Hypermobility and the Ehlers-Danlos Syndromes (EDS)

Quick Facts — New Zealand Focus — Health Professional Version

Includes Generalised Joint Hypermobility (GJH), Hypermobility Spectrum Disorders (HSD), and the associated multi-system conditions

Version 3.1 — August 2026 (interim update: Road to 2026 preliminary findings folded in)

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⚠ Read this first — this document has a use-by date

The international diagnostic criteria for **all** types of EDS and HSD are being replaced. The new framework publishes on **2 December 2026** in the *American Journal of Medical Genetics* (the Ehlers-Danlos Society's own page gives this date; some earlier announcements said 1 December), and it supersedes everything published before it, including the 2017 criteria taught in this document. Best-practice care and management pathways follow in **early 2027**.

This version is therefore deliberately an *interim* document. It teaches the criteria you must use **today**, flags what is likely to change, and will be rewritten once the new framework is out.

🔬 New in this version (v3.1, August 2026): the Road to 2026 preliminary findings

The Road to 2026 research has now been presented in preliminary form, and the July 2026 Global Learning Conference in Dallas has been and gone. **None of it is final** — the binding criteria still publish in December — but the direction of travel is much clearer than it was a few weeks ago. The indicative findings are pulled together in **Section 15**, flagged throughout as *indicative, not final*. The single most important correction to the previous version concerns the kallikrein genetics: the large HEDGE study did **not** confirm KLK15 as a cause of hEDS (see **Section 7**).

Contents

1. Could this be EDS? — when to think of it

If you have a patient with hypermobile joints (see section 3) **and** one or more of the following:

- Unusual or unexplained musculoskeletal pain that doesn't "make sense"
- Recurrent subluxations or dislocations
- Unusual skin — very soft and young-looking, fragile, or hyper-extensible
- An unusual set of symptoms from many body systems that simply don't make sense to be there in one person
- A family history of the same "vague" pattern, usually down the female line
- **A patient where you are beginning to think it is all in their head because there is no such illness**

…then think, *"Could this be EDS?"*

That last bullet has always been the most important one on the page. Section 10 now gives you the numbers behind it.

2. JUST GAPE

Because it's been right in front of you all the time. Adapted from the RCGP/EDS UK Toolkit (Reinhold & Jamieson, 2018). It takes about five minutes.

	Domain	Ask	Examples
JUST	**J**oints and other **S**oft **T**issues	Does the patient (or their family) have a lot of trouble with joints, tendons, ligaments and muscles?	Hypermobility, dislocations, "fibromyalgia", chronic pain, TMJ dysfunction, clicky hips at birth, rotator cuff tendinopathy, tennis/golfer's elbow, RSI, greater trochanteric pain syndrome, ITB syndrome, patellar tendon problems (incl. Osgood-Schlatter), Achilles problems, plantar fasciitis, acquired pes planus
G	**G**ut	Do they have a functional gut disorder or malabsorption?	IBS, intermittent dysphagia or globus, reflux or oesophageal spasm, dyspepsia, nausea, vomiting, recurrent abdominal pain, bloating, constipation, diarrhoea, urgency, fast or slow transit, steatorrhoea, unexplained weight loss, unexplained nutrient deficiencies
A	**A**llergy / **A**topy / **A**utoimmune	Asthma, eczema, hayfever, rhinitis, multiple food/drug/ other allergies or intolerances, itching or urticaria, or more than one autoimmune condition?	Anaphylaxis, chronic urticaria, dermatographia, flushing, thyroid disease, coeliac disease, Crohn's, ulcerative colitis, MS

	Domain	Ask	Examples
P	**P**ostural symptoms	Symptoms on standing that are relieved by lying down?	Light-headedness, palpitations, fatigue, shaking, sweating, breathlessness, fainting, headaches
E	**E**xhaustion	Exhausted much of the time, or can't think as clearly as normal?	Fatigue, "tired all the time", CFS/ME, brain fog, memory/ word-finding/concentration problems that vary day to day, falling asleep after meals

If several domains are positive, it may be EDS or HSD, but it needs proper investigation. Book a second, longer appointment once you've had time to read up and have the diagnostic checklist to hand. This is not a corridor diagnosis.

3. Assessing hypermobility

Beighton Score — the screening threshold

A good start - but it has problems ….

Age group	Positive at
Child to puberty	≥ 6
Age 20–50 years	≥ 5
Age > 50 years	≥ 4

Nine points in total: one point each side for (1) passive dorsiflexion of the 5th MCP beyond 90°, (2) passive apposition of the thumb to the forearm, (3) elbow hyperextension beyond 10°, (4) knee hyperextension beyond 10°; and one point for (5) forward flexion of the trunk with knees straight, palms flat on the floor.

There is a good demonstration video from the Ehlers-Danlos Society — link in section 14. Doing it properly matters; done casually it is unreliable.

Watch this space. The Beighton score is one of the elements explicitly under review for the December 2026 framework. It is a nine-point screen designed in 1973 for epidemiological fieldwork, and it is a poor instrument for the older patient, the stiffened patient, the previously-operated patient, and the patient whose hypermobility is localised rather than generalised. Expect change.

The Five-Point Questionnaire

Use where the Beighton isn't possible, or where superimposed pain and stiffness in an adult will give a falsely low Beighton score.

1. Can you now (or could you ever) place your hands flat on the floor without bending your knees?
2. Can you now (or could you ever) bend your thumb to touch your forearm?
3. As a child, did you amuse your friends by contorting your body into strange shapes, or could you do the splits?
4. As a child or teenager, did your shoulder or kneecap dislocate on more than one occasion?
5. Do you consider yourself "double-jointed"?

"Yes" to ≥ 2 of 5 suggests joint hypermobility, with roughly 80–85% predictive value.

4. The 2017 hEDS criteria — and what is likely to change

Use the official checklist. Download it from the Ehlers-Danlos Society: https://www.ehlers-danlos.com/heds-diagnostic-checklist/

The 2017 criteria require **all three** of:

- **Criterion 1** — Generalised joint hypermobility (Beighton, age-adjusted as above, with the 5PQ as an adjunct)
- **Criterion 2** — Two or more of: (A) systemic features of a connective tissue disorder — a list of twelve, of which five are needed; (B) positive family history in a first-degree relative; (C) musculoskeletal complications (pain in two or more limbs, chronic widespread pain, recurrent joint dislocations or frank instability)
- **Criterion 3** — Exclusion of alternative diagnoses: absence of unusual skin fragility, exclusion of other heritable and acquired connective tissue disorders, exclusion of alternative diagnoses that can produce hypermobility (neuromuscular disease, myopathy, skeletal dysplasia)

Criterion 3 is the one most often skipped, and it is the one that matters most. A 2025 University of Miami registry study of 907 patients referred for hEDS assessment found that among the 178 who met the 2017 criteria, genetic testing identified an alternative or additional diagnosis in 26.4% — a quarter of them — with clinical implications requiring different management. Marfan, Loeys-Dietz, classical and vascular EDS, and osteogenesis imperfecta all matter enormously more than an hEDS label, and all are treatable-by-surveillance in ways hEDS is not.

Investigations

- There are **no** specific or suggestive laboratory or imaging findings that make or exclude hEDS.
- There **are** known genetic mutations for classical (cEDS), vascular (vEDS), and most of the rare types.
- For hEDS there is no clinically usable genetic test — but see section 7. The 2021 line "genetic basis currently not known" now needs softening rather than deleting.
- Clinical diagnosis first, using clinical criteria; genetic confirmation wherever possible and practical.
- **Genetic testing is important in vEDS and should not be delayed** where there is any suspicion.

📦 What is likely to change in December 2026

Nobody outside the committee knows for certain. Based on the published direction of travel of the Road to 2026 project — a Delphi consensus process plus a full literature review since 2017, with interim findings presented at the 2025 International Scientific Symposium in Toronto — the following are widely anticipated:

- **HSD and hEDS look likely to be recombined.** The 2017 split created a two-tier system in which "HSD" was frequently, and wrongly, heard by patients and clinicians alike as "not real enough to be EDS". Final naming and categorisation are still to be confirmed.
- **The Beighton score will be reviewed and probably revised.**
- **Multi-system features may be formally integrated into the criteria** rather than sitting as an informal appendix — which is closer to what the condition actually is.
- **Genes identified between 2017 and 2026 will be factored in**, potentially creating new named types and possibly subdividing hEDS by symptom cluster or genotype.
- **The pathway between diagnosis and management will be spelled out**, in the companion publication due March 2027.

Until 2 December 2026, the 2017 criteria remain the ones to use. Do not pre-empt them with your patients — but it is fair and kind to tell someone with an HSD label that the ground is about to move.

5. The hypermobility spectrum

Some degree of joint hypermobility is common, especially in children, and it is usually benign — no apparently detrimental effects. The clinical trick is to distinguish the benign from the borderline-symptomatic from the overtly syndromic.

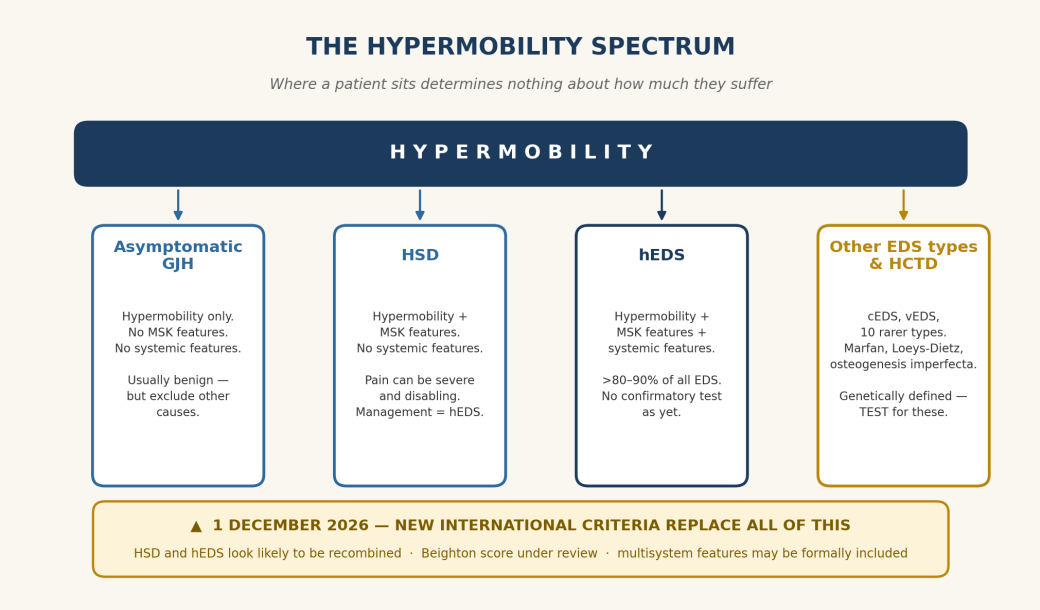

Figure 1. The hypermobility spectrum as currently classified, and the change coming on 1 December 2026.

Phenotype (clinical)	Beighton	Musculoskeletal features	Systemic features
Asymptomatic GJH	Present	Absent	Absent
HSD	Present	Present	Absent
hEDS	Present	Present	Present

Generalised joint hypermobility (GJH) itself runs a spectrum:

1. Asymptomatic (non-syndromic) GJH — hypermobility without other symptoms. Other causes must be excluded.

2. Symptomatic GJH that doesn't meet criteria for hEDS — HSD.

3. A well-defined syndrome — hEDS.

HSD is not a lesser condition. Pain and disability in HSD can equal or exceed that in hEDS. Management is essentially identical. The distinction is taxonomic, not prognostic — and, as noted above, it may not survive December.

Micro- and macro-trauma

Hypermobility leads to repeated micro- and macro-trauma, which over years is a leading cause of pain and, in adulthood, degenerative change. The range runs from hyperextension injury, through mild subluxation, to frank dislocation. Each event causes surrounding collateral damage.

Micro-trauma is often invisible. The patient frequently knows the joint is "out" when nothing is seen clinically or on X-ray. **This is not the patient being unreliable; it is proprioception outperforming radiography.** Macro-trauma is the visible subluxation or dislocation.

6. Classification of the EDS types

EDS are a group of inherited disorders characterised by defects in collagen and connective tissue, mainly affecting ligaments and soft tissues.

- Commonly quoted prevalence is 1:2,500, but this is almost certainly an underestimate; 1:500 to 1:1,000 for the whole hypermobility spectrum is more plausible. **F >>> M** in diagnosed cohorts — around 70–95% female depending on the series. Whether that reflects true prevalence or ascertainment bias is genuinely unsettled and is discussed in section 10.
- The basis is abnormal connective tissue — different collagens and collagen-modifying enzymes in each type.
- **hEDS is by far the commonest (>80–90% of all EDS).**
- cEDS is the next most common.
- vEDS is uncommon but must be recognised because it can be lethal if missed.

Subtype	Abbrev.	Inheritance	Genetics	Protein
Classical	cEDS	AD	*COL5A1*, *COL5A2*	Type V collagen
Vascular	vEDS	AD	*COL3A1*	Type III collagen
Hypermobile	hEDS	AD (apparent)	Not established — see §7	Not established
Ten other rare types	—	Mostly AR	Various	Various

cEDS — classical

- **Major criteria:** skin hyperextensibility with atrophic scarring (especially knees and elbows); generalised joint hypermobility.
- **Minor criteria:** easy bruising; soft doughy skin; skin fragility; molluscoid pseudotumours; subcutaneous spheroids; hernia; epicanthal folds; complications of joint hypermobility; family history in a first-degree relative.

vEDS — vascular

Rare, and the one that kills.

- **Major criteria:** family history of proven vEDS with a *COL3A1* variant; arterial rupture at a young age; spontaneous sigmoid colon perforation without other bowel disease; uterine rupture in the third trimester without prior caesarean or severe perineal tear; carotid-cavernous sinus fistula without trauma.
- **Minor criteria:** bruising unrelated to trauma or in unusual sites; thin translucent skin with easily visible veins; characteristic facial appearance; spontaneous pneumothorax; acrogeria; talipes equinovarus; congenital hip dislocation; hypermobility of small joints; tendon and muscle rupture; keratoconus; gingival recession and fragility; early-onset varicose veins.

If you see arterial rupture, spontaneous bowel perforation, or spontaneous pneumothorax in a young adult or child — consider vEDS as the underlying condition.

Genetic testing is essential. Family cascade screening changes and saves lives. Media coverage and public discussion focus almost entirely on hEDS; vEDS is the one where the diagnosis is time-critical.

7. What's new since 2021 — genetics and mechanism

The 2021 version of this document said flatly that the genetic basis of hEDS is unknown. That is no longer quite right, and the nuance matters.

The kallikrein finding — and why it has been walked back

The 2021 version of this document said flatly that the genetic basis of hEDS is unknown. The story since is a useful lesson in how genetics actually progresses.

In 2025 the Norris Lab at the Medical University of South Carolina, led by Dr Cortney Gensemer (herself an hEDS patient), reported a missense variant in **KLK15** (p.Gly226Asp) segregating with hEDS across a four-generation family and a second mother-daughter family, with roughly a third of a ~200-patient sporadic cohort carrying a rare variant somewhere in the 15-gene kallikrein family. Knock-in mice carrying the familial variant developed connective tissue defects consistent with the human phenotype. It was, reasonably, reported as the first real genetic lead in hEDS.

Then the larger study did not confirm it. The HEDGE study (see Section 15), which whole-genome-sequenced about a thousand hEDS patients, found KLK15 variants were **no more common than in controls.** The honest reading: KLK15 may explain hEDS in a small number of specific families, and the mouse work is real, but it is **not the general cause of hEDS.** A promising single-laboratory finding met a large replication cohort and was substantially narrowed. That is science working as it should — and it is exactly why emerging genetic findings must not drive clinical decisions prematurely.

What this means clinically: still nothing to order. There is no validated kallikrein test, no threshold, no clinical action, and now good reason to think a kallikrein result would not answer the question anyway. Do not order it, do not interpret it, and do not let a patient believe any such result confirms or excludes hEDS.

What HEDGE did establish

Even without a single gene, ***HEDGE delivered something valuable: in over 99% of its participants, the clinical hEDS diagnosis made under the 2017 criteria held up, and fewer than 1% carried a variant pointing to a different connective tissue disorder.*** The condition appears to arise from multiple rare variants and complex interactions rather than one mutation — which is why the field increasingly suspects hEDS is several related conditions rather than one. The fuller picture is in Section 15.

Immune involvement

Several 2025–2026 papers report immune dysregulation in hEDS cohorts. This is early, the mechanisms are not settled, and it is being over-claimed in patient-facing media — and although KLK15 itself did not replicate, the kallikreins are serine proteases with roles in inflammation, so a plausible thread still connects the connective tissue phenotype, the mast cell story, and the dysautonomia story. Watch it; don't yet act on it.

The honest summary for a patient

"Ten years ago we said we had no idea what causes hEDS. We now have one of the world's largest genetic studies telling us two things: made properly, the diagnosis is valid — and there isn't one single gene, so it's probably several related conditions we're still untangling. One gene that looked promising last year, KLK15, didn't hold up in the big study. That's not a step backwards; it's how the science tightens."

8. The comorbidity constellation

This is where the 2021 document was thinnest, and it is where the field has moved most. The Ehlers-Danlos Society's 2026 Global Learning Conference, held in Dallas on 24–26 July 2026, was themed **"Connecting the Stripes"** — precisely this territory: chronic pain, fatigue, dysautonomia, MCAS, gastrointestinal health, neurodivergence, mental health, cardiovascular and urogenital conditions.

The clinical point is simple and often missed: **the joints are frequently not what disables the patient.** For many, it is the autonomic, gastrointestinal and fatigue burden.

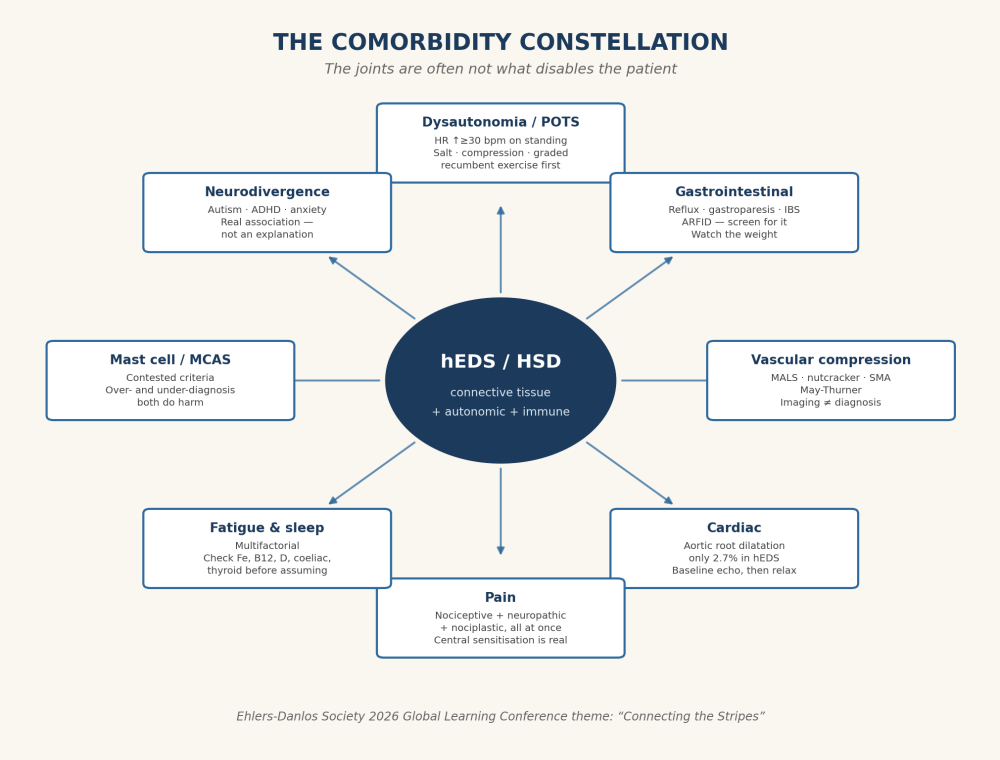

Figure 2. The comorbidity constellation. Each spoke is a common reason a patient with hEDS or HSD is disabled, and none of them is the joints.

Dysautonomia and POTS

Postural orthostatic tachycardia syndrome is common in hEDS. Diagnosis: sustained heart rate increase ≥ 30 bpm within 10 minutes of standing (≥ 40 bpm in adolescents), without orthostatic hypotension, with symptoms for ≥ 3 months, and in the absence of other explanation. A NASA lean test or active stand test in your own rooms is adequate for a working diagnosis; tilt-table is not required to start management.

Current best synthesis: **Lau DH et al., "Postural Orthostatic Tachycardia Syndrome: A State-of-the-Art Review",* Heart, Lung and Circulation *2026;35(2):171–185** — an Australasian review, freshly published, and the most useful single reference for a New Zealand practice. The 2021 NIH expert consensus (Vernino et al.) remains the other standard citation.

Practical management, in order:

1. **Fluid and salt** — 2–3 L/day fluid, 8–10 g/day sodium unless contraindicated

2. **Compression** — waist-high, 30–40 mmHg, abdominal compression matters more than legs

3. **Graded recumbent exercise** — rowing, recumbent cycling, swimming, progressing to upright. Now regarded as first-line non-pharmacological treatment. This is the hardest sell and the highest-yield intervention.

4. **Then medication** — consider ivabradine, low-dose beta-blockade, midodrine, fludrocortisone, pyridostigmine, according to phenotype

5. **Deconditioning is a consequence as well as a contributor.** Framing POTS as "just deconditioning" is both wrong and, in this population, corrosive to the therapeutic relationship.

Note also the substantial post-COVID dysautonomia cohort now presenting, which overlaps heavily and has changed service demand.

Mast cell activation syndrome (MCAS)

The most contested item in this list, and the one where I would urge the most care in both directions.

- There are two competing sets of criteria: a **strict consensus** requiring episodic multisystem symptoms, objective evidence of mast cell mediator release (classically a rise in serum tryptase of ≥ 20% above baseline plus 2 ng/mL during an event), and response to mast-cell-directed therapy; and a **broad consensus** that accepts a clinical picture plus treatment response without the tryptase rise.
- Under the strict criteria, confirmed MCAS is rare. Under the broad criteria, it is common in hEDS cohorts.
- **Both over- and under-diagnosis do harm.** Over-diagnosis leads to escalating restrictive diets, polypharmacy, and an identity built around allergy. Under-diagnosis leaves patients with genuinely disabling flushing, urticaria, GI symptoms and anaphylactoid episodes untreated.
- A reasonable pragmatic path: check tryptase (during and between episodes if you can), consider a trial of H1 + H2 blockade ± a mast cell stabiliser, and judge on response. Refer to immunology where available. Be honest that the field is unsettled.

Gastrointestinal

Very common and frequently the dominant symptom burden: reflux, dysphagia, early satiety, nausea, gastroparesis, bloating, constipation-predominant or mixed IBS, and pelvic floor dysfunction. Mechanisms plausibly include connective tissue laxity of the gut wall and mesentery, autonomic dysfunction of motility, and visceral hypersensitivity.

Two specific cautions:

- **Screen for disordered eating and ARFID (**avoidant/restrictive food intake disorder). Food avoidance driven by pain, nausea and fear is extremely common and is not the same thing as anorexia nervosa — but it produces the same weight loss, and the same risks (see section 12).
- **Unexplained progressive weight loss with post-prandial vomiting should prompt thought about SMA syndrome** — see section 9. Lots of other causes - but this is a biggie in New Zealand press at the moment.

Pain and fatigue

- Pain is frequently mixed nociceptive, neuropathic and nociplastic. Expect all three in one patient.
- Central sensitisation is real and is not a synonym for psychological causation.
- Fatigue in hEDS is multifactorial: sleep disturbance, autonomic load, pain, deconditioning, the metabolic cost of poor proprioception, and anaemia or deficiency where present. Check iron, B12, vitamin D, coeliac serology, thyroid.

Neurodivergence and mental health

There is a consistently reported association between hEDS/HSD and autism and ADHD, and with anxiety. The direction of causation is unresolved and probably multiple. Two things are true at once: (a) this association is real and should inform how you communicate, pace appointments and structure care; and (b) it has been weaponised against patients as evidence that their physical symptoms are psychosomatic. Hold both.

Cardiac — a note of proportion

Patients arrive frightened about their aorta because of what they have read. The reassuring data: a Mayo Clinic cohort of 568 adult EDS Clinic patients found **aortic root dilatation in 2.7% of hEDS and 0.6% of HSD.** Mitral valve prolapse is likewise less common than folklore suggests. A baseline echocardiogram is reasonable; annual imaging in the absence of findings usually is not. This is different from Marfan, Loeys-Dietz and vEDS, where surveillance is essential — which is another reason criterion 3 matters.

9. Abdominal vascular compression syndromes (AVCS)

A radiologist's section, and the most contested territory in New Zealand EDS care in 2026.

Four conditions account for most of what is discussed under this heading. Three sit within a few centimetres of each other just below the diaphragm; the fourth is in the pelvis.

Syndrome	Pinched structure	Pincher	Typical symptoms	The trick
MALS (Dunbar)	Coeliac trunk	Median arcuate ligament	Post-prandial pain, weight loss, nausea	**Dynamic** — worse in expiration, may vanish in inspiration
Nutcracker	Left renal vein	Aorta behind, SMA in front	Left flank/loin pain, haematuria, proteinuria, varicocele, pelvic congestion	**Postural and habitus-dependent** — slim, tall, recent weight loss
SMA syndrome (Wilkie)	Third part of duodenum	The same aortomesenteric angle	Post-prandial pain, vomiting, weight loss — sometimes life-threatening	**A vicious circle** — weight loss thins the fat pad, which worsens compression, which worsens weight loss
May-Thurner (Cockett)	Left common iliac vein	Right common iliac artery against L5	Left leg swelling, varicose veins, DVT, pelvic pain	**Asymmetric by design** — left-leg DVT in a young woman with no risk factors should raise the question

What the radiologist looks for

- **MALS** — CT/MR sagittal: hook- or J-shaped indentation on the upper surface of the coeliac trunk, with post-stenotic dilatation. Doppler: coeliac peak systolic velocity typically > 200 cm/s in expiration, falling on inspiration. *The change is the diagnosis as much as the absolute number.*
- **Nutcracker** — Doppler: peak velocity ratio between narrowed and dilated left renal vein segments, typically > 4–5:1. CT: aortomesenteric angle typically < 35–40°.
- **SMA syndrome** — CT sagittal: aortomesenteric angle < 22° (normal 38–65°), aortomesenteric distance < 8 mm (normal 10–28 mm).
- **May-Thurner** — venous-phase CT: flattening of the left common iliac vein behind the right iliac artery, with collaterals suggesting haemodynamic significance.

Why two radiologists can look at the same patient and disagree

This is the crux, and it is not usually a story about competence or politics.

1. **Modality.** Static CT/MR is geometric. Dynamic colour Doppler measures flow velocity and vessel deformation in real time. Different physics, different questions.

2. **Position and respiration.** Most published criteria assume supine, neutral, inspiratory imaging. These compressions can be dynamic — present in expiration or upright, absent supine. Routine abdominal CT is performed in held inspiration, exactly the phase in which MALS is least visible.

3. **Threshold.** There is no universally agreed cut-off for "pathological". Velocity ratios for nutcracker range from 4:1 to 5:1 across published series. Mild ligamentous indentation of the coeliac is found in roughly **10–25% of asymptomatic people.**

4. **Symptom–imaging mismatch.** Compression meeting criteria can be entirely asymptomatic; classic symptoms can have unimpressive imaging. Collateral drainage differs between individuals. The radiologist sees pixels; the patient is the patient.

5. **Operator dependence.** Dynamic Doppler is exquisitely operator-dependent in a way CT is not. Two skilled sonographers can produce genuinely different studies of the same vessel.

Two radiologists looking at "the same patient" are usually not looking at the same patient. They are looking at the same patient through different instruments, in different positions, on different days, sometimes with different clinical questions in mind. Of course they sometimes disagree.

The hip X-ray paradox

Every doctor in the country already understands this, in a different organ. Take ten patients with bone-on-bone radiographic hip osteoarthritis: some are in agony, some walk in barely symptomatic. Take ten with crippling hip pain: some have dramatic films, some nearly normal ones. Nobody is wrong. The imaging isn't faulty, the radiologist isn't missing something, the patient isn't exaggerating. Partly because the pain could be from the surrounding soft tissues - ligaments and tendons.

Imaging measures anatomy. Clinical medicine measures suffering. The two don't always line up, and the gap is not anyone's fault.

Where the genuine controversy sits

The syndromes themselves are **not** in dispute. Mayo, Cleveland Clinic, Stanford and others run dedicated programmes; surgical decompression for MALS, stenting for May-Thurner, and left renal vein transposition for nutcracker are all performed in selected cases with reasonable outcomes; the peer-reviewed literature on each entity is substantial. A 2023 *Journal of Vascular Surgery* review of more than 250 patients with multiple concurrent compression syndromes reported a strong correlation with EDS and a high rate of co-existing MCAS — and explicitly called for consensus diagnostic criteria, because there aren't any.

What is genuinely contested is narrower than the public conversation suggests:

1. **How often does morphological compression cause clinical symptoms?** Less often than the most enthusiastic operators claim, more often than the most sceptical concede. The honest answer is that we don't know precisely, and it depends on the patient.

2. **What selection criteria predict response to intervention?** Genuinely unsettled. Randomised trials are scarce to non-existent.

3. **Are dynamic ultrasound techniques reliable enough to drive intervention decisions?** Reasonable people disagree. The technique has produced real diagnoses and real over-diagnoses.

4. **Is hEDS associated with increased prevalence of these compressions?** Plausible mechanism, mixed evidence, no consensus. The current New Zealand vascular position is that the literature does not yet support a confirmed link.

None of these legitimate disagreements licenses dismissal of the patient in front of you. A patient with a normal CT and severe post-prandial pain may have MALS the scan didn't capture, or a functional gut disorder, or both, or something else entirely. The clinical task is not to declare the imaging the final word.

"I don't see it on my scan" is not the same as "it isn't there - it's in your head."

Practical advice for the referrer

If you genuinely suspect MALS or one of the others - **say so on the request form.** A routine portal-venous CT abdomen in held inspiration will not answer the question, and reporting it as normal is technically correct and clinically useless. Only a few radiologists will take you seriously - try to find one near you.

10. Being believed — the evidence base for a hard conversation

The 2021 version of this document said "assure the patient you don't think it's all in their head." That instruction was right, and it was based on clinical impression. It is now based on data.

The numbers

Lee & Chopra, Children 2025;12(6):698. A retrospective chart review of 429 patients clinically diagnosed with hEDS at a single US centre (94.8% female, mean age 27.9 years). Patients were asked whether they had previously been told, by physicians who were not board-certified psychiatrists*, that their symptoms were "in their head", that they were "making it up" or attention-seeking, that they might have Munchausen's or a factitious disorder, or had been diagnosed with conversion disorder.

405 of 429 — 94.4% — answered yes to at least one. Only 24 patients reached their hEDS diagnosis without passing through some form of psychiatric mislabelling. Where psychiatric misdiagnosis occurred, diagnostic delay was substantially longer.

The honest caveat, because it matters: this is a single tertiary centre for complex conditions, retrospective, based on patient recall, with obvious selection bias — patients who reached a specialist EDS clinic are precisely the ones whose journey was hardest. The true population figure is certainly lower. **It is still an appalling number**, and no plausible correction for bias makes it a comfortable one. An Australian survey of 152 women with EDS found more than half first noticed symptoms over 15 years before diagnosis, and more than three-quarters received at least one other diagnosis first.

Medicine's track record

We have been here repeatedly, and almost always with women:

- **Peptic ulcer disease.** "Stress and acid" was dogma until Marshall and Warren proved *H. pylori* in 1982. They were ridiculed for years before the Nobel.
- **Endometriosis.** Dismissed as hysteria for the better part of a century. Diagnostic delay is *still* 7–10 years.
- **Multiple sclerosis.** Long misdiagnosed as conversion disorder in young women, until MRI made the lesions visible. The lesions were always there. The instrument wasn't.
- **ACNES (anterior cutaneous nerve entrapment syndrome).** Chronic abdominal pain, predominantly women, repeatedly dismissed as functional or IBS, normal imaging — and it resolves with a small volume of local anaesthetic at the trigger point. Boelens et al. demonstrated this against placebo in a randomised trial (*Ann Surg* 2013). **The block is the proof.** Anaesthetics have a defined duration of action; that is not placebo.

The pattern is consistent: a condition affecting mostly women, producing pain out of proportion to visible findings, attributed to psychology, until an instrument arrives that can see it. MS is the cleanest example because the answer was unambiguous once MRI existed. **The lesion was always there. We just couldn't see it.**

What to actually say

You do not have to endorse every theory a patient brings you in order to take them seriously. The two are not the same thing, and conflating them is what has broken trust in this area.

"I believe your pain is real. I don't yet know what's causing all of it. Some of what you've read I'm not sure about, and I'll tell you honestly which bits. But 'I can't see it' is not the same as 'it isn't there', and I'm not going to treat you as though it is."

That sentence costs nothing and is often the first time the patient has heard it.

Also true, and worth saying plainly

Diagnostic humility runs both ways. Patients in this group are also vulnerable to over-diagnosis, unnecessary and irreversible surgery, escalating polypharmacy, restrictive diets, and expensive private pathways that lead nowhere. Taking someone seriously includes protecting them from harm done in the name of taking them seriously. That is a harder conversation than either dismissal or agreement, and it is the right one.

11. Management

Deal with acute emergencies

- **Vascular rupture** — urgent vascular surgery or interventional radiology
- **Spontaneous pneumothorax** — acute management, chest drain if appropriate
- **Dislocation** — appropriate orthopaedic referral
- **Acute pain** — usual principles

Diagnosis and first conversation

- Give a tentative clinical diagnosis rather than leaving the patient in limbo. Assure them you don't think it's all in their head.
- Offer written information — the Ehlers-Danlos Society site and EDS Aotearoa NZ (section 14).
- Refer to a clinician with experience or interest in EDS, where one is available.
- Care is best led by a multidisciplinary team. In practice this usually means the GP coordinating and referring as needed.
- **Give the patient a copy of the wallet card** for emergency presentations.

Pain

- Usual principles for acute and chronic pain. Start with paracetamol and work up.
- **No specific analgesic has proven advantage in HSD/EDS.**
- Opioids: the honest position is that long-term opioids perform poorly in this population, that abrupt or coercive withdrawal is harmful and has been a live issue in New Zealand this year, and that neither blanket prescribing nor blanket refusal is defensible. Decide with the patient, document the reasoning, and review.
- Low-dose naltrexone, duloxetine, and tricyclics are used by some clinicians with anecdotal benefit and thin evidence. Say so when you offer them.
- Chronic pain services help design an overall strategy where accessible.

Injuries — instability, subluxations, dislocations, tendon and ligament injury

- **Each injury episode is a new trauma on its own merits**, not "part of your condition". Aim to return to the pre-injury state. A dislocation may take less force in EDS, but the associated tissue damage is no less severe — a Bankart lesion is a Bankart lesion.
- **Reduction technique may need modifying.** Severe pain or instability elsewhere in the same limb means standard traction and rotation can injure a second joint — pulling on the wrist or forearm to reduce a shoulder may damage the wrist or elbow. Modify hand position and grip. The force needed is often less. Some patients reduce spontaneously.
- **Protect the skin** — it may be fragile. Use padding.
- Ligament and tendon injuries may heal slowly and recur after progressively less trauma.
- **Subluxations** often respond to gentle mobilisation rather than formal reduction. Some patients do well with osteopathy or skilled musculoskeletal manual therapy — provided the practitioner knows the diagnosis.
- **Splinting and bracing after reduction are important.**
- Early post-trauma physiotherapy is useful, with an EDS-adapted approach.

Long-term joint stabilisation

- **Physiotherapy is the backbone.** See below.
- **External bracing.** Braces and splints help before and after dislocation and for comfort. Hypermobility-specific splints — ring splints for finger hyperextension are the classic example — improve function and reduce pain even without recent subluxation. Neoprene and elastic supports and compression garments provide proprioceptive feedback as well as support. Discuss with physio or OT.
- **Surgery may have sub-optimal outcomes** but can be essential in critical areas — craniocervical instability being the obvious one. When referring, **state explicitly that the patient has EDS**: slow healing, early recurrence, and poor response to local anaesthetic all change the plan.
- **Sclerosant prolotherapy** and other non-surgical stabilisation approaches offer some hope in selected adults. Injected material is thought to induce healing with scarring and shortening, increasing joint stability. The evidence base remains modest; describe it accurately.

Physiotherapy and physical therapies

- Traditional physiotherapy technique needs adapting for EDS, and always needs a wider view of the patient.
- The aim is **empowerment towards self-management**, not dependency.
- Most patients benefit from an individualised, carefully graduated exercise and activity programme. Graduated is the operative word: too fast produces a flare and a lost patient.
- The **Muldowney protocol** is worth considering, at least in part.
- Occupational therapy for independence and social rehabilitation.
- Osteopathy, myofascial release and similar are worth considering.
- **Make sure the practitioner knows the diagnosis** so that techniques can be modified to avoid injury.

Surgery and anaesthesia

- Complications may be increased through slow healing and bleeding tendency. Plan and discuss in an EDS context.
- Recurrence after surgery is common because the ligaments remain abnormal.
- Anaesthetic considerations:
  - Unstable neck — positioning risk
  - **Slow and suboptimal response to local anaesthetic, including epidurals — this is real, well documented, and frequently disbelieved. You will need a higher dose and a longer wait before starting the procedure.**
  - Tourniquets can cause bruising and compartment syndrome
  - Positioning can cause unexpected subluxations, including of the TMJ, during anaesthesia
- Reference: Wiesmann et al., *Orphanet Journal of Rare Diseases* 2014;9:109.

General support

- **Remind patients their disease and their pain are real.** They are likely accustomed to being told there is nothing wrong.
- Support groups and websites offer practical advice for living with painful chronic disease.
- Psychological support helps — offered as adjunct, never as explanation. The distinction is everything, and patients hear it instantly.

Next steps and specialist referral

- Many patients are well managed by GP and physiotherapist with occasional input from a clinician with EDS interest — physician, rheumatologist, geneticist or other.
- Complex or severe cases need a genuine MDT: orthopaedics, neurosurgery, gastroenterology, cardiology, immunology, pain medicine.
- Specific interventions warrant specific referrals — hand therapy, sclerosant prolotherapy, pelvic floor physiotherapy.
- Some rheumatologists and musculoskeletal physicians have real experience. Local availability is variable.
- **Access within the public system remains very limited.** A small number of specialists with EDS expertise work in private practice. Referral generally must come from a doctor.

12. Lifestyle, diet and the weight-loss trap

Many patients are genuinely complex, and prescribing for a myriad of symptoms is complex too — drug interactions and unusual side effects are common enough that "start low, go slow" should be the default.

There does appear to be a role for diet, and many patients report substantial benefit. **The evidence base is weak, largely observational and anecdotal, and I want to be clear about that rather than dress it up.** What patients consistently describe as helping:

- Unprocessed food — fresh, cooked from scratch
- Lower sugar
- A tendency towards lower carbohydrate
- Gluten/grains and dairy are a real problem for some (formally exclude coeliac disease first)
- A low-histamine trial for some, particularly where MCAS is suspected
- Trigger-hunting — the migraine model applied more broadly
- Exercise appropriate to current symptoms and instability — **the best exercise is the one the patient will actually do**
- Some describe the pattern as Paleo-Mediterranean

These won't cure anything. They help set a stable platform and tend to give steadier energy.

⚠ The caution I would now add, which was not in the 2021 version

Restrictive diets in this population carry specific and under-appreciated risk.

- **Disordered eating and ARFID are common** in hEDS, driven by pain, nausea, reflux and fear of symptoms rather than by body image. Every additional exclusion narrows an already narrow diet.
- **Weight loss is not benign here.** The aortomesenteric fat pad is what holds the angle open between the aorta and the SMA. Lose it, and both the left renal vein and the third part of the duodenum can be caught in the same pinch — nutcracker and SMA syndrome from the same mechanism (see section 9). SMA syndrome is self-perpetuating: weight loss thins the pad, compression worsens, vomiting increases, more weight is lost. It can become life-threatening.
- **Therefore: before recommending any exclusion diet, weigh the patient, document it, and monitor it.** If weight is falling, dietitian involvement is not optional. Nutritional adequacy in this group is a safety issue, not a lifestyle one.

Check ferritin, B12, vitamin D and coeliac serology before assuming symptoms are dietary.

13. The New Zealand context in 2026

This has been an extraordinary eighteen months for EDS in New Zealand, and any current NZ clinician should know the outline.

The Health New Zealand guidance

In February 2026, Te Whatu Ora published updated public information on EDS and HSD following what it described as a review of current evidence. The guidance included a "not recommended treatments" section listing interventions including intravenous fluids, opioid analgesia, artificial feeding and vascular abdominal surgery.

Patients and advocates responded immediately, describing the statements as misleading and potentially dangerous, noting that several of the listed treatments are used in complex cases to manage life-threatening complications. Within days, Health New Zealand confirmed the section had been removed pending review. EDS Aotearoa New Zealand sought an urgent meeting, alerted the Health Quality and Safety Commission, and at least two complaints were made to the Health and Disability Commissioner.

Why this matters for your practice: a blanket "not recommended" statement about a class of intervention is a different kind of claim from a statement that the evidence is weak, and it lands very differently on a patient who has been dismissed for a decade. It is worth being aware that many of your EDS patients read this, and read it as official confirmation that the system has decided against them.

Three documents, not one

There is a tendency in public discussion to merge distinct documents into a single "official position". They are not the same and should be considered separately:

1. The **Vascular Society position statement (2024)** — conservative on vascular compression syndromes and EDS, and largely defensible on the current evidence.

2. The **2023 regional review of multi-compression surgery** — also defensible given the state of the trial literature.

3. The **February 2026 Te Whatu Ora website guidance** — the weak link, containing factual errors and blanket discouragement of supportive care, partially withdrawn.

Disagreeing with the third does not require disagreeing with the first two.

All In Her Head

RNZ launched a multi-part investigative podcast by Anusha Bradley on 15 July 2026, following a young New Zealand woman, Rachel, who is starving and around whom clinicians disagree — some saying she needs life-saving surgery, others that the diagnosis doesn't exist. It explores, in RNZ's framing, the grey area where medicine, belief and evidence collide.

Your patients may have listened to it. Many will bring it to you. Having listened yourself is worth more than any position you might take on it. Link in section 14.

Access

- Specialist EDS care in the public system is extremely limited nationally.
- There is no funded EDS clinic in New Zealand.
- A handful of clinicians diagnose and treat EDS; several are in private practice.
- Many patients have faced years of misdiagnosis or of being told the problem is psychological — dozens told RNZ exactly this.
- ACC coverage for EDS-related injury remains inconsistent and is a common source of distress.

This is not a counsel of despair. Most patients can be managed well in primary care with a good physiotherapist, a clinician willing to coordinate, and a doctor who believes them. That last one is free.

14. References and resources

The essentials

Resource	Link
Ehlers-Danlos Society — best overall resource	https://www.ehlers-danlos.com/
New global diagnostic criteria — December 2026	https://www.ehlers-danlos.com/new-global-diagnostic-criteria-2026/
hEDS Diagnostic Checklist (2017 — current until 1 Dec 2026)	https://www.ehlers-danlos.com/heds-diagnostic-checklist/
Assessing joint hypermobility — video	https://www.ehlers-danlos.com/assessing-joint-hypermobility/
EDS Toolkit for GPs (formerly RCGP, now EDS UK)	https://gptoolkit.ehlers-danlos.org/
2026 Global Learning Conference — "Connecting the Stripes"	https://www.ehlers-danlos.com/2026-global-learning-conference/
Wallet card for patients	https://ehlers-danlos.com/wp-content/uploads/walletcard.pdf
This article and others	https://www.matpre.nz/ehlers-danlos

New Zealand

Resource	Link
Ehlers-Danlos Syndromes Aotearoa New Zealand	https://ehlers-danlos.org.nz
EDSANZ media and statements	https://ehlers-danlos.org.nz/media/
Loosely Speaking — NZ support group	https://www.facebook.com/groups/LooselySpeakingNZ
Rare Disorders New Zealand	https://www.raredisorders.org.nz
NZ EDS Guideline 2019 (RDNZ)	https://baynav.bopdhb.govt.nz/media/2677/nz-eds-guideline-v1-2019-nzord-rdnz.pdf
RNZ — *All In Her Head* podcast	https://www.rnz.co.nz/podcast/all-in-her-head
RNZ — HNZ removes EDS advice (Mar 2026)	https://www.rnz.co.nz/news/health/588410/
BOPDHB Grand Round — EDS (2018)	https://vimeo.com/277009100/38da6c1066

Primary literature

Classification and diagnosis

- Malfait F et al. The 2017 International Classification of the Ehlers-Danlos Syndromes. *Am J Med Genet C* 2017;175C:8–26. doi:10.1002/ajmg.c.31552
- Tinkle B et al. Hypermobile Ehlers-Danlos Syndrome. *Am J Med Genet C* 2017;175C:48–69. doi:10.1002/ajmg.c.31538
- Forghani I et al. Hypermobile Ehlers-Danlos Syndrome: diagnostic challenges and the role of genetic testing. *Genes* 2025. PMC12111038

Genetics, biomarker and the Road to 2026

- Gensemer C et al. KLK15 alters connective tissues in hypermobile Ehlers-Danlos syndrome. *iScience* 2025. doi:10.1016/j.isci.2025.113563 — the single-family finding **not** replicated by HEDGE; see https://www.sciencedirect.com/science/article/pii/S2589004225016049
- HEDGE Study (Hypermobile EDS Genetic Evaluation) — https://www.ehlers-danlos.com/hedge/
- Ritelli M et al. Bridging the diagnostic gap for hEDS and HSD: a common extracellular matrix fragmentation pattern in patient plasma as a potential biomarker. *Am J Med Genet A* 2025;197(1). doi:10.1002/ajmg.a.63857 — research-only; not a clinical test
- Halverson CM et al. Comorbidity, misdiagnoses, and the diagnostic odyssey in hEDS. *Genet Med Open* 2023;1(1):100812 — mean 10.4 years to diagnosis, 10.4 prior misdiagnoses
- McGillis L et al. Utilization of the 2017 hEDS criteria by the Toronto GoodHope EDS clinic: a retrospective review. *Am J Med Genet A* 2020;182(3):484–492 — only 15% of prior hEDS patients met 2017 criteria
- Ritelli M et al. Looking back and beyond the 2017 diagnostic criteria for hEDS. *Am J Med Genet A* 2024;194(2):174–194
- Road to 2026 — https://www.ehlers-danlos.com/road-to-2026/

Being believed

- Lee C, Chopra P. The Incidence of Misdiagnosis in Patients with Ehlers-Danlos Syndrome. *Children* 2025;12(6):698. doi:10.3390/children12060698
- Boelens OB et al. Randomized clinical trial of trigger point infiltration with lidocaine to diagnose anterior cutaneous nerve entrapment syndrome. *Ann Surg* 2013;257(5):845–849
- Jackson G. *Pain and Prejudice.* Allen & Unwin, 2019 — the cultural argument, centred on endometriosis, generalises

Comorbidities

- Lau DH et al. Postural Orthostatic Tachycardia Syndrome: A State-of-the-Art Review. *Heart Lung Circ* 2026;35(2):171–185. doi:10.1016/j.hlc.2025.09.004
- Vernino S et al. POTS: State of the science and clinical care from a 2019 NIH Expert Consensus Meeting — Part 1. *Auton Neurosci* 2021;235:102828
- Cardiac defects of hEDS and HSD: a retrospective cohort study (Mayo, n=568). PMC10982405

Vascular compression

- Abdominal Vascular Compression Syndromes: current and future management. *J Vasc Surg* 2023. https://www.jvascsurg.org/article/S0741-5214(23)00201-X/fulltext

Pain, anaesthesia, dislocation

- Chopra P et al. Pain Management in the Ehlers-Danlos Syndromes. *Am J Med Genet C* 2017. doi:10.1002/ajmg.c.31554
- Wiesmann T et al. Recommendations for anaesthesia and perioperative management in patients with EDS. *Orphanet J Rare Dis* 2014;9:109
- Parry J. *Dislocation/subluxation management — "I'm just popping out for a while!"* https://ehlers-danlos.com/wp-content/uploads/Dislocation-Subluxation-Management.pdf
- Local anaesthetic failure in EDS. https://ehlers-danlos.com/wp-content/uploads/local-anesthetic-failure.pdf
- ER Safety Tips for EDS Patients. https://www.painnewsnetwork.org/stories/2017/12/13/er-safety-tips-for-ehlers-danlos-patients

15. The Road to 2026 — indicative findings, and what changes in December

Everything in sections 3, 4, 5 and 6 of this document is written to the **2017** international classification. That classification is being replaced. The new framework publishes on **2 December 2026** in a special issue of the *American Journal of Medical Genetics* (the Ehlers-Danlos Society's page gives this date; some earlier announcements said 1 December). A second publication covering care and management pathways follows in **early 2027**. Both will be open access.

⚠ Everything in this section is INDICATIVE, not final

The findings below were presented in preliminary form — chiefly at the 2025 International Scientific Symposium in Toronto (17–21 September 2025) — and discussed further around the July 2026 Global Learning Conference. They are the direction of travel, not the destination. **Nothing here should be used for diagnosis.** The 2017 criteria remain the standard of care until the new framework publishes. Treat what follows as early notes to watch, and expect at least some of it to shift.

What the two big studies have indicated

HEDGE — the genetics (n ≈ 1,045). The Hypermobile Ehlers-Danlos Genetic Evaluation study whole-genome-sequenced about 1,000 people with hEDS and 45 with HSD from 86 countries, at the Broad Institute. Three indicative messages:

1. **The 2017 criteria, well applied, are catching the right people.** In over 99% of participants the clinical hEDS diagnosis held up. Fewer than 1% carried a variant pointing to a different connective tissue disorder — classical EDS, Marfan, Loeys-Dietz — and those individuals were contacted for follow-up. For a community used to being disbelieved, *"the world's largest genetic study confirmed your diagnosis was right"* is not a small sentence to be able to say.

2. **There is no single hEDS gene.** The signal points to multiple rare variants and complex interactions, not one clean mutation. hEDS increasingly looks like several related conditions producing a similar picture — part of why subtyping is on the table.

3. **KLK15 was not confirmed.** This is the key correction to version 3.0 of this document. HEDGE found KLK15 variants were **no more common in hEDS participants than in controls.** The earlier single-family finding (Section 7) stands as a genuine result in those families and in the mouse model, but it is not the general cause of hEDS — a good, humbling illustration of why a single-laboratory finding must meet a large replication cohort before it changes anything, and a further reason not to order kallikrein testing.

The hEDS/HSD Criteria Review Study — the clinic (n = 326, eight sites including New Zealand). For the first time the criteria have been tested prospectively against a comparison group of non-hypermobile chronic-pain patients. Indicative headlines:

1. **No single measure separates hEDS from HSD.** They appear to sit on one shared biological spectrum. This is the strongest signal yet that the 2017 two-tier split may not reflect a real biological boundary — and the clearest hint that the categories may be recombined or redrawn.

2. **Skin may be under-weighted.** Three or more of four specific skin parameters strongly predicted an hEDS/HSD classification. Skin is in the 2017 criteria but is easily overlooked when attention is on the joints; expect it to carry more weight.

3. **Hypermobility assessment may broaden beyond Beighton.** An expanded four-joint assessment — adding shoulder flexion, forearm rotation, ankle dorsiflexion, and first metatarsophalangeal (big toe) dorsiflexion — is being tested, precisely because the nine-point Beighton misses hypermobility outside its narrow set of joints and performs poorly in older and previously-operated patients. A second real-world validation study began in late 2025.

A possible blood biomarker — early, and research-only

A 2025 study in *AJMG Part A* (Ritelli et al.) found a distinctive pattern of extracellular-matrix fragments in patient plasma — a fibronectin fragment and type I collagen fragments — present in hEDS and HSD but absent in healthy controls and in classical EDS, vascular EDS, rheumatoid arthritis, psoriatic arthritis and osteoarthritis. Hold two things together: it is genuinely interesting, and it is **not a test you can order.** It was measured by Western blot in a research laboratory and needs validation in larger cohorts. Notably, hEDS and HSD showed the *same* pattern — another data point for the "one spectrum, not two conditions" reading.

The structural changes widely anticipated for December

Drawing the above together, the changes most often flagged by those close to the process — all still indicative — are:

- **The Beighton score revised or supplemented**, probably with additional joints.
- **The hEDS/HSD distinction reconsidered**, most likely toward a spectrum or merged category rather than a hard binary.
- **Comorbidities and systemic features given more formal diagnostic weight** — the autonomic, gastrointestinal, skin and related features that Section 8 already treats as central.
- **A clearer diagnostic pathway for non-specialists**, possibly including an interactive digital tool, aimed squarely at the decade-long diagnostic delay.
- **Possible new subtypes** as the genetics matures, likely carved out from what we now call hEDS.

"Will my patients lose their diagnosis?"

On current evidence, almost certainly not in any large numbers. The direction is toward *more* inclusive recognition, not less — the HEDGE <1% figure and the shared-spectrum finding both point that way. The honest caveat: a diagnosis applied loosely, or by a non-specialist, might need reconfirming under any new criteria. That is how criteria revisions work, and it is not a judgement on the patient's suffering. The aim of the review is better recognition, not narrower.

What this means for you, practically

- **Until 2 December 2026**, use the 2017 criteria and the current hEDS checklist. They remain the standard of care and nothing here changes that.
- **After publication**, this version is out of date for diagnosis, and I will rewrite it as version 4.0 once I have read the actual criteria rather than the trailers.
- **If a patient asks now**: *"The criteria are being rewritten and published in December — the first proper update since 2017. It may change how your condition is named and classified. It doesn't change what you're living with, and it doesn't change what we do for you between now and then."*
- **If a patient carries an HSD label**, the spectrum finding is genuinely hopeful: HSD looks likely to be recognised as part of the same picture as hEDS rather than a lesser category. Say so gently, and don't promise a specific outcome — the final naming has not been announced.

A version 4.0 will follow once the new criteria are published and I have read them properly rather than quickly.

Version 3.1 — August 2026. Interim update to version 3.0 (July 2026); supersedes version 2.2
This document is general information for health professionals, not personal clinical advice for any individual patient.

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Whakatāne, New Zealand — August 2026
https://www.matpre.nz/ehlers-danlos