Microvascular decompression of cranial nerves

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Nothing to declare
Objectives

• Summarize the indications
• Describe the procedure
• Apply established monitoring techniques
• Consider investigational methods
Indications for microvascular decompression

• Medically intractable paroxysmal cranial nerve disorders attributable to compression by an aberrant blood vessel loop
• Mainly *hemifacial spasm* or *trigeminal neuralgia*
• Rarely glossopharyngeal neuralgia, paroxysmal vertigo or tinnitus
Hemifacial spasm

• Unilateral attacks of painless but disfiguring irregular clonic facial muscle contractions
• Typically starts in the orbicularis oculi and spreads
• Spontaneous or triggered by facial movements

(www.seattleneurosciences.com)
Trigeminal neuralgia

- Unilateral attacks of severe lancinating facial pain
- Often V2 or V3 distributions
- Spontaneous or triggered by facial touch, chewing, or brushing the teeth
Pathology and pathophysiology

• Usually due to focal nerve compression and demyelination near the root entry zone, but may be caused by central demyelination instead

• Most often attributed to a tortuous artery or vein after excluding aneurysm, tumor, and multiple sclerosis

• Pathophysiologic theories:
  • Ephaptic transmission between demyelinated axons
  • Nuclear hyperexcitability
  • Spontaneous ectopic firing of injured axons
facial nerve
demyelinating lesion

ephaptic transmission
ectopic axonal firing

hyperexcitable nucleus
Treatment

• Medical
  • Carbamazepine, baclofen, gabapentin
  • Botulinum toxin injections
  • Can be sufficient
• Surgery if intractable
Microvascular decompression

- “Jannetta procedure”
- Retromastoid craniectomy
- Microsurgical dissection to expose the nerve and identify the offending vessel
- Mobilize the vessel and insert Teflon pads between it and the nerve
- Hemostasis and closure
Success rates

- Trigeminal neuralgia: 70% long-term cure. Some early successes relapse.

(Barker, et. al, NEJM 1996;334:1078–83)
Neurological complications

• Cranial nerve injury
  • Facial numbness
  • Facial paralysis
  • Hearing loss
  • Extraocular muscle palsy

• Rare
  • Brainstem or cerebral infarction
  • Death
Intraoperative monitoring

• Cranial nerve protection
  • Brainstem auditory evoked potentials (BAEPs)
    • May reduce hearing loss from 8–20% to <2%
  • Free-running and triggered EMG
  • Corticobulbar MEPs
  • Possibly blink reflexes, trigeminal SEPs

• Optimize facial nerve decompression
  • Lateral spread response (LSR)
  • Investigational methods
    • Facial MEPs
    • Other techniques
Lateral spread response (LSR)

• Developed by Møller and Jannetta
• Stimulate one facial nerve branch
  • Normal direct muscle response
  • Abnormal muscle response from another branch
• Ephaptic transmission or nuclear hyperexcitability
• Usually present before and absent after surgery
• Can acutely disappear *during* surgery
  • May identify the offending blood vessel(s)
  • May signify successful decompression
stimulate temporal branch before decompression after

frontalis

mentalisis

Established technique

- Temporal or zygomatic branch stimulation (mandibular branch less effective)
- Cathode proximal, anode 1 cm distal
- Record o. oculi, o. oris, mentalis
- Find best LSR between the orbit and ear canal with 5 Hz 0.1 ms 2–5 mA pulses
- Monitor with 1–2 Hz pulses 20–30% above LSR threshold
- Brief 50 Hz facilitations
• Can suggest effective decompression
• However:
• LSR may be unobtainable
• May disappear before decompression
• May persist after decompression with same or variably lower amplitude
• Influenced by anesthesia, neuromuscular blockade
• Some reports support correlation to early outcome
• Conflicting reports for long-term outcome correlation
• Remains controversial

Possible improvement

- Preoperative facial nerve mapping to find best LSR (usually the temporal branch)
- Cathode distal, anode proximal at mandibular fossa (‘centrifugal’)
- 0.3 ms 5–25 mA pulses
- Record frontalis, o. oculi, o. oris, mentalis
- Initial report suggests greater success and outcome correlation

Facial MEPs

• Presumably due to facial nucleus hyperexcitability:
  • Lower MEP threshold than the non-spasm side
  • Single-pulse MEPs can occur

• Possibly signifying adequate decompression:
  • Threshold elevation?
  • Amplitude reduction?
  • Single-pulse MEP disappearance?

• Needs further study
C3–Cz or C4–Cz

Suggests nuclear hyperexcitability

Could attenuation or disappearance indicate adequate decompression?

Not found in all patients

before decompression

C3–Cz or C4–Cz

after

true decrease?

Orbicularis oculi
Mentalis
Orbicularis oris
Pollicis

Other investigational techniques

- Blink reflex
  - Pulse trains can elicit blink reflexes under anesthesia
  - Could be exaggerated with hemifacial spasm
  - Might attenuate or disappear with decompression
- Blink reflex synkinesis
  - Abnormal lower facial muscle response
  - Might attenuate or disappear with decompression
- Facial F-waves
  - Could be exaggerated with hemifacial spasm
  - Might attenuate or disappear with decompression
Normal awake blink reflexes to supraorbital nerve stimulation

Pulse trains can evoke R1 but not R2 under anesthesia
Blink reflex synkinesis with hemifacial spasm

During surgery:

Would pulse trains evoke R1 synkinesis in o. oris or mentalis muscles?

If so, might synkinesis disappearance signify decompression?

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