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Review

Arm-swing kinematics in Parkinson's disease: A systematic review and meta-analysis☆,☆☆

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ABSTRACT

Background: Parkinson's disease (PD) causes postural instability and gait abnormalities that may be associated with an arm swing reduction.
Objective: To conduct systematic review and meta-analysis to determine the kinematic patterns of arm-swing during gait in people with PD
Methods: A computer literature search of the PubMed, EMBASE, WOS, PEDro, SCOPUS and SciELO databases was conducted. Terms related to PD and arm-swing were combined to find studies that performed a free walking evaluation of the arm-swing of PD patients on or off medication compared to healthy controls. After a standardized evaluation by three examiners, fifteen articles met inclusion criteria. Random effects meta-analysis models were utilized to quantify (1) the arm-swing range of motion (RoM); (2) the arm-swing amplitude; (3) the arm-swing velocity; and (4) the arm-swing asymmetry.
Results: On average, arm-swing RoM (7.07°), amplitude (0.8 cm), and velocity (0.31 m/s) were significantly decreased in PD compared to healthy controls. Healthy subjects had significantly more symmetrical arm-swing (8.16%) than people with PD. Effect sizes were moderate-large.
Conclusions: People with PD have significant differences in RoM, amplitude, velocity, and asymmetry of arm-swing during gait compared to the healthy control group. Medication phase does not significantly influence arm-swing characteristics. Further studies will be needed to determine whether different disease characteristics influence the biomechanics of arm-swing during gait.

1. Introduction

Arm swing reduction and asymmetry during gait in Parkinson's disease (PD) has been widely studied for being considered a prodromal sign of the disease. The arm-swing is an integrated movement in normal human gait, being the result of muscular activity originating in the locomotor circuits of the central nervous system, as demonstrated in different neurophysiological studies with electromyography [1]. The arm-swing has been closely related to lower limb and trunk movement, with gait speed, and with upper limb Range of Motion (RoM) [2]. Arm-swing reduction is clearly related to the dopaminergic depletion in the nigrostriatal circuit and overactivation of inhibitory pathways due to increased sub thalamic nucleus output over the GPi-SNr complex due to basal ganglia pathology. Some authors have shown that the use of dopaminergic medication (L-dopa) and the application of deep brain stimulation in the subthalamic nuclei can significantly improve the reduction of arm-swing and asymmetry [3,4]. The alteration of the relationship between the basal ganglia and the supplementary motor area is related to an erroneous gait motor program, and its altered relationship with the mesencephalic locomotor region and the

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pedunculopontine nuclei results in rigidity and cessation of gait [5]. These alterations result in reduced trunk movement, shorter steps, and asymmetry of the lower limbs during gait. However, no relationship has been shown between arm-swing reduction/asymmetry and gait length, trunk movement, arm RoM or gait parameters in PD [6]. The only fact related to the decrease in arm-swing has been the asymmetry of upper limb RoM between both hemi bodies [7]. This could be due to the somatotopic distribution of the striatum, in which the pathology could affect certain areas to a greater extent [3].

The function of the arm-swing is to facilitate lower limb movements during gait, improve dynamic balance and reduce the energy cost of walking [1], being a relevant feature in subjects with PD, who have shown alterations in lower limb movements during gait, reduced dynamic balance and increased energy cost during gait [8,9]. Despite the existence of studies on the relationship between arm-swing in PD and gait characteristics, there are some discrepancies among the different studies conducted on this topic. To date, the authors of this study are unaware of any other review that unifies the characteristics of arm-swing in PD, being necessary this study for the greater knowledge of this symptom so present in PD. The purpose of the present review and meta-analysis is to determine the kinematic patterns of arm-swing during gait in people with PD to provide a state of the art of the current body of evidence on this important feature present in PD subjects from the early stages.

2. Material and methods

2.1. Design

This review was reported following the PRISMA recommendations for reporting systematic reviews [10] (Supplementary Material). This systematic review and meta-analysis were registered in PROSPERO with the registration number: CRD42021271631.

2.2. Search strategy and database

The following databases were searched on August 2021: PubMed, EMBASE, The Web of Science, PEDro, SCOPUS and SciELO. Several keywords were used in reference to pathology, gait, and arm-swing, combined with Boolean operators (Complete search strategy in Supplementary Material).

2.3. Screening process and eligibility criteria

The titles and abstracts of the studies obtained from the literature search in the different databases were analyzed by two different researchers according to the following inclusion-exclusion criteria. Discrepancies were resolved by a third researcher.

Inclusion criteria: The literature search was limited to cross-sectional studies and clinical trials in English, Spanish that performed an assessment of people with diagnosis of idiopathic PD in on or off phase of medication, compared to healthy controls. To be considered eligible, studies had to present: subjects with a diagnosis of idiopathic PD, assess free walking, include data on kinematics of arm-swing during gait. *Exclusion criteria*: Studies were excluded if variables of interest were not reported, diagnosis of Parkinsonism, presence of freezing, presence of diseases other than PD. If researchers performed an intervention with pre-post gait analysis and included baseline analysis, these studies would be included (including only baseline arm-swing gait measurements). Cross-sectional studies without a control group would be included.

2.4. Data extraction

Standardized methodology was used to extract data from studies that met the inclusion criteria. Data on the first author, year of publication, design, number of patients, demographic data, Hoehn and Yahr (H&Y) scale, Unified Parkinson's Disease Rating Scale (UPDRS) score, medication phase (on-off), time since diagnosis, type of instrumental analysis performed, walking distance (m) and study results (kinematics) were extracted. In addition, means and SD of study results will be obtained. The authors of the included studies were contacted by email, aiming to access possible unclear data. If no answer was received, the data in question were excluded from the analysis.

2.5. Assessment of risk of bias (methodological quality)

To analyze the methodological quality of each individual study, the Newcastle-Ottawa Scale (NOS) [11]. The NOS was developed to assess the quality of nonrandomized studies with its design, content and ease of use directed to the task of incorporating the quality assessments in the interpretation of meta-analytic results. It uses a "star system" in which the study is evaluated through three sections: selection of the study groups; comparability of the groups; ascertainment of the result. It has been shown to have content validity and inter-rater reliability.

2.6. Data synthesis and analysis

Quantitative analysis included studies comparing differences between individuals with PD and healthy controls. The analysis was performed on the variables RoM of the shoulder during gait measured in degrees (°), arm-swing amplitude measured in meters (m), arm-swing velocity, measured in meters/second (m/s), and arm-swing asymmetry, or symmetry, measured in percent (%), obtained using the angle of symmetry described by Zifchock et al. [12].

The baseline gait values reflected in the mean and SD were used to find the comparison values between the PD group and the control group. The mean difference (MD) between groups was used when measurements were collected in the same unit and with comparable assessments; means were converted to the standardized mean difference (SMD), with a 95% confidence interval (CI) to obtain the effect size, or when means were not comparable. A random-effects model was used to determine the overall effect size: in the SMD, an effect size of > 0.8 was considered large, between 0.5 and 0.8 was considered medium, and between 0.2 and 0.5 was considered small [13], and P values < 0.05 were considered statistically significant. The degree of heterogenicity among the studies was estimated by Cochran's Q statistical test (with P values <0.05 considered to be significant) [14] and the inconsistency index (I^2). $I^2 >$ 25% was considered to represent small, $I^2 > 50\%$ medium and $I^2 > 75\%$ large heterogenicity [15]. The I^2 is a complement to the Q test, although it has the same power issues when the number of studies is small [15]. When the Q-test was significant ($P < .1$) and/ or the result of I^2 was $>$ 25%, indicating heterogenicity among the studies, the random-effects model was applied in the meta-analysis. A subgroup analysis was performed according to medication status, establishing the off group, with no effect of dopaminergic mediation, and the on group, under the effect of mediation. The asymmetry was evaluated using a funnel plot in those analyses formed by at least five studies, which indicates the possible risk of publication of small studies with negative results. Studies were analyzed with Review Manager 5.3 statistical software.

3. Results

3.1. Studies selection

A total of 1173 studies were retrieved. Duplicate studies were eliminated, leaving a total of 505 studies, on which a critical reading of the title and abstract was carried out. After first screening, there was a total of 68 studies, which were obtained and read in full text. Finally, 15 studies [16–30] were included in the qualitative analysis, and 11 [16–20,22,23–25,27,30] were included in the quantitative analysis, with a total of 423 subjects. Among them, 244 and 179 participants were

from PD and control groups, respectively. The whole screening process is shown in the PRISMA flow diagram (Fig. 1).Table. 1.

3.2. Characteristics of the included studies

Seventy-seven percent of the studies showed H&Y values, 66% provided UPDRS III information, 62% of the studies stated disease duration, 100% reported the age of the PD group, and 1% did not provide this information on the control group. The characteristics of the included studies are reflected in Table 2.

Several methods were used for gait analysis. Of the selected studies, 3 of them used an accelerometer-based system [20,21,28], 3 used the 3D Kinect video device [17,18,27], one study used 2D video analysis [16]. The remaining studies employed the VICON 3D multicamera video system [19,24,26] and optoelectronic video systems [22,23,25,30]. Data relating to the protocol and marker positions for data extraction are shown in Table 3.

3.3. Quality assessment

The methodological quality, assessed by the NOS scale, showed that 11 of the included studies had poor methodological quality, while 3 [21, 27,29] had fair quality and 2 had good quality [19,20] (Supplementary Material). Only two studies scored in "representativeness of the Cases" [19,26] and three in "selection of Controls" [19,20,25]. All studies met the items "same method of ascertainment for cases and control" and "non-response rate."

3.4. Study results

3.4.1. Arm swing range of motion (°)

Data regarding arm-swing ROM were available from 5 studies [17, 20,22,23,25]. The meta-analysis showed that the arm-swing ROM is approximately 7.07° reduced in PD compared with the healthy groups (MD: - 7.07; 95% CI: - 11.19; - 2.95; p:.08; I2: 45%) (Supplementary Material).

Regarding effect size, we found that the reduction in arm-swing ROM in individuals with PD compared with healthy subjects had a medium effect (SMD: −0.67, 95% CI: −1.06; −0.28, Z: 3.41, P < .001), with low not significant heterogeneity (*p*:.22; I^2: 26%) (Fig. 2). A subgroup analysis was performed according to medication status (off versus on) (Fig. 3). The subgroup analysis showed no significant differences between groups (p:.1), showing a significant reduction in the arm-swing ROM in both groups, being greater the mean reduction of the arm-swing ROM in the off group (MD: −10.84, 95% IC: −18.06; −3.63 vs MD: −4.56, % CI: −8.48; −0.64). The effect size was large in the off group (SMD: −1.06, 95% CI: −1.66; −0.47, Z: 3.15, P < .001), and medium in the ON group (SMD: −0.46, 95% CI: −0.88; −0.03, Z: 2.10, P < .04). There was moderate heterogeneity between subgroups (I^2: 62.4%). The funnel plot presents asymmetry, indicating the risk of publication bias (Fig. 3).

3.4.2. Arm swing amplitude (m)

Data regarding arm-swing amplitude were available in 4 studies [18, 19,23,24]. Subgroup analyses could not be conducted because only 1 study included analysis of amplitude in patients in the off state. The

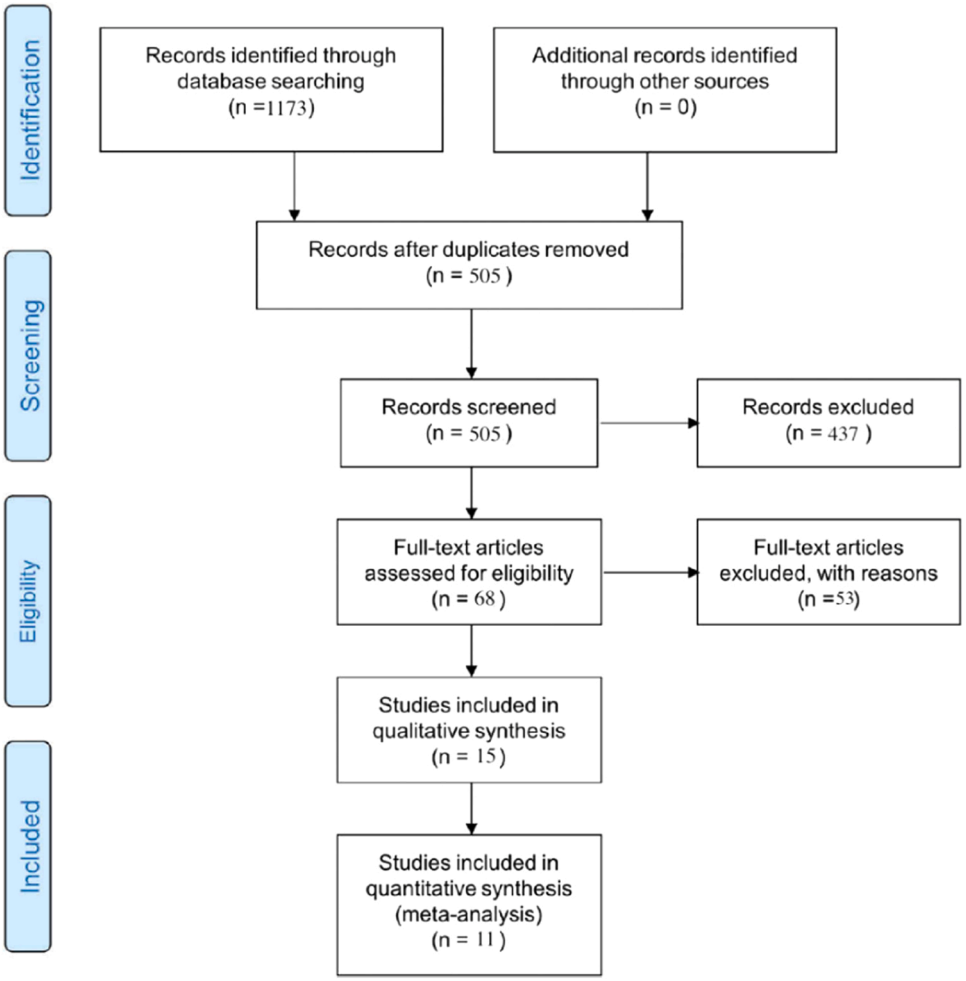

Fig. 1. PRISMA flow diagram.

Table 1
Characteristics of included studies.

n	Design	Sample demographics		Disease duration	H&Y	UPDRS III	FoGQ	ON / OFF
		Healthy	Parkinson Disease					
Behrman et al. [16]	Cross-sectional	n = 8 (2 women) Age = 72.7 (4.5)	n = 8 (2 women) Age = 72.9 (4.7)	11.6 (6.4) years	3 (0.75)	20 (9.4)	NR	ON
Boungiorno et al. [17]	cross-sectional	n = 14 (4 women) Age = 73.5 (6.4)	n = 16 (3 women) Age = 74.9 (7.6)	NR	2.43 (0.51)	NR	NR	ON
Castaño et al. [18]	Cross-sectional	n = 12 Age= 53–73	n = 12 Age= 53–73	NR	I-II	NR	NR	NR
Cole et al. [19]	Prospective observational study	n = 34 Age = 67.65 (2.2)	PD fallers n = 17 Age = 66.2 (1.4) PD non fallers n = 32 Age= 66.9 (2.1)	PD fallers = 6.3 (0.7) years PD non fallers = 3.9 (0.6) years	PD fallers = 1.8 (0.1) PD non fallers = 1.6 (0.2)	PD fallers = 34.5 (2.7) PD non fallers = 26.6 (3.7)	PD fallers = 5 (0.7) PD non fallers = 2.1 (0.6)	ON
Gera et al. [20]	Cross-sectional	n = 15 (7 women) Age = 60.9 (5.8)	n = 17 (13 women) Age = 60.9 (5.8)	8.8 (7.1) years	1.9 (0.3)	23 (14.5)	NR	ON & OFF
Huang et al. [21]	Cross-sectional	n = 8 (7 women) Age = 62.1 (7.3)	n = 8 (7 woman) Age = 63.2 (8.4)	15.5 (13.1) months	1.3 (0.5)	10.5 (4.5)	NR	NR
Isaias et al. [22]	Cross-sectional	n = 10 (7 women) Age = 64 (55–70)	n = 13 (6 women) Age = 64 (52–73)	5 (3–6) years	NR	21 (11–32)	NR	OFF
Koh et al. [23]	Prospective observational study	n = 23 (12 women) Age = 66.37 (5.85) Height = 1.61 (0.07) Weight = 64.26 (14.54)	n = 41 (20 women) Age = 63.13 (8.16) Height = 1.63 (0.09) Weight = 61.32 (9.43)	18.14 (21.91) months	2.05 (0.29)	22.39 (8.45)	NR	ON
Lewek et al. [24]	Cross-sectional	n = 8 (5 women) Age = 61 (12)	n = 12 (9 women) Age = 68 (8)	24 (10) months	1.29 (0.4)	11.25 (0.4)	NR	OFF
Mian et al. [25]	Cross-sectional	n = 13 (3 women) Age = 70 (10) Height = 1.73 (0.1) Weight = 78 (15)	n = 12 (9 women) Age = 71 (6) Height = 1.71 (0.1) Weight = 69 (12)	7.8 (4.3) years	2 (1.5–2.5)	30 (20–51)	NR	OFF
Mirek et al. [26]	Parallel clinical trial	n = 35 (16 women) Age = 64 (7,8)	n = 32 (18 women) Age = 66 (8.7)	5.7 (2.66) years	2.13 (0.45)	NR	NR	ON
Ospina et al. [27]	Cross-sectional	n = 25 (15 women) Age= 46–88	n = 25 (13 women) Age= 45–87	6 (IQR, 1–7) years	16% I, and 84% II	36.8 (13.41)	6.16 (4.74)	NR
Rincon et al. [28]	Cross-sectional	n = 11 Age = 60 (IQR: 51–66)	n = 10 Age = 63.5 (IQR, 53–76)	5 (IQR, 4–5) years	I (10%), 1.5 (20%), and II (70%)	25.8 (10.27)	NR	ON
Roemmich et al. [29]	Cross-sectional	n = 15 Age = 63.5 (8.29) Height = 1.70 (0.11) Weight = 74.2 (14.7)	n = 18 Age = 63.5 (8.93) Height = 1.69 (0.01) Weight = 76.5 (13.7)	NR	NR	22.7 (7.38)	NR	ON
Scandalis et al. [30]	Parallel clinical trial	n = 6 (5 women) Age = 58–67	n = 14 (6 women) Age = 65.5 (48–78)	NR	II-III	NR	NR	OFF

Data reported as median (SD), except for indicated cases. Abbreviations: Fallers: subjects reported as fallers; FoGQ: Freezing of Gait Questionnaire; H&Y: Hoehn and Yahr scale; Non fallers: subjects reported as non-fallers; NR: No Reported; Off: off medication state; On: On medication state; PD: Parkinson Disease

Table 2
Kinematic measurement characteristics.

Study	Type	Device	Measurement characteristics	Walking description
Behrman et al. [16]	2d video system	Peak Video Illustrator motion analysis system (Englewood, Colorado)	Markers at shoulder and elbow joint positions to collect data on left shoulder excursion and elbow joint motion during right limb gait	walked across a 7.5 m indoor level path. Data were collected from the middle 4 m of the walkway.
Boungiorno et al. [17]	3d video system	KinectTM Version 1	Reflective markers to track and record the position of the thumb index finger and the toes	Subjects walked toward the Kinect sensor, which was placed 3.5 m away from the subject's starting point at a height of 0.75 m.
Castaño et al. [18]	3d video system	KinectTM Version 1	Markers on both wrists.	The gait analysis was performed in a corridor 1.5 m wide by 4 m long. Each volunteer walked three times, from the farthest point to the Kinect.
Cole et al. [19]	three-dimensional motion analysis system	Motus 2000; Vicon, Oxford, UK	Markers were attached over specific anatomical landmarks on the trunk (sacrum, sternum, C7 spinous process), arms (lateral border of the acromion, olecranon process of the humerus, radial and ulnar styloids), and head (supra-auricular point, top of the head).	Participants performed six trials consisting of walking barefooted at a self-selected pace along a firm walkway
Gera et al. [20]	Inertial sensors	NR	Both wrists, dorsum of both feet, the sternum and at the fifth lumbar level	Walk seven meters, turn 180 degrees after crossing a marker on the ground and return to the initial starting point. Three trials of this protocol were performed.
Huang et al. [21]	accelerometers	Triaxial G-Link (MicroStrain, Inc.; Williston, VT)	Both forearms (30 cms from the wrist)	Subjects were instructed to walk continuously for about 8 min at a comfortable pace around a rectangular circuit in an indoor hallway approximately 400 m long.
Isaias et al. [22]	three-dimensional motion analysis system	SMART, BTS, Milan, Italy,	Spherical markers (15 mm diameter) attached to fixed bony landmarks.	Three series of six walking trials, at a "preferred", "slow" and "fast" speed, in random order along a 10 m course, following verbal instructions in the absence of external feedback.
Koh et al. [23]	three-dimensional motion analysis system	Qualisys Medical AB, Gothenburg, Sweden	Fifty-two reflective markers were placed on each participant's trunk, arms, forearms, pelvis, thighs, legs, and feet.	Gait was performed on an 8-m long walkway, an overhead body weight support harness, and force platforms in the center of the walkway.
Lewek et al. [24]	three-dimensional motion analysis system	Vicon Nexus, Lake Forest, California	Retroreflective markers were placed bilaterally on the lateral aspect of the fifth metatarsal head, medial and lateral malleoli, medial and lateral femoral condyles, greater trochanters, iliac crests, acromion processes, spinous process of C7, medial and lateral humeral condyles, and the styloid process of the ulna and radius.	Participants walked across a 25-foot walkway. Individuals walked five times. No instructions or feedback regarding arm swing were provided.
Mian et al. [25]	three-dimensional motion analysis system	CODA motion capture system; 6 Cx1 units, Charnwood Dynamics, Rothley, UK	Rigid landmarks in head, acromion processes, lateral epicondyle of the humorous and ulnar styloid. Virtual landmarks in upper back, pelvis, ankle joints and foot	A single walking trial involved the participant walking back and forth along a 6.5-m walkway for 40 s. Participants were instructed to walk at a self-selected speed. Two to 4 normal walking trials were performed. Prior to recorded trials, practice walks were performed.
Mirek et al. [26]	three-dimensional motion analysis system	Vicon Nexus, Lake Forest, California	Head, 7th cervical vertebrae spinous proccess, sternoclavicular joint, 10th thoracic vertebrae, xiphoid process, acromioclavicular joint, middle arm, lateral epicondyle, middle forearm, left thumb, right pinkie, second metacarpal, anterior superior iliac spine, posterior superior iliac spine	Subjects performed gait at a natural, stable speed on a track length of about 15 m, allowing to capture at least 4 cycles of gait.
Ospina et al. [27]	3d video system	KinectTM Version 1	Reference markers at shoulder and wrist	Subjects walked through an interior corridor 4 m long and 1.5 m wide, free of interference and constant light. Subjects were instructed to start at the runner's start and walk at a normal pace towards the camera. Each participant performed three walking trials.
Rincon et [28]	accelerometers	ADXL335, three-axis sensors with a resolution of 3 g	Markers on both wrists.	Each subject was instructed to walk six times at normal pace along an aisle 10 m long and 1.5 m wide.
Roemmich et al. [29]	three-dimensional motion analysis system	Vicon Nexus, Lake Forest, California	Thirty-five passive retroflective markers were placed over bony landmarks according to the Vicon Plug-in-Gait marker set	Each subject performed ten overground gait trials along an 8-m walkway surrounded by a multi-camera optical motion capture system
Scandalis et al. [30]	three-dimensional motion analysis system	Peak Motus, Peak Performance Technologies, Englewood, CO	Markers were placed at the base of the second meta-tarsal of the foot, the most posterior aspect of the lateral heel, on the lateral malleolus, the lateral fibular head of the knee, greater trochanter, angle of the acromion process, lateral epicondyle, distal ulna, seventh cervical vertebrae, sternum, each mid-clavicle, and the forehead between the eyebrows.	Subjects walked along a 40-ft track

NR: No Reported

meta-analysis showed that arm-swing amplitude is approximately 0.08 m lower in PD compared with healthy groups (MD: - 0.08; 95% CI: - 0.09; - 0.07; $p < .001$; I^2: 31%) (Supplementary Material). Regarding effect size, we found that the reduction in arm-swing amplitude in individuals with PD in the on phase of the medication compared to healthy subjects had a large effect (SMD: −1.92; 95% CI: −3.22; −0.62; Z: 2.9; P:.004), with high significant heterogeneity ($P < .001$; I^2: 90%) (Fig. 4). Due to the high heterogeneity between studies, a basic outlier

Table 3
Results from included studies.

Study	Healthy	Parkinson Disease	Results
Swing Range of movement (°)			
Boungiorno et al. [17]	16.1 (7.8)	11 (6.3)	NR
Gera et al. [20]	39.8 (15)	Smaller: 15.7 (7.1) Larger: 29.7 (12.8)	NR NR
Isaias et al. [22]	Smaller: 25.27 (range 2.29–31.8) Larger: 25.71 (range 5.21–38,15)	Smaller: 7.48 (range 1.47–32.0) Larger: 18.19 (range 2.11–29.8)	Most affected: $p < .01$ Least affected: $p < .01$
Koh et al. [23]	Smaller: 28.85 (13.11) Larger: 33.79 (12.88)	Smaller: 20.43 (9.8) Larger: 28.65 (11.53)	Larger: $p = .12$ Smaller: $p = .01$
Mian et al. [25]	Larger: 19.1 (5) Smaller: 23.9 (6.1)	Larger: 11.1 (6.4) Smaller: 18.7 (9.2)	Larger: $p = .004$ Smaller: $p = .004$
Swing Amplitude (meters)			
Castaño et al. [18]	Smaller: 0.22 (0.08) Larger: 0.27 (0.1)	Smaller: 0.1 (0.06) Larger: 0.17 (0.11)	NR
Cole et al. [19]	Fallers: 0.29 (0.02) Non fallers; 0.34 (0.03)	Fallers: 0.22 (0.02) Non fallers; 0.25 (0.02)	Fallers: $p < .05$ Non-fallers: $p < .05$.
Koh et al. [23]	Smaller: 0.28 (0.09) Larger: 0.34 (0.09)	Smaller: 0.20 (0.08) Larger: 0.3 (0.1)	Larger: $p = .16$ Smaller: $p < .01$
Lewek et al. [24]	Larger: 0.74 (0.16) Smaller: 0.67 (0.16)	Larger: 0.78 (0.31) Smaller: 0.46 (0.22)	Larger: $p = .9$ Smaller: $p = .08$
Ospina et al. [27]	Smaller:0.26 (IQR, 0.17–0.33) m Larger: 0.26 (IQR, 0.20–0.34) m	Smaller: 0.16 (IQR, 0.08–0.2) Larger: 0.16 (IQR, 0.09–0.24)	Left: $p = .002$ Right: $p = .006$
Swing Velocity (meters/second)			
Boungiorno [17]	1.2 (0.3)	1 (0.2)	NR
Castaño et al. [18]	Smaller: 0.22 (0.09) Larger: 0.26 (0.07)	Smaller: 0.1 (0.06) Larger: 0.16 (0.09)	NR
Gera et al. [20]	1.68 (0.48)	Smaller: 1.10 (0.7) Larger: 1.40 (0.39)	NR NR
Ospina et al. [27]	Left: 0.25 (IQR, 0.18–0.29) Right: 0.26 (IQR, 0.18–0.31)	Left: 0.16 (IQR, 0.08–0.2) Right: 0.14 (IQR, 0.09–0.21)	Left: $p = .002$ Right: $p = .004$
Scandalis et al. [30]	1.23 (0.16)	0.9 (0.29)	$p < .05$
Swing symmetry (%)			
Gera et al. [20]	14.3 (14.4)	On: 26.7 (16.4) Off: 33.3 (18.6)	NR
Isaias et al. [22]	Smaller: 25.71 (5.21–38.15) Larger: 25.27 (2.29–31.8)	Smaller: 7.8 (1.47–32.06) Larger: 18.19 (2.11–29.8)	NR
Koh et al. [23]	5.4 (4.01)	13.16 (9.54)	$p < .01$
Lewek et al. [24]	5.1 (4)	13.9 (7.9)	NR
Mian et al. [25]	36 (25)	19 (15)	$p = .04$
Castaño et al. [28]	16 (11)	12 (7)	NR

Data reported as median (SD), except for indicated cases. NR: No reported.

removal was performed. The study by Cole et al. [19] was considered outlier because the lower bound of the 95% confidence interval is higher than the upper bound of the pooled effect confidence interval.[31] Meta-analysis without case outliers reduced heterogeneity (p:.47, I^2: 0%), showing that arm-swing amplitude is approximately 0.08 m lower in PD compared to healthy groups (SMD: - 0.08; 95% CI: - 0.12; - 0.04; $p < .001$; I^2: 31%) (Supplementary Material). In terms of effect size, we found that the reduction in arm-swing amplitude in individuals with PD in the on phase of medication compared to healthy subjects had a moderate effect size (SMD: −0.78; 95% CI: −1.24; −0.33; Z: 3.37; $P < .001$), showing no heterogeneity (P:.47; I^2: 0%) (Fig. 5).

3.4.3. Arm swing velocity (m/s)

Data regarding arm-swing velocity were available from 4 studies [17,18,20,30], which compared the arm-swing velocity of PD subjects with that of the healthy control group. The meta-analysis showed that the arm-swing velocity is approximately 0.31 m/s slower in PD compared with the healthy groups (MD: - 0.31; 95% CI: - 0.44; - 0.19; $p < .001$; I^2: 48%) (Supplementary Material). Regarding effect size, we found that the reduction in arm-swing velocity in individuals with PD compared with healthy subjects had a large effect (SMD: −0.95, 95% CI: −1.36; −0.53, Z: 4.45, $P < .001$), with not relevant heterogeneity (p:.85; I^2: 0%) (Fig. 6).

A subgroup analysis was performed according to medication status (off versus on) (Fig. 6). The subgroup analysis showed no significant differences between groups (p:.84), showing a significant reduction in the arm-swing velocity in both groups, with a large effect size: off group (SMD: −0.9, 95% CI: −1,50; −0.31, Z: 2.97, P:.003); ON group (SMD: −0.99, 95% CI: −1,57; −0.41, Z: 3.33, $P < .001$). The mean arm-swing velocity was reduced to a greater extent in the off group (MD: - 0.34; 95% CI: - 0.52; - 0.17; $p < .001$; I^2: 0%) than in the ON group (MD: - 0.28; 95% CI: - 0.46; - 0.10; p:.002; I^2: 77%), without being significant. No heterogeneity was observed between subgroups (p:.84, I^2: 0%).

3.4.4. Arm swing asymmetry (%)

Data regarding arm-swing symmetry were available from 5 studies [20,23–25,28]. The meta-analysis showed that the arm-swing symmetry is approximately 8.16% higher in the healthy control group compared with the PD group (MD: 8.16; 95% CI: 5.63; 10.7; $p < .001$; I^2: 0%) (Supplementary Material). Regarding effect size, we found that the higher arm-swing symmetry in healthy controls compared with PD patients had a large effect (SMD: 0.86, 95% CI: 0.53; 1.19, Z: 5.15, $P < .001$), with no heterogeneity (p:.54 I^2: 0%) (Fig. 7). A subgroup analysis was performed according to medication status (off versus on) (Fig. 7). The subgroup analysis showed no significant differences between groups (p:.54), showing a significant lower arm-swing symmetry in both groups, being greater in the off group (MD: 10.31, 95% IC: 5.5; 15.12 vs MD: 7.34, % CI: 4.35; 10.7). The effect size was large in both groups: off group (SMD: 1, 95% CI: 0.45; 1.55, Z: 3.56, $P < .001$), ON group (SMD: 0.79, 95% CI: 0.38; 1.19, Z: 5.15, $P < .001$). There was no heterogeneity between subgroups (p: .54, I^2: 0%). The funnel plot did not present asymmetry (Figure 8).

4. Discussion

The aim of this systematic review and meta-analysis was to determine the arm-swing kinematic pattern in people with PD, establishing differences between medication states. Despite different types of arm-swing assessments, most studies included subjects with homogeneous characteristics in terms of demographic variables and disease progression (H&Y II-III). It seems that people with PD present a significant decrease in, range, amplitude, velocity, and asymmetry of arm-swing, respect to healthy controls. Even though patients in the off phase present a greater reduction in arm-swing, range, velocity and symmetry, no significant differences were found with respect to patients in the on phase.

The arm-swing during gait minimizes the angular momentum of the body around the vertical axis, reducing energy expenditure by equalizing the torques acting on the Center of Mass (COM) [31]. Studies using electromyography have shown that arm-swing is at least partially actively controlled [32,33], and the inability of coordinated muscle activation, along with stiffness and bradykinesia, may contribute to decreased sway in PD. As there is no counterbalancing arm torque to mitigate the leg torques, the trunk will rotate faster (increased COM mean angular velocity around the vertical axis) [6]. This, in turn,

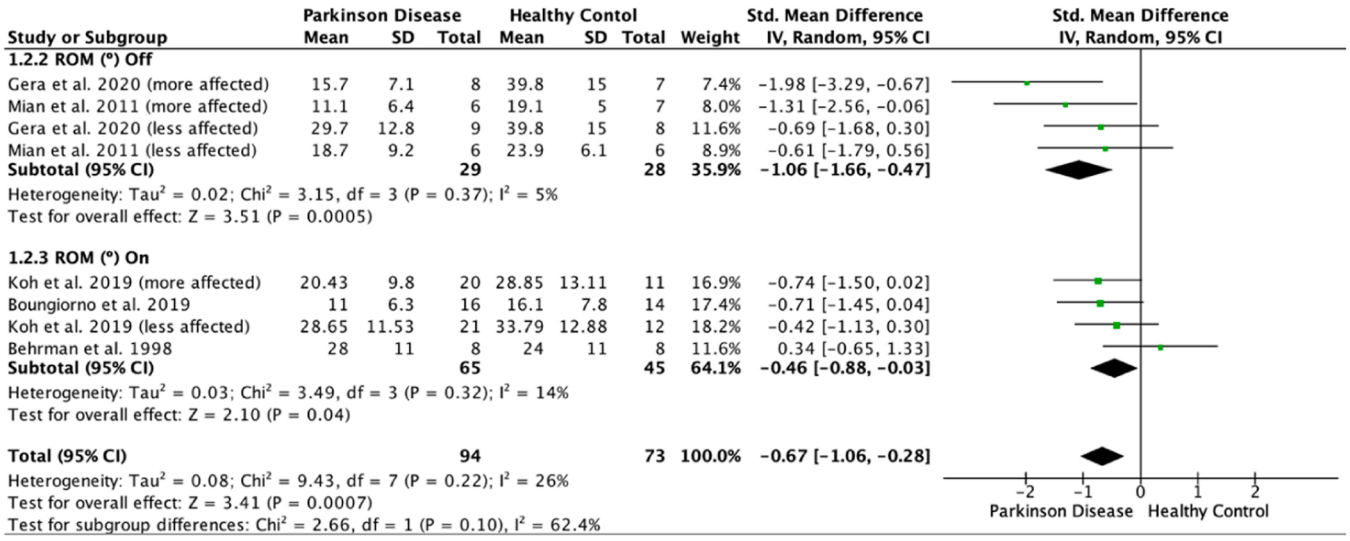

Fig. 2. Forest plot of the results of a random-effects meta-analysis shown as standardized mean differences (SMD) with 95% confidence interval (CI) for arm swing range of motion. Off and on subgroups are reflected, according to medication phase. The shaded square represents the point estimate for each individual study and the weight of the study in the meta-analysis. The diamond represents the overall mean difference of the studies.

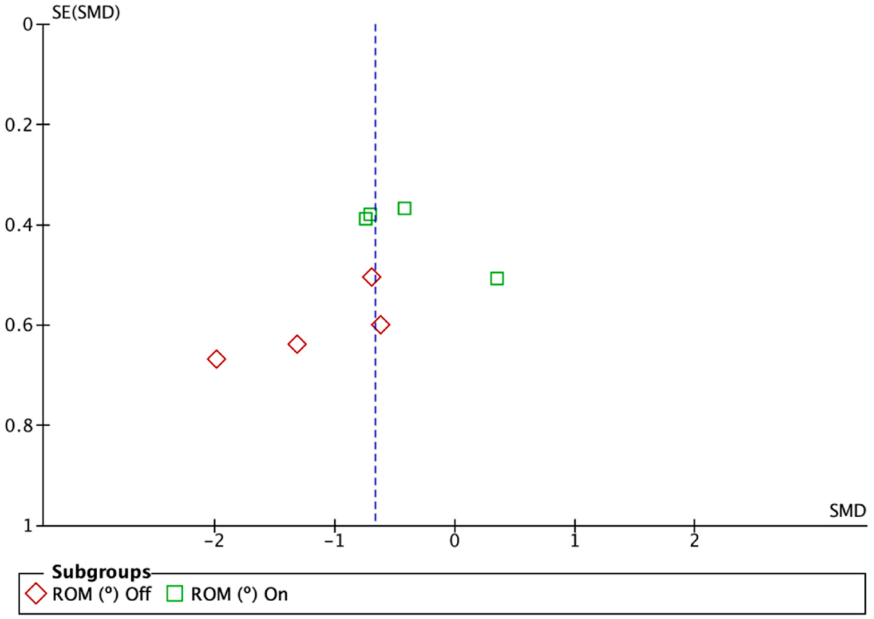

Fig. 3. Arm swing range of motion (°) Funnel Plot. Dispersion of effect sizes. X-axis: observed effect sizes. Y-axis: inversed standard error (higher values on the Y-axis represent lower standard errors). Slight asymmetry, meaning possible publication bias.

increases the mean angular velocity of the COM, acting as an internal perturbation, which will increase the spatiotemporal variability of gait in PD, as a consequence of the inability to effectively apply adaptive responses to these perturbations [34]. Since foot placement at heel moment is based on the predicted COM trajectory, in PD, an alteration of kinematic parameters related to the variation of this trajectory is observed, adapting trunk stiffness strategies, and altering foot placement during gait, relating to a decrease in stride length, and an increase in the coefficient of variation of stride length and stride time [35]. It is suggested that both are compensatory strategies to reduce the loss of balance during heel strike. These facts are directly related to an increased risk of falls, especially when instabilities occur in the medio-lateral axis [36].

4.1. Arm amplitude and ROM (m; °)

The meta-analysis showed a significant reduction in arm-swing RoM and amplitude, as described in patients with PD [1]. Shoulder RoM in the sagittal plane during normal walking ranges between 30°-45° [37] Shoulder and elbow RoM are significant predictors for amplitude of arm-swing in the "most affected" side [7]; Siragy et al. [6] showed that restricted arm-swing reduced average step length and time, while increasing variability for step time.

4.1.1. Arm swing velocity (m/s)

In general, a decrease in the speed of movements is described in PD (bradykinesia), which is also related to arm-swing [38]. Although there are not many studies that specifically analyze the arm-swing velocity, several authors claim to find a significantly lower arm-swing velocity in

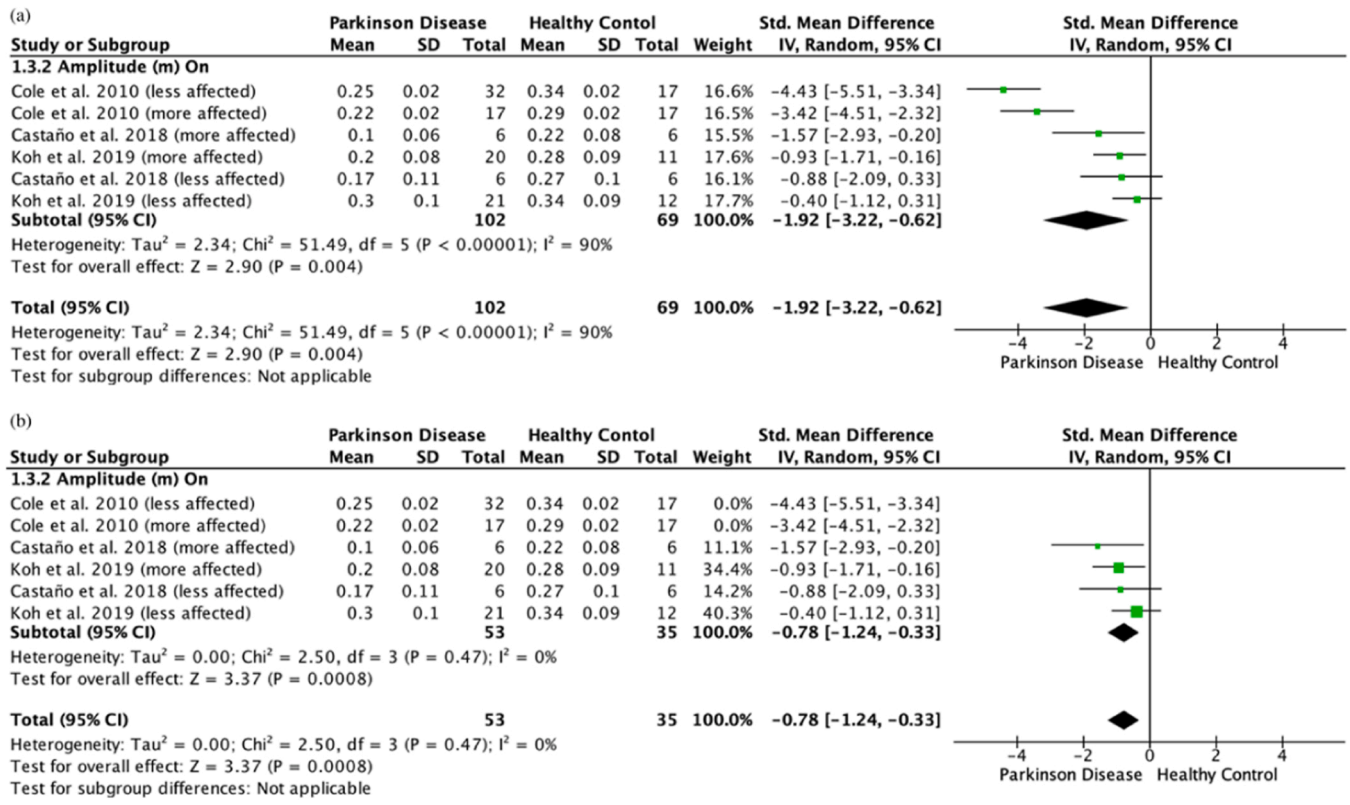

Fig. 4. Forest plot of the results of a random effects meta-analysis shown as standardized mean differences (SMD) with 95% confidence interval (CI) for (a): Arm swing amplitude (m); (b) Arm swing amplitude (m) without outlier study. The shaded square represents the point estimate for each individual study and the weight of the study in the meta-analysis. The diamond represents the overall mean difference of the studies.

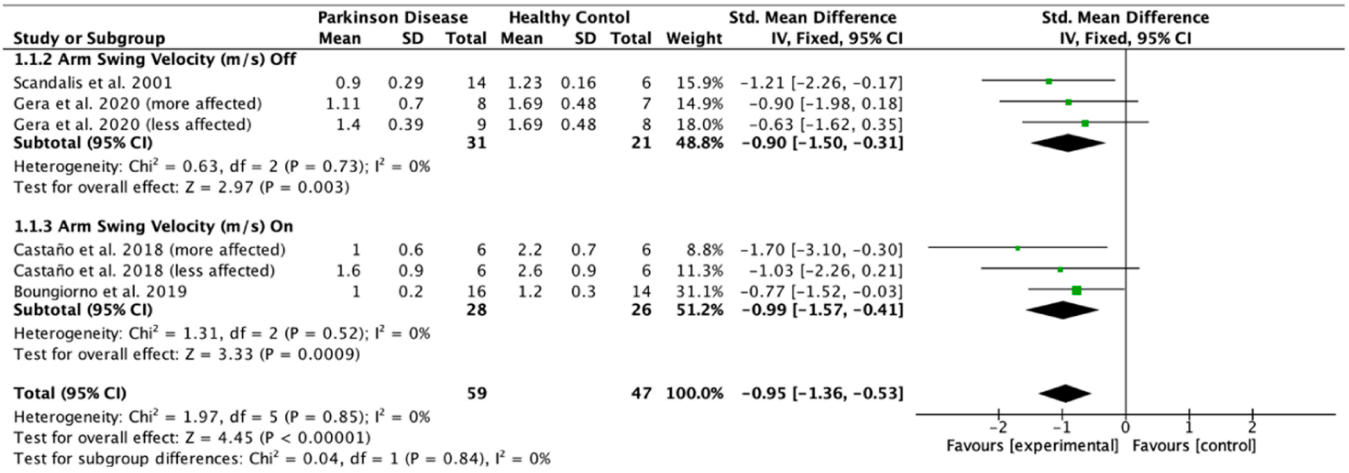

Fig. 5. Forest plot of the results of a random-effects meta-analysis shown as standardized mean differences (SMD) with 95% confidence interval (CI) for arm swing velocity (m/s). Off and on subgroups are reflected, according to medication phase. The shaded square represents the point estimate for each individual study and the weight of the study in the meta-analysis. The diamond represents the overall mean difference of the studies.

people with PD than in healthy controls of the same age [17,20,30]. The arm-swing velocity is lower in the more affected arm [18], which could be a consequence of the asymmetric onset of the disease. The consequences of lower arm-swing velocity, to our knowledge, have not been specifically studied in PD.

4.1.2. Arm swing asymmetry (%)

Significant arm-swing asymmetry was observed in PD subjects compared to healthy controls. Meyns et al. [1], observed that there is a certain degree of arm-swing asymmetry during gait considered physiological, which may increase when dual-tasks are performed during gait [39]. In PD, the arm-swing reduction is accentuated on the more affected side, being the asymmetry in early stages more noticeable than the arm-swing reduction amplitude [21]. This asymmetry has been described to increase as the disease progresses and is related to nigrostriatal denervation, showing a positive correlation between asymmetry and H&Y score in a medication-free state [40]. Mirelman et al. [41] found no association between gait characteristics and arm-swing asymmetry, however, Ospina et al. [27] argue that age influences arm-swing, suggesting that PD patients cannot compensate by

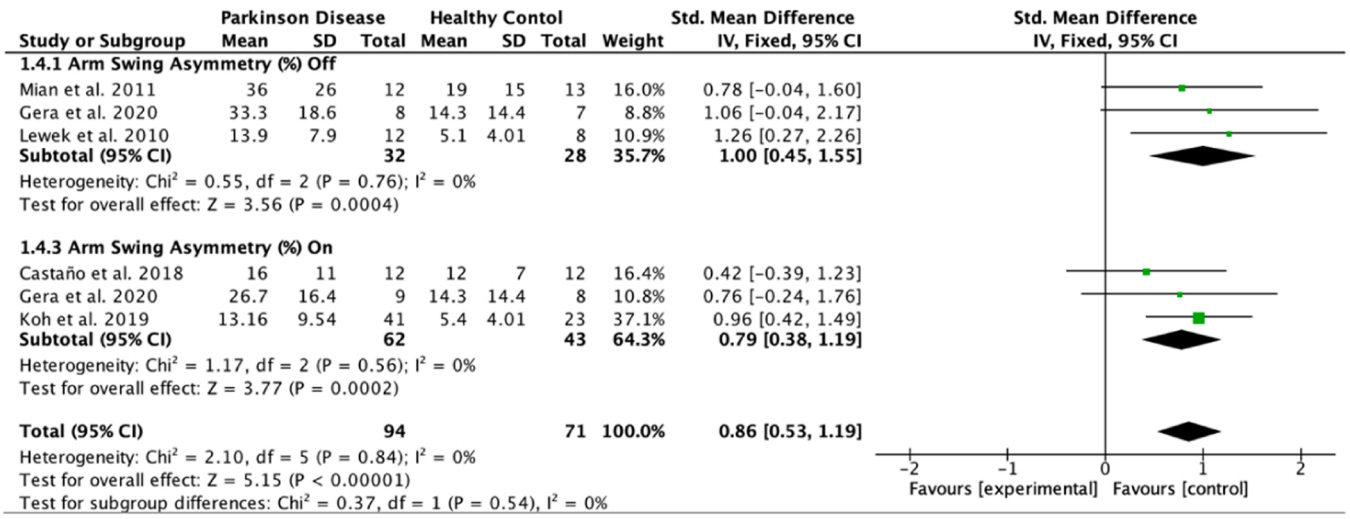

Fig. 6. Forest plot of the results of a random-effects meta-analysis shown as standardized mean differences (SMD) with 95% confidence interval (CI) for arm swing symmetry (%). Off and on subgroups are reflected, according to medication phase. The shaded square represents the point estimate for each individual study and the weight of the study in the meta-analysis. The diamond represents the overall mean difference of the studies.

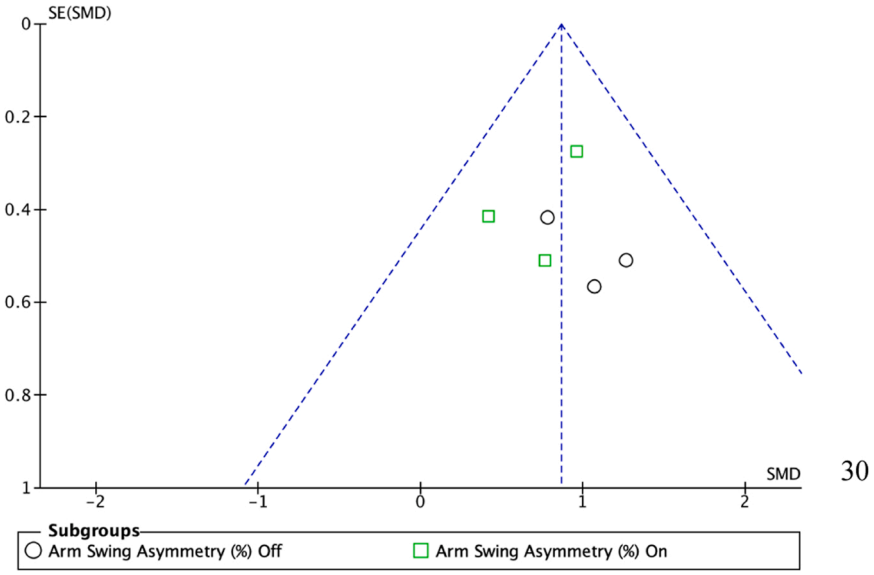

Fig. 7. Arm swing symmetry (%) funnel plot. Dispersion of effect sizes. X-axis: observed effect sizes. Y-axis: inversed standard error (higher values on the Y-axis represent lower standard errors). Slight asymmetry, meaning possible publication bias.

increasing arm movement on the less affected side and, therefore, their movement tends to become more symmetrical as the disease progresses. This may explain why there are differences between patients in the early or in the advanced PD stage.

There is few evidence on treatments aimed at improving arm-swing. According to several authors [1,42,43], when the arm-swing amplitude is increased in people with PD, pelvic movements during gait are facilitated, increasing cadence, walking speed, stride length, and swing phase, and decreasing stride time, double-support time, and stance phase. So, it would be an important goal of treatment. Pharmacological and surgical treatment (deep brain stimulation of the subthalamic nucleus and L-dopa) increases gait speed and arm-swing amplitude, being, in some cases, more effective in the case of combining both treatments [44]. However, according to the results of the present meta-analysis, no significant differences were observed with respect to the arm-swing amplitude, range and velocity in the on and off medication phases. Pharmacological treatment appears to be dependent on where gait takes place, being less effective in terrains that re-create daily situations through dual tasks [45], for which other treatment approaches have been sought. However, although people with PD are able to modify their gait by consciously adapting a certain pattern, it should be avoided in therapy, as it has been shown that conscious walking by increasing arm movement has a negative impact on gait mechanics. This may be due to a dual-task effect, as moving the arms would attract the attention of people with PD as a secondary task. As this population has reduced dual-task ability, they may have difficulty maintaining the gait pattern during conscious arm movement [35]. Yoon et al. [42] increased arm-swing amplitude by having people with PD walk with a 0.45 kg weight in their hands, without any further instruction, thereby increasing gait parameters related to arm-swing amplitude. This therapy could be a valid treatment option that does not require dual tasking, although these authors performed a single session, so its long-term effectiveness would need to be determined. Mainka et al. [43] designed a mobile app that, through sensors, provided arm-swing-dependent closed-loop musical feedback, significantly increasing arm-swing range and velocity, suggesting that the generation

of an external rhythm may help to generate the motor sequences in PD as occurs with gait speed and cadence [46]. The increase in arm-swing amplitude appears to occur when the external sensory cues are auditory, but not visual [47].

Some findings suggest that the absence of arm movement in people with PD varies according to the specific terrain, and therefore, rehabilitation therapies should provide various types of environments similar to real-world terrains [6].

4.1.3. Study limitations

Our current study has several limitations. One of them is that the observed arm-swing parameters have different measurement methods, some were taken with different devices, and have different measurement protocols. The different ways of assessing arm-swing have led to minimize the studies included in the meta-analysis, in order to extrapolate the results as much as possible. PD has a wide spectrum of motor impairments and conditions (on and off medication), which may cause results to vary if it is not a homogeneous population. Most studies included similar stage in H&Y (II-III), but these patients may have high intergroup variability. Therefore, further studies assessing homogeneously with both objective analysis systems and PD subjects with similar characteristics, are needed to confirm our analysis. We recommend that future studies investigate the implications of arm swing in relation to the spatiotemporal parameters of gait, and the performance of studies that perform treatments focused on improving arm-swing during gait in PD, observing whether any treatment could be effective in this regard, as well as its implications on gait, balance, and risk of falls.

5. Conclusion

The present meta-analysis showed that people with PD have significant differences in arm-swing range, amplitude, velocity, and asymmetry during gait compared with the healthy control group. Medication phase does not significantly influence in the arm-swing characteristics. More studies will be needed to determine whether different disease characteristics influence the biomechanics of arm-swing during gait.

CRediT authorship contribution statement

VNL: Design and execution of statistical analysis; writing of the manuscript. FMR and MCT: Review of statistical analysis; review of the manuscript. MDV, PGP, and DFV: Data collection; methods section of manuscript. ACG: conception of tables, data collection and review of the manuscript.

Supplier list

The manuscript submitted does not contain information about medical device(s).

No other products or devices are used for this manuscript.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.gaitpost.2022.08.017.

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