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Understanding Spatial and Temporal Gait Asymmetries in Individuals Post Stroke

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Abstract

Gait asymmetry in spatial and temporal parameters and its impacts on functional activities have always raised many interesting questions in research and rehabilitation. The aim of this topical review is threefold: 1) to examine different equations of asymmetry of gait parameters and make recommendations for standardization, 2) to deepen the understanding of the relationships between sensorimotor deficits, spatiotemporal (step length, swing time and double support time) and biomechanical (kinematic, kinetic, muscular activity) parameter asymmetries during gait and, 3) to summarize the impacts of gait asymmetry on walking speed, falls, and energy cost in individuals post stroke. In light of current literature, we recommend quantifying spatiotemporal asymmetries by calculating symmetry ratios. However, for other gait parameters (such as kinetic or kinematic data), the choice will depend on the variability of the data and the objective of the study. Regardless of the selected asymmetry equation, we recommend presenting the asymmetry values in combination with the mean value of each side to facilitate comparisons between studies. This review also revealed that sensorimotor deficits clinically measured are not sufficient to explain the large variability of spatiotemporal asymmetries (particularly for step length and double support time) in individuals post stroke. Biomechanical analysis has been identified as a relevant approach to understanding gait deviations. Studies that linked biomechanical impairments to spatiotemporal asymmetries suggest that a balance issue and an impaired paretic forward propulsion could be among the important factors underlying spatiotemporal asymmetries. In our opinion, this paper provides meaningful information to aid in better understanding gait deviations in persons after stroke and establishes the need for future studies regrouping individuals post stroke according to their spatiotemporal asymmetries. Furthermore, further studies targeting efficacy of locomotor rehabilitation and the impacts of gait asymmetry on risk of falls and energy expenditure are needed.

Keywords: Stroke; Asymmetry; Gait; Rehabilitation; Sensorimotor impairments

Introduction

Stroke is one of the main causes of adult disability in most countries [1-3]. People living with stroke present several sensorimotor deficits such as contralateral and ipsilateral muscular weakness [4,5], contralateral spasticity [6,7], lack of coordination [8], contralateral impaired sensitivity [9-11], and impaired balance [12]. These sensorimotor deficits are heterogeneous among individuals post stroke and vary according to the size and location of the lesion. After a rehabilitation program, about 11% of individuals post stroke walked with assistance whereas 50% walked independently [13]. In addition to the reduced walking speed, these ambulatory individuals present a gait pattern often characterized by asymmetries in spatial and temporal parameters [14,15]. Walking speed has been shown as an excellent indicator of functional status and quality of life [16]. However, recent studies suggested that the level of asymmetry in different gait parameters could be more relevant than walking speed to understand the degree of paretic leg impairment and the compensatory mechanisms used by individuals post stroke during locomotion [17,18]. Furthermore, these persons are preoccupied by these asymmetries and want to walk 'normally'. One of their main objectives is to present the appearance of a normal gait pattern [19,20]. Therefore, rehabilitation professionals devote a significant portion of their interventions to restoring a more symmetrical gait pattern. Until now, the causes of spatiotemporal asymmetry and its impacts on functional status in individuals post stroke have not been clearly established. This could explain the mitigated efficacy of intervention in reducing gait asymmetry in this population.

In recent years, many studies aiming to better understand gait asymmetry following a stroke have been published. Their results are interesting and need to be summarized and analyzed along with important factors such as functional status and sensorimotor deficits of the individuals, and the index used to quantify the level of asymmetry. The lack of standardization in the quantification of the spatiotemporal asymmetry of gait makes it difficult to compare studies and gain a global comprehension of gait asymmetry in individuals post stroke.

The purpose of this topical review is therefore threefold: 1) to summarize different methods used to report gait asymmetries, to discuss their limits and make recommendations for standardization, 2) to deepen the understanding of the relationships between spatiotemporal asymmetries, sensorimotor deficits, and biomechanical parameter asymmetries (kinematic, kinetic, muscular activity) during

gait, and 3) to summarize the impact of gait asymmetry on walking speed, falls, and energy cost in individuals post stroke.

Quantification of Gait Symmetry

Various equations including variables measured on each side of body (paretic (P) and non-paretic (NP)) are proposed in research literature. The following equations, most commonly reported, used discrete values to quantify asymmetry between sides of different gait parameters. However, it is also possible to quantify regions of deviation in gait symmetry profiles (regarding timing and magnitude) through the use of cross-correlation analysis [21,22].

Difference between sides: (P-NP) or (NP-P) (raw and absolute value)

The difference between sides is a simple way to quantify symmetry. Difference of 0 represents perfect symmetry. With raw difference, the sign of the value indicates the direction of the asymmetry whereas when the absolute value is used, the amplitude of asymmetry is provided.

The asymmetry is not expressed relative to a given value (e.g. the value on one side) which could significantly influence the clinical meaning of the difference. For example, a 5 Newton meter (Nm) difference between sides relative to 60 Nm does not have the same meaning as the same difference relative to 15 Nm. Therefore for parameters with high inter-individual variability, the difference between sides must be used with caution when comparing asymmetry among different individuals. Furthermore, the same precaution holds when asymmetry of different parameters or at different joints are of interest.

Symmetry ratio: (NP/P or P/NP)

Ratios use values obtained from one side over the other. Ratio of 1 represents perfect symmetry. Patterson et al. have compared different equations of spatiotemporal asymmetry (symmetry ratio, symmetry index, log transformation of the symmetry ratio and symmetry angle) to recommend a standardized approach [14]. They found high correlations between these equations ($r \geq 0.97$) and concluded that no expression has a unique advantage over another. Therefore, they proposed to use symmetry ratio because it can be easily interpreted. However, the following limits must be taken in account. First, the distribution of the symmetry ratios might not be normal because the ratio might be artificially inflated when the value of the denominator is low [23,24].

Second, the value of the ratio is affected by direction of the asymmetry and will give a different weighting if an average is calculated. For example, 0.5/1 and 1/0.5 refer to the same level of asymmetry (inverse direction) but the average of both ratios is 1.25 (a bias toward an asymmetry ratio greater than 1) and not a value of 1 which should be the correct interpretation of the average asymmetry of these two subjects (mean value obtained from Equation 2 in Table 1 for an example of this phenomena). To avoid this influence, Patterson et al. recommended computing the ratio of asymmetry by using the greatest value as numerator and indicating the direction of the asymmetry with a sign convention (+ or -) [14]. It is also possible to avoid this bias by quantifying asymmetry with some symmetry indices that use the mean or the sum of each side value as denominator.

Symmetry indices (SI)

Symmetry indices normalize the value of one side or the difference of values between sides according to a reference value in the denominator. This quantification allows inter-individual, inter-segmental, and inter-parameter comparisons. Among various possibilities of SI [14, 17, 25-29], current examples are:

$SI_{NP\ or\ P}$ = [((NP-P)/NP) x 100] or [((NP-P)/P) x 100]

$SI_{highest}$ = [((NP-P)/highest value) x 100]

$SI_{average}$ = [((NP-P)/0.5 (NP+P)) x 100]

SI_{sum} = [((NP-P)/(NP+P)) x 100] or [(NP/(NP+P)) x 100]

A value of 0% represents perfect symmetry for these indices except for the second SI_{sum} equation for which a value of 50% represents perfect symmetry. Limitations of some symmetry indices were analyzed in a study by Zifchock et al. [24]. First of all, if the NP side or P side is used as the denominator, the same limits previously described for symmetry ratio occur when asymmetry varies in directions. To avoid a bias toward a direction of asymmetry as shown in Table 1 (mean value obtained from the Equation 3), it is recommended to use the mean or the sum of each side value as denominator ($SI_{average}$ or SI_{sum}) [24]. Using the $SI_{highest}$ index will also avoid this bias (see Table1, Equation 4).

To our knowledge, no study has compared $SI_{average}$ and SI_{sum}. Further studies comparing these indices are needed. However, when value of one side is positive and value of the other side is negative, which can be observed for angles and angular velocities in gait, the mean or the sum of these two values could be very small and the SI will be inflated. To avoid this situation, Zifchock et al. proposed the use of an angle function symmetry calculation as described in the next paragraph [24].

Symmetry angle (SA): [(45°-arctan (P/NP))/90*100]

This method does not require the choice of a reference value as shown in the previous equation [24]. The result is highly correlated with $SI_{average}$ for strength, kinetic and structural variables whereas for angular velocities, the association is lower ($r \leq 0.63$).

This latter association is lower because some of the $SI_{average}$ values are artificially inflated since some values were positive on one side and negative on the other side. The authors showed that this lower correlation was corrected when these artificially inflated $SI_{average}$ values were removed from the analysis [24]. In light of this result, the SA seems to be appropriate to express asymmetry of kinematic data during gait because it removes the artificial inflation and the choice of a reference variable. For all details on the rationale behind this calculation, see Zifchock et al. [24].

In summary, the choice of the asymmetry equation is not simple and should be based on the type of variables compared between sides according to how they vary. Hence, for spatiotemporal parameters, despite some limitations, it is suggested to quantify asymmetry by calculating symmetry ratios [14]. For other gait parameters (such as kinetic or kinematic data), the choice depends on the variability of the data and the objective. Although SI indices have some limitations, they could be clinically interesting since they are easier to interpret than SA. Regardless of the selected asymmetry calculation, we recommend presenting the asymmetry values in combination with the mean value of each side to facilitate comparisons between studies.

Each side value	S1	S2	S3	S4	S5	S6	Mean
Nonparetic (meter)	0.39	0.59	0.47	0.25	0.40	0.37	0.41
Paretic (meter)	0.25	0.40	0.37	0.39	0.59	0.47	0.41
Equations							
1. NP-P	0.14	0.19	0.10	-0.14	-0.19	-0.10	0.00
2. NP/P	1.56	1.48	1.27	0.64	0.68	0.79	1.07
3. (NP-P)/NP *100	35.90	32.20	21.28	-56.00	-47.50	-27.03	-6.86
4. (NP-P)/highest value*100	35.90	32.20	21.28	-35.90	-32.20	-21.28	0.00
5. (NP-P)/(0.5(NP+P))*100	43.75	38.38	23.81	-43.75	-38.38	-23.81	0.00
6. (NP-P)/(NP+P)*100	21.88	19.19	11.90	-21.88	-19.19	-11.90	0.00
7. NP/(NP+P)*100	60.94	59.60	55.95	39.06	40.40	44.05	50.00
8. (45°-arctan (NP/P))/90)*100	-13.71	-12.07	-7.54	13.71	12.07	7.54	0.00

Table 1: Step length asymmetry values according to different equations. This table presents step length of 6 subjects (fictive data) presenting asymmetries in different directions (S1, S2 and S3 present a higher non-paretic step length and S4, S5, and S6 present a higher paretic step length). Abbreviations: Non-paretic (NP); Paretic (P).

Gait symmetry in Healthy Individuals

Overall, gait of healthy individuals is considered as being symmetrical [30]. However, some authors have shown that healthy individuals present minor asymmetries between the two limbs during walking [31,32]. The differences between sides are variable among the gait parameters and thus a unique value of asymmetry (ex. 10% difference) could not be established. Recent work by Patterson et al. quantified spatiotemporal differences between sides in a large group (more than 80) of healthy individuals in order to determine the threshold value of asymmetry for spatiotemporal parameters during overground gait [14]. They proposed the following definition: individuals are asymmetric when their values fall outside the 95% confidence interval of healthy subjects. Their results establish threshold ratios of asymmetry (highest value/lowest value) of 1.08, 1.05 and 1.04 for step length, stance time and double support time ratio, respectively.

Gait Asymmetry in Individuals Post Stroke

To assess gait asymmetry in individuals post stroke, both level ground and treadmill walking studies (without body weight support) have been analyzed since their results are globally comparable as mentioned by Kautz et al. [33]. Knowing that sensorimotor impairments vary substantially among individuals post stroke, it is expected that large variability will also exist in their level of gait asymmetry. For detailed descriptive reports of asymmetries of spatiotemporal, kinematic, and kinetic gait parameters in individuals with stroke, the readers are invited to consult previous papers [34-36].

Spatiotemporal asymmetries and their relationships

Temporal asymmetry: Studies reported that around 60% of individuals post stroke presented temporal asymmetry [14,15] with stance time [37-39], single stance time [40,41], double support time [14,29] and swing time [25,38-40] being the parameters most often reported. A recent study quantified the correlations between asymmetry of different temporal gait parameters in 161 individuals post stroke [14]. Except for the double support time, the asymmetries of these temporal parameters were highly correlated to each other ($r \geq 0.81$). They concluded that stance time, swing time and double support time might represent different aspects of the gait control and it may be relevant to report each distinctively [14]. However, stance time is composed of two periods of double support time and one period of single stance: the latter corresponding to the contralateral swing phase (Figure 1) and therefore reflects what is happening in all the aforementioned temporal events. For this reason, it is relevant to stipulate that stance time and swing time (contralateral single stance phase) might give redundant information about gait impairments. Indeed, the higher asymmetry ratio observed in swing compared to stance is probably the result of swing phase duration (or percentage) that is lower than stance phase in gait [40,42]. Therefore, the differences in the asymmetry ratio of swing and stance are artificially amplified by the mathematical expression of the asymmetry and thus do not highlight different aspects of gait motor control. One should take into account that an individual presenting a problem of leg oscillation and another individual presenting a problem of stability during paretic stance will both have asymmetry in both swing and stance phase because these gait parameters are related. We therefore recommend reporting double support time asymmetry and swing time (or single stance time) asymmetry distinctively in future studies in individuals post stroke.

With regards to swing phase asymmetry, it is usually observed that values on the paretic side are higher than the non-paretic side [14,29,42-47]. When compared to healthy control individuals, the swing time asymmetry in individuals with stroke is the result of different combinations of asymmetry: augmentation of the paretic swing time, reduction of the non-paretic swing time or both an increased paretic swing time and a decreased non-paretic swing time [25,29,48]. For individuals post stroke with double support time asymmetry, 56% present a longer paretic double support phase [14] which is when the paretic leg is posterior to the non-paretic leg (paretic pre-swing). Very few studies reported the asymmetry of the

double support phases even if these phases are important in gait since they allow the transfer of weight from one leg to the other (stance-to-swing transition) and are seen as stable phases of gait considering that the two feet are on the ground.

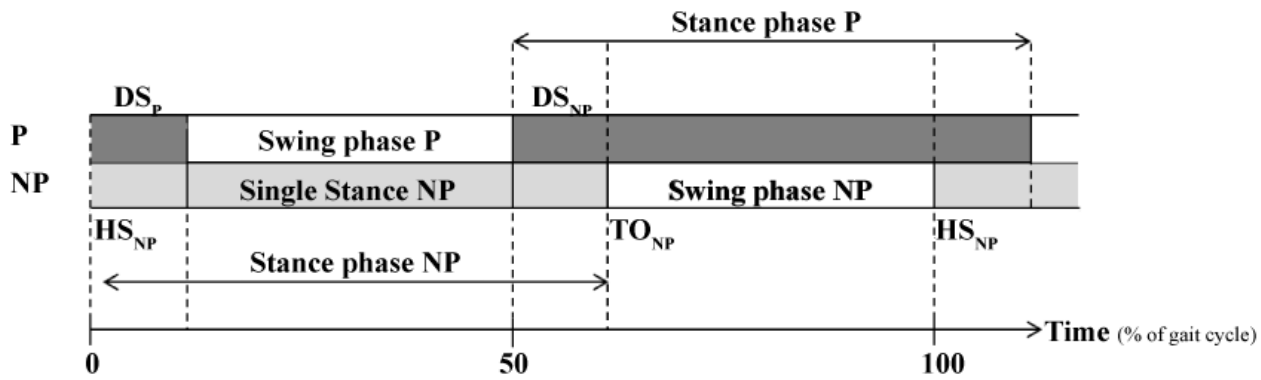

Figure 1: Temporal events of gait. Abbreviations: Paretic double support phase (DS_P); Non-paretic double support phase (DS_{NP}); Paretic Heel Strike (HS_P); Non-paretic Heel Strike (HS_{NP}); Non-paretic Toe off (TO_{NP}).

Spatial asymmetry: Spatial asymmetry in individuals post stroke is mainly concerned with step length asymmetry and is less frequent than in temporal asymmetry (33 % to 49 % vs. 60%) [14,15,27] and more variable. Indeed, among individuals with step length asymmetry, 47% to 76% present a longer step on the paretic side [14,27]. In order to better understand the variability of step length asymmetry, a group of researchers [26] suggested dividing step length into two spatial components, which are the trunk progression (TP) during step and the forward foot placement (FFP) relative to the trunk at heel-strike [26]. This analysis of the step length relative to these two components is very interesting because each component could be explained by specific biomechanical impairments. In Roerdink and Beek's study, individuals with stroke present greater TP during the paretic step than during the non-paretic step. However, the direction of asymmetry in FFP varied in direction among the individuals. Furthermore, step length asymmetry was determined by the sum of the asymmetries in these two components [26]. Reporting these spatial parameters in future studies will certainly help to better explain the biomechanical compensatory strategies that lead to step length asymmetry in individuals post stroke.

Relationship between spatial and temporal asymmetries: A theoretical framework on temporal and spatial asymmetries stipulated that these parameters are not directly related [49]. This statement is supported by empirical data that showed significant but modest correlations between step length ratio and swing time ratio (r = 0.47) and between step length ratio and stance time ratio (r = 0.58) [14]. Recent studies on locomotor adaptation during split-belt treadmill also showed that temporal parameters (phase shift) could be adapted independently of the spatial parameters (center of oscillation shift) [50-52]. These studies support the hypothesis that separated mechanisms are responsible for temporal and spatial gait parameters.

Relationship between Spatiotemporal Asymmetry and Sensorimotor Deficits

In this section, spatiotemporal asymmetries will be analyzed along with the sensorimotor deficits regardless of the equation used to quantify asymmetry.

Temporal asymmetry

Previous studies found that temporal asymmetry correlated with spasticity of the paretic ankle plantarflexors (r = 0.73) [37], isometric dorsiflexor strength (r=0.60) [46], plantarflexor strength (r = 0.33) [46], motor function of the paretic lower extremity (r from -0.53 to -0.88; measured with Chedoke McMaster Stroke Assessment (CMSA), Fugl-Meyer Assessment (FMA), or Brunnstrom's Motor Recovery Stage (BMRS)) [15,28,37,43,53-55], postural sway (r=0.77) [29], and with ankle joint position sense (r = 0.38) [46]. A regression analysis revealed that spasticity of the paretic ankle plantarflexors and motor function of the paretic lower extremity (assessed by FMA) explained 71% of the variance of single stance time asymmetry (with the sensation of the paretic lower extremity, the model explained 76% of the variance) [37]. Another study revealed that paretic dorsiflexion muscle strength and ankle joint position sense explained 51% of the variance of swing time asymmetry [46]. However, other authors who stratified participants according to the level of motor recovery (BMRS) did not find any difference within the group for swing time asymmetry [56]. Lastly, although no correlation has been found between visuospatial neglect and temporal asymmetry, it may be worth considering this aspect because according to Alexander et al. [53] individuals who present this deficit are more likely to present temporal asymmetry [53].

Spatial asymmetry

Relationships between step length asymmetry and sensorimotor deficits are weaker than the relationships between temporal asymmetry and sensorimotor deficits. Step length asymmetry significantly correlated with ankle spasticity (r = 0.75) [37], isokinetic ankle plantarflexor peak torque (r = 0.53), plantarflexor total work (r = 0.53) [37] and isometric plantarflexor strength (r = 0.28) [46], as well as motor function of the paretic lower extremity (r = 0.44) [28,37]. However, other authors who assessed ankle spasticity with the same outcome measure (Modified Ashworth Scale) did not find any significant association between spasticity and step length asymmetry (r = 0.32) [28]. Lastly, a regression analysis revealed that ankle spasticity and work of the knee extensors during an isokinetic maximal

voluntary contraction explained 46% of the variance of the step length asymmetry [37].

Globally, these relationships between sensorimotor deficits and spatiotemporal asymmetries suggested that sensorimotor deficits clinically measured are not sufficient to explain the large variability of spatiotemporal asymmetries in population post stroke. The work of Patterson et al. [38] supports this interpretation. Their study performed on 171 individuals post stroke concluded that spatial and temporal asymmetries were worse in the later stages post stroke, whereas neurological deficit and lower-extremity motor impairment (measured with the National Institutes of Health Stroke Scale and the Chedoke-McMaster Stroke Assessment) were not [38]. The same group of authors also showed that even patients with mild sensorimotor deficits could present considerable temporal asymmetries [15] and sensorimotor deficits could be observed with or without gait asymmetries. Different factors could explain this lack of association such as: 1) the variability of the clinical tools used to assess sensorimotor deficits and their validity to measured capacities required in gait [57] (e.g. isometric strength is not necessarily representative of the muscles' strength during gait), 2) the variability of the equations used to calculate gait asymmetry, and 3) the relative importance of each sensorimotor deficit in gait and its subtasks. For the latter, it has already been proposed that a minimum threshold of sensorimotor function is needed to be able to achieve the different gait subtasks. When one sensorimotor deficit (e.g. muscle strength) has a value under this threshold, it affects the realization of the functional task and could even be a limiting factor preventing an optimal gait pattern [58]. Lastly, 4) the same presentation of spatial or temporal asymmetry could be explained by different deficits. For example, impaired control of the oscillated leg or impaired weight control during stance will both create an asymmetrical swing time. Biomechanical analysis has been identified as a relevant approach to understanding gait deviations [59]. In the next section, this approach will be used to better understand the mechanism underlying spatiotemporal asymmetries in persons post stroke.

Biomechanical Impairments Underlying Spatiotemporal Asymmetries during Gait

Following a stroke, it has been shown that many biomechanical parameters are asymmetrical [34-36,60]. Figure 2 presents the antero-posterior and vertical components of the ground reaction forces (GRF) during self-selected gait in healthy individuals (n=14) and individuals post stroke (n=35) on both sides (paretic and non-paretic). Detailed descriptions of biomechanical modifications in individuals post stroke of joint angles, net joint moments and net joint powers have already been reported [34-36,60].

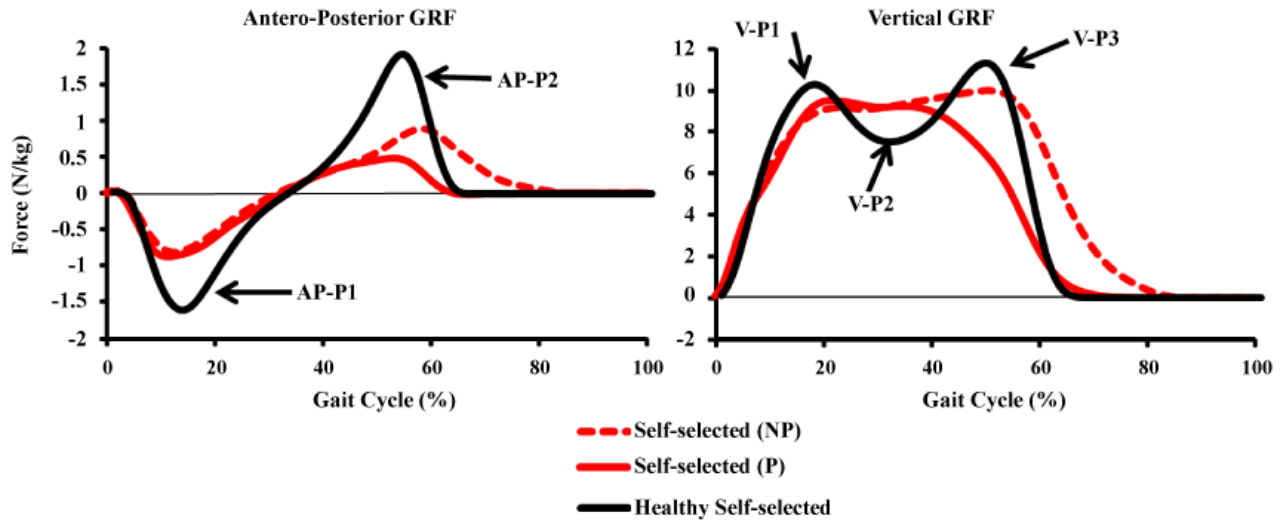

Figure 2: Antero-Posterior and Vertical Ground Reaction Forces. This figure illustrates the mean antero-posterior and vertical components of the ground reaction force (GRF) over a normalised gait cycle in healthy individuals (black line) and individuals post stroke for the paretic side (P; full red line) and non-paretic side (NP; dotted red line) during gait at self-selected speed. The mean (standard deviation) gait speeds for healthy individuals (n=14) and individuals post stroke (n=35) were respectively 1.26 (0.19) m/s and 0.72 (0.26) m/s. Abbreviations: Antero-Posterior (AP); Braking force (negative peak; AP-P1); Propulsive force (positive peak; AP-P2); Vertical (V); First maximal peak (V-P1); Minimum peak between first and second maximum peaks (V-P2); Second maximum peak (V-P3).

However, to date, few studies have considered regrouping the participants according to the direction of the spatial and temporal asymmetries to explain the role of biomechanical parameters in gait asymmetry. This stratification could be particularly important for step length asymmetry, because as opposed to temporal parameters (e.g. swing time), it substantially varied in direction (shorter paretic step or longer paretic step). Although only three studies present biomechanical data according to the direction of step length asymmetry [17,45,54], an examination of the biomechanical changes that could explain temporal and spatial asymmetries following a stroke are presented in this section. To do so, it is relevant to review the main subtasks (or requirements) of gait previously established by Winter [61]. They are: 1) to support the upper body during stance, 2) to control the foot trajectory during swing, 3) to generate the mechanical energy to maintain or to increase the forward velocity, 4) to absorb the mechanical energy to reduce shock or to decrease the forward velocity

of the body, and 5) to ensure balance and safe walking. Optimally, these subtasks should be accomplished using energy conservation measures [34,62].

Biomechanical impairments underlying temporal asymmetry

Swing time or single stance time asymmetries: As previously mentioned, the majority of individuals post stroke present an increased paretic swing time compared to the non-paretic side [14,29,42-47]. The resulting asymmetry could be caused by a reduction of the non-paretic swing time (e.g. due to an early foot contact by the non-paretic side), a prolongation of the paretic swing time, or by a combination of both factors. Using a correlative approach, two studies have assessed the association between asymmetry in swing time and asymmetry in kinetic parameters [45,63]. Kim and Eng [45] found a significant correlation between asymmetry in swing time and asymmetry in the average of the vertical ground reaction force (GRF; $r = 0.678$; $p<0.01$) [45]. Thus, when the swing time on the paretic side is longer than on the non-paretic one, the vertical GRF is smaller on the paretic side. This could reveal a difficulty in controlling balance during paretic stance (Figure 3, link D-5) [45]. Therefore, the non-paretic limb might need to be quickly oscillated to bear weight on the good safe side [40]. This asymmetry in vertical GRF during double support phases is corroborated by other authors [54,64]. A second study found a significant correlation between asymmetry in single stance time and the asymmetry of different parameters of the center of pressure (COP) during single stance [63]. The COP under the paretic side at single stance demonstrated reduced antero-posterior (AP) displacement, AP velocity, and medio-lateral (ML) displacement variability compared to the non-paretic side. These asymmetries were correlated with single stance time asymmetry (AP displacement: $r = 0.76$, AP velocity: $r = 0.55$, and ML variability: $r = 0.59$) [63]. As Kim and Eng, the authors suggested that altered AP COP parameters on the paretic side may represent difficulties in controlling balance and forward progression over the paretic limb during single stance (Figure 3, link D-5). They also suggest that the increased ML displacement variability on the non-paretic side could reflect the difficulty in controlling the oscillation of the paretic leg (Figure 3, link A-2).

The results of a third study revealed a significant negative correlation between the magnitude of the pelvic lateral displacement during gait and the non-paretic swing time (or paretic single stance time) [65]; subjects having the greatest displacements had the shortest paretic single stance time (symmetry was not presented in this paper). Again, a lack of balance control, this time in the frontal plane when bearing on the paretic side was suggested to explain the results (Figure 3, link D-5).

Apart from this potential balance problem, other factors might impair the control of the paretic stance phase and could lead to swing time asymmetry. For example, a lack of hip flexion moment (eccentric action) from middle to late stance on the paretic side seen with reduced thigh movement in extension could prevent normal non-paretic swing and resulted in a shorter non-paretic swing time (Figure 3) [34,66]. Indeed, many studies have observed a reduced hip flexor moment associated with a reduced hip negative power burst (H2) from mid-stance to late stance of the gait cycle in stroke [34,36].

Asymmetry of swing time could also be due to impairment in paretic forward propulsion. The forward propulsion is quantified by the time integral of the positive antero-posterior GRF [67] and ensures the forward progression of the body during gait. This subtask of gait is frequently impaired in individuals post stroke [54,67,68]. As proposed by Olney et al. [34], an impaired paretic forward propulsion during the non-paretic swing could create an early foot contact by the non-paretic side (Figure 3, link C-5) [34]. Also, at paretic double support phase (when the paretic foot is behind the non-paretic foot) a part of the paretic forward propulsion is used to accelerate the paretic leg to swing. Therefore, an impaired paretic propulsion (e.g. caused by a reduced plantarflexor work [69] or moment impulse [67]), could contribute to an increase of the paretic swing duration by reducing the kinetic energy of the leg at toe-off (Figure 3, links C-2 and C-B-A) [25,69].

Therefore, any biomechanical parameters that could lead to a reduced paretic forward propulsion could theoretically lead to swing time asymmetry (e.g. plantarflexor muscles and moment impulse, knee extension moment impulse, hip flexion moment impulse (negative relation), weight bearing distribution, leg extension at push off, etc. see [67,68,70-72] for detailed description of parameters related to forward propulsion). As neurophysiological studies showed that a reduced weight bearing in double support phase could reduce plantarflexor muscle activity [72], this might also explain the association observed by Kim and Eng [45] between swing time asymmetry and weight-bearing asymmetry (Figure 3, link D-C-B).

Lastly, an impaired control of the swing initiation and foot trajectory during paretic swing could also lead to an increased swing time (Figure 3, links A-2 and B-2). For example, in swing initiation, the power delivered to the swing leg is not only created by the ankle push-off but also by the hip flexors muscle that contracts to move the leg upright and forward (pull-off) [73]. Therefore, a reduced hip flexor power could also influence paretic swing time. It is also important to remember that individuals post stroke could present various kinematic strategies during swing phase such as a reduced knee flexion (e.g. caused by hypertonic knee extensors or reduced leg velocity), higher hip abduction, lower dorsiflexion, and hip hiking or circumduction that could lead to an increase paretic swing time (Figure 3, link A-2) [34,74].

Double support time asymmetry: Although some discrepancies exist in the direction of symmetry of double support time in individuals post stroke [14], studies that reported double support time in biomechanical analysis found that the majority of individuals with stroke presented a longer paretic double support time (paretic foot behind) [54,74]. At this critical event, the pushing limb generates energy while the weight-accepting limb absorbs energy.

The factors explaining double support asymmetry could be common to those explaining swing time asymmetry. For example, if the paretic leg generates less energy during late stance and toe-off than the non-paretic leg [25,69], the transition of the weight from the paretic leg to the non-paretic leg is less efficient and slower (Figure 3, links B-4 and C-4). This might result in a longer paretic double support time which could be explained by a delay in the initiation and a decrease in the speed of flexion of the hip during the swing phase [74]. Second, the lack of confidence for paretic weight bearing that leads to a quick advancement of the non-paretic leg immediately after the initial paretic heel strike [40] is also a factor that could contribute to a shorter non-paretic double support time (non-paretic foot behind) (Figure 3, link D-6). Further studies are needed to better understand asymmetries in double support time in individuals post stroke along with the biomechanical parameters and the between and inter-limb compensations.

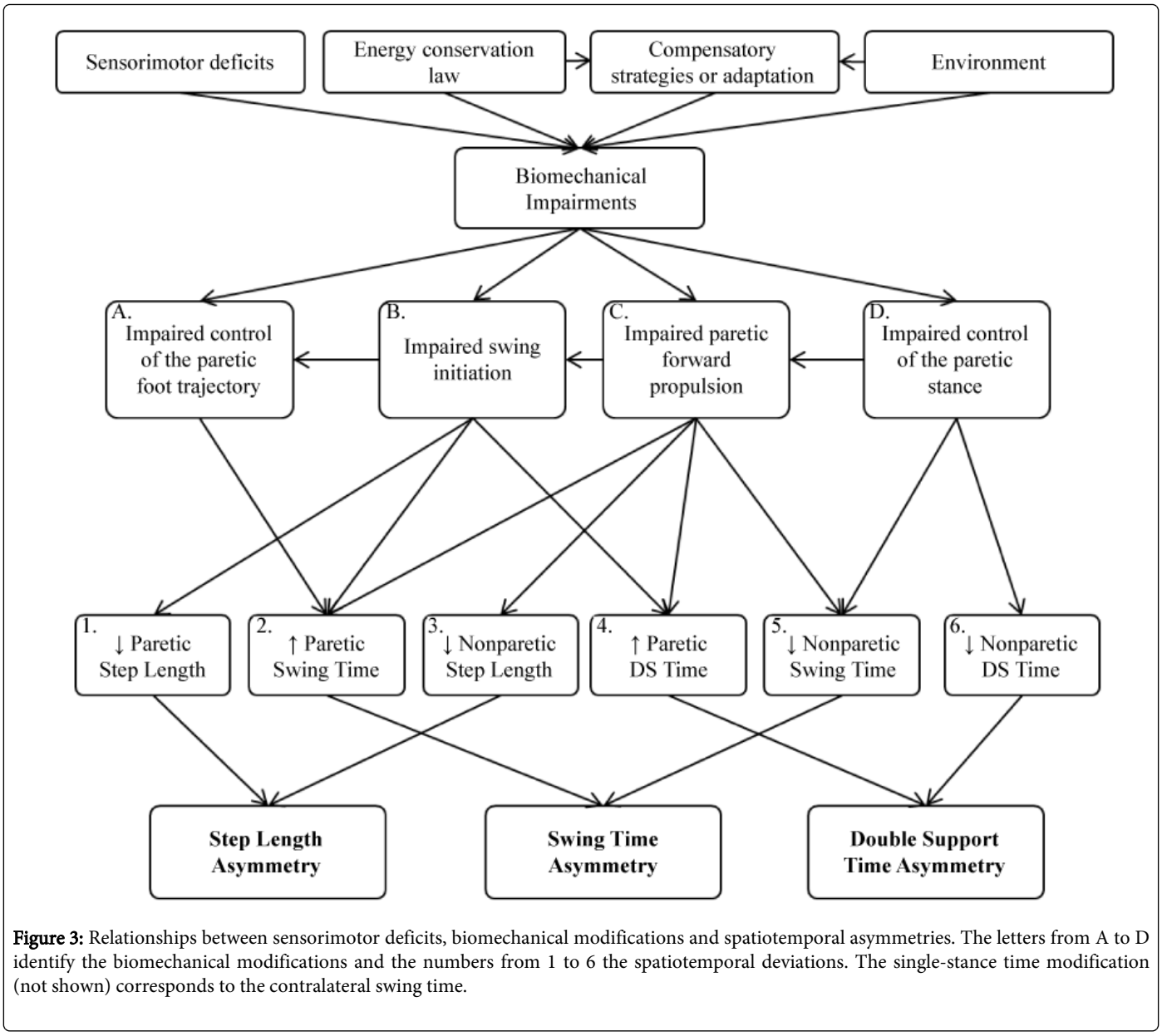

Figure 3: Relationships between sensorimotor deficits, biomechanical modifications and spatiotemporal asymmetries. The letters from A to D identify the biomechanical modifications and the numbers from 1 to 6 the spatiotemporal deviations. The single-stance time modification (not shown) corresponds to the contralateral swing time.

Biomechanical impairments underlying spatial asymmetry

To our knowledge, three studies have analyzed the biomechanical parameters considering the direction of asymmetry (shorter paretic, longer paretic, or equal step length) in an aim to better understand compensatory mechanisms that lead to spatial asymmetry [17,45,54]. The first study did not find significant association between step length asymmetry and average vertical GRF asymmetry for two different groups (longer paretic step and shorter paretic step) [45]. The authors concluded that step length asymmetry is probably the result of different compensatory strategies. However, examining the association between the AP propulsive GRF impulse (which is representative of the forward propulsion) and step length asymmetry, a second study found a significant negative correlation between the two parameters (r=-785) with the AP propulsive GRF impulse explaining 62% of the variance in step length asymmetry [54]. Individuals showing greater decreased paretic forward propulsion compared to the non-paretic side were those having the longer paretic step compared to the non-paretic step (Figure 3). Consistent with these results, a third study that compared AP propulsive GRF impulse of individuals post stroke with healthy controls showed that the paretic AP propulsive GRF impulse during paretic late single stance was reduced in individuals with a longer paretic step and in individuals with symmetrical step whereas it was increased in individuals with a longer non-paretic step [17]. During non-paretic late single stance, individuals post stroke with a longer paretic step had more non-paretic leg AP impulse than controls whereas it was reduced in individuals with a longer non-paretic step. The authors explained this relation by the fact that a greater non-paretic propulsion will cause the trunk to move forward during the paretic swing, thereby increasing the paretic step. As several biomechanical factors could affect the forward propulsion, the authors quantified the joint moment impulses of the hip, knee, and ankle during late single stance phase and double support phase. Briefly, their results showed that all groups (longer paretic, shorter paretic and equal step length) presented a reduced plantarflexion moment impulse

in late single stance and double support phases, which is coherent with the reduced trunk progression during the non-paretic step (when the paretic foot propulses) previously found by Roerdink et al. [26]. However, in individuals with longer paretic step, this observed reduction in plantarflexion moment impulse was more important [17]. In addition, these individuals showed an increase of the non-paretic plantarflexor moment impulse at the late single stance. Individuals post stroke with symmetrical step length compensated for the reduction of the plantarflexor moment impulse by bilaterally increasing the hip flexors moment impulse at the late single-leg stance and at double support. Lastly, stroke individuals with a shorter paretic step length did not present compensation on the non-paretic leg or in other joints and therefore the authors suggest that the reduction in paretic AP impulse at pre-swing decreases the energy delivered to the leg and leads to a reduced paretic step length (Figure 3, link B-1) [17]. These aforementioned results demonstrated that step length asymmetry is representative of different compensatory strategies. It also revealed that walking with symmetrical step length does not imply that the individuals had no impaired paretic forward propulsion. Therefore, this supports the pertinence of partitioning step length into trunk progression and forward foot placement as proposed by Roerdink et al. to better understand underlying impairments and gait compensations of individuals with stroke [26]. Figure 3 summarizes by presenting the potential relationships between biomechanical impairments and spatiotemporal asymmetries.

Relation between Spatiotemporal Asymmetry and Walking Speed, Falls, and Energy Expenditure

Walking speed

There is no consensus on the impact of spatiotemporal asymmetry on gait speed. Many studies have found a significant correlation between temporal asymmetry and walking speed ($r \geq -0.54$) [14,15,29,45,53,55,75] whereas others have found no significant relationship [39]. A point that emerges is that relation between walking speed and temporal asymmetry is not linear since individuals walking at slow speed show a more important association with temporal asymmetry (<0.6m/s) [14,15]. Relation between walking speed and step length asymmetry is generally low (r values ranging from -0.35 to -0.46) [14,54,75] or has not been found significant [15,45]. More studies are required to explain how spatiotemporal asymmetry influences gait speed or vice versa. The initial cause (e.g. impaired paretic propulsion) of the spatiotemporal asymmetry will be important to consider in order to improve symmetry and gait speed. One should keep in mind that spatiotemporal asymmetry (e.g. shorter non-paretic step length) can be improved by reducing paretic step length (instead of increasing non-paretic step length) which will potentially decrease gait speed and have no effect on paretic forward propulsion.

Falls

To our knowledge, until now, no study has established a direct relation between spatiotemporal asymmetry and falls in stroke individuals although some studies have shown a significant relation between balance impairment and temporal asymmetry [29]. Furthermore, one study showed that individuals post stroke with step length asymmetry showed the higher variability in stride time compared to individuals post stroke with symmetrical step length [27]. A higher stride time variability is known to be related for risk of falls [76]. However, we do not know whether asymmetrical gait is more instable than symmetric gait or if individuals adopt an asymmetrical strategy to reduce their instability during gait. The assessment of dynamic stability during gait with new models such as the one proposed by Duclos et al. will help understanding of the relation between gait asymmetry and instability [77].

Energy cost

Some authors have measured the energy cost and the spatiotemporal parameters in individuals post stroke following different interventions (treadmill, botox injection, orthosis) [78-80]. They found that treadmill walking requires higher oxygen consumption when compared with overground (at the same speed) even if the treadmill induced a more symmetrical lower limb angular excursion (with no significant changes in spatiotemporal symmetry) [78]. In a second study, a reduction in energy expenditure was found in individuals post stroke after an Onabotulinum toxin A injection in the spastic hip flexor muscle [79] without changes in spatiotemporal parameters during walking. Lastly, Thijssen et al. [80] found that walking with a lower limb orthosis (orthosisa) (supporting affected hip, knee and ankle) led to a decrease in oxygen consumption and changed spatiotemporal parameters bilaterally without improving symmetry [80]. However, none of the aforementioned studies directly assessed the relationship between changes found in spatiotemporal and energy parameters. Therefore, no assertions can be made about the causal relationship between energy expenditure and spatiotemporal symmetry in stroke individuals.

Future Perspectives

This review clearly demonstrated that future studies are warranted to improve our understanding of gait asymmetry in individuals post stroke. Regarding the quantification of gait asymmetry, there is a necessity for further assessment, and examination of the parameters most affected to attempt to identify the impacts on functional level and recovery. In addition, variability between subjects must be addressed along with several biomechanical parameters. This will allow identification of the parameters that matter most for individuals post stroke, help classify these individuals into different subgroups and find the best locomotor intervention for each subgroup.

Research on gait asymmetry clearly requires a larger number of subjects be enrolled in the study in order to classify participants into subgroups. This will warrant collaboration between researchers. Lastly, since the same gait deviation and asymmetry can be caused by different factors, biomechanical analysis, which can reveal crucial information to pinpoint the cause of gait deviations, should be promoted to help clinicians choose the best intervention and thus improve the gait pattern of their patients. A recent review has suggested that an individualized approach based on the most important limiting factors has the best chance to improve task-oriented gait training interventions [59]. However, as mentioned by the authors, the feasibility of this approach must be demonstrated in futures studies. Emerging technologies (split-belt treadmill, virtual reality, robotic assistance or resistance, etc...) in rehabilitation research will certainly contribute to better understanding the impact of gait asymmetry on gait performance in the stroke population.

Conclusion

The relationships between post stroke sensorimotor deficits, biomechanical parameters and spatiotemporal asymmetries during gait are complex. A better standardization of the equations used to quantify gait parameters' asymmetries could help to create a better global comprehension of gait asymmetry in individuals post stroke. There is also a need to consider multiples factors that interact. Neurophysiological studies certainly could give further insight into the mechanism underlying these asymmetries [72,81]. Clinically, the biomechanical analysis is advantageous in identifying the causes underlying the gait asymmetry and providing useful information to the patient's clinician. Future studies could benefit from regrouping individuals according to their specific spatiotemporal gait asymmetries in order to identify the impairments underlying gait asymmetry and to improve the efficacy of rehabilitation intervention. Furthermore, the impacts of gait asymmetry on risk of falls and energy expenditure require further study.

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