Transcutaneous Spinal Cord Stimulation (tSCS) and Tone Modulation

A research summary for clinical application

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1. Purpose and Scope

This document synthesizes current evidence on how transcutaneous spinal cord stimulation (tSCS) modulates muscle tone in the direction most relevant to pediatric neurorehabilitation: reduction of hypertonia and spasticity, where most of the evidence base sits.

Most of the underlying research comes from adult spinal cord injury (SCI) and multiple sclerosis (MS) populations, with a smaller but growing pediatric literature, largely in cerebral palsy. Where evidence is adult-only, this is flagged explicitly rather than extrapolated silently.

2. Mechanistic Basis of Tone Modulation

tSCS delivers electrical current through surface electrodes over the spinous processes (most commonly lumbar T11–L1, sometimes cervical or combined with coccygeal sites), primarily activating large-diameter Ia afferent fibers in the posterior roots as they enter the spinal cord. This is the same afferent pathway recruited by tendon reflexes and by epidural spinal cord stimulation but achieved non-invasively.

The tone-modulating effect is thought to work through several convergent mechanisms:

- Presynaptic inhibition: sustained afferent volleys from tSCS produce prolonged neurotransmitter depletion at Ia terminals, producing post-activation (homosynaptic) depression — the same phenomenon underlying reduced H-reflex amplitude after stimulation.
- Postsynaptic inhibitory circuit engagement: recent work (Minassian et al., using intrathecal pharmacology and reflex probing) shows that anti-spasticity tSCS transiently restores deficient postsynaptic inhibitory circuit function in people with SCI, correlating directly with reductions in hypertonia, spasms and clonus.
- Supraspinal engagement: tSCS also facilitates corticospinal and propriospinal pathways, meaning it does not act purely as a segmental "damper" — it can simultaneously suppress pathological reflex activity and support voluntary drive, which is why the same modality can, at different parameters, either reduce spasticity or support strength/locomotor training.

Critically, the direction of the tone effect (dampening vs augmenting) depends heavily on stimulation parameters — frequency, intensity relative to motor threshold, electrode montage, and whether

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stimulation is paired with active movement — rather than being a fixed property of "tSCS" as a single intervention.

2.1 Reciprocal inhibition as a specific, measurable target

A more granular mechanism worth highlighting for pediatric application is reciprocal inhibition — the normal process by which activating an agonist muscle's Ia afferents inhibits the motoneuron pool of its antagonist, allowing smooth, coordinated movement rather than co-contraction. This circuit is tested electro-physiologically by conditioning the H-reflex of one muscle with a short-latency prior stimulation of the antagonist's nerve.

This is directly relevant to children with spastic cerebral palsy: a case-control study (12 children with spastic CP vs age-matched controls) found that soleus H-reflex amplitude increased during voluntary dorsiflexion in children with CP, whereas typically developing children showed the expected decrease — a reversal that reflects impaired reciprocal inhibition and helps explain the pathological co-contraction seen clinically in CP gait and posture. Vibratory stimulation produced some inhibitory effect in both groups, but the CP group's baseline dysfunction remained evident.

This gives tSCS a specific, testable target in pediatric hypertonia beyond a generic "reduce tone" framing: the currently recruiting NCT07516067 trial explicitly measures whether tSCS can normalize reciprocal inhibition toward typically-developing values in children with CP, alongside separate measures of H-reflex suppression and motoneuron persistent inward currents. Framing tSCS's target as "restoring a specific, impaired spinal circuit" rather than "generically calming an overactive system" is likely to be more defensible and more precise in clinical or parent-facing communication.

3. Reducing Hypertonia and Spasticity

3.1 Multiple sclerosis and other populations

The same Vienna group has extended the 50 Hz / 30-minute protocol to MS, reporting reduced muscle spasms and clonus duration for around two hours, with clinician-rated lower-limb hypertonia remaining improved at 24 hours post-intervention. A more rigorous 2025 study (Charité, Berlin; German Clinical Trials Register DRKS00023357) used a randomized, sham-controlled, blinded crossover design in 20 people with primary or secondary progressive MS, applying biphasic 50 Hz lumbar tSCS for 30 minutes and assessing bilateral Modified Ashworth Scale sum score as the primary outcome, alongside gait kinematics (inertial measurement units) and EMG-based reflex measures. This is a methodologically stronger design than most of the SCI literature (blinded assessors, sham control, prospective registration) and directly addresses the question of whether tSCS's anti-spasticity effect can be distinguished from placebo/attention effects.

The same paper also situates tSCS against transcutaneous electrical nerve stimulation (TENS), which is already part of established non-pharmacological spasticity guidelines: TENS electrodes sit over the affected peripheral muscle/nerve and activate relatively few fibers, whereas computational modeling indicates lumbar tSCS activates posterior roots across several spinal segments simultaneously — a broader, more centrally-targeted afferent volley. Reported TENS effects on MS spasticity are mixed in the literature, whereas the tSCS effect size in this trial was more consistent,

though the authors caution that tSCS has not yet entered standard clinical guidelines the way TENS has.

Small case series in primary lateral sclerosis have used a closely related montage (T11–T12 and para-umbilical, 50 Hz, 400 µs) with preliminary positive signal on spasticity and mobility measures, though the authors note this needs larger controlled trials.

3.2 Pediatric cerebral palsy

This is the most directly relevant pool of pediatric evidence for a hypertonic population. A 2023 review tracing the field's progression from adult to pediatric applications (Singh, Lucas, Keller, Martin, Behrman, Vissarionov, Gerasimenko) notes that published pediatric tSCS work remains limited in volume but spans CP, spina bifida, and SCI. Across several pilot studies and case reports (children aged roughly 2–13 years, GMFCS levels I–V), tSCS applied at T11/L1 — typically 30–50 Hz, 1 ms biphasic pulses, combined with treadmill training or activity-based locomotor therapy — has been associated with:

- Reduced pathological muscle co-contraction during gait and standing
- Increased passive joint range of motion
- Improved Gross Motor Function Measure (GMFM) scores and walking speed
- Acute improvements in postural control, including bilateral weight-bearing stepping emerging in children who could not previously step

A trial now recruiting at the University of Pittsburgh (NCT07516067), which began enrolling in March 2026, is using a Digitimer DS8R biphasic constant-current stimulator, 30–50 Hz, 1 ms pulse width, with electrode pairs at T11–L1 and over the iliac crests carrying equal, alternating current (biphasic — no fixed anode/cathode role for either electrode), explicitly to characterize changes in spinal excitability, spasticity and motor function in children with CP before, during and after stimulation. Its outcome measures go beyond global spasticity scores: it specifically tests reciprocal inhibition between antagonist muscles (conditioning the tibial-nerve H-reflex with a prior deep-peroneal-nerve stimulation, comparing CP to typically-developing controls), motoneuron persistent inward currents as a direct excitability measure, and whether tSCS shifts these toward typically-developing values — success is pre-defined as a 20% shift in each measure. This suggests the field is moving toward more standardized, mechanistically-grounded pediatric protocols, but rigorous dosing guidance for children specifically is still emerging rather than established.

A separate pilot combined tSCS with short-burst interval treadmill training (SBLTT) in four ambulatory children with CP, now published with concrete outcome data: the Modified Ashworth Scale across four lower-extremity muscles dropped by 1.40 ± 0.22 points with tSCS + SBLTT, compared to only 0.43 ± 0.39 with SBLTT alone, while walking distance (1-minute walk test) was maintained in both conditions and both spasticity and gait gains were sustained at an 8-week follow-up. Children and parents also reported reduced fatigue with the combined intervention. The study explicitly frames tSCS's proposed advantage over existing spasticity treatments: botulinum toxin, baclofen and selective dorsal rhizotomy all work by suppressing or ablating neural/muscular drive, which can reduce tone but often at the cost of strength and with inconsistent functional walking gains unless paired with intensive therapy. tSCS is proposed to work differently — by amplifying sensory feedback to support reorganization of spinal and supraspinal pathways — which is the

rationale for combining it with active training rather than using it as a passive, standalone tone-reduction treatment.

A separate study using a commercial device (SCONE™, SpineX Inc.) examined the acute effects of a single 30-minute transcutaneous spinal neuromodulation session in 12 individuals with CP aged 2 to 50 (GMFCS I–V), with stimulation individually titrated per patient (roughly 15–50 mA across T11/L1, set below each child's threshold). Acute stimulation improved postural and locomotor ability in 11 of the 12 participants, including a 2-year-old (GMFCS IV) achieving bilateral weight-bearing stepping for the first time, and a 10-year-old and a 4-year-old (both GMFCS V) achieving independent head control and supported standing that were not present without stimulation. All participants showed improved coordination between flexor and extensor muscle activity during stepping. As an acute, single-session, uncontrolled case series with a commercial proprietary device, this is hypothesis-generating rather than confirmatory, but it is one of the few pediatric CP datasets spanning the full GMFCS range (I–V) and the widest published pediatric age range (2–50 years) for this intervention.

4. How tSCS Differs From Conventional Anti-spasticity Approaches

This section is included because tSCS is often introduced to families and colleagues alongside, or in place of, established anti-spasticity treatments, and the mechanistic contrast is useful for explaining what tSCS is and is not doing.

- Botulinum toxin (BoNT-A): acts at the neuromuscular junction, blocking acetylcholine release to produce focal, reversible muscle weakness. It reduces tone by reducing the muscle's ability to contract, not by changing spinal excitability — hence its effect is confined to injected muscles and wears off over roughly 3 months, requiring repeat injections.
- Oral or intrathecal baclofen: a systemic or intrathecal GABA-B agonist that dampens excitatory neurotransmission broadly at the spinal cord level. It is not muscle-specific and carries systemic side effects (sedation, weakness, in the oral form) that intrathecal delivery partly mitigates at the cost of an implanted pump.
- TENS: peripheral nerve/muscle-surface stimulation, already part of standard non-pharmacological spasticity guidelines, but activating comparatively few afferent fibers at the site of application.
- tSCS: does not block or ablate anything — it recruits large-diameter posterior-root afferents to transiently drive inhibitory spinal interneuron circuits (and, per Section 2.1, can specifically target reciprocal inhibition), while leaving voluntary motor drive intact and in some paradigms enhancing it. This is the basis for pairing tSCS with active therapy (treadmill training, robotic-assisted gait training) rather than using it as a substitute for injections or medication — the two categories of treatment are not mechanistically equivalent and are not necessarily competing for the same clinical role.

None of the identified literature directly compares tSCS against BoNT-A or baclofen head-to-head in the same trial; the contrast above is mechanistic, drawn from the respective bodies of evidence for each treatment rather than a direct comparative study.

5. Safety and Tolerability

tSCS is generally reported as well tolerated, with adverse events that are minor, transient, and localized rather than systemic. Across published reports the most frequently noted issues are skin redness under the electrodes and discomfort or a tingling/paraesthesia sensation at higher stimulation intensities, both of which resolve without intervention.

Pediatric-specific safety data comes from a pilot study combining cumulative transcutaneous spinal stimulation with locomotor training in three children with SCI (130 total intervention sessions): 88.5% of sessions were free of any adverse effect; reported events were autonomic dysreflexia (5.4% of sessions), skin redness at electrode sites (4.6%), and headache (1.5%) — all managed within session without lasting sequelae. This is reassuring pilot-level evidence, but it comes from a single small study, not a completed formal safety trial. A separate, still-ongoing Johns Hopkins-sponsored protocol (IRB00300695) has safety and feasibility of tSCS in children with incomplete SCI as its explicit primary outcome, over an 8-week gait-training protocol against sham stimulation — meaning pediatric safety is still an open, actively-studied question in the field rather than an established fact, even though the evidence gathered so far points in a reassuring direction.

Because tSCS is entirely non-invasive and surface-electrode based, stimulation can be stopped immediately if any adverse sensation occurs, which is a meaningful practical safety margin compared to implanted (epidural) stimulation systems.

6. Stimulation Parameters Reported Across Indications

The table below consolidates parameters as reported in the literature reviewed. It is a summary of published protocols, not a prescriptive clinical guideline, and pediatric-specific dosing remains far less standardized than adult SCI/MS protocols.

Indication	Electrode placement	Frequency	Intensity	Session length
Hypertonia / spasticity — core protocol (originally established in SCI/MS studies, extended to pediatric CP below)	Electrode pair at T11–L1 (± T11+T12 dual site), second pair over iliac crests or para-umbilical — symmetric biphasic waveform, no fixed anode/cathode roles	50 Hz most replicated; 25–100 Hz range reported	Sub-motor threshold (below level that evokes visible reflex/twitch)	30 min, single or repeated sessions
Locomotor / stepping facilitation	T11 and L1, sometimes + coccyx (Coc1) or cervical (C5)	~25–30 Hz for coordinated flexor/extensor stepping; ~10 Hz favors extensor tone	Supra-threshold, combined with body-weight-supported training	Variable; often paired with gait/treadmill training
Pediatric cerebral palsy (spasticity + motor function)	Electrode pair T11–L1, second pair over iliac crests —	30–50 Hz, 1 ms pulse width, biphasic	Individually titrated, typically sub- to	Single sessions studied; multi-week protocols combined

Indication	Electrode placement	Frequency	Intensity	Session length
	symmetric biphasic pulses, both electrodes carry equal current		supra-threshold depending on goal	with treadmill/AB-LT
kHz-carrier modulated tSCS (comfort-oriented)	Cervical or lumbar, as per indication	5–10 kHz carrier over underlying stimulation frequency	Requires higher charge (~3.8× at posterior root threshold) than conventional	As per protocol; better tolerated, less efficient recruitment

7. Frequency and Waveform Considerations

Frequency is not simply a dial for "more or less effect" — different frequency bands appear to engage different circuits:

- ~10 Hz: associated with enhanced extensor tone during stepping-related tasks in incomplete SCI.
- ~25–30 Hz: associated with better-coordinated flexor/extensor EMG patterns during locomotor tasks.
- ~50 Hz (the most replicated anti-spasticity parameter across the literature, including the pediatric CP studies in Section 3.2): associated with reduced hyperreflexia and clonus suppression.
- ~50–100 Hz at higher amplitude (2–7V range, ~200 µs pulse width): reported for parameter-dependent tone control when electrodes are positioned over L1–L3.

A separate consideration is kHz-range carrier modulation (5–10 kHz superimposed on the underlying stimulation frequency), developed primarily to improve comfort/tolerability, since conventional tSCS at effective intensities can be uncomfortable, particularly relevant for pediatric use. Evidence suggests kHz-modulated tSCS requires substantially higher charge (~3.8× at posterior root reflex threshold) than conventional tSCS, and engages spinal circuits somewhat differently — it produced spinal inhibition in some upper-limb studies without the same recruitment efficiency as conventional stimulation. A separate narrative review notes that the mechanisms underlying these carrier-frequency effects remain poorly understood. This is a relevant comfort/efficacy trade-off to flag for pediatric protocol design, but it has not been studied specifically in children with tone disorders.

8. Summary

- The anti-spasticity/hypertonia-reduction application of tSCS has the strongest and most consistent evidence base, now including blinded, sham-controlled RCT-level data in SCI and MS, and a small but growing, mechanistically-grounded pediatric CP literature.
- A specific, testable mechanistic target — reciprocal inhibition between antagonist muscles — is measurably impaired in children with spastic CP and is a direct outcome measure in the most

methodologically rigorous ongoing pediatric trial, giving tSCS a more precise rationale than a generic "tone normalizer" framing.

- tSCS is mechanistically distinct from botulinum toxin, baclofen, and TENS — it recruits rather than blocks, and its proposed value is in reorganizing spinal/supraspinal circuits when paired with active training, not in replacing focal or systemic antispastic treatments.
- Safety and tolerability data are reassuring so far — reported adverse events are minor, transient and localized, with no serious adverse events reported in any study to date — but this picture is based on small pilot studies; formal pediatric safety/feasibility trials are still ongoing rather than completed, so pediatric safety should be described as well-supported by early evidence rather than as a settled question.
- Directionality of tone effect (whether a given protocol dampens or facilitates tone) is parameter-dependent — frequency, intensity relative to threshold, montage, and pairing with active training — rather than an inherent property of tSCS as a single intervention.

9. References

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