Lothian Spasticity Management Service

A guide to managing spasticity

Does your neuro patient have spasticity?

Is spasticity impacting on function, care, comfort?

Are you looking for advice about best management?

Do you wonder if specialist assessment is needed?

This guide has been devised by the Lothian Spasticity Management Service to support staff in understanding and managing spasticity. We hope it will give you useful information to help your clinical reasoning to develop a management plan for your patient with spasticity.

The Lothian Spasticity Management Service is a multidisciplinary team comprising medical, nursing and physiotherapy specialists. The service aims to provide assessment, advice and, where appropriate treatment for adults with troublesome spasticity with a neurological origin.

Understanding spasticity

Spasticity is a common impairment in neurological conditions such as: Stroke, Multiple Sclerosis, Cerebral Palsy, Acquired Brain Injury, and Spinal Injury.

"Disordered sensorimotor control, resulting from an upper motor neurone lesion, presenting as intermittent or sustained involuntary action of muscles."

(EU – SPASM Group, 2005)

Elements of spasticity to consider

Neurological component

- Increased state of reflex activity is continually present
- Normal feedback mechanism to mediate muscle tone is reduced or absent
- Muscle is constantly contracting

Biomechanical component

- Muscles held in a short position show physiological changes - sacromeres (units of elasticity) are lost and are replaced by connective tissue
- Muscle becomes short and stiff
- There can also be physiological changes in other soft tissues such as tendons, ligaments, joint capsule

Key features of spasticity

- Muscles may look and feel bulky and tight
- You feel resistance when trying to move a body part through its normal range of movement
- Resistance will increase if you try to move the body part more quickly – this is because spasticity is velocity dependant – the faster you try to do the movement, the more resistance you will feel.

There can be confusion between spasticity and contracture. Here are some of the key differences:

Spasticity

- Primary impairment resulting from an upper motor neurone lesion
- Feel resistance to passive stretch
- Velocity dependent i.e. resistance is greater when the body part is moved quickly
- There will be less resistance if the body part is moved slowly and the amount of movement achieved may be greater

Contracture

- Secondary consequence of immobility resulting from physiological changes in muscles and other soft tissues
- Feels as if you have reached a solid point and there is no "give"
- Speed of movement does not influence the amount of movement achieved

Patterns of spasticity

Spasticity can present in different patterns. Thinking about the pattern of spasticity can be useful when considering management options.

Generalised spasticity
Increased muscle tone affecting multiple limbs and/or the trunk
Example:

- tetraparesis with mass abnormal movement patterns

Multi-focal spasticity
Increased muscle tone throughout a limb or limbs affecting multiple joints
Example:

- hemiparesis with upper limb spasticity affecting shoulder, elbow, wrist and fingers and/or lower limb spasticity affecting hip, knee and ankle.

Focal spasticity
Increased muscle tone affecting one part of a limb

Example:

- clenched fist
- flexed elbow
- altered posture of ankle and forefoot.

Associated reaction

- Involuntary and non-functional spontaneous movements, often related to activity e.g. elbow flexion associated with activity such as walking

Spasm

- Sudden involuntary contraction of one or more muscles
- Can be painful
- May be provoked by sensory stimuli e.g. pain, touch
- Can have a negative impact on posture, function, sleep, quality of life

Dystonia

- Involuntary sustained muscle spasms which can force affected body parts into abnormal movements or postures
- Abnormal postures are not fixed - often repetitive, slow, writhing movements occur

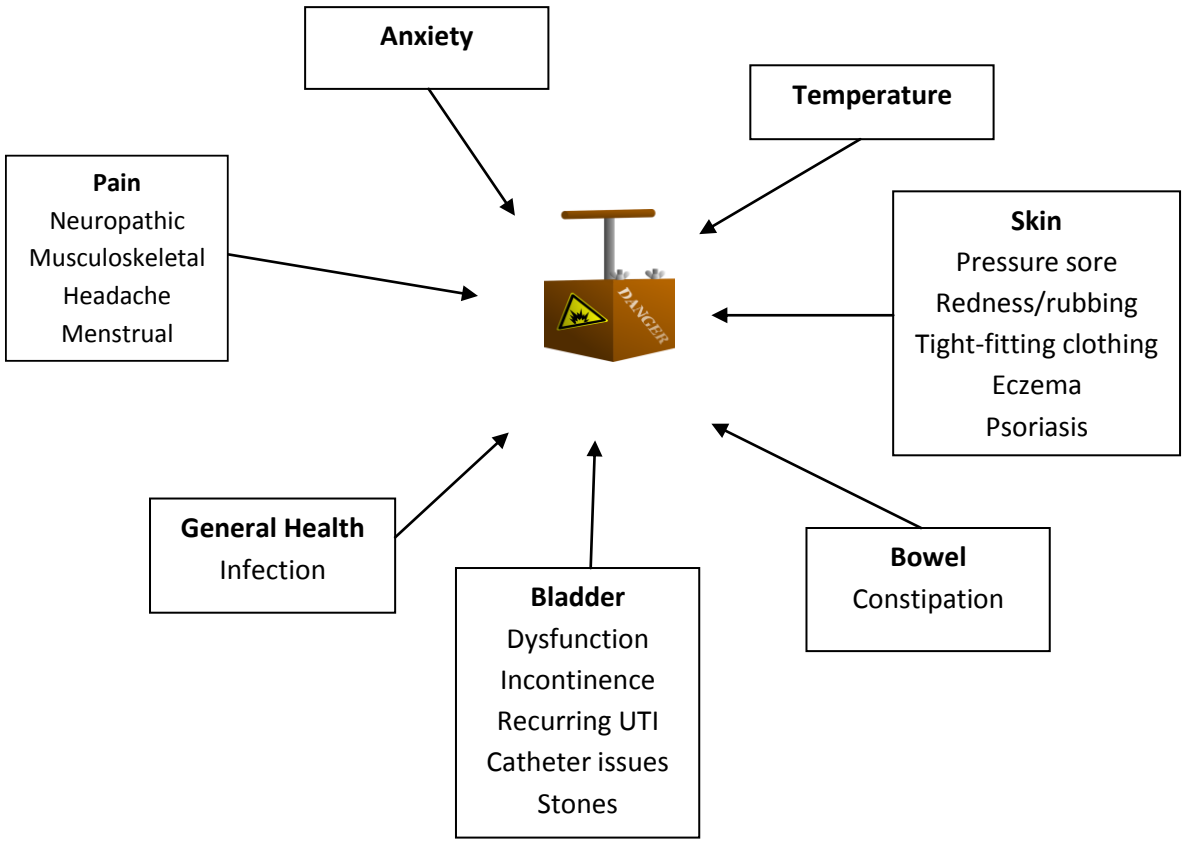
Managing spasticity

Managing spasticity can involve considering a number of different interventions and may involve a number of different members of the MDT. The management plan will take account of information from your assessment of spasticity, the impacts it is having and the patient's goals for treatment.

General spasticity Multi-focal spasticity Spasm	• Consider impacts on patient and set patient-centred goals • Address any **trigger factors** where possible • Implement appropriate **physical treatment strategies** • Consider starting/modifying **oral antispasticity medication**
Focal spasticity Associated reaction Dystonia	• Consider impacts on patient and set patient-centred goals • Address any **trigger factors** where possible • Implement appropriate **physical treatment strategies** • Consider **focal treatment** to specific muscles – this requires referral to a specialist service

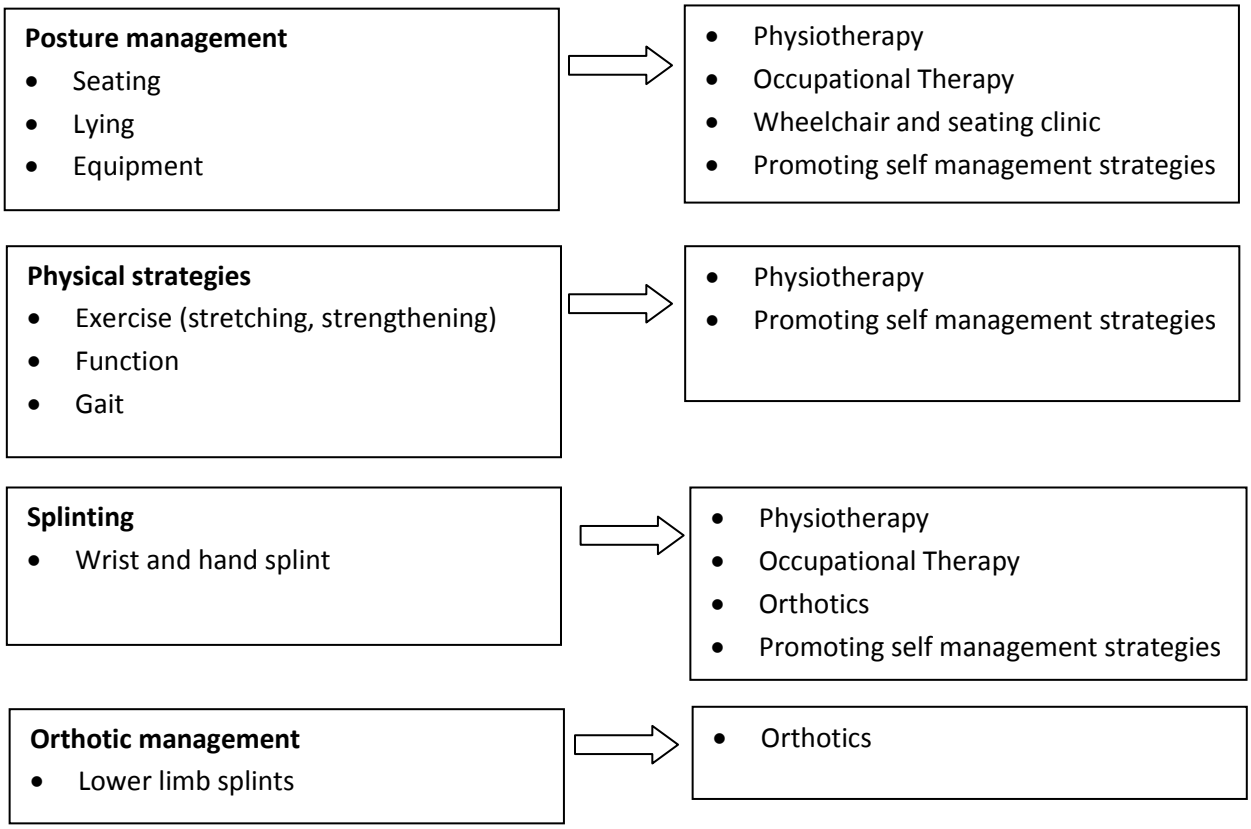
Trigger factors for spasticity

There are various things that can aggravate spasticity. These need to be identified and managed effectively. If these trigger factors are not addressed, other spasticity management strategies may be less effective.



If trigger factors are well addressed and managed there may be less need for medical and physical intervention to manage spasticity.

Physical management strategies



Medication management – oral antispasticity medications

	1st Line	**2nd Line**	**3rd Line**
Medication	**Baclofen**	**Tizanidine**	**Dantrolene**
How it works	Central Action –simulates the GABAB-receptors (therefore inhibits the release of excitatory amino acids.)	Central Action- stimulates presynaptic alpha2-receptors (therefore inhibits the release of excitatory amino acids.)	Peripheral Action- reduces calcium release within skeletal muscle cells.
Starting dose	5mg TID	2mg once daily	25 mg once daily
Dose escalation	Increase by 5mg every 3 days (if in hospital) **or** every week (if in community)	Increase by 2mg every 3 days (if in hospital) **or** every week (if in community)	Increase by 25 mg weekly Titrate down and stop if no benefit in 6-7 weeks
Maximum daily dose	100 mg (in 3 divided doses)	36 mg (in 3-4 divided doses; maximum single dose 12 mg)	400 mg (up to 4 divided doses) *(Usual dose 75 mg TID)*
Peak Concentration	1-3hrs post ingestion	1hr post ingestion	5hours post ingestion
Common side effects	Muscle weakness; sedation; dizziness; nausea,	Drowsiness; sedation; dizziness/light-headedness; hypotension,	Drowsiness; sedation; dizziness/light-headedness; diarrhoea

Considerations/monitoring	**Epilepsy:** can lower seizure threshold **Bladder:** can have effect on bladder muscle and can cause increased urinary problems **Renal impairment:** increased side effect profile	**Liver:** contraindicated in hepatic impairment; can cause changes in liver function – **MUST monitor LFTs monthly for first 4 months** **Renal impairment:** increased side effect profile	**Liver:** can cause Severe Liver Impairment– **MUST monitor LFTs monthly initially then at less frequent regular interval.** Patient must be counselled about signs of liver dysfunction. Those greatest at risk of liver failure- -Higher daily doses (>400mg / day) -Duration of treatment (3-12 month) - Female - Age >30 years - prior history liver disease - on other hepatotoxic medications **Renal impairment:** increased side effect profile **Other:** Makes skin more sensitive to sun – advice to take precautions.
Stopping medication	Avoid abrupt withdrawal – dose should be titrated slowly down. *Abrupt withdrawal can cause a rebound increase in spasticity, hyperactive state, and can precipitate autonomic dysfunction, including hyperthermia, psychiatric reactions and convulsions.*	Avoid abrupt withdrawal – dose should be titrated slowly down. *Abrupt withdrawal can cause rebound hypertension and tachycardia*	Avoid abrupt withdrawal – dose should be titrated slowly down. *Abrupt withdrawal can cause hallucinations, rebound hypertension, tachycardia and seizures*

Medication management - night time spasm

Clonazepam (a benzodiazepine) has muscle relaxant properties which can have a significant beneficial effect in reducing night time spasms
Dose: Normally 500 micrograms taken at night (can be initiated at a lower dose of 250 micrograms if there are concerns around side effects or if patient has reduced renal function). Can be increased in 250/500 microgram increments up to 2 mg if required
Side effects: Amnesia, confusion, dizziness, drowsiness, fatigue, muscle weakness, reduced coordination, restlessness
Considerations: Concurrent use of Baclofen and Tizanidine may increase the risk of CNS depressant side effects, which may affect ability to perform skilled tasks.
Refer to BNF for information about cautions and contraindications

Referral to specialist spasticity management services

Before referring to a specialist spasticity management service, STOP and THINK:

- Does the patient have spasticity arising from a neurological condition?
- Have you considered and managed trigger factors to an optimum level?
- Have you tried appropriate physical management strategies?
- Have you considered/tried oral antispasticity medications (if appropriate)?

Despite all measures taken:

- Are spasticity and/or spasms still having a negative impact on the patient?
- Are there still patient-centred goals that spasticity management could help achieve?

If you answer YES to these questions then referral to a specialist spasticity management service will be appropriate.

You can refer to the **Lothian Spasticity Management Service** by using this referral form *(insert link)*

You can email informal enquiries to the team to this address *(insert email address)*.

The team review and respond to enquiries on *(insert day/time)*