

- The vast majority of Nobel Prize winners in the sciences hold a PhD or its Medical equivalent.
- However, the Nobel Foundation does not require any specific academic degree to win the prize; awards are based on the impact of the discovery and its contribution to mankind rather than to formal credentials.

- I don't claim to have that kind of expertise. However, I do have the following credentials:
- Fellow of the American College of Anesthesiology: FACA
- Diplomate of the American Board of Anesthesiology: DABA
- Doctor of Osteopathic Medicine: DO
- Which makes me a **faca-daba-do**

MICROVASCULAR DECOMPRESSION OF THE TRIGEMINAL NERVE FOR THE TREATMENT OF TRIGEMINAL NEURALGIA

The Jannetta Procedure

Peter Joseph Jannetta (April 5, 1932 – April 11, 2016) was an American Neurosurgeon known for devising microvascular decompression a surgical procedure to treat trigeminal neuralgia. He was the first Walter Dandy Professor of Neurological Surgery at the University of Pittsburgh School of Medicine. Jannetta suffered a head injury after a fall in his home in Pittsburgh and died on April 11, 2016, six days after his 84th birthday.

HISTORY

History

- Tic Douloureux commonly known as Trigeminal Neuralgia, ranks among the oldest recognized neurologic pain syndromes. Its history extends beyond 2000 years, moving from ancient accounts of severe facial pain to modern treatment of neurovascular compression.
- The French term tic douloureaux translates to “painful twitch” which describes an involuntary facial contraction accompanied by intense stabbing pain.

History 2

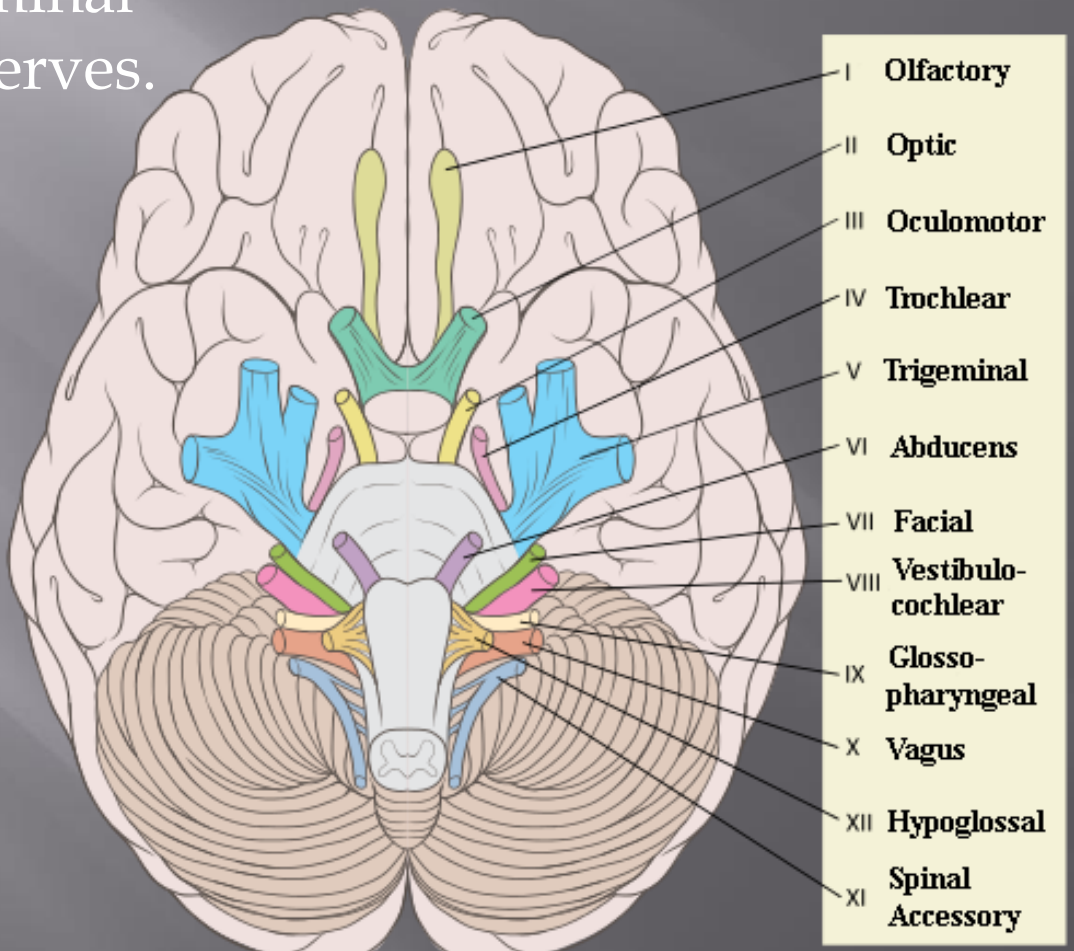
- Early descriptions of this syndrome can be traced to the physician **Aretaeus** (Ἀρεταῖος) of Cappadocia in the 2nd century AD.
- In 1677 the key early clinical account of trigeminal neuralgia was described by the English physician and philosopher **John Locke** (1632-1704)
- John Locke also provided the foundational political and moral theory behind the American evolution in his work "Two Treatises of Government "of 1689.
- In 1891, **Sir Victor Horsely** introduced intracranial surgery for trigeminal neuralgia.
- **Dr. Walter Dandy** made a major discovery during posterior fossa surgery in the 1920's. He noticed blood vessels pressing on the trigeminal nerve near the brain stem and suggested in 1932 that vascular compression might be the cause of trigeminal neuroglia.
- Then in the 1960's , **Peter J. Jannetta** used the operating microscope to show vascular compression of the trigeminal nerve and created modern microvascular decompression.

ANATOMY

Cranial Nerve Roots

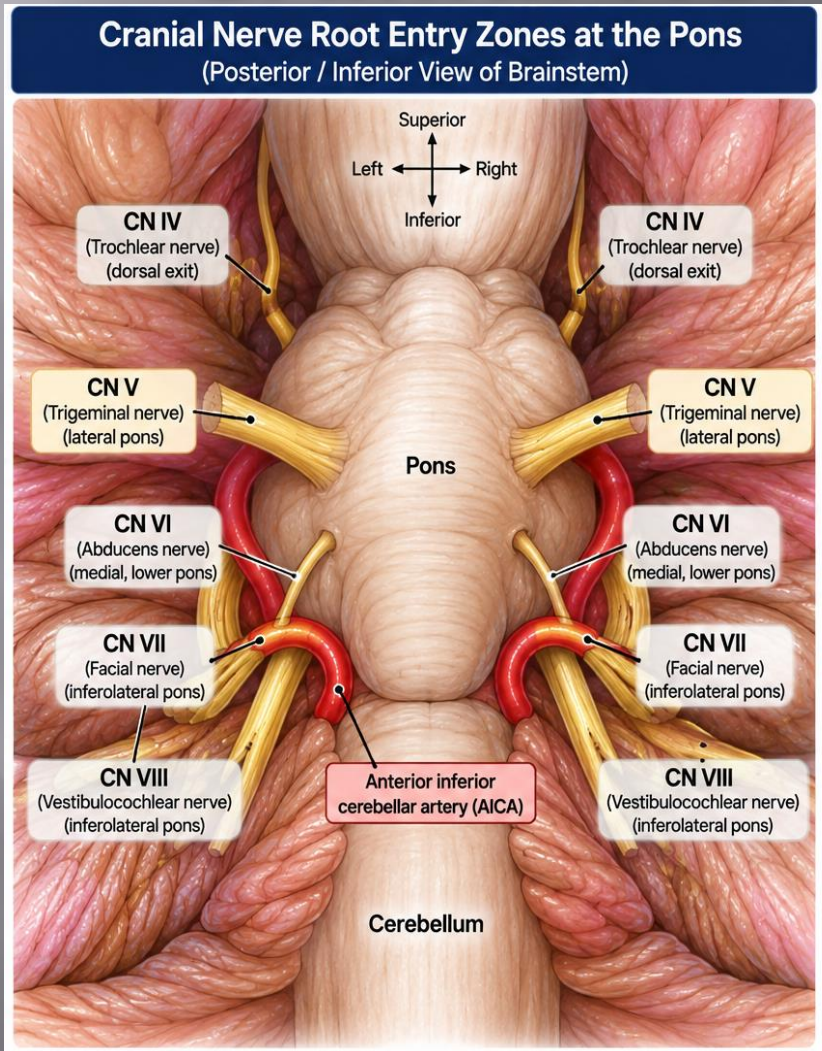
Cranial Nerves

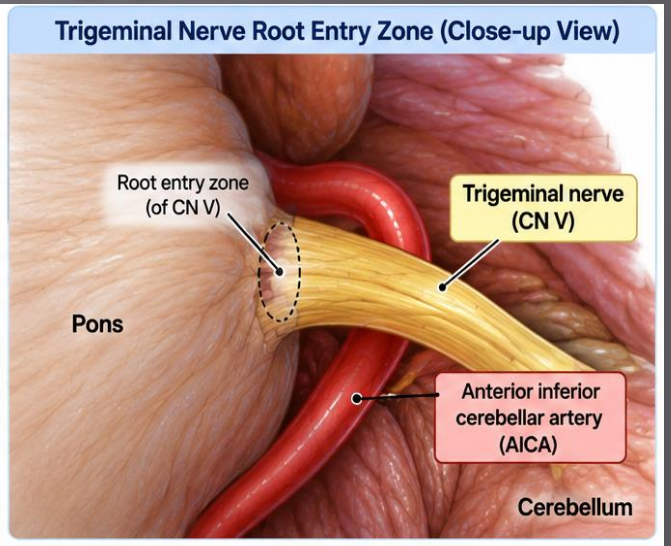
Notice the closeness of the trigeminal and facial nerves.

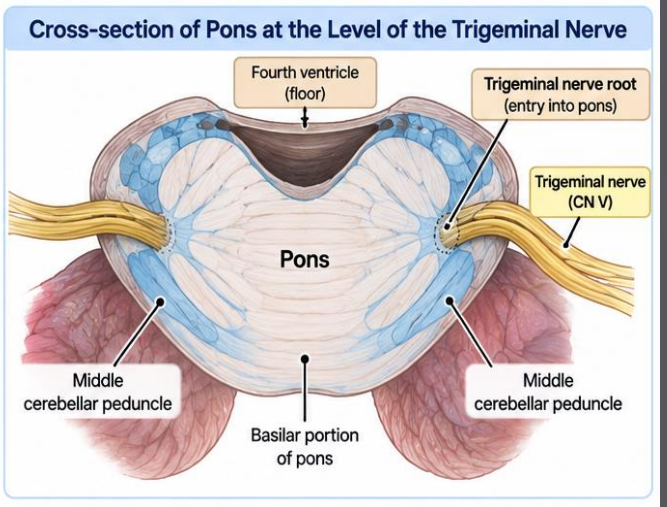


Cranial Nerves

Note the position of the AICA in relation to CN V and VII







TRIGEMINAL NEURALGIA AND HEMI-FACIAL SPASM

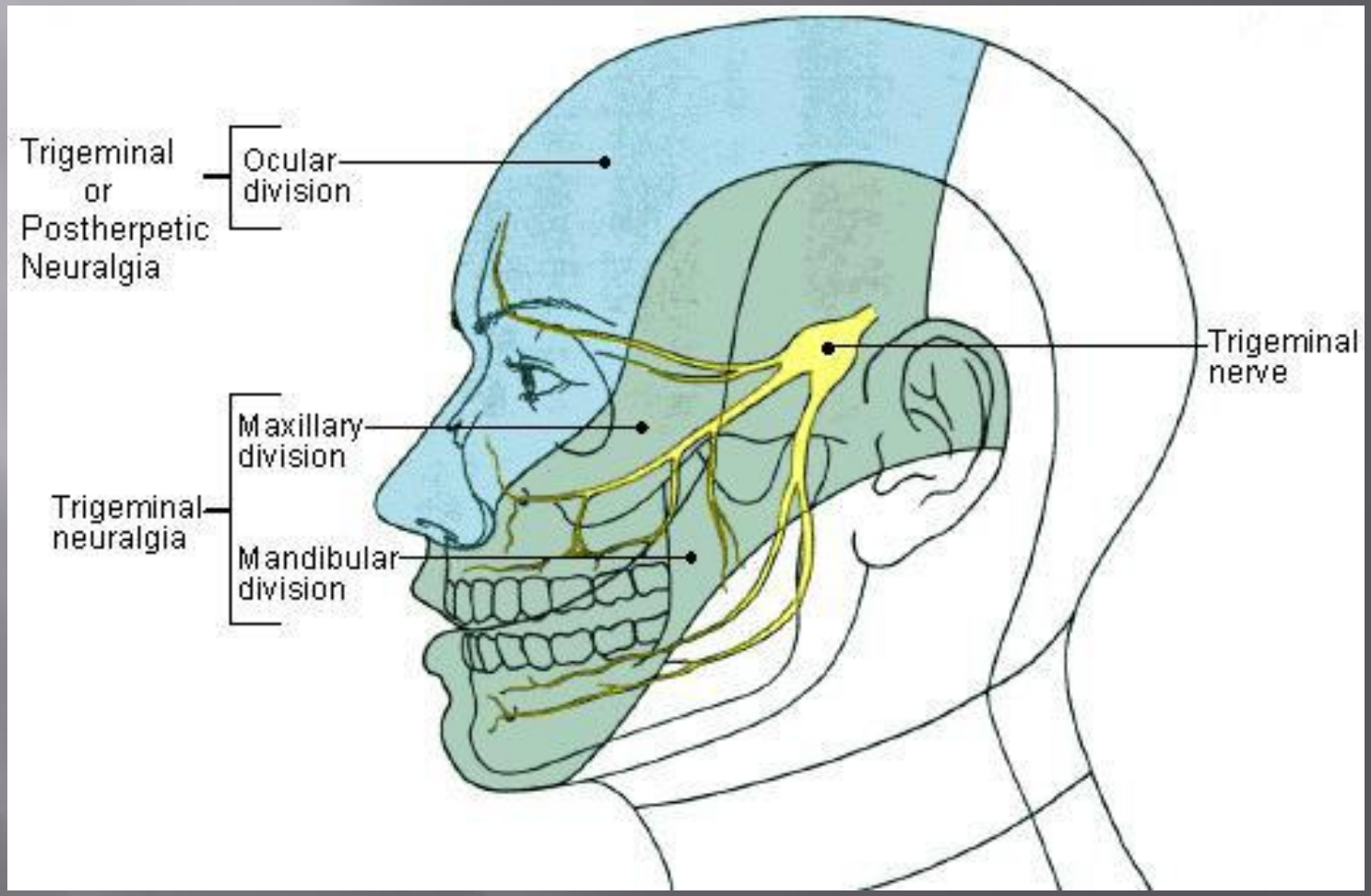
Trigeminal Neuralgia

- Trigeminal neuralgia (tic douloureux): Fifth cranial nerve (V)
  - It is an uncommon disorder occurring in 1 out of every 15,000 people
    - But is the most common of the cranial neuralgias
  - Its incidence is 4.3 per 100,000 people a year
  - Pain classified as *"typical"* and *"atypical"*
    - Typical: pain is intermittent, sudden, and shock like
    - Atypical: pain is constant, burning, and less severe

Trigeminal Neuralgia

- TN occurs in both genders and usually diagnosed in patients over 50
  - The most common chronic facial pain disorders develop at an earlier age than TN and have a female predominance.
    - For example, Migraine is 70% female and has a mean onset of 30 years old.
- TN involves one or more branches of the trigeminal nerve
  - The maxillary branch is involved most often
  - The right side of the face is affected more commonly

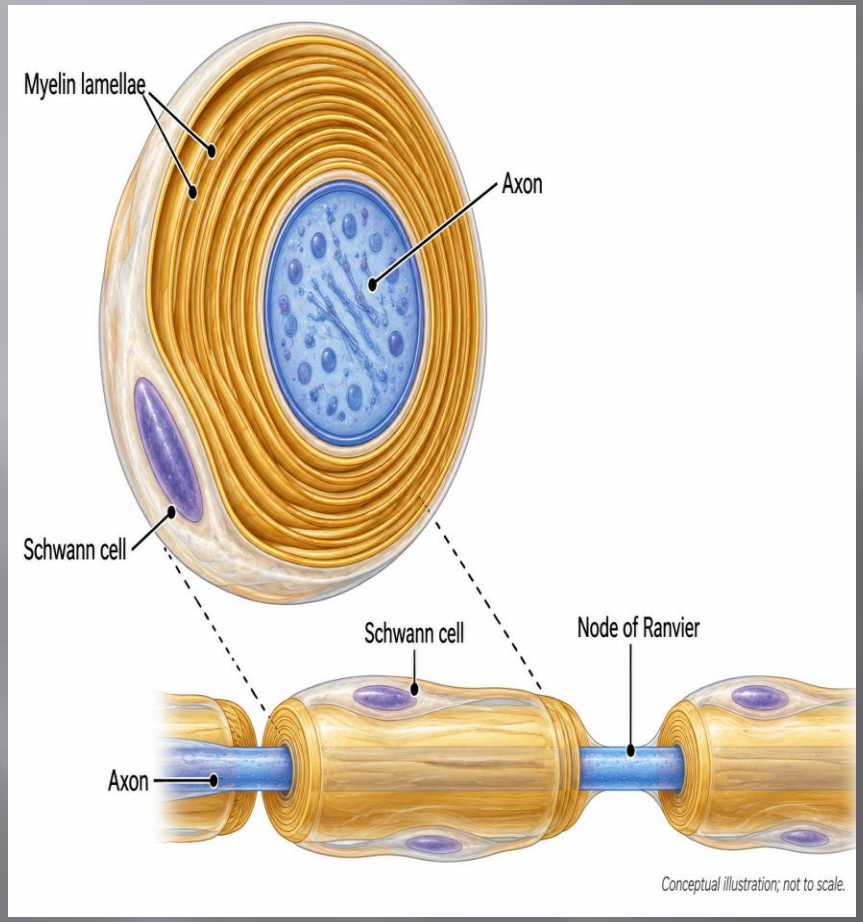
Branches of the Trigeminal Nerve

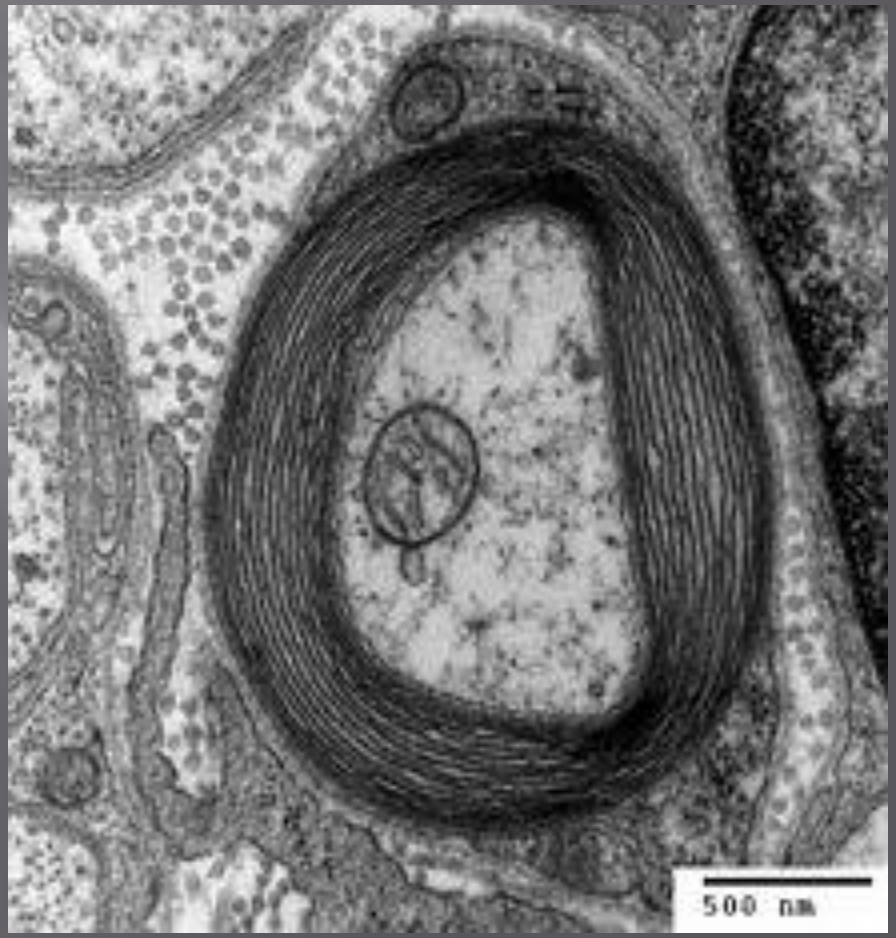


Pathophysiology

- The pathophysiology of TN is believed to be caused by damage to the myelin sheath surrounding the trigeminal nerve root.
- This is caused by the trauma of vascular compression with every heart beat.

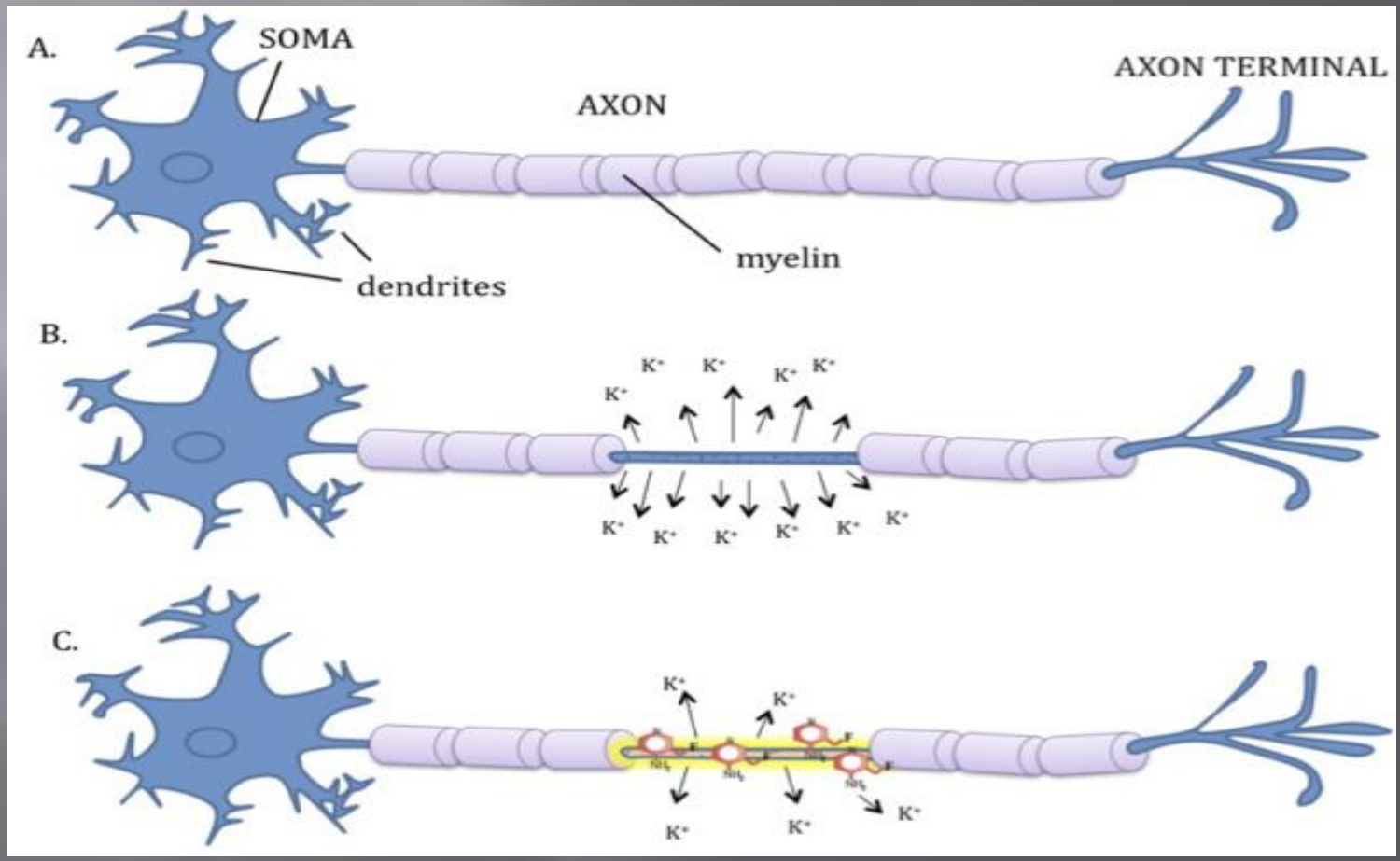
Myelin


Myelin lamellae
Axon
Schwann cell
Schwann cell
Node of Ranvier
Axon
Conceptual illustration; not to scale.


500 nm

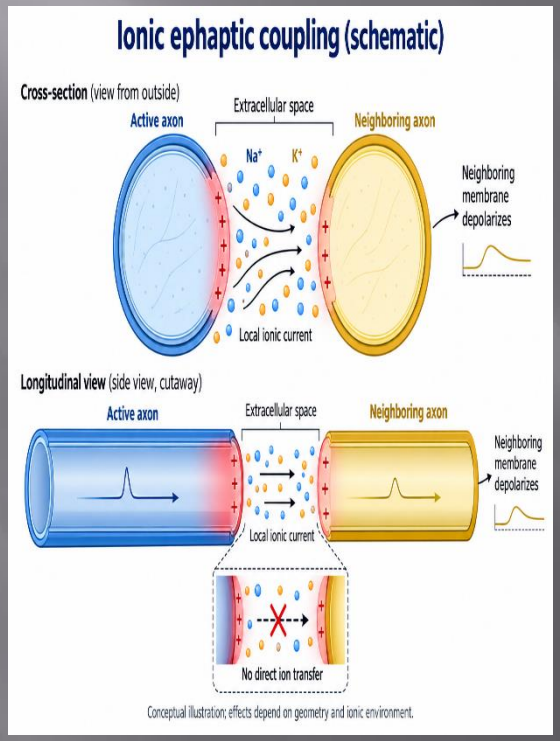
Myelin Function

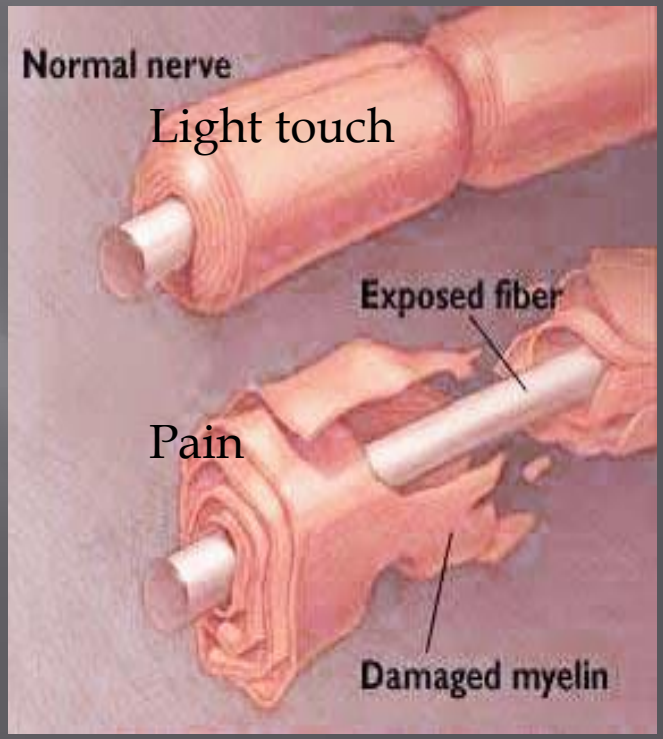
Myelin acts as an electrical insulator which speeds up nerve signal transmission and supports long-term neuron health across the brain and spinal cord.



Demyelination
Ephaptic Transmission

Ephaptic transmission is a mode of communication in the nervous system where neurons influence each other through local extracellular electric fields, rather than via chemical neurotransmitters.





Myelin damage changes light touch fiber to pain fiber

Diagnosis of TN

- Diagnosis based on history and symptoms
  - Intermittent intense electric-shock like pain
  - Pain can be triggered by light touch, putting on make-up, eating, chewing, teeth brushing, shaving, or even a light breeze
  - The pain is nearly always one sided, usually right side
  - TN should be considered with unilateral face pain
  - Pain may occur repeatedly throughout the day
  - Pain lasts from a fraction of a second to two minutes
  - There is no clinically evident neurologic deficit
    - Physical exam is normal
  - MRI can help see vascular compression.

Patient History

- Recurrent episodes of unilateral face pain
  - Pain lasts only seconds and recur
  - Pain rarely occurs during sleep
  - Asymptomatic between episodes
- Trigger zones
  - These are pathognomonic for this disorder
- Because of confusion with Multiple Sclerosis ask about
  - Ataxia, dizziness, focal weakness unilateral vision changes

Differential Diagnosis

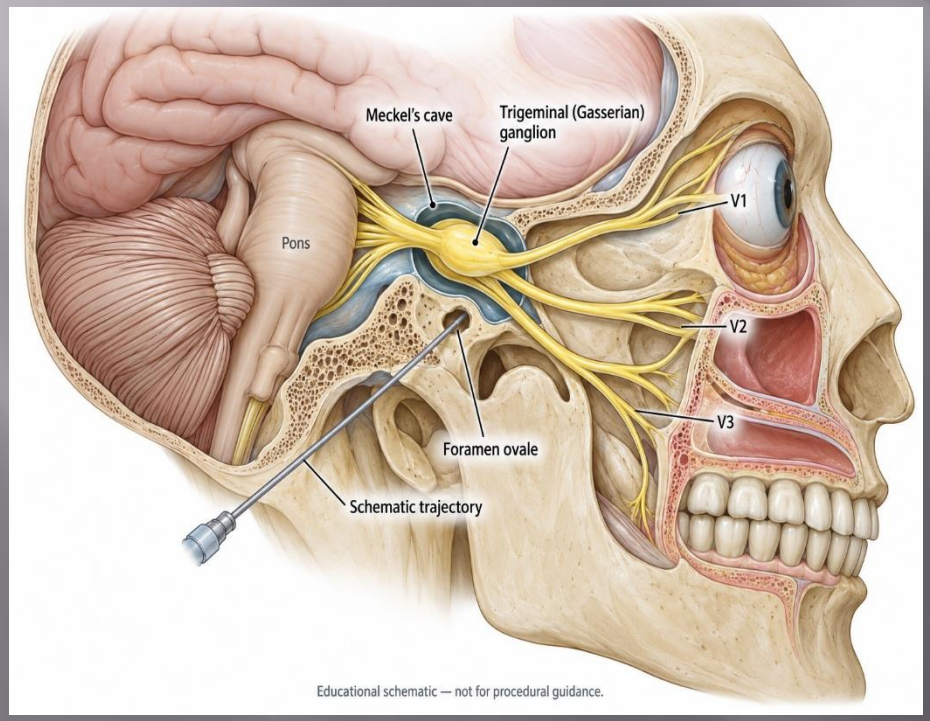
- Cluster headache
- Dental pain
- Intracranial tumors
- Migraine
- Multiple Sclerosis
- Otitis media
- Post Herpetic neuralgia. Shingles (herpes zoster) is a painful viral infection caused by the reactivation of the varicella-zoster virus—the same virus that causes chickenpox.
- Temporal-mandibular joint syndrome

Medical Treatment

- Anticonvulsant drugs are the most frequently used and are the most effective medical treatment.
- Carbamazepine (Tegretol) is the recommended initial treatment.
- Other anticonvulsants have been tried either alone or in combination with Tegretol.
  - Dilantin, gabapentin, clonazepam.
- Non-anticonvulsant medications include
  - Botox, capsaicin, imitrex, baclofen

Invasive Treatment

- Percutaneous:
  - At the gasserian ganglion: Radiofrequency, balloon compression, glycerol injection, gamma knife

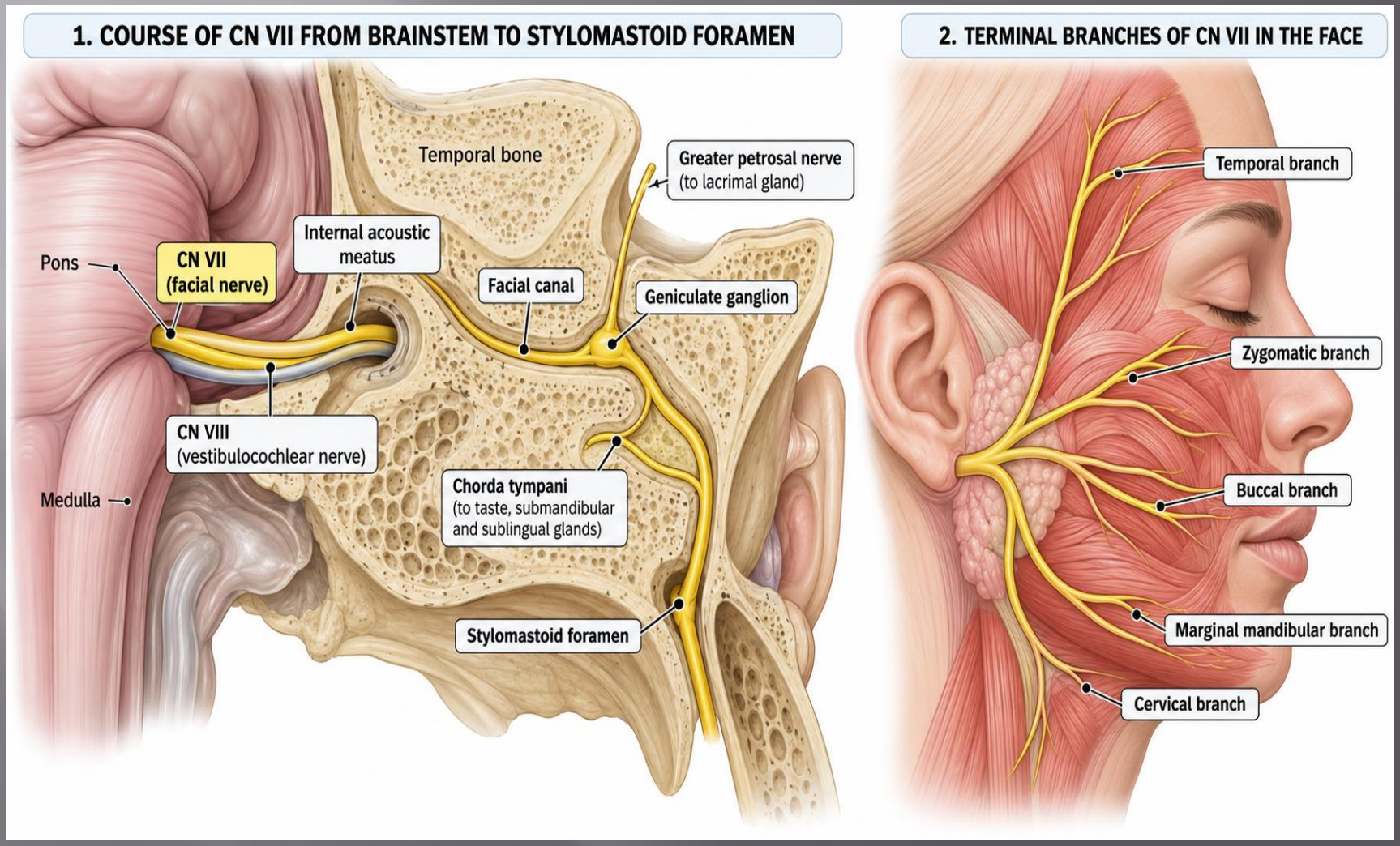




Hemi-facial spasm

- A disease of the seventh cranial nerve (VII)
- It can take two forms
  - Typical: twitching starts in the orbicularis oculi and progresses to the orbicularis oris (lower eyelid to lips)
  - Atypical from lips to eyelids
    - This form is seen only in 2%-3% of patients
- The incidence of hemifacial spasm is 0.8 per 100,000 persons
  - It affects middle-aged or elderly women most frequently
  - It is more common among Asians

Facial Nerve (VII) Anatomy



Hemi-facial Spasm





Figure 1: Patient with pronounced right-sided hemifacial spasm. Involvement of the platysma muscle is clearly visible

Diagnosis of Hemifacial Spasm HFS

- Facial spasm occurring in women over 40
  - In women under 40 suspect MS
- Less than 1% of cases are caused by tumor
- A complete neurologic exam including
  - Electromyography
  - MRI
    - MRI is the most effective screening test for hemifacial spasm.

Treatment of HFS

- Medical treatment consists of anticonvulsants
  - Dilantin and Tegretol

Treatment of HFS

- Botulinum toxin injection into the affected muscles may also be effective.
  - This may require repeated injections every 3-6 months



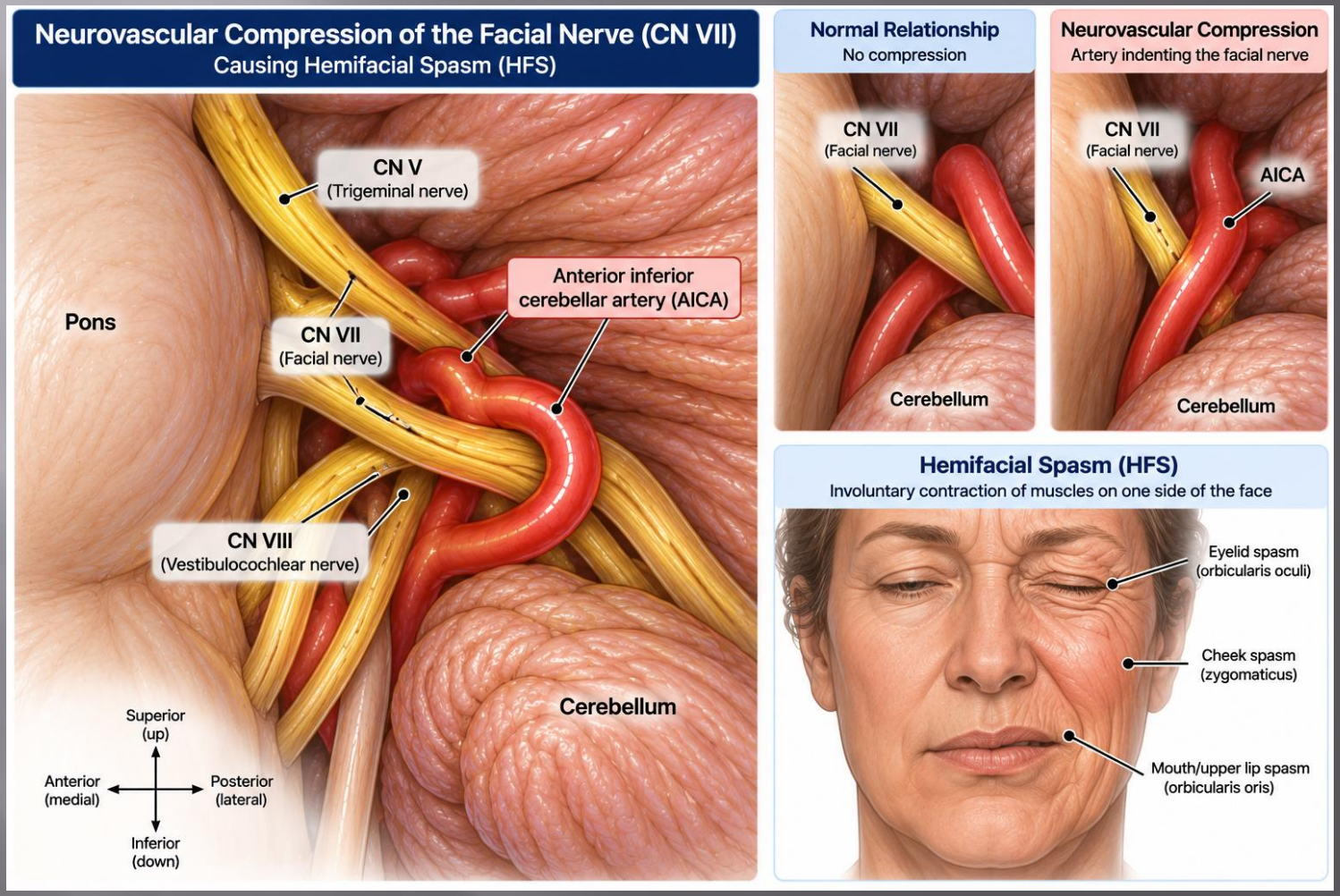
Treatment of HFS

- Invasive treatment for HFS in the early 20th century included
  - Neurolysis: destruction of the nerve branch
  - Injection of the nerve with alcohol
  - Electrical stimulation
- In 1977 Peter Jannetta developed Microvascular decompression of the 7th CN at its root exit using the operating microscope

Treatment Results and Complications

- Microvascular Decompression
  - Excellent results: 80% success with 10% recurrence
- Serious complications (2% of patients)
  - Cerebellar hematoma
  - Brain stem infarction
  - Cerebral infarction (stroke)
  - Hearing loss (VIII)
  - Death

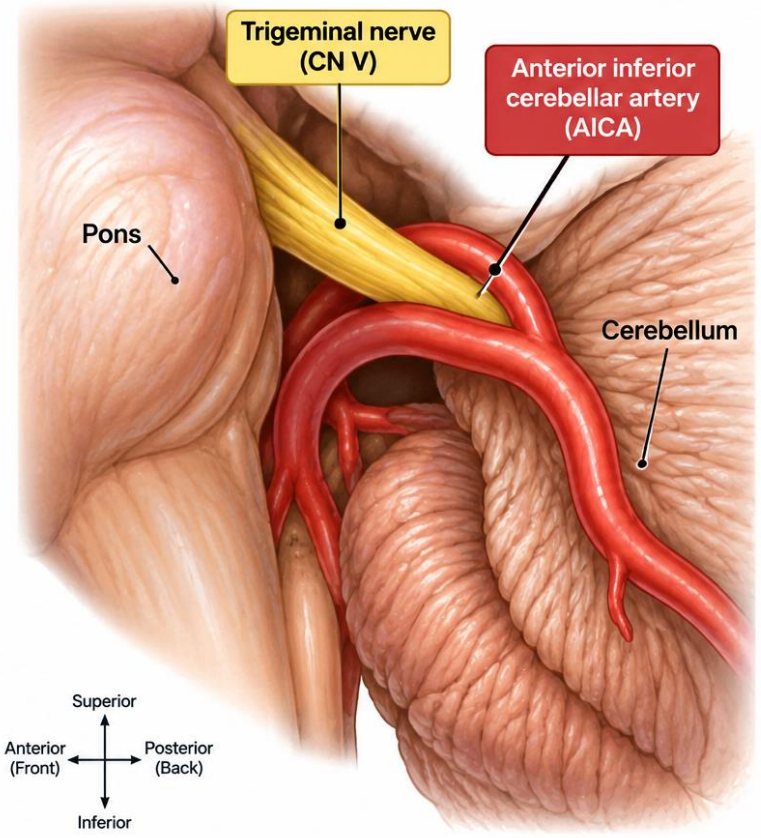
Vascular Compression of CN VII HFS



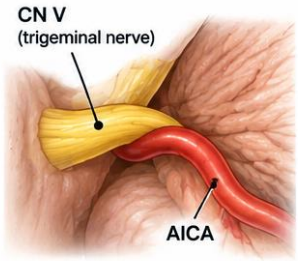
Vascular Compression Trigeminal Nerve

AICA Resting on the Trigeminal Nerve

(Anatomical View – Close-up)

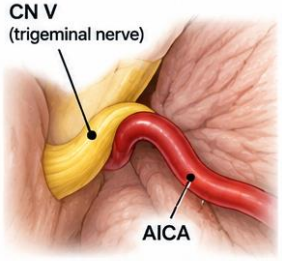


Normal/Non-compressive Contact
(Often seen on MRI)



The AICA can normally contact the trigeminal nerve without causing symptoms.

Compression/Displacement
(More likely to cause symptoms)



When the artery compresses or displaces the trigeminal nerve (especially at the root entry zone), it can lead to trigeminal neuralgia in some people.

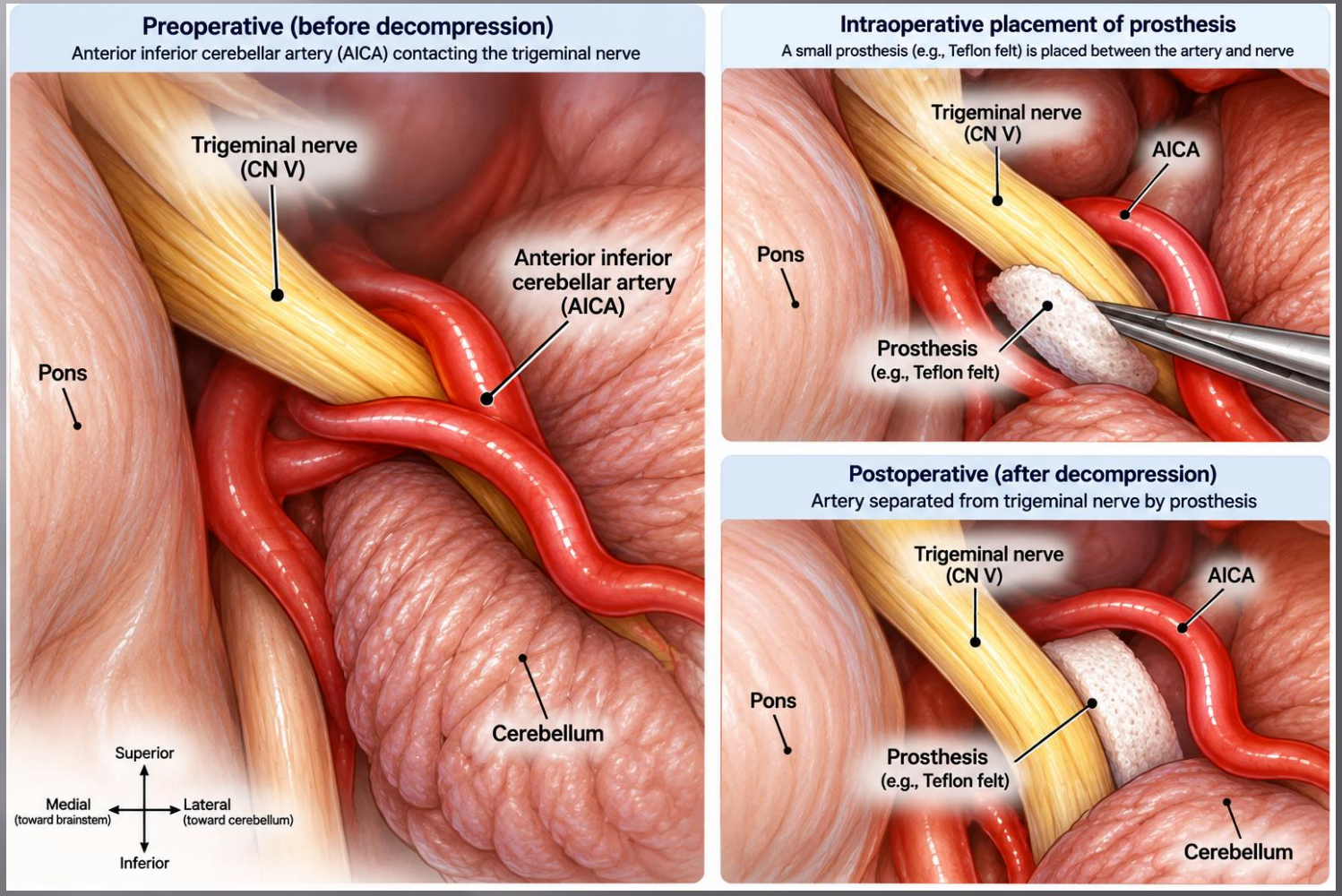
Key Points

- AICA resting on the trigeminal nerve = neurovascular contact.
- Neurovascular contact is relatively common on MRI and does not always cause symptoms.
- Trigeminal neuralgia (if present) typically causes brief, severe, electric-shock-like facial pain, often triggered by touching the face, chewing, brushing teeth, or cold air.
- The superior cerebellar artery (SCA) is more commonly involved in trigeminal nerve compression than AICA.

Bottom line:

"AICA resting on the trigeminal nerve" does not by itself mean you have trigeminal neuralgia or that treatment is needed. The MRI finding should be correlated with your symptoms.

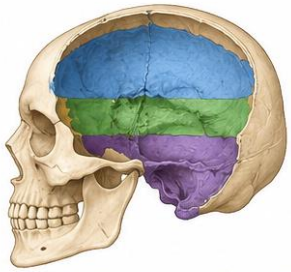
Prosthetic Placement



The Posterior Fossa

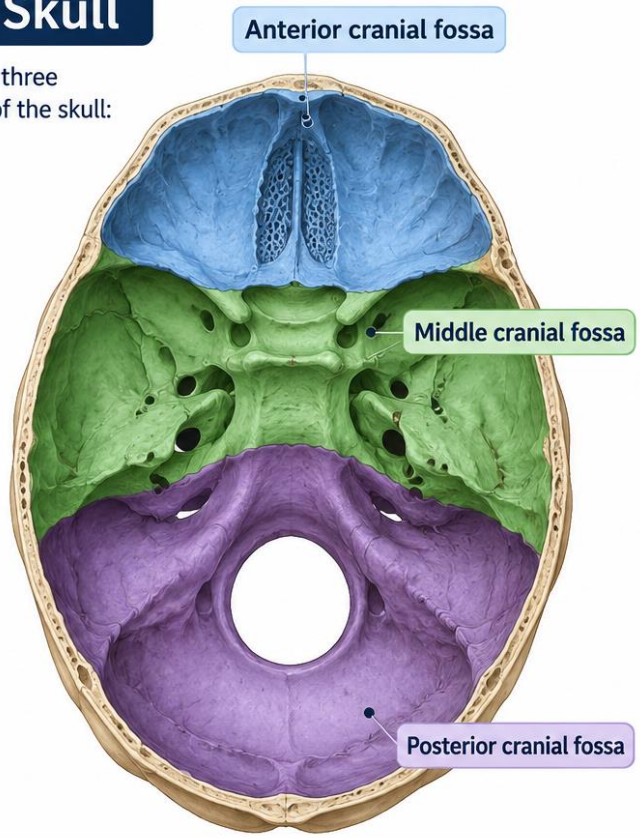
Fossae of the Skull

The cranial cavity is divided into three fossae (depressions) at the base of the skull: anterior, middle, and posterior.



Lateral view (showing fossae)

- Anterior cranial fossa
- Middle cranial fossa
- Posterior cranial fossa



Base of skull (inferior view)

Anterior Cranial Fossa

- **Location:** Frontal bone (anterior), cribriform plate of ethmoid (middle), lesser wings of sphenoid (lateral).
- **Contains:** Frontal lobes of cerebrum, olfactory bulbs and nerves (CN I).
- **Key landmarks:** Cribriform plate, crista galli, foramina for olfactory nerves.

Middle Cranial Fossa

- **Location:** Body of sphenoid (central), greater wings of sphenoid (lateral), temporal bones (lateral).
- **Contains:** Temporal lobes, pituitary gland (in sella turcica), cavernous sinuses, internal carotid arteries, CN III, IV, V_1, V_2, VI.
- **Key landmarks:** Sella turcica, foramen ovale, foramen spinosum, carotid canal.

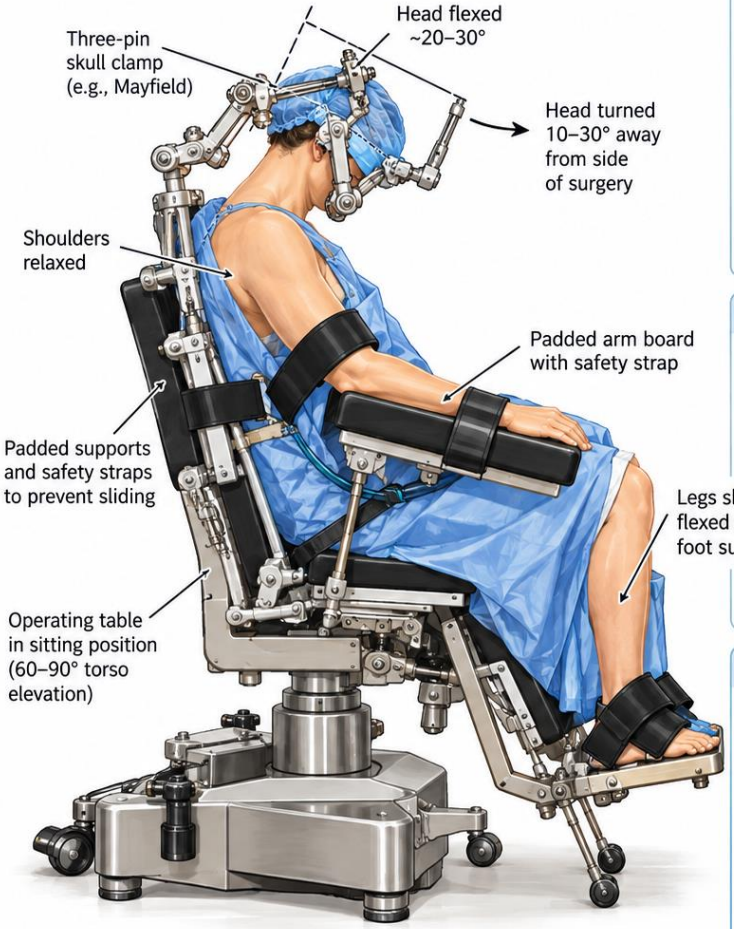
Posterior Cranial Fossa

- **Location:** Occipital bone (posterior), petrous temporal bones (lateral), clivus (anterior).
- **Contains:** Cerebellum, brainstem, cranial nerves VII, VIII, IX, X, X_1, XII.
- **Key landmarks:** Foramen magnum, internal acoustic meatus, jugular foramen, hypoglossal canal.

Sitting Position

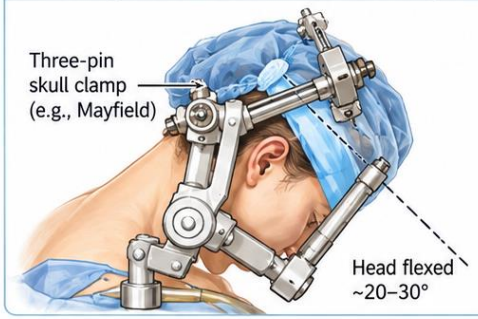
Sitting Position for Neurosurgery

(e.g., posterior fossa surgery, microvascular decompression)

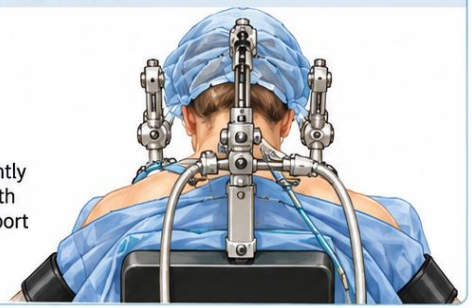


A. Lateral view (typical)

B. Head position and fixation (close-up)



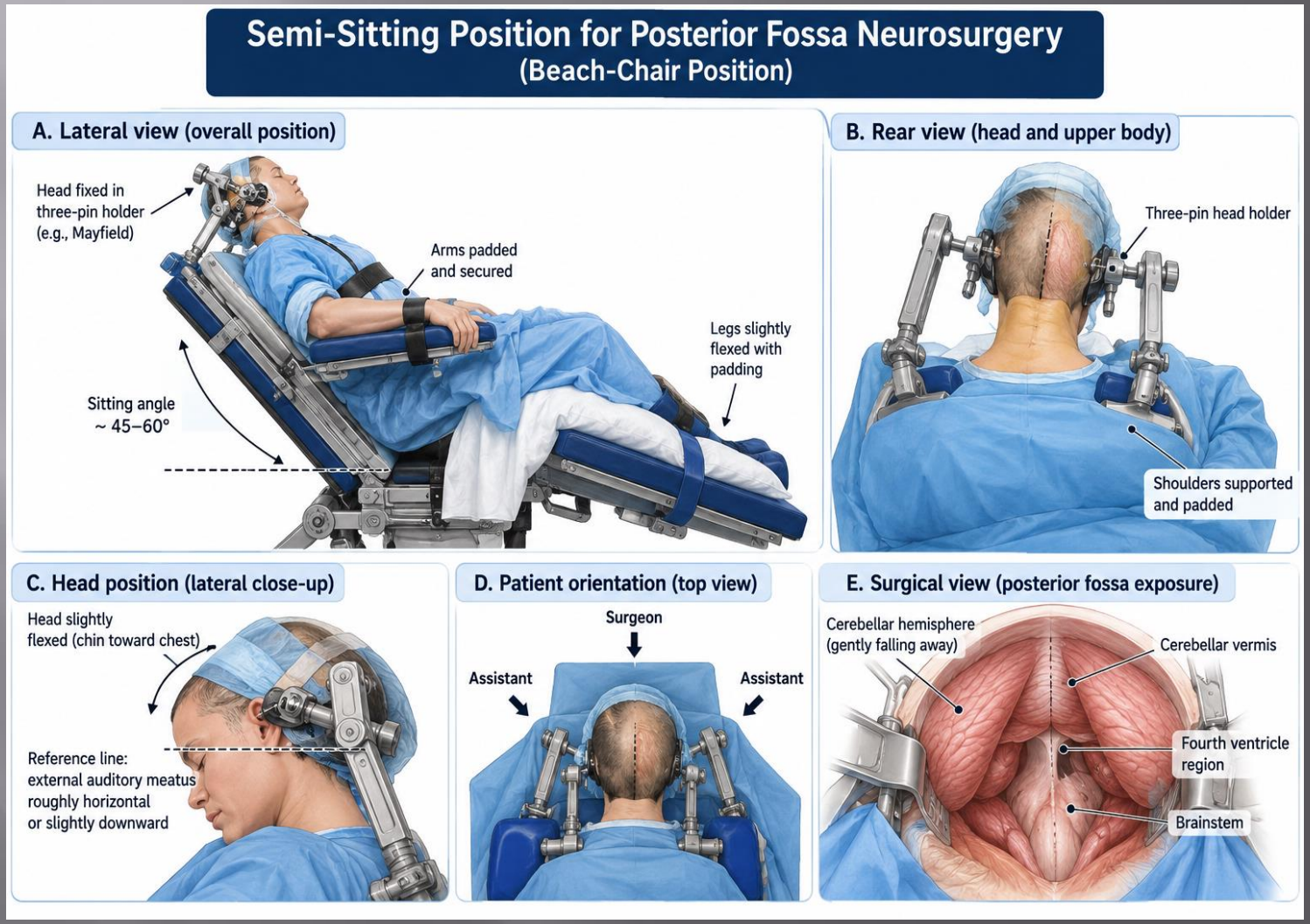
C. Rear view



D. Key points

- Torso elevated ~60–90° (sitting/semi-sitting).
- Head flexed ~20–30° and may be turned 10–30° away from the side of surgery.
- Rigid three-pin skull fixation (e.g., Mayfield).
- Padded supports and safety straps to prevent sliding.
- Arms on padded arm boards, shoulders relaxed.
- Legs slightly flexed with foot support.
- Sitting position increases risk of venous air embolism (often monitored with precordial Doppler or transesophageal echocardiography).

Semi-Sitting Position



Advantages of Sitting Position for Posterior Fossa Surgery

- The sitting position for posterior fossa neurosurgery offers several important surgical advantages, particularly for operations involving the cerebellum, brainstem, fourth ventricle, and cerebellopontine angle (CPA). These benefits must be balanced against the risk of venous air embolism (VAE) and other position-related complications.

Advantages

- Improved surgical exposure
- Provides excellent access to the midline posterior fossa.
- Facilitates approaches to the fourth ventricle, cerebellar vermis, and brainstem.
- Can improve access to the cerebellopontine angle for selected lesions.
- Improved venous drainage and a less congested field

Complications of the Sitting Position

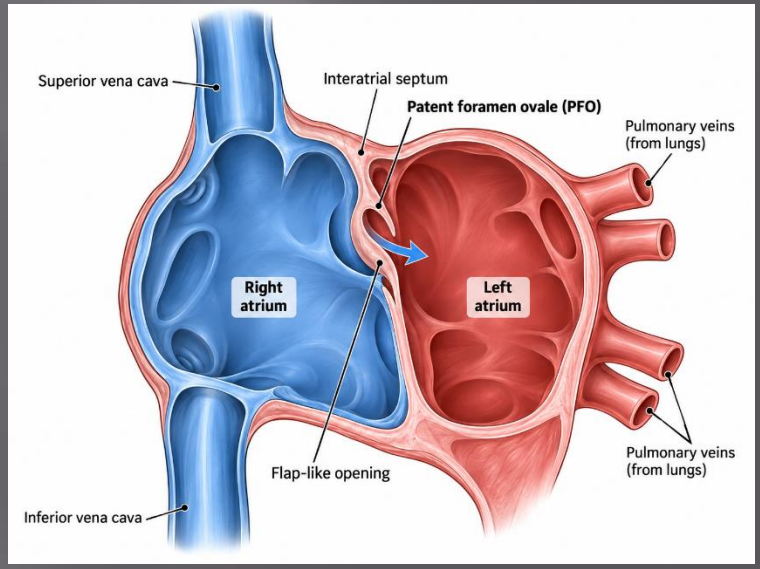
- The sitting position is used for selected posterior fossa, cerebellar, and posterior cervical procedures. It provides excellent surgical exposure and venous drainage, but it carries important anesthetic and neurosurgical risks.
- The three major concerns are **venous air embolism**, **hemodynamic instability**, and complications caused by excessive neck flexion.

Major Complication Venous Air Embolism

- Venous air embolism (VAE)
- Air enters open, non-collapsible veins when the operative site is above the level of the right atrium, potentially causing pulmonary obstruction, hypoxemia, hypotension, and cardiovascular collapse. A paradoxical embolism can cause cerebral or coronary ischemia if air crosses into the arterial circulation through a PFO.

Complications

- **Patient Foramen Ovale**: Venous air enters the arterial circulation, often through a patent foramen ovale (PFO) about 25% **(roughly 1 in 4 adults**), potentially causing stroke or myocardial ischemia. **Cardiac Arrest**: May occur with severe air embolism, cardiovascular compromise, or brainstem manipulation.



Prevention and Monitoring

- Screen for right-to-left cardiac shunts, particularly PFO.
- Use appropriate monitoring for venous air embolism, such as precordial Doppler or transesophageal echocardiography in selected cases, along with end-tidal CO_2 and continuous hemodynamic monitoring.
- Position the head and neck carefully, avoiding excessive flexion and ensuring the airway and venous drainage remain unobstructed.
- Maintain adequate arterial blood pressure and cerebral perfusion; account for the pressure difference between the heart and the elevated brain.
- Protect pressure points and peripheral nerves and recheck the endotracheal tube and vascular access after positioning.
- Have a coordinated anesthesia–surgical plan for immediate management of suspected air embolism

Key Takeaway

- Venous air embolism is the major position-specific concern, while excessive neck flexion and hypotension are important preventable risks. With careful patient selection, positioning, monitoring, and a prepared anesthesia team, the sitting position remains a good option for selected neurosurgical procedures.
- Limit the use of volatile anesthetics: Volatile anesthetics (such as sevoflurane, isoflurane, and desflurane) cause dose-dependent cerebral vasodilation, which increases cerebral blood flow (CBF) and can increase cerebral blood volume (CBV).
- Their effects on the cerebral circulation reflect two opposing mechanisms:
- Direct vascular effect: relaxation of cerebral vascular smooth muscle → vasodilation → increased CBF.
- Metabolic effect: suppression of cerebral metabolic rate of oxygen consumption ($CMRO_2$) → reduced metabolic demand → decreased CBF through metabolic coupling.

Anesthetic Management

- The key concern is that cerebral vasodilation can increase cerebral blood volume and potentially raise ICP, particularly in patients with a brain tumor, cerebral edema, or other space-occupying lesions.
- At higher doses, impaired autoregulation also makes cerebral blood flow more dependent on systemic arterial pressure. Hypotension can therefore threaten cerebral perfusion.
- Volatile anesthetics decrease $CMRO_2$ but directly dilate cerebral vessels. At higher concentrations, vasodilation can predominate, increasing CBF and potentially ICP.
- Therefore, use TIVA (total intravenous anesthesia) without nitrous oxide.

Historical Video

- The following DVD is a presentation of Dr. Peter Jannetta preforming a microvascular decompression.
- I was privileged to have had the opportunity as a fellow in Neurosurgical Anesthesia and Neuro Critical Care at The University of Pittsburgh to work with Dr. Jannetta.
- The surgical team did 650 posterior fossa sitting cases.