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Joint Hypermobility in Chiropractic Practice: Part 2: Manipulation safety and evidence-based rehabilitation

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Narrative: Management of symptomatic joint hypermobility requires careful clinical reasoning, particularly in Chiropractic practice where spinal manipulation is commonly used. While manipulation may provide short-term analgesia through neurophysiological mechanisms, it does not restore ligament restraint and may aggravate instability when applied repeatedly to hypermobile segments.

This review summarises evidence-based Chiropractic management principles, focusing on when manipulation should be avoided, how instability influences clinical safety, and which rehabilitation strategies demonstrate benefit. Progressive strengthening, proprioceptive retraining, motor control exercise, pacing, and education form the core of long-term improvement. Chiropractors play an important role when care is stabilisation-oriented, function-focused, and aligned with modern evidence.

Indexing terms: Chiropractic; Chiropractic adjustment; Chiropractic manipulation; instability; rehabilitation; proprioception; hypermobile Ehlers-Danlos syndrome.

Introduction

Symptomatic joint hypermobility presents a distinct clinical challenge for chiropractors. Unlike conditions defined primarily by restriction or degenerative stiffness, hypermobility disorders are characterised by excessive motion, impaired passive restraint, proprioceptive deficits, and reduced tolerance to mechanical load. (1) As a result, pain patterns are often fluctuating, multifactorial, and poorly served by intervention models focused predominantly on increasing mobility. (2)

> *'... Chiropractors must consider whether the symptomatic region is functioning as an unstable cost centre requiring active control rather than additional mobility...'*

Part 1 of this review outlined the clinical spectrum of hypermobility, mechanisms of instability, and principles of recognition in chiropractic practice. (3) This second paper focuses on management priorities, including manipulation safety, instability-aware clinical reasoning, and evidence-based

rehabilitation strategies.

Manipulation safety in hypermobility

Manual therapy may provide short-term analgesia through neurophysiological modulation of spinal and supraspinal pain processing mechanisms. (4) Spinal manipulation also alters afferent input from paraspinal tissues and reflex neuromuscular activity. (5) Mechanical investigations demonstrate characteristic force-time profiles and spinal tissue loading during high-velocity low-amplitude spinal manipulation procedures. (6) Similar biomechanical findings have been reported in laboratory investigations of spinal manipulation mechanics. (7)

Clinical trials evaluating manual therapy for mechanical neck pain demonstrate short-term improvements in pain and function following spinal manipulation procedures. (8) However, in hypermobility populations, the clinical question is not simply whether manipulation can reduce symptoms transiently, but whether thrust-based interventions meaningfully address the dominant impairment of instability and poor restraint.

Hypermobile patients may experience temporary symptom relief following adjustment or manipulation, yet repeated passive care without stabilisation progression risks reinforcing dependency, symptom cycling, or end-range irritability. (9) Contemporary rehabilitation frameworks for hypermobility emphasise progressive exercise rather than reliance on passive treatment alone. (2)

Evidence syntheses evaluating manual therapy interventions also emphasise that manipulation should be integrated within multimodal care rather than delivered as repeated isolated treatment. (10)

> In already lax segments, increasing motion is rarely the therapeutic priority. Instead, Chiropractors must consider whether the symptomatic region is functioning as an unstable cost centre requiring active control rather than additional mobility. (11)

Contemporary Chiropractic clinical practice guidelines similarly emphasise rehabilitation exercise, patient education, and functional restoration in the management of spinal pain disorders. (12)

Cervical Hypermobility and Neurovascular Safety Constraints

Cervical presentations in hypermobile patients require careful differentiation between benign mechanical pain and symptom profiles constrained by neurovascular or craniocervical instability risk. While most neck pain is not vascular in origin, the clinical consequences of missing cervical arterial dysfunction are high-stakes, and connective tissue disorder phenotypes may introduce additional complexity. (13)

Population-based research has demonstrated that Chiropractic consultation rates preceding vertebrobasilar stroke are similar to those observed in primary care settings. (14) This finding supports the interpretation that early arterial dissection symptoms may drive care-seeking behaviour prior to diagnosis. (15)

Systematic reviews examining the relationship between Chiropractic care and cervical artery dissection have concluded that current evidence does not demonstrate a causal association. (16) Earlier reviews similarly emphasised that rare vascular events require careful differential diagnosis rather than simple attribution of causality. (17)

Contemporary safety reasoning has moved beyond reliance on vertebrobasilar insufficiency provocation tests alone and instead emphasises precautionary history constraints, irritability monitoring, and escalation pathways when cervical arterial dysfunction or upper cervical instability is suspected. (18)

Precautionary history should include transient neurological disturbances, autonomic instability, migraine with aura, recent infection, thrombotic modifiers, minor trauma, and rare differentials such as Eagle syndrome. These do not establish deterministic causality, but they constrain thrust manipulation decision-making in favour of stabilisation-oriented care and referral where indicated. (19)

Table 1. Clinical factors relevant to manipulation safety (Part 2)

Domain	History factors raising precaution	Examination considerations	Clinical implication
Connective tissue disorder phenotype	Lifelong hypermobility, recurrent sprains/subluxations, easy bruising, family history	Excessive cervical range with poor end-range control	Avoid thrust manipulation. Stabilisation-oriented care and referral where indicated
Cervical arterial dysfunction symptom cluster	Dizziness, diplopia, dysarthria, dysphagia, drop attacks, sensory disturbance	Symptoms provoked by sustained positions or rapid movement	Escalation pathways. Avoid provocative cervical procedures
Systemic CAD risk modifiers	Recent acute infection, smoking, pulsatile tinnitus, minor trauma, ipsilateral periorbital or upper neck pain, new progressive headache, partial Horner's syndrome, retinal/ cerebral ischaemic symptoms (29)	Non-specific exam but high-stakes context	Conservative manual care. Consider referral
Pain location red flag	Anterior neck pain is uncommon and concerning	Requires careful differential diagnosis	Heightened vascular precaution
Contextual thrombotic modifiers	Migraine with aura, combined oral contraceptive use, thrombophilia history, smoking	Not deterministic but contributes to vascular context	Not standalone contraindication, but constrains thrust decision-making when combined
Rare differential diagnosis	Eagle syndrome (styloid or stylohyoid ligament carotid compression)	Often missed unless considered	Referral in atypical cases
Mechanistic caution	deep anterior cervical soft-tissue pressure may represent greater risk than thrust procedures	Soft tissue is not automatically low risk	Avoid aggressive anterior techniques

Table note: These factors represent precautionary constraints rather than deterministic predictors. Associations do not confirm causality. In hypermobility phenotypes, excessive cervical motion combined with autonomic symptoms or connective tissue disorder features should heighten precaution, as instability and vascular differentials may be more difficult to exclude clinically (19). For example, a young hypermobile patient presenting with new unilateral headache, anterior neck pain, pulsatile tinnitus, or syncope requesting cervical manipulation represents a context where cervical arterial dysfunction and craniocervical instability must be carefully considered before any thrust intervention (18).

Evidence-based rehabilitation approaches

The strongest evidence for managing symptomatic hypermobility supports rehabilitation strategies emphasising stability, neuromuscular control, endurance, and graded exposure. (2) Hypermobility is therefore rarely a disorder requiring increased mobility. It is more accurately a stability and load tolerance disorder, where excessive motion exists alongside reduced control and poor proprioceptive integration. (20) Contemporary clinical classifications of hypermobility spectrum disorders similarly emphasise impaired connective tissue restraint and neuromuscular control. (1)

Table 2: Rehabilitation Progression Framework

Phase	Goal	Clinical focus	Example exercises	Progression marker
1	Control foundation	Mid-range stability, flare reduction	Isometrics, joint setting, trunk activation	Symptoms stable with low-load tasks
2	Endurance stability	Fatigue-resistant control	Bird-dog holds, scapular endurance drills	Reduced giving-way episodes
3	Sensorimotor integration	Proprioceptive accuracy	Balance drills, perturbation exposure	Improved confidence and control
4	Functional strength	Load tolerance in tasks	Squats, carries, step-down control	Functional activity without flare
5	Resilience and return-to-task	Participation durability	Sport-specific drills, graded impact progression	Sustained stability under demand

Progressive strengthening

Progressive resistance training improves active joint stability by enhancing muscular restraint and increasing tolerance to mechanical load. (21) Randomised trials in children with joint hypermobility syndrome have demonstrated meaningful reductions in knee pain and improvements in function following strengthening programmes. (21)

Structured rehabilitation programmes have also been shown to improve physical capacity and reduce symptom burden in hypermobility populations. (22) Importantly, the therapeutic effect arises not from restricting mobility but from improving the capacity of surrounding musculature to compensate for reduced ligament stiffness.

Engelbert et al. emphasised that strengthening programmes in hypermobility syndromes should prioritise neuromuscular endurance and dynamic joint support rather than maximal force production. (2) Clinically, programmes should begin with low-load mid-range control tasks before progressing toward higher demand functional movements. Early aggressive end-range loading may provoke symptom flares in irritable unstable joints. (9)

Proprioceptive and sensorimotor training

Proprioceptive deficits are well documented in individuals with hypermobility and contribute directly to instability risk. (20) Impaired joint position sense has been demonstrated in patients with hypermobile Ehlers-Danlos syndrome compared with healthy controls. (20) Reduced proprioceptive acuity limits the neuromuscular system's ability to regulate joint position and maintain mechanical stability during functional tasks (23).

Rehabilitation approaches targeting proprioception have demonstrated improvements in symptoms and functional stability. (23) Proprioceptive training programmes can improve joint position awareness and reduce symptom severity in patients with joint hypermobility syndrome. (23)

Clinically, sensorimotor rehabilitation may include balance training, perturbation exposure, closed-chain stability tasks, and coordination exercises that challenge joint control under gradually increasing load. (2)

Motor control and spinal stabilisation

Spinal symptoms in hypermobile patients often reflect impaired stabilisation strategies rather than structural pathology alone. Delayed activation of the transversus abdominis muscle has been demonstrated in individuals with chronic low back pain, highlighting motor control impairment as a central mechanism in spinal dysfunction. (24)

Exercise therapy has strong evidence for improving outcomes in chronic non-specific low back pain, particularly when programmes focus on control, endurance, and progressive functional loading. (25) Spinal stability depends on interaction between passive restraints, muscular control, and neural coordination. (26)

When passive ligamentous support is compromised, as in hypermobility syndromes, neuromuscular control becomes increasingly important in maintaining functional stability. Rehabilitation programmes therefore prioritise endurance-based stabilisation exercises, trunk control training, and progressive functional loading tasks designed to improve the capacity of the active stabilising system. (11)

Kinetic chain load-sharing and the symptomatic cost centre

A clinically useful construct in symptomatic hypermobility is that painful segments may represent the cost centre of task demand rather than the sole driver of dysfunction. In kinetic chain models of human movement, mechanical load is distributed across multiple segments and tissues during functional tasks. (27)

When one link in the system under-contributes because of stiffness, motor control deficit, or fatigue, other regions may compensate by absorbing greater mechanical demand. (28)

In hypermobility syndromes, excessive motion may therefore occur in regions that are repeatedly recruited to compensate for reduced contribution elsewhere in the chain. This phenomenon can increase eccentric braking requirements in vulnerable tissues and may contribute to symptom recurrence. (28)

> Kinetic chain reasoning should be applied as a clinically testable hypothesis rather than a deterministic rule. Functional reassessment following targeted rehabilitation of adjacent regions can help determine whether a symptomatic joint is the primary driver of dysfunction or the site where load failure becomes clinically apparent. Durable improvement depends on graded capacity building across the entire movement system rather than repeated passive care directed at a single segment.

Clinical instability vignettes

Vignette 1:
Multidirectional shoulder instability presenting as rotator cuff tendinopathy

A 19-year-old overhead athlete presents with lateral shoulder pain during serving. She reports slipping sensations rather than traumatic dislocation. History reveals lifelong flexibility and recurrent sprains, raising suspicion of a generalised hypermobility phenotype.

Examination demonstrates excessive external rotation, translation in multiple directions, scapular dyskinesis, and pain on resisted cuff testing. The dominant impairment is instability-driven load failure rather than primary tendinopathy.

Rehabilitation should prioritise scapular control, rotator cuff endurance, proprioceptive retraining, and graded exposure rather than further mobilisation. (28) These rehabilitation priorities are consistent with stability-focused exercise approaches recommended for hypermobility syndromes. (2)

Vignette 2:
Low lumbar instability with short-lived response to manipulation

A 42-year-old man reports episodic low back pain provoked by sustained standing and bending. Manipulation provides only transient relief with rapid recurrence.

Examination demonstrates excessive mid-lumbar motion, reduced trunk endurance, and hip sequencing deficits, with early lumbar substitution during extension tasks.

This pattern aligns with instability-dominant degenerative trajectories described in biomechanical models of spinal stability. (26) Repeated thrust into a lax segment may reinforce symptom cycling, whereas endurance-based motor control rehabilitation is more consistent with evidence-based management of chronic spinal pain. (25)

Vignette 3:
Connective tissue disorder phenotype with cervical neurovascular constraint

A 28-year-old woman presents with chronic neck pain, episodic dizziness, fatigue, recurrent sprains, easy bruising, and orthostatic intolerance. She requests cervical manipulation due to brief prior relief.

Examination reveals marked cervical range with poor restraint rather than restriction. History raises concern for symptomatic connective tissue disorder phenotype with possible craniocervical instability risk.

In this context, HVLA thrust is inappropriate. Contemporary safety framing emphasises cervical arterial dysfunction constraints rather than outdated provocation testing. (18) International manual therapy safety frameworks emphasise structured history screening and escalation pathways when vascular symptoms are present. (19)

Conclusion

Symptomatic hypermobility is not primarily a mobility disorder. It is a stability and load tolerance disorder. Chiropractors contribute most effectively when care is aligned with evidence-based principles: strengthening, proprioceptive retraining, motor control rehabilitation, pacing, and cautious tool selection whether the chiropractor provides it or refers for it. Symptomatic hypermobility is not primarily a mobility problem. It is a stability and load tolerance disorder requiring active restraint, neuromuscular control, and graded capacity building.

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