BJR

RESEARCH

Three-dimensional knee kinematic analysis during treadmill gait

SLOW IMPOSED SPEED *VERSUS* NORMAL SELF-SELECTED SPEED

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Objectives

Whilst gait speed is variable between healthy and injured adults, the extent to which speed alone alters the 3D *in vivo* knee kinematics has not been fully described. The purpose of this prospective study was to understand better the spatiotemporal and 3D knee kinematic changes induced by slow compared with normal self-selected walking speeds within young healthy adults.

Methods

A total of 26 men and 25 women (18 to 35 years old) participated in this study. Participants walked on a treadmill with the KneeKG system at a slow imposed speed (2 km/hr) for three trials, then at a self-selected comfortable walking speed for another three trials. Paired *t*-tests, Wilcoxon signed-rank tests, Mann-Whitney U tests and Spearman's rank correlation coefficients were conducted using Stata/IC 14 to compare kinematics of slow *versus* self-selected walking speed.

Results

Both cadence and step length were reduced during slow gait compared with normal gait. Slow walking reduced flexion during standing (10.6° compared with 13.7°; $p < 0.0001$), and flexion range of movement (ROM) (53.1° compared with 57.3°; $p < 0.0001$). Slow walking also induced less adduction ROM (8.3° compared with 10.0°; $p < 0.0001$), rotation ROM (11.4° compared with 13.6°; $p < 0.0001$), and anteroposterior translation ROM (8.5 mm compared with 10.1 mm; $p < 0.0001$).

Conclusion

The reduced spatiotemporal measures, reduced flexion during stance, and knee ROM in all planes induced by slow walking demonstrate a stiff knee gait, similar to that previously demonstrated in osteoarthritis. Further research is required to determine if these characteristics induced in healthy knees by slow walking provide a valid model of osteoarthritic gait.

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Keywords: Knee, Gait kinematics, Speed

Article focus

- This study aimed to determine the spatiotemporal and kinematic differences induced by a slow imposed walking speed compared with normal walking in a healthy young population.

Key messages

- Imposed slow walking reduces spatiotemporal and kinematic measures compared with normal walking.
- Knee flexion at stance phase is reduced during slow walking.
- Reduced range of movement is detected in all planes, as with osteoarthritic gait.

Strengths and limitations

- This is the first study to measure both the spatiotemporal and 3D kinematic impact of slow imposed walking speed in healthy young adults.
- This measured effect of speed on kinematics potentially provides a target reference for the expected recovery from slow gait in a wide range of knee pathologies to a more physiological profile.
- Limitations: the effect of speed on knee forces was not assessed. Long-term studies utilising force plates will allow detection of both the kinematic and kinetic alterations induced by slow speed.

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Introduction

Historically, tools used to assess knee joint function, such as clinical examination and radiological evidence of disease, are both subjective and based on clinician experience. Furthermore, these modalities do not assess knee function in its most natural weight-bearing state. The evaluation of clinical pathomechanics and the outcomes of interventions, such as total knee arthroplasty for knee osteoarthritis (OA),[1] require consistent and accurate pre- and post-interventional evaluation of knee movement.

Many patient-reported outcome measures (PROMs), such as the Western Ontario and McMaster Universities Osteoarthritis Index[2] and the Knee Osteoarthritis Outcome Score (KOOS),[3] provide standardised and subjective assessment of joint function. However, these PROMs have limitations, such as ceiling effects and functional deficiencies.[4,5] Since the link between knee pathomechanics and degenerative joint pathology has been recognised,[6] gait analysis provides accurate and objective information about joint function for diagnosis and monitoring during the recovery of knee conditions.[6-10]

Gait has been characterised previously in both healthy and OA populations.[8,11] However, healthy controls had a mean age of approximately 62 years in one study[8] and 59 years in another study,[11] which may present a confounding pre-clinical pathological gait in these older populations. Furthermore, while some studies have analysed the effect of speed on spatiotemporal and sagittal kinematics,[12,13] no known studies have assessed the effect of a slow imposed speed on 3D knee kinematics in a normal young healthy population. Characterising this would help us to understand better the impact of knee injuries or degenerative pathologies that inevitably induce slow gait. Comparing the knee kinematics of slow gait with those of normal gait may also provide a target reference for the expected recovery from slow gait, observed in a wide range of knee pathologies,[14,15] to a more physiological profile.

Therefore, the aim of this study was to determine the effect of a slow imposed speed (2 km/hr) on knee kinematics in a normal healthy population, with self-selected comfortable walking as the control. The primary outcome measures included assessing the effects on cadence, step length, flexion during stance, maximum flexion, flexion range of movement (ROM), adduction ROM, rotation ROM, and translation ROM. Our hypothesis was that healthy subjects will exhibit a reduced ROM in all planes (sagittal, coronal and transverse) at a slow walking speed, compared with the self-selected comfortable walking speed.

Materials and Methods

This study received ethics approval as low risk research under the local Human Research and Ethics Committee (LRR 205/15). Subjects were recruited from the authors' university, through an initial lecture presentation outlining the study and then a follow-up email call-out. All subjects participated on a voluntary basis, and provided informed written consent.

To be included in the study, participants were between the ages of 18 years and 35 years, and were familiar with the use of a treadmill. Candidates completed an initial KOOS questionnaire, which screens for evidence of knee orthopathology. The KOOS questionnaire provides a total score between 0 and 100 based on pain, symptoms, activities of daily living, sport, and quality of life, and has been extensively validated.[3,4] A KOOS score of $\geqslant 90$ was sufficient for inclusion in the study.

Potential candidates were excluded if they had previous lower limb surgery, history of chronic ongoing lower limb disease or complications, current knee joint pain (self-reported $>$ 4/10 daily, on visual analogue scale) during activity or at rest, neuropathy or arthritis. Two candidates were excluded from treadmill analysis due to KOOS scores $<$ 90. Three participants were excluded from statistical analysis due to difficulties with the 3D knee kinematics device attachment.

Materials. Shorts were required to be worn to allow attachment of the 3D knee kinematics device. A NordicTrack T13.5 treadmill (2.75 CHP motor) was used (NordicTrack, Sydney, Australia), which measures 51 cm $\times$ 152 cm (width by length).

The KneeKG system (Emovi Inc., Montreal, Canada) was used to measure 3D knee kinematics non-invasively. Development of the KneeKG system originated at the Imaging and Orthopaedics Research Laboratory in Montreal, Canada,[16] in order to reduce the skin movement artefact.[17] Validation studies have proven the KneeKG system to be a useful and reliable kinematic analysis tool.[16,18]

The KneeKG system uses a tibial tracker, attached on the anteromedial aspect, an exoskeletal brace for the femoral tracker (Fig. 1) and a sacral belt. An infra-red camera (Polaris Spectra; Northern Digital Inc., Quebec, Canada) monitors the 3D position of the trackers at a rate of 60 Hz, and a computer enabled with Knee3D suite (Version 2.73.117.1898; Emovi, Inc.) stores 100 data points per dimension (sagittal, coronal, transverse, and anteroposterior translation), for each gait cycle.

Procedure. Height and weight measures were initially recorded. The tibial tracker was then attached on the anteromedial aspect of the tibia. The femoral tracker was placed laterally between the biceps femoris tendon and the iliotibial band, and fixed medially between the adductor tubercle and the posterior aspect of the adductor magnus tendon, as described by Sati et al.[16] Finally, the sacral belt was positioned at the midpoint between the posterior superior iliac spines.

The examiner then completed initialisation, calibration and acquisition of data, previously described by

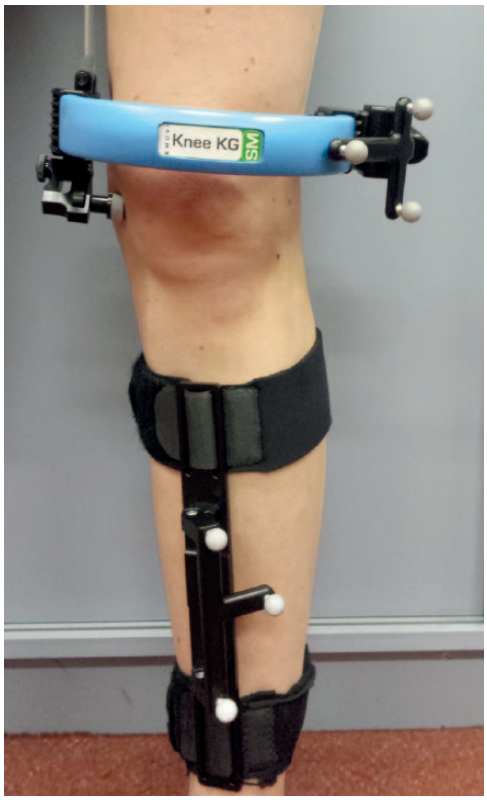

Fig. 1

Photograph showing tibial and femoral trackers positioned for left knee.

Hagemeister et al.[19] The initialisation phase defines the plane of the treadmill using the infra-red reflector probe. The patient calibration method requires the examiner to mark the medial and lateral malleoli in order to define the ankle joint centre. The midpoint of the knee was marked by the medial and lateral femoral epicondyles, and then participants performed a repetitive knee flexion/extension movement for ten seconds, which calculates a mean flexion/extension axis onto which the knee's midpoint is projected to form the knee joint centre. A functional method is used to define the 3D position of the femoral head via a circumduction movement for five seconds, such that the points trace a cone in the coordinate system, the apex of which is the hip joint centre. A slight flexion-hyperextension movement is performed to determine the neutral rotation of the knee at 0° of flexion.

A period of two minutes was allowed for familiarisation with the use of the treadmill. Data were acquired while participants walked barefoot for three trials (45 seconds per trial) at a slow imposed speed (2 km/hr, 0.56 m/sec), the speed also used by Bejek et al[20] in their study assessing walking kinematics and then walked for another three trials at their own self-selected normal speed. This was achieved by having the participant increase the speed in increments of 0.2 km/hr until they reached a comfortable speed, however, in order to eliminate subject bias the speed was not displayed. The device was then removed and the process was repeated on the other knee. Participants completed their session within 45 minutes to 60 minutes. To eliminate bias induced by multiple observers, the same examiner completed every step for each participant.

Spatiotemporal outcomes. The outcomes recorded were participant age, gender, height, weight, preferred speed, cadence, step length, and KneeKG system setup time. Cadence (number of steps per minute) was calculated from the number of cycles walked in the chosen trial (over 45 seconds). Step length (metres per step) was determined from the cadence and the speed using the following equation:[21]

$$Step\ length\ (metres.step^{-1}) = \frac{Speed\ (metres.sec^{-1})}{Cadence\ (steps.minute^{-1})}.60$$

The setup time of the KneeKG system was defined as the start of application of the tibial tracker to the completion of the software calibration phase.

Feedback outcomes. After kinematic data collection, participants completed a feedback sheet reporting on their level of comfort with the KneeKG system (from 0, no discomfort, to 4, severe discomfort) and their level of treadmill experience (from 0, no experience, to 4, weekly use).

Kinematic outcomes. Data regarding sagittal (flexion/extension), coronal (abduction/adduction), axial (internal/external rotation) planes and translational (anteroposterior (AP) movement of the femur relative to the tibia) were acquired. Analysis was conducted by comparing every percentile of the gait cycle between slow and normal walking. This provided data for Figures 2, 3 and 4, which depict the knee angle from 1% to 100%. However, when focusing on key parts of the cycle, each cycle was separated into eight 'zones' as defined in Table I. Data from the dominant knee for each participant were used for the comparisons of spatiotemporal data.

Statistical analysis. The required sample size ($\alpha < 0.05$; power 80%; difference in means, 1°, assuming standard deviations (SD) are 4.5°, as reported by Shabani et al,[10] and equal between groups) was a minimum of 34 subjects. Kinematic data that satisfied the Shapiro-Wilk test for normality were assessed using paired *t*-tests. Paired kinematic data that did not satisfy normality were assessed using the Wilcoxon signed-rank test. Maximum adduction during swing, adduction ROM, external rotation at heel-strike, internal rotation at push-off, and anteroposterior ROM were the variables that did not satisfy normality, and hence were assessed using the Wilcoxon signed-rank test.

Unpaired data were assessed using the Mann-Whitney U test, also known as the Wilcoxon rank-sum test. This does not require the assumption of normal distributions and was applied to demographic data (age, weight,

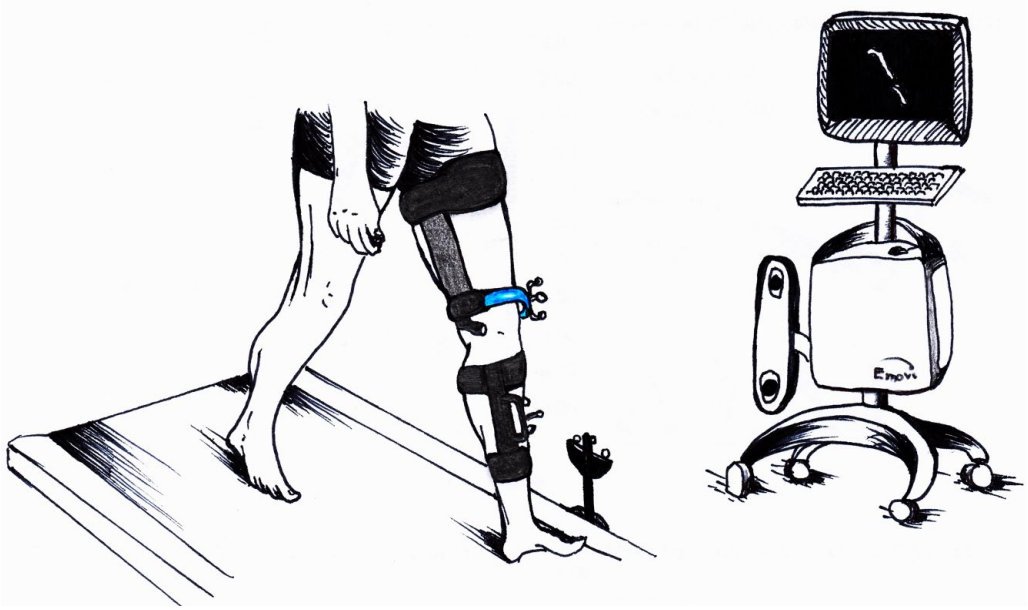

Fig. 2

KneeKG system attachment braces, reference tracker and computer enabled with Knee3D Suite.

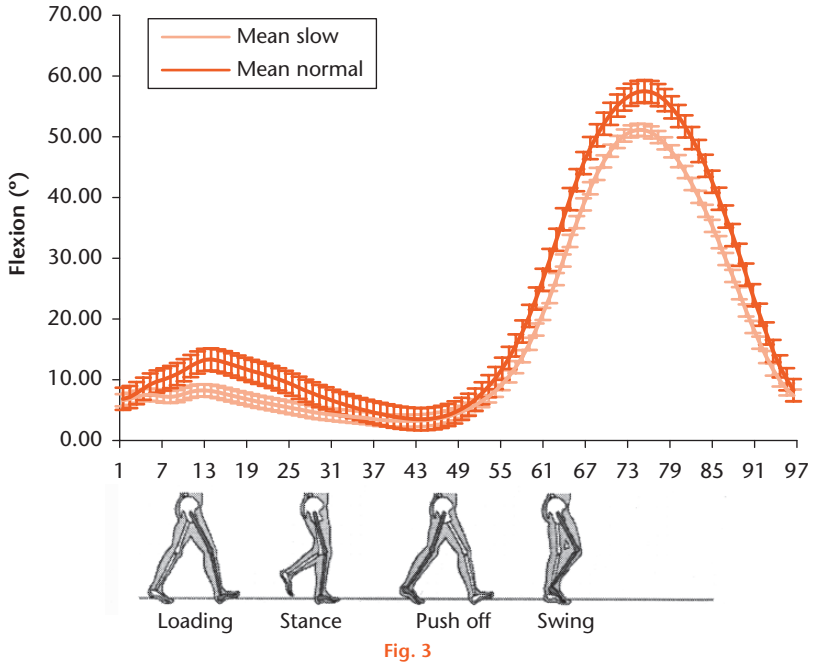

Fig. 3

Mean flexion with standard error of the mean bars.

height, body mass index (BMI), KOOS scores, length of setup time and treadmill experience). Spearman's rank correlation coefficients were used to determine the relationship between spatiotemporal and kinematic features of normal walking. The level of statistical significance was set at $p < 0.05$. Kinematic graphs were plotted in Excel 2010 (Microsoft, Armonk, New York) and statistical analysis was conducted using StataCorp 2015 (Stata Statistical Software; StataCorp LP, College Station, Texas).

Results

Study sample. A total of 51 university students (26 men, 25 women) with healthy knees participated fully in this study. The overall characteristics of the included participants were (mean and SD): age 23.76 years (SD 3.52); height 173 cm (SD 8); body weight 69.34 kg (SD 11.27); and BMI 23.11 kg/m^2 (SD 2.71) (Table II).

Spatiotemporal measures. Participants walked at a mean comfortable walking speed of 0.97 m/sec (SD 0.20; 0.64

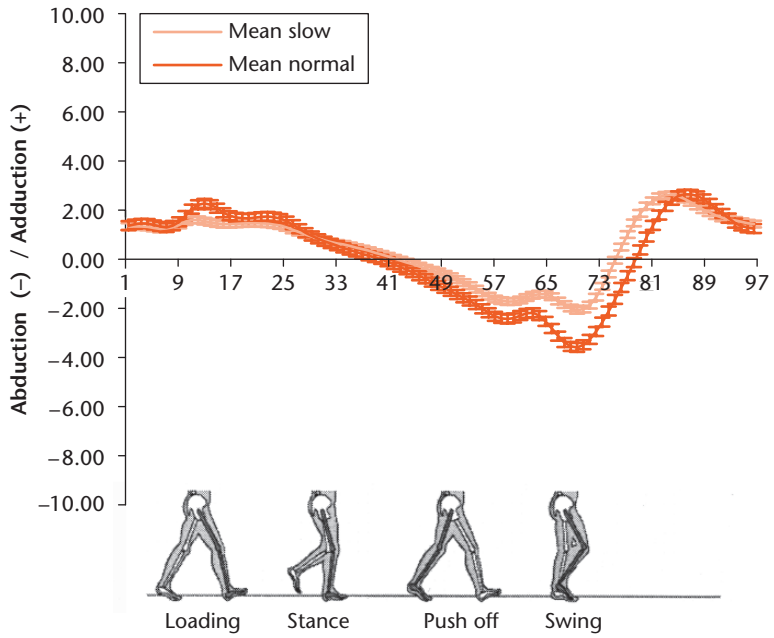

Fig. 4

Mean adduction with standard error of the mean bars.

Table I. Kinematic zones

Zone	% of gait cycle	Phase
1	*1* to *10*	Heelstrike
2	*11* to *30*	Mid-stance
3	*31* to *50*	Terminal stance
4	*51* to *60*	Push-off
5	*61* to *100*	Swing
6	*1* to *60*	Stance phase
7	*11* to *50*	Support
8	*1* to *30*	Loading extended

to 1.61), an increase of 173% from the slow imposed speed. The cadence (steps/min) at slow speed (39.3) compared with at normal speed (47.8) was significantly less ($p < 0.0001$). Step length was also less during slow speed (0.86 m SD 0.1, compared with 1.2 m SD 0.2, $p < 0.0001$) (Table III).

The maximum flexion during swing phase was significantly lower when participants walked at slow speed (54.0° SD 7.2°), compared with normal speed (58.0° SD 5.7°, $p < 0.0001$) (Fig. 2). Significant differences ($p < 0.05$) were sustained over the swing phase from 55% ($p = 0.018$) to 99% ($p = 0.003$) of the gait cycle. Maximum flexion during stance was significantly less at slow speed compared with normal speed (10.6° SD 6.4° and 13.7° SD 6.8° respectively, $p < 0.0001$). Significant differences during the stance phase were sustained over 5% ($p = 0.011$) to 37% ($p = 0.04$) of the gait cycle. No significant differences were detected between the left and right knees for slow compared with normal walking.

Maximum adduction during stance phase was significantly less at slow speed (2.0° SD 3.2°) than at normal speed (2.6° SD 3.7°, $p = 0.0025$) (Fig. 3). Maximum abduction during swing phase was also significantly less at slow imposed speed than at normal speed (-4.7° SD 4.4° and -6.0° SD 5.6° respectively, $p = 0.0001$).

Internal rotation at push-off, using the Wilcoxon signed-rank test, was significantly less at slow speed (-6.0° SD 3.4°) than at normal speed (-7.1° SD 4.1°, $p = 0.0002$) (Fig. 4).

ROM for anteroposterior translation, using the Wilcoxon signed-rank test, was significantly less during slow (8.5 mm SD 3.1) compared with normal (10.1 mm SD 3.8) walking speed ($p < 0.0001$) (Fig. 5).

Anthropometric correlations at normal walking speed. High correlations were found between speed, cadence and step length. Flexion ROM most positively correlated with step length ($r = 0.31$, $p = 0.028$) and speed ($r = 0.27$, $p = 0.053$). Rotation ROM also positively correlated with adduction ROM ($r = 0.32$, $p = 0.022$) (Table IV) at normal walking speed.

Discussion

The most important finding of this prospective study was that participants walking at the slow imposed speed recorded significantly reduced flexion during stance and swing phases of the gait cycle. Adduction during stance phase, swing phase and ROM, as well as internal rotation at push-off and ROM, were all significantly reduced at slow walking compared with the self-selected gait speed. This confirmed our hypothesis that walking at a slow imposed speed results in significantly less ROM in all planes compared with normal walking.

Our study indicated that slow walking creates a reduction in knee flexion during stance, beginning at heelstrike, to a more pathological profile, similar to OA.[8,11,13,20]

Table II. Patient demographics

Characteristic (Mean, SD)	Men	Women	p-value†
Age (yrs)	24.3 (4.0)	23.2 (3.0)	0.26
Height (cm)	177 (6)	168 (7)	< 0.001*
Weight (kg)	74.1 (12.5)	64.4 (7.2)	0.001*
BMI (kg/m^2)	23.5 (3.2)	22.7 (2.1)	0.46
KOOS score (total)	98.1 (2.7)	98.5 (1.7)	0.88
Device discomfort stationary	1.4 (1.0)	1.2 (0.9)	0.65
Device discomfort walking	1.3 (0.8)	1.2 (0.8)	0.44
Treadmill experience	1.9 (0.9)	2.4 (1.0)	0.053
KneeKG system setup time (mins)	9.9 (4.50)	9.1 (3.1)	0.47

†Mann-Whitney *U* test; BMI, Body Mass Index; KOOS, Knee Osteoarthritis Outcome Score; SD, standard deviation; *statistically significant.

Table III. Spatiotemporal data and kinematic 'zones' for slow *versus* normal walking

Mean (SD)	Slow speed	Normal speed	p-value*
Cadence (steps/min)	39.3 (4.0)	47.8 (4.0)	< 0.0001‡
Step length (m)	0.86 (0.1)	1.2 (0.2)	< 0.0001‡
Flexion at heel-strike (°)	6.9 (5.2)	5.7 (5.3)	0.02‡
Max. flexion during stance (°)	10.6 (6.4)	13.7 (6.8)	< 0.0001‡
Max. flexion during swing (°)	54.0 (7.2)	58.0 (5.7)	< 0.0001‡
Max. extension (°)	0.9 (5.4)	0.7 (5.3)	0.69
Flexion-extension ROM (°)	53.1 (8.3)	57.3 (6.6)	< 0.0001‡
Adduction (+) at heel-strike (°)	0.6 (3.0)	1.0 (3.3)	0.04‡
Max. adduction during stance (°)	2.0 (3.2)	2.6 (3.7)	0.0025‡
Min. adduction during swing (°)	-4.7 (4.4)	-6.0 (5.6)	0.0001‡
Max. adduction during swing†	3.5 (4.3)	4.0 (5.0)	0.02‡
Adduction ROM† (°)	8.3 (2.8)	10.0 (3.2)	< 0.0001‡
Ext. rotation at heel-strike† (°)	1.7 (3.0)	2.3 (3.8)	0.01‡
Int. (-) rotation at push-off† (°)	-6.0 (3.4)	-7.1 (4.1)	0.0002‡
Ext. (+) rotation during swing (°)	5.1 (4.4)	6.1 (4.9)	0.016‡
Rotation ROM (°)	11.4 (3.5)	13.6 (3.9)	< 0.0001‡
AP translation ROM (mm)† (°)	8.5 (3.1)	10.1 (3.8)	< 0.0001‡

*paired *t*-test (unless otherwise specified)
†Wilcoxon signed-rank test;
‡statistically significant
SD, standard deviation; ROM, range of movement; Int, internal; Ext, external; AP, anteroposterior; Min, minimum; Max, maximum

Blunting of the knee flexion at stance during slow walking may highlight the absence of a physiological compensatory mechanism in this group; the increased knee flexion at stance phase for higher speeds may allow the spread of knee forces over a greater region of tibiofemoral cartilage. Lelas et al[13] also hypothesise that this knee flexion during stance is required for greater "shock absorption". What seems to aid this is the lower leg inertia observed at higher speeds. Indeed, multiple studies have shown that knee flexion positively correlates with gait speed.[13,22,23]

Moreover, there is a recognised need in the literature to correct abnormal gait kinematics via compensatory mechanisms. Fuentes et al[24] described a "pivot-shift avoidance gait" to avoid rotatory instability in anterior cruciate ligament-deficient patients, at both comfortable and fast speeds. In patients without such mechanisms, the current framework suggests that persistent shifts in intra-articular forces cause cartilage homeostasis to spiral towards cartilage degeneration.[6,7] This is particularly pertinent for patients known to have cruciate ligament injuries whereby the ligamentous instability of the knee, slow gait speed, and the uneven distribution of knee forces may trigger this progression.[10,25-27]

We found that there is a significantly greater maximum adduction angle at higher speeds during the stance phase. Since they are both formulaically related, and since positive correlations are found in the literature,[28,29] adduction angle may be a reasonable surrogate marker of medial knee load. While this may explain why compensatory mechanisms are necessary at higher speeds, without instrumented force plates it is difficult to ascertain the true relationship between speed and increased knee load in our subjects.

The reduced knee ROM in all planes at the slower speed demonstrates a similarity to 'stiff knee' gait, also seen in OA.[8] While the definition of 'stiff knee' gait generally implies a significant reduction in knee flexion during swing phase,[14] studies have shown that OA, similarly to slow walking, not only induces reduced ROM in the sagittal plane, but also in the coronal and transverse planes.[8,30,31] However, without longitudinal studies the temporal relationships between speed, kinematics and orthopathology remain undefined. What also remains unknown is whether the features of pathological gait in OA are an inevitable biomechanical consequence of slow walking,[8,11,13,20] rather than the presence of any

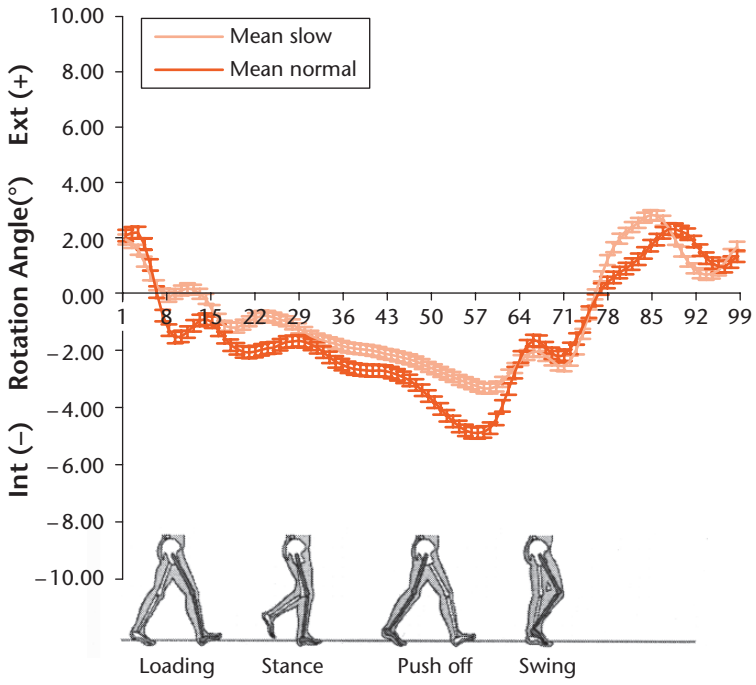

Fig. 5

Mean rotation with standard error of the mean bars.

Table IV. Anthropometric *versus* kinematic correlation coefficients (r) and range of movement (ROM) for normal walking (Spearman's rank correlation coefficients)

	Speed	Cadence	Step length	Flex ROM (°)	Add ROM (°)	Rot ROM (°)	Trans ROM (°)
Speed (m/sec)	1.00						
Cadence (steps/min)	0.73*	1.00					
Step length (m)	0.86*	0.34*	1.00				
Flex ROM (°)	0.27	0.02	0.31*	1.00			
Add ROM (°)	-0.09	-0.03	-0.11	0.09	1.00		
Rot ROM (°)	0.05	0.004	0.15	0.07	0.32*	1.00	
Trans ROM (°)	0.0001	0.15	-0.09	-0.001	0.17	0.24	1.00

*statistically significant ($p < 0.05$)
Flex, flexion; Add, adduction; Rot, rotation; Trans, anteroposterior translation

underlying joint pathology. If most of the kinematic changes in these patients are due to slower walking, rather than arthritis directly, as was hypothesised by Zeni and Higginson,[11] then slow imposed walking in normal subjects may provide a valid model for OA gait. However, more studies would be needed to fully validate if this were a working phenomenon.

There are several limitations identified within this prospective study. First, without kinetic data, it is difficult to extrapolate the compensatory mechanisms that may be observed at higher speeds. Secondly, there is likely to be overestimation of knee joint movement due to soft-tissue micro-movement. Thirdly, participants reported discomfort with the femoral brace which may have affected the results. The alternative is to use skin-mounted trackers but these are known to have unacceptable levels of measurement error, especially of the frontal and axial planes.[32,33] Both the hardware, such as a 'knee sleeve'[34] instead of a brace, and software should be addressed in future gait analysis devices to accommodate more adequately for artefact and variability in subject habitus.

In conclusion, our study showed that slow imposed gait speed reduced the knee ROM in sagittal, coronal and transverse planes, and in flexion during stance, compared with a normal self-selected speed in a young healthy population. Comparing knee kinematics of slow gait to those of normal gait has potentially provided a target reference for the expected recovery from slow gait in injured knees to a more physiological profile. Longitudinal studies would allow monitoring of recovery from slow gait induced by injury, and may potentially characterise timeframes at which early gait adaptation may prevent progression to OA.

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Author Contributions

- N. Mannering: Developed research design, Introduction, Outcomes and discussion, Principle examiner for each participant.
- T. Young: Technical assistance, Critically read, revised and approved the submission of the final manuscript.
- T. Spelman: Statistical analysis for the results, Critically read, revised and approved the submission of the final manuscript.
- P. F. Choong: Senior research coordinator, Critically read, revised and approved the submission of the final manuscript.

Conflicts of Interest Statement

- The authors declare that they have no conflict of interest.