

Original Articles

Treatment of axial postural abnormalities in parkinsonism disorders: A systematic review of pharmacological, rehabilitative and surgical interventions

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ABSTRACT

Axial postural abnormalities (PA) are frequent, highly disabling, and drug-refractory motor complications affecting patients with Parkinson's disease (PD) or atypical parkinsonism. Over the past few years, advances have been reached across diagnosis, assessment, and pathophysiological mechanisms of PA. Nonetheless, their management remains a challenge, and these disturbances are generally overlooked by healthcare professionals, potentially resulting in their worsening and impact on patients' disabilities. From shared consensus-based assessment and diagnostic criteria, PA calls for interdisciplinary management based on the complexity and multifactorial pathogenesis. In this context, we conducted a systematic literature review to analyze the available pharmacological and non-pharmacological treatment options for PA in PD according to the new expert-based classification of axial PA in Parkinsonism. Different multidisciplinary approaches, including dopaminergic therapy adjustment, physiotherapy, botulinum toxin injection, and deep brain stimulation, can improve PA depending on its type and severity. An early, interdisciplinary approach is recommended in PD patients to manage PA.

1. Introduction

Axial postural abnormalities (PA) are common and highly disabling complications observed in Parkinson's Disease (PD) and atypical parkinsonism [1–3]. PA are a part of axial motor symptoms associated with PD and other forms of parkinsonism. They typically appear during the middle or advanced stages of PD and poorly respond to standard dopaminergic therapy [4]. Historically, PA have been classified into four types based on the direction of plane deformities [2,5]: camptocormia (CC) and antecollis (AC) involve the sagittal plane, while Pisa syndrome (PS) and scoliosis affect the coronal plane. The lack of understanding regarding pathogenic mechanisms and the absence of a standardized classification, including clear definitions, cut-off angles, and

measurement methods, have hindered our ability to provide evidence-based treatments to alleviate or prevent the onset of axial PA. Recently, movement disorders experts have reached a consensus on the diagnostic criteria and cut-offs for PA in Parkinsonism [5]. These criteria define CC with "lumbar fulcrum" as having a fulcrum from the spinous processes of L1-sacrum-hip, with a forward trunk flexion of $>30^\circ$, and CC with thoracic fulcrum as having a fulcrum from C7 to T12-L1 and a forward trunk flexion of $>45^\circ$. AC has a forward neck flexion of $\geq 45^\circ$, and PS has a lateral trunk flexion of $\geq 10^\circ$ [5]. The terms "anterior trunk flexion" (with thoracic angles of $\geq 25^\circ$ to $\leq 45^\circ$ or lumbar angles of $>15^\circ$ to $\leq 30^\circ$), "lateral trunk flexion" ($\geq 5^\circ$ to $\leq 10^\circ$), and "anterior neck flexion" ($>35^\circ$ to $\leq 45^\circ$) are recommended to describe milder postural abnormalities [5].

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Despite their prevalence and disabling nature, our understanding of the optimal treatment approaches for these PD features is limited. This represents a significant knowledge gap, considering that CC, PS, AC, or a combination of these conditions affect over 20 % of PD patients during the disease course [1,6,7]. Moreover, it is worth considering that these symptoms are associated with a higher frequency and severity of pain, an increased risk of falls, and ultimately, a lower quality of life [3,8–10].

In this context, we conducted a systematic literature review to analyze the available pharmacological and non-pharmacological treatment options for PA in PD according to the new expert-based classification of axial PA in Parkinsonism. Our review includes oral pharmacological treatments, injection therapies, deep brain stimulation (DBS), spinal surgery, rehabilitative interventions, and orthoses.

2. Methods

The protocol of this systematic review was registered in the PROSPERO database (CRD42023414769). We followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines during the whole process of the review [11]. The Study Quality Assessment and the Levels of Evidence were rated by the Study Quality Assessment Tools and the 5-item Oxford CEBM scale, respectively [12] (<https://www.nhlbi.nih.gov/health-topics/study-quality-assessment-tools>, accessed August. 05, 2023).

2.1. Selection criteria for studies

We included all study designs enrolling participants with PD and PA. We included studies in which PD patients received oral pharmacological treatments, injection therapies, deep brain stimulation, spinal surgery, rehabilitative interventions, and orthoses targeted to improve PA as a standalone treatment or in combination. We defined PA according to the consensus-based nosology and cut-off criteria [5]. We excluded studies on non-human trials, reviews, abstracts, conference proceedings, and protocol papers without data collection.

2.2. Outcomes

The primary outcome was the severity of the PA assessed with clinical or instrumental assessment tools. According to the new Movement Disorders Society (MDS) Task Force Consensus criteria [5] we classified and reported PA when possible; secondary outcomes were measures of other motor and non-motor symptoms (i.e., MDS-Unified Parkinson's Disease Rating Scale, Berg Balance Scale, pain).

2.3. Search strategy

One author searched the MEDLINE (PubMed), Web of Science All databases, and SCOPUS electronic databases from inception until March 2023. The search terms “Parkinson's disease,” “Multiple System Atrophy,” “Progressive Supranuclear Palsy,” “Dementia,” “Lewy body,” “Corticobasal syndrome,” “Axial manifestation,” “Postural syndrome” paired with “Therapeutics,” “Therapy,” “rehabilitation” was used in combination. The whole search strategy for all databases is included in the [supplementary material](#).

2.4. Data collection and analysis

Three reviewer authors independently screened for inclusion based on the title and abstract of all potentially relevant studies using Rayyan online software. Full-text studies were retrieved and independently screened for inclusion. Disagreement was resolved by discussion. The other three independent review authors extracted data from eligible full-text documents. They recorded information on an electronic data sheet extraction form, including the type of the study, sample size, participant population, disease and postural abnormalities duration, Hoehn Yahr

scale, type, details of interventions, type of outcomes, and main findings. Interventions were classified into five categories: pharmacological (oral and injection) treatment, Deep Brain Stimulation (DBS), spinal surgery, rehabilitation, and orthoses.

3. Results

After screening the search results, 99 records were identified for inclusion in the review. The flow of studies through the review is reported in [Fig. 1](#). An overview of the selected studies for each type of intervention is reported in [Tables 1-5](#). In [Fig. 2](#), we displayed an overview of the selected studies, divided by intervention category. In the [supplementary material](#), we described interventions focused on orthoses.

3.1. Pharmacological oral treatment

We found 20 pertinent studies on the effect of oral pharmacological treatment on PA in PD, including 205 patients: 50 % (n = 10) were case reports [13–22], 25 % (n = 5) were prospective interventional before after [23–27], 20 % (n = 4) were case series [28–31], and 1 case-control study [32]. The studies included 80 CC, 59 PS, and 19 AC; no studies divided CC according to a higher (i.e., thoracic fulcrum) or lower (i.e., lumbar fulcrum) spine flexion. In 2 studies, we had an unspecific referral to abnormal posture or a score >0 on item 28 of the UPDRS or sagittal imbalance [23,27]. The median duration of PD and of the PA at time of evaluation were 6.7 years (range 3–11.6 years) and 1 year (range 0.1–6 years), respectively. Outcome measures differed across studies: 12 employed a clinical evaluation (visual estimation) [14,16–22,26,29–31], 3 used the UPDRS/MDS-UPDRS or UDRS [23,26,32], 3 used the angle of spine flexion [24,25,28], 2 using the angle of the dropped head [15,24], and three other types of postural angle measurements [13,25,27]. Only one case-control study evaluated the differences following the exposure to dopamine agonists between patients with and without PA [32]. Six studies reported the cognitive status at baseline.

In 50 % of studies (n = 10), an improvement in PA from a specific drug was reported [15,20,22–29]: levodopa (n = 5, administered in 2 cases oral, in one case intravenously, in 2 cases with intestinal gel infusion), istradefylline (n = 2), selegiline (n = 1), apomorphine (n = 1), Co-ultra micronized palmitoylethanolamide/luteolin (n = 1). In 50 % of studies (n = 11), a worsening effect on PA from a specific drug was reported [13,14,16–19,21,29–32], specifically after the intake of dopamine agonists (n = 7), levodopa/carbidopa/entacapone (n = 1), istradefylline (n = 1), rasagiline (n = 1). One case series reported the onset of PS after the introduction or increase of dosage of a dopaminergic drug (7 cases) and after the withdrawal of pergolide (1 case) [30]. Studies reporting degrees of spine flexion angle showed a median improvement after drug start/removal of 52.1 % for CC (range 30.9–77.8 %), 31.5 % for PS (range 31–37.8 %), and 65.1 % for AC (range 64.4–65.8 %). 7 studies reported pain associated with PA at baseline, and two studies reported an improvement after intervention. For details, see [Table 1](#).

3.2. Pharmacological injection treatment

We found 18 pertinent studies, including a total number of 117 PD patients. CC (or mild forms of anterior trunk flexion) was present in 50 patients, PS (or mild forms of lateral trunk flexion) in 24, and antecollis (or mild forms of anterior neck flexion) in 16. CC and PS were reported in 27 patients with PD. There was a wide range of study designs: 13 were case series/reports, 1 was a goal attainment-controlled study, 1 had a before-after design, one was a controlled parallel group trial, one was a blinded crossover trial, and one was a prospective pilot study.

BoNT serotypes A, including Abobotulinumtoxin [33], Onabotulinumtoxin [34–39], and Incobotulinumtoxin [40,41] was studied in

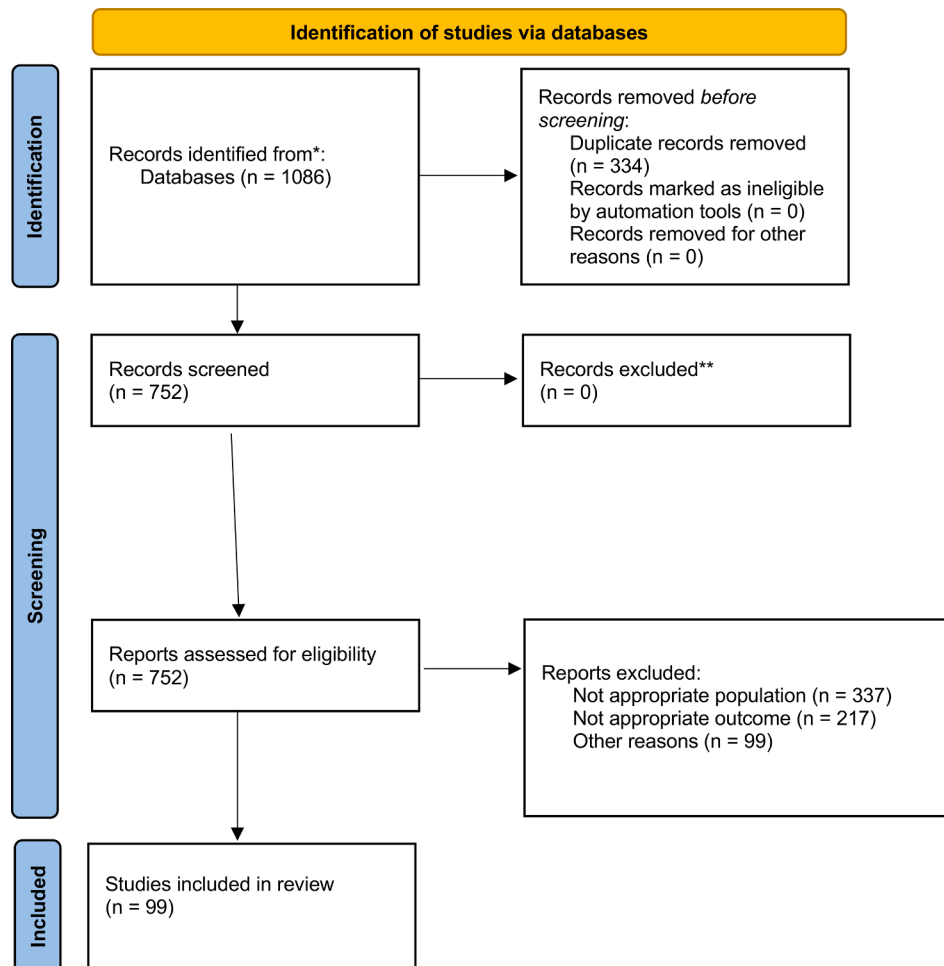


Fig. 1. The flow of studies through the review process.

patients with PD with CC. The injection dose of BoNT is variable among studies and based on the type of BoNT and site of injection. Lidocaine was injected in two studies [42,43] with an injection dose of 50 U per muscle. Four studies used ultrasound-guided BoNT injection, 1 used CT-guided BoNT injection, 3 EMG-guided techniques, and two used a blind/unspecified injection technique.

The muscles injected were bilateral rectus abdominis (n = 6 studies) [34,36,39–41,43], iliopsoas muscles (n = 4 studies) [33,36,40,41], external oblique muscle (n = 4) [38,39,42,43], and internal oblique muscle (n = 1) [43]. Other paraspinal or multifidus muscles [35,37,41] were also injected when camptocormia was associated with PS. Several outcomes, including objective and subjective outcome measurements, were used to evaluate the efficacy of BoNT/Lidocaine injection. The results were mixed among studies: 7 studies showed a moderate to marked improvement of posture after injection [34,37,38,41–43,50]. Four studies reported a significant improvement in pain, abdominal in particular [38–41]. Four studies reported no changes in posture after injection [33,35,36,40]. No other significant changes were reported in these studies in terms of improvement in posture and related reduction of disability and improvement of quality of life (Table 2).

BoNT serotypes A, including Abobotulinumtoxin [44,45], onabotulinumtoxin [37,46] and Incobotulinumtoxin [41,47] were studied in patients with PD with PS. The injection dose of BoNT varies among studies and is based on the type of BoNT and site of injection. One study used ultrasound-guided BoNT injection [46] and 3 studies used EMG-guided injection [41,44,45].

The muscles injected were: thoracic and lumbar paraspinal muscles ipsilateral (n = 6) [37,41,44–47] or contralateral (n = 3) [41,45,46] to

the trunk bending side, bilateral rectus abdominis (n = 1 study) [41], iliopsoas muscles (n = 2 studies) [41,46], external/internal oblique muscle (n = 1) [46], multifidus (n = 1) [41], and quadratus lumborum (n = 1) [47]. Several outcome measurements, including objective and subjective outcome measurements, were used to evaluate the efficacy of BoNT injection. All studies showed a moderate to marked improvement in PS and pain after injection [37,41,44–47]. No other significant changes were reported in these studies regarding the reduction of disability and improvement of quality of life (Table 2).

BoNT serotypes A injection, including Abobotulinumtoxin [48,49] and Onabotulinumtoxin [50,51] was studied in patients with PD with AC. The injection dose of BoNT is variable among studies and based on the type of BoNT and site of injection. Three studies used EMG-guided BoNT injection [48,50,51], while one did not report the method of injection [49]. The muscles injected were unilateral/bilateral Levator scapulae (n = 4) [48–51], Sternocleidomastoid muscle (n = 2) [48,50], splenius capitis (n = 1) [48], Longus collis (n = 1) [51], Anterior/Medial Scalene (n = 2) [49,51]. Several outcome measurements, including objective and subjective outcome measurements, were used to evaluate the efficacy of BoNT injection. Three studies showed a slight improvement in AC and pain. No other significant changes were reported in these studies in terms of improvement in the reduction of disability and improvement of quality of life (Table 2).

3.3. Deep brain stimulation

We found 32 studies on the effect of DBS on PA in PD, including 723 patients. 37.5 % (n = 12) were retrospective observational before-after

Table 1
Studies of pharmacological treatment for axial postural abnormalities in PD.

Study ID/Study design	No. of participants (no. of females)	PD duration – H&Y stage if reported; Cognitive status	Research question Medication status	Type of PA – PA duration	Study outcomes measures	Main results	Follow-up	QA-LE
Takahashi et al. 2022 [23] Prospective interventional Before After	31 (18F)	6.2 ± 4.2–2.4 ± 0.6 Cognitive exclusion criteria: MMSE ≤20	Efficacy and safety of istradefylline for the treatment of PA in PD -Oral Istradefylline at a starting dose of 20 mg/day, increased to 40 mg/day at week 4 if the patient continued to have motor symptoms.	No specific PA reported – NR	Primary: UDRS mean score. Secondary: sub-items of UDRS, MDS-UPDRS III, and adverse drug reactions. Pain status: NR cognitive status: MMSE	Mean change in the UDRS total score was 4.84 (1.97, 7.71; $P = 0.002$), with significant improvements in the neck, right distal arm and hand, and trunk severity scores. Mean change in the MDS-UPDRS part III score was 7.84 (4.34, 11.34; $P < 0.001$). MSE ≥27 was associated with an improvement in the UDRS	24 weeks	Poor –4
Kataoka and Ueno 2017 [24] Prospective Interventional Before After	24 (16F)	ATF: 9.2 ± 7.2. AC : 9.1 ± 7.3.LTF: 5.9 ± 1.8.-ATF: 3.3 ± 1.1.AC: 2.5 ± 0.5 LTF: 2.7 ± 1.2 Cognitive exclusion criteria: NR	Response of abnormal posture to dopamine challenge testing-LD in saline solution (100 ml) intravenously infused over the course of 30 min.	13 patients with ATF (CC) ≥45° –2.6 ± 2.2 4 patients with AC ≥20° –1.5 ± 1.8.7 patients with LTF (PS)-1.8 ± 0.8	Posture angles measured with the use of “Image J” software Pain and cognitive status: NR	The angle of the overall abnormal posture significantly decreased ($p < 0.001$). The angle of the abnormal posture significantly decreased for AF and AC in both natural position (AF $p < 0.001$, AC $p = 0.002$) and in a position with the back averted (AF $p = 0.003$, AC $p = 0.029$), but did not change significantly in patients with LTF ($p = 0.099$) Natural position posture pre-post:AF from 62.6 ± 11.3° to 51.8 ± 13.3° AC from 38.1 ± 12.9° to 13.0 ± 6.3° PS from 19.3 ± 17.2° to 13.7 ± 10.1°	Only evaluation 5–10 min after LD infusion	Fair –4
Yoritaka et al. 2016 [25] Prospective Interventional Before After	20 (8F)	6.3 ± 4.8–2.5 ± 0.4 Cognitive exclusion criteria: NR	Response of CC to selegiline -Participants were administered 5 mg/day of selegiline in the first 8 weeks, and subsequently, 7.5 mg/day for 8 weeks, and for the next 8 weeks, selegiline was discontinued	CC (clinical) – NR	Changes in the degree of AF and the envelope of postural sway (ENV AREA) from baseline to 16 weeks post-treatment studied using Gravicorder (GS-3000 ANIMA). Changes in the ROM AREA (the ratio of the ENV Area with the eye closed to the ENV Area with the eye opened). Changes in total UPDRS part II, part III, and part III-28 (posture scale), lateral flexion, and Visual Analog Scale (VAS) scores. Cognitive status: NR	Twelve of 20 participants showed an improved degree of anteflexion with selegiline, but the changes in the degree of anteflexion and ENV AREA from baseline to the 16th week were not significant. The total UPDRS, UPDRS part II, part III, III-28, and VAS scores improved significantly. VAS reduced from 49.8 ± 25.8: 39.6 ± 36.4	16 weeks of treatment, and 8 weeks after conclusion	Poor
Mensikova et al. 2015 [26] Prospective Interventional Before After	5 (2F)	6.2 ± 3.3 –NR Cognitive exclusion criteria: NR	To assess the effects of apomorphine on CC -Subcutaneous infusion of apomorphine	CC (clinical) –12 years	UPDRS-III; Unified Parkinson’s Dyskinesia Scale (UPDyskS); Clinical Global Impression (CGI) scale of CC Pain and cognitive status: NR	CC had improved in all patients by the fourth week of continuous apomorphine treatment. CGI At twelve months 1.6 ± 0.5; UDPRS-III from 50.8 ± 6.4 to 31.6 ± 4.3; UPDyskS from 50.6 ± 12.7 to 30.8 ± 2.3	1 year	Poor –4

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Table 1 (continued)

Study ID/Study design	No. of participants (no. of females)	PD duration – H&Y stage if reported; Cognitive status	Research question Medication status	Type of PA – PA duration	Study outcomes measures	Main results	Follow-up	QA-LE
Benninger et al. 2015 [27] Prospective Interventional Before After	48 (16F)	22 patients: 4.4 ± 2.4 26 patients: 11.6 ± 5.1 -NR Cognitive exclusion criteria: NR	To quantitatively evaluate the effect of LD on spinal posture. –22 dopa-naive, evaluated before and 3 months after initiation of treatment. 26 patients with motor fluctuations studied during the “off” and “on” states.	Stooped posture, with degree derived from UPDRS item 28 -NA	Spinal mobility, posture, and range of motion evaluated with the device “SpinalMouse.” Trunk angle of inclination (angle between the vertical line from C7 and a line joining C7 to the sacrum). The area under the curve (AUC): a virtual vertical line from C7 and a horizontal line from T12. Pain status: Roland-Morris Disability Questionnaire Cognitive status: NR	In the dopa-naive patients, spinal incline in the upright position was 12.4° ± 1.28° before and 7.6° ± 1.38° after treatment, p = 0.002. Corresponding AUC values were 131.7 ± 8.0 cm ² and 87.1 ± 7.3 cm ² , p < 0.0001. In the response fluctuations patients, spinal incline was 13.3° ± 1.38° in the “off” and 9.3° ± 1.28° in the “on” period, p = 0.015. Corresponding AUC values were 144.6 ± 9.2 cm ² and 103.1 ± 8.2 cm ² , p < 0.0001. Lumbar back pain was reported more often in the dopa-naive group (40.1 %; 9/22 patients) than in the fluctuations group (19.2 %; 5/26 patients; p = 0.027).	3 months in the dopa-naive patients	Fair –4
Morales-Briceno et al. 2019 [28] Case series	(2 M)	Patient 1: 8 Patient 2: 10–3 Cognitive exclusion criteria: NR	Response of CC to Levodopa/carbidopa intestinal gel infusion (LCIG) -LCIG infusion.	2 CC ≥45° 1 PS ≥10° -NR	Thoracic flexion with malleolus method Pain status: NR Cognitive status: Montreal cognitive assessment (MoCA)	MoCA at baseline: patient 1 28; patient 2 27 Degree of truncal flexion at baseline: patient 1 75°; patient 2 45°; Degree of truncal flexion at follow-up: patient 1 30°; patient 2 10° Patient 2 presented also PS = 20°, not changed at follow-up	Patient 1: 12 months Patient 2: 10 months	Fair –4
Fujioka et al 2019 [29] Case series	4 (4F)	Patient 1: 4 Patient 2: 7 Patient 3: 4 Patient 4: 11–3 patients: 3 1 patient: 4 Cognitive exclusion criteria: NR	Effect of istradefylline in combination with DA withdrawal on PA Treatment with Istradefylline after DA withdrawal.	Patient 1 with CC + AC (clinical), 1 year after ropinirole start and 1 year before evaluation. Patient 2 AC, same year after pramipexole start, 5 years before evaluation. Patient 3 PS, 3 years after rotigotine start, 1 month before evaluation. Patient 4 AC, 7 year from pramipexole start, 3 years before evaluation	Clinical evaluation; MRI of paraspinal muscles Pain and cognitive status: NR	Three patients (patient 1, patient 2 and patient 3) with preserved paraspinal muscle volume showed good responses to the treatment regimen at least two months after DA withdrawal. The patient 4, with moderate atrophy of the paraspinal muscles, did not improve	Two months	Poor –4
Fasano et al. 2011 [31] Case series	4 (3 M, 1F)	Patient 1: 5 Patient 2: 5 Patient 3: 7 Patient 4: 5 N/A Cognitive exclusion criteria: NR	Evaluation of reversible PS during rasagiline therapy -Rasagiline 1 mg/day	PS -Within 4 weeks after rasagiline introduction	Clinical evaluation Pain and cognitive status: NR	Rapid improvement of PS within 4 weeks after rasagiline withdrawal	4 weeks	Good-4
Cannas et al. 2009 [30] Case series	8 (6 M, 2F)	8.3 ± 1.8–2.9 ± 0.4 Cognitive exclusion criteria: NR	Evaluation of PS after: – introduction or increase of a dopaminergic drug (7 cases) – after withdrawal of pergolide (1 case) –3 pergolide 1 pramipexole 1	PS – Within 3 months introduction/increase/withdrawal of dopaminergic therapy	Clinical evaluation Pain status: NR Cognitive status: MMSE	MMSE 27.9 ± 1.2 (range 26–30) Improvement within 3 months after reduction/suspension of	3 months	Fair –4

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Table 1 (continued)

Study ID/Study design	No. of participants (no. of females)	PD duration – H&Y stage if reported; Cognitive status	Research question Medication status	Type of PA – PA duration	Study outcomes measures	Main results	Follow-up	QA-LE
			stalevo1 levodopa/Benserazide 1 levodopa-carbidopa CR Withdrawal of Pergolide (n = 1 case)			dopaminergic drug.Case associated with withdrawal of pergolide improved after levodopa dose increase		
Ameghino et al. 2018 [32] Case control	63 Cases with PA (34F) 63 Controls without PA (34F)	7.63 ± 7.83 vs 4.27 ± 3.87P < 0.05 -Cases 2.83 (±0.80) Controls 2.15 (±0.73) Cognitive exclusion criteria: NR	To evaluate the relationship between the treatment with antiparkinsonian pharmacological agents and the development of PA -Cases were more exposed to DAs (74.60 % vs 58.73 %, P = 0.05) and amantadine (30.16 % vs 7.94 %, P < 0.05) than control subjects	21 CC, 22 PS, 4 AC, 12 PS + CC, 3 CC + AC, 1 PS + CC + AC (clinical) -NA	Primary exposure do DAs. Secondary: exposure to other drugs; MDS-UDPRS; H&Y Stage Pain status: NR Cognitive impairment with MMSE ≤26	Cases were more exposed to DAs (74.60 % vs 58.73 %, P = 0.05) and amantadine (30.16 % vs 7.94 %, P < 0.05) than control subjects. Cases presented higher H&Y score (P < 0.05) and higher MDS-UPDRS part III score (29.61 ± 1.39 vs 20.76 ± 10.94, P = 0.05). Cognitive impairment was reported in 16 cases and in 10 controls (P = 0.18)	NA	Fair –3b
Brotini et al. 2021 [22] Case report	1 (F)	5 -NR	To evaluate if the addition of Co-ultramicrozoned palmitoylethanolamide/luteolin (um-PEALut, 700 + 70 mg) to the patient's treatment regimen of CD/LD can reduce the CC onset in OFF -um-PEALut at a dose of 700 + 70 mg added to regular CD/LD	CC only in the OFF state (not painful) -NR	Clinical evaluation Pain status: anamnestic Cognitive status: NR	Reduction of the OFF time, and the OFF states were not associated with the relapse of CC.	4 months	NA
Yasuda et al. 2018 [13] Case report	1 (M)	10 – NR	To assess the period between PS appearance after istradefylline introduction and PS recovery after discontinuation. –20 mg/d istradefylline discontinuation	PS –4 months after istradefylline start	Trunk deviation (no method explained) Pain status: NR Cognitive status: anamnestic	No cognitive impairment at baseline One week after istradefylline stop, PS began to improve, starting from 29°. At 1, 4, 8, and 12 weeks after removing istradefylline, trunk deviation continued to improve as follows:25°, 20°, 15°, and 13°, respectively. Finally, 17 weeks after removing istradefylline, trunk deviation decreased to 12°. This deviation is nearly the same as that before istradefylline administration, and the deviation was most likely the result of a vertebral deformity related to the patient's lumbar spondylosis.	2 years	NA
Mano et al. 2018 [14] Case report	1 (F)	10 -NA	To report a case of dropped head onset after the initiation of DA	AC with previous dull nuchal pain -Six months after start of pramipexole 0.5 mg twice per day	Clinical evaluation Pain status: anamnestic Cognitive status: NR	AC did not improve after increase of levodopa and pramipexole 1.5 mg/day, after switch to ropinirole 6 mg/day, and after switch to rotigotine 13.5 mg/day. No change was obtained with bilateral STN-DBS. AC improved 2 weeks after rotigotine discontinuation, and was absent after six months	6 months	NA

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Table 1 (continued)

Study ID/Study design	No. of participants (no. of females)	PD duration – H&Y stage if reported; Cognitive status	Research question	Medication status	Type of PA – PA duration	Study outcomes measures	Main results	Follow-up	QA-LE
Kataoka et al. 2017 [15] Case report	1 (F)	11—4	Response of dropped head to LCIG	-LCIG via nasoduodenal tube	Dropped head with lumbar pain 2 months	Dropped head angle improvement, evaluated from lateral photographs, both at rest and during the patient's best effort to avert her neck. The angle of dropped head was calculated with the use of "Image J" software by drawing a line between the vertex (Cz) and the seventh spinous process. Pain and cognitive status: NR	The angle in the natural position was 39.39° before the infusion of LCIG, Seven days after starting LCIG improved to 14.04°. On maximum effort to avert the neck, the angle decreased from 28.17 to 9.73°	7 days	NA
Galati et al. 2014 [16] Case report	1 (F)	4 -NR	To evaluate Ropinirole-induced PS	-Ropinirole extended release 4 mg/d for nearly 3 years	PS-2	Clinical evaluation Pain and cognitive status: NR	3 months after ropinirole stop, posture was nearly normal	3 months	NA
Kim et al. 2012 [17] Case report	1 (F)	3 -NR	To evaluate AC induced by pramipexole	-Pramipexole 1 mg three times a day for 6 weeks	AC with pain Three weeks	Clinical evaluation Pain status: anamnestic Cognitive status: NR	Resolution 1 week after the suspension of pramipexole	NA	NA
Solla et al. 2008 [18] Case report	1 (M)	8 -NR	To evaluate LD/CD/entacapone	Induced PS -LD/CD/entacapone tablets (600/150/1200 mg daily)	PS (painless) –2 weeks	Clinical evaluation Pain status: anamnestic Cognitive status: NR	Marked improvement of dystonic posture over the next few days after suspension of LD/CD/entacapone	NA	NA
Suzuki et al 2008 [19] Case report	1 (F)	4 -NR	To evaluate AC induced by pramipexole	-Pramipexole 1 mg three times a day for 11 months	AC – Nearly 11 months	Clinical evaluation Pain and cognitive status: NR	Marked improvement of dystonic posture over the next few days after suspension of pramipexole	NA	NA
Ho et al. 2007 [20] Case report	1 (M)	9	Response of CC to dopamine challenge testing	-CD/LD 50/200 mg + entacapone 200 mg four times a day; ropinirole 3 mg three times a day	CC (only during the off state, painless) – 2 years	Clinical evaluation Pain and cognitive status: anamnestic	Preserved cognitive status Dramatic response of CC to LD	NA	NA
Cannas et al. 2005 [21] Case report	1 (M)	3 -NR	Report of reversible PS during treatment with pergolide	-Pergolide 1 mg three times a day	PS –13 months	Clinical evaluation Pain and cognitive status: NR	Complete resolution of PS three months after withdrawal of pergolide	3 months	NA

Table 2
Studies of BoNT A and lidocaine for treating axial postural abnormalities in PD.

Study ID/ Study design	No. of participants (no. of females) Cognitive status	PD duration (years) -H&Y stage if reported	Total Toxin Dose per Injection Cycle, Method, and Muscle injected	Type of PA – PA Duration (y)-Diagnostic Criteria	Study outcomes measures	Main Results	Follow-up	QA-LE
Yahalom et al. 2023 [49] Case series	1 (M) Cognitive status n.r.	Nr- nr	Abobotulinumtoxin (Dysport) Bilateral Medial Scalene (50 U). Bilateral Levator Scapulae (25 U).Method: n.r.	Anterocollis – n.r. – Anterocollis was evident in sitting and standing position, did not improve in the supine position	Neurological examination Patient Global Impression of Change (PGI-C) Clinician’s Global Impression of Severity (CGI-S) Tsui scale Effect duration and side effects (SEs) of previous treatment	No objective or subjective improvement of anterocollis after BoNT.	n.r.	Poor – 4
Seliverstov et al. 2020 [51] Case series	2 (1 M,1F) Cognitive status n.r.	Patient 1: 11 Patient 2: 8 -n.r.	Onabotulinumtoxin (Botox) Patient 1:Longus Colli: 50 U anterior Scalene: 10 U each side. Levator Scapulae:15 U each side. Patient 2: Longus colli 40 U each side.Method: EMG –guided	Anterocollis –1 y, 5 onths – n.r	Duration of the Improvement after BoNT Side effects after BoNT % of satisfaction after BoNT	Patient 1 at the 4-week follow-up visit after BoNT, showed a reduction of anterior neck pain and improved ability to maintain a more upright head position. She developed a mild dysphagia. Her overall rate of satisfaction with BoNT treatment was 40 % Patient 2 at the 4-week follow-up visit after BoNT improved in neck mobility, and communication abilities, but he had slight difficulties in maintaining an upright neck posture. His overall rate of satisfaction with BoNT was 30 %.	4 weeks and 3 months after the injection.	Poor – 4
Artusi et al. 2019 [46] Prospective, pilot study		60.0 ± 12.0 (36–74) – 3.0 ± 0.6 (2–4)	Onabotulinumtoxin A (Botox) Paraspinal muscle (range): 50 to 75 U Non -paraspinal muscle (range): 25 to 50 U Method US-(Esaote, MyLabTM) EMG-guided (Dantec® Keypoint® G4 Workstation) Ipsilateral and contralateral paraspinal muscles, abdominal muscles, and Iliopsoas muscle (only contralateral) to the bending side	PS – 2.7 ± 2.3 (0 – 7) -a lateral trunk flexion of at least 10° im- proved by passive mobilization and supine positioning	Postural improvement observed two months after BoNT injection. Changes in the degrees of lateral trunk flexion measured with the wall goniometer and “ImageJ”. A cut-off of 5° was used to define a significant improvement. VASAdverse events and side effects related to the procedure (AEs)	The rate of responders was 84.6 % (n = 11/13). The angle of LTF improved by 40 % from 15.7 ± 8.4 to 9.4 ± 11.8 degrees.There was a reduction of pain by 52.2 % in the total VAS score, from 6.9 ± 2.2 to 3.3 ± 1.6.No procedural AEs, sustained bleeding, or cutaneous reactions and no cases of therapy-related adverse effects at the follow-up were observed.	2 months after BoNT injection.	Good – 2c
Matsumoto et al. 2018 [37] Case report	1 (M) Cognitive status n.r	2y – 4	Onabotulinumtoxin (Botox): 50U to the right (ipsilateral) paraspinal muscles from L2 to L4 spinous processes Method: none	CC – 1y axial flexion to the anterior side PS – 1y axial flexion to the right side	Postural improvement by body X-ray and Surface EMG	After BoNT the CC and PS improved and the right abdominal pain was also relieved.A body X-ray showed improvement in the CC.Surface EMG showed increased tonic contraction of the left lumbar paraspinal muscles and reduced tonic contraction of the right lumbar paraspinal muscles.	n.r.	Fair – 4
Todo et al. 2018 [38] Case series	6 (4F, 2 M) Inclusion criteria Mini-Mental	10.6 ± 5.3—3.3 ± 0.52	Onabotulinumtoxin (Botox) (range): 75 to 90 U.Method: sonographic guidance Bilateral	CC – 2.3 ± 0.8 – flexion of the thoracic spine	CA degree before vs.2 weeks after BoNT. Nerng finding after BoNT. VAS	The mean angle of CC improved from median (interquartile range); 38° (23.5°) to 18° (21°),	The length of the follow-up in the study varied across patients. All 6	Poor – 4

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Table 2 (continued)

Study ID/ Study design	No. of participants (no. of females) Cognitive status	PD duration (years) -H&Y stage if reported	Total Toxin Dose per Injection Cycle, Method, and Muscle injected	Type of PA – PA Duration (y)-Diagnostic Criteria	Study outcomes measures	Main Results	Follow-up	QA-LE
	State Examination score ≤ 25		External Abdominal Oblique muscle			p = 0.028. 4/6 patients showed subjective relief.2/6 patients improved painful abdominal contraction from 84 and 98 to 4 and 77.	patients were studied in the short term (2 weeks after the botulinum injection), but for the long-term observation (1 year or more after the injection), only 3 patients were studied.	
Dupeyron et al. 2015 [47] Case report	1	10 years	Incobotulinumtoxin (Xeomin): 50U (T0 and L3 level), left iliocostalis (Ipsilateral) muscle 50 U, left quadratus lumborum (ipsilateral) muscle	PS A tonic lateral flexion of the trunk toward the left side was observed, with a complete resolution in supine position and increased deviation during standing.	Clinical improvement observing pictures	The injection of the ipsilateral iliocostalis muscle did not improve PS, but improved pain. The injection of the ipsilateral quadratus lumborum improved PS.	Follow-up up to 1 year	Fair – 4
Tassorelli et al. 2014 [41] RCT	26 patients n = 13 group A injection of incobotulinum toxin type A (Xeomin) (8F, 5 M) n = 13 group B treated with saline (6F, 7 M) Inclusion criteria Mini-Mental State Examination score > 24	group A (10.2 \pm 8.2), Group B (10.4 \pm 10.1) -Group A (2 in n = 6 patients, 3 in n = 7 patients), Group B (2 in n = 6 patients, 3 in n = 7 patients)	Incobotulinumtoxin A (Xeomin) Maximum 6 sites per patient, with a maximum dose of 50 UI of botulinum toxin per site, with a total dose per patient ranging between 50 and 200 UI. Method EMG-guided Bilateral (ipsilateral or contralateral) Multifidus, Iliopsoas, Rectus Abdominis, inferior thoracic and lumbar paravertebral muscles	PS, CC –Trunk flexion group A: 3.1 \pm 1.9, Trunk flexion Group B 3.0 \pm 1.5 -PS: mild to moderate lateral trunk flexion (Cobb's angle > 10°),CC: anterior trunk flexion	Intensity of low back pain (VAS) UPDRS score FIM score Degree of anterior flexion of the trunk Range of motion of the trunk on the 4 plans Kinematic analysis Lateral inclination in the standing position Anterior flexion in the standing position ROM ipsilateral bending ROM contralateral bending ROM anterior flexion ROM posterior extension	At the end of the treatment period (BoNT/saline + rehabilitation), groups A and B improved significantly in static postural alignment and ROM. Group A showed a significantly more marked reduction in pain score as compared with Group B and a more prolonged efficacy on several clinical and kinematic variables.	At the end of the 4-week rehabilitation program.3 months (t2) after discharge. 6 months after discharge.	Good – 1b
Wijemanne et al. 2014 [39] Case report	1F Cognitive status n.r	7 y – n.r.	Onabotulinumtoxin (Botox): Bilateral Rectus Abdominis: 200 U External (left) Abdominal Oblique:200 U Method:EMG-guided	Dystonic CC – n.r. – (palpable abdominal contractions when standing. Patient could not lie completely flat on her back) (2 yr)	Degree of anterior flexion of the trunk, MDS-UPDRS-III	CC never regressed to her pre-BoNT severity, but it remained improved at 15-20° during the “ON” state (with a motor UPDRS score of 8) compared to 30° in the “OFF” state (with motor UPDRS = 26) The presence of a reduction of right abdominal painful contractions.External Oblique muscle can contribute to truncal flexion.	n.r.	Poor – 4
Furusawa et al. 2013 [42] Before-after	12 (8F, 4 M) Cognitive status n.r	10 \pm 7.7–3.6 \pm 0.7	Lidocaine injections (50 mg of 1 % xylocaine, Astrazeneca) Bilateral External Abdominal Oblique muscle: 50 mg Method: ultrasound guidance	CC – n.r.–CC with upper fulcrum: abnormal truncal flexion at a point between the T12 and L1 vertebrae with flexion angle higher than 40°	Upper CC flexion angle	After a single injection of lidocaine, 8/12 (66.7 %) of patients showed a reduction of flexion angle, on average from 62.1 \pm 13.4 to 54.0 \pm 16.8°. The following repeated injections, led to further improvements: 9/12 patients showed a reduction of flexion angle, on average from 62.1 \pm 13.4 to 49.0 \pm 18.5°. In 8/9 patients, the improvement	90 days	Fair – 4

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Table 2 (continued)

Study ID/ Study design	No. of participants (no. of females) Cognitive status	PD duration (years) -H&Y stage if reported	Total Toxin Dose per Injection Cycle, Method, and Muscle injected	Type of PA – PA Duration (y)-Diagnostic Criteria	Study outcomes measures	Main Results	Follow-up	QA-LE
Furusawa et al. 2012 [43] Case series	5 (4F,1M) Cognitive status n.r	8.2 ± 3.9—2.6 ± 0.8	Lidocaine injections (xylocaine) Bilateral Rectus Abdominis, External and Internal Abdominal muscle. Each muscle was injected 50 mg Method: Ultrasound guidance	CC – n.r. –CC with upper fulcrum: abnormal truncal flexion at a point between the T12 and L1 vertebrae, with flexion angle higher than 40°	CC flexion angle	of posture was maintained after 90 days after first injection. All patients improved posture after injection (in particular the external oblique muscle). There was an improvement, on average, from 49 ± 6.0 to 37.6 ± 10°. One patient showed a mild improvement of posture after the injection into the rectus abdominis.	n.r.	Fair – 4
Santamato et al. 2010 [45] Case report	1 M Cognitive status n.r	n.r. – 3	Abobotulinumtoxin (Dysport): 600 U (100 U/6 sites of injection) (+rehabilitation)Method: EMG recordings Bilateral paraspinal muscles (2–2.5 cm lateral to the spinous processes, T10-L2)	PS – n.r.- marked trunk deviation to the right side	Axial lateral flexion of the trunk angle TDDS VAS	Post-treatment (after 15 days) there was an improvement of PS from 35° to 15°, improvement of 5 points in TDDS, and a reduction of VAS score from 7 to 3.	Fifteen days after the beginning of combined treatment.	Poor – 4
Oyama et al. 2009 [50] Case series	4 F Cognitive status n.r	n.r.- 3	Lidocaine (0.5 %, total 30 ml), MAB (1 % lidocaine 20 ml, mixed with 99.5 % ethanol 10 ml, 15 ml to each side, total 30 ml), and botulinum toxin (BOTOX, total 100–200 units)Method: EMG-guided Sternocleidomastoid, Levator scapulae, Splenius capitis	Dropped head syndrome – from 5 to 15 y – an abnormal ante-flexed posture of the neck on standing/sitting position, whereas curvature of the spine is not present or is relatively mild	Needle EMG Neck CT	Lidocaine injection into Sternocleidomastoid muscle markedly improved dropped head, but the effect was temporary. The effect of botulinum toxin and muscle afferent block was not satisfactory. Lidocaine injection (lidocaine test) could be useful for determining the most affected muscle before using BoNT or muscle afferent block.	3 months	Fair – 4
Colosimo et al. 2009 [36] Case series	2 – n.r. Cognitive status n.r	n.r. – n.r.	Onabotulinumtoxin A (Botox) Total: 800 mU Bilateral deep lumbar portion of Iliopsoas: 300 mU Bilateral Rectus Abdominis: 200 mU Method: CT guidance	CC – n.r. -a reducible forward flexion of the thoracolumbar spine > 45°	n.r.	There was no observed improvement, either objectively or subjectively, in “CC” during the time periods of 1 day, 1 week, and 2 weeks following BoNT injection.	1 day after the procedure. 1 week after the procedure. 2 weeks after the procedure.	Poor – 4
Fietzek et al. 2009 [40] Goal attainment controlled study	10 n = 5 injected in the Iliopsoas (n.r.) n = 5 injected in the rectus abdominis (n.r.) Cognitive status n.r	n.r. – n.r.	Incobotulinumtoxin A (Xeomin) Total (range): 100–300 U (mean dosage 210 ± 50 U) Bilateral Iliopsoas 220 ± 40 U Bilateral Rectus Abdominis 200 ± 63 U Method: ultrasound guidance	CC -patients injected in the Iliopsoas muscle (1.9 ± 0.2), patients injected in the rectus abdominis (3.0 ± 1.4) -n.r.	Timed Up&Go 10 m Goal attainment after 3 weeks after BoNT (functioning, impairment, activity limitations, or participation restrictions)	After 3 weeks of BoNT there was no notable achievement of therapy goals among the patients. None of the patients had successfully reached their therapy goals in full. BoNT in mean dosages of 210 U is not an adequate treatment for CC	3 weeks	Poor – 4
Von Coelln et al. 2008 [33] Case series	3 M Cognitive status n.r	16y,8y,10y – 3	Abobotulinumtoxin A (Dysport) All patients received from 500 to 1,500 MU of BoNT per side at beginning of the treatment and repeated after 4–6 months of interval. Method: ultrasound guidance Unilateral (n = 1 PD) and bilateral (n = 2 PD) injection of the deep portions of the Psoas Major	CC – 3y, 1y, 1.5y – n.r.	Evaluation (physical examination and measuring of body height for quantitative assessment of posture)	–No local complications (hematoma or infection) were reported. All patients complained a mild to moderate weakness of hip flexion at the highest dose of BoNT. Patient 1 consistently reported experiencing improvements lasting for two weeks after each injection. These improvements	2, 4, and 16 weeks after each treatment.	Poor – 4

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Table 2 (continued)

Study ID/ Study design	No. of participants (no. of females) Cognitive status	PD duration (years) -H&Y stage if reported	Total Toxin Dose per Injection Cycle, Method, and Muscle injected	Type of PA – PA Duration (y)-Diagnostic Criteria	Study outcomes measures	Main Results	Follow-up	QA-LE
Van de Warrenburg et al. 2007 [48] Case series	n = 8 PD (3F,5M) n = 1 familial PD (M) Cognitive status n.r	10.5 ± 7 n.r.	Abobotulinumtoxin (Dysport): n = 7 patients Uni/bilateral Levator scapulae: 50–250 U Uni/bilateral Sternocleidomastoid 50–100 U Uni/bilateral Splenius capitis 200 U Method: EMG-guided	Antecollis – 10.5 ± 7.0 – a forward flexion and anterior shift of the neck with prominent cervical paraspinal and Levator scapulae muscles, usually without weakness of residual neck extension	Pain EMG studies	enabled the patient to successfully reach and grasp objects from a previously inaccessible shelf. Notably, height measurements revealed a moderate enhancement in the patient's spontaneous posture, with an increase of 16 cm. Patient 2 showed an improvement in standing and sitting upright for several weeks after injection of BoNT. Patient 3 did not show a relevant improvement of height measurement after BoNT but showed a slightly worsening of spontaneous posture. Most patients did not benefit from these injections; pain reduction was reported in 1 patient An actual improvement of the antecollis in 2 patients	n.r.	Poor – 4
Bonanni et al. 2007 [44] Blinded cross-over trial	9 (5 M,4F) n = 4 BoNT treatment n = 5 placebo MMSE mini mental state examination 20.8 ± 3.3	10.3 ± 7.2 – 2.5 ± 0.46	Abobotulinumtoxin (Dysport): 500 U Method: EMG-guided Paraspinal muscles 2 to 2.5 cm lateral to spinous processes at level L2-L5, ipsilateral to the bending side	PS – 2.4 ± 0.9 – a lateral flexion of the trunk ≥15°	- TDDS – VAS – Goniometric measurement of the lateral displacement	n = 6 patients showed an improvement of lateral banding from 50 % to 85.7 % and reduction of pain. Patients with BoNT treatment improved by an average of 4 points in TDDS score and improved pain by an average of 31.4 mm in VAS scale score from the baseline assessment.	Up to 6 months after BoNT.	Fair – 4
Azher et al. 2005 [34] Case reports	11 (9 M,7F) Cognitive status n.r	11.3 ± 7.6 y – n.r.	Onabotulinumtoxin A (Botox) Total botulinum toxin (range): 300–600 U Method: (n.a.)Rectus Abdominis	CC – 4.5 ± 3.9 -A thoracolumbar spine flexion, >45°	Latency and duration of effect after BoNT Duration and severity of complications	There was a moderate to marked improvement of posture lasting for about 3 to 6 months after injection in 4 patients (one patient reported an improvement of posture by 75 %).	6 months	Poor – 4

Abbreviations: BoNT, botulinum toxin; CC, Camptocormia; CT, Computed tomography; EMG, Electromyography; FIM, Functional Independence Measure; LTF, Lateral trunk flexion; MPMG, Multiple injections point per muscle technique; n.r., not reported; PD, Parkinson's disease; PS, Pisa syndrome; ROM, range of motion; SD, Standard deviation; SPMG, Single point per muscle injection strategy; TDDS, Trunk Dystonia Disability Scale; U, units; UCC, upper camptocormia; UPDRS, Unified Parkinson's Disease Rating Scale; VAS, Visual Analogue Scale; y, years; QA, Quality of Assessment; LE, Levels of Evidence.

Table 3
Studies of Deep Brain Stimulation for treating axial postural abnormalities in PD.

Study ID/Study design	No. of participants (no. of females)	PD duration (years) -H&Y stage if reported	DBS target and settings -Research question	Type of PA – PA Duration	Study outcomes measures	Main results	Follow-up	QA-LE
Guerrero et al. 2022 [64] Case report	1 (M)	5 -NA	Bilateral STN DBS- Efficacy on low back pain and sagittal balance	Not explicated (“Sagittal imbalance”) Prior to PD	Clinical evaluationPain status: anamnestic Cognitive status: NR	Improvement of low back pain (with bilateral lower extremity radiculopathy) and sagittal balance	3 months	NA
Anderson et al. 2019 [65] Case report	1 (M)	7-NA	Bilateral GPi-Long term efficacy of GPi DBS on PS	PS (clinical)-24 months	Lateral bending anglePain status: anamnestic Cognitive status: NR	Improvement from 45 to 25 degrees, significant reduction of severe back pain	48 months	NA
Pandey S. 2017 [66] Case report	1 (F)	7 y-NA	Bilateral STN DBS- Efficacy on UCC	UCC (clinical)-24 months	Clinical evaluationPain status: anamnestic Cognitive status: NR	After DBS there was resolution of CC and back pain	3 months	NA
Akiyama et al. 2017 [67] Case report	1 (F)	12 at DBS16 at Spinal cord stimulation-NA	Bilateral STN-DBS; 4 years later, Spinal cord stimulation (SCS)DBS: Amplitude (V) R 3.5 (bipolar configuration)/ L 3.2, Pw (usec) L 60, R 60, Frequency (Hz) R 60, L 60-To evaluate efficacy of Spinal cord stimulation for painful CC with OS	CC and PS (clinical)-2 y from DBS6 y from SCS	Clinical evaluationPain status: anamnestic (VAS is reported only after SCS) Cognitive status: NR	Painful CC and PS initially improved with STN DBS but reappeared after 2 years. Painful CC and PS improved also after SCS	6 months after SCS	NA
Ekmekci and Kaptan 2016 [68] Case report	1 (F)	11—3	Bilateral STN-DBS- Effects on CC	CC (clinical)-1 y (but with low back pain from 7 years)	Trunk bendingPain status: S-LANNS pain scale Cognitive status: MMSE	Improvement from 60° to < 10° Best improvement achieved in low back pain ($\geq 7/10$ in S-LANNS pain scale)MMSE 26/30 (28/30 at 1 year)	1 year	NA
Ricciardi et al. 2014 [69] Case report	1 (M)	8-NA	Pedunculopontine (PPN) DBS ipsilateral to bending side-To evaluate long-term effect on PS of ipsilateral PPN DBS	PS (clinical)-1 y	Lateral bending angle, UPDRS-IIIPain and cognitive status: NR	6-months after surgery: remarkable improvement of lateral bending, no other relevant changes of UPDRS-III. During the following years, posture progressively worsened as well as his motor and cognitive functions	40 months	NA
Oliveira et al. 2013 [70] Case report	1 (M)	10-NA	Bilateral STN-DBS-To evaluate effect on Dropped Head	AC-108 months	Clinical evaluation Pain status: anamnestic Cognitive status: MMSE; Frontal assessment battery (FAB); Mattis Dementia Rating Scale	No effect on AC and dull nuchal pain MMSE: 30/30FAB: 16/18Mattis Dementia Rating Scale: 138/144	NA	NA
Lyons et al. 2012 [71] Case report	1 (F)	19-NA	Bilateral STN-DBS-To evaluate long-term effects of STN DBS on CC	CC (clinical)-NR	Trunk bending on thoracolumbar regionPain status: anamnestic Cognitive status: NR	Improvement: from 80 to 90° at baseline, to 10-20° on standing in med-off/stim-on 3 months after surgery.Back pain resolved after DBS Sustained effect at 5 years.	5 years	NA
Thani et al. 2011 [72] Case report	1 (F)	13 -NA	Bilateral GPi-DBS – To evaluate effects of GPi DBS on CC	CC (clinical) –2 y	Clinical and visual evaluation, Postambulation sagittal shoulder-hip-knee angle; sagittal head-shoulder-hip angle Pain and cognitive status: NR	At 1 year The 1st angle improved from 133° to 160°, the other from 148° to 170°; complete resolution of CC; improvement in	14 months	NA

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Table 3 (continued)

Study ID/Study design	No. of participants (no. of females)	PD duration (years) -H&Y stage if reported	DBS target and settings -Research question	Type of PA – PA Duration	Study outcomes measures	Main results	Follow-up	QA-LE
Pereira et al. 2010 [73] Case report	1 (F)	15 -NA	Bilateral STN-DBS -To evaluate effects of STN DBS on AC	AC developed after odontoid fracture occurred three weeks after DBS (clinical)-After DBS start	Clinical Pain status: anamnestic Cognitive status: NR	UPDRS part III score from 25 to 14 Development of disproportionate anterocollis and painful spasmodic torticollis, unrelieved by parameter adjustment of the DBS	NA	NA
Yamada et al. 2006 [83] Case report	1 (F)	11 -NA	To evaluate effects of STN DBS on CC	CC (clinical) –5 y	Clinical evaluation Pain and cognitive status: NR	Beneficial effect on CC for over 20 months	20 months	NA
Hellmann et al. 2006 [74] Case report	1 (M)	25 -NA	Bilateral STN DBS -Response to STN-DBS of severe CC in a patient with young-onset PD	CC (clinical) 19 y	Clinical evaluation; UPDRS Pain and cognitive status: NR	OFF-UPDRS score changed from 91 to 58 ON-UPDRS and 47 to 29 The bent spine-and-knees posture was significantly reduced, starting from > 90°	10 months	NA
Micheli et al. 2005 [75] Case report	1 (M)	10 -NA	Bilateral GPi DBS- Response of severe CC	CC (clinical) –2 years	Clinical evaluation Pain and cognitive status: NR	No significant changes at 3 months; slow but sustained improvement of CC at 6 months postoperatively and until 14 months	14 months	NA
Soares et al. 2019 [76] Case series	2 (1F)	Patient 1: 12 Patient 2: 9 –NA Cognitive exclusion criteria: NR	Bilateral STN-DBS -Objective assessment approach in PD with CC that underwent bilateral STN-DBS	CC ≥45° with low back pain -Patient 1: 1.5 y Patient 2: 9 y	UPDRS-III, automated assessment of bending angle and gait parameters (neuroKinect system) Pain status: anamnestic Cognitive status: NR	Patient 1: improvement of 26 points in the UPDRS-III (med ON/stim ON), CC angle (68° before surgery to 38° after surgery) Patient 2: CC (47° before surgery to 9° after surgery) with an improvement of 26 points in the UPDRS-III (med ON/stim ON)	Patient 1 12 months Patient 2 8 months	Fair –4
Capelle et al. 2011 [78] Case series	3 (7 total, but only 3 with PD)	Patient 1: 12 Patient 2: 15 Patient 3: 10 –NA Cognitive exclusion criteria: included but NR	Patient 1 and 2: Bilateral STN-DBS Patient 3: Bilateral GPi- DBS -To evaluate effects of GPi or STN DBS on CC (not only in PD)	CC (clinical) -NR	Burke–Fahn–Marsden (BFMDRS) scale; UPDRS Pain status: VAS Cognitive status: NR	BFMDRS/UPDRS score (OFF) – VAS score of low back pain Patient 1: from 43/8 to 20/6 –from 8 to 6 Patient 2: from 36/12 to 14/12—7 to 6 Patient 3: from 47/9 to 24/6—5 (unchanged)	(Months) Patient 1 16 Patient 2 12 Patient 3 36	Fair-4
Asahi et al. 2011 [77] Case series	4 (2F)	Patient 1: 13 Patient 2: 12 Patient 3: 12 Patient 4: 9- NACognitive exclusion criteria: NR	Bilateral STN-DBS-To evaluate in which types of patients STN-DBS is effective in treating CC	CC (clinical)- Patient 1: 1.8 y Patient 2: 6 y Patient 3: 6 y Patient 4: 7 y	Trunk angle; thoracolumbarparaspinal muscle status calculating paraspinal muscle area and mean CT number (from Computed Tomography) Pain and cognitive status: NR	Patient 1: from 50° to 28° Patient 2: from 40° to 21° Patient 3: from 36° to 23° Patient 4: from 50° to 51°The mean CT number of paraspinal muscle was much smaller in the unchanged patient (range of mean CT number; 17.3–35.0) than in the improved patients (mean CT number; –34.6).	(Months) Patient 1: 18 Patient 2: 21 Patient 3: 40 Patient 4: 24	Good –4

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Table 3 (continued)

Study ID/Study design	No. of participants (no. of females)	PD duration (years) -H&Y stage if reported	DBS target and settings -Research question	Type of PA – PA Duration	Study outcomes measures	Main results	Follow-up	QA-LE
Upadhyaya et al. 2010 [79] Case series	2 (4 total, but only 2 treated with DBS)	Patient 1: NR Patient 2: NR –NA Cognitive exclusion criteria: NR	Bilateral STN DBS Bilateral Gpi DBS -To present representative cases of treating CC in PD.	CC (clinical) -NR	Clinical evaluation Pain and cognitive status: NR	No improvement of CC	Patient 1: 2 years Patient 2: 15 months	Poor –4
Sako et al. 2009 [80] Case series	6 (4F)	9 (range 5–11) -NACognitive exclusion criteria: NR	Bilateral STN DBS- Report of disabling CC alleviated by bilateral STN-DBS	CC > 45° –25 months (range, 12–60 months)	Thoraco-lumbar angle; UPDRSPain and cognitive status: NR	Mean Off-UPDRS III decreased from 48.6 ± 16.6 to 15 ± 9.9; in the “on” phase it decreased from 41 ± 14.7 to 15 ± 9.9. The mean thoracolumbar angle decreased from 73 ± 18.6 to 17 ± 10.3 (postoperative follow-up);	16.8 (range 5–46)	Poor-4
Schabitz et al. 2003 [81] Case series	2 (2 M)	Patient 1: 30 Patient 2: 9- NACognitive exclusion criteria NR	Bilateral STN DBS- Evaluation of focal myopathy of the paraspinalMuscles in trunk forward flexion	CC (clinical, developed during DBS treatment)AC (clinical)-Patient 1 (CC); 4Patient 2 (CC and AC); NA	Clinical evaluation	Patient 1: The PA appeared during the treatment with DBSPatient 2: no response of CC and AC	Patient 1: 15 Patient 2: 3	Poor-4
Margraf et al. 2010 [82] Case control	3 (NA) of 15 cases and 15 controls	PD duration at DBS: NA Authors reported PD duration at evaluation: Patient 1: 22 Patient 2: 41 Patient 3: 19 -NACognitive exclusion criteria: NR	Bilateral STN DBS- To describe theclinical features of CC	CC (clinical, developed during DBS treatment)-5 months 54 months 47 months	Clinical evaluation Pain status: numeric analog scale (NAS) Cognitive status: NR	Development of CC with low back pain during the DBS treatment NAS: NR of every patient	NA	Fair-4
Lai et al. 2021 [53] Observational Retrospective Before-After	36; 25 without CC, 11 with CC (15 of 36F)	10.8 ± 4.4 -NA Cognitive exclusion criteria: NR	Bilateral GPI- DBSAmplitudes (V) L 3.1 ± 0.5/R 3.0 ± 0.6 Frequency (Hz) L 134.1 ± 30.5/R 134.9 ± 30.6 pw (µsec) L 71.3 ± 13.0/R 72.3 ± 12.7 Efficacy on CC	CC (7 TCC-CC ≥30° and 3 UCC-CC ≥45°. 1 had both TCC-CC and UCC-CC)-NR	TCC angle; UCC angle Pain and cognitive status: NR	In TCC-CC group, the TCC angles decreased from 39.1° ± 10.1° to 23.3° ± 8.2° (p = 0.0168); In the UCC-CC group, UCC angles significantly decreased (50.5° ± 2.6° to 39.0° ± 6.7°, p = 0.0124); In patients without CC, a slight but significant deterioration was seen in the TCC angles (from 15.9° ± 5.4° to 17.3° ± 6.6°, p = 0.0308), whereas a non-significant improvement was found in the UCC angles (from 34.2° ± 4.5° to 33.5° ± 5.9°, p = 0.6261) Greater improvement in the TCC angles was found in patients with larger pre-surgical TCC angles during the med-OFF state (p = 0.0001) and better LD responsiveness of the TCC angle (p	7.3 ± 3.3 months	Good-2b

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Table 3 (continued)

Study ID/Study design	No. of participants (no. of females)	PD duration (years) -H&Y stage if reported	DBS target and settings -Research question	Type of PA – PA Duration	Study outcomes measures	Main results	Follow-up	QA-LE
He et al. 2021 [54] Observational Retrospective Before-After	52; 2 with PS (2 of 2 M)	Case 1: 6 Case 2: 9-NA Cognitive exclusion criteria: NR	Bilateral STN DBS Amplitude (V) R 2.5 (bipolar configuration)/ L 2.1, Pw (usec) L 60, R 60, Frequency (Hz) R 130, L 130 Case 2 Amplitude (V) R 2.5/L 2.8, Pw (usec) L 60, R 60, Frequency (Hz) R 130, L 130-Efficacy on PS	PS > 10°-Case 1: 0.5 y Case 2: 2	UPDRS-III score, Pisa angle Pain status: anamnestic Cognitive status: NR	= 0.0043); Improvement of the UCC angles were positively correlated with pre-surgical UCC angles (p = 0.0065) Case 1 (with low back pain): pre DBS Med-Off 59/10°pre DBS Med-On 26/2°, after DBS Med-Off/ Stim-ON 20/2°after DBS Med-On/Stim-ON 18/2° Case 2: pre DBS Med-Off 71/14°pre DBS Med-On 46/14°, after DBS Med-Off/ Stim-ON 43/6°after DBS Med-On/Stim-ON 39/6°	Case 1: 10 months Case 2: 15 months	Fair-4
Liang et al. 2020 [63] Observational Retrospective Before-After	15 DBS with CC ≤3 years (8F)	10.5 ± 4.5–3.2 ± 0.4 Cognitive exclusion criteria: included but NR	Bilateral STN DBS Frequency: 130–170 Hz; Pw: 60–90 μs; Amplitude: 1.5–3.5 V. Efficacy on CC combined with rehabilitation and psychological interventions,	CC ≥15°-2.1 ± 0.9 y	MDS UPDRS-III; CC degree (calculated with android app Max Protractor ver. 1.1.2, an open source software by Maxcom, based on the angle between the long axis of the femur and the upper thoracic plane (bending angle)) Pain status: anamnestic Cognitive status: NR	Most patients experienced various degrees of back pain at baseline MDS-UPDRS, item 3.13 baseline:2.80 ± 0.77 (Med-Off) 1.13 ± 0.52 (Med-On), at follow-up 0.86 ± 0.64 (Med-Off) (<0.001) 0.87 ± 0.64 (Med-On) (<0.05) Degree of CC (°):46.20 ± 8.79 (Med-Off) 14.60 ± 6.09 (Med-On), at follow-up 8.46 ± 7.08 (Med-Off) (<0.001) 8.47 ± 7.08 (Med-On) (<0.001)	6 months	Fair-4
Lai et al. 2021 [52] Observational Retrospective Before-After	36; 10 with CC (7 of 36F)	Reported only for total sample (9.4 ± 3.8)-Range 2–4 Cognitive exclusion criteria: NR	Bilateral STN DBS- Efficacy on CC	CC (8 TCC-CC ≥30°, 2 UCC-CC ≥45°)-NR	Posture analysis: with CamptoAPP based on the method recommended in the consensus statement by Margraf et al; MDS-UPDRS III Pain and cognitive status: NR	All patients in the med-OFF/DBS-ON had significant decrease in both the TCC angle (from 22.6°±16.5° to 16.8 ± 7.0°; p = 0.0069) and the UCC angle (from 33.9°±7.5° to 31.3° ±6.0°, p = 0.0056) at follow-up. Marked improvement in axial symptoms (from 9.2 ± 3.7 to 6.9 ± 3.7, p = 0.0002). In patients with TCC-CC, significant decrease in the TCC angle at follow-up from the baseline med-OFF condition (50.2° ±11.7° vs. 25.3° ±8.3°, p = 0.0004) The 2 patients with UCC-CC similarly showed an	6.0 ± 2.2 months	Fair-2b

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Table 3 (continued)

Study ID/Study design	No. of participants (no. of females)	PD duration (years) -H&Y stage if reported	DBS target and settings -Research question	Type of PA – PA Duration	Study outcomes measures	Main results	Follow-up	QA-LE
Roediger et al. 2019 [55] Observational Retrospective Before-After	158 (NR)	11.8 ± 4.7-NA Cognitive exclusion criteria: NR	Bilateral STN-DBS Amplitude (V) 3.2 ± 0.6 (1.5–5.1) Frequency (Hz) 137 ± 31 (60–210) Pw (µs) 61 ± 4 (60–90)- To quantify the effect of STN DBS on trunk and neck postural angles from video-recordings	3 CC (2 UCC-CC > 45°, 1 TCC-CC > 30°); 2 PS > 10°-NR	The primary endpoint was the global postural angle calculated by the sum of the ventral thoracolumbar, ventral thoracic, ventral cervicaloccipital, lateral thoracolumbar, and lateral cervical-occipital PA. Videoframes were analyzed using “ImageJ”, to calculate the angles. Pain and cognitive status: NR	improvement in the UCC angle at follow-up (from 61.0° to 33.4° and from 51.6° to 47.7°). In the entire sample, higher structural connectivity to the right supplementary motor area (SMA) and right lateral premotor cortex along the dorsal plane (PMd) was associated with larger postsurgical improvements in axial signs and TCC angles after stimulation was turned on. In patients diagnosed with CC, larger improvement in CC angles after STN-DBS was associated with a larger VTA overlap with STN (R = 0.75, p = 0.032). The global postural angle improved by 6.7 % between the pre-surgical MED-ON and post-surgical MED-ON/STIM-ON assessments, from 53.8 ± 21.6° to 50.2 ± 17.2° (p = 0.031). Patients with lower (n = 2) and upper (n = 1) CC respectively improved by 48.1 % in the ventral thoracolumbar angle (36.4 ± 0.0° to 18.9 ± 4.2°) and 13.8 % in the ventral thoracic angle (49.1°–42.3°). Patients with PS (n = 2) improved by 67.5 % in the lateral thoracolumbar angle (16.9 ± 2.0° to 5.5 ± 4.7°).	15.4 ± 11.0 months	Good-2b
Schlenstedt et al. 2019 [56] Observational Retrospective Before-After	192 (62 F)	NR –13.3 ± 5.1 Cognitive exclusion criteria: NR	To investigate the effect of medication and STN-DBS on posture in PD and in subgroups of PD patients with normal posture, impaired (stooped) posture, or clinically diagnosed CC. Second, to study whether the long-term treatment with DBS has a carryover effect on	TCC angle, 157 (81.8 %) TCC-stooped, and 13 (6.8 %) with CC (angle ≥30° in Med-Off). UCC angle, 104 (54.2 %) UCC-stooped, and 40 (20.8 %) with upper CC (UCC angle ≥45° in Med-Off). 9 (4.7 %) had both a	TCC angle UCC angle Pisa angle Pain and cognitive status: NR	For the TCC angles post-DBS, a significant effect of medication (p < 0.001; F = 125.3), a significant effect of stimulation (p < 0.001; F = 40.3), and a medication × stimulation interaction (p = 0.006; F = 7.5)	6–24 months	Good –2b

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Table 3 (continued)

Study ID/Study design	No. of participants (no. of females)	PD duration (years) -H&Y stage if reported	DBS target and settings -Research question	Type of PA – PA Duration	Study outcomes measures	Main results	Follow-up	QA-LE
			posture-Baseline: Med-Off and Med-On conditions. Follow-up: Med-Off/Stim-Off, Med-On/Stim-Off, Med- Off/Stim-On, and Med-On/Stim-On conditions.	clinically diagnosed TCC and UCC. Pisa angle: 120 (62.5 %) had a postural alignment between healthy and posturalchanges, and 2 patients (1.0 %) had a PS ($\geq 10^\circ$)-NR		were found. Medication had an effect additional to stimulation ($p < 0.001$).For the UCC angles, significant effects of medication ($p = 0.033$; $F = 4.6$) and of stimulation ($p < 0.0001$; $F = 15.5$) were found. The medication \times stimulation interaction was not significant for UCC ($p = 0.844$; $F = 0.03$). Stimulation had an effect additional to medication ($p = 0.02$).TCC angle Baseline Med-OFF All 19.4 (7.0) Normal 8.9 (1.6) Stooped 18.8 (4.8) CC 35.9 (5.3) Follow Up Med OFF-Stim ON All 16.6 (6.3) Normal 10.5 (3.5) Stooped 16.2 (5.4) CC 25.6 (8.2) UCC angleBaseline Med-OFF All 40.2 (6.0) Normal 31.7 (2.6) Stooped 40.1 (2.5) CC 48.4 (2.6). Follow Up Med OFF-Stim ON All 39.9 (6.3) Normal 35.8 (6.5) Stooped 39.4 (5.3) CC 44.7 (6.0). Pisa angleBaseline 2.1 (1.9)Follow Up 1.8 (1.4)		
Okazaki et al. 2018 [57] Observational Retrospective Before-After	74; 30 with AF, 16 with scoliosis (37 of 74 F)	AF: 11.8 ± 0.9 Scoliosis: 13.4 ± 1.4 -NACognitive exclusion criteria: included but NR	Bilateral STN-DBS-To investigate the relationship between clinicalcharacteristics and improvement in abnormal postures of PD patients who received STN-DBS	AF (based on data from 62 patients) (C7 sagittal vertical angle > 5 cm) Scoliosis (based on data from 68 patients) (Cobb angle $> 15^\circ$)-NR	Clinical and demographic characteristics, cobb angle and C7SVA (C7 sagittal vertical axis)Pain and cognitive status: NR	AF (29 patients analyzed): In 17 patients, C7SVA was improved by more than 5 cm and in 12 patients, C7SVA improvement was less than 5 cm. Scoliosis (13 patients analyzed): 5 patients presented with improvement of scoliosis over 5° . Patients with improved AF after STN-DBS had thicker abdominal oblique muscle and transverse abdominal muscle than those of patients without improved AF. Patients with	Time of follow-up not explicitly reported	Fair-4

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Table 3 (continued)

Study ID/Study design	No. of participants (no. of females)	PD duration (years) -H&Y stage if reported	DBS target and settings -Research question	Type of PA – PA Duration	Study outcomes measures	Main results	Follow-up	QA-LE
Artusi et al. 2018 [58] Observational Retrospective Before-After	101 patients (39 of 101F). n = 55 TFI (DBS posture improving patients), n = 46 TFNI (DBS posture not improving patients)	Total sample 15.38 ± 4.69 TFI 16.00 ± 4.80 TFNI 14.63 ± 4.49 -Total sample 3.67 ± 0.91 TFI 3.53 ± 0.97 TFNI 3.83 ± 0.80 Cognitive exclusion criteria: included but NR	Bilateral STN-DBS- Efficacy on trunk PA	Abnormal Trunk posture, defined as a score > 0 on the item 28 of the UPDRS in Med-On. CC > 45° (n = 23) PS > 10° (n = 5)-NR	The comparison between Med-Off(baseline) and Stim-On/Med-Off(follow-up) conditions item 28 of the UPDRS (improvement defined as an amelioration of at least 1 point in the abnormal trunk posture) Pain and cognitive status: NR	improved scoliosis were significantly younger at PD onset than those without improvement. 42.7 % improvement in the severity of trunk PA (from 3.86 ± 0.36 to 2.21 ± 0.83; P < 0.001)The subanalysis of patients with CC (n = 23) and PS (n = 5) showed a 42.7 % improvement in abnormal posture severity (from 3.86 ± 0.36 to 2.21 ± 0.83; P < 0.001).	12 ± 4 months	Fair-4
Sakai et al 2017 [59] Observational Retrospective Before-After	14 patients with PD and CC (6F)	13.1 ± 4.9- NACognitive exclusion criteria: included but NR	Bilateral STN-DBS Frequency 130–160 Hz; Pw, 60–120 usec, amplitude, 1.5–3.6 V.-To determine a clinical marker for selecting an appropriate therapy for CC, authors investigated the atrophy and degeneration of paraspinal muscles before surgery	CC ≥ 30° -1.4 ± 1.9 y	Thoraco-lumbar angle (TLA) for CC; MRIs of the thoracolumbar spine for measurements of paraspinal muscle Pain and cognitive status: NR	4 patients with effective DBS (<30° and lasted six months after STN-DBS); 5 partially effective (TLA decreased but persisted > 30° with or without lasting six months after STN-DBS); 5 patients non-effective (no change in TLA after STN-DBS)The cross-sectional area of the lumbar paraspinal muscle with width was significantly larger in the EF group.	6 months	Fair-4
Yamada et al. 2016 [60] Observational Retrospective Before-After	17 (10F)	12.9 ± 6.02- NACognitive exclusion criteria: NR	Bilateral STN-DBS Frequency, 130–160 Hz; Pw, 60–120 μsec; Amplitude, 1.5–3.6 V.-Effects on CC and preoperative factors predictive of postoperative improvement.	CC ≥ 45° 48.3 ± 34.6 months	Photographs to measure thoraco-lumbar angle in Off-Med status Pain and cognitive status: NR	The angle at baseline was 84.0 ± 29.5° and significantly ameliorated 3 months postoperatively (49.8 ± 29.3°) and at the last follow-up (54.8 ± 28.3°).	36.5 ± 17.7 months	Fair-2b
Schulz-Schaeffer et al. 2015 [61] Observational Retrospective Before-After	25 (4F) N = 13 non responders N = 12 responders	All patients 15.4 (3–27) Responders 14.7 (3–27) Non Responders 17 (12–25)- NACognitive exclusion criteria: NR	Bilateral STN-DBS-To identify prognostic factors for the DBS effect on CC	25 CC ≥ 30°; 23 Laterodeviation (clinical)-(months) All patients 35 (8–90) Responders (13 patients) 19.8 (8–61) Non Responders (12 patients) 51.4 (21–90)	Trunk bending angle Pain status: VAS Cognitive status: NR	All patients: From 53.2 (30–90) to 34.8 (0–90) Responders: from 52.7° (30–90) at baseline to 9.6 (0–30) Non responders: from 53.8 (30–90) to 62.1 (40–90) 19 patients reported lumbar back pain before DBS. VAS for pain reduced from 6.9 to 4.2	(months) All patients 30.9 (6–66) Responders 30.0 (7–66) Non responders 31.9 (6–64)	Good-2b
Umemura et al. 2010 [62] Observational Retrospective Before-After	18 (14F)	13.4 (range 5–20)- NACognitive exclusion criteria: NR	Bilateral STN DBS-Effect on relieving PA	8 CC (clinical) 8 PS (clinical)-NR	Degree of postural abnormality according to item 28 of the UPDRS III (score 2: moderate postural abnormality – score 3 or 4: severe postural abnormality) Pain status:	Most patients have mild to-moderate low-back pain at baseline. 13 patients with moderate postural abnormality, 9	Short term: 1 month Long term: 1 year	Fair-4

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Table 3 (continued)

Study ID/Study design	No. of participants (no. of females)	PD duration (years) -H&Y stage if reported	DBS target and settings -Research question	Type of PA – PA Duration	Study outcomes measures	Main results	Follow-up	QA-LE
					anamnesic Cognitive status: NR	improved soon after surgery, but 1 patient deteriorated again. Two patients improved only over a long period after surgery. In 5 patients with severe postural abnormality, 2 improved slightly in the long-term follow-up period after surgery.		

Legend: AC, antecollis; AF, Anteflexion; CC, camptocormia; H&Y, Hoehn and Yahr; LE, Levels of Evidence; MDS-UPDRS, Movement Disorder Society-UPDRS; MMSE: Mini-Mental State Examination; No., number; N.R., not reported; QA, Quality of Assessment; PA, postural abnormalities; PD, Parkinson's Disease; PS, Pisa Syndrome; Pw, Pulse width; UPDRS, Unified Parkinson's Disease Rating Scale; VAS: Visual analogue scale.

studies [52–63], 40.6 % (n = 13) were Case Reports [64–76], 18.75 % (n = 6) were Case Series [77–82], and 1 was a Case-control study [83]. Twenty-seven studies evaluated the effect of bilateral subthalamic DBS (STN-DBS) [52,54–59,61–64,66–68,70,71,73–75,77–84], six studies the effect of bilateral globus pallidus par interna DBS (Gpi-DBS) [53,65,72,76,79,80], and 1 study the effect of DBS targeting the Pedunculopontine nucleus ipsilateral to the bending side of PS [69]. The studies included 200 patients with CC, 24 patients with PS, and 3 cases of AC; in 4 studies, CC patients were divided according to a higher (i.e., thoracic) or lower (i.e., lumbar) spine flexion [52,53,55,56]. In 4 studies, we had an unspecific referral to abnormal posture, a score >0 on item 28 of the UPDRS, or sagittal imbalance to define the PA [57,58,62,64]. The median duration of PD and of PA at DBS (not including two studies in which PA occurred after DBS) was 11.8 years (range 5–25 years) and 2 years (range 0.5–19 years), respectively. The cognitive status at baseline was reported as exclusion criteria in 5 studies, and in 2 studies, there was a report of MMSE. Outcome measures differed across studies, with 16 studies using the angle of spine flexion [52–56,59,61,63,65,68,69,71,74,77,78,81], 13 using a clinical evaluation [64,66,67,70,72,73,75–77,80,83–85], 10 using the UPDRS score [52,54,58,62,63,69,75,77,79,81], 1 using the BFMDRS score [79], and 1 using the Cobb angle and the C7SVA (C7 sagittal vertical axis) [57]. In 81.25 % of studies (n = 26), an improvement in PA was reported. Studies reporting degrees of spine flexion angle showed a median improvement after DBS of 45.8 % for CC (range 0.7 % – 88.9 %) and 62.3 % for PS (range 14.3 % – 80 %). Three case reports, two case series, and one case-control study on 10 DBS-treated patients found no improvement in CC or PS after surgery [67,70,73,80,82,83]. One case report showed posture improvement for six months, with subsequent deterioration [69]. 14 studies reported pain associated with PA at baseline, and nine studies (seven case reports, one before-after, one case series) reported an improvement after intervention. For details, see Table 3.

3.4. Spinal surgery

The total number of patients with PD and PA was 51, including patients with CC, PS, or mild forms of anterior/lateral trunk flexion [84–93]. AC or mild forms of anterior neck flexion have not been reported in the literature as treated with surgery. Most study designs included a case series/report (n = 8) and one retrospective study. The surgical approach seems to have a positive outcome for PA severity, pain, and quality of life in the early stages. However, most studies reported complications after surgery and the need for surgical revision. There was a high heterogeneity of PA assessment tools, which does not

allow for comparison of the treatment effects among studies and thus to calculate the median improvement. For details, see Table 4.

3.5. Standing alone or combined rehabilitative interventions

We found 18 studies on the effect of rehabilitative interventions on PA in PD, including 326 patients. 44 % (n = 8) were RCT [41,94–100], 11 % (n = 2) were Case Reports [45,101], 28 % (n = 5) were Prospective Interventional Before After [102–106], 11 % (n = 2) were Case Series [43,107], and one controlled intervention study [108]. In most studies (n = 13), a follow-up assessment was performed (from 1 to 26 months).

44 % (n = 8) investigated the effects of rehabilitative approaches as standalone treatments [94–97,102,103,107,108]. Half of that were RCTs [94–97]. Rehabilitation combined with botulinum toxin [41,45] or lidocaine [43,105,106] was explored in five studies. Other approaches involved non-invasive brain stimulation (NIBS) [98], osteopathic [99], electroacupuncture [101], galvanic stimulation [100], and prismatic lenses [104] combined with rehabilitation intervention. Three studies included patients with only anterior trunk flexion [97,100,103], and two included patients with CC [43,105], for a total of 53 patients. Only Furusawa and colleagues investigated patients with upper CC [43]. Two studies [95,96] included patients with anterior trunk flexion and lateral trunk flexion for 50 patients. Two studies enrolled patients with LFT (n = 48) [41,102] and seven studies patients with PS (n = 72) [45,98,99,101,102,104–108]. In two studies, we had an unspecific referral to abnormal posture defined as kyphosis [94] and stooped posture [108]. PA duration was reported in 50 % of cases (n = 9) [41,98–103,106,107]. The median duration of PD and PA at the time of evaluation was nine years (range 4–17.2 years) and two years (range 0.35–3.6 years), respectively.

The PA was measured in degrees by using clinical (n = 12) [43,45,95,97,99,100,103–108] or instrumental kinematic analysis (n = 5) [41,94,96,98,102]. One study used x-ray Cobb angle [101]. Other measures explored motor and non-motor (pain) symptoms, balance, mobility, and gait performance. Only five studies included disability and quality of life (FIM, PDQ9, ADL) [41,96–98,103]. The cognitive status measured by the Mini-Mental State Examination (MMSE) was used as the selection criteria for seven studies. Pain effects were measured in nine studies (50 %). An overall decrease in PA severity was reported irrespective of the treatment approaches in the 75 % (n = 3) of the RCTs exploring the effectiveness of rehabilitation treatments. Similarly, a general reduction of the pain severity was reported after rehabilitation treatment. The high heterogeneity of PA assessments does not allow for comparing the treatment effects among studies and thus calculating the

median improvement. No studies focused on anterior neck flexion and AC. One study measured the degree of cervical flexion in patients with anterior neck flexion [96]. For details, see Table 5.

3.6. Orthosis

We found four studies on the use of orthosis on PA in PD, including 32 patients. One study was a Case Series [109], one a Case Report [110], one a cross-sectional study [111], and one a mixed-population uncontrolled clinical trial involving only two patients with PD [112]. The studies included six patients with CC and 26 with anterior trunk flexion. All studies evaluated the effect of different types of orthoses. One study explored the immediate effect of spinal kyphosis-orthosis on anterior trunk flexion and anterior neck flexion. In the remaining three studies, the patients wore the orthoses during daily activities. In one study, a high-frame walker with forearm support was used by three patients [110]. One study used thoracic-pelvic anterior distraction orthoses in a mixed population [112]. One study used a cruciform anterior spinal hyperextension (CASH) associated with daily back extensor strengthening exercise (30 min/day) for four months [109], and one study investigated the effect of spinal kyphosis-orthosis on anterior trunk flexion during gait [111]. One study reported the patients' cognitive status [110]. Two studies included a measure of pain severity among outcomes [110,112]. For details, see the [supplementary material](#). Overall, orthosis was effective in improving balance and gait outcomes. The two studies evaluating pain severity reported an overall reduction in symptom severity [110,112]. The assessment tools used to measure the PA severity and the therapy effects varied considerably among studies. The observational nature of these studies (lacking control treatment) hampers inferring the specific role of such orthosis.

4. Discussion

PA associated with PD calls for interdisciplinary management from the early stage of the disease based on two cardinal issues [2,4]. Firstly, all professionals (i.e., neurologists, psychiatrists, physical therapists, occupational therapists) must share assessment tools and consensus-based criteria [1,113–115]. Secondly, patients should be included in multidisciplinary management based on evidence-based medical treatment (i.e., PD therapy optimization, BoNT, DBS if eligible) and rehabilitation programs according to the type and degree of PA as soon as possible [4]. An algorithmic approach to the management of PA in PD is suggested in Fig. 3.

The etiopathogenesis of PA in PD remains partially unclear. However, the literature supports central (dystonia, proprioceptive and sensorimotor integration deficits, rigidity) and peripheral (spine/soft tissue changes) mechanisms as two mutually non-exclusive pathophysiologic hypotheses [2,116]. Therefore, PA management should be based on the etiopathogenesis of the disturbances involving all contributing mechanisms. Several approaches have been proposed for managing PA in PD, mainly addressing individual aspects of the disorder's pathophysiology. This has led to the fragmentation of interventions that need to consider the multifactorial nature of the disturbances. Furthermore, most studies have not considered specific nosologically entities with precise severity [4,5,113].

Much attention has been devoted to CC (and its manifestations of anterior trunk flexion) and the PS. In contrast, limited attention has been given to AC, for which we need more data on pathophysiology and management. Since last year, the lack of consensus-based criteria for evaluation and classification has hampered the analysis of existing literature regarding the prevention or early treatment of these highly disabling postural disorders.

4.1. Pharmacological oral treatment

The adjustment of pharmacological oral treatment is the first step for

the effective management of PA in PD. Levodopa is the most effective medication for managing PD motor symptoms, including tremors, rigidity, bradykinesia, and gait problems. While levodopa primarily targets these motor symptoms, it may also have some impact on posture and muscle tone, which could improve PA. However, the effectiveness of levodopa in specifically targeting these symptoms can vary among individuals. Istradefylline is an adenosine A2A receptor antagonist that is used as an adjunctive treatment for PD. It works by reducing the "off" time in individuals already on levodopa therapy. Selegiline is a selective monoamine oxidase-B (MAO-B) inhibitor commonly used in the treatment of Parkinson's disease. It helps to increase dopamine levels in the brain by inhibiting the enzyme that breaks down dopamine. While Istradefylline and Selegiline may help improve overall motor symptoms, there is limited specific evidence regarding its effectiveness in addressing PA in PD. Considering the current body of literature, at the onset of a new PA in PD, it can be a reasonable option to try to adjust the pharmacological therapy. The use of selegiline and istradefylline requires further studies. Another important finding that can be taken from the literature is that some cases of PA (especially PS) can appear acutely or sub-acutely after the administration of a new drug, more often a dopamine agonist. In such rare cases, it is possible that the withdrawal of the drug can lead to a resolution of PA; thus, this practice is recommended.

4.2. Pharmacological injection treatment

Botulinum toxin (BoNT) and lidocaine muscle injection have been proposed as a treatment to reduce muscle overactivity in patients with PS, CC, and AC [117]. Despite their high effectiveness in reducing muscle overactivity in focal dystonia, for which it is the recommended treatment [117], BoNT and lidocaine are still debated for their usefulness in treating axial PA [117]. Definite conclusions about the efficacy of BoNT and lidocaine should be drawn with caution, given the scant literature on this topic. Overall, the efficacy of BoNT and lidocaine injection is controversial, and it is still premature to conclude that BoNT is effective and appropriate for these motor complications. There are many reasons, including the poor study design and small sample sizes of previous studies, heterogeneity in muscle selection and injection techniques [4], insufficient data for appropriate doses and types of BoNT, and the need for a standard clinical outcome measurement. Finally, the unclearness of predictive factors of therapy response (i.e., PA duration) hamper the reliability of this approach. However, in the absence of other options and along with the optimization of dopaminergic therapy and physiotherapy, at least for PS, the use of BoNT injection in hyperactive axial muscles on EMG assessment could be considered in carefully selected patients. It should be avoided injections in compensatory paraspinal and nonparaspinal muscles [4]. Further scientific evidence with an adequate sample size is needed by RCTs.

4.3. Deep brain stimulation

A not negligible number of studies (including a high number of patients) have explored the improvement of PA after STN or GPi DBS, and overall, they showed that people with CC and PS could have a remarkable improvement in posture. Also, back pain associated with CC or PS has a good chance of improvement, albeit this last information is provided mainly by case reports. However, it is essential to highlight that these are all retrospective studies (or case series and case reports), and no RCTs or prospective cohort studies are available in the literature to evaluate posture before and after DBS. Moreover, we have scant data on AC, another invalidating PA. The cognitive status of patients was unavailable in most studies, which reported cognitive impairment as an exclusion criterion for treating patients with DBS. Considering available literature evidence, we can conclude that PD patients eligible for DBS to manage motor symptoms and suffer from PS or CC (or a milder form of PA) should be even more encouraged to undergo DBS surgery since posture may improve. Prospective studies, also including patients with

Table 4
Surgical treatments for axial postural abnormalities.

Study ID/Study design	No. of participants (no. of females) Cognitive status	PD duration (years) – &Y stage if reported	Type of PA – PA Duration (y) -Diagnostic Criteria	Study outcomes measuresMain Results	Complication Rate/N. of revisions	Follow-up	QA-LE	
Mei et al. 2022 [87] Case report	1 (M) Cognitive status n.r	7y – n.r.	CC – n.r.- a kyphotic deformity characterized by a marked flexion of the thoracic cage and lumbar spine	Physical examination (pain)CT scan and radiograph (cobb angle)	Postoperative examination showed that the lumbar lordosis was corrected. At the last follow-up, the patient was in good condition and did not report any low back discomfort and resumed his social activities	Revision surgery:1Complications: yes	1 month after surgery 1 month after revision surgery.2 months after revision surgery.3 months after revision surgery6 months after revision surgery1.5 years after the revision surgery	Poor – 4
Farah et al. 2022 [93] Retrospective/prospective case series	12 (6 M,6F) Cognitive status n.r	n.r. – n.r.	CC (n = 5) – n.r. -n.r.PS (n = 3) – n.r.- n.r.	Scoliosis (n = 6) n.r. – n.r.	Oswestry Disability Index (ODI)Radiographic parameters-Pelvic incidence-Pelvic tilt-Lumbar lordosis-Sagittal vertical axis-Coronal tilt	ODI score was significantly decreased from 64 % to 52 % at 1-year of follow-up.Lumbar lordosis was significantly improved from – 16.7° to – 41.4° at 1-year of follow-up. Pelvic tilt wasthe least effectively corrected parameter, with a mean preoperative value of 31.6° vs. 27.8° at 1 year. Sagittal vertical axis was significantly improved from 149.7 mm to 73.6 mm at 1-year of follow-up. Coronal tilt was significantly corrected from 68.2 mm to 22.9 mm at 1-year of follow-up.	Revision surgery: n = 8 patientsn = 3 patients:1 revisionn = 4 patients:3 revisionsComplications: none	40,8 months (mean) [range 12–70]
Nunna et al. 2020 [88] Retrospective case series	3 (2 M,1F) Cognitive status n.r	n.r.- n.r.	Scoliosis – n.r. -n.r.	ODIVAS (back and leg) Radiographic parameters-Pelvic incidence (°)-Pelvic tilt (°)-Lumbar lordosis (°)-Sagittal vertical axis (mm)-Coronal tilt (°)-Coronal balance-Cobb (°)-Sacral slope (°)-Pelvic incidence-Lumbar lordosis (°)	Patient 1: Three-month after surgery, patient showed an improvement of posture: coronal imbalance of 33 mm; sagittal imbalance of 83 mm; lumbar lordosis of 46°; pelvic incidence of 77°; pelvic tilt of 37°; sacral slope of 34°.Improvement of back pain 1 y after surgery. VAS score from “severe” to 0.ODI changed from 22 to 30. Patient 2: Three-month after surgery, patient showed an improvement of posture in sagittal balance of 77 mm, Cobb angle of 20°, and a lumbar lordosis of 46°. Patient reported a resolution of pain after surgery, 1-yr follow up.Patient 3: 6-month FU visit, patient reported an improvement of posture and pain.	Revision surgery (number): 2Complications: none	2 y	Good – 4
Yamato et al. 2020 [89] Retrospective case series	22(6 M,16F) Mental health SRS-22r scores:-	19 y – 3.5 ± 0.6	CC-n.r.-n.r.Scoliosis-n.r.-n.r.Kyphosis following fracture-n.r.-n.r.Kyphosis following spinal fusion -n.r.-n.r.	Radiographic parameters -Thoracic kyphosis (°) -Lumbar lordosis (°)	Improvement of posture with a mean-Thoracic kyphosis from 25.2° to 23.9°- Lumbar	Revision surgery: 36.4 % (n = 8 patients) 16 operationsComplications: yes	5 Years	Good – 4

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Table 4 (continued)

Study ID/Study design	No. of participants (no. of females) Cognitive status	PD duration (years) – &Y stage if reported	Type of PA – PA Duration (y) -Diagnostic Criteria	Study outcomes measuresMain Results	Complication Rate/N. of revisions	Follow-up	QA-LE	
	Preop 2.50 ± 0.54–6 Months:3.08 ± 0.64–1 y: 2.99 ± 0.48–2 y: 2.85 ± 0.73–5 y: 2.60 ± 0.61			-Pelvic tilt (°) -Pelvic incidence (°) -Sagittal vertical axis (mm) -Pelvic obliquity (°) -C7-center of sacral vertical line (mm)-Indoor activities of daily living-Health-related quality of life (HRQOL)	lordosis from 4° to 34.3°- Pelvic tilt from 40.1° to 26.8°- Sagittal vertical axis from 220.1 mm to 94.9 mm- C7- center of sacral vertical line from 66.9 mm to 13.5 mm.9/22 patients showed improvement in quality of life 1 year after surgery. 7/22 patients were previously dependent became independent post-surgery, but 2/22 patients experienced a decline in their ambulatory status, needing wheelchairs instead of the walker or cane they used before the operation.			
Schroeder et al. 2015 [90] Retrospective study	96 (57F,39 M) of whom only seven had sagittal or coronal deformity Cognitive status n.r	n.r.<2 in thirteen patients, 2 in thirty patients2.5 inTwenty-three patients, ≥3 in thirty patients	Spinal stenosisSpondylolisthesisCoronal orsagittal deformity mean sagittal misbalance of 12 cmPrimary coronal deformity of 45° to 55°	VAS (back pain and lower-limb)ODIShort Form-12preoperative and postoperative	Improvement of VAS back pain from 7.4 cm preoperatively to 1.8 cm postoperativelyVAS lower-limb pain improved from 7.7 cm preoperatively to 2.3 cmPostoperativelyODI from 54.1 points to 17.7 points at the time of the latest follow-upShort Form-12 physical component from 26.6 points preoperatively to 30.5 points postoperatively	Revision surgery: 12 %–15.8 % Complications: yes	30.1 months	Poor –4
Sato et al. 2013 [86] Case report	2 (F) Cognitive status n.r.	n.r. – n.r.	Kyphosis – n.r. confirmed with X-ray examinationSpinal scoliosis -n.r. confirmed with X-ray examination	Radiographic parameters C7-plumb (mm) Thoracic kyphosis (°) Lumbar lordosis (°) Pelvic tilt (°) Pelvic incidence (°) Sacral slope (°) Cobb angle (°) C7-plumb (mm) VAS	Improvement of posture and reduction of painPatient 1:- VAS: from 9 before surgery to 2 after surgery.-C7-plumb (SVA) from 193 mm to 56 mm.-Thoracic kyphosis from 24.4° to 29.1°-Lumbar lordosis from – 2.1° (kyphosis) to 29.6°-Pelvic tilt from 44.5° to 30°-Pelvic incidence from 56.7° to 53.3°-Sacral slope from 14.3° to 29.6°- Cobb angle (L1 to L4 level) from 48.2° to 20.1°- C7-plumb (frontal) from 41 mm to 13 mm. Patient 2-VAS from 9 to 1.- C7-plumb from 148 mm to 51 mm.-Thoracic kyphosis from 6.3° to 22.4°-Lumbar lordosis from – 18.2° (kyphosis) to 13.6°-Pelvic	Revision surgery Complications: None	12 months	Poor- 4

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Table 4 (continued)

Study ID/Study design	No. of participants (no. of females) Cognitive status	PD duration (years) – & Y stage if reported	Type of PA – PA Duration (y) -Diagnostic Criteria	Study outcomes measuresMain Results	Complication Rate/N. of revisions	Follow-up	QA-LE
Kawaguchi et al. 2013 [91] Case report	1 (F) Cognitive status n.r	7y – n.r.	Kyphoscoliosis n.r.- n.r.Sagittal and frontal imbalance – n.r.- distances between the C7 plumb line and the superior posterior endplate of S1 vertebralbody andbetween C7 plumb line and frontal center S1 vertical line.	Radiographic parametersSagittal center vertical lineFrontal center vertical line	tilt from 43.7° to 24.8°- Pelvic incidence from 50.7° to 45.1°-Sacral slope (deg.) from 5.4 to 16.8.-Cobb angle (deg.) (L1 to L4) from 30.4 to 12.8-C7-plumb (frontal) (mm) from 21 to 18. The patient showed a reduction of paresthesia, numbness in both legs, C7 sagittal center vertical line and frontal center vertical line.The patient after surgery could stand alone and walk with improved spinal posture.	Revision surgery: noneComplications: yes	n.r. Poor – 4
Wadia et al. 2011 [85] Case reports	2 (M) mental status wasnormal	Patient 1: 13 yPatient 2: 9 y-n.r.	CC – patient 1: 2 y, patient 2: 1 y- a disabling forward flexion of the trunk	VAS Spinal radiographs SF-36 ODI	Patient 1 -Change in VAS back pain score from 10 to 2- Improvement of posture with a change at T3-T12 spinal process level of 12°; a change at T10-L2 spinal process level of 10°; a change at T12-L5 spinal process level of 45°; a change at C7 spinal process level using the sagittal plumb line of 15.4 cm.Patient 2- Improvement of posture with a change at T3-T12 spinal process level of 3°; a change at T5-T12 spinal process level of 5°; a change at T10-L2 spinal process level of 17°; a change at T12-L5 spinal process level of 25°; a change at C7 spinal process level using the sagittal plumb line of 8.9 cm.	Revision surgery (number): 2 (patient 1); 1 (patient 2) Complications: acute myocardial infarction	Patient 1: 5 yearsPatient 2: 3 years Poor – 4
Peek et al. 2009 [84] Case report	1 (M) Cognitive status n.r	n.r. – n.r.	CC – 10 y – a thoracolumbar spine flexion, >45° alleviated by supine positioning	X-ray Neurological assessment	Post-surgery, patients reported a significant improvement of posture and quality of life without back pain.	Revision surgery: 2Presence of surgical complications: Yes	2 years and 5 months Poor – 4

Abbreviations: PA, Axial postural abnormality; ASD, adjacent segment disease; CC, Camptocormia; F, female; FU, Follow-up; n.r., not reported; PD, Parkinson's disease; M, male; MSA, Multiple System Atrophy; ODI, Oswestry disability scores; SF-36, Short Form Health Survey 36; VAS; yr, years; QA, Quality of Assessment; LE, Levels of Evidence.

Table 5
Studies of rehabilitation interventions for axial postural abnormalities in PD.

Study	Study design;n. of patients; Cognitive status	H&Y	PD duration y	Type of PA	Duration of PA (y)	PA measurement; Study outcomes	Rehab Dose/ duration	Type of protocol	Training Effects	FUP	QA-LE
a. Rehabilitation											
Yang et al. 2010 [94] RCT	EG, n = 16 CG, n = 17 Cognitive exclusion criteria n.r.	EG = 2.23 ± 0.53 CG = 2.17 ± 0.72	EG = 4.77 ± 4.83 CG = 5.27 ± 5.55	Kyphosis	n.r.	Electronic Goniometer (degrees) Spatio-temporal gait performance Knee muscle strength Pain and cognitive status n.r.	3d/wk for 4 wks	Downhill TT walking training Grade: 3 %, increases by 1 % progressively (WBS < 40 %) vs conventional therapy	Significant between-group changes in thoracic Kyphosis only at FUP [EG: -2.88°; CG: +3.11°]	1 month	Poor - 2b
Capecchi et al. 2014 [95] RCT	EG, n = 13 (PR + KT) CG, n = 7 Patients' exclusion criteria MMSE ≤ 20	2-4	PR = 9.5 ± 7.4 PR + KT = 11 ± 4.4 CG = 9.6 ± 4.9	ATFLTFUPDRS III >= 2 (posture item)	n.r.	Anatomic references (degrees) anterior and lateral trunk flexion. BBS, TUG Pain and cognitive status n.r.	3d/wk for 4 wks	Active postural reeducation and proprioceptive and tactile stimulation vs no treatment	The EG significant decreases in trunk flexion [PR + KT: -3.2°; CG: 0°] post-treatment and at FUP [PR + KT: -2.7°; CG: +0.5°]; EG significant decrease in trunk inclination only post-treatment [PR + KT: -2.5°; CG: 1°].	1 month	Fair - 2b
Volpe et al. 2017 [96] RCT	EG, n = 15 CG, n = 15 Patients' exclusion criteria MMSE ≤ 24	2-3	EG = 9.4 ± 7.5 CG = 9 ± 7.0	ATFTF	n.r.	Posturography and BAKUPDRS-III, TUG, BBS, FESABC, PDQ-39, Likert scale pain Cognitive status n.r.	5d/wk for 8 wks	Water-based vs. non-water-based	The EG significantly decreased the PA severity compared to the CG after 8 wks. BAK cervical flexion [EG: -62.5 mm; CG + 1,7 mm] BAK dorsal flexion [EG: -22.5 mm; CG: -6.5 mm] lateral inclination [EG: -2.3°; CG: +0,3°] In both groups not significant differences in pain over time.	16 weeks	Good - 1b
Gandolfi et al. 2019 [97] RCT	EG, n = 19; MoCA = 23.68 ± 3.48 CG, n = 18 MoCA = 23.93 ± 3.63 Patients' exclusion criteria MMSE ≤ 24	EG = 3 [1.5; 3] CG = [1.37; 3]	EG = 8.01 ± 5.90 CG = 6.57 ± 4.29	ATF	n.r.	MoCA Freeware software-based program (degrees) UPDRS III, dynamic and static balance, pain, falls, and quality of life assessment.	5 days/wk, 4 wks	Trunk specific exercise program plus home-based self-management vs conventional rehabilitation	The EG significant decreases in anterior trunk flexion [EG: -10°; CG: -2°] post-treatment and FUP [EG: - 10°; CG: -1°] Not significant between-group differences for pain. In both groups pain reduction over time	1 month	Good - 1b
Bartolo et al. 2010 [102] Before-After (Pre-Post) Studies With No Control Group	PA+, n = 22 PA-, n = 22 Patients' exclusion criteria MMSE ≤ 24	1-3	PA+ = 7.9 ± 3.0 PA- = 8.6 ± 4.3	LTF	3.6 ± 2.3	Kinematic behavior of the trunk by an optoelectronic system UPDRS-III Pain and cognitive status n.r.	5d/wk for 4wks	All patients underwent warm-up activities, stretching exercises, strengthening exercises in functional contest, gait training and relaxation exercises.	After treatment significant decreased in trunk flexion [-10° at T1, -9° at FUP] and inclination [-11° at T1, -9° at FUP] in upright standing posture and trunk mobility.	6 months	Fair - 2b
Lee et al. 2017 [103] Case Series	Inpatients, n = 6 Outpatients, n = 3 Cognitive	2-4	1 m-5y	ATF	2 m-10 y	Flexion angle in standing position, UPDRS-II UPDRS III, mH&Y, ADL,	Inpatients twice a day, 5 wks., Outpatients: home-based	Inpatients: back extensor strengthening exercises. Outpatients: home-based	Significant changes in flexion angle (mean	2 m-26 m	Poor - 4

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Table 5 (continued)

Study	Study design;n. of patients; Cognitive status	H&Y	PD duration y	Type of PA	Duration of PA (y)	PA measurement; Study outcomes	Rehab Dose/ duration	Type of protocol	Training Effects	FUP	QA-LE
Lena et al. 2017 [107] Case Series	exclusion criteria n.r. n = 6 [GA, n = 3; GB, n = 3] Exclusion criteria: presence of cognitive impairment	2–3	8.5 ± 12	PS	2.5 ± 1.39	Motor symptoms.Pain and cognitive status n.r. Wall goniometer (degrees)VAS (back pain) UPDRS-IIUPDRS-III Cognitive status n.r.	Outpatients 1 d/w for 3 months. 10 consecutive treatment sessions	exercises and backpack wearing treatment. Postural trunk deviation correction, postural patient's awareness increasing, trunk control and function improvement.	reduction 20.66°) in all sample. Overall, After treatment a significant decreased in LFT.No significant changes in LFT in both GA and GB groups. GA reported significant improvement on back pain, while not significant changes in GB	n.a.	Fair –4
Kawami et al. 2018 [108] Intervention Studies	EG, n = 0, [MMSE = 27.1 ± 1.7]CG, n = 10 [MMSE = 28.1 ± 1.9] Patients' exclusion criteria MMSE < 24	CG = 2-4 4EG = 2–4	EG = 10.1 ± 2.8 CG = 10.8 ± 6.3	Stooped Posture	n.a.	Trunk bending angles, lumbar lordosis, thoracic kyphosis (Spinal Mouse) in comfortable and upright standing position. Pain and cognitive status n.r.	6 d/wk for 4-wks	Postural rehabilitation vs usual home healthcare service	The EG significantly decreased the trunk bending in the comfortable [–6.1° ±3.6] and upright [–6° ±2.2] standing position compared to the CG [+0.4° ±5.2; 0° ±3.6]. The EG significantly decreased the lumbar lordosis in the comfortable [–4.6° ±3.6] and upright [–6° ±5.5] standing position compared to the CG [+4.0° ±9.1; +1° ±7.1].	n.a.	Poor –2b
b. Rehab & Botulinum toxin											
Tassorelli et al. 2014 [41] RCT	EG, n = 13 [VAS = 6.7 ± 1.5]CG, n = 13 [VAS = 5.4 ± 2.5] Patients' exclusion criteria MMSE ≤24	<4	EG = 10.2 ± 8.2 CG = 10.4 ± 10.1	LTB	EG = 3.1 ± 1.9 CG = 3.0 ± 1.5	Kinematic analysis of trunk by optoelectronic system Trunk lateral flexion, UPDRS III, FIM, VAS for pain. Needle EMG protocol testing paraspinal and nonspinal muscles. Cognitive status n.r.	5d/wk for 4 wks	EG = Rehab + iBTA [max 50U/site; total dose 50–200 U] CG = Rehab + saline	The EG significantly decreased the degree of lateral trunk bending post-treatment and at 3-month FUP compared to the CG. The EG significantly decreased the degree of anterior trunk bending only post-treatment compared to the CG. VAS score decreased significantly in both groups. At the end of rehabilitation, the entity of decreased was more marked for EG than CG.	3 and 6 months	Fair –1b
Santamato et. al. 2010 [45] Case Report	n = 1 Cognitive status n.r.	3	5	PS	n.a.	Trunk Dystonia Disability Scale, VAS for pain. Cognitive status n.r.	5d/wk, 3 months; 3d/wk	BTX-A injection [600U] + multidisciplinary rehabilitation program	Fifteen days after the beginning of combined treatment, the patient decreased the trunk	3 months	NA

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Table 5 (continued)

Study	Study design;n. of patients; Cognitive status	H&Y	PD duration y	Type of PA	Duration of PA (y)	PA measurement; Study outcomes	Rehab Dose/ duration	Type of protocol	Training Effects	FUP	QA-LE
									inclination angle by 15°. Effects were maintained at FUP. Fifteen days after the beginning of the combined treatment VAS was decreased from 7/10 to 3/10.		
c. Rehab & Lidocaine injection											
Di Martino et al. 2017 [106] Before-After (Pre-Post) Studies With No Control Group	n = 10 [MMSE = 20.12 ± 1.13]	2.6 ± 0.51	7.1 ± 4.75	PS	0.35 ± 3.55	Wall goniometer (degrees), 3D gait analysis; EMG External Obliquo muscles (EO) and paravertebral muscles. BBS, Camptocormia Questionnaire (CQ), ten-meter walking test, TUG, 6MWT, physiological Cost Index.Pain and cognitive status n.r.	5d/wk for 2 wks	1° week: lidocaine daily injection (50 mg) in EO muscles + a rehabilitative program [stretching, truncal extension, postural control and balance]0.2° week: only rehabilitation program.	Significant reduction LTB after treatment [-8°] and at FUP [-7°].	15 days	Poor - 4
Furusawa et al. 2012 [43] Case Series	N = 12All patients complained of stiffness and pain in the upper abdomen	3.6 ± 0.7	10.00 ± 7.7	Upper Camptocormia	n.r	Software-based program (degrees) Pain and cognitive status n.r.	4-5 days	Repeated lidocaine (50 mg 1 %) injections into the bilateral EO for 4-5 days + trunk extension exercises	Single injection: 8/12 patients improved [-8°] and then subsided. Repeated injection: 9/12 patients [-13°] improvement at 90-days.	Up to 3 months	Good -4
Sakai et al. 2023 [105] Case Series	n = 9 Cognitive status n.r.	3.4 ± 0.5	17.2 ± 7.0	Camptocormia	n.r	Software-based program (degrees) BBS; maximum walking speed; stride lengthPain and cognitive status n.r.	5d/wk, 2 wks	Lidocaine injection into the abdominal external oblique muscles for 5 days in combination with physical therapy for 2 weeks.	A significant reduction in the Total CC [-14.5°], and Upper CC [-7.3°]	n.a.	Fair - 4
c. NIBS & Rehab											
De Icco et al. 2022 [98] RCT	EG, n = 13 Patients with pain (53.8 %) NRS = 3.5 ± 3.4 CG, n = 15 Patients with pain (53.3 %) NRS = 3.1 ± 3.3 Patients' exclusion criteria MMSE ≤24	2-3	EG = 8.7 ± 5.8 CG = 9.8 ± 8.8	PS	EG = 2.8 ± 2.2CG = 3.1 ± 1.7	Kinematic analysis of trunk by optoelectronic system UPDRS-III, FIM, NRS lumbar painCognitive status n.r.	5 d/w for 4 wks	EG: Rehabilitation + real tDCS (bi-hemispheric stimulation over M1, 20 min, 2 mA) for 5 daily sessionsCG: Rehabilitation + sham tDCS (bi-hemispheric stimulation over M1, 20 min, 2 mA) for 5 daily sessions.	The EG decreased the overall LTB decreased [-26.6 %], lateral trunk bending [-25.2 %] and increased the total range of trunk motion [-33.5 %] post-treatment. The overall LTB effects were maintained at FUP. The overall percentage of patients with pain did not change during the study. In the subgroups with pain, the pain improved more in the EG than the CG. The	6 months	Good - 1b

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Table 5 (continued)

Study	Study design;n. of patients; Cognitive status	H&Y	PD duration y	Type of PA	Duration of PA (y)	PA measurement; Study outcomes	Rehab Dose/duration	Type of protocol	Training Effects	FUP	QA-LE
									improvement on NRS was maintained at FUP only in the EG.		
d. Osteopathic Zarucchi et al. 2020 [99] RCT	EG, n = 12 [MMSE = 27.6 ± 3.21] CG, n = 12 [MMSE = 27.5 ± 2.48]	EG = 2.62 ± 0.31 CG = 2.58 ± 0.29	n.a.	PS	EG = 0.68 ± 0.35 CG = 0.83 ± 0.5	Software-based program ATF (degrees), UPDRS, mH&Y, 6MWT, Pain and cognitive status n.r.	1 session/wk; 4 wks	EG: OMT + multidisciplinary Intensive Rehabilitation treatment CG: sham OMT + multidisciplinary Intensive Rehabilitation treatment	The EG decreased the LTB by -3.33° post-treatment.	n.a.	Fair -2b
d. electroacupuncture Lu et al. 2022 [101] Case report	n = 1 Cognitive status n.r.	2.5	4	PS	0.5	Cobb angle (full spine X-ray) Cognitive status n.r.	3d/wk, 4 wks	Electropuncture from the 12 thoracic to the fifth lumbar vertebra	The Cobb angle decreased from 18.14° to 13.41°. After treatment the back pain and tightness decreased.	1 month	NA
e. galvanic stimulation Okada et al. 2015 [100] RCT crossover	EG, n = 4 CG, n = 3 Cognitive status n.r.	3-4	11.28 ± 4.34	ATF	3.11 ± 1.90	Software Digital videocamera (degrees) UPDRS gait subscore Pain and cognitive status n.r.	1 session	EG: real binaural monopolar Galvanic vestibular stimulation (GVS) (20 min) CG: sham binaural monopolar Galvanic vestibular stimulation (GVS) (20 min)	The EG decreased the ATF after GVS in both eyes open [-5.5°] and closed [-8.2°] more than the CG eyes open [-4.4°] and closed [-4.2°].	n.a.	Fair -2b
g. prismatic lens Meglio et al. 2021 [104] Before-After (Pre-Post) Studies With No Control Group	n = 9 VAS = 5.8 ± 2.9	2-3	8.8 ± 3.58	PS	n.a.	Wall goniometer (degrees) VAS for pain Cognitive status n.r.	3 months	Prismatic lenses	Slight not significant improvement in LTB (about 19.5%). A significant reduction of pain over time by 18.6%.	1 month	Poor -4

Legend: RCT, Randomized Controlled Trial; Obs, observational, EG, experimental group; CG, control group; wks, weeks; FUP, Follow-up; °, degrees; PD, Parkinson's Disease; PA, postural abnormalities; CC, camptocormia; AC, antecollis; PS, Pisa Syndrome; No., number; H&Y, Hohen and Yahr; UPDRS, Unified Parkinson's Disease Rating Scale; MMSE, Mini Mental State Examination; MoCA, Montreal Cognitive Assessment; BBS, Berg Balance Scale; TUG, Time UP and Go; BAK, Body analysis Kapture system; FES, Falls Efficacy Scale; PDQ-39, The Parkinson's Disease Questionnaire; ABC, Activities-Specific Balance Confidence Scale; T1, posttreatment; ADL, Activities of Daily Living; m, months; y, year; GA, hyperactivity ipsilateral to the bending side; GB, hyperactivity contralateral to the bending side; VAS, Visual Analogue Scale; FIM, Functional Independence Measure; EMG, Electromyography; BTX, Botulinum Toxin; 6MWT, 6 Minute Walking Test; NRS, Numerical Rating Scale; tDCS, transcranial Direct Current Stimulation; mA, milli Ampere; M1, primary motor area; OMT, Osteopathic Manipulative Treatment; n.r., not reported; NA, not applicable; ATF, Anterior trunk flexion; LTF, lateral trunk flexion; FTF, Forward trunk flexion; *, muscular hyperactivity ipsilateral to the trunk bending side; **, muscular hyperactivity contralateral to the trunk bending side; PR, proprioceptive and tactile stimulation, combined with stretching and postural reeducation; KT, Kinesio Taping; QA, Quality of Assessment; LE, Levels of Evidence.

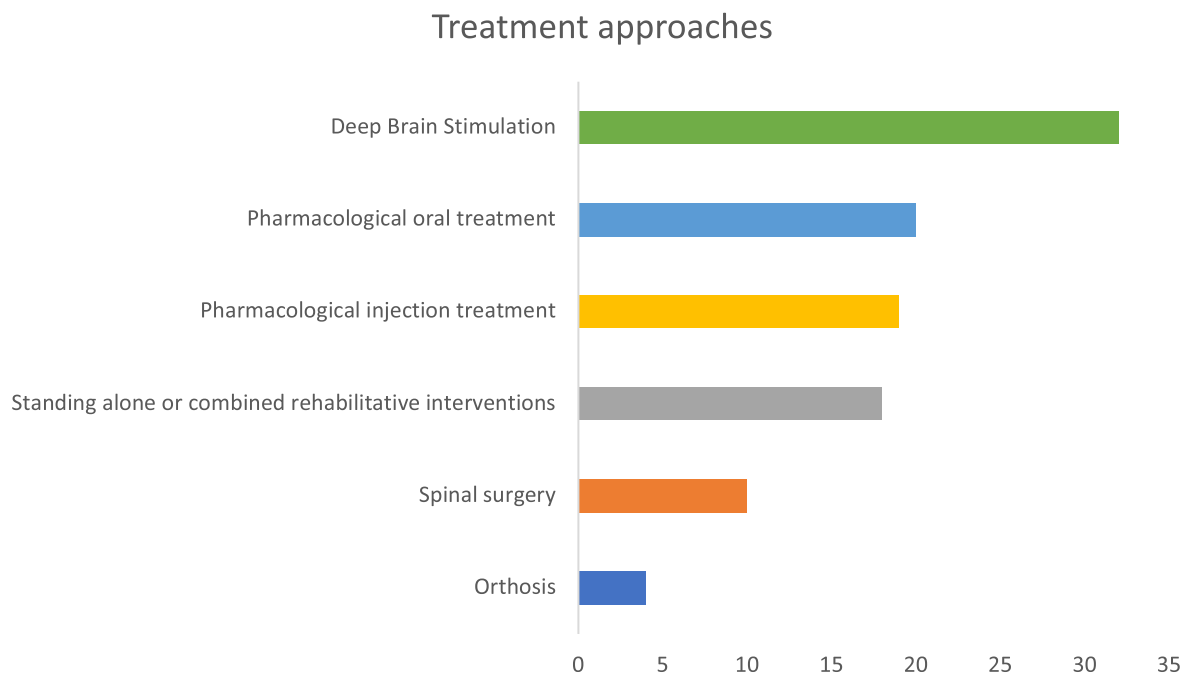


Fig. 2. An overview of the selected studies is presented, divided by intervention category.

antecollis, are needed.

4.4. Spinal surgery

The surgical approach for the management of axial postural abnormalities is arduous in people with PD. The complexity of surgery is mostly linked to the presence of a high rate of perioperative complications, complex drug interactions (anesthetic and levodopa), and the presence of comorbidities [118]. The perioperative complications may include implant pull-out and/or failure, onset of infections, development of proximal junctional kyphosis, pain, increased risk of bleeding, and post-operative aspiration and respiratory failure related to PD-associated upper airway dysfunction [118]. The drug interaction between levodopa and halothane may generate cardiac arrhythmia in PD [118]. Finally, comorbidity, like osteoporosis, strongly associated with PD, may further complicate the surgical intervention with additional surgical complications and revisions. The results of these studies showed, to some extent, an improvement of posture and related pain. However, the surgery revision rate was high in all studies. Therefore, a definite conclusion on the efficacy of spine surgery should be drawn with caution, given the scant literature on this topic and the poor outcome described in the available literature. Schroeder et al. [90] suggested an experience-based treatment algorithm for the lumbar spine in patients with PD presenting spine diseases other than PA (i.e., spinal stenosis). However, data in patients with PD and PA are unsatisfactory because of the small sample size, the lack of a standard clinical outcome measurement, which renders difficult the studies comparison, and the risk of the need for a second surgery. In conclusion, surgery options can be proper for very selected patients, who need to be properly counseled regarding the increased risk of operative complications and closely followed for incipient failure [118].

4.5. Standing alone or combined rehabilitative interventions

The development of specific rehabilitative approaches for PA in PD has been strongly influenced by the difficulties in understanding the pathophysiology of these disorders. However, the literature shows that as our knowledge has improved, there has been a progressive interest in this field of neurorehabilitation. This interest was initially represented

by the description of individual cases or observational studies and has gradually progressed in the last decade to the implementation of methodologically sound randomized controlled trials. Interestingly, the attention towards the rehabilitation of PA has been particularly lively in the European context, specifically in Italy, with the publication of rehabilitative interventions conducted alone or combined with pharmacological approaches [41,95–98,102]. In summary, active movements are more effective than passive ones in determining changes in trunk posture [41,95–98,102]. Based on this theoretical framework, rehabilitation of pathological forward trunk flexion should be focused on stabilization to maintain unconscious self-correction and trunk stabilization during the activities of daily living [97]. The most promising rehabilitative approaches are improving sensorimotor integration processes and enhancing feedforward and cognitive control of postural control (dual tasking) [95–97]. These objectives can also be achieved through water-based treatments, harnessing the benefits of a micro-gravity context on sensorimotor integration processes and muscle tone modulation [96]. In our opinion, biomechanical approaches (i.e., lumbar supports, high-level walking devices, manipulative physiotherapy) can be used as adjuvants in patients for whom such approaches can improve non-motor symptoms such as pain or fear of falling and can provide the patient with improved mobility and overall motor activity. Therefore, these approaches could support the benefits of treatments based on the disorder's pathophysiology rather than being a specific treatment for PAs. To date, there is still scant evidence of treating AC with rehabilitation. Only one study reported a positive outcome on neck flexion after water-based rehabilitation [96]. PA often manifests as a significant challenge for patients with Parkinson's disease and clinicians, and pain becomes an intricate aspect of their experience. The progressive nature of PA in PD can lead to abnormal stress on joints, muscles, and ligaments, triggering pain. Our systematic review supports significant insights into the complex relationship between PA and pain in PD. The rehabilitation interventions, which likely targeted musculo-skeletal imbalances and postural deficits, appear to impact alleviating pain. This correlation suggests that addressing PA through rehabilitation not only improves postural control but also has a positive effect on the associated pain. By establishing this connection, our review underscores the importance of adopting comprehensive strategies targeting postural management and pain reduction, ultimately improving the quality of life

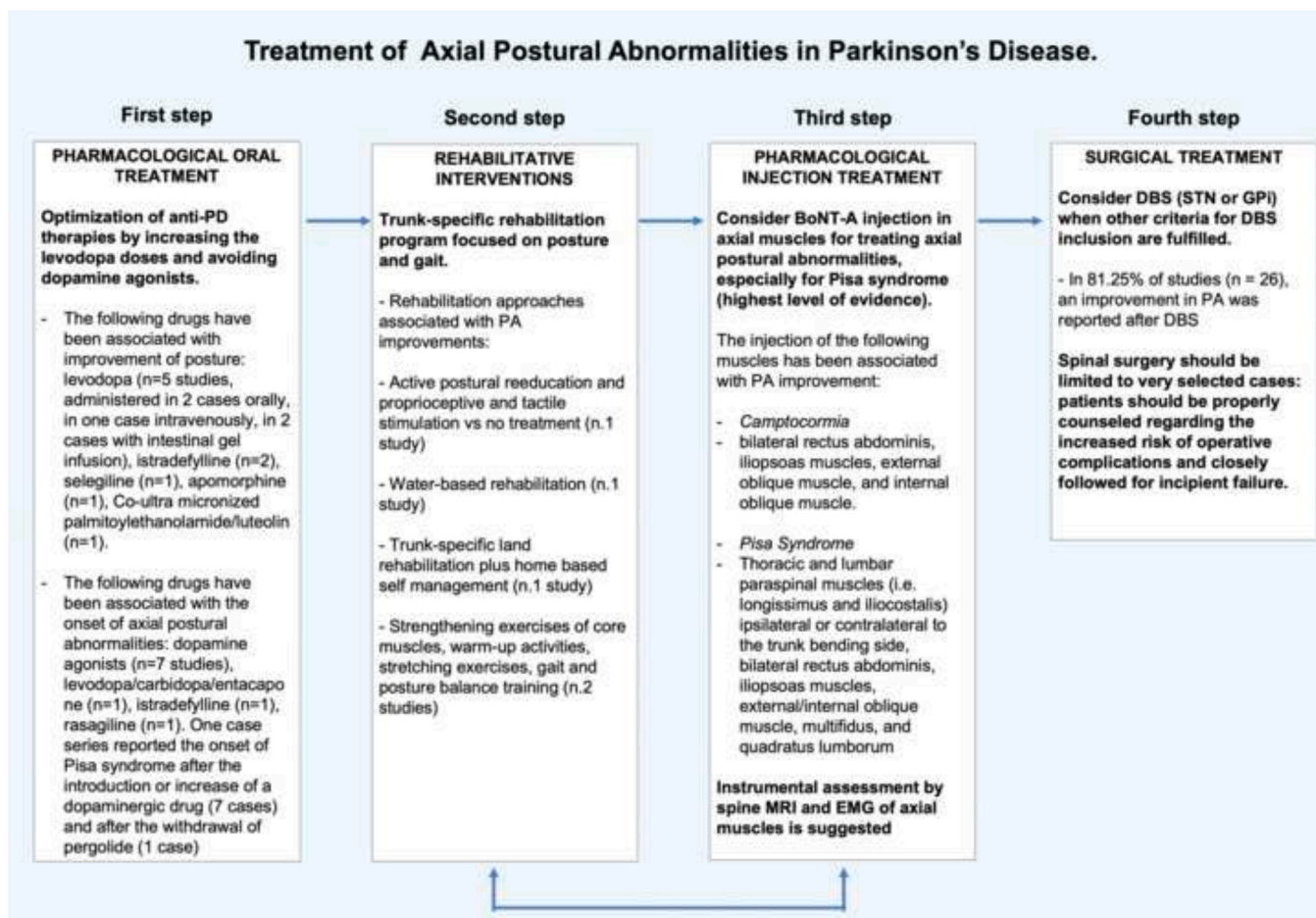


Fig. 3. An algorithmic approach to the management of PA in PD.

in patients with PD. More high-quality research studies with longer follow-ups are warranted to support the application of exercise and other strategies in managing different postural abnormalities.

The primary limitation of this systematic review is the broad period, which could affect the quality of reported interventions. Additionally, coexisting disorders in the patient samples might introduce variability. The considerable heterogeneity in the outcomes used to assess PA treatment effects further constrains the feasibility of a *meta*-analytic approach to evaluate therapeutic impacts.

5. Conclusions

PA are prevalent complications in PD, and their management remains challenging due to their elusive nature. This systematic review highlights the need for dedicated research and clinical focus on addressing PA in PD.

According to our results, we can conclude that the multifactorial nature of PD-associated PA necessitates an interdisciplinary approach involving neurologists, physical medicine and rehabilitation medical doctors, physiotherapists, and other healthcare professionals, who share common, consensus-based assessment and diagnostic criteria which will facilitate accurate evaluation and personalized treatment plans. Indeed, a range of interventions, including medical therapy, physiotherapy, botulinum toxin injections, and deep brain stimulation, offer potential benefits in managing PA. However, the optimal treatment approach should be determined on a case-by-case basis, considering individual patient characteristics, disease stage, symptom severity, and type and severity of PA.

Without standardized guidelines for managing PA, a tailored and

multidisciplinary approach should be adopted from the early phases of the disease. This approach should encompass comprehensive evaluations, regular follow-up, and adjustments to treatment plans to address evolving needs and optimize functional outcomes for patients with PD to manage or possibly prevent PAs.

CRedit authorship contribution statement

Marialuisa Gandolfi: Methodology, Data curation, Formal analysis, Writing – original draft, Writing – review & editing. **Christian Geroin:** Methodology, Data curation, Formal analysis, Writing – original draft, Writing – review & editing. **Gabriele Imbalzano:** Data curation, Formal analysis, Writing – original draft, Writing – review & editing. **Serena Camozzi:** Data curation, Formal analysis, Writing – original draft, Writing – review & editing. **Zoe Menaspà:** Data curation, Formal analysis, Writing – original draft, Writing – review & editing. **Michele Tinazzi:** Conceptualization, Methodology, Writing – review & editing. **Carlo Alberto Artusi:** Methodology, Data curation, Formal analysis, Writing – original draft, Writing – review & editing.

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.

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Appendix A. Supplementary data

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References

- [1] M. Tinazzi, M. Gandolfi, R. Ceravolo, et al., Postural abnormalities in Parkinson's disease: an epidemiological and clinical multicenter study, *Mov Disord Clin Pract.* 6 (7) (2019) 576–585, <https://doi.org/10.1002/mdc3.12810>.
- [2] K.M. Doherty, L. Silveira-Moriyama, B.P. van de Warrenburg, et al., Postural deformities in Parkinson's disease, *Lancet Neurol.* 10 (2011) 538–587, <https://doi.org/10.1016/S1474>.
- [3] C. Geroin, C.A. Artusi, M. Gandolfi, et al., Does the degree of trunk bending predict patient disability, motor impairment, falls, and Back pain in Parkinson's disease? *Front Neurol.* (2020) 11, <https://doi.org/10.3389/fneur.2020.00207>.
- [4] C. Geroin, C.A. Artusi, J. Nonnekes, et al., Axial postural abnormalities in Parkinsonism: gaps in predictors, pathophysiology, and Management, *Movement Disorders.* Published Online (2023), <https://doi.org/10.1002/mds.29377>.
- [5] M. Tinazzi, C. Geroin, R. Bhidayasiri, et al., Task force consensus on nosology and cut-off values for axial postural abnormalities in Parkinsonism, *Mov Disord Clin Pract.* 9 (5) (2022) 594–603, <https://doi.org/10.1002/mdc3.13460>.
- [6] M. Tinazzi, A. Fasano, C. Geroin, et al., Pisa syndrome in Parkinson disease, *Neurology.* 85 (20) (2015) 1769–1779.
- [7] S. Cao, Y. Cui, J. Jin, F. Li, X. Liu, T. Feng, Prevalence of axial postural abnormalities and their subtypes in Parkinson's disease: a systematic review and meta-analysis, *J Neurol.* 270 (1) (2023) 139–151, <https://doi.org/10.1007/s00415-022-11354-x>.
- [8] M. Gandolfi, C. Geroin, M. Gandolfi, I. Maddalena, N. Smania, M. Tinazzi, Do upper and lower camptocormias affect gait and postural control in patients with Parkinson's disease? an observational cross-sectional study, *Parkinsons Dis.* Published Online (2019), <https://doi.org/10.1155/2019/9026890>.
- [9] C. Geroin, N. Smania, F. Schena, et al., Does the Pisa syndrome affect postural control, balance, and gait in patients with Parkinson's disease? an observational cross-sectional study, *Parkinsonism Relat Disord.* 21 (7) (2015) 736–741, <https://doi.org/10.1016/j.parkreldis.2015.04.020>.
- [10] C. Pongmal, M. Fabbri, M. Zibetti, et al., Gait and axial postural abnormalities correlations in Parkinson's disease: a multicenter quantitative study, *Parkinsonism Relat Disord.* 105 (2022) 19–23, <https://doi.org/10.1016/j.parkreldis.2022.10.026>.
- [11] D. Moher, A. Liberati, J. Tetzlaff, et al., Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement, *PLoS Med.* 6 (7) (2009), <https://doi.org/10.1371/journal.pmed.1000097>.
- [12] OCEBM Levels of Evidence — Centre for Evidence-Based Medicine (CEBM), University of Oxford. Accessed December 5, 2022. <https://www.cebm.ox.ac.uk/resources/levels-of-evidence/ocebml-levels-of-evidence>.
- [13] Y. Yasuda, Reversible istradefylline-induced pleurothotonus in a patient with Parkinson's disease: a case report and literature review, *eNeurologicalSci.* 13 (2018) 5–7, <https://doi.org/10.1016/j.ensci.2018.09.001>.
- [14] T. Mano, Camptocormia induced by a dopaminergic agonist, *Clin Neuropharmacol.* 41 (2) (2018) 70–72, <https://doi.org/10.1097/WNF.0000000000000266>.
- [15] H. Kataoka, Y. Sawada, T. Namizaki, N. Shimozato, H. Yoshiji, S. Ueno, Intrajejunal infusion of levodopa-carbidopa gel can continuously reduce the severity of dropped head in parkinson's disease, *Front Neurol.* (2017) 8(OCT), <https://doi.org/10.3389/fneur.2017.00547>.
- [16] S. Galati, J.C. Möller, C. Städler, Ropinirole-induced pisa syndrome in parkinson disease, *Clin Neuropharmacol.* 37 (2) (2014) 58–59, <https://doi.org/10.1097/WNF.0000000000000022>.
- [17] H.J. Kim, B.S. Jeon, S.H. Kim, S.H. Han, Reversible antecollis associated with pramipexole in a patient with Parkinson's disease, *Journal of Clinical Neuroscience.* 19 (6) (2012) 903–904, <https://doi.org/10.1016/j.jocn.2011.09.015>.
- [18] P. Solla, A. Cannas, S. Congia, et al., Levodopa/carbidopa/entacapone-induced acute Pisa syndrome in a Parkinson's disease patient, *J Neurol Sci.* 275 (1–2) (2008) 154–156, <https://doi.org/10.1016/j.jns.2008.07.017>.
- [19] M. Suzuki, T. Hirai, Y. Ito, et al., Pramipexole-induced antecollis in Parkinson's disease, *J Neurol Sci.* 264 (1–2) (2008) 195–197, <https://doi.org/10.1016/j.jns.2007.08.008>.
- [20] B. Ho, R. Prakash, J.C. Morgan, K.D. Sethi, A case of levodopa-responsive camptocormia associated with advanced Parkinson's disease, *Nat Clin Pract Neurol.* 3 (9) (2007) 526–530, <https://doi.org/10.1038/ncpneu0584>.
- [21] A. Cannas, P. Solla, G. Floris, G. Borghero, P. Tacconi, A. Spissu, Reversible Pisa syndrome in Parkinson's disease during treatment with pergolide: a case report [1], *Clin Neuropharmacol.* 28 (5) (2005) 252, <https://doi.org/10.1097/01.wnf.0000183449.04271.05>.
- [22] Brotini S. Palmitoylethanolamide/Luteolin as Adjuvant Therapy to Improve an Unusual Case of Camptocormia in a Patient with Parkinson's Disease: A Case Report - PubMed. Accessed June 30, 2023. <https://pubmed.ncbi.nlm.nih.gov/35096476/>.
- [23] M. Takahashi, T. Shimokawa, J. Koh, et al., Efficacy and safety of istradefylline in patients with Parkinson's disease presenting with postural abnormalities: results from a multicenter, prospective, and open-label exploratory study in Japan, *J Neurol Sci.* (2022) 432, <https://doi.org/10.1016/j.jns.2021.120078>.
- [24] H. Kataoka, S. Ueno, Can postural abnormality really respond to levodopa in Parkinson's disease? *J Neurol Sci.* 377 (2017) 179–184, <https://doi.org/10.1016/j.jns.2017.04.025>.
- [25] A. Yoritaka, H. Mori, N. Hattori, Treatment of mild camptocormia with selegiline in patients with Parkinson's disease, *Eur Neurol.* 76 (1–2) (2016) 35–39, <https://doi.org/10.1159/000447631>.
- [26] K. Mensikova, M. Kaiserova, M. Vastik, S. Kurcova, P. Kanovsky, Treatment of camptocormia with continuous subcutaneous infusions of apomorphine: 1-year prospective pilot study, *J Neural Transm.* 122 (6) (2015) 835–839, <https://doi.org/10.1007/s00702-014-1297-9>.
- [27] F. Benninger, A. Khlebvtovsky, Y. Roditi, et al., Beneficial effect of levodopa therapy on stooped posture in Parkinson's disease, *Gait Posture.* 42 (3) (2015) 263–268, <https://doi.org/10.1016/j.gaitpost.2015.05.015>.
- [28] H. Morales-Briceno, N. Mahant, S. Duma, et al., Levodopa/carbidopa intestinal gel infusion can improve camptocormia in Parkinson's disease, *Parkinsonism Relat Disord.* 65 (2019) 282–283, <https://doi.org/10.1016/j.parkreldis.2019.06.010>.
- [29] S. Fujiyoka, R. Yoshida, K. Nose, et al., A new therapeutic strategy with istradefylline for postural deformities in Parkinson's disease, *Neurol Neurochir Pol.* 53 (4) (2019) 291–295, <https://doi.org/10.5603/PJNNS.A2019.0036>.
- [30] A. Cannas, P. Solla, G. Floris, et al., Reversible Pisa syndrome in patients with Parkinson's disease on dopaminergic therapy, *J Neurol.* 256 (3) (2009) 390–395, <https://doi.org/10.1007/s00415-009-0072-6>.
- [31] A. Fasano, M.A. Di, C. Vitale, et al., Reversible Pisa syndrome in patients with Parkinson's disease on rasagiline therapy, *Movement Disorders.* 26 (14) (2011) 2578–2580, <https://doi.org/10.1002/mds.23918>.
- [32] L. Ameghino, V. Bruno, M. Merello, Postural disorders and antiParkinsonian treatments in Parkinson disease: an exploratory case-control study, *Clin Neuropharmacol.* 41 (4) (2018) 123–128, <https://doi.org/10.1097/WNF.0000000000000285>.
- [33] R. von Coelln, A. Raible, T. Gasser, F. Asmus, Ultrasound-guided injection of the iliopsoas muscle with botulinum toxin in camptocormia, *Movement Disorders.* 23 (6) (2008) 889–892, <https://doi.org/10.1002/mds.21967>.
- [34] S.N. Azher, J. Jankovic, Camptocormia: pathogenesis, classification, and response to therapy, *Neurology.* 65 (3) (2005) 355–359, <https://doi.org/10.1212/01.wnl.0000171857.09079.9f>.
- [35] V.A. Bruno, S.H. Fox, D. Mancini, J.M. Miyasaki, Botulinum toxin use in Refractory pain and other symptoms in Parkinsonism, *Canadian Journal of Neurological Sciences.* 43 (5) (2016) 697–702, <https://doi.org/10.1017/cjn.2016.279>.
- [36] C. Colosimo, F.M. Salvatori, Injection of the iliopsoas muscle with botulinum toxin in camptocormia, *Movement Disorders.* 24 (2) (2009) 316–317, <https://doi.org/10.1002/mds.22249>.
- [37] H. Matsumoto, T. Akahori, K. Hatano, H. Hashida, Botulinum toxin treatment of paraspinal muscles for improving abnormal posture in Parkinson's disease, *Basal Ganglia.* 12 (2018) 1–3, <https://doi.org/10.1016/j.baga.2018.02.002>.
- [38] H. Todo, H. Yamasaki, G. Ogawa, K. Nishida, N. Futamura, I. Funakawa, Injection of onabotulinum toxin a into the bilateral external oblique muscle attenuated camptocormia: a prospective open-label study in six patients with Parkinson's disease, *Neurol Ther.* 7 (2) (2018) 365–371, <https://doi.org/10.1007/s40120-018-0108-x>.
- [39] S. Wijemanne, J. Jimenez-Shahed, Improvement in dystonic camptocormia following botulinum toxin injection to the external oblique muscle, *Parkinsonism Relat Disord.* 20 (10) (2014) 1106–1107, <https://doi.org/10.1016/j.parkreldis.2014.06.002>.
- [40] U.M. Fietzek, F.E. Schroeteler, A.O. Ceballos-Baumann, Goal attainment after treatment of parkinsonian camptocormia with botulinum toxin, *Movement Disorders.* 24 (13) (2009) 2027–2028, <https://doi.org/10.1002/mds.22676>.
- [41] C. Tassorelli, R. De Icco, E. Alfonsi, et al., Botulinum toxin type a potentiates the effect of neuromotor rehabilitation of Pisa syndrome in Parkinson disease: a placebo controlled study, *Parkinsonism Relat Disord.* 20 (11) (2014) 1140–1144, <https://doi.org/10.1016/j.parkreldis.2014.07.015>.
- [42] Y. Furusawa, Y. Mukai, T. Kawazoe, et al., Long-term effect of repeated lidocaine injections into the external oblique for upper camptocormia in Parkinson's disease, *Parkinsonism Relat Disord.* 19 (3) (2013) 350–354, <https://doi.org/10.1016/j.parkreldis.2012.09.008>.
- [43] Y. Furusawa, Y. Mukai, Y. Kobayashi, T. Sakamoto, M. Murata, Role of the external oblique muscle in upper camptocormia for patients with Parkinson's disease, *Movement Disorders.* 27 (6) (2012) 802–803, <https://doi.org/10.1002/mds.24930>.
- [44] L. Bonanni, A. Thomas, S. Varanese, V. Scorrano, M. Onofri, Botulinum toxin treatment of lateral axial dystonia in Parkinsonism, *Movement Disorders.* 22 (14) (2007) 2097–2103, <https://doi.org/10.1002/mds.21694>.
- [45] A. Santamato, M. Ranieri, F. Panza, et al., Botulinum toxin type a and a rehabilitation program in the treatment of Pisa syndrome in Parkinson's disease, *J Neurol.* 257 (1) (2010) 139–141, <https://doi.org/10.1007/s00415-009-5310-4>.

- [46] C.A. Artusi, S. Bortolani, A. Merola, et al., Botulinum toxin for Pisa syndrome: an MRI-, ultrasound- and electromyography-guided pilot study, *Parkinsonism Relat Disord.* 62 (2019) 231–235, <https://doi.org/10.1016/j.parkreldis.2018.11.003>.
- [47] A. Dupeyron, E. Viollet, F. Corioian, C. Gagnard, D. Renard, G. Castelnuovo, Botulinum toxin-a for treatment of Pisa syndrome: a new target muscle, *Parkinsonism Relat Disord.* 21 (6) (2015) 669–670, <https://doi.org/10.1016/j.parkreldis.2015.03.027>.
- [48] B.P.C. Van De Warrenburg, C. Cordivari, A.M. Ryan, et al., The phenomenon of disproportionate anterocollis in Parkinson's disease and multiple system atrophy, *Movement Disorders.* 22 (16) (2007) 2325–2331, <https://doi.org/10.1002/mds.21634>.
- [49] G. Yahalom, E. Atlan, A. Janah, et al., The downside of botulinum injections for anterocollis: a case series and a review of the literature, *Clin Neuropharmacol.* 46 (3) (2023) 89–94, <https://doi.org/10.1097/WNF.0000000000000548>.
- [50] G. Oyama, A. Hayashi, Y. Mizuno, N. Hattori, Mechanism and treatment of dropped head syndrome associated with parkinsonism, *Parkinsonism Relat Disord.* 15 (3) (2009) 181–186, <https://doi.org/10.1016/j.parkreldis.2008.04.040>.
- [51] Y. Seliverstov, S. Arestov, S. Klyushnikov, Y. Shpilyukova, S. Illarioshkin, A methodological approach for botulinum neurotoxin injections to the longus colli muscle in dystonic anterocollis: a case series of 4 patients and a literature review, *Journal of Clinical Neuroscience.* 80 (2020) 188–194, <https://doi.org/10.1016/j.jocn.2020.08.025>.
- [52] Y. Lai, Y. Song, P. Huang, et al., Subthalamic stimulation for camptocormia in Parkinson's disease: association of volume of tissue activated and structural connectivity with clinical effectiveness, *J Parkinsons Dis.* 11 (1) (2021) 199–210, <https://doi.org/10.3233/JPD-202259>.
- [53] Y. Lai, Y. Song, D. Su, et al., Pallidal stimulation as treatment for camptocormia in Parkinson's disease, *NPJ Parkinsons Dis.* 7 (1) (2021), <https://doi.org/10.1038/s41531-020-00151-w>.
- [54] J. He, Z. Cui, S. Li, H. Chen, W. Su, Effect of bilateral subthalamic nucleus deep brain stimulation on Pisa syndrome in Parkinson's disease, *Front Neurol.* 12 (2021), <https://doi.org/10.3389/fneur.2021.739298>.
- [55] J. Roediger, C.A. Artusi, A. Romagnolo, et al., Effect of subthalamic deep brain stimulation on posture in Parkinson's disease: a blind computerized analysis, *Parkinsonism Relat Disord.* 62 (2019) 122–127, <https://doi.org/10.1016/j.parkreldis.2019.01.003>.
- [56] C. Schlenstedt, O. Gavriliuc, K. Boße, et al., The effect of medication and deep brain stimulation on posture in Parkinson's disease, *Front Neurol.* 10 (2019), <https://doi.org/10.3389/fneur.2019.01254>.
- [57] M. Okazaki, T. Sasaki, T. Yasuhara, et al., Characteristics and prognostic factors of Parkinson's disease patients with abnormal postures subjected to subthalamic nucleus deep brain stimulation, *Parkinsonism Relat Disord.* 57 (2018) 44–49, <https://doi.org/10.1016/j.parkreldis.2018.07.014>.
- [58] C.A. Artusi, M. Zibetti, A. Romagnolo, M.G. Rizzone, A. Merola, L. Lopiano, Subthalamic deep brain stimulation and trunk posture in Parkinson's disease, *Acta Neurol Scand.* 137 (5) (2018) 481–487, <https://doi.org/10.1111/ane.12889>.
- [59] W. Sakai, S. Nakane, E. Urasaki, et al., The cross-sectional area of paraspinal muscles predicts the efficacy of deep drain stimulation for camptocormia, *J Parkinsons Dis.* 7 (2) (2017) 247–253, <https://doi.org/10.3233/JPD-160948>.
- [60] K. Yamada, N. Shinjima, T. Hamasaki, J.I. Kuratsu, Subthalamic nucleus stimulation improves Parkinson's disease-associated camptocormia in parallel to its preoperative levodopa responsiveness, *J Neurol Neurosurg Psychiatry.* 87 (7) (2016) 703–709, <https://doi.org/10.1136/jnnp-2015-310926>.
- [61] W.J. Schulz-Schaeffer, N.G. Margraf, S. Munser, et al., Effect of neurostimulation on camptocormia in Parkinson's disease depends on symptom duration, *Movement Disorders.* 30 (3) (2015) 368–372, <https://doi.org/10.1002/mds.26081>.
- [62] A. Umemura, Y. Oka, K. Ohkita, T. Yamawaki, K. Yamada, Effect of subthalamic deep brain stimulation on postural abnormality in Parkinson disease: clinical article, *J Neurosurg.* 112 (6) (2010) 1283–1288, <https://doi.org/10.3171/2009.10.JNS09917>.
- [63] S. Liang, Y. Yu, H. Li, Y. Wang, Y. Cheng, H. Yang, The study of subthalamic deep brain stimulation for parkinson disease-associated camptocormia, *Medical Science Monitor.* 26 (2020), <https://doi.org/10.12659/MSM.919682>.
- [64] J.R. Guerrero, L.S. Bhenderu, K.M. Taghlabi, J.G. Cruz-Garza, C. Saifi, A.H. Faraji, Improvement in sagittal alignment and mechanical low-back pain following deep brain stimulation for Parkinson's disease: illustrative case, *Journal of Neurosurgery: Case Lessons.* 4 (17) (2022), <https://doi.org/10.3171/case22357>.
- [65] B.L. Anderson, R. Ziehmann, X. Huang, J. McInerney, Long-term outcome of globus pallidus internus stimulation for Pisa syndrome, *Cureus.* 11 (1) (2019), <https://doi.org/10.7759/cureus.3838>.
- [66] S. Pandey, Upper camptocormia in Parkinson's disease reversed by bilateral subthalamic deep brain stimulation, *Parkinsonism Relat Disord.* 37 (2017) 123–124, <https://doi.org/10.1016/j.parkreldis.2017.02.013>.
- [67] H. Akiyama, S. Nukui, M. Akamatu, Y. Hasegawa, O. Nishikido, S. Inoue, Effectiveness of spinal cord stimulation for painful camptocormia with Pisa syndrome in Parkinson's disease: a case report, *BMC Neurol.* 17 (1) (2017), <https://doi.org/10.1186/s12883-017-0926-y>.
- [68] H. Ekmecki, H. Kaptan, Camptocormia and deep brain stimulation: the interesting overlapping etiologies and the therapeutic role of subthalamic nucleus-deep brain stimulation in Parkinson disease with camptocormia, *S107, Surg Neuro Int.* 7 (Suppl 4) (2016) S103, <https://doi.org/10.4103/2152-7806.176130>.
- [69] L. Ricciardi, C. Piano, A.R. Bentivoglio, A. Fasano, Long-term effects of pedunculopontine nucleus stimulation for Pisa syndrome, *Parkinsonism Relat Disord.* 20 (12) (2014) 1445–1446, <https://doi.org/10.1016/j.parkreldis.2014.10.006>.
- [70] A. Oliveira, J. Massano, M.J. Rosas, Dropped head syndrome in early-onset Parkinson disease treated with bilateral subthalamic stimulation: clinical, imaging, EMG, and biopsy findings, *Neurological Sciences.* 34 (4) (2013) 593–594, <https://doi.org/10.1007/s10072-012-1089-7>.
- [71] M. Lyons, O. Boucher, N. Patel, B. Birch, V. Evidente, Long-term benefit of bilateral subthalamic deep brain stimulation on camptocormia in parkinson's disease, *Turk Neurosurg.* 22 (4) (2012) 489–492, <https://doi.org/10.5137/1019-5149.JTN.3857-10.0>.
- [72] N.B. Thani, A. Bala, T.E. Kimber, C.R.P. Lind, High-frequency pallidal stimulation for camptocormia in parkinson disease: case report, *Neurosurgery.* 68 (5) (2011), <https://doi.org/10.1227/NEU.0b013e318210c859>.
- [73] E.A.C. Pereira, J. Wilson-MacDonald, A.L. Green, T.Z. Aziz, T.A.D. Cadoux-Hudson, Posterior occipitocervical instrumented fusion for dropped head syndrome after deep brain stimulation, *Journal of Clinical Neuroscience.* 17 (4) (2010) 541–542, <https://doi.org/10.1016/j.jocn.2009.07.114>.
- [74] M.A. Hellmann, R. Djaldetti, Z. Israel, E. Melamed, Effect of deep brain subthalamic stimulation on camptocormia and postural abnormalities in idiopathic Parkinson's disease, *Movement Disorders.* 21 (11) (2006) 2008–2010, <https://doi.org/10.1002/mds.21090>.
- [75] F. Micheli, M.G. Cersósimo, F. Piedimonte, Camptocormia in a patient with Parkinson disease: beneficial effects of pallidal deep brain stimulation: case report, *J Neurosurg.* 103 (6) (2005) 1081–1083, <https://doi.org/10.3171/jns.2005.103.6.1081>.
- [76] C. Soares, M. Vilas-Boas, do, C., Lopes, EM., et al., Automated and objective measures of gait dynamics in camptocormia Parkinson's disease subthalamic deep brain stimulation, *Clin Neurol Neurosurg.* 186 (2019), <https://doi.org/10.1016/j.clineuro.2019.105537>.
- [77] T. Asahi, Y. Taguchi, N. Hayashi, et al., Bilateral subthalamic deep brain stimulation for camptocormia associated with Parkinson's disease, *Stereotact Funct Neurosurg.* 89 (3) (2011) 173–177, <https://doi.org/10.1159/000324907>.
- [78] H.H. Capelle, C. Schrader, C. Blahak, et al., Deep brain stimulation for camptocormia in dystonia and Parkinson's disease, *J Neurol.* 258 (1) (2011) 96–103, <https://doi.org/10.1007/s00415-010-5695-0>.
- [79] C.D. Upadhyaya, P.A. Starr, P.V. Mummaneni, Spinal deformity and Parkinson disease: a treatment algorithm, *Neurosurg Focus.* 28 (3) (2010) 1–7, <https://doi.org/10.3171/2010.1.FOCUS09288>.
- [80] W. Sako, M. Nishio, T. Maruo, et al., Subthalamic nucleus deep brain stimulation for camptocormia associated with Parkinson's disease, *Movement Disorders.* 24 (7) (2009) 1076–1079, <https://doi.org/10.1002/mds.22529>.
- [81] W.R. Schäbitz, K. Glatz, C. Schuhan, et al., Severe forward flexion of the trunk in parkinson's disease: focal myopathy of the paraspinal muscles mimicking camptocormia, *Movement Disorders.* 18 (4) (2003) 408–414, <https://doi.org/10.1002/mds.10385>.
- [82] N.G. Margraf, A. Wrede, A. Rohr, et al., Camptocormia in idiopathic Parkinson's disease: a focal myopathy of the paravertebral muscles, *Movement Disorders.* 25 (5) (2010) 542–551, <https://doi.org/10.1002/mds.22780>.
- [83] K. Yamada, S. Goto, K. Matsuzaki, et al., Alleviation of camptocormia by bilateral subthalamic nucleus stimulation in a patient with Parkinson's disease, *Parkinsonism Relat Disord.* 12 (6) (2006) 372–375, <https://doi.org/10.1016/j.parkreldis.2006.02.003>.
- [84] A.C. Peek, N. Quinn, A.T.H. Casey, G. Etherington, Thoracolumbar spinal fixation for camptocormia in Parkinson's disease, *J Neurol Neurosurg Psychiatry.* 80 (11) (2009) 1275–1278, <https://doi.org/10.1136/jnnp.2008.152736>.
- [85] P.M. Wadia, G. Tan, R.P. Munhoz, S.H. Fox, S.J. Lewis, A.E. Lang, Surgical correction of kyphosis in patients with camptocormia due to Parkinson's disease: a retrospective evaluation, *J Neurol Neurosurg Psychiatry.* 82 (4) (2011) 364–368, <https://doi.org/10.1136/jnnp.2009.176198>.
- [86] M. Sato, T. Sainoh, S. Orita, et al., Posterior and anterior spinal fusion for the Management of Deformities in patients with Parkinson's disease, *Case Rep Orthop.* 2013 (2013) 1–6, <https://doi.org/10.1155/2013/140916>.
- [87] G.L. Mei, H.T. Wei, Y.R. Ma, D. Wan, Surgical correction of kyphosis in patients with camptocormia associated with Parkinson's disease, *A Case Report and Review of the Literature.* *Front Surg.* 9 (2022), <https://doi.org/10.3389/fsurg.2022.822015>.
- [88] R.S. Nunna, R.G. Fessler, Minimally invasive scoliosis correction in parkinson disease: retrospective case series, *Operative Neurosurgery.* 19 (6) (2020) 635–640, <https://doi.org/10.1093/ons/opa187>.
- [89] Yamato Y, Hasegawa T, Togawa D, et al. Extensive Spinal Fusion Surgery in Patients with Parkinson Disease or Atypical Parkinsonism: Time Course of Clinical Outcomes in 5 Years Progress Report. *Spine (Phila Pa 1976).* 2020;45(4):E217-E226. doi:10.1097/BRS.0000000000003246.
- [90] J.E. Schroeder, A. Hughes, A. Sama, et al., Lumbar spine surgery in patients with Parkinson disease, *Journal of Bone and Joint Surgery - American Volume.* 97 (20) (2015) 1661–1666, <https://doi.org/10.2106/JBJS.N.01049>.
- [91] Kawaguchi Y, Nakano M, Seki S, et al. Surgical treatment of lumbar vertebral collapse in a patient with Parkinson's disease: A case report. *European Journal of Orthopaedic Surgery and Traumatology.* 2013;23(SUPPL. 2). doi:10.1007/s00590-012-1145-2.
- [92] H. Koller, F. Acosta, J. Zenner, et al., Spinal surgery in patients with Parkinson's disease: Experiences with the challenges posed by sagittal imbalance and the Parkinson's spine, *European Spine Journal.* 19 (10) (2010) 1785–1794, <https://doi.org/10.1007/s00586-010-1405-y>.

- [93] K. Farah, S. Prost, M. Meyer, et al., Surgery for spinal deformity in Parkinson's disease patients: what are we missing? *Neurochirurgie*. 68 (2) (2022) 183–187, <https://doi.org/10.1016/j.neuchi.2021.08.004>.
- [94] Y.R. Yang, Y.Y. Lee, S.J. Cheng, R.Y. Wang, Downhill walking training in individuals with parkinson's disease: a randomized controlled trial, *Am J Phys Med Rehabil*. 89 (9) (2010) 706–714, <https://doi.org/10.1097/PHM.0b013e3181e721c5>.
- [95] M. Capecchi, C. Serpicelli, L. Fiorentini, et al., Postural rehabilitation and kinesio taping for axial postural disorders in Parkinson's disease, *Arch Phys Med Rehabil*. 95 (6) (2014) 1067–1075, <https://doi.org/10.1016/j.apmr.2014.01.020>.
- [96] D. Volpe, M.G. Giantin, P. Manuela, et al., Water-based vs. non-water-based physiotherapy for rehabilitation of postural deformities in Parkinson's disease: a randomized controlled pilot study, *Clin Rehabil*. 31 (8) (2017) 1107–1115, <https://doi.org/10.1177/0269215516664122>.
- [97] M. Gandolfi, M. Tinazzi, F. Magrinelli, et al., Four-week trunk-specific exercise program decreases forward trunk flexion in Parkinson's disease: a single-blinded, randomized controlled trial, *Parkinsonism Relat Disord*. 64 (May) (2019) 268–274, <https://doi.org/10.1016/j.parkreldis.2019.05.006>.
- [98] R. De Icco, A. Putorti, M. Allena, et al., Non-invasive neuromodulation in the rehabilitation of Pisa syndrome in Parkinson's disease: a randomized controlled trial, *Front Neurol*. 13 (2022), <https://doi.org/10.3389/fneur.2022.849820>.
- [99] A. Zarucchi, L. Vismara, G. Frazzitta, et al., Efficacy of osteopathic manipulative treatment on postural control in Parkinsonian patients with Pisa syndrome: a pilot randomized placebo-controlled trial, *NeuroRehabilitation*. 46 (4) (2020) 529–537, <https://doi.org/10.3233/NRE-203068>.
- [100] Y. Okada, Y. Kita, J. Nakamura, et al., Galvanic vestibular stimulation may improve anterior bending posture in Parkinson's disease, *Neuroreport*. 26 (7) (2015) 405–410, <https://doi.org/10.1097/WNR.0000000000000360>.
- [101] W.J. Lu, J.Q. Fan, M.Y. Yan, K. Mukaeda, L.X. Zhuang, L.L. Wang, Effect of electroacupuncture for Pisa syndrome in Parkinson's disease: a case report, *World J Clin Cases*. 10 (30) (2022) 11023–11030, <https://doi.org/10.12998/wjcc.v10.i30.11023>.
- [102] M. Bartolo, M. Serrao, C. Tassorelli, et al., Four-week trunk-specific rehabilitation treatment improves lateral trunk flexion in Parkinson's disease, *Movement Disorders*. 25 (3) (2010) 325–331, <https://doi.org/10.1002/mds.23007>.
- [103] K.H. Lee, J.M. Kim, H.S. Kim, Back extensor strengthening exercise and backpack wearing treatment for camptocormia in Parkinson's disease: a retrospective pilot study, *Ann Rehabil Med*. 41 (4) (2017) 677–685, <https://doi.org/10.5535/arm.2017.41.4.677>.
- [104] M. Meglio, E. Olivola, M. Santilli, et al., Effects of prismatic lenses on lateral axial dystonia in Parkinson's disease: a pilot study, *Innov Clin Neurosci*. 18 (1–3) (2021) 39–42.
- [105] T. Sakai, S. Nagai, K. Takao, H. Tsuchiyama, K. Ikeda, Effect of intramuscular lidocaine injection with physical therapy on camptocormia in patients with Parkinson's disease who had previously had deep brain stimulation, *J Phys Ther Sci*. 35 (1) (2023) 66–69, <https://doi.org/10.1589/jpts.35.66>.
- [106] S. Di Martino, E. Unti, C. Tramonti, et al., Efficacy of a combined therapeutic approach in the management of Pisa syndrome, *NeuroRehabilitation*. 41 (1) (2017) 249–253, <https://doi.org/10.3233/NRE-171478>.
- [107] F. Lena, E. Iezzi, M. Etoom, et al., Effects of postural exercises in patients with Parkinson's disease and Pisa syndrome: a pilot study, *NeuroRehabilitation*. 41 (2) (2017) 423–428, <https://doi.org/10.3233/NRE-162033>.
- [108] Y. Kawami, K. Marumoto, Y. Shiomi, et al., The effect of comprehensive physiotherapy-based rehabilitation on stooped posture in Parkinson's disease, *J Phys Ther Sci*. 30 (12) (2018) 1440–1445, <https://doi.org/10.1589/jpts.30.1440>.
- [109] B.K. Ye, H.S. Kim, Y.W. Kim, Correction of camptocormia using a cruciform anterior spinal hyperextension brace and back extensor strengthening exercise in a patient with Parkinson disease, *Ann Rehabil Med*. 39 (1) (2015) 128–132, <https://doi.org/10.5535/arm.2015.39.1.128>.
- [110] F.E. Schroeteler, U.M. Fietzek, K. Ziegler, A.O. Ceballos-Baumann, Upright posture in parkinsonian camptocormia using a high-frame walker with forearm support, *Movement Disorders*. 26 (8) (2011) 1560–1561, <https://doi.org/10.1002/mds.23585>.
- [111] G. Jo, J. Park, J. Yoon, yeon,, Kim, H., Kim, SC., Park, SH., Effect of spinal kypho-orthosis to gait and forward flexion in Parkinson disease, *J Phys Ther Sci*. 30 (8) (2018) 988–992, <https://doi.org/10.1589/jpts.30.988>.
- [112] M.P. de Sèze, A. Creuzé, M. de Sèze, J.M. Mazaux, An orthosis and physiotherapy programme for camptocormia: a prospective case study, *J Rehabil Med*. 40 (9) (2008) 761–765, <https://doi.org/10.2340/16501977-0252>.
- [113] A. Fasano, C. Geroïn, A. Berardelli, et al., Diagnostic criteria for camptocormia in Parkinson's disease: a consensus-based proposal, *Parkinsonism Relat Disord*. 53 (2018) 53–57, <https://doi.org/10.1016/j.parkreldis.2018.04.033>.
- [115] M. Tinazzi, M. Gandolfi, C.A. Artusi, et al., Validity of the wall goniometer as a screening tool to detect postural abnormalities in Parkinson's disease, *Parkinsonism Relat Disord*. 69 (2019) 159–165, <https://doi.org/10.1016/j.parkreldis.2019.10.024>.
- [116] C.A. Artusi, E. Montanaro, S. Tuttobene, A. Romagnolo, M. Zibetti, L. Lopiano, Pisa syndrome in Parkinson's disease is associated with specific cognitive alterations, *Front Neurol*. 10 (MAY) (2019) 577, <https://doi.org/10.3389/fneur.2019.00577>.
- [117] W.H. Jost, toxins use of botulinum neurotoxin in Parkinson's disease: a critical appraisal, *Published Online* (2021), <https://doi.org/10.3390/toxins13020087>.
- [118] J.F. Baker, S. McClelland, R.A. Hart, R.S. Bess, Management of spinal conditions in patients with Parkinson disease, e165, *Journal of the American Academy of Orthopaedic Surgeons*. 25 (8) (2017) e157, <https://doi.org/10.5435/JAAOS-D-16-00627>.

Further reading

- [114] N.G. Margraf, R. Wolke, O. Granert, et al., Consensus for the measurement of the camptocormia angle in the standing patient, *Parkinsonism Relat Disord*. 52 (2018) 1–5, <https://doi.org/10.1016/j.parkreldis.2018.06.013>.