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Systematic Review

Effects of Non-Invasive Brain Stimulation on Ataxia Severity, Gait, and Balance in Patients with Cerebellar Ataxia: A Systematic Review and Meta-Analysis

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Highlights

What are the main findings?

- NIBS improves gait function but not balance or disease severity in patients with ataxia.

What are the implications of the main findings?

- tDCS may benefit gait rehabilitation, particularly in patients with spinocerebellar ataxia.
- NIBS effects may vary by disease subtype and stimulation modality, supporting personalized rehabilitation.

Abstract

Background: We aimed to systematically investigate the effects of non-invasive brain stimulation (NIBS) on ataxia severity, gait, and balance in patients with ataxia, and to synthesize current evidence regarding its therapeutic potential. **Methods:** This systematic review and meta-analysis included randomized controlled trials (RCTs) investigating the effects of non-invasive brain stimulation on disease severity, gait function, and balance outcomes in patients with ataxia. SARA and ICARS total scores were used to assess overall ataxia severity. Balance-related outcomes were analyzed separately. Ataxia severity was summarized using scale-specific mean differences. Standardized mean differences were used when different instruments assessed the same outcome construct. Data were extracted using a standardized form, and risk of bias was assessed using the original Cochrane risk-of-bias tool (RoB 1). The primary meta-analyses used random-effects models with restricted maximum likelihood estimation. Embase, Web of Science, Cochrane Library, PubMed, and Scopus were searched from inception to 23 July 2026. **Results:** The systematic review included 13 studies (332 participants); of these, 12 studies contributed to the pooled analysis of ataxia severity, 12 to gait, and 7 to balance. Pooled analyses showed that NIBS significantly reduced ataxia severity (SMD = −0.49, $p = 0.002$), with subgroup benefits observed following electrical stimulation (SMD = −0.29, $p = 0.01$) and in patients with genetic/spinocerebellar ataxia-related ataxia (SMD = −0.38, $p = 0.006$). A significant improvement was also observed in the Scale for the Assessment and Rating of Ataxia score (SMD = −1.39, $p = 0.006$), whereas other measures of ataxia severity showed no significant effects. NIBS also significantly improved gait function (SMD = −0.26, $p = 0.02$), particularly following electrical stimulation (SMD = −0.27, $p = 0.03$) and in patients

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with genetic/SCA-related ataxia (SMD = −0.31, $p = 0.04$). However, NIBS did not significantly improve balance outcomes (SMD = 0.19, $p = 0.25$). **Conclusions:** The findings suggested that NIBS may improve ataxia severity; however, its efficacy in improving balance impairment remains inconclusive. These results support the potential role of NIBS as an adjunctive intervention for selected patients with ataxia, although further high-quality studies are required to establish its clinical applicability.

Keywords: cerebellar ataxia; non-invasive brain stimulation; transcranial direct current stimulation; systematic review; meta-analysis; postural control

1. Introduction

Ataxia comprises a clinically and etiologically diverse spectrum of neurological conditions in which voluntary motor coordination is impaired, largely attributable to dysfunction of the cerebellum and its associated neural circuit [1]. Gait and postural deficits are prominent features [2] that frequently compromise independence and quality of life [3]. Among these deficits, gait and balance instability pose major clinical challenges, as they are associated with falls and reduced daily activity. Neurological deficits progressively worsen as the disease advances, and their severity can be objectively measured using the Scale for the Assessment and Rating of Ataxia (SARA) [4] and the International Cooperative Ataxia Rating Scale (ICARS) [5].

Current management of ataxia primarily focuses on symptom control and functional rehabilitation [1], incorporating pharmacotherapy, physiotherapy, and structured exercise [6]. Notwithstanding that these treatments may lead to some improvement in motor and daily functioning [7], their effects are largely compensatory and may have a limited capacity to directly modulate the underlying neural dysfunction associated with the disease. Treatment responses vary according to disease subtype, severity, and individual neuroplastic potential [3]. Therefore, directly targeting dysfunctional neural circuits may provide a promising approach to ameliorate ataxic motor impairment.

Non-invasive brain stimulation (NIBS) has been shown to facilitate neural recovery and reduce motor dysfunction through neuromodulation [8]. Several non-invasive stimulation strategies—including tDCS, tACS, rTMS, and iTBS—are known to influence the excitability of cortical circuits and associated neuronal populations, enhance synaptic plasticity, and regulate abnormal network activity [9]. NIBS may facilitate residual neural function and adaptive reorganization of cerebello-cortical networks [10]. This may, in turn, improve motor control in patients with ataxia [11].

Randomized controlled trials (RCTs) using various NIBS modalities, including cerebellartDCS, tACS, rTMS, and iTBS, with differing stimulation parameters have yielded inconsistent evidence regarding motor improvement in ataxia [11–15]. Outcome measures (gait, posture, balance, and severity) vary across studies, and despite the common use of SARA and ICARS [4,16], differences in outcome selection, methodology, and follow-up duration impede pooled effect estimation and the identification of responsive domains [5,14]. Small sample sizes reduce statistical power and limit clinical relevance [11,17]. Clinical heterogeneity across ataxia subtypes (hereditary, degenerative, and acquired) further hampers interpretation and generalizability [1]. Given this, the current systematic review with meta-analysis was designed to systematically examine whether NIBS improves motor function and reduces clinical severity among patients with ataxia, focusing on gait mobility, balance and postural control, and global disease severity.

2. Methods

2.1. Protocol and Registration

Ataxia severity and balance were prespecified in the original PROSPERO registration (CRD420261459897). Gait was added as an outcome domain after the initial gait analyses had been conducted. We report this addition as a deviation from the original protocol and interpret the gait findings in Section 3.4 as exploratory. The completed PRISMA 2020 checklist is provided in Supplementary Table S2.

Ataxia severity and balance were the outcome domains specified in the original PROSPERO registration. Gait was not a prespecified outcome and is therefore reported separately as an exploratory outcome in the Supplementary Material. Its inclusion represents a deviation from the registered protocol. Using the Cochrane Risk of Bias tool (RoB 1, 2011) [18], two reviewers independently evaluated the risk of bias in the eight included studies across seven domains. The timing and rationale for this assessment are documented in Supplementary Table S2. The main conclusions focus on the registered outcomes. During manuscript revision, the PROSPERO record was amended to include gait-related outcomes as a separate outcome domain. Because this amendment occurred after the initial gait analyses, these analyses are reported as non-prespecified and exploratory. Gait and balance findings are retained in the main text as separate outcome domains, and gait findings are interpreted as hypothesis-generating.

2.2. Search Strategy

We searched PubMed, Embase, Web of Science, the Cochrane Library, and Scopus from database inception up to 23 July 2026 for eligible English-language publications. The search strategies combined controlled vocabulary, where available, with free-text terms related to ataxia and non-invasive brain stimulation. The complete search strategies as executed in each database, together with the search platforms, database collections, search dates, field tags, Boolean operators, and any restrictions applied, are provided in Supplementary Table S1.

2.3. Inclusion and Exclusion Criteria

Studies were eligible if they included participants of any age with a confirmed diagnosis of ataxia, including hereditary, degenerative, idiopathic, or other forms. The intervention of interest was NIBS, including but not limited to tDCS. Eligible interventions included NIBS alone or in combination with rehabilitation or exercise training. Comparator conditions included sham stimulation, placebo stimulation, or no intervention. Only RCTs were included. Qualifying studies were mandated to include outcome assessments addressing balance, walking ability, or severity of ataxic symptoms. We excluded animal studies; studies involving healthy participants or populations without ataxia; invasive stimulation trials; studies lacking a control group or sufficient data to calculate between-group effects; and conference abstracts, non-randomized controlled trials, and reviews.

2.4. Literature Screening and Data Extraction

Following the literature search, all records were uploaded to EndNote 20, where duplicates were removed. Two reviewers worked independently to screen titles and abstracts, and any record deemed potentially eligible proceeded to full-text assessment against the predetermined selection criteria. The methodological quality of included studies was then evaluated, and relevant data were collected using structured data extraction forms. YZ and CL independently screened titles and abstracts, assessed full-text reports for eligibility, and extracted data using a standardized form. Disagreements were first discussed with reference to the eligibility criteria and original reports. During revision,

YX reviewed disputed decisions and adjudicated disagreements that remained unresolved. The reviewer roles for risk-of-bias assessment are described in Section 2.5.

2.5. Risk-of-Bias Assessment

Two reviewers (YZ and CL) independently assessed the risk of bias in the 13 included studies using the original Cochrane risk-of-bias tool (RoB 1) [19]. We assessed random sequence generation, allocation concealment, blinding of participants and personnel, blinding of outcome assessment, incomplete outcome data, selective reporting, and other sources of bias. Each domain was judged to be at low, high, or unclear risk of bias. The reviewers resolved disagreements through discussion, and a third reviewer (YX) adjudicated unresolved disagreements.

2.6. Statistical Analysis

Meta-analytic pooling was performed via Review Manager 5.4.0 (RevMan, The Cochrane Collaboration, Copenhagen, Denmark) and RStudio 2023.09.1 (Posit Software, PBC, Boston, MA, USA). For the primary synthesis of each continuous outcome, we extracted the mean and standard deviation (SD) for the active stimulation and comparator conditions at the first assessment after completion of the intervention. Effect estimates were based on these post-intervention endpoint values. Later follow-up assessments were kept separate from the primary synthesis. Continuous outcomes were summarized using mean differences (MDs) with 95% confidence intervals (CIs) when studies used the same instrument and units. Standardized mean differences (SMDs), expressed as Hedges' g with a small-sample correction, were used when different instruments assessed a sufficiently comparable outcome construct. Ataxia severity was synthesized separately for SARA and ICARS using MDs in the original scale units. Negative MDs indicated lower ataxia severity in the intervention group than in the comparator group. SARA and ICARS total scores were used to assess overall ataxia severity. The balance-related analysis included measures of balance and functional mobility. Within this analysis, the Timed Up and Go test (TUG) was classified as a measure of functional mobility because it combines rising from a chair, walking, turning, and sitting down. Global ataxia severity, balance performance, postural sway, and gait-related outcomes were considered separately; standardization alone was not regarded as sufficient justification for pooling different constructs. Meta-analyses were performed in R 4.3.x using the meta package (v6.x), with all analyses cross-checked in metafor (v4.x) and RevMan 5.4.0, the latter used for forest-plot display and numerical verification. Ataxia severity was synthesized separately for the Scale for the Assessment and Rating of Ataxia (SARA) and the International Cooperative Ataxia Rating Scale (ICARS). Effects were reported as mean differences (MDs) with 945% confidence intervals (CIs) in the original units of each scale. Negative MDs indicated lower ataxia severity with active stimulation relative to the comparator. When different instruments assessed a sufficiently comparable outcome, we used standardized mean differences (SMDs), calculated as Hedges' g. For syntheses in which pooling was clinically appropriate, we used inverse-variance random-effects models. Between-study variance (τ^2) was estimated using restricted maximum likelihood (REML). This approach allowed for variation in the underlying effects across stimulation modalities, anatomical targets, and treatment schedules. We reported τ^2 and I^2 to describe heterogeneity. Model choice was independent of the observed I^2. Common-effect models were fitted as sensitivity analyses. We summarized each stimulation protocol by modality, anatomical target, stimulation parameters, treatment schedule, and concomitant rehabilitation (Table 1). The exploratory subgroup analyses grouped protocols into electrical and magnetic stimulation. These broad categories included protocols that varied in anatomical target and stimulation parameters. The subgroup estimates therefore describe the protocols represented in each

category and have limited applicability to any individual protocol. The interval between the active and sham sessions was at least 1 week in [20], and the washout period between treatment periods was 12 weeks in [21]. Potential carryover was considered in light of the treatment schedule, the washout interval, and any evidence of period or carryover effects reported by the trial authors.

Table 1. Characteristics of included studies.

Study	Disease Phenotype	N (TG/CG)	Intervention (TG vs. CG)	Stimulation Parameters	Target Region	Balance/Gait-Related Outcomes
Barretto et al., 2019 [17]	Degenerative/unspecified cerebellar ataxia	7 (crossover)	Active tDCS vs. sham tDCS	40 min/session; 5 days; intensity NR	Bilateral motor cortex	SARA; gait/posture;
Benussi et al., 2015 [11]	Degenerative/unspecified cerebellar ataxia	19 (crossover)	Anodal ctDCS vs. sham	2 mA; 20 min; single session	Cerebellar cortex	SARA; ICARS gait/posture; 8MWT;
Benussi et al., 2017 [20]	Degenerative/unspecified cerebellar ataxia	20 (12/8)	Anodal ctDCS vs. sham	2 mA; 20 min/session; 10 sessions	Cerebellar cortex	SARA; ICARS; gait/posture; 8MWT;
Brito et al., 2024 [22]	Hereditary ataxia	10 (crossover)	ctDCS + postural training vs. sham + training	2 mA; 20 min; single session	Cerebellum	Biodex balance
Brito et al., 2024 A [23]	Degenerative/unspecified cerebellar ataxia	22 (11/11)	ctDCS + gait training vs. sham + training	2 mA; 25 min/session; 5 sessions	Cerebellum	TUG; MiniBESTest; SARA
Brito et al., 2026 [24]	Degenerative/unspecified cerebellar ataxia	30 (15/15)	Cerebello-spinal DCS + gait training vs. sham	2 mA; 25 min; 5 sessions	Cerebellum + spinal cord	TUG; MiniBESTest; SARA; balance
Chen et al., 2025 (tACS) [12]	Hereditary ataxia	82 (41/41)	Active tACS vs. sham	70 Hz; 2 mA; 40 min/day; 2 weeks	Bilateral cerebellum	SARA; gait
Chen et al., 2025 A [15]	Hereditary ataxia	38	iTBS vs. sham	3 weeks; 126 pulses/session	Cerebellum	ICARS; gait
França et al., 2020 [25]	Degenerative/unspecified cerebellar ataxia	24 (crossover)	Deep rTMS vs. sham	1 Hz; 90% RMT; 1200 pulses/session	Dentate nucleus	SARA; ICARS; gait/posture; TUG
Maas et al., 2022 [14]	Hereditary ataxia	20 (10/10)	Anodal ctDCS vs. sham	2 mA; 20 min/session; 10 sessions	Cerebellum	posture; SARA gait/stance; 8MWT
Manor et al., 2019 [26]	Hereditary ataxia	20 (10/10)	rTMS vs. sham	20 sessions/4 weeks; MRI-guided	Cerebellum	SARA; COP sway; TUG; gait
Naeije et al., 2023 [21]	Hereditary ataxia	24 (crossover)	Anodal ctDCS vs. sham	20 min/day; 5 days/week; 1 week	Cerebellum	SARA
Sanna et al., 2026 [27]	Degenerative/unspecified cerebellar ataxia	16 (8/8)	Anodal ctDCS vs. sham	2 mA; 20 min/session; 10 sessions	Cerebellar cortex	SARA;ICARS; gait

SARA: Scale for the Assessment and Rating of Ataxia; ICARS: International Cooperative Ataxia Rating Scale; TUG: Timed Up and Go test; 8MWT: 8 m walk test; COP: center of pressure. TG: active stimulation group; CG: sham/control group. tDCS: transcranial direct current stimulation. ctDCS: cerebellar transcranial direct current stimulation. tACS: transcranial alternating current stimulation; rTMS: repetitive transcranial magnetic stimulation; iTBS: intermittent theta burst stimulation.

Pooling was undertaken only when the intervention protocols, comparator conditions, outcome measures, and assessment time points were sufficiently comparable. Sin-

gle-session and repeated-session protocols were considered separately, as were immediate post-intervention and follow-up assessments. When pooling was not justified, individual trial estimates were displayed without a summary diamond and synthesized narratively. For crossover trials, treatment effects and standard errors were obtained from paired analyses reported in the source articles or derived from available paired summary statistics. Paired estimates were pooled using inverse-variance methods. Each participant was counted once in the sample size for a given synthesis. When a cohort provided multiple eligible measurements within one outcome domain, we selected one estimate using the same outcome and time-point selection rule across studies.

3. Results

The database searches identified 1190 records. After removal of 385 duplicates, 805 records were screened by title and abstract, and 651 were excluded. Full texts were sought for 154 reports, of which 7 could not be retrieved. Of the 147 reports assessed for eligibility, 134 were excluded for the reasons shown in Figure 1. Thirteen reports describing thirteen studies were included in the systematic review.

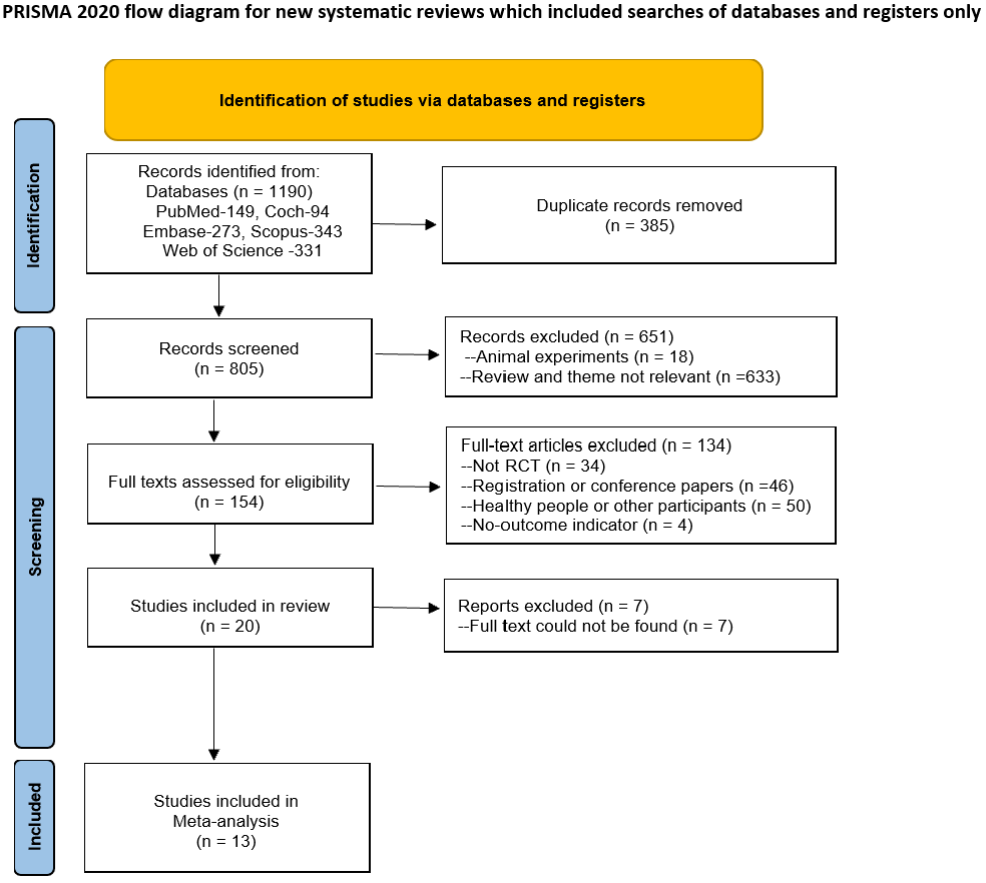


Figure 1. PRISMA 2020 flow diagram showing record identification, screening, report retrieval, eligibility assessment, and study inclusion.

3.1. Study Characteristics

Table 1 summarizes the characteristics of the 13 included studies. The study-level sample sizes listed in Table 1 sum to 332 participants. Five studies used crossover designs and accounted for 84 participants. For these trials, the sample size in Table 1 refers to the number of individuals enrolled in the trial, counted once across treatment periods. The

included studies evaluated transcranial direct current stimulation, transcranial alternating current stimulation, repetitive transcranial magnetic stimulation, intermittent theta-burst stimulation, and cerebello-spinal direct current stimulation.

3.2. Quality Assessment

Methodological quality was determined with the Cochrane Collaboration Risk-of-Bias tool [18]. Adequate random sequence generation and allocation concealment were present in 8 RCTs, indicating a low risk of selection bias. Double-blinding was reported in 12 trials, while the remaining trial had a high risk of bias due to lack of blinding. Blinding of outcome assessment was judged to be at low risk of bias in 12 studies and at unclear risk in 1 study. Selective reporting bias was unclear in four studies, and three studies were judged to have other potential sources of bias. All included studies reported complete outcome data, and the detailed risk-of-bias assessment is depicted in Figures 2 and 3.

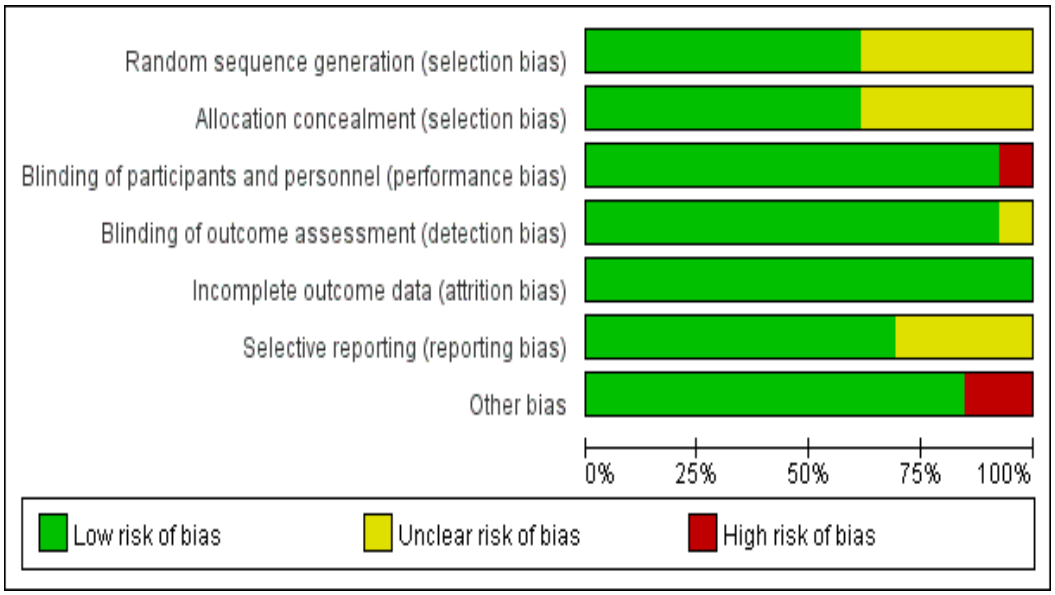


Figure 2. Article quality evaluation form.

3.3. A Meta-Analysis of NIBS on Ataxia Severity in Patients with Ataxia

This meta-analysis evaluated the effects of non-invasive stimulation on ataxia severity in patients with ataxia, including 13 studies and 15 effect estimates. The pooled results demonstrated a significant beneficial effect of non-invasive stimulation on overall ataxia severity (MD = −1.49, 95% CI: −2.46 to −0.53, $p = 0.002$). Ataxia severity was synthesized separately for SARA and ICARS total scores using mean differences (MDs) and fixed-effect inverse-variance models. The SARA analysis included 11 study comparisons and yielded an MD of −1.39 points (95% CI −2.37 to −0.41; $p = 0.006$; $I^2 = 4\%$). The ICARS ($n = 4$) analysis included four study comparisons and yielded an MD of −4.16 points (95% CI −9.17 to 0.84; $p = 0.10$; $I^2 = 0\%$). Negative values indicated lower ataxia severity in the intervention group than in the comparator group (Figure 4).

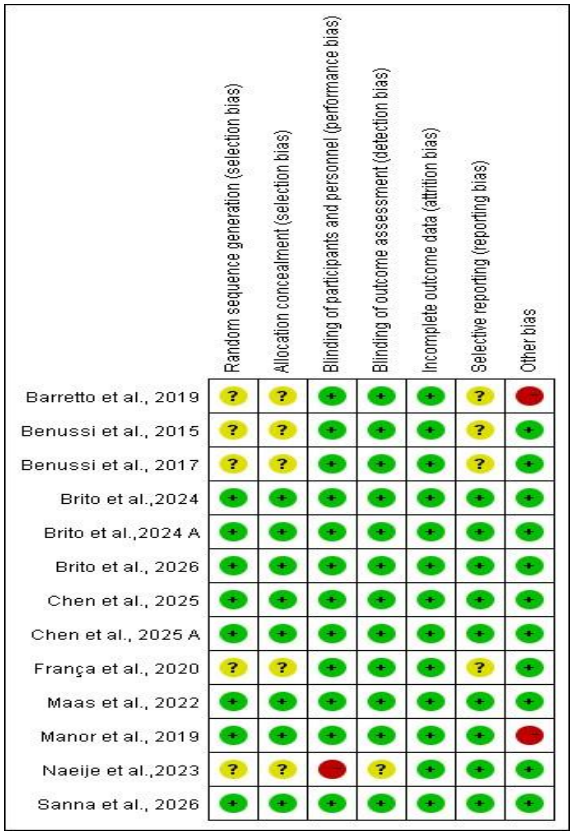


Figure 3. Article quality evaluation form [11,12,14,15,17,20–27].

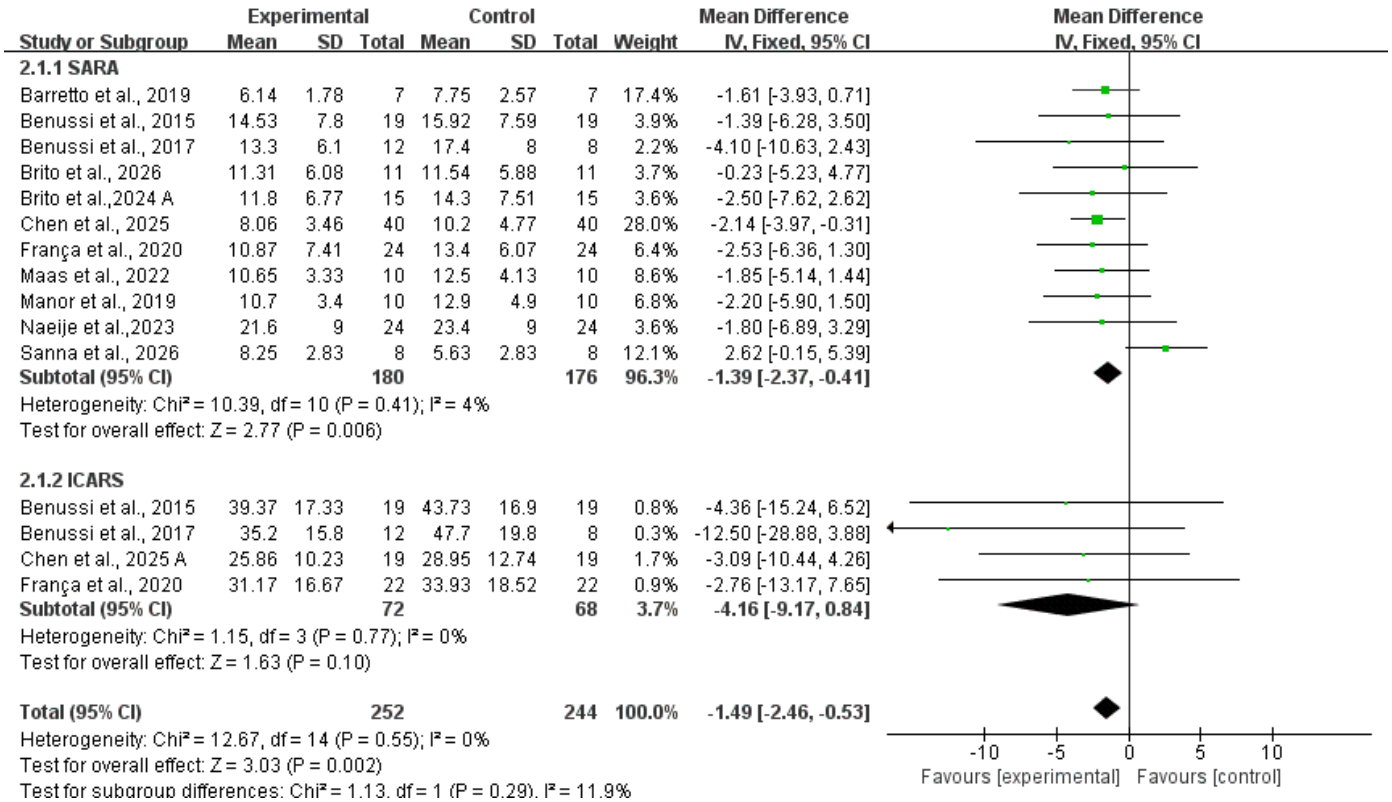


Figure 4. Effects of non-invasive brain stimulation on ataxia severity measured with SARA and ICARS. Pooled mean differences and 95% confidence intervals are presented separately in the original units of each scale. Negative values favor active stimulation [11,12,14,15,17,20,21,23–27].

3.3.1. Disease Type

The exploratory subgroup analysis evaluated ataxia severity by disease etiology (Figure 5). In the genetic ataxia subgroup, which included spinocerebellar ataxia and Friedreich's ataxia, the pooled effect favored NIBS (SMD = −0.38, 95% CI −0.66 to −0.11, p = 0.006). In the subgroup with other degenerative or mixed ataxias, the confidence interval included zero (SMD = −0.23, 95% CI −0.52 to 0.06, p = 0.12). The test for subgroup differences was nonsignificant (p = 0.45), providing insufficient evidence that the effect of NIBS on ataxia severity differed by disease etiology.

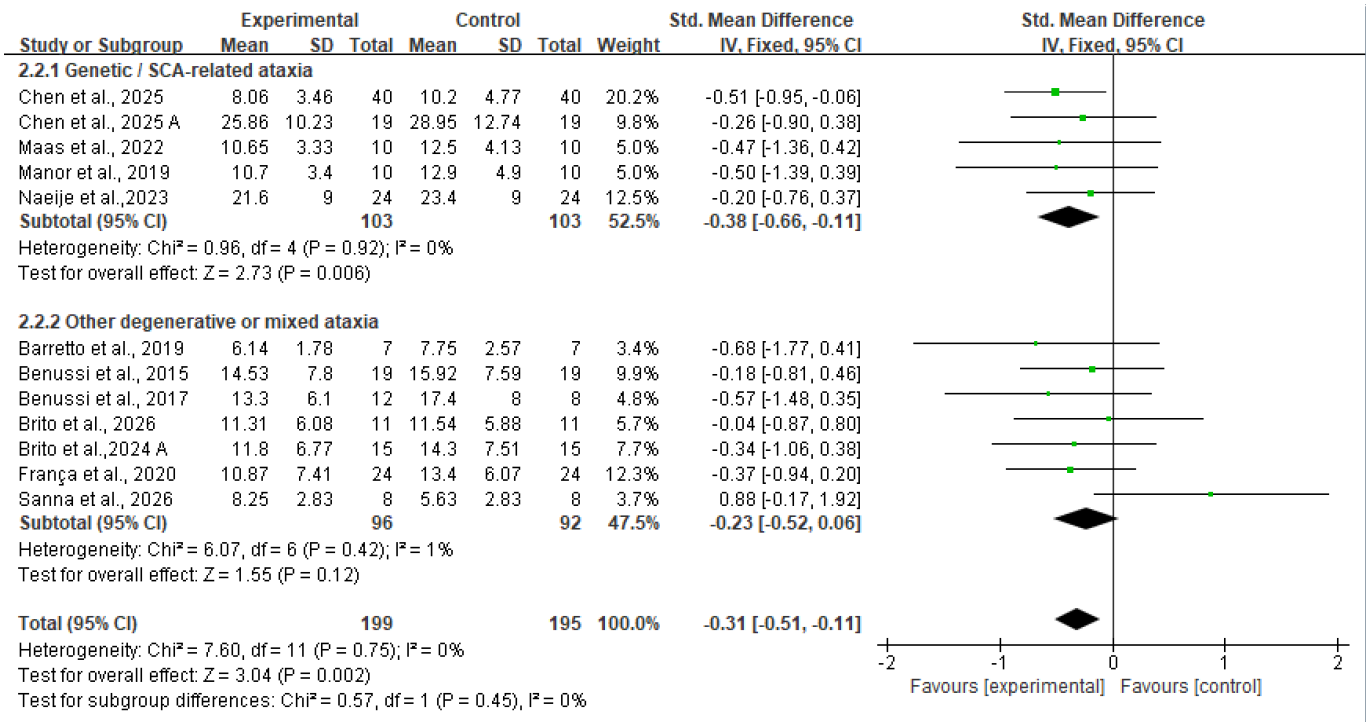


Figure 5. Subgroup analysis by disease type in ataxia severity [11,12,14,15,17,20,21,23–27].

3.3.2. Brain Stimulation Modes

Figure 6 presents an exploratory subgroup analysis of ataxia severity by stimulation modality. The pooled effect within the electrical stimulation subgroup favored active stimulation (SMD = −0.29, 95% CI −0.53 to −0.06, p = 0.01). In the magnetic stimulation subgroup, the confidence interval included zero (SMD = −0.35, 95% CI −0.74 to 0.03, p = 0.07). The test for subgroup differences yielded p = 0.80, providing insufficient evidence that the effect on ataxia severity differed between electrical and magnetic stimulation.

3.4. Exploratory Analysis of Gait Outcomes

This meta-analysis included 12 studies, 13 datasets, and 351 participants (Figure 7). Given the variability in gait-related outcome measures across studies, SMDs were calculated to estimate the pooled effect. Compared with control conditions, NIBS significantly improved gait function in individuals with ataxia (SMD = −0.26, 95% CI: −0.47 to −0.05, I^2 = 9%, p = 0.02), suggesting that NIBS may have beneficial effects on gait performance in this population.

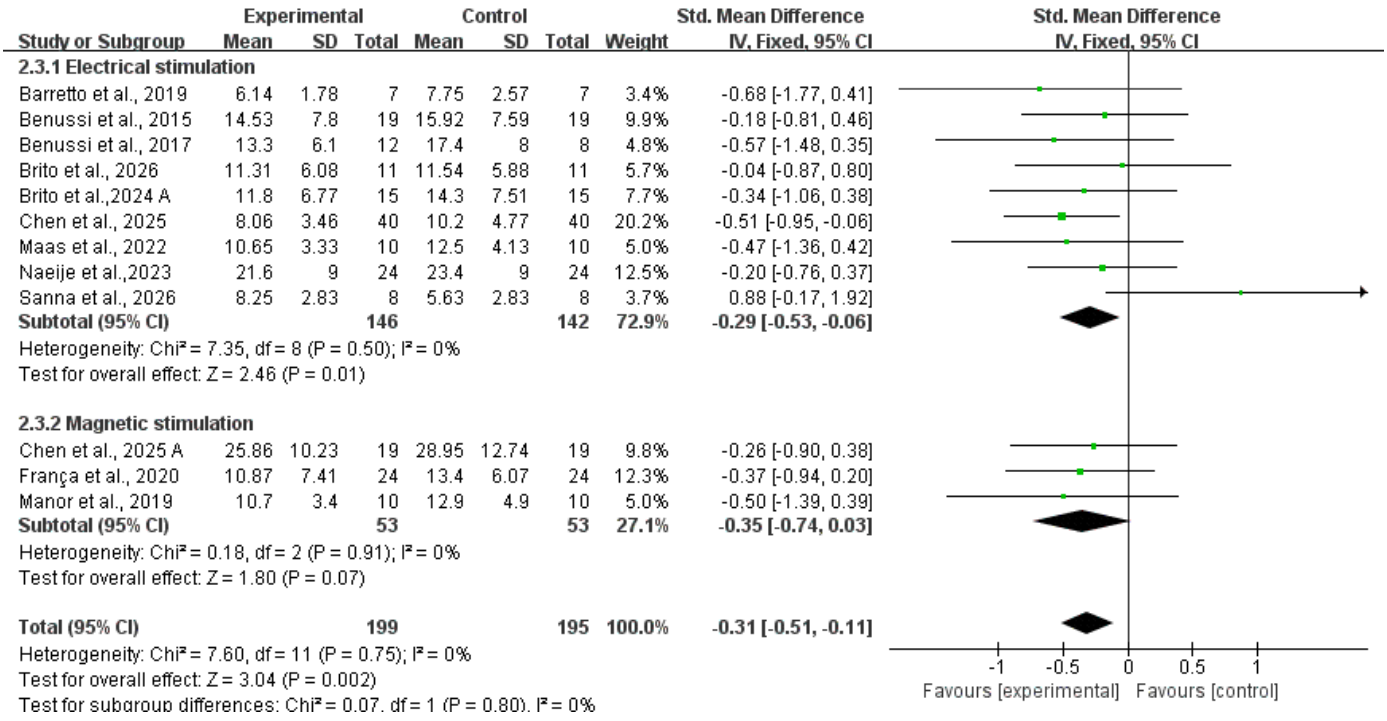


Figure 6. Exploratory subgroup analysis of ataxia severity by stimulation modality (electrical vs. magnetic stimulation) [11,12,14,15,17,20,21,23–27].

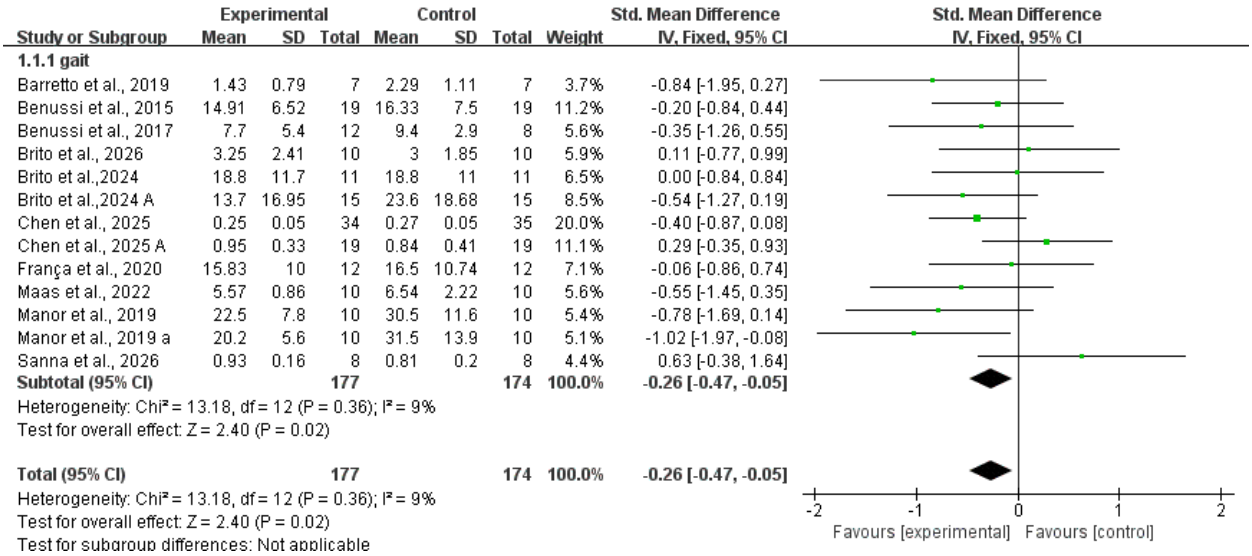


Figure 7. A meta-analysis of NIBS on gait in patients with ataxia [11,12,14,15,17,20,22–27].

3.4.1. Disease Type

Figure 8 presents the exploratory subgroup analysis of gait outcomes by disease etiology. The pooled effect within the genetic/SCA-related ataxia subgroup favored NIBS (SMD = −0.31, 95% CI −0.60 to −0.02, $p = 0.04$). In the subgroup with other degenerative or mixed ataxias, the confidence interval included zero (SMD = −0.20, 95% CI −0.51 to 0.11, $p = 0.20$). The test for subgroup differences yielded $p = 0.62$, providing insufficient evidence that the effect of NIBS on gait differed by disease etiology.

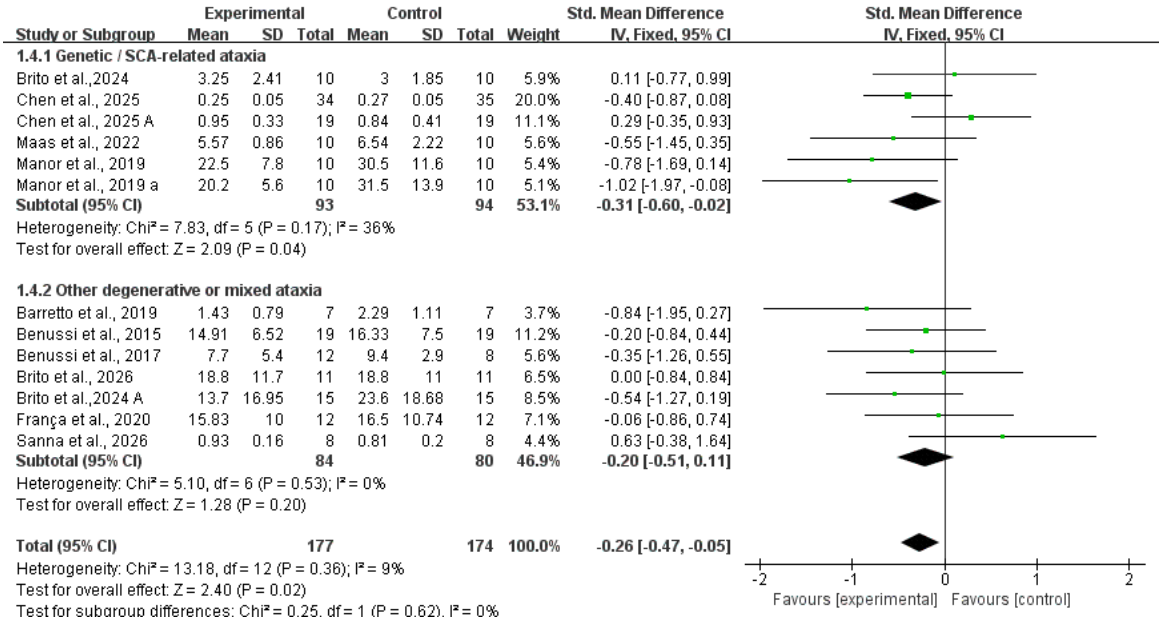


Figure 8. Subgroup analysis of gait-related disease phenotypes [11,12,14,15,17,20,22–27].

3.4.2. Brain Stimulation Modes

Figure 9 presents the exploratory subgroup analysis of gait outcomes by stimulation modality. The pooled effect within the electrical stimulation subgroup favored active stimulation (SMD = −0.27, 95% CI −0.52 to −0.02, p = 0.03). In the magnetic stimulation subgroup, the confidence interval included zero (SMD = −0.23, 95% CI −0.63 to 0.17, p = 0.26). The test for subgroup differences yielded p = 0.86, providing insufficient evidence that the effect on gait differed between electrical and magnetic stimulation.

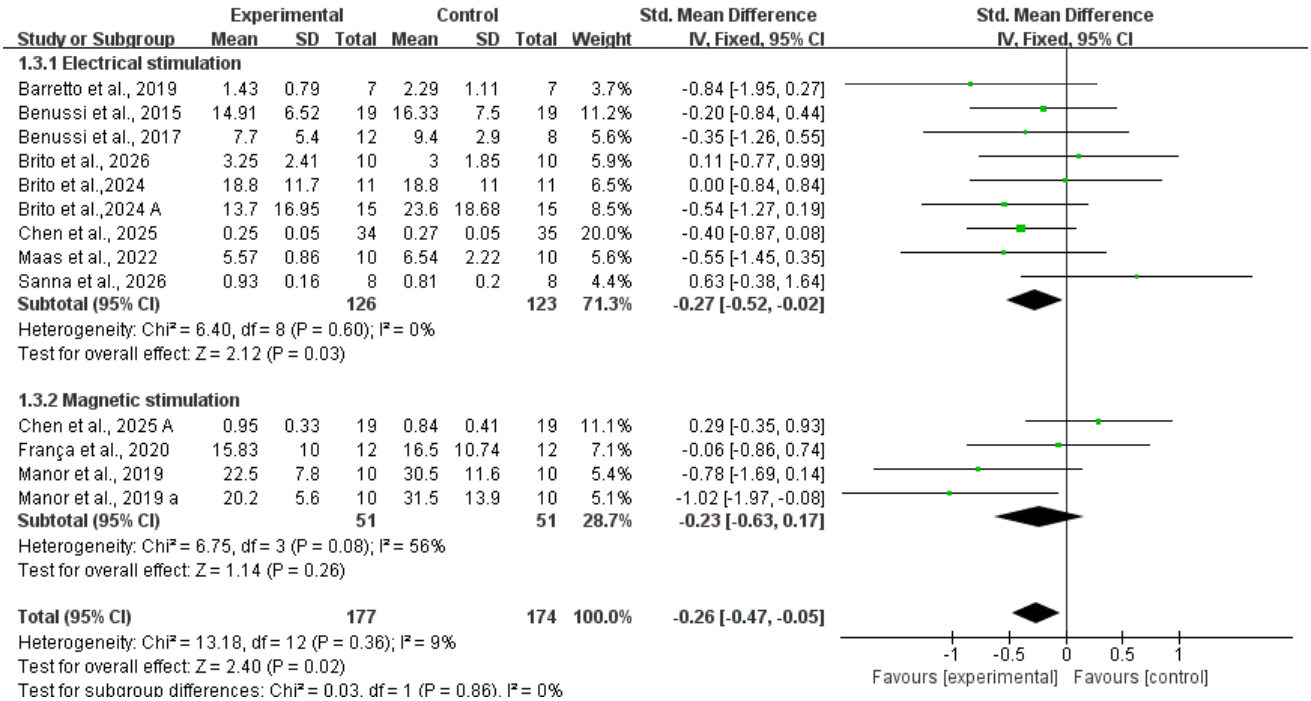


Figure 9. Subgroup analysis of brain stimulation patterns for gait [11,12,14,15,17,20,22–27].

3.5. Meta-Analysis of NIBS on Balance in Patients with Ataxia

To evaluate the effects of NIBS on balance in patients with ataxia, this systematic review and meta-analysis pooled data from seven eligible studies, encompassing 150 participants. The pooled analysis included measures of balance-related performance, including the Berg Balance Scale as a clinical balance measure and the Timed Up and Go test as a functional mobility measure. A fixed-effects framework was adopted because heterogeneity was low (I^2 = 0%, p = 0.48). The pooled point estimate numerically favored the comparator, but the 95% confidence interval included zero. The effect of NIBS on balance therefore remains uncertain (SMD = 0.28, 95% CI: −0.07 to 0.62, p = 0.12) (Figure 10).

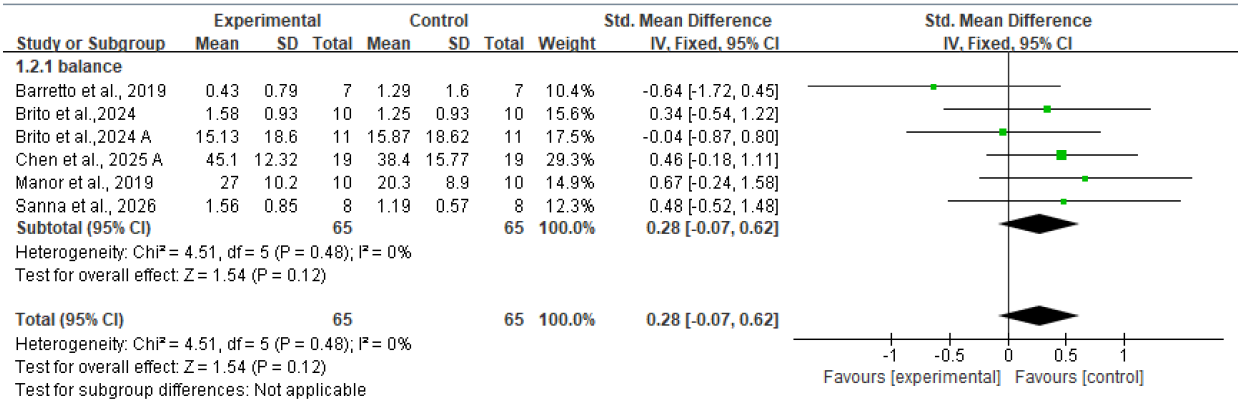


Figure 10. A meta-analysis of nibs on balance in patients with ataxia [15,17,22,23,26,27].

3.6. Assessment of Reporting Bias

The number of independent studies was limited in some outcome-specific meta-analyses. Because ataxia severity, gait, and balance represent distinct outcomes, effect estimates from these domains were considered separately when evaluating the feasibility of funnel-plot assessment. Reporting bias could not be reliably assessed using the available data and remains a potential limitation.

4. Discussion

4.1. Effect of NIBS on Ataxia Severity in Patients with Ataxia

The mechanistic interpretations offered in this section are presented as hypotheses that are consistent with, but not tested by, the present data; changes in cerebello-cortical connectivity, neuronal excitability, and stimulation-induced plasticity are plausible mechanisms. The pooled clinical outcomes do not establish whether these processes mediated the observed effects. The application of NIBS correlated with a notable decline in clinical ataxia severity. The understanding of ataxia as a disorder involving disrupted cerebellar–cortical interactions is growing. This is in contrast to the view of it as isolated cerebellar damage [28]. Modulation of abnormal neural activity and residual network function may contribute to improved motor coordination. tDCS has been reported to modulate neuronal membrane potential, cortical excitability, and synaptic plasticity. One proposed mechanism is that cerebellar tDCS modulates cerebellar–cortical inhibitory interactions in a polarity-dependent manner. If so, this could account for the observed reduction in ataxia severity [29]. Sham-controlled trials have reported improvements in ataxia symptoms following cerebellar tDCS [11], and clinical outcomes may be further influenced by cerebellar spinal tDCS, which can modulate cerebellar–motor cortical connectivity [30]. The present findings are consistent with the hypothesis that the effects of tDCS extend beyond the targeted region to broader cerebello-motor networks, although the trials did not measure network-level activity and this interpretation remains speculative.

The exploratory analyses provided insufficient evidence that the effect of NIBS on ataxia severity differed by disease etiology or stimulation modality. The small number of trials within subgroups and the variation in stimulation protocols limited the precision and clinical interpretation of these comparisons. The broad etiologic subgroups included participants with different diagnoses, disease durations, and levels of baseline severity. The pooled estimates therefore have limited applicability to specific diagnostic subtypes, and the available data were insufficient to determine whether response to stimulation varied across these clinical characteristics. This finding may reflect differences in disease-specific pathophysiology. Patients with SCA may still have some degree of plasticity in their cerebellar–motor networks even as their neurodegeneration progresses. By contrast, disorders such as MSA-C are characterized by more widespread dysfunction of the cerebellum, brainstem, and other areas outside the cerebellum, which may reduce responsiveness to focal cerebellar stimulation [31]. The original difference in statistical significance between the electrical- and magnetic-stimulation analyses does not establish a difference in efficacy between modalities. The included protocols differed in anatomical targets, stimulation parameters, treatment schedules, and concomitant rehabilitation, limiting indirect comparisons. The exploratory subgroup analyses provided insufficient evidence that the effect of NIBS on ataxia severity differed by disease etiology or stimulation modality. Statistical significance within the genetic or spinocerebellar ataxia subgroup, or within the electrical stimulation subgroup, does not establish a difference between subgroups. The formal tests for subgroup differences were nonsignificant, and the estimates were limited by small subgroup samples and variation in stimulation targets, treatment schedules, and concomitant rehabilitation. Modulation of cerebellar–cortical connectivity remains a plausible mechanism for changes in ataxia severity. Whether this mechanism contributes differently across etiologies or stimulation modalities remains an open question.

Available data are hampered by limited sample sizes, phenotypic variability among patients, and protocol heterogeneity across studies. Future studies should consider incorporating disease stratification, neuroimaging, and electrophysiological biomarkers in order to identify patterns of response to treatment and facilitate the development of personalised neuromodulation [29,32,33]. The pooled between-group difference in SARA scores was approximately 1.39 points in favor of NIBS. A recent study estimated a minimal clinically important difference of approximately 1.5 points in individuals with cerebellar ataxia. This estimate provides clinical context, although its applicability to the different populations and follow-up intervals in this review is uncertain. Longitudinal studies also show that the rate of SARA progression varies by spinocerebellar ataxia subtype [34]. Whether the observed short-term differences translate into meaningful or sustained improvements in daily activities, independent mobility, or fall risk remains uncertain.

4.2. Effects of NIBS on Gait Function in Patients with Ataxia

In the present synthesis, NIBS was associated with a small improvement in gait (SMD = −0.26, 95% CI −0.47 to −0.05), estimated from a set of trials that only partially overlaps with those contributing to the severity analysis. Because severity and gait were pooled in separate meta-analyses, these data cannot establish whether the gait effect is independent of the change in overall symptom severity; doing so would require within-trial joint modelling or a formal comparison of effects within the same studies, which the available reports do not permit. Gait impairment in ataxia reflects dysfunction across distributed motor networks rather than isolated cerebellar dysfunction [34], and one hypothesis is that NIBS improves gait by modulating cerebello-motor connectivity involved in movement timing and error correction. The present data do not test this hypothesis [34,35]. Whether changes in cerebello-motor connectivity contributed to the observed gait findings remains to be tested.

The exploratory gait analyses provided insufficient evidence that treatment effects differed by disease etiology or stimulation modality. These comparisons should be interpreted cautiously given the small number of trials and the variation in gait measures and stimulation protocols. This discrepancy may be ascribed to the prevailing engagement of cerebellar–motor circuits in SCA, which exhibit enhanced responsiveness to focal neuromodulation [36]. In contrast, other degenerative ataxias may involve brainstem, basal ganglia, and autonomic dysfunction, potentially reducing the effects of focal cerebellar stimulation [37]. Electrical stimulation was associated with improved gait outcomes, whereas magnetic stimulation yielded no significant effect. This difference may reflect differences in the mechanisms and parameter dependence of the two stimulation modalities, with tDCS providing relatively sustained modulation of excitability, whereas rTMS responses may be more sensitive to stimulation parameters, targeting, and disease characteristics [8,38]. The exploratory gait analyses provided insufficient evidence that treatment effects differed by disease etiology or stimulation modality. Variation in gait measures and stimulation protocols, together with the small number of studies within each subgroup, limited the precision and interpretation of these comparisons. Changes in cerebellar–motor connectivity could influence movement timing and error correction during walking. Whether this mechanism accounts for variation in gait response would require studies that measure neural and gait outcomes together and directly test differences in treatment effects. NIBS, particularly tDCS, may have potential for improving gait function in patients with ataxia. Nevertheless, the available evidence is constrained by limited sample sizes, clinical diversity, and inconsistent gait metrics. Future studies should incorporate quantitative gait analysis, kinematic assessment, and neuroimaging biomarkers to characterize treatment-response patterns and inform individualized neurorehabilitation strategies [31,34,36,39]. Changes in SARA and ICARS total scores reflect overall ataxia severity. These scores include gait and posture items, so effects on individual motor domains require domain-specific measures. The pooled balance-related estimate combines measures of balance and functional mobility, which limits conclusions about postural stability specifically.

4.3. Effects of NIBS on Balance Function in Patients with Ataxia

The pooled estimate for balance did not reach statistical significance (SMD = 0.19, 95% CI −0.13 to 0.52, p = 0.25). This confidence interval is compatible with no effect, with a small-to-moderate benefit, and with a small harm; the analysis, based on seven trials, was underpowered to detect effects of the magnitude plausibly produced by a single NIBS course. The result therefore indicates that the current evidence is inconclusive, not that NIBS fails to affect balance. Balance regulation depends on the integration of multisensory information across cerebellar, vestibular, visual and somatosensory systems, so that modulation of regional excitability alone need not be sufficient to alter a composite balance score. The effect of NIBS on balance-related outcomes remains uncertain, and the available data provide limited insight into the underlying neural mechanisms [40]. Moreover, stimulation-induced neuroplastic changes may require concurrent task-specific rehabilitation to translate neural modulation into meaningful functional gains [7]. Most existing NIBS studies have focused on cerebellar stimulation; however, postural control involves coordinated activity across multiple neural systems, including the cerebellum, motor cortex, sensory integration regions, and vestibular networks. Future studies should therefore explore individualized, network-based stimulation strategies tailored to patients' specific neural dysfunction profiles [41,42].

Therefore, the current evidence does not provide sufficient support for NIBS as a standalone intervention for improving balance function in ataxia. In addition, although the washout intervals in the crossover trials exceeded the expected duration of NIBS after-

effects, carryover effects cannot be entirely excluded. Combining NIBS with task-specific training therefore warrants further investigation. NIBS may enhance neural excitability. It may also enhance neural plasticity. However, neuromodulation alone may be insufficient. It may not be enough to restore the complex sensorimotor integration required for effective postural control. Task-oriented training may help consolidate stimulation-induced plasticity and translate neural changes into functional gains [43]. Incomplete reporting of allocation concealment or assessor blinding in some trials limited our ability to judge the risk of bias. Potential carryover in crossover trials adds uncertainty to the estimated treatment effects and should be considered when interpreting the pooled findings. Ataxia severity and balance were prespecified in the original PROSPERO registration (CRD4202ght). However, the findings should be interpreted with caution because of the small samples, heterogeneity in stimulation protocols, and limited follow-up.

The pooled estimates primarily reflect the first assessment after stimulation. In a pilot trial, Manor et al. reported a greater improvement in SARA scores with rTMS than with sham at one month in a secondary analysis. Maas et al. found no statistically significant between-group difference in SARA change through one year. These findings leave the durability of benefit uncertain. Manor et al. reported no rTMS-related adverse events. Maas et al. reported mild headache and itching in the active group and two focal skin lesions in one participant.

5. Conclusions

NIBS appears to improve clinical severity in individuals with ataxia. Subgroup analyses found that, although electrical stimulation showed potential benefits, the exploratory subgroup findings provided insufficient evidence of differential effects by disease etiology or stimulation modality. These findings require confirmation in adequately powered trials. The gait finding warrants confirmation in trials with prespecified gait outcomes. Further studies are needed to determine whether the observed improvements are clinically meaningful and sustained.

Supplementary Materials: The following supporting information can be downloaded at https://www.mdpi.com/article/10.3390/brainsci16121309/s1, Table S1: Database search strategies for non-invasive brain stimulation in patients with ataxia. Search expressions are reproduced as supplied by the authors; retrieval counts are reported before deduplication.; Table S2: PRISMA checklist.

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