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Hip movement pathomechanics of patients with hip osteoarthritis aim at reducing hip joint loading on the osteoarthritic side

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A B S T R A C T

This study aims at defining gait pathomechanics in patients with hip osteoarthritis (OA) and their effect on hip joint loading by combining analyses of hip kinematics, kinetics and contact forces during gait. Twenty patients with hip OA and 17 healthy volunteers matched for age and BMI performed three-dimensional gait analysis. Hip OA level was evaluated based on plane radiographs using the Tönnis classification. Hip joint kinematics, kinetics as well as hip contact forces were calculated. Waveforms were time normalized and compared between groups using statistical parametric mapping analysis. Patients walked with reduced hip adduction angle and reduced hip abduction and external rotation moments. The work generated by the hip abductors during the stance phase of gait was largely decreased. These changes resulted in a decrease and a more vertical and anterior orientation of the hip contact forces compared to healthy controls. This study documents alterations in hip kinematics and kinetics resulting in decreased hip loading in patients with hip OA. The results suggested that patients altered their gait to increase medio-lateral stability, thereby decreasing demand on the hip abductors. These findings support discharge of abductor muscles that may bear clinical relevance of tailored rehabilitation targeting hip abductor muscles strengthening and gait retraining.

1. Introduction

Osteoarthritis (OA) is a major cause of motor disability. While the etiological factors of idiopathic OA of the lower limb joints remain unclear, a consensus has been reached to define OA as a whole joint disease rather than defining it as being only cartilage-centered [1–3]. The risk factors of OA development and progression can be grouped into non-mechanical (age, obesity, inflammation, genetics) and mechanical factors (muscle weakness, abnormal joint anatomy). Mechanical factors directly relate to abnormal joint load and are considered to be the primary cause of disease progression [4,5]. In contrast to literature related to the pathomechanical factors of knee OA [6–9], literature on hip OA remains scarce and further investigation is thus needed.

Abnormal functional joint mechanics can be documented using three-dimensional motion analysis (3D-MA). So far, 3D-MA studies in patients with hip OA have reported gait dysfunction in terms of kinematics and kinetics, such as decreased hip joint excursion and decreased sagittal and frontal joint moments, as well as decreased joint power [10–13]. The reduced hip power in patients with hip OA is potentially indicative of a strategy for reducing the hip joint load as a result of commonly observed hip muscles weakness [14,15]. As the hip power is a scalar entity, it does not provide information on the functional role of the different muscular structures controlling the hip joint and acting in different plane. The decomposition of the total joint power proposed by Eng and Winter [16] could potentially provide additional insight into the effect of the adopted gait strategy.

Likewise, the mechanical work (i.e. the integral of the decomposed

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power) can be informative on altered contributions of specific muscle groups. As more than 20% of the total work at the hip relates to the frontal plane [16], the calculated work might be of particular interest in patients with hip OA [15] who have hip abductors weakness. So far, only one study compared power and work in patients following total and surface hip replacements [17]. Patients with total hip replacement demonstrated more pronounced hip flexor power absorption. However, the pathomechanics in pre-surgical OA condition remain unexplored. Consequently, there is a need to define the added value of documenting the movement pathomechanics adopted by patients with hip OA using advanced kinematic and kinetic analysis, including projected hip power.

Furthermore, the kinetic parameters in terms of joint moments and powers only indirectly relate to hip joint loading, as they do not account for the effect of muscle forces [18]. Musculoskeletal (MS) models in combination with inverse dynamic simulations of gait allow accounting for these complex relations and thereby provide a non-invasive in vivo analysis of joint loading [19–21]. For patients with hip OA, Lenaerts et al. [18] found increased hip contact forces prior total hip replacement surgery combined with decreased hip adduction and decreased external rotation angles, as well as decreased pelvic obliquity. Moreover, the changes in contact forces could be related to specific changes in joint moments. Specifically, decreased external hip adduction moments were found to decrease the hip contact forces [21,22]. However, previous studies did not relate changes in hip power or work to changes in hip contact forces. Additionally, these previous studies did not compare control versus patients with hip OA, therefore limiting the understanding of how hip pathological condition affects hip loading condition compared to a normal situation.

In summary, there is a clear lack of studies that comprehensively evaluated hip movement pathomechanics (in terms of hip powers, moments and work) during gait in patients with hip OA and related it to hip joint loading. The aim of the current study was to investigate the hip movement pathomechanics related to hip OA comprising hip moment, power and work, as well as hip joint loads. Based on available literature, we hypothesized that patients with hip OA would demonstrate reduced frontal hip moment and power as a consequence of hip abductor muscle weakness that would result in decreased hip contact forces.

2. Methods

2.1. Participants

Twenty patients with hip OA, prior to unilateral total hip replacement surgery, and a group of 17 healthy volunteers matched for age and BMI were included in this cohort study. The local Ethics Committee approved the study protocol and all participants provided written informed consent before engaging in the study. Study patients were recruited from the hip orthopaedic unit and were awaiting for hip replacement. Inclusion criteria for the patients were: aged between 40 and 65 years, a BMI $< 35\ kg/m^2$, unilateral hip OA combined with complaints of hip pain (Tönnis classification grade ≥ 1), no other orthopaedic co-morbidities such as lower limb osteoarthritis or reconstructive joint replacement as well as neuromuscular disease, neurological complications and low-back pain that could affect gait. OA level was evaluated by a single experienced orthopaedic surgeon using pelvic antero-posterior (AP) radiographs for both sides. A maximum of grade 1 on contralateral side, with absence of hip pain, was allowed. Healthy volunteers were recruited based on verbal screening and were included if they did not report either any musculoskeletal, neuromuscular disease or previous joint replacement. They as well had to be pain-free at the lower limbs.

2.2. Motion assessment procedure

A single experienced assessor collected all data to minimize the error associated with marker placement. All participants performed a minimum of three barefoot level walking trials on a 10 m walkway with two force platforms embedded in the central part of the walkway to ensure that patients reached steady-state walking velocity before striking the force platform. All trials were performed at a self-selected pace with the only instruction not to rush and to walk such as in daily life.

2.3. Instrumentation

Passive reflective markers with a diameter of 14 millimetres were affixed bilaterally to each participant according to the total body Plug-in-Gait model (Vicon. Oxford. UK), with the exclusion of arms and head. Three-dimensional motion of the markers was measured by 15 cameras of an optoelectronic system (Vicon Motion Systems, UK) at a frequency of 100 Hz. Synchronized ground reaction forces and moments were registered at 1500 Hz via the two embedded force platforms (AMTI, Watertown, MA, USA) and were used for kinetic data calculation and gait cycle definition.

2.4. Kinematic and kinetic analyses

Anthropometric measures such as height, weight, bilateral leg length, and knee and ankle width were collected for computation of the kinematic and kinetic data. Leg length was measured from antero-superior iliac spine to internal malleolus and ankle and knee widths were measured as the distance between the malleoli and the femoral condyles respectively, using a Vernier calliper. Kinematic and kinetic variables were obtained for each trial using a rigid segment linked model and inverse dynamics equations [23] (Plug-in-Gait software package, Vicon Motion Systems, UK). Three Knee Alignment Device (KAD) [24] calibration trials were collected to define the knee flexion/extension axis, thereby minimizing cross-talk between knee sagittal and frontal plane kinematics. The KAD trial resulting in the least excursion of the frontal knee motion during swing phase was retained for calibrating the remaining dynamic trials. Prior to kinematic and kinetic computations, the marker position data were filtered using Woltring's smoothing spline with a mean squared error of $15\ mm^2$ [25]. Additionally, a 6 Hz second order Butterworth low-pass filter was applied to the ground reaction forces and moments of the force platforms. Multiplanar joint angles were calculated using Cardan method, as well as internal net moments and powers of the hip joint were calculated. Furthermore, powers of the hip joint were calculated for the three different anatomical planes as the dot product of the net joint moment and the decomposed angular velocity for the specific anatomical plane. Subsequently, absolute mechanical work for each plane was calculated as the time integration of the respective power with respect to time [16]. Peak power bursts and mechanical work phases were defined following the power profile introduced by Eng and Winter [16]. However, the frontal power generation phases originally comprising two phases were merged into a unique work phase (wH2F), as both relate to power generation by the hip abductors (Fig. 1). All kinetic data were normalized to body mass. Trials were discarded in the presence of inaccuracy of force plate data or missing markers. Walking velocity was computed for each trial based on stride length and time as identified based on the foot marker trajectories.

2.5. Musculoskeletal modelling

A musculoskeletal model with 14 segments, 19 ° of freedom and 88 musculotendon actuators and including two wrapping surfaces around each hip joint, to account for the effect of the hip joint capsule, [26] was used. All simulations were generated using the standard workflow

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