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DISSERTATION

The Impact of Hip Abductor Muscle Status on In Vivo Joint Loads through Kinematics and Muscle Activity 51 Months Following Total Hip Arthroplasty

zur Erlangung des akademischen Grades
Doctor medicinae (Dr. med.)

vorgelegt der Medizinischen Fakultät
Charité – Universitätsmedizin Berlin

von

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Datum der Promotion: 25.11.2022

Preface

Partial results and findings based on this dissertation were accepted for presentation at the Virtual EFORT Congress - VEC^{hybrid} 2021 (June 30 to July 2, 2021):

- Tobias Winkler, Hilmi Cem Dinc, Adam Trepczynski, Carsten Perka, Georg N. Duda, Philipp Damm. Intraoperatively Occurring Muscle Damage Determines Postoperative Muscle Performance Resulting In Higher In Vivo Joint Loading After Total Hip Arthroplasty.

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List of abbreviations

%BW	Percent of the body weight
Abd	Abduction
Add	Adduction
ADL	Activity of daily living
ALA	Anterolateral approach
BMI	Body mass index
CCD	Caput-collum-diaphyseal
CHS	Contralateral heel strike
cm	Centimeter
cm^3	Cubic centimeter
CT	Computed tomography
CTO	Contralateral toe off
CTW™	Cementless Tapered Wedge
DAA	Direct anterior approach
DLA	Direct lateral approach
EMG	Electromyography
EQ-5D-3L	EuroQol-5D-3L
Ext	Extension
Flex	Flexion
FR	Fat ratio
F_{res}	Resultant force
GLmax	Gluteus maximus muscle
GLmed	Gluteus medius muscle
GLmin	Gluteus minimus muscle
HHS	Harris Hip Score
HRA	Hip resurfacing arthroplasty
HS	Heel strike
IPMA	Instant of peak muscle activity
M-mode	Motion mode
Max	Maximum
MIS	Minimally invasive surgery
mm	Millimeter

MSM	Musculoskeletal model
MVIC	Maximum voluntary isometric contraction
N/A	Not available
N	Newton
OA	Osteoarthritis
OECD	Organisation for Economic Co-operation and Development
p	p-value
PA	Posterior approach
PROM	Patient reported outcome measure
ROM	Range of motion
r_s	Spearman's rank correlation coefficient
SD	Standard deviation
sEMG	Surface electromyography
SENIAM	Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles
TFL	Tensor fasciae latae muscle
THA	Total hip arthroplasty
TMV	Total muscle volume
TO	Toe off
VAS	Visual analog scale
WOMAC	Western Ontario and McMaster Universities Osteoarthritis Index

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Abstract

Background: Well-established clinical scores show that total hip arthroplasty (THA) in primary hip osteoarthritis alleviates pain and markedly improves the performance of activities of daily living (ADLs). However, objective measurements show that THA patients' movement and electrophysiological patterns do not match those of healthy age-matched individuals. Surgical incision as well as intraoperative soft tissue traction and compression cause iatrogenic damage of the hip muscles, which is associated with their atrophy and fatty degeneration. An unfavorable muscle status may negatively affect joint loads. An improper in vivo hip joint resultant contact force (F_{res}) may shorten an implant's lifespan and also determine functional outcome following THA. This retrospective analysis aimed to identify whether kinematics and electrophysiological activity mediate the impact of structural muscle impairment on kinetics.

Materials and methods: In order to determine the F_{res}, instrumented femoral prostheses were implanted via a direct lateral approach. Nine patients (two females, seven males) participated in synchronous recordings of load patterns and surface electromyography along with three-dimensional mapping of motion sequences at a mean of 51 months (period: 35-64 months) postoperatively. The hip movement patterns of five ADLs (level walking, ascending stairs, descending stairs, standing up, sitting down) and the electrophysiological activity of the hip abductors gluteus maximus muscle, gluteus medius muscle, and tensor fasciae latae muscle (TFL) were assessed and correlated with both the hip abductor muscle status (total muscle volume [TMV], fat ratio [FR]) evaluated by postoperative computed tomography images and the in vivo F_{res}.

Findings: Across all ADLs, the results yield high inter-individual variability. Compared to asymptomatic control groups in the literature, this study's patients produced reduced extension and lower sagittal range of motion (ROM) in level walking, while stair negotiation resulted in higher flexion and greater ROM in the sagittal plane. Particularly TFL activity patterns are shaped by irregularities and hyperactivity. TMV and FR have an effect on both motion patterns in the sagittal and frontal planes and shape and timing of muscle activity. Furthermore, compensatory movement strategies and abnormal muscle activity may lead to not only higher but also lower hip joint loads.

Interpretation: The data do not provide conclusive evidence of muscle damage affecting joint loads via atypical movement and electrophysiological patterns. Overall, however, the results support the hypothesis that structural impairment of hip abductors may lead to the

development of pathomechanical movement patterns and irregular muscle activity, which in turn may adversely affect hip joint loads.

Abstract (German)

Fragestellung: Gängige klinische Scores zeigen, dass die Implantation einer Hüfttotalendoprothese bei primärer Coxarthrose die Schmerzen der Patienten bedeutend lindern und die Ausführung von Aktivitäten des täglichen Lebens merklich verbessern kann. Ergebnisse objektivierbarer Messmethoden zeigen jedoch, dass weder die Bewegungsmuster noch die Muskelaktivität dieser Patienten denen gesunder Gleichaltriger entspricht. Die Implantation einer Hüfttotalendoprothese führt entweder über ein Schnitt- oder ein Quetschtrauma zu einer iatrogenen Schädigung der Hüftmuskulatur, was mit deren Atrophie und Verfettung einhergeht. Ein abträglicher Muskelstatus kann sich ungünstig auf die Hüftgelenksbelastung auswirken. Die resultierende Hüftkontaktkraft ist ein bedeutender Faktor für die Haltbarkeit einer Hüfttotalendoprothese, die das funktionelle Ergebnis eines endoprothetischen Ersatzes mitbestimmt. Das Ziel dieser retrospektiven Analyse war es, das Verständnis für die auf die in vivo resultierende Hüftgelenksbelastung wirkenden Zusammenhänge zwischen periartikulärer Muskelschädigung, pathologischen Bewegungsabläufen und irregulärer Muskelaktivität zu erweitern.

Material und Methodik: Zwecks in-vivo-Bestimmung der Hüftkontaktkräfte erfolgte per transglutealem Zugang die Implantation von instrumentierten Hüfttotalendoprothesen. Neun Patientinnen und Patienten (zwei weiblich, sieben männlich) nahmen zum durchschnittlichen Zeitpunkt von 51 Monaten (Zeitraum: 35-64 Monate) postoperativ an synchronen Belastungsmessungen, dreidimensionalen Bewegungserfassungen und Oberflächen-Elektromyographie-Messungen teil. Die Bewegungsmuster der Hüfte von fünf Aktivitäten des alltäglichen Lebens (ebenes Gehen, treppauf Gehen, treppab Gehen, Aufstehen, Hinsetzen) sowie die Muskelaktivität der Hüftabduktoren (M. gluteus maximus, M. gluteus medius, M. tensor fasciae latae) wurden erfasst und jeweils mit dem anhand von postoperativen computertomographischen Aufnahmen evaluierten Muskelstatus (Gesamtvolumen, prozentuale Verfettung) und der Hüftkontaktkraft korreliert.

Ergebnisse: Über alle Aktivitäten hinweg ergab sich aus den Messergebnissen eine hohe interindividuelle Streuung. Im Vergleich zu symptomlosen Kontrollgruppen aus der Literatur zeigte sich beim Gehen eine reduzierte Extension und ein geringerer Bewegungsumfang in der Sagittalebene. Beim Treppengang hingegen erfolgten eine höhere Flexion und ein größerer Bewegungsumfang in der Sagittalebene. Insbesondere

die Aktivitätsmuster des M. tensor fasciae latae waren von Unregelmäßigkeiten und Überaktivität geprägt. Die Daten zeigen auf, dass Muskelvolumen und -verfettung sowohl die Hüftbewegung in der Sagittal- und Frontalebene als auch die elektrophysiologische Form und den Zeitablauf von Muskelaktivität beeinflussen. Die Ergebnisse weisen ferner darauf hin, dass beeinträchtigte Bewegungsabläufe und gestörte Muskelaktivität nicht nur eine Erhöhung, sondern auch eine Verminderung der Hüftkontaktkraft bewirken können. **Schlussfolgerung:** Die Daten liefern keine stichhaltigen Beweise für einen durchgehenden Effekt einer Muskelschädigung über atypische Bewegungsabläufe und elektrophysiologische Signale auf die Gelenkbelastungen. Jedoch bekräftigen die Ergebnisse insgesamt die Hypothese, dass eine strukturelle Beeinträchtigung der Hüftabduktoren zur Entstehung von pathomechanischen Bewegungsmustern und unregelmäßiger Muskelaktivität führen kann, was sich wiederum ungünstig auf die Hüftkontaktkräfte auswirken kann.

1. Introduction

1.1 Total hip arthroplasty

1.1.1 Medical indications, epidemiology, socio-economic implications

Total hip arthroplasty (THA) is considered a highly successful surgical procedure [1,2], in which an artificial acetabulum and a femoral prosthesis replace a hip joint in its entirety. THA is considered a therapeutic measure of last resort [3], mainly for patients suffering from end-stage osteoarthritis (OA) [4-7] in whom conservative treatments like oral analgesia, physical therapy, exercise, and other measures have failed to produce a desired outcome [6,7]. Although to a lesser extent, a variety of musculoskeletal conditions – such as hip fractures [4], autoimmune disorders like rheumatoid arthritis [8], chronic inflammatory conditions such as ankylosing spondylitis [9,10] and septic arthritis [11], some benign and malignant conditions that lead to bone tumors [12], developmental dysplasia of the hip [13,14], and osteonecrosis [4] – are additional indications for THA.

Being the primary indication for THA in the majority of the cases [15], OA is one of the most common musculoskeletal disorders in the world [16]. It is a leading source of disability, placing an immense socio-economic burden on public health [17-20]. Incidence of hip OA is higher in women than in men at all ages, increasing more rapidly especially between the ages from 50 to 75 and peaking at around 75 years of age [21]. The late complications of hip OA include joint failure due to the disintegration of the articular surfaces of the acetabulum and the femoral head, which also interferes with neighboring structures [22] and leads to hip pain and functional constraints [15,17] accompanied by a restriction of daily activities [21]. Engaging in a relieving posture or compensatory gait mechanics as a result of hip pain may lead to advanced complications of musculoskeletal integrity [23,24] and further decrease patients' ability to undertake activities of daily living (ADLs) [24]. Furthermore, walking disability by cause of OA-related pain has been linked to comorbidities like mental distress [25-27], cardiovascular disease [28], incident diabetes [29], and a greater risk of all-cause mortality [28,30]. Although the pathological mechanism underlying OA is still an object of investigation, a biopsychosocial model has been proposed to explain the origin of pain [21], and a multifactorial genesis involving an interplay of biomechanical alterations and biochemical processes is suspected [4,21,23,27,31]. Globally, THA procedures are in excess of one million per year [32], and the quota is expected to

continue rising substantially [33,34]. Between 2005 and 2014, significant increases in hip replacement utilization rates were recorded in a greater part of the member countries of the Organisation for Economic Co-operation and Development (OECD), with the United States, Switzerland, and Germany leading the field with respect to frequency at more than 200 per 100,000 population [33]. The United States Agency for Healthcare Research and Quality reported a 2.9% average annual increase of partial or total hip arthroplasty between 2003 and 2012, also registering hip replacement as the fourth most common operation for both women and men in 2012 [35]. Based on a revised projection of data from the years 2000 to 2014, the volume of primary THA in the United States is going to grow to 635,000 in 2030 [36], confirming the worldwide trend towards rising THA procedures in countries with a high income [21]. According to the German Federal Statistical Office, the implantation of an artificial hip joint was the sixth most frequently performed surgery in Germany in 2018 for women and men [37]. The aging population in most OECD member countries, amongst them especially people reaching the age of 80 years and beyond, is believed to be a leading cause for the surge in hip replacements [38], also because of the higher incidence of OA in older patients [7].

Socio-economic implications of THA indications are projected to become an even larger burden than they already are at present because of the continuing growth of OA as one of the most prevalent diseases [18] in populations of high-income countries [21]. Along with the expanding demand for hip replacement in younger patients [4] as well as the progressive interest in a more active lifestyle and higher quality of life [39], an increasing demand for revision surgeries due to implant failure [33,40] is expected. In their systematic review and meta-analysis, Evans et al. estimate the timeframe for the need of revision surgery to be 15 to 20 years in about three-quarters and 25 years in more than half of the patients [40]. All-component revision (41.1%) is the predominant technique used in revision THA, while implant instability and dislocation (22.5%), mechanical loosening (19.7%), and infection (14.8%) are its prevalent causes [41]. Therefore, it is not only essential to provide for comprehensive preventive measures in order to delay a potential THA as far as individually feasible but also to extend the longevity of hip prostheses by optimizing implant design and to constantly improve pre- and intra-surgical procedures in conjunction with offering appropriate postoperative care.

1.1.2 Functional outcome, determinants

The successful outcome of THA is assumed to rest on multiple interdependent factors [42] and is frequently assessed by implant survival and patient-reported outcome scores [32]. While implant survival, patient reported outcome measures (PROMs), and clinical scores have regularly been used as indicators for the success of THA, uniting the learnings from kinetic and kinematic computations [43], as well as integrating the results of radiographic imaging [44-47] and electrophysiological recordings [48] have increasingly become objects of research in assessing the functional outcome of surgical treatments [43]. Comparing the postoperative results by the means of patient surveys might be complicated as they often rely on subjective impressions [49,50]. Methods like gait analysis [49,50] and measuring internal joint loading conditions contribute to a more objectifiable interpretation of functional short- and long-term outcomes. The number of reports evaluating physical function following THA is growing in the light of technological advances [43]. Various research institutions focusing on the investigation of human locomotion examine the synergy of biomechanical components making up specific activities and work on establishing musculoskeletal models (MSMs) with the acquired data to improve implant design and therapeutic efforts [51,52]. Measures employed in biomechanical research capture possible abnormalities in the patterns of joint loads, kinematics, and muscle activity which have either existed before THA as part of an underlying pathology, for example OA, or developed as a consequence of surgical procedures [53-56].

Patients undergoing THA are subject to trauma of the periarticular tissue, in addition to an incision or excision of the joint capsule [57], an irritation of synovia, and potential nerve damage [58-61]. Capsular management is a relevant aspect of THA [62] because the preservation of capsular structures may influence the functional outcome. An experimental study by van Arkel et al. showed that all capsular ligaments served as primary restraints at certain points of the full range of motion (ROM) of the hip joint, thus acting against excessive hip rotation and consequential impingement and instability [63]. Soft tissue injuries are associated with a heightened risk of dislocation [64-68], which is a rather rare condition in healthy individuals and almost only triggered by high impact trauma [69]. Despite evidence revealing that capsular repair in THA reduces the risk of dislocation [70-73], the preference for either capsulotomy or capsular repair appears to be dependent on the experience [68] and the training of the respective surgeon [74].

Weber et al. report subclinical damage in the sciatic, femoral, and obturator nerves with an incidence of 70% in the short postoperative term, which the authors attribute to THA-related trauma [60]. In general, surgical trauma may range from tension caused by leg lengthening, traction injury with leg manipulation, traction due to inappropriate retractor placement, direct trauma, strictures by trochanteric wires, extruded cement, and compression by postoperative hematoma to unexplained reasons [61,75]. Contrary to these findings, however, is evidence by Farrell et al., who report motor nerve palsy to be an uncommon complication after THA with a prevalence of only 0.17%, although the authors also mention that many patients may have had undiagnosed subtle nerve injuries [76]. Other findings note a prevalence of 1% to adequately reflect the common notion of surgery-related nerve palsy in OA-patients [77], with revision THA yielding a higher prevalence of 7.6% [78]. Abitbol et al. evaluated the incidence of gluteal nerve injury by using electromyography (EMG) and state that EMG-detected gluteal nerve injuries may assist in the interpretation of gait abnormalities [59], as the gluteus maximus muscle (GLmax) is innervated by the inferior gluteal nerve and the gluteus medius muscle (GLmed), gluteus minimus muscle (GLmin), and tensor fasciae latae muscle (TFL) are innervated by the superior gluteal nerve [79]. It appears that clinical assessment alone does not sufficiently describe the incidence of THA-related nerve damage, while EMG produces electrophysiological indications for nerve lesions [58-60,80].

Soft tissue deficiency and compromised muscle function as part of THA are not only increasing susceptibility to hip dislocations [81] but can also lead to gait abnormalities such as Trendelenburg gait [82,83] and other gait pathologies [55,84]. As a consequence of surgical intervention, the muscles and tendons encompassing the hip joint suffer trauma brought forth by the chosen approach [57]. By and large, there is no consensus on which surgical approach should be utilized as standard, and the debate over which is the best approach continues on the grounds that no surgical approach has proven to be universally superior over other approaches [53,57,85-87]. Regardless of this fact, the posterior approach (PA), the direct anterior approach (DAA), the anterolateral approach (ALA), and the direct lateral approach (DLA) are the approaches used most often in THA [53,85-88]. A systematic review and meta-analysis conducted by Miller et al. reported less pain, better documented function, lower rates of revision, instability, and infection, but higher rates of nerve damage in patients undergoing primary THA employing the DAA as opposed to the PA [89,90]. While a retrospective review by Angerame et al. found the DAA and PA not to differ

significantly in early revision rates (defined as less than five years), the DAA was shown to correlate with a significantly higher early revision rate in view of femoral component loosening [91]. The PA, on the other hand, had a higher incidence of hip joint dislocation [91], with reported rates varying from 0% to 6.4% – depending on whether soft tissue repair was applied or not [66]. Hip instability is a complication more common in the PA and likely a result of approach-related posterior soft tissue damage as well as of less preferable positioning of prosthesis components [91,92]. Yet, as assessed by all PROMs recorded in the Dutch Arthroplasty Register prior to and three months after THA, both the DAA and PA are superior to the ALA and the DLA [93]. A cohort study comparing PROMs between the DLA and PA found the PA to be preferable over the DLA because of a significant functional benefit in the early postoperative phase and the PA not generating an increased revision risk after adjusting for patient and surgical factors [94]. The DLA is affiliated with a higher occurrence of gait abnormalities [95], which are viewed as an aftereffect of abductor weakness caused by the splitting of the GLmed and GLmin [53] or intraoperative injuries to the inferior gluteal nerve [48]. Abductor weakness accounted for by GLmed myotomy is also regarded as a potential complication of the ALA [85]. It is associated with postoperative limp and lower patient satisfaction, yet also with the advantages of a reduced dislocation rate and favorable acetabular display during surgery in comparison with the PA [85]. Developments in healthcare economics [96], its high popularity among patients [97], and the continuously rising volume for both primary and revision THA [34] have led to a trend towards minimally invasive surgery (MIS) [1]. Citing lower surgical duration and blood loss but also slightly lower clinical scores and a higher risk of iatrogenic nerve palsy, a meta-analysis by Migliorini et al. found no conclusive advantages of MIS over the more traditional approaches as of yet [98]. Not least because soft tissue injuries and muscle damage are relevant measures of outcome [32,34], further studies will have to investigate whether limited visibility of the operating site in MIS puts in jeopardy the evidenced durability of THA [1,99] and if functional recovery following MIS eventually reaches satisfactory levels [100].

Each approach in THA entails the retraction of periarticular tissue and the splitting or release of certain muscles and tendons [53,85,101-105]. This procedure is associated with fatty degeneration and thus compromises periarticular hip muscle status [47,104,106-108]. Further potential complications are hip instability, aseptic loosening, nerve palsy, intraoperative, short- and long-term periprosthetic fractures [53,109-111], and periprosthetic joint infection

[112,113]. Depending on the endpoints implemented in order to assess the functional outcome of THA comparing the various surgical approaches with each other, it is critical to select the outcome measures accordingly. The combination of certain outcome measures practically always allows for endpoints that favor one approach over the other. Ideally, both surgeon and patient are aware of the risks associated with THA and different surgical approaches, while the surgeon is experienced with various approaches, so that the most appropriate one suiting the patient's individual anatomy can be chosen [89,91].

Unsatisfactory functional outcomes of THA can be attributed to a multifactorial etiopathology brought about by parameters only partly elaborated in this investigation. Presently, orthopedic treatments involving surgical procedures on joints ultimately result in a certain degree of muscular fatty degeneration ensuing from iatrogenic muscle damage, which is virtually inevitable [45,47,83,101,114]. Iatrogenic muscle impairment and the accumulation of fat in muscles was shown to significantly correlate with higher in vivo hip joint contact forces in ADLs [115-117], while gait parameters [118] and muscle activity were also affected following THA [119]. The following chapter surveys the current state of biomechanical research with respect to the role of the structural integrity of the hip abductor musculature in ADLs.

1.2 Biomechanics of the hip joint

1.2.1 Kinetics

Calculations of hip joint loads have been undertaken as early as more than a century ago when Koch first formulated his laws of bone architecture in 1917, which mathematically confirmed the doctrine of the functional form of bone by Wolff [120]. Supported by a mathematical basis, Koch argued that Wolff's findings would lead to a broader acknowledgement of their relevance for the prevention and therapy of osseous defects [120] – and they did. In 1966, Rydell measured in vivo hip joint contact forces using an instrumented prosthesis for the first time ever and showed the actual forces acting upon the hip joint to be higher than previous calculations had assumed [121,122]. Although Rydell's account was based on only two patients' recordings, at that time, the collected measurements provided innovative knowledge about in vivo joint loads during gait [121]. The waveform of the resultant force (F_{res}) which Rydell illustrated for one gait cycle resembles the waveforms obtained with instrumented implants today (please refer to

figure 1 below) [121]: “In the stance-phase two maximum points with a low point between occur similarly to the vertical force curve describing the action between the foot and the ground. (…) The first maximum point appears immediately after toe-off, and the second immediately before heel-strike.” With about 160 to 300 percent of the body weight (%BW), the F_{res} collected during gait was close to the F_{res} evidenced nowadays. Rydell suggested that inter-individual differences in the F_{res} were due to the distinctive execution of tasks and the patients’ individual anatomical properties [121,122].

In the same year, Paul advanced the understanding of internal joint loads by presenting a biomechanical model to determine the F_{res} and moment acting on the hip joint with the help of combining the assessments of measured ground reaction forces and photographs [123]. Paul implemented the aspect of muscle pull also discussed by Pauwels in 1935 [124] into this model by further utilizing synchronously recorded electrophysiological measurements, which led to estimations of peak joint forces during gait of 390%BW [123]. Nevertheless, Paul argued that the results should be viewed as curves of the upper and lower limits of joint loads because of the narrow evidence supporting his theory concerning the exact amount of force exerted on the femoral head by periarticular muscles [123]. In the following years, in vivo measurements of hip joint contact forces were reported by Häggström in 1974 [125], English in 1979 [126], and Goodman in 1980 [127]. Between 1985 and 1990, a research group of scientists at Case Western Reserve University published data on the F_{res} affecting the hip joint, resulting, among others, in publications by Davy et al. in 1988 [128] and by Kotzar et al. in 1991 [129]. Bergmann et al. were the first to gather detailed gait data coupled with measurements of in vivo joint forces and moments, producing the most comprehensive data set at that time [130,131]. In order to simulate realistic loading conditions at the hip joint sufficient enough for preclinical testing of hip prostheses, individual MSMs based on the obtained in vivo data and radiological images of each patient were created [132] and further enhanced [133]. A new generation of instrumented implants was presented by Damm et al. in 2010 [134], resulting not only in the acquisition of in vivo joint load and moment components [116,117,135-140] but also in a fundamental insight regarding in vivo friction moments [137,141-144]. Determined by the respective ADL carried out, internal loads at the hip joint were shown to individually exceed as much as 400%BW [116]. Low-impact activities like ergometer cycling produced peak joint loads of only between 69 to 121%BW [138], whereas high impacts caused by stumbling led to contact forces as high as 870%BW [145].

With regard to meeting the demands of a constantly growing younger patient population [33] and the subsequent requirement of prolonging implant lifetime to endure particularly younger patients' more active lifestyle [4,146,147], the construction and consistent further development of hip implants involve the consideration of various biomechanical parameters [134]. The adequate application of contact forces is an integral part, and the accurate measurement of the in vivo situation provides relevant indications for the avoidance of mechanical failure when testing new implants in preclinical trials under conditions simulating those found in vivo [134,137]. Additionally, knowledge about in vivo forces serves as a critical point of reference when developing MSMs and validating computerized simulations for the estimation of physiological loading conditions [133,148].

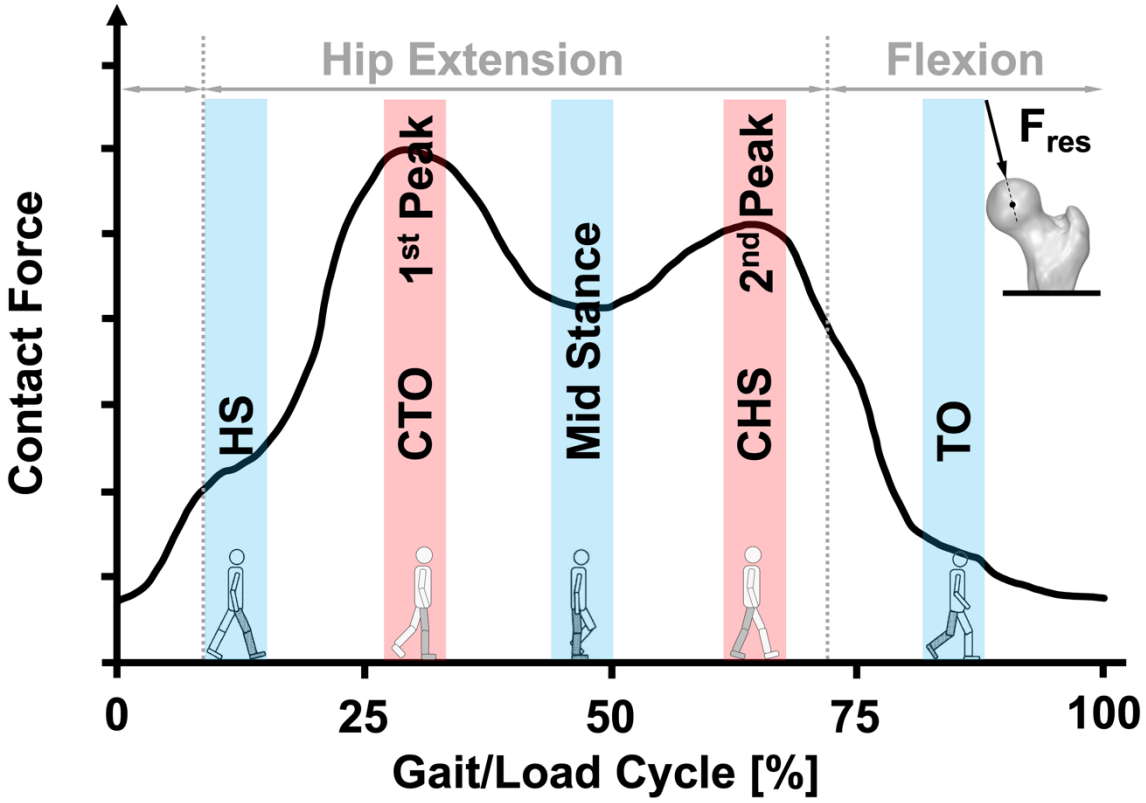

Figure 1: Exemplary illustration of gait phases of the right lower extremity and respective in vivo hip joint resultant contact force in level walking (Julius Wolff Institute for Biomechanics and Musculoskeletal Regeneration – archive). Highlighted leg = Operated leg. Colored belts = Gait phases. HS = Heel strike. CTO = Contralateral toe off. CHS = Contralateral heel strike. TO = Toe off. Black line = Resultant force. F_{res} = Resultant force.

In vivo joint loads at the hip accumulate as an aggregate of forces brought about and mediated by inertial parameters and gravitational pull, the integrity of soft tissue such as capsular ligaments and tendons [149], as well as individual and agonist-antagonist muscle activity (co-contraction) [150]. According to the results of a computerized simulation of level walking, however, muscle contributions to hip joint loads are responsible for 95% of the forces exerted [151]. Correa et al. found that the hip-spanning GLmax, GLmed, iliopsoas

muscle, and hamstrings played the major role in the makeup of the resultant contact force [151]. Shape and timing of the F_{res} as seen in figure 1 correspond to the respective muscles' contributions [151]:

- the GLmax is most responsible for joint loads in early stance, taking place from around heel strike (HS) to contralateral toe off (CTO), when both legs are touching the ground;
- the GLmed adds the largest part of contact forces throughout stance, lasting from around HS to contralateral heel strike (CHS);
- the iliopsoas muscle upholds the F_{res} throughout the swing phase, lasting from about toe off (TO) to HS; and
- the hamstrings contribute during the late swing and early stance phases, taking place from shortly before HS to CTO.

By implication of the aforementioned, the GLmax and GLmed are the leading muscles in the formation of the 1st peak F_{res} [151]. With reference to investigating the influence of hip abductor weakness on joint loads in level walking, past models have employed the results of ground reaction force plates and found the GLmed to be most influential in articular loading [152]. Earlier research utilizing an MSM has also identified the GLmed as a leading muscle in the contribution to ground reaction forces, naming it as the main supporting muscle during mid stance and classifying the GLmax and GLmed as having major shares in the 1st peak resultant ground reaction force [153].

Of the ADLs assessed in this investigation, gait is the best researched. For simplification purposes and because level walking and stair negotiation are similar in that they are locomotor ADLs with resembling double-peak load patterns, level walking serves as an example to elaborate the hip joint biomechanics of gait and gait-related ADLs. Thus, as this chapter introduces hip joint kinetics by the example of gait, the following subchapters on kinematics and muscle activity also give an outline of hip joint biomechanics as relevant mainly for locomotor ADLs. With sit-to-stand and stand-to-sit, however, this investigation also examines two non-locomotor ADLs with single peak load patterns, of which accounts in the literature are particularly scarce. For this reason, the figures below merely serve to provide an overview of essential sit-to-stand (please refer to figure 2) and stand-to-sit (please refer to figure 3) hip biomechanics. A more detailed analysis of these non-locomotor ADLs follows in the results and the discussion of this investigation.

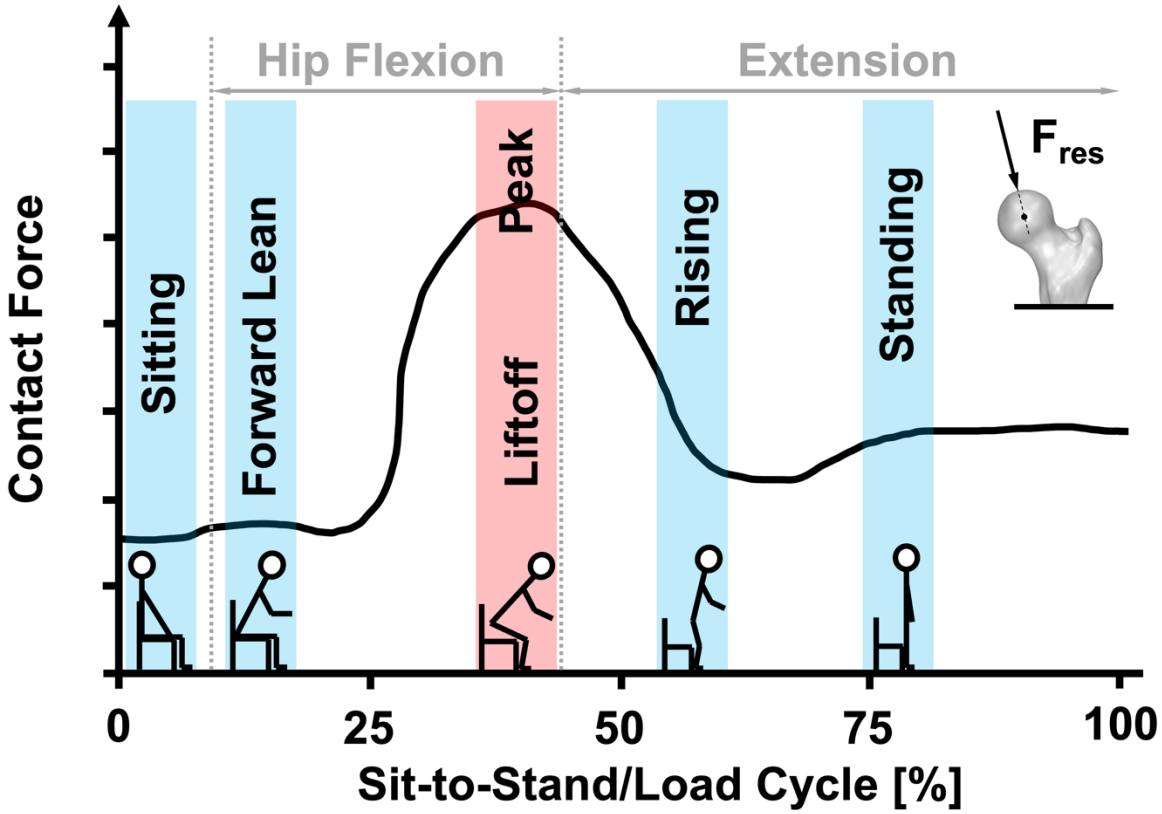

Figure 2: Exemplary illustration of motion phases and in vivo hip joint resultant contact force in standing up (Julius Wolff Institute for Biomechanics and Musculoskeletal Regeneration – archive). Colored belts = Motion phases. Black line = Resultant force. F_{res} = Resultant force.

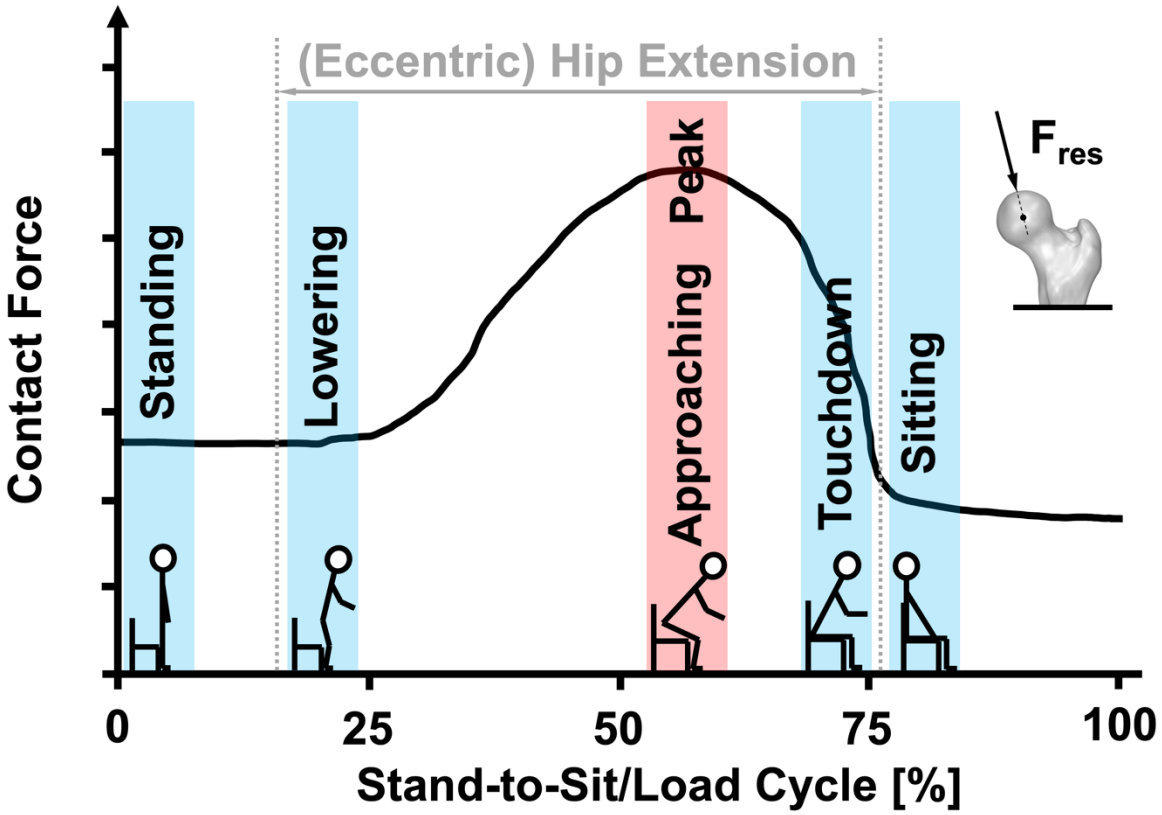

Figure 3: Exemplary illustration of motion phases and in vivo hip joint resultant contact force in sitting down (Julius Wolff Institute for Biomechanics and Musculoskeletal Regeneration – archive). Colored belts = Motion phases. Black line = Resultant force. F_{res} = Resultant force.

The question whether the findings above can be validated against in vivo data on hip joint loads was partially answered by Damm et al., who report increased contact forces in patients with gluteal muscle impairment [116,117]. To which extent individual kinematics and hip abductor muscle activity in a variety of ADLs are involved in the formation of in vivo joint loads, however, had not yet been determined.

1.2.2 Kinematics

Human locomotion is an intricate interaction of various active and passive components, which can be altered by conscious intents or unconscious neural patterns, so that individual kinematic results may be achieved. The mechanical nature of ADLs is that all movements are interwoven by means of a kinetic/kinematic chain [122,149,154-157], subjecting the hip joint to constant changes in loading, mediated by static ligamentous structures and dynamic muscle-tendon components [149]. The GLmax works as a stabilizer of the hip joint in the sagittal plane, whereas the GLmed, GLmin, and TFL secure the hip joint in the frontal plane [79], especially during walking when the ipsilateral leg is in the stance phase and the contralateral leg is in the swing phase [149,158]. Upright standing alone may serve as an example of the human body's intersegmental connectedness because it requires the smooth interplay of many more structures aside from the hip abductor muscles examined within the scope of this study. As for active components involved in weight-bearing standing, for instance, the triceps surae and tibialis anterior muscles of the shank work as antagonists in order to preserve the upper ankle joint's position in the sagittal plane and, similarly, the quadriceps femoris muscle of the thigh secures the knee joint [79]. Autochthonous back muscles, also called spinal erectors, stabilize the spinal column [79]. Passive components not only involved in maintaining a straight posture but also in the execution of other ADLs include various ligaments such as the iliofemoral ligament, which is the body's strongest ligament, preventing hyperextension of the hip joint [149] and also assisting the GLmed and GLmin in stabilizing the pelvis in the frontal plane during gait [79]. Together with the pubofemoral ligament, which prevents disproportionate hip abduction and extension, and the ischiofemoral ligament, which also restricts excessive extension and internal rotation, these three ligaments make up the main body of the external hip joint capsule [149]. Other passive components are the fibrous structures of the knee joint's dorsal articular capsule and the collateral ligaments of the knee and ankle joints, preserving their respective joints' stability in the frontal plane [79]. Active components may also passively influence the extent of a joint's movement. This is exemplified by the

hamstrings limiting maximum hip flexion, thereby reducing the hip joint's ROM when standing upright [79,159]. This is also called passive insufficiency [159]. Active insufficiency, on the other hand, occurs when a muscle spanning across two or more joints cannot contract further as to produce a certain muscle tension or maximum ROM in the joints involved [160]. Other potential factors altering joint ROM are osseous impingement and soft tissue restraint; here, bones, muscles, and connective tissues like body fat, skin, but also scar tissue may narrow joint movement through the physical space they take up [63,79,161]. In addition to the aspects mentioned above, a physiological gait pattern depends, among other things, on individual anatomy. Factors that have a bearing on the outcome here are, for example, leg length (anatomical and functional) [149], the anatomical and mechanical axes of the leg (e.g., genu varum, genu valgum), and the caput-collum-diaphyseal (CCD) angle. Other determinants are potential rotational malpositions of the femoral neck (coxa antetorta, coxa retrotorta) and possible hip dysplasia (transverse and sagittal angle of the acetabular plane) [79].

The upper thigh may be moved within the three planes of the hip joint's center, thus resulting in the hip joint's three degrees of freedom and six principal angular motions, as assessed with the neutral zero method by Debrunner [79,162,163]:

1. extension and flexion ranging from about 20° to 130°-140° and taking place in the sagittal plane;
2. abduction and adduction ranging from about 50° to 30° during hip extension and ranging from about 80° to 20° during hip flexion of 90°, taking place in the frontal plane; and
3. external and internal rotation ranging from about 40° to 50° during hip flexion of 90° and ranging from about 40° to 30° during hip extension, taking place in the transverse plane [79].

It should be noted that the abovementioned values pertaining to the achievable ROM are merely reference values collected in medical examination settings. They show high variability between individuals and are usually not realized in ADLs [149].

Although a reduction of hip joint ROM may be attributable to advanced age to some extent, any considerable loss of joint mobility until at least the age of 74 should be treated as abnormal [164]. This might be the case with individuals suffering from medical impairments, for instance OA [23] or THA-related soft tissue injury [165]. In their systematic

review on functional deficiencies following THA, Kolk et al. highlight lower hip sagittal and frontal ROM as well as lower peak hip flexion, extension, and adduction in comparison with healthy controls [165]. The authors further underscore lower hip sagittal ROM as well as lower peak hip flexion and extension compared to the contralateral limb [165]. Patients having undergone THA are reported to have favorable postoperative clinical outcomes and improved gait kinematics when compared to their presurgical levels [166]. Yet, there are numerous accounts on kinematic parameters not returning to healthy individuals' standard levels, leaving patients with residual gait impairments in the long run. Besides pointing out kinetic irregularities such as lower ipsilateral hip abductor moments, Beaulieu et al. found lower hip sagittal ROM and compensatory adaptations at both ankle joints in THA patients at an average of 10.6 months postoperatively [118]. A long-term follow-up study by Bennett et al. even found patients to exhibit pathomechanical gait patterns 10 years after THA, observing reduced hip and knee sagittal ROM, hip frontal plane ROM, and lower maximum hip extension, with decreased velocity and step length compared to asymptomatic individuals [167].

Abductor weakness may become manifest in altered gait kinematics, for example in Trendelenburg gait, in which a patient walking on the affected leg tilts his or her upper body towards the affected side (ipsilateral lateroflexion of the trunk) [168]. In an attempt to counterbalance gravitational force and in order to compensate for the missing moment generated by the hip abductors in healthy individuals, the opposite hip is kept from giving in to the downwards pull and declining outside of the horizontal line in the frontal plane [149]. As mentioned before, abductor weakness may originate from or persist despite THA [166]. In any case, the implantation of hip prostheses is accompanied by an altered hip joint geometry and subsequently different muscle moment arms [150], which in turn might facilitate the realignment of joint kinematics [149]. Symptomatic individuals' compensatory motions allocate the concomitantly arising forces that are properly proportioned in asymptomatic individuals in a now inefficient technique [168] throughout both the affected and unaffected limb's hip, knee and ankle joints, as well as spine and pelvis [23,118,155]. In fact, the body's plan of action of upholding the alignment of lower limb joints may turn out to be more to its detriment than to its benefit, and these unconsciously adopted kinematic patterns promote an emergence of OA in other joints, possibly resulting from irregular loading conditions [155].

The data on individual muscles' contributions to hip joint loads by Correa et al. [151] mentioned previously are in line with findings examining the functional capacity of individual muscles and muscle groups in realizing a fluent gait pattern. In accordance with this digitized model, the weakening of hamstrings and the iliopsoas muscle strongly affects normal walking, while the removal of other muscles (i.e., hip and knee extensor muscles) does not change overall kinematics [168]. Notably, the absence of GLmed activity renders physiological gait non-executable [168], which expressly underlines the crucial role of this hip abductor and pelvic stabilizer in the emergence of gait pathologies [149]. Other accounts suggest the TFL to be the main hip abductor throughout the stance phase [158]. Gottschalk et al. distinguish three anatomically and functionally different segments of the GLmed that are primarily active from HS until CTO, with the middle segment merely initiating abduction during early stance and the anterior and posterior segments stabilizing the hip by locking the femoral head into the acetabulum through pelvic rotation [158]. The GLmin is assumed to stabilize the hip in a similar manner from CTO to TO [158]. As a central muscle contributing to hip joint loads in early stance (between HS and CTO) [151], the GLmax performs as an extensor and external rotator of the hip joint during load acceptance and forward propulsion [169]. The upper fibers of the GLmax, however, were found to act much like the GLmed in hip abduction during both level walking [170] and stair negotiation, unlike the lower fibers, which function as the primary hip extensor in ascending stairs [171].

In spite of a lack of consensus in the literature on how exactly muscles join forces to harmonize kinematics in level walking and other ADLs [172], the GLmed, GLmin, and TFL are recognized as the principal hip abductors due to their anatomical configuration [151,152,158,168,173]. Their structural integrity constitutes a fundamental part in physiologic gait [149,169]. On that account, the influence of muscle impairment on ADLs had earlier been investigated by correlating hip abductor muscle status to peak in vivo joint loads [115-117]. As stated above, the mediating role of individual kinematics on hip joint contact forces is likely determined by muscular integrity to a non-negligible extent. To date, research on this question has been based on theoretical considerations, and the kinematic gap between muscle impairment and in vivo hip joint loads has remained open.

1.2.3 Periarticular muscle activity

The significance of structurally affected muscle tissue in the formation of altered contact forces and the emergence of respective compensatory kinematic patterns is documented by various studies making use of ground force plates for the assessment of kinetics and a motion capture system involving a certain set of markers and cameras for the evaluation of kinematics [174]. Radiological examination methods diagnosing predictors of muscle denervation and ensuing muscle dysfunction such as atrophy, hypertrophy, and fat replacement are also involved in the interpretation of the postoperative hip abductor weakness [45,175,176]. In contrast to passive structures such as bones, tendons, and capsular ligaments, active structures like muscles exhibit electrical activity, which can be quantified and graphically displayed [177].

Along with the ongoing sophistication of gait analysis, complementary electrophysiological measurements have emerged as an easy-to-use procedure to assess dynamic muscle action and timing [178]. Surface electromyography (sEMG) is a popular method because of its rather uncomplicated applicability in gait laboratory settings [179] as opposed to fine wire needle electrodes, which necessitate skin penetration and may cause discomfort [177]. Nonetheless, the utilization of sEMG has to be considered mindfully, as surface electrodes have certain limitations pertaining to their reliability [178,179]. Among the shortcomings associated with sEMG are amplitude cancellation, crosstalk, and spatial variability of muscle activity [180]. The aspect of amplitude cancellation, however, is negligible in the context of investigations targeting muscle activation times and activation pattern shapes – both of which are regularly the objective of gait analyses [180]. Moreover, crosstalk and spatial variability of muscle activity can be essentially reduced when attaching electrodes suitably [178,181]. Supplementary to simultaneously acquired kinetic and kinematic data, sEMG provides useful insights about the relative intensity level of individual muscles' activity in dynamic activities [182], in addition to their onset and cessation times [183]. Therefore, sEMG constitutes a feasible solution aiming at the identification of pathomechanical strategies adopted by patients with abductor muscle weakness due to OA- and THA-related abductor muscle impairment.

Dwyer et al. found that hip OA-related abductor muscle weakness leads to an increased activation of both the ipsilateral and contralateral GLmed in level walking and stepping tasks [184]. Increased GLmed and TFL activity during gait was also shown in patients

diagnosed with hip OA shortly before undergoing THA [185] and after receiving varus osteotomy of the femur [186]. Vogt et al. observed delayed GLmed activity onset times in patients shortly following THA, indicating abnormal hip abductor recruitment patterns when compared to healthy age-matched adults [183]. The authors point out approach-related surgical trauma, explicitly mentioning the ALA and the transgluteal Hardinge approach (the DLA), respectively, among the risk factors endangering the function and integrity of the GLmed [183]. The research of Chomiak et al. agrees with these findings, as the authors registered electrophysiological signs for nerve lesions affecting the GLmax, GLmed, and TFL, arising from approach-related nerve injury, even though muscle activity was not found to be associated with the clinical outcome [88]. A longitudinal study by Long et al. conducted kinetic, kinematic, and dynamic electrophysiological measurements of patients prior to THA and in time intervals up until two years after surgery, providing interesting results: most preoperative EMGs revealed abnormal patterns, which had all normalized by one and two years after THA [56]. Force plate measurements, however, displayed increased postoperative joint loads, pointing to prolonged ipsilateral hip muscle weakness despite nondescript EMG recordings and the absence of clinical evidence [56]. Abductor muscle weakness is compensated by higher activity of other muscles [168], which involves the inefficient substitution and loading of muscles that are less active under normal circumstances [152]. This, in turn, produces lopsided joint moments [168], possibly explaining the contradictory results mentioned above.

Sims et al. propose that increased GLmed activity may stem from the central nervous system's inability to appropriately rate the muscular force demanded for the execution of an ADL [187]. Hurley explains that pathological afferent signals from the ipsilateral leg to the central nervous system may wrongfully be recognized as originating bilaterally, which sets off a neuromuscular malfunction in both legs that can eventually lead to bilateral arthritic changes [188]. Another element to consider is muscle fatigue, which is described as a consequence of weak muscles operating at their upper limits and subsequently failing to uphold neuromuscular protective mechanisms, thus allowing for atypical kinematic patterns and making the joint vulnerable to disruptive movements [188]. Although it is unclear whether the shift to irregular abductor muscle activity patterns is causative of hip OA or if neuromuscular deficits arise secondarily, altered muscle activity may aggravate disease progression as a result of higher joint loads [183,187]. Excessive loads caused by abductor weakness may lead to a vicious cycle of advancing exhaustion and

deterioration due to compensating the either inadequately low, disproportionately high, or generally irregular activity of impaired abductor muscles [168]. This notion is confirmed by Meyer et al., who advise against taking up postoperative gait training before having adequately strengthened the affected muscles with a view to reducing hip joint contact forces [189].

The relationship of electrophysiological activity to the force generated by a muscle depends on physiological processes, individual macro- and microanatomy, and technical considerations [190]. In consequence of the sole rationale that EMG signal amplitude increases together with the intensity of a muscle contraction, assertions concerning individual muscles' contribution to the emerging forces at a respective joint can only be made in a qualitative, not quantitative manner [190]. Hence, despite the advantages of EMG recordings, any current EMG measurements fall short of being perfectly conducive to an accurate calculation of individual muscles' actual moments acting on a respective joint. Past studies have correlated the results of EMG assessments with impact forces in gait and stepping tasks using force plates [184], utilized neuromusculoskeletal models with simulated EMG measurements to associate individual muscle contribution with contact forces in gait [151], and used computerized simulations to deduce muscle forces and consecutive joint loads from exploratory kinetic, kinematic, and electrophysiological recordings [191]. Based on the premise that earlier state-of-the-art computational models have been developed employing input of multiscale biophysical models, experimental EMG evidence [192], and data from ground reaction force plates [193], all evaluations regarding musculoskeletal forces and moments are only very elaborate estimations of real circumstances at best [194,195]. Regardless of these shortcomings, in vivo EMG still most accurately represents underlying muscle activity in healthy and pathological populations [174,192,196]. To that end, the synchronous measurement of in vivo joint loads and electrophysiological activity feeds into the development process of more refined computerized simulations by expanding knowledge about contact forces and muscle activity as they actually come into being.

1.3 Study objective and hypotheses

The objective of this study was to investigate the long-term functional outcome of THA and to examine the impact of GLmax, GLmed, and TFL status on postoperative in vivo hip joint loading through kinematics and muscle activity.

Although previous research found higher short-term [116] and long-term [117] in vivo hip joint loads to be associated with muscle status, correlations of kinematic abnormalities and irregularities in muscle activity with in vivo hip joint contact forces of a patient cohort as large as the one in this study have not yet been discussed in the existing literature. For this reason, radiological, kinetic, kinematic, and electrophysiological recordings of individuals with instrumented hip joint implants compiled at an average of 51 months after THA were analyzed in retrospect. The study participants were submitted to motion capture analyses in order to examine hip joint kinematics during various locomotor and non-locomotor ADLs, while the electrophysiological activity of their hip abductor muscles (GLmax, GLmed, and TFL) was also recorded. Simultaneously, in vivo joint loads were quantified in real time.

First, it was hypothesized that muscle damage has an effect on individual hip joint kinematics, which consequently impact on in vivo hip joint loads. Second, it was hypothesized that muscle deterioration influences individual muscle activity, which thereupon interferes with the in vivo contact forces of the hip.

With regard to the concomitant measurement of nine THA patients' in vivo hip joint contact forces spanning across five different ADLs, the data inspected within the framework of this study is unmatched. While there are limitations as to how this data can be interpreted, this investigation may serve as a basis of discussion for future research and propose ideas for pre-, intra-, and postoperative therapeutic measures.

2. Materials and methods

2.1 Ethics statement and study design

This study was approved by the Charité Ethics Commission (EA2/057/09) and registered at the German Clinical Trials Register (DRKS-ID: DRKS00000563). Prior to participation in this multicenter, non-interventional study, written informed consent was obtained by all patients. Data acquisition took place at Julius Wolff Institute for Biomechanics and Musculoskeletal Regeneration and Berlin-Brandenburg Center for Regenerative Therapies of Charité – Universitätsmedizin Berlin, Germany. The implantations of the instrumented hip prostheses essential to this investigation were performed by the same orthopedic surgeon at Sana Kliniken Sommerfeld in Kremmen, Germany.

At an average of 51 months after THA, data on individual in vivo hip joint kinetics, kinematics, and electrophysiological muscle activity were recorded by means of utilizing an instrumented hip implant for kinetics, conducting gait analysis for kinematics, and using sEMG measurements for electrophysiological muscle activity, following a prearranged protocol. Physical examinations were conducted by a board certified orthopedic surgeon and clinical scores were collected [115,117]. Individual periarticular hip muscle status was determined by assessing computed tomography (CT) scans according to a fixed classification system [115,116,197,198]. Published work of our research group already investigated the impact of muscle status on postoperative joint loading in a longitudinal study [115-117]. These publications include the outcomes of the physical examinations mentioned above and the results of the Harris Hip Score (HHS), the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), the EuroQol-5D-3L (EQ-5D-3L), and the visual analog scale (VAS) as an instrument of measurement for pain intensity [115,117].

2.2 Patients

Nine patients (n = 9), all of whom had an indication for undergoing primary THA due to degenerative OA of the hip joint, were included in this study. Patients had to meet certain criteria in order to be considered for this study: degenerative primary hip arthritis as a defined clinical indication for total hip replacement surgery, a minimum age of 50 and a maximum age of 65 years, and the motivation to take part in a long-term clinical study

which includes attending continuous data recording sessions. They also had to submit their informed written consent. The only exclusion criteria were previous THA on the ipsilateral side and the presence of active implants, such as cardiac pacemakers, cochlear implants, or neurostimulators.

The demographic characteristics of the patient population in this study were previously published by our research group and are summarized in table 1 below [115-117]. The measurements were conducted at an average of 51 months (50.7 months) after THA, with the earliest examination being 35 months postoperatively and the latest being 64 months postoperatively (±10.7 months). The patients' study names indicate the operated leg, i.e., R refers to the right operated side, L refers to the left operated side.

Table 1: Demographic characteristics of individual patients at 51 months after total hip arthroplasty. THA = Total hip arthroplasty. BMI = Body mass index.

Subject	H2R	H3L	H4L	H5L	H6R	H7R	H8L	H9L	H10R
Months after THA	64	62	60	57	50	47	43	38	35
Age [years]	67	65	55	67	72	56	59	57	55
Body height [cm]	172	168	178	168	176	179	178	181	163
Body weight [kg]	84	83	82	78	83	87	95	126	100
BMI [kg/m^2]	29	30	26	28	27	27	30	38	38
Sex [f/m]	m	m	m	f	m	m	m	m	f

2.3 Total hip arthroplasty and instrumented implant

Despite the wide array of surgical techniques, all patients received their THA procedure by means of a transgluteal DLA [199]. An instrumented "Cementless Tapered Wedge" (CTW™) hip prosthesis (Merete Medical GmbH, Berlin, Germany) was used as the femoral component and a cross-linked ultra-high-molecular-weight polyethylene inlay (Durasul, Zimmer GmbH, Winterthur, Switzerland) as the acetabular component. Detailed accounts of the implant's design and instrumentation have been discussed in past publications [134,135,137,200,201]. Past measurements of joint loads with instrumented hip implants performed by our research group have also been extensively discussed in the literature [116,130,131,135-140,145,202-204], including a major part of the results pertaining to the kinetic data collected 51 months after THA [115,117].

2.4 Kinematic recordings

Simultaneously to the kinetic measurements performed at our gait laboratory, subjects were recorded on video and with a motion capture system in order to obtain kinematic data for the assessment of commonly performed ADLs. The dynamic activities included within the context of this study were level walking at the respective subject's preferred walking speed, ascending and descending stairs without holding on to the banister, and standing up from and sitting down on a chair in an intuitive fashion and a bilateral, symmetrical manner. The setup of the motion capture system included ten high-speed, low-latency cameras (Vicon Motion Systems Limited, Oxford, UK), which were located in various positions of the room. The patients were equipped with 71 retroreflective markers, attached to predetermined anatomical landmarks on the head, trunk, pelvis, arms, thighs, shanks, and feet (please refer to figure 4 below) [205,206]. Each subject's height and weight were documented in order to be implemented into an MSM which is described in earlier publications [132,133,207]. Utilizing postoperative CT scans, this MSM employs patient-specific skeletal anatomies to establish individual hip joint centers and axes in order to assess hip joint kinematics [132,133,207]. Trials of each patient in each ADL were averaged according to the F_{res} by using a modified dynamic time warping algorithm [208].

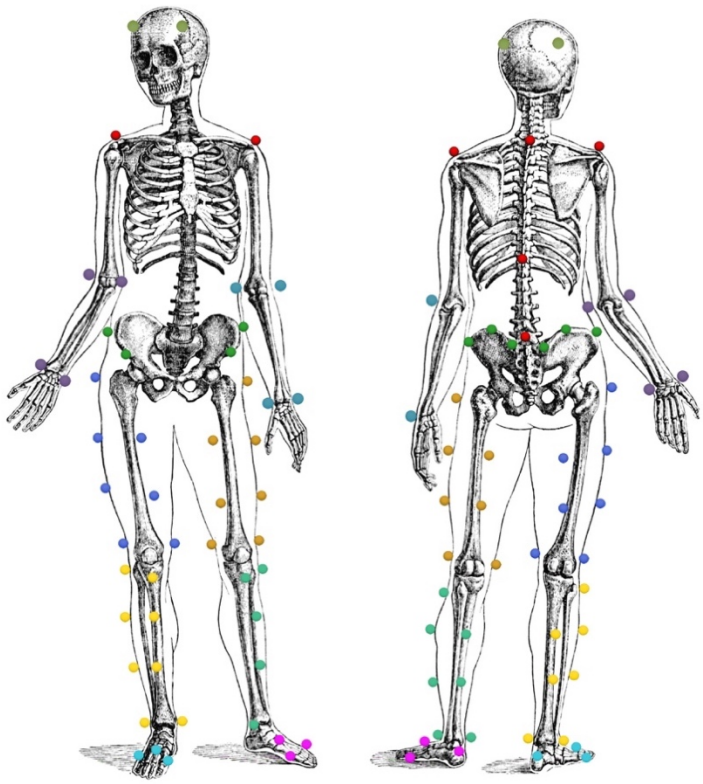

Figure 4: Placement of retroreflective markers on anatomical landmarks for kinematic recordings [205,206,209].

2.5 Muscle activity recordings

Individual muscle activity was measured using a system of wireless sEMG electrodes (Myon 320, Myon AG, Schwarzenberg, Switzerland). sEMG transmitters were placed over the GLmax, GLmed, and TFL of the subjects' operated legs, per protocol according to the sensor locations on individual muscles recommended by the Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles (SENIAM) project [181].

Because data on muscle status was available for the three periarticular hip muscles GLmax, GLmed, and TFL, these muscles' electrophysiological recordings were selected for inclusion in this retrospective investigation. Although the muscle status for the GLmin was also available and was assessed in earlier investigations [115-117], this muscle's activity was not measured via sEMG due to its high crosstalk with the GLmed and because of its anatomical position, being situated deeper under the skin and being covered by the GLmed.

sEMG data were collected at 9600 Hz, rectified and passed through a fourth-order band-pass Butterworth filter with a 10 Hz cutoff using custom scripts in MATLAB (MathWorks, Natick, MA, United States). In order to analyze patients' inter-individual differences, the ensemble averaged sEMG signals of each patient in each ADL were normalized using the peak muscle activity in millivolt as the reference point and expressed as the percentage of such peak muscle activity.

2.6 Periarticular muscle status

Pelvic CT scans were obtained on the day of individual kinematic and kinetic analyses and assessed by determining the muscles' fatty degeneration and individual hip abductor muscle status in accordance with an established classification system [115,116,197,198], ultimately leading to the results used in this investigation. Of the acquired data on GLmax, GLmed, and TFL status, total muscle volume (TMV) was documented in cubic meters (cm^3) and fat ratio (FR) was documented in percent (%). Earlier publications of our research group present the short- and long-term impact of muscle status on postoperative joint loading and describe the evaluation of the hip abductor muscle status of the patients in this study in further detail [115-117].

To stay within the limits of this investigation, the abovementioned muscle status indicators were used as parameters in our analyses and correlated with kinetic, kinematic, and electrophysiological marks of interest (please see chapter 2.7 Points of interest).

2.7 Points of interest

The points of interest chosen for the correlation analysis in this study are based upon relevant reference marks that are also discussed in the literature [116,117,130,131,135-140,145,203,204].

A structural impairment of periarticular hip muscles contributes to a rise of peak in vivo hip contact forces [117]. Hence, muscle status and kinematics realized at the 1st and 2nd (where applicable) peak F_{res} were correlated to investigate the impact of muscle damage on the performance of ADLs. Starting from the premise that potential gait abnormalities affect in vivo contact forces, peak joint loads were also correlated to concurrent kinematics. The muscles central to this investigation are mainly responsible for hip joint movement in the sagittal and frontal planes, which is why these two planes of motion and their respective angular motions extension/flexion (sagittal plane) and abduction/adduction (frontal plane) were examined. Since standing up and sitting down are tasks in which movement predominantly takes place in the sagittal plane [210-212], the focus lay exclusively on hip extension and flexion in these two ADLs.

It was hypothesized that structural muscle impairment influences dynamic joint movement. Thus, muscle status and maximum dynamic hip extension, flexion, abduction, and adduction were correlated. Along these lines, muscle status and dynamic hip ROM, which is defined as the amplitude of the maximum joint excursions produced during each ADL, were also correlated. Some maximum dynamic hip joint excursions occur around or close to the points of the gait cycle at which the operated limb's stance phase either begins (CTO) or ends (CHS). These are also instants in temporal proximity to the peak resultant forces. Other maximum dynamic hip joint excursions are realized at or around HS and TO, which is when the respective F_{res} is relatively low.

Muscle status and GLmax, GLmed, and TFL activity, as recorded during peak in vivo contact forces, were correlated in order to investigate the impact of muscle damage on muscle activity. Peak contact forces were also correlated to concurrent muscle activity,

so that the hypothesis of irregular muscle activity affecting in vivo joint loads could be put to the test.

The point in time at which a particular muscle reaches its peak activity during a normalized gait/movement cycle is defined as the instant of peak muscle activity (IPMA). Based on the assumption that the F_{res} at the hip joint is influenced by the timing of peak abductor muscle activity, the IPMA and the co-occurring joint loads were correlated. Additionally, it was hypothesized that anatomical muscle impairment alters the timing of muscle activity. For this reason, muscle status and the IPMA were correlated.

2.8 Statistical analysis

Statistical analyses were conducted using SPSS Statistics (version 25, IBM, Armonk, NY, United States). Individual hip joint kinematics and muscle activity were reported alongside mean and standard deviation. Tables illustrate the distributions among the respective points of interest for comparison purposes. In view of the low number of participants in this study (n = 9), the samples were considered to be distributed non-parametrically and presented as such. All correlations were performed applying Spearman's rank correlation coefficient (r_s). A correlation coefficient greater than or equal to .50 was regarded as a large effect size [213,214] and respective results are highlighted and included in the summary of the principal findings. An α smaller than or equal to .05 (two-tailed) was regarded as significant in determining the p-value's significance level. The data in this investigation are cross-sectional. Thus, the resulting correlation coefficients and p-values shall be interpreted as being descriptive rather than confirmatory.

3. Results

3.1 In vivo joint loads

The peak joint loads utilized in this investigation were first assessed within the framework of another study by our research group [115-117], while the joint loads achieved during maximum joint excursions and at the respective IPMA were additionally determined in the context of this study. All of the above were used for correlation analysis, as addressed previously in detail (please see chapter 2.7 Points of interest). Individual load patterns are shown in figure 5 below. The joint loads recorded at the points of interest and selected for correlation analyses are shown in tables 2, 3, 4, 5, and 6 below.

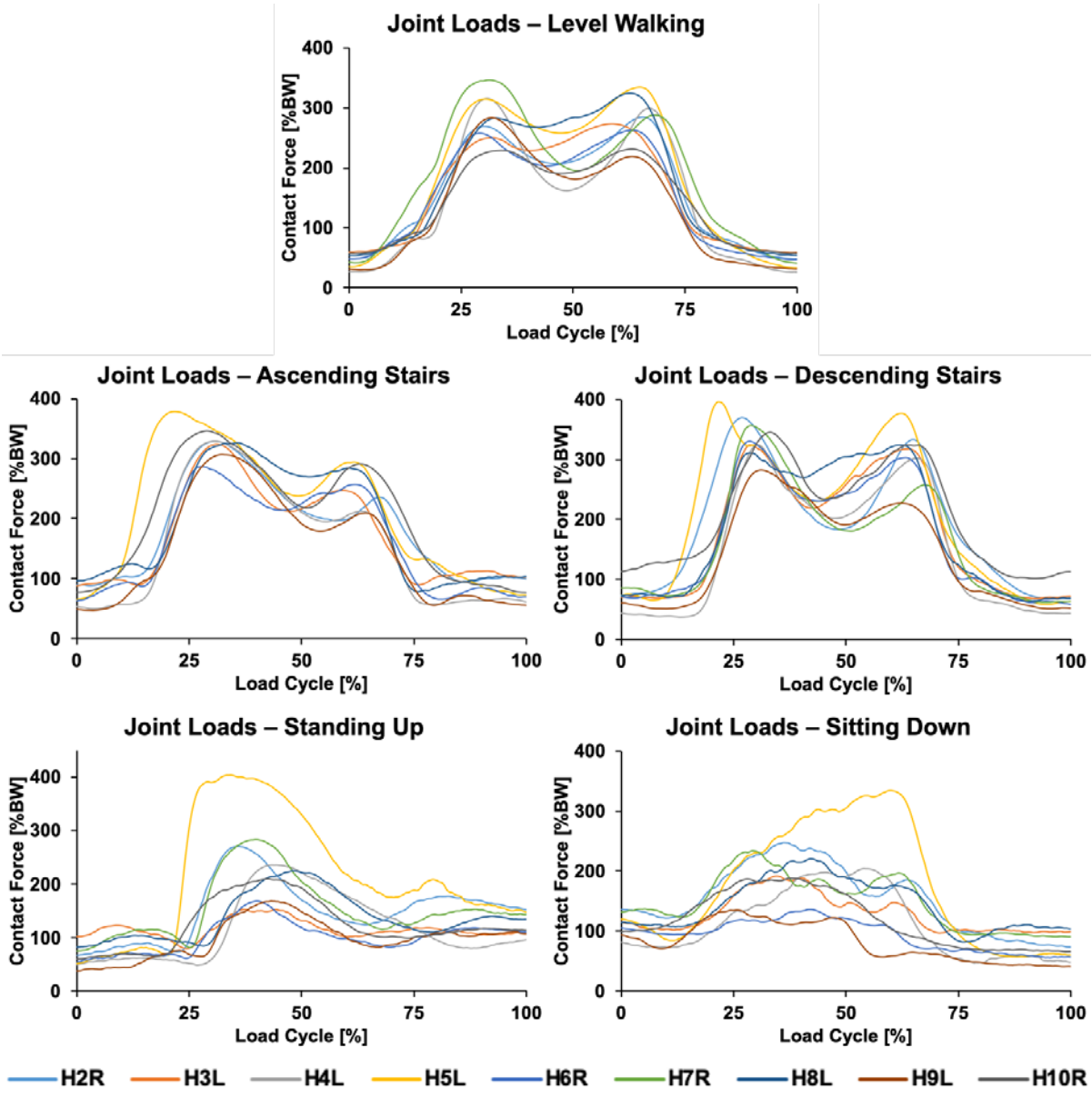

Figure 5: Individual load patterns in activities of daily living. Contact force in [%BW].

Table 2: Individual joint loads in <u>level walking</u> at various points of interest. Joint loads in [%BW] and [(N)]. 1st Peak = 1st peak resultant force. 2nd Peak = 2nd peak resultant force. Max = Maximum. Ext = Extension. Flex = Flexion. Abd = Abduction. Add = Adduction. IPMA = Instant of peak muscle activity. GLmax = Gluteus maximus muscle. GLmed = Gluteus medius muscle. TFL = Tensor fasciae latae muscle. N/A = Not available.

		Point of Interest								
		1st Peak	2nd Peak	Max Ext	Max Flex	Max Abd	Max Add	IPMA GLmax	IPMA GLmed	IPMA TFL
Patient	H2R	269 (2225.1)	284 (2350.5)	221 (1824.5)	49 (405.6)	74 (614.7)	251 (2077.0)	227 (1876.3)	143 (1185.1)	98 (808.9)
	H3L	250 (2045.1)	273 (2239.3)	217 (1778.6)	71 (584.2)	70 (572.4)	273 (2234.6)	165 (1352.3)	187 (1529.4)	185 (1514.4)
	H4L	316 (2547.4)	299 (2410.6)	241 (1945.4)	28 (222.6)	52 (421.3)	293 (2365.8)	194 (1562.0)	202 (1632.0)	168 (1352.4)
	H5L	314 (2397.0)	334 (2545.3)	297 (2266.5)	35 (268.9)	103 (784.2)	314 (2392.8)	272 (2077.5)	201 (1532.5)	235 (1790.2)
	H6R	258 (2110.1)	263 (2150.5)	231 (1887.5)	112 (912.1)	69 (567.9)	247 (2023.1)	183 (1500.4)	68 (554.8)	248 (2025.1)
	H7R	346 (2971.4)	288 (2468.4)	264 (2266.6)	150 (1286.3)	41 (354.9)	322 (2763.1)	322 (2766.8)	322 (2766.8)	N/A
	H8L	283 (2631.0)	324 (3016.3)	287 (2670.9)	53 (494.4)	76 (711.3)	318 (2954.8)	193 (1798.1)	147 (1364.5)	206 (1916.4)
	H9L	283 (3490.5)	218 (2692.7)	167 (2065.1)	109 (1339.4)	33 (406.5)	217 (2681.7)	272 (3349.1)	200 (2470.9)	148 (1819.7)
	H10R	229 (2242.3)	231 (2265.5)	167 (1638.8)	98 (964.3)	70 (686.7)	225 (2203.3)	187 (1833.7)	191 (1874.6)	189 (1854.4)

Table 3: Individual joint loads in <u>ascending stairs</u> at various points of interest. Joint loads in [%BW] and [(N)]. 1st Peak = 1st peak resultant force. 2nd Peak = 2nd peak resultant force. Max = Maximum. Ext = Extension. Flex = Flexion. Abd = Abduction. Add = Adduction. IPMA = Instant of peak muscle activity. GLmax = Gluteus maximus muscle. GLmed = Gluteus medius muscle. TFL = Tensor fasciae latae muscle. N/A = Not available.

		Point of Interest								
		1st Peak	2nd Peak	Max Ext	Max Flex	Max Abd	Max Add	IPMA GLmax	IPMA GLmed	IPMA TFL
Patient	H2R	329 (2721.4)	236 (1950.8)	139 (1149.4)	98 (810.3)	140 (1157.1)	180 (1491.2)	320 (2643.7)	293 (2422.2)	234 (1937.9)
	H3L	325 (2657.8)	248 (2028.4)	171 (1401.4)	99 (810.1)	105 (857.2)	295 (2418.1)	227 (1859.5)	286 (2340.6)	109 (889.3)
	H4L	329 (2653.8)	212 (1712.3)	175 (1409.9)	54 (434.9)	50 (403.2)	257 (2077.9)	200 (1613.7)	280 (2260.0)	60 (481.9)
	H5L	378 (2884.2)	294 (2244.4)	159 (1213.1)	78 (593.1)	242 (1848.9)	321 (2447.8)	364 (2773.7)	362 (2760.9)	77 (588.1)
	H6R	287 (2346.7)	257 (2102.9)	123 (1009.4)	84 (686.2)	80 (652.1)	257 (2098.6)	287 (2346.7)	88 (720.9)	97 (794.8)
	H8L	327 (3038.2)	284 (2644.0)	205 (1907.8)	109 (1016.2)	103 (961.3)	309 (2874.1)	273 (2537.8)	291 (2703.3)	272 (2527.9)
	H9L	307 (3785.7)	210 (2585.0)	146 (1795.4)	61 (754.8)	56 (688.9)	306 (3767.5)	72 (887.5)	47 (585.6)	214 (2642.3)
	H10R	347 (3401.0)	291 (2859.3)	238 (2332.7)	101 (986.1)	289 (2836.2)	282 (2764.1)	297 (2916.7)	341 (3345.0)	85 (829.6)

Table 4: Individual joint loads in descending stairs at various points of interest. Joint loads in [%BW] and [(N)]. 1st Peak = 1st peak resultant force. 2nd Peak = 2nd peak resultant force. Max = Maximum. Ext = Extension. Flex = Flexion. Abd = Abduction. Add = Adduction. IPMA = Instant of peak muscle activity. GLmax = Gluteus maximus muscle. GLmed = Gluteus medius muscle. TFL = Tensor fasciae latae muscle. N/A = Not available.

		Point of Interest								
		1st Peak	2nd Peak	Max Ext	Max Flex	Max Abd	Max Add	IPMA GLmax	IPMA GLmed	IPMA TFL
Patient	H2R	370 (3059.0)	333 (2755.3)	192 (1588.9)	88 (730.7)	101 (832.4)	333 (2754.7)	216 (1788.9)	196 (1618.3)	125 (1030.2)
	H3L	325 (2659.0)	318 (2603.0)	146 (1193.1)	79 (649.0)	95 (774.4)	318 (2602.1)	131 (1073.9)	109 (892.5)	72 (593.7)
	H4L	324 (2616.5)	302 (2439.2)	253 (2038.5)	48 (389.5)	65 (520.9)	317 (2557.8)	60 (484.3)	90 (724.4)	291 (2349.7)
	H5L	396 (3024.5)	377 (2880.3)	239 (1823.0)	60 (457.2)	91 (694.2)	377 (2875.2)	372 (2838.8)	386 (2947.8)	64 (489.2)
	H6R	331 (2705.7)	304 (2483.4)	241 (1969.1)	68 (553.6)	96 (786.9)	297 (2428.6)	106 (868.8)	102 (832.9)	103 (845.4)
	H7R	356 (3057.0)	258 (2212.0)	151 (1293.2)	86 (741.8)	78 (668.0)	248 (2128.8)	356 (3053.8)	78 (667.2)	N/A
	H8L	311 (2894.7)	325 (3019.6)	162 (1510.6)	79 (732.6)	64 (598.7)	310 (2885.1)	103 (961.7)	94 (873.5)	306 (2843.0)
	H9L	282 (3480.0)	227 (2801.1)	219 (2698.8)	57 (702.4)	66 (816.3)	223 (2754.8)	128 (1578.9)	211 (2596.8)	156 (1926.0)
	H10R	345 (3381.0)	324 (3177.7)	282 (2769.0)	104 (1018.3)	161 (1575.2)	323 (3167.8)	174 (1704.1)	147 (1440.5)	123 (1207.7)

Table 5: Individual joint loads in standing up at various points of interest. Joint loads in [%BW] and [(N)]. Peak = Peak resultant force. Max = Maximum. Ext = Extension. Flex = Flexion. IPMA = Instant of peak muscle activity. GLmax = Gluteus maximus muscle. GLmed = Gluteus medius muscle. TFL = Tensor fasciae latae muscle. N/A = Not available.

		Point of Interest					
		Peak	Max Ext	Max Flex	IPMA GLmax	IPMA GLmed	IPMA TFL
Patient	H2R	271 (2240.0)	170 (1408.6)	208 (1723.0)	145 (1202.7)	128 (1060.2)	211 (1745.4)
	H3L	156 (1255.0)	117 (960.7)	116 (949.1)	156 (1273.8)	115 (941.7)	116 (953.2)
	H4L	236 (1907.2)	96 (774.8)	229 (1844.3)	230 (1854.7)	112 (904.3)	188 (1519.6)
	H5L	404 (3079.8)	160 (1221.1)	390 (2978.5)	275 (2101.1)	297 (2263.1)	286 (2184.6)
	H6R	168 (1374.9)	117 (960.1)	127 (1037.4)	102 (832.1)	95 (780.9)	85 (691.4)
	H7R	284 (2435.3)	144 (1232.2)	225 (1933.0)	127 (1089.8)	132 (1136.4)	N/A
	H8L	225 (2088.2)	134 (1244.2)	175 (1630.0)	128 (1186.3)	168 (1560.2)	134 (1243.1)
	H9L	168 (2076.2)	107 (1322.9)	134 (1651.3)	92 (1138.5)	96 (1188.8)	108 (1330.3)
	H10R	210 (2055.9)	114 (1119.1)	187 (1833.6)	209 (2051.3)	195 (1910.4)	68 (667.4)

Table 6: Individual joint loads in <u>sitting down</u> at various points of interest. Joint loads in [%BW] and [(N)]. Peak = Peak resultant force. Max = Maximum. Ext = Extension. Flex = Flexion. IPMA = Instant of peak muscle activity. GLmax = Gluteus maximus muscle. GLmed = Gluteus medius muscle. TFL = Tensor fasciae latae muscle. N/A = Not available.

		Point of Interest					
		Peak	**Max Ext**	**Max Flex**	**IPMA GLmax**	**IPMA GLmed**	**IPMA TFL**
Patient	**H2R**	247 (2047.8)	136 (1127.5)	160 (1320.2)	162 (1336.0)	180 (1489.1)	123 (1014.8)
	H3L	192 (1567.9)	115 (945.8)	139 (1139.6)	184 (1507.2)	186 (1520.6)	101 (825.8)
	H4L	205 (1648.9)	82 (661.6)	204 (1645.6)	183 (1477.0)	182 (1467.2)	74 (595.4)
	H5L	334 (2545.0)	120 (918.7)	329 (2508.2)	210 (1603.5)	301 (2297.7)	156 (1193.9)
	H6R	137 (1117.4)	105 (862.0)	110 (903.8)	95 (780.7)	118 (963.4)	118 (964.9)
	H7R	234 (2009.8)	132 (1133.1)	196 (1684.7)	133 (1139.2)	179 (1535.4)	N/A
	H8L	220 (2044.6)	114 (1059.6)	173 (1609.2)	113 (1049.6)	200 (1859.9)	219 (2033.8)
	H9L	135 (1663.1)	93 (1149.3)	121 (1497.5)	132 (1629.6)	74 (908.0)	82 (1007.0)
	H10R	188 (1846.1)	99 (973.7)	172 (1686.2)	147 (1442.3)	185 (1817.6)	183 (1791.9)

3.2 Muscle status

The data pertaining to individual hip abductor muscle status used in this investigation were evaluated and published previously by our research group [115-117]. As discussed earlier (please see chapter 2.7 Points of interest), individual TMV and FR were correlated with various points of interest. Individual muscle status is listed in table 7 below.

Table 7: Individual muscle status. Total muscle volume in [cm^3]. Fat ratio in [%]. GLmax = Gluteus maximus muscle. GLmed = Gluteus medius muscle. TFL = Tensor fasciae latae muscle. TMV = Total muscle volume. FR = Fat ratio.

		GLmax		**GLmed**		**TFL**	
		TMV	**FR**	**TMV**	**FR**	**TMV**	**FR**
Patient	**H2R**	282.1	13.5	97.7	11.7	23.2	17.5
	H3L	245.0	23.0	144.9	18.3	55.0	8.1
	H4L	272.3	10.3	121.0	6.3	23.9	7.7
	H5L	193.3	28.2	121.9	23.4	19.1	7.0
	H6R	263.4	13.5	119.1	16.4	24.6	4.3
	H7R	347.7	18.2	145.5	16.0	28.7	18.3
	H8L	321.3	25.2	141.5	26.4	49.7	14.3
	H9L	334.4	21.6	164.6	19.2	25.3	8.4
	H10R	246.5	19.5	105.9	16.5	31.3	12.7

3.3 Kinematics

Kinematic measurements at various predetermined points during the gait cycle yielded individually different results for all parameters and activities observed. Due to a recording error, no kinematic measurements were available for subject H7R in ascending stairs. The numerical values recorded at individual points of interest for level walking, ascending and descending stairs, as well as standing up and sitting down are listed in the tables below. Also illustrated below are individual kinematic patterns.

3.3.1 Level walking

In level walking, maximum extension ranged from -4.7° (H2R) to -19.7° (H10R) with a mean of -9.7° ±4.8°. At the 2nd peak F_{res}, which was around TO, extension ranged from -0.2° (H3L) to -14.9° (H10R) with a mean of -6.9° ±4.4°.

In most subjects, maximum flexion occurred around HS, ranging from 22.4° (H9L) to 37.5° (H4L) with a mean of 29.6° ±5.2°. At the 1st peak F_{res}, which was around CTO, flexion ranged from 10.5° (H10R) to 30.1° (H4L) with a mean of 21.1° ±5.9°. Maximum abduction was achieved during the swing phase, ranging from -2.6° (H6R) to -15.5° (H7R) with a mean of -7.9° ±3.8°.

Maximum adduction occurred during the stance phase and lay between 5.7° (H5L) and 14.5° (H4L) with a mean of 8.3° ±2.6°. Subject H4L produced an adduction of 13.6° at the 1st peak F_{res} and of 10.8° at the 2nd peak F_{res}. All other subjects' range in the frontal plane lay between 2.6° (H10R) and 7.9° (H2R) at the 1st peak F_{res}, and -2.2° (H7R) and 8.7° (H8L) at the 2nd peak F_{res}.

Sagittal ROM lay between 32.9° (H2R) and 48.8° (H4L) with a mean of 39.3° ±5.5°. A mean of 16.2° ±3.9° was measured in the frontal plane, ranging from 11.8° (H6R) to 22.7° (H7R).

Individual kinematic patterns and numerical values of interest are shown in figure 6 and table 8 below.

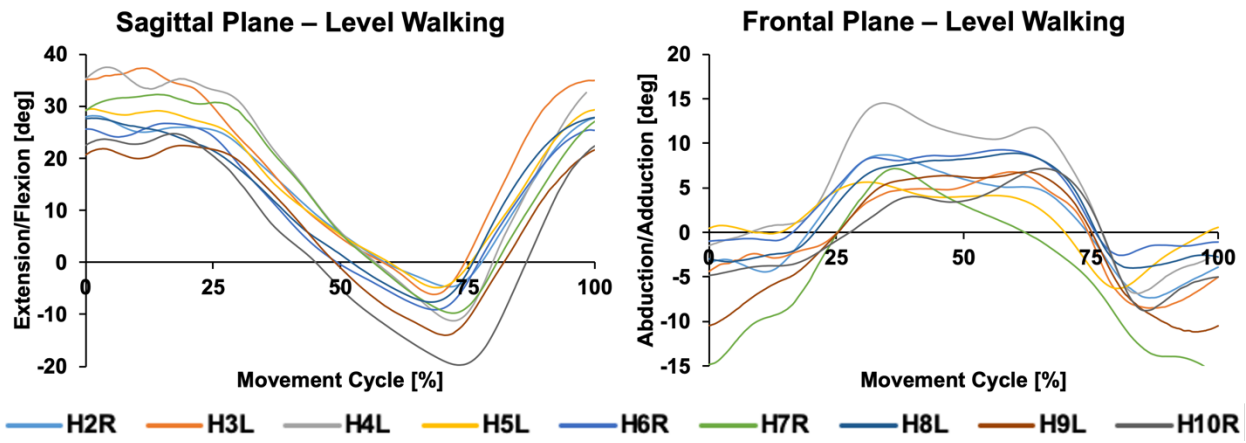

Figure 6: Individual kinematic patterns in level walking. Kinematics in [deg].

Table 8: Individual kinematics in level walking. All numerical values in [deg]. 1st Peak = 1st peak resultant force. 2nd Peak = 2nd peak resultant force. ROM = Range of motion. SD = Standard deviation.

		H2R	H3L	H4L	H5L	H6R	H7R	H8L	H9L	H10R	Mean (SD)
1st Peak	Sagittal Plane	22.6	22.3	30.1	22.8	19.9	27.6	15.8	18.1	10.5	21.1 (5.9)
	Frontal Plane	7.9	3.7	13.6	5.7	7.5	4.9	7.1	4.3	2.6	6.4 (3.3)
2nd Peak	Sagittal Plane	-2.9	-0.2	-8.5	-3.4	-7.3	-8.4	-6.2	-10.8	-14.9	-6.9 (4.4)
	Frontal Plane	4.7	6.8	10.8	2.7	8.7	-2.2	8.7	6.8	6.9	6.0 (3.9)
Maximum	Extension	-4.7	-6.2	-11.3	-4.9	-9.0	-9.8	-7.7	-14.0	-19.7	-9.7 (4.8)
	Flexion	28.2	37.4	37.5	29.5	26.7	32.3	27.9	22.4	24.7	29.6 (5.2)
	Abduction	-7.4	-8.4	-6.9	-6.3	-2.6	-15.5	-4.0	-11.2	-8.8	-7.9 (3.8)
	Adduction	8.7	6.8	14.5	5.7	9.3	7.2	8.9	6.8	7.2	8.3 (2.6)
ROM	Sagittal Plane	32.9	43.6	48.8	34.4	35.8	42.0	35.6	36.4	44.4	39.3 (5.5)
	Frontal Plane	16.1	15.2	21.4	12.0	11.8	22.7	13.0	18.0	16.0	16.2 (3.9)

3.3.2 Ascending stairs

Ascending stairs produced a similar kinematic pattern in the sagittal plane, as maximum extension occurred during or briefly after TO and maximum flexion occurred during or directly before HS. Only subject H9L displayed an actual extension with a maximum of -0.6°, while all other subjects produced a mean extension of 9.1° ±6.6°. The frontal plane yielded a mean abduction of -13.4° ±8.4°, ranging from -4.5° (H5L) to -28.6° (H8L).

Subject H2R was measured with a maximum adduction of 40.2° briefly after HS, with all other subjects producing a maximum adduction in the range of 3.7° (H3L) to 17.1° (H5L) (mean of 12.2° ±12.1°). The sagittal ROM showed a mean of 58.1° ±3.1°. The frontal ROM in subject H2R was 47.5°, while the mean frontal ROM in all subjects was 25.6° ±12.1°. Individual kinematic patterns and numerical values of interest are shown in figure 7 and table 9 below.

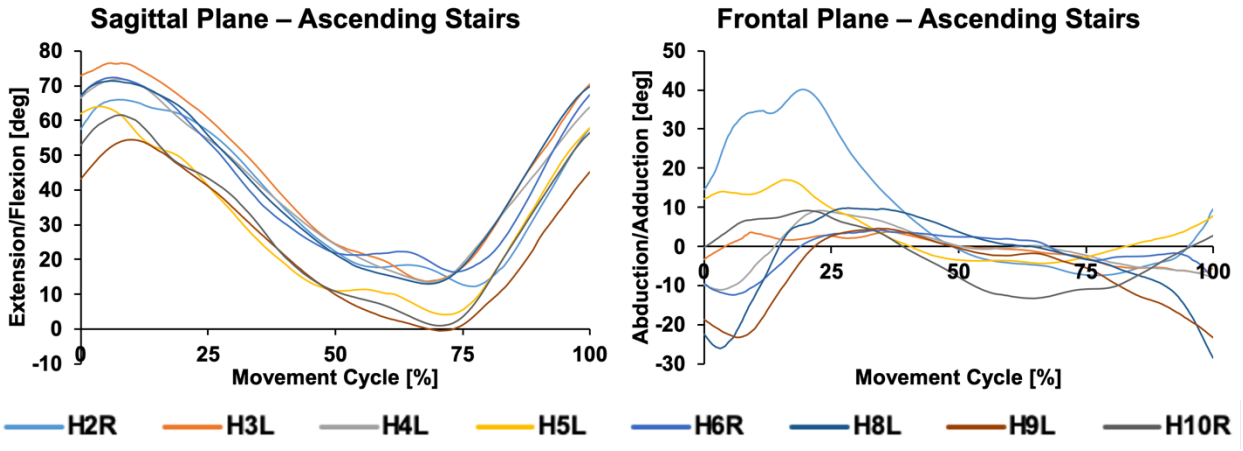

Figure 7: Individual kinematic patterns in ascending stairs. Kinematics in [deg].

Table 9: Individual kinematics in ascending stairs. All numerical values in [deg]. 1st Peak = 1st peak resultant force. 2nd Peak = 2nd peak resultant force. ROM = Range of motion. SD = Standard deviation. N/A = Not available.

		H2R	H3L	H4L	H5L	H6R	H7R	H8L	H9L	H10R	Mean (SD)
1st Peak	Sagittal Plane	49.5	52.2	47.5	46.5	49.7	N/A	39.1	31.4	39.4	44.4 (7.1)
	Frontal Plane	20.7	2.4	7.5	12.7	3.3	N/A	9.4	4.2	5.6	8.2 (6.1)
2nd Peak	Sagittal Plane	17.9	20.0	15.2	10.0	22.3	N/A	15.4	1.6	4.7	13.4 (7.3)
	Frontal Plane	-5.0	-0.7	0.1	-3.9	1.6	N/A	0.4	-1.8	-13.1	-2.8 (4.7)
Maximum	Extension	12.2	13.7	13.3	4.1	16.5	N/A	12.9	-0.6	0.9	9.1 (6.6)
	Flexion	65.9	76.5	71.9	64.0	72.3	N/A	71.4	54.4	61.5	67.2 (7.2)
	Abduction	-7.3	-6.8	-11.1	-4.5	-12.4	N/A	-28.6	-23.4	-13.2	-13.4 (8.4)
	Adduction	40.2	3.7	9.1	17.1	4.1	N/A	9.7	4.6	9.2	12.2 (12.1)
ROM	Sagittal Plane	53.6	62.8	58.6	59.9	55.8	N/A	58.5	55.0	60.6	58.1 (3.1)
	Frontal Plane	47.5	10.5	20.3	21.5	16.5	N/A	38.4	28.0	22.4	25.6 (12.1)

3.3.3 Descending stairs

Descending stairs produced no actual extension, yielding a mean maximum extension of 13.4° ±3.8°, ranging from 8.5° (H10R) to 18.1° (H3L). Mean maximum flexion was shown to average 42.6° ±6.3°, ranging from 32.4° (H9L) to 50.7° (H3L). Mean maximum abduction was -10.0° ±6.0, ranging from -4.9° (H2R) to -24.5° (H7R). As the only subject, H7R yielded an actual abduction in maximum adduction at -2.6°, with other subjects' maximum adduction ranging from 0.5° (H3L) to 8.5° (H8L). Sagittal and frontal ROM showed means of 29.2° ±3.5° and 14.2° ±4.0°, respectively. Individual kinematic patterns and numerical values of interest are shown in figure 8 and table 10 below.

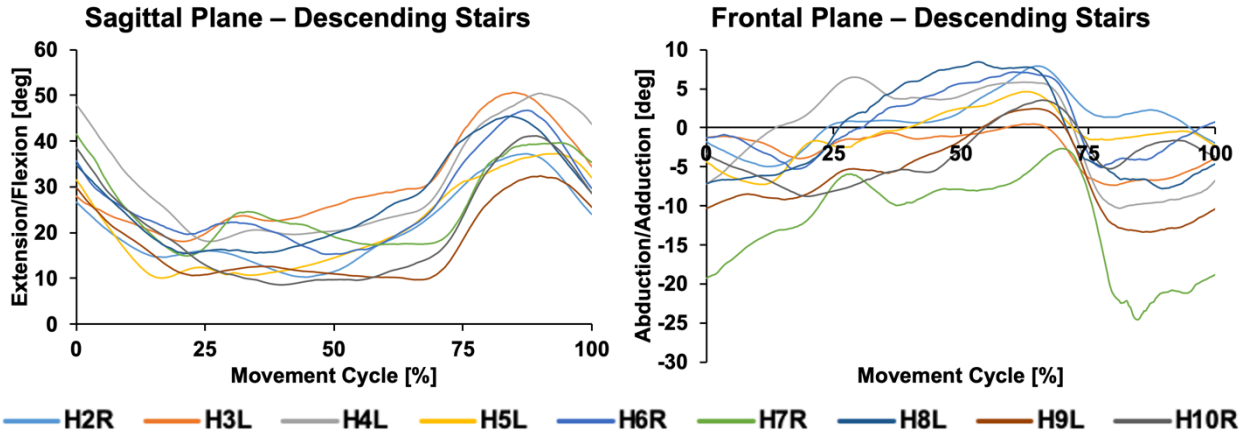

Figure 8: Individual kinematic patterns in descending stairs. Kinematics in [deg].

Table 10: Individual kinematics in descending stairs. All numerical values in [deg]. 1st Peak = 1st peak resultant force. 2nd Peak = 2nd peak resultant force. ROM = Range of motion. SD = Standard deviation.

		H2R	H3L	H4L	H5L	H6R	H7R	H8L	H9L	H10R	Mean (SD)
1st Peak	Sagittal Plane	15.9	22.6	19.4	11.9	22.1	21.8	16.2	12.0	9.9	16.9 (4.9)
	Frontal Plane	0.8	-1.4	6.3	-1.7	-0.4	-6.0	1.5	-5.4	-6.3	-1.4 (4.1)
2nd Peak	Sagittal Plane	20.5	29.6	24.7	19.6	20.4	17.5	26.6	10.1	12.4	20.1 (6.3)
	Frontal Plane	7.9	0.5	5.7	4.6	7.0	-3.3	7.8	2.3	3.2	4.0 (3.7)
Maximum	Extension	10.2	18.1	18.1	10.0	15.3	14.9	15.4	9.7	8.5	13.4 (3.8)
	Flexion	37.3	50.7	50.5	37.2	46.7	41.4	45.5	32.4	41.2	42.6 (6.3)
	Abduction	-4.9	-7.4	-10.3	-7.3	-5.3	-24.5	-7.8	-13.4	-8.8	-10.0 (6.0)
	Adduction	7.9	0.5	6.5	4.6	7.1	-2.6	8.5	2.4	3.5	4.3 (3.7)
ROM	Sagittal Plane	27.1	32.6	32.4	27.2	31.4	26.6	30.1	22.7	32.7	29.2 (3.5)
	Frontal Plane	12.8	7.9	16.8	11.9	12.4	21.9	16.2	15.8	12.4	14.2 (4.0)

3.3.4 Standing up

In standing up, maximum flexion occurred shortly before peak contact forces, averaging 91.2° ±14.4° and ranging between 72.9° and 113.6°. At peak joint loads, kinematic measurements showed a mean flexion of 85.4° ±12.6°, ranging from 68.4° to 101.1°. Individual kinematic patterns and numerical values of interest are shown in figure 9 and table 11 below.

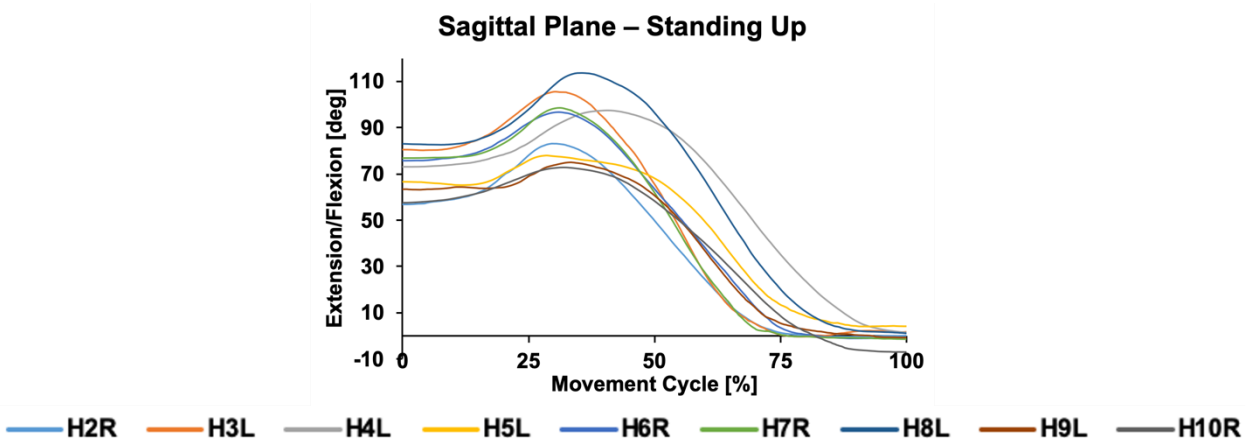

Figure 9: Individual kinematic patterns in standing up. Kinematics in [deg].

Table 11: Individual kinematics in standing up. All numerical values in [deg]. Peak = Peak resultant force. ROM = Range of motion. SD = Standard deviation.

		H2R	H3L	H4L	H5L	H6R	H7R	H8L	H9L	H10R	Mean (SD)
Peak	Sagittal Plane	79.6	100.4	96.8	76.6	87.4	88.9	101.1	69.3	68.4	85.4 (12.6)
Maximum	Extension	-0.2	-0.4	1.3	4.2	-1.2	-1.4	1.0	-0.7	-6.8	-0.5 (2.9)
	Flexion	83.1	105.5	97.4	77.9	96.6	98.5	113.6	74.9	72.9	91.2 (14.4)
ROM	Sagittal Plane	83.3	105.9	96.1	73.7	97.8	99.9	112.5	75.5	79.7	91.6 (13.9)

3.3.5 Sitting down

Sitting down generated flexion patterns, in which most subjects reached their maximum after peak contact forces were achieved. Flexion at peak joint loads ranged from 54.5° to 96.3°, averaging 72.8° ±14.5°. Maximum flexion, however, lay between 70.3° and 111.2°, with a mean of 88.9° ±13.8°. Individual kinematic patterns and numerical values of interest are shown in figure 10 and table 12 below.

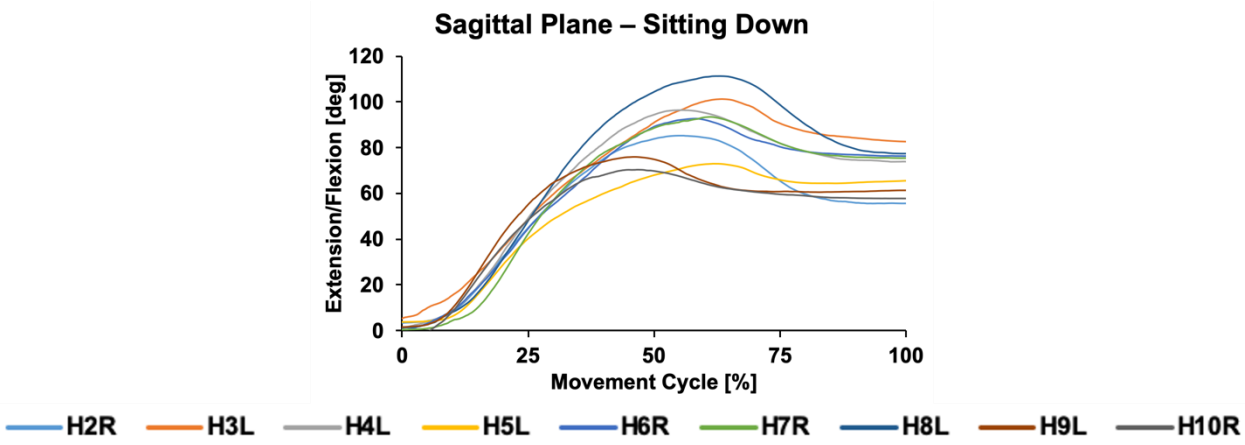

Figure 10: Individual kinematic patterns in sitting down. Kinematics in [deg].

Table 12: Individual kinematics in sitting down. All numerical values in [deg]. Peak = Peak resultant force. ROM = Range of motion. SD = Standard deviation.

		H2R	H3L	H4L	H5L	H6R	H7R	H8L	H9L	H10R	Mean (SD)
Peak	Sagittal Plane	69.2	67.7	96.3	72.7	78.3	54.5	93.5	56.5	66.9	72.8 (14.5)
Maximum	Extension	3.4	5.2	1.3	3.9	0.0	0.6	1.2	1.6	-4.1	1.5 (2.7)
	Flexion	85.2	101.5	96.4	73.0	92.8	93.4	111.2	76.0	70.3	88.9 (13.8)
ROM	Sagittal Plane	81.8	96.3	95.1	69.1	92.8	92.8	110.0	74.4	74.3	87.4 (13.3)

3.4 Muscle activity

Muscle activity recordings at various predetermined points during the gait cycle produced individually different results for all parameters and activities observed. Due to a recording error in subject H7R, there were no data on TFL activity in any of the ADLs and also no data on muscle activity in ascending stairs. The numerical values recorded at individual points of interest for level walking, ascending and descending stairs, as well as standing up and sitting down are listed in the tables below. Also illustrated below are individual muscle activity patterns.

3.4.1 Level walking

A remarkable finding in level walking was that, apart from H2R and H6R, the respective inter-individual IPMA was identical (H7R and H10R) or very similar. In all subjects, GLmax activity peaked shortly before the 1st peak F_{res} at a mean of 24.0% ±2.7% of the gait cycle. Upon joint loads reaching their 1st peak, GLmax activity varied considerably from 17% to 63% with a mean of 44.4% ±17.4%. As contact forces approached the 2nd peak F_{res}, GLmax activity decreased even further to an average 18.8% ±11.0%. Although GLmax activity generally dropped throughout the gait cycle after hitting its peak, H3L, H4L, H5L and H9L recorded surges in activity briefly after the 2nd peak and at around TO. The average GLmax activity level of 46% ±13% in H10R was the highest among all subjects, with H10R also being the only subject whose GLmax activity never dropped below 36%. GLmed activity resembled GLmax activity with the exception of the late maxima in subjects H2R and H6R at 75% and 81% of the gait cycle, respectively. Despite these two subjects achieving maximum GLmed activity around TO, however, both H2R and H6R produced substantial activity spikes briefly before the 1st peak F_{res}, while H2R came close to achieving maximum GLmed activity. Shortly before the 1st peak F_{res} is also where all other subjects reached maximum GLmed activity with a mean of 35.3% ±24.5%. As the F_{res} reached its 1st peak, GLmed activity fell to a mean of 45.1% ±12.5%. Upon reaching the 2nd peak F_{res}, GLmed activity decreased even more, averaging 26.7% ±9.3%, only to experience activity surges around TO similar to the GLmax.

TFL activity patterns were very different among all subjects, nonetheless, producing comparable instants of peak muscle activity almost immediately before the 1st peak F_{res}. The mean IPMA was found to be at 22.2% ±4.1% of the gait cycle. Only H2R achieved

peak TFL activity earlier than any other subject, reaching its maximum at 13% of the gait cycle. Subjects H4L and H10R exhibited TFL activity that was only substantially present at its IPMA, falling to levels below 20% activity immediately after reaching peak muscle activity. TFL activity patterns in H2R, H3L, and H5L resembled those recorded for the GLmed in the same patients. TFL activity in H6R is notable in that it had the highest mean activity with 74% ±25% and an activity pattern that is unique among all subjects. A constantly close-to-maximum relative activity was recorded throughout the trials, only dropping in mid stance, reaching its low around the 2nd peak F_{res} and abruptly rising again to high levels after TO.

Individual muscle activity patterns and numerical values of interest are shown in figure 11 and table 13 below.

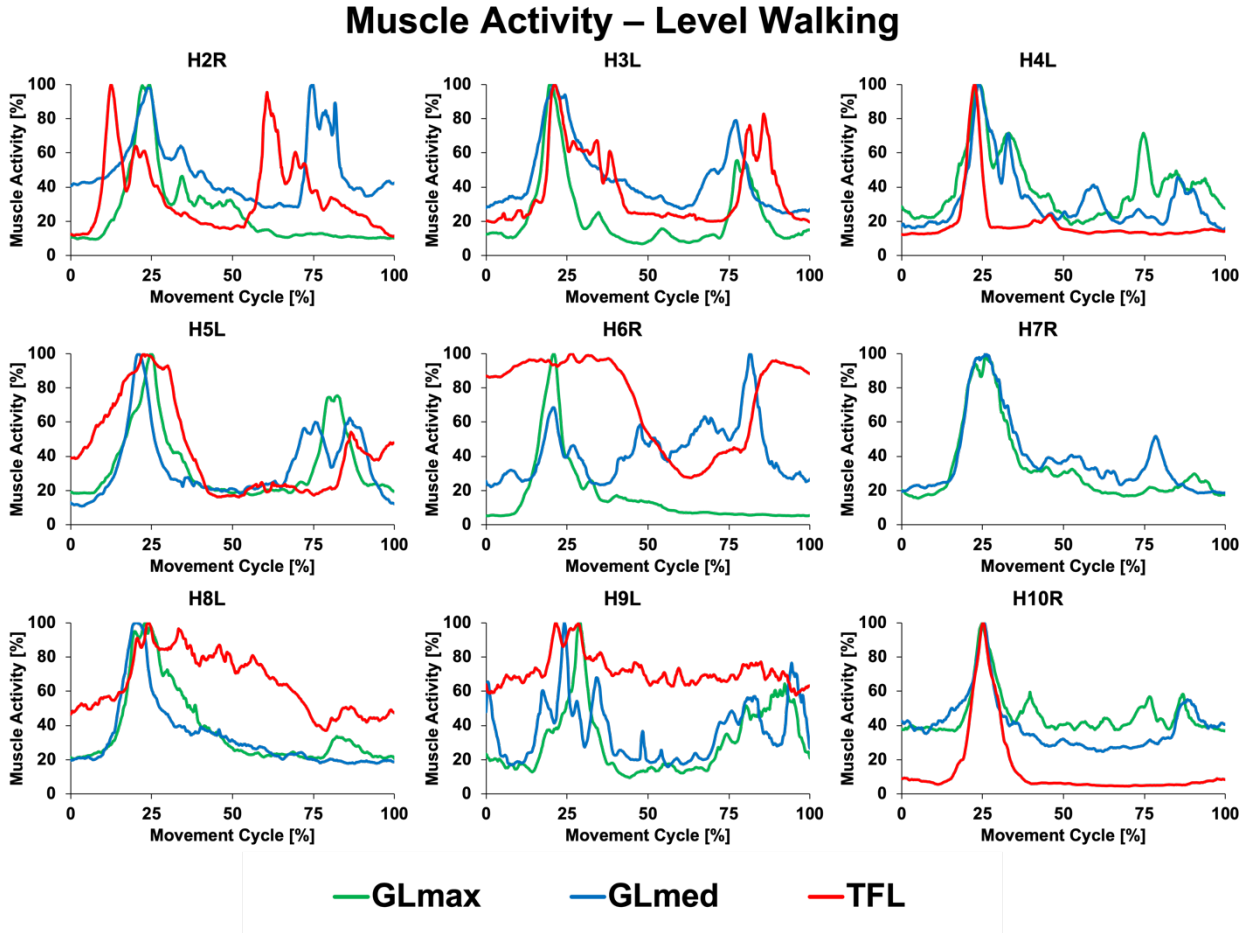

Figure 11: Individual muscle activity patterns in level walking. Muscle activity in [%].

Table 13: Individual muscle activity in level walking. All numerical values in [%] of the peak electrophysiological activity (points of interest: 1st peak resultant force, 2nd peak resultant force) or movement cycle (point of interest: IPMA). 1st Peak = 1st peak resultant force. 2nd Peak = 2nd peak resultant force. IPMA = Instant of peak muscle activity. GLmax = Gluteus maximus muscle. GLmed = Gluteus medius muscle. TFL = Tensor fasciae latae muscle. SD = Standard deviation. N/A = Not available.

		H2R	H3L	H4L	H5L	H6R	H7R	H8L	H9L	H10R	Mean (SD)
1st Peak	GLmax	31	17	63	45	23	61	62	54	42	44.4 (17.4)
	GLmed	57	55	43	27	39	69	39	38	41	45.1 (12.5)
	TFL	28	60	16	88	95	N/A	90	80	16	59.2 (34.1)
2nd Peak	GLmax	11	10	23	20	7	17	23	14	44	18.8 (11.0)
	GLmed	30	29	19	22	49	24	23	19	26	26.7 (9.3)
	TFL	48	24	14	23	28	N/A	68	64	5	34.2 (23.3)
IPMA	GLmax	24	20	24	25	21	26	23	29	25	24.0 (2.7)
	GLmed	75	22	24	21	81	26	20	24	25	35.3 (24.5)
	TFL	13	22	23	23	27	N/A	24	22	25	22.2 (4.1)

3.4.2 Ascending stairs

In ascending stairs, the instant of peak GLmax activity for all but two subjects was measured between 19% and 28% of the movement cycle, which is at or shortly before the 1st peak F_{res}. H8L and H9L achieved peak GLmax activity at 49% and 87% of the movement cycle, respectively, raising the mean to 34.7% ±23.1%. GLmed activity patterns in all individuals but H6R and H9L were similar to those of the GLmax, averaging an IPMA of 30.1% ±21.0%. With the exception of H6R and H9L, who displayed an IPMA of 76% (TO) and 3%, respectively, GLmed reached peak activity between 18% and 39% of the movement cycle in all other subjects. As was the case in level walking, TFL activity patterns were highly variable amongst subjects. With no common activity pattern distinguishable, TFL activity mostly peaked briefly before or after the 2nd peak F_{res}, apart from H9L (24%) and H10R (6%). Individual muscle activity patterns and numerical values of interest are shown in figure 12 and table 14 below.

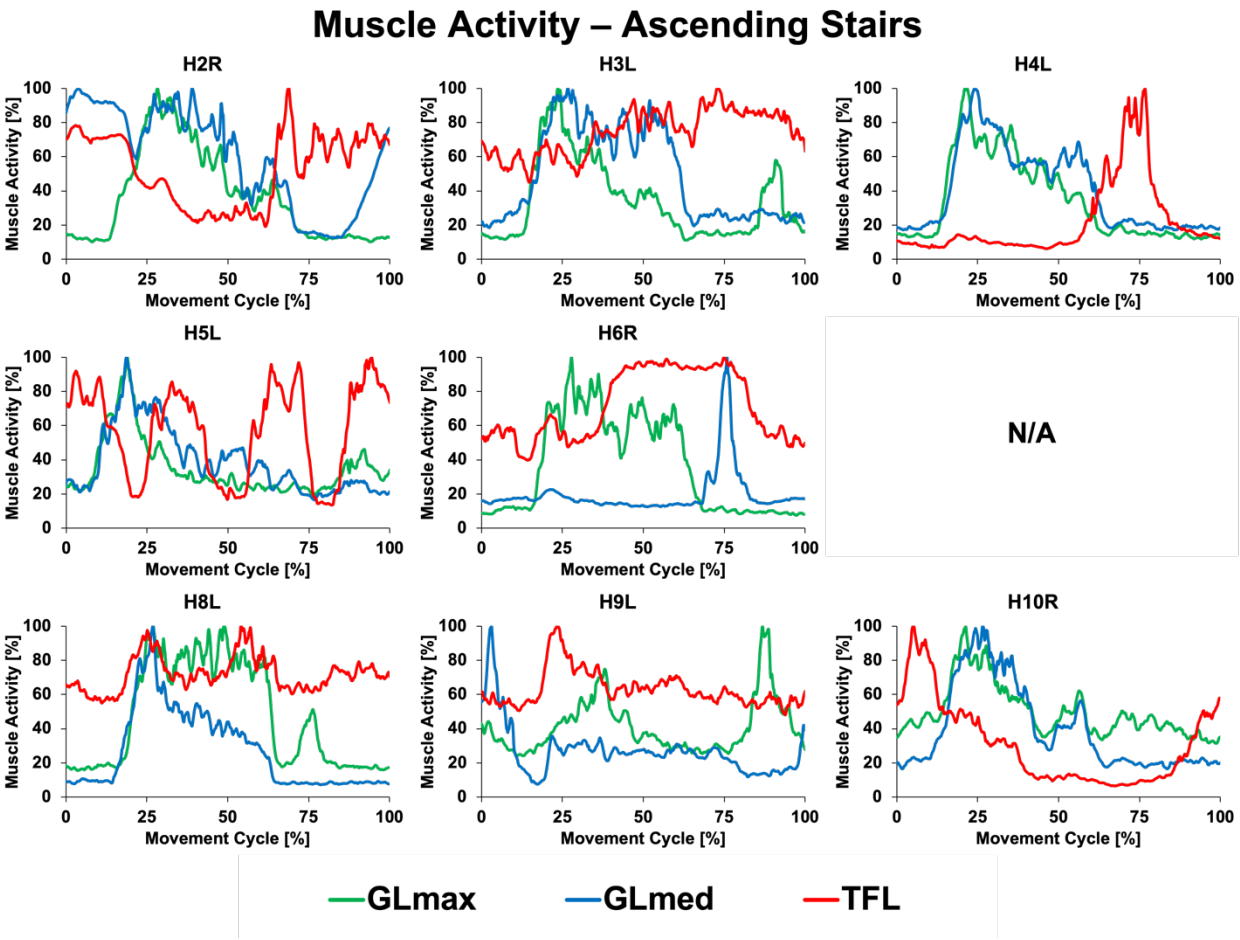

Figure 12: Individual muscle activity patterns in ascending stairs. Muscle activity in [%]. N/A = Not available.

Table 14: Individual muscle activity in ascending stairs. All numerical values in [%] of the peak electrophysiological activity (points of interest: 1st peak resultant force, 2nd peak resultant force) or movement cycle (point of interest: IPMA). 1st Peak = 1st peak resultant force. 2nd Peak = 2nd peak resultant force. IPMA = Instant of peak muscle activity. GLmax = Gluteus maximus muscle. GLmed = Gluteus medius muscle. TFL = Tensor fasciae latae muscle. SD = Standard deviation. N/A = Not available.

		H2R	H3L	H4L	H5L	H6R	H7R	H8L	H9L	H10R	Mean (SD)
1st Peak	GLmax	91	64	72	60	100	N/A	86	51	78	75.3 (16.4)
	GLmed	86	77	76	68	17	N/A	50	31	85	61.3 (25.9)
	TFL	45	57	10	18	50	N/A	71	73	30	44.2 (23.4)
2nd Peak	GLmax	31	21	16	22	57	N/A	77	28	34	35.7 (21.0)
	GLmed	46	65	24	36	14	N/A	28	28	21	32.7 (16.2)
	TFL	88	78	44	72	93	N/A	82	60	8	65.7 (28.0)
IPMA	GLmax	28	23	21	19	28	N/A	49	87	21	34.7 (23.1)
	GLmed	39	27	24	18	76	N/A	27	3	27	30.1 (21.0)
	TFL	69	73	77	95	75	N/A	54	24	6	59.0 (30.0)

3.4.3 Descending stairs

In descending stairs, GLmax activity reached peak levels at a mean of 32.8% ±23.0% of the movement cycle. Yet, H3L and H9L both produced their IPMA at around TO, with all other subjects falling within the range of 18% to 29%. Complementary to the GLmax, maximum GLmed activity occurred at an average of 30.8% ±24.3% of the movement cycle, with H6R and H9L achieving their respective IPMA shortly after the 2nd peak F_{res} at 80% (TO) and 66% (CTO), respectively. Analogously to the previous ADLs above, the TFL did not yield a distinct activity pattern among the entire group of subjects, yet among one specific cluster: the TFL in H3L, H6R, and H9L showed resembling activity patterns with 73% to 81% averages, never dropping below an activity of 50%. Individual muscle activity patterns and numerical values of interest are shown in figure 13 and table 15 below.

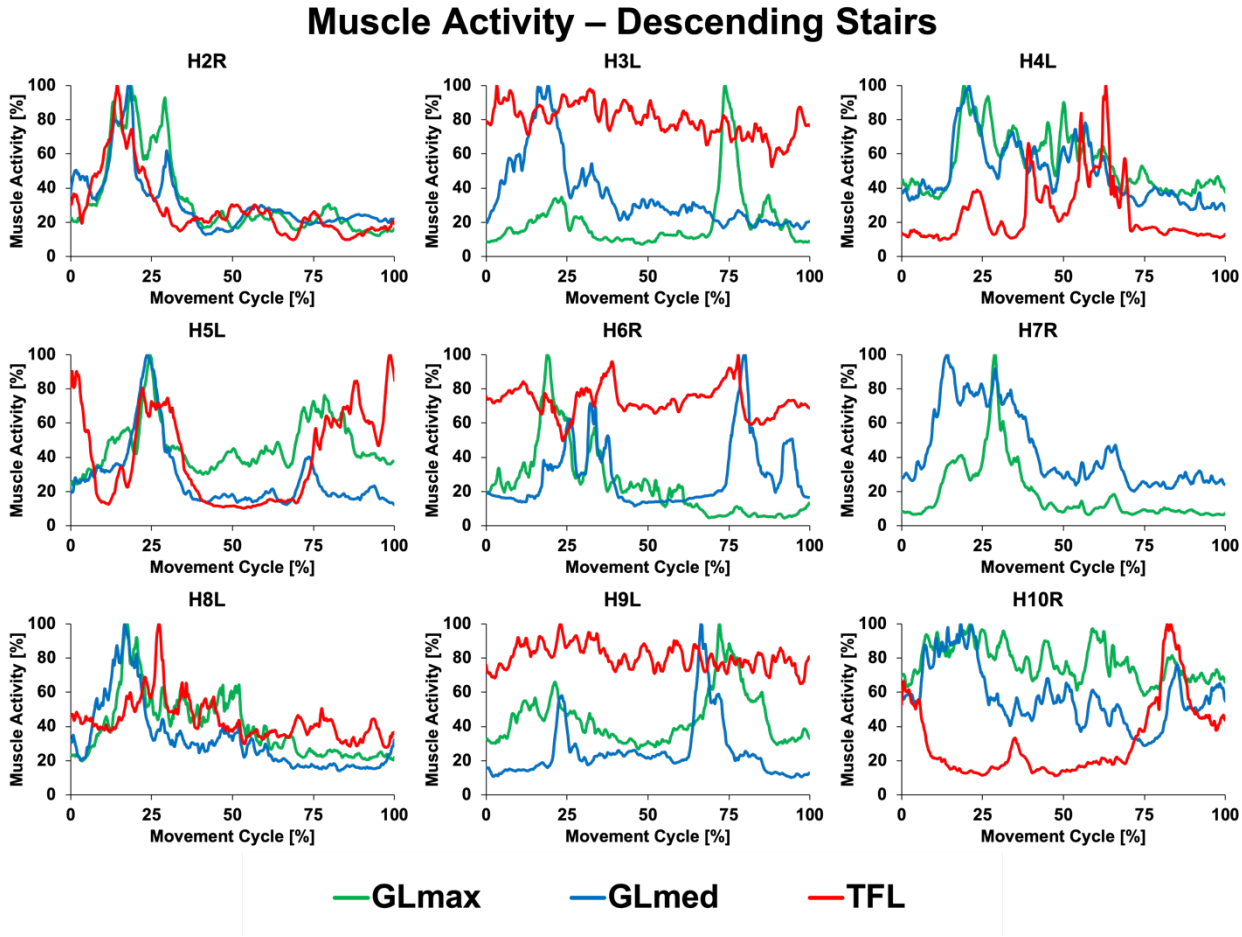

Figure 13: Individual muscle activity patterns in descending stairs. Muscle activity in [%].

Table 15: Individual muscle activity in descending stairs. All numerical values in [%] of the peak electrophysiological activity (points of interest: 1st peak resultant force, 2nd peak resultant force) or movement cycle (point of interest: IPMA). 1st Peak = 1st peak resultant force. 2nd Peak = 2nd peak resultant force. IPMA = Instant of peak muscle activity. GLmax = Gluteus maximus muscle. GLmed = Gluteus medius muscle. TFL = Tensor fasciae latae muscle. SD = Standard deviation. N/A = Not available.

		H2R	H3L	H4L	H5L	H6R	H7R	H8L	H9L	H10R	Mean (SD)
1st Peak	GLmax	73	22	57	74	25	95	62	40	84	59.3 (25.6)
	GLmed	35	43	59	86	29	89	43	19	42	49.6 (24.2)
	TFL	29	92	20	80	77	N/A	73	90	22	60.5 (31.0)
2nd Peak	GLmax	25	11	47	42	11	10	27	38	72	31.3 (20.7)
	GLmed	27	31	41	21	16	35	22	21	41	28.4 (9.2)
	TFL	13	79	39	15	76	N/A	37	72	19	43.7 (28.2)
IPMA	GLmax	19	74	19	24	19	29	18	72	21	32.8 (23.0)
	GLmed	18	19	21	23	80	14	17	66	19	30.8 (24.3)
	TFL	14	4	63	99	78	N/A	27	23	82	48.7 (36.0)

3.4.4 Standing up

In standing up, four subjects were recorded with an identical or similar respective intra-individual IPMA. The GLmax, GLmed, and TFL peaked at 25% of the movement cycle in H5L, while H3L, H4L, and H7R produced relatively adjacent maxima of muscle activity (39% to 56%, 50% to 77%, and 63% to 65%, respectively). With the exception of the TFL in H10R, H5L is also the subject with the lowest recorded IPMA in all muscles. All individuals' muscles' particular mean IPMA was recorded in the second half of the standing up task, well past peak joint loads were achieved: the GLmax averaged at 56.9% ±18.3%, the GLmed at 54.7% ±16.8%, and the TFL at 51.2% ±33.1% of the movement cycle. In general, activity levels of the GLmax and GLmed were higher on average throughout the whole motion of standing up than they were during stair ascent, with almost no single prominent activity bursts of these muscles. Individual muscle activity patterns and numerical values of interest are shown in figure 14 and table 16 below.

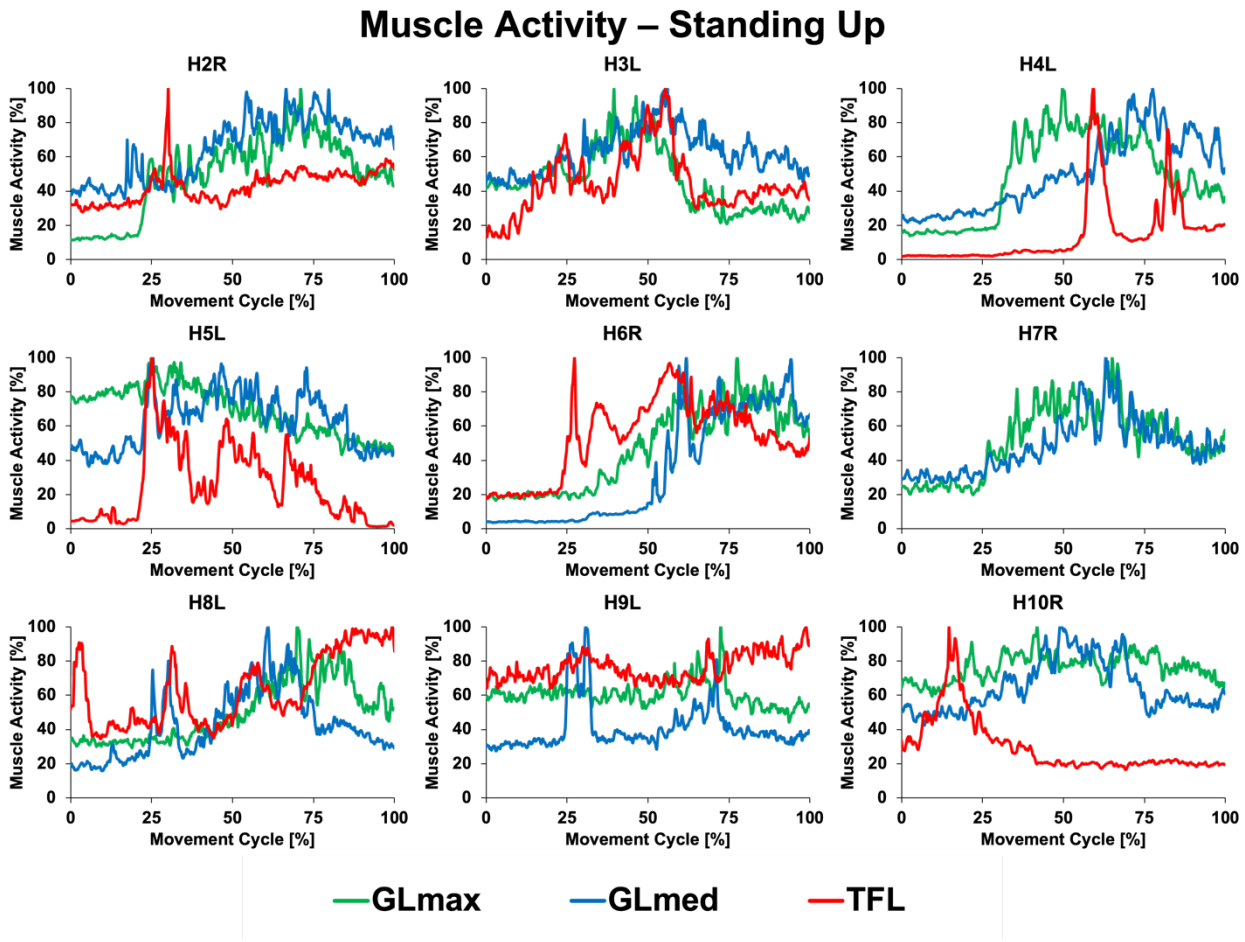

Figure 14: Individual muscle activity patterns in standing up. Muscle activity in [%].

Table 16: Individual muscle activity in standing up. All numerical values in [%] of the peak electrophysiological activity (point of interest: peak resultant force) or movement cycle (point of interest: IPMA). Peak = Peak resultant force. IPMA = Instant of peak muscle activity. GLmax = Gluteus maximus muscle. GLmed = Gluteus medius muscle. TFL = Tensor fasciae latae muscle. SD = Standard deviation. N/A = Not available.

		H2R	H3L	H4L	H5L	H6R	H7R	H8L	H9L	H10R	Mean (SD)
Peak	GLmax	48	78	87	96	29	64	48	62	84	66.1 (21.9)
	GLmed	51	83	48	68	8	44	65	34	78	53.3 (23.3)
	TFL	37	44	5	37	56	N/A	44	72	20	39.4 (20.5)
IPMA	GLmax	71	39	50	25	77	65	70	72	42	56.9 (18.3)
	GLmed	67	56	77	25	62	63	61	31	50	54.7 (16.8)
	TFL	30	55	59	25	27	N/A	99	99	15	51.2 (33.1)

3.4.5 Sitting down

In sitting down, two GLmax and GLmed activity patterns were identifiable: H4L, H5L, H7R, H8L, H9L, and H10R produced continuous activity levels with less and smaller activity bursts. H2R, H3L, and H6R generated more and larger activity surges in both or either one of the two muscles. Only in H6R did all muscles' activity levels drop to 20% or lower after the process of sitting down, whereas in all other subjects, muscle activity levels remained relatively high or volatile even after having sat down. H3L, H4L, and H7R had a similar intra-individual respective IPMA for the GLmax and GLmed, with H6R, H8L, and H10R showing a resembling intra-individual particular IPMA for the GLmed and TFL. Both the GLmax and GLmed achieved adjacent activity levels at peak joint loads in H4L, H6R, H9L, and H10R. The GLmax and TFL, on the other hand, displayed coinciding activity levels in H2R and H3L. Individual muscle activity patterns and numerical values of interest are shown in figure 15 and table 17 below.

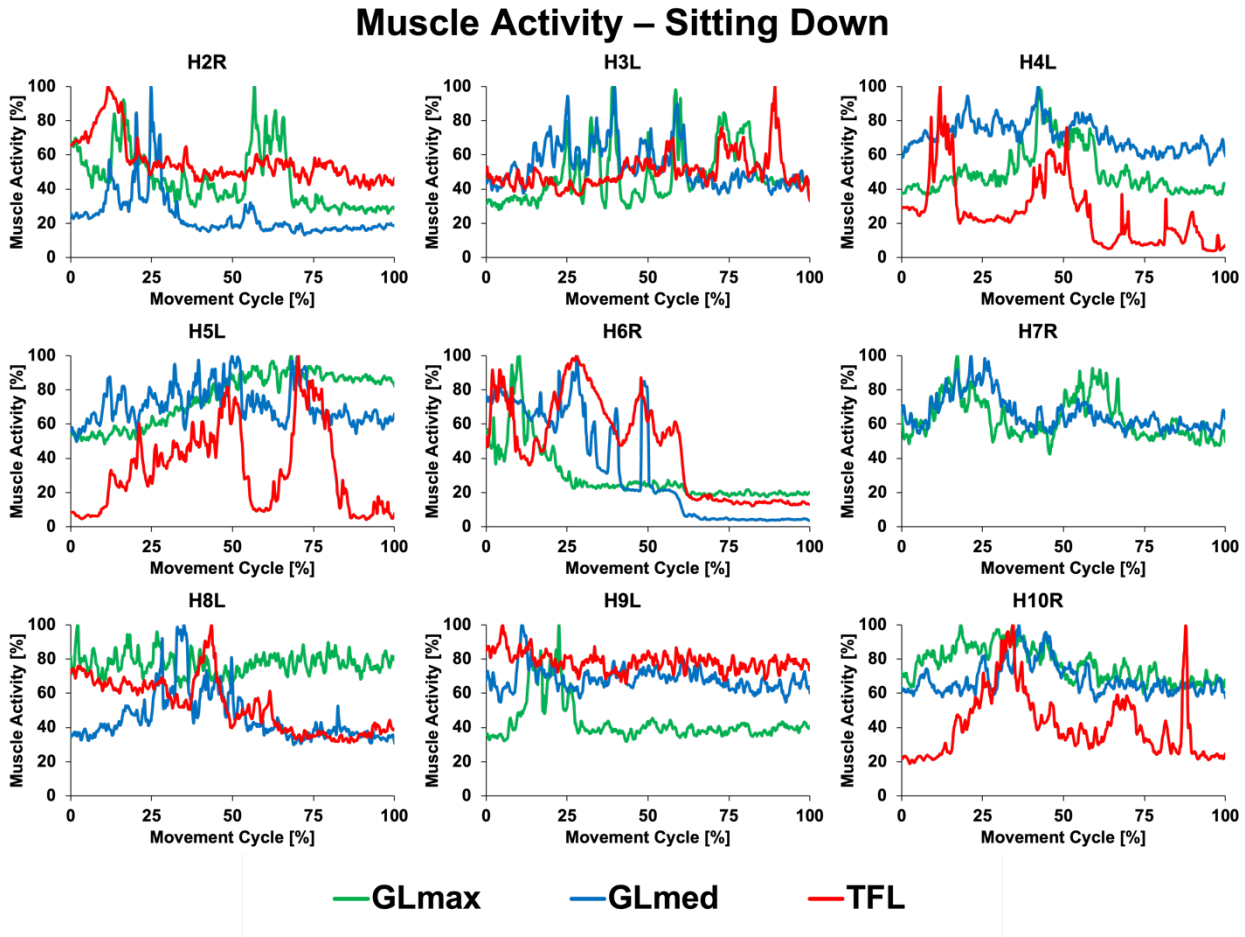

Figure 15: Individual muscle activity patterns in sitting down. Muscle activity in [%].

Table 17: Individual muscle activity in sitting down. All numerical values in [%] of the peak electrophysiological activity (point of interest: peak resultant force) or movement cycle (point of interest: IPMA). Peak = Peak resultant force. IPMA = Instant of peak muscle activity. GLmax = Gluteus maximus muscle. GLmed = Gluteus medius muscle. TFL = Tensor fasciae latae muscle. SD = Standard deviation. N/A = Not available.

		H2R	H3L	H4L	H5L	H6R	H7R	H8L	H9L	H10R	Mean (SD)
Peak	GLmax	40	44	74	84	24	60	76	60	94	61.9 (22.5)
	GLmed	15	61	79	68	23	79	58	64	91	59.6 (25.2)
	TFL	49	45	30	11	48	N/A	90	81	61	51.9 (25.8)
IPMA	GLmax	57	39	42	68	10	17	2	22	18	30.7 (22.1)
	GLmed	25	40	42	50	28	21	35	11	36	32.1 (11.8)
	TFL	11	89	12	70	28	N/A	43	5	34	36.7 (30.0)

3.5 Hypothesis I

Impact of hip abductor muscle status on hip joint kinematics

3.5.1 Correlation of muscle status and kinematics at peak in vivo contact forces

Total Muscle Volume

The only significant correlation of TMV and kinematics at peak joint loads was found between the frontal plane at the 2nd peak in stair descent and the GLmed (r_s = -0.68*). The TFL showed effects on the frontal plane at the 1st peak F_{res} in level walking (r_s = -0.62) and the 1st peak F_{res} in stair ascent (r_s = -0.64). Moreover, an effect of the TFL could also be observed on the sagittal plane at the 1st peak in level walking (r_s = -0.53). The GLmax was found to have an effect on the sagittal plane at the 1st peak of stair ascent (r_s = -0.52). The complete array of correlations between TMV and kinematics at peak in vivo contact forces is presented in table 18 below.

Table 18: Correlation of total muscle volume and kinematics at peak in vivo contact forces. TFL = Tensor fasciae latae muscle. 1st Peak = 1st peak resultant force. 2nd Peak = 2nd peak resultant force. Peak = Peak resultant force. r_s = Spearman's rank correlation coefficient. p = p-value. Significance level * = 0.05.

		Total Muscle Volume					
		Gluteus Maximus		Gluteus Medius		TFL	
		r_s	p	r_s	p	r_s	p
Walking 1st Peak	Sagittal Plane	0.03	0.932	0.07	0.865	-0.53	0.139
	Frontal Plane	0.17	0.668	-0.40	0.286	-0.62	0.077
Walking 2nd Peak	Sagittal Plane	-0.33	0.381	-0.07	0.865	-0.05	0.898
	Frontal Plane	-0.02	0.966	-0.23	0.546	0.18	0.637
Stairs Up 1st Peak	Sagittal Plane	-0.52	0.183	-0.31	0.456	-0.05	0.911
	Frontal Plane	0.14	0.736	-0.43	0.289	-0.64	0.086
Stairs Up 2nd Peak	Sagittal Plane	-0.19	0.651	-0.24	0.570	0.12	0.779
	Frontal Plane	0.21	0.610	0.33	0.420	0.24	0.570
Stairs Down 1st Peak	Sagittal Plane	0.10	0.798	0.25	0.516	0.33	0.381
	Frontal Plane	0.02	0.966	-0.27	0.488	-0.22	0.576
Stairs Down 2nd Peak	Sagittal Plane	-0.25	0.516	-0.13	0.732	0.23	0.546
	Frontal Plane	-0.05	0.898	-0.68*	0.042	-0.42	0.265
Stand Up Peak	Sagittal Plane	0.15	0.700	0.25	0.516	0.40	0.286
Sit Down Peak	Sagittal Plane	-0.30	0.433	-0.42	0.265	-0.28	0.460

Fat Ratio

No significant correlations were found between FR and kinematics at peak joint loads. Yet, the GLmax was found to have an effect on the frontal plane at the 1st peak F_{res} in level walking (r_s = -0.53). Furthermore, the GLmed showed effects on the sagittal plane at the 1st peak F_{res} in level walking and stair ascent (r_s = -0.57 and -0.52, respectively). Finally, the TFL showed an effect on the sagittal plane in sitting down (r_s = -0.50). The complete array of correlations between FR and kinematics at peak in vivo contact forces is presented in table 19 below.

Table 19: Correlation of fat ratio and kinematics at peak in vivo contact forces. TFL = Tensor fasciae latae muscle. 1st Peak = 1st peak resultant force. 2nd Peak = 2nd peak resultant force. Peak = Peak resultant force. r_s = Spearman's rank correlation coefficient. p = p-value.

		Fat Ratio					
		Gluteus Maximus		Gluteus Medius		TFL	
		r_s	p	r_s	p	r_s	p
Walking 1st Peak	Sagittal Plane	-0.35	0.356	-0.57	0.112	-0.08	0.831
	Frontal Plane	-0.53	0.139	-0.47	0.205	-0.17	0.668
Walking 2nd Peak	Sagittal Plane	0.32	0.406	0.15	0.700	-0.05	0.898
	Frontal Plane	-0.32	0.406	-0.03	0.932	-0.35	0.356
Stairs Up 1st Peak	Sagittal Plane	-0.33	0.420	-0.52	0.183	-0.36	0.385
	Frontal Plane	0.14	0.736	0.02	0.955	0.43	0.289
Stairs Up 2nd Peak	Sagittal Plane	-0.24	0.570	-0.29	0.493	-0.19	0.651
	Frontal Plane	-0.17	0.693	0.05	0.911	0.45	0.260
Stairs Down 1st Peak	Sagittal Plane	-0.32	0.406	-0.28	0.460	-0.13	0.732
	Frontal Plane	-0.28	0.460	-0.20	0.606	-0.22	0.576
Stairs Down 2nd Peak	Sagittal Plane	0.02	0.966	-0.03	0.932	-0.12	0.765
	Frontal Plane	-0.22	0.576	-0.08	0.831	-0.10	0.798
Stand Up Peak	Sagittal Plane	0.00	1.000	0.00	1.000	0.07	0.865
Sit Down Peak	Sagittal Plane	-0.17	0.668	-0.03	0.932	-0.50	0.170

3.5.2 Correlation of muscle status and maximum joint excursions

Total Muscle Volume

The GLmax was found to correlate significantly with maximum abduction in stair ascent (r_s = -0.74*). GLmax also showed effects on maximum abduction in stair descent (r_s = -0.55). The GLmed was found to have an effect on both maximum abduction and adduction in stair descent (r_s = -0.65 and -0.60, respectively). Additionally, the TFL was shown to have an effect on adduction in stair ascent (r_s = -0.57). The complete array of correlations between TMV and maximum joint excursions is presented in table 20 below.

Table 20: Correlation of total muscle volume and maximum joint excursions. TFL = Tensor fasciae latae muscle. r_s = Spearman's rank correlation coefficient. p = p-value. Significance level * = 0.05.

		Total Muscle Volume					
		Gluteus Maximus		Gluteus Medius		TFL	
		r_s	p	r_s	p	r_s	p
Walking	Maximum Extension	-0.28	0.460	-0.20	0.606	-0.30	0.433
	Maximum Flexion	-0.18	0.637	0.07	0.865	-0.08	0.831
	Maximum Abduction	-0.40	0.286	-0.43	0.244	-0.28	0.460
	Maximum Adduction	0.38	0.308	-0.38	0.308	-0.10	0.798
Stairs Up	Maximum Extension	-0.24	0.570	-0.10	0.823	0.17	0.693
	Maximum Flexion	-0.29	0.493	0.00	1.000	0.26	0.531
	Maximum Abduction	-0.74*	0.037	-0.17	0.693	-0.48	0.233
	Maximum Adduction	0.10	0.823	-0.48	0.233	-0.57	0.233
Stairs Down	Maximum Extension	0.00	1.000	0.10	0.798	0.20	0.606
	Maximum Flexion	-0.20	0.606	-0.05	0.898	0.45	0.224
	Maximum Abduction	-0.55	0.125	-0.65	0.058	-0.35	0.356
	Maximum Adduction	-0.03	0.932	-0.60	0.088	-0.32	0.406
Stand Up	Maximum Flexion	0.22	0.576	0.32	0.406	0.45	0.224
Sit Down	Maximum Flexion	0.27	0.488	0.32	0.406	0.47	0.205

Fat Ratio

The GLmax correlated significantly with adduction in level walking (r_s = -0.75*). The GLmed showed an effect on adduction in level walking (r_s = -0.55). The TFL was found to show an effect in abduction in level walking (r_s = -0.58), as well as in adduction (r_s = 0.50) in stair ascent. The complete array of correlations between FR and maximum joint excursions is presented in table 21 below.

Table 21: Correlation of fat ratio and maximum joint excursions. TFL = Tensor fasciae latae muscle. r_s = Spearman's rank correlation coefficient. p = p-value. Significance level * = 0.05.

		Fat Ratio					
		Gluteus Maximus		Gluteus Medius		TFL	
		r_s	p	r_s	p	r_s	p
Walking	Maximum Extension	0.28	0.460	0.12	0.765	-0.02	0.966
	Maximum Flexion	-0.15	0.700	-0.40	0.286	-0.05	0.898
	Maximum Abduction	0.00	1.000	0.18	0.637	-0.58	0.099
	Maximum Adduction	-0.75*	0.020	-0.55	0.125	0.02	0.966
Stairs Up	Maximum Extension	-0.33	0.420	-0.33	0.420	-0.43	0.289
	Maximum Flexion	-0.24	0.570	-0.29	0.493	-0.36	0.385
	Maximum Abduction	0.10	0.823	-0.24	0.570	-0.38	0.352
	Maximum Adduction	0.21	0.610	0.10	0.823	0.50	0.207
Stairs Down	Maximum Extension	-0.27	0.488	-0.25	0.516	-0.20	0.606
	Maximum Flexion	-0.32	0.406	-0.28	0.460	-0.23	0.546
	Maximum Abduction	0.03	0.932	0.03	0.932	-0.33	0.381
	Maximum Adduction	-0.13	0.732	0.03	0.932	-0.12	0.765
Stand Up	Maximum Flexion	0.05	0.898	0.03	0.932	0.17	0.668
Sit Down	Maximum Flexion	-0.05	0.898	-0.02	0.966	0.12	0.765

3.5.3 Correlation of muscle status and dynamic range of motion

Total Muscle Volume

The GLmax was found to significantly correlate with sagittal ROM in stair ascent and descent (r_s = -0.76* and -0.67*, respectively), as well as the frontal ROM in stair descent (r_s = 0.87**). The GLmax also correlated with frontal ROM in level walking (r_s = 0.63) and stair ascent (r_s = 0.64). The TFL was found to significantly correlate with sagittal ROM in standing up (r_s = 0.67*), while also showing an effect in sitting down (r_s = 0.57). The complete array of correlations between TMV and dynamic ROM is presented in table 22 below.

Table 22: Correlation of total muscle volume and dynamic range of motion. TFL = Tensor fasciae latae muscle. ROM = Range of motion. r_s = Spearman's rank correlation coefficient. p = p-value. Significance level * = 0.05, ** = 0.01.

		Total Muscle Volume					
		Gluteus Maximus		Gluteus Medius		TFL	
		r_s	p	r_s	p	r_s	p
Walking	Sagittal ROM	-0.05	0.898	0.17	0.668	0.43	0.244
	Frontal ROM	0.63	0.067	0.28	0.460	0.02	0.966
Stairs Up	Sagittal ROM	-0.76*	0.028	0.19	0.651	0.40	0.320
	Frontal ROM	0.64	0.086	-0.21	0.610	-0.19	0.651
Stairs Down	Sagittal ROM	-0.67*	0.050	-0.45	0.224	0.37	0.332
	Frontal ROM	0.87**	0.002	0.25	0.516	-0.05	0.898
Stand Up	Sagittal ROM	0.27	0.488	0.20	0.606	0.67*	0.050
Sit Down	Sagittal ROM	0.30	0.433	0.27	0.488	0.57	0.112

Fat Ratio

Only the TFL was found to significantly correlate with frontal ROM in stair ascent (r_s = 0.81*), while also showing effects in level walking (r_s = 0.58). The GLmed showed an effect in frontal ROM in level walking (r_s = -0.55). The complete array of correlations between FR and dynamic ROM is presented in table 23 below.

Table 23: Correlation of fat ratio and dynamic range of motion. TFL = Tensor fasciae latae muscle. ROM = Range of motion. r_s = Spearman's rank correlation coefficient. p = p-value. Significance level * = 0.05.

		Fat Ratio					
		Gluteus Maximus		Gluteus Medius		TFL	
		r_s	p	r_s	p	r_s	p
Walking	Sagittal ROM	-0.33	0.381	-0.33	0.381	-0.10	0.798
	Frontal ROM	-0.40	0.286	-0.55	0.125	0.58	0.099
Stairs Up	Sagittal ROM	0.33	0.420	0.19	0.651	-0.29	0.493
	Frontal ROM	0.14	0.736	0.19	0.651	0.81*	0.015
Stairs Down	Sagittal ROM	-0.10	0.798	-0.08	0.831	-0.37	0.332
	Frontal ROM	-0.43	0.244	-0.37	0.332	0.48	0.187
Stand Up	Sagittal ROM	-0.07	0.865	-0.02	0.966	0.27	0.488
Sit Down	Sagittal ROM	-0.12	0.765	-0.07	0.865	0.18	0.637

Impact of hip joint kinematics on in vivo hip joint contact forces

3.5.4 Correlation of kinematics and peak in vivo contact forces

The sagittal plane correlated significantly with the 1st peak F_{res} in level walking (r_s = 0.72*). Moreover, there was a significant correlation between the frontal plane and the 2nd peak in stair ascent (r_s = 0.71*). Furthermore, the frontal plane was found to have an effect on the 2nd peak F_{res} in stair descent (r_s = 0.55). The complete array of correlations between kinematics and peak in vivo contact forces is presented in table 24 below.

Table 24: Correlation of kinematics and peak in vivo contact forces. 1st Peak = 1st peak resultant force. 2nd Peak = 2nd peak resultant force. Peak = Peak resultant force. r_s = Spearman's rank correlation coefficient. p = p-value. Significance level * = 0.05.

		Sagittal Plane		Frontal Plane	
		r_s	p	r_s	p
Joint Load	Walking 1st Peak	0.72*	0.030	0.40	0.286
	Walking 2nd Peak	0.43	0.244	-0.08	0.831
	Stairs Up 1st Peak	-0.17	0.693	0.71*	0.047
	Stairs Up 2nd Peak	0.02	0.955	-0.17	0.693
	Stairs Down 1st Peak	-0.22	0.576	-0.28	0.460
	Stairs Down 2nd Peak	0.33	0.381	0.55	0.125
	Stand Up Peak	-0.12	0.765	—	—
	Sit Down Peak	0.17	0.668	—	—

3.5.5 Correlation of maximum joint excursions and concomitant in vivo contact forces

Maximum abduction showed effects on joint loads in level walking (r_s = 0.55) and stair descent (r_s = 0.52). The complete array of correlations between maximum joint excursions and concomitant in vivo contact forces is presented in table 25 below.

Table 25: Correlation of maximum joint excursions and concomitant in vivo contact forces. Max = Maximum. Ext = Extension. Flex = Flexion. Abd = Abduction. Add = Adduction. r_s = Spearman's rank correlation coefficient. p = p-value.

			Max Ext		**Max Flex**		**Max Abd**		**Max Add**	
			r_s	p	r_s	p	r_s	p	r_s	p
Joint Load	**Walking**	Max Ext	0.47	0.205	—	—	—	—	—	—
		Max Flex	—	—	-0.45	0.224	—	—	—	—
		Max Abd	—	—	—	—	0.55	0.125	—	—
		Max Add	—	—	—	—	—	—	0.10	0.798
	Stairs Up	Max Ext	-0.21	0.610	—	—	—	—	—	—
		Max Flex	—	—	0.12	0.779	—	—	—	—
		Max Abd	—	—	—	—	0.33	0.420	—	—
		Max Add	—	—	—	—	—	—	0.07	0.867
	Stairs Down	Max Ext	-0.32	0.406	—	—	—	—	—	—
		Max Flex	—	—	-0.03	0.932	—	—	—	—
		Max Abd	—	—	—	—	0.52	0.154	—	—
		Max Add	—	—	—	—	—	—	0.27	0.488
	Stand Up	Max Flex	—	—	-0.20	0.606	—	—	—	—
	Sit Down	Max Flex	—	—	0.02	0.966	—	—	—	—

3.6 Hypothesis II

Impact of hip abductor muscle status on hip abductor muscle activity

3.6.1 Correlation of muscle status and muscle activity at peak in vivo contact forces

Total Muscle Volume

The TMV of the GLmax showed effects on muscle activity at the 1st peak F_{res} in level walking (r_s = 0.53). Both the GLmed and TFL showed effects on muscle activity at the 1st peak F_{res} in stair ascent (r_s = -0.50 and 0.62, respectively), while the TFL also correlated with muscle activity at the 2nd peak F_{res} in stair descent and the peak F_{res} in sitting down (r_s = 0.60 and 0.52, respectively). The complete array of correlations between TMV and muscle activity at peak in vivo contact forces is presented in table 26 below.

Table 26: Correlation of total muscle volume and muscle activity at peak in vivo contact forces. TFL = Tensor fasciae latae muscle. 1st Peak = 1st peak resultant force. 2nd Peak = 2nd peak resultant force. Peak = Peak resultant force. r_s = Spearman's rank correlation coefficient. p = p-value.

		Total Muscle Volume					
		Gluteus Maximus		Gluteus Medius		TFL	
		r_s	p	r_s	p	r_s	p
Muscle Activity	Walking 1st Peak	0.53	0.139	-0.10	0.798	0.07	0.867
	Walking 2nd Peak	0.03	0.932	-0.47	0.205	0.19	0.651
	Stairs Up 1st Peak	0.12	0.779	-0.50	0.207	0.62	0.102
	Stairs Up 2nd Peak	0.33	0.420	0.33	0.420	-0.07	0.867
	Stairs Down 1st Peak	0.20	0.606	0.22	0.576	0.29	0.493
	Stairs Down 2nd Peak	-0.32	0.406	-0.20	0.606	0.60	0.120
	Stand Up Peak	-0.48	0.187	-0.15	0.700	0.33	0.420
	Sit Down Peak	-0.20	0.606	0.15	0.700	0.52	0.183

Fat Ratio

The FR of the GLmed showed an effect on muscle activity at the 1st peak F_{res} in level walking (r_s = -0.65). Another strong correlation was found with the TFL in sitting down (r_s = 0.69). The complete array of correlations between FR and muscle activity at peak in vivo contact forces is presented in table 27 below.

Table 27: Correlation of fat ratio and muscle activity at peak in vivo contact forces. TFL = Tensor fasciae latae muscle. 1st Peak = 1st peak resultant force. 2nd Peak = 2nd peak resultant force. Peak = Peak resultant force. r_s = Spearman's rank correlation coefficient. p = p-value.

		Fat Ratio					
		Gluteus Maximus		Gluteus Medius		TFL	
		r_s	p	r_s	p	r_s	p
Muscle Activity	Walking 1st Peak	-0.05	0.898	-0.65	0.058	-0.31	0.456
	Walking 2nd Peak	0.23	0.546	-0.18	0.637	0.38	0.352
	Stairs Up 1st Peak	-0.48	0.233	-0.38	0.352	0.31	0.456
	Stairs Up 2nd Peak	0.10	0.823	0.24	0.570	-0.02	0.955
	Stairs Down 1st Peak	0.08	0.831	-0.08	0.831	-0.26	0.531
	Stairs Down 2nd Peak	0.05	0.898	-0.45	0.224	-0.48	0.233
	Stand Up Peak	0.28	0.460	0.33	0.381	0.00	1.000
	Sit Down Peak	0.45	0.224	-0.12	0.765	0.69	0.058

3.6.2 Correlation of muscle status and the instant of peak muscle activity

Total Muscle Volume

The TMV of the GLmax and the IPMA of the GLmax were found to correlate significantly in stair ascent (r_s = 0.83**), in addition to showing effects in standing up (r_s = 0.63). The GLmed showed effects in stair ascent (r_s = -0.64), while the TFL was also shown to have an effect on its respective IPMA in stair ascent (r_s = -0.57). The complete array of correlations between TMV and the IPMA is presented in table 28 below.

Table 28: Correlation of total muscle volume and the instant of peak muscle activity. TFL = Tensor fasciae latae muscle. IPMA = Instant of peak muscle activity. r_s = Spearman's rank correlation coefficient. p = p-value. Significance level ** = 0.01.

		Total Muscle Volume					
		Gluteus Maximus		Gluteus Medius		TFL	
		r_s	p	r_s	p	r_s	p
IPMA	Walking	0.45	0.224	-0.40	0.286	0.10	0.823
	Stairs Up	0.83**	0.010	-0.64	0.086	-0.57	0.139
	Stairs Down	-0.13	0.732	-0.02	0.966	-0.43	0.289
	Stand Up	0.63	0.067	-0.33	0.381	0.36	0.385
	Sit Down	-0.45	0.224	-0.28	0.460	0.36	0.385

Fat Ratio

The FR of the GLmed and the IPMA of the GLmed correlated significantly in level walking (r_s = -0.67*) and standing up (r_s = -0.82**), while the TFL and its IPMA displayed a

significant correlation in stair ascent (r_s = -0.71*). The complete array of correlations between FR and the IPMA is presented in table 29 below.

Table 29: Correlation of fat ratio and the instant of peak muscle activity. TFL = Tensor fasciae latae muscle. IPMA = Instant of peak muscle activity. r_s = Spearman's rank correlation coefficient. p = p-value. Significance level * = 0.05, ** = 0.01.

		Fat Ratio					
		Gluteus Maximus		Gluteus Medius		TFL	
		r_s	p	r_s	p	r_s	p
IPMA	Walking	0.07	0.865	-0.67*	0.050	-0.40	0.320
	Stairs Up	0.02	0.955	-0.31	0.456	-0.71*	0.047
	Stairs Down	0.28	0.460	0.10	0.798	-0.48	0.233
	Stand Up	-0.47	0.205	-0.82**	0.007	0.29	0.493
	Sit Down	0.05	0.898	0.12	0.765	-0.24	0.570

Impact of hip abductor muscle activity on in vivo hip joint contact forces

3.6.3 Correlation of muscle activity and peak in vivo contact forces

GLmax activity was found to have a significant effect on the 1st peak F_{res} in level walking (r_s = 0.75*), while additionally having an effect on the 1st peak F_{res} in stair descent (r_s = 0.60). Moreover, GLmed activity was shown to have an effect on the 1st peak F_{res} in stair ascent (r_s = 0.64). The complete array of correlations between muscle activity and peak in vivo contact forces is presented in table 30 below.

Table 30: Correlation of muscle activity and peak in vivo contact forces. TFL = Tensor fasciae latae muscle. 1st Peak = 1st peak resultant force. 2nd Peak = 2nd peak resultant force. Peak = Peak resultant force. r_s = Spearman's rank correlation coefficient. p = p-value. Significance level * = 0.05.

		Muscle Activity					
		Gluteus Maximus		Gluteus Medius		TFL	
		r_s	p	r_s	p	r_s	p
Joint Load	Walking 1st Peak	0.75*	0.020	0.05	0.898	-0.02	0.955
	Walking 2nd Peak	0.33	0.381	-0.33	0.381	0.12	0.779
	Stairs Up 1st Peak	-0.14	0.736	0.64	0.086	-0.26	0.531
	Stairs Up 2nd Peak	0.38	0.352	-0.17	0.693	-0.17	-0.167
	Stairs Down 1st Peak	0.60	0.088	0.40	0.286	-0.12	0.779
	Stairs Down 2nd Peak	0.22	0.576	-0.03	0.932	-0.19	0.651
	Stand Up Peak	0.35	0.356	0.00	1.000	-0.14	0.736
	Sit Down Peak	0.25	0.516	-0.07	0.865	-0.31	0.456

3.6.4 Correlation of the instant of peak muscle activity and concomitant in vivo contact forces

The IPMA of the GLmax was found to correlate significantly with concomitant contact forces in level walking (r_s = 0.77*), standing up (r_s = -0.85**), and sitting down (r_s = 0.87**). The IPMA of the GLmed correlated significantly with concurrent joint loads in sitting down (r_s = 0.80**). Finally, the IPMA of the TFL correlated significantly with contact forces in level walking (r_s = 0.83**), while also showing effects in stair ascent (r_s = -0.55). The complete array of correlations between the IPMA and concomitant in vivo contact forces is presented in table 31 below.

Table 31: Correlation of the instant of peak muscle activity and concomitant in vivo contact forces. TFL = Tensor fasciae latae muscle. IPMA = Instant of peak muscle activity. r_s = Spearman's rank correlation coefficient. p = p-value. Significance level * = 0.05, ** = 0.01.

		IPMA					
		Gluteus Maximus		Gluteus Medius		TFL	
		r_s	p	r_s	p	r_s	p
Joint Load	Walking	0.77*	0.016	-0.27	0.488	0.83**	0.010
	Stairs Up	-0.40	0.320	0.07	0.867	-0.55	0.160
	Stairs Down	0.35	0.356	0.45	0.224	-0.26	0.531
	Stand Up	-0.85**	0.004	-0.37	0.332	0.14	0.736
	Sit Down	0.87**	0.002	0.80**	0.010	0.43	0.289

3.7 Summary of the correlation analysis

3.7.1 Hypothesis I

Impact of hip abductor muscle status on hip joint kinematics

The *correlation of total muscle volume and kinematics at peak in vivo contact forces* showed larger TMV of the GLmed to be significantly correlated with frontal plane kinematics as there is an increasing shift from lower hip adduction towards abduction in stair descent at the 2nd peak F_{res} (r_s = -0.68*, p = 0.042). No significant correlation was found between FR and kinematics at peak in vivo contact forces.

The *correlation of muscle status and maximum joint excursions* showed significant effects in both the TMV and FR. Larger TMV of the GLmax correlates significantly with higher maximum hip abduction in stair ascent (r_s = -0.74*, 0.037). Additionally, higher FR of the

GLmax, correlates significantly with higher maximum hip adduction in level walking (r_s = -0.75*, p = 0.02).

The *correlation of muscle status and dynamic range of motion* also showed significant effects in both TMV and FR. Larger TMV of the GLmax is significantly correlated with lower sagittal plane hip ROM in stair ascent (r_s = -0.76*, p = 0.028) and descent (r_s = -0.67*, p = 0.05) but also with higher frontal plane ROM in stair descent (r_s = 0.87**, p = 0.002). Larger TMV of the TFL was found to significantly correlate with higher sagittal plane ROM in standing up (r_s = 0.67*, p = 0.05). Higher FR of the TFL was found to significantly correlate with higher frontal plane hip ROM in stair ascent (r_s = 0.81*, p = 0.015).

All principal findings regarding the impact of muscle status on kinematics are summarized in table 32 below.

Table 32: Impact of muscle status on kinematics. Results in regular font indicate correlation coefficients with a strong effect size ($r_s \geq 0.5$). Results in bold indicate statistical significance. ↑ = Larger/higher. ↓ = Smaller. TMV = Total muscle volume. FR = Fat ratio. GLmax = Gluteus maximus muscle. GLmed = Gluteus medius muscle. TFL = Tensor fasciae latae muscle. ROM = Range of motion. Flex = Flexion. Abd = Abduction. Add = Adduction.

	1st Peak	2nd Peak	Maximum	ROM
↑ TMV GLmax	↓ Flex – Stairs Up	—	**↑ Abd – Stairs Up**	↑ Frontal – Walking
	—	—	↑ Abd – Stairs Down	**↓ Sagittal – Stairs Up**
	—	—	—	↑ Frontal – Stairs Up
	—	—	—	**↓ Sagittal – Stairs Down**
	—	—	—	**↑ Frontal – Stairs Down**
↑ TMV GLmed	—	—	**↑ Abd – Stairs Down**	—
	—	—	**↓ Add – Stairs Down**	—
↑ TMV TFL	↓ Flex – Walking	—	↑ Abd – Stairs Up	**↑ Sagittal – Stand Up**
	↓ Add – Walking	—	—	↑ Sagittal – Sit Down
	↓ Add – Stairs Up	—	—	—
↑ FR GLmax	↓ Add – Walking	—	**↓ Add – Walking**	—
↑ FR GLmed	↓ Flex – Walking	—	↓ Add – Walking	↓ Frontal – Walking
	↓ Flex – Stairs Up	—	—	—
↑ FR TFL	↓ Flex – Sit Down	—	↑ Abd – Walking	↑ Frontal – Walking
	—	—	↑ Add – Stairs Up	**↑ Frontal – Stairs Up**

Impact of hip joint kinematics on in vivo hip joint contact forces

The *correlation of kinematics and peak in vivo contact forces* showed 1st peak joint loads in level walking to rise significantly with increasing hip flexion (r_s = 0.72*, p = 0.03). At the 1st peak F_{res} in stair ascent, higher hip adduction was found to significantly correlate with higher contact forces (r_s = 0.71*, p = 0.047).

All principal findings regarding the impact of kinematics on in vivo joint loads are summarized in table 33 below.

Table 33: Impact of kinematics on in vivo contact forces. Results in regular font indicate correlation coefficients with a strong effect size ($r_s \geq 0.5$). Results in bold indicate statistical significance. ↑ = Higher. ↓ = Lower. F_{res} = Resultant force. Flex = Flexion. Abd = Abduction. Add = Adduction.

	1st Peak F_{res}	**2nd Peak F_{res}**	**F_{res} at Maximum Abd**
↑ Flex	**↑ Walking**	—	—
↑ Abd	—	—	↓ Walking
	—	—	↓ Stairs Down
↑ Add	**↑ Stairs Up**	↑ Stairs Down	—

3.7.2 Hypothesis II

Impact of hip abductor muscle status on hip abductor muscle activity

The *correlation of muscle status and the instant of peak muscle activity* showed larger TMV of the GLmax to correlate significantly with a later IPMA in stair ascent (r_s = 0.83**, p = 0.01). Higher FR of the GLmed was shown to significantly correlate with an earlier IPMA in level walking (r_s = -0.67*, p = 0.05) and standing up (r_s = -0.82**, p = 0.007). Similarly, higher FR of the TFL was found to correlate significantly with an earlier IPMA in stair ascent (r_s = -0.71*, p = 0.047).

All principal findings regarding the impact of muscle status on muscle activity are summarized in table 34 below.

Table 34: Impact of muscle status on muscle activity. Results in regular font indicate correlation coefficients with a strong effect size ($r_s \geq 0.5$). Results in bold indicate statistical significance. ↑ = Higher/later. ↓ = Lower/earlier. TMV = Total muscle volume. FR = Fat ratio. GLmax = Gluteus maximus muscle. GLmed = Gluteus medius muscle. TFL = Tensor fasciae latae muscle. IPMA = Instant of peak muscle activity.

	1st Peak Muscle Activity	2nd Peak Muscle Activity	IPMA
↑ TMV GLmax	↑ Walking	—	**↑ Stairs Up**
	—	—	↑ Stand Up
↑ TMV GLmed	↓ Stairs Up	—	↓ Stairs Up
↑ TMV TFL	↑ Stairs Up	↑ Stairs Down	↓ Stairs Up
	↑ Sit Down	—	—
↑ FR GLmed	↓ Walking	—	**↓ Walking**
	—	—	**↓ Stand Up**
↑ FR TFL	↑ Sit Down	—	**↓ Stairs Up**

Impact of hip abductor muscle activity on in vivo hip joint contact forces

The *correlation of muscle activity and peak in vivo contact forces* showed increased GLmax activity at the 1st peak F_{res} in level walking to correlate significantly with higher joint loads ($r_s = 0.75^*$, $p = 0.02$).

The *correlation of the instant of peak muscle activity and concomitant in vivo contact forces* showed a later IPMA of the GLmax to significantly correlate with higher contact forces in level walking ($r_s = 0.77^*$, $p = 0.016$) and sitting down ($r_s = 0.87^{**}$, $p = 0.002$), while also significantly correlating with lower joint loads in standing up ($r_s = -0.85^{**}$, $p = 0.004$). A later IPMA of the GLmed is significantly correlated with higher joint loads in sitting down ($r_s = 0.80^{**}$, $p = 0.01$), and a later IPMA of the TFL correlates significantly with higher contact forces in level walking ($r_s = 0.83^{**}$, $p = 0.01$).

All principal findings regarding the impact of muscle activity on in vivo joint loads are summarized in table 35 below.

Table 35: Impact of muscle activity on in vivo contact forces. Results in regular font indicate correlation coefficients with a strong effect size ($r_s \geq 0.5$). Results in bold indicate statistical significance. ↑ = Higher/later. ↓ = Lower. TMV = Total muscle volume. FR = Fat ratio. GLmax = Gluteus maximus muscle. GLmed = Gluteus medius muscle. TFL = Tensor fasciae latae muscle. IPMA = Instant of peak muscle activity. F_{res} = Resultant force.

	1st Peak F_{res}	**F_{res} at IPMA**
↑ Muscle Activity GLmax	**↑ Walking**	—
	↑ Stairs Down	—
↑ Muscle Activity GLmed	↑ Stairs Up	—
↑ IPMA GLmax	—	**↑ Walking**
	—	**↓ Stand Up**
	—	**↑ Sit Down**
↑ IPMA GLmed	—	**↑ Sit Down**
↑ IPMA TFL	—	**↑ Walking**
	—	↓ Stairs Up

4. Discussion

4.1 Patients

Compared to previous reports of primary THA patients, the average age of subjects in this investigation (61.4 ±6.4 years) was considerably lower [83,118,215]. Because patients are projected to be progressively younger and more physically active [216-218] at the time of their primary THA, this study intended to examine a preferably younger generation of patients who were following a comparatively active lifestyle. Both the mean height of 173 ±7 cm and mean body mass index (BMI) of 30.3 ±4.6 were greater than in other studies [83,118,215]. Along with age, BMI may have had an effect on the fat content of the hip abductor muscles [197], potentially also affecting hip joint kinematics and muscle activity [219,220]. In order to preserve the physical integrity of both the hip joints and their periarticular musculature, healthy individuals as well as hip OA and THA patients are encouraged to maintain a normal BMI and remain non-obese, as a higher BMI is linked with increases of GLmed and GLmax activity [219], along with increased fatty degeneration of hip muscles [197]. In addition to this, Lerner et al. observed altered gait patterns in asymptomatic obese individuals, implying that obesity is linked to hip abductor muscle weakness [220]. An increased BMI was further found to significantly correlate with higher hip joint contact forces [221] and a higher susceptibility to hip OA [222]. Besides, different kinetic, kinematic, and muscle activation patterns are reported to exist for women and men [219,223-230]. Yet, this investigation did not specifically test for gender differences among parameters. The variable range of test dates of 35 to 64 months after the patients in this study underwent THA is unlikely to have decisively contributed to inter-individual differences in the recordings. A systematic review found postoperative kinematic measurements comparing patients with healthy individuals to not have been influenced much by the duration of postoperative follow-up intervals [165]. Gait kinematics between the ages of 54 through 79 years seem to be homogenous, and a decline of essential kinematic parameters does not manifest until after the age of 80 years [167]. Moreover, Ng et al. found that, although the HHS improves significantly following THA, the score levels off after 18 months [231].

4.2 Kinematics

The literature offers a rising number of studies investigating kinematics in patients suffering from pathologies of the musculoskeletal system such as OA, and the short-term

condition of symptomatic individuals who underwent THA. Nevertheless, there is still only very limited postoperative evidence of long-term gait and gait-related, as well as other everyday life activities' kinematic patterns to compare the results of this investigation to. This study did not include healthy controls and, therefore, yields the results of a cross-sectional retrospective analysis of a unique cohort of nine patients with instrumented implants. In spite of that, the patients in this investigation attained values that are similar to those of other studies assessing long-term kinematic outcomes of THA patients and working with a comparable methodology.

4.2.1 Level walking

Beaulieu et al. analyzed lower limb gait biomechanics of healthy subjects and THA patients 10.6 months after surgery and concluded that surgical intervention does not restore normal kinematic conditions [118]. Our THA patients reached similar mean peak extension, flexion, and adduction as the patients of Beaulieu et al. [118] Insofar as a comparative statistical analysis between THA patients and healthy individuals is concerned, almost all of the abovementioned mean values found by Beaulieu et al. are significantly lower than in healthy controls [118]. Because our patients' results are similar to those of the patients of Beaulieu et al., the performance of our patients may possibly also be classified as poorer than the performance of healthy individuals.

A longitudinal study conducted by Agostini et al. assessed kinematic parameters 3, 6, and 12 months after THA, showing continuous improvement of the mean dynamic sagittal ROM [55]. Notably, the produced hip joint kinematic parameters are considerably lower than the values found by Beaulieu et al. and in our study [55,118]. This, however, might be explained by Beaulieu et al. and our research group applying analogous methods for kinematic assessment, whereas Agostini et al. did not utilize a system with reflective body markers but a hip joint goniometer [55].

Bennett et al. obtained spatiotemporal and kinematic specifications of age-differentiated patient groups ten years following THA and compared their results with the outcomes of a healthy control group [167]. They found that mean dynamic sagittal ROM is reduced across all age groups and inferred that pathomechanical gait patterns persist in the long run [167]. Moreover, none of their five patient groups managed to perform an actual mean peak extension, which is in marked contrast to their controls [167] and our THA patients,

who all reached an actual hip extension throughout walking trials. Bennett et al. also found decreased self-selected velocity and step and stride lengths in THA patients compared to their control group, which might come as a consequence of insufficient hip extension ability [167]. Our patients produced a similar self-selected walking speed, suggesting that their hip extension and, for that matter, sagittal ROM might also be substandard in comparison with healthy individuals of other studies and rather resemble those studies' respective patient groups' kinematic measurements.

In their longitudinal study matching THA patients' pre- and postoperative gait measurements against those of a healthy control group, Foucher et al. propose that, although gait variables of THA patients improved 12 months after surgery, postoperative dynamic sagittal ROM, as well as flexion and abduction moments correlate significantly with their respective preoperatively measured values, indicating gait adaptations to have formed before surgery at least to some extent [166]. The authors do not mention the exact numerical results, but their study's postoperative measurements of dynamic sagittal ROM recordings also yielded lower values than those recorded in healthy individuals.

A study by Kiss et al. which looks into the differences in functional outcome of a DLA and an ALA compared with healthy individuals revealed superior results when opting for an ALA [232]. ALA patients showed even larger ROM than healthy controls [232]. Peculiarly, our patients' measurements do not even roughly match those of the DLA patients in that study [232], despite the circumstance that they were also operated employing a DLA. As is the case with the assessment of the functional outcome following THA in the literature, however, methodological approaches vary appreciably between publications, which means that respective results should be interpreted carefully.

4.2.2 Ascending stairs

While studies dealing with the long-term functional outcome of hip arthroplasties using gait analysis as part of their methodology are already scarce, investigations examining patients' stair ascent and descent are yet much rarer.

A study by Queen et al. compared the hip joint kinematics of healthy controls to those of patients who received either THA or hip resurfacing arthroplasty (HRA) at an average of 18 months prior to enrollment in the study [233]. The authors found no difference in sagittal

ROM of THA patients and healthy controls, while the ROM of HRA patients was slightly lower [233]. Our patients, in turn, produced a larger sagittal ROM than all their groups. A possible explanation of this could be that the stairs in our trials were higher, which may have required our patients to produce a greater hip flexion, explaining the rather large difference in sagittal ROM. To that effect, the test subjects of the study by Queen et al. showed lower peak flexion angles than our patients [233]. Remarkably, both THA and HRA patients ascended stairs with significantly higher hip flexion than controls, which is in line with the general observation that hip OA and hip arthroplasty patients are likely to present with a flexion contracture or extension deficit, respectively [118,155,165,234-236]. Our patients' mean peak extension is similar to that found in THA and HRA patients of the aforementioned study [233]. Strikingly, the group of healthy individuals had a significantly larger mean extension [233], again underlining the common extension deficit in hip arthroplasty patients. The commensurability of our results pertaining to extension angles in stair ascent is given due to the circumstance that hip extension angles are less influenced by step height than hip flexion angles. Accordingly, our findings may be deemed compatible with those discussed above. Interestingly, our patients' frontal plane kinematics during stair ascent differ substantially from those of the above subjects [233], accentuating a considerably larger frontal ROM. This is unlikely a result of higher kinematic variability in the frontal plane between our patients and those of Queen et al. Instead, the results are most likely incommensurable owing to contrasting methods in kinematic assessment. Depending on the model used for the computerized transfer of the markers applied to anatomical bony landmarks, individual studies' results might differ significantly [205].

A handful of investigations which do not provide actual measurements but compared hip joint kinematics of THA patients during stair ascent to those of healthy controls, also found no significant differences in sagittal ROM between the groups [237-239]. One study by Lamontagne et al. found larger peak extension angles in patients, as well as no disparities in flexion in both groups [240]. Notably, these results are nearly inverse to the findings of Queen et al. [233] Peak abduction among most tested groups is comparable [233,237,238,240], whereas Lamontagne et al. found lower frontal ROM and peak adduction in THA patients when compared to asymptomatic individuals [237,238].

4.2.3 Descending stairs

Our patients' mean peak extension is similar to that found by Queen et al. in THA and HRA patients, while their control group yielded a higher mean peak extension [233]. The healthy individuals in their study produced a peak mean flexion which is not only lower than that of their THA and HRA patients [233] but also the peak mean flexion found in our patients. Hence, a flexion contraction or extension deficit might be the cause of sagittal plane differences in patients and controls, precisely as found in level walking and stair ascent.

In keeping with the abovementioned possible explanation for divergences in frontal plane hip joint kinematic measurements among different studies, our patients' mean peak abduction and adduction disagree with the measurements of Queen et al. [233] Still, the literature agrees in that there is no noteworthy divergence in frontal plane ROM and peak abduction and adduction between patients and healthy individuals [233,237,238,240].

4.2.4 Standing up

The literature on hip joint kinematics of THA patients executing standing and sitting tasks is rarest when compared with the availability of the literature on the other ADLs included in this investigation. On the grounds that only one study that provided numerical values for their THA patients' kinematic recordings was identified, comparing the actual measurements in this investigation with other publications' results was not feasible. Because pre- and postoperative kinematics have shown to be correlated significantly [166], however, referring to the measurements of patients suffering from hip OA serves as a necessary and also viable alternative. Eitzen et al. provide an account of patients suffering from mild-to-moderate hip OA in contrast with healthy individuals rising from a chair [241]. Despite using the same seat height as was done in the trials of our investigation and the fact that Eitzen et al. also did not install armrests, their hip OA patients and controls exhibited respective sagittal ranges of motion about 30° lower than our THA patients [241]. Naturally, hip sagittal joint excursion in standing up is limited to a very low hip extension, while hip flexion is predominant in the process of rising from a chair.

One explanation for the relatively large discrepancies between our findings and those of Eitzen et al. is methodological variability. In any case, a comparison with other studies' measurements on THA patients' hip joint excursions has to be conducted with caution. A

cross-investigational association of kinematic measurements in standing up and sitting down is challenging not only because of potentially different methodologies applied for the recording and procession of kinematics (e.g., differing placement of markers, diverging location of the hip joint center) [155,205]. It is also challenging in that comparing measurements across studies might lead to false conclusions stemming from possibly different seat heights used or variant anthropometric characteristics among the subjects, altered foot positioning at the initiation of movement, and an implementation of auxiliary agents such as the use of armrests [242]. Regardless of the lack of a healthy control group or studies implementing analogous methodologies, the key outcomes of the investigations considered give some indication of pathomechanical hip joint kinematics.

For example, Shiomoto et al. investigated pre- and postoperative hip joint kinematics by use of continuous radiographic imaging and a three-dimensional-to-two-dimensional model-to-image registration technique, observing an improvement of hip flexion from before THA to 62 months postoperatively [243]. But despite a significant improvement, THA patients' sagittal plane kinematics did not reach the hip flexion attained by healthy individuals [243]. Both the results of their THA patients and their control group differ decidedly from our THA patients' measurements, who were measured with a larger mean hip flexion. This may likely be attributed to having utilized different methods for kinematic assessment.

A study by Lamontagne et al. comparing lower-limb joint kinematics of THA patients at an average of 317 days after THA to those of a healthy control group found sagittal ROM to be lower in THA patients than in healthy individuals, perhaps as a consequence of the lower peak flexion also detected [244]. A study assessing the influence of the sagittal hip joint angle at seat-off (shortly before rising from a chair) on hip joint contact forces found joint loads to rise almost linearly as hip flexion increases [245]. Given that pre- and postoperative kinematics were shown to correlate significantly [166] and that patients with mild-to-moderate hip OA display lower hip flexion when compared to controls [246], it follows that the decrease in hip flexion [244] could be a residual movement pattern which patients established prior to THA as a strategy to reduce mechanical demands and compensate muscle weakness [118,247]. It may also be a mechanism that circumstantiated preoperatively in order to scale down joint loads on the operated side in an attempt to reduce pain [118].

4.2.5 Sitting down

Conforming with standing up, Lamontagne et al. found sitting down to be associated with lower sagittal ROM and peak flexion in THA patients as opposed to asymptomatic controls [244]. Akin to the results found in standing up [244], it could be speculated that the lower flexion in sitting down might also be a compensatory method adopted prior to THA to deal with muscle weakness or a response aimed at minimizing hip joint contact forces and avoiding pain [118,247].

4.2.6 Summary

Across various studies, nearly all postoperative groups of patients fell short of meeting healthy controls' hip dynamic sagittal plane ROM standards in level walking due to, most commonly, either lower peak hip extension or, less frequently, flexion, or both, confirming the notion in the literature that postoperative kinematics do not return to normal levels [55,118,165-167,232,248]. Measurements in stair negotiation yielded patients mostly matching sagittal and frontal plane ROM levels of control groups in the literature, yet the former appears to happen at the cost of altered peak extension and/or flexion [233,237,238,240]. Despite the literature on hip joint kinematics following THA in standing and sitting tasks being scarce [165], investigations agree in that they found lower sagittal plane ROM and peak flexion [243,244]. Increased flexion in gait and stair negotiation is likely a manifestation of a flexion deformity or an extension deficit [118,155,165,234-236], respectively. Lower flexion in standing and sitting tasks, in turn, may have emerged as a strategy to reduce joint loads [118,245].

To a great extent, our results in level walking and stair negotiation compare well with the picture in the literature, presumably because we assessed the hip joint kinematic measurements of other investigations that used analogous methods. However, depending on the employed methodology and possibly even the task under investigation, numerical results for kinematic recordings vary markedly across investigations [249,250], as was found to be the case in standing and sitting tasks. A possible solution to this issue could be to take into consideration the apparent relative performance gap between any THA patients and healthy controls of one study and then to cautiously weigh the results of studies against each other. In spite of the above, this approach can only serve as a rough approximation to the issue of methodological discordance among publications. Providing for comparative analyses in kinematic assessments should be a core theme of

the scientific community so that discrepancies in the interpretation of different investigations' results can be avoided in the future. On the whole, our findings blend in well with the existing literature.

4.3 Muscle activity

Regardless of the ongoing sophistication of computerized simulations emulating muscle activity through MSMs, in vivo electrophysiological recordings most genuinely reflect patients' actual muscle activity materialized in ADLs [174,192,196]. Nevertheless, the literature on hip abductor muscle activity in ADLs following THA is scarce, and the prevailing scarcity of investigations with analogous normalization methods of dynamic movements further complicates examining studies in contrast with one another [251]. The context of carrying out ADLs with dynamic and submaximal muscle activity plays a crucial role as this measurement setup determines the inferences that can be drawn about how impairments of the muscles surrounding the hip joint may influence patients' daily performances.

As this investigation did not include a reference group or sEMG recordings from different time intervals, the sEMG data of the GLmax, GLmed, and TFL are analyzed descriptively and are also compared with the findings of other studies which examined the same muscles. For the purpose of drawing comparisons to these investigations, the discussion of hip abductor muscle activity in this chapter is limited to parameters of qualitative evidence such as potential irregularities regarding muscle activation timing and pattern shapes within the five ADLs for which our patients were measured. Overall, gait is most likely the best researched ADL [174,252], which is also why the emphasis in the majority of research on muscle activity in day-to-day activities is on level walking. It would be desirable for other ADLs such as stair negotiation and standing and sitting tasks to receive more attention in future studies.

Our sEMG results yielded high inter-individual variability across all muscles and ADLs, which might partly be due to the relatively small sample size of nine patients. On the other hand, the literature addresses variability as a common observation in EMG recordings [253]. Agostini et al. provide an extensive account on normative EMG data during gait, noting not only inter-individual variability of EMG signals but also pronounced intra-individual variability in muscle activation timing [253]. Walking speed was found to have an

effect on the amplitude of electrophysiological recordings [254]. Decreases in gait velocity were shown to increase the onset timing and sequences of muscle activation [255,256]. Slower walking speed also appears to raise inter-individual variability in muscle activation, which could be explained by a longer length of stay within each of the different gait phases and, consequently, an increase of individual muscles' contribution to locomotion [255]. Faster walking speed, in turn, appears to influence muscle recruitment and produce some kind of automatic locomotion pattern, generating more uniform and shorter sequences of muscle activation [255]. This framework further complicates comparing EMG recordings among studies investigating healthy and pathological populations, and normalizing gait cycles by the various subphases of gait could counterbalance potential differences in muscle activation times transpiring due to the aforementioned context [255].

As mentioned earlier, our THA patients' walking speed compares well with the substandard gait velocity of other investigations' THA patients. Thus, the inter-individual variability in our sEMG recordings is not an exception when examined against the background of the literature. Another important determinant playing into the formation of variable EMG signals is individual joint position [174,247,257,258]. Perry brings up that the altered functional anatomy of the musculoskeletal system affects muscle torque [174]. When the respective individual joint angle changes, the position of bony structures and muscle-tendon components also changes, which in turn alters the extent of muscle effort needed to realize a certain movement. In other words, this changes the emerging muscle force accordingly [174]. Murray et al. point out the probability of visually observable variability in pelvic motion during gait to be linked directly with variability in hip abductor muscle activity [254]. Given that our patients also exhibited large inter-individual differences in kinematic measurements, the circumstance of joint position having an effect on EMG signals may further explain the considerable variability of our sEMG recordings.

4.3.1 Level walking

In level walking, the main burst of GLmax activity was recorded beginning shortly before HS, which is in conformity with this muscle's function as a main extensor of the hip joint [169,171]. The initial activation of the GLmax reflects the recruitment of that muscle and an associated hip extensor moment in the late swing phase in preparation for making contact with the ground (HS) and the ensuing loading response [171,252]. Throughout load acceptance between HS and CTO [151], GLmax activity rose until peaking in all patients

shortly before CTO and the 1st peak F_{res}, which is when the GLmax exerts the most force required for the forward propulsion of the body [169]. After the IPMA, GLmax activity swiftly dropped, although another main burst of activity was registered shortly after the 2nd peak F_{res} in five patients. Around the 2nd peak F_{res}, the ipsilateral hip and knee, respectively, were in an extended position. Trepczynski et al. found that the knee 2nd peak F_{res} is accompanied by a knee extension moment of the quadriceps femoris muscle that is countered by a knee flexion moment mainly generated by the gastrocnemius muscles [259]. Probably, a similar constellation is applicable for the hip joint, in which a co-contraction of hip extensors, such as the GLmax and the hamstrings, and hip flexors, like the iliopsoas and rectus femoris muscles, contributes substantially to the emergence of the 2nd peak F_{res}. For this speculation to be validated, however, the collected in vivo data would have to be matched with in silico modelling using the MSM applied by Trepczynski et al. [259] Still, as muscle activity contributes as much as 75% to knee joint contact forces [259], assuming a similar situation for the hip joint does not seem unreasonable.

In accordance with the results of Agostini et al., who assessed the timing of muscle activity in THA patients [55], our subjects showed two main bursts of GLmed activity between HS and CTO and between TO and mid swing. These findings also comply with Gottschalk et al., who showed the GLmed to be active during early gait in healthy adults [158], and even more so with Shiavi et al., who provide EMG profiles of asymptomatic test subjects additionally exhibiting GLmed activity during the swing phase of gait [255]. Like Agostini et al. [55], we found several patients to generate a third activity burst of the GLmed between mid to late stance up until the swing phase. Although a number of healthy controls were also recorded producing a third GLmed activity burst, Agostini et al. report this third burst of GLmed activity to appear during the swing phase in THA patients, as opposed to appearing during late stance and pre swing in the control group [55]. This delayed activity burst was reproduced by THA patients on the contralateral side at the latest follow-up of 12 months postoperatively, which the authors interpret as a compensatory strategy to offset gait asymmetry [55]. In general, Agostini et al. found THA patients to display prolonged bursts of activity relative to a healthy control group, especially early (3 months postoperatively) after replacement surgery [55]. Because a control group or data from earlier time intervals were not included in this investigation, such an observation could not be made. In spite of this, the duration of GLmed activity bursts of our THA patients resonates with both the above investigation's control and long-

term follow-up (12 months postoperatively) THA group [55], confirming our test subjects' longstanding rehabilitation from surgical intervention.

As is the case with the IPMA of the GLmax, peak muscle activation times of the TFL were recorded shortly before CTO and the 1st peak F_{res}, with another activity burst occurring between late stance and the swing phase in half of the recorded data. However, there is considerable variability among our patients in TFL activity pattern shapes. The results of a study on the clinical and functional outcome of patients who received a thrust plate prosthesis show a TFL activation pattern with two activity bursts on the operated side and three activity bursts on the non-operated side and in healthy controls [260]. In the same investigation [260], the TFL on the operated side was recorded with prolonged activation timing that is also distinctly visible in four of our THA patients. Hence, half of our patients were recorded generating a similar prolonged biphasic pattern as found in the study mentioned above [260], while the other half was recorded analogously producing the first yet shortened activity burst, with an additional and similarly short burst of TFL activity between CHS and TO. Two of our subjects only generated the first activity burst, however, in conjunction with the GLmax and GLmed. It is unlikely that this simultaneous occurrence of peak muscle activity was due to crosstalk as it was not repeated in any of the trials investigating other ADLs. Nonetheless, the first TFL activation is present in all of our patients' recordings, which is a finding in line with the literature [260-262]. Interestingly, three of our patients exhibited constant firing of the TFL. This observation is associated with a prolonged stance phase [56], the latter being indicative of lower gait velocity [255]. Then again, Shrader et al. do not report any differences in TFL activity between pre- and postoperative THA and RHA groups, as well as healthy controls [263].

In their longitudinal study on the functional recovery of THA patients, Long et al. show that any irregular upper and lower GLmax, GLmed, and TFL activity recorded preoperatively had returned to normal 12 months after surgery, along with postsurgically rehabilitated spatiotemporal gait characteristics [56]. A study on the functional recovery of THA and RHA patients 3 months after surgery shows there are deficits relative to healthy controls in that, preoperatively, premature and out-of-phase GLmax and GLmed activity were recorded [263]. Postoperatively, these muscles' activity assumed typical phasic patterns, although activation timing was slightly extended in the THA group [263]. In their case study of a 60-year-old male patient who underwent THA by means of a DAA, Chopra

et al. describe preoperative hyperactivity of the GLmax, GLmed, and TFL on the operated side that ceased one year postoperatively but was still displayed by the non-operated side's muscles, which points towards muscle weakness and confirms the simultaneously recorded compensatory movement patterns electrophysiologically [261].

Considering our patients' mean time interval of 51 months since having undergone THA and that even the shortest postoperative duration in our cohort is already 35 months, it seems likely that some of our patients may have generated normal or close to normal activation sequences of some muscles, while others were affected by increased and prolonged muscle activation. Steens et al., on the other hand, offer a different account in that they observed increased GLmax, GLmed, as well as TFL amplitudes and prolonged activation timing extending into mid stance 2.1 years after surgery (the follow-up range was nine months to five years postoperatively) [260]. The authors attribute their findings to these muscles' support of the hip in avoiding Trendelenburg gait [260]. Our data show five patients with such an extended GLmax activity, six patients with a lengthy GLmed activity, and four patients with a continued TFL activity. The literature establishes the GLmed and TFL as the main hip abductors [151,152,158,168,173]. The upper part of the GLmax was also shown to function much like an abductor [170,171], which might explain their prolonged activity well into the stance phase in some of our patients.

4.3.2 Ascending stairs

A completely new picture emerged in stair ascent, as all muscles produced markedly extended temporal patterns compared with level walking. This observation complies with Chopra et al., who remark muscle activity in stair activities to be higher than in gait owing to an increased hip ROM, which in turn puts increased demands on muscle force in order to successfully accomplish stair negotiation [261]. Lyons et al. also found increased upper and lower GLmax, GLmed, and TFL activity during stair ascent in comparison with level walking [171], while Shrader et al. report out-of-phase GLmax activity in THA patients [263]. Stair ascent is a more challenging task than level walking in that it places even higher stress on maintaining pelvic balance [171,263].

Of the eight patients whose electrophysiological activity was measured (H7R is missing due to a recording error), six patients exhibited extended biphasic activation patterns of the GLmax and five patients of the GLmed. This biphasic pattern consisted of two activity

bursts succeeding each other seamlessly: the first burst, which also included the IPMA, surfaced between late swing and CTO, and the second burst appeared no earlier than mid stance and no later than CHS. The first activity burst of the GLmax reflects the recruitment of its lower fibers for the initiation of contact of the ipsilateral limb with the step ahead and the subsequent hip extension for the elevation of the body. The second GLmax activity burst had a noticeably lower amplitude, likely representing the stabilization of the extended limb against the swing of the contralateral limb towards the next step, along with the upper fibers' contribution to pelvic balance in the frontal plane [171]. Presumably because the GLmed is predominantly active during loading response [171,264] and as the GLmed consists of up to three functionally distinguishable parts of which the posterior part extends the hip [265], its sEMG signal took on a similar pattern as that of the GLmax. Despite our inability of assessing the different segments of muscles, our patients' muscle activity patterns match the findings of Lyons et al., who also recorded initial upper and lower GLmax, as well as GLmed activity beginning before HS and stretching into mid stance [171]. Lin et al. recorded muscle activity in healthy young adults and found GLmax activity in general agreement with our recordings and the literature [266]. Dwyer et al. investigated muscle activity in end-stage OA patients and healthy controls, revealing increased GLmed activity amplitudes in OA patients carrying out stepping tasks [184]. The authors propose this observation to be suggestive of pathological changes of muscular anatomy and ensuing muscle weakness [184], which is a theory that is corroborated by our findings.

Peculiarly, two of our patients with monophasic and otherwise only relatively weak GLmed activity patterns displayed almost mirror-inverted GLmed activity sequences with their respective IPMA taking place either around late swing (H9L) or around TO (H6R). In healthy individuals, the GLmed was shown to be inactive during the swing phase [264]. Our patients' irregular and out-of-phase muscle recruitment may have originated prematurely to counter muscle weakness and ensure hip frontal plane stabilization in the pre and early swing phases. Interestingly, the IPMA of the GLmax in H9L also fell in the time period of early swing, which possibly either constitutes an isometric contraction and concomitant extensor moment of the GLmax [266] or an excessive response of the upper fibers of the GLmax acting in hip frontal plane stabilization [171].

Analogously to level walking, our patients did not produce a characteristic TFL activity pattern. Quite the contrary, inter-individual TFL activity eminently varied between mono-, bi-, and triphasic activation sequences. Except for H8L, all patients displayed increased TFL activity when GLmax and GLmed activity were lower. Although these findings compare well with the literature, Lyons et al. report only little change in TFL activation timing relative to level walking [171]. We did not make the same observation, perhaps because the subjects in our study were THA patients and not healthy individuals. Chopra et al. report postoperative improvement of preoperatively increased TFL activity, even though aphasic activation patterns of the GLmax and GLmed were still present [261]. Additionally, our data show high TFL activity in almost all patients around the early swing phase. This is an observation in conformity with Gottschall et al., who found TFL activity to be highest during initial swing as an impression of the maximum hip flexion achieved and needed for thigh ascent at this point in time [262].

4.3.3 Descending stairs

Contrary to stair ascent, stair descent did not produce an entirely different set of muscle activity patterns than level walking. In fact, stair descent brought forth sEMG recordings which show patterns that resemble variations of the activity sequences yielded in level walking. When looking at the kinematic patterns our patients generated in level walking and stair ambulation, it is striking how similar they appear to be in the sagittal plane between gait and stair ascent yet how similar they actually are in the frontal plane of level walking and stair descent. Despite the seemingly resembling sagittal plane movement patterns in level walking and stair ascent, however, the latter produced a much greater ROM (almost by 20°) and is a more demanding ADL than level walking, which is an aspect the literature agrees upon [171,261,263,267]. In contrast, frontal plane kinematic patterns are not far from being congruent, with only relatively little deviation from their actual ROM (by 2°). This is also reflected by individual GLmax and GLmed activity, which, as in level walking, reached their respective IPMA between HS and CTO in seven patients.

At this point of the movement cycle, GLmax activity represents the eccentric hip extension of its lower fibers and the abductor moment of its upper fibers during load acceptance of the ipsilateral limb [171,266]. Like the upper GLmax, the eccentric abduction of the GLmed stabilizes the hip in the frontal plane during loading response between HS and CTO [171,266]. In three of our patients, this even continued throughout the stance phase, possibly

indicating muscle weakness [184]. Lyons et al. found muscle activity during stair descent to be universally lower than during gait and stair ascent, concluding this ADL to be independent of hip extension [171]. While this finding may be true for healthy individuals, it remains unclear whether this assertion holds true for OA and THA patients. Our data suggest hip extensor action through GLmax activity in most of our subjects, likely resulting from eccentric and isometric contraction in order to stabilize the ipsilateral limb in the sagittal plane during load acceptance between HS and CTO. Unlike Lyons et al., our patients' electrophysiological activity was not gathered by the use of fine wire needle electrodes, which limited us to assessing whole muscle activity as opposed to differentiating between the functionally variable upper and lower segments of the GLmax [171]. Therefore, the GLmax activity recorded in our subjects may have emerged from upper GLmax fibers, which are reported to function much like the GLmed in the frontal plane [171].

Strangely, the same two patients who displayed atypical muscle activity in stair ascent exhibited out-of-phase GLmed activity bursts at about the same time in pre (H9L) and early swing (H6R), potentially to ensure hip frontal plane stabilization and as a consequence of structural disintegration [184]. H9L additionally generated peak GLmax activity analogously to his GLmax IPMA in stair ascent, which possibly reflects eccentric hip extension [266]. It may also have been an out-of-phase attempt of the upper GLmax fibers to stabilize the hip in the frontal plane [171]. GLmax and GLmed activity in H9L differed decidedly in level walking. H6R, on the other hand, produced consistent yet abnormal patterns of either GLmax or GLmed activity throughout level walking, stair ascent, and stair descent. Chopra et al. found preoperatively abnormal GLmax and GLmed activity in stair negotiation to persist after THA, whereas irregular TFL activation patterns were found to improve [261].

Complementary to level walking and stair ascent, stair descent did not yield uniform TFL activity across subjects, instead producing rather individual activation sequences without following a distinct pattern. However, there is correspondence between patients' individual TFL activation patterns compared to stair ascent. H3L, H6R, and H9L generated continuous TFL activity, and again, H5L displayed three prolonged activity bursts. This might point towards compensatory hyperactivity of the periarticular hip muscles which originated presurgically when OA first became symptomatic [184,187,268,269].

Besides this, six of the eight patients with recorded TFL activity exhibited TFL firing between late stance and early swing, which is in line with the literature and corresponds with this muscle's function as a hip flexor and its preparation for the swing phase [171,262].

4.3.4 Standing up

A healthy adult rises from a sitting to a standing position about 60 times per day [270], which makes it one of the most frequent ADLs and thereby a skill of substantial clinical significance [271]. Although gait and gait-related activities remain the predominantly studied movement patterns [174,252], the literature provides an increasing number of investigations on the fundamental movement of sit-to-stand in healthy individuals. However, studies on pathological populations or postoperative accounts are either rare or not available. In spite of rising from a sitting to a standing position being one of the most common ADLs, no studies on postoperative electrophysiological assessment of the gluteal muscles and the TFL were found to compare the findings of this investigation with.

On another note, different anthropometric features between individuals, contrasting foot positioning at movement initiation, the use of auxiliary agents (armrests etc.), and seat height are significant factors in the determination of the joint angles realized, the total excursion of body segments, and the torque built up in the lower limbs' joints [211,242], all of which may in turn influence the formation of electrophysiological signals [174,247,257,258]. For this reason, the findings of this study are the result of its methodological setting, which has to be taken into consideration when comparing the outcomes found in the literature.

Our patients generated less variable sEMG signals in standing up in that the GLmax and GLmed produced similar activity patterns, while the TFL also yielded less variable activity sequences than in level walking and stair ambulation. Nonetheless, TFL activity was volatile in comparison to GLmax and GLmed activity. While there were almost no activity bursts during the initial phase of rising from a chair, the forward inclination of the trunk preceding seat-off was accompanied by some low and steady GLmax, GLmed, and TFL activity. Presumably, this rudimentary electrophysiological activity of the hip muscles was some sort of anticipatory state of being active in preparation for initiating the transfer of forward momentum during seat-off, in which the body moves anteriorly [211,272]. Preparatory muscle activity was previously reported while executing compound movements such as rising from a chair, and it was identified as a means to adjust individual posture from a

stable to a less stable position [273]. An explanation for the increased activation of the GLmax which may be observed in almost half of our patients (H3L, H5L, H9L, and H10R) might be a lack of horizontal momentum provided by the hip flexors. Vander Linden et al. performed simultaneous kinetic, kinematic, and electrophysiological measurements on healthy elderly adults, finding that the generation of horizontal momentum by the hip flexors is crucial in attaining an upright position [274]. In case of diminished horizontal momentum due to insufficient hip flexion, the hip extensors increase activity in order to compensate for insufficient hip flexion [274]. This short phase (about 25% of the movement cycle) involved peak TFL activity during maximum trunk and hip flexion in half of our subjects. It was followed by the buttocks' liftoff from the chair and an ensuing drop of TFL activity, probably as a result of the hip extension transitioning the body towards upright standing [211,272]. There was a gradual increase of muscle activity as the extension of the hip continued, with the majority of patients reaching peak GLmax and GLmed activity and half of the patients generating peak TFL activity within this stretch. Upon approaching the final standing position and thus closing in on full hip extension and stabilization of the pelvis in the frontal plane, GLmax and GLmed activity declined slowly, reflecting the decrease and subsequent cessation of hip extension velocity [211]. Any residual muscle activity after this point in time likely represents the anterior-posterior and lateral sway during quiet stance, which is an observation made in an investigation on whole-body movements during sit-to-stand [211].

Other studies investigating the muscle activity of healthy younger (mean ages of 21 to 27 years) [272,275,276] and elderly adults (mean ages of 69 to 70 years) [276,277] report GLmax and GLmed activity waveforms essentially in conformity with our findings. In a study assessing the functional aspects of healthy elderly subjects (mean age of 74 years) while rising from a chair, Dehail et al. showed the GLmax to be the most frequently activated muscle among eight muscles of the lower limb, accentuating its relevance in trunk and hip extension [278]. Dehail et al. also recorded peak GLmax activity at about the same time as was done in our patients [278], which suggests regular GLmax activity in our subjects. Munton et al. analyzed both asymptomatic and arthritic individuals' GLmax signals, which the authors not only found to be in keeping with one another and the existing literature on normal subjects at the time but also with our results [279].

4.3.5 Sitting down

Going from a standing into a sitting position is an essential ADL [280], and failure to master this critical task is associated with an increased risk of falling [281]. Complementary to standing up from a sitting position, there are no accounts of post-THA EMG assessments of the gluteal muscles and the TFL in the literature. Only a small number of studies examine the stand-to-sit movement in healthy and/or other pathological populations. One study assessing the sEMG signals of six lower limb muscles in healthy individuals and patients suffering from arthritis found no differences in the signals' phasic patterns, besides also noting them to be a mirror-inverted version of what they were found to be in standing up [279]. Despite resembling kinematic patterns across patients, our results only show such a reversed phasic pattern in one patient (H6R). Another study investigating both standing up and sitting down, however, points out that, although both tasks yield similarities in muscle activation, they also produce noticeable differences – yet in the activation of muscles that are not part of our investigation [281].

Assuming a sitting position is initiated through trunk flexion and synchronous eccentric contraction of the hip extensors until the thighs and buttocks touch the seat [281], which is reflected by increasing GLmax activity found in all and peak GLmax activity observed in five of our patients during that phase. Both the GLmed and TFL also showed major activity bursts, although no uniform activation pattern became apparent. Presumptively, their activity is due to their role in maintaining pelvic balance (GLmed and TFL) and assisting in trunk flexion and the stabilization of the knee in the sagittal plane (TFL). By the time of having taken a sitting position and while extending the trunk towards assuming vertical posture, muscle activity ceased only in H6R. All other patients still displayed vigorous activity in either one, two, or all of the three lower limb muscles examined. Conceivably, the ongoing muscle activity is based on co-contractions due to the extension of the trunk [281]. Our patients' GLmax activity can be characterized by a sustained pattern throughout all phases of the movement and thus is consistent with the findings of previous studies [279,281]. Because the literature provides no accounts of recorded GLmed and TFL activity, we are unable to match our data accordingly.

4.3.6 Summary

Investigations on the functional outcome after THA incorporating other, more rigorous ADLs than level walking are scarce in the literature. The few accounts published do not

yield long-term postoperative results or they use different methods of normalization, which makes it difficult to evaluate our findings relative to the findings of other research and put our results in context. Despite our lack of discerning a difference regarding the amplitude relative to maximum performance capacity, we can distinguish the modalities (monophasic, biphasic, triphasic etc.) and temporal patterns of hip muscle activation generated by the GLmax, GLmed, and TFL during the most common ADLs. Except for the abovementioned variability in a limited number of our THA patients, GLmax and GLmed phasic patterns in all ADLs were mostly typical and in agreement with the literature. While for level walking identifying distinct bursts of muscle activation is viable, stair ascent and descent occurred to cause more individual challenges among our test participants, which resulted in a more variable electrophysiological outcome in the hip muscles, with muscles elementary for the execution of the movement (GLmax and GLmed) displaying unmistakable activity patterns. In spite of the controlled experimental setting and limited exposure to potential confounding factors, stair ambulation produced higher variability than level walking, probably because stair negotiation is a more demanding task [171,263,267]. The activation of the GLmax and GLmed during level walking is in accordance with the findings of a study quantifying the individual muscles' contribution to whole body support during gait [153], underlining these muscles' relevance in the accomplishment of this relevant day-to-day task. The arthroplasty surgery of the participants in this investigation had been performed at least 35 months before data acquisition, which is an aspect that likely contributed to the similarity of our findings regarding GLmax and GLmed activity with the results of studies assessing these muscles' activity in patients with a recovery time of at least 12 months or even asymptomatic individuals [55,56]. Some of our patients may have recovered better than others, adding to the variability of our sEMG recordings. Still, TFL activity was found to deviate most noticeably from conventional patterns in view of its inconsistent phases of activation and hyperactivity in up to half of our patients, which is a known attendant circumstance in hip joint pathologies [185,187]. In order to evaluate the degree of electrophysiological irregularities among the hip muscles affected by hip OA, TFL activity could thus serve as an important reference in future investigations.

Joint position has an effect on sEMG signals [174,247,257,258], and visual inspection of individual muscle activity waveforms reveals that there are ADL-specific relative activity sequences and temporal patterns for the periarticular hip musculature. In the context of

discussing varying sEMG profiles in the more demanding ADLs under investigation like stair ascent and descent, it appears that the count of activity bursts or muscle activation onset and cessation times, respectively, matter more than attempting to outline one unitary sequencing pattern for each muscle in all test participants. Moreover, prolonged phasic muscle activity, as also found in a number of our patients, is associated with muscle weakness [184,269] and may lead to muscle stiffness [268]. Furthermore, both earlier and delayed onset times of muscle activation were found in subjects suffering from pain [268,282-285]. With regard to pain being a leading symptom of hip OA [15,22,32] and the patients in this study having been diagnosed with symptomatic hip OA prior to having undergone THA, pain is a circumstance that may have had a lasting effect on their kinematic [235,286] and electrophysiological parameters [184,185,269].

When normalizing the sEMG recordings of the patients in this study, the aim was to reduce variability. For this reason, the relative intensity of the ADL-related muscle activity, which was acquired by normalizing the absolute values in millivolt to the peak muscle activity of the task under investigation, was deemed to be the most adequate normalization technique [287-290]. It is not an indicator of how active a respective muscle is relative to its potential absolute maximum activity, as is intended to be produced, for example, within the framework of a maximum voluntary isometric contraction (MVIC) [174,291]. In an MVIC, the recorded electrophysiological signals are normalized to the produced reference value, yet while carrying out dynamic tasks, the activation of individual muscles may not only be submaximal but also exceed 100% of the reference value realized in an MVIC [291,292]. Dynamic activities, such as the ADLs in this study, do not produce the highest but the most appropriate muscle activity possible [288,292]. It is imperative to take this determining factor into account when analyzing muscle activity, which may also be evaluated in terms of individual activation timing [293]. The TFL, for example, was shown to be very active throughout all ADLs in an out-of-phase manner. While this observation does not quantify the TFL motor units recruited, it conveys information about TFL temporal patterns. The electrophysiological data discussed in this investigation indicates irregularities in muscle activity timing and pattern shapes. But this information cannot be directly linked to possible inferences about muscle force [174,279,294] or applied as evidence of any potential muscle weakness [259]. As follows, due to the sEMG recordings in this study being of qualitative and not quantitative nature, they do not imply normal muscle strength.

Long-term postoperative follow-up investigations involving electrophysiological assessments of THA patients are rare. In addition, accounts of THA patients carrying out other tasks than gait and gait-related ADLs, such as standing up from and sitting down on a chair, are missing in the current research. Hence, the results of this sEMG analysis fill a part of this gap in the existing literature.

4.4 Impact of hip abductor muscle status on in vivo hip joint loads through hip joint kinematics and hip abductor muscle activity

The purpose of this study was to investigate the effect of hip abductor muscle impairment on hip joint kinematics and hip abductor muscle activity in the long term and also to determine the longstanding influence of hip joint movement patterns and periarticular hip muscle activity on the formation of hip joint contact forces. In earlier publications, our research group already offered a first account on the short- and long-term impact of THA on hip abductor muscle status and of hip abductor muscle status on in vivo hip joint loads [116,117].

In response to the significant decrease of GLmin volume in the short (3 months) and long run (50 months), our THA patients' TFL volume was found to increase significantly [115]. Rodríguez-Roiz et al. argue that TFL hypertrophy following THA may be a reaction of hip overloading succeeding gait irregularities [295]. Müller et al. suggest a functional interdependence of the hip's muscle groups, finding TFL cross-sectional area to rise as a consequence of GLmed impairment secondary to THA using a DLA [296]. Sutter et al. share this notion in that they attribute the increase of TFL volume to this muscle taking over a large part of the function of structurally impaired hip abductors, thus interpreting post-THA TFL hypertrophy to be compensative [297]. Similarly, Pumberger et al. report a significant compensatory hypertrophy of the TFL microarchitecture six months postoperatively in two groups of patients who underwent THA either with a DLA or via MIS by means of an ALA [107]. Additionally, both GLmax and GLmed volume were also shown to increase markedly following THA, with long-range hypertrophy almost reaching statistical significance [115]. In the GLmed, however, our subjects' postoperative volume growth is also associated with fatty infiltration [115], indicating iatrogenic and/or preoperative muscle damage [45,47,54,83,108,296-298]. The positive volume changes of the GLmax after THA are consistent with the findings of Rasch et al., who report an increase of GLmax volume two years postoperatively [299]. The results of Uemura et al., whose patients even

experienced significant growth of GLmax and GLmed cross-sectional area at a mean of four years after hip replacement, imply that a longer recovery period may lead to significant volume changes of the affected muscles [300]. Interestingly, the long-term effects of OA and THA on the anatomy of the hip abductor muscle group reveal a structural interconnectedness that also translate into a functional interplay between kinematics, kinetics, and electrophysiological activity. The points of interest described in chapter 2.7 and chosen for statistical analysis are discussed below.

4.4.1 Hypothesis I

Impact of hip abductor muscle status on hip joint kinematics

Our data support the understanding that the structural integrity of the GLmax, GLmed, and TFL is vital for the realization of hip joint movements in all of the five ADLs investigated, especially in level walking, stair ascent, and stair descent. As expected, only little associations were found with the 2nd peak F_{res}, while correlations with a strong effect size ($r_s \geq 0.5$) were found with the 1st peak F_{res}, along with the other points of interest maximum joint excursions and dynamic ROM. As pointed out earlier, the GLmax, GLmed, and TFL perform their main tasks during or around the 1st peak F_{res}. For this reason, it comes as no surprise that their respective TMV and/or FR has an impact on hip joint movement during the formation and the occurrence of these peak joint loads. Nevertheless, the literature and the measurements show that the activation of the muscles under investigation is particularly critical for the time interval between HS and CHS, which is why not only peak contact forces but also other instants in the course of a full movement cycle are reasonable marks of interest.

Even though level walking and stair ambulation seem very much alike in that they are ADLs which necessitate propulsive movement, the effect of individual muscles' status in those locomotive sequences has to be differentiated. While gait demands continuous forward propulsion of the body [301] and stability of the hip within the frontal plane [168], stair climbing further increases the difficulty level of these locomotion requirements. It produces a larger sagittal ROM [261] and raises joint loads compared to level walking [117], generating large concentric powers due to high extensor moments [266] and emphasizing the hip abductor group's purpose of maintaining pelvic balance [262]. The results of this study reflect these circumstances insofar as potential deficiencies became more apparent in stair climbing than in any other ADL.

TMV includes both fat-free muscle mass and adipose tissue, with the latter being expressed as the percentage of fat volume in TMV. Despite the evidence that fatty infiltration is a known negative predictor of functionality [197,302] and has been shown to persist in THA patients in the long run [115,117], TMV was found to be a stronger determinant of resultant kinematics than FR. One potential explanation for this could be that composition and strength of aging muscle deteriorate irrespective of muscle quantity [303]. This is an aspect which may have affected the functional outcome of the recordings at disposal. However, this suggestion cannot be sufficiently substantiated by the underlying data because muscle strength was not assessed. In spite of this, increases of both indicators of muscle status largely predict kinematics as assessed by their impact on maximum joint excursions and dynamic ROM.

In particular, hip dynamic ROM is mostly affected by an increase of GLmax TMV in that a significant decrease of sagittal plane ROM in stair negotiation and a significant increase of frontal plane ROM in stair descent were found. There are trends for increases of frontal ROM in level walking and stair ascent. Upon visual inspection, half of the patients showed a somewhat regular locomotion pattern, whereas the other half limped noticeably and moved along with an anterior tilt of the upper body. The latter is associated with hip extensor muscle weakness and hip flexor contraction [169,252,261] as well as identified as a compensatory mechanism resulting from reduced peak hip extension and/or sagittal hip ROM [235,236,304]. According to Shrader et al., irregularities in GLmax electrophysiological activity, in addition to unfavorable upper body posture, may cause a performance decrease pertaining to the lower sagittal ROM in stair climbing [263]. Another key finding regarding the GLmax is that an increase of TMV is associated with higher maximum abduction in stair ascent and descent, which indicates that the GLmax hypertrophy previously mentioned likely emerged as an immediate result of the fatty degeneration of the GLmin. It is possible that the GLmax received more of the workload [152] which, under physiological circumstances, is mainly assumed by the other gluteal muscles and the TFL in more rigorous ADLs, leading to the hypertrophy of the GLmax. Yet, as no distinction was made between upper and lower GLmax status, it cannot be determined to what extent the upper GLmax, which performs as a hip abductor [170,171], ultimately contributed to these effects.

An increase of TFL TMV also affects dynamic ROM, however, to a lesser degree. In the TFL, larger TMV significantly correlates with higher sagittal plane ROM in standing up, while showing a trend for higher sagittal plane ROM in sitting down. An increase of TFL FR significantly correlates with higher frontal plane ROM in stair ascent, conceivably because of the increased maximum adduction also made out in the same ADL. The fact that frontal plane kinematics are sensitive to an increase in TFL FR shows the important role the TFL plays [158], especially during the exertion of more demanding ADLs [171,261,263]. Other key findings are that increases in TFL TMV are associated with higher abduction in stair ascent and that an increase of GLmed TMV significantly correlates with higher abduction and lower adduction in stair descent. This is in line with the literature on GLmed and TFL function, which accentuates and identifies these muscles as the hip joint's primary abductors [151,152,158,168,173].

Increases of GLmed TMV and GLmax and GLmed FR link with decreases of maximum adduction in stair descent and level walking, respectively. Throughout the 1st peak F_{res}, larger TMV and higher FR also correlate strongly with lower adduction in level walking and stair ascent, besides lower flexion in level walking, stair ascent, and sitting down. Lower hip adduction at these critical points of interest hints at decreased adductor moment as well as abductor muscle weakness and thus at an adaptation of abnormal frontal plane kinematics [165]. It might further have been an attempt to compensate insufficient abductor muscle strength and/or to reduce hip joint contact forces [166,235]. Despite this consideration being only theoretical, decreased hip flexion in sitting down might also come as a result of trying to lower joint loads, since contact forces were found to increase in a nearly linear manner as hip flexion in standing up increases [245]. Reduced self-selected walking speed evident in long-term follow-ups of THA patients was not found to be the cause of lower hip flexion [165]. Nonetheless, lower flexion may be indicative of pain-avoidance strategies, persistent hip flexor weakness, or individual concerns regarding the stress put on the ipsilateral limb [118]. However, the latter is unlikely to be the case because the patients in this study had a recovery time of approximately 50 months and their clinical scores yielded satisfactory results overall [115,117]. Upon assessment through the VAS, seven of nine patients reported no pain and only two patients noted very low pain, which was interpreted as merely transitory [115,117].

On the whole, the findings of this investigation clearly illustrate that muscle impairment interferes with physiological movement.

Impact of hip joint kinematics on in vivo hip joint loads

The 1st and 2nd peak F_{res} are critical instants of the gait cycle as they pose the points in time in which the highest loads are exerted on the hip joint during locomotion [116,117,131,137,305]. The 1st peak F_{res} emerges around CTO, ushering in the single-limb stance phase of the ipsilateral limb, and the 2nd peak F_{res} occurs around CHS, initiating the final stage of the gait cycle (please refer to figure 1). In our patients, higher hip flexion at the 1st peak F_{res} is significantly correlated with higher joint loads. The 2nd peak F_{res} shows a similar but slightly weaker correlation with the sagittal plane of the hip joint: lower hip extension moderately ($r_s = 0.43$) [213,214] correlates with higher contact forces. Hip OA and THA patients can display hip flexion contractures or, probably more appropriate, hip extension deficits [118,155,165,234-236]. This fact pattern is reflected by lower hip extension in ADLs. Given that hip arthroplasty patients were shown to develop biomechanical strategies throughout the progression of OA aiming at pain reduction and that the arising pathomechanical patterns persist following surgery [118,165,306], our patients' decreased hip extension in various ADLs may be interpreted correspondingly. Hip flexion loosens the capsular and ligamental constriction imposed on femoral head and acetabulum, consequently lowering the hip joint's exposure to compressive forces [307], which may temporarily relieve patients from coxalgia. Persistent holding of this position, however, may lead to an atrophy of active and passive structures responsible for hip extension, adding to the potential for developing hip flexor deformities or extension deficits [118], in addition to the influence of approach-related scar tissue. It is also possible that patients try to reduce periarticular hip muscle load, which can be construed as a compensatory technique attempting to offset hip muscle weakness [118,235,247].

Moreover, the results show that an increase of hip adduction correlates significantly with higher 1st peak hip contact forces in stair ascent and that there is a similar association with higher joint loads in level walking and also the 2nd peak F_{res} in stair descent. To that effect, it is interesting to note that higher maximum hip abduction in level walking and stair descent correlate strongly with lower joint loads. Level walking and stair negotiation entail single-limb stance phases, in which the hip abductor muscles mainly contribute to a balanced pelvis [149,308]. Lamontagne et al. found that THA patients yield different

movement patterns than healthy controls [237]. Deficient hip joint kinematics become particularly apparent while transitioning from double- to single-limb support, which roughly translates to the instants of the gait cycle when the 1st and 2nd peak F_{res} come into being (please refer to figure 1) and the hip abductor muscles do most of their work [237]. The authors of the above study speculate that the lower abduction moment they found in THA patients is a consequence of surgical approach-related structural impairment of the GLmed [237]. On the other hand, compensatory strategies to offset mechanical deficiencies, such as the ones found by Lamontagne et al. [237], might have originated before THA. Hurwitz et al. report decreased hip adduction moment in hip OA patients as opposed to healthy controls, which they interpret to be the result of a compensative routine, in which affected individuals shift their upper body over their hip joint with the goal of either reducing joint forces and pain, offsetting movement limitations, or counterbalancing strength deficits of the hip abductors [235]. Along these lines, Foucher et al. ascertained that the locomotion patterns of arthritic individuals are altered before undergoing surgery and persist thereafter [166]. The authors show pre- as well as postoperative gait kinematics and peak external moments in THA patients to be significantly correlated [166].

The findings support the understanding that the hip abductor muscles facilitate pelvic stability in the frontal plane and the idea that their anatomical properties may either promote or impede physiological locomotion [149,168,170,171,308]. A redeployment of muscle volume in and a fatty infiltration of the hip abductor group occur to serve as predictors for altered joint loads under the biomechanical conditions attained while conducting routine tasks. A possible mechanism behind this outcome is that there is an increase of adipose tissue in the hip abductors, which is to the disadvantage of functionally active muscle mass (contractile muscle fibers), thus compromising muscle quality and strength and also frontal plane balance [197,309,310]. Various publications identify the GLmax and GLmed as significant contributors to the formation of hip contact forces and ground reaction forces, respectively [149,151-153,187,311-313]. The findings of Damm et al. are in line with the aforesaid, as they report abductor muscle impairment to be paralleled by increased in vivo hip joint loads [116,117].

Notwithstanding that muscle strength and external moments were not assessed, hence preventing the establishment of a causal relationship between muscle weakness and the

results of this study, our data suggest that muscle impairment and movement patterns which are less than optimal lead to higher hip joint loads.

4.4.2 Hypothesis II

Impact of hip abductor muscle status on hip abductor muscle activity

Taking into consideration that the periarticular hip muscles investigated mainly account for the three-dimensional movement of the hip joint, especially hip extension, abduction, internal rotation, and pelvic stabilization [151,152,158,168,170,171,173], it is not surprising that, for the most part during locomotion, they reach maximum activity between HS and CHS. Accordingly, and as is the case with hip joint kinematics, most associations between muscle status and muscle activity are at the 1st peak F_{res}. In addition, there are correlations with a strong effect size between muscle status and muscle activity at the IPMA, which, for the best part of the movement cycle, is realized shortly before or after the 1st peak F_{res}. Interestingly, an increase of GLmax TMV significantly correlates with a later IPMA in stair ascent, also correlating with a later IPMA in standing up, whereas increases of GLmed and TFL TMV correlate with an earlier IPMA in stair ascent. Furthermore, an increase of GLmed FR significantly correlates with an earlier IPMA in level walking and standing up, while an increase of TFL FR significantly correlates with an earlier IPMA in stair ascent.

Dieterich et al. found an earlier activation onset of the hip abductors in symptomatic hip OA patients and patients with chronic hip pain when compared with healthy controls [268]. Yet, it is difficult to apply the inferences of this finding to the results of this study one-to-one, not least because our hip OA patients underwent THA and the aforementioned authors' measurement setup for muscle activation consisted of motion mode (M-mode) ultrasound [268]. Nonetheless, EMG measurements of patients with patellofemoral pain also show an earlier onset of muscle activity, hinting at a compensatory strategy caused by longstanding pain [285]. This notion is paralleled by Zacharias et al., who report trends with moderate to large effect sizes for an earlier peak activation of GLmin in hip OA patients [314]. On the other hand, there are reports of subjects experiencing pain, whose electrophysiological recordings demonstrate delayed muscle activation onset contrary to asymptomatic individuals, which also implies the emergence of offsetting mechanisms to counter pain [282-284]. Aside from the finding that the relative timing of muscle activation is altered while in pain [268,282-285] and after having undergone THA [55,183] and that there seems

to be no universal pattern resulting in either a clearly earlier or strictly delayed onset of electrophysiological activity, it remains elusive whether deviations from healthy controls' measurements are a cause or a consequence of the underlying pathology [282,284].

There is also debate about the origin of adaptations to pain with respect to movement and muscle activity. Proceeding from various instances of the nervous system, individual motor units may ultimately be activated in a different intensity or sequence in order to counter the activation of muscle fibers which contribute to the emergence of pain [315]. An sEMG system using a single pair of electrodes for each muscle, however, is not sufficient for mapping this spatial redistribution of muscle activity [316,317]. On that account, it can only be speculated that our THA patients may have produced a similar adaptation to pain preoperatively and that it prevailed postoperatively. The dominant nature of pain catalyzes an adaptation to different muscle recruitment patterns, which results in less painful kinematic output relatively fast [316]. But the dissipation of pain does not automatically restore pre-pain muscle recruitment patterns [316]. Intriguingly, one factor contributing to this phenomenon could be the individual perception of pain: after subjects with a particular sensation of vulnerability are exposed to pain, they maintain their protective movement strategies even when pain has disappeared [318]. In a prior publication, our research group presented the postoperative clinical scores of the THA patients in this investigation, noting satisfactory results in HHS, WOMAC, EQ-5D-3L, and VAS, all of which document patient-reported pain [117]. Despite the inability to compare pre- and postoperative clinical scores and PROMs because all subjects in this study were exclusively surveyed 51 months postoperatively, all our patients had a medical indication for undergoing THA due to symptomatic OA. Pain [15,22,32], morphologic changes in soft tissue [54,298,319], gait abnormalities [235,246,309,320,321], and electrophysiological activity being different from standard or norm [184,185,187,269] in OA patients are reported in the literature. For this reason, it can be concluded that, since our patients suffered from pain, they were engaged in antalgic movement to some extent and displayed irregular muscle activity patterns to a certain degree before they underwent THA. Because no kinetic, kinematic, and electrophysiological data were collected before our patients underwent THA, however, we refer to other publications which found correlating pre- and postoperative recordings. Preoperatively assessed muscle weakness [165,309], kinetics [165,166], and kinematics [165,166] were shown to persist postoperatively, which is why there is reason to suppose that this also holds true for our THA patients.

The subjects in this study may have adopted compensatory strategies prior to undergoing THA to avoid pain and offset muscle weakness arising from either disuse atrophy or neuromuscular deficits [184,322]. The constant pain-related disuse of certain muscles during the progression of OA leads to diminished contractile muscle mass, fatty infiltration, and reduced muscle strength [54,188,322-324]. Hip pain patients' prolonged phasic muscle activity, which may also be noticed in our patients, is not only associated with muscle weakness [184,269] but it may also alter muscle architecture and lead to muscle stiffness, thereby modifying the anatomical features of the muscles in question [268]. Despite the observation that muscle fat content [197] and muscle wasting increase with age [325], hip OA and THA patients were found to have more atrophy of type I (slow twitch) and especially type II (fast twitch) muscle fibers than age-matched controls [326,327]. In addition to inducing such morphological changes, continuous protective muscle activity impairs physiological muscle function (e.g., providing a range of dynamic activation variability and the capability of swiftly reacting to task-specific mechanical demands), consequently facilitating atypical kinematic patterns that promote an exacerbation of the prevalent arthritic changes and leading to OA in other joints [188,268,269].

Fatty infiltration of the musculature is not necessarily accompanied by decreased muscle mass of the respective muscles, yet it appears to materialize at the cost of muscle quality [328]. Proceeding on the basic premise that muscle mass is a measure for strength and physical function [323,329,330] and that fatty infiltration is a criterion for muscle impairment [108,114,197,302,310,331], it seems possible that the remaining contractile muscle mass undergoes a functional transition. Ling et al. speculate that, as muscle quality decreases, motor units' range of function shifts from a rather coordinative scope to one regulating pure force generation, which results in less neuromuscular control and leads to an aggravation of OA [332].

By contrast, our THA patients' lasting muscle impairment may have emerged as a direct result of muscle trauma from surgical intervention [45,47,83,108,296]. Supposedly, our patients' postoperative increase of GLmax TMV is not only the outcome of increased neuromuscular recruitment and improved overall hip function but also a compensatory response to hip abductor impairment and weakness following THA. This idea is supported by MSMs which enable computational simulations of hip abductor weakness and show a functional compensation by the GLmax [152,168]. Moreover, our patients' significant TFL

hypertrophy in the long view indicates that this hip abductor compensates for the significant long-term atrophy and impairment of the GLmin [115,117]. The TFL likely takes on a considerable part of the smallest gluteal muscle's task as a principal hip abductor in the performance of ADLs [158,173,297]. It is unclear to which extent an intermuscular shift of specific functions takes place, yet the significant long-term increase of TFL volume and the trends for lasting growth of GLmed and GLmax volume [115] support the idea of the hip muscles' functional interdependence [133,333].

Additionally, denervation injuries following THA have been reported in the literature [57,60,76,77,88,104]. However, there is no consensus on which surgical approach is most damaging to the branches of the gluteal nerves. Although all surgical approaches in THA may cause nerve lesions, transgluteal approaches, such as the DLA, appear to be responsible for the majority of denervation injuries affecting the GLmed [57,88]. The TFL is most sensitive to denervation in THA because it is believed to be innervated solely by the inferior branch of the superior gluteal nerve, but this structure is only seldom injured as a result of using a transgluteal approach [88]. An injury of nerval structures can lead to atrophy and subsequent structural disintegration of the innervated muscle tissue by initiating an irreversible transformation of contractile muscle mass into smaller and less contractile muscle fibers and non-contractile fibrous connective tissue [114]. Being a feared complication following THA, motor nerve palsy is a severe yet particularly rare condition [76,77,80]. While Farrell et al. found that most of the THA patients who incurred nerve palsy did not fully recover, they amount to merely 0.17% of the cases [76]. Chomiak et al. argue that, in a clinical setting, only EMG recordings which assess contingent fibrillation potentials of resting muscles or muscles performing an MVIC can effectively identify neuronal damage [88]. In contrast, Abitbol et al. developed a proprietary scoring system which evaluates phasic activation, spontaneous electrical activity, and recruitment [59]. None of our patients showed signs for motor nerve palsy, and it can be assumed that any potential injuries of the inferior and superior gluteal nerves and their branches likely took a subclinical course and resolved by the time long-term measurements at an average of 51 months after THA had begun [59,60,77]. Because our patients' electrophysiological activity was exclusively recorded by using surface electrodes and while they were conducting various ADLs (an MVIC was not performed), any observation of irregularities pertaining to neuronal damage would have been incidental.

Clinical symptoms of THA patients with lesions of the superior gluteal nerve operated through a DLA do not correlate with EMG recordings [334]. Regardless of the absence of pronounced clinical symptoms or electrophysiological irregularities suggestive of gluteal nerve injury, however, it is possible that morphological changes in our patients' hip muscle architecture are long-term consequences of nerve lesions sustained perioperatively [104,114,175]. Unis et al. found the TFL to atrophy in 61%, hypertrophy in 12%, and incur fatty infiltration in 42% of the patients who underwent THA via MIS by means of a modified ALA [104]. Chomiak et al. reinforce these findings by asserting that the inferior branch of the superior gluteal nerve, which innervates the TFL, is at particular risk of complete lesion during THA using an ALA [88]. In line with this, Roy et al. suggest GLmed atrophy and abductor weakness to be due to superior gluteal nerve damage in THA with a DLA [175]. It is believed that parameters other than direct nerve injury such as trochanteric nonunion, blunt muscle trauma, detachment of the gluteal flap, inappropriate retractor placement, damage caused by leg lengthening, and chronic nerve injury prior to THA are at the bottom of functional impairments and muscle weakness [334,335]. Since our patients' electrophysiological activity was not recorded before they underwent THA, the possibility of preoperatively present chronic nerve lesions cannot be ruled out entirely.

Impact of hip abductor muscle activity on in vivo hip joint loads

Our results show that an increase of GLmax and GLmed activity correlates strongly with higher 1st peak hip contact forces. Also, peak hip abductor muscle activity correlates significantly with higher concomitant in vivo contact forces. As discussed earlier, the GLmax, GLmed, and TFL are mainly active while performing their principal tasks during the time interval immediately before and shortly after CTO, which is when the ipsilateral limb accepts the remaining body weight from the contralateral limb and thereupon carries the body throughout the stance phase of the gait cycle [158,171,252]. In the stance phase, the investigated muscles facilitate a stabilization of the pelvis in the frontal plane, especially during mid stance. After a continuous hip extension of the ipsilateral limb, the weight is shifted back from the ipsilateral to the contralateral limb and the 2nd peak F_{res} materializes around the time of CHS [149,237,308] (please refer to figure 1). Not surprisingly, therefore, the sEMG activity of the GLmax, GLmed, and TFL realized at the occurrence of the 2nd peak F_{res} correlates only moderately, at the most, with concomitant joint loads.

The significant correlation between higher GLmax activity and increasing 1st peak F_{res} in level walking is a key finding that confirms the above. This is echoed by a strong correlation in stair descent for the same muscle and for the GLmed in stair ascent. In a like manner, a later IPMA of the GLmax correlates significantly with higher joint loads in level walking and sitting down but also with lower joint loads in standing up. Correspondingly, a later IPMA of the GLmed in sitting down correlates significantly with an increase in the concomitant F_{res}. Analogously to the GLmax, a later IPMA of the TFL correlates significantly with higher contact forces in level walking. However, a later IPMA of the TFL also associates with lower contact forces in stair ascent. Looking at it the other way around, an earlier IPMA of the TFL associates with higher contact forces in stair ascent, which is a remarkable finding that is addressed in detail below.

The different effects of muscle activity on contact forces could be due to a number of reasons. For example, inconsistent findings may be due to the fact that the sEMG electrodes utilized while recording only covered a certain area on the surface of the investigated muscles. While great care to comply with the guidelines of the SENIAM project was taken during the measurements [181], recording the electrophysiological activity of functionally different parts of the GLmax, for instance, as would have been possible with fine wire needle electrodes, was technically not feasible [171]. Another possible explanation for differing joint loads could be irregularities in muscle activation timing. As discussed earlier, especially the TFL, which is known to be affected when joint pathologies are present [185,187], displayed inconsistent patterns of electrophysiological activity throughout all ADLs. Nevertheless, the major reason for earlier peak TFL activity to associate with higher joint loads in stair ascent may be the emergence of relatively high adduction moments which occur in the course of alternating single leg stance phases. In ADLs like standing up or sitting down, high adduction moments can be avoided because both the ipsilateral and contralateral leg are consistently in contact with the ground. Level walking is an ADL which allows for a compensatory shift of the torso over the hip joint as a means to reduce joint loads and to counteract any potential abductor weakness, thereby decreasing adduction moments [235]. In contrast, ADLs which generate relatively high adduction moments during regular intervals of single leg stance phases, such as stair ascent, place an equally high demand on the TFL and likely involve a compensatory activation of additional, small muscles close to the hip joint, thus leading to higher joint loads.

The forces exerted on the hip joint are considerably determined by muscle force [149,151,152,187,191,311-313]. Although there is no straight relationship between muscle force and EMG signal amplitude [252,259], the intensity of a muscle contraction is expressed by the latter [177,294]. Our electrophysiological data reflect the relative intensity of the peak dynamic muscle activity realized during ADLs. The peak signal amplitude reached during level walking was found to amount to roughly one third of what was achieved in a test intending to produce maximum potential muscle activity [171,177]. Partly for this reason, it can be assumed that the positive correlations between muscle activity and the 1st peak F_{res} as well as the significant increase of the F_{res} at the IPMA allow for the careful deduction that higher hip abductor muscle activity is indicative of increased muscle forces acting on the hip joint. The supposition of increased muscle activity leading to higher joint contact forces further relies on Foucher et al. noting net agonist and antagonist muscle activity to be mirrored by external moments and external moments to correlate with joint contact forces [166]. Other accounts also remark that an altered electrophysiological activity of muscles is an aspect that is relevant in the formation of joint loads [259,269].

Muscle force is a function of contraction type, velocity, and individual muscle fibers' length, the latter depending on joint position [174,190,247,258]. Thus, muscle activation may be determined by individual kinematics [174,247,257,258]. Hagio et al. found activation profiles of individual muscles to correspond with the force directions produced in an experimental setup of changing hip and knee joint angles [258]. The authors suggest that a selective recruiting of those lower limb muscles generates the task-specific force required [258]. While both intentional and unintentional changes in the recruitment of muscle fibers result in different mechanical actions yielding short-term benefits like avoiding painful instances during movement or compensating strength deficits [235], long-term modifications of muscle activity and adjustments of load distribution can bear undesirable aftereffects [316]. A change of force direction, coming from altered intramuscular activation, may permanently reshape individual movement and thus put at risk the long-term integrity of passive structures such as joints and ligaments [316].

Electrophysiological recordings alone do not provide sufficient evidence for the purpose of determining muscle forces [179,259,294], which are found to mainly define joint contact forces [149,151,152,187,191,311-313]. Yet, even if a combination of diagnostic and experimental methods was utilized, assessing human in vivo muscle forces would not be feasible at

this point in time [192-195,336,337]. The results of muscle activity recordings depend on a number of factors, including extrinsic parameters like electrode placement or the use of auxiliary agents but also intrinsic parameters such as the nature of the task under investigation or varying anthropometric characteristics among individuals [174,180,190,257,338]. The general understanding of muscle activation timing and the relative intensity of the electrophysiological activity in OA and THA patients improves with the continuous assessment of these groups in the literature. There is a scarcity of publications on the EMG patterns of hip OA and THA patients conducting various ADLs, however, which makes it difficult to come to a firm conclusion regarding the findings of this study.

As noted above, the patients in this investigation displayed irregular muscle activation timing and pattern shapes in some measure. Hence, it can be assumed that our patients' hip joint contact forces differ from those of asymptomatic age-matched controls. As measuring in vivo joint contact forces of healthy individuals is not feasible at the current state of science, however, it cannot finally be determined whether the hip joint loads measured are disproportionately high or low. In spite of that, the results show that dynamic and maximum electrophysiological activity of the GLmax, GLmed, and TFL have an impact on peak hip contact forces. By and large, the activation patterns of the muscles investigated are in agreement with the respective load patterns of the F_{res} realized. Nonetheless, in order to confirm whether physiological levels of the F_{res} are exceeded or perhaps not even achieved, further investigations should validate MSMs that utilize data from in vivo hip joint load measurements against in vivo recorded EMG data [193,305,339].

4.4.3 Summary

The objective of this study was to investigate the long-term functional outcome of THA and to examine the impact of GLmax, GLmed, and TFL status on postoperative in vivo hip joint loading through kinematics and muscle activity. It was hypothesized that an impairment of the hip musculature would lead to the emergence of pathomechanical locomotion patterns and irregular hip muscle activity, consequently causing an effect on in vivo hip joint loads. The measurement setup of the data in this study is unique in the literature. For the first time, in vivo hip joint contact forces which were measured during rigorous and vital ADLs like level walking, stair negotiation, and sit-to-stand/stand-to-sit were coupled with synchronous recordings of in vivo hip joint kinematics and electrophysiological signals. In addition to these simultaneously recorded in vivo kinetic,

kinematic, and electrophysiological measurements, correspondent muscle volumes and fat ratios of the GLmax, GLmed, and TFL were utilized for a retrospective analysis.

Although the literature does not attribute muscle weakness to a single cause [174,188,252,340], the outcome of this investigation confirms muscle impairment to be strongly associated with compensatory movement patterns and irregular muscle activity. Furthermore, the results support the understanding that hip joint kinematics and hip abductor muscle activity are strong predictors of in vivo hip contact forces. This study identifies contingent repercussions on THA patients' hip joint loads caused by potential abnormalities in hip joint kinematics and irregularities in muscle activation timing and pattern shapes. It can be concluded that hip joint movement in the sagittal and frontal planes as well as hip abductor muscle activation onset and cessation times depend on the structural integrity of the GLmax, GLmed, and TFL. Regardless of the original cause of impaired muscle function, a reduction of muscle tissue quality induces a worse functional outcome following THA [108,176].

The statistical results in this investigation are descriptive and not confirmatory. As follows, the findings of this study may not be entirely sufficient in establishing a causal chain between muscle impairment and altered joint loads brought about exclusively through kinematics and muscle activity. Nevertheless, the results corroborate the interdependence of these biomechanically and medically relevant parameters for the realization of ADLs. Even though some trends in agreement with the stated hypotheses may not be statistically significant, they yield moderate to strong effect sizes which could possibly become statistically significant with a larger cohort. This aspect makes them relevant enough to conduct further research into this topic. Overall, the findings agree with the notion that the structural integrity of the GLmax, GLmed, and TFL plays a pivotal role in the performance of ADLs.

4.5 Clinical implications

The present data, which were gathered at an average of 51 months after THA, allow for an elaboration on the periarticular hip muscles' involvement in the emergence of pathomechanical kinematic and muscle activity patterns, which in turn determine the formation of hip joint contact forces. In sum, the results show that, despite the relatively

long postoperative period, our THA patients' overall performance of locomotor and non-locomotor ADLs is not normal.

On the one hand, postoperative reports show favorable clinical outcomes and improved quality of life in patients who underwent THA [50,147,231]. Our THA patients' long-term postoperative clinical scores (HHS, WOMAC, EQ-5D-3L, and VAS), which were presented by our research group in an earlier publication, provided good results [117]. On the other hand, a preferable outcome assessed by clinical scores and PROMs does not automatically translate into an equally desirable biomechanical execution of ADLs [49,236]. Even though the postoperative situation and satisfactory clinical scores of our subjects are likely referable to their longstanding recovery period, the abnormal kinematics and muscle activity they produced during testing might be consequences of pain-avoidance strategies adopted prior to having undergone surgery [118,166,185,269,286]. When in pain, individuals present with compensatory adaptations to their movement patterns and abnormal muscle activation [166,184,185,235,268,269,282-286,316]. The literature suggests that kinematic deficits and atypical electrophysiological activation in THA patients, however, do not originate from pain experienced in the course of functional assessments but that they reflect a persistence of preoperative strategies aimed at averting pain when it was actually present [118,166,185,269].

Preoperatively, OA patients produce irregular patterns of motion and weight-bearing even in the absence of pain [304,341]. This being the case, iatrogenic muscle impairment is likely only one of several factors which determine the postoperative functional abilities of THA patients. Considering that pathomechanical adaptations predominantly surface during fatigue [56,188] or while performing rather demanding functional tasks such as walking with increased velocity [342] or stair ascent [263,343], unlearning these harmful motion patterns and reestablishing physiological kinematics with the aid of specific exercises is vital [344]. If they remain untreated, low-key compensatory strategies allow for a subsistence of detrimental offsetting movements even during less demanding ADLs [344], which facilitates the progression of muscle weakness secondary to selective disuse of certain muscle segments of the hip abductor group [235].

A systematic review by Loureiro et al. reports generalized muscle weakness in the affected limb of OA patients, especially in hip and knee flexors and extensors as well as

hip abductors and adductors [340]. Rasch et al. and Sicard-Rosenbaum et al. assessed THA patients' muscle strength at a mean of two years following THA and singled out the hip abductors as the weakest muscle group [309,345]. Muscle weakness is associated with altered muscle morphology, muscle activation, and gait abnormalities [118,168,175,184,188,189,215,248,261,302,327,329], all of which were found in the participants of this study.

It has been suggested that the increased activity of the GLmed in ADLs is due to persistent muscle weakness [56,184,260]. Hence, GLmed hyperactivity could possibly be the effect of a neuromuscular compensatory technique aiming at offsetting hip abductor muscle weakness. Investigators also found an increase of hip abductor muscle activity and a concomitant reduction of weight-bearing in the operated leg [56,184,260]. Dwyer et al. theorize that the inverse relationship between muscle activity and ipsilateral contact forces is a protection mechanism aiming at preventing pain through joint load reduction [184]. However, increased muscle activation may also signify rising muscle forces and can raise joint loads unphysiologically high [187,269].

Prolonged phasic muscle activity, as also present in our patients, is not only associated with muscle weakness [184,269] but it may also lead to muscle stiffness [268]. Arokoski et al. report a relatively larger decrease of type II (fast twitch) muscle fibers in OA patients compared to type I (slow twitch) muscle fibers, with the latter being associated with increased muscle stiffness [323]. Thus, even if baseline muscle volume was achieved through conditioning and resistance training, it is questionable whether original muscle quality can be recovered.

Experimental research suggests that certain motor units of a motor unit pool are selectively activated while others involved in the emergence of pain are inhibited, which reorganizes motor unit recruitment and ultimately changes the force angle in another direction [315]. Depending on which motor unit recruitment strategy the central nervous system deems most advantageous, patients might perform ADLs either under the condition of pain reduction or with the biomechanically most efficient type of locomotion [315]. While the main benefit of antalgic movement is the avoidance of pain, long-term effects of altered muscle recruitment are inefficient load distribution and less variability in locomotion patterns, consequently jeopardizing the integrity of other structures [316].

Another possible explanation for the emergence of pathomechanical locomotion patterns could be an anomaly of the neurosensory system [174,187,346]. The neurosensory system may be compromised as a result of iatrogenic muscle impairment from THA [183,341,347]. This would entail at least a partial loss of a patient's spatial hip joint perception because proprioception is a main function of soft tissue and contributes to the proper performance of movement [177,183,188,348].

The potentially deleterious effects of muscle weakness on the autonomy of those involved are well documented. Smith et al. point out that particularly older individuals rely on a reallocation of muscle force generation when they are affected by muscle weakness [349]. The arising reorganization of motor unit recruitment exposes this age group to a downward spiral of the consequences of muscle-disuse atrophy [349]. This eventually promotes vulnerability and dependence on others, which may finally necessitate affected individuals to seek institutionalization [349]. Additionally, muscle weakness and loss of proprioception are acknowledged as indicators for an increased risk of falling [25,345,350]. Irrespective of the pathogenesis of muscle weakness, muscle damage contributes to the emergence of abnormal patterns of locomotion and irregular muscle activity, which thereupon have an effect on joint loads.

Although the compensatory locomotion patterns adopted by hip OA and THA patients aim at alleviating pain, reducing joints loads, and offsetting muscle weakness, protective movement strategies facilitate the manifestation of concomitant effects (i.e., hip flexion contracture and/or hip extension deficit) and have unfavorable long-term consequences such as pain aggravation, expedited progression of OA in the affected joint, and spreading of OA to other joints – both ipsilateral and contralateral – as well as to spine and pelvis [118,149,155,188,189,235,351].

According to Kovalak et al., who performed functional and radiographic assessments of THA patients at an average of 14 months after surgery, fatty atrophy of the gluteal muscles does not only compromise the functional architecture of the operated hip but also of the non-operated hip [108]. Earlier publications of our research group yield complementary findings, adding that muscle impairment affects in vivo hip joint contact forces [116,117]. The muscular asymmetries between the operated and the non-operated

limb, which are found to persist for at least two years after THA, seem to contribute to the pathogenesis and/or progression of OA at the contralateral hip joint [108,300].

In this fashion, hip abductor weakness also translates into disproportionate muscle and contact forces in joints below the hip [23,149,168]. Commonly, patients suffering from hip OA and recipients of THA are particularly prone to developing or progressing in knee OA [286,352-354]. Inordinate hip adduction during ADLs, for instance, induces a dynamic knee valgus, which is a known risk factor for soft tissue injuries of the knee [355]. When compared to healthy individuals, both hip OA and THA patients were also found to move with increased knee flexion and ankle dorsiflexion, which are compensatory mechanisms offsetting restricted hip extension and enabling patients to keep up the forward propulsion of the body during the terminal stance phase of the affected hip's side [23,267]. The relevance of the hip musculature during the stance phase is emphasized through the striking finding that nearly all strong and significant associations of our correlation analysis ensued from either the 1st peak F_{res}, maximum dynamic hip joint excursions, or the IPMA. In keeping with the findings of a systematic review [165], our THA patients exhibited substandard maximum dynamic hip extension while executing locomotor ADLs. In fact, hip extension is identified as a key parameter in gait and stair negotiation [263,356] as well as in future disease progression [356]. Notably, Shrader et al. ascribe the lack of hip extension to postoperative weakness of the GLmax [263], which affirms the understanding of the hip adductor muscle group's functional interdependence.

Compensatory locomotion patterns in THA patients emerge from an inefficient distribution of muscle forces throughout the kinetic/kinematic chain [122,149,154-157,232,267]. The altered mechanical actions and subsequent inadequate allocation of loads generally affect both the ipsilateral and contralateral limb, bringing about interlimb asymmetries in kinematic and kinetic parameters [56,155,232,238,260,286,341,346,347,352,354,357,358]. In the literature, there is no common position on the question whether time-distance parameters, however, such as cadence, velocity, step length, stride length, and stance phase duration are significantly altered between the affected and the unaffected limb [119,232,236,260,341,343,358-361]. Colgan et al. and Kyriazis et al. suppose that the shorter stance phase they observed in the operated limb of THA patients is due to pain and muscle weakness [236,360]. Follow-ups beyond the period shortly after surgery may eventually be more revealing as Kyriazis et

al. found their patients' spatiotemporal parameters to catch up with the ones of healthy controls in the long run (8 to 10 years after THA) [360].

Symmetry of various spatiotemporal parameters, in turn, might actually be the result of an offsetting process of the non-operated limb aligning itself to the operated side's limb in order to avoid non-linear movement and to accomplish more efficient locomotion patterns [167]. With regard to counteracting any adverse effects of temporospatial imbalance, increased pelvic rotation is assumed to be another compensation strategy [232]. On that note, it is in agreement with present research that aberrant movement of the ipsilateral limb is associated with countervailing movement of the contralateral limb, at least in the short term [232,238,260,286,358,361]. Yet, there are a number of studies which report that symmetry in kinematic parameters may be achieved on a long-term basis [56,167,358,361]. For this reason, the execution of ADLs may not uncommonly look somewhat symmetrical, but the forces within the joint and/or ground reaction forces provide information about the actual asymmetry between the affected and unaffected limb [346]. Accordingly, the balance of evidence on kinematic and weight-bearing asymmetry points towards THA patients relieving the operated limb and loading the non-operated limb [56,238,260,286,341,346,347,354,357]. It appears that particularly weight-bearing asymmetry between the operated and non-operated limb is a major issue pertaining to the functional outcome following THA.

Operative treatment should always be considered cautiously and assessed on an individual basis, and holistic thinking can be advantageous for both patients and healthcare professionals [21]. This proves to be of value mainly for patients suffering from mild to moderate OA who did not yet elect to undergo THA and may eschew hip replacement in favor of conservative treatment. However, weighing for or against operative or non-operative intervention is less of an option for patients with end-stage OA or OA patients with a rather active lifestyle [22,147,248]. When indicated, THA yields worse clinical outcomes if postponed, and it is the only intervention in hip OA patients that effectively halts pain and an advancement of compensatory strategies in locomotor and non-locomotor ADLs, also slowing down the progression of OA to other joints [22,147,248]. Hip abductor muscles in OA patients were shown to be structurally and functionally impaired even before undergoing surgery [319,324]. Still, potential preoperatively existent muscle impairment is likely worsened by soft tissue injuries attributable to THA. An interconnection between soft tissue injuries sustained during THA and a poor

postoperative outcome is evident in the literature [47,53,95,176]. Comparisons between various surgical approaches concerning their overall postoperative functional outcome have not shown superiority of one approach over the other, and any differences reported are questioned with regard to their clinical relevance [53,85,87,98,176,362,363]. Yet, because all of the participants in this study were operated with a DLA, the results were certainly influenced by the possible consequences of this surgical technique. Of the approaches assessed in their study (PA, DLA, ALA, and anterior approach), Agten et al. identified the DLA to cause the most damage to and fatty muscle atrophy in the hip abductor muscles and tendons [47]. Tadross et al. point out that the DLA brings about a decline in hip abductor strength and frontal plane ROM [176]. Although the definition of a lateral approach is interpreted rather liberally in the literature, the results of studies assessing the repercussions of transgluteal approaches show that, due to their disruptive effects on the gluteal muscle compartment, they have a larger impact on diminished hip abductor strength and abductor-related irregularities in locomotor and non-locomotor ADLs than other approaches [53,95,176,364].

The balance of evidence suggests a multifactorial etiology in the pathogenesis of OA [4,21,23,31,32]. Patients display very individual compensatory methods for their functional impairments following THA [155], which underlines the importance of considering multidisciplinary approaches in individual pre- and rehabilitation programs. Reardon et al. call for attention to preoperative training programs aimed at improving the physical properties of the hip muscles [327]. The authors outline reversal of muscle atrophy and weakness as primary goals of prehabilitation [327]. A systematic review and meta-analysis examining the effects of prehabilitation on the postoperative outcome found significantly less pain, better overall function, and shorter hospitalization in THA patients [365]. Such training regimens should include exercises which contribute to reversing any potentially detrimental neuromuscular control adaptations (i.e., altered levels and onset times of muscle activation [183,187] or especially a reorganization of motor unit recruitment [315,316]) and enable a neuromuscular reeducation of proper hip mechanics [366]. A concept of early rehabilitation after THA, on the other hand, may incorporate the theory of cross-education, in which the unaffected limb is trained while the affected limb remains immobilized for as long as medically prescribed [367]. The intention of implementing cross-education into rehabilitation is to stimulate interlimb neural circuits, preserve bilateral strength, and reestablish symmetry [367]. Furthermore, an implementation of biofeedback

into rehabilitation protocols might assist in breaking these patterns as THA patients present with established movement strategies [368,369]. A novel procedure to attain a more satisfactory postoperative functional outcome of THA may be the future use of allogeneic placenta-derived, mesenchymal-like adherent cells [370]. Preliminary results of clinical trials show that these cells improve muscle strength when compared to the results of a control group who received a placebo, thereby lessening the complications of iatrogenic muscle damage [370].

Our results show that total volume and fatty degeneration of the GLmax, GLmed, and TFL have a significant impact on hip joint kinematics and hip abductor muscle activity. As kinematics and muscle activity also have an influence on joint loads, the findings of this investigation support the conception that the structural integrity of the muscles encompassing the hip joint is of utmost importance not only for the limitation of OA progression to other parts of the body but also for the lifespan of a hip implant. Loureiro et al. note that, because end-stage hip OA patients are known to compensate loads between limbs with the intent of averting pain, the ensuing muscle-disuse atrophy might put the long-term success of a hip implant at risk due to hip joint moments correlating with bone mineral density [340]. McCrory et al. also point out that inadequate joint loads may jeopardize the success of a hip prosthesis [341]. Lenaerts et al. stress that adequate joint loading with respect to bone remodeling and implant fixation are critical elements to consider in avoiding prosthesis failure [306]. Long et al. highlight even subclinical muscle weakness to be partly responsible for increased loosening of hip implants among more active THA patients [56].

Actual in vivo hip joint contact forces were found to considerably exceed the results of pre-clinical testing methods, which implies that implant manufacturers rely on testing protocols that do not simulate in vivo conditions [203,230]. In order for the industry to provide a growing patient base [33,34] and its increasing demands [4,39] with continuously improved prosthesis designs and longer implant survival rates, however, existing ISO standards should be revised and complemented by the results of in vivo measurements, as the circumstances require [137,203,230]. By nature, facilitating large-scale in vivo measurements is difficult, which complicates collecting extensive in vivo kinetic data on various ADLs in order to give an account for the high inter-individual variability produced by sophisticated computational modelling [230]. Regardless, in vivo measurements are essential in validating

MSMs and their contributions to the knowledge of the actual processes taking place within the joint are unparalleled. The measured original joint loading conditions are largely determined by muscle forces, and simultaneous recordings of kinematics and muscle activity can give information prerequisite for MSMs in predicting probabilistic muscle forces and joint loads [192,193,371].

Several studies contradict each other in terms of the role of certain muscles during gait. For example, Anderson et al. assert that the major share of support at mid stance is provided by the GLmed and GLmin [153]. Van der Krogt et al. assert that the GLmed is crucial for the execution of proper gait [168], and Correa et al. and Valente et al. found the GLmed to be a main contributor to hip joint contact forces [151,152]. Gottschalk et al., however, declare that the TFL fundamentally controls the weight-bearing process during walking and that the GLmed and GLmin mainly stabilize the hip during early and mid to late gait, respectively [158]. Anderson et al. call attention to the identification of the correct muscle forces due to their significance in computational models, as MSMs express the estimated contribution of each muscle in the emergence of ground reaction forces [153]. Yet, the fact that musculoskeletal geometry is very individual complicates modelling computed simulations [372]. Frigo and Crenna lay emphasis on the fact that contemporary models are unable to factor in the inter-individual variety of anthropometric and functional specifications [179]. Capturing individual muscle features such as fiber length and type with the intent of determining in vivo muscle force is too invasive a procedure to be performed as a matter of usual practice [132,172,194,336]. But innovative technological solutions to this issue might render possible the non-invasive assessment of muscle properties on a larger scale [373,374]. The discussion of these different aspects highlights the pertinence of choosing appropriate properties for MSMs and further underlines the relevance of extended in vivo validations in a clinical setting [172,192].

4.6 Limitations

Several limitations of this study can be acknowledged and shall be addressed. This investigation presents the kinematic measurements and electrophysiological recordings of an internationally unprecedented number of individuals with instrumented hip joint implants. Yet, the limited quantity of patients also confines the findings of this retrospective analysis to being exploratory in nature and the results regarding our hypotheses as descriptive rather than conclusive.

Despite efforts to minimize inter-individual differences by attempting to provide for an optimal measurement setup and testing protocol, the recordings produced quantitative and qualitative evidence with relatively high inter-individual variability. On the one hand, this might be because the data were not corrected for the potential influence of age, BMI, and sex on muscle status, kinematics, muscle activity, and contact forces [197,219-221,223-229]. On the other hand, however, a sample size of nine subjects is unlikely to yield consistent results across a variety of ADLs, especially when taking into account that even sample sizes markedly larger than the cohort in this study bring forth great variability in testing results [230].

51 months after hip replacement is the only point in time at which comprehensive data of three-dimensional motion capture and sEMG data in synchronicity with concomitant in vivo joint hip loads and contemporary CT scans of our THA patients' operated hip were collected. In view of these circumstances, there is no complete functional baseline data set of our patients before they underwent surgery, so interpreting the data in contrast with earlier postoperative intervals was not feasible. There were also no corresponding measurements of the contralateral hip or a reference group to compare the recordings of the ipsilateral hip to. Instead of being restricted by the outcome of a cross-sectional analysis of exclusively the operated hip of THA patients, the inclusion of earlier postoperative data acquisition times, the contralateral hip, and asymptomatic individuals as controls may have strengthened the findings and shed light on the functional progress of recovery from THA. Nevertheless, the kinematic and electrophysiological recordings available compare well with the postoperative measurements of other studies evaluating the functional outcome of THA patients with a longstanding rehabilitation period.

In spite of the fact that joint moments may give information on muscular contribution to the formation of joint contact forces [149,166,235,375,376], that joint moments were found to produce considerably different loading profiles across a variety of ADLs [230], and that the various single force components acting upon the hip joint do not always reach their respective maximum or minimum around the instant of the F_{res} [137], joint moments or the separate constituents of the in vivo F_{res} were not assessed. Analogous measurements generated large quantities of in vivo kinetic, kinematic, and electrophysiological data on an unprecedented number of locomotor and non-locomotor ADLs. Given the complexity of placing the results of this analysis in the context of the existing literature, incorporating

individual hip moment and force components into this study would have gone beyond the scope of this investigation.

4.7 Outlook on further research

Partially owed to the limitations of this study, a causal link between hip abductor muscle impairment and altered in vivo hip joint loads through kinematics and electrophysiological activity was not established. The multifaceted interconnectedness of muscle deterioration and the in vivo F_{res} is too complex an issue to be exhausted within the context of a single investigation. However, this study confirms the notion that an impairment of the hip abductor musculature has an impact on hip joint kinematics and GLmax, GLmed, and TFL activity. In turn, the compressive forces within the hip joint are largely dependent on the hip movement patterns and the electrophysiological activity of the hip abductors emerging during the performance of locomotor and non-locomotor ADLs. This analysis revealed certain elements which require consideration by investigators in future projects.

On the grounds that no preoperative measurements on hip joint kinematics and hip abductor muscle activity were acquired and because extensive longitudinal data and analogous recordings of asymptomatic individuals are unavailable thus far, the long-term functional outcome of THA patients and their situation relative to a reference group or their presurgical baseline condition cannot be tracked. Therefore, prospective investigations should include individual baseline data and measurements from earlier postoperative points in time as well as involve a healthy age-matched control group. In doing so, an extensive functional improvement may be assessed over a protracted period of time and evaluated in comparison to the situation of an asymptomatic reference group.

Furthermore, THA patients were found to offset muscle weakness by adopting compensatory strategies in the execution of locomotor and non-locomotor ADLs [55,118,149,155,166,232,237,261,267,358]. Despite the successful alleviation of pain and the absence of severe functional impairments as present in end-stage OA, these postoperatively persisting counterbalancing techniques involve the development and/or progression of all manner of asymmetries between the operated and non-operated limb [56,155,232,236,238,260,286,341,346,347,352,354,357,358,361]. In order to clinically address potential interlimb asymmetries in temporospatial characteristics, kinematic parameters, electrophysiological recordings, and weight-bearing measurements, forthcoming studies

should further collect longitudinal data of both the affected and unaffected limb.

Assessing the muscle status of a specific muscle group or selected single muscles alone may not be sufficient when evaluating the impact of muscle impairment on the functional outcome following THA. The quality of muscular examinations may be enhanced by factoring in the influence of muscles directly involved in certain tasks under investigation not as agonists but as antagonists. This is specifically relevant for compound movements, in which the complete motion is performed by muscles operating within kinetic/kinematic chains that are vital components of virtually all ADLs [122,149,154-157,377]. For this reason, the muscles assessed for their structural status and electrophysiological activity should be extended to also include antagonist muscles in future research.

By the same token, various publications have identified co-contractions to assist in the stabilization of and impact on the overall compressive forces acting on joints [136-138,150,191,259,348,378]. An increased presence of co-contractions, however, may be indicative of gait disturbances, suggesting improper muscle activity and/or muscle weakness [179,191,378]. This investigation did not examine the influence of agonist-antagonist muscle activity, which is why the role of muscle co-contractions in the formation of in vivo hip joint loads remains unclear. Upcoming projects should implement the electrophysiological signals of the antagonist musculature in order to clarify the functional capacity of muscle co-contractions.

In addition to the above suggestions, a number of critical questions and particular issues arose throughout the course of this study, and they could not be answered or solved with the data at disposal. Nonetheless, the data supplement fundamental information, which may be applied in the validation of subject-specific MSMs our research group is currently working on. The validation of these computational models against in vivo measurements substantiates their findings and will enable investigators to engage in matters pertaining to the effects of, for instance, muscle force [52,133,148,192,193,259,305,339,371].

Besides, this study's findings grant a factual foundation against which researchers and clinicians alike may track and oversee the success or ineffectiveness of a rehabilitative measure or other medical procedures in patients who had a long-term recovery period after having undergone THA.

Lastly, and after thorough review of the existing literature, the following observations about THA patients can be made:

- The functional outcome following THA appears to be contingent upon both acute soft tissue trauma induced surgically and chronic muscle deterioration dating back to the period before having undergone THA.
- Kinematic abnormalities and electrophysiological irregularities seem to emerge from a combination of compensatory motion patterns and muscle weakness.
- Compensatory motion patterns are likely a product of habitual pain-avoidance strategies and neuromuscular reorganization.
- Muscle weakness and poor muscle quality conceivably originate from a conjunction of muscle-disuse atrophy and iatrogenic muscle damage.
- The macroanatomical determination of muscle status reveals a redeployment of muscle volume in the hip abductor group. Yet, in order to conclusively clarify the state of muscle quality a microanatomical analysis involving an assessment of muscle fiber types would have to be performed.

The questions these observations give rise to shall be answered within the framework of further research.

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Statutory declaration

“I, Hilmi Cem Dinc, by personally signing this document in lieu of an oath, hereby affirm that I prepared the submitted dissertation on the topic “The Impact of Hip Abductor Muscle Status on In Vivo Joint Loads through Kinematics and Muscle Activity 51 Months Following Total Hip Arthroplasty” (title in German: “Der Einfluss von Muskelstatus auf die In-vivo-Gelenkbelastungen durch Kinematik und Muskelaktivität 51 Monate nach Implantation einer Hüfttotalendoprothese”) independently and without the support of third parties, and that I used no other sources and aids than those stated.
All parts which are based on the publications or presentations of other authors, either in letter or in spirit, are specified as such in accordance with the citing guidelines. The sections on methodology (in particular regarding practical work, laboratory regulations, statistical processing) and results (in particular regarding figures, charts and tables) are exclusively my responsibility.

Furthermore, I declare that I have correctly marked all of the data, the analyses, and the conclusions generated from data obtained in collaboration with other persons, and that I have correctly marked my own contribution and the contributions of other persons (cf. declaration of contribution). I have correctly marked all texts or parts of texts that were generated in collaboration with other persons.

My contributions to any publications to this dissertation correspond to those stated in the below joint declaration made together with the supervisor. All publications created within the scope of the dissertation comply with the guidelines of the ICMJE (International Committee of Medical Journal Editors; www.icmje.org) on authorship. In addition, I declare that I shall comply with the regulations of Charité – Universitätsmedizin Berlin on ensuring good scientific practice.

I declare that I have not yet submitted this dissertation in identical or similar form to another Faculty.

The significance of this statutory declaration and the consequences of a false statutory declaration under criminal law (Sections 156, 161 of the German Criminal Code) are known to me.”

Date Signature

Curriculum vitae

Mein Lebenslauf wird aus datenschutzrechtlichen Gründen in der elektronischen Version meiner Arbeit nicht veröffentlicht.

Acknowledgements

At this point, I would like to thank everyone who made it possible for me to take up work on this dissertation and who supported me in any way during its completion.

First and foremost, I want to thank all the patients who are part of this study, as the knowledge gained would not have been attainable without their cooperation and consent.

I would like to thank Professor Dr. Tobias Winkler for his guidance and for giving me the opportunity to complete my doctoral thesis at the intersection of musculoskeletal surgery and biomechanics.

I would also like to thank Dr.-Ing. Philipp Damm for his supervision and professional insight throughout the process.

Special thanks go to Dr. rer. nat. Alwina Bender, Lorenz Schmidt-Bleek, and Vivian Waldheim for their time and patience while helping me to get acquainted with the software and hardware necessary for data acquisition and processing. The countless hours in the computer laboratory would not have been the same without their company and support.

The conversations with Dr. rer. medic. Adam Trepczynski added to my biomechanical understanding, and I thank him for sharing his expertise with me.

I am also extending my thanks to Sónia Alves for the interdisciplinary exchanges we had.

I wish to express my sincerest gratitude to my beloved father, who encouraged me to become a physician, and to my grandfather, whose legacy inspired me to write this dissertation.

From start to finish, my dear partner was always the puffer I needed, and my appreciation for her is beyond words.