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Abstract: Background: Although approximately one-third of stroke survivors suffer abnormal foot posture and this can influence mobility, there is very little objective information regarding the foot and ankle after stroke.

Objective: As part of a programme of research examining foot and ankle biomechanics after stroke, we investigated multi-planar kinematics and the relationship with function.

Methods: In a single assessment session, static foot posture (Foot Posture Index); mobility limitations (Walking Handicap Scale) and multi-segment foot and ankle kinematics during stance phase of walking were measured in 20 mobile chronic stroke survivors and 15 sex and age-matched healthy volunteers.

Results: Compared to the healthy volunteers, the stroke survivors demonstrated consistently reduced range of motion across most segments and planes, increased pronation and reduced supination, disruption of the rocker and the timing of joint motion. Changes in pronation /supination were associated with limited walking ability.

Conclusions: Our findings highlight structural and movement deficiencies in the intrinsic foot segments and joints in all three planes which do not support common clinical practices that focus on correction of sagittal ankle deformity and assumed excessive foot supination. Some of these abnormalities were associated with limitation in functional ability. Biomechanical abnormalities of foot and ankle are modifiable and there is potential for clinical studies and future developments of interventions to help prevent or treat these abnormalities which may improve functional ability post stroke.

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Dear Sir/Madam

I would like to confirm that all authors were fully involved in the study and preparation of the manuscript and that the material within has not been and will not be submitted for publication elsewhere.

I would like also to confirm that there is no any commercial interest of the authors relevant to the subject of the manuscript.

Yours faithfully,

Dr Saeed Forghany

DECLARATION OF INTEREST: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

The effect of stroke on foot kinematics and the functional consequences

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Abstract

Background: Although approximately one-third of stroke survivors suffer abnormal foot posture and this can influence mobility, there is very little objective information regarding the foot and ankle after stroke.

Objective: As part of a programme of research examining foot and ankle biomechanics after stroke, we investigated multi-planar kinematics and the relationship with function.

Methods: In a single assessment session, static foot posture (Foot Posture Index); mobility limitations (Walking Handicap Scale) and multi-segment foot and ankle kinematics during stance phase of walking were measured in 20 mobile chronic stroke survivors and 15 sex and age-matched healthy volunteers.

Results: Compared to the healthy volunteers, the stroke survivors demonstrated consistently reduced range of motion across most segments and planes, increased pronation and reduced supination, disruption of the rocker and the timing of joint motion. Changes in pronation /supination were associated with limited walking ability.

Conclusions: Our findings highlight structural and movement deficiencies in the intrinsic foot segments and joints in all three planes which do not support common clinical practices that focus on correction of sagittal ankle deformity and assumed excessive foot supination. Some of these abnormalities were associated with limitation in functional ability. Biomechanical abnormalities of foot and ankle are modifiable and there is potential for clinical studies and future developments of interventions to help prevent or treat these abnormalities which may improve functional ability post stroke.

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Introduction

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5 Regaining safe, independent mobility is a priority for many stroke survivors and is a
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7 primary goal in stroke rehabilitation [1]. There is an extensive literature about the
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9 mechanisms of hemiplegic gait (via biomechanics) and the rehabilitation of walking
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11 after stroke, but this concentrates on hip and knee movements; there is a particular
12
13 paucity of information about foot and ankle function. As the foot is the only source of
14
15 direct contact with the ground, its function is important in weight bearing tasks and
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17 there is some evidence of foot and ankle problems after stroke: Foot deformities
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19 which could influence walking have been reported in ~50% of people with chronic
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21 stroke [2], while approximately 30% suffer abnormal, asymmetric foot posture while
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23 standing (with almost equal numbers of pronation and supination abnormality) [3],
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25 and foot posture abnormality is associated with walking limitations [3].
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34 Most literature on foot and ankle abnormalities to date focuses in the sagittal plane
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36 [4-7]. During the stance phase of walking, the most reported deviations at the ankle
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38 are initial contact with the foot (rather than the heel), reduced plantarflexion after
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40 initial contact, dorsiflexion during midstance and plantarflexion at toe off [8-10], while
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42 varus foot deformities are said to be common in swing phase [5]. However these
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44 descriptions are based on a single segment model of the foot and ankle (in which the
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46 foot and ankle are assumed to act as a single unit) and do not take movement in the
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48 frontal and transverse planes into account [5, 8]. This reliance on a single segment,
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50 uni-planar foot model ignores the coupling between foot joints and movements
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52 across planes of motion [11] which are influenced by extrinsic and intrinsic multi-joint
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54 muscles that are affected by stroke [12], and evidence that stroke affects the
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1 rearfoot, midfoot and forefoot during walking [6,12,13]. This limited information
2 creates the risk of incorrect diagnosis and inappropriate clinical interventions. For
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4 example, using the single segment foot model to describe the commonly reported
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6 abnormality of a plantarflexed ankle (equinus) and excessive midfoot dorsiflexion
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8 (the so called midfoot break) may lead to motion at the tarso-metatarsal and midfoot
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10 joints to be interpreted as ankle motion, inferring use of an ankle-foot orthosis,
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12 whereas a foot orthosis would more effectively address abnormal intrinsic foot
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14 movements.
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22 Thus, we undertook a programme of research

- 23 • To examine foot and ankle biomechanics after stroke using a tri-planar multi-
24 segment model
- 25 • To explore the impact of any abnormalities on functional walking ability

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31 The findings regarding kinematics (movement patterns) are reported here. Data
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33 regarding the intrinsic and external forces (the kinetics), muscle activity and impact of
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35 neuromuscular impairments will be reported separately.
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41 **Methods**

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44 Subsequent to ethical approval from the University and National Health Service,
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46 stroke survivors who could walk independently with or without an assistive device for
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48 at least 10m were recruited from in and out-patient stroke services of the local
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50 hospital and a stroke support group. Healthy sex and age-matched (with 5 years)
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52 volunteers were also recruited from stroke participants' relatives and staff of the
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54 university and their relatives. For each participant, static foot posture (Foot Posture
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56 Index (FPI) [14]); mobility limitations (Walking Handicap Scale [15]); multi-segment
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1 foot and ankle kinematics and spatio-temporal gait parameters during stance phase
2 were recorded in a single measurement session at the University's gait analysis
3 clinic.
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9 A ten camera Qualysis Proreflex system (Qualisys Medical, 2003, 100Hz) was
10 used to obtain the kinematic data. Eighteen reflective markers were attached to the
11 forefoot, midfoot, rearfoot and shank on affected side of stroke survivors and the
12 same sides of healthy control group (Table 1). The Calibrated Anatomical System
13 Technique (CAST) was adopted to establish a suitably anatomical four segmental
14 model of foot and shank [16]. Participants walked barefoot along the walkway while
15 the Qualysis system tracked the movements of the reflective markers indicating the
16 movement of the foot and ankle segments. A minimum of ten walking trials were
17 collected. Data from the markers were smoothed (4th order Butterworth, low-pass
18 filter with 6 Hz cut off) and individual segment coordinate systems were defined using
19 the anatomical markers and joint centre calculations with the positive X-axis to the
20 right, positive Y-axis facing anteriorly, and positive Z-axis pointing superiorly. Vertical
21 velocity of the midpoint between the heel and toe markers derived the point of initial
22 contact (IC) and toe off (TO) of stance phase [17]. After detection of gait events,
23 processed kinematic data were normalised to 100 percent of stance phase to enable
24 averaging across trials. The variables measured were maximums, minimums and
25 range of motion of the rearfoot (i.e. the calcaneus relative to the shank), the midfoot
26 (i.e. the midfoot relative to the rearfoot) and the forefoot (i.e. the forefoot relative to
27 the midfoot) in the three anatomical planes (sagittal, transverse and frontal) for the
28 affected side of participants with stroke and the matched sides of healthy volunteers.
29 Angular rotations were derived for each trial (Visual3d software (C-motion, USA)).
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2 SPSS 16.0 was used to compare the data for the stroke and healthy control groups
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4 using independent t-tests. For clarity, only the comparisons which showed a
5
6 statistically significant difference are presented. Binary logistic regression determined
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8 whether kinematic abnormalities in the affected foot and ankle predict walking ability
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10 after stroke with functional walking ability (household or community level, measured
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12 by the Walking Handicap Scale [15]) as the dependent variable. The independent
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14 variables were the kinematic variables that showed significant differences between
15
16 the stroke and control groups ($p < 0.05$).
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25 **Results**

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27 Twenty stroke survivors were recruited; 7 men, mean age 65.0 ± 10.2 years, the
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29 right side was affected for 8 survivors and median time after stroke 6.9 months, IQR:
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31 10.4 months) and 15 healthy age and sex matched volunteers, which provided 20
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33 side-matched feet; 10 men, mean age 67.1 ± 8.6 years. The mean affected Foot
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35 Posture Index while standing of stroke survivors showed no overall difference to the
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37 healthy volunteers (2.5 ± 1.8 versus 3.4 ± 1.8 , $p = 0.17$) but the stroke survivors walked
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39 more slowly ($p < 0.001$, 0.77 ± 0.26 (95% CI 0.64 to 0.89) vs. 1.10 ± 0.14 (95% CI
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41 1.03 to 1.17) $\text{m}\cdot\text{s}^{-1}$) with a shorter stride length ($p < 0.001$, 0.92 ± 0.25 (95% CI 0.80
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43 to 1.03) vs. 1.24 ± 0.14 (95% CI 1.17 to 1.31) m) and spent approximately 29%
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45 longer in double limb support phase ($p = 0.007$, 0.25 ± 0.06 (95% CI 0.23 to 0.28) vs.
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47 0.36 ± 0.12 (95% CI 0.30 to 0.42) s). The stroke survivors were categorised into two
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49 groups according to their walking ability; household ($n=9$, 45%) or community
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51 walkers ($n=11$, 55%). Household walkers walked more slowly than the community
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53 walkers ($p < 0.005$, 0.60 ± 0.20 (95% CI 0.44 to 0.75) vs. 0.91 ± 0.23 (95% CI 0.75 to
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1.06) m.s⁻¹). The comparisons of foot kinematics are detailed below and the data for all significant comparisons are detailed in Table 2.

Whole foot movement (Table 2)

Kinematic patterns of the affected foot in the sagittal plane were similar to the control group with three distinct phases of dorsi/ plantarflexion (Figure 1A). However, the stroke survivors demonstrated less range of motion. In the first phase, they showed 1.8° less plantarflexion which was of shorter duration. In the second phase, the affected side exhibited a longer period of dorsiflexion but comparable range of movement and maximum dorsiflexion. The final phase of plantarflexion showed less overall movement and reduced maximum plantarflexion at toe off. In the frontal plane (figure 2A), the stroke and control groups showed three similar phases of inv/eversion. For the stroke group, the foot was slightly more everted during most of stance phase, although the differences were not significant. In the transverse plane (figure 3A), foot motion showed less similarity between the stroke and control groups than the sagittal and frontal planes. The general pattern was of abduction followed by adduction but the stroke group showed 4° less overall movement (primarily from reduced adduction) and later maximum abduction.

Rearfoot movement (Table 2)

Kinematic patterns of the affected rearfoot in the sagittal plane were similar to the control group with three phases of dorsi/ plantarflexion (figure 1B). Initial plantarflexion was reduced by 2.1° but the greatest differences occurred in the last phase of stance (the propulsion phase), where range of plantarflexion was reduced and the rearfoot was less plantarflexed at toe off. In the frontal plane (figure 2B), the

1 overall pattern was similar between groups but the stroke group showed 3° less total
2 range of movement and greater maximum eversion. Similar to the sagittal plane, the
3
4 most significant differences were in the last part of stance where the rearfoot was
5 showed less inversion particularly at toe off. For both groups, the smallest rearfoot
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7 movements were in the transverse plane (figure 3B), with two phases of
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10 abd/adduction but the stroke group showed less overall movement (by ~2.6°) than
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12 the control group. Initial contact started from a more adducted position and the first
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14 phase of motion involved less abduction at a later time was and less movement
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16 during the adduction phase.
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24 *Mid-foot movement (Table 2)*

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26 Kinematic patterns of the affected mid-foot showed a similar pattern of sagittal
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28 plane motion to the control group (figure 1C). Although the stroke group
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30 demonstrated greater mid-foot dorsiflexion during most of stance phase, none of the
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32 comparisons were significant. In the frontal plane (figure 2C), both the stroke and
33
34 control groups showed three phases of inv/eversion during stance phase but no
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36 comparisons were significant except the range of the final inversion movement,
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38 which was reduced in stroke. In the transverse plane (figure 3C), the stroke and
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40 control groups had a similar pattern of abd/adduction of the mid-foot and no
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42 comparisons were significant.
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51 *Forefoot movement (Table 2)*

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53 Kinematic patterns of the affected forefoot in the sagittal plane (figure 1D) were
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55 consistent with that for the control group with three phases of dorsi/ plantarflexion.
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57 No significant differences were found in the first (plantarflexion) and second
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1 (dorsiflexion) phases but there was later maximum dorsiflexion and less motion (2.7°)
2 during the final (plantarflexion) phase in the stroke group. Both groups showed three
3 phases of forefoot inv/eversion in the frontal plane (figure 2D) but none of the
4 comparisons were significant although stroke survivors tended to be more inverted
5 during most of stance. In the transverse plane (figure 3D), stroke survivors tended to
6 show greater abduction over the whole of stance phase with the main differences
7 (less motion and less adduction) in late stance.
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20 *Association between foot kinematics and walking ability*

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23 The results of the binary regression models revealed that rearfoot movements were
24 most closely related to walking ability in that stroke survivors with a less plantarflexed
25 (odds ratio=1.30, p=0.005) or inverted rearfoot (odds ratio=1.70, p=0.004) at toe off
26 or less adducted rearfoot in late stance (odds ratio=0.65, p=0.02) were more likely to
27 be limited to walking indoors (so called household walkers). The coefficient of
28 determination (Pseudo R-Square) indicated that the final regression models
29 explained 31%-46% of variance in the stroke group's walking ability. None of the
30 other parameters entered in to the models showed a significant influence on walking
31 ability
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48 **Discussion**

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50 Our findings provide new insight into the nature and functional importance of
51 deviations of foot and ankle biomechanics after stroke and highlight structural and
52 movement deficiencies in the intrinsic foot segments and joints in all three planes
53 which are not detectable using a single segment foot model. To our knowledge, this
54 is the first biomechanics study using a multi-segment foot and ankle in stroke
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1 survivors and so comparisons are limited. Nevertheless the inter-segment rotations
2 in our control group were generally consistent with previous reports [18] and the
3 stroke group showed grossly similar movement patterns to the control group of
4 healthy older adults, although there were differences in the absolute angular
5 positions and range and timing of kinematic events similar to reports of
6 musculoskeletal pathologies [19,20]. Further research to better understand the
7 influence of neuromuscular impairments and the role of other aspects of
8 biomechanics would further enhance our understanding of the mechanisms of gait
9 abnormalities post-stroke and inform clinical practice.
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25 Some of the abnormal kinematic parameters were associated with limited function.
26 Such abnormalities can be modified using footwear and orthotics and our data
27 suggest the prescription, design and evaluation of footwear and orthoses should
28 consider the intrinsic joints of the foot as well as the ankle. However the
29 abnormalities we identified contrast with common clinical beliefs and practices.
30 Traditionally clinical management of foot and ankle problems after stroke focuses on
31 impairments of dorsiflexion [12]. The current findings do not support this approach;
32 we found the range of ankle, rearfoot and midfoot dorsiflexion to be normal during
33 stance phase and the primary abnormality was a reduced range and timing of
34 rearfoot plantarflexion after initial contact and in late stance indicated defective heel
35 and forefoot rocker functions and difficulties of the body to 'roll' forward. Rollover
36 orthotics and/or footwear adaptations may aid forward progression [21].
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57 Significant decreases in rearfoot inversion and adduction in late stance were also
58 seen, resulting in a less supinated foot during the propulsion (late) phase of stance.
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Supination of the foot during late stance is an important mechanism to produce a stiff lever against which body weight is transferred from one leg to the other in double stance and enables forward propulsion [22,23]. This is supported by our finding that increased rearfoot pronation was associated with limited walking ability. The external support offered by orthotics may stabilise the rearfoot and may be the mechanism by which orthotics improve the efficiency and speed of walking after stroke [24, 25]. Further research is needed to examine the effect of orthotics and/or footwear adaptations on foot biomechanics and function after stroke.

Our results indicate that stroke survivors have, overall, less movement in the foot during stance phase and a more pronated foot; the reduction in overall range of movement being due to decreases in range of supination movements. This contrasts with the clinical belief that equinovarus (supinated/inverted foot) is the primary foot deformity after stroke [4,12] and suggests that, in most cases, orthotics or footwear adaptations should limit rather than promote pronation. This indicates that adaptations to the medial side of the foot (such as arch supports or shoe/heel wedges) should be used rather than the more commonly prescribed lateral adaptations. Moreover the use of lateral foot adaptations may be detrimental [26]. Further research is needed establish optimal designs of orthotics and their efficacy. As expected stroke survivors walked more slowly than healthy volunteers which may be a confounding factor associated with the observed kinematic differences [27, 28]. Alternatively, the impaired position, movement and function of the foot as it interfaces with the floor could limit gait speeds. If speed was the cause of the observed differences, we might expect a systematic effect, or even offset, in the data, and this was not the case. Consequently we favour the latter explanation.

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3 The main limitation of this study, as with most gait laboratory studies, is the
4 convenience sampling, small sample size and large numbers of variables studied.
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6 However, our sample was recruited primarily from consecutive admission/
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8 attendances to in- and out-patient stroke services in a large inner city hospital and
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10 the inclusion criteria were broad to capture a pragmatic sample, furthermore the
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12 participants were typical of other research studies [5,8] and so we feel the
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14 participants were reasonably representative of stroke patients with enduring walking
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16 limitations. As this is the first study of its type, there was no existing data with which
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18 to undertake a sample size calculation and so the sample size was determined by
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20 the resources available. Again, it is typical of studies in the field but nevertheless
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22 some of the insignificant comparisons may have been type II errors from an
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24 insufficient sample size; further research using this data to calculate the sample size
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26 is needed to replicate the findings. Despite the relatively small sample size, some
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28 comparisons were significant and were sufficiently strong to overcome the potential
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30 for Type II errors and demonstrate a significant influence functional mobility, which is
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32 clearly important. Further research with a sample size powered to detect functionally
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34 and well as statistically significant differences is warranted.
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46 In this study, we used the participants' position when standing as the zero degree
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48 (0°) reference position of the joints against which any changes in position were
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50 calculated. Differences in absolute angles between the stroke and healthy controls
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52 participants are therefore partly dependent on the static foot posture. We feel this
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54 was justified as we have reported no differences in the Foot Posture Index for both
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56 groups.
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Conclusions

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3 Our findings highlight structural and movement deficiencies in the intrinsic foot
4 segments and joints in all three planes which are not detectable using a single
5 segment foot model and do not support common clinical practices that focus on
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8 correction of sagittal ankle deformity and assumed excessive foot supination. Some
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11 of these abnormalities were associated with limitation in functional ability. The
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14 findings will improve in the clinical management of the foot and ankle in people with
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17 stroke. Biomechanical abnormalities of foot and ankle are modifiable and there is
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20 potential for clinical studies and future developments of interventions to help prevent
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23 or treat these abnormalities which may improve functional ability post stroke. This
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26 might include novel therapeutic strategies such as innovative design of orthoses,
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29 other forms of conservative treatment and surgical corrections. This information will
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32 also enable clinicians to target the foot and ankle appropriately during rehabilitation
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35 and prescribe, design and evaluate footwear, foot orthoses and other forms of
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38 therapy and surgical corrections more effectively.
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Conflict of interest

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42 None.
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Table 1: Anatomical and tracking markers in our multi-segments foot and shank model

Segments	Anatomical (Calibration) markers	Tracking markers
Shank	1- two markers on the medial and lateral femur epicondyles 2- two markers on the most medial and lateral aspects of malleolus	1- a cluster of four markers on the distal and anterior aspect of shank
Rearfoot	1- A cluster of four markers on calcaneus (two markers on the bisection line of the posterior aspect of heel, distally and proximally (ICAL and SCAL, respectively). Two markers on the medial and lateral aspects of heel at the same distance from the posterior bisection line (MCAL and LCAL, respectively)	1- the same as calibration markers
midfoot	1- a cluster of three markers (one marker on the navicular tuberosity (NAV), one marker on the cuboid tuberosity (CUB) and one marker on the dorsal aspect of second cuneiform (SCN2))	1- the same as calibration markers
Forefoot	1- SCN2 2- one marker on the distal head of first metatarsal (D1MT) 3- one marker on the distal head of fifth metatarsal (D5MT)	1- D1MT 2- the head of second metatarsal (D2MT) 3- D5MT
Total foot	1- two markers on the most medial and lateral aspects of malleolus 2- D1MT 3- D5MT	1- SCAL 2- D1MT 3- D5MT

Table 2. Mean and standard deviation movements of each foot segment in each plane

(NB. For clarity, only the significant comparisons between stroke survivors and healthy age-matched controls are shown)

Parameter	Mean \pm sd for Stroke survivors	Mean \pm sd for healthy volunteers	P value (95%CI) of mean difference between stroke and healthy data
REARFOOT MOTION - SAGITTAL PLANE			
Range of movement during initial plantarflexion	3.3° \pm 2.1°	5.4° \pm 2.5°	P < 0.007 (-3.6 to -0.6)
Range of plantarflexion during late stance	11° \pm 4.6°	15.6° \pm 4.5°	P < 0.003 (-7.5 to -1.7)
Plantarflexion at toe off	-3° \pm 6.9°	-8.8° \pm 4.3°	P < 0.003 (2.1 to 9.4)
REARFOOT MOTION - FRONTAL PLANE			
Total range of movement	8.9° \pm 3.2°	12° \pm 3.3°	P < 0.006 (-5.1 to -0.9)
maximum eversion	3.5° \pm 2.1°	2.3° \pm 1.5°	P < 0.05 (-0.06 to 2.3)
Range of inversion during late stance	8.8° \pm 3.4°	12° \pm 3.4°	P < 0.006 (-5.3 to -1.0)
Inversion at toe off	-5.4° \pm 4.4°	-9.6° \pm 3.4°	P < 0.002 (1.7 to 6.8)
REARFOOT MOTION – TRANSVERSE PLANE			
Total range of movement	6.4° \pm 2.6°	9.0° \pm 4.9°	P < 0.04 (-5.1 to -0.09)
Adduction at initial contact	-2.7° \pm 2.5°	-1° \pm 2.2°	P < 0.03 (-3.2 to -0.2)

Maximum abduction	$1.3^{\circ} \pm 2.7^{\circ}$	$3.3^{\circ} \pm 3.2^{\circ}$	P < 0.05 (-3.8 to -0.03)
Timing of maximum abduction	$39\% \pm 25\%$	$25\% \pm 16\%$	P < 0.04 (0.9% to 28%)
Range of movement during the adduction phase	$6.1^{\circ} \pm 2.9^{\circ}$	$9.0^{\circ} \pm 4.9^{\circ}$	P < 0.03 (-5.5 to -0.3)

FOREFOOT MOTION - SAGITTAL PLANE

Timing of maximum dorsiflexion	$98\% \pm 2.6\%$	$96\% \pm 2.1\%$	P < 0.01 (0.6% to 3.9%)
Range of final plantarflexion phase	$1.9^{\circ} \pm 2.1^{\circ}$	$4.6^{\circ} \pm 3.3^{\circ}$	P < 0.008 (-4.8 to -0.8)

FOREFOOT MOTION – TRANSVERSE PLANE

Range of the final adduction phase	$1.3^{\circ} \pm 1.8^{\circ}$ versus	$3.1^{\circ} \pm 1.9^{\circ}$	P < 0.009 (-3.1 to -0.5)
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WHOLE FOOT – SAGITTAL PLANE

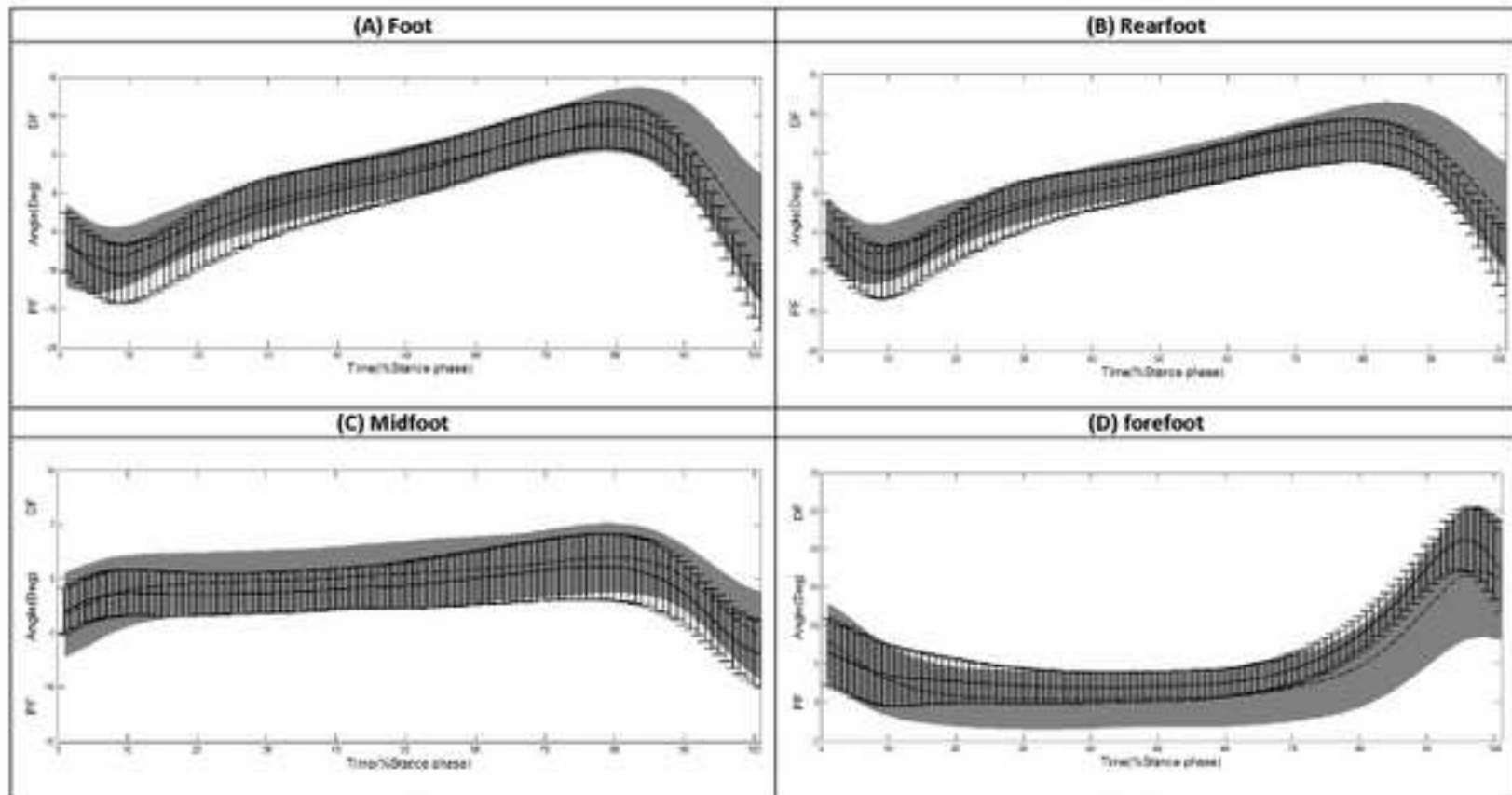
Total range of movement	$20.2^{\circ} \pm 3.7^{\circ}$	$23.4^{\circ} \pm 4.7^{\circ}$	P < 0.02 (-6.0 to -0.6)
Range of movement during initial plantarflexion	$2.5^{\circ} \pm 1.7^{\circ}$	$4.3^{\circ} \pm 2.3^{\circ}$	P < 0.007 (-3.2 to -0.6)
Timing of the 1st phase (plantarflexion)	$6.8\% \pm 2.8\%$	$9.0\% \pm 1.7\%$	P < 0.006 (-3.6% to -0.7%)
Timing of 2nd phase (dorsiflexion)	$73.9\% \pm 5.7\%$	$69.7\% \pm 2.1\%$	P < 0.003 (1.5% to 7.0%)
Range of plantarflexion during late stance	$15.9^{\circ} \pm 6.5^{\circ}$	$23.1^{\circ} \pm 4.7^{\circ}$	P < 0.001 (-10.9 to -3.6)
Plantarflexion at toe off	$-5.9^{\circ} \pm 8.4^{\circ}$	$-14^{\circ} \pm 3.8^{\circ}$	P < 0.001 (3.9 to 12.2)

WHOLE FOOT – TRANSVERSE PLANE

Total range of movement	$9.5^{\circ} \pm 4.6^{\circ}$	$13.5^{\circ} \pm 5.0^{\circ}$	P < 0.01 (-7.0 to -0.9)
Timing of maximum abduction	$59.1\% \pm 25\%$	$35.5\% \pm 25\%$	P < 0.003 (8.6% to 38.6%)
Range of movement of adduction phase	$9.4^{\circ} \pm 4.8^{\circ}$	$13.5^{\circ} \pm 5.0^{\circ}$	P < 0.01 (-7.3 to -1.0)

7. Figure(s)
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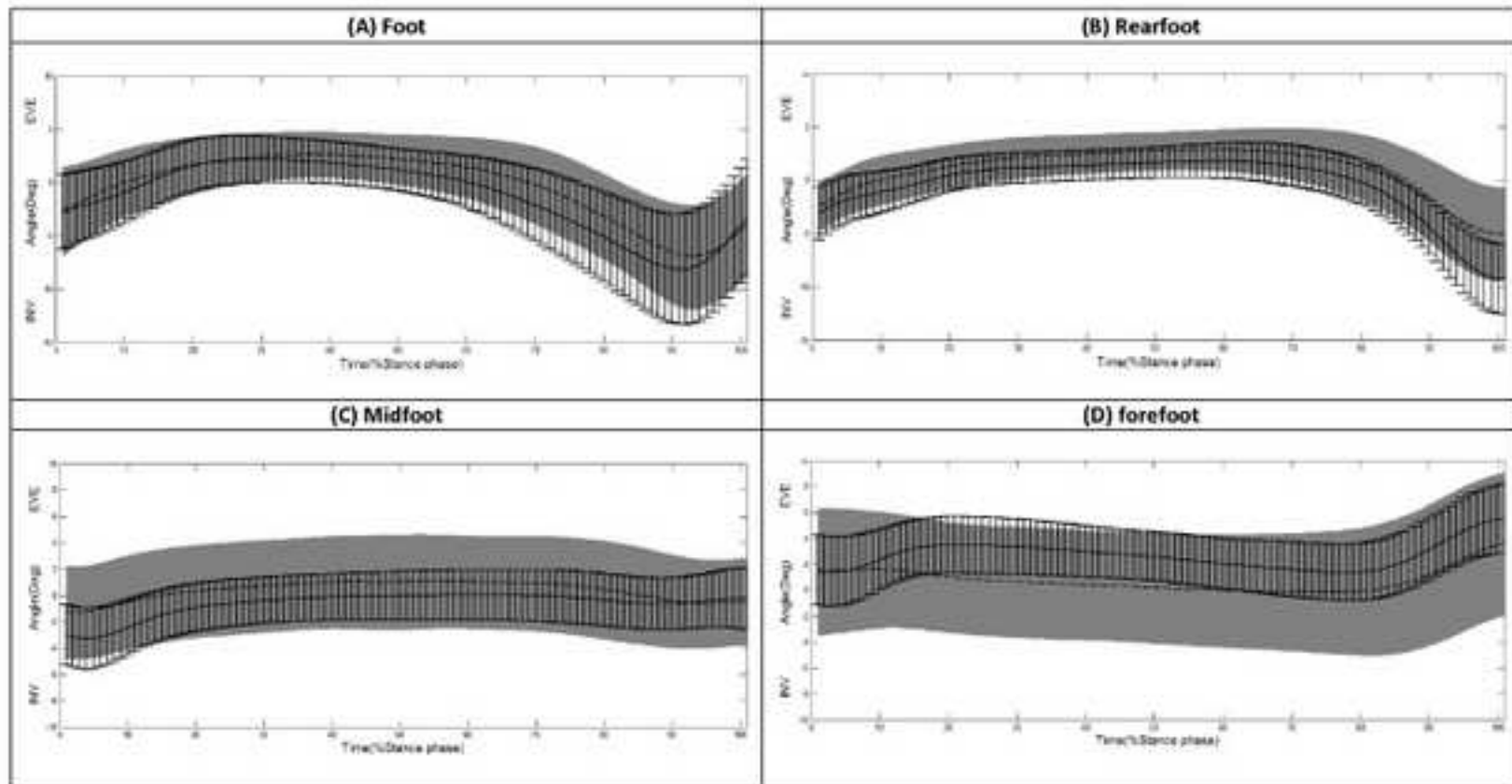
Figure1 : Sagittal plane movements of multi-segment foot and ankle during stance phase in stroke (Mean = Dash line; \pm 1SD= Grey band) and control (Mean = Solid line; \pm 1SD= Error bar) participants. DF = Dorsiflexion, PF = Plantarflexion



7. Figure(s)

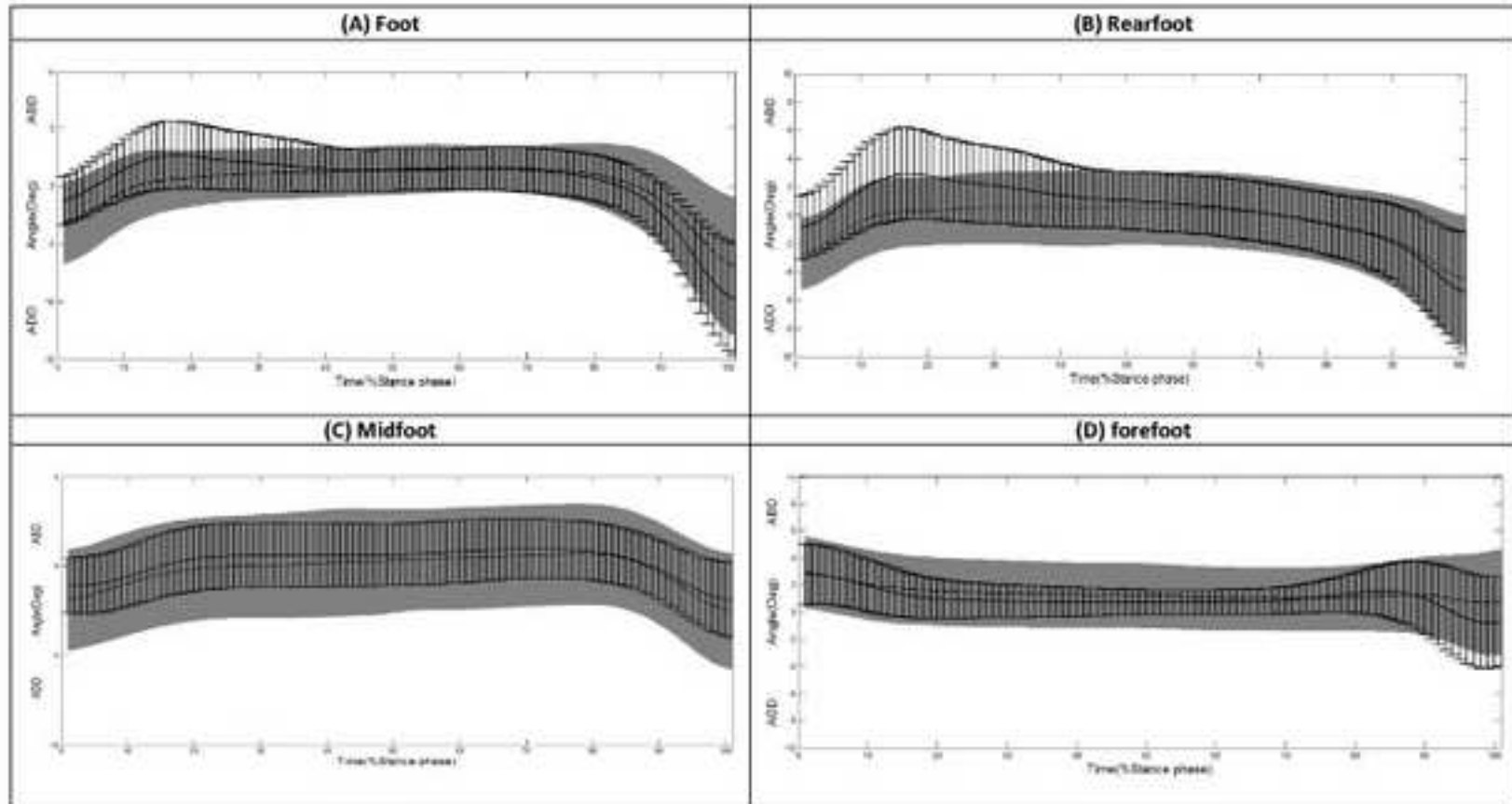
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Figure 2: Frontal plane movements of multi-segment foot and ankle during stance phase in stroke (Mean = Dash line; \pm ISD= Grey band) and control (Mean = Solid line; \pm ISD= Error bar) participants. EVE = Eversion, INV = Inversion



7. Figure(s)
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Figure 3: Transverse plane movements of the multi-segment foot and ankle during stance phase in stroke (Mean = Dash line; \pm 1SD= Grey band) and control (Mean = Solid line; \pm 1SD= Error bar) participants. ABD = Abduction, ADD = Adduction



*Research Highlights

- Stroke survivors showed movement deficiencies in the intrinsic affected foot joints
- Stroke survivors showed reduced range of motion across most segments and planes
- Stroke survivors showed increased pronation and reduced supination
- Stroke survivors showed disruption of the rocker and the timing of joint motion
- The changes in pronation and supination were associated with limited walking ability