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Prevalence of Equinovarus Deformity in Stroke Survivors: A Cross-Sectional Study

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Abstract

Background: Equinovarus, where the foot points down and turns inward, is one of the commonest ankle and foot problems after stroke, yet we have little specific prevalence data for it.

Objective: To estimate how many stroke survivors with spasticity have equinovarus, and to look at ankle range of motion (ROM) and Modified Ashworth Scale (MAS) grade in relation to it.

Methods: We analyzed cross-sectional data from 62 stroke survivors at a tertiary centre in Latur (age 29 to 65 years, MAS 2 to 4), with goniometric ROM recorded for both ankles. Prevalence was estimated with exact binomial confidence intervals. Groups were compared with Mann–Whitney U, Fisher's exact and Wilcoxon signed-rank tests, with Holm adjustment across the four movements, and an exploratory logistic regression was added.

Results: Equinovarus was present in 34 of 62 participants (54.8%; 95% CI 41.7 to 67.5%; exact binomial test against 50%, $p = 0.526$). Sex (male 63.4% vs female 38.1%, Fisher $p = 0.0667$), age ($p = 0.542$), affected side ($p = 1.000$) and MAS grade (exact $p = 0.607$) were not significantly linked to the deformity. On the affected side, dorsiflexion was on average 15.4° lower than on the unaffected side ($p = 3.69 \times 10^{-9}$), plantarflexion was 4.4° higher ($p = 6.32 \times 10^{-5}$) and eversion was 4.9° lower ($p = 7.39 \times 10^{-6}$). Inversion did not differ ($p = 0.635$).

Conclusion: A little over half of the stroke survivors with spasticity in this sample had equinovarus. The loss of dorsiflexion and eversion was large enough to matter clinically, but we could not show that age, sex, side or MAS grade predicts who is affected. The sample is small and the criterion used to label equinovarus was not recorded, so these findings should be read as provisional.

Keywords: Stroke, Equinovarus, Ankle, Goniometry, Spasticity, Prevalence

Introduction

A stroke, or cerebrovascular accident (CVA), happens when blood flow to part of the brain is suddenly interrupted and neurological function is lost.[1] The interruption can come from a blocked vessel (ischemic stroke) or a ruptured one (haemorrhagic stroke). Survival after the acute event has improved over the years, so more and more people now live with what the stroke leaves behind, and stroke remains a leading cause of long-term disability.[2]

The numbers are large. In 2021 an estimated 93.8 million people were living with stroke, with ischemic stroke accounting for 65.3% of cases and subarachnoid haemorrhage for 5.8%.[2] Once the acute phase is over, most of the motor problems come down to weakness and spasticity. Sensory loss adds to this, and proprioceptive deficits in particular are linked to poorer postural control, weaker protective reflexes and

reduced joint movement, balance and gait. Walking and balance rely heavily on the ankle and foot, so problems there can limit a person's independence far more than their size would suggest.[2]

Lance described spasticity in 1980 as a motor disorder with a velocity-dependent increase in tonic stretch reflexes and exaggerated tendon jerks, caused by hyperexcitability of the stretch reflex and forming one part of the upper motor neuron syndrome.[3] Guidance on managing post-stroke spasticity has since been brought together in a modified scoping review, which shows how common and how clinically important the problem is.[3] In practice, spasticity rarely comes alone. It usually travels with weakness, poor selective movement control and changes in how the muscle itself behaves mechanically.[3]

Spastic paresis is among the most frequent complications of stroke. When an acquired brain injury damages the corticospinal pathways, the muscles that should produce force receive too little command, and this shortfall is what is called paresis. Over time the muscle and tendon change as well, with lower elasticity and strength. Force generation suffers, muscle power falls, and gait and weight transfer become limited.[4]

Spasticity in the muscles of the ankle and foot is very common, and it often leads to deformities such as equinus, varus, equinovarus and striatal toe.[5] Of these, spastic equinovarus is the one seen most often. It is the commonest deformity of the ankle and foot complex in spastic hemiplegia and is part of the typical hemiplegic gait pattern.[5] The foot sits in plantarflexion and inversion. The usual cause is spasticity of the plantar flexors and invertors (typically the gastrocnemius–soleus complex and tibialis posterior), with little or no contribution from the dorsiflexors. This separates it from simple varus deformity, where tibialis anterior spasticity plays only a minor part, if any.[5] How the deformity looks in a given patient depends on how severe the spasticity and weakness are, which is why a careful clinical examination is needed to work out the main cause.[5]

The ankle has two main jobs: absorbing impact and moving the body forward. It is also the key joint in the ankle strategy people use to stay balanced.[6] Foot deformities that could interfere with walking have been reported in about 50% of chronic stroke patients, and close to 30% stand with an abnormal, asymmetric foot posture, which is linked to walking limitations.[6,7] In a cross-sectional study of 184 patients, 42% had an ankle–foot deformity. Pes equinovarus was the most frequent (48%), followed by pes equinus (22%), claw toe (19.5%) and pes varus (10.4%).[7] Those with deformities had a longer disease duration and clearly higher lower limb spasticity, yet their ambulation, postural control, balance and daily living activities did not differ significantly from patients without deformities.[7]

The reason this posture matters becomes clearer when the gait cycle is considered. In swing, limited dorsiflexion together with plantarflexion and inversion can reduce foot clearance, and patients may compensate by circumducting the leg or hitching the hip. At initial contact, a foot that lands on its outer border or forefoot gives a smaller, less stable base, which can make weight acceptance and balance harder. For these reasons equinovarus is treated as a significant clinical problem and not just a cosmetic one.[7]

Equinovarus also matters because something can be done about it. Tarsal fusion has been reported to improve gait capacity in chronic stroke patients with pes equinovarus.[8] In a cohort of 64 post-stroke patients who had surgery for spastic equinovarus, every foot was brought to a plantigrade position, nearly half of the patients could stop using orthoses or assistive devices, and ambulation scores improved regardless of age, sex or how long ago the stroke occurred.[9]

The effects are not purely mechanical. A qualitative study of 13 community-dwelling chronic stroke survivors identified three themes: the impact on mobility and balance, worries about “standing out”, and the kind of help received.[10] Participants described pain, sensory deficits and weakness that limited their

walking and made them more afraid of falling. They also spoke of damage to body image and self-esteem, and felt that clinical care concentrated on the lower limb in general while missing the particular problems of the foot and ankle.[10]

Muscle imbalance adds to the picture. In 20 hemiparetic patients with foot varus, compared with 16 healthy controls, video gait analysis and surface EMG showed the calf muscles firing too early in swing and a marked asymmetry between the two dorsiflexors, mostly because the extensor digitorum longus was less active.[11] An interdisciplinary group has also published guidance on assessing and treating spastic equinovarus foot after stroke, a sign of how complex the problem is clinically.[12]

Two measures sit at the centre of this study. Ankle range of motion (dorsiflexion, plantarflexion, inversion and eversion) can be measured with a goniometer, using standard procedures that have been described in detail.[13] Spasticity is graded on the Modified Ashworth Scale (MAS), an ordinal scale that runs from 0, no increase in muscle tone, to 4, where the part is rigid in flexion or extension. Grade 1+ means a slight increase in tone with a catch, followed by minimal resistance through less than half of the range. The strengths and limits of the Ashworth and Modified Ashworth Scales as measures of spasticity have been reviewed,[14] and the reliability of the MAS between and within raters has been examined in a systematic review and meta-analysis.[15]

More advanced tools exist. Dynamic EMG adds value when assessing equinus and equinovarus foot deviation after stroke, although several barriers limit how often it is used.[16] An expert panel has validated international recommendations for managing post-stroke equinovarus foot deformity using the Delphi method,[17] and the ways of judging how well equinovarus surgery works on the gait of hemiplegic patients have also been reviewed.[18] Where instrumented analysis is not available, grading spasticity clinically and measuring the ankle with a goniometer is still the most practical way to identify and describe the deformity.

Procedure

Permission was taken from the Institutional Ethical Committee of Maharashtra institute of Physiotherapy, Latur. The subjects were selected according to inclusion and exclusion criteria. The subjects were informed about procedure. Then consent was taken from the subjects. After obtaining consent we were able to observe the prevalence of equinovarus deformity in stroke survivors by Assessing the spasticity by MAS and range of motion of ankle joint by GONIOMETER. The necessary data was taken by the subject and was written on the data collection sheet.

Assessment of Spasticity of Ankle Joint

The Modified Ashworth Scale (MAS) was used to assess the degree of increase in muscle tone around the ankle joint bilaterally by passively moving the ankle through its range of motion and grading the resistance felt according to the MAS grading criteria.[13]

Assessment of Ankle Dorsiflexion and Plantarflexion

The dorsiflexion and plantarflexion of ankle joint was measured with the help of universal goniometer. The participants were positioned comfortably in supine lying on the plinth, with the lower limb supported and the ankle in a neutral position. The fulcrum of the goniometer was placed over the later aspect of the lateral malleolus. The stationary arm was aligned parallel to the lateral midline of the fibula, directed toward the head of the fibula, the moving arm was aligned parallel to the fifth metatarsal.[14]

Assessment of Ankle Inversion and Eversion

The inversion and eversion of ankle joint was measured with the help of universal goniometer. The participants were positioned comfortably in supine lying on the plinth, with the lower limb supported and

the ankle in a neutral position. The fulcrum of the goniometer was placed over the anterior aspect of the ankle joint, midway between the medial and lateral malleoli. The stationary arm was aligned to the anterior midline of the tibia, while the moving arm was aligned to the anterior midline of the second metatarsal.[14] After that collected data was written on data collection sheet and stored in Microsoft excel, then result was analyzed by using Microsoft excel. After data analysis, the results were interpreted, and the discussion and conclusion were drawn based on the study findings.

Data Recording and Statistical Analysis

All measurements were systematically entered into the master chart and then analyzed statistically to evaluate the study variables according to the objectives of the research. Data were collected by using a structure proforma, entered in MS excel sheet and was analyzed by using SPSS 24.0 (IBM, USA). Qualitative data were expressed in term of proportion and quantitative data were expressed in term of mean and standard deviation. After data collection proportion test was used to interpretate the data. A p value of <0.05 was considered as statistically significant whereas a p value <0.001 was considered as highly significant.

RESULT

Participants

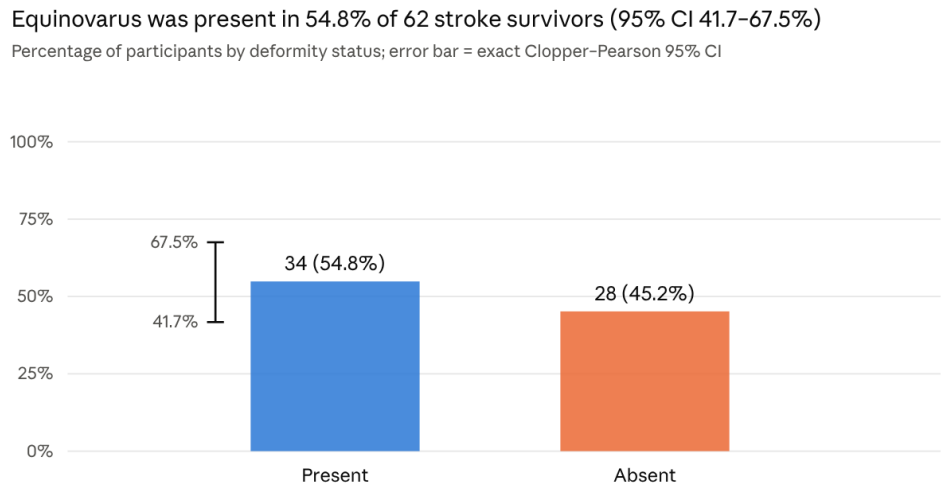
Sixty-two stroke survivors were analysed (41 men, 21 women; 48 right-sided and 14 left-sided; MAS 2 in 35, MAS 3 in 17, MAS 4 in 10).

Table 1. Characteristics by equinovarus status

Characteristic	All (n = 62)	Present (n = 34)	Absent (n = 28)	Test and exact p
Age, mean ± SD (years)	51.1 ± 10.7	51.6 ± 11.7	50.6 ± 9.7	Mann–Whitney U = 519.5, p = 0.542 (Welch t, p = 0.737)
Age, median (IQR)	52.5 (42.0–60.0)	54.5 (41.5–62.0)	52.0 (42.0–58.5)	
Male, n (%)	41 (66.1)	26 (76.5)	15 (53.6)	Fisher p = 0.0667 (χ^2 p = 0.058)
Female, n (%)	21 (33.9)	8 (23.5)	13 (46.4)	
Right side affected, n (%)	48 (77.4)	26 (76.5)	22 (78.6)	Fisher p = 1.000
Left side affected, n (%)	14 (22.6)	8 (23.5)	6 (21.4)	
MAS 2, n (%)	35 (56.5)	18 (52.9)	17 (60.7)	Freeman–Halton p = 0.607; Armitage trend p = 0.357
MAS 3, n (%)	17 (27.4)	9 (26.5)	8 (28.6)	
MAS 4, n (%)	10 (16.1)	7 (20.6)	3 (10.7)	

Prevalence of equinovarus

Bar Graph 1. Prevalence of equinovarus deformity among 62 stroke survivors



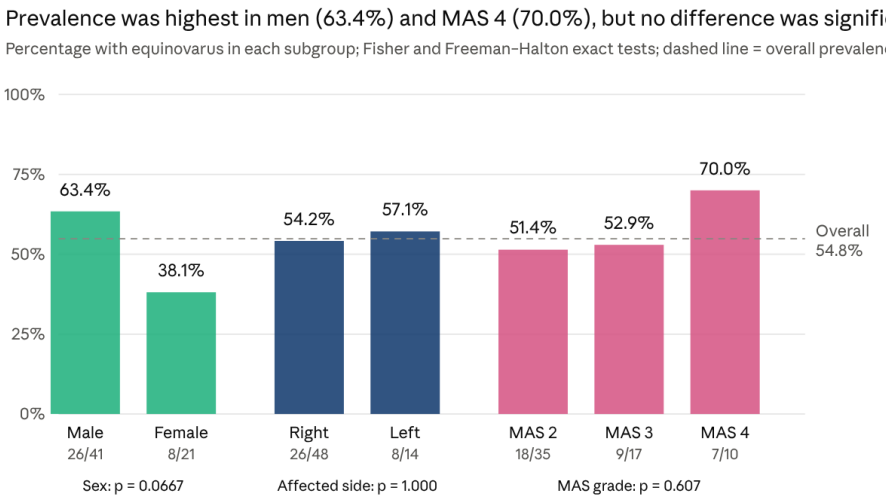
Equinovarus was present in 34 of 62 participants (54.8%) and absent in 28 (45.2%). The exact Clopper–Pearson 95% CI was 41.7–67.5% (Wilson 42.5–66.6%). An exact binomial test against 50% gave p = 0.526, so the data neither support nor refute a prevalence different from one half.

Prevalence by sex, side and MAS grade

Table 2. Prevalence by subgroup (exact 95% CI)

Subgroup	Present / n	Prevalence (%)	95% CI	Effect size	Exact p
Overall	34 / 62	54.8	41.7–67.5		0.526 (vs 50%)
Male	26 / 41	63.4	46.9–77.9	OR male vs female 2.82 (0.95–8.34); Cramér's V 0.24	0.0667
Female	8 / 21	38.1	18.1–61.6		
Right side	26 / 48	54.2	39.2–68.6	OR right vs left 0.89 (0.27–2.95); V 0.03	1.000
Left side	8 / 14	57.1	28.9–82.3		
MAS 2	18 / 35	51.4	34.0–68.6	V 0.13	0.607
MAS 3	9 / 17	52.9	27.8–77.0		
MAS 4	7 / 10	70.0	34.8–93.3	MAS 4 vs ≤ 3: OR 2.16	0.490

Bar Graph 2. Prevalence of equinovarus by sex, affected side and MAS grade



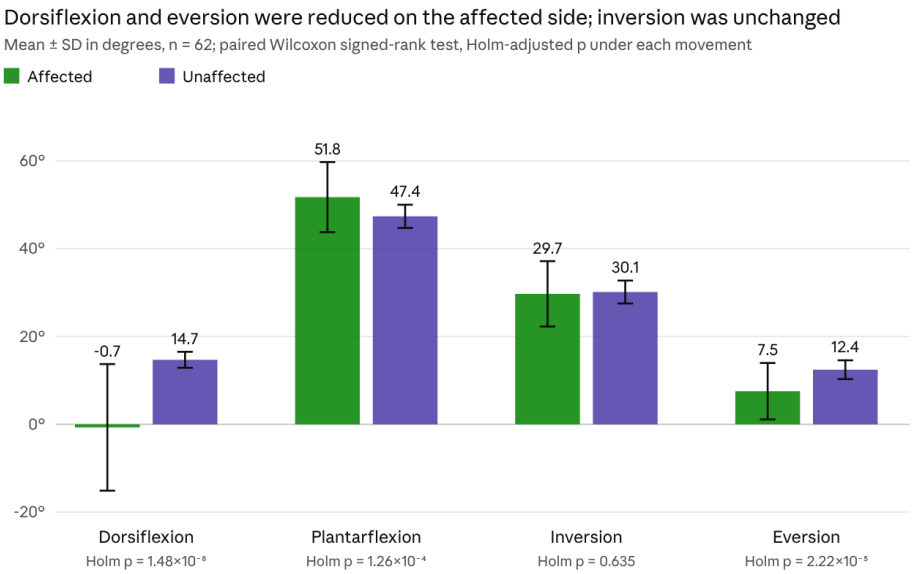
No factor reached $p < 0.05$. The sex difference (63.4% vs 38.1%) was the largest but its interval for the odds ratio includes 1.

Affected versus unaffected limb (all 62 participants)

Table 3. Ankle ROM in degrees, paired comparison

Movement	Affected, mean ± SD (median)	Unaffected, mean ± SD (median)	Mean difference (95% CI)	Wilcoxon W	Exact p	Holm p	Rank-biserial r
Dorsiflexion	−0.7 ± 14.4 (−2.5)	14.7 ± 1.8 (15.0)	−15.4 (−19.0, −11.8)	136.5	3.69×10^{-9}	1.48×10^{-8}	−0.86
Plantarflexion	51.8 ± 8.0 (50.5)	47.4 ± 2.7 (48.0)	+4.4 (2.2, 6.5)	390.0	6.32×10^{-5}	1.26×10^{-4}	0.59
Inversion	29.7 ± 7.5 (30.5)	30.1 ± 2.6 (30.0)	−0.4 (−2.3, 1.4)	851.0	0.635	0.635	0.07
Eversion	7.5 ± 6.4 (7.0)	12.5 ± 2.1 (12.5)	−4.9 (−6.7, −3.1)	322.5	7.39×10^{-6}	2.22×10^{-5}	−0.66

Bar Graph 3. Ankle ROM of the affected and unaffected limb.



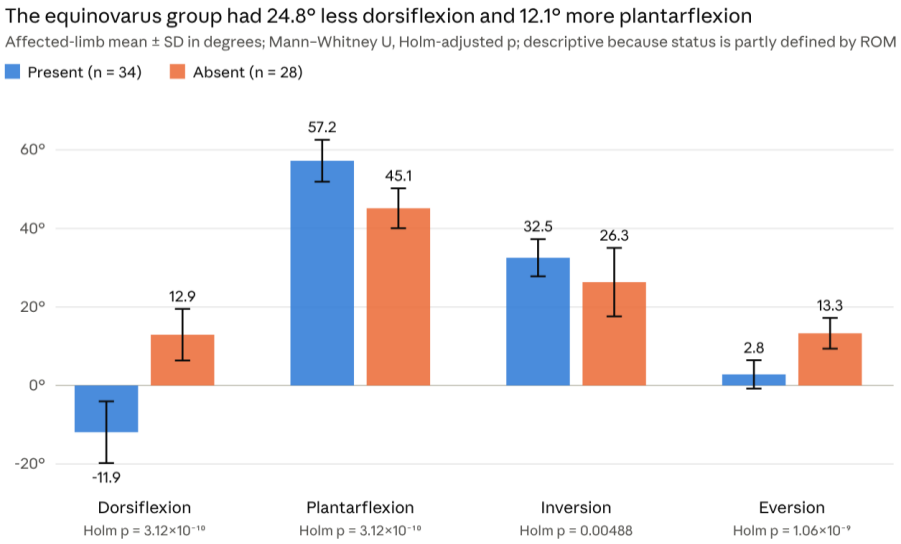
Paired t-tests agreed in every case ($p = 3.59 \times 10^{-12}$, 1.27×10^{-4}, 0.651, 9.66×10^{-7}). Within the equinovarus-present group (exploratory, unadjusted), the affected limb differed by −26.1° in dorsiflexion ($p = 3.58 \times 10^{-7}$), +9.6° in plantarflexion ($p = 5.59 \times 10^{-7}$), +2.4° in inversion ($p = 0.0129$) and −9.5° in eversion ($p = 2.28 \times 10^{-6}$). Within the absent group no movement differed significantly ($p = 0.072$ to 0.367).

Affected-limb ROM by equinovarus status

Table 4. Affected-limb ROM, present vs absent (descriptive; see Section 4 on circularity)

Movement	Present, mean ± SD (median)	Absent, mean ± SD (median)	Mean difference (95% CI)	Hodges–Lehmann	Mann–Whitney U	Exact p	Holm p	r (rank-biserial)	Cohen's d
Dorsiflexion	−11.9 ± 7.9 (−10.5)	12.9 ± 6.6 (14.5)	−24.8 (−28.4, −21.2)	−24.0	16.0	7.79×10^{-11}	3.12×10^{-10}	−0.97	−3.40
Plantarflexion	57.2 ± 5.3 (57.0)	45.1 ± 5.1 (46.0)	+12.1 (9.5, 14.7)	12.0	934.0	9.08×10^{-11}	3.12×10^{-10}	0.96	2.31
Inversion	32.5 ± 4.7 (33.0)	26.3 ± 8.7 (29.0)	+6.2 (2.6, 9.8)	4.0	675.0	0.00488	0.00488	0.42	0.91
Eversion	2.8 ± 3.6 (2.0)	13.3 ± 3.9 (13.0)	−10.5 (−12.4, −8.6)	−11.0	38.5	5.29×10^{-10}	1.06×10^{-9}	−0.92	−2.79

Bar Graph 4: Affected-limb ROM by equinovarus status



MAS, AGE and ROM

Higher MAS was associated with less affected-side dorsiflexion (Spearman $\rho = -0.488$, $p = 5.79 \times 10^{-5}$; Holm 2.32×10^{-4}) and less eversion ($\rho = -0.364$, $p = 0.0036$; Holm 0.011), but not significantly with plantarflexion ($\rho = 0.237$, $p = 0.064$) or inversion ($\rho = 0.227$, $p = 0.076$). Age was unrelated to ROM ($|\rho| \leq 0.13$, all $p \geq 0.31$) and to MAS ($\rho = 0.020$, $p = 0.876$).

Discussion

A stroke is a sudden loss of neurological function caused by an interruption of blood flow to the brain,[1] and it weighs heavily on people and health systems around the world.[2] Spasticity is one of its commonest after-effects, and guidance on managing it has been published.[3] At the ankle, spastic overactivity changes how muscle and tendon behave. Elasticity, strength and power drop, and walking and weight transfer suffer as a result.[4] Equinovarus is usually named as the most frequent deformity in spastic hemiplegia. It comes mainly from spasticity in the plantar flexors and invertors, and how it looks in a given person depends on how severe the spasticity and weakness are.[5] In our sample, 34 of 62 stroke survivors had it, which is 54.8% (95% CI 41.7 to 67.5%). That is a bit over half, and it sits comfortably with what the literature describes. Structural foot deformity after stroke has been studied before,[6] but the closest comparison is a cross-sectional series of 184 patients, in which 42% had an ankle–foot deformity and 48% of those had pes equinovarus.[7] The two numbers are not directly comparable, though. That series gives the proportion among every patient it assessed, which comes to roughly one in five for equinovarus, whereas everyone in our sample already had spasticity (MAS 2 or higher). The same series also found spasticity to be significantly higher in patients with deformity,[7] so a group picked for spasticity should show a higher figure, and ours does. We also need to be straight about how precise our estimate is. The confidence interval runs about 13 percentage points either side, so the real prevalence could be anywhere from roughly 42% to 68%. Why it matters: severe cases can end up needing surgery, and tarsal fusion has been reported to improve gait capacity in chronic stroke patients with pes equinovarus.[8]

We went looking for what set the equinovarus group apart and found nothing convincing. Age ($p = 0.542$), affected side ($p = 1.000$), MAS grade (exact $p = 0.607$) and sex ($p = 0.0667$) were all non-significant. Nothing in the literature behind this thesis suggests an age or sex effect either. The closest thing is a

surgical cohort in which correction of spastic equinovarus worked irrespective of age, sex or how long ago the stroke had happened,[9] but that tells us how well treatment works, not who develops the deformity, so it neither supports nor contradicts our result. Sex came nearest to significance: 63.4% of men had the deformity against 38.1% of women. But the confidence interval for the odds ratio (0.95 to 8.34) includes 1, and the adjusted result ($p = 0.0405$) slid to $p = 0.0728$ once we reclassified one discordant record. That is a question for a bigger study, not a finding. MAS grade did not separate the groups either, even though prevalence rose from 51.4% at MAS 2 to 70.0% at MAS 4. Only 10 people had MAS 4, and since everyone was already spastic, there was little spread in tone to work with. MAS did track real loss of movement, though. Higher grades went with less dorsiflexion ($\rho = -0.488$) and less eversion ($\rho = -0.364$), which fits what we said earlier about spasticity severity shaping the deformity and being higher in affected patients. A tone grade on its own, then, does not tell you who has a deformed foot posture.

Whether any of this changes daily life is a different question. People who have had a stroke say that foot and ankle problems hold back their mobility and balance, yet clinical care tends to focus on the lower limb as a whole.[10] On the other hand, the 184-patient series found no significant differences in walking, postural control, balance or daily activities between patients with and without deformity. We did not measure function, so we cannot say whether equinovarus made a practical difference to our participants.

The movement measurements told a clear story. People with equinovarus had a mean dorsiflexion of −11.9°, while those without averaged +12.9°. They also had 12.1° more plantarflexion, 10.5° less eversion and 6.2° more inversion. That is the classic plantarflexion-plus-inversion pattern and fits overactive plantar flexors and invertors. The inversion gain was smaller and less certain ($p = 0.00488$; $r = 0.42$) than the eversion loss, partly because six people without deformity had unusually low inversion (2 to 15°). Muscle imbalance around the ankle may play a part in the inversion too. Earlier gait work in hemiparetic patients with foot varus found premature calf activity and reduced extensor digitorum longus activity during swing,[11] though our data cannot test that. One caution matters a great deal. The “present” label appears to follow dorsiflexion, so the dorsiflexion and plantarflexion differences mostly show that the groups were labelled consistently. They are not independent evidence. Pooling all 62 participants, inversion did not differ between limbs ($p = 0.635$), but that hides two opposite patterns. In the present group the affected limb tended to have more inversion (+2.4°), and in the absent group less (−3.9°, $p = 0.072$). Averages across mixed groups can cancel real differences out.

Statistical and clinical significance are not the same thing, and the results show why. The pooled dorsiflexion deficit of 15.4° (26.1° in the present group; $p = 3.69 \times 10^{-9}$) is far larger than the few degrees by which goniometry usually varies, so it matters clinically as well as statistically, although we did not test reliability. The pooled plantarflexion difference of 4.4° ($p = 6.32 \times 10^{-5}$) is statistically significant but about the size of measurement error, so its clinical value is doubtful. The 9.6° difference within the present group is more convincing. A pooled inversion difference of 0.4° ($p = 0.635$) is unimportant either way. For sex, an odds ratio of 2.8 would be a big deal if real, but with a 95% CI of 0.95 to 8.34 and $p = 0.0667$ we simply cannot tell, and a wide interval is not the same as no effect. We also ran many tests, so one nominal $p < 0.05$ (the adjusted sex effect) could easily be chance, and it did not hold up in the sensitivity analysis.

To sum up, equinovarus was common in stroke survivors with spasticity, it showed up mainly as lost dorsiflexion and eversion, and MAS grade alone did not pick it out. Interdisciplinary guidance on assessing and treating spastic equinovarus foot already exists,[12] and routine goniometry of the ankle is a practical

way to find the patients who need it. Our findings are provisional. We would like to see them confirmed in a larger sample.

Conclusion

In this study of 62 stroke survivors with spasticity, equinovarus was present in 34 people, which is 54.8% (95% CI 41.7 to 67.5%). In plain terms, a little over half of the patients we examined had the deformity, although the true figure could reasonably lie anywhere between about 42% and 68%.

Where the deformity was present, the loss of movement was large. Dorsiflexion on the affected side averaged 15.4° less than on the unaffected side, and eversion averaged 4.9° less. These are differences big enough to matter in a patient's posture and walking, not just on paper. Plantarflexion was 4.4° higher on the affected side, but that gap is close to the size of ordinary measurement error, so we would not read much into it. Inversion showed no overall difference between the two sides.

We could not show that age, sex, affected side or MAS grade tells us who is likely to have equinovarus. Men had the deformity more often than women (63.4% against 38.1%), but this was not statistically convincing, and it depended on a single record, so it remains a question for future work. MAS grade, on its own, did not identify the deformity, even though higher grades went with less dorsiflexion and less eversion. Measuring ankle movement directly with a goniometer was the more reliable way to spot the problem.

These findings should be treated as provisional. The sample is small and comes from one centre, every participant already had spasticity, the rule used to label equinovarus was not written down, and a few records need to be checked against the source forms. A larger, multi-centre study with a clear definition of equinovarus and reliable goniometric measurement would show whether these patterns hold up.

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