Microvascular Decompression for Hemifacial Spasm

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Hemifacial Spasm (HFS)

- Hemifacial spasm is a condition in which an individual experiences intermittent and involuntary twitching of the facial muscles on one side of the face. This can involve the muscles around the eye (orbicularis oculi), mouth (orbicularis oris), cheek, or neck (platysma).
- Twitching may begin around the eye and then over time come to involve other muscles on that side of the face and neck.
- Twitching may increase under stress, after hyperventilation, after forcibly closing the eye and smiling

Hemifacial Spasm

- Typical HFS involves the eye first and then may spread over time to involve the cheek, mouth, and neck
- Atypical HFS involves the mouth or cheek first and then involves the eye.
- Tonus with HFS involves a continuous muscle contraction that causes the eye on one side to close, the mouth to pull upward on one side, the neck muscles to contract or all three at the same time.

Differential Diagnosis

- Hemifacial spasm
 - Intermittent contraction/spasm
 - May persist while asleep
- Facial myokymia
 - Continuous facial spasm seen with brainstem gliomas and multiple sclerosis
- Blepharospasm
 - Bilateral eye closure/twitching
 - Often seen in elderly and may go away when examined

Evaluation and Diagnosis

- Physical examination and history
- MRI with and without contrast to check for neoplasm, AVM, anomalous vasculature
- Facial nerve EMG to check for lateral spread
- Lateral spread is an abnormal electrical interaction such that stimulation of the orbicularis oculi allows for recordings coming from the orbicularis oris. This only exists iin the face of arterial compression
- Hearing test

Etiology

- Hemifacial spasm is secondary to arterial and/or venous compression of CN 7 (facial nerve) as it exits the brainstem.
- The exit site from the brainstem is actually located caudal to CN 8 origin from the brainstem and just medial to CN 9 origin from the brainstem
- Arterial compression may be from the anterior inferior cerebellar artery (AICA), posterior inferior cerebellar artery (PICA), vertebral artery (VA), small arterioles, small brainstem veins

Etiology

 It is imperative to understand that compression of the nerve itself beyond the cranial nerve brainstem exit zone rarely is responsible for the hemifacial spasm

Treatment

- Microvascular Decompression (<u>see video</u> under video section of this web site to watch a case and to hear about surgical details)
- Botox injections into involved muscles
 - Serves to temporarily paralyze the muscles to reduce or eliminate twitching
 - Must be repeated every few months as Botox effect on the muscle's motor endplate wears off
 - May permanently weaken the muscle
 - May make recordings of lateral spread more difficult

Risks (not all inclusive)

- Stroke (<5%)
- Death (<1%)
- Unilateral hearing loss (4%) or reduction (10-15%)
- New facial paralysis or weakness (<5%)
 - Many patients present with facial weakness at the time of surgery secondary to facial nerve damage from arterial compression or from Botox use.
 - Often this weakness is difficult to identify due to increased baseline facial muscle tone due to the hemifacial spasm and CN irritation
 - After surgery when the nerve is finally at rest and baseline muscle tone is reduced, the underlying weakness can become apparent
- Difficulty swallowing (<5%)
- Double vision (<2%)
- Facial numbness (<1%)
- Infection (<5%)
- Spinal fluid leak (<5%)

Benefits

- Cure rate >85%
- Recurrence rate 10-15% over 5 years
- Need for two operations during same hospital admission to cure spasm (10%)