Active Postural Stabilization in Pediatric Neuromuscular Hypotonia via Ungrounded Reaction Wheel Momentum Exchange

Preliminary Working Draft & Scientific Preprint

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Table of contents

1 Abstract . . . 2
2 Introduction: Active Assist-as-Needed vs. Passive Spinal Bracing . . . 2
3 Pathophysiological Modeling of Pediatric CMT1B (`MPZ` Demyelination) . . . 3
4 Pre-Simulation Real2Sim System Identification & "No Sim Cheating" Discipline . . . 4
  4.1 Zero-Torque Real2Sim Identification Protocol . . . 4
  4.2 "No Sim Cheating" Simulation Discipline . . . 5
5 Control Architecture: Trapezoidal DMI Cueing, APA Intention Gating & 3-Stage Graduated Washout . . . 5
  5.1 Trapezoidal DMI Pulse Shaping (Digital 1-Finger-Rule) . . . 5
  5.2 Dual-IMU Jerk APA & Pelvic Weight-Shift Intention Gating . . . 6
  5.3 3-Stage Graduated Failsafe Washout . . . 6
6 Multi-Granularity Hindsight Telemetry Evaluation (Levels G1–G4) . . . 7
  6.1 Simulation Benchmark Summary . . . 8
7 Conclusion . . . 8
8 Correspondence & Inquiries . . . 8

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This manuscript is an unrefereed preliminary working draft and scientific preprint undergoing formal peer review and clinical evaluation. It documents active research, Software-in-the-Loop (SITL) simulations, and embedded control architectures conducted at Balance Bionics for pediatric postural support. It does not constitute medical advice or a certified medical device specification. Direct technical correspondence and clinical inquiries to torben@balancebionics.com.

1 Abstract

Pediatric neuromuscular disorders—specifically hereditary demyelinating neuropathies such as Charcot-Marie-Tooth Type 1B (CMT1B / `MPZ` mutation), muscular hypotonia, and mild cerebral palsy—severely disrupt axial trunk control during critical neurodevelopmental stages. In demyelinating neuropathies, peripheral motor and sensory nerve conduction velocity drops drastically ($v_c \leq 15\,\mathrm{m/s}$ vs. normal $\geq 50\,\mathrm{m/s}$), producing sensorimotor loop latencies up to 200 ms, coupled with a severe distal-to-proximal muscle weakness gradient ($0.2\times$ ankle vs. $1.0\times$ hip stiffness). Conventional orthotic management relies on passive rigid spinal corsets, which immobilize trunk kinematics, inducing paraspinal disuse atrophy, learned non-use, and secondary deformities. Conversely, abrupt robotic actuators risk triggering spastic startle reflexes or destructive oscillations when applied to latency-delayed neural loops.

Here we present **Mitti**, an untethered, wearable neuro-postural assistive platform utilizing ungrounded direct-drive reaction wheels integrated into an ergonomic pelvic harness. Mitti introduces three key control paradigms: (1) a **Digital 1-Finger-Rule Trapezoidal DMI (Dynamic Movement Intervention) pulse envelope** that delivers smooth tactile micro-cues ($0.5 - 1.5\,\mathrm{Nm}$) without square-wave shock; (2) a dual-IMU **Anticipatory Postural Adjustment (APA) and pelvic weight-shift intention detector** that differentiates voluntary movement ($30 - 80\,\mathrm{ms}$ prior to trunk tilt) to widen control deadbands ($1.6\times$) and suppress counter-torques, while narrowing deadbands ($0.6\times$) upon detecting abrupt stumble precursors; and (3) a **3-stage graduated failsafe deceleration protocol** ($0 - 200\,\mathrm{ms}$ exponential washout, $200 - 500\,\mathrm{ms}$ active zero-torque brake, $500 - 1000\,\mathrm{ms}$ hardware disconnect) ensuring postural equilibrium during faults.

To ensure safety prior to closed-loop therapy, patient-specific MuJoCo digital twins are calibrated using **non-actuated `LOGGING`-mode Real2Sim system identification** with zero active motor torque. Multi-granularity hindsight telemetry evaluation (Levels G1–G4) across extensive Software-in-the-Loop (SITL) physics sweeps confirms up to 34% reduction in posture sway variance during dynamic perturbation without muscle fatigue or orthotic resistance against intentional movement.

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2 Introduction: Active Assist-as-Needed vs. Passive Spinal Bracing

Acquisition of axial postural control is the foundational motor milestone of human motor development. Independent upright sitting, standing, and bipedal locomotion depend directly on the central nervous system's (CNS) capacity to coordinate paraspinal, abdominal, and pelvic muscle groups against gravitational torque. In pediatric neuromuscular pathologies, axial muscular hypotonia and delayed proprioceptive feedback lead to severe postural instability, trunk sagging, and asymmetrical spinal loading.

For over a century, clinical orthopedics has addressed pediatric spinal instability primarily through passive mechanical bracing (e.g., Boston, Milwaukee, or thoracolumbosacral orthoses [TLSO]). Although rigid corsets prevent immediate gravitational collapse and decelerate scoliosis progression, their biomechanical mechanism is fundamentally static: 1. **Mechanical Immobilization**: External plastic shells enforce rigid skeletal alignment, transferring body weight directly through compressive contact pads rather than active muscle contraction. 2.

Learned Helplessness (Learned Non-Use): The central nervous system reduces efferent motor drive to paraspinal muscles because external plastic sustains the gravitational load. Over prolonged wear, this results in progressive disuse atrophy of the erector spinae and multifidus muscles. 3. **Impaired Functional Exploration**: Rigid restriction eliminates the child's natural sensorimotor exploration of their functional stability envelope, impeding proprioceptive adaptation and active balance recovery.

To overcome these fundamental limitations, Mitti pioneers the **Assist-as-Needed (AAN)** active neuro-postural paradigm. Rather than locking the torso in plastic, Mitti provides dynamic, non-restrictive assistive micro-torques only when kinematic deviation exceeds individual physiological thresholds. The tactile cues act as an analog to a pediatric physical therapist's light fingertip stabilization—the "1-Finger-Rule"—stimulating mechanoreceptors and muscle spindles to prompt the child's own muscular self-recruitment.

Crucially, Mitti eliminates external ground-reaction linkages. By mounting high-speed, direct-drive slotless BLDC reaction wheels at the pelvic level, the system exchanges angular momentum ($H = I\omega$) directly with the patient's torso:

$$\tau = I\frac{d\omega}{dt}$$

where I is the flywheel moment of inertia ($\approx 1.25 \times 10^{-4}\,\mathrm{kg} \cdot \mathrm{m}^2$) and ω is flywheel angular velocity (rad/s). This creates localized, pure free-torque couples without crutches, heavy leg linkages, or grounded gantries, permitting full, unencumbered freedom of movement.

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3 Pathophysiological Modeling of Pediatric CMT1B (`MPZ` Demyelination)

Charcot-Marie-Tooth disease Type 1B (CMT1B) is an autosomal dominant demyelinating hereditary motor and sensory neuropathy caused by point mutations in the myelin protein zero (`MPZ`) gene located on chromosome 1q22. In Schwann cells, `MPZ` encodes a major structural transmembrane glycoprotein essential for compacting peripheral myelin sheaths. Pathological mutations impair myelin integrity, leading to segmental demyelination, onion bulb formation, and profound slowing of action potential conduction along peripheral axons.

In healthy pediatric cohorts, peripheral motor nerve conduction velocity (MNCV) exceeds $50\,\mathrm{m/s}$. In children with severe CMT1B, MNCV drops dramatically to:

$$v_c \leq 15\,\mathrm{m/s}$$

often plummeting below $10\,\mathrm{m/s}$. When combined with length-dependent peripheral conduction across sensory afferents (Ia/II spindle fibers) and motor efferents (α-motoneurons), the total sensorimotor closed-loop delay expands to:

$$\tau_{\mathrm{delay}} \approx 200\,\mathrm{ms}$$

(compared to $30 - 40\,\mathrm{ms}$ in healthy children).

Furthermore, length-dependent axonal degeneration produces a pronounced **distal-to-proximal stiffness and strength gradient**. Distal ankle musculature (tibialis anterior,

gastrocnemius-soleus complex) undergoes early denervation and atrophy, whereas proximal pelvic and lumbar core musculature retains relative integrity:

$$k_{\text{ankle}} \approx 0.2 \cdot k_{\text{nominal}}$$

$$k_{\text{hip/trunk}} \approx 1.0 \cdot k_{\text{nominal}}$$

This combination of 200 ms conduction delay and severe distal ankle weakness creates severe control vulnerabilities: * **Inverted Pendulum Instability**: Because ankle stiffness is insufficient to stabilize the center of mass (CoM) via an ankle strategy, the child relies on compensatory hip and trunk strategy. * **Destructive Reflex Resonance**: If an active robotic actuator delivers an instantaneous square-wave torque impulse $\tau(t) = \tau_0 \cdot u(t)$, the child's delayed stretch reflex activates 200 ms after perturbation onset. If the actuator has already de-asserted its torque, the delayed endogenous contraction creates an over-correcting counter-swing, initiating destabilizing limit cycles or startle-induced postural collapse.

Consequently, robotic assistive inputs must be carefully shaped to avoid inducing reflex startle, as detailed in Section 5.

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4 Pre-Simulation Real2Sim System Identification & "No Sim Cheating" Discipline

A primary challenge in deploying active robotic devices in vulnerable pediatric cohorts is parameter tuning. Standard robotic identification algorithms apply energetic chirp or multi-sine torque excitations to extract frequency-response functions. In children with neuromuscular hypotonia or CMT1B, applying unverified active torques risks inducing sudden falls, spinal hyperextension, or distress.

Mitti resolves this ethical and clinical dilemma through a strict **Pre-Simulation Real2Sim System Identification** protocol operating exclusively in non-actuated **`LOGGING` Mode**.

4.1 Zero-Torque Real2Sim Identification Protocol

Prior to closed-loop assistive therapy, the child wears the Mitti harness in non-actuated `LOGGING` mode. The actuator motor phases are completely de-energized (gate driver PWM disabled, zero motor current). The dual-IMU central hub samples tri-axial spine dynamic acceleration, pelvic dynamic acceleration, and angular velocities at 100 Hz.

During a 5-minute unassisted sitting and standing assessment, natural postural sway and voluntary reach exploration are recorded. The unforced trunk dynamics are modeled as a damped inverted pendulum:

$$I_{\text{trunk}}\ddot{\theta}(t) + B_{\text{joint}}\dot{\theta}(t) + \big(K_{\text{joint}} - mgh\big)\theta(t) = 0$$

where: * I_{trunk} is the child's upper-body moment of inertia ($\text{kg} \cdot \text{m}^2$), * B_{joint} is the effective viscous damping ($\text{N} \cdot \text{m} \cdot \text{s/rad}$), * K_{joint} is the passive musculoskeletal joint stiffness ($\text{N} \cdot \text{m/rad}$), * m is upper-body mass (kg), and h is center-of-mass height (m).

By performing recursive least squares (RLS) and spectral analysis on the unforced 100 Hz kinematics, patient-specific values for K_{joint}, B_{joint}, natural frequency ω_n, and sway damping ratio ζ are determined. These empirical values are automatically injected into the MuJoCo

XML digital twin (`simulation/models/humanoid_imu.xml`), establishing an individualized biomechanical model before any active torque is commanded.

4.2 "No Sim Cheating" Simulation Discipline

To guarantee that Software-in-the-Loop (SITL) simulation results translate accurately to physical hardware without sim-to-real transfer gaps, all MuJoCo simulation environments enforce strict physical constraints: 1. **Actuator Saturation & Bandwidth**: Motor torque is capped at $\tau_{\text{peak}} = 1.5\,\text{Nm}$ and $\tau_{\text{cont}} = 0.5\,\text{Nm}$; flywheel rotational speed is capped at $\omega_{\max} = 500\,\text{rad/s}$ (4775 RPM). 2. **Discrete Execution Loop**: Physics steps execute at 1000 Hz, while firmware sensing and control loops run at 100 Hz ($\Delta t = 10\,\text{ms}$) with simulated 2 ms ESP-NOW communication latency and 2% packet drop rate. 3. **Sensor Noise Injection**: Gaussian noise ($\sigma_a = 0.05\,\text{m/s}^2, \sigma_\omega = 0.02\,\text{rad/s}$) and baseline gravity leakage matching ICM-42688-P MEMS IMU characteristics are superimposed on simulated sensors. 4. **Thermal Constraints**: Actuator coil heating is tracked via an empirical thermal RC network; sustained torque must keep surface temperature below 41° C in compliance with IEC 60601-1.

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5 Control Architecture: Trapezoidal DMI Cueing, APA Intention Gating & 3-Stage Graduated Washout

The Mitti firmware control architecture balances rapid stabilization against involuntary stumbles with non-interference during voluntary motor action.

5.1 Trapezoidal DMI Pulse Shaping (Digital 1-Finger-Rule)

To deliver proprioceptive cues without triggering spastic startle or delayed reflex resonance in CMT1B patients (200 ms latency), corrective torque is modulated via a non-blocking trapezoidal envelope per axis (`RAMP_UP` → `PLATEAU` → `RAMP_DOWN`):

```
      Torque (Nm)
         ^
τ_target|           +---------------+
        |          / |               | \
        |         /  |               |  \
        |        /   |               |   \
      0 +-------+----+---------------+----+----------------> Time (ms)
             RAMP_UP     PLATEAU     RAMP_DOWN   REFRACTORY
             (50ms)       (D1)        (50ms)     (T_refr)
```

1. **`TRAP_RAMP_UP` (50 ms)**: Torque rises linearly from 0 to τ_{target}. This gradual rise time is shorter than the 200 ms neural delay but gentle enough to avoid high-frequency mechanical shock:

$$\tau(t) = \tau_{\text{target}} \cdot \frac{t - t_{\text{start}}}{50\,\text{ms}}$$

2. **`TRAP_PLATEAU` (D_1 ms)**: Torque remains isometric at τ_{target} for duration D_1 (typically $100 - 150\,\text{ms}$), providing sustained mechanoreceptor stimulation.
3. **`TRAP_RAMP_DOWN` (50 ms)**: Torque decays linearly back to zero, preserving sign continuity and preventing rebound jerks.

4. **`TRAP_REFRACTORY` ($T_{\text{refractory}}$ ms)**: An un-bypassable quiet interval measured strictly from the end of `RAMP_DOWN` prevents sensory habituation and allows mechanoreceptors to repolarize.

5.2 Dual-IMU Jerk APA & Pelvic Weight-Shift Intention Gating

A critical failing of conventional posture controllers is opposing voluntary patient movement. In normal voluntary movement (such as reaching forward or taking a step), the CNS initiates feedforward **Anticipatory Postural Adjustments (APAs)**: the pelvis shifts weight laterally $30 - 80\,\text{ms}$ prior to trunk movement. Conversely, an unintended stumble or fall lacks this pelvic precursor, displaying sudden linear jerk and differential angular acceleration at the trunk.

Mitti distinguishes these states in real time (100 Hz): 1. **Band-Limited Spine Jerk**:

$$j_{\text{raw}}[n] = \frac{a_{\text{spine}}[n] - a_{\text{spine}}[n-1]}{\Delta t}$$

$$j_{\text{spine}}[n] = \alpha_j j_{\text{raw}}[n] + (1-\alpha_j) j_{\text{spine}}[n-1], \quad \alpha_j = 0.4$$

2. **Differential Angular Acceleration**:

$$\Delta\omega_{\text{roll}}[n] = \omega_{\text{spine, roll}}[n] - \omega_{\text{pelvis, roll}}[n]$$

$$\Delta\dot{\omega}_{\text{raw}}[n] = \frac{\Delta\omega[n] - \Delta\omega[n-1]}{\Delta t}$$

$$\Delta\dot{\omega}[n] = \alpha_j \Delta\dot{\omega}_{\text{raw}}[n] + (1-\alpha_j)\Delta\dot{\omega}[n-1]$$

3. **Pelvic Lateral CoP Proxy**: Gravity-compensated lateral pelvic acceleration a_{lat} is monitored:

$$0.15\,\text{m/s}^2 \le |a_{\text{lat}}| \le 2.5\,\text{m/s}^2$$

When detected, an APA window is latched for 250 ms (`INTENTION_VOLUNTARY_APA`). During this window, the controller deadband is scaled by $1.6\times$ (`deadband_multiplier = 1.6`), actively suppressing counter-torques and granting the child complete freedom to move. 4. **Unintended Fall Precursor**: If high jerk ($|j_{\text{spine}}| \ge 15.0\,\text{m/s}^3$) or buckling angular acceleration ($|\Delta\dot{\omega}| \ge 250^\circ/\text{s}^2$) occurs without a preceding pelvic APA, the system immediately latches `INTENTION_UNINTENDED_FALL_PRECURSOR`, scaling the deadband to $0.6\times$ (`deadband_multiplier = 0.6`) for rapid stabilization before catastrophic tilt ($> 15^\circ$) occurs.

5.3 3-Stage Graduated Failsafe Washout

To ensure patient safety upon communication timeout ($> 100\,\text{ms}$ ESP-NOW loss), sensor anomaly, or thermal excursion, Mitti executes a graduated deceleration protocol rather than an abrupt E-stop torque drop: * **Stage 1 ($0 - 200$ ms)**: Exponential torque washout:

$$\tau(t) = \tau_0 \cdot \exp\left(-\frac{t}{\sigma}\right), \quad \sigma \approx 65\,\text{ms}$$

* **Stage 2 (200 − 500 ms)**: Active zero-torque motor braking, dumping residual flywheel momentum safely. * **Stage 3 (500 − 1000 ms)**: Physical hardware gate driver disable via GPIO disconnect.

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6 Multi-Granularity Hindsight Telemetry Evaluation (Levels G1–G4)

To evaluate therapeutic efficacy and maintain medical device traceability (IEC 62304 / ISO 14971), Mitti establishes a 4-level hindsight telemetry framework:

Level	Name	Temporal Resolution	Core Metrics & Objectives
Level G1	Raw Sensor Dynamics	100 Hz sample-by-sample	Dual-IMU accelerations, angular rates, filtered jerk j_{spine}, differential acceleration $\Delta\dot{\omega}$, and FOC phase currents.
Level G2	Biomechanical Event Segmentation	10 − 50 Hz event windows	Classification of movement states: Quiet Stance, Voluntary APA Window, Trapezoidal DMI Delivery, and Stumble Precursor Events.
Level G3	Real2Sim Counterfactual Simulation	Session batch replay	Replaying recorded patient kinematic logs through the calibrated MuJoCo digital twin to evaluate counterfactual control settings (e.g. alternate deadband multipliers, pulse durations) risk-free.
Level G4	Clinical Neuroplasticity Index	Longitudinal multi-week	Active Intervention Rate (impulses/hour), Postural Stability Quotient, and longitudinal reduction in required assistive

Level	Name	Temporal Resolution	Core Metrics & Objectives
			torque, demonstrating endogenous motor recovery.

6.1 Simulation Benchmark Summary

In extensive MuJoCo SITL benchmark sweeps across simulated pediatric subjects with CMT1B (200 ms latency, $0.2 \times$ ankle stiffness) subjected to lateral impulse perturbations ($0.5 - 1.5$ Nm): 1. **Sway Variance Reduction**: Active Trapezoidal DMI stabilization achieved a **34.2% reduction in coronal sway variance** compared to passive baseline (`LOGGING` mode). 2. **Absence of Reflex Oscillation**: While step-function torque pulses induced secondary oscillations in 78% of trials due to 200 ms reflex delay, Trapezoidal DMI envelope pulse shaping eliminated reflex overshoot in 100% of trials. 3. **Zero Resistance During Voluntary Action**: In voluntary reaching trials preceded by pelvic weight shift, APA intention gating prevented counter-torque application in 98.4% of voluntary attempts, verifying that assistive intervention remains strictly non-restrictive.

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7 Conclusion

Mitti demonstrates that active, ungrounded reaction wheel micro-torques paired with bio-inspired intention gating and trapezoidal pulse shaping offer a compelling, active alternative to passive pediatric spinal bracing. By tailoring control dynamics to the 200 ms neural latencies and distal stiffness gradients of demyelinating neuropathies such as CMT1B, and calibrating digital twins via non-actuated Real2Sim identification, the system establishes a safe, neuroplasticity-enhancing platform for pediatric neurorehabilitation.

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