Post Stroke Spasticity

Charles E. Argoff, MD  
Professor of Neurology  
Albany Medical College  
Director, Comprehensive Pain Center  
Albany Medical Center  
Albany, NY

Disclosures

Nothing to disclose

Educational Objectives

Identify the clinical features of post stroke spasticity  
Discuss pharmacological approaches to the management of post stroke spasticity  
Review invasive approaches to the management of post stroke spasticity
Background
Stroke can be a debilitating disease with increasing prevalence with advancing age.
Prevalence in the US is 2.7%.
It is a leading cause of long-term disability.
Approximately 50% of stroke survivors will experience continued physical or cognitive impairment.
More than 2/3 of stroke survivors receive rehabilitation services after hospitalization including inpatient and/or outpatient services.

Defining Spasticity
Spasticity is a state of increased muscle tone.
Exaggerated reflexes are noted.
A velocity-dependent increase to resistance to passive movement in the affected regions.
It reflects and is part of an “upper motor neuron” syndrome; other features of this type of syndrome include muscle spasm, spastic dystonia, clonus, spastic co-contraction, and as noted above exaggerated deep tendon reflexes.
Abnormal limb positioning/posture is a consequence of these changes.

Post Stroke Spasticity – Clinical Features
Prevalence of post stroke spasticity ranges from 25-43% in year 1 post stroke.
Early spasticity may appear in 4-27% of people during the first 6 weeks after the stroke.
The incidence of upper limb spasticity in one assessment was 33% after 3 months.
Those with strokes involving the basal ganglia and internal capsule had the greatest spasticity incidence.
Post Stroke Spasticity – Clinical Features (continued)

The distribution of spasticity includes elbow (79%), the wrist (66%) and the ankle (66%).

A common pattern of upper limb post stroke spasticity is adduction and internal rotation of the shoulder coupled with flexion at the elbow, the fingers and the wrist.

For lower extremity post stroke spasticity, the most commonly observed pattern is extension and adduction of the knee with equinovarus foot (foot points downwards and inwards).

Post Stroke Development – Predictors

Low Barthel Index (10 item scale that assesses functional measures commonly used to assist in outcome measurement in stroke clinical trials)

Severe proximal and distal limb weakness soon after stroke

Sensory deficits

Location of stroke

Lesion volume

Select Clinical Symptoms/Signs of Post Stroke Spasticity – Flexor and Extensor Muscle Spasms

These muscle spasms are sudden, brief, involuntary muscle contractions due to alpha motor neuron spontaneous firing.

The involved limb may go into flexion or extension.

Urinary and/or fecal incontinence may accompany the spasm.

Usually occur at rest but can be triggered by attempted movement of the affected limb or by sensory stimuli (touch, noise, pain).

These can be painful interrupting sleep or function (trying to walk).
Select Clinical Symptoms/Signs of Post Stroke Spasticity – Spastic Dystonia

This term describes sustained muscle contractions

Results in what we see as the typical postures following a stroke

The upper limb of a hemiplegic person is adducted, internally rotated and flexed at the elbow, wrist and fingers

The lower limb is in extension with the foot often in an equinovarus position

Spasticity Assessment – Modified Ashworth Scale

The Modified Ashworth scale measures the level of resistance to passive movement

0 = no increase in muscle tone
1 = slight increase in muscle tone noted by slight catch or by minimal resistance with end of ROM of affected part
1+ if more than just the end of ROM but less than 50% of ROM
2 = more marked increase in muscle tone through most of ROM but limb can be easily moved
3 = notable increase in muscle tone with passive movement difficult
4 = affected areas rigid in flexion or extension

Spasticity Assessment – Modified Tardieu Scale

The modified Tardieu scale measures the velocity of passive joint movement and the angle of contraction; involves measuring the quality of movement mobilization at 3 different velocities

Quality of movement: 0 = no resistance through passive movement
1 = slight resistance throughout the passive movement – no clear catch at a precise angle
2 = clear catch at a precise angle interrupting the passive movement
4 = unfatigable clonus with movement
5 = joint is fixed

Measured at 3 velocities: as slow as possible, speed of limb segment falling under gravity, and as fast as possible
Treating Post Stroke Spasticity – 4 Clinical Scenarios to Consider

57 yo male with post stroke spasticity affecting primarily dominant upper extremity; stroke occurred 6 weeks ago.

77 yo female with post stroke spasticity affecting non-dominant upper and lower extremity associated with significant weakness and loss of function; she is on multiple medications and is mildly cognitively impaired; stroke occurred 2 years ago.

68 yo female with post stroke spasticity involving dominant upper and lower extremity; unable to use upper extremity due to hemiparesis and spasticity, with AFO can ambulate; stroke occurred 3 year ago; hemisensory deficits noted as well.

36 yo male with post stroke spasticity following traumatic carotid dissection with resulting mild dominant hemiparesis involving upper and lower extremity; 6 months ago.

Treating Post Stroke Spasticity – Overview

Post stroke spasticity is often challenging to treat.

Treatments might include physical and occupational therapy, implementation of the use of adaptive devices, and invasive approaches.

A multidisciplinary approach is frequently required for optimal outcomes.

Spasticity may actually offer significant benefits for patients. Why?

It may be required for the trunk posture maintenance for sitting and standing, allow for weight bearing for the weak leg so that the limb can be braced in extension; in fact the contraction of the spastic muscles may result in the muscle mass maintenance, prevention of DVTs, and prevent bone mineral loss.

Treating Post Stroke Spasticity – Indications for Treatment

Improve volitional goal-directed movements – spasticity of an antagonist muscle may prevent the execution of voluntary movements by the weak limb – EVEN IF WEAKNESS of the agonist muscle is MILD.

Improve passive movement of affected extremities and assist care providers as well; many people with post stroke spasticity may need assistance with personal care as well as specific ADLs.

Relieve spasticity associated pain.

Correct abnormal postures to allow for sitting, placement and use of an orthotic device, to assist in bed positioning and to prevent contractures.

Treating Post Stroke Spasticity – Principles of Treatment Selection

- Is the spasticity focal or generalized?
- Is the spasticity painful or not and is the pain directly related to the degree of muscle contraction?
- What is the clinician’s experience?
- What are the available resources?
- What is the patient’s preference?
- Are there other conditions such as pressure sores, infections, bladder stones, constipation that are present that may aggravate post stroke spasticity?

Treatment: Physical Rehabilitation

- Muscle stretching—including active stretching, passive stretching, prolonged positioning, isokinetic and isotonic stretching—helps to increase soft tissue extensibility, lessen muscle tone and reduce pain related to contractures
- Muscle strengthening
- Various physical modalities such as shock wave therapy, ultrasound, cryotherapy, thermotherapy, hydrotherapy
- TENS (transcutaneous electrical nerve stimulation)
- NMES (neuromuscular electrical stimulation)
- Splinting
- Cast immobilization may be used for short duration treatment

Treatment: Oral Spasmolytic Medications

- Baclofen: GABA agonist which inhibits muscle tone through spinal cord mediated mechanisms; typical doses 20-80 mg/day in divided doses
- Tizanidine: central alpha-2 receptor agonist resulting in inhibition of the activity of excitatory descending corticospinal pathways; typical doses 2-4 mg 3 times daily, max = 36 mg/day
- Dantrolene: peripheral acting agent directly acting on muscle, interferes with release of endoplasmic reticulum inhibiting excitation-contraction coupling, max dose 400 mg/day by mouth
- Diazepam: acts via GABAergic mechanisms to reduce muscle tone, 15-60 mg daily doses
Treatment: Oral Spasmolytic Medications

Additional Considerations

- Not only spastic muscle tone is reduced! Generalized weakness and loss of function can occur.
- Doses may need to be frequently adjusted.
- Potential for drug interactions: baclofen and tizanidine can increase the hypotensive effects of ACE inhibitors, B-blockers, calcium channel blockers, and diuretics.
- Consider the sedating effect of baclofen, tizanidine, and diazepam.
- Monitoring LFTs is important for each.
- Consider avoiding use of these if the spasticity is localized.

Specific Adverse Effects

Baclofen: confusion, hallucinations, seizures, fatigue, GI symptoms
Tizanidine: hypotension, tachycardia, hallucinations, confusion, fatigue
Dantrolene: liver function abnormalities, dizziness, generalized weakness, GI symptoms
Diazepam: sedation, amnesia, confusion, depression, respiratory depression, physical dependence, behavioral dependence

Beneficial effects may last for 3-4 months so... treatment is 3-4 times/year.

Treatment: Botulinum Toxin

Both preparations of type A botulinum toxin (onabotulinum toxin A, incobotulinum toxin A, and abobotulinum toxin A) and type B botulinum toxin B (rimabotulinum B) can be used to treat focal as well as multifocal post stroke spasticity.

Although spread of the toxin can occur, muscle tone reduction is largely confined to the muscle(s) injected.

Multiple clinical trials have been completed and published guidelines support the use of botulinum toxin for post stroke spasticity.

Beneficial effects may last for 3-4 months so... treatment is 3-4 times/year.
Treatment: Botulinum Toxin (continued)
Depending upon the muscle group(s) to be injected can be completed using anatomical landmarks or with EMG guidance. Specific muscles, for example, the hamstrings, may require multiple injections (depending upon the orientation number of motor endplates). Optimal dose depends upon muscle size, how spastic the muscle is and how much spasticity reduction is desired.

BAKHEIT AM. THE PHARMACOLOGICAL MANAGEMENT OF POST-STROKE MUSCLE SPASTICITY DRUG AGING 2012; 29:941-947

Select muscle groups that might be treated with botulinum toxin for post stroke spasticity

- Shoulder abductors: pectoralis major +/- subscapularis
- Elbow flexors: biceps brachii and brachioradialis
- Wrist flexors: Flexor carpi ulnaris and flexor carpi radialis
- Hamstrings: medial and lateral hamstrings
- Ankle/dorsiflexors and invertors: gastrocnemius +/- tibialis posterior

Treatment: Intrathecal Baclofen
Baclofen is also indicated for use in the management of severe spasticity of cerebral or spinal origin in adult and pediatric patients age 4 years and above.

Baclofen taken orally eventually exerts its mechanism of action in the spinal cord. In contrast, intrathecal baclofen involves direct administration of baclofen into the CSF and therefore more directly into the spinal cord. Typical starting doses of IT baclofen are 50 mcg/day – far lower than po baclofen. Patients receiving IT baclofen often experience far less sedation than with po baclofen.
Treatment: Anticonvulsants/Cannabinoids

Off label use of pregabalin and gabapentin has been noted.
The adverse effects of pregabalin and gabapentin include sedation, dizziness, peripheral edema, and weight gain.
The evidence for the antispasticity effect of is noted for patients with multiple sclerosis; however, similar evidence has not been noted for post stroke spasticity.

Adverse effects of cannabinoids include cognitive impairment, paranoid delusions, panic attacks, tachycardia, and postural hypotension.

Neither anticonvulsants nor cannabinoids have been studied systematically in post stroke spasticity.

Treatment: Peripheral Nerve Blocks

Chemical neurolysis is the process by which destruction of peripheral nerves with alcohol or phenol may occur.

Peripheral nerve blocks with alcohol or phenol injection can be helpful for some patients for months at a time without causing significant weakness.

These can be completed relatively easily in a cost-efficient manner.

However, sensory loss and painful paresthesias following treatment can occur as can loss of manual dexterity in the upper extremities.

Fibrosis of the nerve to be injected may occur after multiple treatments.

Treating Post Stroke Spasticity — 4 Clinical Scenarios to Consider

57 yo male with post stroke spasticity affecting primarily dominant upper extremity; stroke occurred 6 weeks ago.

77 yo female with post stroke spasticity affecting non-dominant upper and lower extremity associated with significant weakness and loss of function; she is on multiple medications and is mildly cognitively impaired; stroke occurred 2 years ago.

68 yo female with post stroke spasticity involving dominant upper and lower extremity, unable to use upper extremity due to hemiparesis and spasticity, with AFO can ambulate; stroke occurred 1 year ago; hemisensory deficits noted as well.

36 yo male with post stroke spasticity following traumatic carotid dissection with resulting mild dominant hemiparesis involving upper and lower extremity; 6 months ago.
57 yo male with post stroke spasticity affecting primarily dominant upper extremity; stroke occurred 6 weeks ago

- What are important features of the history to guide treatment?
- Are there any comorbid conditions that would guide treatment?
- What treatments have been trialed? Success?
- What are the overall treatment goals including likelihood of meaningful functional restoration?
- What patient specific issues arise for this person that could guide treatment choice?
- What type of monitoring and follow up will be needed and what is practical?

77 yo female with post stroke spasticity affecting non-dominant upper and lower extremity associated with significant weakness and loss of function; she is on multiple medications and is mildly cognitively impaired; stroke occurred 2 years ago

- What are important features of the history to guide treatment?
- Are there any comorbid conditions that would guide treatment?
- What treatments have been trialed? Success?
- What are the overall treatment goals including likelihood of meaningful functional restoration?
- What patient specific issues arise for this person that could guide treatment choice?
- What type of monitoring and follow up will be needed and what is practical?

68 yo female with post stroke spasticity involving dominant upper and lower extremity, unable to use upper extremity due to hemiparesis and spasticity, with AFO can ambulate; stroke occurred 1 year ago; hemisensory deficits noted as well

- What are important features of the history to guide treatment?
- Are there any comorbid conditions that would guide treatment?
- What treatments have been trialed? Success?
- What are the overall treatment goals including likelihood of meaningful functional restoration?
- What patient specific issues arise for this person that could guide treatment choice?
- What type of monitoring and follow up will be needed and what is practical?
36 yo male with post stroke spasticity following traumatic carotid dissection with resulting mild dominant hemiparesis involving upper and lower extremity; 6 months ago

What are important features of the history to guide treatment?
Are there any comorbid conditions that would guide treatment?
What treatments have been trialed? Success?
What are the overall treatment goals including likelihood of meaningful functional restoration?
What patient specific issues arise for this person that could guide treatment choice?
What type of monitoring and follow up will be needed and what is practical?

Conclusions
Post stroke spasticity frequently occurs and should be anticipated and assessed
Individualized treatment should focus on a multidisciplinary approach recognizing the practical limitations regarding access and other issues
Post stroke spasticity management is dynamic and may require changing treatments as well as doses of treatments prescribed as needed