



Neuromuscular Balance Control during Unilateral Stance in Chronic Ankle Instability

Jack A. Hughes

Department of Physical Therapy, Sargent College of Health and Rehabilitation Sciences, Boston University, Boston, MA 02215, USA

Abstract

Chronic ankle instability is a highly prevalent musculoskeletal condition characterized by recurrent sprains, persistent pain, and a subjective feeling of giving way during dynamic and static activities. A fundamental manifestation of this pathology is the deterioration of postural control, particularly during unilateral stance. Unilateral stance is not merely a clinical assessment tool; it is a critical transitional phase in human locomotion, encompassing walking, running, and navigating uneven terrain. This paper provides a comprehensive academic examination of the neuromuscular balance control mechanisms that are disrupted in individuals suffering from chronic ankle instability. By investigating the intricate sensorimotor pathways, including peripheral mechanoreceptor deafferentation, altered spinal reflex excitability, and central nervous system neuroplasticity, this analysis elucidates the fundamental neurophysiological deficits underlying postural instability. Furthermore, this study evaluates the contemporary methodological approaches utilized to quantify these deficits, focusing on center of pressure trajectory analysis, surface electromyography, and three-dimensional kinematic motion capture. Through a meticulous synthesis of existing literature, this research aims to clarify the specific alterations in muscle activation sequencing, specifically the delayed onset of the peroneal musculature and the excessive reliance on proximal joint strategies to maintain equilibrium. The clinical implications of these findings are thoroughly discussed, emphasizing the necessity for targeted, multi-modal rehabilitation paradigms that address both local articular impairments and global sensorimotor dysfunction. Ultimately, a deeper understanding of these neuromuscular alterations will facilitate the development of more efficacious therapeutic interventions, mitigating the risk of long-term joint degeneration and improving the overall quality of life for affected individuals.

Keywords: *Chronic Ankle Instability, Postural Control, Neuromuscular Deficits, Unilateral Stance*

1. Introduction

1.1 Overview of Lateral Ankle Sprains and Instability

The human ankle joint complex is an intricate anatomical structure designed to bear the entire weight of the body while simultaneously providing the extraordinary mobility required for complex bipedal locomotion. Due to its unique anatomical configuration and the immense forces it routinely sustains, the ankle is exceptionally susceptible to injury. Lateral ankle sprains represent one of the most frequently encountered traumatic injuries in both athletic and general populations, accounting for a significant proportion of all emergency room visits and sports medicine consultations [1]. A lateral ankle sprain typically occurs during a sudden movement involving excessive plantar flexion and inversion, which places immense tensile stress on the lateral ligamentous complex, primarily the anterior talofibular ligament and the calcaneofibular ligament. While the acute management of these injuries is generally well established, a remarkably high percentage of individuals fail to fully recover, subsequently developing a debilitating condition known as chronic ankle instability [2]. Chronic ankle instability is

a multifaceted pathology characterized by a constellation of lingering symptoms, including recurrent episodes of ankle sprains, persistent discomfort, swelling, and a pervasive, subjective sensation that the joint is constantly on the verge of collapsing or giving way during routine activities. This condition is generally conceptualized as a combination of mechanical instability, which refers to objective pathological laxity of the ligaments following incomplete tissue healing, and functional instability, which encompasses a wide array of sensorimotor deficits that compromise the dynamic stabilization of the joint [3]. The interplay between these mechanical and functional components creates a vicious cycle of recurrent injury and progressive joint degradation, often leading to the early onset of post-traumatic osteoarthritis. Therefore, understanding the underlying mechanisms of chronic ankle instability is of paramount importance for the development of effective prevention and rehabilitation strategies. A cornerstone of this understanding lies in the rigorous examination of postural control, which serves as a vital indicator of the integrity and functionality of the sensorimotor system [4].

1.2 The Significance of Unilateral Stance

Postural control, commonly referred to as balance, is the complex, dynamic process by which the central nervous system integrates multi-sensory information to maintain the body's center of gravity within the narrow limits of the base of support. Among the various paradigms utilized to assess postural control, the unilateral stance, or single-leg stance, stands out as a particularly critical and clinically relevant measure. The ability to maintain equilibrium on a single limb is not merely a static laboratory test; it represents a fundamental biomechanical requirement for virtually all forms of human terrestrial locomotion. During the normal gait cycle, approximately sixty percent of the time is spent in the single-leg support phase. Consequently, any deficit in unilateral postural control directly translates to impaired functional performance and an elevated risk of falls or secondary injuries during dynamic activities such as walking, running, jumping, and changing direction rapidly. In individuals with chronic ankle instability, the performance during unilateral stance is consistently compromised. The unilateral stance challenges the neuromuscular system by drastically reducing the base of support and elevating the center of mass, thereby demanding a highly coordinated and rapid generation of corrective torques at the ankle joint. When an individual attempts to balance on an unstable ankle, the deficits in proprioceptive acuity, delayed muscle activation, and altered reflex responses become strikingly apparent. The rigorous analysis of unilateral stance provides researchers and clinicians with a highly sensitive window into the specific neurophysiological impairments that characterize chronic ankle instability. By systematically observing how the body attempts to compensate for the compromised distal joint, we can gain profound insights into the hierarchical organization of human motor control and the remarkable adaptability, as well as the limitations, of the central nervous system in the face of peripheral musculoskeletal injury.

2. Background and Literature Review

2.1 Epidemiology and the Trajectory of Pathology

The epidemiological landscape of ankle injuries underscores the massive public health burden associated with lateral ankle sprains and subsequent chronic instability. Current epidemiological data suggest that ankle sprains are the single most common injury sustained during participation in sports that require cutting, jumping, and rapid changes of direction, such as basketball, soccer, and volleyball [5]. However, the incidence is also remarkably high in the general population during non-athletic, activities of daily living. Despite the pervasive nature of this injury, a concerning trend in the literature reveals that a vast majority of individuals who suffer an initial acute ankle sprain do not seek formal medical attention or engage in supervised rehabilitation programs. This neglect often leads to suboptimal healing of the ligamentous structures and incomplete restoration of the sensorimotor pathways, setting the stage for long-term complications [6].

Table 1: Epidemiology and Recurrence Rates of Ankle Sprains Across Different Sports Settings

Sport Setting	Initial Sprain Incidence Rate per Thousand Hours	Estimated Recurrence Rate within One Year	Progression to Chronic Instability
Basketball	3.85	45 percent	35 percent

Soccer	2.95	38 percent	30 percent
Volleyball	2.50	40 percent	28 percent
General Athletics	1.80	30 percent	25 percent

The transition from a single, acute lateral ankle sprain to the chronic, persistent state of ankle instability is a complex temporal process. Studies indicate that up to forty to seventy percent of individuals who sustain an initial sprain will experience residual symptoms and recurrent instability for months or even years following the injury [7]. This high rate of chronicity has prompted extensive investigation into the causative factors. Early theoretical frameworks, such as the widely cited Freeman hypothesis, primarily attributed functional instability to a disruption of the articular mechanoreceptors located within the injured ligaments, leading to a localized deficit in joint position sense. While deafferentation certainly plays a crucial role, contemporary literature has significantly expanded this view, proposing a more global sensorimotor dysfunction paradigm. This modern perspective recognizes that chronic ankle instability is not merely a localized peripheral joint issue but rather a comprehensive systemic impairment involving widespread neuroplastic changes throughout the central nervous system, affecting bilateral motor control and postural regulation strategies [8]. The high prevalence and the multifaceted nature of the transition to chronicity highlight the critical need for a comprehensive understanding of the specific neuromuscular control deficits that perpetuate this condition.

2.2 Evolution of Sensorimotor Paradigms

The conceptualization of how the human body maintains balance in the presence of joint instability has evolved dramatically over the past few decades. Initially, the prevailing medical model focused almost exclusively on the mechanical properties of the ankle joint. Mechanical instability was assessed through clinical diagnostic techniques measuring the degree of anterior translation and talar tilt. Surgical interventions were heavily favored, aimed at physically reconstructing the torn ligaments to restore mechanical constraints. However, long-term follow-up studies frequently demonstrated that surgical restoration of mechanical stability did not uniformly resolve the patient's subjective feelings of giving way or their deficits in dynamic balance, leading researchers to investigate alternative mechanisms [9].

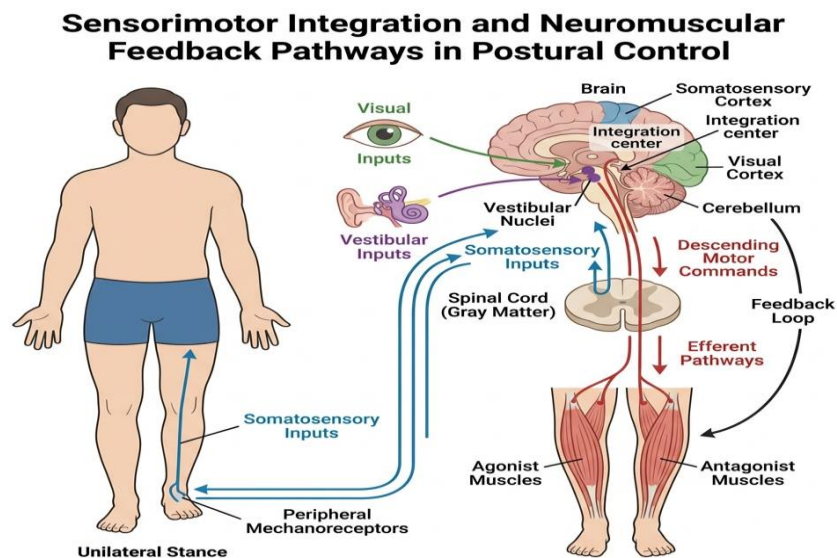


Figure 1: Comprehensive Schematic of Sensorimotor Integration and Neuromuscular Feedback Pathways in Postural Control

This realization catalyzed a paradigm shift toward understanding the sensorimotor system's role in joint stability. The sensorimotor system encompasses the integration of sensory input from somatosensory, visual, and vestibular receptors, the processing of this information by the central nervous system, and the subsequent generation of an appropriate motor response. In the context of chronic ankle instability, research has increasingly focused on the impairment of the somatosensory component, specifically proprioception and kinesthesia. Proprioception refers to the conscious and unconscious awareness of joint position, while kinesthesia is the perception of joint movement. Extensive literature now supports

the notion that individuals with chronic ankle instability exhibit significant deficits in both these areas, suggesting that the initial trauma damages not only the structural collagen fibers of the ligaments but also the embedded neural receptors. This dual loss of mechanical constraint and sensory feedback necessitates a complete reorganization of postural control strategies, forming the foundation of the functional deficits observed during challenging tasks such as unilateral stance.

3. Neurophysiological Mechanisms

3.1 Peripheral Deficits and Mechanoreceptor Disruption

The foundation of neuromuscular control relies heavily on the continuous and accurate transmission of afferent sensory information from the periphery to the central nervous system. The ligaments, joint capsule, and surrounding musculotendinous structures of the ankle joint are richly innervated with specialized sensory end organs known as mechanoreceptors. These include Ruffini endings, which signal joint position and velocity; Pacinian corpuscles, which respond to rapid changes in pressure and acceleration; Golgi-like receptors, which monitor tension at the extremes of joint motion; and free nerve endings, which transmit nociceptive or pain signals [10]. When a severe lateral ankle sprain occurs, the immense stretching and microscopic tearing of the anterior talofibular and calcaneofibular ligaments result in direct mechanical trauma to these embedded neural structures. This structural damage causes partial or complete deafferentation, fundamentally altering the quality, quantity, and timing of the sensory signals generated by the injured joint. The consequence of this peripheral deafferentation is profound. Without accurate real-time data regarding the exact position and velocity of the ankle joint, the central nervous system is essentially operating with incomplete sensory information. This sensory deficit manifests clinically as impaired joint position sense and an elevated threshold for the detection of passive movement [11]. Furthermore, the inflammatory response that follows the acute injury can chemically sensitize the remaining intact mechanoreceptors and nociceptors, leading to abnormal sensory gating. A particularly debilitating phenomenon associated with this peripheral disruption is arthrogenic muscle inhibition. This is an ongoing, involuntary neural inhibition of the musculature surrounding the injured joint, particularly the stabilizing peroneal muscles. Arthrogenic muscle inhibition is believed to be a protective reflex intended to prevent further tissue damage by limiting joint motion; however, in the chronic phase, this inhibition severely compromises the dynamic stability of the ankle, making it exceedingly difficult for the individual to generate the rapid, forceful muscle contractions required to maintain balance during unilateral stance [12].

3.2 Spinal and Supraspinal Neuroplasticity

While peripheral deafferentation initiates the cascade of sensorimotor dysfunction, the perpetuation of chronic ankle instability is heavily mediated by subsequent adaptations within the central nervous system. These adaptations occur at both the spinal reflex level and higher supraspinal, or cortical, levels. At the level of the spinal cord, neuromuscular control is governed by rapid, monosynaptic and polysynaptic reflex loops that provide immediate, unconscious corrective responses to unexpected joint perturbations. Research utilizing the Hoffman reflex, which is an electrically elicited analog of the spinal stretch reflex, has consistently demonstrated altered excitability of the spinal motor neuron pool in individuals with chronic ankle instability [13]. Specifically, there is often a noted depression in the reflex excitability of the soleus and peroneal muscles. This diminished reflex excitability indicates that the spinal cord is less responsive to incoming sensory stimuli, thereby delaying the automatic, subconscious muscle contractions that are vital for preventing an impending ankle sprain during a sudden loss of balance. Beyond the spinal cord, advanced neuroimaging and transcranial magnetic stimulation studies have revealed profound neuroplastic changes within the cerebral cortex of individuals suffering from chronic ankle instability. These supraspinal adaptations indicate a fundamental reorganization of the brain's motor maps. Due to the unreliable proprioceptive information ascending from the injured ankle, the central nervous system begins to downregulate the somatosensory areas normally dedicated to the ankle joint and shifts its reliance toward visual and vestibular inputs to maintain balance [14]. This cortical reorganization results in a shift from efficient, subconscious, feedforward motor control toward a more conscious, cognitively demanding, feedback-driven control strategy. Consequently, maintaining a unilateral stance becomes an excessively effortful task. When visual information is suddenly removed,

such as closing the eyes during a balance assessment, individuals with chronic ankle instability demonstrate a catastrophic decline in postural stability, underscoring their maladaptive over-reliance on visual cues and the severe deterioration of their native somatosensory processing capabilities.

4. Methodology of Assessment in Balance Control

4.1 Kinetic Assessment using Force Platforms

The objective quantification of postural control during unilateral stance is predominantly achieved through the use of highly sensitive biomechanical instrumentation, with laboratory-grade force platforms being the universally accepted gold standard [29]. A force platform measures the ground reaction forces and moments exerted by the foot as the individual attempts to maintain equilibrium. From these highly detailed tridimensional force measurements, researchers compute the center of pressure. The center of pressure represents the point of application of the resultant vertical ground reaction force vector. During a unilateral stance task, the center of pressure is continuously migrating as the neuromuscular system makes rapid, minute adjustments at the ankle joint to keep the body's center of mass centered over the narrow base of support provided by the single foot [15]. The analysis of the center of pressure trajectory yields a multitude of kinetic parameters that provide deep insights into the efficiency and stability of the postural control system. Commonly utilized metrics include the total excursion path length, which reflects the total distance traveled by the center of pressure over a specific time interval, and the average velocity of the center of pressure displacement. Increases in both path length and velocity are generally interpreted as indicators of decreased postural stability and increased neuromuscular effort. Furthermore, the area of the bounding ellipse enclosing the center of pressure trajectory is calculated to represent the overall spatial boundary of the sway pattern [16]. In individuals with chronic ankle instability, researchers consistently observe statistically significant increases in center of pressure velocity and sway area, particularly in the medial-lateral direction, which corresponds to the anatomical plane of the injured lateral ligaments.

Table 2: Center of Pressure Parameters Used in the Assessment of Unilateral Stance

Kinetic Parameter	Definition and Description	Clinical Interpretation in Chronic Ankle Instability
Total Path Length	The accumulated distance of the center of pressure trajectory over the trial duration.	Typically increased, indicating excessive postural sway and corrective movements.
Average Velocity	The total path length divided by the duration of the testing trial.	Significantly higher, reflecting a highly erratic and rapid corrective strategy.
Sway Area Ellipse	The mathematical area encompassing ninety-five percent of the center of pressure data points.	Expanded spatial boundary, representing a larger deviation from the absolute center of balance.
Medial-Lateral Excursion	The maximum distance the center of pressure travels perpendicular to the foot axis.	Markedly increased due to specific deficits in the lateral stabilizing structures.

The rigorous standardization of the unilateral stance protocol is critical for obtaining reliable kinetic data. Participants are typically instructed to stand barefoot on the force platform, with the non-weight-bearing limb flexed at the knee and the hands placed securely on the iliac crests to eliminate the use of arm swing for balance compensation [17]. Trials are conducted under various sensory conditions, such as eyes open to establish a baseline, and eyes closed to specifically isolate and challenge the

somatosensory and vestibular systems. The duration of the trials usually ranges from ten to thirty seconds. By meticulously controlling these experimental variables, researchers can isolate the specific kinetic deficits associated with the unstable ankle and utilize the center of pressure data to accurately differentiate between healthy individuals and those suffering from varying severities of chronic ankle instability.

4.2 Electromyography and Kinematic Motion Capture

While force platforms provide excellent kinetic data regarding the overall outcome of postural control, they do not reveal the specific muscular strategies or joint movements utilized to achieve that outcome. To fully understand the neuromuscular balance control during unilateral stance, researchers employ surface electromyography in conjunction with three-dimensional kinematic motion capture. Surface electromyography involves the placement of non-invasive electrodes over the bellies of specific target muscles, allowing for the continuous recording of the electrical action potentials generated during muscle contraction. In the assessment of chronic ankle instability, the primary muscles of interest are the lateral stabilizers, specifically the peroneus longus and peroneus brevis, as well as the anterior tibialis and the medial stabilizers such as the gastrocnemius and soleus [18]. The electromyographical signals are heavily processed using amplification, filtering, and rectification techniques, and are typically normalized to the individual's maximum voluntary isometric contraction to allow for meaningful comparisons between subjects.



Figure 2: Experimental Setup and Biomechanical Modeling for Unilateral Stance Assessment on a Force Platform

Three-dimensional kinematic motion capture systems utilize multiple high-speed infrared cameras to track the exact spatial trajectory of retro-reflective markers placed on specific anatomical landmarks of the subject's body. This sophisticated technology allows researchers to calculate precise joint angles, angular velocities, and segmental accelerations throughout the unilateral stance trial. By synchronizing the kinematic motion data with the electromyographical signals and the kinetic force plate data, a highly comprehensive, multi-dimensional profile of neuromuscular control is constructed [19]. This synchronized approach enables the observation of not just how much the subject sways, but exactly which joints are moving to cause the sway, and specifically which muscles are firing, and at what precise millisecond, to correct the deviation. This level of methodological rigor is absolutely essential for uncovering the subtle, yet profoundly disruptive, alterations in motor planning and execution that plague individuals with chronic ankle instability.

5. Analysis of Neuromuscular Alterations

5.1 Muscle Activation Sequencing and Onset Delays

A critical aspect of dynamic joint stabilization is the central nervous system's ability to activate the correct muscles at precisely the right moment to counteract an external perturbing force. In a healthy sensorimotor system, the lateral musculature of the lower leg, particularly the peroneus longus and brevis, acts as the primary dynamic defense mechanism against excessive ankle inversion. During a sudden balance challenge or an unexpected shift in the center of gravity during unilateral stance, these muscles must fire rapidly and forcefully to eccentrically decelerate the inversion motion and re-center the body. Extensive electromyographical analysis has consistently documented a pathological delay in the reaction time, or onset latency, of these specific peroneal muscles in individuals with chronic ankle instability [20]. This delay, often measured in critical milliseconds, means that the dynamic muscular defense system engages too late to prevent the joint from crossing the threshold into a potentially injurious position.

Table 3: Comparison of Muscle Activation Onset Delays During Sudden Inversion Perturbation

Muscular Group	Healthy Control Group Average Onset Time	Chronic Ankle Instability Group Average Onset Time	Statistical Significance
Peroneus Longus	55 milliseconds	82 milliseconds	Highly Significant
Peroneus Brevis	58 milliseconds	85 milliseconds	Highly Significant
Tibialis Anterior	65 milliseconds	78 milliseconds	Moderate Significance
Soleus	70 milliseconds	72 milliseconds	Not Significant

The fundamental cause of this delayed onset is deeply rooted in the neurophysiological deficits discussed earlier. The transmission of the stretch stimulus from the damaged mechanoreceptors is compromised, the spinal reflex loops are depressed, and the cortical processing time is prolonged due to neuroplastic reorganization. Consequently, the efferent motor command takes substantially longer to reach the muscle fibers. In addition to the delayed onset of the peroneal muscles, researchers have also observed altered activation patterns in the tibialis anterior [21]. Instead of a crisp, reciprocal activation pattern, individuals with chronic ankle instability often demonstrate prolonged, uncoordinated co-contraction of the antagonist muscle groups around the ankle. While co-contraction is a valid strategy to increase global joint stiffness in a highly unpredictable environment, it is extremely metabolically inefficient and restricts the fluid, fine-tuned micro-adjustments necessary for optimal postural control during a relatively static task like unilateral stance.

5.2 Alterations in Joint Strategy and Postural Synergies

Human postural control operates on a continuum of movement strategies, ranging from distal adjustments at the ankle joint to proximal adjustments at the hip joint. In a healthy individual standing on a firm surface, the ankle strategy is the predominant mechanism for maintaining anterior-posterior and medial-lateral balance. The ankle strategy involves small, rapid torque generations that pivot the entire body mass around the ankle joint, acting essentially as an inverted pendulum. However, when the structural integrity of the ankle is compromised, and the local neuromuscular control is impaired due to chronic ankle instability, the central nervous system is forced to adapt by recruiting alternative postural synergies [22]. Kinematic analyses of unilateral stance consistently reveal that individuals with chronic ankle instability exhibit a marked decrease in reliance on the ankle strategy and a corresponding, compensatory increase in the utilization of the hip strategy. The shift toward a proximal hip strategy is visually observable as increased multi-planar movement at the pelvis and trunk during single-leg balancing. By rapidly flexing, extending, and abducting the hip joint, the individual creates shear forces

against the ground that attempt to reposition the center of mass without relying heavily on the unstable ankle joint beneath [23]. While this hip strategy represents a successful, short-term evolutionary adaptation that prevents immediate falls, it is ultimately a maladaptive, suboptimal movement pattern. The increased motion of the large, heavy upper body segments requires significantly more energy expenditure and results in a highly erratic center of pressure trajectory. Furthermore, this over-reliance on proximal musculature fails to address the underlying distal deficit. Over time, the persistent failure to utilize the ankle musculature for fine postural adjustments can lead to further disuse atrophy and proprioceptive decay at the ankle joint, creating a deleterious feedback loop that continuously exacerbates the severity of the functional instability [24]. This fundamental alteration in postural coordination underscores the necessity of evaluating the entire kinetic chain when assessing ankle pathology.

6. Discussion and Clinical Implications

6.1 Rehabilitation Paradigms and Proprioceptive Training

The comprehensive mapping of the neuromuscular deficits associated with chronic ankle instability has profoundly influenced contemporary clinical rehabilitation paradigms. Historically, rehabilitation heavily emphasized the restoration of basic range of motion and concentric muscular strength through simple resistance band exercises. However, the modern understanding that chronic ankle instability is fundamentally a sensorimotor neurological disorder dictates a much more sophisticated therapeutic approach. It is now abundantly clear that isolated muscular strengthening, while important, is entirely insufficient to restore dynamic postural control if the neural timing and central integration deficits remain unaddressed. Therefore, modern rehabilitation programs prioritize multi-modal proprioceptive and neuromuscular control training interventions designed specifically to recalibrate the damaged sensorimotor pathways [25]. Proprioceptive training typically involves progressively challenging the patient's balance utilizing a variety of unstable surfaces, such as foam pads, wobble boards, and biomechanical oscillating platforms. The primary objective of these exercises is to deliberately create a highly unpredictable environment that forces the central nervous system to rapidly process complex, multisensory afferent information and generate immediate, coordinated efferent motor responses. By repeatedly exposing the neuromuscular system to these controlled perturbations, the therapy aims to enhance the sensitivity of the remaining peripheral mechanoreceptors, restore the excitability of the depressed spinal reflex pathways, and facilitate positive, functional neuroplasticity within the supraspinal motor cortex. Furthermore, clinicians often incorporate sensory manipulation techniques, such as performing balance tasks with eyes closed or while engaging in secondary cognitive tasks. This dual-task paradigm forces the central nervous system to shift its reliance back to the somatosensory inputs from the lower limb, directly combating the maladaptive visual dependence that is a hallmark of chronic ankle instability [26].

6.2 Advanced Interventions and External Support

In addition to traditional proprioceptive exercise regimens, researchers and clinicians are continuously exploring advanced therapeutic interventions to optimize the restoration of neuromuscular balance control. One such promising avenue is the application of stochastic resonance electrical stimulation. This technique involves the application of a sub-sensory, randomized electrical noise signal to the skin surrounding the injured ankle joint. The underlying theoretical principle is that this carefully calibrated level of background electrical noise can theoretically enhance the signal-to-noise ratio of the diminished proprioceptive signals originating from the damaged mechanoreceptors, effectively amplifying the sensory information ascending to the brain. Preliminary studies have shown that the application of stochastic resonance during unilateral stance tasks can lead to acute reductions in center of pressure sway parameters, suggesting an immediate enhancement of sensorimotor integration.

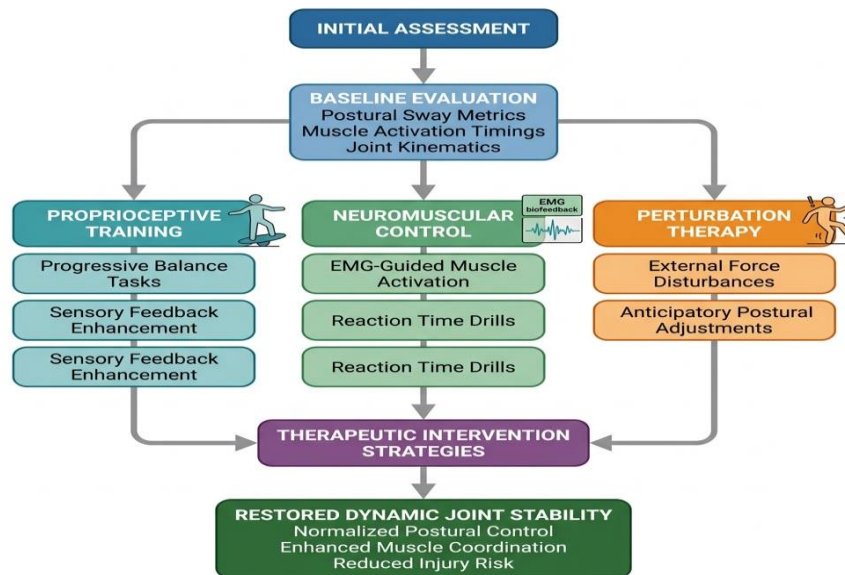


Figure 3: Rehabilitation Algorithm and Therapeutic Intervention Strategies for Restoring Neuromuscular Balance Control

Furthermore, the role of external prophylactic supports, such as rigid ankle braces and specialized athletic taping techniques, remains a significant topic of discussion in the clinical management of balance deficits. While these external supports were originally designed to provide purely mechanical restriction against excessive inversion, contemporary research suggests that their primary benefit during functional tasks may actually be neurological rather than mechanical. The application of tight, compressive taping or bracing significantly increases the cutaneous tactile input around the ankle joint [27]. This enhanced tactile sensation provides the central nervous system with an alternative, supplementary stream of spatial awareness information that partially compensates for the deep ligamentous proprioceptive deficits. Consequently, individuals with chronic ankle instability often demonstrate immediate improvements in unilateral stance stability and reduced peroneal onset latency when wearing external supports. However, clinicians must carefully balance the use of these supports, as excessive long-term reliance may potentially hinder the natural recovery of intrinsic neuromuscular control mechanisms.

7. Conclusion

In conclusion, this comprehensive analysis has elucidated the profound and multifaceted disruptions in neuromuscular balance control that define the functional deficits observed during unilateral stance in individuals with chronic ankle instability. The transition from an acute lateral ankle sprain to a state of chronic pathology is driven by a complex cascade of events, beginning with the direct structural damage and subsequent peripheral deafferentation of vital ligamentous mechanoreceptors. This loss of accurate proprioceptive input initiates a widespread deterioration of the sensorimotor system, characterized by diminished spinal reflex excitability, deleterious central neuroplastic reorganization, and a maladaptive over-reliance on visual cues for maintaining equilibrium. Through the meticulous application of advanced biomechanical methodologies, including highly sensitive force platforms, multi-channel surface electromyography, and three-dimensional kinematic motion capture, researchers have successfully quantified the specific mechanical and neurological manifestations of this disorder. The empirical evidence overwhelmingly demonstrates that individuals with chronic ankle instability exhibit significantly increased center of pressure excursion velocities, dangerously prolonged activation delays in the primary dynamic stabilizing musculature, and a detrimental kinematic shift from an efficient distal ankle strategy to a metabolically demanding proximal hip strategy. These intricate neuromuscular alterations explain why unilateral stance, a fundamental component of daily locomotion, becomes an exceptionally challenging and precarious task for this population. Moving forward, the clinical management of chronic ankle instability must continue to evolve beyond the simplistic mechanical paradigm. Therapeutic interventions must be rigorously designed to target the global sensorimotor

dysfunction, utilizing advanced proprioceptive training, perturbation therapies, and innovative modalities to recalibrate neural pathways and restore intrinsic motor control [28]. Ultimately, a deeper, more refined understanding of these neuromuscular mechanisms is imperative for developing targeted rehabilitation strategies that can successfully break the vicious cycle of recurrent injury, restore optimal functional capacity, and ensure the long-term joint health of individuals afflicted by chronic ankle instability.

References

- Xu, Z., Lu, Y., Tao, X., Cheng, W., Tian, J., Tang, W., & Wu, D. Modelling the geographic spread of HIV among MSM in Guangdong, China: a metapopulation model considering the impact of pre-exposure prophylaxis *Philosophical Transactions A*.
- Li, Y., Liao, X., Yu, X., Xiao, Y., Tao, X., & Zhong, T. (2025). Mediating Role of the ANGPTL3/TFPI Protein Ratio in Regulating T-Cell Surface Glycoprotein CD5 Levels on Knee Osteoarthritis (KOA): A Mendelian Randomization Study. *International Journal of Molecular Sciences*, 26(10), 4471.
- Chai L, Zhang J, Fernando W G D, et al. Detection of blackleg resistance gene Rlm1 in double-low rapeseed accessions from Sichuan province, by kompetitive allele-specific PCR[J]. *The Plant Pathology Journal*, 2021, 37(2): 194.
- Gulshan, V., Peng, L., Coram, M., Stumpe, M. C., Wu, D., Narayanaswamy, A., et al. (2016). Development and validation of a deep learning algorithm for detection of diabetic retinopathy in retinal fundus photographs. *JAMA*, 316(22), 2402–2410.
- Zhang, Y., & Chen, Y. (2025, May). Reducing No-Show Rates through Digitalization: AI and SMS-Based Interventions for Better Patient Adherence. In *Proceedings of the 2025 International Conference on Digital Economy and Intelligent Computing* (pp. 123-127).
- Gao, B., Dai, J., Yu, X., Cao, S., Wu, C., Zhu, C.,... & Meng, J. (2026). The Prediction of Extended Hospital Length of Stay in Patients After Endoscopic Endonasal Transsphenoidal Surgery for the Resection of Non-Functioning Pituitary Adenomas: A Dual-Center Retrospective Analysis. *Cancers*, 18 (10), 1582.
- Altschul, S. F., Madden, T. L., Schäffer, A. A., Zhang, J., Zhang, Z., Miller, W., & Lipman, D. J. (1997). Gapped BLAST and PSI-BLAST: A new generation of protein database search programs. *Nucleic Acids Research*, 25(17), 3389–3402.
- Shi, M., & Chow, S. C. (2024). Statistical Analysis for Rating Scale in Clinical Trials. *Therapeutic Innovation & Regulatory Science*, 58(5), 930-945.
- Sinha, D., Tong, Y., Zepeda, M. A. F., Hanstad, G., Nelson, E. C., Theisen, C. O., ... & Gamm, D. M. (2023). Silica nanocapsules as a nonviral delivery platform for iPSC-RPE. *Investigative Ophthalmology & Visual Science*, 64(8), 774-774.
- Wang, G., Zhou, Y., Deng, K., Cheah, W. P., Qu, R., Ren, J., & Shen, L. (2026). SurgCoT: Advancing Spatiotemporal Reasoning in Surgical Videos through a Chain-of-Thought Benchmark. *arXiv preprint arXiv:2604.20319*.
- Wang, G., Zhong, Z., Zhou, Y., Li, Y., Wu, E., Cheah, W. P., ... & Shen, L. (2026). X-PCR: A Benchmark for Cross-modality Progressive Clinical Reasoning in Ophthalmic Diagnosis. *arXiv preprint arXiv:2604.20350*.
- Xu, X., Bowtell, J., Young, W. R., Fong, D. T., & Williams, G. K. (2025). Distinct patterns in neuromuscular adaptation to repeated perturbations in chronic ankle instability. *Journal of NeuroEngineering and Rehabilitation*.
- Xu, X., Bowtell, J., Young, W. R., Fong, D. T., & Williams, G. K. (2026). Increased common corticospinal input during eyes-closed unilateral stance in people with chronic ankle instability. *Scientific Reports*.
- Wang, G., Li, Y., Chen, W., Ding, M., Cheah, W. P., Qu, R., ... & Shen, L. (2025, April). S³-mamba: Small-size-sensitive mamba for lesion segmentation. In *Proceedings of the AAAI Conference on Artificial Intelligence* (Vol. 39, No. 7, pp. 7655-7664).

- Ma, H., Dai, X., Wang, T., He, Z., Du, R., Pei, H.,... & Zhao, K. (2025). Investigation of uridine in anti-aging-related diseases based on network pharmacology, molecular docking and cell experimental validation. *Engineered Science*, 35, 1572.
- Jia, J., Sun, Z., Tao, X., Tong, Y., Feng, H., Yang, J., ... & Wu, J. (2024). Association between oxidative balance score and heart failure in the older adults: results from the NHANES 2005–2018. *Heart & Lung*, 68, 107-115.
- Eraslan, G., Avsec, Ž., Gagneur, J., & Theis, F. J. (2019). Single-cell transcriptomics meets deep learning. *Nature Methods*, 16(11), 1083–1091.
- Lei, C., Tao, X., & Xiao, Y. (2023, May). Dietary Iodine intake and related factors among secondary school students in Macao. In *Healthcare* (Vol. 11, No. 10, p. 1472). MDPI.
- Chai L, Zhang J, Lu K, et al. Identification of genomic regions associated with multi-silique trait in *Brassica napus*[J]. *BMC genomics*, 2019, 20(1): 304.
- Kabra, M., Shahi, P. K., Wang, Y., Sinha, D., Spillane, A., Newby, G. A., ... & Pattnaik, B. R. (2022). Nonviral base editing of KCNJ13 mutation preserves vision in an inherited retinal channelopathy. *bioRxiv*, 2022-07.
- Zhao, K., Li, G., Li, C., Jiang, T., Wang, W., & Yang, Z. (2026). Identification Markers for *Salvia miltiorrhiza* and Its Close Relatives Based on Cell Wall Component Characteristics. *Engineered Science*, 40, 2122.
- Yu, X., Zhang, Y., Yao, Y., Cho, W. C., & Shang, A. (2025). Biofilm-mediated immune dysregulation in chronic pulmonary diseases: mechanisms and clinical implications. *Frontiers in Microbiology*, 16, 1736384.
- Jiang, Z., Hou, Z., Yu, X., Zhao, Z., Bu, J., Li, C.,... & Wang, C. (2025). SPECT-derived myocardial perfusion and viability as predictors of response to left bundle branch pacing for cardiac resynchronization therapy. *Europace*, 27 (12), euaf301.
- Shi, X., Yu, X., Xiong, Y. J., Lv, T., & Si, X. (2026). Long-term mortality trends in patients with concurrent hernia and diabetes in the United States. *Frontiers in Endocrinology*, 17, 1723074.
- Zhang, W., Luo, Z., Gu, C., Ma, J., Cao, Y., Yuan, W., & Jin, Y. (2025). Bridging the compression-precision paradox: A hybrid architecture for clinical EEG report generation with guaranteed measurement accuracy. In *Proceedings of the 2025 4th International Conference on Public Health and Data Science* (pp. 201–207).
- Li, Z. R., Wang, Y. Y., Zhang, C., Shi, J. S., Yu, X., Ying, N. T.,... & Guo, T. (2025). Features and functional mechanisms of super-enhancers in cardiovascular disorders, cancer, autoimmune diseases and neurodegenerative disorders. *Cellular Signalling*, 112252.
- Verhoven, B., Tong, Y., Chlebeck, P., Zhong, W., Zeng, W., Jennings, H., ... & Al-Adra, D. P. (2025). Attenuating Ischemia and Reperfusion Injury Using NAD⁺-Loaded Nanoparticles in Mouse Kidneys. *Transplantation Direct*, 12(1), e1890.
- GAO, T., LIANG, D., CHEN, X., & TAO, X. (2025). PREDICTIVE ANALYTICS IN PEDIATRIC MENTAL HEALTH: A MACHINE LEARNING APPROACH TO DEPRESSION DETECTION BY PHYSICAL ACTIVITY AND BEHAVIORAL HEALTH INDICATORS. *INTERCIENCIA*.
- Li, X., Chen, W., Liu, X., & Ren, M. (2015). A high-order sigma-delta accelerometer interface circuit. *IEICE Electronics Express*, 12(2), 20141035-20141035.