Connective Tissue Disorders & Hypermobility

A Regenerative Approach to Lasting Stabilization

Dr. Mulvaney frequently sees patients with connective tissue disorders for whom standard treatment approaches, including surgery, don't always serve them as well as they could, or who are living with pain that significantly constrains their quality of life and ability to exercise. His practice has had considerable success treating these patients by addressing the core problem: underlying ligamentous laxity, treated with comprehensive prolotherapy to the affected body part.

What Are Connective Tissue Disorders?
In a connective tissue disorder (CTD) — including Ehlers-Danlos Syndrome (EDS) and generalized hypermobility spectrum disorders — the collagen a person genetically produces is a looser, more relaxed variety than the standard type. Collagen is the structural rebar of the body. It is found in skin, hair, bone, muscle, tendons, ligaments, and fascia. Having a looser collagen type may offer a competitive advantage in certain sports, but it also means joints, ligaments, and the surrounding soft tissue architecture are prone to premature wear and instability.

When someone with a CTD sustains an injury — whether to the spine, a shoulder, a hip, or any other joint — that injury must be evaluated through the lens of their connective tissue disorder. Standard treatment assumptions do not apply. The injury exists within a body-wide context of ligamentous laxity, and treatment must reflect that.

The most commonly affected areas reflect the joints that bear the greatest load or demand the most dynamic range of motion — but any joint in the body can be affected:

- Cervical and lumbar spine — premature degenerative disc disease and facet arthropathy
- Thoracic spine — rib instability, costovertebral joint laxity
- Shoulders — multi-directional instability, rotator cuff involvement
- Hips — labral stress, femoroacetabular laxity

Why Surgery Often Fails in CTD Patients
The current evidence strongly cautions against surgical intervention as a first-line approach for CTD-related joint pain. Several key reasons:

- Poor tissue healing — abnormal collagen structure leads to impaired surgical repair and prolonged recovery
- Adjacent segment failure — stabilizing one joint without addressing systemic laxity often loads neighboring joints, creating new instability
- Anesthesia and medication sensitivities — many CTD patients have heightened autonomic responses and altered drug metabolism
- High recurrence rates — without addressing underlying ligamentous laxity, surgical repair is frequently followed by re-injury or instability at the same or adjacent sites

Our Approach: Treating the Entire Functional Unit
We do not treat pieces and parts — we treat the entire functional unit.

We use 15% dextrose prolotherapy — an ultrasound-guided, injection-based regenerative treatment that works by focusing the body's own repair mechanisms to precisely correct ligamentous laxity. A carefully placed solution is delivered at the ligamentous insertion points, initiating a controlled healing cascade that promotes collagen remodeling and structural tightening.

When treating the cervical spine, for example, we treat from the base of the skull to the top of the thoracic spine — every level, both sides — to restore stability to that entire functional unit. When treating the lumbar spine, we treat the entire lumbosacral complex — the sacroiliac ligaments, iliolumbar ligaments, and all relevant ligamentous attachments — not just the site of reported pain. When treating a shoulder, we address the full array of ligaments and tendon insertions surrounding the joint, not the glenohumeral joint alone.

This global approach is essential in CTD patients because systemic laxity means no joint is truly isolated. Addressing only one focal area leaves the surrounding soft tissue architecture unsupported and vulnerable.

The Role of Fascia

Emerging research has highlighted fascia — the continuous web of connective tissue that surrounds and connects muscles, ligaments, tendons, and organs — as a central player in CTD pathophysiology. In hEDS and hypermobility spectrum disorders, fascia undergoes pathological remodeling: it thickens in some areas, loses its ability to glide smoothly between layers, and becomes a source of pain through irritation of the dense network of nerve fibers it contains.

Our primary target with prolotherapy is the ligamentous insertion — the anchor point where ligament meets bone — because this is where structural instability originates. Fascia responds secondarily as the ligaments tighten and the mechanical environment normalizes. By restoring ligamentous integrity first, we allow the entire soft tissue system, fascia included, to re-establish proper tension, glide, and load distribution.

Standard CTD Treatment Protocol

- 3 treatment sessions, spaced approximately one month apart
- Each session targets the full ligamentous complex of the affected region(s)
- Ultrasound guidance used to ensure precise needle placement
- No driver needed — you will walk in and walk out of each session, with only a few days of decreased activity afterward
- Reassessment at conclusion; additional cycles offered for complex or multi-region cases

- Complementary physical therapy and neuromuscular re-education encouraged between sessions

What Patients Can Expect

Most patients experience a temporary increase in soreness at the treatment sites for 24–72 hours following each session — this is a normal part of the healing process. Gradual improvement in stability and reduction in pain is typically observed between sessions, with cumulative benefit continuing for weeks to months after the final treatment. Following your last session, we will schedule a follow-up appointment at 8 weeks to give your body adequate time to complete its healing response and allow us to fully assess your progress.

Selected References

Wang TJ, Stecco A, Hakim AJ, Schleip R. Fascial Pathophysiology in Hypermobility Spectrum Disorders and Hypermobile Ehlers–Danlos Syndrome: A Review of Emerging Evidence. Int J Mol Sci. 2025;26:5587.

Zoppi N, et al. Dermal Fibroblast-to-Myofibroblast Transition Sustained by Avβ3 Integrin-ILK-Snail1/Slug Signaling Is a Common Feature for Hypermobile EDS and HSD. Biochim Biophys Acta Mol Basis Dis. 2018;1864:1010–1023.

Wang TJ, Stecco A. Fascial Thickness and Stiffness in Hypermobile Ehlers-Danlos Syndrome. Am J Med Genet C Semin Med Genet. 2021;187:446–452.

Malfait F, et al. The 2017 International Classification of the Ehlers-Danlos Syndromes. Am J Med Genet C Semin Med Genet. 2017;175:8–26.

Kumar B, Lenert P. Joint Hypermobility Syndrome: Recognizing a Commonly Overlooked Cause of Chronic Pain. Am J Med. 2017;130:640–647.

Questions about whether prolotherapy is right for you? Ask Dr. Mulvaney at your next appointment.