The Effect of Body Mass Index on the Biomechanics Before and After Total Knee Arthroplasty

by

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Abstract

Osteoarthritis (OA) is prevalent, commonly affecting weight-bearing joints, such as the knee. In Canada, approximately 40% of Canadians aged 50 to 64 are diagnosed with OA, of which 38% experience knee pain. When conservative treatments do not help those with end-stage knee OA, total knee arthroplasty (TKA) is an elective surgical option that can provide pain relief and improve knee function. Individuals living with obesity (Body Mass Index [BMI] > 30.0 kilogram/meter squared [kg/m^2]) are at increased risk for developing knee OA compared to normal weight people, partly due to altered knee kinematics and kinetics. Approximately 7 million Canadians were living with obesity in the year 2013.

Quantitative gait analyses via three-dimensional (3D) motion capture have shown that individuals living with obesity or OA have altered knee kinematics and kinetics compared to those of normal weight or those without OA. However, there is a paucity of research on the effect of obesity and OA on knee biomechanics after TKA, particularly in individuals living with Class II obesity (BMI 35.0-39.99 kg/m^2). Understanding the impact of BMI on knee kinematics and kinetics after TKA may inform surgical and rehabilitation protocols to improve individual outcomes.

The purpose of this prospective matched series was to evaluate the impact of obesity on time series and parameters of kinematics (angles of flexion-extension, abduction-adduction, internal-external rotation) and external kinetics (non-normalized and normalized moments to body weight of flexion-extension, abduction-adduction), and spatio-temporal parameters (velocity, cadence, stride length) at pre-TKA, post-TKA, and change from pre-to-post TKA, in 20 adults aged 50 to 70 years. Comparisons were made between 5 females/5 males with class II

obesity (OB group) and 10 age- and sex-matched normal/overweight adults (BMI 18.5-29.9 kg/m^2 [N/OW group]) using 3D motion capture on level ground.

Participants received a 3D gait assessment within one-month before and 12-weeks after TKA. Optoelectronic motion capture and synchronized floor-embedded force platforms collected motion and forces during 10-meter walking at a self-selected speed. A hybrid biomechanical model was created, with inertial properties of body segments estimated via the participant's body height and weight. The data were first screened and interpolated using Eva Real-Time Software and then processed using Visual3D.

To evaluate group differences in kinematic, kinetic and spatio-temporal parameters at pre- and post-TKA, a Wilcoxon rank-sum test was used to compare group medians and interquartile ranges. To evaluate changes over time from pre- to post-TKA between groups in kinematic, kinetic and spatio-temporal parameters, a two-way repeated measures analysis of variance was performed.

The mean BMI for the OB group was 37.1 kg/m^2 (standard deviation [SD] 1.79), and 27.0 kg/m^2 (SD 2.15) for the N/OW group ($p=0.0005$, 95%CI [29.4, 34.8]). Both groups were age and sex matched, with the OB group having mean age of 64.2 years (SD 6.04) with 5 females and 5 males, and the N/OW group having a mean age of 66.8 years (SD 2.86) with 5 females and 5 males. Twenty participants completed the preoperative gait assessment, with 17 (85%) participants (8OB, 9N/OW) completing post-TKA assessment.

At pre-TKA, the OB group had greater maximum flexion angle during swing phase ($p=0.05$) and greater non-normalized extension moment during swing phase ($p=0.03$) compared to the N/OW group. At post-TKA, the OB group had greater maximum adduction angle during stance ($p<0.001$), greater maximum non-normalized moment in adduction ($p<0.001$), greater minimum flexion non-normalized moment during stance phase ($p=0.01$), and greater maximum extension non-normalized moment during swing phase ($p<0.001$), compared to the N/OW group. From pre- to post-TKA, the OB group increased in adduction angle whereas the N/OW group decreased in adduction angle ($p=0.02$) and the OB group decreased in extension non-normalized moment whereas the N/OW group increased in extension non-normalized moment ($p=0.02$) during swing. The groups did not differ in any spatio-temporal parameters either pre- or post-TKA.

While future work is warranted, this thesis contributes to the knowledge of the impact of obesity on the knee time series and parameters of kinematics and kinetics, and spatio-temporal parameters of level walking in adults undergoing TKA. Exploring this relationship can further our understanding of how these factors interact and may allow us to individualize rehabilitation approaches to address modifiable gait differences or to accommodate differing recovery trajectories to improve patient outcomes.

Preface

This thesis is an original work by Christopher Aaron Wayne. The research project, of which this thesis is a part, received ethics approval from the University of Alberta Ethics Board, Project Name “Impact of Body Mass Index on Gait Biomechanics after TKA”, ID Pro00070695, 07/10/2017-02/11/2019. No part of this thesis has been previously published.

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Chapter 1 Introduction

1. Statement of the Problem

Osteoarthritis (OA) is a prevalent chronic musculoskeletal condition, affecting more than 12% of Canadians aged 15 or older.[1–3] Osteoarthritis is a leading factor of disability, causing chronic pain, loss of function and a decrease in quality of life.[4–6] Weight-bearing joints, especially the knee, are commonly affected.[2] Of those diagnosed with OA, approximately 29% experience knee OA.[7] In Canada, like many other countries, the prevalence of knee OA is increasing, creating socioeconomic and economic public health burdens.[8]

Knee OA is a multifactorial disease with the strongest risk factors being previous joint injury (pooled odds ratio [OR] 2.83, 95% CI 1.91-4.19) and obesity (pooled OR 2.10, 95% CI 1.82-2.42).[9–13] Adults living with moderate knee OA are typically treated conservatively with physical therapy, medication and/or weight management.[14–17] However, if the OA does not respond to conservative treatment, total knee arthroplasty (TKA) is an elective surgical option.[18] Total knee arthroplasty replaces deteriorated joint structures of the knee with artificial femoral, tibial, and sometimes patellar, implants. The primary goal of a TKA is to relieve pain, restore knee function, and improve quality of life.[19,20]

Total knee arthroplasty is cost effective and effective for those with end-stage knee OA.[21,22–23] In 2016-2017, 67,169 TKAs were performed in Canada, an increase of 16% since 2012-2013.[24] Adults living with obesity (body mass index [BMI] > 30 kilograms/meter squared [kg/m^2]) who received a TKA are at increased risk (adjusted OR 2.32 [95% CI 1.52-3.53])[25] for knee OA compared to those of normal weight who received a TKA.[19] In 2013, approximately seven million Canadian adults were living with obesity.[26] Both the prevalence of obesity and demand for TKA is increasing.[27,28] Currently, up to 59% of TKA adult patients in Alberta undergoing TKA are living with obesity.[29]

Limited evidence suggests that adults living with obesity have increased knee pain and worse knee function compared to adults of normal weight.[30] Further, current evidence suggests that having a high BMI pre-TKA is associated with functional limitations post-TKA, more postoperative complications and need for revision TKA.[31,32–3637,38] However, the overall impact of BMI on patient outcomes after TKA is unclear. Although those with a high BMI appear to report similar clinical improvement at three months post-TKA compared to those of normal weight, they also report lower absolute function and higher absolute pain both before and after TKA.[39–41] A 2015 review article by Rodriguez-Merchan found 16 studies that reported no adverse association between obesity and TKA outcomes, while 24 studies and 3 systematic reviews reported lower TKA outcomes in adults living with obesity.[42]

Improved physical functioning (walking) is one of patients' primary expectations after TKA.[43] The impact of obesity on TKA joint biomechanics during walking is not clearly defined.[30] Altered gait biomechanics are seen between adults 1) living with obesity compared to those of normal or overweight (BMI 18.5-29.99 kg/m^2) without knee OA or TKA;[31,44–51] and 2) living with TKA compared to those without TKA, in quantitative gait analysis.[20,52–55] However, the effect of obesity on knee joint kinematics, kinetics and spatio-temporal gait parameters after TKA, particularly in adults living with class II obesity (BMI 35.00-39.99 kg/m^2)[56] with knee OA and TKA is unclear. Biomechanical evaluation of gait is needed to determine the impact of class II obesity on walking after TKA.

1.1 Limitations of Current Evidence

The paucity of evidence regarding gait in individuals with obesity who undergo TKA, particularly in those with higher classes of obesity, is partially due to the difficulty in performing standard motion capture analyses because of increased soft tissue artifact. This requires alteration of usual marker placements/approaches to ensure accuracy of the obtained gait data. Further, soft tissue artifact creates challenges in accurately palpating anatomical landmarks,[57,58] which is also critical for obtaining high-quality gait data.

In addition, most studies evaluating gait after TKA have been performed at least six months post-TKA.[20,59] Typically, primary recovery after TKA occurs within three months (12-weeks) of surgery, with continued recovery up to six to 12 months.[60,61] The first month after surgery is considered the subacute period, when function is at the lowest level and pain at the highest.[62] Thus, evaluation of gait three months after TKA may provide important information regarding changes in gait post-TKA. If knee biomechanics are negatively affected by obesity during level ground walking, we can consider strategies to address modifiable gait differences in those living with obesity after their TKA. For example, this information may inform rehabilitation and/or surgical approaches to improve recovery post-TKA in a growing sub-set of the TKA patient population.

1.2 Study Aims

The primary aim is to compare knee biomechanics in kinematics and kinetic time series and parameters, and spatio-temporal gait parameters at pre-TKA, 12-weeks post-TKA, and change from pre- to post-TKA, in adults with class II obesity as compared to age- and sex-matched adults who are normal/overweight. Understanding the impact of obesity on these parameters before surgery and during the post-acute recovery phase when gait parameters are modifiable[63–67] may inform whether clinical decisions such as targeted rehabilitation and/or modification of surgical approaches (e.g., personalized medicine) are needed. We expect to find between-group differences in kinematic and kinetic time series, knee joint angles (e.g., flexion-extension, abduction-adduction, internal-external rotation), knee joint moments (e.g., flexion-extension, abduction-adduction), and spatio-temporal parameters during level ground walking.

1.3 Thesis Outline

This thesis will consist of the following chapters: Chapter 2 contains a review of the existing literature of gait and human body dynamics. Chapter 2 will also review the features of obesity, OA, and TKA and how these factors may impact gait. Chapter 3 will discuss the methods and materials of the work; specifically, the experimental setup and procedure, data processing and parameter extraction and the experimental data analyses used. Chapter 4 will present the results, with the overall goal of comparing the trends in kinematic and kinetic time series, and kinematic, kinetic and spatio-temporal parameters of gait between participant groups. Chapter 5 provides a discussion of the results within the context of currently published evidence and the strengths and limitations of the study. Chapter 6 offers a summary of the work and concludes the findings with recommendations for future work.

Chapter 2 Literature Review

2.1 Overview

This chapter reviews existing literature on gait, obesity and OA. It will also review how gait is affected by obesity and OA generally, and then focus on the impact of obesity on gait in adults undergoing TKA.

2.2 Gait

Bipedal locomotion gait in humans is an autonomous motor activity,[68] requiring the use of the central nervous system, the peripheral nervous system and the musculoskeletal system.[69] The autonomous motor activity in humans can also be overridden with volitional input.[70] When walking, there are various joint stresses and loads on the body, depending on the phase of the gait cycle, walking speed and inclination, and hardness of the surface.[68] Gait analysis assesses an individual's ability and efficiency to move through space by ambulation.[8,54,71]

2.2.1 Human body dynamics

The study of motion of the body segments in relation to internal and external forces (muscle moments) is known as human body dynamics. The forces acting on muscles or joints are measured two different ways. Direct measurement is an invasive procedure requiring force transducers to be inserted directly into the muscles and joints. Indirect measurement is non-invasive and uses estimations from anthropometric measurements, body kinematics and body kinetics and is more commonly reported in gait literature.

2.2.2 Anthropometric measurements

The human body is segmented into the head, trunk, arms, and legs connected by frictionless joints, with each segment having dynamics based upon its mass/volume, shape, and tissue density/distribution. On average, the upper body accounts for 13% of the total body weight, the head, neck and trunk account for 47% of the total body weight, and the lower body accounts for 31% of the total body weight.[72] Researchers have obtained anthropometric measures through living and cadaver human beings. These parameters were calculated through direct measurement, mechanical torsion, chronophotography, photogrammetry, radiographs, stereophotogrammetry, computed tomography, magnetic resonance imaging, x-ray fluoroscopy, and computer assisted techniques.[72] Combining the anthropometric properties of each sub-segment with kinematic data allows for assessing the dynamics of each body segment.

2.2.3 Kinematics

The description of the movement of body segments, termed kinematics, is required to obtain human body dynamics such as with gait.[73] Different devices have been created for this purpose and are described below.

2.2.3.1 Kinematic measurement devices

Several different devices can track human motion and are typically separated into three different categories: visual systems (marker-based, non-marker based, combinatorial), non-visual systems (e.g., mechanical, inertial), and robotic-aided systems. Visual systems are considered the "gold standard" for tracking human motion,[74] and were the focus of this thesis, so the review herein focuses on this category of kinematic devices.

Visual systems use sensor technology (e.g., cameras) to track physical markers attached to bony landmarks on the body, a method known as marker-based tracking. The main advantage to using visual systems is the low error in the tracking of each marker (less than 1 millimetre

[mm]) as well as reduced data processing time. Several commercial technologies for visual motion capture systems are available.

Visual tracking systems can be classified as **active, passive, non-marker-based or combinatorial**. In an **active system**, the physical marker emits light that can be detected by the camera. Active systems (e.g., Optotrack, CODA, and Polaris) have higher accuracy and sampling rates. In a **passive system**, the cameras emit infrared light that reflects off the infrared markers and is captured by the cameras. Passive systems (e.g., VICON and Qualisys) can be wireless, but the equipment can be bulky. Both systems convert two-dimensional (2D) marker positioning into three-dimensional (3D) marker positioning by using data from two or more cameras.

Other systems include **non-marker-based visual tracking systems**, which require no markers during visual tracking. This type of system uses high speed (high sampling rate) and high pixel cameras to capture data in either 2D or 3D. The main advantage to using non-marker-based systems is that there are no measurement errors associated with markers. This includes errors: a) in the accuracy of marker placement on anatomical landmarks, b) of marker displacement when soft tissue under the markers moves, and, c) when markers are occluded or fall off during experimentation.[75,76] Although considered ideal, non-marker-based systems are expensive, require many computing resources and are not fully validated in terms of their accuracy. Finally, the last type of visual system, which attempts to overcome the limitations of both marker-based and non-marker-based systems, is **combinatorial tracking**. In this system, both markers and high-resolution video cameras are used. Marker-based motion capture systems, as used in this thesis, were explored in greater depth.

2.2.3.2 Marker-based motion capture systems

Marker-based systems can use individual markers placed either directly on anatomical landmarks or on a plate (with three or more markers attached to each plate) that is placed on a body segment. The movement of the markers is recorded by the motion capture system. Marker type and placement have varied in published research (Appendix A). Markers in a marker-based motion capture system can be used for 1) both segment definition and tracking, 2) tracking only, 3) segment definition only, and 4) virtual marking for both tracking and segment definition. Typically, a marker set is derived using a combination of these approaches.

A marker's location in 3D space is obtained using a pre-defined frame of reference. A global coordinate system (X, Y, Z) is defined. Then, a local coordinate system (x, y, z) is defined using the instantaneous position vectors of at least three markers relative to the global coordinate system. Joint angles are calculated based on the markers attached to the two segments of interest using the local coordinate system. Joint angles of flexion-extension, abduction-adduction, and internal-external rotation are typically of interest.[77]

2.2.4 Kinetics

The study of forces causing movement is called kinetics.[73] The motion of a segment is a result of the forces acting on the segment, which can be external or internal. **External forces** include forces due to gravity, ground reaction or external forces. **Internal forces** include forces due to muscles and ligaments. [78]

2.2.4.1 Link-segment model of measurement

To calculate the dynamics of each segment, the human body can be represented by a link-segment model and free-body diagrams. This requires accurate measurements of segment masses, center of masses, joint centers, and moments of inertia. Common parameters are described where *m* is the center of mass of each segment, *I* is the moment of inertia about the center of mass, *Rx,Ry* is a resultant force at each joint, and *M* is the resultant moment between two segments. The assumptions to develop such a model are described in Appendix B.

2.2.4.2 Kinetic devices: force platforms

Devices developed to measure the forces exerted by the human body use force transducers, which produce an electrical signal in proportion to an applied force. Different types of force transducers include strain gauge,[79] piezoelectric sensors,[80] piezoresistive sensors, and capacitance gauges.[81] A combination of force transducers can be used to measure forces and moments of force in multiple directions along three axes.

Force platforms are common, commercially available devices used for force-sensing of foot placement. Two common force platforms include a flat plate supported by either a centrally instrumented pillar or by four triaxial transducers. Force platforms can measure the 3D components of a single equivalent force applied to its point of application, also known as center of pressure, as well as vertical forces. **Center of pressure** is defined as the centroid of all external forces acting on the plantar surface of the foot.[82] Force platforms are also classified as single-pedestal or multi-pedestal models. Multi-pedestal models use more than one force platform.

2.2.5 Spatio-temporal Measurement

The study of movement can also be described in terms of distance (spatial; step length, stride length, and step width) and time (temporal; toe off times) qualities. Commonly, both of these parameters are included in analysis of movement.

2.2.5.1 Spatio-temporal devices

Spatio-temporal devices can measure the distance travelled or the time elapsed. Devices that measure distance can be described as **contact** (e.g., tape measure,[83] surveyor's wheel, meter stick[84]) and **non-contact** devices (e.g., electronic distance meter, laser rangefinder,[85] GPS[86]). Devices to measure time include stopwatch,[87] timing gates,[88] electric clock, and atomic clocks. Commonly commercially available devices, such as accelerometers,[89] integrate both distance and time devices together.

2.2.6 Human gait

Human gait is cyclic in nature with a repetitive pattern.[90] As such, the sequences for walking can be described by specific parameters.

2.2.6.1 Gait Cycle

The gait cycle consists of two phases – stance and swing. The stance phase, when the foot is in contact with the ground, represents the first 60% of the gait cycle.[91] The main components of stance include heel strike, mid-stance and toe-off. The swing phase makes up the remaining 40% of the gait cycle and consists of initial swing, mid-swing and terminal swing. Typically, maximum flexion-extension moment loads of the knee occur during level walking after heel strike in loading response, with estimations of three to four times the body weight during slow and normal walking.[92] However, variations in gait style can change peak knee loads.[93] Variations in gait style occur due to changes in the location of the center of mass and base of support, shifting of posture, and an increase/decrease in toe clearance during swing. Further, changing stride width and length, muscular strength and functional mobility have also caused changes in gait style.[94]

2.2.6.2 Gait parameters

During human gait, many parameters can be collected from the hip, knee and ankle. Herein, the focus was knee kinematics, knee kinetics, and kinematic-derived spatio-temporal parameters.

Kinematic gait parameters of the knee

A common kinematic parameter (Appendix C) is knee range of motion (ROM). Knee ROM is the angular distance between the femur and shank segments. There are three types of ROM: passive, active and functional. Passive ROM is the degree to which the knee may be moved passively to the endpoints in the range of motion without muscle contraction. Active ROM is the degree to which the knee can be moved by muscle contraction. Functional ROM is the minimum degree to which the knee can be moved to perform activities of daily living. Normal knee ROM is considered to be approximately 135 degrees.[95,96] Measuring knee ROM is important because total knee ROM can correlate to functional performance while ROM restrictions can occur due to injury or previous surgery.[97] Most functional activities such as stair climbing and ambulation require approximately 117 degrees of active ROM.[95,98] Another useful measure is knee excursion, which indicated the amount of free knee joint angulation used during an activity and calculated by subtracting the minimum value from the maximum value.[99] Further, knee ROM can also be described as direction specific such as maximum knee extension and maximum knee flexion.

Other common kinematic parameters can also be useful when analyzing gait. Peak knee flexion angle can measure toe clearance during swing as well as the ability of the quadriceps to absorb force. This can be useful to determine the impact of various conditions on knee flexion. For example, in knee OA, knee flexion is commonly reduced in loading response, and a greater magnitude of knee flexion occurs during mid-stance gait period.

Knee kinematics are also measured in terms of abduction-adduction angles and internal-external rotation angles throughout the gait cycle. These are also of clinical interest as peak knee adduction angle is a common measure for knee OA, with adults with knee OA having greater adduction angles than those without knee OA. Similarly, internal-external rotation angles are measures of the "screw-home" mechanism of the knee as well as measures of knee instability; adults with knee OA commonly have a neutral position compared to normal/healthy adults, who have more internal rotation.[100]

Kinetic gait parameters of the knee

The most common kinetic parameters (Appendix C) include (1) knee adduction moment, which rotates the tibia medially on the femur, and the (2) knee flexion moment, which is a measure of the compressive force across the tibiofemoral joint when the quadriceps contract.[101] External peak knee adduction moment is a measure of medial compartment loading and progression of knee OA, with knee OA having a higher external peak knee adduction moment compared to normal/healthy adults. Peak knee extension moment and flexion moment are also measures of medial compartment loading, and knee OA, with adults with knee OA having reduced internal peak knee extension moment and flexion moments.[102] Further, those with increased BMI have lower internal knee extension moment in late stance.[103] Finally, changes in knee internal-external moments may be indicative of degenerative articular cartilage.[104]

Spatial temporal gait parameters

Spatial (distance) parameters of gait include step length and stride length, which can help determine if a mobility issue is unilateral or bilateral. For example, a decrease in step length may occur on only on the affected side. Muscle weakness (e.g., hamstring muscle bracing), manifested by the muscle not allowing the knee to fully extend, could occur unilaterally (step length) or bilaterally (stride length).[105,106]

Temporal (time) parameters include: (1) cadence, (2) speed and velocity, (3) single limb support and (4) double limb support. Cadence measures ambulatory activity while speed/velocity can measure patient's joint issues or broader health status, with slower velocities found in those wanting to decrease joint forces and moments or unable to generate higher forces and moments due to muscle weakness.[47,107,108] Importantly, speed/velocity can also influence step/stride length and overall kinetics. Single and double limb support can also indicate a joint issue or more global health status. People with joint problems or in poorer health, spend less time in single limb support and more time in double limb support than their healthier counterparts.[109] In knee OA, it is commonly seen that people have reduced walking speed, and shorter stride length compared to healthy individuals.[110] A summary of the spatio-temporal gait parameters is provided in Appendix D.

2.2.7 Factors of interest affecting gait

Two factors that affect gait speed and stability that are of particular interest in this project are obesity and OA.[53,108] The first line of management of symptomatic knee OA (i.e., pain and functional limitations) is conservative therapy such as exercise, pharmacotherapy, bracing and/or weight management. However, if non-operative treatment is not effective, TKA is a viable and effective surgical option. Current evidence is limited on the impact of obesity on the biomechanics of gait after TKA. Gait is modified in those living with obesity, and those with OA, including those who undergo TKA relative to healthy, normal weight individuals, but the relationship amongst these variables is complex and requires further investigation. For example, a cross-sectional study of 157 participants reported that participants with a higher BMI and knee OA had greater peak knee compressive forces ($p=0.0006$) and greater shear forces ($p=0.004$) independent of knee alignment and that external knee adduction alignment was associated with greater external peak knee adduction moments ($p<0.0001$), independent of BMI.[111]

2.3 Obesity

2.3.1 Prevalence and measurement

Obesity is abnormal or excessive body fat accumulation that typically poses a health risk to an individual.[56] The most common method used to measure obesity is BMI (weight/ height^2). Skinfolds, waist-to-hip ratios and waist circumference are less commonly used in current published evidence.[112–114] Body mass index does not always reflect an individual's physical fitness as it only considers their weight and height ratio; thus, BMI does not differentiate between people with high body fat and those with high muscle mass.[115,116] Further, it also does not differentiate between different levels of adiposity based on age and sex.[117] Thus, BMI can overestimate adiposity in some instances whereas underestimate in others. The BMI ranges have been developed as an indicator of disease. The ranges are as follows: underweight (<18.5 kg/m^2), normal weight (18.5-24.9 kg/m^2), pre-obesity (25.0-29.9 kg/m^2), obesity class I (30-34.9 kg/m^2), obesity class II (35.0-39.9 kg/m^2) and obesity class III (>40 kg/m^2).[117,118] Body mass index remains the most commonly reported measure of obesity in published research evidence and, for the proposed work, BMI was used as to classify obesity.[115,118,119]

According to the World Health Organization in 2014, 13% of the world's adult population (11% men and 15% of women) were obese (based upon BMI), with the prevalence of worldwide obesity more than doubling between 1980 and 2014.[120] In Canada, the prevalence of adults who are overweight or living with obesity is 25.4%, a prevalence rate increase of 17.5% since 2003.[121,56] In a trend analysis of BMI data in Canada from 2000 to 2011,[122] it was found that the percent change of those with obesity increased by over 15%, with larger increases in obesity occurring in the higher classes of obesity. For example, the percent increase in adults living with class II obesity over the 10-year period was 28.6%. This suggests that not only are more Canadians becoming obese, but that obesity in the higher classes of obesity is substantially increasing.[121]

2.3.2 Associated chronic conditions

Obesity is associated with numerous health conditions and morbidities. Associations have been found between obesity and the incidence of cardiovascular disease (e.g., heart disease, stroke, coronary artery disease, congestive heart failure), diabetes, musculoskeletal disorders (e.g., OA), cancer (e.g., endometrial, breast, ovarian, prostate, liver, gallbladder, kidney, and colon), lower back pain, asthma and inflammation.[120,121,123124] Moreover, obesity leads to poor psychosocial health such as poor quality of life[125], negative self-image, and weight stigma by others.[112,126–128]

2.3.3 Biomechanical effects

The gait characteristics of adults living with obesity as compared to those who are normal/overweight have not been fully investigated,[48,129,130] particularly in those with knee OA. Limited evidence supports that adults with obesity have significant differences in kinematic, kinetic, and spatio-temporal parameters compared to normal/overweight adults (Appendix E).[48,105,131–145]

Gait evaluation of kinetics suggests that individuals with obesity have increased ground reaction forces[46,138,141,146,147] (more specifically, two to three times the normal force between the ground and the individual[148]), greater internal knee extension moment,[108] greater internal rotation moment in early stance,[108] greater instantaneous vertical loading rate,[132] and greater cumulative knee adductor load[108] compared to those of normal weight. Some studies have normalized moments to body weight, which obscures some of the difference in non-normalized moments at the knee between those with obesity and those of normal weight.[141] Individuals living with obesity also have reduced knee ROM during stance,[67,149–151] less knee flexion angle during heel strike,[108] greater knee adduction angle in early stance,[46,108] and less knee abduction angle at terminal stance.[108] Finally, individuals living with obesity have shorter stride length,[46,48,108] slower walking velocity,[48,49,108] greater step width (due to need for increased base of support or increased soft tissue in the thigh),[46,48,49] lower cadence,[49] longer stance phase (increased time in double support phase) and shorter swing phase.[141,147,152,153]

All of these gait modifications may be an attempt for the individual living with obesity to increase overall stability.[147,153] For example, during stance, the hip and knee are less flexed in the flexion-extension plane in those with obesity than those of normal weight. Further, to increase stability in the abduction-adduction plane, those with obesity may increase step width to reduce postural sway by increasing their base of support.[154–158] Others have suggested that these gait modifications may also be an attempt to reduce the muscle activity (metabolic cost) required to keep the body upright, thereby altering the pressure and force through the body.[50,147] Individuals with obesity who display these altered gait parameters may be at high risk of developing biomechanical pathology or knee OA.[50,147] Despite the substantial work done in people with obesity, albeit much of it performed in people with lower classes of obesity, there is much less evidence about the impact of obesity in people with concurrent knee OA or those who undergo TKA.

2.4 Osteoarthritis

2.4.1 Definition/diagnosis

Osteoarthritis is a chronic and progressive[159] degenerative joint disease[160,161] in which cartilage and bone degenerate, causing inflammation, pain, injury, loss of function and impaired repair responses.[5,162,163] Weight bearing joints (hips, knees, spine) and the hands are most commonly affected.[164] Osteoarthritis is accompanied by pain and disability, which negatively affects quality of life.[5] According to recent OA guidelines,[165] the diagnosis of OA requires an assessment of (1) the impact of pain on daily functioning, (2) pain severity, onset, duration, location, spread, quality, interference, triggers, type of pain, (3) ongoing pharmacological and non-pharmacological treatments, (4) current inflammation and joint damage, and (5) pain-related biological, psychological and social factors. More specifically, the diagnosis of knee OA, according to both American College of Rheumatology and European Union League Against Rheumatism, requires: (1) pain and tenderness, (2) crepitus on knee motion, (3) age of 40 years or older, (4) radiography that shows joint space narrowing, osteophytes, subchondral bone sclerosis, and subchondral bone cysts, and (5) synovial fluid with a white blood cell count less than 2000/mm.[3,166,167]

2.4.2 Epidemiology/risk factors

Osteoarthritis has a multifactorial etiology with systemic and local mechanical factors associated with its development.[159,160,168–170171] Systemic factors associated with OA include: older age, female sex, menopause, genetics, poor nutrition and obesity, and low or high bone density. Local mechanical factors associated with OA include: joint damage and deformity, muscle weakness, and repetitive joint loading. Of these factors, previous joint injury and abnormal stresses from the aging process have the strongest associations with the presence of OA.[160]

Obesity is, however, an established risk factor in the development and progression of knee OA,[9–13,172] with the risk of developing knee OA increasing 1.3-6.0 fold in obese versus non-obese people.[173–175] Others have suggested that obesity is also a causal factor for accelerating the progression of knee OA.[176,177] Obesity causes a shift in the loading compartment of the knee, thereby increasing joint friction between the tibia and femur and accelerating knee degeneration.[178] These changes due to obesity can result in excessive axial loads and higher sheer and compressive forces[137,179] that degenerate knee joint structures (bone mineral density, cartilage[180–183]) by creating bias loading towards the medial aspect of the knee, especially in adducted alignment.[184,185]

2.4.3 Gait abnormalities

Knee OA can affect both joint kinetics and kinematics. A common measure of force through the knee for evaluating risk of knee OA is the external knee adduction moment. The external knee adduction moment is a surrogate measure for the medial tibiofemoral contact force; as it increases in magnitude, the joint loading becomes greater in the medial compartment of the knee.[186,187] Particularly important is the external peak knee adduction moment as it has been associated with the progression of OA as well as its severity.[188,189]

Abnormalities in the flexion-extension plane with knee OA include 1) increased knee flexion angles and internal moments during stance phase, which are associated with anterior knee pain;[63,190] and 2) reduced knee flexion after heel strike, impairing shock absorption of impact loading by the quadriceps.[191] Reduced knee flexion may be a consequence of quadriceps avoidance gait, a mechanism developed to minimize pain while walking, and that may persist post-TKA.[63,67] Quadriceps avoidance gait is depicted when there is an internal flexion moment during single support or a lack of change between internal flexion and extension moments throughout the stance phase.[192] Although movement in the abduction-adduction and flexion-extension planes appears associated with OA disease progression,[53] less is known about the impact of knee OA in the internal-external rotation plane. It appears that those with mild knee OA have neutral position compared to those who are healthy have internal tibial rotation.[100,178]

The impact of obesity on gait parameters also appears to be affected by the presence and severity of knee OA. On average, older adults (mean 70±7.8 years) with end-stage knee OA have restricted knee flexion during stance, which is manifested by limited overall knee ROM during the gait cycle, lower internal flexion moment during the first half of stance, lower magnitude of overall internal flexion moment, and a higher external adduction moment compared to asymptomatic adults.[193] The progression of knee OA has sometimes been found to be associated with higher joint loading[137,178] and adduction-adduction knee alignment.[194,195] Less is known about how gait changes after a TKA is performed for the treatment of end-stage OA.

2.5 Total Knee Arthroplasty

Total knee arthroplasty is an elective surgical procedure that replaces deteriorated structures (bone, cartilage, ligaments) of the knee with tibial and femoral implants and sometimes patellar resurfacing or implant. It is a cost-effective procedure that provides pain relief and functional gains in patients with severe knee OA.[196–198]

2.5.1 Prevalence

Although individuals older than 55 years are currently the largest group of individuals receiving TKA in Canada, a recent trend suggests that patients younger than 55 years will account for almost one-third of arthritic cases by 2030; this younger age group may be the fastest growing group requiring a TKA.[199] The demand and volume for TKA is increasing exponentially worldwide.[32,200] In 2016-2017, 67,169 TKAs were performed in Canada, an increase of 16% since 2012-2013.[24] In the United States, TKA is among the most common major surgical procedure performed, in part, due to the aging population.[200] In the year 2012, almost 2/3 of patient cases for over 650,000 TKAs performed in the USA were for females, which also aligns with the greater incidence of OA in the female population.[201,202] In Canada, reasons for increases in TKA also appear associated with increasing prevalence of obesity, longevity of life, and improved surgical technique.[43,203]

2.5.2 Gait

Knee OA has been associated with gait abnormalities for which TKA provides a treatment option.[53] TKA can improve tibiofemoral loading by decreasing medial compartment load and reducing adduction-abduction motion.[53,204,205] Improving knee alignment is important for knee loading and implant survival.[206,207] Evidence from a systematic review of 19 articles suggested that TKA led to increases in knee maximum flexion angle.[53]

The biomechanics between those who receive a TKA compared to those who have non-symptomatic OA remains different, and limited evidence provides mixed reviews as to the extent that TKA improves the biomechanics of gait (Appendix F). In one systematic review, authors noted that individual studies were difficult to compare due to methodological differences and heterogeneity of gait parameters evaluated.[52] For example, Komnik et al., 2015, systematically reviewed 87 articles to evaluate kinematic and kinetic parameters in the post-TKA period as well as examining methodological approaches.[54] Knee adduction moments were measured in 29 (33.3%) articles and the vertical ground reaction force was measured in 31 articles (35.6%). Level walking was performed in seven articles (8%) and 35 (40.2%) articles assessed at least two joints. This level of heterogeneity among studies makes it difficult to determine the degree to which TKA can impact gait.

However, a systematic review of 11 studies and 268 participants indicated that knee ROM was reduced during the gait cycle, including knee flexion during swing in those who were six-months after TKA relative to those with asymptomatic knees across most studies.[52] Further, three systematic reviews reported reduced knee ROM, reduced knee flexion during stance, and altered knee kinetics after TKA relative to healthy adults.[52,53,67] In addition, several studies investigating post-TKA adults reported that increased knee flexion angles and internal knee flexion moments during stance phase were associated with implant failure[208,209] and anterior knee pain[63,190] in those with stable implants compared to those with unstable implants.

A longitudinal study of 42 individuals undergoing TKA who underwent gait analysis at one week pre-TKA and one year post-TKA found that the overall knee flexion angle, early stance internal knee flexion moment and late stance internal knee extension moments all increased, while overall and mid stance external knee adduction moment magnitudes decreased after TKA.[210] Furthermore, they noted that medial knee joint loading was reduced, and that most gait parameters moved towards normal knee patterns, with the exception of the rotation moment post-TKA.[210]

Another study of 103 individuals with TKA reported that many participants' gait pattern did not improve after their TKA surgery.[211] They found that, while only 1 out of 29 individuals were within normal range for knee stance flexion preoperatively, this only improved to 9 out of 28 individuals at 12 months post-operatively. These authors suggested that gait assessment could potentially be used to guide post-operative rehabilitation in an attempt to improve gait parameters.[211] These varying results suggest that further evaluation of gait post-TKA is still required.

2.6 Total Knee Arthroplasty, Obesity and Gait

2.6.1 Prevalence and risk factors

As the number of individuals with obesity and OA increases, it is expected that the use of total joint arthroplasty will become more prevalent,[212,213] with the estimated TKA volume increasing more than the total hip arthroplasty volume.[214] In a cohort study of 7,512 individuals, Leyland et al., 2016 found that individuals who were overweight or obese, had a higher hazard for requiring TKA compared to normal weight BMI individuals. Hazard ratios (HR) for requiring TKA ranged from 1.41 (95% CI 1.27–1.57) for overweight individuals (BMI 25.0 to 29.9 kg/m^2) to 2.67 (95% CI 2.34–3.04) for those with class III obesity (BMI > 40 kg/m^2) when they were compared to those of normal weight (BMI 18.5- 24.99 kg/m^2).[172] Further, they found that the population attributable risk of obesity for knee OA–related TKA was 31.0%.

Currently, surgeons performing a TKA consider ligamentous instability, axis deviations[215] and avoidance of implant malalignment as they prepare for TKA.[216] However, when performing TKAs for individuals with obesity, these technical challenges are compounded by higher rates of wound healing complications,[213] superficial and deep infections,[13,34,217–219] higher revision and overall complication rates,[34,220,221] increased length of hospital stay,[222] and poor functional outcomes.[223] Thus, many surgeons have pre-determined a BMI cut-off whereby individuals above this cut-off score are strongly discouraged or even refused to undergo TKA until their BMI is optimized.[224,225] Yet many individuals with obesity commonly believe that a TKA is crucial for weight loss as their pain and loss of knee function limit their ability to exercise and lose weight while awaiting surgery.[226]

2.6.2 Gait by BMI Classification

Some studies investigating the effect of BMI on the outcomes of TKA have considered BMI a dichotomous (classifying individuals into obese or not using the BMI cutoff of $30kg/m^2$),[216,227] as opposed to a continuous variable. However, other studies have stratified obesity by BMI classes to discriminate amongst patients based on severity of obesity, with those with *morbid obesity* and *super obesity* more likely to experience complications compared to those with lower levels of obesity.[228,229] There is a paucity of literature that has investigated how obesity affects the gait of individuals undergoing TKA, when considering higher classes of obesity. Limited evidence is summarized below.

2.6.3 Class I obesity

Studies investigating gait and class I obesity (BMI 30.0 to 34.99 kg/m^2) typically have small sample sizes.[216,230] In 21 individuals with class I obesity [mean $33.3kg/m^2$ (SD 1.8)] awaiting TKA, higher absolute vertical ground reaction forces were found compared to overweight individuals; these differences did not remain significant after adjusting for body weight.[230] Further, these authors suggested there are also biomechanical gait differences related to sex differences and body weight in individuals with severe knee OA. It was found that men had higher peak external knee adduction moment, impulse, and peak ground reaction force compared to women.[230] However, the authors acknowledged the limited sample size may have limited their conclusions.[230]

Despite small samples, there is some evidence to suggest that individuals with class I obesity, especially females, walk slower before and after TKA.[231] Boonefoy-Mazure et al., 2017 found that improvements in gait velocity and knee ROM after TKA were similar between individuals with obesity compared to normal/overweight individuals, but also noted that individuals with obesity had a lower postoperative level of gait speed and knee ROM compared to those of normal weight.[216]

2.6.4 Class II and greater obesity

Individuals living with class II (BMI 35.0-39.99 kg/m^2) and class III obesity have not frequently been investigated, which is partly due to concerns regarding soft tissue artifact (based on excess adipose tissue) affecting data collection.[230,232] Bonnefoy-Mazure et al. 2017[216] reported that persons with BMI class II and class III had approximately 15 degrees less knee flexion angle (~40°) at baseline and 10 degrees less knee flexion angle (~42°) one year after surgery during mid-swing compared to those of normal weight. More research on gait before and after TKA in these sub-sets of individuals living with higher classes of obesity is needed.

2.7 Gaps in Knowledge and Anticipated Outcomes

Clear gaps in knowledge related to outcomes in adults with obesity undergoing TKA have been identified. Most research on post-TKA gait biomechanics has not focused on adults with Class II obesity, but rather on healthy or overweight adults. Given the high prevalence of obesity in this patient population, understanding how obesity affects gait is important to determine if there are opportunities to improve postoperative outcomes in those living with obesity. This information could facilitate development of rehabilitation interventions. For example, different joint motion limitations or moment patterns that affect gait between those with/without obesity or between sexes may require targeted rehabilitation pre- and post-TKA, facilitating the development of personalized medicine approaches. Debbi et al., 2015[233] recommended that more studies are needed in the early post-operative period (i.e., within three months of TKA) when patient rehabilitation gait patterns can be most influenced. At three months post-TKA, most patients are transitioning from the acute recovery phase to functional activities, so gait patterns may be less amenable as patients adapt to their usual postoperative gait.

Further, examining recovery by BMI category may indicate the need for setting realistic post-operative expectations regarding lower and slower recovery for those living with obesity. Improved walking is one of individuals' primary expectations after TKA, regardless of obesity.[234] A systematic review of physical therapy effectiveness post-TKA reported that gait re-education interventions, directed at the general TKA patient population were associated with improved long-term walking performance.[235] Thus, it is possible that gait re-education could also be effective if implemented for patients living with obesity. Finally, if forces through the knee joint are significantly increased in people with obesity, 1) surgical approaches or prosthesis designs may require further investigation, or 2) the use of gait aids may be encouraged for a longer postoperative period or permanently on uneven surfaces to offload implant forces and potentially improve implant longevity.

The proposed work explores a current gap in clinical knowledge and provides reasoning to why further research is warranted. This preliminary work will provide more knowledge to assist in developing future studies and potential interventions to improve outcomes following TKA in this growing sub-set of patients.

Chapter 3 Methods and Materials

3.1 Overview

This chapter focuses on the methods and materials used in this study. It includes study objectives, study design, the experimental set up and procedure, data processing and parameter extraction, the data analysis performed, and study constraints.

3.2 Objective

To determine whether 10 adults (5 males/5 females) with class II obesity (BMI 35.0-39.99 kg/m^2) differ in gait patterns on level ground, using 3D motion capture, compared to 10 age- and sex-matched adults who are normal/overweight (BMI 14.5-29.99 kg/m^2) in terms of

a) kinematic and kinetic time series
    a. graphical representation of the knee joint angles over the gait cycle in flexion-extension, abduction-adduction, and internal-external rotation.
    b. graphical representation of the knee moments, both non-normalized and normalized to body weight, over the gait cycle in flexion-extension and abduction-adduction.
b) kinematic parameters
    a. knee joint angles in
        i. flexion-extension (initial, loading, minimum, maximum, range),
        ii. abduction-adduction (minimum, maximum) and,
        iii. internal-external rotation (minimum, maximum).
c) kinetic parameters
    a. knee non-normalized moments (minimum, maximum) in flexion-extension and abduction-adduction.

b. knee normalized moments (minimum, maximum) in flexion-extension and abduction-adduction, and

d) spatio-temporal parameters

We performed these afore-mentioned group comparisons at:

a) Pre-TKA (within 4 weeks before TKA),
b) Post-TKA (12 weeks after TKA), and
c) Change from pre- to post-TKA.

3.3 Methods

3.3.1 Study Design

This was a prospective matched-series study using a convenience sample of eligible participants who provided written informed consent. Participants attended two sessions at the Glenrose Rehabilitation Hospital in the Syncrude Centre for Motion and Balance (gait lab) – one session within four-weeks prior to their scheduled surgery for TKA (hereafter referred to as "pre-TKA") and another session at approximately 12-weeks after TKA (hereafter referred to as "post-TKA"). Ethical approval was obtained in June 2017 from the University of Alberta Ethics review board, ID Pro00070695.

3.3.2 Participants and Recruitment

Participants were recruited from August 2017 to November 2017 at the Edmonton Bone and Joint Clinic. All participants received similar pre- and peri-operative care in hospital and at the clinic as the Edmonton zone follows a standardized perioperative clinical pathway.

Height and weight were measured by clinic staff as part of patients' standardized preoperative care using a calibrated medical scale. A clinic staff member pre-screened potential participants for BMI criteria and then received assent from potential participants to allow the researcher to discuss the study with them. As it was anticipated that little change in weight would occur during the wait time to surgery (1-5 months), the weight recorded at this clinic visit was used to calculate participant BMI and assign participants to their respective groups.

Participants were eligible if they were 1) between 50 to 70 years of age, 2) had class II obesity or were normal/overweight, 3) booked for bicondylar TKA, 4) lived within metro Edmonton, and 5) were able to walk 10 meters independently without walking aids. Exclusion criteria consisted of 1) previous lower limb surgeries within one year of enrollment (including previous joint arthroplasties), 2) did not speak English, or 3) any medical condition preventing functional testing (e.g., neuromuscular disorders).

The researcher explained study requirements and provided potential participants with an information letter to review. All willing and eligible participants provided signed informed consent (Appendix G).

Initially, 10 participants (5 males, 5 females) with a BMI between 35.0-39.99 kg/m^2 (hereafter referred to as the "OB group") were enrolled. Then, each OB participant was matched based on sex and age (within 5 years) to a participant with a BMI between 18.5-29.99 kg/m^2 (hereafter referred to as the "N/OW group").

All further data collection occurred at the Glenrose Rehabilitation Hospital in the Syncrude Centre for Motion and Balance. A sample size of 20 participants was calculated to be sufficient to obtain results that are comparable to other gait studies and to account for a potential attrition rate of 15% (Appendix H).

3.4 Experimental setup

3.4.1 Gait Equipment

Three-dimensional knee joint kinematics were measured using an eight-camera optoelectronic **motion capture system** (Eagle Digital Camera, Motion Analysis Corporation, Santa Rosa, CA, USA) recorded at a collection rate of 120 Hertz (Hz). Kinetics were measured using three **force plates** (AMTI, Watertown, MA, USA) embedded within a three-meter walking surface, recorded at a collection rate of 2,400 Hz and synchronized with the motion analysis system. Real-time collection of motion capture data was completed through the Eva Real-Time Software (EVaRT; Version 5.0). Visual3D software (C-Motion, Germantown, MD, USA) allowed for the calculation of kinematic, kinetic and spatio-temporal parameters. Microsoft Excel (Version 16.12) was used for data output and plotting of kinematic and kinetic time series data.

Calibration of the **motion capture cameras** consisted of: 1) a calibration square equipped with four retroreflective markers (Motion Analysis Corporation) to define the XYZ axes of the global coordinate system, and 2) a rigid 500-mm calibrating wand equipped with three retroreflective markers (Motion Analysis Corporation) for capture of medium to large sized volumes. Based on the manufacturer's specifications, the mean error in the position of the markers captured by the motion capture system was one mm (standard deviation [SD] 1 mm). The motion capture system was pre-installed at the Syncrude Centre for Motion and Balance.

Body segments were tracked using 42 retroreflective markers in a newly derived lower-body marker set modified for participants with obesity derived from Helen Hayes[236] and Dalhousie University's Hatfield et al., 2011[236] (Appendix I). Placement of the markers (Figure 3-1) were as follows:

1) Four separate rigid plates[237] were placed bilaterally on the lateral mid-thigh and mid-shank and secured to the body by elasticized bands. Each plate had four markers attached on top of the elasticized band to ensure the markers would not slip.

2) One rigid plate with four markers was placed posteriorly to the sacrum and attached using an elasticized belt.

3) Eleven individual markers were placed bilaterally, and secured by tape, on the anterior superior iliac spine, greater trochanter, medial and lateral femoral epicondyles, tibial tuberosity, fibular head, medial and lateral malleolus, between the second and third metatarsal, and fifth metatarsal, and calcaneus.

An intra-rater test-retest reliability pilot evaluation was completed on a male test subject (BMI 39.9 kg/m^2) to ensure anatomical landmarks were reliably palpated and that no marker plates slipped (Appendix J). This test showed no differences in kinematic, kinetic, and spatio-temporal parameters between gait sessions.

Figure 3-1. Male (BMI 39.9kg/m^2) with protocol marker set attached.

3.5 Experimental procedure

3.5.1 Calibration

Before the arrival of each participant to the Syncrude Centre for Motion and Balance, the motion analyst/kinesiologist calibrated the motion capture system. A calibration square was used to determine the global coordinate system and to register all camera sensors within the collection volume. This ensured that the system could calculate the triangulation of the markers. Relative distances between cameras, and between the origin of the room and each camera were established. The calibration also ensured that the software system could detect and limit image distortion due to the camera lens. The global coordinate system origin was defined by aligning the square on the corner of the first of three force platform for a five-second trial. The coordinate system was defined as a positive y-axis pointing in the forward progression for walking, positive x-axis towards the right of the participant, and positive z-axis up.

A calibration wand was then used to provide the capture volume of the cameras with the information needed to calculate the 3D position and distortion maps. This calibration procedure was performed within the areas directly above and surrounding the force platforms. The motion analyst ensured that there were adequate data and that the 3D space calculation and distortion were at acceptable levels (~6000 frames ± 10%). The location of the force platforms in the global coordinate system was already established in the Syncrude Centre for Motion and Balance lab's system. The local coordinate system was defined using anatomical landmarks with respect to the individual markers.

3.5.2 Participant measurements

Upon arrival at the Syncrude Centre for Motion and Balance, the participant's body weight was re-measured using a weight scale, with the participants barefoot and wearing spandex shorts that were provided by the researcher. The participant's body mass was used to calculate normalized kinetic measurements.

Markers were then placed on the lower-extremities and pelvis. Marker placement was performed by the primary researcher (CW) at pre- and post-TKA sessions; a kinesiologist with more than 10 years of gait analysis experience verified the marker accuracy. Following marker placement, participants stood in anatomical position for a static calibration trial to: a) capture the relative positions of the markers on their body, b) reconstruct any markers that were occluded during the experimental trial, c) develop template models in EVaRT software to define body segments, and d) label markers automatically across trials and participants. During this preparatory phase, a photograph was taken as a backup for marker locations in the event that any markers detached and required re-attachment, as well as for reference for the post-TKA evaluation.

3.5.3 Walking Trials

Participants were instructed to walk at a self-selected speed for all walking trials, which were performed barefoot on level ground, as per usual gait laboratory procedure. Each participant was required to perform multiple walking trials (median 12 trials, range 6-17 trials per participant). The total walking duration to complete the session was less than 10 minutes, with participants being able to rest as needed between trials.

For each trial, participants walked 10 meters in a straight line on a level walkway across the camera capture volume and three force plates. Three successful walking trials by each participant were selected for data analysis. A trial was successful if (a) two subsequent footsteps had been cleanly placed on two out of the three force plates, (b) their instantaneous gait velocity was continuously within 5% of their average self-selected gait velocity, and (c) there were no visibly obvious alterations to their stride to contact the force plate. If participants had visibly altered gait speed (due to stumbling or lockout of the surgical knee) or did not step completely on the force plate, the walking trial was discarded, and another walking trial performed. In circumstances where participants were only able to step on one force plate per trial due to a short step length, six walking trials were selected for that participant for data analysis. Further, within each trial, three foot strikes on the force plate were required to ensure proper representation of ground reaction force on each foot: two trials from one foot, one trial from the other foot. Lastly, the spatio-temporal gait parameters of speed were calculated from the product of cadence and stride length. Stride length was calculated by the distance between the proximal end position of the foot at ipsilateral heel strike to the proximal end position of the foot at the next ipsilateral heel strike.

3.6 Data Processing and Parameter Extraction

3.6.1 Motion capture data

After the unlabeled x,y,z-coordinate time series data were captured by the cameras, the data were processed using the EVaRT software. The markers were labelled, and the data were then screened and interpolated for missing data. The anatomical labels for each marker were checked manually to ensure that they did not disappear throughout each gait trial. If markers were missing (e.g., due to occlusion) during the data collection period, a cubic spline interpolation operation was performed using data points on either side of the data collection gap.[238] This method required sufficient data before and after the gap to accurately estimate the cubic spline. The spline fill was used if the gap was relatively small (less than 10 missing points). If markers that were part of a rigid body were occluded, a rigid-body fill was performed. This method required that only one of several markers placed on a rigid body were occluded and that the other visible markers could be used to reconstruct the missing marker data. When a significant portion of the data were missing for a marker (greater than 10 missing points), a virtual marker was reconstructed using data from the three markers (either rigid bodies or individual markers) nearest to the missing marker. No filtering was done to the raw data due to 120 Hz being an appropriate sampling frequency from a power spectrum analysis. The complete motion capture data were then exported into a C3D file format.

The C3D file was then imported into Visual3D. A standing calibration trial was loaded into the Visual 3D workspace. A hybrid biomechanical model, with both 6 degrees of freedom and 3 degrees of freedom, was used, which included anthropometric characteristics (segment mass, center of mass location, moments of inertia) and segment modeling (Appendix K). The three gait trials were assigned to the model and explored. Event processing was then completed for each foot to visually ensure that the ground reaction force matched with heel strike and toe-off.

Kinematic and kinetic time series data that had 3D coordinates of each marker in the motion capture system were collected to identify a trend represented by the sequence of observations. Then, angular kinematics (knee flexion-extension [initial, loading, maximum, minimum, range], adduction-abduction [minimum, maximum], and internal-external rotation [minimum, maximum]) were calculated by determining the relative orientation of the proximal segment to the distal segment. These were computed for three complete gait cycles in one trial in one gait session. Angular kinematics were based on a Cardan rotation sequence of x- (flexion-extension), y- (adduction-abduction), and z- (medial-lateral rotation) rotations. The Cardan sequence for x,y,z joint angle calculation was as follows: (1) Right leg: x = lateral, y = anterior, z = up, and (2) Left leg: x = medial, y = anterior, z = up.

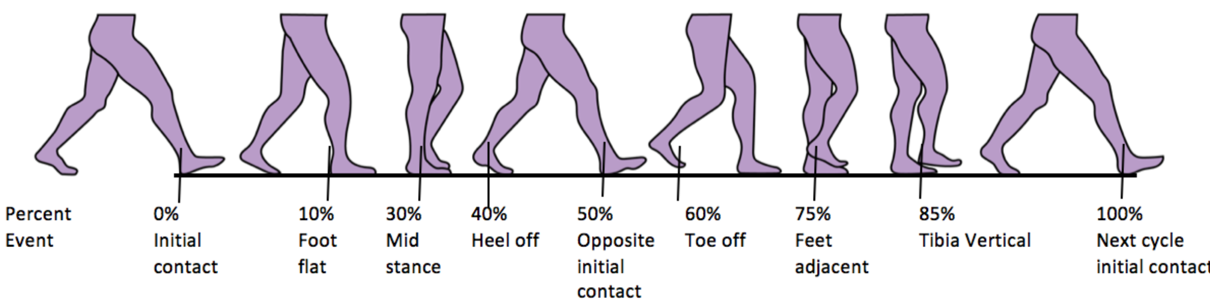
The kinetics of the biomechanical model were determined using inverse dynamic analysis. Inverse dynamic analysis uses the kinematics of the biomechanical model and the location, magnitude, and direction of ground reaction forces. Internal knee joint moments (flexion-extension [minimum, maximum] and adduction-abduction [minimum, maximum]) were obtained using the Newton-Euler inverse dynamics formula.[239] The proximal segment coordinate system was used as the reference for joint moments. Kinetics were calculated for three complete gait cycles in one gait session. Analysis was primarily focused on the stance phase where the knee was weight-bearing, although the entire gait cycle was observed. Knee moments that were not normalized to body weight (non-normalized moments) were presented first as it allowed body mass to be a distinguishing factor between participant groups (i.e., absolute loading on the knee through the implant).[141] Knee moments were then normalized to body weight (Newton meter per kilogram [Nm/kg]) and presented as patterns of loading on the knee. Finally, spatio-temporal parameters (velocity, cadence, stride length) were computed from force platform and kinematic data.

Kinematic and kinetic time series data as well as kinematic and kinetic parameters were then exported to Microsoft Excel for preparation for analysis. The values for the kinematic, kinetic, and spatio-temporal parameters were averaged for three walking trials per participant in Microsoft Excel and tabulated. The participant-averaged data were used for time and group comparisons. Kinematic and external kinetic time series data were graphed into two separate waveforms: 1) individual trials per participant by group, and 2) group means.

3.6.2 Parameters

Kinematic and Kinetic Time Series Data

Kinematic and kinetic time series data were visually described and trends between the study groups were also compared. Kinematic time-series were also described relatively to a range of existing time series data from a population of normal weight adults (BMI between 18.5 kg/m^2 and 24.99 kg/m^2) with no musculoskeletal conditions (normative values). The following phases were defined to aid in identifying a particular part of the gait cycle in Figure 3-2.



Periods	Loading response	Mid-stance	Terminal stance	Pre-swing	Initial swing	Mid-swing	Terminal swing
Phase	Stance				Swing		
Cycle	One cycle						

Figure 3-2. The gait cycle. The occurrence of 8 events from heel contact at 0% gait cycle to the subsequent heel contact at 100% gait cycle.

Gait events and periods

1) Initial contact: The instance of time when the foot/heel makes contact with the ground. Usually seen with an extended knee and neutral ankle.
2) Loading response: When the lower limb is attempting to absorb the shock caused by ground reaction forces and stabilize the lower limb to bear body weight. The knee flexes rapidly.

3) Mid-stance: The knee is being extended with the contralateral foot leaving the ground and continues as the body weight travels along the length of the foot until it is aligned with the forefoot. The function is to stabilize weight bearing.
4) Terminal stance: The time from when the heel rises until the contralateral foot makes contact with the floor. The knee has achieved maximum extension. The function is to continue to stabilize weight bearing and to maximize step length.
5) Pre-swing: The time from when the contralateral foot has initial contact to the ipsilateral toe-off. Body weight is transferred onto the contralateral limb and the knee undergoes passive flexion. The function is for the knee to prepare for swing.
6) Initial swing: The time from the instant the foot leaves the ground until maximum knee flexion occurs- demonstrated by the swinging limb is directly under the body and opposite to the stance limb. The function is for foot clearance for limb advancement.
7) Mid-swing: The time from when the swinging limb is opposite to the stance limb to when the tibia is vertical. The knee is undergoing passive extension and the function is for limb advancement.
8) Terminal swing: The tibia goes beyond perpendicular and the knee fully extends. The function is for limb advancement and preparation for stance.

Kinematics parameters

The following parameters were defined:

- Initial: The angle of the knee in the first frame when the heel is in contact with the floor (0% gait cycle).
- Loading: The angle of the knee in the frame where the forefoot (ball of the foot, just below the 4^{th} and 5^{th} metatarsal heads) is flat in contact with the floor (2-10% gait cycle).

This frame is also identified as the moment when the ankle moves from plantarflexion to dorsiflexion.

- Minimum (min): The smallest angle of the knee within the entire gait cycle.
- Maximum (max): The largest angle of the knee within the entire gait cycle.
- Range: The difference between the maximum and minimum angles, also known as the ROM of the knee.

Clinically significant differences for kinematics were considered at a threshold of ± 5 degrees.[240]

Kinetic parameters

The following parameters were defined:

- Minimum (min): The smallest knee moment within the entire gait cycle.
- Maximum (max): The largest knee moment with in the entire gait cycle.

Clinically significant differences for normalized kinetics were set at a threshold of 0.13 Nm/kg.[240]

Spatio-temporal parameters

The following parameters were defined:

- Velocity: the quotient of the distance traveled by the time.
- Cadence: the quotient of the number of steps taken per minute.
- Stride length: the distance between the position (e.g., heel strike) of one foot to the next similar position in the same foot (e.g. next heel strike).

Clinically significant differences for speed is 0.10-0.20 m/s[241], cadence value of less than 100 step/min[242,243] and 20% change in stride length.[244]

3.7 Experimental Data Analysis

To evaluate group differences in kinematic, kinetic and spatio-temporal parameters at pre- and post-TKA, a Wilcoxon rank-sum test was used to compare group medians and interquartile ranges. The Wilcoxon rank-sum test provided a more conservative estimate due to the small sample size and the potential for data to be skewed. However, as data did meet normality assumptions, group means with standard deviations were also reported.

To evaluate changes over time from pre- to post-TKA between groups in kinematic, kinetic and spatio-temporal parameters, a two-way repeated measures Analysis of Variance (ANOVA) was performed. In these analyses, the 'between subject' factor was group (N/OW or OB) and the 'within subject' factor was time (pre-TKA or post-TKA). The two-way ANOVA analyses were used to determine kinematic and kinetic: (1) main effect of time (i.e., are there differences between pre- and post-TKA parameters?), (2) main effect of group (i.e., are there differences between groups [OB or N/OW]?), and (3) interaction effects (i.e., are there differences in patterns of change between groups over time?).

Values were considered significant at two-tailed $p < 0.05$. If there were no significant values, trends in the data were described qualitatively. If outliers (>1.5 SD from the mean group value) were present, a sensitivity analysis was performed to determine if the outlier data altered the group data. In the sensitivity analysis, outliers were removed, and statistical analysis repeated. Finally, to determine if velocity contributed to the changes in kinematic and kinetic parameters over time, a one-way repeated measures ANOVA with the factor being "group (N/OW or OB)" was used for three trials within each gait session for all participants. All statistical data analyses were performed using SPSS (v.23, IBM Corp, USA) and STATA (v.13, Collage Station, Texas, USA).

3.8 Constraints

There were several possible methodological and external constraints. Methodological constraints included the lack of prior research examining the effect of obesity, particularly class II obesity, on gait biomechanics after TKA. This meant that there was also sparse information on standardized modelling of segments for adults with class II obesity. To overcome this constraint, a case study was performed prior to data collection to test the intra-rater reliability of marker placement and reproducibility of the gait testing protocol (Appendix J).

Potential external constraints included: a) feasibility of recruiting participants, particularly those with class II obesity, in a timely fashion, b) inability for patients waiting for TKA, particularly those living with Class II obesity, to perform the gait assessments as planned (both pre- and post-TKA), c) delays in time to surgery following preoperative gait assessment, and d) postoperative complications delaying patient recovery and preventing their participation in the follow-up gait assessment.

Chapter 4 Results

4.1 Overview

This chapter presents the results obtained from conducting the experiment in Chapter 3. It includes the results regarding recruitment and follow-up as well as the estimation of time series data depicted in graphs. The non-parametric estimates and parametric estimates of the pre-TKA, post-TKA and change from pre- to post-TKA kinematics, kinetics and spatio-temporal parameters are reported.

4.2 Recruitment and Follow-Up

A total of 52 adults between the ages of 50-70 years preparing to undergo TKA were approached to participate in the study. Of these, 20 (38%) participants agreed to participate and provided consent; all participants were recruited within four months (August to November 2017).

Ten participants (5 males, 5 females), in the OB group had a mean BMI of 37.1 kg/m^2 (SD 1.79) and a mean age of 64.2 years (SD 6.04). Another 10 participants were recruited (5 males, 5 females) for the N/OW group, which had a mean BMI of 27.0 kg/m^2 (SD 2.15) and a mean age of 66.8 years (SD 2.86). Group matching for age (within 5 years) and sex were done; thus no group differences existed in age or sex ($p>0.05$). Both groups were assessed a median of 11 days (IQR 6-16) before surgery and the median follow-up assessment was 105 days (IQR 97-110). There was no difference in the length of hospital stay between the two groups (N/OW 3 days (IQR 2-4); OB 2.5 days (IQR 2-4) ($p=0.95$, 95%CI [1.90, 5.56]). All the participant demographic characteristics are presented in Table 4-1.

Seventeen (85%) participants completed the 12-week post-TKA assessment. Two participants (both female sex, 1 from OB group of 59.8 years and 1 from N/OW group of 68.9

years) had complications (knee swelling) post-TKA and were unable to complete their 12-week post-TKA evaluation. Another OB group participant (female sex, 64.0 years) was lost to follow-up. Data from 20 participants were available for pre-TKA assessment (baseline evaluation), but only 17 (8 OB; 9 N/OW) participants were available for 12-week post-TKA evaluation. All participants retained similar weight between recruitment and both the pre- and post-TKA assessments.

Table 4-1. Pre-TKA Demographic Characteristics for Participants by Group.

Characteristic	Pre-TKA			
	N/OW (n=10)	OB (n=10)	95% CI	p-value
Sex (#,% female)	5 (50)	5 (50)		
Mean Age (SD)	66.8 (2.86)	64.2 (6.04)	64.5, 69.0	0.45
Mean Height in meters (SD)	1.69 (0.10)	1.71 (0.09)	1.60, 1.76	0.70
Mean Weight in kilograms (SD)	**76.5 (13.0)**	**112.3 (9.92)**	**66.6, 86.5**	***0.0005**
Mean BMI in kg/m^2 (SD)	**27.0 (2.15)**	**37.1 (1.79)**	**25.4, 28.7**	***0.0002**
Mean Pre-TKA assessment time in days (SD)	9.30 (5.40)	14.1 (10.0)		0.34
Length of Stay (days)	3 (2-4)	2.5 (2-4)	1.90, 5.56	

Note: BMI = body mass index. SD=Standard Deviation. CI= Confidence interval. p-values based on independent t-tests for continuous variables and chi-squared tests for sex. Significant differences (p<0.05) denoted with an asterisk (*) and bolded.

4.3 Time Series Analysis of Kinematics and Kinetics

4.3.1.1 Pre-TKA Knee Joint Angle

Flexion-extension angle of the knee

The time series data for the pre-TKA individual trials and group means (ensembled) of flexion-extension angles of the knee (hereafter flexion-extension angle) are depicted in Figure 4-1 and Figure 4-2. From the ensembled data, both groups showed a similar trend to each other. However, both groups deviated between each other for the entire gait cycle, with the OB group having more knee flexion angle. Further, deviations from normative values in healthy adults (hereafter normative values) include the N/OW group at loading response and the OB group at mid- to terminal stance and terminal swing.

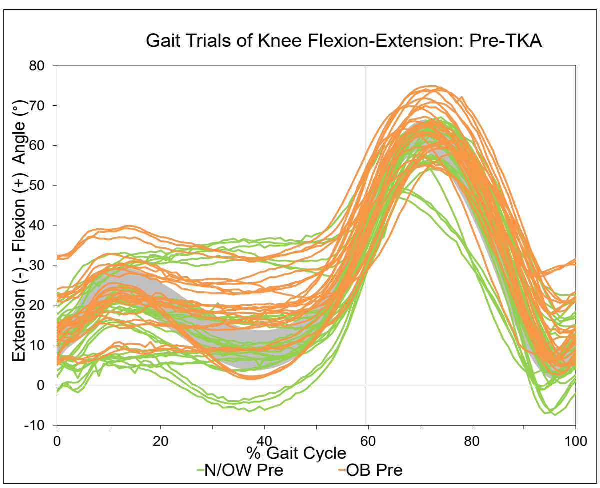


Figure 4-1. The time series of pre-TKA flexion-extension angle of the knee gait trials for N/OW (green) and OB (orange) groups over stance (0-60% gait cycle) and swing (61-100% gait cycle). Different traces represent trial data across participants. Extension and flexion is denoted by negative and positive values, respectively. Grey bands represent normative values in healthy adults.

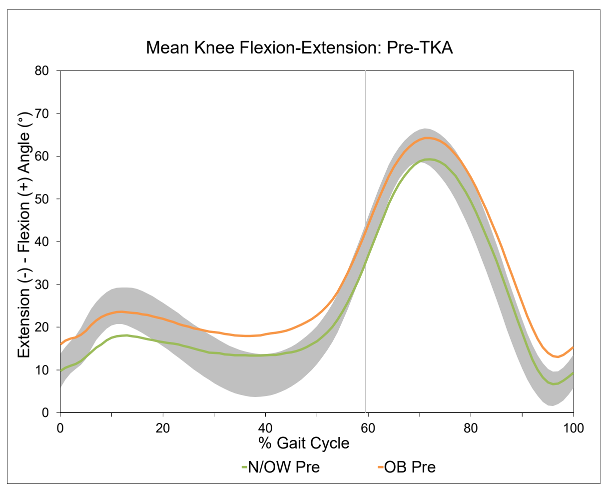


Figure 4-2. The time series of pre-TKA across-participant mean flexion-extension angle of the knee for N/OW (green) and OB (orange) groups over stance (0-60% gait cycle) and swing (61-100% gait cycle). Extension and flexion is denoted by negative and positive values, respectively. Grey bands represent normative values in healthy adults.

Abduction-adduction angle of the knee

The time series data for the pre-TKA individual trials and group means of abduction-adduction angles of the knee (hereafter abduction-adduction angle) are depicted in Figure 4-3 and Figure 4-4. From the ensembled data, both groups showed a similar trend to each other. However, both groups deviated between each other for the entire gait cycle except during mid-swing, with the N/OW group having greater adduction angle. To add, deviations from normative values include the N/OW group at loading response and terminal swing.

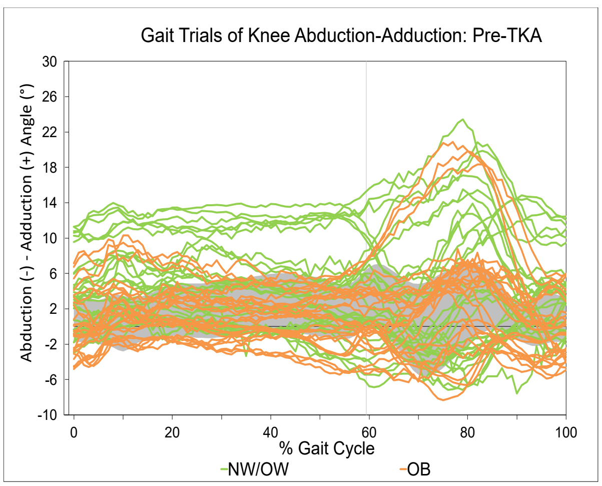


Figure 4-3. The time series of pre-TKA abduction-adduction angle of the knee gait trials for N/OW (green) and OB (orange) groups over stance (0-60% gait cycle) and swing (61-100% gait cycle). Different traces represent trial data across participants. Abduction and adduction is denoted by negative and positive values respectively. Grey bands represent normative values in healthy adults.

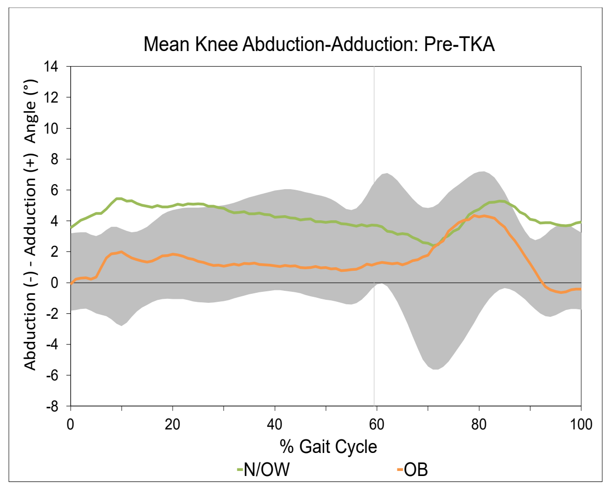


Figure 4-4. The time series of pre-TKA across-participant mean abduction-adduction angle of the knee for N/OW (green) and OB (orange) groups over stance (0-60% gait cycle) and swing (61-100% gait cycle). Abduction and adduction is denoted by negative and positive values respectively. Grey bands represent normative values in healthy adults.

Internal-external rotation angle of the knee

The time series data for the pre-TKA individual trials and group means of internal-external rotation angles of the knee (hereafter internal-external angle) are depicted in Figure 4-5 and Figure 4-6. From the ensembled data, both groups showed a similar trend to each other. However, both groups deviated between each other at mid- to late-stance and during the swing period: the N/OW group had less external rotation angle during mid- to late-stance, but greater external rotation in terminal swing.

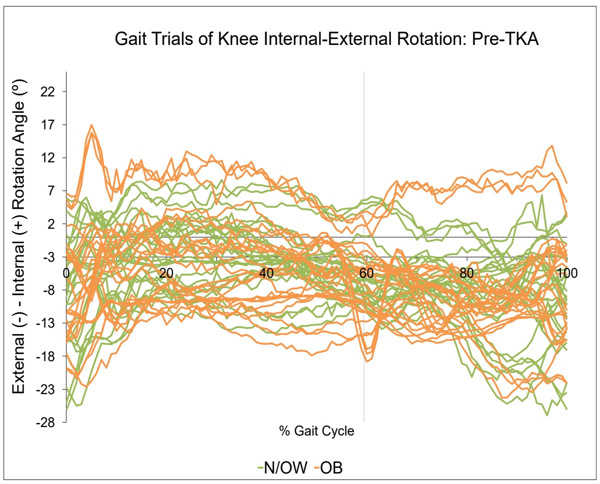


Figure 4-5. The time series of pre-TKA internal-external rotation angle of the knee gait trials for N/OW (green) and OB (orange) groups over stance (0-60% gait cycle) and swing (61-100% gait cycle). Different traces represent trial data across participants. External and internal rotation is denoted by negative and positive values respectively.

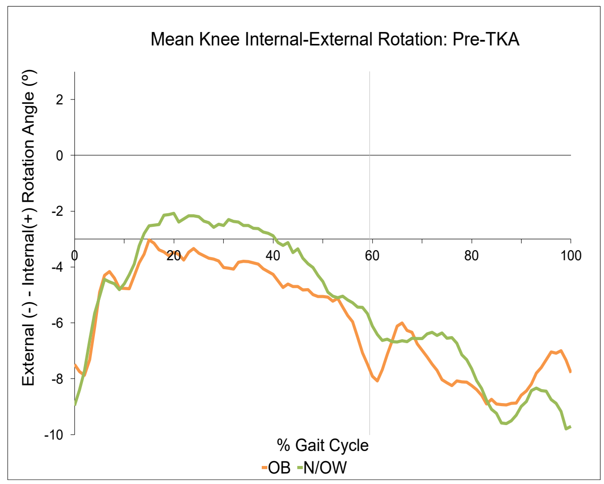


Figure 4-6. The time series of pre-TKA mean internal-external rotation angle of the knee for N/OW (green) and OB (orange) groups over stance (0-60% gait cycle) and swing (61-100% gait cycle). External and internal rotation is denoted by negative and positive values respectively.

4.3.1.2 Post-TKA Knee Joint Angle

Flexion-extension angle of the knee

The time series data for the post-TKA individual trials and group means of flexion-extension angle are depicted in Figure 4-7 and Figure 4-8. From the ensembled data, both groups showed a similar trend to each other but are deviated throughout the entire gait cycle, with the OB group having more knee flexion angle. In addition, deviations from normative values include the OB group at mid- to late- stance and both groups at terminal swing.

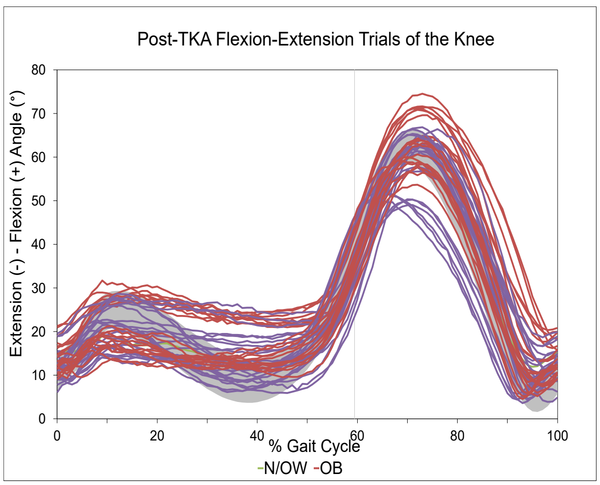


Figure 4-7. The time series of post-TKA knee angle of the knee gait trials for N/OW (purple) and OB (burgundy) groups over stance (0-60% gait cycle) and swing (61-100% gait cycle). Different traces represent trial data across participants. Extension and flexion is denoted by negative and positive values respectively. Grey bands represent normative values in healthy adults.

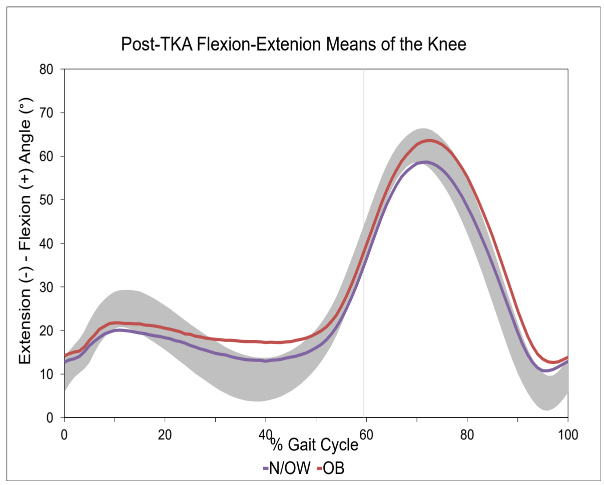


Figure 4-8. The time series of post-TKA across participant mean flexion-extension knee angle of the knee for N/OW (purple) and OB (burgundy) groups over stance (0-60% gait cycle) and swing (61-100% gait cycle). Extension and flexion is denoted by negative and positive values respectively. Grey bands represent normative values in healthy adults.

Abduction-adduction angle of the knee

The time series data for the post-TKA individual trials and group means of abduction-adduction angle are depicted in Figure 4-9 and Figure 4-10**.** From the ensembled data, both groups showed a similar trend to each other. However, both groups deviated between each other for the entire gait cycle except during mid-swing, with the OB group having greater knee adduction angle. To add, deviations from normative values include the OB group at loading response and terminal swing.

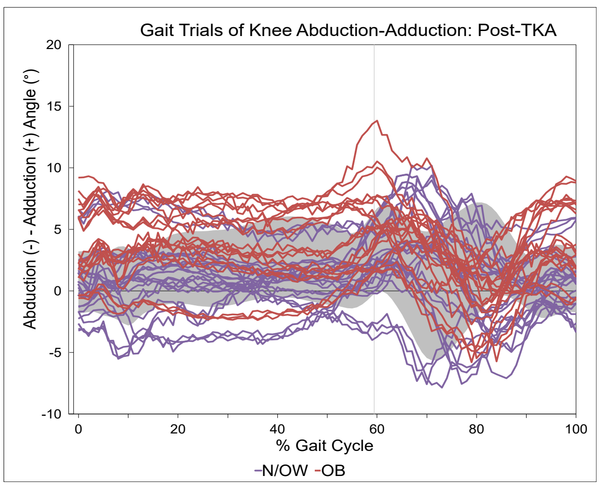


Figure 4-9. The time series of post-TKA Abduction-adduction angle of the knee gait trials for N/OW (purple) and OB (burgundy) groups over stance (0-60% gait cycle) and swing (61-100% gait cycle). Different traces represent trial data across participants. Abduction and adduction is denoted by negative and positive values respectively. Grey bands represent normative values in healthy adults.

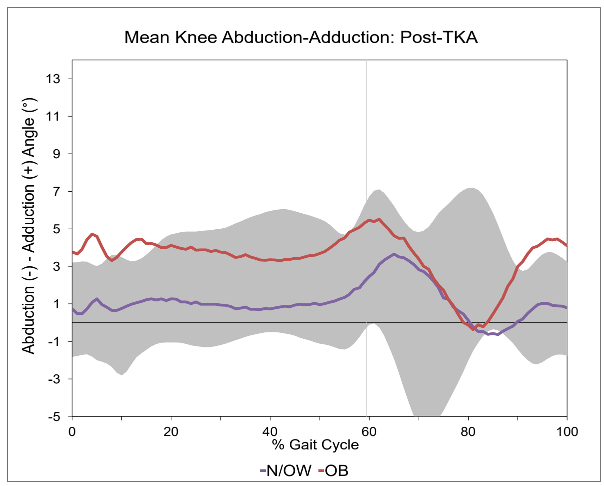


Figure 4-10. The time series of post-TKA across-participant mean abduction-adduction angle of the knee for N/OW (purple) and OB (burgundy) groups over stance (0-60% gait cycle) and swing (61-100% gait cycle). Abduction and adduction is denoted by negative and positive values respectively. Grey bands represent normative values in healthy adults.

Internal-external rotation angle of the knee

The time series data for the post-TKA individual trials and group means of internal-external angle are depicted in Figure 4-11and Figure 4-12. The ensembled data showed that both groups showed a similar trend to each other during stance phase. However, the groups to deviate from each other with the OB group showed less external rotation angle. During the swing period, the OB group showed one external rotation angle peak during terminal swing, whereas the N/OW group showed two external rotation angle peaks during initial and terminal swing.

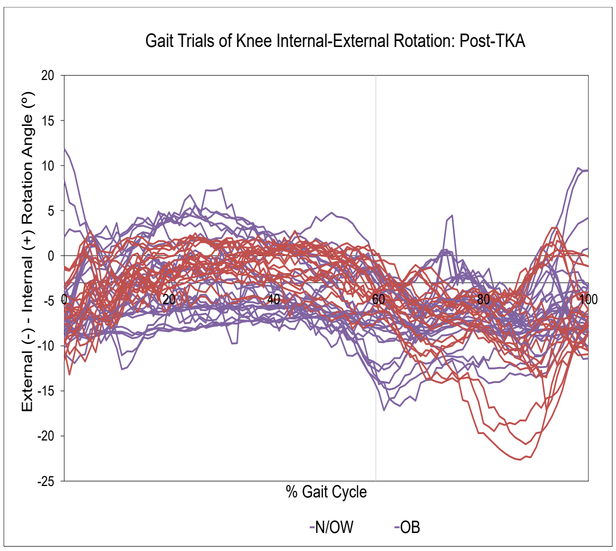


Figure 4-11. The time series of post-TKA internal-external knee angle of the knee gait trials for N/OW (purple) and OB (burgundy) groups over stance (0-60% gait cycle) and swing (61-100% gait cycle). Different traces represent trial data across participants. External and internal rotation is denoted by negative and positive values respectively.

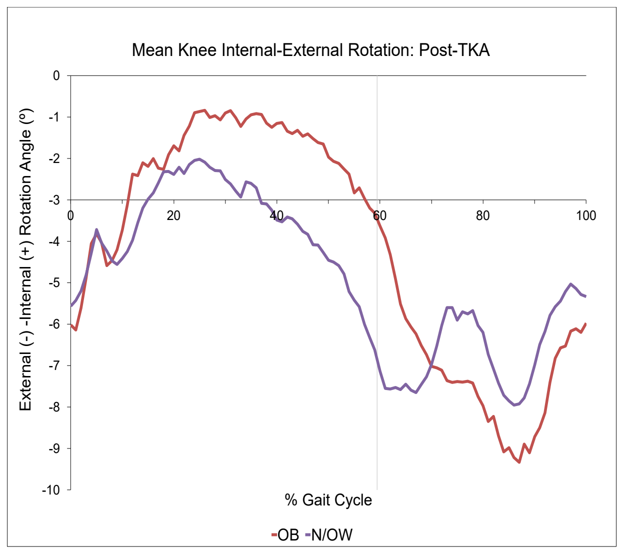


Figure 4-12. The time series of post-TKA across-participant mean internal-external rotation angle of the knee for N/OW (purple) and OB (burgundy) groups over stance (0-60% gait cycle) and swing (61-100% gait cycle). External and internal rotation is denoted by negative and positive values respectively.

4.3.1.3 Change from Pre- to Post- TKA Knee Joint Angle

Flexion-extension angle of the knee

The time series data for the change from pre- to post-TKA group means of flexion-extension angle are depicted in Figure 4-13**.** All evaluations showed a similar trend to each other and to normative values. However, the OB group showed less change in flexion-extension from pre- to post-TKA whereas the N/OW group moved more towards normative values in the loading response gait period from pre- to post-TKA. Deviations from normative values include the pre- and post-TKA OB groups at mid- to terminal stance and terminal swing and the pre-TKA N/OW group at loading response.

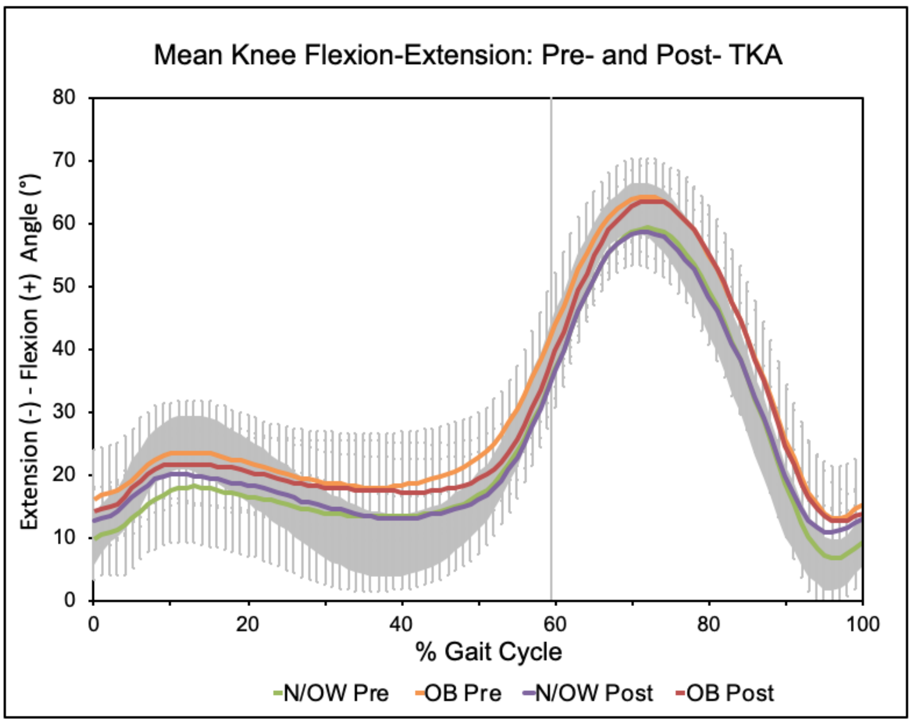


Figure 4-13. The time series of across-participant mean flexion-extension angle of the knee for N/OW pre- (green), OB pre- (orange), N/OW post- (purple) and OB post- (burgundy) TKA over stance (0-60% gait cycle) and swing (61-100% gait cycle). Extension and flexion is denoted by negative and positive values respectively. Grey bands represent normative values in healthy adults. Error bars denote one standard deviation.

Abduction-adduction angle of the knee

The time series data for the change from pre- to post-TKA group means of abduction-adduction angle are depicted in Figure 4-14. The results show differing trends between group from pre- to post-TKA; the OB group transitioned from a less adducted knee pre-TKA to a more adducted knee post-TKA, whereas the mean N/OW group transitioned from a more adducted knee pre-TKA to a less adducted knee post-TKA. Deviations from normative values include the pre-TKA N/OW group and the post-TKA OB group at loading response and terminal swing.

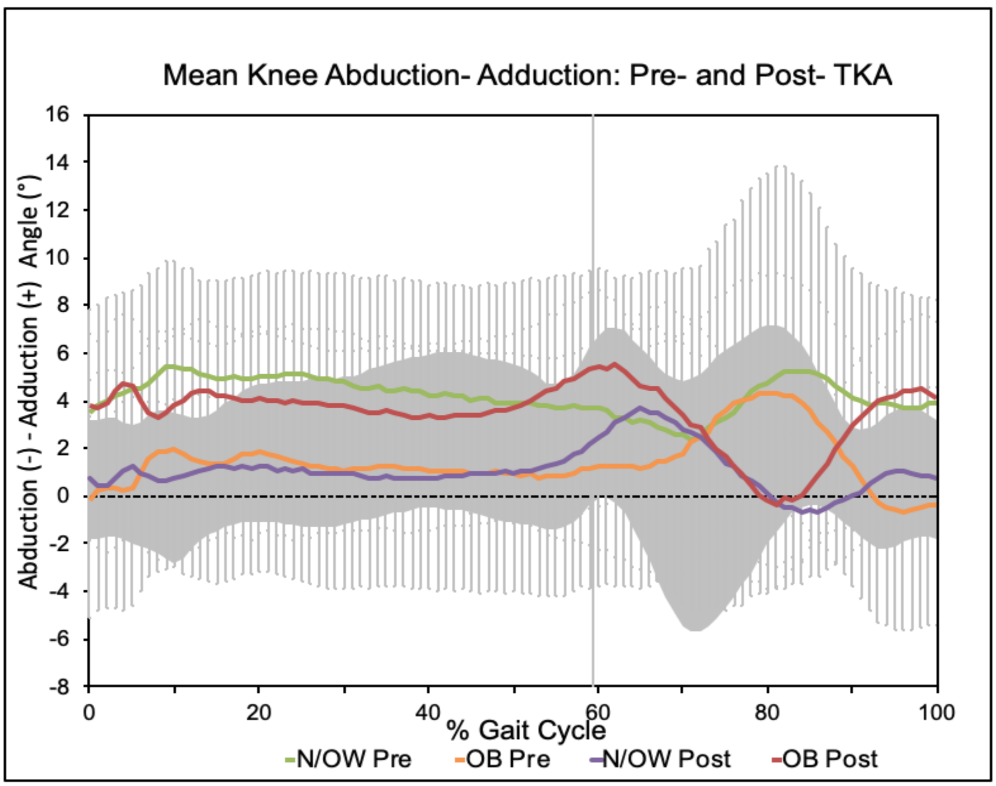


Figure 4-14. The time series of across-participant mean abduction-adduction angle of the knee for N/OW pre- (green), OB pre- (orange), N/OW post- (purple) and OB post- (burgundy) TKA over stance (0-60% gait cycle) and swing (61-100% gait cycle). Abduction and adduction is denoted by negative and positive values respectively. Grey bands represent normative values in healthy adults.

Internal-external rotation angle of the knee

The time series data for the change from pre- to post-TKA group means of internal-external angle are depicted in Figure 4-15. The N/OW group trend is very similar both pre- and post-TKA in both stance and swing. In contrast, the OB group moved towards less external rotation in stance pre- to post-TKA.

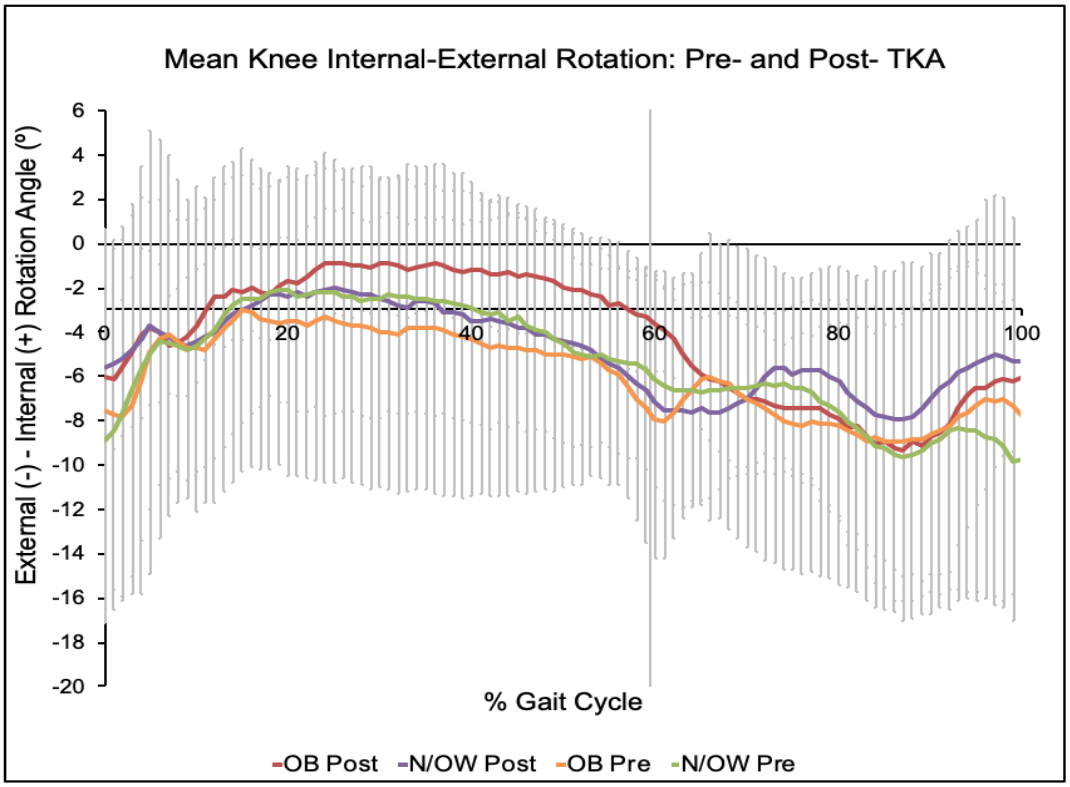


Figure 4-15. The time series of across-participant mean internal-external angle of the knee for N/OW pre- (green), OB pre- (orange), N/OW post- (purple) and OB post- (burgundy) TKA over stance (0-60% gait cycle) and swing (61-100% gait cycle). External and internal rotation is denoted by negative and positive values, respectively. Error bars denote one standard deviation.

4.3.2.1 Pre-TKA Knee Non-Normalized Moment

Flexion-extension knee non-normalized moment

The time series data for the pre-TKA individual trials and group means of flexion-extension non-normalized knee moments (hereafter flexion-extension non-normalized moments) are depicted in Figure 4-16 and Figure 4-17. From the ensembled data, both groups show a similar trend to each other in stance phase but are deviated, with the OB group had a greater flexion non-normalized moment throughout. However, in the swing phase, the OB group had a greater extension non-normalized moment in terminal swing compared to the N/OW group.

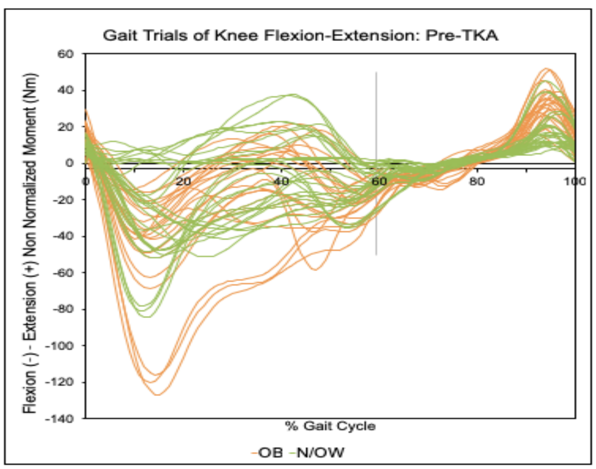


Figure 4-16. The time series of pre-TKA flexion-extension moment of the knee gait trials for N/OW (green) and OB (orange) groups (non-normalized) over stance (0-60% gait cycle) and swing (61-100% gait cycle). Flexion and extension is denoted by negative and positive values respectively.

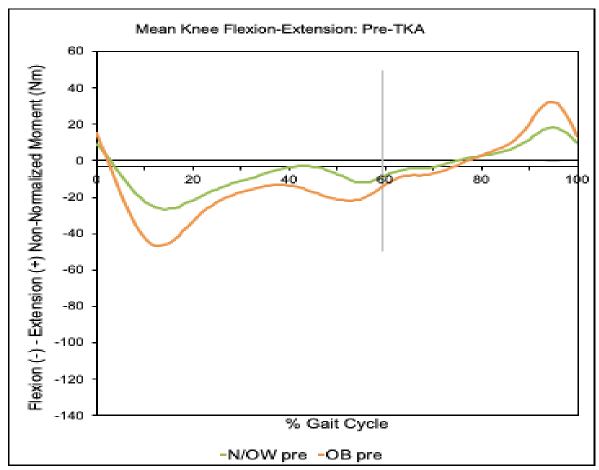


Figure 4-17. The time series of pre-TKA across-participant mean flexion-extension moment of the knee for N/OW (green) and OB (orange) groups (non-normalized) over stance (0-60% gait cycle) and swing (61-100% gait cycle). Flexion and extension is denoted by negative and positive values respectively.

Abduction-adduction knee non-normalized moment

The time series data for the pre-TKA individual trials and group means of abduction-adduction non-normalized knee moments (hereafter abduction-adduction non-normalized moments) are depicted in Figure 4-18 and Figure 4-19. The ensembled data showed that both groups showed a similar trend to each other except deviating in stance with the OB group having greater adduction non-normalized moment.

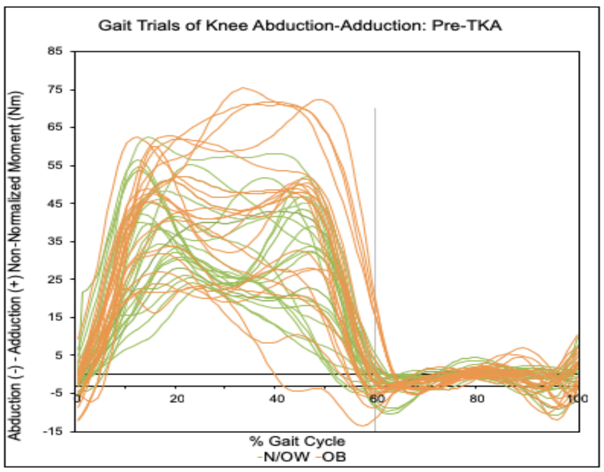


Figure 4-18. The time series of pre-TKA abduction-adduction moment of the knee gait trials for N/OW (green) and OB (orange) groups (non-normalized) over stance (0-60% gait cycle) and swing (61-100% gait cycle). Abduction and adduction is denoted by negative and positive values respectively.

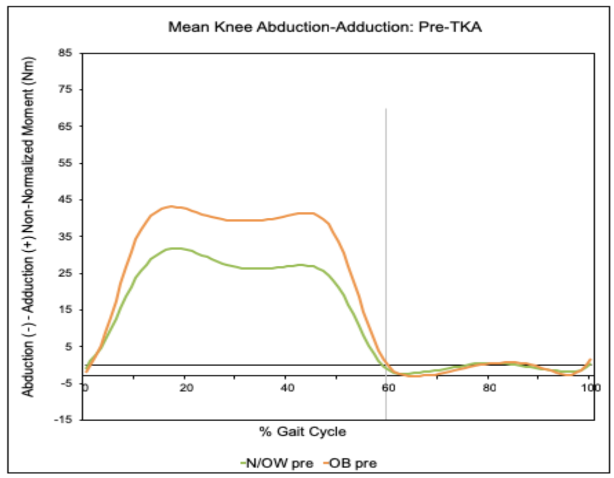


Figure 4-19. The time series of pre-TKA across-participant mean abduction-adduction moment of the knee for N/OW (green) and OB (orange) groups pre-TKA (non-normalized) over stance (0-60% gait cycle) and swing (61-100% gait cycle). Abduction and adduction is denoted by negative and positive values respectively.

4.3.2.2 Post-TKA Knee Non-Normalized Moment

Flexion-extension knee non-normalized moment

The time series data for the post-TKA individual trials and group means of flexion-extension non-normalized moments are depicted in Figure 4-20 and Figure 4-21. The ensembled trends are similar to those found in the pre-TKA time period with the OB group had greater flexion non-normalized moment in stance and greater extension non-normalized moment in terminal swing compared to the N/OW group.

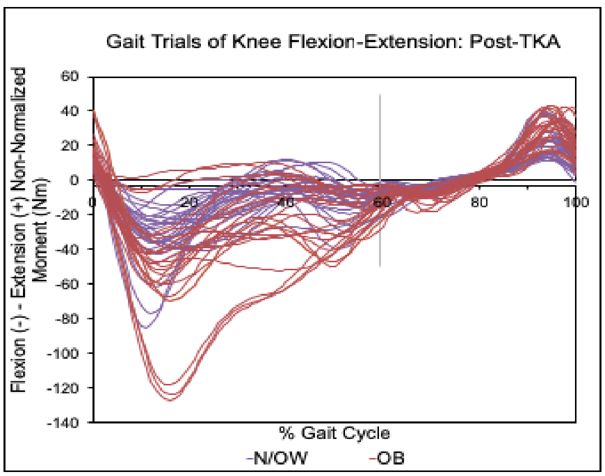


Figure 4-20. The time series of post-TKA flexion-extension moment of the knee gait trials for N/OW (purple) and OB (burgandy) groups (non-normalized) over stance (0-60% gait cycle) and swing (61-100% gait cycle). Flexion and extension is denoted by negative and positive values respectively.

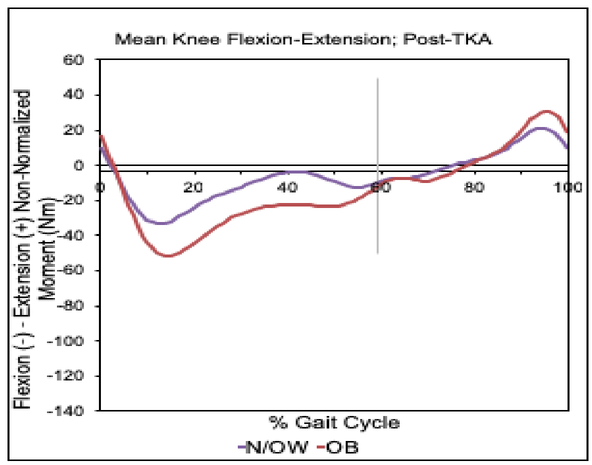


Figure 4-21. The time series of post-TKA across-participant mean flexion-extension knee moment for N/OW (purple) and OB (burgandy) groups (non-normalized) over stance (0-60% gait cycle) and swing (61-100% gait cycle). Flexion and extension is denoted by negative and positive values respectively.

Abduction-adduction knee non-normalized moment

The time series data for the post-TKA individual trials and group means of abduction-adduction non-normalized moments are depicted Figure 4-22 and Figure 4-23. The ensembled trends between groups are similar in loading response and swing phase. However, the OB group increased in adduction non-normalized moment during mid- to terminal stance whereas the N/OW group decreased.

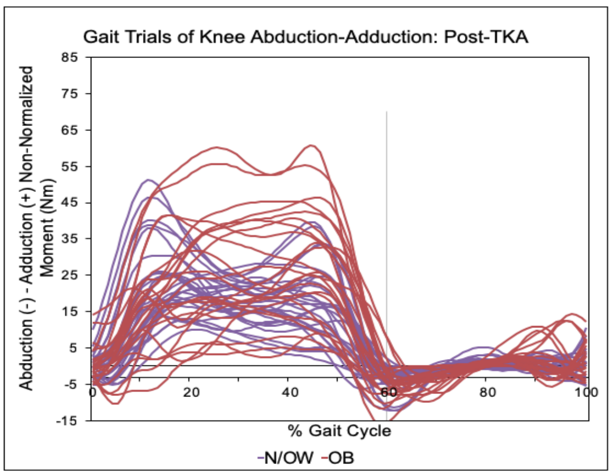


Figure 4-22. The time series of post-TKA abduction-adduction moment of the knee gait trials for N/OW (purple) and OB (burgandy) groups (non-normalized) over stance (0-60% gait cycle) and swing. Abduction and adduction is denoted by negative and positive values respectively.

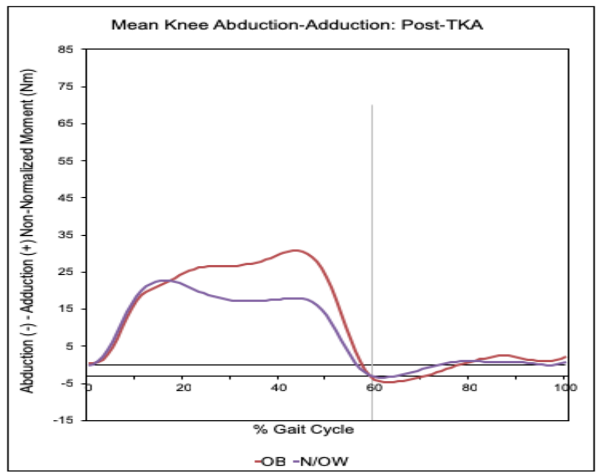


Figure 4-23. The time series of post-TKA across-participant mean abduction-adduction moment of the knee for N/OW (purple) and OB (burgandy) groups post-TKA (non-normalized) over stance (0-60% gait cycle) and swing (61-100% gait cycle). Abduction and adduction is denoted by negative and positive values respectively.

4.3.2.3 Change from Pre- to Post-TKA Knee Non-Normalized Moment

Flexion-extension knee non-normalized moment

The time series data for the change from pre- to post-TKA group means of flexion-extension non-normalized moment are depicted in Figure 4-24**.** The ensembled data showed that all four groups had a similar trend throughout the gait cycle but deviated from each other at loading response and terminal swing. Further, both groups increased in flexion non-normalized moment in loading response. To add, the N/OW group slightly increased in extension non-normalized moment in terminal swing from pre- to post-TKA.

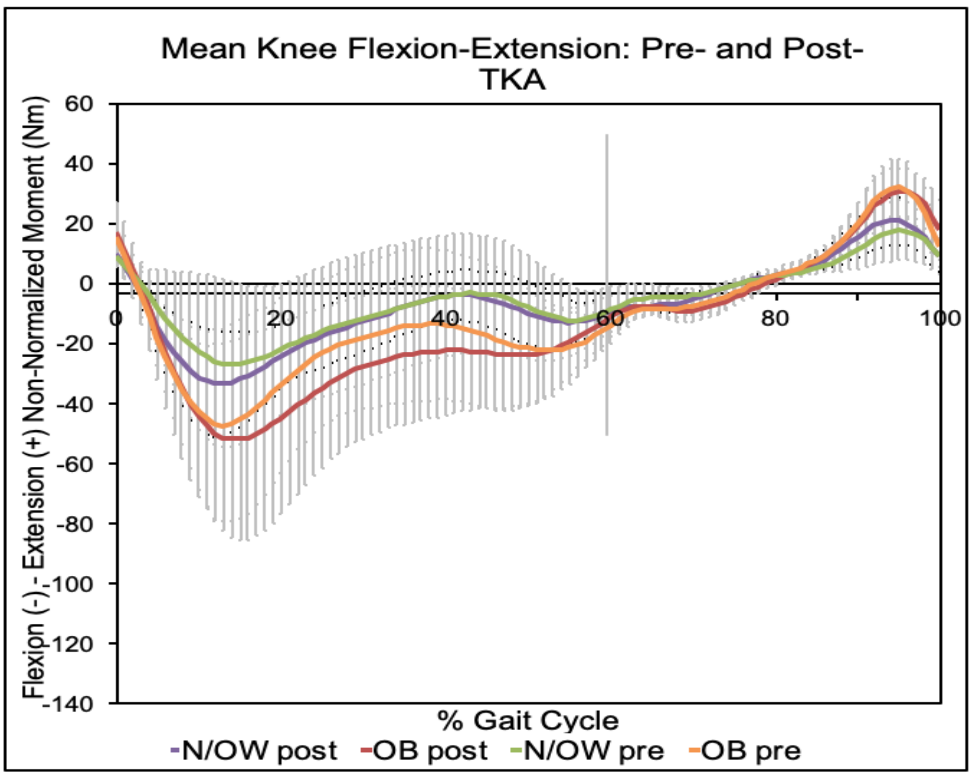


Figure 4-24. The time series of across-participant mean flexion-extension moment, not normalized to body weight, of the knee for N/OW pre- (green), OB pre- (orange), N/OW post- (purple) and OB post- (burgundy) TKA over stance (0-60% gait cycle) and swing (61-100% gait cycle). Flexion and extension is denoted by negative and positive values respectively. Error bars denote one standard deviation.

Abduction-adduction non-normalized moment of the knee

The time series data for the change from pre- to post-TKA group means of abduction-adduction non-normalized moment are depicted in Figure 4-25. All groups deviated from each other in stance phase and showed a similar trend from the ensembeld data except for the post-TKA OB group, which lacked a first peak. This suggested a lack of knee adduction non-normalized moment in the post-TKA OB group. To add, both groups decreased in magnitude from pre- to post-TKA suggests that the knee adduction non-normalized moment decreased after TKA in both groups.

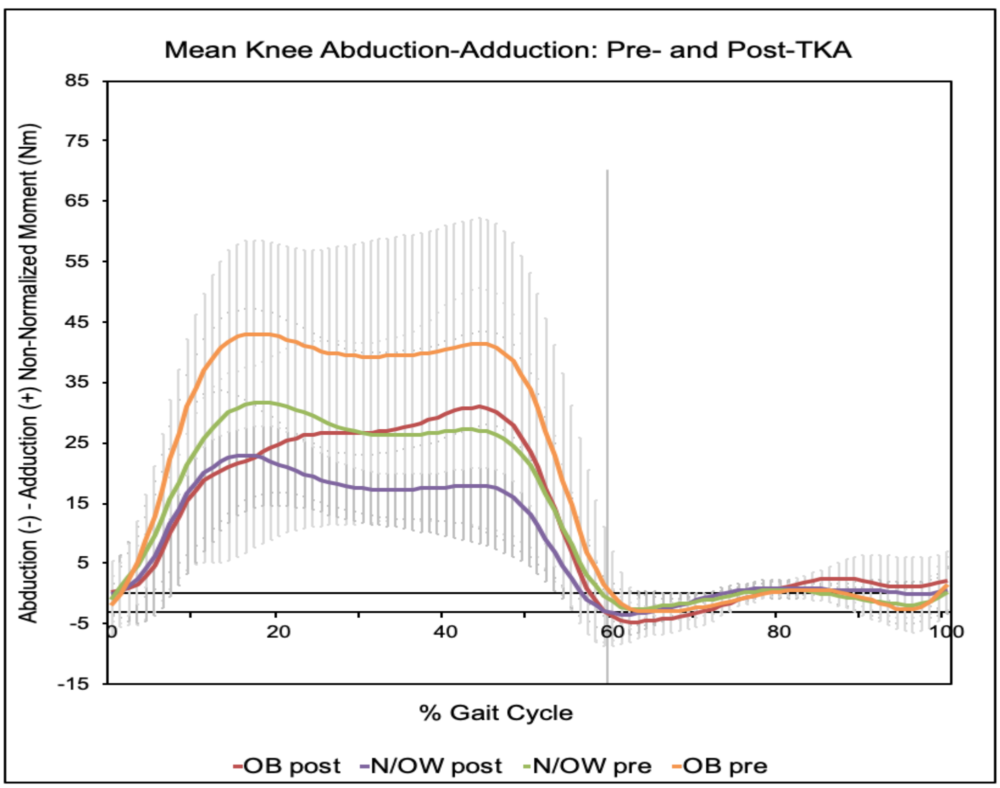


Figure 4-25. The time series of across-participant mean frontal knee moment, not normalized to body weight, for N/OW pre- (green), OB pre- (orange), N/OW post- (purple) and OB post- (burgundy) TKA over stance (0-60% gait cycle) and swing (61-100% gait cycle). Abduction and adduction is denoted by negative and positive values respectively. Error bars denote one standard deviation.

4.3.3.1 Pre-TKA Knee Normalized Moment

Flexion-extension normalized moment

The time series data for the pre-TKA individual trials and group means of flexion-extension normalized to body weight knee moments (hereafter flexion-extension normalized moments) are depicted in Figure 4-26 and Figure 4-27. Both groups showed similar trends to each other throughout the entire gait cycle. However, slight deviations occurred during mid- to terminal stance and terminal swing.

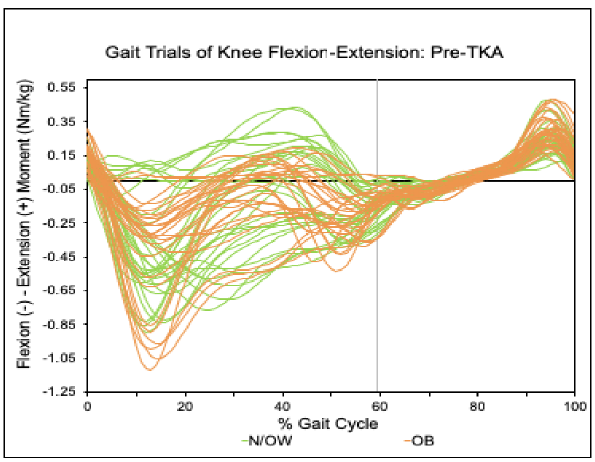


Figure 4-26. The time series of pre-TKA flexion-extension knee normalized moment of the knee gait trials for N/OW (green) and OB (orange) over stance (0-60% gait cycle) and swing (61-100% gait cycle). Flexion and extension is denoted by negative and positive values respectively.

Figure 4-27. The time series of pre-TKA across-participant mean flexion-extension knee normalized moment for N/OW (green) and OB (orange) over stance (0-60% gait cycle) and swing (61-100% gait cycle). Flexion and extension is denoted by negative and positive values respectively.

Abduction-adduction normalized moment

The time series data for the pre-TKA individual trials and group means of abduction-adduction knee moments normalized to body weight (hereafter abduction-adduction normalized moments) are depicted in Figure 4-28 and Figure 4-29. Both groups showed similar trends to each other, with exceptions in deviations in mid- to terminal stance, where the N/OW group had greater adduction normalized moment.

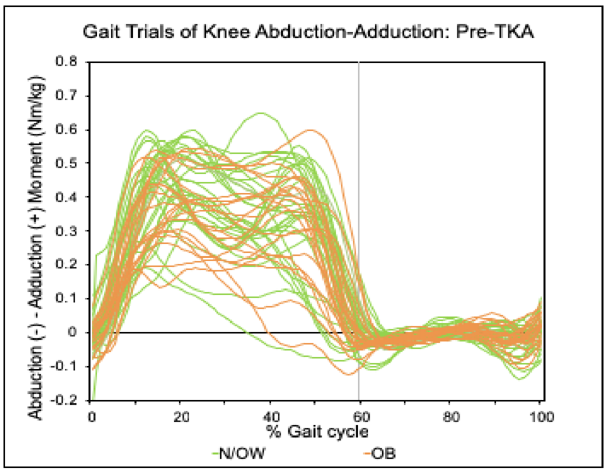


Figure 4-28. The time series of pre-TKA abduction-adduction normalized moment of the knee gait trials for N/OW (green) and OB (orange) over stance (0-60% gait cycle) and swing (61-100% gait cycle). Abduction and adduction is denoted by negative and positive values respectively.

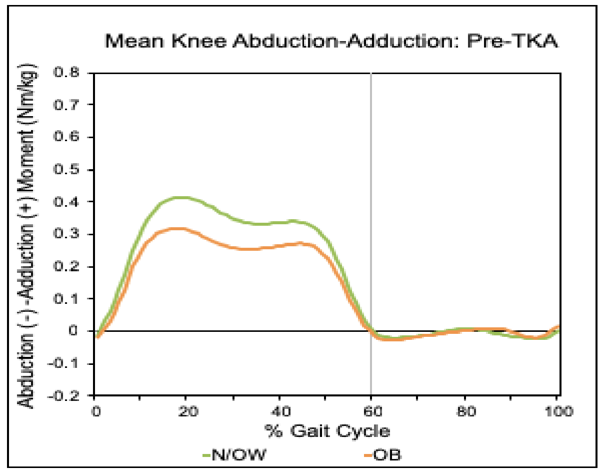


Figure 4-29. The time series of pre-TKA across-participant mean abduction-adduction normalized moment of the knee for N/OW (green) and OB (orange) pre-TKA over stance (0-60% gait cycle) and swing (61-100% gait cycle). Abduction and adduction is denoted by negative and positive values respectively.

4.3.3.2 Post-TKA Knee Normalized Moment

Flexion-extension normalized moment

The time series data for the post-TKA individual trials and group means of flexion-extension, normalized to body weight, knee moments (hereafter flexion-extension normalized moments) are depicted in Figure 4-30 and Figure 4-31. Both groups showed similar trend to each other with the exception in mid- to terminal stance, where the OB group had greater flexion moment compared to the N/OW group.

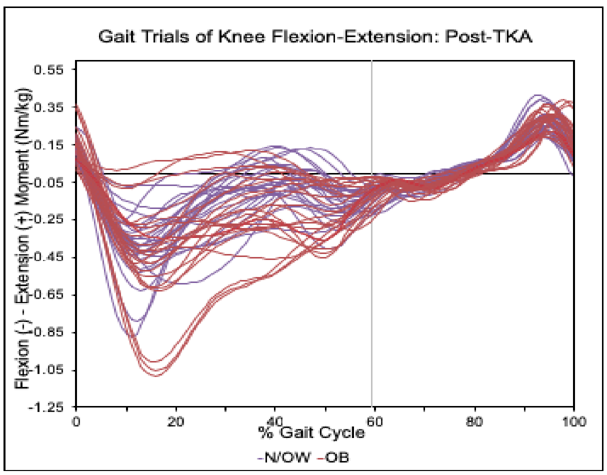


Figure 4-30. The time series of post-TKA flexion-extension normalized moment of the knee gait trials for N/OW (purple) and OB (burgundy) over stance (0-60% gait cycle) and swing (61-100% gait cycle). Flexion and extension is denoted by negative and positive values respectively.

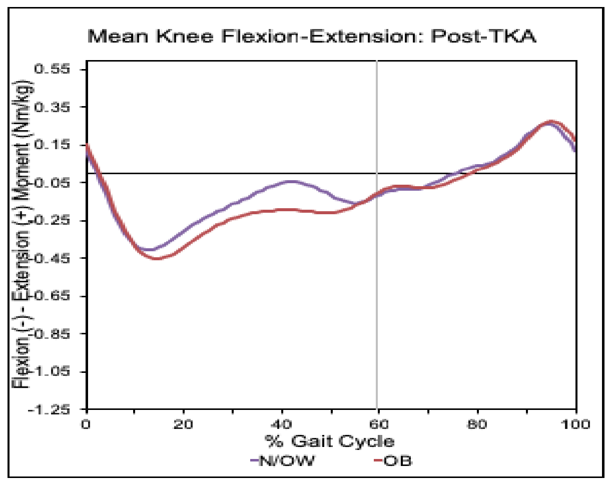


Figure 4-31. The time series of post-TKA across-participant mean flexion-extension normalized moment of the knee for N/OW (purple) and OB (burgundy) post-TKA over stance (0-60% gait cycle) and swing (61-100% gait cycle). Flexion and extension is denoted by negative and positive values respectively.

Abduction-adduction normalized moment

The time series data for the post-TKA individual trials and group means of abduction-adduction normalized moments are depicted in Figure 4-32 and Figure 4-33. Similar to pre-TKA individual trial findings, both groups showed similar trends in comparison to each other. However, there is deviations during loading response and terminal stance between groups, with the N/OW group had greater adduction normalized moment in loading response but less adduction normalized moment in terminal sstance.

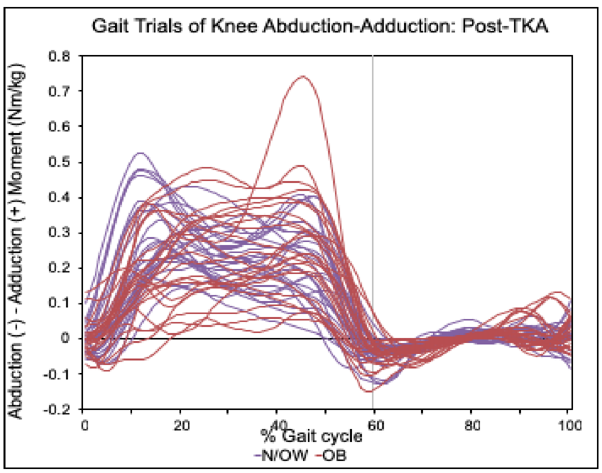


Figure 4-32. The time series of post-TKA abduction-adduction normalized moment of the knee gait trials for N/OW (purple) and OB (burgundy) over stance (0-60% gait cycle) and swing (61-100% gait cycle). Abduction and adduction is denoted by negative and positive values respectively.

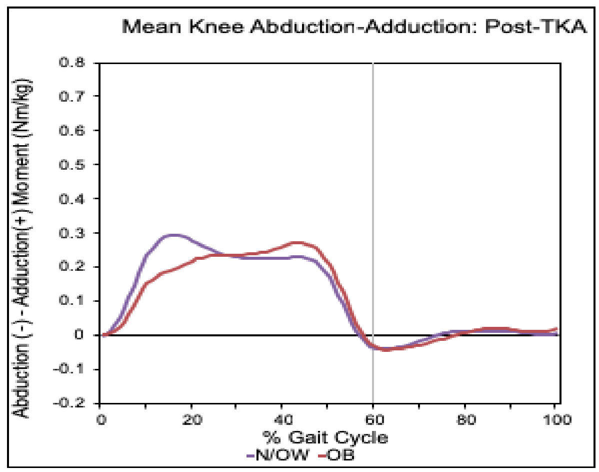


Figure 4-33. The time series of post-TKA across-participant mean abduction-adduction normalized moment of the knee for N/OW (purple) and OB (burgundy) post-TKA over stance (0-60% gait cycle) and swing (61-100% gait cycle). Abduction and adduction is denoted by negative and positive values respectively.

4.3.3.3 Change from Pre- to Post-TKA Knee Normalized Moment

Flexion-extension normalized moment

The time series data for the change from pre- to post-TKA group means of flexion-extension normalized moment are depicted in Figure 4-34. All groups showed a similar trend to each other throughout the gait cycle. However, during mid- to terminal stance, the OB pre- and post-TKA groups deviate from each other, with the post-TKA OB group having greater flexion normalized moment.

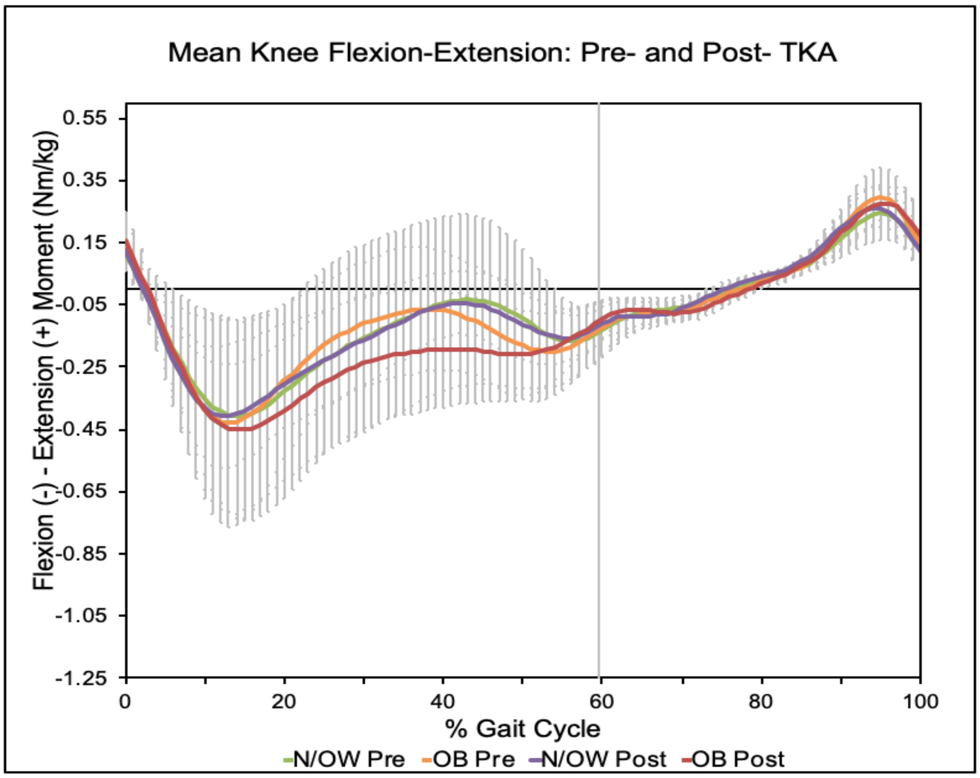


Figure 4-34. The time series of across-participant mean flexion-extension normalized moment of the knee for N/OW pre- (green), OB pre- (orange), N/OW post- (purple) and OB post- (burgundy) TKA over stance (0-60% gait cycle) and swing (61-100% gait cycle). Flexion and extension is denoted by negative and positive values respectively. Error bars denote one standard deviation.

Abduction-adduction normalized moment

The time series data for the change from pre- to post-TKA group means of abduction-adduction normalized moment are depicted in Figure 4-35. Similar to the abduction-adduction non-normalized moment findings of change pre- to post-TKA, all groups showed similar trends to each other, except for the post-TKA OB group lacking a first peak. Furthermore, both groups deviated from pre- to post-TKA, suggesting that in both groups, knee adduction normalized moment decreased after TKA.

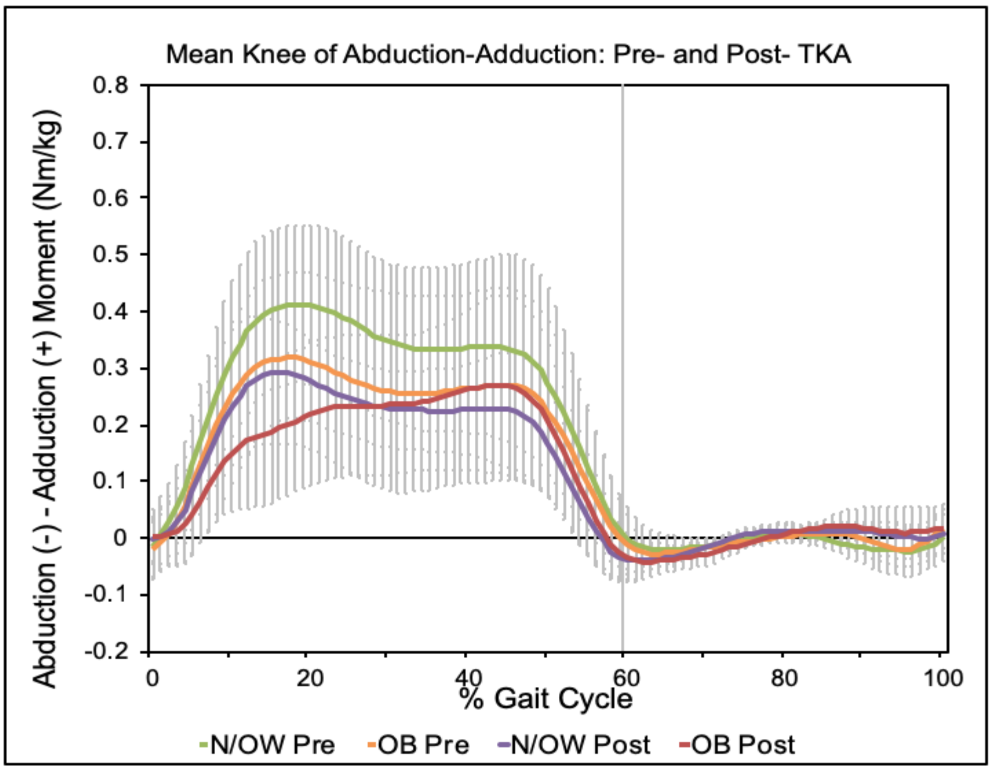


Figure 4-35. The time series of across-participant mean abduction-adduction normalized moment of the knee for N/OW pre- (green), OB pre- (orange), N/OW post- (purple) and OB post- (burgundy) TKA over stance (0-60% gait cycle) and swing (61-100% gait cycle). Abduction and adduction is denoted by negative and positive values respectively. Error bars denote one standard deviation.

4.4 Kinematic Analysis

4.4.1 Pre-TKA Knee Joint Angle

Flexion-extension angle of the knee

The descriptive kinematic parameters for the pre-TKA comparisons of flexion-extension angle are shown in Table 4-2. The OB group had larger median flexion angles during initial contact event (p=0.15) and loading response period (p=0.25) suggesting that the OB group has greater flexion of the knee than the N/OW group pre-TKA, but these were not statistically significant findings. Further, the OB group had larger median maximium (p=0.05) and minimum (p=0.18) values in stance phase and swing phase (maximium p=0.05; minimum 0.10), compared to the N/OW group, suggesting that the OB group remained in more knee flexion throughout the gait cycle.

Abduction-adduction angle of the knee

The descriptive kinematic parameters for the pre-TKA group comparisons of abduction-adduction angle are shown in Table 4-2. The OB group, in comparison to the N/OW group, had a lower median minimum (p=0.44) and maximum value (p=0.66), suggesting that the OB group had less knee adduction throughout the gait cycle, but these findings were not statistically significant.

Internal-external rotation angle of the knee

The descriptive kinematic parameters for the pre-TKA group comparisons of internal-external angle are shown in Table 4-2. Although the N/OW group had larger median minimum and maximum rotation values compared to the OB group (p=1.0), and a more externally rotated angle during mid-swing period (p=0.63), these findings were not statistically significant.

4.4.2 Post-TKA Knee Joint Angle

Flexion-extension angle of the knee

The descriptive kinematic parameters for the post-TKA group comparisons of flexion-extension angle are shown in Table 4-2. The OB group had larger median flexion angles compared to the N/OW group, suggesting that the OB group still had greater knee flexion throughout the gait cycle post-TKA, but none of these findings were statistically significant ($p>0.05$).

Abduction-adduction angle of the knee

The descriptive kinematic parameters for the post-TKA group comparisons of abduction-adduction angle are shown in Table 4-2. Unlike the pre-TKA findings, the post-TKA results show that the OB group, in comparison to the N/OW group, had a larger median maximum adduction angle ($p=0.0005$). This suggests that the OB group had greater knee adduction at the initial swing gait period as well as less knee adduction during mid swing gait period in comparison to the N/OW group.

Internal-external rotation angle of the knee

The descriptive kinematic parameters for the post-TKA group comparisons of internal-external angle are presented in Table 4-2. Unlike the pre-TKA findings, the post-TKA results show that the OB group had a larger median minimum value ($p=0.18$) and maximum median value ($p=0.21$) than the N/OW group, but these findings did not attain statistical significance.

4.4.3 Change from Pre- to Post-TKA

Changes in kinematic parameters in the flexion-extension, abduction-adduction, and internal-external rotation angles of the knee between pre- and post-TKA by group were summarized (Table 4-2). An interactional effect was found in the maximum knee abduction-adduction angle ($p=0.02$), suggesting that the groups followed different patterns over time; the OB group increased in knee adduction over time whereas the N/OW group decreased in knee adduction from pre- to post-TKA. A group effect was found in the maximum flexion-extension angle ($p=0.04$) indicating that the OB group had significantly higher maximum knee flexion than the N/OW group at both pre- and post-TKA. In all other kinematic parameters, there were no significant changes over time or between groups ($p>0.05$). The only clinically significant difference was the change in maximum adduction angle in the OB group from pre- to post-TKA.

Table 4-2. Changes from Pre- to Post- TKA Kinematics Across Participants by Groups.

Changes from Pre- to Post-TKA Kinematics		N/OW Mean (SD)			OB Mean (SD)					
		Pre	Post	95%CI	Pre	Post	95%CI	p-main	p-int	p-grp
Flx-Ext Stance °	Initial	10.1 (6.80)	13.2 (3.36)	8.99-14.4	16 (8.80)	15.4 (4.90)	12.0-19.4	0.54	0.36	0.10
	Loading	19.1 (10.5)	20.1 (4.05)	15.8-23.5	24.3 (8.88)	23.6 (6.16)	20.0-27.9	0.94	0.69	0.19
	Min	8.52 (7.99)	11.7 (4.30)	6.93-13.3	15.1 (9.67)	14.9 (5.25)	11.0-19.0	0.53	0.48	0.07
	Max	**59.7 (5.65)**	**59.7 (5.89)**	**56.9-62.5**	**65.4 (6.20)**	**65.4 (6.89)**	**62.1-68.8**	**0.99**	**0.98**	***0.04**
	Range	53.5 (6.21)	50 (5.4)	48.8-54.7	53.4 (8.58)	52.7 (4.29)	49.5-56.6	0.23	0.43	0.61
Flx-Ext Swing °	Min	6.19 (8.14)	9.77 (4.28)	4.71-11.2	12.0 (8.84)	12.7 (6.61)	8.34-16.4	0.35	0.53	0.12
	Max	**59.7 (5.65)**	**59.7 (5.89)**	**56.9-62.5**	**65.5 (6.20)**	**65.4 (6.89)**	**62.0-68.8**	**0.99**	**0.98**	***0.04**
Abd-Add °	Min	-0.18 (5.46)	-1.87 (4.02)	-3.38- 1.33	-2.42 (3.84)	-1.30 (2.06)	-3.50- -0.24	0.85	0.36	0.54
	Max	**7.61 (8.42)**	**5.72 (3.29)**	**3.55-9.79**	**6.11 (6.40)**	**13.9 (4.89)**	**6.40-13.7**	**0.11**	***0.02**	**0.17**
Int-Ext Rot °	Min	-13.8 (6.45)	-7.47 (4.34)	-13.7- -7.53	-12.5 (7.41)	-10.8 (5.82)	-15.0- -8.20	0.12	0.36	0.53
	Max	0.32 (5.07)	-1.36 (7.12)	-3.50-2.49	0.19 (8.11)	3.20 (6.26)	-2.10-5.51	0.79	0.34	0.33

Note: SD= Standard Deviation. Flx-Ext= flexion-extension. Abd-Add= abduction-adduction. Int-Ext rot= internal-external rotation. p-main = p-value of main effect (time). p-int = p-value of interaction (time*group). p-grp= p-value of group effect. p-values based on two-way repeated measures ANOVA. Significant differences in two-way repeated measures ANOVA ($p<0.05$) denoted with an asterisk (*) and bolded

4.5 Kinetic Analysis

4.5.1.1 Pre-TKA Knee Non-Normalized Moments

The descriptive kinetic parameters for the pre-TKA group comparisons of flexion-extension and abduction-adduction non-normalized moment are shown in Table 4-3. The median maximum flexion-extension non-normalized moment was significantly greater in the OB group (p=0.03) than the N/OW group. The OB group also had larger median minimum and maximum abduction-adduction non-normalized moments during loading response phase of gait (p=0.16), and greater median knee abduction non-normalized moment during initial swing phase (p=0.09) compared to the N/OW group, but these findings did not attain statistical significance.

4.5.1.2 Post-TKA Knee Non-Normalized Moments

The descriptive kinetic parameters for the post-TKA group comparisons of flexion-extension and abduction-adduction non-normalized moment are shown in Table 4-3. The median maximum flexion-extension non-normalized moment (p=0.01), median minimum flexion-extension non-normalized moment (p=0.003) and median maximum abduction-adduction non-normalized moment (p=0.006) were significantly different between groups with the OB group having higher non-normalized moments than the N/OW group. All non-normalized parameters of the knee were higher in the OB group relative to the N/OW group regardless of statistical significance.

4.5.1.3 Pre- to Post-TKA Knee Non-Normalized Moments

Changes in flexion-extension and abduction-adduction non-normalized moments between pre- and post-TKA by group were summarized (Table 4-3). There was a significant reduction in the mean maximum abduction-adduction non-normalized moment from pre- to post-TKA (p=0.003). Further, the OB group had a significantly higher mean maximum flexion-extension non-normalized moment than the N/OW group (p=0.02).

Table 4-3. Pre- to Post-TKA Changes in Kinetics, Not Normalized to Body Weight, Comparisons for Participants by Groups.

Pre- to Post-TKA Kinetics Non-normalized		N/OW Mean (SD)			OB Mean (SD)					
		Pre	Post	95%CI	Pre	Post	95%CI	p-main	p-int	p-grp
Flx-Ext	Min	-33.5 (24.6)	-35.1 (17.4)	-44.6- -23.9	-49.9 (32.0)	-54.7 (32.2)	-68.7– -35.6	0.39	0.68	0.17
Nm	Max	**21.3 (10.4)**	**21.7 (8.37)**	**17.0 - 26.1**	**33.6 (9.34)**	**33.0 (9.0)**	**28.6 - 38.1**	**0.93**	**0.69**	***0.02**
Abd-Add	Min	**35.8 (12.3)**	**22.7 (6.64)**	**23.4 - 35.1**	**48.6 (14.9)**	**31.1 (18.9)**	**29.8-49.8**	***0.003**	**0.62**	**0.05**
Nm	Max	-4.50 (2.9)	-3.78 (2.93)	-5.56- -2.72	-6.10 (2.7)	-5.79 (1.61)	-7.08 - -4.78	0.21	0.58	0.16

Note: SD= Standard Deviation. Flx-Ext= flexion-extension. Abd-Add= abduction-adduction. p-main = p-value of main effect (time). p-int = p-value of interaction (time*group). p-grp= p-value of group effect. p-values based on two-way repeated measures ANOVA. Significant differences in two-way repeated measures ANOVA (p<0.05) denoted with an asterisk (*) and bolded.

4.5.2.1 Pre-TKA Knee Normalized Moments

Knee moments, normalized to body weight, in flexion-extension and abduction-adduction were summarized for pre-TKA assessments for both groups (Table 4-4). Although the N/OW group had slightly greater median minimum flexion-extension moments, minimum and maximum abduction-adduction moments compared to the OB group, none of these normalized values were statistically significant.

4.5.2.2 Post-TKA Knee Normalized Moments

Knee moments, normalized to body weight, in flexion-extension and abduction-adduction were summarized for post-TKA assessments for both groups (Table 4-4). Although the OB group had slightly greater median minimum and maximum flexion-extension moment, and minimum abduction-adduction moment relative to the N/OW group post-TKA, none of these normalized findings attained statistical significance.

4.5.2.3 Pre- to Post-TKA Knee Normalized Moments

Changes in kinetic parameters in flexion-extension and abduction-adduction normalized moment between pre- and post-TKA by group were summarized (Table 4-4). The mean maximum flexion-extension normalized moment significantly changed from pre- to post-TKA ($p<0.001$) between groups ($p=0.017$) with the OB group demonstrating significantly higher mean maximum flexion normalized moment than the N/OW group. Further the pattern of change was different between groups with the N/OW group reducing their maximum flexion-extension normalized moments from pre- to post-TKA, while the OB group reported an increase in the normalized moment post-TKA. No clinically significant differences were found.

Table 4-4. Pre- to Post-TKA Changes in Knee Moments, Normalized to Body Weight, for Participants by Groups.

Pre- to Post- Kinetics Normalized		N/OW Mean (SD)			OB Mean (SD)					
		Pre	Post	95%CI	Pre	Post	95%CI	p-main	p-int	p-grp
Flx-Ext Nm/kg	Min	-0.48 (0.30)	-0.43 (0.16)	-0.58- -0.33	-0.46 (0.35)	-0.44 (0.31)	-0.62- -0.28	*0.000	0.14	0.14
	Max	**0.28 (0.08)**	**0.21 (0.06)**	**0.23 - 0.31**	**0.31 (0.08)**	**0.33 (0.06)**	**0.27 - 0.35**	***0.000**	***0.017**	***0.017**
Abd-Add Nm/kg	Min	-0.05 (0.03)	-0.06 (0.03)	-0.45- -0.18	-0.05 (0.03)	-0.06(0.02)	-0.07 - -0.04	0.137	0.34	0.97
	Max	0.37 (0.32)	0.32 (0.08)	0.04-0.07	0.32 (0.15)	0.30 (0.13)	0.24 - 0.38	0.171	0.40	0.87

Note: SD= Standard Deviation. p-main = p-value of main effect (time). p-int = p-value of interaction (time*group). p-grp= p-value of group effect. p-values based on two-way repeated measures ANOVA. Significant differences in two-way repeated measures ANOVA ($p<0.05$) denoted with an asterisk (*) and bolded.

4.6 Spatio-temporal Parameter Analysis

4.6.1 Pre-TKA, Post-TKA, and Change from Pre- to Post-TKA Spatio-Temporal parameters

Spatial-temporal parameters for both participant groups, including velocity, cadence and stride length for pre-TKA and post-TKA, and change from pre- to post-TKA (Table 4-5) were summarized. At pre-TKA and post-TKA, there were no significant difference in velocity, cadence, or stride length between group ($p>0.05$). In the changes from pre- to post-TKA, the N/OW group increased slightly in velocity, cadence and stride length over time whereas the OB group slightly decreased over time, but none of these group differences or changes over time attained statistical significance. No clinically significant differences were found.

Table 4-5. Pre- to Post-TKA Changes in Spatio-Temporal parameters for participants by groups.

Pre- to Post-TKA Spatio-temporal Parameters	N/OW Mean (SD)			OB Mean (SD)					
	Pre	Post	95%CI	Pre	Post	95%CI	p-main	p-int	p-grp
Velocity (m/s)	1.06 (0.27)	1.10 (0.24)	0.97-1.21	1.14 (0.26)	1.10 (0.14)	1.01-1.23	0.78	0.29	0.81
Cadence (step/min)	110.3 (12.1)	113.5 (9.3)	106.6-117.1	116.5 (8.29)	113.1 (7.25)	110.7-118.9	0.96	0.10	0.50
Stride Length (m)	1.14 (0.19)	1.19 (0.17)	1.08-1.25	1.17 (0.20)	1.16 (0.1)	1.08-1.25	0.60	0.46	0.99

Note: SD= Standard Deviation. p-main = p-value of main effect (time). p-int = p-value of interaction (time*group). p-grp= p-value of group effect. p-values based on two-way repeated measures ANOVA.

Chapter 5 Discussion

5.1 Overview

This chapter discusses the results of the study and their potential clinical significance. Firstly, main findings will be summarized. These important findings at pre-TKA, post-TKA and changes from pre- to post-TKA will then be discussed in the context of other published evidence. Finally, strengths and limitations of the study will be discussed along with future directions in light of these findings.

5.2 Main findings

The importance of gait biomechanics during pre- and post-surgical time periods is relevant to TKA outcomes of knee pain, patient satisfaction and implant longevity.[53] This study evaluated the effects of class II obesity in adults undergoing TKA (OB group) on changes in time-series and parameters of gait kinematics and kinetics, and spatio-temporal parameters relative to normal or overweight adults undergoing TKA (N/OW group). In addition, the kinematics of both of these groups were compared to that of a normative group (i.e., healthy adults of normal weight without knee OA.

At pre-TKA, the OB group had greater maximum flexion angle during swing phase and greater extension non-normalized moment during swing phase compared to the N/OW group. In time series analysis, both groups demonstrated similar patterns of gait, with changes noted from normative values in their initial loading response and terminal stance phase. Post-TKA, the OB group had greater maximum adduction angle during stance, greater maximum non-normalized moment in adduction, greater minimum flexion non-normalized moment during stance, and greater maximum extension non-normalized moment during swing, compared to the N/OW group. In changes from pre- to post-TKA, the OB group increased in adduction angle postoperatively, whereas the N/OW group decreased in adduction angle after surgery. Although both groups primarily remained within normative values for abduction-adduction angles at both pre- and post-TKA, the pattern of change demonstrated increased adduction angle at heel strike for the OB group post-TKA and for the N/OW group at pre-TKA. In addition, the OB group decreased in maximum extension normalized moment post-TKA, whereas the N/OW group increased in maximum extension normalized moment after surgery. The groups did not differ for pre-TKA, post-TKA or in changes from pre-TKA to post-TKA in any spatio-temporal parameters.

5.3 Pre-TKA

Pre-TKA, both groups demonstrated similar gait parameters, except that the OB group had significantly greater flexion angle and extension non-normalized moment during swing phase compared to the N/OW group. This could suggest that in the non-weight bearing swing phase, there is muscle weakness associated with the inactivity of the quadriceps and increased demands on the hamstrings.[245] These results also suggest that the OB group has greater magnitude of knee moment compared to the N/OW group. These findings agree with Bonnefoy-Mazure et al., 2017[107] who found significantly greater maximum flexion angle during the swing phase in 120 participants with severe OA one week before TKA. Browning and Kram 2007[141] also found greater flexion-extension knee moments in 20 participants with obesity (BMI 30 to 43 kg/m^2). However, neither of these studies are directly comparable to the current study as Bonnefoy-Mazure et al.[96] did not include adults with obesity and Browning and Kram 2007[141] did not include adults with knee OA. Thus, the current study results may suggest that both pathologies lead to changes in gait prior to TKA, with severe OA contributing to the changes in flexion-extension angle and obesity contributing to the changes in flexion-extension moments.

Our time series data would support this hypothesis: neither group followed normative values during the loading response and terminal stance gait periods in the flexion-extension angle. Thus, both groups appeared to walk with a straight-legged posture (less knee flexion during loading response and less knee extension during mid-stance to terminal stance gait periods), similar to what others have reported for those with end-stage OA.[246,247]

5.4 Post-TKA

5.4.1 Abduction-adduction

Post-TKA, both groups, again demonstrated similar gait parameters to each other with the following exceptions: the OB group had significantly greater knee adduction angle and maximum adduction non-normalized moment compared to the N/OW group after surgery. Thus, it appears that the OB group became more adducted after TKA in the abduction-adduction plane. In addition, although both groups had a reduction in adduction non-normalized moment during loading response, the OB group had a greater shift of the maximum non-normalized moment in abduction-adduction from loading response (first peak) to terminal stance (second peak) compared to the N/OW group. There is evidence to suggest that knee adduction moment is reduced post-TKA, but only if knee alignment is brought more to neutral and uncertainty remains as to whether knee adduction moment is restored to normative values.[53] Our time series results show that the first peak of knee adduction non-normalized moment does decrease in both groups, but that the OB group has a significantly higher second peak knee adduction non-normalized moment in comparison to the N/OW group. Thus, it is possible that the OB group may have ongoing knee symptoms following surgery that occur during terminal stance or that they may be unable to maintain neutral knee alignment post-TKA with the resulting change in the pattern of moment of the knee.[53,207,248,249] In contrast, an abstract proceeding by Outerleys et al., 2017,[250] showed that at one-year post-TKA, adults with class II obesity did not differ in knee abduction-adduction angles or moments compared to those of normal/overweight. These differential findings between groups may also be related to the timing of the post-TKA gait evaluation. Most gait studies to date have not evaluated post-TKA gait until six-months or later, so it is possible that some of the differences observed in the current study are related to the period of recovery after TKA. Perhaps the OB group would show similar postoperative gait to the N/OW group at six months or greater post-TKA if the current group differences in gait are due to a slower recovery trajectory post-TKA. Since there is currently no evidence to support this hypothesis, further work is required to determine if postoperative timing of gait evaluation influenced our results.

For the most part, these findings in abduction-adduction agree with current evidence. Bonnefoy-Mazure et al., 2017[107] found that, post-TKA, participants had significantly greater knee adduction angle in comparison to healthy adults, while several studies evaluating gait in participants with OA found that the participants with OA had greater knee adduction angle and higher knee adduction moments compared to healthy control participants.[251–253] Finally, Lai et al., 2008[105] also reported that the maximum knee adduction angle was significantly greater in participants with obesity (BMI 33 kg/m^2) compared to healthy weights while Sheehan et al., 2013[47] found that those who are overweight (BMI>25 kg/m^2) had an increase of knee adduction angle compared to those of healthy weight (BMI<25 kg/m^2). Again, current evidence is limited as none of these previous studies had participants with obesity who underwent TKA.[251–253] Our results are, however, contrary to those of Mills, et al., 2016[252] who found that the knee adduction moment did not differ between those with OA compared to those who are healthy. Since that study did not stratify for OA disease severity and did not include participants with obesity, it is difficult to generalize their findings to the study cohort.

Despite these findings in abduction-adduction, the maximum knee adduction moment was only significantly different between groups when it was non-normalized, suggesting that the differences may only be due to a higher weight in the OB group, and not due to an altered walking pattern. In contrast, Maclean et al., 2016[108] found no difference in knee adduction non-normalized moment in eight participants with obesity (BMI 35.5 SD 5.69) compared to those of healthy BMI. Since their study did not include either participants with knee OA or those who had TKA, the group differences found in our study may be related to knee OA that required TKA.

In summary, these findings in abduction-adduction suggest that the OB group had increase in their knee adduction angle typically found in those with TKA or obesity as well as increases in knee adduction moment that is typically seen in those with OA. This would result in the OB group having experienced a greater medial compartment load that can lead to degeneration of cartilage and bone area pre-TKA,[148] but may also be associated with increased prosthesis wear post-TKA. Again, these results may signify adjustment of gait to reduce moment about the knee[105] or a different pattern of recovery between groups. Our evaluation three months post-TKA may not capture the final post-TKA gait patterns between groups as Outerleys et al. 2017,[250] found no differences in gait at one-year post-TKA between those of normal/overweight or those with class II obesity. Future work should consider multiple postoperative assessments to determine how gait might change over time in patients with obesity.

5.4.2 Flexion-extension

Post-TKA, both groups had similar gait parameters in flexion-extension, except that the OB group had significantly greater minimum flexion non-normalized moment during stance, and maximum extension non-normalized moment during swing compared to the N/OW group. This would suggest that the OB group experienced greater absolute forces through the knee, resulting in greater loading of the quadriceps and hamstrings. The greater flexion-extension moments in the OB group are of some concern, as they have been known to be associated with higher risk of tibial component loosening and the presence of anterior knee pain.[63,208] Sosdian et al., 2014[53] also found that participants with a TKA had significantly greater flexion-extension moments compared to healthy individuals. However, Sosdian et al., 2014[53] did not investigate gait in participants with obesity and noted there are few longitudinal gait studies investigating how flexion-extension moments might change over time after a TKA. Our results are contrary to studies investigating gait and OA that found that no difference in knee flexion-extension moments in those with knee OA compared to those without knee OA,[252,253] even when considering participants with class I obesity.[230] Further, our results are also in contrast to an abstract proceeding by Outerleys et al., 2017,[250] which showed that at one-year post-TKA, adults with class II obesity do not differ in knee flexion-extension angles or moments compared to those of normal/overweight.

Since, after normalization to body weight, there were no substantial group differences noted in our study, it may be possible that the differences in the flexion-extension normalized moment may simply be due to BMI. Browning & Kram 2007[141] also found greater flexion-extension knee moments in participants with obesity. As participants with obesity have greater body mass, they require a greater maximum extension moment in the quadriceps to decelerate, absorb the forces and accept the body weight in order to stabilize and prevent the knee from collapsing, relative to the N/OW group.[148] Thus, there may be increased demands on the hamstrings due to quadriceps weakness resulting in pain avoidance gait (extension moment is present throughout stance) for subsequent initial contact and weight bearing.[52,246] Further, other evidence suggests that abnormal kinetic patterns in flexion-extension may be due to abnormal muscle function, biomechanical effects on other joints, prosthetic failure/difficulties, and functional abilities.[52] Limited evidence suggests that the abnormal moment pattern similar to that seen in our study may be due to a lack of ACL and reduction in knee proprioception.[52]

Clearly, more work is needed to determine the impact of gait after TKA as it is plausible that gait differences among people undergoing TKA are multi-factorial, with obesity being one factor of interest. As Sosdian et al., 2014[53] suggested, more longitudinal gait analyses after TKA, particularly when postoperative gait evaluations commence during the recovery period, are warranted. It is currently unclear as to whether gait continues to change over time postoperatively and the influence that obesity might play in post-TKA gait over the long-term.[216] Longitudinal analyses might also inform whether gait is modifiable post-TKA, if assessed early, and could lead towards people with TKA moving towards more normative gait values with appropriate gait training and/or rehabilitation.

5.5 Change from Pre- to Post- TKA

Both groups followed a similar pattern of change in gait from pre- to post-TKA in most parameters. The exceptions were maximum extension normalized and non-normalized moment during swing phase and maximum adduction non-normalized moment during stance phase. In the maximum extension normalized and non-normalized moments, the OB group decreased in magnitude from pre- to post-TKA whereas the N/OW group increased in magnitude over the two evaluations. Further, in the maximum adduction non-normalized moment during stance, both groups decreased the mean adduction moment from pre- to post-TKA. However, the OB group's adduction non-normalized moment remained significantly higher than the N/OW group adduction non-normalized moment at both gait evaluations. Similar to other limited studies evaluating gait after TKA, it appears that TKA contributes to the lowering of the maximum adduction normalized moment regardless of obesity,[254,255] but our findings suggest that those with obesity experience less absolute effect on knee joint kinetics than those of normal weight. The lack of comparative studies in changes from pre- to post-TKA kinetics also warrants further investigation as this would also inform if rehabilitation or surgical (e.g., prosthesis design) approaches could improve gait outcomes in those with obesity who undergo TKA.

5.6 Spatio-temporal parameters

Interestingly, we found no significant changes in velocity, cadence, or stride length compared to the N/OW group or over time, although the N/OW group slightly increased their velocity and cadence from pre- to post-TKA while the OB group slightly decreased these parameters over time. Neither the group differences nor the changes over time were statistically significant. The OB group did not appear to decrease the magnitude of knee joint moments during gait by walking with a slower velocity to increase their contact time with the floor, reduce their cadence to decrease the number of step impacts with the floor, or reduce their stride length to decrease the moment arm, which is common in those with obesity.[145,250]

Studies comparing obese and normal/overweight individuals reported that individuals living with obesity typically have a slower walking speed, reduced cadence, and shorter stride length.[49,48, 211, 256,136] Further, Bonnefoy-Mazure et al., 2017[107] also found significant differences in velocity, cadence, and stride length when comparing their knee OA group to a healthy control group at one week pre-TKA and three months post-TKA. Again, Bonnefoy-Mazure et al., 2017[107] did not include patients living with obesity, but did have a larger sample size of 120 participants relative to our small pilot study of 20 participants. In contrast, in an abstract proceeding by Outerleys et al., 2017,[250] found no difference in gait velocity in 71 participants grouped into class II obesity and healthy/overweight BMI who were not significantly different one week before and 12 months after TKA. Our study highlights the need for more research, including longitudinal gait analyses, investigating the effect of obesity on spatio-temporal parameters in those undergoing TKA.

5.7 Strengths

This study has several strengths. Our participants were age- and sex-matched to control for any sex or age differences between groups. We also successfully recruited appropriate candidates for the study within four months, with all participants completing their pre-TKA assessment. Further, most participants (85%) were able to complete gait assessments postoperatively, which was earlier post-TKA than has usually been performed in previous studies.[20,59,107,211,254,255] In addition, there were concerns that participants might not be able to walk without gait aids at the post-TKA assessment. This study demonstrated that a gait evaluation during the early post-TKA time point was feasible, as all of the participants who attended the post-TKA evaluation successfully completed their gait assessment without a gait aid. Perhaps, one of the more notable strengths was that the initial post-TKA gait evaluation occurred at three months post-TKA. This time period may be a clinically informative time period to investigate gait patterns, as they may be modifiable, and thus, amenable to rehabilitation interventions. Lastly, we were able to collect comprehensive gait data in participants living with class II obesity, which is not a frequently studied population.[53]

5.8 Limitations

Sex and age

Despite its strengths, this study does have some limitations. The small sample size may have resulted in some gait differences between groups being overlooked due to the substantial individual variation seen within both groups. Thus, the ability to detect significant group differences is difficult and may not have truly existed- differences possibly could have resulted from measurement error (estimations in anthropometrics or marker placement inaccuracies) or sampling error (convenience sample: mean BMI of N/OW group being classified as overweight as opposed to normal weight). However, from our results, the amount of variation and patterns of differences in both groups was similar, and likely a large sample size is needed to evaluate group differences in a meaningful way.

Although we matched for sex and age groups, we were unable to perform sub-analyses to see if further gait differences occur within sex and age sub-groups. These factors would be important to study as these factors might have influenced the findings of our study.

With regard to sex, we found that there is a difference in adipose tissue distribution in men living with class II obesity compared to women living with class II obesity; Men had greater adipose tissue in the pannus, whereas women had greater adipose tissue in the thighs. Subsequently, the accuracy of marker placement may have been different between sexes, with more potential for inaccuracy on the medial and lateral femoral epicondyles of women and on the ASIS of men; this, in turn could impact kinematics and kinetics. This finding highlights a limitation in using BMI as a measurement of obesity. The BMI classification does not account for age and sex, the location or amount of body fat, or an individual's health, which makes it a poor measurement of obesity. We matched participants on sex so that groups would remain comparable. In subsequent work, other ways to measure obesity such as waist circumference, waist-to-hip ratios, skin-folds, or DEXA scans, in addition to BMI, will likely improve accuracy in the evaluation of gait in people living with obesity.

In addition, a higher cadence and shorter stride length is found in women compared to men.[257,258] Similarities in spatio-temporal parameters between groups may have been due to the small sample size with both sexes represented, reducing the opportunity to find group differences. However, these findings might also be due to the early postoperative assessment where participants in both groups were still recovering from their surgery and not at their peak post-TKA functional status.

With regard to age, the study investigated adults between 51 and 70 years of age. Walking velocity and stride length is known to vary with age, and therefore the study participants could have varied in walking velocity due to age. As the N/OW group was on average, slightly older than the mean OB group, the similarities between groups in spatiotemporal parameters may have been due to the increased mean age in N/OW group. However, with only a mean group difference in age of 2.6 years, our group comparisons are likely generalizable to this age group. A large sample with more representation of various age groups could better inform the impact of age and obesity on gait after TKA.

Obesity and related factors

Soft tissues artifact in participants living with obesity made motion capture challenging. Accuracy in locating the bony landmarks on participants and placing the markers on the same locations at pre- and post-TKA assessments required extra time, and likely perfect accuracy of the marker placement was not achieved. Inaccuracies in finding anatomical landmarks were most likely to occur for the bilateral anterior superior iliac spine on the pelvis, as well as the medial and lateral femoral epicondyles as described above.[259] Firstly, if the markers were placed on the femoral condyle instead of the femoral epicondyle in the OB group, significant differences in maximum and minimum flexion-extension moments could occur merely due to marker placement and not related to group differences. For example, difficulty in palpating the medial and lateral femoral epicondyles in a swollen knee post-TKA was likely associated with one participant's data (3 trials of 111 trials, 2.7%) in the post-TKA evaluation being classified as an outlier (greater than 1.5 SD from the mean group value). This participant had approximately 15° of knee abduction angle in mid-swing and 10° internal rotation angle more than any other participant. Secondly, if the anatomical landmarks had inaccuracies, then subsequent knee axes would also be inaccurate, affecting the knee angles and moments. This could alter our results to show different pathologies such as crouch gait (only a positive sagittal moment in stance) or stiff-knee gait (greater external flexion moment and smaller external extension moment) that were related to differences in accuracy of marker placement between groups rather than group differences related to obesity.[260]

To reduce the likelihood in landmark inaccuracies, we took still images of the marker locations on the participants at the pre-TKA assessment to use as a reference for the post-TKA assessment to assist in palpating the landmarks at the second gait assessment. Our mixed model of marker placement was novel and developed in collaboration with Dalhousie University in Halifax, Nova Scotia. Furthermore, we performed a case study to test intra-rater and test-retest reliability with reliable and repeatable results.

Normative data are currently not available in healthy age-matched controls for the marker set used in this study. The normative comparative values used in the study were based on a Helen Hayes marker set in healthy weight adults. Our marker set contained elements of this marker set, in addition to marker plates similar to those used at Dalhousie University. This makes comparisons to other gait studies challenging. Although normative values are not directly comparable, comparisons were made with the findings from Dalhousie University, with agreement noted between sites. Obtaining normative values in adults with obesity using new marker approaches are needed in order to accurately evaluate gait in this patient population. In particular, normative values at the hip and knee are needed.

We focused solely on the knee as this was the joint of interest, but it would be interesting to evaluate lower body gait patterns as well as trunk stability. We made the choice to focus on the knee overtly. Adults living with obesity frequently have an abdominal pannus, which can affect both gait as well as the accurate measurement of hip movement. For example, the pannus can physically impede hip flexion; thus, the movement and moments seen at the knee may be influenced by the limited hip flexion particularly with swing. Further, the pannus makes it very difficult to place markers and assess movement of the hip itself. Because of the methodological challenges as well as the fact that we were primarily interested in the knee, all of our evaluation focused on the knee. Further work in gait evaluation is needed to determine how to accurately assess lower extremity gait in people living with the higher classes of obesity.

Moreover, in determining segment mass, normative anthropometric data were used; we used Dempster's regression equations and data, based on a segment being a percent of total body mass. Those who are overweight and living with obesity may not have a distribution of body mass that is proportionate to Dempster's data, and there may be individual or sex variations in mass distribution. Thus, there may subsequently be errors in estimation of segment mass and subsequent center of mass positions.[261] Despite this limitation, the Dempster's regression equations and data are widely used by biomechanists and provide a quick estimation of segment weights. Further work is required to determine how the use of normative anthropometric data affects the assessment of kinetics in those living with obesity.

Assessment

We also only performed one post-operative assessment; more postoperative assessments might allow us to determine the trajectory of gait changes over time. Most gait studies post-TKA have only assessed gait one time, but generally when all postoperative gait changes are expected to have stabilized. In our cohort, multiple postoperative assessments might have proven useful during the recovery phase to determine if the groups changed their gait parameters over time or at different rates postoperatively.

Lastly, as per usual motion capture analysis, ambulation was done in a lab environment, which may not be reflective of functional walking in a free-living environment. Participants were walking on a flat surface with no obstacles. Thus, we did not assess natural variations in walking velocity, which could affect the kinematics and kinetics of the knee. However, these natural variations would also make group comparisons difficult, and as data are currently lacking in this patient group, we wanted to undertake the evaluation in the controlled environment. We did instruct the participants to walk at a natural velocity and selected the gait trials that visually represented the participant's usual walking velocity and pattern in the laboratory setting.

Chapter 6 Conclusions

Improved walking ability is one of the primary goals of people undergoing TKA.[262,263] A recent systematic review found that patients benefitted from walking interventions post-TKA.[66] However, few studies to date have evaluated how obesity, and in particular class II obesity, affects walking after TKA. The current lack of evidence prevents the development or implementation of optimal interventions or therapy, as we lack information on what gait differences occur in this group of patients. Our work evaluated gait before and after TKA in adults with and without obesity to gain empiric knowledge about gait differences. We hope that this knowledge will inform future gait evaluations in this patient group, and ultimately lead to improved rehabilitation and, potentially, surgical approaches.

Gait was quantified using 3D motion capture technology for 20 individuals pre- and post-TKA. Time series data captured patterns of gait kinematics and kinetics across a gait cycle. Kinematic parameters showed that adults with obesity have altered flexion-extension angles at pre-TKA, and abduction-adduction angles at post-TKA compared to adults of normal/overweight. Our findings suggest that knee alignment may be affected by both TKA and obesity. Kinetic parameters indicated that adults with obesity have altered non-normalized moments in flexion-extension at pre- and post-TKA and abduction-adduction in post-TKA compared to adults of normal/overweight. The lack of findings in normalized moments suggest that these differences may primarily be due to BMI. Spatio-temporal parameters revealed that adults with obesity did not have significant differences in velocity, cadence, and stride length compared to adults of normal/overweight either pre- or three months post-TKA.

These results provide insights into what aspects of gait differ between groups and may suggest that rehabilitation can be specifically targeted in adults with obesity who have gait abnormalities. Primarily, this work has contributed to the mechanistic understanding of gait, which will inform future research.

6.1 Recommendations for Future work

Further development and investigation in study design, parameters investigated, and real-world applications is required to provide greater evidence of how gait affects outcomes after TKA in those living with obesity.

Firstly, further development of gait data collection, procedures, and analysis are needed to evaluate gait in people living with obesity. Specifically, anthropometrics determined from a sample of adults living with obesity would be helpful rather than using Dempster's equation.[264] This would require imaging individuals and adjusting anthropometric parameters before each gait session, direct measurements of each body segment with subsequent adjustment in the analysis, or the development of normative anthropometric data for people living with obesity. This would allow for more accurate comparisons amongst studies. Further, an obesity specific marker set needs to be further validated and tested for reliability to allow better evaluation of gait in people with obesity. Finally, a better measure of obesity than the most commonly used BMI needs to be determined. Waist circumference, waist-to-hip ratios, skin-folds, or DEXA scans in addition to BMI, would give a more accurate estimation of an individual's level of obesity. This would also allow consideration of the impact of distribution of adipose tissue and subsequent impact on gait.

Secondly, a larger sample size with multiple post-TKA assessments is needed to make conclusive statements about gait differences between these groups following TKA. Due to the substantial variation in kinematic and kinetic parameters in both groups, a larger sample size would provide more data to detect potential differences, increase confidence levels, more accurately identify potential outliers, and help to identify if statistical differences are clinically relevant. In particular, a larger sample size would allow for investigations and in-depth analysis of the association between age and sex on kinematic and kinetic parameters.

The impact of sex and age on gait parameters in people with obesity should be included, which requires a larger sample size of males and females within each five-year age grouping as post-TKA gait differences may vary by age and sex. In addition, participants with obesity who do not have knee OA could be used as a comparison group to compare gait parameters to differentiate between gait associated with knee OA/TKA and that related to knee OA/TKA in the presence of obesity.

Longitudinal gait assessment would also inform how gait changes over time between groups. We do not know if our reported group differences would be the same if they were measured at six months or one year post-TKA. Multiple time assessments would allow us to see the trajectory of change in gait after TKA to determine when gait retraining might be most effective. In addition, long-term follow-up might allow determination of how gait relates to reported pain and functional levels, which would assist in developing comprehensive rehabilitation interventions.

Thirdly, in real world applications, the measurements of repetitive loading of the knee in a real environment would be insightful to determine how gait impacts daily physical activity. This could include functional tasks and gait on uneven ground as this would be more representative of daily living. These additional evaluations would also inform rehabilitation practices as well as perhaps being useful to promote increased physical activity post-TKA in adults who would benefit from more movement.

Fourthly, knowledge of mechanical alignment of the knee obtained through x-rays and type of surgical fixation would be useful. The images can provide a better view of alignment issues as soft tissue can conceal the true orientation of the femur for knee abduction-adduction. This would help to identify implant position, to create a biomechanically friendly prosthetic knee, thereby preventing accelerated implant wear and loosening.[265]

Fifthly, measuring hip and trunk movement during gait and the relationship to knee biomechanics during gait would also be useful. This may require the development of novel methods of motion capture due to the challenge of accurately measuring trunk and hip movements when there is a large abdominal pannus.

The current findings have added empiric knowledge regarding motion capture analyses in adults with obesity who undergo TKA. These preliminary findings suggest that there are many unanswered questions that still require further exploration and evaluation. It is probable that there are clinical implications, which could lead to the development of rehabilitative and surgical approaches that may improve patient outcomes following TKA.

Further work is required to determine if adults should undergo gait retraining or other rehabilitative interventions post-TKA to improve their gait outcomes, and hopefully achieve their functional goals. However, this preliminary work was an important first step in identifying gait differences between adults of N/OW and those with class II obesity who undergo TKA.

References

1. Birtwhistle R, Morkem R, Peat G, et al. Prevalence and management of osteoarthritis in primary care: an epidemiologic cohort study from the Canadian Primary Care Sentinel Surveillance Network. *C Open*. 2015. doi:10.9778/cmajo.20150018.
2. MacDonald K V., Sanmartin C, Langlois K, Marshall DA. Symptom onset, diagnosis and management of osteoarthritis. *Heal Reports*. 2014.
3. O', Donnell S, Lagacé C, McRae L, Bancej C. Life with arthritis in canada: A personal and public health challenge. *Chronic Dis Can*. 2011;31(3):135-136.
4. O'Donnell S, Lagacé C, McRae L, Bancej C. Life with arthritis in canada: A personal and public health challenge. *Chronic Dis Can*. 2011;31(3):135-136.
5. Bombardier C, Hawker G, Mosher D. The Impact of Arthritis in Canada : Today and Over the Next 30 Years. *Arthritis Alliance of Canada*. 2011;Fall. http://www.arthritisalliance.ca.
6. Felson DT, Lawrence RC, Dieppe PA, et al. Osteoarthritis: New insights - Part 1: The disease and its risk factors. In: *Annals of Internal Medicine*. Vol 133. ; 2000:635-646. doi:10.7326/0003-4819-133-8-200010170-00016.
7. Macdonald K V, Sanmartin C, Langlois K, Marshall DA. Symptom onset, diagnosis and management of osteoarthritis. *Stat Canada*. 2014;25(9):10-17.
8. Henriksen M, Creaby MW, Lund H, Juhl C, Christensen R. Is there a causal link between knee loading and knee osteoarthritis progression? A systematic review and meta-analysis of cohort studies and randomised trials. *BMJ Open*. 2014;4(7):e005368. doi:10.1136/bmjopen-2014-005368.
9. Lee R, Kean WF. Obesity and knee osteoarthritis. *Inflammopharmacology*. 2012;20(2):53-58. doi:10.1007/s10787-011-0118-0.

10. Coggon D, Reading I, Croft P, McLaren M, Barrett D, Cooper C. Knee osteoarthritis and obesity. *Int J Obes*. 2001;25(5):622-627. doi:10.1038/sj.ijo.0801585.

11. Blagojevic M, Jinks C, Jeffery A, Jordan KP. Risk factors for onset of osteoarthritis of the knee in older adults: a systematic review and meta-analysis. *Osteoarthr Cartil*. 2010;18(1):24-33. doi:10.1016/j.joca.2009.08.010.

12. Felson DT, Anderson JJ, Naimark A, Walker AM, Meenan RF. Obesity and knee osteoarthritis. The Framingham Study. *Ann Intern Med*. 1988;109(1):18-24. doi:10.1059/0003-4819-109-1-18.

13. Kerkhoffs GMMJ, Servien E, Dunn W, Dahm D, Bramer J a M, Haverkamp D. The influence of obesity on the complication rate and outcome of total knee arthroplasty: a meta-analysis and systematic literature review. *J Bone Joint Surg Am*. 2012;94(20):1839-1844. doi:10.2106/JBJS.K.00820.

14. Zhang W, Moskowitz RW, Nuki G, et al. OARSI recommendations for the management of hip and knee osteoarthritis, Part II: OARSI evidence-based, expert consensus guidelines. *Osteoarthr Cartil*. 2008. doi:10.1016/j.joca.2007.12.013.

15. Pendleton A, Arden N, Dougados M, et al. EULAR recommendations for the management of knee osteoarthritis: report of a task force of the Standing Committee for International Clinical Studies Including Therapeutic Trials (ESCISIT). *AnnRheumDis*. 2000;59:936-944.

16. Jordan KM, Arden NK, Doherty M, et al. EULAR Recommendations 2003: An evidence based approach to the management of knee osteoarthritis: Report of a Task Force of the Standing Committee for International Clinical Studies Including Therapeutic Trials (ESCISIT). *Ann Rheum Dis*. 2003;62(12):1145-1155. doi:10.1136/ard.2003.011742.

17. Hochberg MC, Altman RD, April KT, et al. American College of Rheumatology 2012 recommendations for the use of nonpharmacologic and pharmacologic therapies in osteoarthritis of the hand, hip, and knee. *Arthritis Care Res*. 2012;64(4):465-474. doi:10.1002/acr.21596.

18. Badley EA immunity R and EMD. Arthritis in Canada . An ongoing Challenge. *Heal Canada*. 2003. doi:H39-4/14-2003E.

19. Şentürk İ, Kanatlı U, Ataoğlu B, Esen E. Gait analysis after total knee arthroplasty: comparison of pre- and postoperative characteristics. *Cukurova Med J (Çukurova Üniversitesi Tıp Fakültesi Dergisi)*. 2017;42(1):92-92. doi:10.17826/cutf.280133.

20. Arnold JB, Mackintosh S, Olds TS, Jones S, Thewlis D. Improvements in knee biomechanics during walking are associated with increased physical activity after total knee arthroplasty. *J Orthop Res*. 2015;33(12):1818-1825. doi:10.1002/jor.22969.

21. Hawker G, Wright J, Coyte P, et al. Health-related quality of life after knee replacement. *J Bone Joint Surg Am*. 1998;80(2):163-173. http://www.ncbi.nlm.nih.gov/pubmed/9486722.

22. Boyd AD, Ewald FC, Thomas WH, Poss R, Sledge CB. Long-term complications after total knee arthroplasty with or without resurfacing of the patella. *J Bone Joint Surg Am*. 1993;75(5):674-681. http://www.ncbi.nlm.nih.gov/pubmed/8501082.

23. Beal MD, Delagramaticas D, Fitz D. Improving outcomes in total knee arthroplasty — do navigation or customized implants have a role ? *J Orthop Surg Res*. 2016:1-7. doi:10.1186/s13018-016-0396-8.

24. Canadian Institute for Health Information. Hip and Knee Replacements in Canada, 2016-2017. *Can Jt Replace Regist*. 2018;(June):1-50.

25. Shadyab AH, Li W, Eaton CB, LaCroix AZ. General and Abdominal Obesity as Risk

Factors for Late-Life Mobility Limitation After Total Knee or Hip Replacement for Osteoarthritis Among Women. *Arthritis Care Res*. 2018. doi:10.1002/acr.23438.

26. Janssen I. The Public Health Burden of Obesity in Canada. *Can J Diabetes*. 2013. doi:10.1016/j.jcjd.2013.02.059.

27. Canadian Institute for Health. Hip and Knee Replacements in Canada: Canadian Joint Replacement Registry 2015 Annual Report. 2015;(September):1-63. https://secure.cihi.ca/free_products/CJRR_2015_Annual_Report_EN.pdf.

28. Bryan D, Parvizi J, Austin M, et al. Obesity and total joint arthroplasty. A literature based review. *J Arthroplasty*. 2013;28(5):714-721. doi:10.1016/j.arth.2013.02.011.

29. Beaupre LA. Personal Communication.

30. Torres-Claramunt R, Hinarejos P, Leal-Blanquet J, et al. Does Obesity Influence on the Functional Outcomes of a Total Knee Arthroplasty? *Obes Surg*. 2016. doi:10.1007/s11695-016-2233-x.

31. Ritter MA, Davis KE, Meding JB, et al. The effect of alignment and BMI on failure of total knee replacement. *J Bone Joint Surg Am*. 2011;93(17):1588-1596. doi:10.2106/JBJS.J.00772.

32. Lim CT, Goodman SB, Huddleston JI, et al. Weight Gain after Primary Total Knee Arthroplasty is Associated with Accelerated Time to Revision for Aseptic Loosening. *J Arthroplasty*. 2017. doi:10.1016/j.arth.2017.02.026.

33. Salih S, Sutton P. Obesity , knee osteoarthritis and knee arthroplasty : a review. *BMC Sport Sci Med Rehabil*. 2013;5(25):25-30.

34. Rajgopal V, Bourne RB, Chesworth BM, MacDonald SJ, McCalden RW, Rorabeck CH. The Impact of Morbid Obesity on Patient Outcomes After Total Knee Arthroplasty. *J*

Arthroplasty. 2008;23(6):795-800. doi:10.1016/j.arth.2007.08.005.

35. Si H, Zeng Y, Shen B, et al. The influence of body mass index on the outcomes of primary total knee arthroplasty. *Knee Surgery, Sport Traumatol Arthrosc*. 2015;23(6):1824-1832. doi:10.1007/s00167-014-3301-1.

36. Liao C-D, Huang Y-C, Lin L-F, Huang S-W, Liou T-H. Body Mass Index and Functional Mobility Outcome Following Early Rehabilitation After a Total Knee Replacement: A Retrospective Study in Taiwan. *Arthritis Care Res (Hoboken)*. 2015;67(6):799-808. doi:10.1002/acr.22474.

37. Jasper LL, Jones CA, Mollins J, Pohar SL, Beaupre LA. Risk factors for revision of total knee arthroplasty: a scoping review. *BMC Musculoskelet Disord*. 2016;17(1):182. doi:10.1186/s12891-016-1025-8.

38. Electricwala AJ, Jethanandani RG, Narkbunnam R, et al. Elevated Body Mass Index Is Associated With Early Total Knee Revision for Infection. *J Arthroplasty*. 2016;32(1):3-6. doi:10.1016/j.arth.2016.05.071.

39. Vincent HK, Vincent KR. Obesity and inpatient rehabilitation outcomes following knee arthroplasty: A multicenter study. *Obesity*. 2008. doi:10.1038/oby.2007.10.

40. Jones CA, Cox V, Jhangri GS, Suarez-Almazor ME. Delineating the impact of obesity and its relationship on recovery after total joint arthroplasties. *Osteoarthr Cartil*. 2012;20(6):511-518. doi:10.1016/j.joca.2012.02.637.

41. Collins JE, Donnell-Fink LA, Yang HY, et al. Effect of Obesity on Pain and Functional Recovery Following Total Knee Arthroplasty. *J Bone Joint Surg Am*. 2017. doi:10.2106/JBJS.17.00022.

42. Rodriguez-Merchan EC. Review article: Outcome of total knee arthroplasty in obese

patients. *J Orthop Surg*. 2015. doi:10.1177/230949901502300124.

43. Hawker G, Bohm ER, Conner-Spady B, et al. Perspectives of Canadian stakeholders on criteria for appropriateness for total joint arthroplasty in patients with hip and knee osteoarthritis. *Arthritis Rheumatol*. 2015;67(7):1806-1815. doi:10.1002/art.39124.

44. Cheatham W. Do Patient Factors and Prehabilitation Improve Outcomes After Total Knee Arthroplasty? *Top Geriatr Rehabil*. 2013;29(1):17-25.

45. DeVita P, Hortobágyi T. Obesity is not associated with increased knee joint torque and power during level walking. *J Biomech*. 2003;36(9):1355-1362. doi:10.1016/S0021-9290(03)00119-2.

46. Lai PPK, Leung AKL, Li ANM, Zhang M. Three-dimensional gait analysis of obese adults. *Clin Biomech*. 2008;23(SUPLL.1):2-6. doi:10.1016/j.clinbiomech.2008.02.004.

47. Sheehan KJ, Gormley J. The influence of excess body mass on adult gait. *Clin Biomech*. 2013;28(3):337-343. doi:10.1016/j.clinbiomech.2013.01.007.

48. Runhaar J, Koes BW, Clockaerts S, Bierma-Zeinstra SMA. A systematic review on changed biomechanics of lower extremities in obese individuals: A possible role in development of osteoarthritis. *Obes Rev*. 2011;12(12):1071-1082. doi:10.1111/j.1467-789X.2011.00916.x.

49. Page Glave A, Dibrezzo R, Applegate DK, Olson JM. The effects of weight and activity on select kinematic gait variables in adult females. *J Phys Educ Sport*. 2013;13(4):471-478. doi:10.7752/jpes.2013.04075.

50. Spyropoulos P, Pisciotta JC, Pavlou KN, Cairns MA, Simon SR. Biomechanical gait analysis in obese men. *Arch Phys Med Rehabil*. 1991;72(13):1065-1070. http://www.ncbi.nlm.nih.gov/pubmed/1741658.

51. Malatesta D, Vismara L, Menegoni F, Galli M, Romei M, Capodaglio P. Mechanical external work and recovery at preferred walking speed in obese subjects. *Med Sci Sports Exerc*. 2009;41(2):426-434. doi:10.1249/MSS.0b013e31818606e7.

52. McClelland JA, Webster KE, Feller JA. Gait analysis of patients following total knee replacement: A systematic review. *Knee*. 2007;14(4):253-263. doi:10.1016/j.knee.2007.04.003.

53. Sosdian L, Dobson F, Wrigley T V., et al. Longitudinal changes in knee kinematics and moments following knee arthroplasty: A systematic review. *Knee*. 2014;21(6):994-1008. doi:10.1016/j.knee.2014.09.009.

54. Komnik I, Weiss S, Fantini Pagani CH, Potthast W. Motion analysis of patients after knee arthroplasty during activities of daily living--a systematic review. *Gait Posture*. 2015;41(2):370-377. doi:10.1016/j.gaitpost.2015.01.019.

55. Akbari Shandiz M, Boulos P, Saevarsson SK, Yoo S, Miller S, Anglin C. Changes in knee kinematics following total knee arthroplasty. *Proc Inst Mech Eng Part H J Eng Med*. 2016;230(4):265-278. doi:10.1177/0954411916632491.

56. Navaneelan T, Janz T. Adjusting the scales: obesity in the Canadian population after correcting for respondent bias. *Heal a Glance*. 2014;53(17):1929-1930. doi:10.1787/health_glance-2009-en.

57. Fu XY, Zelik KE, Board WJ, Browning RC, Kuo AD. Soft tissue deformations contribute to the mechanics of walking in obese adults. *Med Sci Sports Exerc*. 2015;47(7):1435-1443. doi:10.1249/MSS.0000000000000554.

58. Lerner ZF, Board WJ, Browning RC. Effects of an obesity-specific marker set on estimated muscle and joint forces in walking. *Med Sci Sports Exerc*. 2014;46(6):1261-

1267. doi:10.1249/MSS.0000000000000218.

59. Paterson KL, Sosdian L, Hinman RS, et al. Effects of sex and obesity on gait biomechanics before and six months after total knee arthroplasty: A longitudinal cohort study. *Gait Posture*. 2018. doi:10.1016/j.gaitpost.2018.01.014.

60. Kennedy DM, Stratford PW, Riddle DL, Hanna SE, Gollish JD. Assessing Recovery and Establishing Prognosis Following Total Knee Arthroplasty. *Phys Ther*. 2008. doi:10.2522/ptj.20070051.

61. Mistry JB, Elmallah RDK, Bhave A, et al. Rehabilitative Guidelines after Total Knee Arthroplasty: A Review. *J Knee Surg*. 2016;29(3):201-217. doi:10.1055/s-0036-1579670.

62. Stevens-Lapsley JE, Schenkman ML, Dayton MR. Comparison of self-reported knee injury and osteoarthritis outcome score to performance measures in patients after total knee arthroplasty. *PM R*. 2011;3(6):541-549. doi:10.1016/j.pmrj.2011.03.002; 10.1016/j.pmrj.2011.03.002.

63. Smith AJ, Lloyd DG, Wood DJ. Pre-surgery knee joint loading patterns during walking predict the presence and severity of anterior knee pain after total knee arthroplasty. *J Orthop Res*. 2004;22(2):260-266. doi:10.1016/S0736-0266(03)00184-0.

64. Bade MJ, Stevens-Lapsley JE. Early high-intensity rehabilitation following total knee arthroplasty improves outcomes. *J Orthop Sports Phys Ther*. 2011;41(12):932-941. doi:10.2519/jospt.2011.3734.

65. Ahmed AR, Abd-Elkader SM, Al-Obathani KS. Effect of a 6-week rehabilitation program on gait parameters after total knee arthroplasty. *Saudi Med J*. 2010;31(9):1032-1035. doi:0' [pii].

66. Artz N, Elvers KT, Lowe CM, Sackley C, Jepson P, Beswick AD. Effectiveness of

physiotherapy exercise following total knee replacement: systematic review and meta-analysis. *BMC Musculoskelet Disord*. 2015;16(1):15. doi:10.1186/s12891-015-0469-6.

67. Milner CE. Is gait normal after total knee arthroplasty? Systematic review of the literature. *J Orthop Sci*. 2009;14(1):114-120. doi:10.1007/s00776-008-1285-8.

68. Lyytinen, T., Bragge, T., Liikavainio, T., Vartiainen, P., Karjalainen, P., Arokoski J. The Impact of Obesity and Weight Loss on Gait in Adults. *Stud Mecahnobiology, Tissue Eng Biomater*. 2015;(16):125-147.

69. Vaughan CL. Theories of bipedal walking: An odyssey. In: *Journal of Biomechanics*. Vol 36. ; 2003:513-523. doi:10.1016/S0021-9290(02)00419-0.

70. Rajasekaran V, López-Larraz E, Trincado-Alonso F, et al. Volition-adaptive control for gait training using wearable exoskeleton: Preliminary tests with incomplete spinal cord injury individuals. *J Neuroeng Rehabil*. 2018. doi:10.1186/s12984-017-0345-8.

71. Astephen JL, Deluzio KJ, Caldwell GE, Dunbar MJ, Hubley-Kozey CL. Gait and neuromuscular pattern changes are associated with differences in knee osteoarthritis severity levels. *J Biomech*. 2008;41(4):868-876. doi:10.1016/j.jbiomech.2007.10.016.

72. Lu TW, Chang CF. Biomechanics of human movement and its clinical applications. *Kaohsiung J Med Sci*. 2012;28(2 SUPPL.):S13-S25. doi:10.1016/j.kjms.2011.08.004.

73. Vaughan, Christopher; Davis, Brian; O'Connor J. *Dynamics of Human Gait*. Champaign, Illinois: Human Kinetics Publishers; 1992.

74. Schurr SA, Marshall AN, Resch JE, Saliba SA. Two-Dimensional Video Analysis is Comparable to 3D Motion Capture in Lower Extremity Movement Assessment. *Int J Sports Phys Ther*. 2017.

75. Cappello A, Cappozzo A, La Palombara PF, Lucchetti L, Leardini A. Multiple anatomical

landmark calibration for optimal bone pose estimation. *Hum Mov Sci*. 1997. doi:10.1016/S0167-9457(96)00055-3.

76. Reinschmidt C, Van Den Bogert AJ, Nigg BM, Lundberg A, Murphy N. Effect of skin movement on the analysis of skeletal knee joint motion during running. *J Biomech*. 1997. doi:10.1016/S0021-9290(97)00001-8.

77. Rodgers MM, Cavanagh PR. Glossary of biomechanical terms, concepts, and units. *Phys Ther*. 1984;64:1886-1902.

78. Cappozzo A. Gait analysis methodology. *Hum Mov Sci*. 1984;3(1-2):27-50. doi:10.1016/0167-9457(84)90004-6.

79. Chockalingam N, Giakas G, Iossifidou A. Do strain gauge force platforms need in situ correction? *Gait Posture*. 2002. doi:10.1016/S0966-6362(02)00017-6.

80. Change ND. Introduction to piezoelectric force sensors. *DYTRAN Gen Cat Instrum Handb*. 1995.

81. Dobrzynska JA, Gijs MAM. Polymer-based flexible capacitive sensor for three-axial force measurements. *J Micromechanics Microengineering*. 2013. doi:10.1088/0960-1317/23/1/015009.

82. Lugade V, Kaufman K. Center of pressure trajectory during gait: A comparison of four foot positions. *Gait Posture*. 2014. doi:10.1016/j.gaitpost.2014.07.001.

83. Borstad JD. Measurement of Pectoralis Minor Muscle Length: Validation and Clinical Application. *J Orthop Sport Phys Ther*. 2008. doi:10.2519/jospt.2008.2723.

84. Stevens SS. Measurement and man. *Science (80-)*. 1958. doi:10.1126/science.127.3295.383.

85. Unnikrishnan R, Hebert M. Fast Extrinsic Calibration of a Laser Rangefinder to a Camera.

Robotics. 2005.

86. Gps A, Gps L, Gps A. 1 . GLOBAL POSITIONING SYSTEM (GPS) OVERVIEW. *Glob Position Syst Overv*. 1994. doi:10.1016/j.lpm.2011.07.018.

87. Peters DM, Fritz SL, Krotish DE. Assessing the reliability and validity of a shorter walk test compared with the 10-Meter Walk Test for measurements of gait speed in healthy, older adults. *J Geriatr Phys Ther*. 2013. doi:10.1519/JPT.0b013e318248e20d.

88. van Loo MA, Moseley AM, Bosman JM, de bie RA, Hassett L. Inter-rater reliability and concurrent validity of walking speed measurement after traumatic brain injury. *Clin Rehabil*. 2003. doi:10.1191/0269215503cr677oa.

89. Dudzik M, Tomczyk K, Jagiello AS. Analysis of the error generated by the voltage output accelerometer using the optimal structure of an artificial neural network. In: *Proceedings of the 2018 19th International Conference on Research and Education in Mechatronics, REM 2018*. ; 2018. doi:10.1109/REM.2018.8421789.

90. Loudon JK (Janice K, Swift M, Bell S. The clinical orthopedic assessment guide. *SciTech B News*. 2008. doi:10.1111/j.1440-6055.2007.00596.x.

91. Kharb A, Saini V, Jain Y, Dhiman S. A review of gait cycle and its parameters. *IJCEM Int J Comput Eng Manag*. 2011;13(July):78-83.

92. Lerner ZF, Haight DJ, DeMers MS, Board WJ, Browning RC. The effects of walking speed on tibiofemoral loading estimated via musculoskeletal modeling. *J Appl Biomech*. 2014;30(2):197-205. doi:10.1123/jab.2012-0206.

93. DeMers MS, Pal S, Delp SL. Changes in tibiofemoral forces due to variations in muscle activity during walking. *J Orthop Res*. 2014;32(6):769-776. doi:10.1002/jor.22601.

94. Porto HC Del, Pechak CM, Smith DR, Reed-Jones RJ. Biomechanical Effects of Obesity

on Balance. *Int J Exerc Sci*. 2012;5(4):301-320. http://digitalcommons.wku.edu/cgi/viewcontent.cgi?article=1465&context=ijes.

95. Boone DC, Azen SP. Normal range of motion of joints in male subjects. *J Bone Jt Surg - Ser A*. 1979. doi:10.2106/00004623-197961050-00017.

96. Roach KE, Miles TP. Normal hip and knee active range of motion: The relationship to age. *Phys Ther*. 1991. doi:10.1093/ptj/71.9.656.

97. Bade MJ, Kohrt WM, Stevens-Lapsley JE. Outcomes Before and After Total Knee Arthroplasty Compared to Healthy Adults. *J Orthop Sport Phys Ther*. 2010. doi:10.2519/jospt.2010.3317.

98. Steultjens MPM, Dekker J, Van Baar ME, Oostendorp RAB, Bijlsma JWJ. Range of joint motion and disability in patients with osteoarthritis of the knee or hip. *Rheumatology*. 2000. doi:10.1093/rheumatology/39.9.955.

99. Myles CM, Rowe PJ, Walker CRC, Nutton RW. Knee joint functional range of movement prior to and following total knee arthroplasty measured using flexible electrogoniometry. *Gait Posture*. 2002. doi:10.1016/S0966-6362(01)00198-9.

100. Bytyqi D, Shabani B, Lustig S, Cheze L, Karahoda Gjurgjeala N, Neyret P. Gait knee kinematic alterations in medial osteoarthritis: Three dimensional assessment. *Int Orthop*. 2014. doi:10.1007/s00264-014-2312-3.

101. Creaby MW. It's not all about the knee adduction moment: The role of the knee flexion moment in medial knee joint loading. *Osteoarthr Cartil*. 2015;23(7):1038-1040. doi:10.1016/j.joca.2015.03.032.

102. Gök H, Ergin S, Yavuzer G. Kinetic and kinematic characteristics of gait in patients with medial knee arthrosis. *Acta Orthop Scand*. 2002. doi:10.1080/000164702321039606.

103. Kaufman KR, Hughes C, Morrey BF, Morrey M, An KN. Gait characteristics of patients with knee osteoarthritis. In: *Journal of Biomechanics*. ; 2001. doi:10.1016/S0021-9290(01)00036-7.

104. Andriacchi TP, Mündermann A. The role of ambulatory mechanics in the initiation and progression of knee osteoarthritis. *Curr Opin Rheumatol*. 2006. doi:10.1097/01.bor.0000240365.16842.4e.

105. Lai PPK, Leung AKL, Li ANM, Zhang M. Three-dimensional gait analysis of obese adults. *Clin Biomech*. 2008. doi:10.1016/j.clinbiomech.2008.02.004.

106. Malanga G, DeLisa JA. Clinical observation. In: *Gait Analysis in the Science of Rehabilitation*. ; 1998. doi:10.1227/01.NEU.0000342798.92438.48.

107. Bonnefoy-Mazure A, Armand S, Sagawa Y, Suvà D, Miozzari H, Turcot K. Knee Kinematic and Clinical Outcomes Evolution before, Three Months and One Year Following Total knee Arthroplasty. *J Arthroplasty*. 2016;32:793-800. doi:10.1016/j.arth.2016.03.050.

108. Maclean KFE, Callaghan JP, Maly MR. Effect of obesity on knee joint biomechanics during gait in young adults. *Cognet Med*. 2016:1-16. doi:10.1080/2331205X.2016.1173778.

109. O. L, A. M, G. S, R. D, Y. B, A. E. The effect of a biomechanical gait-training device on gait patterns of obese knee osteoarthritis patients. *Rheumatol (United Kingdom)*. 2014;53:i132. doi:http://dx.doi.org/10.1093/rheumatology/keu112.004.

110. Al-Zahrani KS, Bakheit AMO. A study of the gait characteristics of patients with chronic osteoarthritis of the knee. *Disabil Rehabil*. 2002. doi:10.1080/09638280110087098.

111. Messier SP, Pater M, Beavers DP, et al. Influences of alignment and obesity on knee joint

loading in osteoarthritic gait. *Osteoarthr Cartil*. 2014;22(7):912-917. doi:10.1016/j.joca.2014.05.013.

112. Lee YS. Consequences of childhood obesity. *Ann Acad Med Singapore*. 2009. doi:10.1007/s12098-011-0489-7.

113. Janssen I, Katzmarzyk PT, Ross R. Waist circumference and not body mass index explains obesity-related health risk. *Am J Clin Nutr*. 2004. doi:10.1093/ajcn/79.3.379.

114. Centers for Disease Control and Prevention. Defining adult overweight and obesity. Centers for Disease Control and Prevention. doi:https://www.cdc.gov/obesity/adult/defining.html.

115. Centers of disease control. Body mass index: Considerations for practitioners. *Cdc*. 2011.

116. Xu X, Mirka GA, Hsiang SM. The effects of obesity on lifting performance. *Appl Ergon*. 2008. doi:10.1016/j.apergo.2007.02.001.

117. World Health Organization. Obesity and overweight. Fact sheet N°311. *Fact sheet N*. 2013. doi:http://www.who.int/mediacentre/factsheets/fs311/en/.

118. Queensland Goverment. Using Body Mass Index. *Nutr Educ Mater Online*. 2013:1-3. http://www.health.qld.gov.au/masters/copyright.asp%5Cnhttp://www.health.qld.gov.au/nutrition/resources/hphe_usingbmi.pdf.

119. Prentice AM, Jebb SA. Beyond body mass index. *Obes Rev*. 2001. doi:10.1046/j.1467-789x.2001.00031.x.

120. WHO. Obesity and overweight: Fact sheet No 311. 311. http://www.who.int/mediacentre/factsheets/fs311. Published 2016.

121. Ogilvie KK, Eggleton A. Obesity in Canada: A whole-of-society approach for a healthier Canada. 2016;(March):56.

http://www.parl.gc.ca/content/sen/committee/421/SOCI/Reports/2016-02-25_Revised_report_Obesity_in_Canada_e.pdf.

122. Twells LK, Gregory DM, Reddigan J, Midodzi WK. Current and predicted prevalence of obesity in Canada: a trend analysis. *C open*. 2014;2(1):E18-26. doi:10.9778/cmajo.20130016.

123. Monteiro R, Azevedo I. Chronic inflammation in obesity and the metabolic syndrome. *Mediators Inflamm*. 2010. doi:10.1155/2010/289645.

124. Public Health Agency of Canada. Obesity in Canada: A joint report from the public health agency of Canada and the Canadian Institute for health information. *Public Heal Agency Canada*. 2011:62. doi:HP5-107/2011E-PDF.

125. Dixon JB. The effect of obesity on health outcomes. *Mol Cell Endocrinol*. 2010;316(2):104-108. doi:10.1016/j.mce.2009.07.008.

126. Whitaker R, Wright J, Pepe M, Seidel K, Dietz W. Predicting Obesity in Young Adulthood From Childhood. *Massachusetts Med Soc*. 1997. doi:10.1056/NEJM199709253371301.

127. Beck AR. Psychosocial Aspects of Obesity. *NASN Sch Nurse*. 2016. doi:10.1177/1942602X15619756.

128. Carrière G. Parent and child factors associated with youth obesity. *Heal reports / Stat Canada, Can Cent Heal Inf = Rapp sur la sant?? / Stat Canada, Cent Can d'information sur la sant??* 2003.

129. Nantel J, Mathieu ME, Prince F. Physical activity and obesity: Biomechanical and physiological key concepts. *J Obes*. 2011;2011. doi:10.1155/2011/650230.

130. Wearing SC, Hennig EM, Byrne NM, Steele JR, Hills AP. The biomechanics of restricted

movement in adult obesity. *Obes Rev*. 2006;7(1):13-24. doi:10.1111/j.1467-789X.2006.00215.x.

131. Vartiainen P, Bragge T, Lyytinen T, Hakkarainen M, Karjalainen PA, Arokoski JP. Kinematic and kinetic changes in obese gait in bariatric surgery-induced weight loss. *J Biomech*. 2012;45(10):1769-1774. doi:10.1016/j.jbiomech.2012.05.002.

132. Pamukoff DN, Lewek MD, Blackburn JT. Greater vertical loading rate in obese compared to normal weight young adults. *Clin Biomech*. 2016;33:61-65. doi:10.1016/j.clinbiomech.2016.02.007.

133. Cimolin V, Vismara L, Galli M, Grugni G, Cau N, Capodaglio P. Gait strategy in genetically obese patients: A 7-year follow up. *Res Dev Disabil*. 2014;35(7):1501-1506. doi:10.1016/j.ridd.2014.04.005.

134. Freedman Silvernail J, Milner CE, Thompson D, Zhang S, Zhao X. The influence of body mass index and velocity on knee biomechanics during walking. *Gait Posture*. 2013;37(4):575-579. doi:10.1016/j.gaitpost.2012.09.016.

135. Lerner ZF, Board WJ, Browning RC. Effects of obesity on lower extremity muscle function during walking at two speeds. *Gait Posture*. 2014;39(3):978-984. doi:10.1016/j.gaitpost.2013.12.020.

136. DeVita P, Rider P, Hortobágyi T. Reductions in knee joint forces with weight loss are attenuated by gait adaptations in class III obesity. *Gait Posture*. 2016;45:25-30. doi:10.1016/j.gaitpost.2015.12.040.

137. Harding GT, Hubley-Kozey CL, Dunbar MJ, Stanish WD, Astephen Wilson JL. Body mass index affects knee joint mechanics during gait differently with and without moderate knee osteoarthritis. *Osteoarthr Cartil*. 2012;20(11):1234-1242.

doi:10.1016/j.joca.2012.08.004.

138. Vismara L, Romei M, Galli M, et al. Clinical implications of gait analysis in the rehabilitation of adult patients with “Prader-Willi” Syndrome: a cross-sectional comparative study (“Prader-Willi” Syndrome vs matched obese patients and healthy subjects). *J Neuroeng Rehabil*. 2007;4:14. doi:10.1186/1743-0003-4-14.

139. Segal NA, Yack HJ, Khole P. Weight, Rather Than Obesity Distribution, Explains Peak External Knee Adduction Moment During Level Gait. *Am J Phys Med Rehabil*. 2009;88(3):180-191. doi:10.1097/PHM.0b013e318198b51b.

140. De Souza SAF, Faintuch J, Valezi AC, et al. Gait cinematic analysis in morbidly obese patients. *Obes Surg*. 2005;15(9):1238-1242. doi:10.1381/096089205774512627.

141. Browning RC, Kram R. Effects of obesity on the biomechanics of walking at different speeds. *Med Sci Sports Exerc*. 2007;39(9):1632-1641. doi:10.1249/mss.0b013e318076b54b.

142. DeVita P, Hortobágyi T. Obesity is not associated with increased knee joint torque and power during level walking. *J Biomech*. 2003. doi:10.1016/S0021-9290(03)00119-2.

143. Messier SP, Davies a B, Moore DT, Davis SE, Pack RJ, Kazmar SC. Severe obesity: effects on foot mechanics during walking. *Foot ankle Int / Am Orthop Foot Ankle Soc [and] Swiss Foot Ankle Soc*. 1994;15(1):29-34. doi:10.1177/107110079401500106.

144. Glave A, Brezzo R, Applegate D, Olson J. The effects of obesity classification method on select kinematic gait variables in adult females. 2014;54:197-202.

145. Ko SU, Stenholm S, Ferrucci L. Characteristic gait patterns in older adults with obesity-Results from the Baltimore Longitudinal Study of Aging. *J Biomech*. 2010;43(6):1104-1110. doi:10.1016/j.jbiomech.2009.12.004.

146. Browning RC, McGowan CP, Kram R. Obesity does not increase external mechanical work per kilogram body mass during walking. *J Biomech*. 2009;42(14):2273-2278. doi:10.1016/j.jbiomech.2009.06.046.

147. DeVita P, Hortobagyi T. Obesity is not associated with increased knee joint torque and power during level walking. *J Biomech*. 2003;36(9):1355-1362. doi:10.1016/S0021-9290(03)00119-2.

148. Henriksen M, Simonsen EB, Alkjær T, et al. Increased joint loads during walking - A consequence of pain relief in knee osteoarthritis. *Knee*. 2006;13(6):445-450. doi:10.1016/j.knee.2006.08.005.

149. Saari T, Tranberg R, Zügner R, Uvehammer J, Kärrholm J. Total knee replacement influences both knee and hip joint kinematics during stair climbing. *Int Orthop*. 2004;28(2):82-86. doi:10.1007/s00264-003-0525-y.

150. Levinger P, Menz HB, Morrow AD, Feller JA, Bartlett JR, Bergman NR. Lower limb biomechanics in individuals with knee osteoarthritis before and after total knee arthroplasty surgery. *J Arthroplasty*. 2013;28(6):994-999. doi:10.1016/j.arth.2012.10.018.

151. Yoshida Y, Mizner RL, Snyder-Mackler L. Association between long-term quadriceps weakness and early walking muscle co-contraction after total knee arthroplasty. *Knee*. 2013;20(6):426-431. doi:10.1016/j.knee.2012.12.008.

152. Hills AP, Hennig EM, Byrne NM, Steele JR. The biomechanics of adiposity - Structural and functional limitations of obesity and implications for movement. *Obes Rev*. 2002. doi:10.1046/j.1467-789X.2002.00054.x.

153. Nantel J, Brochu M, Prince F. Locomotor strategies in obese and non-obese children. *Obesity*. 2006. doi:10.1038/oby.2006.206.

154. Błaszczyk JW, Cieślinska-Świder J, Plewa M, Zahorska-Markiewicz B, Markiewicz A. Effects of excessive body weight on postural control. *J Biomech*. 2009. doi:10.1016/j.jbiomech.2009.03.006.

155. Cruz-Gómez NS, Plascencia G, Villanueva-Padrón LA, Jáuregui-Renaud K. Influence of obesity and gender on the postural stability during upright stance. *Obes Facts*. 2011. doi:10.1159/000329408.

156. Son SM. Influence of Obesity on Postural Stability in Young Adults. *Osong Public Heal Res Perspect*. 2016. doi:10.1016/j.phrp.2016.10.001.

157. Melzer I, Oddsson LIE. Altered characteristics of balance control in obese older adults. *Obes Res Clin Pract*. 2016. doi:10.1016/j.orcp.2015.05.016.

158. Cieślińska-Świder J, Furmanek MP, Błaszczyk JW. The influence of adipose tissue location on postural control. *J Biomech*. 2017. doi:10.1016/j.jbiomech.2017.06.027.

159. de Carvalho RT, Santos Neto DB, Chammas V, Arrebola LS, Colombo ML, Scalizi Junior C. Influence of body mass index in revision total knee arthroplasty. *Acta Ortop Bras*. 2015;23(6). doi:10.1590/1413-785220152306140199.

160. Felson DT. Epidemiology of hip and knee osteoarthritis. *Epidemiol Rev*. 1988;10(1):1-28. doi:10.1038/nrrheum.2010.191.

161. Sarzi-Puttini P, Cimmino MA, Scarpa R, et al. Osteoarthritis: An overview of the disease and its treatment strategies. *Semin Arthritis Rheum*. 2005;35(1 SUPPL. 1):1-10. doi:10.1016/j.semarthrit.2005.01.013.

162. Kraus, V. B.; Blanco, Francisco; Englund, Martin; Karsdal, Morten; Lohmander S. Call for Standardized Definitions of Osteoarthritis and Risk Stratification for Clinical Trials and Clinical Use. *Osteoarthr Cartil*. 2015;23(8):1233-1241.

doi:10.1016/j.joca.2015.03.036.Call.

163. International ORS. Definition of OA. https://www.oarsi.org/research/standardization-osteoarthritis-definitions.

164. Huskisson EC, Dieppe PA, Tucker AK, Cannell LB. Another look at osteoarthritis. *Ann Rheum Dis*. 1979;38(5):423-428. http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1000386&tool=pmcentrez&rendertype=abstract.

165. Geenen R, Overman CL, Christensen R, et al. EULAR recommendations for the health professional's approach to pain management in inflammatory arthritis and osteoarthritis. *Ann Rheum Dis*. 2018;77(6):797-807. doi:10.1136/annrheumdis-2017-212662.

166. Salehi-Abari I. Autoimmune Diseases and Therapeutic Approaches Open Access 2016 ACR Revised Criteria for Early Diagnosis of Knee Osteoarthritis. *Autoimmune Dis Ther Approaches*. 2016. doi:10.14437/2378-6337-3-118.

167. Zhang W, Doherty M, Peat G, et al. EULAR evidence-based recommendations for the diagnosis of knee osteoarthritis. *Ann Rheum Dis*. 2010. doi:10.1136/ard.2009.113100.

168. Arden N, Nevitt MC. Osteoarthritis: Epidemiology. *Best Pract Res Clin Rheumatol*. 2006;20(1):3-25. doi:10.1016/j.berh.2005.09.007.

169. Michael JW, Schluter-Brust KU, Eysel P. The Epidemiology, Etiology, Diagnosis, and Treatment of Osteoarthritis of the Knee. *Dtsch Arztebl Int*. 2010;107(9):152-162. doi:10.3238/arztebl.2010.0152.

170. Brandt KD, Radin EL, Dieppe P a. Yet more evidence that osteoarthritis is not a cartilage disease. *Ann Rheum Dis*. 2006;65:1261-1265. doi:doi: 10.1136/ard.2006.058347.

171. Silverwood V, Blagojevic-Bucknall M, Jinks C, Jordan JL, Protheroe J, Jordan KP.

Current evidence on risk factors for knee osteoarthritis in older adults: A systematic review and meta-analysis. *Osteoarthr Cartil*. 2015. doi:10.1016/j.joca.2014.11.019.

172. Leyland KM, Judge A, Javaid MK, et al. Obesity and the Relative Risk of Knee Replacement Surgery in Patients With Knee Osteoarthritis: A Prospective Cohort Study. *Arthritis Rheumatol*. 2016;68(4):817-825. doi:10.1002/art.39486.

173. Grotle M, Hagen KB, Natvig B, Dahl FA, Kvien TK. Obesity and osteoarthritis in knee, hip and/or hand: an epidemiological study in the general population with 10 years follow-up. *BMC Musculoskelet Disord*. 2008;9:132. doi:10.1186/1471-2474-9-132.

174. Yusuf E, Nelissen RG, Ioan-Facsinay A, et al. Association between weight or body mass index and hand osteoarthritis: a systematic review. *Ann Rheum Dis*. 2010;69(4):761-765. doi:10.1136/ard.2008.106930.

175. Oliveria S, Felson D, Cirillo P, Reed J, Walker A. Body weight, body mass index, and incident symptomatic osteoarthritis of the hand, hip, and knee. *Epidemiology*. 1999;10(2):161-166. doi:10.1097/00001648-199903000-00013.

176. Andriacchi TP. Valgus alignment and lateral compartment knee osteoarthritis: A biomechanical paradox or new insight into knee osteoarthritis? *Arthritis Rheum*. 2013;65(2):310-313. doi:10.1002/art.37724.

177. Felson DT. Osteoarthritis as a disease of mechanics. *Osteoarthr Cartil*. 2013;21(1):10-15. doi:10.1016/j.joca.2012.09.012.

178. Andriacchi TP, Mündermann A, Smith RL, Alexander EJ, Dyrby CO, Koo S. A framework for the in vivo pathomechanics of osteoarthritis at the knee. *Ann Biomed Eng*. 2004;32(3):447-457. doi:10.1023/B:ABME.0000017541.82498.37.

179. Harding GT, Dunbar MJ, Hubley-Kozey CL, Stanish WD, Astephen Wilson JL. Obesity

is associated with higher absolute tibiofemoral contact and muscle forces during gait with and without knee osteoarthritis. *Clin Biomech*. 2016;31:79-86. doi:10.1016/j.clinbiomech.2015.09.017.

180. Janicka A, Wren TAL, Sanchez MM, et al. Fat mass is not beneficial to bone in adolescents and young adults. *J Clin Endocrinol Metab*. 2007. doi:10.1210/jc.2006-0794.

181. Taes YEC, Lapauw B, Vanbillemont G, et al. Fat mass is negatively associated with cortical bone size in young healthy male siblings. *J Clin Endocrinol Metab*. 2009. doi:10.1210/jc.2008-2501.

182. Ding C, Cicuttini F, Scott F, Cooley H, Jones G. Knee structural alteration and BMI: A cross-sectional study. *Obes Res*. 2005. doi:10.1038/oby.2005.47.

183. Andriacchi TP, Koo S, Scanlan SF. Gait Mechanics Influence Healthy Cartilage Morphology and Osteoarthritis of the Knee. *J Bone Jt Surg*. 2009;91(Supplement 1):95-101. doi:10.2106/JBJS.H.01408.

184. Bennell KL, Bowles K-A, Wang Y, Cicuttini F, Davies-Tuck M, Hinman RS. Higher dynamic medial knee load predicts greater cartilage loss over 12 months in medial knee osteoarthritis. *Ann Rheum Dis*. 2011;70(10):1770-1774. doi:10.1136/ard.2010.147082.

185. Miyazaki T, Wada M, Kawahara H, Sato M, Baba H, Shimada S. Dynamic load at baseline can predict radiographic disease progression in medial compartment knee osteoarthritis. *Ann Rheum Dis*. 2002;61:617-622. doi:10.1136/ard.61.7.617.

186. Hunter DJ, Niu J, Felson DT, et al. Knee alignment does not predict incident osteoarthritis: The Framingham osteoarthritis study. *Arthritis Rheum*. 2007. doi:10.1002/art.22508.

187. Lim BW, Kemp G, Metcalf B, et al. The association of quadriceps strength with the knee

adduction moment in medial knee osteoarthritis. *Arthritis Rheum*. 2009. doi:10.1002/art.24278.

188. Miyazaki T, Wada M, Kawahara H, Sato M, Baba H, Shimada S. Dynamic load at baseline can predict radiographic disease progression in medial compartment knee osteoarthritis. *Ann Rheum Dis*. 2002. doi:10.1136/ard.61.7.617.

189. Sharma L, Hurwitz DE, Thonar EJMA, et al. Knee adduction moment, serum hyaluronan level, and disease severity in medial tibiofemoral osteoarthritis. *Arthritis Rheum*. 1998. doi:10.1002/1529-0131(199807)41:7<1233::AID-ART14>3.0.CO;2-L.

190. Berti L, Benedetti MG, Ensini A, Catani F, Giannini S. Clinical and biomechanical assessment of patella resurfacing in total knee arthroplasty. *Clin Biomech*. 2006;21(6):610-616. doi:10.1016/j.clinbiomech.2006.01.002.

191. Syed IY, Davis BL. Obesity and osteoarthritis of the knee: hypotheses concerning the relationship between ground reaction forces and quadriceps fatigue in long-duration walking. *Med Hypotheses*. 2000;54(2):182-185. doi:10.1054/mehy.1999.0013.

192. Henriksen M, Alkjaer T, Lund H, et al. Experimental quadriceps muscle pain impairs knee joint control during walking. *J Appl Physiol*. 2007. doi:10.1152/japplphysiol.01105.2006.

193. Deluzio KJ, Astephen JL. Biomechanical features of gait waveform data associated with knee osteoarthritis. An application of principal component analysis. *Gait Posture*. 2007. doi:10.1016/j.gaitpost.2006.01.007.

194. Cerejo R, Dunlop DD, Cahue S, Channin D, Song J, Sharma L. The influence of alignment on risk of knee osteoarthritis progression according to baseline stage of disease. *Arthritis Rheum*. 2002. doi:10.1002/art.10530.

195. Hunter DJ, Sharma L, Skaife T. Alignment and osteoarthritis of the knee. In: *Journal of*

Bone and Joint Surgery - Series A. ; 2009. doi:10.2106/JBJS.H.01409.

196. Finks J, Osborne N, Birkmeyer JD. Trends in Hospital Volume and Operative Mortality for High-Risk Surgery — NEJM. *N Engl J Med*. 2011;364(22):2128-2137. doi:10.1056/NEJMsa1010705.

197. Krummenauer F, Wolf C, Gunther K, Kirschner S. Clinical Benefit and Cost Effectiveness of Total Knee Arthroplasty in the Older Patient. *Eur J Med Res*. 2009;14:76-84. doi:10.1186/2047-783X-14-2-76.

198. Losina E, Walensky RP, Kessler CL, et al. Cost-effectiveness of total knee arthroplasty in the United States: patient risk and hospital volume. *Arch Intern Med*. 2009;169(12):1113-21; discussion 1121-2. doi:10.1001/archinternmed.2009.136.

199. Hootman JM, Helmick CG. Projections of US prevalence of arthritis and associated activity limitations. *Arthritis Rheum*. 2006;54(1):226-229. doi:10.1002/art.21562.

200. Cram P, Lu X, Kates SL, Singh JA, Li Y, Wolf BR. Total Knee Arthroplasty Volume, Utilization, and Outcomes Among Medicare Beneficiaries, 1991-2010. *JAMA*. 2012;308(12):1227. doi:10.1001/2012.jama.11153.

201. Kurtz S, Ong K, Lau E, Mowat F, Halpern M. Projections of primary and revision hip and knee arthroplasty in the United States from 2005 to 2030. *J Bone Joint Surg Am*. 2007;89(4):780-785. doi:10.2106/JBJS.F.00222.

202. Hame SL, Alexander RA. Knee osteoarthritis in women. *Curr Rev Musculoskelet Med*. 2013;6(2):182-187. doi:10.1007/s12178-013-9164-0.

203. Rand JA, Trousdale RT, Ilstrup DM, Ws, Harmsen W. Factors affecting the durability of primary total knee prostheses. *J Bone Jt Surg Am*. 2003;85-A(2):259-265. http://www.google.com/patents?hl=en&lr=&vid=USPAT4462120&id=dp51AAAAEBAJ

&oi=fnd&dq=Total+Knee+Prostheses&printsec=abstract%5Cnhttp://jbjs.org/article.aspx?articleID=25788%5Cnhttp://www.ejbjs.org/cgi/content/abstract/85/2/259.

204. Zhao D, Banks SA, Mitchell KH, D'Lima DD, Colwell CW, Fregly BJ. Correlation between the Knee Adduction Torque and Medial Contact Force for a Variety of Gait Patterns. *Anticancer Res*. 1991;11(4):1609-1612. doi:10.1002/jor.

205. Halder A, Kutzner I, Graichen F, Heinlein B, Beier A, Bergmann G. Influence of Limb Alignment on Mediolateral Loading in Total Knee Replacement. *J Bone Jt Surgery-American Vol*. 2012;94(11):1023-1029. doi:10.2106/JBJS.K.00927.

206. Ritter MA, Davis KE, Davis P, et al. Preoperative malalignment increases risk of failure after total knee arthroplasty. *J Bone Joint Surg Am*. 2013;95(2):126-131. doi:10.2106/JBJS.K.00607.

207. Werner FW, Ayers DC, Maletsky LP, Rullkoetter PJ. The effect of valgus/varus malalignment on load distribution in total knee replacements. *J Biomech*. 2005;38(2):349-355. doi:10.1016/j.jbiomech.2004.02.024.

208. Hilding MB, Lanshammar H, Ryd L. Knee joint loading and tibial component loosening. RSA and gait analysis in 45 osteoarthritic patients before and after TKA. *J Bone Joint Surg Br*. 1996;78(1):66-73.

209. Hilding MB, Ryd L, Toksvig-Larsen S, Mann A, Stenström A. Gait affects tibial component fixation. *J Arthroplasty*. 1999;14(5):589-593. doi:10.1016/S0883-5403(99)90082-7.

210. Hatfield GL, Hubley-Kozey CL, Astephen Wilson JL, Dunbar MJ. The Effect of Total Knee Arthroplasty on Knee Joint Kinematics and Kinetics During Gait. *J Arthroplasty*. 2011;26(2):309-318. doi:10.1016/j.arth.2010.03.021.

211. Rahman J, Tang Q, Monda M, Miles J, McCarthy I. Gait assessment as a functional outcome measure in total knee arthroplasty: a cross-sectional study. *BMC Musculoskelet Disord*. 2015;16:66. doi:10.1186/s12891-015-0525-2.

212. George J, Klika AK, Navale SM, Newman JM, Barsoum WK, Higuera CA. Obesity Epidemic: Is Its Impact on Total Joint Arthroplasty Underestimated? An Analysis of National Trends. *Clin Orthop Relat Res*. 2017. doi:10.1007/s11999-016-5222-4.

213. Woon CYL, Piponov H, Schwartz BE, et al. Total Knee Arthroplasty in Obesity: In-Hospital Outcomes and National Trends. *J Arthroplasty*. 2016;31(11):2408-2414. doi:10.1016/j.arth.2016.04.028.

214. Derman PB, Fabricant PD, David G. The Role of Overweight and Obesity in Relation to the More Rapid Growth of Total Knee Arthroplasty Volume Compared with Total Hip Arthroplasty Volume. *J Bone Jt Surgery-American Vol*. 2014;96(11):922-928. doi:10.2106/JBJS.L.01731.

215. Lozano LM, López V, Ríos J, et al. Better outcomes in severe and morbid obese patients (BMI > 35 kg/m2) in primary Endo-Model rotating-hinge total knee arthroplasty. *ScientificWorldJournal*. 2012;2012:249391. doi:10.1100/2012/249391.

216. Bonnefoy-Mazure A, Martz P, Armand S, et al. Influence of Body Mass Index on Sagittal Knee Range of Motion and Gait Speed Recovery 1-Year After Total Knee Arthroplasty. *J Arthroplasty*. 2017;32(8):2404-2410. doi:10.1016/j.arth.2017.03.008.

217. Odum SM, Springer BD, Dennos AC, Fehring TK. National Obesity Trends in Total Knee Arthroplasty. *J Arthroplasty*. 2013;28(8, Supplement):148-151. doi:http://dx.doi.org/10.1016/j.arth.2013.02.036.

218. Malinzak RA, Ritter MA, Berend ME, Meding JB, Olberding EM, Davis KE. Morbidly

Obese, Diabetic, Younger, and Unilateral Joint Arthroplasty Patients Have Elevated Total Joint Arthroplasty Infection Rates. *J Arthroplasty*. 2009;24(6 SUPPL.):84-88. doi:10.1016/j.arth.2009.05.016.

219. Namba RS, Paxton L, Fithian DC, Stone M Lou. Obesity and perioperative morbidity in total hip and total knee arthroplasty patients. In: *Journal of Arthroplasty*. Vol 20. ; 2005:46-50. doi:10.1016/j.arth.2005.04.023.

220. Samson AJ, Mercer GE, Campbell DG. Total knee replacement in the morbidly obese: A literature review. *ANZ J Surg*. 2010;80(9):595-599. doi:10.1111/j.1445-2197.2010.05396.x.

221. Schwarzkopf R, Thompson SL, Adwar SJ, Liublinska V, Slover JD. Postoperative complication rates in the "super-obese" hip and knee arthroplasty population. *J Arthroplasty*. 2012;27(3):397-401. doi:10.1016/j.arth.2011.04.017.

222. Prohaska MG, Keeney BJ, Beg HA, et al. Preoperative body mass index and physical function are associated with length of stay and facility discharge after total knee arthroplasty. *Knee*. 2017;24(3):634-640. doi:10.1016/j.knee.2017.02.005.

223. Foran JRH, Mont M a, Etienne G, Jones LC, Hungerford DS. The outcome of total knee arthroplasty in obese patients. *J Bone Joint Surg Am*. 2004;86-A(8):1609-1615. doi:86/8/1609 [pii].

224. Adhikary SD, Liu W-M, Memtsoudis SG, Davis CM, Liu J. Body Mass Index More Than 45 kg/m2 as a Cutoff Point Is Associated With Dramatically Increased Postoperative Complications in Total Knee Arthroplasty and Total Hip Arthroplasty. *J Arthroplasty*. 2016;31(4):749-753. doi:10.1016/j.arth.2015.10.042.

225. Baker P, Petheram T, Jameson S, Reed M, Gregg P, Deehan D. The association between

body mass index and the outcomes of total knee arthroplasty. *J Bone Joint Surg Am*. 2012;94(16):1501-1508. doi:10.2106/JBJS.K.01180.

226. Dowsey MM, Liew D, Stoney JD, Choong PF. The impact of pre-operative obesity on weight change and outcome in total knee replacement: A PROSPECTIVE STUDY OF 529 CONSECUTIVE PATIENTS. *J Bone Jt Surg - Br Vol*. 2010;92-B(4):513-520. doi:10.1302/0301-620X.92B4.23174.

227. George J, Piuzzi NS, Ng M, Sodhi N, Khlopas AA, Mont MA. Association Between Body Mass Index and Thirty-Day Complications After Total Knee Arthroplasty. *J Arthroplasty*. 2017. doi:10.1016/j.arth.2017.09.038.

228. D'Apuzzo MR, Novicoff WM, Browne JA. The John Insall Award: Morbid Obesity Independently Impacts Complications, Mortality, and Resource Use After TKA. *Clin Orthop Relat Res*. 2015;473(1):57-63. doi:10.1007/s11999-014-3668-9.

229. Werner BC, Evans CL, Carothers JT, Browne JA. Primary Total Knee Arthroplasty in Super-obese Patients: Dramatically Higher Postoperative Complication Rates Even Compared to Revision Surgery. *J Arthroplasty*. 2015;30(5):849-853. doi:10.1016/j.arth.2014.12.016.

230. Paterson KL, Sosdian L, Hinman RS, et al. The influence of sex and obesity on gait biomechanics in people with severe knee osteoarthritis scheduled for arthroplasty. *Clin Biomech*. 2017;49(August):72-77. doi:10.1016/j.clinbiomech.2017.08.013.

231. Pua YH, Seah FJ, Seet FJ, Tan JW, Liaw JS, Chong HC. Sex Differences and Impact of Body Mass Index on the Time Course of Knee Range of Motion, Knee Strength, and Gait Speed After Total Knee Arthroplasty. *Arthritis Care Res*. 2015;67(10):1397-1405. doi:10.1002/acr.22584.

232. Benoit DL, Ramsey DK, Lamontagne M, Xu L, Wretenberg P, Renström P. Effect of skin movement artifact on knee kinematics during gait and cutting motions measured in vivo. *Gait Posture*. 2006;24(2):152-164. doi:10.1016/j.gaitpost.2005.04.012.

233. Debbi EM, Bernfeld B, Herman A, et al. Frontal plane biomechanics of the operated and non-operated knees before and after unilateral total knee arthroplasty. *Clin Biomech*. 2015;30(9):889-894. doi:10.1016/j.clinbiomech.2015.07.013.

234. de Achaval S, Kallen MA, Amick B, et al. Patients' expectations about total knee arthroplasty outcomes. *Heal Expect*. 2016;19(2):299-308. doi:10.1111/hex.12350.

235. Artz N, Elvers KT, Lowe CM, Sackley C, Jepson P, Beswick AD. Effectiveness of physiotherapy exercise following total knee replacement: systematic review and meta-analysis. *BMC Musculoskelet Disord*. 2015;16:15. doi:10.1186/s12891-015-0469-6.

236. Collins TD, Ghoussayni SN, Ewins DJ, Kent JA. A six degrees-of-freedom marker set for gait analysis: Repeatability and comparison with a modified Helen Hayes set. *Gait Posture*. 2009;30(2):173-180. doi:10.1016/j.gaitpost.2009.04.004.

237. Cappozzo A, Cappello A, Croce UD, Pensalfini F. Surface-maker cluster design criteria for 3-D bone movement reconstruction. *IEEE Trans Biomed Eng*. 1997;44(12):1165-1174. doi:10.1109/10.649988.

238. C-Motion. Visual 3D. *C-Motion Res Biomech*. 2012:1. http://www.c-motion.com/.

239. Vaughan CL, Davis BL, Christopher L, Connor JCO, O'Connor JC. *Dynamics of Human Gait*.; 1992. doi:10.1094/PHP-2007-0719-08-RV; (2002), SAS Institute. Version 9.2. SAS Institute IncSloderbeck, P.E., Michaud, J.P., Whitworth, R.J., (2008) Sorghum Insect Management., , Kansas State University Extension Publication MF-742; Sparks, A.N., A review of the biology of the fall armyworm (1979) The Florida Entomologist, 62, pp. 82-

87; Stichler, C.S., McFarland, M., Coffman, C., (1997) Irrigated and Dryland Grain Sorghum Production: South and Southwest Texas, , http://lubbock.tamu.edu/files/2011/10/b604.

240. Wilken JM, Rodriguez KM, Brawner M, Darter BJ. Reliability and minimal detectible change values for gait kinematics and kinetics in healthy adults. *Gait Posture*. 2012;35(2):301-307. doi:10.1016/j.gaitpost.2011.09.105.

241. Bohannon RW, Glenney SS. Minimal clinically important difference for change in comfortable gait speed of adults with pathology: A systematic review. *J Eval Clin Pract*. 2014. doi:10.1111/jep.12158.

242. Tudor-Locke CE, Barreira T V., Brouillette RM, Foil HC, Keller JN. Preliminary Comparison of Clinical and Free-Living Measures of Stepping Cadence in Older Adults. *J Phys Act Heal*. 2013. doi:10.1123/jpah.10.8.1175.

243. Tudor-Locke C, Han H, Aguiar EJ, et al. How fast is fast enough? Walking cadence (steps/min) as a practical estimate of intensity in adults: a narrative review. *Br J Sports Med*. 2018. doi:10.1136/bjsports-2017-097628.

244. Elble RJ, Thomas SS, Higgins C, Colliver J. Stride-dependent changes in gait of older people. *J Neurol*. 1991. doi:10.1007/BF00319700.

245. Piazza SJ, Delp SL. The influence of muscles on knee flexion during the swing phase of gait. *J Biomech*. 1996. doi:10.1016/0021-9290(95)00144-1.

246. Øiestad BE, Juhl CB, Eitzen I, Thorlund JB. Knee extensor muscle weakness is a risk factor for development of knee osteoarthritis. A systematic review and meta-analysis. *Osteoarthr Cartil*. 2015. doi:10.1016/j.joca.2014.10.008.

247. Mizner RL, Snyder-Mackler L. Altered loading during walking and sit-to-stand is affected

by quadriceps weakness after total knee arthroplasty. *J Orthop Res*. 2005. doi:10.1016/j.orthres.2005.01.021.

248. Zhao D, Banks SA, D'Lima DD, Colwell CW, Fregly BJ. In vivo medial and lateral tibial loads during dynamic and high flexion activities. *J Orthop Res*. 2007. doi:10.1002/jor.20362.

249. El-Hawary R, Roth SE, King GJW, Chess DG, Johnson J a. Load balance in total knee arthroplasty: an in vitro analysis. *Int J Med Robot*. 2006. doi:10.1002/rcs.

250. Outerleys, J; Dunbar, M; Hubley-Kozey, C; Ikeda, D; Astepen Wilson J. The Effect of Body Mass Index on Gait Mechanics and Muscle Activity Before and After Total Knee Arthroplasty Surgery. 2017.

251. Telfer S, Lange MJ, Sudduth ASM. Factors influencing knee adduction moment measurement: A systematic review and meta-regression analysis. *Gait Posture*. 2017. doi:10.1016/j.gaitpost.2017.08.025.

252. Mills K, Hunt MA, Ferber R. Biomechanical deviations during level walking associated with knee osteoarthritis: A systematic review and meta-analysis. *Arthritis Care Res*. 2013. doi:10.1002/acr.22015.

253. Van Tunen JAC, Dell'Isola A, Juhl C, et al. Association of malalignment, muscular dysfunction, proprioception, laxity and abnormal joint loading with tibiofemoral knee osteoarthritis - A systematic review and meta-analysis. *BMC Musculoskelet Disord*. 2018. doi:10.1186/s12891-018-2202-8.

254. Hatfield GL, Stanish WD, Hubley-Kozey CL. Three-dimensional biomechanical gait characteristics at baseline are associated with progression to total knee arthroplasty. *Arthritis Care Res*. 2015;67(7):1004-1014. doi:10.1002/acr.22564.

255. Biggs, Paul Robert; Whatling, Gemma Marie; Wilson, Chris; Metcalfe, Andrew John; Holt CA. Which Osteoarthritic Gait Features Recover Following TOtal Knee Replacement Surgery? 2017.

256. Milner CE, Meardon SA, Hawkins illian L, Willson JD. Walking velocity and step length adjustments affect knee joint contact forces in healthy weight and obese adults. *J Orthop Res*. 2018. doi:10.1002/pi.5316.

257. McKean KA, Landry SC, Hubley-Kozey CL, Dunbar MJ, Stanish WD, Deluzio KJ. Gender differences exist in osteoarthritic gait. *Clin Biomech*. 2007;22(4):400-409. doi:10.1016/j.clinbiomech.2006.11.006.

258. Ko S uk, Tolea MI, Hausdorff JM, Ferrucci L. Sex-specific differences in gait patterns of healthy older adults: Results from the Baltimore Longitudinal Study of Aging. *J Biomech*. 2011. doi:10.1016/j.jbiomech.2011.05.005.

259. Thewlis D, Richards J, Bower J. Discrepancies in knee joint moments using common anatomical frames defined by different palpable landmarks. *J Appl Biomech*. 2008. doi:10.1123/jab.24.2.185.

260. Steele KM, van der Krogt MM, Schwartz MH, Delp SL. How much muscle strength is required to walk in a crouch gait? *J Biomech*. 2012. doi:10.1016/j.jbiomech.2012.07.028.

261. Matrangola SL, Madigan ML, Nussbaum MA, Ross R, Davy KP. Changes in body segment inertial parameters of obese individuals with weight loss. *J Biomech*. 2008. doi:10.1109/ACSAT.2013.56.

262. Kahn TL, Schwarzkopf R. Does Total Knee Arthroplasty Affect Physical Activity Levels? Data from the Osteoarthritis Initiative. *J Arthroplasty*. 2015;30(9):1521-1525. doi:10.1016/j.arth.2015.03.016.

263. Harding PA, Holland AE, Hinman RS, Delany C. Physical activity perceptions and beliefs following total hip and knee arthroplasty: a qualitative study. *Physiother Theory Pract*. 2015;31(2):107-113. doi:10.3109/09593985.2014.959581.

264. Clauser CE, McConville JT, Young JW. Weight, Volume, and Center of Mass of Segments of the Human Body. *AMRL Tech Rep No 69-70*. 1969. doi:AMRL-TR- 69-70 (AD 710 622).

265. Rivière C, Iranpour F, Auvinet E, et al. Alignment options for total knee arthroplasty: A systematic review. *Orthop Traumatol Surg Res*. 2017. doi:10.1016/j.otsr.2017.07.010.

266. Benedetti MG, Catani F, Leardini A, Pignotti E, Giannini S. Data management in gait analysis for clinical applications. *Clin Biomech*. 1998;13(3):204-215. doi:10.1016/S0268-0033(97)00041-7.

267. Cappozzo A, Catani F, Della Croce U, Leardini A. Position and orientation in space of bones during movement: anatomical frame definition and determination. *Clin Biomech*. 1995;10(4):171-178. doi:10.1016/0268-0033(95)91394-T.

268. Kadaba MP, Ramakrishnan HK, Wootten ME. Measurement of Lower-Extremity Kinematics during Level Walking. *J Orthop Res*. 1990;8:383-392. doi:10.1002/jor.1100080310.

269. Davis RB, Ounpuu S, Tyburski D, Gage JR. A gait analysis data collection and reduction technique. *Hum Mov Sci*. 1991;10(5):575-587. doi:10.1016/0167-9457(91)90046-Z.

270. Manal K, McClay I, Stanhope S, Richards J, Galinat B. Comparison of surface mounted markers and attachment methods in estimating tibial rotations during walking: An in vivo study. *Gait Posture*. 2000;11(1):38-45. doi:10.1016/S0966-6362(99)00042-9.

271. Ko S uk, Ling SM, Winters J, Ferrucci L. Age-related mechanical work expenditure

during normal walking: The Baltimore Longitudinal Study of Aging. *J Biomech*. 2009;42(12):1834-1839. doi:10.1016/j.jbiomech.2009.05.037.

272. Vartiainen P, Bragge T, Lyytinen T, Hakkarainen M, Karjalainen PA, Arokoski JP. Kinematic and kinetic changes in obese gait in bariatric surgery-induced weight loss. *J Biomech*. 2012;45(10):1769-1774. doi:10.1016/j.jbiomech.2012.05.002.

273. Tanikawa H, Tada M, Harato K, Okuma K, Nagura T. Influence of Total Knee Arthroplasty on Patellar Kinematics and Patellofemoral Pressure. *J Arthroplasty*. 2016. doi:10.1016/j.arth.2016.06.044.

274. Forrester SE. Selecting the number of trials in experimental biomechanics studies. *Int Biomech*. 2015;2(1):62-72. doi:10.1080/23335432.2015.1049296.

275. Horsak B, Schwab C, Clemens C, et al. Is the reliability of 3D kinematics of young obese participants dependent on the hip joint center localization method used? *Gait Posture*. 2018. doi:10.1016/j.gaitpost.2017.09.029.

276. Kainz H, Carty CP, Modenese L, Boyd RN, Lloyd DG. Estimation of the hip joint centre in human motion analysis: A systematic review. *Clin Biomech*. 2015. doi:10.1016/j.clinbiomech.2015.02.005.

277. Fiorentino NM, Atkins PR, Kutschke MJ, Foreman KB, Anderson AE. In-vivo quantification of dynamic hip joint center errors and soft tissue artifact. *Gait Posture*. 2016. doi:10.1016/j.gaitpost.2016.09.011.

278. Dempster WT. Space requirements of the seated operator: geometrical, kinematic, and mechanical aspects other body with special reference to the limbs. *WADC Tech Rep*. 1955. doi:AD 087892.

Appendices

Appendix A: Marker placement assumptions, marker type and common placement

Retroreflective markers are used according to a standard arrangement (marker set). There has been a lack of standardized procedure for gait analysis both for single patient and large sample of patients.[266] Capello et al, 1995[267] suggested the following for marker point selection:

1. Sufficient markers must be visible to the cameras at any given time.
2. Light emitted or reflected from markers should be oriented within the field of view of a sufficient number of cameras.
3. The distance between three markers associated with each body segment and the offset of any marker from the line joining the other two should be sufficiently large so that error propagation from reconstructed marker coordinates to the bone orientation in space will be minimal.
4. The relative movement between markers and underlying bone should be minimal.
5. Mounting the markers on the experimental subject should be easy
6. It should be possible to place markers despite the presence of appliances such as orthoses, prostheses or external fracture fixators.

One of the first recognized biomechanical marker set was developed at the Helen Hayes hospital (described by Kadaba et al., 1990[268]). It has become the basis to which most modern marker sets have been derived from.[236] In a systematic review of 87 full text articles, the most common frequently named marker-set of 26 (34.7%) was the Vicon Plug-In Gait model and Helen Hayes model whereas 36 (48%) articles did not refer to a specific marker-set. [268] Most common anatomical landmarks include (Table 2 below): anterior superior iliac spine, posterior superior iliac spine, greater trochanter of the femur, lateral malleolus, lateral epicondyle of femur, sacrum, fibular head, and base of 1st and 5th metatarsal heads.

Methodology of marker placement. There are many different marker sets that have been proposed. A summary marker placement placed on anatomical landmarks can be found in the table 1.

Table A-1. A summary of methodology of marker placement by various studies.

Protocol of marker placement	Author
David	Vismara at al, 2007
Clarke	Messier et al. 1994
Villar	de Souza et al., 2005
Modified Helen Hayes	Vartiaien et al. 2012
Holdm and coworker	Lai et al 2008
Davis, Ounpuu, Tyburski, Gage	Cimolin et al 2014
Obesity	Lerner et al 2013
Lerner	DeVita et al 2016
Cleveland clinic full-body	Messier et al 2014
Sharma	Sharma et al 1998

References from systematic reviews of Komnik 2015 and Kadaba 1990

Most common methodology of marker placement on anatomical landmarks.

Table A-2. A summary of marker placement on anatomical landmarks of the body by various studies.

Marker Placement	Authors
Lateral aspect of foot (lateral and medial malleoli)	Segal et al., 2009, Glave et al., 2014; Vartiaien et al. 2012, Lai et al 2008, (Silvernail et al 2013), Lerner et al 2013, Browning et al 2007, DeVita and Hortobagyi 2003, Tanikawa et al 2016, Arnold et al 2015
Crest of tibia	Segal et al., 2009;
Lateral condyle of femur (femoral tracking device)	Segal et al., 2009, Glave et al., 2014, Lai et al 2008, Pamukoff et al 2016, Lerner et al 2013, Browning et al 2007, DeVita and Hortobagyi 2003, Arnold et al 2015
Sacrum extension*	Segal et al., 2009*; Glave et al., 2014, Lai et al 2008
Upper thoracic extension*	Segal et al., 2009;
Anterior superior iliac spine	Glave et al., 2014, Ko et al 2009, Vartiaien et al. 2012, Lai et al 2008. Pamukoff et al 2016, Lerner et al 2013, DeVita et al 2016, Browning et al 2007. Arnold et al 2015
External border of greater trochanter	Glave et al., 2014, Vartiaien et al. 2012, Lai et al 2008, (Silvernail et al 2013). Browning et al 2007, DeVita and Hortobagyi 2003, Tanikawa et al 2016, Arnold et al 2015
Base of 5th toe, 1th and 5th metatarsal heads	Glave et al., 2014, Ko et al 2009; Vartiaien et al. 2012, Lai et al 2008. Pamukoff et al 2016, Silvernail et al 2013, Lerner et al 2013, Browning et al 2007, DeVita and Hortobagyi 2003, Arnold et al 2015
Calcaneus (posterior)	Glave et al., 2014, Ko et al 2009, Vartiaien et al. 2012, Lai et al 2008, Lerner et al 2013, DeVita et al 2016, Arnold et al 2015
Posterior superior iliac spine	Ko et al 2009, Lerner et al 2013, Arnold et al 2015
Iliac Crest	Ko et al 2009, Silvernail et al 2013
Medial and lateral knees	Ko et al 2009, Vartiaien et al. 2012, Silvernail et al 2013
Biceps femoris	Vartiaien et al. 2012
Gastrocnemius	Vartiaien et al. 2012
Lumbar vertebrae	Vartiaien et al. 2012
Marker clusters to sacrum and shank	Lerner et al 2013, DeVita et al 2016
End of fibular head	Tanikawa et al 2016

*extensions = markers mounted on a board fixed to the skin
$ lateral wands over mid femur and mid tibia (Ko et al 2009, Arnold et al 2015)
% virtual markers of ASIS, medial epicondyle, fibular head, tibial tubercle, medial malleolus, 2nd metatarsal, heel (Harding et al 2012)

Marker attachment: anatomical vs cluster. Markers (preferably balls) for motion capture can be placed anatomically or in clusters directly on the skin or on fixtures attached to the body segment (e.g. clothing). The fixtures can be rigid to reduce photogrammetric error effects or non-rigid. Non-rigid marker placement does not compensate for artifacts due to the relative movement between the skin and bone. Thereby using skin markers or non-rigid fixtures may compensate for these artifacts.

Conventional gait analysis consists of placing single markers on anatomical segments such as the trunk, pelvis, thigh, shank, and foot.[269] However, placing markers on segments has its limitations, the biggest of which is when the segments are large curved areas as well as movement of soft tissue over the bone during movement,[266] which result in errors in ROM of joints and joint angles.[236] To overcome these limitations, clusters of markers on a fixed board placed away from the bony landmarks have found better tracking.[237,267,270]

Appendix B: Link-segment model assumptions

1) Each of the segments has a fixed mass. The mass is considered a point mass located at the segments' center of mass.
2) The location of the center of mass should not change during the movement of the segment.
3) The joint centers are considered to be ball-and-socket or hinged joints.
4) The moment of inertia of each segment about its mass center is constant during movement.
5) The length of each segment remains constant during the movement of the segment. The length of each segment is determined by the distance between the proximal and distal joints of each segment.

Appendix C: Common kinematic and kinetic parameters of gait.

Table D-1. A summary of kinematic and kinetic parameters of the knee by various studies.

Kinematic Parameters	**Authors**
ROM at knee (°)	Vismara at al, 2007, Ko et al 2009, Glave et al. 2013, Harding et al 2012, Arnold et al 2015, Debbi et al 2015
Knee flexion-extension angle (initial contact, early stance, toe off)	DeVita et al 2016, Harding et al 2012, Arnold et al 2015, Vartiaien et al. 2012, Messier et al 2014
Knee abduction-adduction angle (max, peak)	Debbi et al 2015, Lai et al 2008
Kinetic Parameters	**Authors**
Knee adduction-abduction moment in stance (peak)	Runhaar et al, 2011, Harding et al 2012, Messier et al 2014, Debbi et al 2015, Vismara at al, 2007, Vartiaien et al. 2012
Knee flexion-extension moment	Vartiaien et al. 2012, Harding et al 2012, Arnold et al 2015, Ko et al 2009
Knee rotation moment	Harding et al 2012

Appendix D: Common spatio-temporal parameters of gait.

Table E-1. A summary of spatiotemporal parameters used by various studies of gait.

Spatiotemporal Parameters	**Authors**
Cadence (steps/min)	Vismara at al, 2007; Vartiaien et al. 2012; Glave et al., 2013; De Souza et al 2005, Arnold et al 2015, Debbi et al 2015
Swing time (% gait cycle)	Vartiaien et al. 2012; Arnold et al 2015
Double support time (% gait cycle)	Vartiaien et al. 2012;
Duration of stance phase (as % of gait cycle)	Vismara at al, 2007; Lai et al 2008, Harding et al 2012, Arnold et al 2015
Duration of single support (as % of gait cycle)	Vismara at al, 2007; Debbi et al 2015
Stride length (m)	Vismara at al, 2007; Glave et al., 2014; DeVita et al 2016, Harding et al 2012, De Souza et al 2005, Arnold et al 2015, Debbi et al 2015
Walking Velocity (m/s)	Vismara at al, 2007; Glave et al., 2014; Ko et al 2009; Glave et al., 2013; Lai et al 2008, DeVita et al 2016, Harding et al 2012, De Souza et al 2005, Arnold et al 2015
Step width (m)	Glave et al., 2014; Vartiaien et al. 2012; Glave et al., 2013; Browning et al 2007
Stride width (cm)	Ko et al 2009
Support base	De Souza et al 2005

Appendix E: Biomechanical parameter measurements in studies with obesity**.**

Table F-1. A summary of studies of obesity and biomechanics.

Author	n (OB vs N)	Age (OB vs N)	BMI (OB vs N)	Motion assessed	Joints assessed	Orientation	Measurement methods (flash rate)
Vismara et al. 2007[138] Italy	14 vs 20	29.4 vs 30.2	39.2 vs 21.4	Gait- SS	Hip, knee, ankle/foot	All joints in sagittal plane and toe-out angle	Motion analysis system (100 Hz)
Segal et al. 2009[139] USA	40 vs 19	49.2 vs 48.7	35.8 vs 22.8	Gait- SS	Knee, ankle/foot	Knee joint in coronal plane and toe-out angle	Motion analysis system (60 Hz)
De Souza et al 2005[140] Brazil	34	47.2	40.1	Gait- SS	-	Spatial and temporal measurements and toe-out angle	-
Vismara et al 2006	10 vs 10	26.7 vs 29.4	36.1 vs 20.9	Gait- S	Hip, knee, ankle	All joints in sagittal plane	Motion analysis system
Browning and Kram [141]2007 USA	10 vs 10	Young Adults	35.6 vs 22.1	Gait- S	Hip, knee, ankle	All joints in sagittal plane	High speed video (200 Hz)
DeVita and Hortobagyi 2003[147] USA	21 vs 18	39.5 vs 20.8	42.3 vs 22.7	Gait- S	Hip, knee, ankle	All joints in sagittal plane	High speed video (60 Hz)
Messier et al 1994[143] USA	16 vs 13	30.0 vs 35.2	41.4 vs 20.8	Gait- S	Ankle/foot	Coronal plane and toe-out angle	High speed video (100 Hz)
Runhaar et al 2011[48] SR	10+	19+	Various	Various	Hip, knee, ankle		
Glave et al 2014[49] USA	12OW 10	32.25 vs 37.8	31.42 vs 21.71	Gait- SS	Knee	Sagittal, frontal	Motion analysis system
Ko et al, 2009[271] USA	34 vs 74OW vs 56	68.79 vs 67.08 vs 68.88	>=30, >=25-30, <25	Gait –SS and S-max speed walking)	Hip, knee, ankle	Sagittal, frontal	Motion analysis system (60Hz)
Vartianien 2012[272] FIN	13	45.5	42.2	Gait- S	Hip, knee	Sagittal, frontal	High speed camera
Lai et al 2008[46] CHINA	14 vs 14	35.36 vs 27.57	33.06 vs 21.33	Gait- SS	Hip, knee, ankle	Sagittal, frontal	Motion analysis system (60Hz)
Pamukoff et al 2016[132] USA	15 vs 15	21.2 vs 20.4	33.5 vs 21.6	Gait- SS	Knee	Sagittal, frontal	Motion analysis system (100 Hz)
Cimolin et al 2014 [133] Italy	8	28.7	44.2	Gait- SS	Knee	Sagittal	Motion analysis system (100 Hz)
Silvernail et al 2013[134] USA	10 vs 10 vs 10	23.8OB vs 22.6OW vs 23N	34.4OB vs 26.9OW vs 22.4N	Gait- SS	Knee	Sagittal, frontal	Motion analysis system (120 Hz)
Lerner et al 2013[135]USA	5 vs 9	35 vs 26	35 vs 22.1	Gait- S	Muscle forces	Sagittal	Motion analysis system (100 Hz)
DeVita et al 2016[136] USA	10	42.8	43.2	Gait SS & S	Knee	Sagittal	Motion analysis system

Harding et al 2012 [137]CAN	81 vs 95 OW vs 68N	55.9OB, 53OW, 51H	34.9OB, 27.6OW, 22.8H	Gait- SS	Knee	Sagittal	Motion analysis system (100 Hz)
Browning et al 2007[141]	10 vs 10	31/26 vs 31/25	37/34 vs 21/23	Gait- S	Knee	Sagittal, frontal	High speed video

SS= self-selected speed, S= standardized speed.

Appendix F: Gait journal articles investigating TKA.

Table G-1. A summary of articles that investigated TKA and gait.

Author	n	Age	BMI	Motion assessed	Joints assessed	Orientation	Measurement methods (flash rate)
Tanikawa et al 2016[273]	5 (3M, 2F)	92±6.5yrs (cadavers)	-	Patellofemoral pressure, patella offset, patella tilt	Knee	Sagittal	Motion analysis system (120 Hz)
*Komnik et al 2015[54]	Various	Various	Various	Gait (standardized and self-selected speed)	Knee	Sagittal, frontal, transverse	Motion analysis system
Arnold et al 2015[20]	17	67.8	31.8	Gait (self-selected speed)	Knee	Sagittal	Motion analysis system (100Hz)
*Sosdian et al 2014[53]	Various	Various	Various	Various	Knee	Sagittal	
Debbi et al 2015[233]	50	65.9	33.5	Gait (self-selected speed)	Knee	Sagittal	Motion analysis system (100Hz)
Shandiz et al 2016[55]	9	44-82yr	20-38	Gait	Knee	Sagittal	CT scanner, Fluoroscopy

Appendix G: Informed Consent

INFORMATION SHEET

Title of Project: Impact of Body Mass Index on Gait Biomechanics and Patient Reported Outcomes after TKA: A Pilot Study

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Background: In Canada there are increasing numbers of patients who have knee arthritis that may lead to the need for a knee replacement. We are also seeing an increase in the number of patients with knee arthritis and obesity who need a knee replacement. Early reports suggest that patients who live with obesity and have a knee replacement may not have the same outcomes as patients who do not live with obesity and have a knee replacement.

Purpose of Research: We would like to know if patients who live with obesity and receive a knee replacement walk differently than people who do not live with obesity and have a knee replacement. We would also like to know if these two groups of patients report different outcomes after their knee replacement.

Procedures: You are being asked to participate in this study because you are going to have knee replacement surgery and you meet our study requirements. We would like to ask you questions about how you manage your daily activity and how much pain you have before surgery and then again at 6, 12 and 26 weeks after your surgery.

We would also like to measure how you walk in a gait (walking) clinic located at the Glenrose Rehabilitation Hospital (Glenrose). You will attend 3 walking assessments at the Glenrose. The first will be before your surgery. You will walk over a 10 meter surface and your walking will be recorded. Then at twelve weeks after your operation, we would like you to return to the Glenrose to have your walking (gait) examined in the same way as it was before your operation. At 26 weeks after your surgery, we would like you to return to the Glenrose for a final walking assessment. This assessment will be done on a treadmill with your body weight supported by a harness. This will allow you to walk on surfaces like you would use outdoors. At each of these visits, we will also have you complete the questions about how you manage your daily activities and how much pain you have. We will also have you complete the questionnaires when you have attend the planned visit with your surgeon at the Edmonton Bone and Joint Clinic at six weeks after your operation. Each of the appointments at the Glenrose will take approximately 1 hour.

Possible Benefits: There may be no direct benefits to you by participating in this study. You are providing orthopaedic surgeons and other health professionals with information to help them make decisions regarding how to provide the best care for patients who have knee replacement surgery. The results of this study should help to improve care and outcomes for patients who have knee replacement surgery.

Possible Risks: There are few additional risks with taking part in this study. The 2 gait assessments (preoperatively and at 12 weeks after your surgery) are done on a flat surface in the walking lab. You will be able to rest as often as needed and can stop walking at any time. The final walking assessment will be done on a treadmill, but you will wear a harness so that you will not fall. You will decide the speed of walking on the treadmill and will be able to rest as often as needed. You will also be able to stop the walking assessment at any time.

Confidentiality: If you agree to participate in this part of the follow-up study, data will be collected from your medical records, interviews, and from the data collected in the walking assessments. Your Alberta Health Care Number is needed so that the study can obtain your information from your medical records for this study.

Any personal information that you provide for this study will be kept confidential. The program staff will not know any of your responses. Anything that is published from the results of this study will not contain your name. Data will be kept in written, hard-copy and password-protected electronic formats. Printed materials will be stored in a locked filing cabinet in the Collaborative Orthopaedic REsearch (CORe) Office located at the University of Alberta.

The information will be maintained for a minimum of five years after the study is completed and will be stored in CORE (in paper and electronic format) at the University of Alberta. After that time, all paper and electronic formats will be destroyed in a way that ensures privacy and confidentiality. The information from this study may be used to plan future research, but if we do this it will have to be approved by a Research Ethics Board. You may request a report of the research findings and your own results at any time by contacting one of the study investigators.

By consenting to this study, you are giving permission to the research team to access your personal and health information that is needed for the study.

You are free to withdraw from the study at any time.

Parking: You will be given a parking pass for all appointments at the Glenrose so that you do not have to pay for your parking.

Please contact the following investigators if you have any questions or concerns.

Lauren Beaupre, PT PhD 780-492-8626

Allyson Jones, PT PhD 780-492-2020

The plan for this study has been reviewed for its adherence to ethical guidelines by a Health Research Ethics Board at the University of Alberta. For questions regarding participant rights and ethical conduct of research, contact the Research Ethics Office at 780-492-2615. This office has no direct involvement with this project.

CONSENT FORM

Title of Project: Impact of Body Mass Index on Gait Biomechanics and Patient Reported Outcomes after TKA: A Pilot Study

Principal Investigators:
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Dr. Allyson Jones, Departments of Physical Therapy and School of Public Health, University of Alberta
Co-Investigators:
Dr. Albert Vette, Department of Mechanical Engineering, University of Alberta
Dr. Mary Forhan, Departments of Occupational Therapy and Rehabilitation Medicine, University of Alberta
Dr. John Spence, Department of Physical Education and Recreation, University of Alberta
Dr. Sanja Schreiber, Research Associate, Alberta Health Services
C Wayne, MSc Student, Rehabilitation Sciences, University of Alberta

Please circle your answers:

Do you understand that you have been asked to be in a research study?	Yes	No
Have you read and received a copy of the attached information sheet?	Yes	No
Do you understand the benefits and risks involved in taking part in this research study?	Yes	No
Have you had an opportunity to ask questions and discuss this study?	Yes	No
Do you understand that you can quit taking part in this study at any time?	Yes	No
Has how we will keep the data confidential been explained to you?	Yes	No
Do you understand who will have access to your health information?	Yes	No
Do you wish to donate your discarded tissue at time of revision surgery if applicable?	Yes	No

I agree to take part in this study. Yes No

Signature of Research Participant	Printed Name	Date
Patient Healthcare Number	Daytime Phone	Additional Phone
Signature of Investigator/Delegate	Printed Name	Date
Witness Signature	Printed Name	Date

Appendix H: Sample Size

Twenty participants (10 males/10 females) were recruited and assessed pre-TKA. This sample size was determined based on:

1) a study by Forrester, 2015[274] who found that 3 gait trials in experimental biomechanical studies using a paired t-test, has a power of 0.8 with a middle effect size if there are 20 or more participants (using a two-tailed p-value ≤ 0.05).
2) similar gait studies investigating obesity and TKA that reported statistically significant differences in kinematic, kinetic and spatio-temporal outcomes.[20,134,138,139,143] Typically, 5-20 participants were included in these studies.

Future studies should recruit more than 20 participants, as shown by our power calculation from our sample as shown in Table H-1.

Table H-1. Power and effect size calculations

	Power	**Effect Size**
Temporal Spatial	0.06 – 0.08	0.05-0.22
Kinematics	0.35-1	0.18-1.98
Kinetics	0.56-0.99	0.59-1.3

Appendix I: Model marker placement

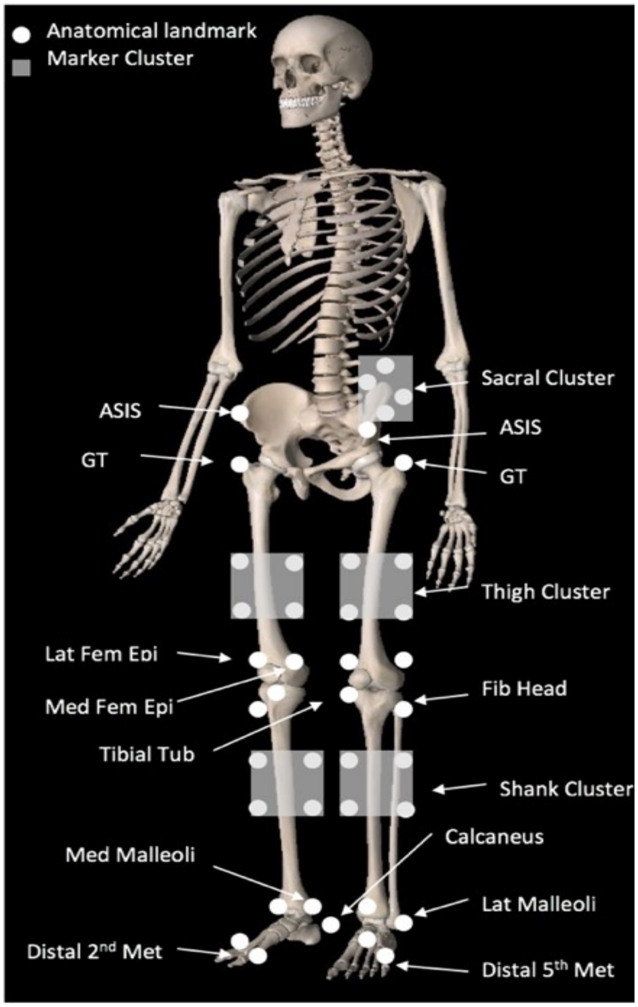

Figure I-1. Hybrid marker set.

This marker set is a hybrid marker set, with a basis from the Helen Hayes marker set and cluster plates from the Dalhousie University's Hatfield et al., 2011[236] marker set. The marker set is similar and comparable to the marker set used in the Glenrose Rehabilitation Hospital in the Syncrude Centre for Motion and Balance, with the only difference being in the tracking of the thigh segment.

Appendix J: Intra-rater test-retest reliability pilot evaluation

To determine if 3D capturing of data, time series gait representations, and gait parameter extraction of kinematics, kinetics and spatio-temporals, in adults living with class II obesity was feasible, we completed an intra-rater test-retest reliability case study. We assessed marker placement, reliability of repeated sessions of marker placement, and visibility of markers with a male volunteer living with class II obesity. The researcher and kinesiologist placed markers on the volunteer, had the volunteer complete the gait session, and then removed the markers. After one hour, the process was repeated. The data were collected and analyzed using the methods found in Chapter 3. The trends of the time series data were graphically compared. Then, kinematic, kinetic, and spatio-temporal parameters were compared using a Wilcoxon signed-rank test. Quantification of the movement between anatomical markers, plate markers, and virtual markers was completed.

The results were as follows: The researcher and a kinesiologist were able to palpate and place markers on the anatomical landmarks of the volunteer in two different gait sessions. There was no difference between kinematic, kinetic and spatio-temporal parameters between gait sessions (z-value > 0.05). Anatomical markers and marker plates remained visible to the cameras except for the anterior superior iliac spine (ASIS) markers, which would periodically disappear in some frames. When virtual markers of the ASIS were projected to create a pelvis, an approximate 2-3mm change was found. Moreover, waveform data and trends of curves were similar to those found in other gait studies.

Appendix K: Model construction: defining linked segments

Anatomical markers were placed on anatomically bony landmarks near segment endpoints to define the segment coordinate system. Tracking markers were placed on convenient locations not obstructed by participant's movement and had minimal tissue movement. A hybrid marker set was created, which contained markers from the Helen Hayes marker set as well as cluster plates used in the Dalhousie University marker set. The joints were defined using 3 degrees of freedom but tracked using 6 degrees of freedom. This allowed for accurate tracking of the pelvis. As the soft tissue artifact would occasionally occlude anterior superior iliac spine (ASIS) markers, this would cause the pelvis to disappear. However, by using the thigh cluster plate to track the thigh segment, the ASIS markers could be reconstructed allowing the pelvis to be tracked.

The proximal location of the femur and hip joint center of the pelvis was determined from the Helen Hayes formula. Marker placement on the greater trochanter was determined functionally by getting the participant to internally and externally rotate their leg.[275–277] Lower body target identifications were selected for each segment. The knee joint center was defined as along the line of the medial/lateral knee targets placed halfway between two targets. Segment mass was determined using Dempster's data.[278] Segment geometric shape was a cone, with location of mass centers and moments of inertia calculated relative to the participants mass and height.

For the knee, both the thigh and shank segments were used to calculate knee joint angles; the thigh segment was the reference segment (proximal segment). To calculate knee joint moments, the segment coordinate system of the thigh was used (proximal segment). Each segment was defined by at least 3 markers (non-collinear) to allow for the calculation of 6 degrees of freedom. Segmentation of the lower body was determined as follows by Table K-1 and landmark definitions by Table K-2.

Table K-1. Segment definitions

Segment	Proximal Joint	Distal joint	Tracking
Thigh	Joint: Right hip	Lateral: lateral femoral epicondyle Medial: medial femoral epicondyle	Thigh cluster plate markers 1-4 (6 DOF)
Shank	Lateral: lateral femoral epicondyle Medial: medial femoral epicondyle	Lateral: lateral malleolus Medial: medial malleolus	Calibration targets for tracking
Foot	Lateral: lateral malleolus Medial: medial malleolus	Medial: fifth metatarsal Medial: 2^{nd} metatarsal	Calibration targets for tracking
Pelvis	Lateral: right iliac Medial: left iliac	Lateral: right hip Medial: left hip	Calibration targets for tracking

Note: starting point = reference, end point = landmark is on a line, lateral object = landmark is on a plane. DOF = degree of freedom.

Table K-2. Landmark definitions

Landmark	
Knee Joint	Starting point: lateral femoral epicondyle Ending point: medial femoral epicondyle Offset by percent (1.0 = 100%): axial 0.5
Iliac	Starting point =left (right) ASIS End point = sacrum Lateral object = right (left) ASIS
Hip	Starting point = right (left) ASIS End point = left (right) ASIS Lateral object = sacrum
Sacrum	Starting point= pelvic plate marker 3 End point= pelvic plate marker 4 Lateral object: pelvic plate marker 1