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"Postural Alignment First, Symptom Tailored" Integrated Pathway as a Multimodal Peripheral Intervention Strategy for Pain and Cervical Disease (Part 1)

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Abstract

Conventional treatments for chronic cervical diseases often rely on symptom-targeted interventions such as pharmacologic analgesia and neurointervention, while upstream biomechanical and neuromodulatory imbalances remain under addressed.

This review introduces the "Postural Alignment First, Symptom Tailored" (PAST) Integrated Pathway-a novel, structured, multimodal framework that prioritizes postural realignment before symptom-based therapy.

The PAST pathway operates through a three-stage, five-tier model built on the principle of "Structure First, Functional Integration, and Tailored Intervention." It targets postural-fascial-neural dysfunctions contributing to pain, dizziness, tinnitus, and sleep disorders. Key focuses include: 1) the central neurophysiological mechanisms of postural control; 2) dysfunctions of proprioception and autonomic regulation; 3) biomechanical-autonomic coupling patterns; and 4) hierarchical classification of peripheral stimulation techniques.

By incorporating multimodal assessment tools and closed-loop feedback systems, the PAST pathway provides a structured approach that bridges the structural-functional divide in neuromusculoskeletal management. It presents a low-risk, scalable model with strong translational potential in perioperative and chronic pain care.

Keywords

Pain, Cervical diseases, PAST pathway, Multimodal evaluation system, Hierarchical management process

Introduction

The relationship between postural dysfunction and pain/neurological disease

Postural control is not only about static aesthetics and movement performance, but also the core element of maintaining the balance between musculoskeletal-neurological load and pain regulation.

In the process of human upright evolution, the "tension continuous frame" composed of the skull, thorax, pelvis and lower limbs, places the earlobe, acromion, anterior superior iliac spine and lateral malleolus in a straight line in the sagittal plane, which is considered the anti-gravity posture with the lowest energy and most stable mechanics [1].

However, the static trend of modern lifestyles (prolonged desk work and screen use) leads to a combination of postural abnormalities: forward head posture (FHP), increased thoracic kyphosis and anterior pelvic tilt. These changes disrupt the structural biomechanical balance. FHP can increase cervical shear forces, cause persistent neck flexor elongation and lead to compensatory overactivation of the shoulder girdle muscles. Ultimately, this results in chronic myofascial tension, ligament strain and local ischaemia, which activate peripheral pain receptors, leading to acute or chronic pain [2].

At the muscle-fascia level, FHP places the deep neck flexors (longus capitis, longus colli) in a state of prolonged stretch, reducing muscle spindle sensitivity and weakening cervical spine stability. It also activates superficial muscles such as the upper trapezius and levator scapulae, creating a 'pain-tension-dysbalance' cycle [3]. Studies show that the average FHP (craniovertebral) angle in patients with chronic neck pain is approximately 5.3-8.2 degrees lower than in healthy people, and there is a significant positive correlation between FHP severity and Visual Analogue Scale (VAS) scores (r = 0.45-0.62) [4].

At an epidemiological level, a multinational data analysis of over 40,000 samples has shown that sitting for more than 6 hours a day with poor posture (including rounded shoulders and head forward) increases the risk of neck and shoulder pain by 82% and is closely associated with chronic pain in other areas such as low back pain and migraine [5]. This type of posture-related chronic pain has been identified as a core etiological factor in the 'musculoskeletal pain spectrum', which is beginning to replace the traditional 'single-point injury model'.

Therefore, acute and chronic episodes of neck, shoulder, low back and knee pain should be included in a comprehensive assessment of postural chain and muscle control strategies. Correcting postural imbalance at its source can not only reduce the frequency of pain episodes, but also significantly improve the long-term maintenance rate of the intervention.

The central mechanism of neck postural imbalance and polysystemic symptoms of cervical origin overlap

Recent studies have also shown that cervical disorders are also closely related to postural imbalance, and the core pathological basis is the dysfunction of the coupling between neck proprioception and the central arousal regulatory network [5-7].

Cervicogenic vertigo (CGD): Studies show that the main pathological basis is the abnormal input of neck proprioception and the decoupling of the central integration system (especially the vestibular-cerebellar network). In patients with chronic neck pain, the reactivity of the cerebellar tonsil (fastigial nucleus) to multimodal proprioceptive and vestibular signals during task states is reduced, leading to delayed postural adjustment and increased balance fluctuations [6]. This process not only manifests as dizziness and gait instability, but is also closely related to the level of arousal regulated by the reticular formation of the vestibular system and the microcirculation of the cochlea [7].

Further evidence suggests that "vestibulo-reticulo-collic pathway dysfunction" is the common central basis for various "cervical non-specific symptoms". This network plays a central role in the regulation of gravity adaptation, muscle tone and arousal thresholds. Once the perceptual input is misaligned, due to abnormal posture or deep cervical muscle fibrosis, this can lead to excitation of the locus coeruleus and increased sympathetic activity, which manifests as fragmented sleep, nocturnal awakenings and subjective difficulty falling asleep - known as non-idiopathic sleep disorders [8]. In addition, some patients experience low-frequency tinnitus associated with sympathetic otic microcirculation, a pathological pattern known as cervicogenic tinnitus [9]. Therefore, CGD is not just a simple "vestibular imbalance", but also an expression of a coupling disorder between the cerebellum, vestibule and reticular system. It is only through precise postural proprioceptive regulation that the abnormal firing mode of this cross-coupling pathway can be disrupted and the symptoms completely alleviated.

Cervicogenic headache (CEH): A disorder of posture, muscle and nerve coupling: CEH is a chronic disorder that originates in the cervical spine (particularly the C1-C3 segments) and is perceived as a headache along the trigeminal nerve pathway. Extensive imaging and electromyographic studies have shown that head extension (forward head posture) leads to compensatory activation of superficial muscle groups such as the upper trapezius, levator scapulae and sternocleidomastoid, while reducing tension in deep neck flexors (such as the longus colli), resulting in neural compression and proprioceptive distortion [3,10]. The facet joints and associated soft tissues of the C1-C2 segment provide the anatomical basis for cervicogenic headaches.

In addition, the asymmetric muscle tension caused by FHP can be transmitted via the spinal trigeminal nucleus-supraoptic system to the thalamus and central pain modulation areas, causing "central sensitisation" of chronic pain [11]. Postural intervention studies (such as postural re-education combined with deep neck flexor training) have been shown to significantly reduce CEH symptoms and improve headache intensity and frequency.

Sleep disorders associated with cervical spine disease: the intersection of antigravity imbalance and wakefulness regulation: Sleep disorders associated with cervical spondylosis refer to chronic sleep disturbances caused by postural and neural network imbalances in the cervical spine. The main pathophysiological mechanisms include: fibrosis of the musculus longus colli and anterior scalene muscles compressing the sympathetic chain in the neck, leading to a sustained increase in sympathetic tone at night (manifested as an increase in the LF/HF ratio of HRV), which is significantly associated with fragmented sleep and abnormally prolonged REM duration [12]. Cervical pain itself may activate the locus coeruleus-hypothalamic arousal system, increasing micro-awakenings and disrupting sleep structure, rather than simply manifesting as 'difficulty falling asleep'. Functional imaging studies have shown that individuals with FHP show a significant reduction in cerebellar vermis and dorsal raphe nucleus activation during task states, affecting sleep position regulation and proprioceptive-cerebellar coordination, suggesting a direct central pathway linking postural regulation function to sleep quality [13]. It should be emphasised that this type of sleep disorder is often misdiagnosed

as "anxiety insomnia" or "primary insomnia", making monotherapy with conventional sedatives (such as benzodiazepines) ineffective. If the status of the kinetic chain and sympathetic tone is not corrected first, routine hypnotics (such as benzodiazepines) may mask upstream abnormalities and chronic insomnia may persist.

Common Mechanisms of Postural Disorders: Current research generally agrees that the vestibulocerebellar-cervical muscular network plays a role as an "amplifier of dysregulation" in various cervical disorders. This network coordinates the body's perception of gravity (from the semicircular canals), head and neck position (from the somatosensory system) and arousal state (from the reticular system). When posture becomes unbalanced or muscle groups compensate, the closed loop of sensory-motor-neurological regulation is disrupted. The above indicate the occurrence of several related symptoms: cervical vertigo, headaches, tinnitus and sleep disturbances may occur alternately or simultaneously [7,14].

Problems with current diagnosis and treatment

Limitations of the current conventional approach: Symptom orientation *vs*. postural origin: Currently, conventional treatment strategies for cervical spondylosis and chronic somatic pain still focus on "symptomatic pain relief", integrating non-pharmacological analgesia, neurointerventional procedures and multidisciplinary physiotherapy. However, while these interventions may alleviate pain and suppress inflammatory pathways in the short term, they often overlook potential upstream causes: -- postural imbalance and [15] neuromuscular dysregulation. This 'source neglect' leads to difficulties in achieving long-term stable therapeutic outcomes, with recurrence rates as high as 30-50%.

Further research suggests [16] that static postural deviation, abnormal pelvic tilt and inadequate deep neck flexor (DNF) function may synergistically induce 'sympathetic sensitisation' in conjunction with a hyperactive autonomic nervous system. This is manifested by an increased LF/HF ratio in HRV (heart rate variability), which increases pain input and arousal load, thereby promoting chronic disease progression. In addition, while neurological interventions or pharmacological treatments may be effective for some patients, they do not fundamentally restore the imbalance in the coupling between the 'kinetic chain and the neural network'. As a result, symptoms often return after the intervention is removed.

The future paradigm of diagnosis and treatment urgently needs to shift to a 'mechanism integration orientation', i.e. restoring the balance of postural tension and the basis of neuro-fascial plasticity prior to pain control, in order to truly break the chain of disease progression.

Drug therapy: A systematic review shows that the analgesic effect of non-steroidal anti-inflammatory drugs (NSAIDs) for chronic neck and shoulder pain typically occurs within 2 to 6 weeks, but there is no clear evidence of functional improvement beyond 12 weeks [17]. In addition, the gastrointestinal (e.g. ulcer bleeding), cardiovascular (e.g. thrombotic events) and renal risks associated with NSAIDs limit their long-term use. Although opioids can rapidly reduce visual analogue scale (VAS) scores in acute exacerbations, their long-term use has been shown to be associated with dependence, increased tolerance, cognitive impairment and sleep disturbances (such as REM suppression and increased awakenings), and clinical guidelines no longer recommend their long-term use for chronic non-cancer pain [18].

Interventional treatment: precise block and repeatable radiofrequency: For chronic neck and shoulder pain, interventional treatments such as selective nerve root blocks (SNRBs) and transforaminal epidural injections can provide short-term relief. Systematic reviews show that selective injections at levels [19], C6-T1 can relieve radicular pain for 2 to 12 weeks and improve functional scores such as NDI, but their analgesic benefits typically diminish after 6 months, suggesting the need to combine them with functional exercises to maintain efficacy.

Radiofrequency ablation (RFA) is a common treatment for chronic small joint pain. A double-blind RCT confirmed that mid-branch radiofrequency can maintain a relatively stable reduction in VAS for 3-6 months after surgery, but the effect varies widely between individuals and there are potential complications (such as head-down syndrome, accelerated degeneration of adjacent segments)[20].

However, all current interventions are mainly focused on short-term symptom control and fail to fundamentally correct the upstream causes, such as the imbalance in the posture-power chain. Therefore, it is recommended to prioritise the evaluation of interventional indications after postural alignment has been achieved to avoid structural-functional disconnection.

Physical/rehabilitative treatment: postural re-education and musculoskeletal integration: Compared with localised muscle strengthening exercises, Global Postural Re-education (GPR) and the Modified McKenzie Method have shown superior efficacy in improving chronic neck pain. High-quality randomised controlled trials suggest that GPR can effectively restore muscle chain tension and sagittal alignment. Training over 10 weeks significantly reduced pain scores, increased cervical range of motion and improved dynamic balance [21] while standing.

In addition, coordination of the foot-pelvis-spine-cranium kinetic chain has been shown to be closely related to upper limb load transfer pathways and neck strain. Biomechanical studies indicate that lower limb alignment imbalances (such as excessive pronation and hip internal rotation) significantly increase the incidence of shoulder and neck pain [22], suggesting that an overall alignment strategy should include foot support, pelvic tilt regulation and thoracic expansion intervention.

Vestibular-proprioceptive integration training has also been incorporated into the management protocol for cervicogenic vertigo [23], aiming to improve centre of gravity control by strengthening deep neck muscles and the eye-head-trunk coordination mechanism. A prospective study showed that vestibular training can reduce patients' Dizziness Handicap Inventory (DHI) scores by approximately 19 points and decrease balance area by up to 30%, suggesting an empirical basis in dynamic stability and subjective symptom control [23].

Acupuncture, dry needling and traditional techniques: Systematic reviews have shown that acupuncture has moderate- to high-strength evidence for the adjuvant treatment of chronic neck pain [24], with analgesic and functional improvement effects lasting at least 3 months and good safety.

Dry needling is beneficial for myofascial trigger point activation and short-term pain relief, particularly for cervicogenic headache [25], but its additive effect as an adjunct to routine rehabilitation is not clear, suggesting that precise indications should be evaluated.

Craniosacral therapy (CST) is a manual therapy that has shown moderate benefits for improving cervicogenic headache and sleep quality in some randomised controlled trials [26], but the overall quality of the evidence is not high and further large-sample studies are needed to confirm this.

The Current Diagnostic and Treatment Model Ignores the Causes of "Postural, Neural and Fascial Imbalances"

Single point thinking based on pain thresholds leads to a disconnect between structure and function: Most disciplines still focus on peripheral pain perception and the inflammatory signalling cascade ("analgesia-inflammation-blockade-rf" chain), emphasising downstream pain nerve blockade, while neglecting the "posture-proprioception-fascial network" that can continuously amplify nociceptive input. For example, epidural steroid injections (ESIs) can reduce pain in the early stages [27], but their effects diminish rapidly after three months and offer no improvement in postural parameters.

Recent cross-sectional studies have shown that in patients with chronic non-specific neck pain, vagal activity significantly decreased and the HRV LF/HF ratio increased when standing [28]; this autonomic abnormality was positively correlated with neck pain intensity, functional impairment and cervical spine mobility [29], suggesting that postural imbalance enhances the pain memory circuit through sympathetic upregulation.

Lack of perspective on the chain of forces: The human body's "foot pelvis spine skull" forms a continuous tension frame; the shift in lower limb alignment can alter cervical stability through pelvic tilt and spinal curvature. A systematic review of over 100,000 samples in 2024 [30] showed that excessive internal rotation of the lower limbs and excessive foot inversion significantly increased the risk of low back pain (OR 1.42 to 1.88), highlighting the biomechanical effects of lower limb imbalance cascading upwards.

At the same time, patients with neck pain have impaired gait and balance indicators, confirming the dynamic chain coupling between the neck, pelvis and lower limbs [31]. However, current guidelines rarely require a cross-regional assessment of the impact of the feet or pelvis on cervical spine disease, resulting in uncorrected underlying tension and easy recurrence of symptoms.

Low sensitivity to autonomic coupling: Postural imbalance may continuously increase sympathetic activity, manifested as an elevated HRV LF/HF ratio; however, HRV monitoring has not been incorporated into routine neck pain assessment. Comparative studies show that in patients with chronic neck pain, HRV indicators are significantly correlated with pain intensity and cervical spine mobility [28], yet this reversible physiological window is often overlooked in clinical practice.

Sleep neglect of sympathetic coupling: Cervical postural imbalance can continuously activate the sympathetic chain [32], manifesting as increased LF/HF HRV and decreased nocturnal vagal tone; failure to investigate cervical sympathetic coupling is often misdiagnosed as "primary insomnia" or "anxiety disorder", leading to the sole use of sedatives and hypnotics. Experimental studies have confirmed that partial sleep restriction (5 hours per night) can significantly reduce HF power and increase LF/HF, making it more likely to induce nocturnal sympathetic overactivity compared to fragmented wakefulness; large cohorts of nurses also found a moderate negative correlation between [33], subjective decline in sleep quality, and increased low-frequency HRV and LF/HF ratios. If the cervical dynamic chain imbalance is not simultaneously corrected, monotherapy alone will not reverse the autonomic tension imbalance, but will mask the cause and delay the timing of intervention.

Lack of multidisciplinary quantitative indicators: Current guidelines still rely primarily on imaging findings of nerve compression or disc degeneration for disease classification. However, this "structure-dominated" approach overlooks dynamic chain parameters and autonomic nervous system indicators that are closely related to functional status. For example, [34] anterior cervical lordosis (FHP), deep neck flexion endurance (DNF) and heart rate variability (HRV) have independent predictive value in predicting pain intensity and disease duration. Studies show that even in the absence of significant MRI abnormalities, patients may experience persistent pain due to imbalances in FHP and HRV [31-33]. Some patients with obvious cervical degeneration may not report symptoms, representing a "symptom-structure mismatch" phenomenon [34]. Therefore, there is an urgent need to incorporate FHP angles, DNF endurance and HRV metrics into the assessment pathway, reconstructing a cross-system, multi-dimensional functional assessment framework to more accurately identify abnormalities in the postural-neuromuscular-fascial-autonomic coupling.

Taken together, these findings suggest that fundamental relief of cervical conditions cannot be achieved by relying solely on pain relief or local intervention without a holistic approach to the autonomic sleep of the motor chain.

Clinical consequences of inadequate assessment and intervention of "postural, neural and fascial imbalances" in the current diagnostic and treatment modality

Prolonged pain and high recurrence rates: The current model of intervention, which focuses on pain relief, often fails to include restoration of sagittal balance and kinetic chains, resulting in difficulty in maintaining long-term pain relief [35]. Studies show that the recurrence rate of conventional drug and physiotherapy approaches is as high as 35-50% within 6-12 months of discontinuation [31-35]; however, interventions incorporating Global Posture Re-education (GPR) have shown potential to reduce the recurrence rate of pain [36].

Multiple system symptoms "specialisation" leads to overlapping treatment: Cervicogenic dizziness (CGD), primary insomnia and intractable tinnitus are often managed by different specialties (e.g. neurology, psychiatry, otolaryngology) and receive concurrent interventions such as analgesics, hypnotics, antidepressants or nerve blocks [37]. Multidisciplinary care can help reduce medication use and associated adverse effects. If these conditions are uniformly incorporated into the postural-sympathetic coupling model of early intervention, clinical medication requirements may be reduced and overall compliance improved.

Increasing economic burden and social costs: Frequent and repetitive neurointerventions (such as epidural injections and radiofrequency ablation) and long-term medication costs impose a significant economic burden on patients and the health care system. Health economic simulations have shown [38] that each reduction in pain recurrence over a 2-year period can save treatment costs and work loss. This underlines the urgency and economic rationale for incorporating 'posture-neuromuscular-fascial integration' at an early stage in the intervention pathway.

Neurophysiological mechanisms of postural maintenance

Mechanical goals and framework: The 'upright-chest-column' posture is not only aesthetically significant, but also the least energy consuming anti-gravity structural configuration that has evolved in the process of human upright evolution. The mechanism for maintaining this posture has the following anatomical and biomechanical advantages

Optimisation of cervical-cervical alignment: Studies have shown that the biomechanical properties of the cervical spine differ between the sexes, which may affect neck loading and postural stability [39].

The relationship between spinal motion and thoracic function has been shown to influence respiratory function and postural stability [40].

Continuous tension along the lower limb-pelvis-cranium axis: The pelvis is held slightly forward and the alignment of the lower limbs (calcaneus-fibula-femur-sacrum-occipital bone) forms a structural system similar to a 'tension column', allowing the ground reaction force to be transmitted upwards along the fascia-bone continuum, avoiding local overloading or cross-compensation [40].

This body shape is the evolutionary product of "energy conservation-shear resistance-load distribution", and maintaining its stability is an important mechanical basis for alleviating chronic cervical disease and pain.

Neurophysiological Tertiary Loop

Multimodal sensory coupling: millisecond error detection: The human body monitors postural deviation through four channels: vision, vestibular, proprioception and fascial tension.

1) The mechanism of multimodal sensory coupling is at the heart of maintaining human postural stability. Postural perception does not rely on a single sensory pathway, but consists of a 'postural error correction network' that integrates information from multiple sources, including visual, vestibular, somatosensory and myofascial tension receptors. This process of information integration can achieve sub-second or even millisecond accuracy:

2) Vestibular-cervical-visual coordination: The

vestibular system plays a key role in postural control and its reaction speed is crucial for maintaining balance [41]. At the same time, proprioception of the neck muscles may influence postural control and sympathetic nerve activity through fast neural pathways [42].

3) Fascia-myosacral tension integration: Fascia may be involved in postural control and movement regulation through receptors and neural pathways, and may be involved in the rapid coordination of thoracic tension in respiratory movements, suggesting that the fascial system not only participates in the stretch sense, but also assumes the function of motor reflex regulation [43].

Visual weight and external reference: The visual system plays an important compensatory role in postural control, which can be weighted when sensory information is inconsistent, as an important compensatory pathway for standing balance [44].

This "millisecond time window + multimodal synchronisation" mode is the physiological basis of advanced human postural control and provides a neural basis for perception-posture-movement training in the PAST pathway.

The brainstem-cerebellum-cortex tri-level integration network: the central feedforward regulator of postural control (**Table 1**)

The maintenance of the "upright-chest-column" antigravity posture relies not only on peripheral feedback pathways, but also on the feedforward motor intention integration mechanism of the brainstem-cerebellum-cortex triad (anticipatory postural adjustment, APA). The system consists of three closed-loop regulatory axes:

1) Vestibulo-cerebellar loop (VCL)

After entering the lateral vestibular nucleus (LVN) from the semicircular canals, vestibular signals are differentiated along two pathways:

On the way up, it reaches the fastigial nucleus of the cerebellum and forms a closed-loop prediction module;

On the way down, the external vestibular spinal tract (LVST) directly regulates the extensor alpha motor elements (α-MN) of the spinal anterior horn.

The fastigial LVN circuit can be activated approximately 100-150 ms before the occurrence of the movement intention, and the activation threshold of the thoracic dorsiflexors and extensors can be adjusted in advance, significantly improving the accuracy of the feedforward response of the anti-gravity posture [45].

In addition, the LVN and the gigantocellular reticular nucleus (GRN) of the brainstem have an inhibition-depression mechanism that jointly determines the dynamic recruitment threshold of the α-MN in the thoracic and lumbar segments. This mechanism can achieve a dynamic balance between maintaining postural stability and action transfer [46].

Table 1: The brain regions/spinal segments directly related to posture control.

Rank	Primary Structure	Features	Key Pathways
Cortex	Auxiliary motor area (SMA), frontal-parietal posture network, primary motor cortex (Brodmann 4) [61]	Postural intention and anticipatory postural adjustment (the command centre for conscious postural adjustment activates the anterior horn cells of the C5-T1 spinal cord to control the shoulder and neck muscles).	Cortico-reticular bundle
Epencephal	Ventral, dorsal nucleus, vestibular cerebellum [48,62]	The error is predicted, the extensor muscle tone is coordinated and the contraction strength of the anti-gravity trunk muscle groups (erector spine and trapezius) is adjusted in real time.	Top nucleus-vestibular nucleus-LVST
	Basal ganglia [63]	Automate the movement programme to create unconscious postural habits.	Nigrostriatum
Brainstem	Lateral/inferior vestibular nuclei, giant cell reticular nucleus [64]	Postural balance reflex, muscle tone regulation (integrating balance information from the inner ear, increasing neck and back extensor muscle tone through the vestibular spinal tract and maintaining upright posture against gravity);	LVST, MVST, RST
	[63,64] Brainstem reticular formation	It is responsible for regulating muscle tone, transmitting postural maintenance signals to the spinal cord through the reticular spinal tract, and inhibiting excessive flexor tension.	
Medulla Spinalis	C1-C3 (deep neck muscles), C3-T6 (thoracic and dorsal intrinsic muscles), L2-S1 (pelvic and lower limb extensor muscles), T6-L1 (basic rhythmic control network for postural maintenance) [51,65].	Postural pattern generator, anti-gravity muscular element; basic rhythm control network for maintaining posture, forming feedback loop with brainstem.	γ-loop, Ia/Ib-front Angle integration

2) Cortico-reticulospinal loop

Postural motor intentions are projected downwards from the prefrontal-isoloid-cingulate cortex via the dorsal and ventral reticulospinal tracts (dorsal/ventral RST):

At the same time, it increases the drive of the axial extensor muscles;

It also inhibits the reflex excitability of the contralateral flexor muscle group.

The TMS-MEP study confirmed that the activation of the RST pathway was significantly earlier than that of the corticospinal tract by about 120 ms, which is a typical source of APA initiation signals [47].

3) Cooperative vestibular-reticular discharge mechanism

During high demand antigravity movements such as "quick head lift" or "chest extension", synergistic discharges between LVN and GRN may occur in conjunction with RST output from the lumbar extension segment, triggering a compound discharge wave (gamma burst) driven by γ motor neurons. This mechanism can increase thoracic and back extensor muscle tension by over 20% in a very short time, resulting in a rapid and stable upright response [48,49].

Spinal Postural Pattern Generator (PPG)

In the "chest-up-column" posture, the spinal cord not only acts as a conduction centre for downward commands, but also has the ability to generate autonomous postural patterns. Studies have shown that

The C1-C3 segments control the deep neck flexors and head extensors, which can be regulated by rapid perception and reflex to coordinate head position control;

The C3-T6 segment contains intersegmental horizontal connections between the anterior horn motor elements of several thoracic back extensor muscles (such as the multifidus, sacrospinalis and longus). Under input from the anterior lateral system (ALS) and the reticulospinal projection, it can achieve rhythmic discharge patterns that control mild extension of the thoracic vertebrae and external rotation of the manubrium of the sternum, thereby forming the typical 'chest thrust' posture [48,50].

Based on studies of animal models and human transcutaneous/dural spinal cord electrical stimulation (tSCS/eSCS), rhythmic field potentials locked to the standing phase have been recorded in the C3-T6 region, suggesting an independent mechanism for postural schema generation, independent of cortical drive, defined as the "spinal postural pattern generator (PPG)" [51].

More importantly, in the state of transient cortical inhibition, PPG activation by spinal cord electrical stimulation can still maintain baseline extensor muscle tone, further confirming its function as a semi-autonomous central pattern generator (CPG) and may be involved in the neural implementation of the "posture-first" stage of the PAST pathway [52].

Autonomic nervous endocrine coupling mechanism: neural pathway of postural regulation and muscle group activity: During the maintenance of the antigravity posture, the autonomic nervous system not only regulates the circulatory and metabolic states, but also directly influences the mechanical properties of the trunk extensor muscles and the respiratory rhythm. Postural maintenance relies not only on the passive structure of the musculoskeletal system, but also on the coordinated regulation of the autonomic and endocrine systems. Recent studies have shown that mild sympathetic excitation (an increase in plasma norepinephrine [NE] of about 10-20%) can activate the sympathetic-adrenal axis, which, through the action of β_2-adrenergic receptors on skeletal muscle membranes, upregulates actin myosin, upregulates actin-myosin, upregulates actin-myosin (F-actin-myosin) ATPase activity, increases fatigue resistance and isometric tension holding capacity of the extensor muscles, while increasing baseline levels of fascial tension to maintain a 'chest-up stance' [53-55].

Increased parasympathetic-vagal activity can stabilise thoracic volume and ventilatory efficiency by inhibiting excessive contraction of the intercostal muscles and thorax, thus preventing 'chest locking' caused by anxiety or shallow breathing and helping to maintain coordinated postural and respiratory regulation [56]. Maintaining a forward head posture and thoracic flexion for prolonged periods (such as over 6 hours of daily office work or screen time) can lead to a sympathetic-parasympathetic imbalance, with an increased LF/HF ratio in nocturnal HRV of at least 30%, indicating a trend towards sympathetic dominance, and showing a significant positive correlation with subjective neck pain scores (VAS) and sleep fragmentation (PSQI) [57].

This mechanism explains why it is necessary to first reconstruct the baseline of the postural-autonomic nervous coupling in order to avoid a secondary functional disconnection caused by "over-analgesia/sedation".

Plasticity regulation and the pathogenesis of chronic postural disorders: Long-term postural abnormalities not only result in mechanical imbalance, but also lead to a plasticity reconstruction disorder of the brain-cerebellum-spinal cord-muscle spindles circuit, which is the underlying central mechanism of many chronic cervical disorders.

1) Vestibular-cerebellar-spinal cord loop plasticity decreased in smartphone addicts

Functional Magnetic Resonance Imaging (fMRI) and Transcranial Magnetic Stimulation (TMS) studies have shown [58] that the postural control circuit can be significantly affected when the forward bending posture is maintained for more than 4 hours daily for a period of 12 weeks: Synaptic post-inhibition between the cerebellar peduncle (fastigial nucleus) and the vestibular nuclei (LVN) decreases by about 25%; the reticulospinal tract (RST) shows increased inhibition in the posterior spinal cord, leading to an increased threshold for postural error feedback; the area representing the thoracic and dorsal muscles in the motor cortex is significantly reduced, characterised by delayed recruitment of electromyographic signals and increased contraction thresholds. These changes suggest that prolonged head-down posture may induce a functional hypo-reactive state centred on the reorganization of cerebellocortical representational maps.

2) Severe thoracic kyphosis and muscle spindle degeneration

Imaging and muscle biopsy studies have shown that in patients with severe thoracic kyphosis, the trapezius and erector spinae muscles show the following changes: decreased muscle spindle density; and a significant reduction in the sensitivity of γ motor neurons (γ-MNs) to muscle spindle tension receptors and receptor density [54-59]. This suggests that degeneration of peripheral proprioceptors may inversely weaken the central perceptual map, leading to impairments in bidirectional plasticity in the "posture-muscle spindle-cortex" loop.

3) Risk of misdiagnosis of cervical sympathetic chain compression and sleep disorders

Chronic fibrosis of deep muscle groups such as the longus capitis and trapezius, together with adhesions in the sympathetic chain (communicating sympathetic trunk), can compress sympathetic afferent pathways: an increased LF/HF ratio in nocturnal HRV (≥ 2.5) leads to decreased sleep efficiency, fragmented REM sleep and a lowered pain threshold, often misdiagnosed as primary insomnia or anxiety disorders. Studies have shown that following interventions such as postural reconstruction (e.g. PAST path), HRV metrics and sleep efficiency can return to near baseline levels within 4 weeks, indicating a high degree of reversibility of autonomic nervous function [60].

Foot-pelvis-spine-cranium dynamic chain: an upward stable base with multi-segmental tension coupling

Lower limb alignment determines upward tension conduction: A meta-analysis (n ≈ 102,000) [66] shows that excessive foot pronation (overpronation) and hip internal rotation are significantly associated with the risk of low back pain. This suggests that altered lower limb alignment, through pelvic tilt and lumbar rotation, may create 'stress hotspots' in the spinal tension chain, ultimately weakening cervical stability. Sagittal plane gait intervention and anterior pelvic tilt correction can effectively reverse this chain tension migration process.

Pelvic-thoracic coupling promotes longitudinal alignment [67]: The anterior pelvic tilt of 5°-10° is considered to be the best anatomical state for synergistic extension of the thoracic vertebrae and external rotation of the sternal handle, which helps to construct the cranio-sacral sagittal symmetry structure. It has been found that there is a coupling relationship between the posture of the thoracic spine and the flexibility of the lower limbs.

The remote redistribution of fascial spiral tension along the thoracic-pelvic chain increases the flexibility and stability of the lower limbs.

Fascial spiral and cranio-cervical stability coupling [68]: The Ruffini terminals in the thoracic and dorsal fascia are important tension sensors; changes in their compliance can directly regulate the accuracy of vestibulo-cerebellar error prediction. Studies have shown that lower limb orthoses (e.g. improved FPI6 scores), when coordinated with thoracic extension, can increase the sensitivity of the fascia-cerebellar proprioceptive feedback pathway, thereby improving the ability to fine-tune head position and posture.

Evidence for neural plasticity and multimodal intervention: Multimodal postural sense calibration (such as closed-chain visual-vestibular-neck training) can reduce the centre-of-gravity-foot pressure projection offset by approximately 35% within 4 weeks, showing significant efficacy for cervicogenic vertigo [69]. Another randomised controlled trial confirmed that [70], combined with isometric training of the deep cervical flexors over 10 weeks, can reduce NDI scores by 14 points, significantly better than single-site training. In addition, percutaneous electrical stimulation applied to the C3-T6 spinal segments can restore sitting balance without cortical drive, suggesting that this region may provide the neural "base" for postural control. In a 12-week integrated orthopaedic intervention, the comorbidity rate of low back and neck pain decreased by approximately 38% (RR ≈ 0.62), and HRV LF/HF also decreased in parallel, suggesting that the autonomic nervous system also benefits from the rebalancing of the force lines.

Clinical and scientific implications: From symptomatic pain relief to closed-loop assessment of the structural-functional nervous system

Need for improvement in assessment dimensions: Current clinical assessment of chronic pain and cervical

spine disorders often focuses on subjective scales (such as VAS, NDI). However, an increasing number of studies suggest that single dimensions struggle to capture "upstream mechanisms" such as multisegmental tension coupling and autonomic nervous system imbalance. Therefore, it is essential to systematically introduce the following assessment modules: lower limb alignment parameters (such as sole pressure distribution, foot-knee axis deviation); pelvic anteversion angle and spinal sagittal plane balance indicators (such as SVA, PI-LL mismatch); thoracocervical dynamic alignment (such as displacement angle, movement symmetry); and autonomic nervous system indicators (such as LF/HF ratio in HRV) to assess the state of sympathetic-vagal coupling [71]. These quantitative dimensions form a multidimensional data support for the 'posture-movement-neural axis'.

Multimodal transformation of the intervention paradigm: In the acute phase, 'drug/neurointervention' can quickly block abnormal discharge pathways. However, for patients in the chronic phase, the focus should be on functional recovery and neural plasticity. The following combined intervention strategies are recommended: postural strength chain training (such as GPR, pelvic reconstruction, closed chain training); sensory rebalancing (such as vestibular-visual-proprioceptive integration training); neuromodulation techniques (such as tSCS, HRV resonance training) [72]. Studies have shown that these combined approaches significantly reduce pain recurrence rates, promote autonomic recovery and improve quality of life [68,73].

Future direction: intelligence and closed-loop remodelling

With the development of wearable technology, the fusion of an inertial measurement unit (IMU) + surface electromyography (sEMG) + HRV module has become the basis of an individualised postural neurological monitoring platform:

It can track the recruitment of movement units and postural compensation in real time, support feedback adaptation during training, and construct a neuro-biomechanical closed-loop control system. In the future, such platforms are expected to provide an 'individual postural-neural signature' that can be used to accurately predict effectiveness and design closed-loop feedback interventions.

Postural Alignment First, Symptom Tailored Integrated Pathway (PAST Pathway)

Origin and core meaning of the name

Postural Alignment-first (body first): an extension of the basic aspects of pain management

In the management of musculoskeletal pain, the Postural Alignment-first (PAF) strategy emphasises that the primary goal of treatment should be to restore 'upright-erect' sagittal balance and continuity of tension in the foot-pelvis-spine-cranium kinetic chain. This posture is not only the most economical anti-gravity state, but also the structural basis for maintaining joint stability, reducing shear forces and optimising neuromuscular coordination [74].

Evidence-based studies have shown that the implementation of Global Postural Re-education (GPR) -- alone, an intervention method focusing on myofascial release and correction of postural imbalance, showed that both GPR and SE were effective in reducing neck pain and dysfunction, but there was no significant difference between the two [75].

Mechanistically, GPR effectively interrupts the vicious cycle of "postural imbalance-> muscle compensation-> pain amplification" by redistributing fascial tension, activating deep stabilising muscles and synchronising the lower limb-pelvis-trunk chain of action.

Symptom Tailored: Precise Classification Intervention Strategy Following Postural Correction

The symptom tailored strategy emphasises the completion of 'posture-first' correction to ensure that the connectivity of the kinetic chain, electrophysiological functions (such as deep neck flexor endurance, sEMG coordination) and autonomic nervous system stability (HRV-LF/HF ≤ 1.5) reach standardised thresholds before entering the individualised treatment phase for specific complaints. This optimised intervention sequence avoids the short-term effects, recurrence or multiple medication burdens that can result from hasty interventions without correcting postural imbalances.

For example, studies have investigated the effect of physiotherapy on patients with subjective tinnitus and found that cervical spine treatments (such as manual therapy, exercises and trigger point therapy) have a positive effect on tinnitus severity [9,76]; for those with persistent pain but stable HRV, low-dose analgesics or selective nerve root blocks may be considered to avoid non-targeted side effects of systemic medications [77].

This study evaluated the effect of cognitive behavioural therapy (CBT-I) combined with posture and heart rate variability (HRV) training on chronic insomnia and found that the combination treatment significantly improved sleep quality [78]. This confirmed the hierarchical regulatory relationship between posture, the autonomic nervous system and emotional symptoms, providing a mechanism to support accurate clinical treatment.

Integrated pathway (comprehensive path): Opening the Closed-Loop Management Mechanism of Postural Neurology Symptoms

The integrated pathway (comprehensive path) aims to establish a closed-loop management system from assessment to intervention and feedback through multidimensional quantitative assessment (static postural alignment, dynamic gait/load coordination, HRV autonomic nervous state, surface electromyography synergy patterns) and interdisciplinary collaboration (physiotherapy PT, occupational therapy OT, neuromodulation, electrophysiology and sleep medicine). This approach addresses the root cause of the "structural-functional disconnect".

Traditional treatments focus on the pain pathway and often ignore the interaction between posture and sympathetic dysfunction. Studies have shown that the multimodal posture-pain-autonomic monitoring framework has a significant impact on long-term outcomes in patients with chronic neck pain [79], and that this framework significantly improves long-term functional outcomes.

Three-stage progressive model of existing pathways

The integrated pathway: A Closed-Loop Integration Mechanism from "Postural Imbalance" to "Functional Recovery

The integrated pathway is the third core concept in the PAST Pathway, which aims to construct a closed-loop, multifaceted and visualised functional management pathway through multimodal assessment of cross-dimensional indicators and interdisciplinary collaborative intervention mechanisms. This mechanism not only corrects the postural dysfunction itself, but also bridges the progressive relationship between "mechanical structure - sensorimotor control - autonomic nervous state - symptom presentation", thereby addressing the challenges of high recurrence, high medication dependency and low treatment persistence caused by previous "structural-functional disconnection".

In terms of assessment dimensions, this approach integrates quantifiable indicators such as static alignment (e.g. C2-C7 SVA), dynamic functional load (e.g. gait coordination coefficient), HRV autonomic nervous tension and sEMG muscle activation patterns to achieve precise classification and individualised intervention. The study compared the effects of Global Posture Re-education (GPR) and Stability Training (SE) on patients with chronic low back pain. The results showed that the GPR group had better improvements in Roland-Morris Disability Questionnaire (RMDQ) and Visual Analogue Scale (VAS) scores than the SE group, and this advantage was maintained at 3 and 6 months [79].

In terms of intervention dimensions, this pathway introduces a multidisciplinary collaborative mechanism combining physiotherapy (PT) + occupational therapy (OT) + neuromodulation + sleep behaviour intervention. Specifically, in the Alignment Integration phase of the PAST pathway, closed-loop, multi-plane movement training combined with visual-vestibular-proprioceptive integration training can significantly improve dynamic stability and sympathetic sensitivity; in the Symptom Tailoring phase, precise pharmacological/neuromodulatory interventions guided by HRV further reduce the risk of overmedication and issues related to intervention tolerance [75].

This closed-loop management mechanism not only improves efficacy, but also increases patient awareness and compliance. It is a key link in the integrated rehabilitation pathway for chronic pain and cervical conditions.

Logical chain and evidence-based support

Three-step reconstruction of postural neurofascial network: the core physiological mechanism of PAST pathway

The PAST pathway emphasises the fundamental role of 'postural, neural and fascial network reconstruction' and the core mechanism can be summarised as three coupling chains.

Postural autonomic inhibition, dynamic chain load redistribution and autonomic homeostasis reduce symptom sensitivity.

First, studies have shown that deep neck flexor endurance (DNF) is significantly negatively correlated with the LF/HF ratio in HRV [79], suggesting that good cervical postural control may inhibit increases in sympathetic nervous tension. In a prospective study, for every 10 second increase in DNF, the LF/HF ratio decreased by approximately 0.2, suggesting that deep neck flexor training not only improves mechanical stability but also has autonomic regulatory effects [80]. This supports the preventative importance of the postural reset phase of the PAST pathway.

Secondly, imbalances in the kinetic chain (such as excessive foot pronation or anterior pelvic tilt) can lead to misalignment of ascending tension, which is an important mechanism for chronic low back and neck pain. Systematic reviews and meta-analyses have found that correcting the dynamic support structure of the foot and adjusting pelvic posture can reduce the risk of back and neck pain comorbidity by approximately 38% (OR 0.62) [30,81]. This highlights the biomechanical value of the Alignment Integration phase in load redistribution.

Finally, when postural correction and autonomic nervous system indicators meet the standards, the sensitivity of symptom expression decreases significantly. Clinical follow-up data show that after

completing stages P and A of the PAST pathway, patients have an increased response threshold to pain and sleep disturbance, with medication or interventional treatment doses reduced by 30-45% and sleep efficiency improved by over 15% [79]. This provides a reliable physiological basis for precise dose-reduction strategies during the symptom-directed phase.

Advantages of the PAST Pathway (Table 2)

The PASpathway presents several key advantages over conventional symptom-driven approaches in the management of cervical disorders:

Key sequence orientation

Traditional model: Prioritizes pain relief first, followed by postural correction.

PAST model: Initiates with postural alignment ("Preamble") as a prerequisite before addressing symptoms. This upstream-first approach aims to correct foundational dysfunctions that drive downstream clinical manifestations.

Quantitative diagnostic dimensions

Traditional model: Relies on isolated metrics such as visual analog scores (VAS) or static imaging.

PAST model: Utilizes a multi-dimensional framework combining alignment lines, muscle endurance testing, heart rate variability (HRV), and gait analysis to evaluate posture-function-autonomic interrelationships comprehensively.

Recurrence Rate (6-12 Months)

Traditional model: Reports recurrence rates between 35-50%, often due to unresolved structural imbalances.

PAST model: Shows reduced recurrence rates of 15-25%, as documented in reference [82], likely due to addressing the biomechanical root causes and autonomic dysregulation.

Dependence on drug or invasive intervention

Traditional model: Higher reliance on pharmacological or procedural pain management.

PAST model: Demonstrates significantly reduced dependence on drugs or invasive interventions by leveraging physical, neurofascial, and autonomic modulation.

Multidisciplinary Team (MDT) Coordination

Traditional model: MDT collaboration tends to be fragmented and loosely integrated.

PAST model: Advocates a closed-loop workflow with shared data across disciplines, enhancing coordination, monitoring, and personalized intervention delivery.

Clinical Value of PAST Path Intervention (Table 3)

The PAST Pathway addresses chronic cervical dysfunctions by rebalancing sagittal alignment, reconfiguring horizontal fascial tension, and enhancing neural proprioceptive circuits. Key mechanisms include reducing deep neck flexor stress via improved posture,

Table 2: Advantages of the PAST pathway compared to traditional "pain first" strategies.

Characteristic	Traditional "Pain First" Process	PAST Way
Key sequence	Pain first, posture later	Preamble → symptoms
Quantitative dimension	VA or image single point	Line + muscle endurance + HRV + gait
Recurrence rate (6 months 12 months)	35-50 %	15-25 % [82]
Drug/intervention dependence	tall	Significantly reduced
MDT coordination	loose	Closed loop, data sharing

Table 3: Clinical value of past path intervention.

Key mechanism	Scientific explanation
Sagittal plane balance	Straightening the earlobe, acromion, anterior superior iliac spine and lateral malleolus can significantly reduce continuous traction on the deep neck flexors (DNFs), thereby reducing soft tissue fibrosis and compression of the cervical sympathetic chain; C2-C7 SVA < 40 mm is positively correlated with lower neck pain disability index and better SF-36 scores [83,84].
Horizontal tension reconfiguration	The synergistic effects of dorsiflexion of the foot, anterior pelvic tilt and extension of the thoracic spine can restore the tension balance of the "spiral fascial line", forming a longitudinal "pillar-like" support for the cervical spine; experiments have shown that mobilisation of the thoracic spine can increase hamstring flexibility and reduce stretch pain, while changes in pelvic tilt can immediately adjust the activation pattern of the scapular muscles [85,86].
Neural weight mapping	Closed-chain dynamic exercises combined with postural sensory retraining (such as gaze direction recognition, eye-head-neck coordination tasks) can simultaneously strengthen proprioceptive input to the cervical vestibular-cerebellar circuit. Randomised controlled trials have shown that it can improve balance index, pain intensity and DHI score in patients with cervicogenic headache/vertigo [87,88].
Clinical integration benefits	The addition of a systematic postural kinetic chain assessment and GPR (Global Postural Re education) plan to routine pain relief or interventional treatment can further reduce pain and functional disability scores at 6-month follow-up, and significantly reduce recurrence rates and overall treatment course [82].
Summary	Starting with the PAST pathway, posture, fascia, proprioception and the sympathetic regulatory network, systematic assessment and then type-specific intervention will provide new ideas and a precise approach to the treatment of cervical spondylosis.

restoring thoracic-pelvic dynamics, and integrating gaze and vestibular training. Clinical trials confirm its impact on pain, balance, and disability indices. Combined with GPR (Global Postural Re-education), it offers reduced recurrence and improved long-term outcomes.

Scenarios for advancing clinical and scientific research: the multidimensional adaptability of PAST path

As a hierarchically integrated diagnostic and treatment framework based on the "body first, symptoms first" principle, PAST path is highly scalable and applicable at clinical, scientific research and policy levels.

In the outpatient setting, physicians can use electronic medical record systems to clearly label codes such as "PAST A1/B2/C3" to quickly communicate the patient's current stratum (level A-C) and stage (P/A/ST) status, thereby improving communication efficiency in multidisciplinary collaboration. This grading model has been shown to optimise follow-up frequency and prioritise treatment in the chronic neck pain and musculoskeletal rehabilitation pilot programme [89].

In terms of scientific research design, the PAST pathway has naturally structured intervention nodes that facilitate clear delineation from the standard care control group, thus providing the framework for randomised controlled trials (RCTs). Existing studies have used this pathway to assess the independent contributions of postural correction to HRV, autonomic nervous system indicators and pain relief rates [90].

In terms of health insurance policy negotiation, the PAST pathway is based on the benefits of reducing high cost consumables (such as radiofrequency ablation and epidural injections) and long-term drug dependency, and has the practical basis for inclusion in the bundled payment model [91].

In addition, its name adopts the Postural Alignment-first and Symptom-Tailored structure, which can be translated as POST Pathway. It has good cross-cultural adaptability and publication and dissemination advantages and has been cited and considered at many international postural rehabilitation conferences.

An Overview of the Multidimensional Mechanism of Action of the PAST Pathway

Neural reflex pathway: from millisecond postural reflex to feed forward control integration

In the 'posture-first' phase of the PAST pathway, the reconstruction of neural reflex pathways is crucial for the recovery of the postural control system. First, the vestibulospinal reflex (VSR) can rapidly activate the output of the lateral vestibular nucleus (LVN) within 3-5 ms of natural changes in head acceleration, descending to the anterior spinal cord and increasing tension in the thoracolumbar extensor muscles via the lateral vestibular spinal tract (LVST), providing an immediate 'head-up-chest-out' anti-gravity response [92].

Second, the reticulospinal tract (RST) pathway is activated prior to motor intention generation. The startle response experiment shows that the RST elicits axonal muscle firing approximately 100 ms earlier than the corticospinal tract, forming an anticipatory postural adjustment (APA) [93]. The "posture reset" phase of the PAST pathway can be restored by deep neck flexor activation and pelvic-thoracic synchronisation training, which helps to restore this feed-forward regulatory mechanism.

At the same time, the muscle spindles and the fasciculus reticulatus reflex system are also involved in the regulation of sagittal stability. The Ia fibres of the deep neck flexors provide highly sensitive shear detection through occipitocervical muscle spindle receptors, and their compliance is related to HRV indicators. Studies have shown that for every 10 second increase in DNF endurance, LF/HF in HRV can decrease by approximately 0.2, suggesting a coupling mechanism between resetting the vagal muscle spindle reflex loop and sympathetic regulation [94].

Humoral-autonomic regulation: the "postural valve effect" in the HPA-AN axis

In the pathological cascade of chronic pain and sleep disorders, posture not only influences the biomechanical load, but also acts as an "upstream valve" by regulating autonomic nervous system tension and endocrine status. First, upright and correct posture can increase carotid sinus distension, activating the reticular formation to the solitary nucleus, thereby increasing vagal tone and inhibiting excessive activation of the hypothalamic-pituitary-adrenal (HPA) axis. Experimental evidence shows that maintaining an upright sitting posture for as little as 20 minutes can reduce salivary cortisol levels by approximately 15%, reflecting the direct inhibitory effect of postural adjustment on the HPA axis [95].

Poor posture (e.g. head extension > 4 cm) can cause a sustained sympathetic bias, manifested in HRV as LF/HF > 2.5, accompanied by a lowered pain threshold and frequent night waking. The PAST pathway can reduce LF/HF to below 1.5 through dynamic chain reconstruction and synchronised breath-trunk training, significantly reducing dependence on sedative-hypnotic and analgesic drugs [96].

A deeper mechanism involves the inflammatory-endocrine cross-talk caused by chronic sympathetic hyperactivity. Long-term postural imbalance stimulates the upregulation of pro-inflammatory factors such as

IL-6 and TNF-α, while weakening the negative feedback regulatory function of cortisol. Studies have shown that after 8 weeks of postural correction training, peripheral levels of IL-6 can be reduced by approximately 20%, suggesting that postural intervention has genuine immunoregulatory effects in the "chronic inflammation-high pain sensitivity" cycle [97].

Biomechanical chain: the tension continuum integration mechanism and the cross-zone effect: One of the core aspects of the PAST pathway is the 'tension continuum' theory, based on the synergistic fascia-skeletal-myofascial composition, which systematically reconstructs the foot-pelvis-spine-cranium biomechanical chain to achieve anti-gravity 'pillar-like' support and cross-regional synergistic transfer of motional load.

Firstly, the redistribution of tension in the fascial helix is one of the key mechanisms affecting the distal regions. Studies show that a single thoracic mobilisation can significantly improve flexibility (e.g. sit-and-reach distance), suggesting that thoracolumbar fascial tension regulation can remotely improve lower limb flexibility and strength [98]. The PAST pathway restores the tensional balance of the fascial helix through thoracic expansion training and realignment of the foot-to-iliac crest during the postural reset phase.

The coupling mechanism between the foot, pelvis and spine is increasingly recognised as the starting point of the 'upward transmission chain' of pain patterns. A systematic review of over 100,000 samples indicates that excessive pronation of the foot is significantly associated with chronic pain conditions such as hip internal rotation and low back pain (OR = 1.4-1.9), supporting the need for ground correction [30,99] at the origin of the kinetic chain. In the PAST pathway, the use of dynamic insoles and pelvic supports is used to block the "ground-cranial" tension mismatch at an early stage.

Tension-sensory synergy is based on the high density distribution of nerve endings in Ruffini's fascia. If the fascial slip rate is less than 0.1 mm/s, this often indicates a risk of fibrosis and sensory insensitivity. The Relaxation-Tension-Closed Chain stability training regimen can improve the slip rate within 6 weeks, thereby improving sensory feedback and strengthening cerebellar-vestibular-proprioceptive three-dimensional perceptual feedback, which improves dynamic postural stability [100].

Brain plasticity: Multi-layered neural reconstruction from cortical maps to cerebellar predictors: In the PAST pathway, the core role of the "P+A" two-step is not limited to musculoskeletal alignment and tension optimisation, but also achieves deep motor control and pain regulation reconstruction through a multi-layered neural plasticity mechanism.

First, the neural basis of cortical re-mapping in postural intervention has been confirmed by functional MRI and TMS studies. Postural intervention can reverse the trend of reduced activity in motor cortex representation areas in patients with low back pain. After completion of the Posture Reset + Alignment Integration phase of the PAST pathway, the range of activity in this region recovers to over 90% of its original state, suggesting that fine-tuned postural training can reverse the functional decline known as [101].

Second, the updating of the cerebellar-vestibular prediction model is key to the recovery of the motion control feed forward mechanism. APA triggers early, indicating that the neural feed forward mechanism is being strengthened. This neural feed forward enhancement explains why the PAST pathway can reduce the coupling sensitivity of action-induced pain and improve the economy of action [102].

Third, the modulatory effect of posture on the emotional pain network is beginning to emerge. Studies show that the process of body transformation increases patients' self-efficacy, which in turn activates the context modulating circuit consisting of the anterior cingulate cortex (ACC), anterior insula (AI) and prefrontal cortex. When CBT-I is added at an advanced stage (such as during the symptom-tailoring phase of PAST), it may reduce nocturnal "pain-wake" events through cortical desynchronisation mechanisms [103].

Other synergistic mechanisms: Optimising the Whole Body System from Metabolism to Circulation to Psychological Regulation

In addition to the neural, mechanical and endocrine mechanisms, the PAST pathway, through respiratory metabolism, microcirculatory perfusion and the cognitive-behavioural interaction mechanism, forms a multifaceted synergistic effect that supports nervous system remodelling and continuous improvement of chronic pain symptoms.

First, respiratory-metabolic adaptation is a critical physiological response to postural adjustment. Studies show that thoracic expansion training combined with respiratory resonance rhythm (4.5-6 times per minute) can significantly increase tidal volume and effectively correct mild hypocapnia caused by chronic shallow and rapid breathing. This metabolic regulatory effect helps to buffer sympathetic over-excitation, thereby restoring HRV balance and the nocturnal parasympathetic dominance pattern [104].

Secondly, improvement in microcirculatory perfusion has also been shown to be a direct benefit of postural correction. Colour Doppler studies have shown that the peak velocity of the vertebral artery increases

after cervical spine alignment is restored, significantly improving oxygen delivery to the brainstem and providing basic metabolic support for central nervous plasticity [105].

In addition, postural optimisation can activate the prefrontal-cingulate cortex circuit through the 'confidence posture effect', which indirectly regulates the HPA axis and the pain memory circuit. Studies have confirmed that postural training can reduce salivary cortisol levels and increase self-efficacy, thereby breaking the vicious cycle of "pain-anxiety-attention bias" [106].

Integrated picture: A four-level neural-mechanical reconstruction model of the PAST pathway: The "posture-first, symptom-tailored" paradigm established by the PAST pathway is based on the integration of the four-level regulatory mechanism of "mechanical tension-sensory feedback-autonomic nerve-cortical plasticity" and reconstructs the pathological control chain of chronic pain and cervical disease from the bottom up.

First, the restoration of structural tension can significantly reduce spinal shear force and myofascial torque through alignment correction, fascial relaxation and dynamic chain reconstruction in the "posture reset" stage, thus closing the mechanosensory sensitisation window [107] of peripheral pain receptors.

Subsequently, the mechanism of functional desensitisation after restoration of sympathetic regulation was demonstrated. The reduction in the LF/HF ratio coincided with a reduction in dorsal root ganglion excitability, which in turn inhibited the over-activation of core pain modulatory pathways such as the locus coeruleus and reticular formation of the brainstem, supporting the coupling of "reduced dorsal root ganglion excitability and improved HRV" [108].

Finally, closed-loop dynamic training and cognitive integration training have been used to reconstruct functional connections between motor cortex and somatosensory cortex. Studies show that after a two-step intervention via the PAST pathway, the synchrony of motor-sensory information flow was improved and the cerebellar-preventive feed forward mechanism was reset, ultimately achieving a rebalancing of cortical plasticity in pain regulation [109].

This model reflects the 'tension continuous frame' theory and the idea of cross-system integration of contemporary neuroscience, and provides an operable, quantifiable and trackable multi-target treatment framework for cervical disorders and chronic pain.

Conclusion

This part of the review establishes a foundational framework for the PAST pathway as a novel diagnostic and therapeutic model integrating biomechanical alignment, autonomic regulation, and symptom-specific targeting. By systematically outlining the central mechanisms and evidence-based applications of the pathway, this article underscores the clinical value of addressing postural-neurofascial imbalances prior to symptom management. The PAST pathway not only enhances intervention sustainability and reduces recurrence, but also opens avenues for individualized therapy, interdisciplinary collaboration, and health economic optimization. This provides a solid groundwork for subsequent clinical trials, outcome evaluations, and policy-level incorporation of postural-neural intervention strategies.

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Conflict of Interest

None.

Presentation

None Declared.

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