INTRODUCTION TO HYPERMOBILITY

This New Zealand-based review is to help clinicians to recognise joint hypermobility and to consider possible diagnoses and management approaches when in day-to-day practice they see patients with one or more of the following:

- Joint hypermobility (double-jointed), especially when associated with musculoskeletal pain
- Recurrent subluxations/dislocations
- Unusual skin – especially skin fragility or hyperextensible skin
- Unusual bruising or bleeding

The spectrum of Hypermobility disorders includes GJH (Generalised Joint Hypermobility), HSD (Hypermobility Spectrum Disorder), hEDS (Hypermobile Ehlers-Danlos Syndrome) and the other EDS (Ehlers-Danlos Syndromes) types. They are considered together here for simplicity and because of potential diagnostic overlap at first sight. This broad-based overview sets out to provide the clinician with tools to place patients in a presumptive diagnostic category based on clinical features and to develop an initial management plan.

RED FLAGS

vEDS – Vascular EDS$^{1,2,10}$

- Arterial rupture or unusual bleeding in a child or young adult. Aorta, other large vessels and small vessels can be involved.
- See How to Save a Life$^{1,2}$ and vEDS reference article$^{10}$

All EDS – Anaesthetics and Surgery

- Local anaesthetic – local, regional or epidural has less effect and slower onset in EDS$^{27,28}$
  - May need more anaesthetic and longer wait before beginning procedure
- Surgical issues
  - Prone to bleeding
Any clinician planning surgery or anaesthesia in a patient with hypermobility or EDS should carefully read Wiesmann et al: *Recommendations for anaesthesia and perioperative management in patients with Ehlers-Danlos Syndrome(s)*

### Assessment

#### History

- History of hypermobility (double-jointed) – now or historically at any stage of life
- Hyperextensible (stretchy) skin
- Musculoskeletal pain (joint and muscle pain)
- History of one or more of these – this list is not exhaustive
  - Enquire about family history – all EDS has a genetic component
  - Repeated dislocations (often of more than one joint) - often “spontaneous” with less than usual trauma
  - Easy bruising – especially in children where the history is of no trauma but there are obvious bruises. Is in the differential for non-accidental injury.
  - Vessel rupture or unusual internal bleeding in a young person
  - Unusual or severe prolapses and hernias or bowel perforation in younger people
  - Associated symptoms that may be present (but are not diagnostic) include (but not limited to): fatigue, headaches, sleep disturbance, autonomic dysfunction (especially POTS – Postural Orthostatic Tachycardia Syndrome), irritable bowel, and Mast Cell Activation Syndrome

#### Examination – See “Aids to Diagnosis”

- Usual basic clinical examination
- Check for hypermobility - Check Beighton Score
  - If $\geq 4/9$, hypermobility is present (depends on age)
  - If positive, look for other EDS features to differentiate GJH, HSD and EDS
  - In adults, if too stiff or painful to do Beighton Score, use “Five Point Questionnaire” - see *Aids to Diagnosis*
  - Look for other causes of hypermobility e.g. neurological, trauma etc.
- Check skin for hyperextensibility – especially in cEDS. See *Aids to Diagnosis*
  - 3cm stretch at neck, elbow, and knee. 1.5cm forearm. 1 cm palm (thenar eminence).
- Check skin for atrophic scarring - see *Aids to Diagnosis*
- Assess joints and tender areas – typical inflammatory signs absent in face of significant pain

#### Investigations

- There are no specific or suggestive laboratory findings
- There are specific known genetic mutations/abnormalities for cEDS and vEDS (and most of the rare types) but not for hEDS. Clinical diagnosis is made first using criteria outlined in followed by genetic confirmation wherever possible and practical.

#### Differential diagnosis – a few suggestions (not exhaustive)

- Other causes of hypermobility such as constitutional, Marfan’s Syndrome, Trisomy 21, previous injury, neuropathic joints, osteogenesis imperfecta
- Vessel rupture – all other congenital and acquired causes including trauma
- Joint and muscle pain
Other arthropathies usually are typically inflammatory in nature, have more swelling and often have suggestive radiology or laboratory

Fibromyalgia

- Bruising (esp. in children) – other vasculopathies and clotting defects, e.g. von Willerbrands
- Stretchy skin - there may be other causes but in the context of hypermobility think of cEDS

Indications for transfer or initial specialised assessment

- Acute vascular rupture is a medical emergency – consult vascular surgeons
- All others – a clinician well versed in EDS if available
- Support services – as this can be a chronic, debilitating and painful condition, a multidisciplinary approach over the long term is important

AIDS TO DIAGNOSIS

BEIGHTON SCORE – Assessment tool for hypermobility

1 point for each side for 1-4 and 1 point for 5. Total 9. If ≥ 4/9, hypermobility is present (From 7).

See 3 for video.

THE FIVE-POINT QUESTIONNAIRE

Use in cases where it is not possible to do Beighton Score or where superimposed pain and stiffness in an adult will give a falsely low Beighton Score – See 7

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”?

A “yes” answer to ≥ 2/5 questions suggests joint hypermobility with 80–85%

SKIN - HYPEREXTENSIBILITY
SKIN - SCARRING

Hypermobile EDS            Classical EDS
A                          D
B                          E
C                          F

(From 7)

FACIAL FEATURES IN vEDS

Small Ear Lobes
Prominent Eyes
Narrow Nose
Thin Lips
Small Chin
MANAGEMENT

ACUTE EMERGENCIES

- Vascular rupture – appropriate vascular surgery or interventional radiology referral
- Dislocations – appropriate orthopaedic referral
- Acute pain – usual principles

DIAGNOSIS

- Give a tentative clinical diagnosis. Assure the patient you don’t think it’s all in their head – this is often what they have been told for a long time.
- Offer patient information on Ehlers-Danlos.com website and support groups – See References

REFERRAL

- If available refer to a clinician with experience in EDS – geneticist, rheumatologist, etc.
- A multidisciplinary team best leads care. Practically this often means the primary care doctor will refer to the appropriate specialist as and when needed, e.g. orthopaedic surgeon, neurosurgeon.

PAIN – INITIAL (ACUTE)

- Usual principles for initial management of acute or chronic pain
  - Usually start with paracetamol and work up as needed
  - There are no specific analgesics proven to have significant advantage in HSD/EDS
- Bracing or splinting can be helpful

INJURIES – INSTABILITY, SUBLUXATIONS, DISLOCATIONS, TENDON & LIGAMENT INJURIES – INITIAL EMERGENCY AND ACUTE (SHORT TERM) STRATEGIES

- Each individual injury episode (dislocation) should be treated as a new trauma on its own merits and not just passed off as “part of your condition”. Aim is to return to the pre-injury state. Even though a dislocation may be due to less force than in someone without EDS, the associated tissue effects and damage will likely be no less severe, e.g. Bankart lesion in shoulder.
- Dislocation - initial reduction
  - Because those with EDS may have severe pain, instability or injury elsewhere in the same region, some typical traction and twisting movements used for reduction may need to be modified to prevent injury elsewhere from the procedure itself, e.g. with shoulder dislocation pulling from the wrist or forearm may injure wrist or elbow. May need to modify hand position and grip. The force needed to reduce may be less than that needed for non-EDS. Some patients can “spontaneously reduce”.
  - Protect skin – it may be fragile. Use padding if needed.
  - Ligament and tendon injuries may take longer to heal and may recur after relatively less trauma after the first episode
- Subluxations may respond well to gentle manipulation rather than actual reduction
- Splinting and bracing are important after reduction
- Immediate post-trauma physiotherapy is used it is important to take into account the somewhat different approach needed in EDS.
- Appropriate rehabilitation is aimed at return to independent living
JOINT INSTABILITY AND PAIN – LONG TERM STRATEGIES TO STABILISE

After an initial injury some of the joints may become prone to repeat injury from relatively minor trauma

- Physiotherapy – see below
- External bracing
  - Braces and splints can be useful to prevent or after dislocation/subluxation as well as for comfort
  - In the first instance splinting is best discussed with and accessed through a local physiotherapy department.
  - Specific hypermobility-focused splint types can improve function and reduce pain even in the absence of current or recent subluxation/dislocation. As an example, finger splints along the lines of ring splints can be very helpful to prevent finger hyperextension to improve overall finger stability.
  - Neoprene or elastic supports and compressive clothing are also used and can be useful as they allow proprioceptive feedback as well as support and pain relief. Discuss options with physical therapist or occupational therapist.
  - Orthotic referral where appropriate
- Surgery may have a sub-optimal outcome but may be important in critical areas, e.g. craniocervical instability. When referring to or discussing with an orthopaedic surgeon, neurosurgeon, etc., be clear that the patient has EDS where issues may include slow healing, early recurrence and poor response to local anaesthetic agents.
- Non-surgical approaches to joint stabilisation such as prolotherapy offer some hope and may be appropriate in selected circumstances in adults. Materials (chemical/sclerosant) injected into ligaments are thought to induce healing with scarring and shortening which in turn may increase joint stability and thereby reduce pain and subluxation. Not yet widely available.

PHYSIOTHERAPY

- As there are limited management options, physiotherapy is key
- Traditional techniques of physiotherapy often need adapting for EDS and always require taking a wider view of the patient
- Physiotherapy should be aimed at empowering the patient towards self-management
- Aim for social rehabilitation training for independence with help of occupational therapists where needed
- Appropriate strategies:
  - Education and reassurance
  - Most patients will benefit from an individualised, carefully graduated exercise and activity programme
  - Physiotherapists can also advise on posture correction, proprioception training, splints/supports/taping, relaxation techniques, hydrotherapy, gentle manual techniques, including Myofascial release and pain management strategies
  - For dislocation and subluxation management, see “I’m popping out for a while”35
  - The Muldowney Protocol is an EDS specific programme that can take over a year to work through with a musculoskeletal therapist (6-9 months in younger patients). Not all patients can tolerate this and there are few physiotherapy departments that are likely to be able to provide a service over this length of time at this stage. Currently there is no evidence to show this is more beneficial than physiotherapy strategies listed above, but it may be worth investigating.
SURGERY AND ANAESTHESIA

- Surgical complications may be increased due to slow healing and potential for bleeding. Appropriate strategies should be planned and discussed in EDS context.
- Recurrence after surgery may occur because of the inherently abnormal ligaments.
- Some issues with anaesthesia:
  - Unstable neck may be an issue with positioning
  - Slow and suboptimal response to local anaesthetic incl. epidurals
  - Tourniquet can cause bruising and compartment syndrome
  - Positioning can cause unexpected subluxations including temporomandibular joint during anaesthesia

Any clinician planning surgery or anaesthesia in a patient with hypermobility or EDS should carefully read Wiesmann et al: *Recommendations for anaesthesia and perioperative management in patients with Ehlers-Danlos Syndrome(s)*

GENERAL SUPPORT

- Support groups offer advice on how to cope with day-to-day living with a painful chronic disease that may significantly disrupt life
- Chronic pain teams may help to design an overall pain management strategy
- Psychology support can be helpful

Request Help

A NOTE ON COORDINATING CARE

- Many patients can be managed by their GP and physiotherapist with occasional input from an EDS specialist
- Some with complex or severe issues will need a truly multidisciplinary team that may include orthopaedics, neurosurgery, gastroenterology, etc.
- Appropriate referrals for specific interventions, e.g. hand therapist, prolotherapy

ADVICE

- Best all-round web advice: Ehlers-Danlos Society
- Some geneticists, rheumatologists and musculoskeletal specialists have experience in diagnosing and managing EDS. Local availability will likely be variable.
- The New Zealand EDS Working Group that is part of NZORD (New Zealand Organisation for Rare Disorders) may be able to offer some resources in the near future. This may include a list of Health Professionals with EDS knowledge and experience. See NZORD.

GENERALISED JOINT HYPERMOBILITY (GJH)

There is a spectrum of GJH

1. Asymptomatic (non-syndromic) GJH – hypermobility without other symptoms. Other causes, e.g. neurological deficit must be excluded.
2. Symptomatic GJH that doesn’t meet criteria for hEDS – HSD
3. A well-defined syndrome - hEDS
Joint hypermobility can lead to micro and macro trauma, which in the long run can be a leading cause of pain and in adulthood degenerative changes. This can range from hyperextension injury to mild subluxation through to frank dislocation. Each event leads to surrounding collateral damage.

**HSD – HYPERMOBILITY SPECTRUM DISORDER**

- Common
- Hypermobility and significant additional symptoms are typically limited to the musculoskeletal system
- Pain may be significant and debilitating
- Types
  - Generalised HSD – G-HSD
    - GJH
    - Musculoskeletal manifestations – Similar to hEDS
  - Peripheral HSD – P-HSD
    - Hypermobility in hands and feet only
    - Musculoskeletal manifestations – Similar to hEDS
  - Localised HSD – L-HSD
    - Hypermobility in a single joint
    - Musculoskeletal manifestations – Similar to hEDS
  - Historical – history of one of the above usually with 5-Point Questionnaire

**EDS – EHLENS-DANLOS SYNDROMES**

EDS (Ehlers-Danlos Syndromes) are a group of inherited disorders characterised by defects in collagen mainly affecting the ligaments and soft tissues

- Relatively rare – about 1:5000. F>M
- Basis is abnormal collagen – different types of collagen in each of the EDS types
- The commonest (>80-90%) is hEDS – genetic basis currently not known
- cEDS and vEDS are uncommon – have a known genetic basis (see Table below)
  - All the rest are very rare but genetic basis is clear

There are 13 types of EDS with significant overlap in features

**CLINICAL CLASSIFICATION OF EDS**

<table>
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<tr>
<th>EDS SUBTYPE</th>
<th>ABBREV</th>
<th>INHERITANCE</th>
<th>GENETICS</th>
<th>COLLAGEN</th>
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<td>cEDS</td>
<td>AD</td>
<td>COL5A1, COL5A2</td>
<td>Type 5</td>
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<td>AD</td>
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<tr>
<td>10 other types</td>
<td>See Ref 6,7</td>
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<td></td>
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</table>

Ver 5.2 - 20/07/17
hEDS – HYPERMOBILE EDS

- Most common
- See hEDS Diagnostic Checklist for details and how to apply criteria - includes a downloadable pdf
  - **Criterion 1** - Main feature (must be present)
    - Generalised Joint Hypermobility (Beighton Score ≥ 4/9 over age 50; ≥ 5/9 in young adults; ≥ 6/9 in children and adolescents)
  - **Criterion 2** (at least 2 features)
    - Feature A – Generalised connective tissue disorder (at least 5/12 present)
      - Soft, velvety skin
      - Mild skin hyperextensibility (not as much as cEDS)
      - Unexplained striae
      - Piezogenic papules (nodules on side of feet)
      - Recurrent/multiple hernias
      - Atrophic scarring
      - Arachnodactyly
      - Arm span-Height ratio: ≥ 1.05
      - Pelvic floor prolapse
      - Dental crowding
      - Mitral valve prolapse
      - Aortic root dilatation
    - Feature B
    - Family history (autosomal dominant but can appear to skip)
    - Feature C (at least 1) - see details in checklist
      - Musculoskeletal pain
      - Chronic widespread pain ≥ 3 months
      - Recurrent joint dislocations/instability in absence of significant trauma (may also occur with trauma)
  - **Criterion 3** – All must be present
    - No skin fragility
    - Reasonable exclusion of other connective tissue disorders e.g. Marfan’s
    - Exclusion of other disorders that could cause GJH

To diagnose hEDS all 3 criteria must be satisfied.

- Other features that can occur (not an exhaustive list)
  - Sleep disturbance, chronic fatigue, POTS, functional GIT disorders, unusual hernias, internal hernias, dysautonomia, Raynaud’s, MCAS, some cardiac features, osteoarthritis secondary to joint instability, headaches, TMJ dysfunction, increased gynaecological presentations, pelvic floor dysfunction, anxiety, depression. Multiple other features that affect quality of life may be part of the spectrum.
  - Overall effect on life may range from severe (bed-ridden) to relatively minor

cEDS – CLASSICAL EDS

- Relatively common
- **Major criteria**
  - Skin features - hyperextensible skin, atrophic scarring (esp. knees & elbows)
  - Generalised Joint Hypermobility
- **Minor criteria**
To diagnose cEDS:

- Criterion 1 – Skin features

  Plus

- Criterion 2 – GJH &/or at least 3 minor criteria

Needs confirmation with genetic testing if possible.

vEDS – VASCULAR EDS

- Rare and dangerous
- **Major criteria**
  - Family history proven vEDS
  - Arterial rupture at young age
  - Spontaneous colon perforation in absence of other disease
  - Uterine rupture without predisposing cause
  - Carotid-cavernous sinus fistula without trauma
- **Minor criteria**
  - Bruising not related to trauma or in unusual sites
  - Thin, translucent skin with easily visible veins
  - Characteristic facial appearance
  - Spontaneous pneumothorax
  - Acrogenic
  - Talipes equinovarus
  - Congenital hip dislocation
  - Hypermobility of small joints
  - Tendon and muscle rupture
  - Keratoconus
  - Gingival recession and fragility
  - Early onset varicose veins

To diagnose need a family history with arterial rupture or dissection younger than 40 or any of the other major features

Genetic testing is important

ALL OTHER TYPES – See Ref 7
UNUSUAL SYMPTOMS THAT CAN BE ASSOCIATED WITH EDS – esp. hEDS

- Dysautonomia (autonomic dysfunction) such as POTS (postural orthostatic tachycardia syndrome)
- MCAS (mast cell activation syndrome) masquerading as unusual allergies
- GIT symptoms including constipation
- Raynaud’s

A Note on Genetics

Of the three most common forms of EDS, vEDS and cEDS have an established genetic basis. Determining the exact molecular basis for these conditions can be useful for diagnosis, management and reproductive decision-making. It is, however, not mandatory that every person with a clinical diagnosis of EDS undergoes molecular analysis.

To arrange for an assessment of the genetic basis for a person with a vEDS or cEDS phenotype, a referral can be made to Genetic Health Services NZ through any one of their three hubs centered in Christchurch, Wellington or Auckland. See the GHSNZ website for details on how to lodge a referral and request for assessment at www.genetichealthservice.org.nz

Rarer forms of EDS can also be assessed in a similar manner on a case-by-case basis.

For hEDS (by far the most common), although there are clear familial (autosomal dominant) components underpinning the disorder, a defined molecular basis has not been discovered. A referral of such individuals can still be made to GHSNZ, but beyond a confirmation of the clinical diagnosis little specific laboratory investigation can be offered.

Note that genetic services criteria for acceptance are such that assessment is not guaranteed, especially for hEDS.

This document has been edited by Matthew Preston (Radiologist) for New Zealand EDS Working Group
July 2017
Email: matpre@gmail.com
1. **HOW TO SAVE A LIFE** - a condensed list of life-saving surgical and post-operative suggestions for patients with Vascular Ehlers-Danlos Syndrome (a bit dated but succinct) [http://edstoday.org/urgent/](http://edstoday.org/urgent/)


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**General References**


For Physiotherapists


For Patients and Doctors Seeking Advice


33. Loosely speaking – New Zealand support Facebook page – type “Loosely Speaking” into the Facebook search box. This is a closed group – so ask to join. Link is https://www.facebook.com/search/top/?q=loosely%20speaking%20%20ehlers%20danlos%20nz%20support%20group

34. NZORD – New Zealand Organisation for Rare Disorders at http://www.nzord.org.nz - includes Specialist Directory


36. Genetic and Rare Diseases Information Centre at https://rarediseases.info.nih.gov/diseases/6322/ehlers-danlos-syndromes

37. Myths and facts - for patients - at http://edstoday.org/myths-and-facts/
Appendix 1 – Flowcharts

"Unspecified" Heritable Disorder of Connective Tissue

- Marfan Syndrome
- Loys–Dietz
- Ehlers Danlos Syndromes
- Osteogenesis imperfecta

Cardiology review with echocardiogram

Assessment of bone health

(Re) Consider the underlying genetic diagnosis

Hypermobility child
Belmont ≥ 4/9

Check for functional symptoms of "joint hypermobility syndrome" and formulate a management plan

Anxiety

Disabling Fatigue

Joint instability episodes

Soft tissue injuries

Widespread musculoskeletal pain

Osteopenia

Paediatric patients with symptoms suggestive of EDS

Symptoms suggestive of EDS presenting to the GP

Joint hypermobility +/- pain
- Belmont Criteria ≥3/9

Cutaneous features
- Skin hyperextensibility
- Atrophic scars
- Severe skin laxity
- Soft, doughy, velvety skin
- Thin, translucent skin
- Excessive/equatorial bruising or bleeding

Unexplained vessel or hollow organ rupture

- +/- Family history of EDS

Joint hypermobility +/- pain
- Not family history of EDS
- Photodamaged or other systemic CTD manifestations

Hypermobility EDS (hEDS)
- Generalised joint hypermobility
- Skin hyperextensibility and atrophic scarring
- AND/OR other skin features [https://ehlers-danlos.com/heds-diagnostic-checklist]

Classical EDS
- Skin hyperextensibility and atrophic scarring
- AND Generalised joint hypermobility OR other skin features

Classical-like EDS
- Skin hyperextensibility, velvety skin but NO atrophic scarring AND
- AND Generalised joint hypermobility
- AND Easy/spongy connective tissue

Vascular EDS
- Family history of EDS
- Arterial rupture/dissection <65 years, spontaneous pneumothorax

Cardiac-valvular EDS
- Severe progressive cardiac-valvular problems
- AND Generalised joint hypermobility OR other skin features

Hypermobility Spectrum Disorder and hEDS

See Management appendix

?EDS – refer to General Paediatrician

- Consider differential diagnosis: see Appendix...
- Clarify EDS phenotype: see
  [http://ehlers-danlos.com/eds-types]
- Appropriate onward referral for:
  1. Investigations
  2. Management appendix...
- Genetic testing: see box 6.
### APPENDIX 2 – WORKING GROUP MEMBERS (Alphabetical)

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<th>ORGANISATION</th>
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<tr>
<td>BURLING; Fraser</td>
<td>Rheumatologist Auckland</td>
<td>Rheumatologist</td>
<td><a href="mailto:backtolife@iconz.co.nz">backtolife@iconz.co.nz</a></td>
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<tr>
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<td><a href="mailto:Rachel.Callear@huttvalleydhb.org.nz">Rachel.Callear@huttvalleydhb.org.nz</a></td>
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<tr>
<td>CAMPBELL-STOKES; Priscilla</td>
<td>Hutt Valley DHB</td>
<td>Paediatric Rheumatologist Hutt Valley DHB</td>
<td><a href="mailto:Priscilla.Campbell-Stokes@huttvalleydhb.org.nz">Priscilla.Campbell-Stokes@huttvalleydhb.org.nz</a></td>
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<tr>
<td>CRAWFORD; Lisa</td>
<td>NZORD</td>
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<tr>
<td>PRESTON; Matthew</td>
<td>BOPDHB; Eastbay Radiology</td>
<td>Medical - Radiologist</td>
<td><a href="mailto:matpre@gmail.com">matpre@gmail.com</a></td>
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<tr>
<td>ROBERTSON; Stephen</td>
<td>Department of Women’s and Children’s Health Dunedin School of Medicine University of Otago</td>
<td>Curekids Professor of Paediatric Genetics</td>
<td><a href="mailto:Stephen.robertson@otago.ac.nz">Stephen.robertson@otago.ac.nz</a></td>
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### HYPERMOBILITY AND EDS – PATHWAY DOCUMENT LIST – To follow

This current document is an overview of Hypermobility and EDS. Several of the areas covered require more detailed information to guide management in more detail. As they become available the additional documents will be added alongside this one.

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<td>Overview and basic management guidelines with extensive references</td>
<td>Current – this document</td>
<td>Matthew Preston</td>
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