Chemodenervation With Botulinum Toxins & Phenol For Spasticity & Dystonia

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Objectives

For patients who have dystonias and spasticity:
• Be able to assess their abnormal movements; and
• Be able to treat the abnormal movements, especially with nerve blocks and botulinum toxin muscle injections.
Retrocollis involving: B/L SCM & Posterior Paracervical Muscles.

A. Alfaro, Spasticity & Dystonia: Dx & Tx.
25yr woman after an MVC on 12/23/17
Pre- & Post-Injections: Bupivacaine; then Phenol to Spinal Accessory Nerve
CERVICAL DYSTONIA: TREATMENT WITH PHENOL TO SPINAL ACCESSORY NERVES
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• INTRODUCTION: Cervical dystonia (CD) was treated with botulinum toxin or phenol motor point blocks; however, phenol injections to spinal accessory nerves (SAN) were not reported.

• OBJECTIVE: To assess the effects of phenol blocks to SAN on head rotation. Since SAN innervate sternocleidomastoid and upper trapezius muscles, a block of the ipsilateral SAN should increase head rotation ipsilaterally.

• METHODS: For CD, 13 patients had a total of 17 phenol injections to SAN. For patients with torticollis, three had left SAN blocks, and one had a right SAN block. SAN were identified at the proximal 1/3 of the sternocleidomastoid muscle; when stimuli <1.0 mA produced strong contractions of sternocleidomastoid and/or upper trapezius muscles, phenol 5% was injected via a hypodermic needle. Goniometry for head rotation was recorded pre- and post-injections.

• RESULTS: For 3 patients, head rotation was initially left 21.7, and right 59 degrees; and post-injection left 44.6 and right 55.1 degrees. Post-injection, head rotation left increased 23.2 degrees (107%) and right was -3.9 degrees (-6.6%). Phenol injected averaged 1.2 ml/patient at a stimulus of 0.5mA. One patient had a right SAN phenol block with 0.8 ml at 1 site; head rotation increased right 49% and left 24%. Post-injection, 2 of 17 injections caused upper trapezius weakness.

• CONCLUSIONS: For patients who have CD with torticollis, head rotation increased in the direction of the SAN that was blocked with phenol without a significant decreased contralateral head rotation. This is the first report of phenol SAN blocks; more data will determine the role of phenol SAN blocks for the treatment of CD.
Oromandibular Dystonia: Jaw closed for 4 years despite B/L masseter muscle injections with onabotulinumtoxinA 150, 200 & 300 Units in total @ 3 month intervals.
Oromandibular Dystonia: Jaw closed for 4 years

9/18/14 Pre- & Post- Phenol Injections to Masseters, Medial Pterygoid & Temporalis Muscles Bilaterally

54 yr Man
4/1/17 bilateral pontine & cerebellar infarcts.
Basilar artery thrombosis & S/P endovascular thrombectomy.
Unable to open right side of mouth.

EMG Right Masseter muscle at rest.

EMG right masseter during jaw opening

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Pre and Post-Bupivicaine Injections to Right Masseter Muscle

Dx Post-Stroke: Right nasolabial fold weakness due to CN VII lesion; and dystonia of right masseter muscle that is innervated by CN V.
DYSTONIAS

- Associated with lesions in the basal ganglia, but can involve lesions in the cerebellum and brainstem.
- Includes cervical dystonia (torticollis, laterocollis, anterocollis, retrocollis), hemifacial spasm, blepharospasm, oromandibular dystonia, and limb dystonia.
- About 1 per 10 cases are associated with “trauma.”
- Average of 5 years before diagnosed, and 2 years before treated.
- Dx with PE & EMG. EMG with continuous or bursts of MUP, and is NOT velocity dependent with muscle stretching.
- Dystonias can be due to genetic disorders, especially for younger patients. Carbidopa/levodopa may be Tx.
For spastic muscles: EMG shows motor unit potentials at rest, and more so with stretch.

- When stretching muscles, intrafusal fibers (muscle spindles) are stimulated & send impulses via Ia afferents to AHC.
- AHC then send efferent impulses via alpha motoneurons to muscles, & via gamma fibers to intrafusal fibers.
- Phenol blocks Ia afferents, gamma efferents, and alpha motoneurons.
- Muscles then contract.
Spastic Elbow Flexor Muscles

**PE:** No volitional movement; with passive stretch a firm brachioradialis & MAS 3/4; MAS 4/4 at about -45 degrees elbow extension with a contracture likely at that joint angle; spastic muscles are BR, BB & Brachialis; skin breakdown with blisters. **Tx:** oral medications; injections with botulinum toxin to muscles or phenol to nerves; stretch & splint?
Modified Ashworth Scale

to assess spasticity via resistance to passive muscle stretch.

• 4 Very Severe; fixed end point.
• 3 Severe resistance to stretch
• 2 Moderate resistance to stretch
• 1+ Mild with a “catch”
• 1 Mild
• 0 No resistance to stretch

• Ashworth
Spastic Elbow Flexor Muscles

Modified Tardieu Pendulum Test
Record:
1. the elbow angle when released against gravity;
2. then note Ashworth Score.

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Injection to Musculocutaneous Nerve To Decrease Spasticity For Elbow Flexors
What muscles also flex the elbow & are innervated by the radial nerve & median nerve?
ABSTRACT:
Objectives: To assess changes in elbow flexor spasticity after phenol injections to musculocutaneous nerves (MCN), and to identify post-injection complications.
Design: A retrospective assessment of phenol injections to MCN for patients who have elbow flexor spasticity due to central nervous system disorders. For a subgroup of post-stroke patients, elbow flexor spasticity was assessed with a Modified Ashworth Scale (MAS) from 0 (no spasticity) to 4 (severe spasticity) pre- and post-injections, and analyzed with a t-test.
Setting: Outpatient clinic in a rehabilitation hospital.
Participants: Men and women who have spasticity for elbow flexor muscles.
Interventions: Injections to MCN with Phenol 5% in sterile water.
Main Outcome Measures: Change in spasticity with a MAS (0-4), dose of phenol injected, number of sites injected, electrical current (mA) to stimulate MCN, and complications due to injections.
Results: For 196 MCN injections with phenol, 106 injections were for stroke patients (48 women and 58 men) and their data (mean + standard deviation) was: age 63.6 + 12.9 years for men and 68.0 + 13.7 years for women; 0.54 + 0.16 mA to stimulate the MCN for injections; 1.3 + 0.56 sites injected; 0.67 + 0.53 ml phenol injected; and MAS pre-injection 3.2 + 0.3, and post-injection 0.9 + 0.7. The MAS post-injection was significantly less (p<0.05) than pre-injection with a t-statistic 28.1. Of 196 MCN injected with phenol, one patient had transient numbness in the distribution of the lateral antebrachial cutaneous nerve.
Conclusions: Phenol injections to MCN are an effective, safe and inexpensive treatment to decrease elbow flexor spasticity for post-stroke patients.
Intrinsic Finger Flexor Spasticity

- Adducted fingers.
- Ulnar nerve innervates palmer interossei.
- Ulnar & median nerves innervate FPB.
Post-phenol injection to ulnar nerve:

Stage 2 decubitus

Stage 3 decubitus
“Clenched Fist” for a 34 year old woman who had a stroke in 2013 with right spastic hemiparesis.

8/7/17

A. Alfaro

A. Alfaro
Bupivacaine Injections To The Anterior Interosseus Nerve Decreased Spasticity For The Flexor Digitorum Profundus Muscle
Since EMG shows evidence for spasticity for finger flexor muscles, what should we do initially?

1. Nothing.
2. Rx P.O. Baclofen &/or Tizanidine.
3. Rx a splint &/or place an object in her hand to prevent finger flexion.
4. Rx exercises to stretch finger flexor muscles.
5. Inject botulinum toxin into finger flexor muscles (i.e. FDS & FDP).
6. Diagnostic block of anterior interosseus nerve. If fingers then extend, inject phenol onto the AIN or botulinum toxin into FDP muscle.
Oral Baclofen


Abstract

A **double blind crossover trial of baclofen against placebo in elderly stroke patients was discontinued because the drug produced an unacceptably high level of drowsiness.** In a subsequent study baclofen 10 mg was given orally to 12 elderly stroke patients, and drug concentrations measured from a series of plasma samples. A group of healthy subjects given the same dose in a previous study were used as controls. Elderly patients took longer to achieve peak plasma baclofen concentrations, but healthy controls had higher peak values and eliminated the drug more rapidly; areas under the curve were similar in the two groups. Simulations based on mean data suggest that increased drowsiness in the elderly was probably not due to changes in the drug's pharmacokinetic behaviour.

- N=80 patients; stroke >>TBI
- Independent variables: BoNT; TZD; placebo.
- Dependent variables: wrist & finger flexor spasticity:
  - MAS > 3 pre-Treatment for 3 groups.
  - Disability Assessment Scale (DAS): to assess dressing; hygiene; cosmesis; pain; or combination of these.

- Adverse Events
  - Somnolence: 4 = 20% 10=47.6% 2=10.5%
  - Tired/fatigue: 4 = 20% 2 = 9.5% 2=10.5%
  - Liver enzymes: 0 2= 9.5% 0

• **MAS wrist flexors:**
  - Wk 3 BoNT > TZD=Pl (-1.55, -0.25, -0.67).
  - Wk 6 BoNT > TZD=Pl (-1.32, -0.22, -0.68).

• **MAS finger flexors:**
  - Wk 3 BoNT > TZD=Pl (-1.45, -0.65, -0.17)
  - Wk 6 BoNT > TZD=Pl (-1.37, -0.39, -0.26)
  - Higher dose of BoNT produced > MAS.

• **DAS:** wk 6 BoNT vs TZD p=0.2; DAS cosmesis domain
  BoNT > TZD=PL (-1, +0.12, -0.16) p<0.003
Comparisons of Oral Medications


• If using oral spasmolytics, consider the following:
  – All have side effects (e.g. weakness, sedation);
  – TZD is less effective than onabotulinumtoxinA for ULS.
  – Muscle weakness for baclofen > TZD.
  – TZD & clonidine are centrally acting alpha 2 adrenergic agonist; do not use together; both may lower BP.
  – Check liver enzymes when using TZD, baclofen or dantrolene.
Hip Adductor Spasticity Ashworth 3+/4 for a patient who has Multiple Sclerosis
Hip Adductor Spasticity: Ashworth 0/4
Spastic Paraplegia

- PE: describe her gait.
  - Ankle plantar flexed?
  - Foot drop?
  - Ankle inverted?
  - Stiff knee?
  - Hip hiking?
  - Narrow base of support (hip adducted)?
Spastic Paraplegia: Post-Injections

- To decrease spasticity and improve gait, injections included:
  - Obturator nerve to widen base of support;
  - Tibial nerve to ankle plantar flexors;
  - Tibialis Posterior muscle.
ABSTRACT:
Objectives: To assess changes in hip adductor spasticity after phenol injections to obturator nerves (ON), and to identify post-injection complications.
Design: A retrospective assessment of phenol injections to ON for patients who have hip adductor hypertonicity due to central nervous system disorders. For a subgroup of post-stroke patients, spasticity was assessed with a Modified Ashworth Scale (MAS) from 0 (no spasticity) to 4 (severe spasticity) pre- and post-injections, and analyzed with a t-test.
Setting: Outpatient clinic in a rehabilitation hospital.
Participants: Men and women who have hypertonicity for hip adductor muscles.
Interventions: Injections to ON with Phenol 5% in sterile water.
Main Outcome Measures: Change in spasticity with a MAS (0-4), dose of phenol injected, number of sites injected, electrical current (mA) to stimulate ON, and complications due to injections.
Results: For 391 ON injections, 376 were with phenol and 15 with lidocaine. This included 62 stroke patients (29 women and 33 men), who had a total of 74 phenol injections, the mean + standard deviation was: age 69.6 + 14.1 years; 0.54 + 0.13 mA to identify ON; 1.5 + 0.67 sites injected; 1.18 + 0.82 ml injected; and MAS pre-injection 2.9 + 0.62, and post-injection 0.67 + 0.70. The MAS post-injection was significantly less (p<0.05) than pre-injection with a t-statistic 20.47. Of 376 ON injected with phenol, no patient had paresthesias in the distribution of the ON; one patient had transient numbness at the medial leg in the area of the saphenous nerve.
Conclusions: Phenol injections to ON are an effective and safe approach to decreasing hip adductor spasticity for post-stroke patients.
Post-Stroke Abnormal Gait with Ankle Clonus, a Foot Drop & Genu Recurvatum
Post-stroke Foot Drop, Genu Recurvatum & Sustained Ankle Clonus

MMT: 0/5 ankle DF; 4/5 KE; 3+ HF.

• Tx for sustained ankle clonus?
  – Do nothing.
  – Rx oral tizanidine or baclofen.
  – Inject botulinum toxin into gastrocnemius & soleus when she is an outpatient.
  – Inject Phenol onto tibial nerve to ankle plantar flexors.

• Which orthotic should be Rxed for her foot drop & genu recurvatum?
  – KAFO
  – AFO with double metal uprights.
  – AFO with tamarac hinges to DF the ankle, & a posterior pin to control plantar flexion.
Ankle Foot Orthosis (AFO): Stabilize Ankle; Dorsi-flexor.

Off the shelf AFO

Custom Made AFO
Stance Phase Pre- & Post-Lidocaine to Soleus & Gastrocnemius Muscles
Post-Stroke Abnormal Gait with a Genu Recurvatum
Post-Stroke Genu Recurvatum & Foot Drop with a MAFO and Heel Lift

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Post-Stroke Genu Recurvatum: AFO with Tamarac Hinges and a Heel Lift

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TBI with equinovarus deformity. Why no clonus?

Treatments? Chemodenervate muscles? Phenol nerve blocks?

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Post-Injections to Tibial Nerve to Ankle Plantar Flexors & Tibialis Posterior Muscles

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Prevalence of Spasticity

- 65% Spinal Cord Injury
- 23% Traumatic Brain Injury
- 58% Stroke (52% have ≥ 1 joint contracture)
- 84% Multiple Sclerosis
- 73% Adult Cerebral Palsy
Are stroke patients treated for spasticity?

- Up to 79% of stroke patients in rehabilitation have upper limb spasticity.
- About 58% were treated for spasticity.

MOA

• Phenol: blocks la afferents & gamma efferents (to “sedate” intrafusal muscle fibers); blocks alpha motor neurons.
  – Onset: immediate. Duration: Weeks to years.
• Botulinum Toxins: Block the release of Ach.
  Onset: about two weeks. Duration: 3 months.
Why Treat Spasticity?

• Improve function.
  – Use of limb;
  – Transfer &/or walk better.

• Improve hygiene.

• Improve appearance (cosmesis).

• Prevent or reduce pain & contractures.
  – Contractures are iatrogenic.
Orthopedic Surgery

- Treat spasticity before doing elective surgery, e.g. TKR or THR.
- Surgery is a stressor that increases.
- The post-operative increase in spasticity will impair function/recovery.
- For ankle plantar flexion spasticity, do a gastrocnemius resection. Do not do tendon achilles lengthening (TAL).
Algorithm-Based Therapy for Spasticity & Dystonia

• PE to identify SAD.
• Treatments.
  – Phenol nerve blocks to improve function, prevent contractures, reduce pain.
  – Botulinum toxins.
  – Oral Medications (tizanidine, baclofen, dantrolene); Cannabis, Marijuana; ITB; surgery (tendon lengthening &/or transplants).
  – Neurotomy.
Thanks for not duplicating my slides

The slides in this presentation are the property of Dr. Abraham Alfaro. They have been presented in prior lectures and publications, and will appear in a book expected to be published in 2019.