Hypermobility and Hypomobility

Hypermobility

Hypermobility is an increased degree of motion at a joint.
Hypermobility can occur at one joint or several joints, or can be generalized throughout the body. It can range from mild joint laxity to extreme mobility or joint instability.

Women and children tend to be more flexible than men and the elderly.

Racial origin can have an influence on ROM.

Hypermobility and compensatory weakness can be created in the body by soft tissue tightness and hypomobility in another area.

Physiological hypermobility occurs in some body types and occupations and during pregnancy.

The hormone **relaxin**, secreted during pregnancy, allows joint capsules and ligaments to become hypermobile to facilitate labour and delivery.

Joints in the pelvis, ankle, and feet, remain hypermobile for up to 6 months post partum.

Hypermobility itself doesn't mean P and dysfunction, persons with joint laxity may be at risk for musculoskeletal symptoms and injuries including sprain, tendinitis, OA, and entrapment neuropathies such as Carpal tunnel syndrome.

Hypermobility syndrome occurs in up to 5% of those with hypermobility; the person experiences symptoms including mm and joint P, overuse syndromes, such as Tendinitis, abdominal mm weakness, hyper extensible skin, and mitral valve prolapse.

Certain pathologies predispose people to hypermobility.

- (RA**) Rheumatoid arthritis**, a systemic autoimmune disorder characterized by inflammation and destruction of connective tissue, leading to hypermobility of the affected joints
- Ehlers-Danlos syndromes, are a group of inherited disorders, characterized by joint hyper mobility and sometimes dislocation, skin hyperextensibility, increased bruising and tissue fraugility

- **Marfan's syndrome**, is an inherited disorder with fragmentation of elastin, leading to joint hyper mobility, elongated bones, aortal widening, mitral valve prolapse and changes in the eye.

Medical

Damage to a joint is sufficient, a hypermobile joint may be treated with injections to sclerose the joint capsule or vertebral disc, surgical shortening of ligaments or mm's crossing the joints.

Fixation externally with splints or internally with pins, plates.

Causes

Compensation: due to postural dysfunction elsewhere in the body.

Increased flexibility: due to body type, occupation.

Hormonal influences: during pregnancy.

Joint trauma: sprains, strains or dislocation.

Pathologies and conditions: causing joint laxity such as RA, E-D syndromes, MS, and peripheral nerve lesions.

Symptoms

The affected joint has a greater than normal ROM.

Joint capsule is lax.

MM's crossing the joint may be H+ in an attempt to support the joint

CONTRAINDICATIONS

Do not mobilize a hypermobile joint.

Do not stretch muscles that cross a hypermobile joint past the accepted range for that joint.
**Please read Assessment, make point form notes of what you will see in observation, palpation, and testing

TREATMENT

No techniques used specific on the hypermobile joint. Hypermobility is combined with compensatory hypomobility at another joint proximal and distal to the hypermobile joint.

Joint play is used on the hypomobile joints.
TP's may be present in mm's that cross a hypermobile joint, these are treated with mm stripping or ischemic compressions, followed by heat, no stretch.

EXAMPLE: Hyperkyphotic treatment, the client has reduced thoracic mobility and protracted shoulders. Protraction causing the GH joint turning inferiorly, allowing the humeral head to slip inferiorly. All rotator cuff mm's need to work harder to keep the humeral head in place, the joint capsule stretches and the GH joint becomes hypermobile.

SELF-CARE
Strengthening program is **key** with hypermobile joints.

Start with isometric exercises, progressing to isotonic exercises.

Refer out to physiotherapy, chiropractor or physician if needed.

HYPOMOBILITY

Hypomobility is loss of motion at a joint, including the loss of normal joint play movements.

Hypomobility can occur at one joint or several joints.

Joints on the dominant side tend to be more hypomobile than non dominant.

Hypomobile joints can lead to strained mm's, peripheral nerve compression, tendinitis, and decreased ability to perform ADL's.

CAUSES

Compensation: due to postural dysfunction.

Decreased flexibility: body type or occupation.

Intra-articular and extra-articular adhesions: joint trauma, surgery, immobilization, prolonged bed rest and wheelchair use.

Surgical fixation: pins, screws, plates, rods, surgical shortening of ligaments or mm's that cross the joint.

Pathologies and conditions causing contractures: Dupuytren's contracture, frozen shoulder, ankylosing spondylitis, PN lesions, and CN lesions.

SYMPTOMS

The affected joint has a reduced ROM.

Stiffness and P are present.

The joint capsule may be fibrosed, shortened fascia, scar tissue or contractures.

Nutrition is reduced to the articular surfaces of the joint.

Myofascial P syndromes, TP's are present un the mm's that cross the joint.

CONTRAINDICATIONS

Do not attempt to mobilize a hypomobile joint that has been surgically repaired with metal appliances.

Where ligaments have been surgically shortened, do not restore full ROM of the affected joint in the direction that will stretch the repaired ligament.

**Please read Assessment, make point form notes of what you will see in observation, and testing.

TREATMENT

Hydro: Heat is used to make the soft tissue crossing the hypomobile joint more flexible.

Treat compensatory structures first, DDB

GSM

Fascial techniques

TP treatment, followed by heat and stretch.

Passive stretching

Frictions, followed by a stretch 30 sec+, and Ice up to 5 mins.

Joint play on the hypomobile joint, increasing grades of oscillations with progress.

SELF-CARE

Heat is applied to soft tissues.

Passive self stretching 30-3-3 to maintain flexibility.

Refer to physiotherapy, chiropractor, or physician if needed