

# Medication for the management of spasticity in seating

Dr. Alison Hatfield,  
Consultant in Rehabilitation Medicine,  
St.Mary's Hospital, Portsmouth

# Introduction

- What is spasticity?
- Why is spasticity important?
- Approaches to spasticity management
  - Oral medication
  - Botulinum toxin
  - Invasive treatments
- Conclusions



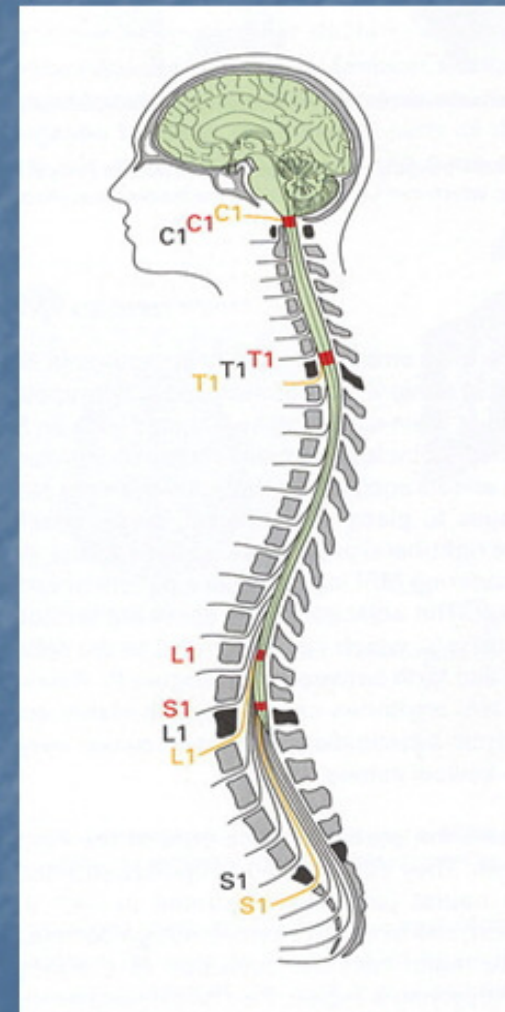
# What is spasticity?

- 'Spasticity is a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex as one component of the upper motor neurone syndrome'
- Lance 1980



# Who gets spasticity?

- Occurs in conditions where there has been an upper motor neuron lesion
- Multiple sclerosis
- Cerebral palsy
- Traumatic brain injury
- Stroke
- Spinal cord injury





# Evolution of spasticity after injury

- Disruption of descending pathways
- Muscle flaccidity
- In acute care- patients lie immobile with muscle groups often in shortened position for long periods of time.
- Subacute- at several weeks plastic neural reaarangements, sprouting of fibres, emergence of abnormal excessive reflex responses to peripheral inputs

What effects does spasticity  
have on seating?



# Seating and spasticity

- Reduced stability and difficulty seating
- Pressure ulcers
- Pain
- Spasms
- Contracture development



# What problems can spasticity cause in fitting a wheelchair?

- Sliding out of their wheelchair- posterior tilted pelvis, tight hamstrings, knees flexed
- Tight hip adduction or windsweeping
- Spasms of feet, difficulty reaching the footplates
- Getting in & out of the wheelchair, standing transfers or hoist
- Spasticity may vary so one set up might not solve the variable nature of their posture



# Should we treat spasticity?

- Severity
- Impact on activities of daily living
- Pain
- Relation to position- suitability of equipment
- But it can aid transfers and walking in patients with severe lower limb weakness
- And it can aid grip in an otherwise paralysed upper limb







# Treatment for spasticity

- Multidisciplinary
- Patient and carers
- Nursing and medical
- Physiotherapy
- Occupational therapy
- Wheelchair service team
- Orthotist



# Review of medical problems

- Underlying condition, general medical illness
- Bladder- Urinary tract abnormalities
- Bowels- Constipation
- Skin- Pressure sores, nail fold infections
- DVT
- Heterotopic ossification
- Pain





# Medical treatments for spasticity

- Oral medication
- Intramuscular botulinum toxin
- Intrathecal pumps
- Phenol
- Ablative surgery



# Oral treatment



- Antispasticity agents: baclofen, tizanidine, dantrolene
- Analgesics
- Anticonvulsants



# Baclofen

- Agonist of the inhibitory neurotransmitter GABA
- Originally used as an anticonvulsant- anti seizure activity low but found to be effective on spasticity
- Acts at the level of the spinal cord to inhibit calcium intake which impedes release of excitatory neurotransmitters



# Baclofen dosing

- After oral dose peak level at 2-3 hours, half life 3-4 hours
- Initial dose of 2.5-10mg per day  
maintenance of 20-90mg per day in three divided doses; doseage needed often increases over months



# Tizanidine

- Derivative of clonidine
- Acts centrally as an alpha- 2 adrenergic agent to modulate the release of excitatory neurotransmitters
- Half life 2-3 hours
- Shown to reduce the spasticity in adults with SCI and MS





# Doses of Tizanidine...

- Given in doses of 4-8mg per day up to 8-24 mg per day
- Side effects sedation (40-50% of individuals), dizziness, dry mouth, hypertension, liver damage





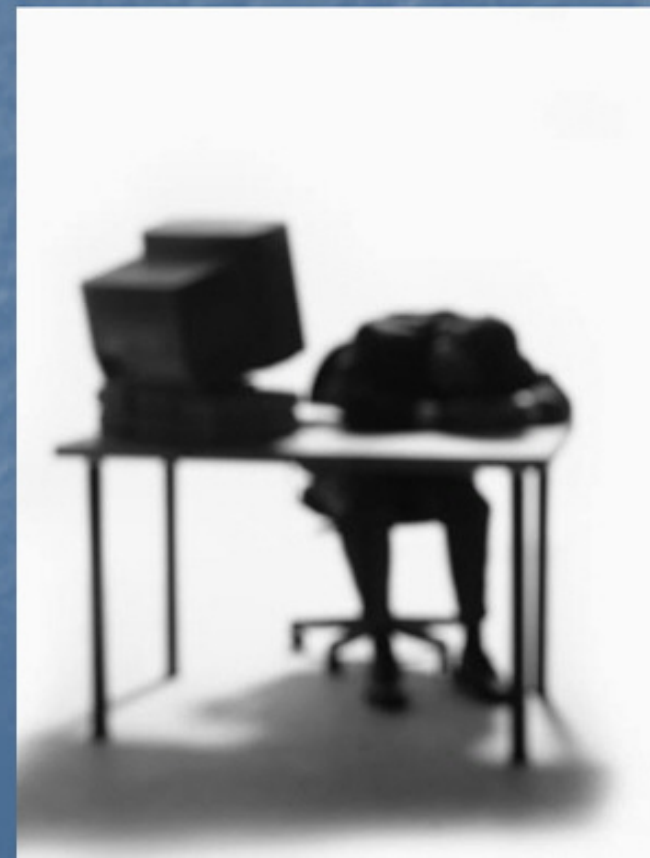
# Dantrolene

- Acts on the muscle
- Decreases the force of muscle contraction
- Studies in children with CP reduction in strength, spasticity lowered, mild or no functional improvement
- Drowsiness less common than with baclofen but still in 25% of children with significant medication to effect their spasticity
- Fatigue, weakness, vomiting, diarrhoea, hepatotoxicity (less than in adults)



# Diazepam

- Benzodiazepine ?facilitates GABA-A receptor neuronally mediated inhibition
- These receptors are located throughout the Central nervous system
- Lethargy more evident than effects on spasticity
- Variation in responsiveness, some intolerable sedation at low doses





# Administration of Diazepam

- Start 0.1-0.2mg per kg per day working up to a max of 0.8mg per kg per day in 2-3 divided doses
- Can be given orally, buccally, nasally, rectally, IM or IV
- Dependence risk



# Other oral medication

- Gabapentin, Pre- gabalin
- Carbamazepine, Vigabatrin
- Clonidine, Orphenadrine
- Clonazepam
- Magnesium



# Cannabinoids





# Spasticity or contracture?

- 50 year old female progressive multiple sclerosis
- Dysphagia, low body weight, pressure ulcer over her ischial tuberosity
- Dysarthria, variable ability to indicate yes/no
- Loss of range of movement at hips and knees
- ? Will need leg amputation as foot stuck under ischial tuberosity
- Pain
- Unable to seat, difficulty managing position in bed



# What did we do?

- Admission to rehabilitation unit
- MDT assessment
- Review of range of movement/ spasticity versus contracture over several days
- Review nutrition- ? Mental capacity, PEG tube inserted
- Trial of baclofen, analgesia for pain
- Review of equipment and care package
- Some spasticity reduced by improvement in general medical condition; Significant underlying contracture
- Has not needed amputation, sits for short periods up to 2 hours. Modular seating system used occasionally.
- Does not like hoist and being transferred out of bed.





# **LESS WRINKLES IN ONLY MINUTES**

• 83% saw less  
wrinkles & fine lines\*



[www.dermitage.com](http://www.dermitage.com)

\* Home-use study of 270





# Botulinum toxin: History

- Protein produced by the anaerobic bacterium *Clostridium botulinum*
- Earliest reports of botulism from the Roman Empire
- First accurate description by Justinus Kerner 1817





# Sources of infection



- One of the most common culprits in food-borne botulism is home-canned food, especially vegetables such as asparagus, green beans, and peppers.



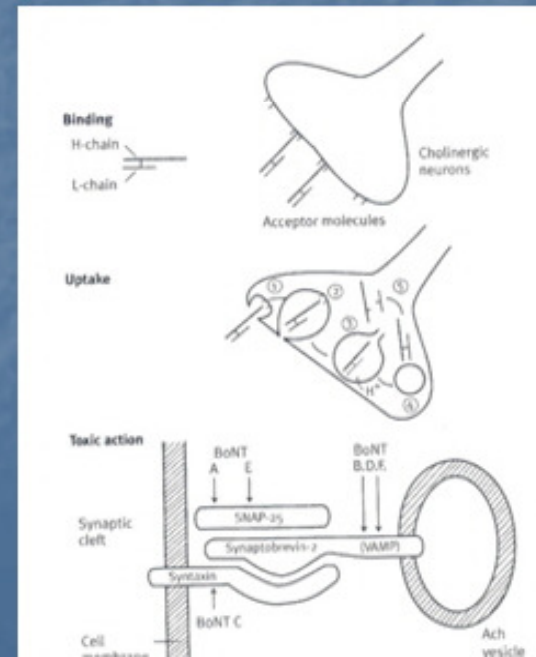
# Uses of botulinum toxin

- 1920s first concentrated into a relatively pure form
- 1940-1970s attempts to develop for biological warfare
- 1970s strabismus in primates
- 1980s strabismus, dystonias, hemifacial spasm in humans
- 1989 spasticity



# Botulinum toxin action

- Only works on the motor system
- No dysaesthesia
- Attaches to the presynaptic neurone
- Inhibits release of Acetylcholine
- Prevents depolarization
- Reversible





# Botulinum toxin A

- Most potent serotype
- Very avidly binds in the muscle
- 2 different preparations available:
  - Dysport produced by Ipsen
  - Botox produced by Allergan



# Botulinum toxin: side effects

- Very well tolerated
- Weakening of injected muscle
- Diffusion into adjacent muscles
- Dysphagia
- Localized autonomic failure



# Why use botulinum toxin instead of tablets?





# Botulinum toxin: patient selection

- Focal spasticity
- Physiotherapy assessment
- Goals of treatment and patient consent
- Define muscles to inject
- Dose per muscle



# Hip adductor spasticity

- 40 year old female progressive multiple sclerosis
- Dysarthria, cognitive impairment
- Enjoys sitting out in group sessions, going out on trips with her mother
- Positioning in wheelchair and carrying out personal care increasingly difficult because of spasticity and contractures



# Intrathecal treatment



- Began in 1960s with use of intrathecal phenol to ablate spasticity and painful muscle spasms of lower extremities in patients with SCI or MS
- Intrathecal phenol also ablates bladder function
- ITB began in Europe in 1985, baclofen was injected into the spinal fluid of a 4 year old child with severe spasticity secondary to near drowning





# Other local treatments

- Phenol
- Lignocaine
- Ethanol



# Other treatments

- Intrathecal phenol
- Ablative surgery- the last option as irreversible- selective lesions in the nerve roots to reduce neural input and reduce flexor and stretch reflexes
- Neural stimulation



# A case of severe spasticity

- 32 year old female, single parent of 2 children
- Spinal cord injury road traffic accident complete T6 injury
- Knees flex, hips flex
- Thrown out of w/c by spasms
- Pressure sore on foot, backache
- Baclofen, dantrolene- too sedating
- Baclofen pump- eroded thro' skin



What can we do next?



# Treatment

- Iliopsoas injection botulinum toxin under ultrasound guidance
- Tenotomies
- Retrial of baclofen pump
- Management of wounds
- Nutrition, smoking advice
- Positioning



# New developments spasticity management.

- Botulinum toxin and electrical stimulation- to enhance the effect of botulinum toxin on upper limb spasticity after stroke
- Electrostimulation



# Conclusions.

- Spasticity can sometimes be useful but can interfere with a range of activities which include the ability to sit comfortably
- There are a range of medical treatments which need to be tailored to the individual's needs
- Treat exacerbating factors
- Use the least invasive treatment first
- Involve the multidisciplinary team



# Thank you

