Potential contributing factors to upper limb associated reactions in people with acquired brain injury: an exploratory study

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ABSTRACT

Purpose: To determine which potential contributing factors are associated with upper limb associated reaction (AR) expression in individuals with acquired brain injury (ABI).

Methods: Forty-two participants underwent three-dimensional motion analysis at self-selected walking speed to generate the AR outcome measure, quantifying their upper limb kinematic deviation compared to healthy controls. Clinical assessment included: upper and lower limb hypertonicity, spasticity and strength, balance, dynamic walking stability, arm and leg function, anxiety, arm pain/discomfort, and fear of falling.

Results: Significant, moderate-to-strong correlations ($r = 0.42–0.74, p < 0.05$) existed between upper limb ARs and both hypertonicity and spasticity of the upper limb muscles and the knee extensors. Significant, moderate correlations to ARs ($r = 0.42–0.59, p < 0.05$) existed for balance, dynamic stability, upper limb strength, and arm function. The severity of AR was significantly different between those with and without hypertonicity of the four tested upper limb muscles, elbow and long finger flexor spasticity, knee extensor spasticity, and reduced dynamic stability ($p < 0.05$; effect sizes $\geq 0.80$). However, these contributing factors were not present in all participants.

Conclusions: Associated reactions are complex and multi-factorial. There were several significant correlations indicating that factors may influence AR severity. While positive upper motor neuron syndrome features should be prioritised for clinical assessment, these factors are not prerequisites for ARs.

IMPLICATIONS FOR REHABILITATION

- Upper limb associated reactions are a complex and multi-factorial phenomenon.
- Upper limb muscle hypertonicity and spasticity should be prioritised for assessment; however, they are not prerequisites for associated reactions.
- Hypertonicity and spasticity should be differentiated as they may have differing relationships to associated reactions.
- Knee extensor hypertonicity and spasticity, postural stability, upper limb strength, and arm function may also be contributing factors to consider.

Introduction

Associated reactions (ARs) are a movement dysfunction frequently observed in the hemiplegic upper limb following acquired brain injury (ABI). These involuntary movements commonly occur in response to effortful exertions during functional tasks, causing visibly awkward, uncomfortable, hemiplegic limb postures, which often adversely impact walking [1], dynamic balance [2,3], functional upper limb use [2,3], activities of daily living performance [1], and self-esteem by adding to the stigma of disability [4]. Associated reactions are prevalent, reported in up to 88% of people with stroke [5–8], and are a key treatment focus for physical rehabilitation and pharmacological management [9,10]. Whilst the literature lacks estimates of prevalence in people with other causes of ABI, anecdotally, it is expected to be similar to a stroke.

People with ABI may present with a myriad of potential co-occurring contributing factors. Literature exploring the relationship of potential contributing factors to ARs is inconclusive and often contradictory, providing little guidance to clinicians regarding the key priorities for assessment and management. Overall, the studies investigating relationships between ARs and potential contributing factors have tended towards low participant numbers, averaging 19 participants [7,8,11–16], demonstrated selection biases, and incorporated different AR outcome measures. This limits conclusive interpretation and dissemination.

While some literature has classified ARs as being unrelated to spasticity (i.e., stretch-insensitive muscle overactivity) [17,18], others have included ARs alongside a diverse set of phenomena under the umbrella term of “spasticity” [19]. Whether this means...
that spasticity is a causative factor for ARs remains unclear. Five studies suggest ARs have a key relationship with spasticity in people with stroke [11–16]. Conversely, two other studies found no significant relationships between ARs and spasticity [7,8]. Importantly, the definition of spasticity utilised, and the associated selection of muscle tone outcome measure may influence the interpretation of findings. Of note, all the aforementioned studies employed the Ashworth Scale or Modified Ashworth Scale (MAS) to broadly quantify spasticity. However, these scales align more closely with the multi-dimensional spasticity definition of Pandyan et al. [20] assessing the “...intermittent or sustained involuntary activation of muscles...” and quantifying the hypertonicity or resistance to passive movement [21]. Therefore, these studies may actually indicate a relationship between ARs and hypertonicity. Hypertonicity may be differentiated from the uni-dimensional construct of true velocity-dependent spasticity as defined by Lance [22,23]. Whilst there is no perfect clinical assessment tool for spasticity, quantification of the velocity-dependent nature requires the use of the clinical Modified Tardieu Scale (MTS), which has yet to be reported in AR research. Therefore knowledge regarding differential relationship of velocity-dependent spasticity to ARs remains unknown [21]. Further research is required to determine whether it is upper limb hypertonicity, spasticity, or both positive features of the Upper Motor Neuron Syndrome (UMNS) that influence ARs or whether ARs can exist in the absence of these features.

Evidence surrounding other potential contributing factors is more ambiguous. Contracture, or loss of passive joint range, may be another factor related to AR presentation [2], given the commonly reported stereotypical flexed joint position causing accompanying soft tissue shortening [2,24,25]. However, Ada et al. [7] refuted this relationship, with only 13 of the 24 people with stroke and ARs demonstrating a loss of passive elbow range of motion. Two other studies reported opposite findings for Fugl–Meyer upper limb scale scores. Boissy et al. [14] reported ARs to only occur in people with severe upper limb motor deficits. In contrast, Hwang et al. [26] counterintuitively showed people with better Fugl–Meyer assessment scores had greater upper limb ARs as quantified by extent of the AR musculature electromyographic and torque recordings. Similarly, confusion exists for upper limb strength. One study suggests a notable relationship to ARs with significant between-group differences in elbow flexion strength in those with severe and moderate ARs [14], whereas another reported a weak, non-significant relationship [13]. Further, ARs may theoretically result from a lack of background postural control causing pathological postural fixation in the absence of stability [27]; however, the relationship between ARs and postural control has yet to be formally investigated.

Other hypothesised contributing factors may also include physical factors such as balance dysfunction [28,29], impaired gait performance [30,31], or lower limb factors such as hypertonicity or spasticity [32,33] and reduced muscle strength and power [31,34]. There may also be other non-physical factors contributing to AR expression. Fear of falling is common after ABI [35,36] and may influence gait parameters [37,38] and similarly impact associated upper limb parameters. Additionally, pain and anxiety, which may influence spasticity presentation [39], could also influence the expression of an AR [40]. As such, there is a myriad of potential contributing factors to the AR phenomenon. The extent to which each of these factors influences how an AR manifests has yet to be determined.

Identifying which of these factors contribute to ARs is challenging, particularly in the presence of severe and complex movement disorders. Further, previous investigations have tendency to focus on a single contributing impairment when exploring AR relationships, rather than a range of potential factors. The main contributing factors may vary between individuals, both within and between diagnoses. However, an understanding of which factors are most strongly related to ARs is important to refine assessment processes and provide a better understanding of the AR phenomenon.

Uncertainty regarding the contributing factors to ARs may be partly due to the inadequacy of current AR assessment methods. Historically, AR assessment has occurred in stationary, seated testing positions and focussed on the elbow joint [41]. Given that three-dimensional motion analysis (3DMA) is the criterion-reference method for assessing movement [42], recent work has used this methodology to describe the nature and extent of ARs [43]. A key finding of this previous research was the extent of contribution to ARs of the other upper limb joints, not just solely the elbow joint [43]. Further, seated tests for the assessment of gait-related ARs lacked ecological validity [41,44]. A new, more ecologically-valid, dynamic outcome measure of AR kinematics during walking has since been developed [45]. This current study proposes the first evaluation of ARs that is 1) ecologically valid for walking with robust clinimetric properties and, 2) includes a range of potential contributing factors including both physical and psychological impairments.

Therefore, the aim of this exploratory study was to determine which potential contributing factors are most strongly associated with the expression of upper limb ARs during walking in a cohort of individuals with ABI.

Methods

This study was approved by the Human Research Ethics Committees of Epworth Healthcare (HREC 648-14) and the University of the Sunshine Coast (S/17/1006). All participant recruitment and testing were performed at Epworth Hospital, a major brain injury rehabilitation centre in Melbourne, Australia. All participants provided written informed consent prior to assessment. The funders played no role in the design, conduct, or reporting of this study.

Participants

Participants included a convenience sample of individuals with adult-onset, non-progressive ABI (including traumatic brain injury, stroke, or stable cerebral neurosurgical condition following neurosurgery for benign tumour/s) and apparent hemiplegia who had an AR in any part of the hemiplegic upper limb during walking as diagnosed by the participants’ treating team who used visual observation for the subjective diagnosis. For inclusion, participants needed to be able to walk ≥10 m, barefoot, with no gait aid or hands-on assistance. All participants were assessed during a single two-hour session. Participants with an orthopaedic upper limb injury, contracture impacting normal arm swing during walking, upper limb deficits as a result of a lower motor neuron injury, or a flare upper limb were excluded. A comparative healthy control dataset was utilised in this study as outlined elsewhere [43,45]. Briefly, they were injury-free adults without comorbidities affecting their walking or upper limbs. To obtain a criterion-reference measure of ARs during walking, participants underwent 3DMA testing, walking at their self-selected speed. Self-selected, comfortable paced walking was included due to its important predictive functional value [46,47]. To examine the correlations between a range of potential contributing factors and ARs, they also
undertook a battery of clinical tests reflecting potential contributing factors to ARs.

**Measurement of ARs**

The detail surrounding the development of criterion-reference outcome measures for abnormal upper limb movement in people with ARs was outlined in a prior study [45]. These measures were included to quantify ARs in this study. The upper limb kinematics data collection methodology has previously been described [43,45] and was performed using a 13-camera Optitrack 3DSA system sampling at 120 Hz and Motive:Body software V1.9.0 (NaturalPoint, Inc., Corvallis OR, USA). Data were modelled using Visual3D V6.01.15 (C-motion, Inc., Germantown, MD USA) and exported to custom-written LabVIEW software for analysis (National Instruments, Texas, USA). Details of the customised model have previously been reported [43].

Data were extracted over the 101 points of the gait cycle for each participant, for six upper limb joint axes including: shoulder flexion/extension, shoulder abduction/adduction, shoulder internal/external rotation, elbow flexion/extension, forearm pronation/supination, and wrist flexion/extension. From this, composite scores were devised to quantify the upper limb AR and then classify abnormality relative to a reference healthy control dataset [43,45]. Previous work has recommended the inclusion of three outcome measures to holistically quantify ARs [45]; the Arm Posture Score arithmetic mean, Kinematic Deviation Score worst axis (KDSw), and the number of impaired joints. However, for the purpose of this study, the KDSw was used as the primary AR outcome measure due to its ability to inherently classify abnormality at each joint axis and the overall upper limb AR with 98% accuracy [45]. The KDSw quantifies the arithmetic mean difference between the participant’s data and a comparable healthy cohort at each point of the gait cycle, similar to the Arm Posture Score [48,49]. However, it also scales this difference to the standard deviation (SD) about the mean of the healthy cohort data by classifying abnormality as scores outside the 95% confidence interval (i.e., >1.96SD). A KDS is attributed to each joint axis reflecting the extent of AR at that axis. The worst of these six joint axes then becomes the representative score used, as it has been shown to be more sensitive in classifying abnormality (compared to an average of the six axes) [45]. A higher KDSw indicates a worse AR.

**Clinical testing**

A neurorehabilitation physiotherapist with more than 12 years of clinical experience conducted all testing (author MBK). The Supplementary Appendix Table details information regarding each potential contributing factor, the test employed to quantify it, the testing positions (where applicable), and the corresponding analysis methods. Briefly, the contributing factors and corresponding tests included were:

1. **Contracture** of shoulder internal rotation, elbow flexion, wrist flexion, and finger flexion movements measured by passive range of motion.
2. **Hypertonicity** of the hemiplegic shoulder internal rotators, elbow flexors, wrist flexors, and long finger flexors for the upper limb and gastrocnemius, soleus, and knee extensors in the lower limb, measured by the MAS [21,50].
3. **Spasticity** of the hemiplegic shoulder internal rotators, elbow flexors, wrist flexors, and long finger flexors for the upper limb and gastrocnemius, soleus, and knee extensors in the lower limb, measured by the MTS [21,22,51].
4. **Upper limb strength** measured by the hemiplegic handgrip strength as a representative test for overall upper limb strength [52].
5. **Arm function** measured by the Arm Activity Measure [53].
6. **Lower limb strength** measured by a load cell which quantified the hemiplegic lower limb’s maximal isometric voluntary contraction during a single leg press test [54].
7. **Leg function** measured by self-selected walking velocity during a 10m walk test [55].
8. **Balance** measured by the Short Form Berg Balance Scale [56].
9. **Dynamic stability** measured by the lateral centre of mass (COM) displacement during walking [57].
10. **Fear of falling** measured by the Short Falls Efficacy Scale International [58].
11. **Anxiety** measured by the anxiety subsection of the Hospital Anxiety and Depression Scale [59].
12. **Arm pain or discomfort** during walking measured by the Visual Analogue Scale [60].

**Statistical analysis**

For all analyses, correlations were performed to evaluate the extent of association between each potential contributing factor and the AR measure during walking. Tests of Skewness and Kurtosis confirmed normality. Where outcomes were normally distributed, Pearson correlation coefficients (r) were calculated to quantify the extent of associations between these outcomes. For those that were not normally distributed, Spearman correlation coefficients (ρ) were used. Correlation strength was classified according to Evans’ guidelines as very strong (r ≥ 0.8), strong (0.6–0.79), moderate (0.4–0.59), weak (0.2–0.39) or very weak (<0.2) [61]. Based on the sample size included in this study (n = 42), with an α of 0.05 and β of 0.2, we were powered to detect correlation coefficients of r ≥ 0.42 as statistically significant [62]. This is at the lower end of the threshold for a moderate strength correlation (r = 0.40 to r = 0.60) as defined by Evans [61].

For impairments where there was the ability to dichotomise the group into two independent samples (e.g., the specific contributing factor being present vs. absent, normal vs. abnormal, or low vs. high), further analyses were performed to determine if the feature had an impact on AR presentation. t-Test calculations and Cohen’s “d” effect size (ES) were used to determine the statistical difference and magnitude of the mean KDSw between the two groups (i.e., those with and without the contributing factor). p-Values of <0.05 were considered to be significant and ES deemed small (0.20), moderate (0.50) or large (≥0.80) [63]. Additionally, Chi-squared analyses were performed to explore the relationship between hypertonicity and spasticity of each of the upper limb muscles and the AR at the corresponding upper limb joint axis using the specific axis KDS value (e.g., the relationship between hypertonicity or spasticity of the shoulder internal rotators and the AR present at the shoulder joint axis). Given finger kinematics could not reliably be performed using 3DMA, the relationship between the long finger flexors was evaluated with the wrist joint axis. For the Chi-squared analyses, each upper limb joint axis KDS for each participant was classified as present or absent. Abnormality or presence of AR was defined as a score of >1.96 (SD) indicating ≥95% confidence that on average, across the gait cycle, the joint axis mean was abnormal [45].

To perform the analyses for this study, a statistical analysis website incorporating LabVIEW, Matlab, Python, and Highcharts [64], Microsoft Excel 2013 and a website for Chi-square analyses were used [65].
**Results**

**Participant characteristics**

The demographic data for the 42 participants are presented in Table 1. All participants had a stable non-progressive upper motor neuron lesion with an apparent hemiplegia. Where any bilateral deficits were present, the hemiplegic upper limb was evaluated.

### Table 1. Participant characteristics (n = 42).

| Characteristics                        | Range |
|----------------------------------------|-------|
| Injury type (n)                        | CVA (25), TBI (15), NS (2) |
| Sex (male/female)                      | 26/16 |
| Age (years)                            | 48.4 ± 16.7 |
| Weight (kg)                            | 80.03 ± 16.05 |
| Height (cm)                            | 172.3 ± 8.4 |
| Time Post Injury (years)               | 6.2 ± 5.7 |
| Self-selected gait velocity (m/s)      | 0.85 ± 0.29 |
| Hemiplegic side associated reaction (L/R) | 26/16 |
| Upper limb function (ArMA scores)      | Passive subsection 5.1 ± 3.9 0–14 |
|                                      | Active subsection 31.2 ± 14.6 6–50 |

Values are mean ± SD unless indicated. Higher ArMA scores indicate poorer function (passive subsection available scores 0–32 and active subsection scores 0–52).

CVA: cerebrovascular accident (stroke); TBI: traumatic brain injury; NS: cerebral damage post neurosurgery; ArMA: arm activity measure.

Most data were normally distributed, except for the outcome measure scales (Arm Activity Measures A and B, Short Form Berg Balance Scale, Short Falls Efficacy Scale International, Hospital Anxiety and Depression Scale) which therefore underwent non-parametric correlation analyses. All KDSw scores were normally distributed, therefore, parametric analyses were used for between-group differences.

Table 2 outlines all correlation and between-group analyses for the potential contributing factors.

### Table 2. Correlations and between-group differences of potential contributing factors.

| Correlation to KDSw | Number (%) of people with feature | Mean KDSw of those with feature | Number (%) of people without feature | Mean KDSw of those without feature | Between-group difference |
|---------------------|-----------------------------------|---------------------------------|--------------------------------------|-----------------------------------|-------------------------|
| **Upper limb contracture** |                                     |                                 |                                      |                                   |                         |
| Shoulder external rotation | r = −0.37*                       | 0                              | 42 (100)                             | 6.05 (3.00)                      |                         |
| Elbow extension       | r = −0.53*                        | 1 (2.4)                        | 13.17*                               | 41 (97.6)                        | 5.88 (2.82)             |
| Wrist extension       | r = −0.53*                        | 1 (2.4)                        | 10.12*                               | 41 (97.6)                        | 5.95 (2.97)             |
| **Upper limb hypertonicity (MAS score)** |                       |                                 |                                      |                                   |                         |
| Shoulder internal rotators | r = 0.45*                        | 26 (61.9)                      | 6.90 (3.17)                          | 16 (38.1)                        | 4.68 (2.15)             | <0.01b 0.82c |
| Elbow flexors         | r = 0.53*                         | 30 (71.4)                      | 6.70 (3.14)                          | 12 (28.6)                        | 4.43 (1.90)             | <0.01b 0.87c |
| Wrist flexors         | r = 0.44*                         | 27 (64.3)                      | 6.91 (3.19)                          | 15 (35.7)                        | 4.50 (1.85)             | <0.01b 0.92c |
| Long finger flexors   | r = 0.74*                         | 24 (57.1)                      | 7.17 (2.88)                          | 18 (42.9)                        | 4.03 (1.72)             | <0.01b 1.49g |
| **Upper limb spasticity (MTS score)** |                       |                                 |                                      |                                   |                         |
| Shoulder internal rotators | r = 0.31*                        | 26 (57.1)                      | 6.61 (3.02)                          | 18 (43)                          | 5.30 (2.90)             | 0.08 0.44 |
| Elbow flexors         | r = 0.40*                         | 25 (59.5)                      | 6.93 (3.28)                          | 17 (40)                          | 4.76 (2.00)             | <0.01b 0.80d |
| Wrist flexors         | r = 0.34*                         | 22 (52.4)                      | 6.74 (3.10)                          | 20 (48)                          | 5.30 (2.76)             | 0.06 0.49 |
| Long finger flexors   | r = 0.65*                         | 20 (47.6)                      | 7.66 (2.66)                          | 22 (52)                          | 4.59 (2.55)             | <0.01b 1.18e |
| **Lower limb hypertonicity (MAS score)** |                       |                                 |                                      |                                   |                         |
| Gastrocnemius         | r = 0.23                         | 42 (100)                      | 6.05 (3.00)                          | 0 (0)                            | NA                      |                         |
| Soleus                | r = 0.11                         | 28 (66.7)                      | 6.07 (3.42)                          | 14 (33.3)                        | 6.02 (2.02)             | 0.48 0.02 |
| Knee extensors        | r = 0.46                         | 13 (31.0)                      | 7.37 (3.55)                          | 29 (69.0)                        | 5.46 (2.87)             | 0.05 0.59 |
| **Lower limb spasticity (MTS score)** |                       |                                 |                                      |                                   |                         |
| Gastrocnemius         | r = −0.11                        | 31 (73.8)                      | 5.97 (3.12)                          | 11 (26.2)                        | 6.28 (2.79)             | 0.38 0.10 |
| Soleus                | r = 0.01                         | 22 (52.4)                      | 6.20 (3.31)                          | 20 (47.6)                        | 6.92 (10.12)            | 0.44 0.10 |
| Knee extensor         | r = 0.42*                        | 9 (21.4)                       | 8.98 (2.79)                          | 33 (78.5)                        | 5.35 (2.50)             | <0.01b 1.37f |
| **Other**             |                                     |                                 |                                      |                                   |                         |
| Upper limb strength (kg/kg) | r = −0.44*                       |                                 |                                      |                                   |                         |
| Passive arm function  | r = 0.21                         |                                 |                                      |                                   |                         |
| Active arm function   | r = 0.59*                        |                                 |                                      |                                   |                         |
| Lower limb strength (kg/kg) | r = −0.36*                       |                                 |                                      |                                   |                         |
| Leg function – gait velocity (m/s) | r = −0.39*                       | 11 (26.2)                      | 7.53 (3.90)                          | 31 (73.8)                        | 5.53 (2.48)             | 0.07 0.61 |
| Balance               | r = −0.42*                        |                                 |                                      |                                   |                         |
| Dynamic stability (cm) | r = 0.44*                        | 26 (63.4)                      | 6.80 (3.27)                          | 15 (36.6)                        | 4.64 (1.97)             | <0.01b 0.80e |
| Fear of falling       | r = 0.28                         | 10 (23.8)                      | 4.71 (2.58)                          | 32 (76.2)                        | 6.47 (3.03)             | 0.04b 0.63 |
| Anxiety               | r = −0.07                        | 12 (28.6)                      | 5.71 (2.81)                          | 30 (71.4)                        | 6.19 (3.06)             | 0.32 0.16 |
| Arm pain/discomfort during walking | r = 0.11                        | 17 (40.5)                      | 6.51 (2.86)                          | 25 (59.5)                        | 5.91 (3.06)             | 0.26 0.20 |

r: Pearson’s correlation coefficient; ρ: Spearman’s rho coefficient; KDSw: Kinematic Deviation Score worst axis; SD: standard deviation; MAS: Modified Ashworth Scale; MTS: Modified Tardieu Scale.

Bold correlation values indicate at least a moderate relationship (r ≥ 0.40).

*No SD could be calculated as there is only one participant in the group; *indicates p values with a statistically different between-group KDSw for the independent samples (p < 0.05); **indicates large Cohen’s ‘d’ effect sizes; †denotes missing data whereby for dynamic stability data was missing for one participant as both ASIS and PSIS markers were not visible for any three-dimensional motion analysis trials.

*Denotes a statistically significant association (p < 0.05).
Upper limb hypertonicity

There was a strong correlation between the KDSw and MAS of the long finger flexors \( (r = 0.74) \) and a moderate relationship for the other three muscle groups \( (r = 0.44–0.53) \). Forty participants (95.2%) had hypertonicity in at least one of the four upper limb muscle groups tested. Significant between-group differences and large ES in KDSw existed for the presence of hypertonicity in each of the upper limb muscle groups \( (p < 0.05; \ ES > 0.82) \), demonstrating the impact of hypertonicity on overall AR presentation. At an individual joint axis level however, this impact was less clear, with hypertonic shoulder internal rotators \( \chi^2 (1, n = 42) = 0.16, \ p = 0.69 \) and wrist flexors \( \chi^2 (1, n = 42) = 1.41, \ p = 0.23 \) both showing no association to abnormal upper limb movement during walking at their corresponding joint axis. However, a significant association existed for hypertonic elbow flexors \( \chi^2 = 4.67, \ p < 0.05 \) and long finger flexors to their corresponding joint axis during walking \( \chi^2 = 5.79, \ p < 0.05 \).

Upper limb spasticity

Only the MTS score of the long finger flexors had a strong relationship to the KDSw \( (r = 0.65) \) and the elbow flexors had a moderate relationship to KDSw \( (r = 0.40) \). Thirty-six participants (85.7%) had upper limb spasticity in at least one of the four muscle groups. The participants presenting with spasticity in the long finger \( (ES = 1.18) \) and elbow flexor muscle \( (ES = 0.80) \) groups had a significantly \( (p < 0.05) \) higher KDSw and larger ES than those without (Table 2). This difference did not exist for shoulder internal rotators \( (ES = 0.44) \) and wrist flexors \( (ES = 0.49) \). At an individual joint axis level there was no significant association between the spastic shoulder internal rotators \( \chi^2 (1, n = 42) = 1.64, \ p = 0.20 \), elbow flexors \( \chi^2 (1, n = 42) = 2.19, \ p = 0.14 \), and wrist flexors \( \chi^2 (1, n = 42) = 1.63, \ p = 0.20 \) and the AR at their corresponding joint axis during walking. However, there was a significant association between spastic long finger flexors and AR at the wrist joint during walking \( \chi^2 (1, n = 42) = 5.52, \ p < 0.05 \).

Lower limb hypertonicity

There was a weak, non-significant correlation between soleus hypertonicity and KDSw and a moderate, significant correlation to KDSw for knee extensor hypertonicity \( (r = 0.46) \). Given that all participants presented with gastrocnemius muscle hypertonicity, correlational analysis could not be performed. Hypertonicity of gastrocnemius (100%) and soleus (66.7%) were prevalent in the cohort but had little impact on the AR expression. There was no between-group difference in KDSw in those with and without soleus hypertonicity. Hypertonicity of the knee extensors was less prevalent, occurring in 13 participants (31.0%). However, its presence lead to a significantly greater AR \( (p < 0.05; \ ES = 0.59) \).

Lower limb spasticity

The correlations to KDSw were weak for spasticity of gastrocnemius \( (r = -0.11) \) and soleus \( (r = 0.01) \) and moderate for the knee extensors \( (r = 0.24) \). Spasticity of gastrocnemius (73.8%) and soleus (52.3%) were prevalent in the cohort but once again had little impact on the AR with no between-group differences in those with and without spasticity. Spasticity of the knee extensors was also less common than hypertonicity, occurring in nine participants (21.4%). However, when knee extensor spasticity was present, it was associated with a significantly worse AR \( (p < 0.05; \ ES = 1.37) \).

Other contributing factors

Of the remaining 10 correlations performed, active upper limb function, dynamic stability, upper limb strength, and balance had moderate relationships to ARs \( (r ≥ 0.42) \). The other factors had very weak-to-weak, non-significant correlations. The presence of reduced dynamic stability \( (ES = 0.80) \) and fear of falling \( (ES = 0.63) \) demonstrated a significant difference in AR severity \( (p < 0.05) \), whereas community ambulation ability, anxiety, and pain or discomfort did not.

Discussion

This study found that ARs during walking are complex, multi-factorial, and cannot be attributed to a single or small number of contributing factors. Whilst there were several significant relationships, few were rated more than moderate. Long finger flexor hypertonicity and spasticity did account for 54.7% and 42.3%, respectively, of the variability in AR presentation during walking. However, the remaining factors accounted for less than 35% of the variability, highlighting that each predictive factor only makes a small contribution. For those factors that could be classified dichotomously (e.g., present vs. absent, normal vs. abnormal, or low vs. high), a large proportion of the cohort exhibited deficits in these factors and for the majority, this was associated with a more severe AR. However, there were still participants with a significant AR in the absence of these impairments.

An important contribution of this study to the field of ARs is the provision of further understanding regarding the impact upper limb positive UMNS features have on AR expression during walking. Prior literature has historically been inconclusive with some suggesting a relationship to spasticity [11–16] and others showing no relationship [7,8], with spasticity only quantified via the Ashworth Scale or MAS. This is the first study to differentiate hypertonicity and spasticity as distinct aspects of the UMNS and explore their potentially different relationships to ARs. Advances in the field of spasticity have led to this distinction whereby the MAS measures hypertonicity or resistance to passive movement [21] and the MTS is the appropriate clinical measure of velocity-dependent spasticity [21,22]. This study demonstrates that hypertonicity and spasticity of the upper limb muscles were both prevalent. Participants that had either positive UMNS feature (hypertonicity or spasticity), presented with a more severe AR during walking. Hypertonicity appeared to play a somewhat greater role in AR expression compared with spasticity. Hypertonicity of all four of the assessed upper limb muscle groups had a significant, positive relationship to AR severity. For spasticity, however, this relationship and influence existed for only the long finger flexors and elbow flexors and not for the shoulder internal rotators and wrist flexors. Long finger flexor positive features of the UMNS demonstrated the strongest relationship of all the studied contributing factors. Whether the presentation of distal positive UMNS features is an indicator of more severe overall impairment, or whether there is some direct influence of this muscle group on ARs, is unknown. Importantly, however, hypertonicity or spasticity presence is not indicative of AR causation as commonly, ARs occurred at specific joint axes, in the absence of these impairments. For example, 24% and 37% of people with elbow joint ARs did not have elbow flexor hypertonicity or spasticity, respectively.
hypertonicity and spasticity also occurred without joint axis-specific ARs, with 43% and 45% of participants with shoulder internal rotator hypertonicity and spasticity respectively, without an AR at this axis. Similar findings were identified for the wrist joint. Contracture rarely featured in this cohort, making it an unlikely contributor to gait-related ARs. These findings have implications for neurorehabilitation clinical practice, emphasising that whilst not a pre-requisite for ARs during walking, positive UMNS features should be prioritised for impairment-based assessment of people with ABI. Careful assessment is therefore required as not all individuals with ARs possess these factors and nor should it be assumed that all individuals with these impairments have ARs.

Targeted pharmacological management (e.g., Botulinum Neurotoxin) should be reserved for those with confirmed positive UMNS features in the relevant muscles and a corresponding joint axis AR.

Distinct from this, negative features of the UMNS also demonstrated a relationship to ARs. Reduced grip strength scores, which were used as a surrogate measure of global upper limb strength [52,66], and higher Arm Activity Measure scores, indicating poorer active arm function, were both related to AR expression. The impact on AR severity could not be established given the lack of specified cut off scores for normative grip strength or arm function. However, there does appear to be an important relationship with ARs. It is unknown whether the participants have an AR due to the strength and function deficits or whether the AR impairs strength and arm function. Additionally, given that arm swing during walking is fairly passive with small requirements for active movement or strength [67,68], these deficits may not play a direct role in AR expression. They may rather reflect a more severe overall brain injury, which may also cause more severe ARs.

A common clinical understanding of ARs is that they are a pathological form of fixation when the person with ABI lacks a background of postural control [27]. Additionally, given the effort-dependent nature of ARs, the effort related to maintaining postural stability whilst walking may elicit the ARs. Whilst not indicative of causative factors, the findings from this study align with this theory. Poorer balance and dynamic stability, represented by lower Short Form Berg Balance Scale scores and increased lateral COM displacement during walking respectively, were moderately associated with a worse AR. Ex-post facto analysis showed that those with worse dynamic stability walked significantly slower \( r = -0.66, p < 0.05 \), which is well established in prior literature [69]. However, given the weak relationship between walking speed and ARs, dynamic stability may contribute a unique aspect to the expression of AR not captured by walking speed alone.

Lower limb impact on ARs was mixed. Lower limb positive UMNS features were more strongly associated with ARs than negative features. Despite our cohort’s widespread prevalence of gastrocnemius and soleus hypertonicity and spasticity, no significant relationships with ARs were observed. Less prevalent knee extensor hypertonicity and spasticity had a moderate relationship with ARs. Their presence increased AR severity, potentially via the increased effort required to flex the knee for the swing phase during walking in the presence of knee extensor hypertonicity and/or spasticity. With respect to the lower limb negative UMNS features, despite our cohort’s deficits in both lower limb strength and leg function, they had weak relationships with ARs. This indicated that participants had sufficient lower limb strength and capacity to walk 10 m independently at a self-selected speed and thus had little influence on ARs. At faster walking speeds, with greater power and effort required, a stronger relationship may exist. The relationship between the lower limbs and arm swing during walking is an ongoing contention [70] and unfortunately, this study cannot provide further insight into the relationship between the lower limb and ARs. Of note, for lower limb strength, we evaluated predominantly the knee extensors using the leg press strength test. Given the prevalence of ankle plantar flexor deficits in people with ABI [71,72], and their importance for walking [73], assessment of ankle plantar flexor strength may have yielded different results.

Pain or discomfort during walking was present in 40%, anxiety in 29%, and fear of falling in 24% of the cohort. However, these factors were not significantly associated with ARs, possibly because participants self-selected their walking speed. Results may differ if evaluated under conditions of greater demand. Confounding results for fear of falling based on established cut-points were observed; participants with a high vs. low-moderate fear of falling demonstrated a significantly less severe AR, with no walking speed difference. It is unknown, however, whether there is a specific combination of factors that make individuals more likely to present with an AR. For example, individuals with anxiety, fear of falling, and upper limb hypertonicity may be more likely to have an AR during walking than with someone with pure hypertonicity. This requires further exploration.

Limitations

This was the first comprehensive investigation into a range of hypothesised contributing factors for ARs. There are other potential contributing factors that may warrant future investigation. Only a select group of muscles were evaluated for hypertonicity and spasticity in the upper and lower limbs. The muscle groups assessed were selected given they are most commonly affected in people with ABI [72,74,75]. However, future research should consider more extensive upper muscle evaluation and inclusion of the hamstrings and adductor muscles in the lower limbs. More broadly, additional assessment items for evaluation in future research may include pinch grip, Fugl–Meyer upper and lower limb assessments, pain remote from the arm, sensation, coordination, and inattention.

A mixed ABI diagnostic group was included, and impairments may vary between diagnoses. However, only individuals with ARs as a result of stable, non-progressive upper motor neuron lesions were included, the mixed cohort aligns with the International Consensus Statement for the management of upper limb disorders of tone [51], and it enabled a relatively large cohort compared to prior studies.

Contributing factors that possess a notable relationship to AR may reflect greater brain injury severity rather than a direct impact on the AR. We were unable to include a measure of the severity of neurological injury because no uniform measure of severity exists for a mixed ABI group. For example, the National Institutes of Stroke Health Scale is the recommended measure for stroke severity [76], and incorporates physical impairments such as motor performance of the upper and lower limbs. In contrast, the Glasgow Outcome Scale and length of post-traumatic amnesia are the two standard measures of traumatic brain injury severity [77,78], but only measure arousal, orientation, and memory. Measures of brain injury severity and its contribution to ARs seem implicit to investigate. However, scales quantifying cognition are likely irrelevant as predictors of ARs. We included the Arm Activity Measure as a global measure of upper limb function, and unsurprisingly there was a negative relationship with ARs. Nevertheless, the Arm Activity Measure is itself dependent on the same physical contributing factors included in this study.
In this study, analysis of ARs included the shoulder, elbow, forearm, and wrist. However, data from the finger joints were unable to be captured. This may pose a potential issue in the assessment of ARs given that a clenched fist is a commonly observed pattern in people with upper limb spasticity [74], physiotherapists frequently rate the fingers as being implicated in ARs [79], and, this study illustrated a strong relationship between long finger flexor positive UMNS features and ARs. However, 3DMA is the criterion-reference available and at this stage provides the most accurate, objective data possible. Refined 3DMA finger data is an important target for future research.

We assessed people who could walk unaided at their self-selected walking speed, relationships may differ during other tasks, such as sitting to standing, walking with assistance, walking at different speeds, or adding cognitive loading.

**Conclusion**

Associated reactions are complex and multi-factorial. This study did not identify a single or small number of contributing factors that were able to account for the whole AR presentation during walking. Where strong relationships did exist between ARs and specific features (i.e., upper limb spasticity and hypertonicity), there was still considerable variability. Whilst positive features of the UMNS in the upper limb may be prioritised for assessment, importantly, these factors are not essential requirements for ARs. Thorough clinical AR assessment is required ensuring factors are not overlooked and that all possible contributors are identified and addressed.

**Author contributions**

Primary Author MBK performed study design, all data collection, cleaning, analysis, processing and interpretation, and drafting the manuscript; GW and RC contributed to study design, data analysis and interpretation, and critical revision of the manuscript; BFM assisted in data collection of the clinical cohort, developed the upper limb model and analysis program for the three-dimensional motion analysis, assisted in data processing and critical review of the manuscript. KJB and JO contributed to the study design and critical revision of the manuscript.

**Ethics approval and consent to participate**

This research is approved by the Human Research Ethics Committees of Epworth Healthcare (HREC 648-14) and the University of the Sunshine Coast (S/17/1006). All subjects that were invited, consented to do so and provided written informed consent prior to assessment.

**Disclosure statement**

Each of the authors has read and concurs with the content in the final manuscript. The material within has not been and will not be submitted for publication elsewhere except as an abstract.

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