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Joint Hypermobility in Chiropractic Practice: Spectrum, Recognition, Spinal and Extremity Laxity, and Evidence-Based Care

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Narrative: Joint hypermobility is common and frequently under-recognised in Chiropractic practice. This review defines joint laxity and hypermobility, presents mobility as a clinical spectrum, outlines major conditions associated with symptomatic hypermobility, and summarises evidence-based assessment and management strategies relevant to chiropractors.

Hypermobility exists on a continuum from asymptomatic increased range to disabling instability. Reliable tools include the Beighton score, validated screening questionnaires, and limb-specific assessments. Hypermobility predisposes to recurrent dislocations, patellar instability, chronic ankle sprains, and secondary overload syndromes such as rotator cuff tendinopathy.

Spinal laxity also occurs in connective tissue disorders and degenerative lumbar instability. Evidence supports rehabilitation focused on strengthening, proprioception, pacing, and education.

Spinal manipulation may provide short-term analgesia but repeated thrust techniques in unstable joints may worsen symptoms and disability trajectories over time. Chiropractors play an important role in recognising hypermobility, avoiding inappropriate mobilisation of unstable segments, and delivering evidence-based care centred on functional stability.

Indexing terms: Chiropractic; joint hypermobility; hypermobility spectrum disorder; Ehlers-Danlos syndrome; manipulation; adjustment, rehabilitation; instability.

Introduction

Joint hypermobility is common in the general population. For many people, it causes no problems at all. They are simply flexible. Others, however, experience pain, instability, repeated injury, and long-term disability. In these patients, hypermobility becomes clinically important. This is especially relevant in chiropractic practice, where patients often present with persistent musculoskeletal pain that does not fit simple mechanical patterns. (1)

'... Recognising hypermobility early allows chiropractors to avoid inappropriate thrust techniques, prioritise stabilisation, and refer appropriately when systemic risk is suspected...'

Hypermobility is not a diagnosis by itself. It is a physical trait. Its

meaning depends on context. Some individuals have increased joint motion without symptoms, while others develop complex syndromes involving connective tissue, proprioceptive impairment, fatigue, and multisystem complaints. (2) These symptomatic forms are now grouped under modern classifications of hypermobility-related disorders. (1)

For Chiropractors, recognising hypermobility is essential. It influences clinical reasoning, risk assessment, and treatment planning. Spinal and extremity manipulation may provide short-term pain relief, but in unstable joints, repeated mobilisation may worsen irritability and functional outcomes over time. (3) Understanding when mobility is adaptive, and when it signals instability, is therefore a core clinical skill.

This review provides a practical and evidence-based framework for Chiropractors. It defines laxity and hypermobility, explains the spectrum of joint mobility, outlines conditions associated with symptomatic hypermobility, and discusses assessment and management options supported by peer-reviewed research.

Definitions

Joint Laxity and Joint Hypermobility

Joint laxity refers to excessive passive movement within a joint due to reduced stiffness of supporting structures such as ligaments, capsules, or connective tissue restraints. It is primarily a structural concept. Laxity is important because it reduces passive stability, meaning that joints rely more heavily on muscular control to remain aligned during movement. (4)

Laxity is clinically relevant in extremity and spinal disorders. In the shoulder, laxity contributes to multidirectional instability and recurrent subluxation. (5) In the knee, laxity can predispose to patellar instability. (6) In the lumbar spine, segmental laxity plays a role in degenerative instability trajectories. (7) Joint laxity is therefore not simply 'extra movement', it is a biomechanical vulnerability.

Joint hypermobility describes the ability of a joint to move beyond expected physiological limits, accounting for age, sex, and ethnicity. (2) It is a clinical finding rather than a disease. Hypermobility may be localised to one region or generalised across multiple joints.

Some hypermobility is benign. However, when hypermobility is accompanied by pain, instability, fatigue, or functional impairment, it becomes part of a broader clinical syndrome. Modern classification recognises this spectrum through hypermobility spectrum disorders and hypermobile Ehlers-Danlos syndrome. (1) The distinction matters. A flexible dancer with no symptoms is not the same as a patient with recurrent dislocations and chronic pain.

Hypermobility spectrum disorders and hypermobile Ehlers-Danlos syndrome

The 2017 classification of the Ehlers-Danlos syndromes clarified diagnostic criteria for hypermobile EDS and positioned hypermobility spectrum disorders as symptomatic hypermobility that does not meet hEDS criteria. (1, 8) Hypermobility spectrum disorders are common in musculoskeletal practice and often present with pain, instability, proprioceptive impairment, and reduced functional tolerance. (2)

Subluxation

Medical and Chiropractic concepts

The term subluxation has different meanings in different clinical contexts. In medicine and orthopaedics, subluxation refers to a partial dislocation where joint surfaces lose normal anatomical alignment. This is a structural diagnosis, commonly associated with trauma or connective tissue disorders, and is often confirmed radiologically. (9)

In Chiropractic, the WHO guideline definition describes subluxation as:

> a lesion or dysfunction in a joint or motion segment in which alignment, movement integrity and/or physiological function are altered while contact between joint surfaces remains intact. It is described as a functional entity that may influence biomechanical and neural integrity, and the purported displacement is not necessarily visible on static imaging. (10)

This distinction is critical in hypermobility care. In unstable hypermobile joints, the clinical problem is not a fixed displacement to correct. The problem is insufficient restraint and poor control. Management must prioritise stability rather than repeatedly increasing motion.

Clinical relevance for Chiropractors

Hypermobility challenges traditional assumptions in manual therapy. More motion is not always better. In symptomatic patients, the goal is often control, not mobility.

Spinal manipulation may reduce pain through neurophysiological mechanisms, including modulation of pain sensitivity. (11) However, manipulation does not restore ligament stiffness or passive restraint. (3) In hypermobile and unstable joints, repeated end-range mobilisation may contribute to worsening irritability and functional decline over time. (3) Recognising hypermobility early allows chiropractors to avoid inappropriate thrust techniques, prioritise stabilisation, and refer appropriately when systemic risk is suspected.

Mobility as a clinical spectrum and conditions associated with hypermobility

Joint mobility is not best understood as normal versus abnormal. In reality, movement exists along a continuum. Some joints move more than expected, some less, and both extremes can generate clinical problems.

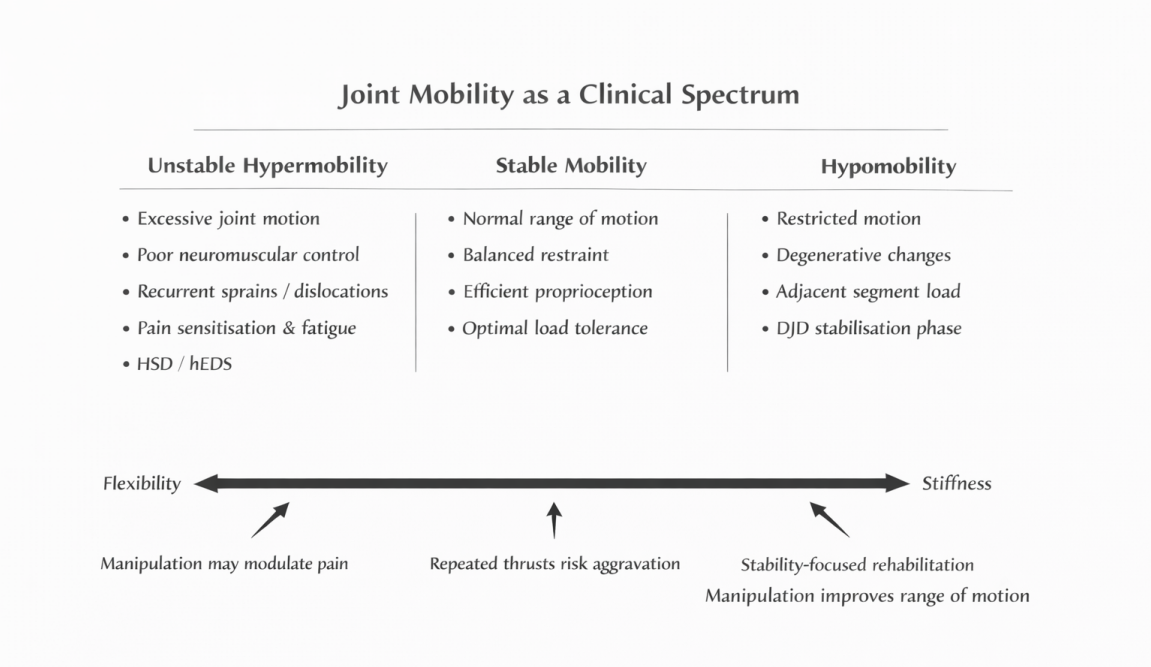
At one end of this spectrum lies unstable hypermobility. Here, joint motion exceeds normal limits and is accompanied by reduced connective tissue restraint and impaired neuromuscular control. Patients often describe joints that feel unreliable. Sprains recur easily. Episodes of giving way or dislocation may occur without major trauma. (2)

In the middle lies stable mobility. This is the zone of healthy function. Joints move freely but remain well supported by passive structures and coordinated muscle control. Load tolerance is high. Symptoms are minimal.

At the opposite end lies hypomobility. Restricted motion is often linked with stiffness, protective guarding, or degenerative adaptation. In the spine, this may contribute to segmental overload and adjacent compensation over time. (7)

These states often coexist. Hypermobile patients frequently develop stiff protective regions, especially in the spine. (3) Excess motion in one segment may coexist with restriction in another. This complexity explains why simplistic approaches, such as treating every painful area as a site of restriction, can fail in hypermobile care.

Figure 1: Joint mobility as a clinical spectrum (unstable hypermobility to stable mobility to hypomobility).



Hypermobility itself is common, particularly in childhood and adolescence. In many individuals it is a normal variant. (12) The clinical challenge arises when hypermobility is accompanied by symptoms. Modern classification recognises a spectrum of symptomatic disorders, including hypermobility spectrum disorders and hypermobile Ehlers-Danlos syndrome. (1, 8)

Hypermobility spectrum disorders describe symptomatic hypermobility without full hEDS criteria. (2) Presentations are common in musculoskeletal practice and may include persistent pain, recurrent injury, instability sensations, and reduced tolerance to sustained load. (2) Proprioceptive impairment and reduced neuromuscular control often become key drivers of symptoms. (4)

Hypermobile EDS represents the more complex end of the spectrum, with broader systemic involvement. (8) Autonomic dysfunction and orthostatic intolerance are well described and contribute to dizziness, syncope, exercise intolerance, and fatigue. (13, 14) Psychological comorbidity may also be more frequent, including anxiety vulnerability in some cohorts (15, 16) These associations do not imply symptoms are psychological in origin. They reflect broader systemic complexity.

Not all hypermobility is inherited. Joint laxity can also be acquired after injury or physiologic change, including pregnancy-related ligamentous change. (17) Repetitive end-range stretching may worsen restraint in predisposed individuals. This is clinically relevant because flexibility-focused programmes can unintentionally increase instability.

Extremity laxity

Hypermobility is often most visible in the extremities. Patients rarely present complaining of 'hypermobility'. Instead, they present with repeated injuries, persistent tendon pain, or joints that do not feel stable. In many cases, laxity is the underlying driver.

Joint laxity reduces passive restraint and shifts the burden of stability onto the neuromuscular system. (4) When control is insufficient, joints begin to fail under load. Recurrent extremity problems are therefore often part of an instability pattern.

Recurrent dislocations and joint instability

In hypermobility disorders, dislocations and subluxations may occur with minimal force and may recur across the lifespan. The shoulder is a common example. Generalised joint hypermobility is associated with multidirectional glenohumeral instability, and patients may describe slipping or clunking during ordinary activity. (5)

Patellar instability is another hallmark. In hypermobile populations, recurrent patellar subluxation or dislocation is common, particularly in adolescence, and hypermobility may influence outcomes. (6) Chronic ankle sprains and functional instability are also common and can lead to long-term activity limitation. (18)

Tendinopathy as a secondary overload syndrome

A frequent clinical mistake is to treat tendon pain in hypermobile patients as a local tissue problem. Often, it is not. Hypermobility increases demand on dynamic stabilisers. Muscles and tendons must work harder to compensate for passive laxity. Over time, this can contribute to overload syndromes, tendinopathy, and persistent pain.

Rotator cuff related shoulder pain illustrates this. Instability can increase shear forces across the cuff during arm elevation. (19) In hypermobile patients, tendon pain may reflect a failure of stability rather than simple overuse. The same principle applies at the knee, where patellofemoral pain commonly coexists with instability and altered tracking. Rehabilitation must address control, not just symptoms.

Proprioception and neuromuscular control

Proprioceptive deficits are well documented in hypermobile populations. (20) Reduced joint position sense limits protective control and increases injury risk. Enhancing proprioceptive input through targeted rehabilitation can reduce symptoms and improve function. (4) See table 1.

Table 1: Clinical factors relevant to hypermobility recognition (Part 1)

Extremity condition	Hypermobility contribution	Clinical implication	Key references
Multidirectional shoulder instability	Reduced passive restraint increases translation	Stabilisation more important than mobilisation	(5)
Rotator cuff related shoulder pain	Dynamic stabiliser overload due to instability	Treat control deficit alongside tendon pain	(19)
Patellar instability and recurrent dislocation	Ligament laxity and altered tracking	Strength and hip control essential	(6)
Chronic ankle instability	Impaired restraint and proprioceptive deficit	Balance and neuromuscular training required	(18)
Widespread pain with hypermobility	Overload plus sensitisation risk	Pain-aware, multidisciplinary care may be needed	(21)

Extremity manifestations are among the most disabling aspects of symptomatic hypermobility. Pain is often not caused by restriction. It is caused by excess motion without control.

Spinal Laxity, Degenerative Instability, and Disability Trajectories

Hypermobility is often discussed in relation to the extremities. However, spinal laxity is equally important. Chiropractors frequently manage spinal pain, and hypermobility in spinal segments changes both diagnosis and treatment risk.

Spinal stability reflects a balance between passive restraints, muscular control, and neural coordination. (22) Painful segments in hypermobile patients may represent the point of load failure within a broader kinetic chain rather than the sole source of dysfunction. (23) When passive support is compromised, the system relies more heavily on active control. In practice, painful segments in hypermobile patients may represent the point of load failure within a broader kinetic chain rather than the sole source of dysfunction.

Spinal hypermobility in heritable disorders

In hypermobile EDS and related disorders, spinal pain is common and may be accompanied by fatigue and broader pain burden. (8, 24) Proprioceptive impairment also affects spinal control and may contribute to recurrent pain episodes. (20)

Upper cervical involvement requires special caution. Neurological and spinal manifestations of EDS have been described, and suspected craniocervical instability requires referral rather than thrust manipulation. (25)

Degenerative joint disease and instability

A common misconception is that degeneration always produces stiffness. Degenerative spinal change often passes through an instability phase in which excessive motion contributes to pain recurrence and disability. (7) This is clinically relevant in older adults and in disabled populations where instability and deconditioning interact.

Motor control impairment is strongly linked with chronic spinal pain. Hodges and Richardson demonstrated inefficient deep trunk stabilisation associated with low back pain. (26) Exercise therapy has strong evidence for non-specific chronic low back pain and aligns with stabilisation-oriented care. (27)

In both heritable and degenerative contexts, excess motion may be the problem, not restriction.

Clinical recognition and assessment of hypermobility

Hypermobility is frequently missed in routine musculoskeletal care. Many patients are treated for isolated pain sites without recognition of the underlying stability problem.

Recognition begins with history. Patients often describe lifelong patterns rather than acute onset problems. They may recall being 'double-jointed' as children, with symptoms increasing over time. (2)

History features suggestive of symptomatic hypermobility include recurrent sprains, repeated instability episodes, or dislocations with minimal trauma. (5) Pain may be diffuse, shifting, and accompanied by tendon complaints that do not resolve with standard care. (19) Fatigue and autonomic symptoms may be prominent in hEDS. (13) Psychological distress may also occur, including anxiety vulnerability. (15)

Examination

The Beighton score is widely used to assess generalised joint hypermobility. (28) However, Beighton alone has limitations. It does not capture limb-specific instability, proprioception, or functional control deficits. Clinical assessment should include movement quality, mid-range control, and stabiliser endurance.

The 5-part questionnaire is a useful screening adjunct, particularly in adults who may have stiffened with age. (29) Limb-specific tools such as the Lower Limb Assessment Score improve detection of lower extremity hypermobility. (30) Proprioceptive deficits are well documented and support including joint position sense and balance testing in assessment. (20)

Referral awareness is essential when systemic risk is suspected, including severe autonomic symptoms or suspected cervical instability. (13, 25)

Table 2: Clinical tools for recognition and assessment

Tool or domain	What it assesses	Clinical use	Key references
Beighton score	Generalised joint hypermobility	Screening, especially in youth	(28)
5-part questionnaire	Historical hypermobility traits	Useful in adults and subtle cases	(29)
LLAS	Limb-specific hypermobility	Relevant for knee and ankle instability	(30)
Proprioceptive testing	Joint position awareness deficits	Guides rehab priorities	(20)
Motor control assessment	Functional stability	Identifies control impairment	(26)

Manipulation

Short-term benefit, contraindications, and long-term risk

Detailed discussion of manipulation safety and rehabilitation strategies is presented in Part 2 of this review. Spinal adjustment / manipulation is central to Chiropractic practice. In people with hypermobility, it raises a clinical question. When does mobilisation relieve pain safely, and when might it aggravate an unstable system?

Manual therapy can provide short-term symptom improvement through neurophysiological mechanisms. Manipulation influences pain processing and may reduce guarding and pain sensitivity. (11) These responses explain immediate relief in some hypermobile patients.

The difficulty is that pain relief does not necessarily indicate improved stability.

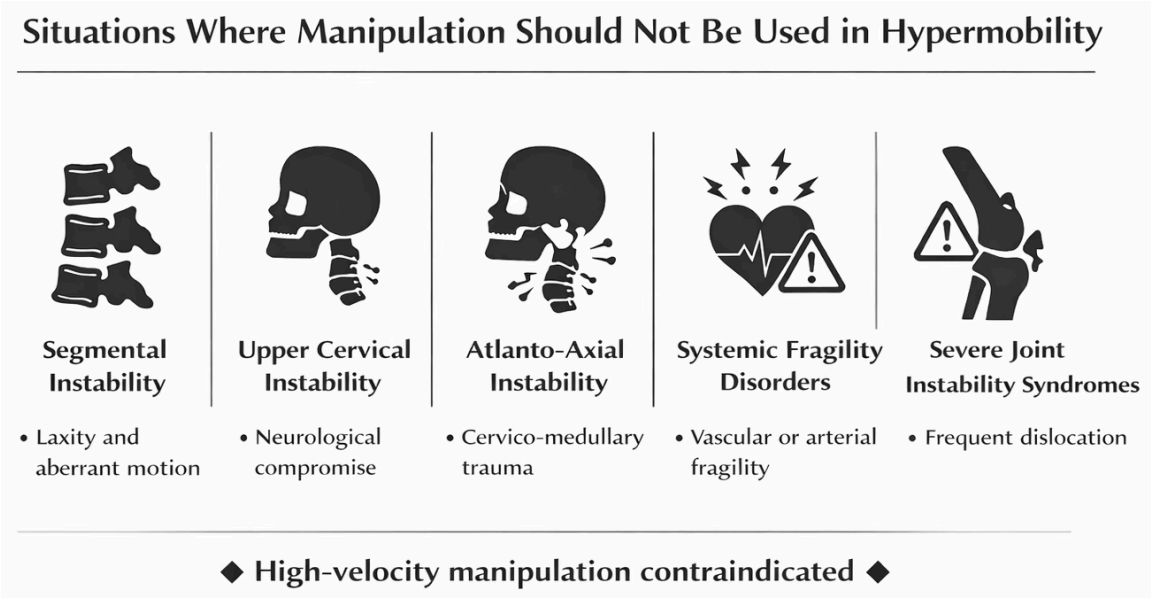
> Manipulation / adjustment does not restore ligament stiffness or passive restraint. (3) This does not mean manipulation has no role in care, but rather that its effects should be interpreted primarily as short-term symptom modulation rather than structural correction. In symptomatic hypermobility, excess motion without adequate neuromuscular control is often the primary impairment. (3)

Caution is required where restraint is severely compromised. Suspected craniocervical instability is a major contraindication to high-velocity cervical manipulation/adjustment and requires referral. (25) Degenerative lumbar instability is also relevant. Degeneration can include an instability phase during which excessive motion contributes to pain recurrence and disability. (7) In such contexts, repeated mobilisation at unstable levels may increase irritability rather than improve function.

Autonomic dysfunction also complicates care. Orthostatic intolerance and syncope warrant careful screening and conservative approaches. (13) Recurrent dislocation syndromes indicate that passive restraint has already failed, and further mobilisation is unlikely to address the core impairment. (5)

This does not remove the Chiropractor's role, it refines it. Manual therapy may still be useful when applied selectively to coexisting hypomobile regions or for symptom modulation when applying a 'kinetic chain' approach to care (covered in part 2). The long-term focus should shift toward stabilisation and active rehabilitation.

Figure 2: Situations where manipulation should not be used in hypermobility.



Evidence-Based Rehabilitation and Management

The most effective interventions in symptomatic hypermobility improve control, strength, and functional tolerance. Hypermobility is not primarily a mobility problem. It is a stability problem.

Strength and resistance training

Progressive strengthening improves active stability and reduces symptoms. Trials in children with hypermobility have shown benefits from strengthening and proprioceptive programmes, including reduced knee pain and improved function. (31)

Proprioceptive and sensorimotor rehabilitation

Proprioceptive deficits are common and clinically meaningful. (20) Enhancement of proprioception has been shown to improve symptoms in hypermobility syndromes. (4) Balance training, perturbation work, and closed-chain control are central.

Motor control and spinal stabilisation

Deep stabiliser impairment is associated with chronic low back pain. (26) Exercise therapy has strong evidence in non-specific low back pain and aligns with stabilisation-oriented care for

instability-driven presentations. (27) These principles map onto spinal stability models emphasising passive, active, and neural subsystems. (22)

Multidisciplinary care and pacing

Multidisciplinary approaches combining physical and cognitive-behavioural strategies have improved disability outcomes in hypermobility-related conditions. (32) Psychological therapies have evidence in chronic and recurrent pain, supporting pacing and function-oriented recovery. (33) Pain management frameworks in EDS emphasise coordinated strategies over passive-only care. (21)

What is not supported

Passive-only care does not address the underlying impairment. Stretching programmes aimed at increasing flexibility may be inappropriate in already hypermobile patients. Repeated thrust manipulation/adjustment into lax segments may provide short-term analgesia but does not restore restraint and may worsen irritability over time. (3)

Chiropractic care is most effective when it includes education, graded strengthening, proprioception, pacing, and load management.

Conclusion and Practice Recommendations

Joint hypermobility is common, but its clinical meaning varies widely. For some individuals it is benign. For others, it drives persistent pain, instability, recurrent injury, and disability. Recognising this difference is essential in Chiropractic practice.

Hypermobility is best conceptualised as a spectrum from unstable hypermobility to stable mobility to hypomobility. Extremity laxity increases risk of recurrent dislocation and secondary overload syndromes such as tendinopathy, including rotator cuff related pain and patellar instability. Spinal laxity may occur in heritable connective tissue disorders and during degenerative instability trajectories.

Validated screening tools and careful history and examination improve detection. Autonomic dysfunction and suspected cervical instability require conservative decision-making and appropriate referral.

Manipulation may provide short-term analgesia via neurophysiological mechanisms. (11) However, manipulation does not restore ligament stiffness or passive restraint. (3) In unstable joints, repeated end-range mobilisation may worsen irritability and functional outcomes over time. (3) Evidence supports stabilisation-oriented rehabilitation, including progressive strengthening, proprioceptive retraining, pacing, and patient education.

Hypermobility is not primarily a mobility problem. It is a stability problem. Chiropractors who recognise this early can improve safety and outcomes by prioritising control, confidence, and long-term function.

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