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**ISOKINETIC STRENGTH OF KNEE EXTENSOR MUSCLE  
AND KNEE RANGE OF MOTION IN PATIENTS AFTER TOTAL  
KNEE ARTHROPLASTY**

**Master thesis**  
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# CONTENTS

<b>ABBREVIATIONS</b> .....	4
<b>LIST OF PUBLICATIONS</b> .....	5
<b>1. INTRODUCTION</b> .....	7
<b>2. REVIEW OF LITERATURE</b> .....	8
2.1. Osteoarthritis of the knee .....	8
2.1.1. Possibility of osteoarthritis treatment .....	9
2.2. Knee extensor strength deficit in patients with knee osteoarthritis .....	10
2.2.1. Age-related changes in muscle function .....	10
2.2.2. Quadriceps muscle strength in knee osteoarthritis .....	11
2.3. Total knee arthroplasty in knee osteoarthritis .....	11
2.3.1. Goals and indications for total knee arthroplasty .....	12
2.3.2. Types of prostheses used for total knee arthroplasty .....	12
2.3.4. Complications after total knee arthroplasty .....	13
2.3.5. Postoperative management after total knee arthroplasty .....	14
2.4. Muscle strength testing .....	14
<b>3. OBJECTIVES OF THE STUDY</b> .....	17
<b>4. MATERIALS AND METHODS</b> .....	18
4.1. Subjects .....	18
4.2. Study design.....	19
4.3. Methods .....	20
4.3.1. Isokinetic Dynamometry.....	20
4.3.2. Knee range of motion .....	21
4.3.3. Pain measurement .....	22
4.3.4. Statistical evaluation of the data .....	23
<b>5. RESULTS</b> .....	24
5.1. Isokinetic strength of knee extensor muscles .....	24
5.1.1. Unilateral TKA group.....	24
5.1.2. Bilateral TKA group .....	34
5.2. Knee ROM.....	36
5.2.1. Unilateral TKA group.....	36
5.2.2. Bilateral TKA group .....	36
5.3. Pain .....	38

5.3.1. Unilateral TKA group.....	38
5.3.2. Bilateral TKA group.....	38
<b>6. DISCUSSION</b> .....	<b>40</b>
6.1. Isokinetic contraction of knee extensor muscles .....	40
6.2. Knee ROM.....	47
6.3. Knee pain .....	48
<b>CONCLUSIONS</b> .....	<b>49</b>
<b>REFERENCES</b> .....	<b>50</b>
<b>SUMMARY IN ESTONIAN</b> .....	<b>57</b>
<b>ACKNOWLEDGEMENTS</b> .....	<b>59</b>

## ABBREVIATIONS

AAOS	– American Academy of Orthopaedic Surgeons
BMI	– body mass index
BL	– bilateral
BM	– body mass
KE	– knee extensor muscles
MHC	– myosin heavy chain
MVC	– maximal voluntary contraction
OA	– osteoarthritis
RFD	– rate of peak torque development (dF/dt)
ROM	– range of motion
TKA	– total knee arthroplasty
UL	– unilateral

## LIST OF PUBLICATIONS

1. **Djuško V., Gapeyeva H., Buht N., Mäeots T., Pääsuke M., Ereline J., Peterson K., Haviko T.** (2005) Pre-and postoperative changes of isokinetic strength of knee extensor muscles and knee range of motion in patients with total knee arthroplasty. *Acta Kinesiologiae Universitatis Tartuensis*, 10: 48-62.
2. **Gapeyeva H., Pääsuke M., Ereline J., Djuško V., Buht N., Mäeots T., Haviko T.** (2004) Quadriceps femoris strength in women with osteoarthritis before knee arthroplasty. Abstracts of 14th European Congress of Physical and Rehabilitation Medicine. Vienna, p. 56.
3. **Djuško V., Buht N., Mäeots T., Gapeyeva H., Haviko T., Pääsuke M.** (2004) Pre- ja postoperatiivsed muutused reie nelipealihase isokineetilise jõu ja põlveliigese liikuvuse näitajates ühepoolse hilise gonartroosiga patsientidel. Konverentsi ``*Teadus, sport ja meditsiin*`` kogumik. Kääriku, lk. 110
4. **Mäeots T., Gapeyeva H., Ereline J., Buht N., Djuško V., Haviko T., Pääsuke M.** (2004) Reie neliepealihase ja põlveliigese funktsionaalne seisund kahepoolse goanrtroosiga patsientidel enne ja pärast teist endoproteesimist. Konverentsi ``*Teadus, sport ja meditsiin*`` kogumik. Kääriku, lk. 122
5. **Gapeyeva H., Buht N., Mäeots T., Djuško V., Ereline J., Pääsuke M., Haviko T.** (2004) Isomeetiline jõud ja tahteline aktivatsioon reie nelipealihasel põlveliigese endoproteesimisel gonartroosiga patsientidel. Tartu Ülikooli Arstiteaduskonna aastakonverents, 6.-8. oktoober 2004, *Eesti Arst*, 9: 609
6. **Gapeyeva H., Pääsuke M., Ereline J., Djuško V., Buht N., Mäeots T., Haviko T., Maasalu K., Kolts I.** (2003). Effect of endoprosthesis on isokinetic strength of knee extensor muscles. In: H. Ring and N. Soroker (eds.) 2nd World Congress of the International Society of Physical and Rehabilitation Medicine. Bologna: Monduzzi Editore, pp. 281-284. [ISI Proceedings]

7. **Djuško V.** (2003) Reie nelipealihase isokineetilise jõu näitajad ühepoolse hilise gonartroosiga naispatsientidel enne põlveliigese endoproteesimist. Kehakultuuriteaduskonna üliõpilaste V teadusliku konverentsi teesid. Tartu, Tartu Ülikool, lk. 7.
  
8. **Gapeyeva H., Djuško V., Mäeots T., Buht N., Ereline J., Pääsuke M., Haviko T.** (2003) Reie nelipealihase funktsionaalsed omadused enne põlveliigese endoproteesimist naistel vanuses 52-79 aastat. VIII Russian National Congress ``*People and their health*``. Sankt-Petersburg (vene keeles).

# 1. INTRODUCTION

The knee is the most commonly affected weight-bearing joint in as many as one-third of people 63-94 years old. The function of the knee joint is closely linked to the integrity of joint structures, ligaments and the coordinated action of surrounding muscles (Aquino *et al*, 2002; Baker & McAlindon, 2000; Felicetti *et al*, 2004). In patients with knee osteoarthritis (OA), knee extensors strength deficit is a clinical feature that has been described previously (Fisher & Pendergast, 1997; Slemenda *et al*, 1997; Pap *et al*, 2004). Knee extensors strength decrease may result from the pain of osteoarthritis, however, some researchers have suggested that quadriceps strength deficit precedes the onset of knee OA, and is itself a risk factor for the development of knee OA (Slemenda *et al*, 1997).

In the early phase of knee OA, the most of patients can be managed by conservative means such as weight loss, exercises, use of a walking stick and anti-inflammatory analgesic drugs. The predominant indication for surgical treatment is persistent pain and serious disturbance of the activities of daily life against conservative treatment (Yasuda, 1997; Maurer *et al*, 1999).

Total knee arthroplasty (TKA) is one of the common operations performed for end-stage osteoarthritis. Today, about 300,000 total knee replacements are done annually in the United States, according to the American Academy of Orthopaedic Surgeons (2006).

Involvement of the knee extensor muscles group in knee osteoarthritis is receiving research interest. Numerous of publications concerned the peak torque, the concentric ratio flexors/extensors, with comparison between males and females, sport specialities, young and old people.

In the present study the strength and power deficit of knee extensor muscles and changes of knee joint range of motion in two groups of subjects was estimated before and after total knee arthroplasty. Our attention was focused on the knee extensor muscles because this muscle has the greatest strength decline in knee OA patients compared with the other lower extremity muscles. Results of the present study can use physiotherapists and other specialists in rehabilitation management of patients after joint replacement.

## 2. REVIEW OF LITERATURE

### 2.1. Osteoarthritis of the knee

Osteoarthritis (OA) is a primarily non-inflammatory, degenerative joint disease (McAlindon *et al*, 1992; Hurley, 1999; Gür *et al*, 2003; Kwiatkowski & Plominski, 2004; Gerwin *et al*, 2006). This condition usually occurs late in life, affecting large weight-bearing joints such as the knee and the hip. Traditionally, osteoarthritis was only associated with the elderly, but up to a third of the population over the age of 45 years complain of symptoms related to osteoarthritis (Gür *et al*, 2003; Wilson *et al*, 2006). Osteoarthritis affects over 14 million individuals and over 60% of adults over 50 years of age (Guccione *et al*, 1994; Lawrence *et al*, 1994; Maurer *et al*, 1999). It is particularly disabling when the knees are affected, because it limits the ability to walk, to rise from a chair, and to use stairs. Because 30 to 40% of persons over age of 60 years have knee OA, it is likely to contribute greatly to disability in the general population (Wilson *et al*, 2006; Hauselmann *et al*, 1996; Maurer *et al*, 1999; Hurley, 1997; Gür *et al*, 2003).

The World Health Organization has reported that knee OA is more prevalent in women than men (Murray & Lopez, 1997; Borrero *et al*, 2006). Physical disability arising from pain and loss of functional capacity reduces quality of life and increases the risk of further morbidity and mortality (Maurer *et al*, 1999).

According to the Kellgren-Lawrence scale there are four stages of OA (Link *et al*, 2003):

- **Grade 0**: normal knee joint, no loss of cartilage and no deformation;
- **Grade I**: some loss of articular cartilage, if severe loss of cartilage, joint space narrows, osteophytes may be seen;
- **Grade II**: bone hardening (sclerosis) and cysts, changes in bone density (whitening of bone on x-ray);
- **Grade III**: some deformations on edge of bone, rough edges, increased joint narrowing;
- **Grade IV**: complete loss of joint space, definite deformity of bone ends, changes in joint shape mean the bone contour has been altered.



Knee OA has many known endogenous and exogenous risk factors. The most important endogenous risk factors are (Kwiatkowski, 2004):

- age;
- gender;
- race;
- and inborn proneness.

The exogenous factors include (Kohatsu & Schurman, 1990; Kwiatkowski, 2004):

- obesity;
- joint overuse;
- and structure disturbances after trauma.

The risk factors may possibly accumulate (Chitnavis *et al*, 2000; Kwiatkowski, 2004). Knee OA is associated with a variety of pathophysiologic deficits, including joint instability, reduced joint range of motion, and disuse atrophy of the quadriceps muscle. The results of study of van der Esch *et al* (2005) supported the premise that biomechanical factors play a role in the degeneration of the osteoarthritic knee joint. Structural change particularly at the patellofemoral compartment, may change the biomechanical relation (O'Reilly *et al*, 2005).

### **2.1.1. Possibility of osteoarthritis treatment**

Modern medicine is able to offer numerous nonsurgical and surgical options of OA treatment (AAOS).

Nonsurgical options of OA treatment include (Pangano *et al*, 2005):

- using of orthotics;
- continued nonoperative therapy, including oral anti-inflammatory medications (NSAIDs) and intra-articular steroid injections and other minimally invasive procedures to help diminish the OA individual's pain and foster restoration of function.

Alleviation of pain and inhibition of inflammation are the primary goals of pharmacotherapy of osteoarthritis (OA). These therapeutic goals can almost always be accomplished by the use of analgesics and nonsteroidal anti-inflammatory drugs (NSAID) (Bradley *et al*, 1991). However, no clinical studies exist, which can positively confirm prevention, slowing down or reversal of any advanced joint cartilage destruction by any individual medication. Disease modifying therapy is still in its

infancy; discovery and development of novel therapeutic targets and agents are an extremely difficult task, currently challenging many pharmaceutical companies and academic institutions (Steinmeyer & Kontinen, 2006).

Surgical options of OA treatment include (Haspl, 2005):

- unicompartmental arthroplasty;
- high tibial osteotomy;
- autologous chondrocyte transplantation;
- hyaluronic acid injections;
- total knee arthroplasty (TKA).

## **2.2. Knee extensor strength deficit in patients with knee osteoarthritis**

### **2.2.1. Age-related changes in muscle function**

The marked decrease in skeletal muscle strength and size with aging is a multifactorial syndrome which may be attributable in part to:

- (a) biological changes of aging itself;
- (b) the accumulation of acute and chronic diseases;
- (c) the assumption of a sedentary life style,
- (d) and selective or generalized nutritional inadequacies (Fiatrone & Evans, 1993; Jones & Rose, 2004).

Maximal muscle strength is achieved in the second or third decade of life and then declines with age. Muscles of the upper extremities, such as handgrip muscles or elbow flexors and extensors, tend to change less with age than muscles of the lower extremities (Bazzucchi *et al*, 2004). Muscle strength decreases approximately 30% on average between the ages of 50 and 70 years, with even more dramatic strength losses after age of 80 (Lynch *et al*, 1999).

The ability to develop muscle power diminishes with age to an even greater extent than muscle strength (Porter, 2006). The ability of skeletal muscles to generate power is important for the performance of many daily activities, ranging from instrumental tasks (e.g., bathing, dressing, cooking, etc.) to recreational activities (Krivickas *et al*, 2001).

### **2.2.2. Quadriceps muscle strength in knee osteoarthritis**

Muscle has an integral role in the structure and function of joints. The quadriceps is the primary stabilizer of the knee, affording protection of the articular structures. In patients with knee osteoarthritis, knee extensor muscles strength decrease is a clinical feature that has been described previously (O'Reilly *et al*, 1997; Pap *et al*, 2004). Knee extensor muscle strength in older persons with knee OA may be reduced by up to one third compared with age-matched controls (Slemenda *et al*, 1997; Brandt *et al*, 1999). These strength declines are thought to primarily result from the atrophy of type IIB fibers, which are responsible for the rapid production of power (Maurer *et al*, 1999).

The motor and sensory functions of the muscle are intimately linked, and deficits in the sensorimotor functions of KE muscle have been identified and proposed as possible factors in the pathogenesis of knee OA or as consequences of the disease (Hurley, 1998). Several researchers suggest that the knee extensor muscles strength decrease is a primary risk factor for knee pain, disability, and progression of joint damage in persons with osteoarthritis of the knee (Mc Alindon *et al*, 1993; Slemenda *et al*, 1997; Maurer *et al*, 1999).

Several studies have shown that the degree of KE muscles strength decline correlates with the degree of knee pain and the degree of physical disability (Gür *et al*, 2003). In daily activities such as ascending and descending stairs, standing up from a chair, or sitting down in a chair, the knee extensor muscle and hamstring groups contract concentrically or eccentrically to control limb movement and to prevent joint overloading.

### **2.3. Total knee arthroplasty in knee osteoarthritis**

With an aging population and a larger percentage of individuals at all ages focused on a pain-free active life, osteoarthritis of the knee and its management has become a major economic concern in healthcare today. This attitude is the main impetus for the increasing demand for surgical intervention involving total joint arthroplasty (Maurer *et al*, 1999; Vijay *et al*, 2002).

Total knee arthroplasty is one of the most common operations performed for end-stage (III-IV stage according to Kellgren-Lawrence scale) OA in older individuals.

TKA provides gradual pain reduction and progressive improvement in the functional capacity and health-related quality of life (Walsh *et al*, 1998; Parent & Moffet, 2002; Stevens *et al*, 2003).

94-97% of patients can expect a good to excellent clinical result. This clinical result encompasses minimal to no pain, the ability to walk more than 1,5 km, increased range of motion, and patient satisfaction with the procedure. These results generally hold up 5- and 10-year follow-ups, with about 1% failure rate per year. Thus, one can expect about 90% of success with these procedures at 10 years (Dietz & Gekeler, 1981; Tankersley *et al*, 1997).

### **2.3.1. Goals and indications for total knee arthroplasty**

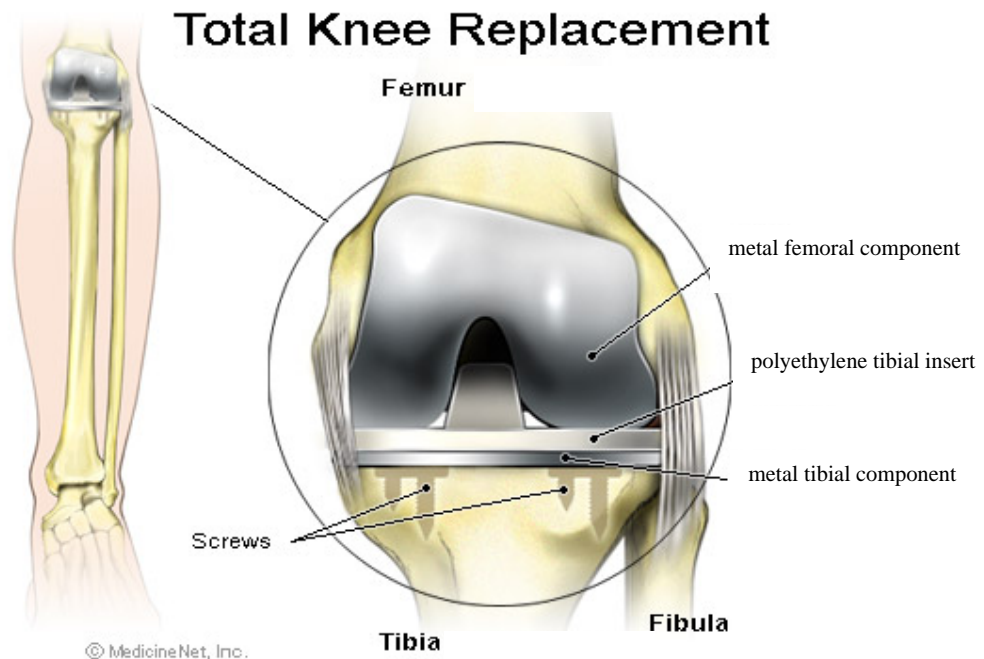
The main indication is to relieve pain caused by arthritis. Secondary goals are to correct deformity, and to restore function. More specifically, candidates for knee replacements have severe degenerative changes of their knee joint seen on radiographs and have failed multiple methods of nonoperative treatment to relieve their pain. These methods include anti-inflammatory medications, the use of a cane, decreased activity, loss of weight when indicated, as well as interarticular corticosteroid injections. These methods should be tried 3-6 months before a knee arthroplasty (Tankersley *et al*, 1997; Yasuda, 1997; Maurer *et al*, 1999).

The goals of knee arthroplasty are:

1. To restore a pain free joint
2. To restore range of motion
3. To allow function that approaches normal for a patient (Tankersley *et al*, 1997).

### **2.3.2. Types of prostheses used for total knee arthroplasty**

All prosthesis used in TKA has a metal femoral component, a metal tibial component, and a polyethylene tibial insert that is snapped into the tibial component. (**Figure 1**).



**Figure 1.** A total knee replacement scheme. <http://www.medicinenet.com>.

The types of prosthesis for TKA are the following:

- cruciate retaining, also known as cruciate-sparing;
- cruciate substituting, also known as cruciate-sacrificing or posterior-stabilized;
- and mobile-bearing, also known as rotating-platform (Haspl, 2005).

In this study a tricompartmental mobile-bearing knee endoprosthesis were used.

#### **2.3.4. Complications after total knee arthroplasty**

Most common complications after TKA are (AAOS): infection, deep venous thrombosis, aseptic loosening, patellofemoral maltracking. Patellofemoral maltracking is the most common complication following TKA. It can be avoided by paying careful attention during surgery to maintaining the normal Q-angle (the angle between the axis of the extensor mechanism and the axis of the patellar tendon). Anything that increases the Q-angle will increase the likelihood of maltracking.

Extensor mechanism complications are the most commonly reported reasons for revision surgery after TKA and are a frequent source of postoperative morbidity (Parker *et al*, 2003).

### **2.3.5. Postoperative management after total knee arthroplasty**

The main objectives of rehabilitative protocol are: improvement of the preoperative clinical state, prevention and management of the common postoperative problems and complications. Objectives require the improvement of the function of the operated knee (good articular excursion, muscular strengthening and recovery of ambulation and gait pattern), as well as the reduction of pain (Cademartiri & Soncini, 2004).

Usually, mobility training for bed, dressing, transfers and ambulation is started within 24 to 48 hours postoperatively, depending on surgical procedures and comorbidities. The acute care goals inherently are aimed at discharge, which is achieved when the patient is ambulatory, usually with a walker or cane. This occurs in 5 to 10 days, and discharge may be to the home or a rehabilitation unit (Brimer, 1999).

The transition to the intermediate postoperative phase of rehabilitation occurs after 2 to 3 weeks and continues for 4 to 12 weeks. The goals are functional independence in all activities of daily living. Ideally, knee range of motion of 0° to 5° of extension and 110° to 120° of flexion will be obtained. Therapeutic exercises should be focused on attaining optimal muscle strength and endurance, ambulation over a variety surfaces and distances, and safe static and dynamic balance (Brimer, 1999).

Weight-bearing status is one aspect of TKA rehabilitation that may need further research because of potential risk. Generally, partial to full weight bearing using a walker for assistance should be started within 1 to 2 postoperative days, depending on the orthopedist surgical assessment and procedure (Kauffman, 2000).

## **2.4. Muscle strength testing**

Muscle strength testing has been the most often applied approach in testing muscle function in general, as well as functional movement abilities (Mirkov *et al*, 2004). Voluntary measures of strength are affected by degree of effort. Previous study showed that in OA patients, as in other patient groups, effort may be influenced by pain and psychologic outlook (O'Reilly *et al*, 2005).

There are many different ways to measure muscle strength in the laboratory. Isometric evaluation of muscle behaviour, which measures a muscle's maximum

capacity to produce static force, has proceeded with different and often opposite results. One of the major limitations of such tests is that they are not specific to the performance of most human movements that require dynamic activation of musculature through a movement range. During isometric testing a muscle exerts force but does not change in length. Isometric contractions are common in activities such as yoga and stretching.

For dynamic tests, increasing the speed of movement significantly influences the ability of muscles to generate force; output is much reduced at high speed (flyswatters are very light weight to allow rapid swings). However, speed of movement has much less effect on eccentric test results (Enoka, 2001). When the goal of the testing is to make precise strength measurements, velocity of movement must be precisely controlled (because of the dramatic effect of muscle contraction speed on force production). The most commonly tested movement is knee extension, but many other muscular actions (e.g., hip flexion and abduction, shoulder flexion) can be measured with isokinetic dynamometer. (Svetlize, 1991). Knee extension is accomplished primarily by contraction of the quadriceps femoris muscles, which consist of the rectus femoris and vasti medialis, intermedius and lateralis muscles. Knee flexion is produced by the hamstring muscle group, which consists of the biceps femoris, semitendinosus, and semimembranosus muscles (Worrell *et al*, 1989). These muscle groups are tested in the seated position, however the alternative positions (lying) are also recommended for isokinetic exercises and assessment of the KE and flexor muscles. Regardless of age and activity, the bilateral relationships of the quadriceps and hamstring muscle groups tend to be within 5 to 10% of each other. Isokinetic strength of these muscle groups is also frequently reported relative to body weight.

Isokinetic dynamometry allows assessing the application of force through all of part of a joints range of motion. Isokinetic dynamometry enables the rapid and reliable quantification of force or torque (Perrin, 1993). An isokinetic contraction is a refinement of the controlled motion concept. The isokinetic contraction is dynamic, but the speed of the motion is held constant by a special device. In this way, resistance is in direct ratio to the varying force applied through the full course of a natural movement (Svetlize, 1991).

Different angular velocities are using during isokinetic testing procedure. In a case study, Chow (1999) tested female subjects at angular velocities ranging from 0,43 6,98 rad·sec<sup>-1</sup> (20-400°/s). A combination of knee torque and geometry of the knee

(obtained from knee radiographs) was used to determine the different knee joint forces. All knee joint forces were found to decrease with increasing isokinetic speed.

People involved in isokinetic testing of muscular performance should be aware that the peak torque occurs later in the range of motion with increasing angular velocity, especially when testing weak muscle groups. In high angular velocities this may become a problem since the limb may pass the optimal joint position for muscular performance, and the record peak torque may not represent the subjects maximal torque capacity (Kannus & Beynnon, 1993).

Isokinetic dynamometry has widespread applications in rehabilitation and training of muscle function and also in assessment of dynamic muscle function in both clinical research and sports environments (Baltzopoulos, 1995; Gleeson & Mercer, 1996). Multiple factors, including type of muscle fiber, size of the muscle, length and speed of the muscle at contraction, age and gender, affect the magnitude of strength generated (Gaines & Talbot, 1999).



### **3. OBJECTIVES OF THE STUDY**

The general objective of the study was to evaluate the knee extensor muscle function and knee active flexion range of motion in patients with knee OA scheduled for unilateral total knee arthroplasty (UL TKA) and in patients scheduled for bilateral total knee arthroplasty (BL TKA) before, 3, 6 and 12 months after surgery and compare the results of two groups of patients and to healthy age- and gender matched control subjects.

**More specifically the present study had the following aims:**

- 1) To study the changes in isokinetic PT and power output of knee extensor muscle of involved leg in patients with knee OA after TKA and to compare the data of two groups of patients and to control subjects.
- 2) To investigate the changes in isokinetic explosive force production (maximal rate of force development (MRFD) and rate of force development at 30° degrees (RFD<sub>30</sub>) and at 60° degrees of knee extension (RFD<sub>60</sub>)) of KE muscles of involved leg in patients with knee OA following TKA and to compare the data of two groups of patients and to controls.
- 3) To assess the recovery of knee range of motion in patients with knee OA after UL and BL TKA and to compare the data of two groups of subjects.
- 4) To evaluate the changes in knee pain in patients with knee OA after UL and BL TKA.

## 4. MATERIALS AND METHODS

### 4.1. Subjects

Twelve patients with idiopathic OA of knee joint volunteered in the study. Three groups of subjects were studied: subjects with knee OA who had undergone a primary UL TKA, subjects who had the UL TKA of one leg previously and was scheduled for a BL TKA and older adults without knee pathology as a control group (**Table 1.**).

The UL TKA group included 6 patients (3 males and 3 females, age ranged 48-74 years) who were scheduled to undergo unilateral tricompartmental TKA for idiopathic OA of the knee. The BL TKA group included 6 patients (3 males and 3 females, age ranged 49-74). Knee OA symptoms duration and time between operations in patients are represented in **Table 2.**

All prosthesis were implanted according to a standard procedure with a medial parapatellar approach and cemented insertion of the patellar, tibial and femoral components. The indications for arthroplasty were clinical OA of the knee in end stage. Patients reported pain and functional disability during activities of daily living prior to surgery. Subjects were recruited from the Department of Traumatology and Orthopaedics, University of Tartu, and referred by an orthopedic surgeon who diagnosed the OA. Potential subjects for the TKA group were excluded if they had evidence of: 1) musculoskeletal impairments, other than the TKA, that limited function in the lower extremity to be tested; 2) uncontrolled blood pressure; or 3) neurological disorders.

The control group comprised 6 age- and gender-matched volunteers (3 males, 3 females, age ranged 49-74 years). Controls were sedentary and did not exercise regularly. All subjects gave written informed consent that was approved by the Ethics Committee of the University of Tartu.

**Table 1.** Anthropometric characteristics and age of the subjects (mean±SE).

<b>SUBJECTS</b>	<b>N</b>	<b>Age (yr)</b>	<b>Height (cm)</b>	<b>Body mass (kg)</b>	<b>BMI (kg·m<sup>-2</sup>)</b>
<b>Unilateral TKA group</b>	6	58,7±4,0	166,0±0,1	78,1±10,1	28,0±2,2
<b>Bilateral TKA group</b>	6	57,9±4,2	165,0±0,1	90,1±4,2	33,1±1,3
<b>Controls</b>	6	58,0±4,2	165,0±0,1	73,6±5,7	27,0±1,5

**BMI** – body mass index.

**Table 2.** Symptoms duration in patients before operation.

<b>UL TKA group</b>	<b>symptoms duration before IK TKA (yr)</b>	<b>BL TKA group</b>	<b>symptoms duration before IK TKA (yr)</b>	<b>time between operations (yr)</b>
1. K. M. (f)	2	1. O. A. (f)	3	2
2. P. E. (f)	3	2. T. L. (f)	7	3
3. A. M. (f)	6	3. K. T. (f)	3	2
4. M. A. (m)	9	4. P. E. (m)	6	1
5. L. K. (m)	10	5. L. H. (m)	4	2
6. J. O. (m)	10	6. K. H. (m)	6	1
<b>mean</b>	<b>6.6</b>		<b>4.8</b>	<b>1.8</b>

UL – unilateral group; BL – bilateral group. IK – involved knee = scheduled for TKA knee joint; f- female; m- male.

**Notes:** for BL group involved knee- knee scheduled for TKA, uninvolved knee – knee with previous TKA.

## 4.2. Study design

All measurement were performed during period of 2002-2006 in Laboratory of Kinesiology and Biomechanics, University of Tartu at the day before TKA and also three, six and twelve months after surgery. Control group subjects were tested once.

Subjects were given instructions before collecting the data, and the testing using isokinetic dynamometer was demonstrated. This was allowed by a practice sessions to familiarize the subjects with procedures. Firstly tested leg in patients was the involved leg of patients (in bilateral group involved leg was leg scheduled for TKA). The determination of leg dominance in control group was based on a kicking preference. The uninvolved leg in patients was tested initially following the testing of involved leg. Involved leg of patients in UL and BL groups was leg with knee scheduled for TKA, and uninvolved leg in BL TKA group was leg with previously replaced knee.

### **4.3. Methods**

#### **4.3.1. Isokinetic Dynamometry**

All tests were performed on the computer controlled isokinetic dynamometer Cybex II (U.S.A.) (Picture 2.). Quadriceps strength was tested on a day before TKA and when subjects were not fatigued from prior physical activity. Subjects were positioned sitting with the hip and knee at 100 and 90° angles respectively, and were instructed to grip the handles of the seat during the testing. The thigh, pelvis, and trunk were stabilized with belts. An adjustable lever arm was attached to the subjects leg by a padded cuff just proximal to the lateral malleolus. The axis of rotation of the dynamometer arm was positioned just lateral to the lateral femoral epicondyle. Concentric isokinetic tests were used. Subjects did not have prior experience with the isokinetic dynamometer and were familiarized with testing procedures by performing 3 consecutive warm-up trials for each angular velocity, one of which was a maximal contraction.

The unilateral maximal voluntary isokinetic concentric strength (peak torque, PT), maximal rate of force development (RFD), rate of force development at 30° of knee extension (RFD<sub>30</sub>) and at 60° degrees of knee extension (RFD<sub>60</sub>) of the KE muscles (gravity-corrected values), and power output at maximal PT (N) were recorded using a modified Cybex II dynamometer at angular velocities of 2.09 rad·s<sup>-1</sup> and 3.14 rad·s<sup>-1</sup>. One trial with the highest PT value from three attempts and data of N from this trial were used for future analysis. The order of speed was designed from slower to faster. A one minute rest was allowed between each attempt. The same researcher conducted all tests, and the subjects were verbally encouraged to exert maximal efforts.



**Figure 2.** Isokinetic dynamometer.

#### **4.3.2. Knee range of motion**

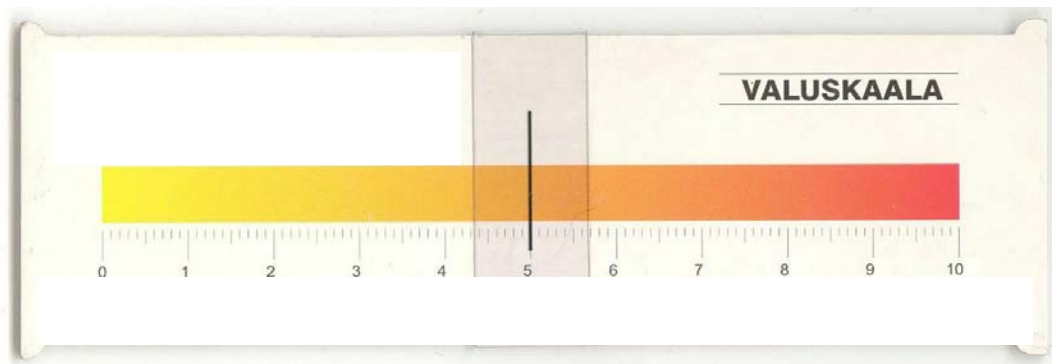
Knee active ROM was measured using Gollehon Extendable Goniometer (*Lafayette Instrument, USA*) (Picture 3.). All measurements were conducted before, three and six months after TKA. All measurements were conducted before, three and six months after TKA.



**Figure 3.** Measurement of knee active flexion ROM.

### 4.3.3. Pain measurement

A numeric rating scale (Picture 4.) (visual analogue scale) was used to quantify knee pain (Hassan *et al*, 2002). TKA subjects were asked to verbally rate the pain in and around the knee on a scale from 0 to 10, where 0 represented no pain and 10 represented the worst pain. All measurements were conducted before, three and six months after TKA.



**Figure 4.** Visual analogue scale.

#### **4.3.4. Statistical evaluation of the data**

Data were means and standard errors of mean (SE). Student's *t*-criterion was used to calculate significance of difference between parameters of OA knees and controls. One-way analysis of variance (ANOVA) followed by Bonferroni post hoc comparisons were used to test for differences between groups of patients before and after TKA. A level of  $p < 0.05$  was selected to indicate statistical significance.

## 5. RESULTS

### 5.1. Isokinetic strength of knee extensor muscles

#### 5.1.1. Unilateral TKA group

*Isokinetic strength of KE muscles* (PT) at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  (**Figure 5A**) of the involved leg in OA patients was lower than of uninvolvement leg prior to UL TKA, 3, 6 and 12 months after surgery. The difference was significant ( $p < 0.05$ ) 3 and 6 months after UL TKA. Also, the significant difference ( $p < 0.05$ ) in PT between KE muscles of the involved leg in patients and dominant leg of controls has been observed. Difference between the dominant and nondominant leg of controls was not statistically significant ( $p > 0.05$ ).

PT of KE muscles at angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  (**Figure 6A**) of the involved leg in OA patients was also lower than of uninvolvement leg prior to UL TKA, 3, 6 and 12 months after surgery. The difference was significant ( $p < 0.05$ ) 3 and 6 months after UL TKA. Also, the significant difference ( $p < 0.05$ ) in PT between KE muscles of the involved leg in patients and controls has been observed. Difference between the dominant and nondominant leg of controls was not statistically significant ( $p > 0.05$ ).

*Power output of KE muscles* (N) in patients with OA and in control group subjects at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  are represented in **Figure 7A**. N of the involved KE muscles was lower than of the uninvolvement leg prior to UL TKA, 3, 6 and 12 months after surgery. The difference was significant ( $p < 0.05$ ) 3, 6 and 12 months after UL TKA. Also, the significant difference ( $p < 0.05$ ) in N between KE muscles of the involved leg in patients and dominant leg of controls has been observed. N of KE muscles of the uninvolvement leg at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  did not differ significantly from controls ( $p > 0.05$ ). Difference between the dominant and nondominant leg of controls was not statistically significant ( $p > 0.05$ ).

Values for N of KE in patients with knee OA and in control subjects at angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  are presented in **Figure 8A**. N of the KE muscles of the involved leg in patients was lower than of uninvolvement leg prior to UL TKA, 3, 6 and 12 months after surgery. The difference was significant ( $p < 0.05$ ) 3 and 6 months after UL TKA. Also, a significant difference ( $p < 0.05$ ) in N between KE muscles of the involved leg in patients and controls has been observed. N of KE muscles of the uninvolvement leg in OA



patients did not differ significantly ( $p>0.05$ ) from controls. There was noted no difference between the dominant and nondominant leg values of N of controls at this angular velocity.

*Isokinetic maximal rate of force development* (MRFD) of KE muscles at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  is represented in **Figure 9A**. MRFD of the involved leg in OA patients was lower than of uninvolved leg prior to UL TKA, 3, 6 and 12 months after surgery. The difference was statistically significant ( $p<0.05$ ) only 6 months after UL TKA at both angular velocities. Also, the significant difference ( $p<0.05$ ) in MRFD between KE muscles of the involved leg in patients and controls was observed 6 months postoperatively. There was noted no difference between the dominant and nondominant leg of controls ( $p>0.05$ ).

MRFD of KE muscles at angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  is represented in **Figure 9B**. MRFD of the involved leg in OA patients was lower than of uninvolved leg prior to UL TKA, 3, 6 and 12 months after surgery. The difference was statistically significant ( $p<0.05$ ) only 6 months after UL TKA. MRFD between KE muscles of the involved leg in patients and controls did not differ statistically significantly ( $p>0.05$ ). There was found no difference ( $p>0.05$ ) between the dominant and nondominant leg of controls at this angular velocity.

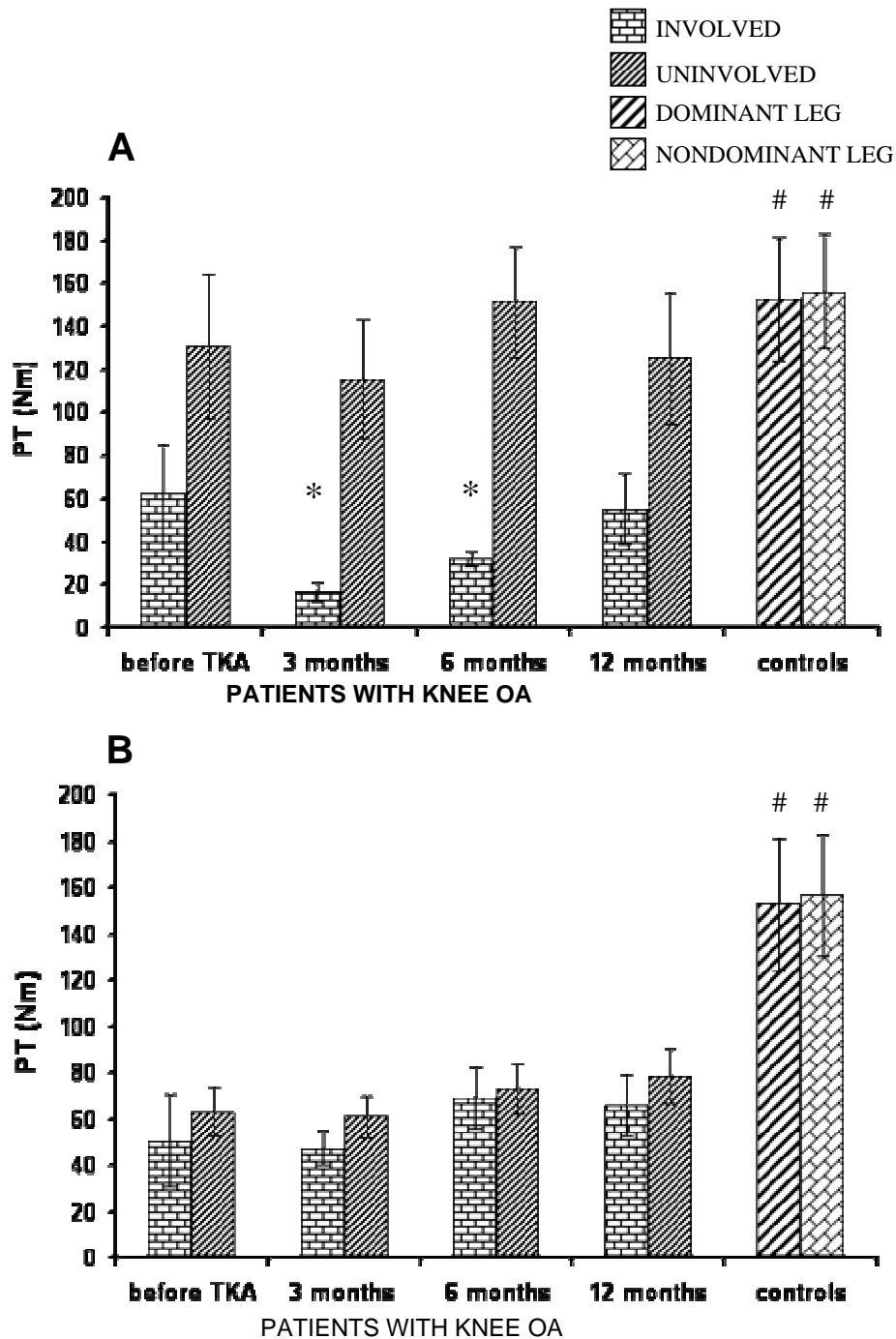
*Values of rate of force development RFD at 30° from beginning of concentric contraction* ( $\text{RFD}_{30}$ ) at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  in OA patients and in controls is represented in **Figure 10A**.  $\text{RFD}_{30}$  of the involved leg in OA patients was lower than of uninvolved leg prior to UL TKA, 3, 6 and 12 months after surgery. The difference was statistically significant ( $p<0.05$ ) 3 and 6 months after UL TKA. Values of  $\text{RFD}_{30}$  of KE muscles of the involved leg in patients and in controls did not differ significantly ( $p>0.05$ ) as well as the dominant and nondominant leg of controls.

$\text{RFD}_{30}$  at angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  is represented in **Figure 10B**.  $\text{RFD}_{30}$  of the involved leg in OA patients was lower than of uninvolved leg prior to UL TKA, 3, 6 and 12 months after surgery. The difference was statistically significant ( $p<0.05$ ) 3 and 6 months after UL TKA. Values of  $\text{RFD}_{30}$  of KE muscles of the involved leg in patients and in controls did not differ significantly ( $p>0.05$ ) as well as the dominant and nondominant leg of controls.

*Rate of force development of KE muscles at 60° from beginning of concentric contraction* ( $\text{RFD}_{60}$ ) at angular velocity of  $60^\circ/\text{s}$  is represented in **Figure 11A**.  $\text{RFD}_{60}$  of the involved leg in OA patients was lower than of uninvolved leg prior to UL TKA, 3, 6

and 12 months after surgery. The difference was statistically significant ( $p < 0.05$ ) 3 and 6 months after UL TKA. The significant difference ( $p < 0.05$ ) in  $RFD_{60}$  between KE muscles of the involved leg in patients and controls was observed 3 and 6 months after UL TKA. There was noted no difference between the dominant and nondominant leg of controls was not statistically significant ( $p > 0.05$ ).

$RFD_{60}$  at angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  is represented in **Figure 11B**.  $RFD_{60}$  of the KE of involved leg in OA patients was lower than of uninvolved leg prior to UL TKA, 3, 6 and 12 months after surgery. The difference was not statistically significant ( $p > 0.05$ ). The difference in  $RFD_{60}$  between KE muscles of the involved leg in patients and in controls was statistically significant ( $p < 0.05$ ) 3 months after surgery. There was found no significant difference between the dominant and nondominant leg of controls ( $p > 0.05$ ).



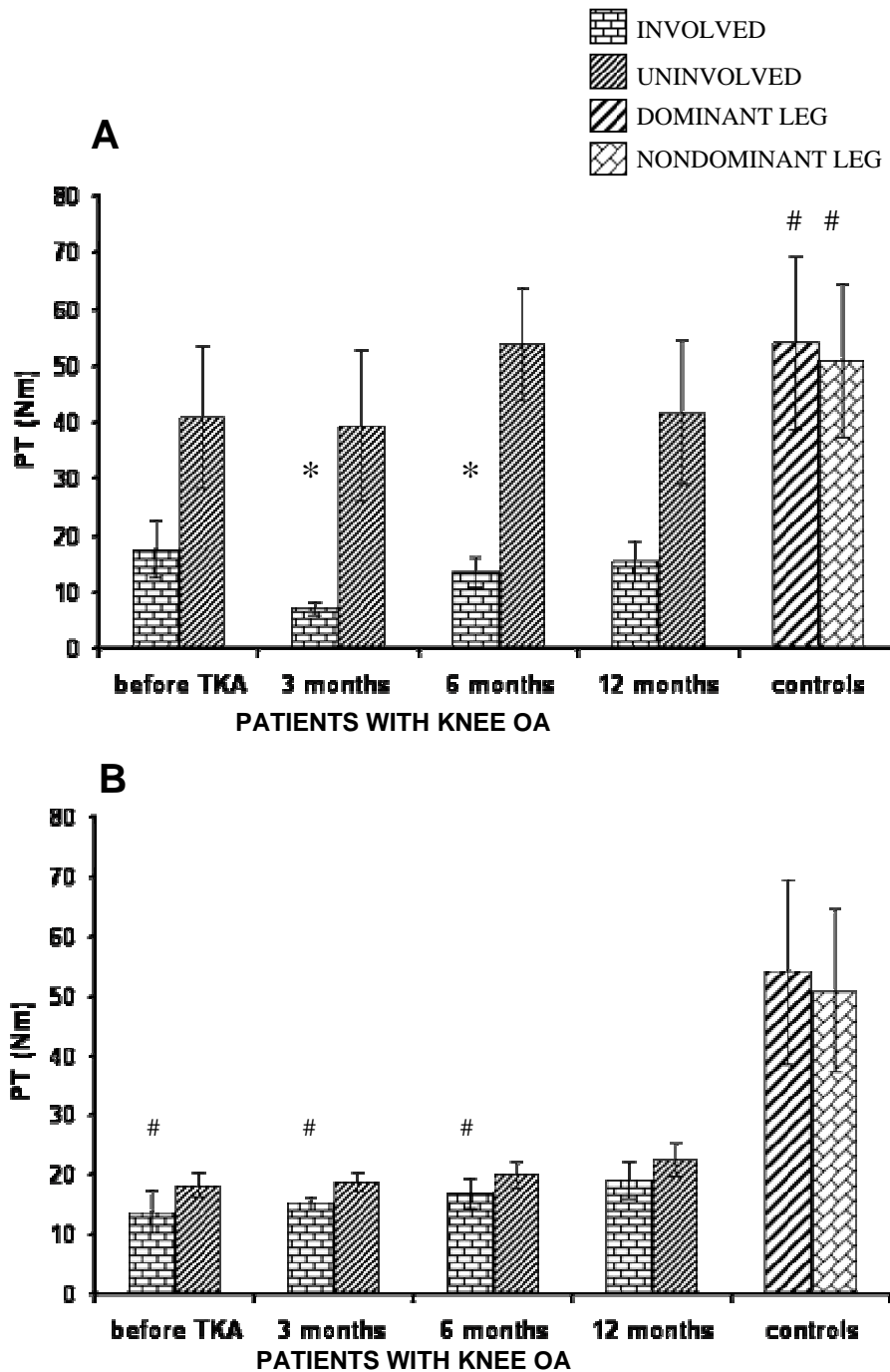
**Figure 5.** Isokinetic peak torque (PT) of the knee extensor muscles at the angular velocity of  $2.09 \text{ rad}\cdot\text{sec}^{-1}$  in patients with knee osteoarthritis before, 3, 6 and 12 months after total knee arthroplasty (TKA) and in controls (mean $\pm$ SE).

\*  $p < 0.05$  as compared with uninvolved leg;

#  $p < 0.05$  as compared the involved leg of patients to dominant and nondominant leg of controls.

Involved leg - scheduled for TKA leg.

**A** - patients with unilateral TKA, **B** - patients with bilateral TKA.



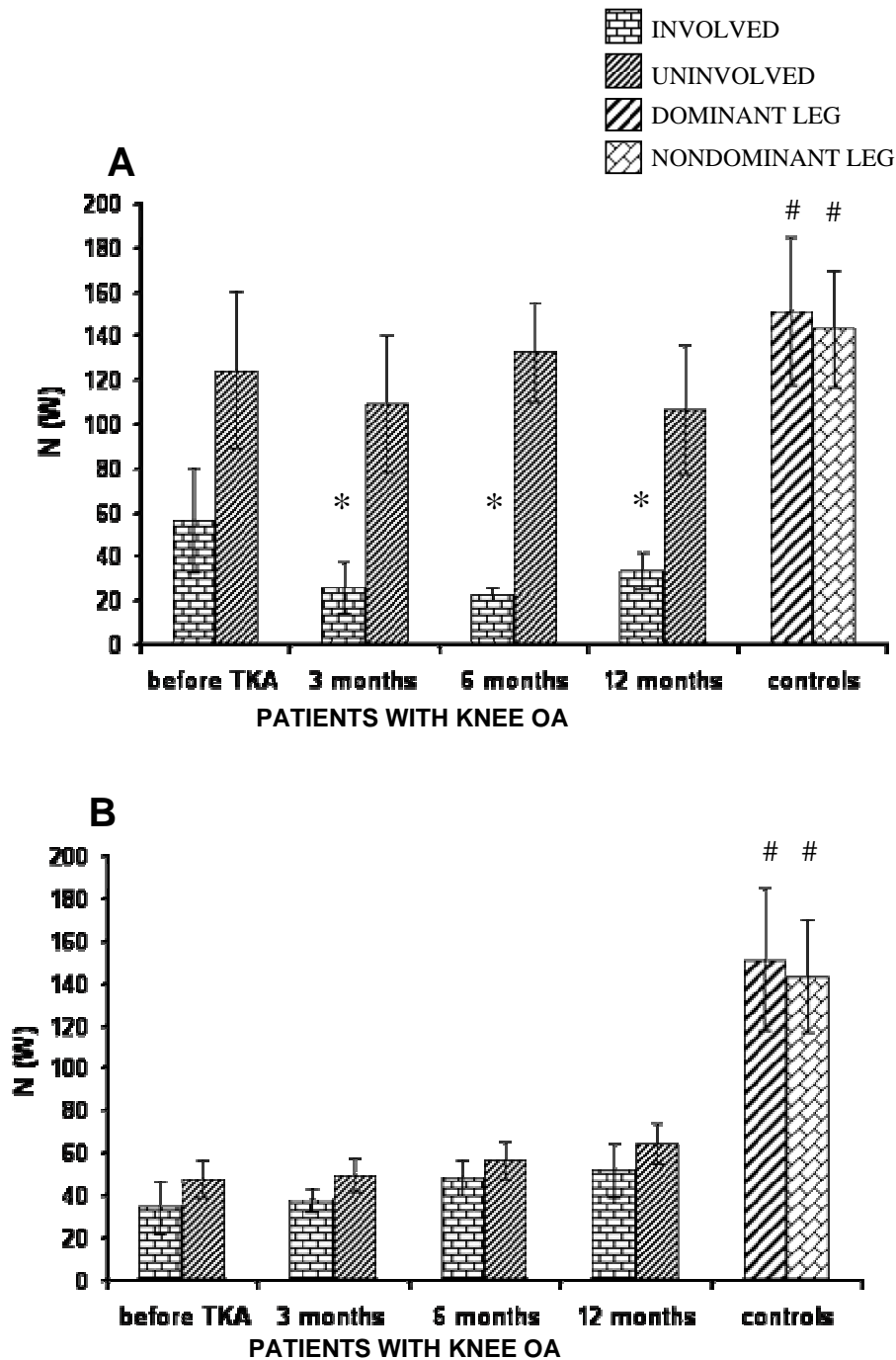
**Figure 6.** Isokinetic peak torque (PT) of the knee extensor muscles at the angular velocity of  $3.14 \text{ rad}\cdot\text{sec}^{-1}$  in patients with knee osteoarthritis before, 3, 6 and 12 months after total knee arthroplasty (TKA) and in controls (mean $\pm$ SE).

\*  $p < 0.05$  as compared with uninvolved leg;

#  $p < 0.05$  as compared the involved leg of patients to dominant and nondominant leg of controls.

Involved leg - scheduled for TKA leg.

A - patients with unilateral TKA, B - patients with bilateral TKA.



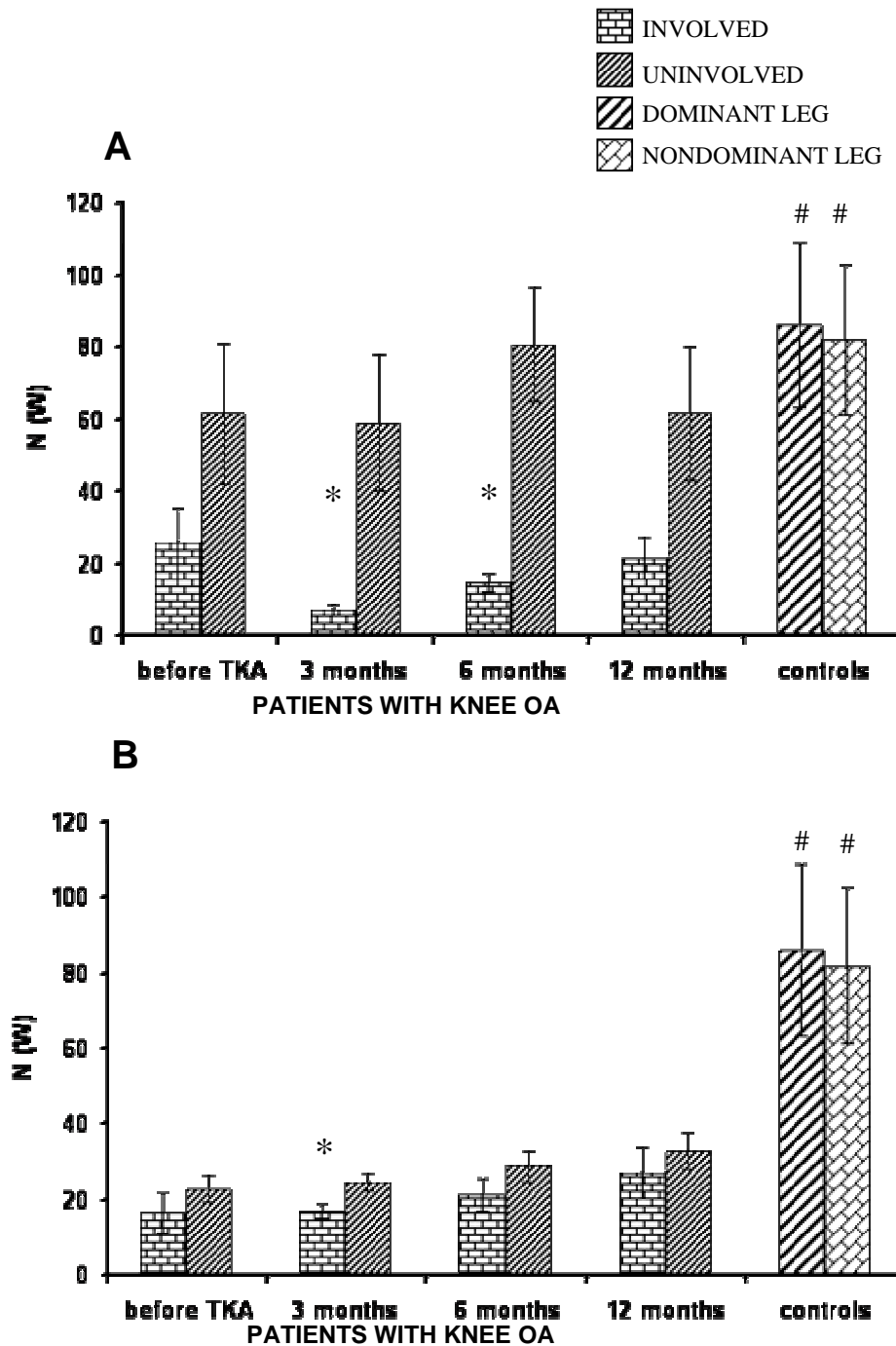
**Figure 7.** Power output (N) at maximal isokinetic peak torque of the knee extensor muscles at the angular velocity of  $2.09 \text{ rad}\cdot\text{sec}^{-1}$  in patients with knee osteoarthritis before, 3, 6 and 12 months after total knee arthroplasty (TKA) and in controls (mean $\pm$ SE).

\*  $p < 0.05$  as compared with uninvolved leg;

#  $p < 0.05$  as compared the involved leg of patients to dominant and nondominant leg of controls.

Involved leg - scheduled for TKA leg.

**A** - patients with unilateral TKA, **B** - patients with bilateral TKA.



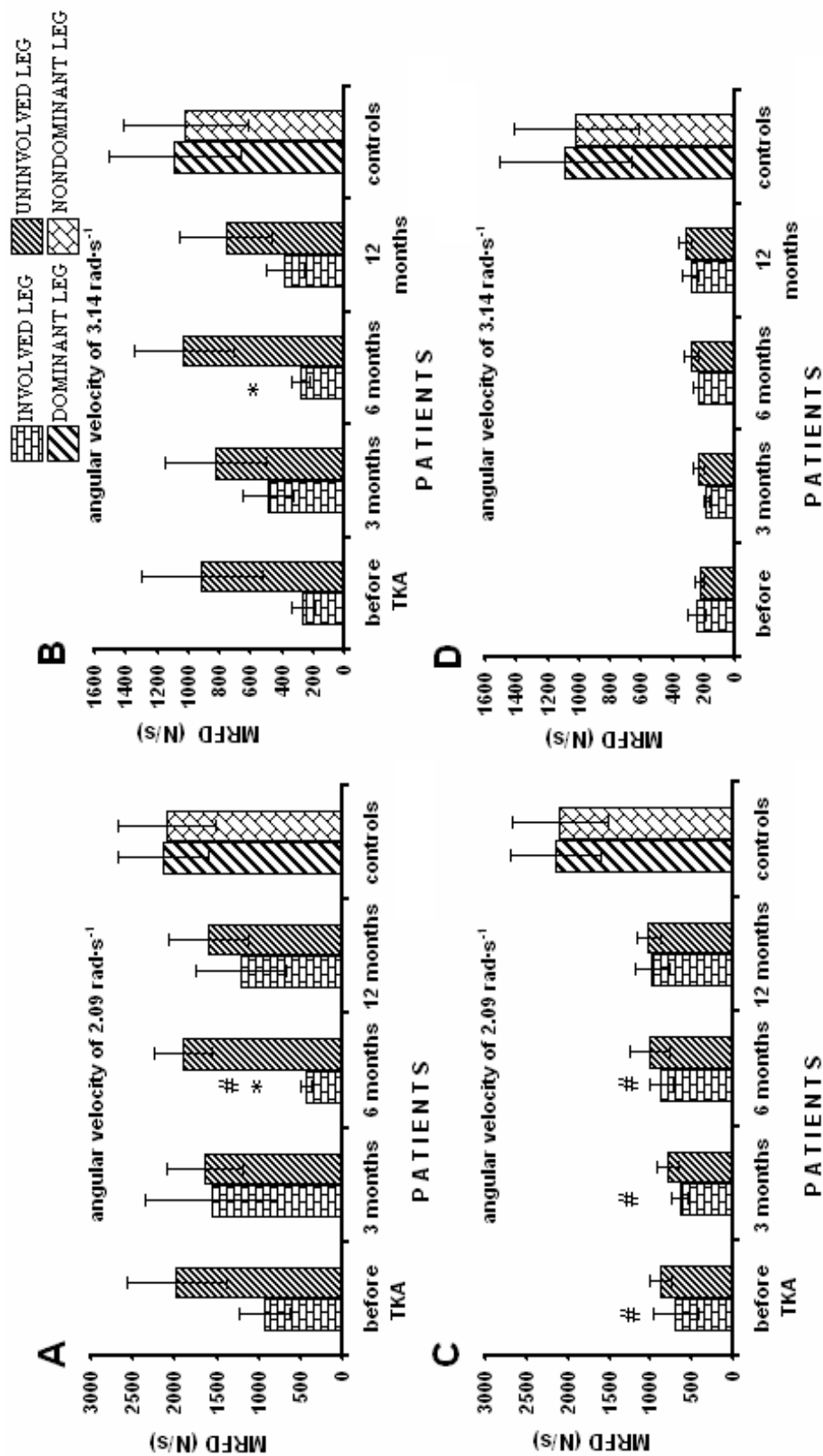
**Figure 8.** Power output (N) at maximal isokinetic peak torque of the knee extensor muscles at the angular velocity of  $3.14 \text{ rad}\cdot\text{sec}^{-1}$  in patients with knee OA before, 3, 6 and 12 months after total knee arthroplasty (TKA) in controls (mean $\pm$ SE).

\*  $p < 0.05$  as compared with uninjured leg;

#  $p < 0.05$  as compared the involved leg of patients to dominant and nondominant leg of controls.

Involved leg - scheduled for TKA leg.

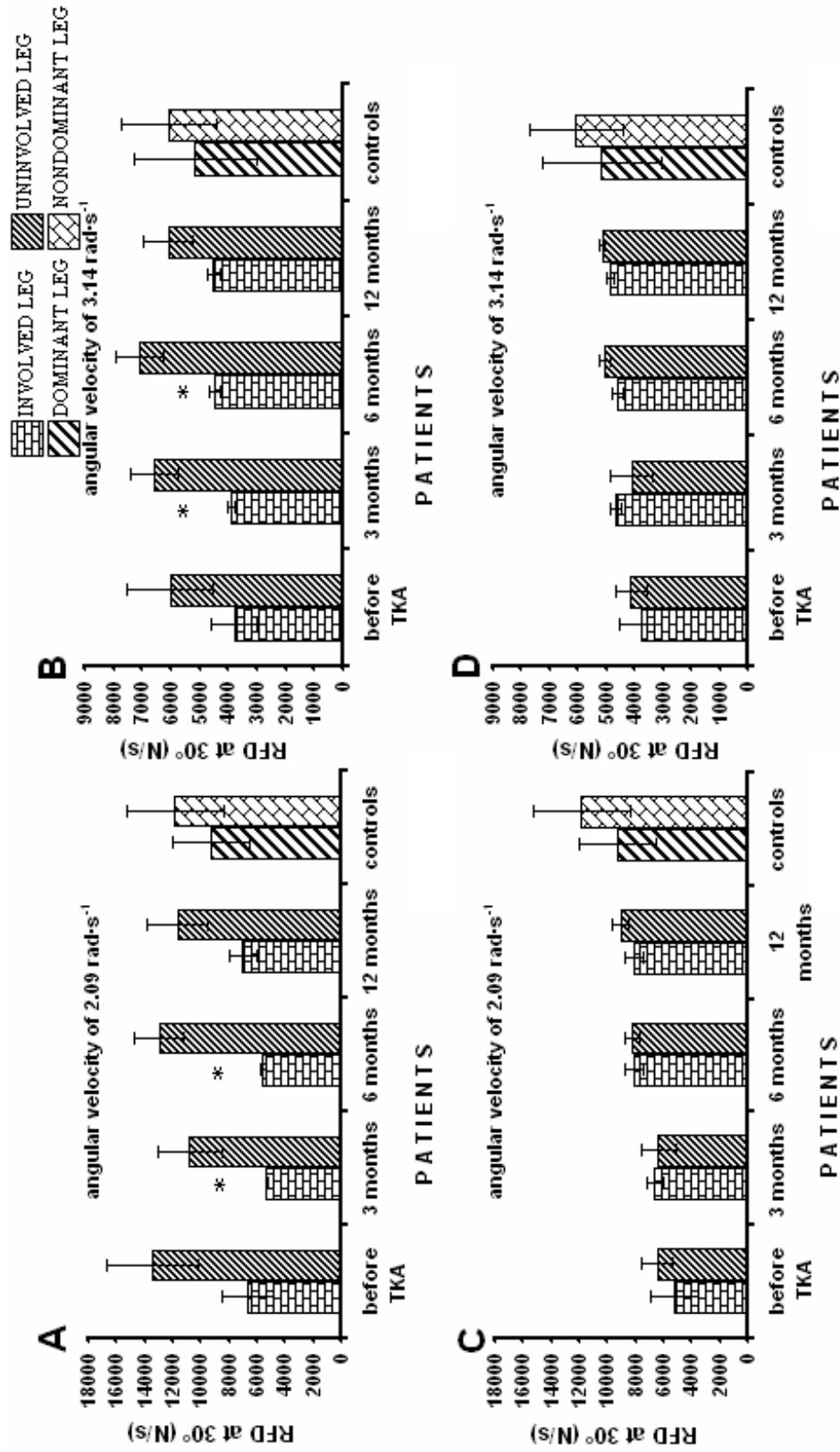
A - patients with unilateral TKA, B - patients with bilateral TKA.



**Figure 9.** Maximal rate of force development (MRFD) of the knee extensor muscles at the angular velocity of 2.09 rad·s<sup>-1</sup> and at 3.14 rad·s<sup>-1</sup> in patients with knee OA before, 3, 6 and 12 months after total knee arthroplasty (TKA) and in controls (mean±SE). **A, B** - patients with unilateral TKA, **C, D** - patients with bilateral TKA. Involved leg - leg with scheduled for TKA knee.

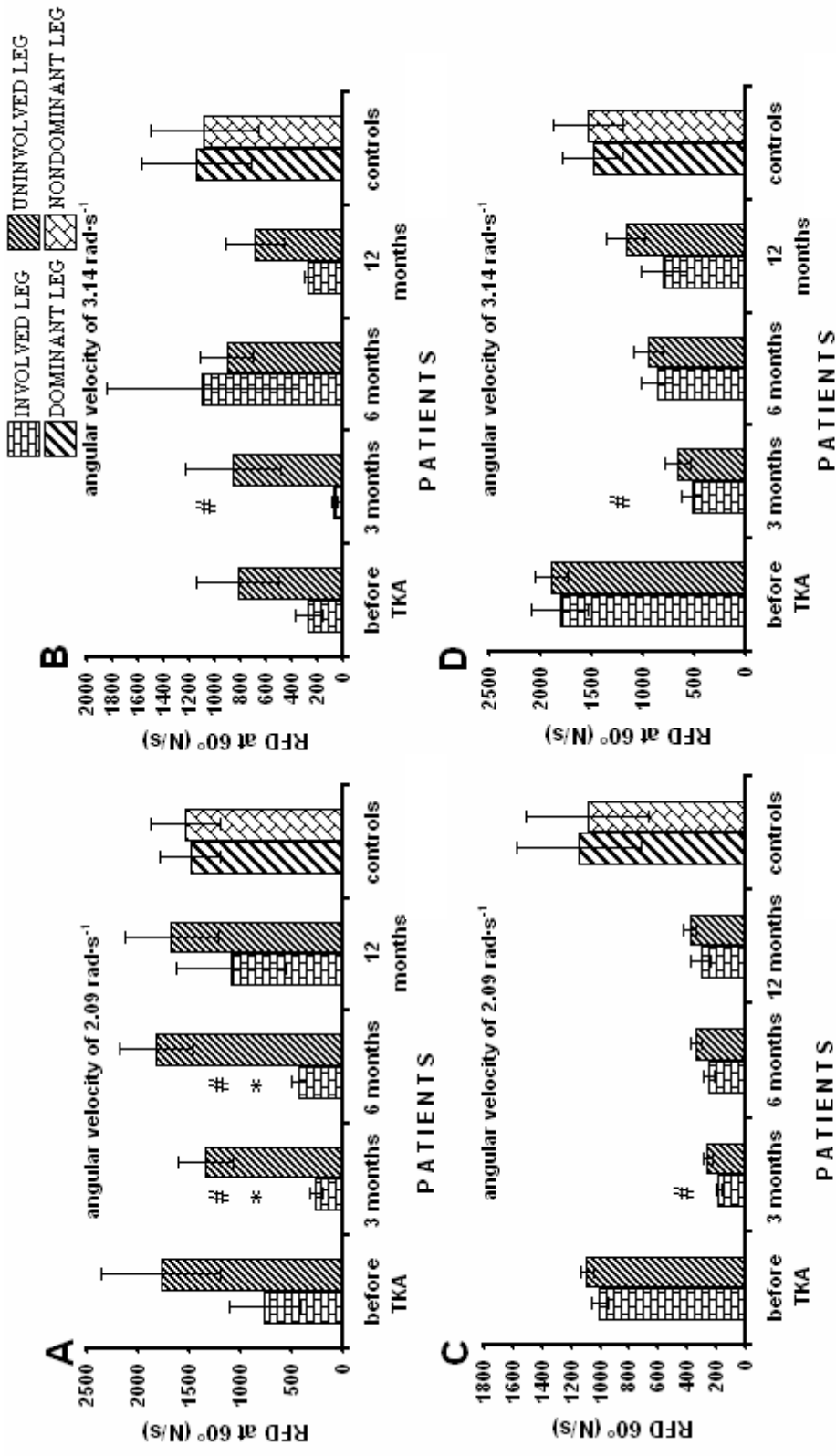
\* p<0.05 as compared with uninvolved leg.

# p<0.05 as compared the involved leg of patients to dominant and nondominant leg of controls.



**Figure 10.** Rate of force development (RFD) at 30 degrees of knee extension at the angular velocity of 2.09 rad·s<sup>-1</sup> and at 3.14 rad·s<sup>-1</sup> in patients with knee OA before, 3, 6 and 12 months after total knee arthroplasty (TKA) and in controls (mean±SE). **A, B** - patients with unilateral TKA, **C, D** - patients with bilateral TKA. Involved leg - leg with scheduled for TKA knee. \* p<0.05 as compared with uninvolved leg.





**Figure 11.** Rate of force development (RFD) at 60 degrees of knee extension at the angular velocity of 2.09 rad·s<sup>-1</sup> and at 3.14 rad·s<sup>-1</sup> in patients with knee OA before, 3, 6 and 12 months after total knee arthroplasty (TKA) and in controls (mean±SE). **A, B** - patients with unilateral TKA, **C, D** - patients with bilateral TKA. Involved leg - leg with scheduled for TKA knee. \* p<0.05 as compared with uninvolved leg. # p<0.05 as compared the involved leg of patients to dominant and nondominant leg of controls.

### 5.1.2. Bilateral TKA group

*Isokinetic strength of KE muscles* (PT) at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  (**Figure 5B**) of the involved leg in UL TKA patients was lower than of uninvolved leg prior to BL TKA, 3, 6 and 12 months after surgery. There was noted no significant difference ( $p>0.05$ ) between involved and uninvolved leg of patients. The significant difference ( $p<0.05$ ) in PT between KE muscles of the involved leg in patients and controls has been observed. Difference between the dominant and nondominant leg of controls was not statistically significant ( $p>0.05$ ).

PT of KE muscles at angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  (**Figure 6B**) of the involved leg in UL TKA patients was also lower than of uninvolved leg prior to BL TKA, 3, 6 and 12 months after surgery. The difference was not statistically significant ( $p>0.05$ ). The significant difference ( $p<0.05$ ) in PT between KE muscles of the involved leg in patients and controls has been observed before BL TKA, 3 and 6 months after surgery. Difference between the dominant and nondominant leg of controls was not statistically significant ( $p>0.05$ ).

*Power output* (N) of KE muscles in patients with UL TKA and in control group subjects at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  are represented in **Figure 7B**. N of the involved KE muscles was lower than of the uninvolved leg prior to BL TKA, 3, 6 and 12 months after surgery where the difference was not significant ( $p>0.05$ ). The significant difference ( $p<0.05$ ) in N between KE muscles of the involved leg in patients and controls was found before and after surgery. Difference between the dominant and nondominant leg of controls was not statistically significant ( $p>0.05$ ).

Values for N of KE in patients with UL TKA and in control subjects at angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  are presented in **Figure 8B**. N of the KE muscles of the involved leg in patients was lower than of the uninvolved leg prior to BL TKA, 3, 6 and 12 months after surgery. The difference was significant ( $p<0.05$ ) 3 months after BL TKA. Also, the significant difference ( $p<0.05$ ) in N between KE muscles of the involved leg in patients and controls has been observed. There was noted no difference between the dominant and nondominant leg of controls ( $p>0.05$ ) at this angular velocity.

*Isokinetic maximal rate of force development* (MRFD) of KE muscles at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  is represented in **Figure 9C**. MRFD of the involved leg in UL TKA patients was lower than of uninvolved leg prior to BL TKA, 3, 6 and 12 months after surgery. The difference was not statistically significant ( $p>0.05$ ). The

significant difference ( $p < 0.05$ ) in MRFD between KE muscles of the involved leg of patients and controls was observed before, 3 and 6 months after BL TKA. There was noted no difference between the dominant and nondominant leg of controls ( $p > 0.05$ ) at this angular velocity.

MRFD of KE muscles at angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  is represented in **Figure 9D**. MRFD of the involved leg in UL TKA patients was lower than of uninvolvement leg prior to BL TKA, 3, 6 and 12 months after surgery. The difference was not statistically significant ( $p > 0.05$ ). Values of MRFD of KE muscles of the involved leg in patients and in controls did not differ significantly ( $p > 0.05$ ) as well as the dominant and nondominant leg of controls.

*Values of RFD<sub>30</sub>* at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  in UL TKA patients and in controls is represented in **Figure 10C**. RFD<sub>30</sub> of the involved leg in UL TKA patients was lower than of uninvolvement leg prior to TKA, 3, 6 and 12 months after surgery. The difference was not statistically significant ( $p > 0.05$ ). Values of RFD<sub>30</sub> of KE muscles of the involved leg in patients and in controls did not differ significantly ( $p > 0.05$ ) as well as the dominant and nondominant leg of controls.

RFD<sub>30</sub> at angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  is represented in **Figure 10D**. RFD<sub>30</sub> of the involved leg in UL TKA patients was lower than of uninvolvement leg prior to BL TKA, 3, 6 and 12 months after BL TKA. The difference was not statistically significant ( $p > 0.05$ ). Values of RFD<sub>30</sub> of KE muscles of the involved leg in patients and in controls did not differ significantly ( $p > 0.05$ ) as well as the dominant and nondominant leg of controls.

*Values of RFD<sub>60</sub>* at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  is represented in **Figure 11C**. RFD<sub>60</sub> of the involved leg in UL TKA patients was lower than of uninvolvement leg prior to BL TKA, 3, 6 and 12 months after surgery. The difference was not statistically significant ( $p > 0.05$ ). Values of RFD<sub>60</sub> of KE muscles of the involved leg in patients and in controls differed significantly ( $p < 0.05$ ) 3 months after BL TKA. There was found no difference ( $p > 0.05$ ) between the dominant and nondominant leg of controls at this angular velocity.

RFD<sub>60</sub> at angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  is represented in **Figure 11D**. RFD<sub>60</sub> of the involved leg in UL TKA patients was lower than of uninvolvement leg prior to BL TKA, 3, 6 and 12 months after surgery. The difference was not statistically significant ( $p > 0.05$ ). Values of RFD<sub>60</sub> of KE muscles of the involved leg in patients and in controls

differed significantly ( $p < 0.05$ ) 3 months after surgery. There was noted no difference between the dominant and nondominant leg of controls ( $p > 0.05$ ).

## **5.2. Knee ROM**

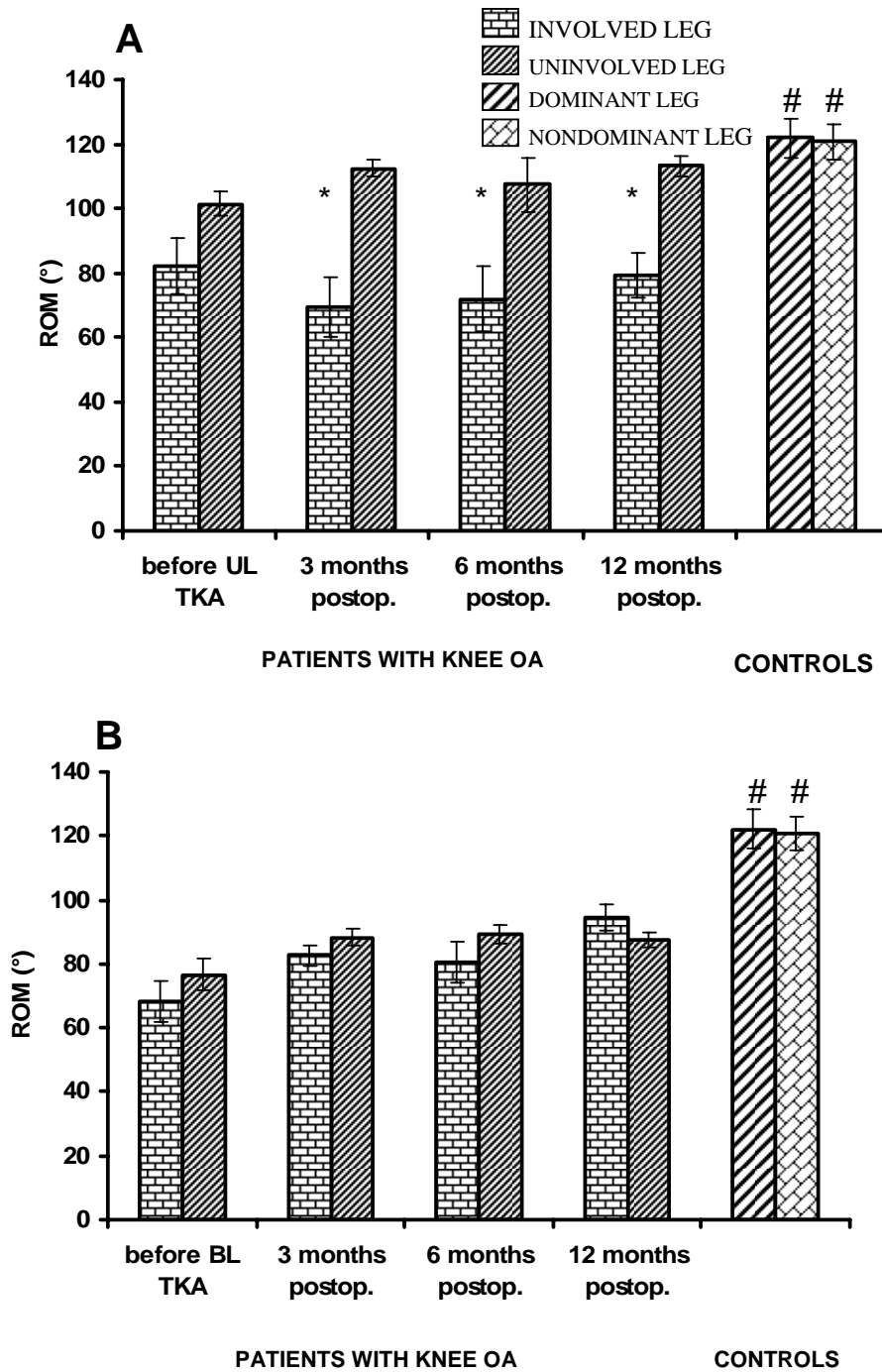
***Knee active extension ROM.*** Patients with knee OA had deficit of knee extension ranged 7-10° before TKA and there were noted no significant differences in this characteristic 3, 6 and 12 months after TKA. Below represented results of knee active flexion ROM.

### **5.2.1. Unilateral TKA group**

**Figure 12A** shows the values for active ROM of knee joint flexion in patients with OA prior to surgery, 3, 6 and 12 months after surgery and in control group subjects. Knee flexion active ROM of involved leg in OA patients was lower 3, 6 and 12 months after TKA as compared with uninvolved leg and with controls ( $p < 0.05$ ). There were no significant differences ( $p > 0.05$ ) in knee flexion active ROM between the dominant and nondominant leg of controls.

### **5.2.2. Bilateral TKA group**

**Figure 12B** represents the values for active ROM of involved leg in BL TKA patients. Knee flexion active ROM was lower prior to BL TKA, 3, 6 and 12 months after surgery as compared with uninvolved leg and to controls ( $p < 0.05$ ). ROM of knee flexion between involved and uninvolved leg did not differ significantly ( $p > 0.05$ ). There were no significant differences ( $p > 0.05$ ) in knee flexion active ROM between the dominant and nondominant leg of controls.



**Figure 12.** Active range of motion (ROM) of knee flexion in patients with knee osteoarthritis before, 3, 6 and 12 months after total knee arthroplasty (TKA) and in controls (mean±SE).

\*  $p < 0.05$  as compared with uninvolved leg;

#  $p < 0.05$  as compared the involved leg of patients to dominant and nondominant leg of controls.

Involved leg - scheduled for TKA leg.

A - patients with unilateral TKA, B - patients with bilateral TKA.

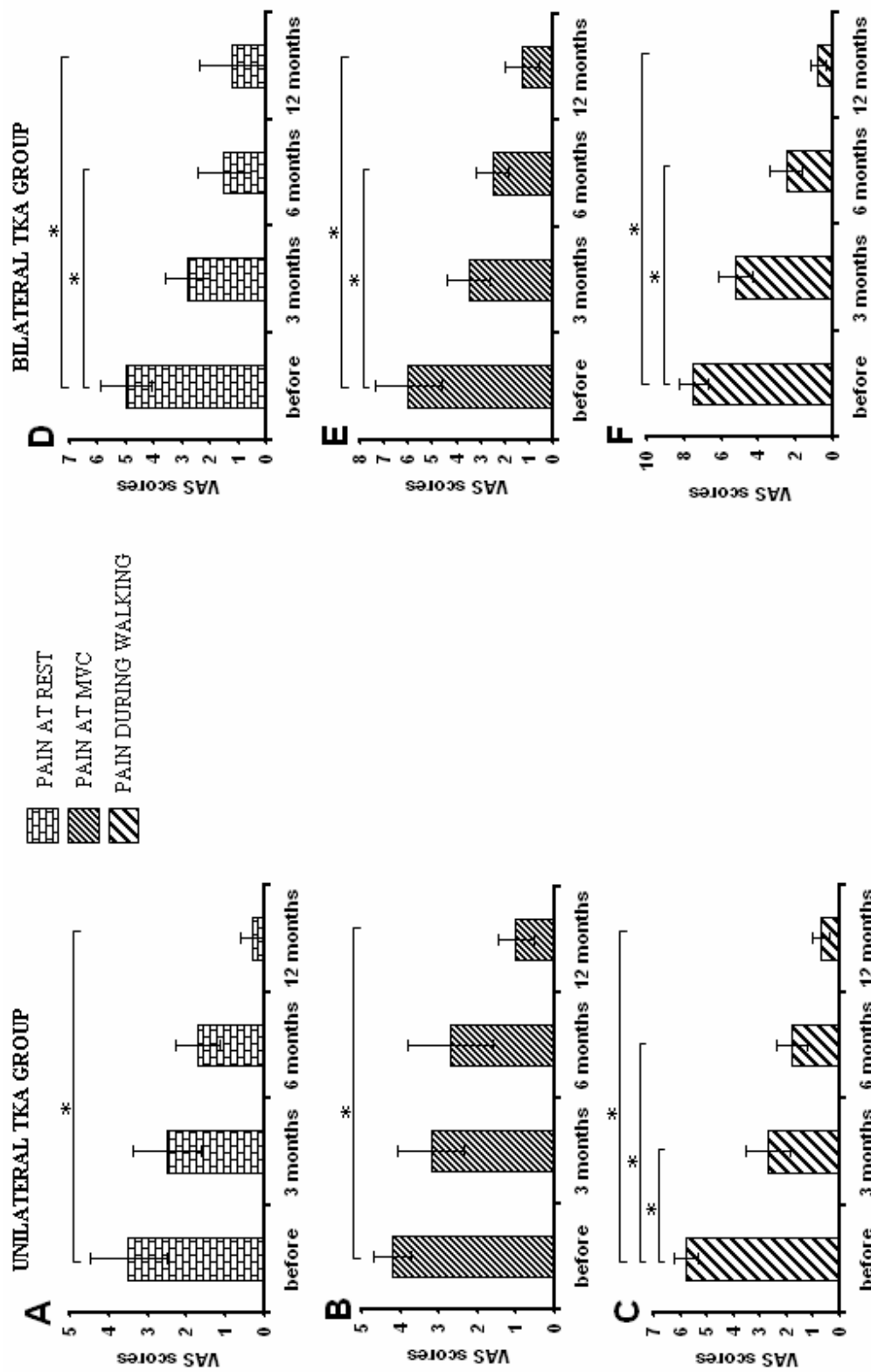
## **5.3. Pain**

### **5.3.1. Unilateral TKA group**

VAS scores for pain (at rest and at maximal voluntary contraction (MVC) and during walking) in patients with UL TKA are presented in **Figure 13, A-C**. There was observed a significant decrease of pain postoperatively 3, 6 and 12 months. Pain at rest, at maximal voluntary contraction and during walking was significantly ( $p < 0.05$ ) lower 12 months after surgery. Pain during walking was significantly ( $p < 0.05$ ) lower 3, 6 and 12 months after TKA as compared with preoperative data.

### **5.3.2. Bilateral TKA group**

VAS scores for pain (at rest and at maximal voluntary contraction (MVC) and during walking) in patients with BL TKA are presented in **Figure 13, D-F**. In bilateral group a significant decrease of pain was found 3, 6 and 12 months postoperatively. Patients reported significant ( $p < 0.05$ ) pain decrease at rest, at maximal voluntary contraction and during walking was observed 6 and 12 months after surgery.



**Figure 13.** Knee pain assessed by visual analogue scale (VAS) at rest, at maximal voluntary contraction (MVC) and during walking in patients with knee OA before, 3, 6 and 12 months after TKA (mean±SE). **A, B, C** - patients with unilateral TKA, **D, E, F** - patients with bilateral TKA. \*  $p < 0,05$  as compared with preoperative data.

## 6. DISCUSSION

### 6.1. Isokinetic contraction of knee extensor muscles

Muscle strength usually refers to maximum force (recorded by a dynamometer) or torque (recorded by isokinetic dynamometer) of the tested muscle group (Sale, 1991). A number of studies have considered recording muscles ability for explosive force (or torque) production as an additional class of strength tests.

In this study isokinetic contraction peak torque (PT) and isokinetic maximal rate of force development (RFD) of KE muscles in patients with knee OA was measured prior to unilateral and bilateral TKA and 3, 6 and 12 months after surgery. Data of the involved leg of both groups of patients were compared with the uninvolved leg and to controls (of dominant and nondominant leg).

Results of present study indicated that isokinetic contraction PT and RFD of KE muscles of the involved leg in patients scheduled for TKA were significantly lower prior to TKA, 3, 6 and 12 months after TKA at angular velocities of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  and  $3.14 \text{ rad}\cdot\text{s}^{-1}$  compared with uninvolved leg. In UL TKA and BL TKA patients the isokinetic contraction PT of KE muscles of the involved leg was found to be lower as compared to controls (dominant and nondominant leg). Numerous studies have demonstrated the KE muscles strength decrease in OA patients (Slemenda *et al*, 1997; Mizner *et al*, 2003; Stevens *et al*, 2003; Pap *et al*, 2004). Pain and failure of volitional activation of the KE muscles are causes of decreased muscle force in OA patients in comparison to controls (Van Baar *et al*, 1998; Mizner *et al*, 2003).

It is well known that the ability of human muscles to develop tension is dependent on the angular velocity of movement. Our data indicated that in subjects with TKA the isokinetic contraction PT deficit seems to be more distinct at  $2.09 \text{ rad}\cdot\text{s}^{-1}$  rather than at  $3.14 \text{ rad}\cdot\text{s}^{-1}$ . Concentric contractions at slow- and intermediate-velocity differ by recruitment of the motor units. At slow-velocity concentric contraction the high resistance induced by isokinetic dynamometer implies maximal muscle activation throughout the ROM, and a large amount of both slow and fast motor units are recruited. Whereas, during intermediate-velocity concentric contraction the lower resistance induced by isokinetic dynamometer restrict considerable muscle activation that is involved primarily only at the beginning (agonists) and at the end (antagonists) of range of motion (Wrigley & Grant, 1998).



In the present study we observed isokinetic contraction PT deficit of KE muscles of involved leg in UL TKA patients at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  prior to TKA 53 and 60% as compared with the uninvolved leg and to controls respectively. In BL TKA group the deficit was 20% compared with uninvolved leg and 68% compared to controls. At higher angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  isokinetic contraction PT deficit of KE muscles of the involved leg of UL TKA group before surgery was 57 and 68%, respectively as compared with the uninvolved leg and to controls. In BL TKA group the deficit was 23 and 75% respectively. This is in agreement with data of Fisher & Pendergast (1997) who found 72% decline in KE isokinetic contraction strength in OA subjects at angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  in comparison to healthy controls. Madsen *et al* (1995) found that in knee OA patients, isokinetic strength was reduced by 40%.

In this study the deficit of isokinetic MRFD of involved leg KE muscles at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  was 53% compared with uninvolved leg and 57% compared to controls for UL TKA group before TKA. For BL TKA group this deficit was 21 and 68%, respectively. MRFD at higher angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  was 72 and 76% compared with uninvolved leg and to controls in UL TKA group. There was no deficit of MRFD for BL TKA group as compared with uninvolved leg, compared to controls the deficit was 78%, respectively. The capacity of explosive force production declines drastically with increasing age, even more than maximal muscle strength. The decreased ability to develop force rapidly in older people seems to be associated with a lower capacity for neuromuscular response in controlling postural sway (Izquierdo *et al*, 1999). In knee osteoarthritis, and also after TKA, the neuromuscular system undergoes various adaptations during gait and other activities (Bizzini *et al*, 2003). With this we can explain so drastic decrease of explosive strength of involved leg of OA patients. Izquierdo and colleagues (1999) found that in 70-year old men the isometric maximal rate of force development on the force-time curve was lower than in 20-year old men group as much as 64%. Contractile RFD depends on the following factors: the level of neural activation, muscle size, and fiber-type (MHC isoform) composition, the range of the effect of inhibitory neurons and the elastic properties (compliance) of muscles and tendons (Grimby *et al*, 1981; Harridge, 1996; Aagaard *et al*, 2002). Harridge and colleagues (1995) found that the maximal joint moment recorded during fast isokinetic limb movements was positively related to the relative content of type II myosin heavy chain (MHC) isoforms in the quadriceps [vastus lateralis] muscle. And based on these observations, they concluded that contractile RFD is a major determinant of the

maximal force and velocity that can be achieved during fast limb movements. RFD is inherently of major importance for athletes engaged in sports that involve an explosive type of muscle action. However, RFD may also play an important role in other populations. For example, in the elderly individual, the ability to exert a rapid rise in muscle force may reduce the incidence of falls related to the impaired control of postural balance with increasing age (Bassey *et al*, 1992; Aagaard *et al*, 2002).

In the present study deficit of isokinetic RFD of involved leg KE muscles at 30 degrees of knee extension ( $RDF_{30}$ ) at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  in UL TKA group was 51% compared with uninvolved leg and 45% compared to controls. For BL TKA group this deficit was 20 and 57%, respectively.  $RDF_{30}$  deficit of the involved leg at higher angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  was 37 and 38% comparing with uninvolved leg and to controls in UL TKA group. For BL TKA group this deficit was 9 and 48%, respectively.

During dynamic contraction conditions, RFD is influenced by the specific force-velocity and length-tension properties of the respective muscles involved in the movement (Aagaard *et al*, 2002).

In the present study the deficit of isokinetic RFD of involved leg KE muscles at 60 degrees of knee extension ( $RDF_{60}$ ) at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  was 58% compared with uninvolved leg and 51% compared to controls. For BL TKA group this deficit was 25 and 65%, respectively.  $RDF_{60}$  deficit of involved leg at higher angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  was 68 and 77% compared with uninvolved leg and to controls in UL TKA group. For BL TKA group this deficit was 33 and 85%, respectively. Results of present study is not in agreement with results of study of Pääsuke *et al* (2003) who demonstrated the ability to develop isometric force of the knee extensor muscles rapidly did not differ significantly in middle-aged and elderly women.

***Changes of isokinetic contraction parameters of KE muscles three months after TKA.*** We found increase of isokinetic PT deficit of KE muscles of involved leg at angular velocities of  $2.09 \text{ rad}\cdot\text{s}^{-1}$   $3.14 \text{ rad}\cdot\text{s}^{-1}$  for UL TKA and for BL TKA groups. For UL TKA group at lower angular velocity the deficit was 86 and 89% compared with uninvolved leg and to controls. For BL TKA group the deficit was not so evident compared with uninvolved leg (23%) and greater (70%) compared to controls. Several studies have demonstrated KE weakness early after TKA and it was concluded that levels of strength deficit are greater than can be accounted for by muscle atrophy alone (Slemenda *et al*, 1997; Stevens *et al*, 2003; Mizner *et al*, 2003; Pap *et al*, 2004).

Investigations of postoperative changes suggest that patients lose approximately half of their preoperative KE muscles strength in the first month after surgery (Perhonen *et al*, 1992; Stevens *et al*, 2003). Perhaps the most commonly held belief as to why patients are weak early after surgery is that the pain associated with surgical trauma evokes failure of voluntary muscle activation, also known as muscle inhibition (Mizner *et al*, 2003).

Results of present study are in agreement with Mizner *et al* (2005) who found that patients who had undergone TKA experienced a profound loss of KE strength (62%). The loss of strength was also explained by a combination of failure of voluntary muscle activation and atrophy; however, the increased activation failure after TKA was not explained by increased pain.

The results of present study is not in agreement with the study of Lorentzen *et al* (1999) who demonstrated a bilateral significant increase in KE muscles strength (14-18%) in the operated leg three to six months after TKA. Lorentzen *et al* (1999) also found that the knee pain during the muscle strength measurements decreased significantly from the preoperative level within three months after TKA. This is an important factor for the evaluation of muscle strength after TKA.

In this study the deficit of isokinetic MRFD of involved leg KE muscles at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  was 6% compared with uninvolved leg and 28% compared to controls for UL TKA group at three months after TKA. For BL TKA group this deficit was 19 and 70%, respectively. MRFD of KE muscles of involved leg at higher angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  was 42 and 56%, compared with uninvolved leg and to controls in UL TKA group. For BL TKA group this deficit was 22 and 84%, respectively. Examination of morphological alterations of aged human quadriceps femoris muscles with and without injury can provide some insight into the cause of the force decline observed prior to and following TKA. Aging contributes to a decrease in the size of the fast glycolytic, type II muscle fibers, with osteoarthritis increasing the extent of these changes. A decrease in the total number of type I and II muscle fibers as well as atrophy of type II muscle fibers may be largely responsible for the decreased force-production ability of elderly individuals and therefore, may be an effective target of rehabilitation protocols. Following TKA and elderly patient may need not only to overcome age-related deficits in force production but also to counter muscular strength decline attributable to the osteoarthritic disease process (Fiatrone & Evans, 1993; Gaines & Talbot, 1999; Lewek *et al*, 2001).

In the present study the deficit of isokinetic RFD of KE muscles of involved leg at 30 degrees of knee extension ( $RFD_{30}$ ) at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  was 51% compared with uninvolved leg and 56% compared to controls for UL TKA group at three months after TKA. For BL TKA group we found that involved leg KE muscles were 3% stronger than of uninvolved leg, but values were 45% lower than dominant leg of controls. Deficit of  $RFD_{30}$  of KE muscles at higher angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  was 41 and 36% compared with uninvolved leg and to controls in UL TKA group. For BL TKA group this deficit was lower 13 and 24%, respectively.

The deficit of isokinetic RFD of KE muscles of involved leg at 60 degrees of knee extension ( $RFD_{60}$ ) at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  was 81% compared with uninvolved leg and 83% compared to controls for UL TKA three months after TKA. For BL TKA group this deficit was 20 and 66%, respectively. Deficit of  $RFD_{60}$  of KE muscles of involved leg at higher angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  was 93 and 95% compared with uninvolved leg and to controls for UL TKA group. For BL TKA group this deficit was greater (31 and 85%, respectively).

***Changes of isokinetic contraction parameters of KE muscles six months after TKA.*** Six months after surgery we also found a significant deficit of isokinetic contraction PT of KE muscles of involved leg for both groups. At lower angular velocity ( $2.09 \text{ rad}\cdot\text{s}^{-1}$ ) the deficit for UL TKA was 79 and 80% compared with uninvolved leg and to controls, respectively. At higher angular velocity ( $3.14 \text{ rad}\cdot\text{s}^{-1}$ ) this deficit was 74 and 75% compared with uninvolved leg and to controls. For BL TKA group isokinetic PT deficit at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  was not so evident 6 months after TKA in comparison with uninvolved leg (6%), but comparing to controls the deficit was significant (66%). At higher angular velocity the deficit was 16 and 69% comparing with uninvolved leg and to controls in this group.

The deficit of isokinetic MRFD of KE muscles of involved leg at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  was 78% compared with uninvolved leg and 80% compared to controls for UL TKA group six months after surgery. For BL TKA group this deficit was 14 and 60%, respectively. MRFD deficit of KE muscles of involved leg at higher angular velocity of  $3.14 \text{ rad}\cdot\text{s}^{-1}$  was 73 and 75% compared with uninvolved leg and to controls for UL TKA group. For BL TKA group this deficit was 17 and 79%, respectively.

The deficit of RDF of KE muscles of involved leg at 30 degrees of knee extension ( $RDF_{30}$ ) at angular velocity of  $2.09 \text{ rad}\cdot\text{s}^{-1}$  was 58% compared with

uninvolved leg and 56% compared to controls for UL TKA group six months after surgery. For BL TKA group we found that values of RFD<sub>30</sub> of KE of involved leg were 2% lower than of uninvolved leg and 32% lower than dominant leg of controls. RFD<sub>30</sub> at higher angular velocity of 3.14 rad·s<sup>-1</sup> was 37 and 27% comparing with uninvolved leg and to controls in UL TKA group. For BL TKA group this deficit was lower (9 and 24%, respectively).

The deficit of RFD<sub>60</sub> of KE muscles of involved leg at 60 degrees of knee extension (RFD<sup>60</sup>) at angular velocity of 2.09 rad·s<sup>-1</sup> was 77% compared with uninvolved leg and 73% compared to controls for UL TKA six months after surgery. For BL TKA group this deficit was 9 and 44%, respectively.

***Changes of isokinetic contraction parameters one year after TKA.*** One year after surgery strength deficit of KE muscles of involved leg was still remarkable for both groups at both angular velocities. For UL TKA group isokinetic contraction PT deficit at lower velocity of 2.09 rad·s<sup>-1</sup> was 56 and 65% comparing with uninvolved leg and controls, respectively. For BL TKA the deficit was 16 and 66% comparing with uninvolved leg and to controls. At angular velocity of 3.14 rad·s<sup>-1</sup> PT deficit for involved leg was 62 and 71% for UL TKA group and this deficit was 13 and 65% for BL TKA group, respectively as compared with uninvolved leg and controls.

Result of present study is in agreement with Mizner *et al* (2003) who found that KE muscle force deficit was 40% compared with KE muscles force in age-matched subjects without knee disease existed a year after surgery. Silva *et al* (2003) found a 32% strength deficit of KE muscles during isometric testing in patients two years after TKA.

The present study indicated that the deficit of MRFD of KE muscles of involved leg at angular velocity of 2.09 rad·s<sup>-1</sup> was 24% compared with uninvolved leg and 44% compared to controls for UL TKA group one year after surgery. For BL TKA group this deficit was 4 and 55%, respectively. MRFD deficit of KE muscles of involved leg at higher angular velocity of 3.14 rad·s<sup>-1</sup> was 51 and 66% comparing with uninvolved leg and to controls in UL TKA group. For BL TKA group this deficit was 13 and 75%, respectively.

Decreased KE muscle force production is a major impairment following TKA. Knee extension force deficit of 30% to 40% compared with knee extension force in age-matched subjects without knee disease have been reported to exist a year or more after surgery (Berman *et al*, 1991; Bolanos *et al*, 1998; Walsh *et al*, 1998).

In this study the deficit of RFD<sub>30</sub> of KE muscles of involved leg at angular velocity of 2.09 rad·s<sup>-1</sup> was 41% compared with uninvolved leg and 42% compared to controls one year after surgery. For BL TKA group we found that values of involved leg were 11% lower than of uninvolved leg and 32% lower than of dominant leg of controls. RFD<sub>30</sub> deficit of KE muscles of involved leg at higher angular velocity of 3.14 rad·s<sup>-1</sup> was 26% comparing with uninvolved leg and to controls for UL TKA group. For BL TKA group this deficit was 6 and 21%, respectively.

The deficit of RFD<sub>60</sub> of KE muscles of involved leg at angular velocity of 2.09 rad·s<sup>-1</sup> was 36% compared with uninvolved leg and 30% compared to controls one year after surgery. For BL TKA group this deficit was 32 and 49%, respectively. RFD<sub>60</sub> deficit of KE muscles of involved leg at higher angular velocity of 3.14 rad·s<sup>-1</sup> was 62 and 77% comparing with uninvolved leg and to controls in UL TKA group. For BL TKA group this deficit was 20 and 74%, respectively. Handel *et al* (2005) tested TKA subjects three years after surgery and found in the group of patients with TKA 16% strength deficit as compared to control group. He concluded that the deficits of knee strength in patients after TKA are more distinct in high velocities of motion.

TKA predictably reduces knee pain, but it has had limited success in restoring KE muscles force-generating capacity and function to that of age-matched people without osteoarthritis (Mizner *et al*, 2003).

## 6.2. Knee ROM

Knee ROM is an important variable in determining the clinical outcome (Maloney & Schurman, 1992; Schurman *et al*, 2005). According to different researchers, normal knee active flexion ROM is about 130°. We found that active flexion ROM of involved knee in OA patients was significantly lower prior to UL TKA, 3, 6 and 12 months after surgery as compared with contralateral leg and to controls. Mean active flexion ROM value prior to UL TKA was in patients 82° for the involved knee and 101° for the uninvolved knee.

**Three months** after TKA the mean value of knee active flexion ROM for UL TKA group of patients was 69.5° for the involved knee. **Six months** after TKA knee active flexion of involved leg ROM was almost on the same level as 3 months after UL TKA (mean 71°). **One year** after UL TKA knee active flexion ROM of involved leg almost matched preoperative level (mean 79°) but was still remarkably lower than active flexion ROM of uninvolved leg and ROM of dominant and nondominant leg of controls. In this study we observed significant differences in knee ROM within UL TKA group of patients 6 months after surgery (45-100°). This data is in agreement with the study of Ryu *et al* (1993), who measured ROM 4 years after TKA and found significant differences between the subjects (90°-130°). Myles *et al* (2002) found that decreased knee ROM still persists 18 months after TKA. Rowe *et al* (2005) found that in two thirds of patients before TKA (total amount of patients was 50) had preoperative active flexion ROM of more than 90°, 83% of them had reduced flexion postoperatively. The remaining one third had preoperative active flexion ROM of 90 degrees or less, 85 % of them had improved flexion postoperatively. This data is similar with our study. In UL TKA group of subject's active flexion ROM was decreased after TKA. In BL TKA group ROM increased significantly after TKA.

Mean knee active flexion ROM value **prior to BL TKA** was in patients 68.5° for the involved knee and 76.5° for the uninvolved knee. **Three months** after BL TKA the mean value of knee active flexion ROM for UL TKA group of patients was 82.5° for the involved knee, and 88° for the uninvolved knee. **Six months after** BL TKA knee ROM was almost on the same level as 3 months after UL TKA (mean 80.5°). **One year** after BL TKA knee ROM of involved leg significantly exceeded the preoperative level (mean 94.5°) and was remarkably greater than ROM of uninvolved leg (mean 87.5°).

Although TKA offers excellent pain relief and contributes to the overall well-being of the patients, these results suggest that it also leads to a reduced range of active and functional motion in the majority of patients. This is associated with a lower-than-normal physical quality of life. The design of implants and rehabilitation programmes should be taken into account so that better range of motion and quality of life can be achieved for patients.

### **6.3. Knee pain**

In the present study indicated pain relief in patients of UL TKA group of 24-44% 3 months after surgery, 36-70% 6 months after surgery and 77-92% 12 months after TKA as compared with preoperative data.

In BL TKA group pain level at rest, at maximal volitional contraction and during walking was significantly lower compared with preoperative level 3, 6 and 12 months after surgery. Pain was lower at rest (44%) and at maximal volitional contraction (42%) and during walking (31%) lower comparing with preoperative level 3 months after surgery. At six months pain level was 70% lower at rest, 59% lower at MVC and 67% lower during walking comparing with preoperative data. One year after TKA percentage was 76, 90 and 80%, respectively.

Total knee replacement is thus a valid treatment in those cases in which degenerative joint disease necessitates a radical solution.

In our series of patients, we found that although there was progressive recovery of isokinetic strength of involved KE muscles, a considerable imbalance remained between the involved and uninvolved legs and between involved leg and dominant and nondominant leg of controls. The results of this study enable to plan more critically the rehabilitation programme for OA patients scheduled for TKA.



## CONCLUSIONS

1. A significant decrease in isokinetic concentric PT and N of knee extensor muscles has been observed in the involved leg of patients pre- and postoperatively in comparison with uninvolved leg and to control subjects at both angular velocities. Isokinetic concentric strength of uninvolved leg of patients of BL TKA group was lower than of uninvolved leg of UL TKA group before and after surgery.
2. Isokinetic concentric PT matched the preoperative level in UL TKA group twelve months after surgery and in BL TKA group three months after surgery. For both groups of patients the values of isokinetic concentric PT and N at both angular velocities were significantly lower than of dominant and nondominant leg of controls twelve months after surgery (53-87%).
3. Value of isokinetic maximal rate of force development (MRFD) of the knee extensor muscles of the involved of patients with UL and BL TKA matched the preoperative level 6 months after surgery but were significantly lower than of control group subjects one year after surgery (44-78%).
4. Values of rate of force development of the knee extensor muscles of involved leg at angular velocity of 2.09 rad·sec<sup>-1</sup> at 30 and 60° of knee extension were significantly lower in UL TKA group three and six months postoperatively as compared with uninvolved leg.
5. Knee active flexion ROM of involved leg in UL TKA group of patients was significantly lower than of uninvolved leg three, six and twelve months after surgery. In BL group knee ROM of involved leg matched the level of uninvolved leg three months after surgery, and differences between involved and uninvolved leg were not so evident. Involved knee active flexion ROM of both groups of patients were significantly lower than of controls dominant and nondominant leg before and twelve months after surgery.
6. Decrease of knee pain in UL TKA and in BL TKA groups 3 months after surgery. Patients of the UL TKA group reported a significant decrease of pain twelve months after knee surgery at rest, at MVC and during walking. Patients of the BL TKA group reported a significant decrease of pain at six and at twelve months after surgery at rest, at MVC and during walking.

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## SUMMARY IN ESTONIAN

### REIE NELIPEALIHASE ISOKINEETILISE JÕU JA PÕLVELIIGESE LIIKUVUSE NÄITAJAD PATSIENTIDEL PÄRAST PÕLVELIIGESE ENDOPROTEESIMIST

Käesoleva uurimistöö eesmärgiks oli uurida reie nelipealihase ja põlveliigese funktsionaalset seisundit hilise gonartroosiga patsientidel enne, kolm, kuus ja kaksteist kuud pärast ühe- ja kahepoolse põlveliigese endoproteesimist ning võrrelda uuritud näitajaid haigel ja tervel jalal, samuti tervetest indiviididest moodustatud kontrollrühmaga. Käesolevad uuringud viidi läbi Tartu Ülikooli Kinesioloogia ja biomehaanika laboratooriumis ajavahemikul 2002-2006.

Töö ülesanneteks oli hinnata: 1) reie nelipealihase maksimaalset isokineetilist jõumomenti (PT), võimsust (N), maksimaalset jõugradienti ning jõugradienti 30° ja 60° põlveliigese ekstensioonil nurkkiirustel 2.09 rad·s<sup>-1</sup> ja 3.14 rad·s<sup>-1</sup>; 2) põlveliigese aktiivset liikuvust painutusliigutusel; 3) valu põlveliigeses pre- ja postoperatiivselt puhkeolekus, kõnnil ja reielihaste pingutamisel.

Vaatlusalused moodustati kolm rühma. Esimese rühma vaatlusalusteks olid hilise gonartroosiga naised (3) ja mehed (3) vanuses 48 kuni 74 aastat, keda uuriti vahetult enne ning kolm kuud, kuus kuud ja kaksteist kuud pärast ühepoolset totaalset põlveliigese endoproteesimist (ÜP TPE grupp). Teise rühma vaatlusalusteks olid ühepoolse totaalse põlveliigese endoproteesiga naised (3) ja mehed (3) vanuses 49 kuni 74 aastat, keda uuriti vahetult enne kontralateraalse põlveliigese endoproteesimist ning kolm kuud, kuus kuud ja kaksteist kuud pärast operatsiooni (KP TPE grupp). Kontrollrühma moodustasid samas vanuses 3 naist ja 3 meest kellel ei olnud ortopeedilisi ja neuroloogilisi haigusi.

Uurimistöös kasutati reie nelipealihase isokineetilise jõu näitajate määramiseks Cybex II (*Lumex Inc.*, USA) moderniseeritud dünamomeetrit. Põlveliigese aktiivse liikuvuse (ROM) mõõtmisel kasutati mehaanilist goniomeetrit *Gollehon Extendable Goniometer* (*Lafayette Instrument*, USA). Valu (0-kuni 10 punkti) hinnati visuaalse analoog skaala (Hassan *jt*, 2002) abil.

Uuringu põhjal järel dati:

1. Gonartroosiga patsientidel olid reie nelipealihase isokineetiline jõumoment ja võimsus mõlemal nurkkiirustel haigel jalal pre- ja postoperatiivselt oluliselt väiksemad kontralateraalse jalaga ja kontrollrühma näitajatega võrreldes. Gonartroosiga patsientidel KP TPE grupis isokineetiline jõumoment ja võimsus tervel jalal oli oluliselt madalamad võrreldes terve jalaga ÜP TPE grupis enne kui ka pärast operatsiooni.
2. Jõumomendi ja võimsuse näitajate taastumine preoperatiivsele tasemele oli ÜP TPE grupi patsientidel aeglasem võrreldes kahepoolse gonartroosiga patsientidega (vastavalt 12 ja 3 kuud). Nii ÜP kui ka KP TPE grupi patsientide isokineetilise jõu näitajad olid oluliselt väiksemad võrreldes kontrollrühma domineeriva ja mittedomineeriva jalaga kaksteist kuud pärast operatsiooni (53-87%).
3. Gonartroosiga patsientidel jõugradient nurkkiirustel  $2.09 \text{ rad}\cdot\text{s}^{-1}$  ja  $3.14 \text{ rad}\cdot\text{s}^{-1}$  haigel jalal taastunud preoperatiivsele tasemele kuus kuud pärast endoproteesimist. Võrreldes kontrollrühmaga oli nimetatud näitaja oluliselt madalam (44-78%) aasta pärast operatsiooni.
4. Reie nelipealihase isokineetiline jõugradient mõlemal nurkkiirustel  $30$  ja  $60^\circ$  põlveliigese ekstensioonil oli ÜP TPE grupi patsientidel haigel jalal oluliselt väiksem kolm ja kuus kuud postoperatiivselt võrreldes terve jalaga.
5. ÜP TPE grupi patsientidel oli põlveliigese aktiivne liikuvus painutusliigutusel haigel jalal kolm, kuus ja kaksteist kuud pärast endoproteesimist oluliselt väiksem võrreldes terve jalaga. KP TPE grupi patsientidel ei olnud olulist erinevust põlveliigese aktiivsel liikuvusel painutusliigutusel haigel jalal võrreldes varem opereeritud põlvega ning liikuvus taastus preoperatiivsele tasemele kolm kuud pärast endoproteesimist. Mõlema rühma põlveliigese ROM oli võrreldes kontrollgrupiga oluliselt madalam nii enne kui ka 12 kuud pärast operatsiooni.
6. ÜP ja KP TPE grupi patsientidel oli valu vähenenud kolm kuud pärast endoproteesimist. ÜP TPE grupi patsiendid täheldasid olulist valu vähenemist kaksteist kuud pärast endoproteesimist nii puhkeolekus kui ka tahtelisel pingutusel ning kõnnil, samas KP TPE grupi patsiendid olid valu vähenes oluliselt kuus ja kaksteist kuud pärast põlveliigese endoproteesimist puhkeolekus, pingutusel ja kõndimisel.

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