What is the clinical entity TN?

- Unilateral recurrent episodic intense facial pain
  - "electric-shock", "stabbing", "lancinating", "shooting", "burning", "excruciating"
  - Abrupt onset following physical stimulation of ipsilateral 'trigger zone'
- Typically assoc'd with cramping of facial muscles
  - "Tic Douloureux" = painful twitch
- The “Suicide disease”

What is Atypical TN?

- Also known as Trigeminal Neuralgia Type 2 (TN2)
- Constant, aching, burning pain
  - of lower intensity than TN1
- Bilateral TN - simultaneous involvement of both sides of face is rare

What is going on pathologically?

- Empirical evidence suggests 95% TN due to neurovascular compression **CLASSIC TN**
  - Wearing away of myelin sheath (both de- & dys- myelination have been shown)
    → Impaired nociceptive system
    → Ephatic crosstalk / Ignition hypothesis
- 5% MS plaque / brainstem infarct, CPA mass, perineural process **SYMPTOMATIC TN**

What might we find on imaging?

- Routine MR sequences
  - Normal brain
- High resolution T2 MR (CISS/FIESTA):
  - Vascular loop (artery) @ CNS REZ
  - ± CNS nerve atrophy
- MRA
  - Source images most helpful for vascular loop @ REZ

Disclosure

- The graphics in this presentation were created by Amirsys.
- Dr Glastonbury has received royalties from Amirsys and Elsevier
Most often arterial
- Superior cerebellar
- Anterior inferior (AICA) & posterior inferior (PICA)

Sometimes venous
- Superior petrosal vein & its tributaries

13-60% asymptomatic vascular contact with CN5 at surgery; up to 78% MRI
3-17% of TN decompressive surgeries find no vascular compression
CN9-12 often in contact with vertebral @
- Rarely have syndromes other than CN9

Be careful that trigeminal symptoms are frequently all called “TN”!
Always consider other causes of facial pain:
- TMJ, dental or sinus disease
- Multitude of trigeminal nerve pathologies may manifest with pain / dysesthesias

Three twins: CN5¹, CN5² & CN5³ (V1/V2/V3)
Sensory - face, conjunctiva, sinuses, teeth, tongue, external aspect of TM
- PLUS anterior + middle fossa meninges
Motor to most of jaw muscles
Complex brainstem nuclei
Largest cisternal cranial nerve
Complex extracranial branches
**Motor nucleus**

**Sensory nuclei**

- Mesencephalic
- Primary sensory proprioceptive
- Chief sensory
- Facial touch
- Spinal tract
- Pain & temperature

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**CN5 Branches**

5¹: Ophthalmic - Nasociliary, frontal lacrimal

5²: Maxillary - PPF, zygomatic, infraorbital, palatine

5³: Mandibular Masticator, mylohyoid, 4 sensory nerves

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**Symptomatic Trigeminal Neuralgia**

- MS, Infarct, Lyme disease
- Leptomeningeal infection, inflammation, tumor & CPA masses
- Perineural, tumor or process inflammation

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**TN Fast Facts**

- TN is a specific neurologic disorder
  - **TN1**: Unilateral, CN5¹ / CN5³ intense episodic pain
  - Specific triggers may be identified
  - Tends to worsen in frequency/severity over time
- **TN2**: Atypical TN = constant aching pain
- **Classic** = No established etiology
  - Vascular compression at CN5 REZ implicated
  - SCA most often, AICA, PICA, sup petrosal vein
- **Symptomatic** = Lesion / mass / tumor etc

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**‘Red Flag’ Clinical Symptoms of TN**

- Sensory changes
- Deafness or inner ear balance issues
- Poor response to medical therapy
- Prior skin or oral neoplasm
- Isolated to CN5¹ or bilateral TN
- Optic neuritis, family history of MS
- Onset <40 years age
Key Radiology Take Home Points

- Look for vascular loop @ REZ (T2 & MRA)
  
- Not until after you have evaluated the entire course of CNS brainstem nuclei to deep face
  
Exclude Symptomatic TN (brainstem lesion / CNS mass / perineural process) first!
  
You must know your CNS nerve anatomy!

References


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Imaging for Hemifacial Spasm (HFS)

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Assistant Professor
University of Virginia Health System

Disclosures
• None

OBJECTIVES
• Understand HFS, the work up and treatment options
• Review relevant facial nerve anatomy
• Characterize the imaging appearance of vascular compression of the facial nerve in patients with HFS

1893 by Edouard Brissaud
• “A case of a 35-year-old woman with clonic contractions (secousses cloniques) in the right face. All the facial muscles were involved, including the frontalis, the orbicularis oculi, the zygomaticus, and the platysma”.

What is Hemifacial Spasm?
• Hemifacial spasm is a neuromuscular disorder characterized by frequent involuntary contractions (spasms) of the muscles on one side (hemi-) of the face (facial).

Hemifacial Spasm
• HFS is most commonly caused by vascular compression of the facial nerve.
• Other etiologies
  – Intracranial masses
  – Facial nerve injury
  – Brain stem infarct
  – Demyelinating plaque

The Many Faces of Hemifacial Spasm: Differential Diagnosis of Unilateral Facial Spasms
Toby C. Yaltho, MD1 and Joseph Jankovic, MD*
Parkinson’s Disease Center and Movement Disorders Clinic, Department of Neurology, Baylor College of Medicine, Houston, Texas, USA.
Hemifacial Spasm

- HFS is most commonly caused by vascular compression of the facial nerve.
- Other etiologies
  - Intracranial masses
  - Facial nerve injury
  - Brain stem infarct
  - Demyelinating plaque

Vascular Cause

- Vessels
  - Vertebrobasilar artery
  - Posterior inferior cerebellar artery (PICA)
  - Anterior inferior cerebellar artery (AICA)

Variations in vascular compression

- 1 vessel – multiple contacts
- Multiple vessel with contacts
- Adhesions and other variations

Hemifacial Spasm, A Neurosurgical Perspective
Park et al, JKNS, 2007

HFS work up – 3 step

- Clinical history & Neurological examination
- Electromyography (EMG) to distinguish from other abnormal facial movement disorders such as blepharospasm, tics, partial motor seizures, synkinesis, Meige’s syndrome, and neuromyotonia
- MRI

Role of MRI - Confusion

- Ability to detect vascular compression of the facial nerve with MRI AND also the significance of vascular compression by MRI
- Advances in imaging techniques allows for determination of culprit vessel in almost all patients
- Exclude other structural causes and confounding diagnosis

Vascular Cause

- Vessels
  - Vertebrobasilar artery
  - Posterior inferior cerebellar artery (PICA)
  - Anterior inferior cerebellar artery (AICA)

Role of MRI - Confusion

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Protocol features

- Ax T1, T2 FS, T1 FS post, T2 SPACE/CISS/SSFP
- Cor T1, STIR, T1 FS post
- WB: Ax FLAIR
- MRA TOF COW
Protocol – Sample Coverage

- Superior: top of CC
- Inferior: bottom of mandible
- Anterior: tip of nose
- Posterior: back of CC

3D SSFP sequences

- Axial
- Coronal
- Sagittal

HFS – Treatment Options

Botulinum inj
- Local botulinum toxin injection into the orbicularis oculi or lower facial muscles is a nonsurgical palliative option that can reduce the severity of HFS.

HFS – Treatment Options

MVD
- Only microvascular decompression offers the potential for cure of HFS with success rates exceeding 90%....


Facial nerve nucleus-Motor

- Muscles of facial expression
- Ventral pons
- Fibers loop around abducens nuclei causing facial colliculus
- Exit in CP cistern at ponto medullary junction

Proximal Facial Nerve Anatomy

Raghavan et al, Imaging of Facial Nerve, NICNA

Proximal Facial Nerve Anatomy

Step by step approach

- Confirm laterality
- Exclude other structural causes
- Identify facial nerve
- Identify the offending vessel (almost always an artery – Vertebrobasilar, PICA or AICA)
- Point of contact - Proximal/ Middle/ Distal
- If proximal – delineate it more precisely
- Single or multiple vessels/ point of contact
- Define contact: contact +/- displacement

MR Images

Case 1

Case 2

Case 3
Case 3


Case 4

Future directions


Further Reading


THANKS

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Imaging for Facial Reanimation

Loss of facial nerve function can be devastating to patients. The field of facial plastics has developed many methods of improving the appearance and function of the face in patients with facial paresis, and in this session we will discuss imaging evaluation in preparation for one of these methods – gracillis muscle transfer.

The most common cause of facial paresis is Bell’s palsy. Typically patients with Bells experience a rapid onset and resolve spontaneously. A small percentage will be left with persistent Bells palsy and the first and most important role of the radiologist in patients with facial paresis is distinguishing those patients with more ominous causes of facial paresis from patients with persistent Bells. Features which suggest mass lesions as a cause of facial paresis include recurrent or progressive course of weakness, history of prior parotid or skin cancer, mass lesion in the parotid, multiple cranial neuropathies and ipsilateral adenopathy. Patients with any of these features must have high quality imaging through the entire course of the facial nerve in order to evaluate it fully for the presence of perineural tumor spread, vascular lesion, or benign tumor such as facial nerve schwannoma.

An additional role of the radiologist in the patient with mass lesion as the cause of facial palsy is identification of the extent of disease. In a patient with parotid cancer and perineural tumor spread, accurate delineation of the extent of disease is required for pre-operative planning. In particular it is important for the surgeon to know whether temporal bone drilling will be required to achieve a clean margin. This can also be helpful for pre-operative planning of cable grafts to bridge any defects in the facial nerve.

A variety of surgical approaches can improve the appearance and quality of life for patients with facial paresis. Lid weights (made of gold or platinum) can reduce corneal exposure. Fascial lata slings may improve facial tone and elevate the corner of the mouth and nasolabial fold. Gracillis muscle grafts can significantly improve the voluntary smile of patients and radiologists can be very helpful in their placement.

The gracillis graft features a gracillis muscle from the thigh, which comes with an artery, two veins and an obturator nerve. It is placed in the face, extending from the temple region to the corner of the mouth. The obturator nerve is anastamosed to either a cross face nerve graft (brought over during a prior surgery) or to a branch of the ipsilateral trigeminal nerve. Unlike most other head and neck flaps, there is no skin brought with the gracillis graft, and it is buried deep to the skin of the face. For this reason, features such as skin turgor and capillary refill cannot be used to monitor the integrity of the venous and arterial anastomosis. Bedside Doppler can be used for the arterial pedicle, but gives no information on the venous pedicle.

We do ultrasound on these grafts, the morning after the procedure. We identify the site of venous anastomosis by identifying the venous coupler. We evaluate the artery and vein both above and below the venous coupler and ensure that there is good color flow, Doppler signal and compressibility of the vein.
Gracillis grafts are typically anastamosed to the facial artery and vein. In some patients, prior surgery or injury in this region renders the status of these vessels uncertain. In these patients we perform preoperative CTA and CTV to identify alternative sites of vascular anastomosis.
Get Off My Nerves: Perineural Tumor Spread in H&N Cancer

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Goals for ASNR 2015 Symposium

- Basics
  - clinical scenarios, presentation
  - involved nerves
- Imaging strategies, modality, issue of Brain imaging, importance of fat-suppression
- All I can do in 20 minutes:
  - show some basics
  - major involved, some rare nerves
  - avoidance of pitfalls

Perineural Tumor Spread (PNS)

- Definition: dissemination of tumor from the primary site along tissue planes of the neural sheath
- Small vs. large nerve PNS
- Clinical settings:
  - salivary gland-parotid, minor salivary gland (palate)
  - mucosal (SCCa)-palate/RMT, nasopharynx (via MS, PPF)
  - skin (SCCa, desmoplastic melanoma)
  - previously treated-forgotten disease*
- Symptoms: pain, paresthesias, motor denervation, but up to 40% may be asymptomatic
- Implications: serious finding associated with decreased survival. Detection will often affect treatment

Perineural Tumor Spread (PNS)

- Failure to recognize PNS (big problem)
  - common pitfall in head and neck imaging
  - good way to get sued
  - guarantees disease recurrence
- Because may be asymptomatic, up to radiologist to think of PNS and detect it
- Rarely, site to which tumor spreads perineurally may present prior to detection of primary cancer. Therefore, must consider PNS whenever lesion is seen in Meckel’s cave, pterygopalatine fossa, or cavernous sinus

PNS bad, but not universally deadly, as in this typical scenario

66-y/o man with Hx/o right cheek SCCa, disappeared with topical 5FU, subsequent paresthesias dismissed by ENT because scope was neg. Ultimately developed parotid met, resected, XRT, but had progressive multiple cranial neuropathy

Outside report: “Worrisome for right parotid met, no facial PNS.”

11/10

Fast forward, admission to MDACC 7/12

IMPRESSION:
“This is very advanced perineural tumor spread, I would say extreme.”

2/15, post chemo

IMPRESSION:
“...treatment response has been miraculous.”
PNS Anatomic Considerations

- Trigeminal nerve V
  - V₁ ophthalmic
  - V₂ maxillary
  - V₃ mandibular
- Facial nerve VII
- Connections between V and VII
- Spinal nerve, other unusual nerve

Perineural Tumor Spread - Imaging

- Widening/destruction of or excessive enhancement within neural foramina (oval, rotundum, palatine, stylomastoid foramen/descending facial canal, vidian canal)
  - CT better for bone destruction (late finding)
- Loss of normal fat density (CT)/T1 signal intensity (MR) or excessive enhancement/widening of the pterygopalatine fossa
- Enlargement/excessive enhancement within cavernous sinus or Meckel’s cave
- MR technique: 16-18 cm FOV, 3 mm slices, fat-suppressed, post contrast T1-weighted images

What should a normal pterygopalatine fossa look like?

What should a normal Meckel’s Cave look like?

58-y/o man, s/p resection SCCa left medial forehead, locally recurrent 5 months later, with PNS on supratrochlear branch V₁, extending to frontal nerve
72-y/o man presented with left forehead numbness, subsequently developing fullness. Imaging (Brain MR) allegedly normal. Biopsy of left eyebrow region SCCa. Post-bx developed diplopia attributable clinically to left abducens palsy. Note tumor going through superior orbital fissure.

Nasociliary Nerve

- Branch of V1
- Provides cutaneous sensory fibers to skin of lateral nose, and sensory innervation from the frontal dura, sphenoid and ethmoid sinus mucosa, nasal mucosa, and medial canthus


77-year old woman with recurrent SCCa, left medial canthal region

(PNS) Anatomic Considerations

- Maxillary nerve, V2-sensory to the mid-face, palate, sinonasal region, upper oral cavity. Common pathway to PPF, foramen rotundum, cavernous sinus, Meckel’s cave
- Mandibular nerve, V3-sensory to lower face and oral cavity, motor innervation to muscles of mastication. Common pathway to foramen ovale, Meckel’s cave
- Antegrade PNS-Meckel's cave to cavernous sinus or downward along V3, Cavernous sinus anteriorly along V2, PPF along palatine or infraorbital nerves
- Facial nerve, generally from primary parotid lesions or lesions that secondarily extend into the parotid

Fair-skinned 74-y/o male with left cheek melanoma, and V2 hypesthesia
62-y/o man, now with left V₂ paresthesias following Mohs surgery for left cheek SCCa. Recurrence with infraorbital nerve PNS.

61-y/o woman, lesion removed from right cheek, told keratoacanthoma (it wasn’t), or there was lurking SCCa. Then lesion recurred, radiologist said scar vs. mass, no mention of infraorbital nerve vs. PNS when PPF was OK. Lesion progressed, now horrible.

3 years later, no evidence of disease.

64-y/o female presents with sudden onset of left cheek numbness. Unsuccessfully treated for sinusitis. PE confirmed V₂ sensory abnormality and was otherwise normal.

Palatal/maxillary alveolar ridge ACCa, PNS to PPF, rotundum.
(PNS) Anatomic Considerations

- Maxillary nerve, \( V_2 \)-sensory to the mid-face, palate, sinonasal region, upper oral cavity. Common pathway to PPF, foramen rotundum, cavernous sinus, Meckel’s cave

- Mandibular nerve, \( V_3 \)-sensory to lower face and oral cavity, motor innervation to muscles of mastication. Common pathway to foramen ovale, Meckel’s cave

- Antegrade PNS-Meckel’s cave to cavernous sinus or downward along \( V_3 \), Cavernous sinus anteriorly along \( V_2 \), PPF along palatine or infraorbital nerves

- Facial nerve, generally from primary parotid lesions or lesions that secondarily extend into the parotid

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Prior left buccal SCCa, now recurrent to masticator space, and then PNS along \( V_3 \), into Meckel’s cave

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Inferior Alveolar Nerve

- Branch of mandibular nerve (\( V_3 \))
- Provides sensory innervation to lower gingiva and teeth, and cutaneous sensory innervation to the chin via the mental nerve
- Enters the mandible through the mandibular foramen
- At risk for PNS from lower lip primaries or any tumor that invades the mandible
- May be involved in downhill or antegrade PNS
56-y/o man presenting with left “numb chin” syndrome, outside radiologist interpreted MR as osteomyelitis. Dx: lymphoma

Recurrent left lower lip SCCa with proven PNS along the mental/inferior alveolar nerve to level of main trunk V3

Auriculotemporal Nerve

- Branch of mandibular nerve (V3)
- Arises just below foramen ovale
- Provides cutaneous innervation to lateral face, preauricular, external ear, and TMJ
- Courses through stylomandibular tunnel and parotid gland
- ATN and therefore V3 at risk for PNS in 1° or 2° malignancies of parotid gland and skin cancers in its cutaneous distribution
- Evaluation of all parotid malignancies should include foramen ovale and proximal course of V3

Pre-auricular carcinoma, invasive of parotid, with PNS along auriculotemporal nerve to foramen ovale
**PNS-Anatomic Considerations-Facial Nerve**

- Generally related to parotid pathology, either primary parotid malignancy, or lesions, generally skin cancers, that secondarily invade the parotid, at diagnosis or at recurrence.
- Less commonly, skin cancers that have not yet invaded the parotid.
- Beware the subdermal skin cancer, that is difficult to detect clinically.
- When is “Bell’s Palsy” a Bell’s Palsy?
- Must evaluate:
  - peripheral branches
  - SMF
  - descending canal

**How Can Cancer Access the Facial Nerve Perineurally?**

- Via peripheral branches and back into main trunk
- Directly into the stylomastoid foramen
- Back along GSPN (to follow)

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**Relationship Between CN 5 and 7**

- Distal branches of V serve as conduits for small branches of VII and IX. These represent real or potential routes of PNS:
  - Potential sources of PNS:
    - chorda tympani-tongue, SM/SL glands
    - LSPN (runs with auriculotemporal nerve)-parotid gland
  - Greater superficial petrosal nerve (GSPN)
    - Branch of CN7 originating in nervus intermedius
    - Preganglionic fibers, motor root of SP ganglion
    - Post ganglionic supply to palate, nasal, lacrimal
  - Potential for perineural tumor spread quite real
Course of the GSPN

Facial hiatus
Geniculate ganglion
Vidian canal
Foramen lacerum

43-y/o woman with right facial pain and numbness. Dx: ACCa of the hard palate with PNS to the PPF and vidian nerve

69-y/o man with ACCa left nasomaxillary. Spread to PPF facilitates PNS to vidian and rotundum
62-y/o woman with several year history of left ear discomfort and placement of tympanostomy tube for treatment of eustachian tube dysfunction. More recently developed left trigeminal sensory neuropathy, and oh yeah, just noticed she’s NOT TEARING FROM THE LEFT EYE. Outside brain MR read as cavernous sinus meningioma, patient referred for proton therapy.

Repeat imaging obtained primarily for XRT planning. How to make a very long story short…?

Dx: ACC NP

Spinal, Uncommon Nerve PNS

- Spinal
  - great auricular nerve
  - supraclavicular nerve

- Uncommon
  - small cranial nerves

Great Auricular Nerve (GAN)

- Superficial branch of the superficial cervical plexus.
- GAN provides sensory innervation to the skin over the parotid and lower pre-auricular region.
- GAN leaves plexus, courses over and around the SCM (Erb’s point) and then upward toward the ear.
- Has communicating branches with the facial nerve within the parotid gland, and with the auricular branch of the vagus nerve
- GAN at risk for PNS in its cutaneous distribution

68-y/o man with prior skin resections, now with recurrent SCCa left parotid, and PNS along auriculotemporal and greater auricular nerve

72-y/o man with multiply recurrent SCCa left face, prior extensive Rx for V1 PNS to cavernous sinus

Now with recurrence over the left lower parotid region
Supraclavicular Nerves

- Branches of cervical plexus
- Formed by twigs from C3, C4 spinal nerve ventral rami
- Provide sensory innervation to skin over the clavicle, anteromedial shoulder, upper chest
- Anterior, posterior, intermediate branches

Rare Nerves—advanced, slow growing malignancies can spread to 3rd, 6th cranial nerves, maybe others, if cavernous sinus involved

- Advanced recurrent lacrimal ACCa
- Advanced recurrent facial melanoma, already had cavernous sinus disease

Conclusion

- Perineural tumor spread is a very serious and potentially life-threatening complication of head and neck cancer
- Because it may be asymptomatic at presentation or masked at recurrence due to prior therapy, it is critical that the radiologist make the diagnosis
- Diagnosing PNS requires careful attention to imaging technique and a solid understanding of the relevant neuro-anatomy