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## Chapter

# Complications after Total Knee Arthroplasty: Stiffness, Periprosthetic Joint Infection, and Periprosthetic Fracture

*Atthakorn Jarusriwanna and Chaturong Pornrattanamaneewong*

## Abstract

Total knee arthroplasty (TKA) is one of the most successful surgical procedures with effective treatment in patients suffering from end-stage knee osteoarthritis. The goal of the operation is to improve pain, correct the deformity, and increase function. However, complications after surgery are the important factors related to dissatisfied TKA. Stiffness, periprosthetic joint infection (PJI), and periprosthetic fracture are among the most common complications following TKA and usually raise issues as concern points for both patients and the surgeons. Each complication needs precise assessment and specific care to prevent further serious issues. In this chapter, the authors will focus and describe all of these three frequent complications in details from their definition to management.

**Keywords:** total knee arthroplasty, complications, stiffness, periprosthetic joint infection, periprosthetic fracture

## 1. Introduction

There is approximately 20 percent of the patients with dissatisfaction following TKA whether pain or having postoperative problems [1, 2]. In the previous literatures, reporting of complications and adverse events after TKA was not standardized with several definitions being proposed. Healy et al. published a list of 22 TKA complications and their standardized definitions including these three common problems, which were endorsed by The Knee Society to improve quality measurement and consistent with ICD-9 codes [3]. Stiff TKA could produce pain and diminish functional ability, whereas PJI and periprosthetic fracture might cause severe morbidity. Early detection and appropriate management are the key success to resolve these problems which would enhance patient's outcome and improve satisfaction.

## 2. Stiffness

Normal knee range of motion (ROM) ranges from 0 to 140 degrees, while achievement of postoperative ROM from 0 to 110 degrees can be defined as success

TKA [4]. In general, a minimum of 90 degrees of knee flexion is required for functional recovery in daily activities, as 83 degrees of knee flexion is required for going up and down stairs and 93 degrees for sitting, which were demonstrated by a biomechanical study [5, 6]. Stiffness after TKA has variable incidence, ranging from 1.3 to 5.3 percent, but some literature proposed up to 60 percent of patients who suffered from stiff TKA [7]. These variables may cause by a variety of definitions as there was absolutely no consensus on degrees of knee flexion limitation defined as stiff TKA. The standardized definition by the TKA Complications Workgroup of The Knee Society described that limitation of ROM as reported by the patient with physical examination showed extension restriction to 15 degrees short of full extension or flexion less than 90 degrees were defined as stiffness. However, this definition could not be applicable if the preoperative arc of motion is less than 75 degrees [3].

## **2.1 Factors**

The factors or etiologies related to stiff TKA could be categorized into three phases: preoperative, intraoperative, and postoperative periods [8]. Each period also has its specified causes and different management. Surgeons should evaluate the patients carefully for the proper treatment.

### *2.1.1 Preoperative period*

Preoperative ROM limitation is the most important risk factor for postoperative stiffness [4, 9]. Patients with a greater degree of preoperative ROM had superior postoperative ROM and functional scores, with less complications. Only 71.4 percent of patients with preoperative ROM less than 90 degrees would achieve postoperative ROM at least 90 degrees, while more than 90 percent of patients with preoperative ROM greater than 90 degrees could perform ROM more than 90 degrees postoperatively [10]. Lee et al. demonstrated that 33 percent of patients with preoperative ROM less than 50 degrees developed either superficial or deep infection, as well as skin necrosis after the operation, whereas only 13 percent of patients with preoperative ROM between 50 and 90 degrees suffered from these complications [11]. The cause of stiffness before surgery is also one of the considerable factors for postoperative stiffness. The same study by Lee et al. showed patients with osteoarthritis or rheumatoid arthritis had greater postoperative ROM than patients with prior infectious arthritis or traumatic arthritis significantly [11]. Patients with younger age, absence of diabetes mellitus, and lower preoperative walking limitations were found to be the additional predictors with better postoperative ROM [12]. Moreover, obesity might be another factor influencing postoperative ROM. Järvenpää et al. proposed patients with body mass index (BMI) greater than 30 kg/m<sup>2</sup> had poorer postoperative ROM at 1-year follow-up approximately 6 degrees than patients with less BMI [13].

### *2.1.2 Intraoperative period*

At the time of surgery, technical errors during the bone cut, soft tissue procedure, and implantation, which relate to an imbalance in flexion and extension gaps, are the most frequent causes of postoperative stiffness. All of these conditions may result in limitation of motion both flexion and extension after TKA (**Table 1**) [4, 9].

Bone cut	Soft tissue procedure	Implantation
<ul style="list-style-type: none"> <li>• Insufficient posterior tibial slope or creation of anterior tibial slope</li> <li>• Inaccurate joint line level which alters the joint line and the patella, either patella alta or patella baja</li> <li>• Inadequate osteophytes resection</li> <li>• Insufficient bone cut, either proximal tibia or distal femur, especially the posterior condyle</li> </ul>	<ul style="list-style-type: none"> <li>• Inappropriate tension of posterior cruciate ligament (in case of cruciate-retaining prosthesis)</li> <li>• Inadequate soft tissue release, especially deep medial collateral ligament (MCL) for varus knee and iliotibial band for valgus knee</li> </ul>	<ul style="list-style-type: none"> <li>• Improper size of the prosthesis, especially incorrect choice of larger tibial insert</li> <li>• Malposition of the femoral component, either excessive hyperflexion or hyperextension</li> <li>• Malrotation of the prosthesis may also cause the problem of patellofemoral kinematics</li> </ul>

**Table 1.**  
 The intraoperative conditions which result in stiff TKA.

### 2.1.3 Postoperative period

There are several factors causing stiffness following TKA in this period, including inadequate rehabilitation and poor patient motivation, deep infection, arthrofibrosis, complex regional pain syndrome (CRPS), associated stiffness or pain derived from the adjacent joints or spine that alters knee motion, and heterotopic ossification (HO) [14]. Adequate postoperative pain management is essential in improving functional recovery and achieving rehabilitation protocol, especially knee motion enhancement [15]. Deep infection or PJI is one of the conditions leading to difficulty in ROM with chronic dull pain. It should be considered, especially in patients who developed stiffness after achieving adequate ROM [4, 14]. The details of this condition are described later in this chapter.

Arthrofibrosis after TKA is the most common cause of stiffness with an incidence ranging from 1.2 to 17 percent [9]. The etiology is multifactorial and the exact pathophysiology is unclear. Patients with poor preoperative ROM, higher complexity surgery, and a history of previous knee surgery increase the risk of excessive fibrous tissue formation after TKA. The theory of developing arthrofibrosis is disruption of cytokines and growth factors signaling cell growth, differentiation, and death, resulting in uncontrolled proliferation of fibroconnective tissue [16]. The histology is characterized by metaplasia of calcified tissue, myofibroblasts, and excessive fibrosis, with the increasing number of macrophages and lymphocytes in the periarticular tissue [17, 18]. The clinical manifestation is broad spectrum, from a localized lesion to a generalized involvement of the entire joint, and results in the formation of extensive extra-articular fibrous tissue.

Recently, there is no gold standard for diagnosis of arthrofibrosis, and also no effective method to prevent the idiopathic arthrofibrosis after TKA, apart from patient education and early mobilization [4].

## 2.2 Treatment

Initial evaluation of stiff TKA to assess the causes is necessary before management. A correct diagnosis leads to correct treatment. The evaluation should review back to the preoperative status of the patient, especially the risk factors mentioned above. The radiological examination should perform in case of suspicious mechanical problems from surgical errors of bone cut and implantation. Do not hesitate to work up for PJI if infection or wound-related complications that predispose the patient to infection are suspected [4, 9].

There are various treatment options for stiff TKA: manipulation under anesthesia (MUA), arthroscopic arthrolysis, open arthrolysis, and revision surgery [4].

### *2.2.1 Manipulation under anesthesia (MUA)*

The purpose of MUA is to break immature adhesions within the knee in patients who disadvantage of self-training or regular rehabilitation programs and accelerate the initial rehabilitation process [19]. This procedure should be performed within 6–8 weeks after initial TKA before the development of mature adhesions which increases the likelihood of complications after MUA, especially periprosthetic fractures or rupture of the extensor mechanism [20]. Aggressive rehabilitation is necessary to prevent further and recurrent stiffness. A systematic review by Fitzsimmons et al. showed a mean gain in knee motion from 30 to 47 degrees after MUA [7].

### *2.2.2 Arthroscopic arthrolysis*

Arthroscopic arthrolysis is a minimal invasive surgery that resects fibrosis directly in the suprapatellar pouch, medial and lateral gutters, and also in the intercondylar groove [4]. The indication of this operative procedure is painless, stiff TKA after non-progression of conservative treatment for 3 months. Disadvantage is inadequate arthrolysis because of poor access to the posterior structure and the area above the suprapatellar pouch [9]. A systematic review demonstrated improvement of overall ROM between 18.5 and 60 degrees, which also achieved 30.8–42 degrees even performing arthroscopic arthrolysis after 1 year of index TKA [7].

### *2.2.3 Open arthrolysis*

Open lysis of adhesions is recommended in case of severe ROM limitation which impedes the use of arthroscope without component malposition and after the failure of conservative treatment. This operative procedure can provide a broad assessment of the knee joint and fibrosis resection should be performed meticulously. However, exposure to the joint may be difficult from adhesions and need further operative technique, for example, tibial tubercle osteotomy, quadriceps snip, or VY-plasty [4, 9]. A systematic review by Fitzsimmons et al. showed an average increasing of ROM between 19 and 31 degrees after open arthrolysis [7].

### *2.2.4 Revision surgery*

This is the final treatment option reserved for stiffness from surgical errors that need to be corrected. Accurate analysis of the errors is required for planning the revision correctly to meet the patient's satisfaction [4].

## **3. Periprosthetic joint infection (PJI)**

PJI is a serious complication and is considered one of the most common causes of revision surgery following the failure of primary TKA [21]. The incidence of PJI after primary knee replacement is ranging from 0.85 to 2.2 percent [22], with a higher rate up to 9 percent in revision cases [23]. Despite a small incidence of infection following TKA, the trend of revision due to PJI was rising by 2.5-fold in the past decade [22].

This problem illustrates an increasing and substantial treatment burden to both orthopedic surgeons and the patients, as well as the health service system.

A systematic review and meta-analysis by Kunutsor et al. showed patients with smoking, BMI >30 kg/m<sup>2</sup>, diabetes, depression, steroid use, previous joint surgery, and frailty were the significant risk factors associated with the long-term developing PJI [24]. A study by Rosteijs et al. demonstrated the most common pathogen found in PJI after TKA was methicillin-susceptible *Staphylococcus aureus* (MSSA) which occurred in 28.2 percent of patients, followed by coagulase-negative *Staphylococcus* (CoNS), methicillin-resistant *Staphylococcus epidermidis* (MRSE), *Streptococcus*, ampicillin-susceptible *Enterococcus faecalis*, and methicillin-resistant *Staphylococcus aureus* (MRSA) with the frequency of 16.4, 13.2, 9.1, 7.1, and 6.6 percent, respectively. However, up to 17.8 percent of patients could not identify any pathogens [25].

### 3.1 Diagnosis

Recently, there is no gold standard for the diagnosis of PJI [21]. The Musculoskeletal Infection Society (MSIS) and the Infectious Diseases Society of America (IDSA) have previously developed criteria to standardize the definition of PJI in 2011 and 2013 [26, 27], together with an International Consensus Meeting on PJI in 2013 [28]. The latest consensus in 2018 proposed a new scoring-based definition for PJI after emerging of new diagnostic tests. Two positive cultures of the same organism or the presence of a sinus tract were considered as major criteria and a definite diagnosis of PJI. The minor criteria consisted of laboratory tests either serum or synovial fluid which were weighted differently. An elevated serum C-reactive protein (CRP) or D-dimer received 2 points, whereas an elevation of erythrocyte sedimentation rate (ESR) weighted 1 point. Furthermore, an elevated synovial white blood cell (WBC) count or leukocyte esterase (LE) was considered 3 points. The other diagnostic tests for synovial fluid were a positive alpha-defensin, an elevated synovial polymorphonuclear (PMN) percentage, and synovial CRP which took 3, 2, and 1 point, respectively. Patients with a total score of equal or greater than 6 were suggested infected, while a score between 2 and 5 was classified as inconclusive and required further intraoperative diagnostic score to fulfill the definition, and a score of 0 to 1 was defined as no infection.

The intraoperative diagnostic score consisted of positive histology, purulence, and a single positive culture which scored 3, 3, and 2 points, respectively. In combination with the inconclusive preoperative diagnostic score, patients with an overall score of equal or greater than 6 were considered infected, whereas a score between 4 and 5 was inconclusive and need further molecular findings, and a score of 3 or less was defined as aseptic (**Table 2**). The threshold of each laboratory test is detailed in **Table 3**. The sensitivity and specificity of this new scoring system are 97.7 and 99.5 percent, respectively, which is higher sensitivity than the previous diagnostic criteria [29].

### 3.2 Treatment

Management of PJI includes surgical intervention and medical treatment, especially antibiotics therapy, with the goals of eradicating the infection, minimizing pain by restoring the function of the infected joint before performing the revision arthroplasty, as well as reducing morbidity and mortality of the patients [30]. Tsukayama et al. classified characteristics of infection after TKA into four types with the guidance of surgical options among these scenarios (**Table 4**) [31].

Major criteria (at least one of the following)		Decision
1. Two positive cultures of the same organism		Infected
2. Sinus tract with evidence of communication to the joint or visualization of the prosthesis		
Minor criteria (Preoperative diagnosis)	Score	Decision
Serum		
1. Elevated CRP or D-dimer	2	≥6 Infected 2-5 Inconclusive (possibly infected)* 0-1 Not infected
2. Elevated ESR	1	
Synovial		
1. Elevated synovial WBC count or LE	3	≥6 Infected 2-5 Inconclusive (possibly infected)* 0-1 Not infected
2. Positive alpha-defensin	3	
3. Elevated synovial PMN percentage	2	
4. Elevated synovial CRP	1	
Intraoperative diagnosis	Score	Decision
Inconclusive preoperative score* or dry tap with		
1. Positive histology	3	≥6 Infected 4-5 Inconclusive ≤3 Not infected
2. Positive purulence	3	
3. Single positive culture	2	

*Modified from Parvizi et al. The 2018 Definition of Periprosthetic Hip and Knee Infection: An Evidence-Based and Validated Criteria. J Arthroplasty. 2018;33(5):1309-14.e2.*

**Table 2.**  
The 2018 International Consensus Meeting on Musculoskeletal Infection scoring-based definition for PJI.

Laboratory test	Acute (<90 days)	Chronic (>90 days)
1. Serum CRP (mg/L)	100	10
2. Serum D-dimer (ng/mL)	860	860
3. Serum ESR (mm/h)	—	30
4. Synovial WBC count (cells/μL)	10,000	3,000
5. Synovial alpha-defensin (signal-to-cutoff ratio)	1	1
6. Synovial PMN (%)	90	80
7. Synovial CRP (mg/L)	6.9	6.9

*Modified from Parvizi et al. The 2018 Definition of Periprosthetic Hip and Knee Infection: An Evidence-Based and Validated Criteria. J Arthroplasty. 2018;33(5):1309-14.e2.*

**Table 3.**  
The threshold of laboratory test of the minor criteria.

#### 4. Periprosthetic fracture

Periprosthetic fracture after TKA is found increasingly in recent years due to a large number of performed TKAs and growing of geriatric population. This serious complication is impact to the quality of life and functional recovery of the patients, which is recognized to develop high morbidity and mortality [32]. The incidence

Type and definition	Characteristics	Treatment options
I: Positive intraoperative culture	<ul style="list-style-type: none"> <li>• A positive culture of an intraoperative specimen during a revision arthroplasty for aseptic loosening</li> </ul>	<ul style="list-style-type: none"> <li>• Antibiotics alone, without further operation</li> </ul>
II: Early postoperative infection		
<ul style="list-style-type: none"> <li>• Superficial</li> </ul>	<ul style="list-style-type: none"> <li>• Occurs within 1 month after joint replacement</li> <li>• Local inflammation of acute onset</li> <li>• No sinus tract</li> <li>• No extension through capsule</li> </ul>	<ul style="list-style-type: none"> <li>• Cultures of the tissues or drainage fluid</li> <li>• Débridement of the soft tissue</li> <li>• Wound closure/antibiotic beads (remove after 2 weeks)</li> <li>• 2–6 weeks of antibiotic therapy</li> </ul>
<ul style="list-style-type: none"> <li>• Deep</li> </ul>	<ul style="list-style-type: none"> <li>• Occurs within 1 month after joint replacement</li> <li>• Local inflammation of acute onset</li> <li>• No sinus tract</li> <li>• Extension through capsule</li> </ul>	<ul style="list-style-type: none"> <li>• Cultures of the tissues or drainage fluid</li> <li>• Arthrotomy, synovectomy, and débridement of all infected soft tissue</li> <li>• Exchange of polyethylene insert</li> <li>• Wound closure/antibiotic beads (remove after 2 weeks)</li> <li>• 4–6 weeks of antibiotic therapy</li> </ul>
III: Acute hematogenous	<ul style="list-style-type: none"> <li>• Occurs more than 1 month after joint replacement</li> <li>• Local inflammation of acute onset</li> <li>• No sinus tract</li> <li>• Extension through capsule</li> <li>• Represents hematogenous seeding of the joint from another primary site of infection</li> </ul>	<ul style="list-style-type: none"> <li>• Cultures of the tissues or drainage fluid</li> <li>• Arthrotomy, synovectomy, and débridement of all infected soft tissue</li> <li>• Exchange of polyethylene insert</li> <li>• Wound closure/antibiotic beads (remove after 2 weeks)</li> <li>• 6 weeks of antibiotic therapy</li> </ul>
IV: Late chronic	<ul style="list-style-type: none"> <li>• Occurs more than 1 month after joint replacement</li> <li>• Insidious onset, usually no fever or leukocytosis</li> <li>• Sinus tract may be observed</li> <li>• Extension through capsule</li> </ul>	<ul style="list-style-type: none"> <li>• Cultures of the tissues or drainage fluid</li> <li>• Débridement and removal of all prosthetic components and bone cement</li> <li>• Applying an antibiotic cement spacer</li> <li>• 6 weeks of antibiotic therapy</li> </ul>

**Table 4.**  
*Tsukayama classification and treatment options.*

of fracture following TKA varies from 0.3 to 5.5 percent in primary knee replacement and has been reported as high as 30 percent in revision knee surgery [33, 34]. The most common site of fracture is a supracondylar area of the distal femur which occurs ranging from 0.3 to 2.5 percent [32, 35], followed by patellar periprosthetic fracture, especially in the resurfaced patella, with an incidence around 0.68 percent. However, the true incidence of this type of fracture may be obscured from undetected and asymptomatic patients [36]. The least common pattern is a proximal tibial fracture which affected approximately 0.3 to 0.5 percent [37]. Most frequently, periprosthetic fracture results from low-energy trauma, and osteoporosis is considered a significant predictor of fracture risk [38]. The other predisposing factors are any causes that affected bone quality, for example, prolonged corticosteroid



use, inflammatory joint diseases, especially rheumatoid arthritis, and patients with neurological and musculoskeletal problems, which have a high risk of falls [32, 35]. Iatrogenic causes from surgical procedure including anterior femoral notching, or alteration of anterior femoral cortex during bone preparation of distal femur is theorized to be an association of supracondylar femoral fracture after TKA [35]. A biomechanical study by Lesh et al. revealed a reduction of torsional strength and bending strength of the distal femur by 39.2 and 18 percent, respectively, after the full-thickness cortical defect was created [39]. However, the clinical outcome is still controversial [32, 40, 41]. The risk factors of periprosthetic tibial fractures in TKA are the use of long tibial stems, cementless press-fit fixation, malalignment of tibial component, and previous osteotomy of the tibia [37]. All other predisposing factors are detailed in **Table 5** [42].

Medical factors	Surgical factors		
	Femur	Tibia	Patella
<ul style="list-style-type: none"> <li>• Osteoporosis</li> <li>• Prolonged corticosteroid use</li> <li>• Inflammatory joint diseases e.g., rheumatoid arthritis</li> <li>• Neurological and musculoskeletal problems e.g., epilepsy, parkinsonism, myasthenia gravis, poliomyelitis, cerebral palsy</li> </ul>	<ul style="list-style-type: none"> <li>• Anterior femoral notching</li> <li>• Component malposition</li> <li>• Poorly reamed bone</li> <li>• Stress shielding</li> <li>• Box cut for posterior stabilized (PS) implants</li> </ul>	<ul style="list-style-type: none"> <li>• Use of long tibial stems</li> <li>• Cementless press-fit fixation</li> <li>• Intramedullary referencing</li> <li>• Malalignment</li> <li>• Osteolysis</li> <li>• Sclerosing subchondral bone</li> <li>• Tibial tubercle osteotomy</li> </ul>	<ul style="list-style-type: none"> <li>• Excessive bony resection</li> <li>• Central peg</li> <li>• Press-fit implants</li> <li>• Lateral release</li> <li>• Fat pad excision</li> <li>• Maltracking</li> <li>• Cement heat necrosis</li> </ul>

**Table 5.** Predisposing factors associated with periprosthetic fractures after TKA.

## 4.1 Classification

### 4.1.1 Femur

There were several classification systems described for supracondylar periprosthetic fracture of femur. Rorabeck et al. developed a classification that described fracture configuration and integrity of prosthesis to guide appropriate management of each fracture pattern. The key factors considered in the classification were the fracture displacement and the prosthesis stability [43, 44]. This classification later was widely known as “Lewis and Rorabeck classification” (**Table 6**).

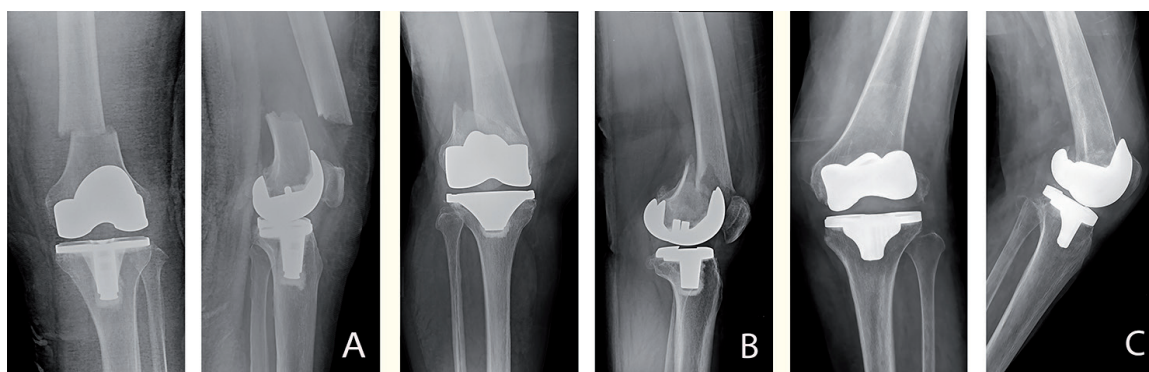
The Lewis and Rorabeck classification recommended nonoperative treatment for type I classification [44]. However, Su et al. suggested surgical management in any type of fracture because of the high complication rate and further displacement in case of conservative treatment. An alternative classification was developed and proposed to characterize the fracture line in relation to the component for help in choosing among surgical options (**Figure 1; Table 7**) [45].

Type	Characteristics
I	Undisplaced fracture; prosthesis intact
II	Displaced fracture; prosthesis intact
III	Displaced or undisplaced fracture; prosthesis loosening or failing e.g., significant instability or polyethylene wear

**Table 6.**  
*Lewis and Rorabeck classification.*

Type	Characteristics
I	Fractures are proximal to the femoral component
II	Fractures originate at the proximal end of the component and extend proximally
III	Any part of the fracture line is distal to the upper edge of the component's anterior flange

**Table 7.**  
*Su classification.*



**Figure 1.**  
*Anteroposterior and lateral radiographs showing periprosthetic fracture of TKA (A) Su classification type I; Lewis and Rorabeck classification type II (B) Su classification type II; Lewis and Rorabeck classification type II (C) Su classification type III; Lewis and Rorabeck classification type III.*

#### 4.1.2 Tibia

The Mayo classification described by Felix et al. (also known as Felix classification) is widely recognized to assess periprosthetic tibial fractures following TKA [46]. Fractures are classified into four types based on location and proximity to the prosthesis and each type is subcategorized by stability and whether the fracture occurred intraoperatively or postoperatively. The details are described in **Table 8**.

#### 4.1.3 Patella

The widely used classification for periprosthetic patellar fractures is the classification proposed by Goldberg et al. which is characterized by fracture configuration, stability of patellar component, and integrity of extensor mechanism [47]. The newer classification described by Ortiguera and Berry focused similarly on the stability of patellar components and integrity of extensor mechanism but differently on the quality of residual bone stock (**Tables 9 and 10**) [36].

Type	Characteristics
I	Fractures are located at the tibial plateau
II	Fractures occur inferior to the tibial plateau adjacent to the prosthetic stem
III	Fractures occur distal to the tibial stem
IV	Fractures involve the tibial tubercle
Additional subtype	Characteristics
A	A fracture with a stable prosthesis on radiographs
B	Fractures with radiographic evidence of component loosening
C	Intraoperative fractures

**Table 8.**  
*Mayo (Felix) classification.*

Type	Characteristics
I	Fractures are located in the periphery of the patella and do not involve the patellar component and the extensor mechanism
II	Fractures disrupt the implant-bone composite or the extensor mechanism
III	Fractures involve the inferior pole of the patella
• IIIA	• With ruptured patellar ligament
• IIIB	• Intact patellar ligament
IV	Patellar fractures accompanied by patellofemoral dislocation

**Table 9.**  
*Goldberg classification.*

Type	Characteristics
I	A stable implant and intact extensor mechanism
II	A stable implant with disruption of the extensor mechanism
III	Loose patellar component
• IIIA	• With reasonable bone stock
• IIIB	• With poor bone stock (<10 mm thickness or marked comminution)

**Table 10.**  
*Ortiguera and Berry classification.*

## 4.2 Treatment

Fracture treatment options in each component are related on their classified types. For supracondylar femoral fracture, Lewis and Rorabeck classification recommended nonsurgical treatment in type I, whereas treatment options either closed reduction and fixation with an intramedullary nail or open reduction and internal fixation with a plate could be performed in type II. Type III fracture requires revision of the prosthesis using a long stem or structural allograft [44]. Su et al. suggested reduction with antegrade or retrograde intramedullary nail, or sometimes a fixed-angle device for Su classification type I fracture. Su classification

type II requires management with either a fixed-angle device or retrograde supracondylar nail, and type III fracture may be managed with either a fixed-angle device or revision arthroplasty with a stemmed femoral component. However, if loosening is identified in any classification types, revision TKA with a femoral stem is recommended [45].

Felix et al. proposed a treatment algorithm for periprosthetic tibial fractures related to their classification. For type IA, nondisplaced IIA, and IIIA fracture, nonoperative treatment with protected weight-bearing is required. If displacement is observed in type IIA and IIIA fracture, closed reduction with casting or open reduction with internal fixation is recommended. Any loosening types (IB, IIB, and IIIB) should be treated with revision arthroplasty. In case of intraoperative fracture (subcategory C), bracing with protected weight-bearing can be treated in any type if the fracture is stable and nondisplaced. However, in unstable fracture pattern or displaced fracture, further surgical management is required. Type IC fracture may be treated by screw fixation and/or a long-stemmed tibial prosthesis to bypass the fracture site. Type IIC fracture can be managed with bone grafting at the cortical defect and bypassing the fracture site with a long tibial stem. Type IIIC fracture can be treated with either closed reduction and casting or open reduction with internal fixation [46].

For treatment of patellar periprosthetic fracture, Ortiguera and Berry suggested nonoperative treatment for type I fracture. If patients developed extensor mechanism disruption with a well-fixed implant (type II), open reduction with internal fixation of the displaced fragment, or alternatively, patellectomy with advancement and repair of the extensor mechanism is recommended. Operative treatment for type IIIA fracture required revision of the patellar component or component resection with patelloplasty, whereas implant removal with patellectomy is recommended for type IIIB fracture [36].

In the elderly, physiologic changes of bone, especially a high rate of bone resorption, result in diminishing bone mass and strength [48]. Osteoporosis workup and treatment are necessary in addition to fracture management in patients with periprosthetic fracture after TKA.

## 5. Conclusions

This chapter concludes with the principle, classification, and management of three typical conditions, which are considered serious and unsatisfied results after TKA. Causes of stiff TKA divide into three different periods and each period needs specific management, but the most important risk factor for postoperative stiffness is the limitation of preoperative ROM. Patient education and motivation either before or after surgery are necessary to prevent further problems and meet the patient's satisfaction. An exploration of new diagnostic tests enhances the accuracy of PJI diagnosis and the latest scoring-based definition achieved more sensitivity than the previous criteria. Major criteria of two positive cultures of a similar pathogen or the presence of a sinus tract to the knee joint can diagnose PJI. If a diagnosis has not been made, the further investigation of minor criteria, including serum and synovial laboratory tests, would have been collected preoperatively. An inconclusive diagnosis from the minor criteria needs furthermore investigation from intraoperative findings. Periprosthetic fractures are principally classified from the anatomy of fracture site. The most common is a femoral supracondylar fracture and the surgical

options depend on fracture location and configuration. Finally, the goal of treatment among these three conditions is return to ambulation with a well-function knee prosthesis.

### **Conflict of interest**

The authors declare no conflict of interest.

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