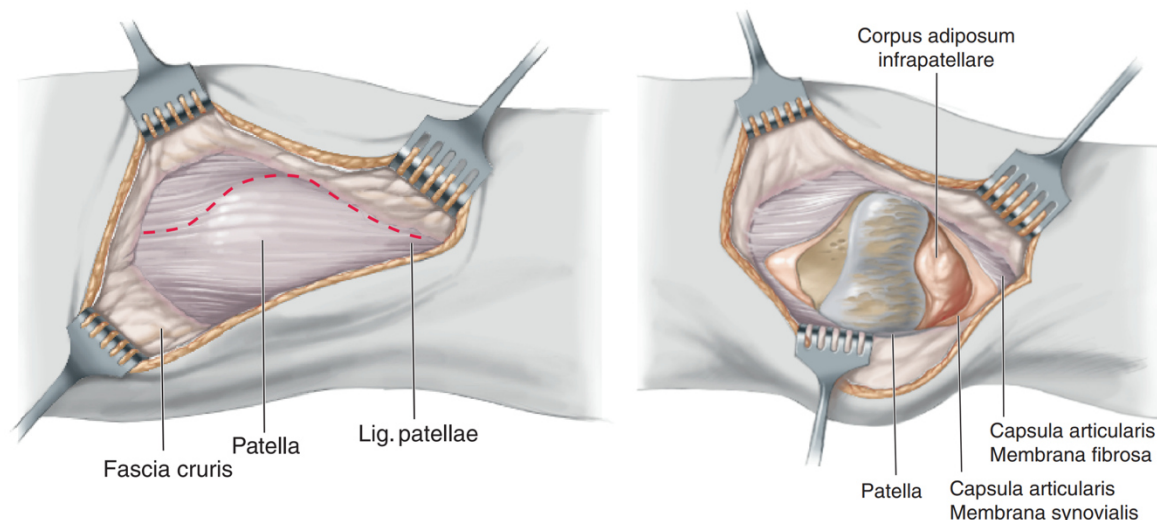


Figure 14. Parapatellar approach, medial cutting line (left) and view after parapatellar approach performed with patella folded away (right); Source: (53)

Then, surgery is continued using one of two standard techniques, the tibia-first technique, or the femur-first technique. The advantage of the femur-first technique is that the femur and tibia can be prepared independently of each other, thus ensuring maximum bone-saving. In addition, the removal of the resected tibia is significantly easier due to the already cut femur. However, ligament stabilization requires more experience because the preparation of the two bones is independent of each other and therefore the risk of possible error sources is greater. (53)

In the tibia-first technique, the tibia is resected first, although removal of the resected tibial sheath is more difficult because the femoral condyles are still present. Spacers or active balance systems can be used to help balance the knee joint. If precise balancing of the knee joint ligaments is not ensured, the femoral incision based on the resected tibia may result in a wrong resection. (53)

In the following, the femur-first technique is described, although the procedure may vary depending on the implant. (53)

After successful arthrotomy, the patella is everted and the knee is dislocated by flexing and soft tissue release. (35)

A meniscus resection can be performed as far as visible, and the anterior cruciate ligament and osteophytes can be removed. If the posterior cruciate ligament is contracted, it may be necessary to balance it. If the lift-off of the tibial plateau in flexion is not feasible, resection of the posterior cruciate ligament may be necessary, requiring the use of a posterior stabilized prosthesis. (53)

Then, the femoral medullary canal is opened above the notch using a drill, and a medullary guide rod is inserted and aligned. By using a guide, the bone cut of the femur is performed and the fit of the implant is assessed with the help of a trial implant. (53)

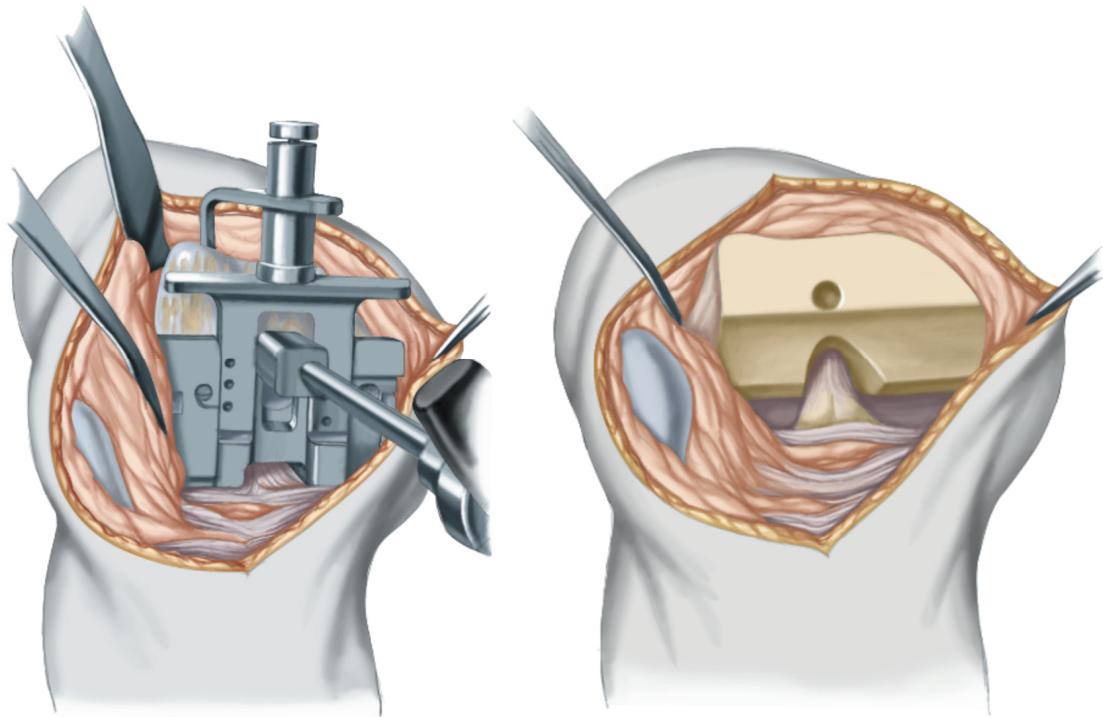


Figure 15. Femoral alignment (left) and ready cut femur (right); Source: (53)

Subsequently, the referencing of the tibial section is performed via intra- or extramedullary alignment. Intramedullary alignment is suitable when a stem is used, or the tibial anterior edge is not easily visualized. The tibial medullary canal is opened at the base of the anterior cruciate ligament and an intramedullary alignment rod is inserted. In extramedullary alignment, a rod is aligned parallel to the tibial anterior edge through the center of the ankle joint to the second ray. (53)

Now the tibial resection is determined via a feeler system and performed using a guide. The aim of the osteotomy is to create an equally wide extension and flexion gap shown in figure 16. (53)

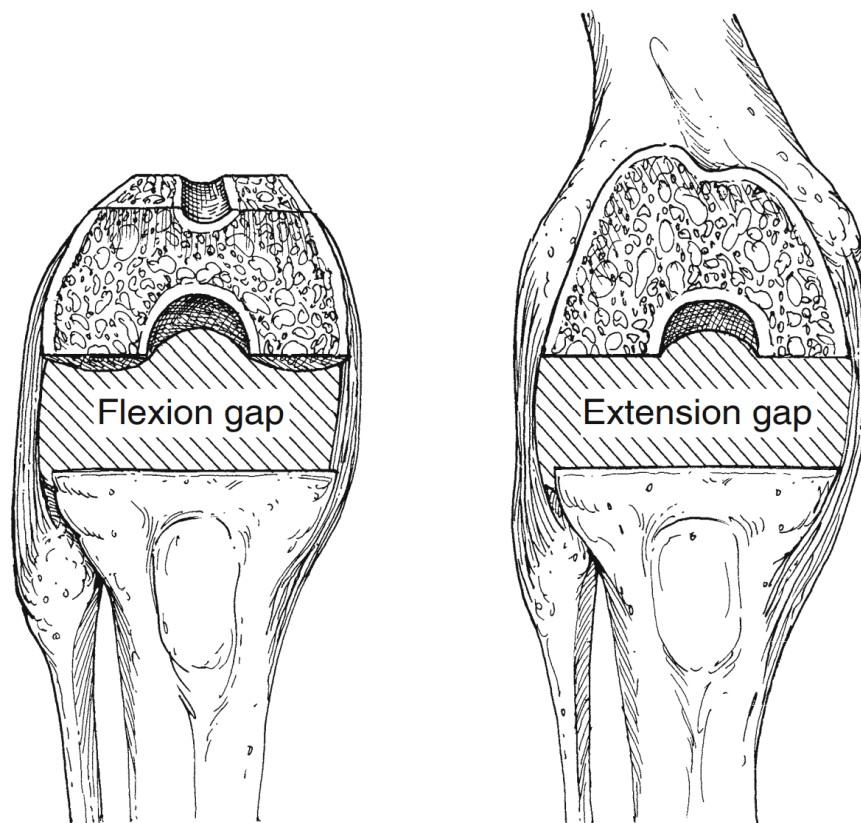


Figure 16. Equal flexion and extension gap after femoral and tibial resection; Source: (54)

After all bone incisions have been made, the remnants of the menisci can be removed and the ligament alignment can be checked and adjusted using spacers. (53)

The bone holes for the anchoring elements of the tibial plateau are then drilled using a template and a test implant can be inserted. It is important that stable ligament conditions are ensured in full extension and 90° flexion and that the full range of motion is possible. (53)

When resurfacing the patella, it is important that the new bone-patella component composite is about as thick as the original patella and medialized. The tracking of the patella can be assessed by the “no thumb” test. (35,52)

Finally, the original prosthesis can be implanted, the stability parameters rechecked and, if the desired result has been achieved, the wound closed. (35)

1.3.6 Complications

Like all arthroplasty procedures, TKA is associated with potential risks caused by the surgical and anesthetic procedures in general or by the placement of the implant itself. (27)

A distinction is made between systemic (medical) and local (orthopedic) complications. During hospitalization after primary TKA, systemic complications have an incidence of 8.31 %, while local complications occur with a frequency of 2.78 %. (55)

Among systemic complications, with 4.6 %, the cardiovascular system is most commonly affected by thrombosis, tachycardia, blood pressure fluctuations, pulmonary embolism, or myocardial events. Urinary tract infections, another common complication, account for 1.2% especially in patients with catheter insertion. (42)

A list of intraoperative and postoperative surgical complications associated with primary TKA during hospitalization is shown in table 5.

Table 5. Intraoperative and postoperative surgical complications associated with primary TKA during hospitalization; Source: (56)

Complication	Frequency (%)
Wound hematoma/postoperative bleeding	0.86
Fracture	0.15
Nerve damage	0.10
Malposition of the implant	0.03
Dislocation of the implant	0.03
Misalignment of the patella	0.02
Vascular lesion	0.02
Other	0.80

According to a systemic review by Pabinger et al. (57) the worldwide overall 10-year revision rate of TKA is 6.2 % ranging from 4.9-7.8 %.

In a worldwide analysis, Sadoghi et al. (58) revealed that aseptic loosening (29.8 %), septic loosening (14.8 %), pain without other reason (9.5 %), wear (8.3 %), instability (6.2 %), implant breakage (4.7 %), and periprosthetic fracture (3 %) are the most common causes for revision surgery in TKA.

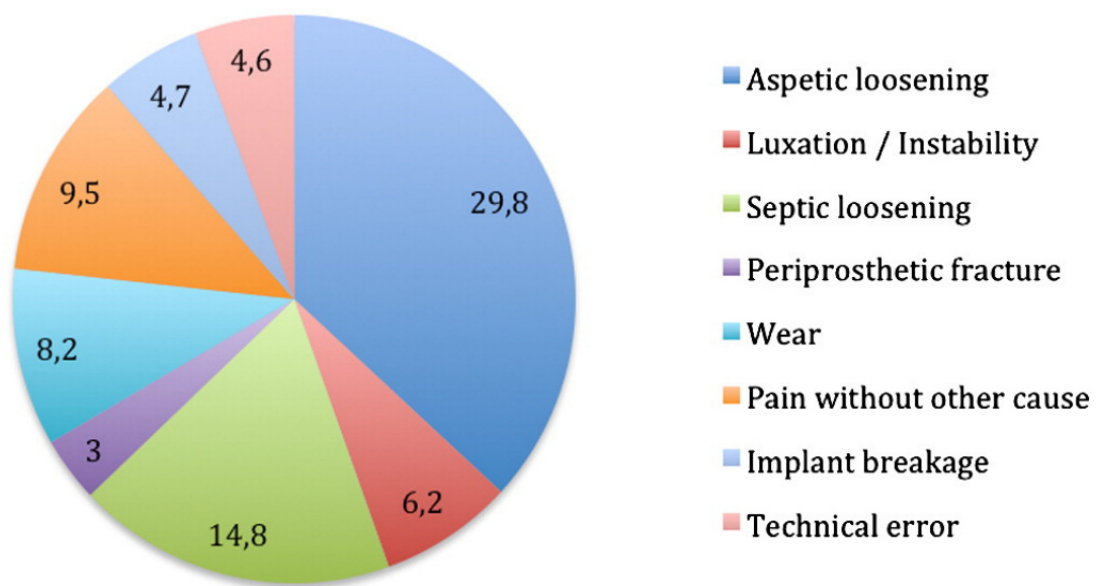


Figure 17. Causes of revision surgery after TKA in percent (%); Source: (58)

2 MATERIAL AND METHODS

2.1 Background and Study Aim

Total knee arthroplasty (TKA) is one of the most frequently performed orthopedic surgeries, usually due to knee osteoarthritis (35). Osteoarthritis is usually seen in elderly people with obesity being a significant risk factor, as shown in a large number of previous studies (15,17,59-61).

Given the increasing number of obese patients who are likely to require TKA (62), it is important to investigate whether there is a difference in clinical outcome compared with non-obese patients to ensure appropriate counseling.

The debate about the clinical outcome of TKA in obese patients has remained controversial to this day, since studies dedicated to this field have arrived at different conclusions, as shown in a review article by Rodriguez-Merchan et al. (63). This controversy leads to the discussion whether there should be a BMI cutoff for TKA, usually set at a BMI of 40 kg/m² (morbidly obese), due to a deteriorated outcome and higher risks, as recently discussed in a clinical faceoff between Thomas K. Fehring and Nicholas J. Giori (64).

This study aims to evaluate the clinical outcome of TKA in relation to BMI and to determine, whether obesity is associated with less beneficial clinical outcomes.

2.2 Study Design

In this study, a retrospective data analysis, approved by the Medical University of Graz (EK number 30-253 ex 17/18), was performed, based on a pre-existing dataset of 432 patients (484 implants) who received TKA at Bad Radkersburg State Hospital collected by the Department of Orthopedics and Trauma of the Medical University of Graz. The pre-existing dataset, which includes a variety of pre- and postoperative knee scores used in this study, was collected through clinical examination or via telephone when patients were unable to attend an on-site appointment.

By reviewing the postoperative x-rays of each implant from the hospital's database, MEDOCS, the type of patella replacement (no patella replacement, polyethylene, metal-backed) was determined and added to the dataset.

358 patients (392 implants) received primary TKA with an ACS[®] implant with metal-backed patella resurfacing between 2003 and 2011 at Bad Radkersburg State Hospital.

The variables weight and height were then added and used to calculate the BMI of patients with an ACS[®] implant with metal-backed patella resurfacing by evaluating the preoperative anesthesia evaluation.

In order to compare the scores depending on the BMI, the patients were divided into three groups: the non-obese group with a BMI under 30 kg/m², the obese group with a BMI of 30 kg/m² to 39.99 kg/m², and a morbidly obese group with a BMI higher than 40 kg/m².

Nine scores of included implants (3x Tegner preoperative, 4x Tegner postoperative, 1x KSS, 1x WOMAC) showed inconsistencies like values above the maximum number of points of the score. These scores were deleted and not included in the evaluation, which is why the number of evaluated scores does not always correspond to the number of implants. Implants missing more than five scores were excluded from the study.

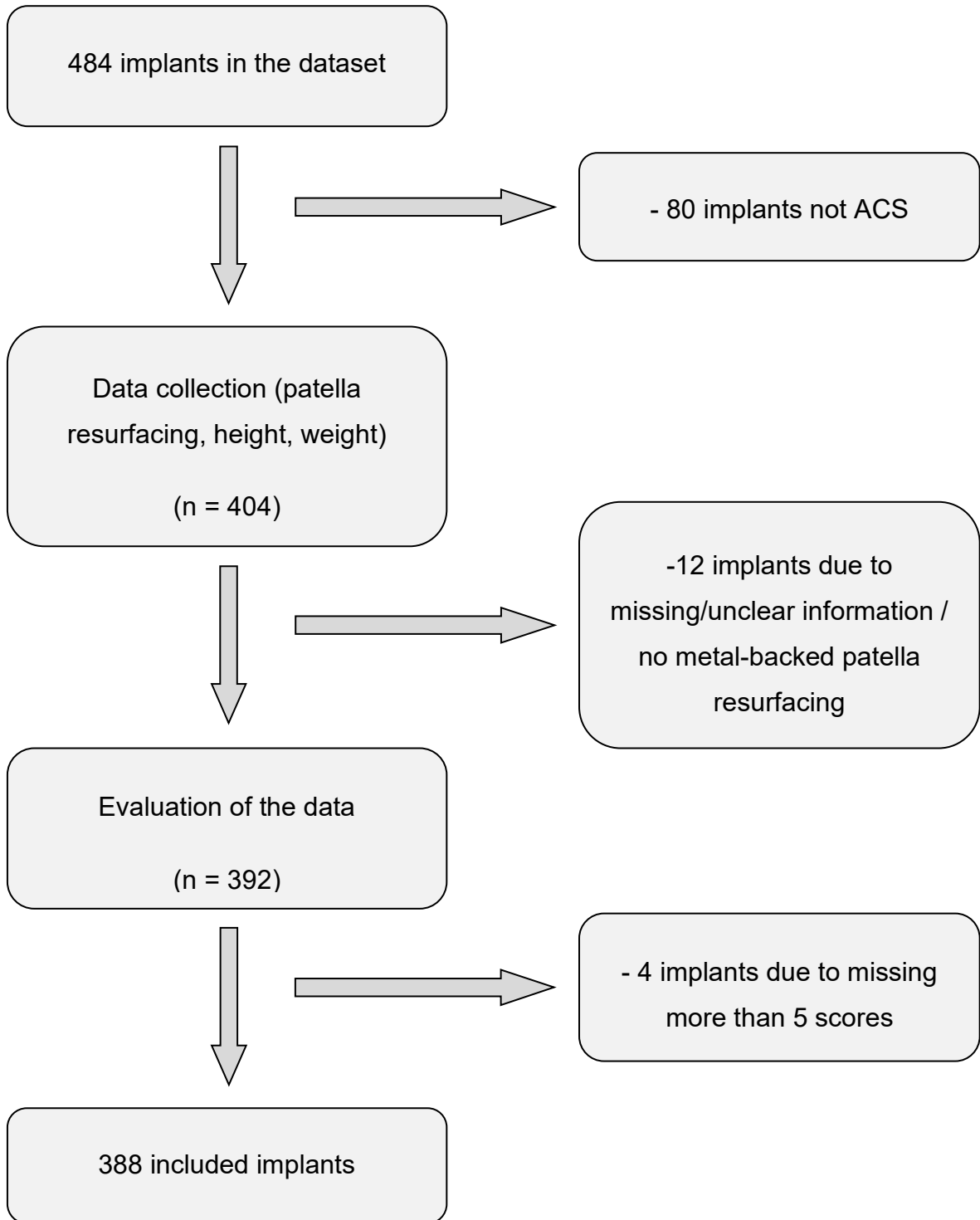


Figure 18. Flow chart of patients inclusion

2.3 Outcome Measures

In order to evaluate the clinical outcome of TKA, a variety of well-established scores, as well as the fact if a revision had to be performed, was used. In the following, the scores used in this study are described.

2.3.1 Clinical Scores

2.3.1.1 Knee Society Score

The Knee Society Score (KSS) is an overall knee rating system that is divided into two scores (knee score and functional score) to prevent the problem of poor knee scores due to patient frailty. (65)

To draw deductions, the knee score assesses the knee joint itself using three main parameters (pain, stability, and range of motion), as well as flexion contracture, extension lag, and misalignment. A maximum of 100 points can be achieved with a stable (negligible anteroposterior and mediolateral instability), well-aligned, pain-free knee with 125° of range of motion. (65)

The functional score evaluates the patient's ability to walk and climb stairs, with deductions made for walkers. A person who can walk an unlimited distance and climb and descend a staircase normally without holding onto a railing archives a perfect score of 100 points. (65)

2.3.1.2 Tegner Activity Scale

The Tegner activity scale is a score based on work and sports activities to measure knee function which ranges from 0 (sick leave or disability pension due to knee problems) to 10 (competitive sports on a very high level) (66). A score of 5 to 10 can only be achieved through participation in recreational or competitive sports (67).

In this study, an adapted version for the German regions (Tegner-G), developed by Wirth, Meier, Koch, and Swanenburg (68), was used. The Tegner-G scale is easy to administer, short in length, and provides a reliable and valid tool to measure the outcome for patients with TKA (66).

2.3.1.3 12-item Short Form Survey

The 12-item Short Form Survey (SF-12) is a shorter, yet valid, alternative to the 36-item Short Form Survey (SF-36), to measure the general health status of the patient. 12 items subset of those in the SF-36 are used to measure 8 health concepts (physical functioning, role limitations due to physical health problems, bodily pain, general health, vitality (energy/fatigue), social functioning, role limitations due to emotional problems, and mental health). (69)

From these items two summary measures, the SF-12 physical (PCS-12) and mental (MCS-12) component summary scales are obtained. The PCS-12 and the MCS-12 are then transformed to have a mean of 50 and a standard deviation of 10 in the general U.S. population, meaning that all scores above 50 are above average, and all scores below 50 are below average. (69)

Table 6. SF-12 measurement model; Source: (69)

Item	Scale	Summary Measures
Moderate activities	Physical Functioning	Physical Health (PCS)
Climbing several flights of stairs		
Accomplishing less than you would like to	Role-Physical	
Limited in the kind of activities		
Pain interferes with normal work	Bodily Pain	
In general, would you say your health is ...	General Health	
Having a lot of energy	Vitality	Mental Health (MSC)
Health interferes with social activities	Social Functioning	
Accomplishing less than you would like	Role-Emotional	
Not carrying out activities as carefully as usual		
Feeling calm and peaceful	Mental Health	
Feeling downhearted and blue		

2.3.1.4 Western Ontario and McMaster Universities Osteoarthritis Index

The Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) is a health questionnaire designed to measure symptoms and physical disability in patients with knee or hip OA, to assess changes in health status as a result of treatment intervention. (70)

The WOMAC assesses three dimensions using 24 questions (5 pain, 2 stiffness, and 17 physical function). An ordinal scale of 0 (“none”) to 4 (“extreme”) is used for the Likert version of the WOMAC, with lower scores indicating lower levels of symptoms or physical disability, resulting in a maximum total score of 96. (71)

The score is then normalized to a 100 point scale. (72)

The WOMAC score was validated for paper, telephone, computer mouse and touch-screen administration. (72)

2.3.1.5 Range of Motion

To assess the active range of motion (ROM) of the knee a 30.5 cm goniometer is usually used, providing a reliability coefficient of 0.99. To ensure that the test is reproducible and thus comparable, all measurements should be performed with the patient in the same position (supine) using the same size and design of goniometer. (73)

2.3.1.6 Visual Analogue Scale

The Visual Analogue Scale (VAS) is a commonly used tool to assess pain (74). It consists of a 10 cm line, anchored by two verbal descriptors representing two extremes of pain (usually “no pain” and “worst imaginable pain”). The patient is asked to place a mark on the line to indicate the pain intensity. The score is determined by using a millimeter scale, measuring from the zero anchor to the patient’s mark, providing 101 levels of pain intensity. (74,75)

In this study a centimeter scale was used, resulting in a score from 0-10 instead of 0-100.

2.4 Statistical Methods

The data (patient data, day of the surgery, implant, follow-up, clinical scores, revision) from the pre-existing dataset was collected in Excel (Microsoft, Redmont, WA) and supplemented with the variables: patella resurfacing, weight, size, and BMI.

The scores and the revision rate were tested for normality using the Shapiro-Wilk test. To determine whether there are significant differences between the BMI groups, three different statistical tests were used. The Kruskal-Wallis test was used to assess the scores because these are non-normally distributed, unconnected samples with more than 2 groups. Furthermore, the Mann-Whitney-U test was used for the pairwise comparison between groups of a score when the Kruskal-Wallis test showed a significant difference between groups. The p-values for the Mann-Whitney-U test were then corrected using the Bonferroni correction.

To assess the revision rate, the Chi-squared test was used, which is suitable for binary data in unconnected samples.

P-values ≤ 0.05 were considered significant. All statistic calculations were performed with IBM SPSS Statistics version 26 (IBM, Armonk, NY).

3 RESULTS

3.1 Patient Characteristics

We included 388 implants (357 patients) with a mean age of 65.5, 68.3 % being female. They all received an ACS[®] TKA implant with metal-backed patella resurfacing at Bad Radkersburg State Hospital between 2003 and 2011.

Table 7. Baseline characteristics of the overall population (n = 388); * one value is missing

Characteristics	Total (n = 388)
Age (years)	65.5
Female sex n (%)	265 (68.3)
Follow-up (years)	10.1
BMI (kg/m ²)	30.9
Revision n (%)	41* (10.6)

All patients underwent clinical and radiological examination shortly after surgery, and various knee scores were collected after a mean follow-up of 10.1 (4.6-12.3) years.

Table 8. Baseline characteristics of the pre- and postoperative scores

Score	n	Mean \pm SD	Median	Minimum	Maximum
Preoperative					
VAS	388	7.7 \pm 1.3	8	0	10
Tegner	382	3.0 \pm 1.3	3	0	10
Postoperative 10 years					
VAS	387	1.9 \pm 1.9	2	0	10
Tegner	381	2.8 \pm 1.2	3	0	7
KSS-Knee	387	83.6 \pm 15.3	89	2	100
KSS-Function	386	67.8 \pm 24.9	70	0	100
WOMAC	386	81.7 \pm 14.9	85.6	21.9	100
ROM-Flexion	388	102.8 \pm 14.0	100	35	135
PCS-12	382	37.3 \pm 10.3	36.9	13.9	61.3
MCS-12	382	52.8 \pm 10.9	55.8	15.7	70.4

3.1.1 BMI Subgroups

The implants were divided into 3 subgroups depending on their BMI. The non-obese group with a mean BMI of 27.1 ± 2.2 kg/m², the obese group with a mean BMI of 33.7 ± 2.8 kg/m² and the morbidly obese group with a mean BMI of 43.4 ± 3.3 kg/m². It should be said that the average BMI of the non-obese group is ≥ 25 kg/m² and thus the majority of the patients in this group are overweight and not normal weight, according to the WHO BMI classification.

Table 9. Baseline characteristics of the BMI subpopulations; * one value is missing

Characteristics	Non-obese (n = 193)	Obese (n = 174)	Morbidly Obese (n = 21)
Age (years)	66.7 ± 8.7	64.6 ± 7.6	62.2 ± 9.4
Female sex n (%)	125 (64.8)	124 (71.3)	16 (76.2)
Follow-up (month)	121 ± 10	121 ± 8	121 ± 8
BMI (kg/m ²)	27.1 ± 2.2	33.7 ± 2.8	43.4 ± 3.3
Revision n (%)	23* (12.0)	15 (8.6)	3 (14.3)

193 Implants (49.7 %) were in the **non-obese group** with a mean age of 66.7 ± 8.7, 64.8 % being female.

Table 10. Baseline characteristics of the pre- and postoperative scores in the non-obese group and test for normality

Score	n	Mean ± SD	Median	Minimum	Maximum	Normality
Preoperative						
VAS	193	7.7 ± 1.4	8	0	10	0.000
Tegner	192	3.1 ± 1.3	3	0	10	0.000
Postoperative 10 years						
VAS	193	1.9 ± 1.9	1	0	9	0.000
Tegner	192	2.9 ± 1.1	3	0	7	0.000
KSS-Knee	192	84.7 ± 14.2	89	32	100	0.000
KSS-Function	191	70.6 ± 23.1	70	0	100	0.000
WOMAC	192	82.5 ± 13.8	87.1	37.9	100	0.000
ROM-Flexion	193	103.1 ± 13.3	100	60	135	0.000
PCS-12	190	38.0 ± 9.9	37.2	15.2	61.3	0.110
MCS-12	190	53.3 ± 10.9	56.5	15.7	70.4	0.000

174 Implants (44.8 %) were in the **obese group** with a mean age of 64.6 ± 7.6 , 71.3 % being female.

Table 11. Baseline characteristics of the pre- and postoperative scores in the obese group and test for normality

Score	n	Mean \pm SD	Median	Minimum	Maximum	Normality
Preoperative						
VAS	174	7.7 ± 1.4	8	3	10	0.000
Tegner	169	2.9 ± 1.2	3	0	6	0.000
Postoperative 10 years						
VAS	173	1.9 ± 2.0	2	0	10	0.000
Tegner	168	2.7 ± 1.2	3	0	6	0.000
KSS-Knee	174	82.2 ± 16.6	88	2	100	0.000
KSS-Function	174	65.6 ± 25.7	70	5	100	0.000
WOMAC	174	81.2 ± 16.2	85.3	21.9	100	0.000
ROM-Flexion	174	102.9 ± 14.5	100	35	135	0.000
PCS-12	171	36.6 ± 10.8	35.5	13.9	61.2	0.008
MCS-12	171	53.0 ± 10.3	55.9	25.2	68.4	0.000

21 Implants (5.4 %) were in the **morbidly obese group** with a mean age of 62.2 ± 9.4 , 76.2 % being female.

Table 12. Baseline characteristics of the pre- and postoperative scores in the morbidly obese group and test for normality

Score	n	Mean \pm SD	Median	Minimum	Maximum	Normality
Preoperative						
VAS	21	7.8 ± 0.9	8	6	9	0.002
Tegner	21	2.9 ± 1.1	3	1	5	0.055
Postoperative 10 years						
VAS	21	2.1 ± 1.7	2	0	6	0.013
Tegner	21	2.5 ± 1.5	2	1	7	0.002
KSS-Knee	21	85.7 ± 12.7	91	54	98	0.001
KSS-Function	21	59.6 ± 31.6	55	5	100	0.050
WOMAC	20	78.6 ± 12.7	75.8	48	100	0.417
ROM-Flexion	21	98.1 ± 16.2	100	45	120	0.001
PCS-12	21	36.6 ± 9.4	36.1	22.5	54.8	0.498
MCS-12	21	46.4 ± 14.1	47.4	19	65	0.167

3.2 Analysis of Clinical Scores

All clinical scores from the pre-existing dataset were compared in relation to the BMI. Except for Tegner postoperative with a p-value of 0.046, no significant difference was found between the scores with respect to BMI.

Table 13. Analysis of the outcome scores with respect to the BMI group

Score	Non-obese		Obese		Morbidly Obese		p-value
	n	Mean ± SD	n	Mean ± SD	n	Mean ± SD	
Preoperative							
VAS	193	7.7 ± 1.4	174	7.7 ± 1.4	21	7.8 ± 0.9	0.923
Tegner	192	3.1 ± 1.3	169	2.9 ± 1.2	21	2.9 ± 1.1	0.186
Postoperative 10 years							
VAS	193	1.9 ± 1.9	173	1.9 ± 2.0	21	2.1 ± 1.7	0.762
Tegner	192	2.9 ± 1.1	168	2.7 ± 1.2	21	2.5 ± 1.5	0.046
KSS-Knee	192	84.7 ± 14.2	174	82.2 ± 16.6	21	85.7 ± 12.7	0.509
KSS-Function	191	70.6 ± 23.1	174	65.6 ± 25.7	21	59.6 ± 31.6	0.097
WOMAC	192	82.5 ± 13.8	174	81.2 ± 16.2	20	78.6 ± 12.7	0.362
ROM-Flexion	193	103.1 ± 13.3	174	102.9 ± 14.5	21	98.1 ± 16.2	0.681
PCS-12	190	38.0 ± 9.9	171	36.6 ± 10.8	21	36.6 ± 9.4	0.383
MCS-12	190	53.3 ± 10.9	171	53.0 ± 10.3	21	46.4 ± 14.1	0.091

To further evaluate between which groups there is a significant difference for Tegner postoperatively, a pairwise comparison was performed. The corrected p-value showed that there is no significant difference between groups for Tegner postoperative.

Table 14. Pairwise comparison of the postoperative Tegner score with corrected p-value

	p-value	Corrected p-value
Morbidly Obese - Obese	0.170	0.510
Morbidly Obese - Non-obese	0.035	0.105
Obese - Non-obese	0.089	0.267

3.3 Analysis of Revision Rate

As with the clinical scores, there was no significant difference ($p = 0.495$) in revision rate between the BMI groups with a revision rate of 12.0 %, 8.6 % and 14.3 % in the non-obese, the obese and morbidly obese group respectively. Nevertheless, it should be noted that the overall revision rate of 10.6 % is above average.

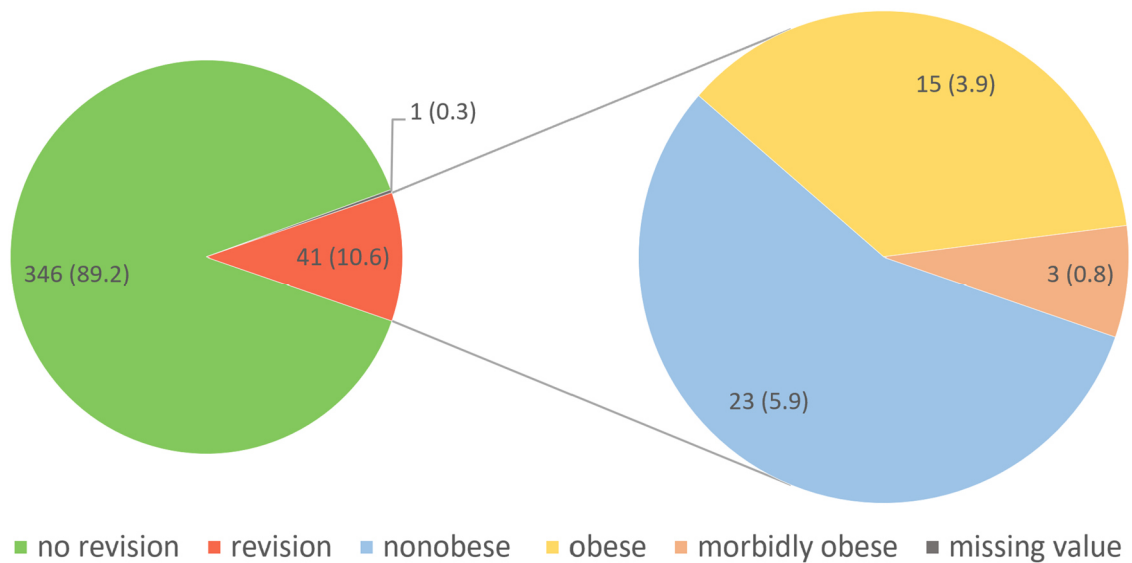


Figure 19. Overview of revision rate n (%) separated in BMI groups

4 DISCUSSION

This retrospective study compared the clinical outcome of primary TKA with an ACS® implant and metal-backed patella resurfacing in relation to the BMI and showed that there is no significant difference between the three BMI groups (non-obese [BMI < 30 kg/m²], obese [BMI 30.00-39.99 kg/m²], morbidly obese [BMI > 40 kg/m²]).

According to the World Health Organization (WHO), overweight and obesity in adults is defined as a BMI of ≥ 25 kg/m² and ≥ 30 kg/m² respectively caused by abnormal fat accumulation that may affect health. (76)

In many parts of the world, especially in high-income countries, obesity is increasing at an alarming rate: about 2 billion people are overweight and one-third of them are obese (77). In 2015, the GBD 2015 Obesity Collaborators (78) found that obesity has doubled in more than 70 countries since 1980 and has steadily increased in most others.

An increase in BMI leads to an almost exponential increase in the risk of knee osteoarthritis, as shown in a meta-analysis by Zhou et al. (79).

Moreover, it is technically challenging, takes longer, and requires greater exposure of the surgical access to perform TKA in obese patients. (80)

According to the review article by Kulkarni et al. (28), short- and medium- term complication rates are potentially higher in obese patients undergoing TKA, with even higher rates in super obese patients, leading to significantly longer hospital stays. Kulkarni et al. also noted that compared with only 7 % of non-obese patients undergoing TKA, 30 % of obese patients had at least three comorbidities (e.g., diabetes, coronary artery disease, hyperlipidemia, hypertension, sleep apnea), leading to an increased risk of perioperative complications.

The debate about the clinical outcome of TKA in obese patients remains controversial since studies dedicated to this field reveal different conclusions in their results. This is shown in a review article by Rodriguez-Merchan et al. (63), who concluded that 16 studies found no negative association between obesity and TKA

outcomes, whereas 24 studies reported inferior TKA outcomes in obese patients. In addition, 3 systematic reviews demonstrated that obesity negatively affects the outcome, complication rate, implant survival, and cost of TKA.

Hakim et al. (81) is the study most comparable to ours because they chose the same grouping with a mean follow-up of 10.6 ± 2.8 years, but the outcome of TKA was assessed by using only the KSS knee and function score. In contrast to our study, they found a significant difference in KSS knee score between the BMI < 29.99 kg/m² group and the BMI > 40 kg/m² group ($p = 0.046$) and between the BMI 30-39.99 kg/m² group and the BMI > 40 kg/m² group ($p = 0.030$). Similarly, the KSS function score showed a significant difference between the BMI < 29.99 kg/m² group and the BMI > 40 kg/m² group ($p = 0.011$) and the BMI 30-39.99 kg/m² group and the BMI > 40 kg/m² group ($p = 0.001$) as well. Compared to our study, their data is normally distributed and has a much lower standard deviation, which may lead to a non-significant result in ours, despite similar mean values of the KSS knee and function score. Nevertheless, Hakim et al. emphasize that despite the increased risk of perioperative complications after TKA in morbidly obese patients, outcomes are similar to those in obese patients, and they should therefore continue to enjoy the benefits of TKA.

Collins et al. (82) analyzed the outcome of 445 consecutive primary TKRs using Press Fit Condylar Sigma prosthesis after 6 months, 18 months, 3 years, 5 years and 9 years regarding the BMI divided into two groups (non-obese [BMI < 30 kg/m²], Obese [BMI \geq 30 kg/m²]). The obese group was further classified into two groups (mildly obese [BMI 30-35 kg/m²], highly obese [BMI \geq 35 kg/m²]) to show the effect of increasing obesity on outcome. Collins et al. found a small but significant negative impact on clinical outcome (KSS knee and function), with highly obese patients having lower function scores than non-obese patients, however, significant improvements in outcome were maintained in all groups nine years after TKR. Regarding the overall complication rates and implant survival no difference between non-obese and obese patients was found. In summary, this study suggests that there is no reason to limit access to TKR in obese patients given the relief of symptoms after TKR and the low perioperative complication and revision rates in both groups.

In a prospective study, Bourne et al. (83) examined the effects of sex, age, diagnosis, and obesity in 728 consecutive patients undergoing 843 primary TKAs on changes in preoperative and postoperative outcomes (Knee Society clinical rating, WOMAC, and MCS-12) with a mean follow-up of 9.5 (5-11) years. For BMI comparison, patients were divided into 7 groups (underweight, normal, overweight, class I, class II, class III and class IV). The results of this study showed that BMI had little effect on the change in scores. The underweight group showed a significant difference in all scores, but this group consisted of merely 4 implants, and must therefore be interpreted with caution. Class III (n = 86) and Class IV (n = 15) showed a significant difference in the change in WOMAC score. Revision was required for 13 implants (1.5 %). Overall, this study took a different approach to analyzing the outcome of TKA in relation to BMI by comparing only the change in outcome and not the absolute values as in our study. Nevertheless, their suggestion is consistent with our results, which concluded that obesity should not be a discriminating factor in determining which patients should enjoy the benefits of TKA.

Gaillard et al. (84) compared the survival and outcomes of primary TKAs with a posterior-stabilized, cementless, rotating-platform implant regarding the BMI divided into four groups (BMI < 25 kg/m², 25 kg/m² ≤ BMI < 30 kg/m², 30 kg/m² ≤ BMI < 35 kg/m², BMI ≥ 35 kg/m²). 880 implants were analyzed with an average follow-up of 61.7 (12-146) months. In terms of clinical outcome, higher levels of obesity were associated with lower maximum flexion angle (p < 0.001), KSS knee score (p < 0.001), and functional score (p = 0.005). Postoperative patient satisfaction (p = 0.9), total rate of complications (p = 0.9) and implant revision (p = 0.9), showed no significant difference.

Järvenpää et al. (85) compared the clinical outcome of 48 patients and 52 knees who had TKA regarding their BMI divided into two groups (non-obese [BMI < 30 kg/m²] and obese [BMI ≥ 30 kg/m²]). After a mean follow-up of 10.8 (9-12) years the obese group showed significantly lower ROM-values (p = 0.016), KSS knee score (p = 0.01), WOMAC pain (VAS 0-100) (p = 0.021), WOMAC stiffness (p = 0.006), WOMAC physical function (p = 0.003), KSS knee score (p = 0.01). For KSS function a p-value of 0.051 was determined. In contrast to our study, they concluded that obesity has a negative effect on the outcome of TKA.

In a prospective observational study of 289 patients who received TKA, Jones et al. (86) showed that there was no significant difference in WOMAC pain ($p = 0.017$) and WOMAC function ($p > 0.20$) between four BMI groups ($< 25.0 \text{ kg/m}^2$, $25.0\text{-}29.9 \text{ kg/m}^2$, $30.0\text{-}34.9 \text{ kg/m}^2$, $\geq 35.0 \text{ kg/m}^2$) after a follow-up of 3 years. The scores were also assessed after 6 months, with a significant difference in WOMAC function ($p = 0.01$) between groups. They therefore concluded that severe obesity can be considered an independent risk factor for slow recovery over three years after TKA. This finding could explain the different conclusions in studies regarding the outcome of TKA in relation to BMI, as it is possible that the outcome is highly dependent on the time of the assessment.

In a prospective cohort study with 157 patients Li et al. (87) showed that there is no significant difference in ROM ($p = 0.912$) after a follow-up of 90 days between three groups (normal [BMI $18.0\text{-}24.9 \text{ kg/m}^2$], overweight [BMI $25.0\text{-}29.9 \text{ kg/m}^2$] and the obese [BMI $\geq 30.0 \text{ kg/m}^2$]) which underwent primary TKA, with 115.2 ± 5.8 , 115.3 ± 6.1 and 115.9 ± 5.2 degree of flexion respectively. Moreover, no significant difference was found in VAS ($p > 0.05$) between groups after 90 days. The ROM was also assessed after 3, 15, and 30 days postoperatively, showing a significantly higher ROM in the obese group ($104.4 \pm 8.5^\circ$) than in the normal ($98.9 \pm 8.9^\circ$, $p = 0.010$) and overweight ($97.7 \pm 7.8^\circ$, $p = 0.001$) groups. The VAS was also assessed after 1, 2, 3, 15 and 30 days. The VAS in rest was 2.0 ± 0.7 in the obese group after one day and therefore significantly lower than in the normal (2.2 ± 0.6 , $p = 0.043$) and overweight (2.3 ± 0.6 , $p = 0.010$) groups. No significant difference in the incidence of complications was shown.

Regarding the revision rate in TiN coated TKR, Mohammed et al. (47) described a revision rate of 4.9 % with a follow up of 10 years and Louwerens and Hockers et al. (88) reported a revision rate of 6 %, which is much lower than ours (10.6 %). This may be due to the fact that our study was not performed by the same hospital in which the surgery took place, allowing a more critical analysis. Another reason for the high revision rate could be the use of metal-backed patella resurfacing, which should be further investigated.

In conclusion, this study showed that there is no significant difference in knee function, pain, and revision rate after primary TKA with an ACS® implant with metal-backed patella resurfacing by means of various well-established knee scores in relation to the BMI. Although the results of the above-mentioned studies differ in part from those of this study, their summary is largely the same as ours, namely that after TKA there is a strong improvement in symptoms even in obese patients. Therefore, they should also receive TKA regardless of their BMI.

However, it should be noted that there are some limitations. Despite the high number of included patients, the group sizes are uneven with few patients in the morbidly obese group, making the comparison to the morbidly obese group less valid due to the smaller numbers. Furthermore, it should be taken into consideration that a retrospective study design is generally not the most valid method and since this study is based on a pre-existing dataset, and the added values (height, weight and resulting BMI) were obtained from medical records and anesthesia protocols, the circumstances under which the data was obtained could not be accounted for.

Nevertheless, the strengths of this study are the single-center cohort, the high number of patients included, the wide variety of scores, and the long mean follow-up of 10.1 years.

5 CONCLUSIONS

In conclusion, this study has revealed that there is no significant difference in knee function, pain, and revision rate after primary TKA with an ACS® implant with metal-backed patella resurfacing by means of various well-established knee scores in relation to the BMI. Therefore, a BMI limit for performing TKA cannot be supported, as it has no impact on the result.

6 REFERENCES

1. Platzer W. Taschenatlas Anatomie in 3 Bänden Band 1 Bewegungsapparat. 11th ed. Stuttgart: Thieme; 2013.
2. Schünke M, Schulte E, Schumacher U. Prometheus LernAtlas der Anatomie Allgemeine Anatomie und Bewegungssystem. 2nd ed. Stuttgart: Thieme; 2007.
3. Drake RL, Vogl AW, Mitchell AWM. Gray's Anatomy for Students. 4th ed. Philadelphia: Elsevier; 2020.
4. Anderhuber F, Pera F, Streicher J. Waldeyer - Anatomie des Menschen Lehrbuch und Atlas in einem Band. 19th ed. Berlin: De Gruyter; 2012.
5. Aumüller G, Aust G, Engele J, Kirsch J, Maio G, Mayerhofer A, et al. Duale Reihe Anatomie. 3rd ed. Stuttgart: Thieme; 2014.
6. Thompson JC. Netter's Concise Orthopaedic Anatomy. 2nd ed. Philadelphia: Saunders Elsevier; 2010.
7. Gaillard R, Shabani B, Ballis R, Neyret P, Lustig S. Anatomy, Physiology, and Biomechanics of the Native Knee. In: Rodríguez-Merchán EC, Oussedik S, editors. Total Knee Arthroplasty A Comprehensive Guide Cham: Springer; 2015. p. 1-25.
8. Gilroy AM, MacPherson BR, Wikenheiser JC, Schuenke M, Schulte E, Schumacher U. Atlas of Anatomy. 4th ed. New York: Thieme; 2020.
9. Jerosch J, Heisel J, Tibesku CO. Knieendoprothetik Indikationen, Operationstechnik, Nachbehandlung, Begutachtung 2nd ed. Berlin Heidelberg: Springer; 2015.
10. Schünke M. Topografie und Funktion des Bewegungssystems Funktionelle Anatomie für Physiotherapeuten. 3rd ed. Stuttgart: Thieme; 2018.
11. Pereira D, Ramos E, Branco J. Osteoarthritis. Acta Médica Portuguesa 2015;28(1):99-106.
12. Niethard FU, Pfeil J, Biberthaler P. Duale Reihe Orthopädie und Unfallchirurgie. 8th ed. Stuttgart: Thieme; 2017.
13. Sharma L. Osteoarthritis of the Knee. The New England Journal of Medicine 2021;384(1):51-9.
14. Cui A, Li H, Wang D, Zhong J, Chen Y, Lu H. Global, regional prevalence, incidence and risk factors of knee osteoarthritis in population-based studies. eClinicalMedicine 2020;29-30:100587.

15. Felson DT, Lawrence RC, Dieppe PA, Hirsch R, Helmick CG, Jordan JM, et al. Osteoarthritis: new insights. Part 1: the disease and its risk factors. *Annals of Internal Medicine* 2000;133(8):635-46.
16. Haq I, Murphy E, Dacre J. Osteoarthritis. *Postgraduate Medical Journal* 2003;79(933):377-83.
17. Silverwood V, Blagojevic-Bucknall M, Jinks C, Jordan JL, Protheroe J, Jordan KP. Current evidence on risk factors for knee osteoarthritis in older adults: a systematic review and meta-analysis. *Osteoarthritis and Cartilage* 2015;23(4):507-15.
18. O'Neill TW, McCabe PS, McBeth J. Update on the epidemiology, risk factors and disease outcomes of osteoarthritis. *Best Practice & Research Clinical Rheumatology* 2018;32(2):312-26.
19. Ruchholtz S, Wirtz DC. *Orthopädie und Unfallchirurgie essentials Intensivkurs zur Weiterbildung*. 2nd ed. Stuttgart: Thieme; 2013.
20. Rodríguez-Merchán EC, Oussedik S. The Arthritic Knee: Etiology and Patterns of Disease. In: Rodríguez-Merchán EC, Oussedik S, editors. *Total Knee Arthroplasty A Comprehensive Guide* Cham: Springer; 2015. p. 27-37.
21. Michael JW, Schlüter-Brust KU, Eysel P. The epidemiology, etiology, diagnosis, and treatment of osteoarthritis of the knee. *Deutsches Ärzteblatt International* 2010;107(9):152-62.
22. Thieme Verlag. *Endspurt Klinik Orthopädie, Unfallchirurgie Skript 8*. 1st ed. Stuttgart: Thieme; 2015.
23. Waldt S, Eiber M. Erkrankungen der Gelenke. In: Vogl TJ, Reith W, Rummeny EJ, editors. *Diagnostische und Interventionelle Radiologie*. Berlin Heidelberg: Springer; 2011. p. 1221-68.
24. Antony J. Automatic quantification of radiographic knee osteoarthritis severity and associated diagnostic features using deep convolutional neural networks [PhD Thesis on the Internet]. Dublin: Dublin City University; 2018 Available from: https://www.researchgate.net/publication/339941257_PhD_Thesis_Automatic_quantification_of_radiographic_knee_osteoarthritis_severity_and_associated_diagnostic_features_using_deep_convolutional_neural_networks.
25. Thorlund JB, Juhl CB, Roos EM, Lohmander LS. Arthroscopic surgery for degenerative knee: systematic review and meta-analysis of benefits and harms. *The BMJ* 2015;350:h2747.
26. Shan L, Shan B, Suzuki A, Nouh F, Saxena A. Intermediate and long-term quality of life after total knee replacement: a systematic review and meta-analysis. *The Journal of Bone and Joint Surgery. American Volume*. 2015;97(2):156-68.

27. Seidlitz C, Kip M. Introduction to the Indications and Procedures In: Bleß H-H, Kip M, editors. *White Paper on Joint Replacement Status of Hip and Knee Arthroplasty Care in Germany*. Berlin: Springer; 2018. p. 1-14.
28. Kulkarni K, Karssiens T, Kumar V, Pandit H. Obesity and osteoarthritis. *Maturitas* 2016;89:22-8.
29. Gao J, Xing D, Dong S, Lin J. The primary total knee arthroplasty: a global analysis. *Journal of Orthopaedic Surgery and Research* 2020;15(1):190.
30. Adie S, Harris I, Chuan A, Lewis P, Naylor JM. Selecting and optimising patients for total knee arthroplasty. *The Medical Journal of Australia* 2019;210(3):135-41.
31. Lützner J, Lange T, Schmitt J, Kopkow C, Aringer M, Böhle E, et al. S2k-Leitlinie: Indikation Knieendoprothese Evidenz- und konsensbasierte Indikationsstellung in der Knie Endoprothetik (EKIT-Knie). *Der Orthopäde* 2018;47(9):777-81.
32. Van Manen MD, Nace J, Mont MA. Management of primary knee osteoarthritis and indications for total knee arthroplasty for general practitioners. *The Journal of the American Osteopathic Association* 2012;112(11):709-15.
33. Hsu H, Siwiec RM. Knee Arthroplasty. [updated 2022 Sep 4]. In: StatPearls [Internet]. Treasure Island: StatPearls Publishing; 2023 [cited 2023 May 21]. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK507914/>.
34. Gemayel AC, Varacallo M. Total Knee Replacement Techniques. [updated 2022 Sep 4]. In: StatPearls [Internet]. Treasure Island: StatPearls Publishing; 2023 [cited 2023 May 21]. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK538208/>.
35. Varacallo M, Luo TD, Johanson NA. Total Knee Arthroplasty Techniques. [updated 2022 Nov 3]. In: StatPearls [Internet]. Treasure Island: StatPearls Publishing; 2023 [cited 2023 May 21]. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK499896/>.
36. Li N, Tan Y, Deng Y, Chen L. Posterior cruciate-retaining versus posterior stabilized total knee arthroplasty: a meta-analysis of randomized controlled trials. *Knee Surgery, Sports Traumatology, Arthroscopy* 2014;22(3):556-64.
37. Kolisek FR, McGrath MS, Marker DR, Jessup N, Seyler TM, Mont MA, et al. Posterior-stabilized versus posterior cruciate ligament-retaining total knee arthroplasty. *The Iowa Orthopaedic Journal* 2009;29:23-7.
38. Own image. Medical University of Graz - Department of Orthopedics and Trauma 2023.
39. Flören M, Reichel H. Implantate. In: Wirtz DC, editor. *AE-Manual der Endoprothetik Knie*. Berlin Heidelberg Springer; 2011. p. 57-71.

40. Halder AM, Köhler S. Indikation, Untersuchungen, Aufklärung und Planung der Knieendoprothese. In: Wirtz DC, editor. AE-Manual der Endoprothetik Knie. Berlin Heidelberg Springer; 2011. p. 85-108.
41. Capella M, Dolfin M, Saccia F. Mobile bearing and fixed bearing total knee arthroplasty. *Annals of Translational Medicine* 2016;4(7):127.
42. Reichel H, Bieger R. Endoprothetik. In: Kohn D, Günther K-P, Scharf H-P, editors. Spezielle orthopädische Chirurgie. München: Elsevier; 2016. p. 270-304.
43. Implantcast GmbH. TiN-coating [Internet]. 2023 [cited 2023 May 18]. Available from: <https://www.implantcast.de/en/company/technology/tin-coating/>
44. Hauer G, Leitner L, Ackerl M, Klim S, Vielgut I, Ehall R, et al. Titanium-Nitride Coating Does Not Result in a Better Clinical Outcome Compared to Conventional Cobalt-Chromium Total Knee Arthroplasty after a Long-Term Follow-Up: A Propensity Score Matching Analysis. *Coatings* 2020;10(5):442.
45. Mezger PR, Creugers NH. Titanium nitride coatings in clinical dentistry. *Journal of Dentistry* 1992;20(6):342-4.
46. Galetz MC, Seiferth SH, Theile B, Glatzel U. Potential for adhesive wear in friction couples of UHMWPE running against oxidized zirconium, titanium nitride coatings, and cobalt-chromium alloys. *Journal of Biomedical Materials Research Part B: Applied Biomaterials* 2010;93(2):468-75.
47. Mohammed A, Metcalfe A, Woodnutt D. Medium-term outcome of titanium nitride, mobile bearing total knee replacement. *Acta Orthopaedica Belgica* 2014;80(2):269-75.
48. Thomas P, Weik T, Roider G, Summer B, Thomsen M. Influence of Surface Coating on Metal Ion Release: Evaluation in Patients With Metal Allergy. *Orthopedics* 2016;39(3 Suppl):24-30.
49. Sonntag R, Reinders J, Kretzer JP. What's next? Alternative materials for articulation in total joint replacement. *Acta Biomaterialia* 2012;8(7):2434-41.
50. van Hove RP, Brohet RM, van Royen BJ, Nolte PA. No clinical benefit of titanium nitride coating in cementless mobile-bearing total knee arthroplasty. *Knee Surgery, Sports Traumatology, Arthroscopy* 2015;23(6):1833-40.
51. Implantcast GmbH. Mobile-Bearing system ACS® MB system [Internet]. 2023 [cited 2023 May 18]. Available from: <https://www.implantcast.de/en/for-medical-professionals/products/standard/-/tumour-prosthetics/knee-endoprosthesis/primary-endoprosthesis-knee/acsr-mb/>

52. Scuderi GR. The Basic Principles. In: Scuderi GR, Tria AJ, editors. *Knee Arthroplasty Handbook Techniques in Total Knee and Revision Arthroplasty*. New York: Springer; 2006. p. 1-6.
53. Röhrig H. Operation der Kniegelenksendoprothese. In: Wirtz DC, editor. *AE-Manual der Endoprothetik Knie*. Berlin Heidelberg Springer; 2011. p. 127-41.
54. Tria AJJ. Instrumentation in Total Knee Arthroplasty. In: Scuderi GR, Tria AJ, editors. *Knee Arthroplasty Handbook Techniques in Total Knee and Revision Arthroplasty*. New York: Springer; 2006. p. 7-24.
55. Pulido L, Parvizi J, Macgibeny M, Sharkey PF, Purtill JJ, Rothman RH, et al. In Hospital Complications After Total Joint Arthroplasty. *The Journal of Arthroplasty* 2008;23(6 Suppl):139-45.
56. Weißer M, Zerwes U, Krupka S, Schönfelder T, Klein S, Bleß H-H. Status of Healthcare. In: Bleß H-H, Kip M, editors. *White Paper on Joint Replacement Status of Hip and Knee Arthroplasty Care in Germany*. Berlin: Springer; 2018. p. 41-89.
57. Pabinger C, Berghold A, Boehler N, Labek G. Revision rates after knee replacement. Cumulative results from worldwide clinical studies versus joint registers. *Osteoarthritis and Cartilage* 2013;21(2):263-8.
58. Sadoghi P, Liebensteiner M, Agreiter M, Leithner A, Böhler N, Labek G. Revision surgery after total joint arthroplasty: a complication-based analysis using worldwide arthroplasty registers. *The Journal of Arthroplasty* 2013;28(8):1329-32.
59. Suri P, Morgenroth DC, Hunter DJ. Epidemiology of osteoarthritis and associated comorbidities. *PM&R: The Journal of Injury, Function and Rehabilitation* 2012;4(5 Suppl):10-9.
60. Jinks C, Jordan K, Croft P. Disabling knee pain--another consequence of obesity: results from a prospective cohort study. *BMC Public Health* 2006;6:258.
61. Blagojevic M, Jinks C, Jeffery A, Jordan KP. Risk factors for onset of osteoarthritis of the knee in older adults: a systematic review and meta-analysis. *Osteoarthritis and Cartilage* 2010;18(1):24-33.
62. Friedman RJ, Hess S, Berkowitz SD, Homering M. Complication rates after hip or knee arthroplasty in morbidly obese patients. *Clinical Orthopaedics and Related Research* 2013;471(10):3358-66.
63. Rodriguez-Merchan EC. Review article: Outcome of total knee arthroplasty in obese patients. *Journal of Orthopaedic Surgery* 2015;23(1):107-10.

64. Ricciardi BF, Giori NJ, Fehring TK. Clinical Faceoff: Should Orthopaedic Surgeons Have Strict BMI Cutoffs for Performing Primary TKA and THA? *Clinical Orthopaedics and Related Research* 2019;477(12):2629-34.
65. Insall JN, Dorr LD, Scott RD, Scott WN. Rationale of the Knee Society clinical rating system. *Clinical Orthopaedics and Related Research* 1989;248:13-4.
66. Swanenburg J, Koch PP, Meier N, Wirth B. Function and activity in patients with knee arthroplasty: validity and reliability of a German version of the Lysholm Score and the Tegner Activity Scale. *Swiss Medical Weekly* 2014;144:w13976.
67. Tegner Y, Lysholm J. Rating systems in the evaluation of knee ligament injuries. *Clinical Orthopaedics and Related Research* 1985;198:43-9.
68. Wirth B, Meier N, Koch PP, Swanenburg J. Entwicklung und Evaluation einer deutschen Version der Tegner Aktivitätsskala zur Erfassung der Funktion nach einer Verletzung des vorderen Kreuzbands. *Sportverletzung Sportschaden* 2013;27(1):21-7.
69. Ware JE, Keller SD, Kosinski M. SF-12: How to score the SF-12 physical and mental health summary scales. 2nd ed. Boston: Health Institute, New England Medical Center; 1995.
70. McConnell S, Kolopack P, Davis AM. The Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC): a review of its utility and measurement properties. *Arthritis Care & Research* 2001;45(5):453-61.
71. Collins NJ, Misra D, Felson DT, Crossley KM, Roos EM. Measures of knee function: International Knee Documentation Committee (IKDC) Subjective Knee Evaluation Form, Knee Injury and Osteoarthritis Outcome Score (KOOS), Knee Injury and Osteoarthritis Outcome Score Physical Function Short Form (KOOS-PS), Knee Outcome Survey Activities of Daily Living Scale (KOS-ADL), Lysholm Knee Scoring Scale, Oxford Knee Score (OKS), Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), Activity Rating Scale (ARS), and Tegner Activity Score (TAS). *Arthritis Care & Research* 2011;63(11 Suppl):208-28.
72. Wright RW. Knee injury outcomes measures. *The Journal of the American Academy of Orthopaedic Surgeons* 2009;17(1):31-9.
73. Gabr A, Tansey R, Haddad FS. Outcome Measures in Total Knee Arthroplasty. In: Rodríguez-Merchán EC, Oussedik S, editors. *Total Knee Arthroplasty A Comprehensive Guide* Cham: Springer; 2015. p. 79-87.
74. Williamson A, Hoggart B. Pain: a review of three commonly used pain rating scales. *Journal of Clinical Nursing* 2005;14(7):798-804.
75. Jensen MP, Karoly P, Braver S. The measurement of clinical pain intensity: a comparison of six methods. *Pain* 1986;27(1):117-26.

76. World Health Organization. Obesity and overweight [Internet]. 2021 June 9 [cited 2023 May 18]. Available from: <https://www.who.int/en/news-room/fact-sheets/detail/obesity-and-overweight>
77. Seidell JC, Halberstadt J. The global burden of obesity and the challenges of prevention. *Annals of Nutrition and Metabolism* 2015;66(Suppl 2):7-12.
78. Afshin A, Forouzanfar MH, Reitsma MB, Sur P, Estep K, Lee A, et al. Health Effects of Overweight and Obesity in 195 Countries over 25 Years. *The New England Journal of Medicine* 2017;377(1):13-27.
79. Zhou ZY, Liu YK, Chen HL, Liu F. Body mass index and knee osteoarthritis risk: a dose-response meta-analysis. *Obesity* 2014;22(10):2180-5.
80. Salih S, Sutton P. Obesity, knee osteoarthritis and knee arthroplasty: a review. *BMC Sports Science, Medicine and Rehabilitation* 2013;5(1):25.
81. Hakim J, Volpin G, Amashah M, Alkeesh F, Khamaisy S, Cohen M, et al. Long-term outcome of total knee arthroplasty in patients with morbid obesity. *International Orthopaedics* 2020;44(1):95-104.
82. Collins RA, Walmsley PJ, Amin AK, Brenkel IJ, Clayton RA. Does obesity influence clinical outcome at nine years following total knee replacement? *The Journal of Bone and Joint Surgery. British Volume.* 2012;94(10):1351-5.
83. Bourne RB, McCalden RW, MacDonald SJ, Mokete L, Guerin J. Influence of patient factors on TKA outcomes at 5 to 11 years followup. *Clinical Orthopaedics and Related Research* 2007;464:27-31.
84. Gaillard R, Gaillard T, Denjean S, Lustig S. No influence of obesity on survival of cementless, posterior-stabilised, rotating-platform implants. *Archives of Orthopaedic and Trauma Surgery* 2017;137(12):1743-50.
85. Järvenpää J, Kettunen J, Soininvaara T, Miettinen H, Kröger H. Obesity has a negative impact on clinical outcome after total knee arthroplasty. *Scandinavian Journal of Surgery* 2012;101(3):198-203.
86. Jones CA, Cox V, Jhangri GS, Suarez-Almazor ME. Delineating the impact of obesity and its relationship on recovery after total joint arthroplasties. *Osteoarthritis and Cartilage* 2012;20(6):511-8.
87. Li H, Gu S, Song K, Liu Y, Wang J, Wang J, et al. The influence of obesity on clinical outcomes following primary total knee arthroplasty: A prospective cohort study. *The Knee* 2020;27(3):1057-63.
88. Louwerens JKG, Hockers N, Achten G, Sierevelt IN, Nolte PA, van Hove RP. No clinical difference between TiN-coated versus uncoated cementless CoCrMo mobile-bearing total knee arthroplasty; 10-year follow-up of a randomized controlled trial. *Knee Surgery, Sports Traumatology, Arthroscopy* 2021;29(3):750-6.