

Patient-Centered Approach to Matching Symptoms to Interventions for Headache Disorders - The Old and the New

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Objectives

- Review the presentations of several types of common headache disorders
- Review advances in our understanding of the pathophysiology of certain headache disorders
- Describe current approaches to managing headache disorders through a case-based approach

Goals of Migraine Management

Relieving	Relieving symptoms and <u>restoring function</u>
Reducing	Reducing headache <u>frequency and severity</u>
Reducing	Reducing headache-related <u>disability</u>
Preventing	Preventing <u>disease progression</u>

- **Acute Treatment**

- Achieve rapid pain relief during an attack, improve function and reduce disability.

- **Preventative treatment**

- Reduce migraine attack frequency, severity and disability.

Silberstein SD. *Continuum* (Minneapolis Minn). 2015;21(4 Headache):973-989.

Silberstein SD. *Neurology*. 2000;55(6):754-762.



RAPID AND
CONSISTENT FREEDOM
FROM PAIN
ASSOCIATED
SYMPTOMS WITHOUT
RECURRENCE



RESTORED ABILITY TO
FUNCTION



MINIMAL NEED FOR
REPEAT DOSING OR
RESCUE MEDICATIONS.



REDUCTION IN
HEALTHCARE
RESOURCE USE



MINIMAL OR NO AEDS



ALL PEOPLE WITH
MIGRAINE SHOULD BE
OFFERED A TRIAL OF
MIGRAINE SPECIFIC AS
NEEDED TREATMENT.

American Headache Society Goals of Acute Treatment of Migraine

“Migraine Acute Treatment Tool Box”

Nonspecific Agents

- NSAIDS
- Acetaminophen
- Combination Analgesics
- Anti-emetics
- Muscle relaxants
- Opioids

Migraine Specific

Ergotamines

- Dihydroergotamine IV/NS/IM (Trudhesa)
- Oral ergotamine

Triptans

- Sumatriptan, naratriptan, zolmitriptan, frovatriptan, eletriptan, almotriptan, rizatriptan

Specific NSAIDS

- Diclofenac Potassium oral solution
- Celexocib Oral Solution

Ditans

- Lasmitidan

Gepants

- Ubrogепant, Rimegepant

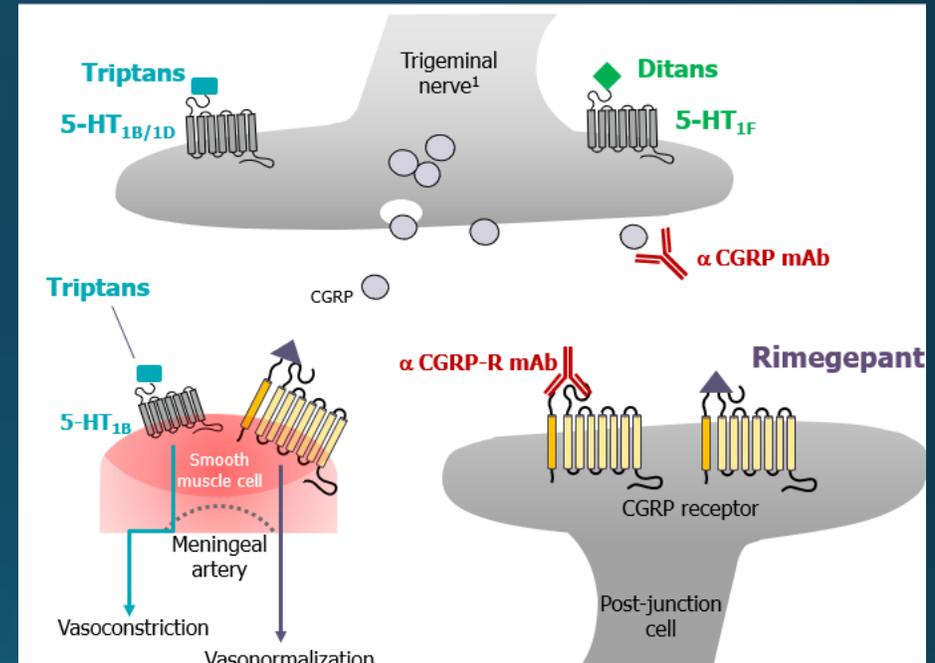
Mechanism of Action

Triptans predominantly exert an agonist action at 5-HT_{1B} and 5-HT_{1D} receptors

Ditans exert an agonist effect on the 5-HT_{1F} receptors

Anti CGRP monoclonal antibodies bind and inhibit either the CGRP receptor or CGRP itself

Gepants are small molecules that bind to and antagonize the CGRP receptor, without causing any vasoconstrictive effects.



Dihydroergotamine (DHE)

~40% 2h Pain freedom*

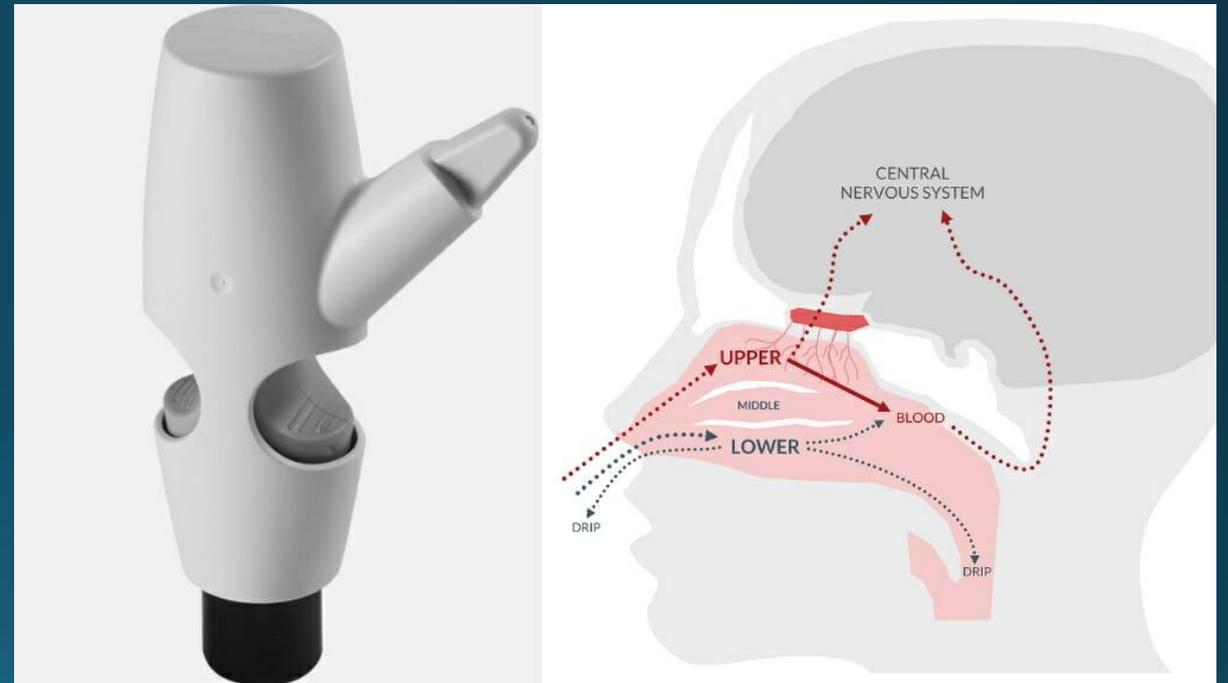
- Nasal

- Old = Migranal
- New POD device = TRUDHESA
 - FDA approved
 - Drug levels similar to IV

- Parenteral

- 1mg SC/IM
- 1mg IV q8h or continuous IV
- A/E = Nausea

- NOT within 24h of a triptan



<https://clinicaltrials.gov/ct2/show/NCT03557333>

Clinical Efficacy

	Ubrogepant	Rimegepant	Lasmiditan	TRIPTANS	DHE NS
Dosing	50 mg, 100 mg tabs	75 mg ODT	50 mg, 100 mg tabs	Oral, NS, IM	0.5 mg per acutation
Pain freedom	21.2% 100mg vs 20.5% 50mg vs 13% placebo	21% 75 mg ODT vs 11% placebo	28% 100mg 31% 200mg vs 21% placebo	30-50% tabs 50% NS 70% Suma IM	47% vs 33% placebo
Pain Relief	61% vs 61.4% vs 49% placebo	59% vs 43% placebo	55.3% 200mg 54% 100mg vs 45% placebo.	30-60%	61% experienced lasting relief for 24 hrs
AE	Nausea, sedation @ less than 4%	Nausea, sedation @ less than 4% ***sucrolose	No driving 8 hrs dizziness with 200mg dose at 17%	Flushing, chest tightness, jaw tightness, sedation, contraindicated with active vascular disease	Life-threatening peripheral ischemia with coadministration of with potent CYP3A4 inhibitors including protease inhibitors and macrolide antibiotics.

Zavegepant

- Phase 2/3 data show good results
- 5 mg dose not statistically different than placebo
- 10 mg and 20 mg doses both statistically superior to placebo for: 2-h pain freedom (22.5%, 23.1% vs 15.5%) and Freedom from most bothersome sx's (41.9%, 42.5% vs 33.7%)
- Adverse events: dysgeusia, nasal discomfort

Medications with evidence for prevention

Established efficacy ^b		Probably effective ^c	
Oral	Parenteral	Oral	Parenteral
Candesartan	Eptinezumab	Amitriptyline	OnabotulinumtoxinA + CGRP mAb ^{d,e}
Divalproex sodium	Erenumab	Atenolol	
Frovatriptan ^f	Fremanezumab	Lisinopril	
Metoprolol	Galcanezumab	Memantine	
Propranolol	OnabotulinumtoxinA ^d	Nadolol	
Timolol		Venlafaxine	
Topiramate			
Valproate sodium	rimegepant	Oral preventative + Onabotulinum Type A	

Atogepant

- FDA approval: 9/28/21, after guidelines published
- Indication: Preventive treatment of episodic and chronic migraine in adults
- Route of administration: Oral tablet
- Dosing: 10 mg, 30 mg, or 60 mg daily, with or without food
- 3.7 to 4.2 decrease in monthly migraine days over 12 weeks
- AEs (>4% and more common than placebo)
 - Nausea
 - Constipation
 - Fatigue

Rimegepant

- FDA approval: 5/21/21
- Indication: Preventive treatment of episodic migraine in adults
- Route of administration: Oral tablet
- Dosing: 75 mg ODT every other day, with or without food
- 4.3 day reduction in migraines during weeks 9 to 12.
- AEs (>2% and more common than placebo)
 - Nausea
 - Abdominal pain/dyspepsia

	Erenumab	Fremanezumab	Galcanezumab	Eptinezumab
FDA Approval	May 2018	September 2018	September 2018	April 2020
Target	CGRP -R	CGRP	CGRP	CGRP
Route of Administration	SQ autoinjector	SQ Autoinjector Prefilled Syringe	SQ Autoinjector Prefilled Syringe	Infusion 30 minutes
Dosing	70 mg, 140 mg	225 mg Q monthly OR 675 Q 90 days	240 mg loading, 120 mg SQ thereafter	100 mg IV OR 300 mg IV
Cost	\$575/month	\$575/month or \$1,725 quarterly	\$575/month	\$1495/100 mg + infusion center costs
Common AE	Constipation, muscle spasm	Injection site reaction	Injection site reaction	Injection site reaction, hypersensitivity

Monoclonal antibodies

Table 2. Efficacy outcomes in phase II and phase III clinical trials using mAbs anti-CGRP for the prevention of **chronic migraine**.

	Change in migraine days			% of participants achieving 50% migraine days reduction
	active	placebo	Δ (p-value)	
Erenumab (Phase II) (70, 140 mg)	-6.6	-4.2	-2.4 (<0.0001)	Active 70 mg: 40%* Active 140 mg: 41%* Placebo: 23%
Galcanezumab (Phase III) (120, 240 mg)	-4.8 -4.6	-2.7	-2.1 -1.9 (<0.001)	Active: 28% Placebo 15%
Fremanezumab	Phase IIb (675/225, 900 mg)	-4.2	-1.8 -1.9 (0.0023)	Active: 53.0%; 55.0%* Placebo: 31.0%
	Phase III (225, 675 mg ^{**})	-4.6 -4.3	-2.5 -1.8 (<0.001)	Active: 41.0%; 38.0%* Placebo: 18.0%
Eptinezumab (Phase III) (100, 300 mg)	-7.7 -8.2	-5.6	-2.1 -2.6 (<0.0001)	Active: 57.6%; 61.4%* Placebo: 39.3%

*Statistically significant difference compared to placebo

**One single injection quarterly

Prevention for Chronic Migraine

- Topiramate
- Valproic acid
- Beta- blockers
- TCAs
- Onabotulinum Type A
- Erenumab 70 mg or 140 mg SQ monthly
- Fremanezumab 225 mg SQ monthly or 675 mg SQ every 90 days
- Galcanezumab 240 mg SQ loading dose, 120 mg SQ monthly
- Eptinezumab 100mg or 300mg IV SQ 90 days
- Atogepant – under FDA review currently.

Ailani J et al. *Headache*. 2021;61(7):1021-1039.

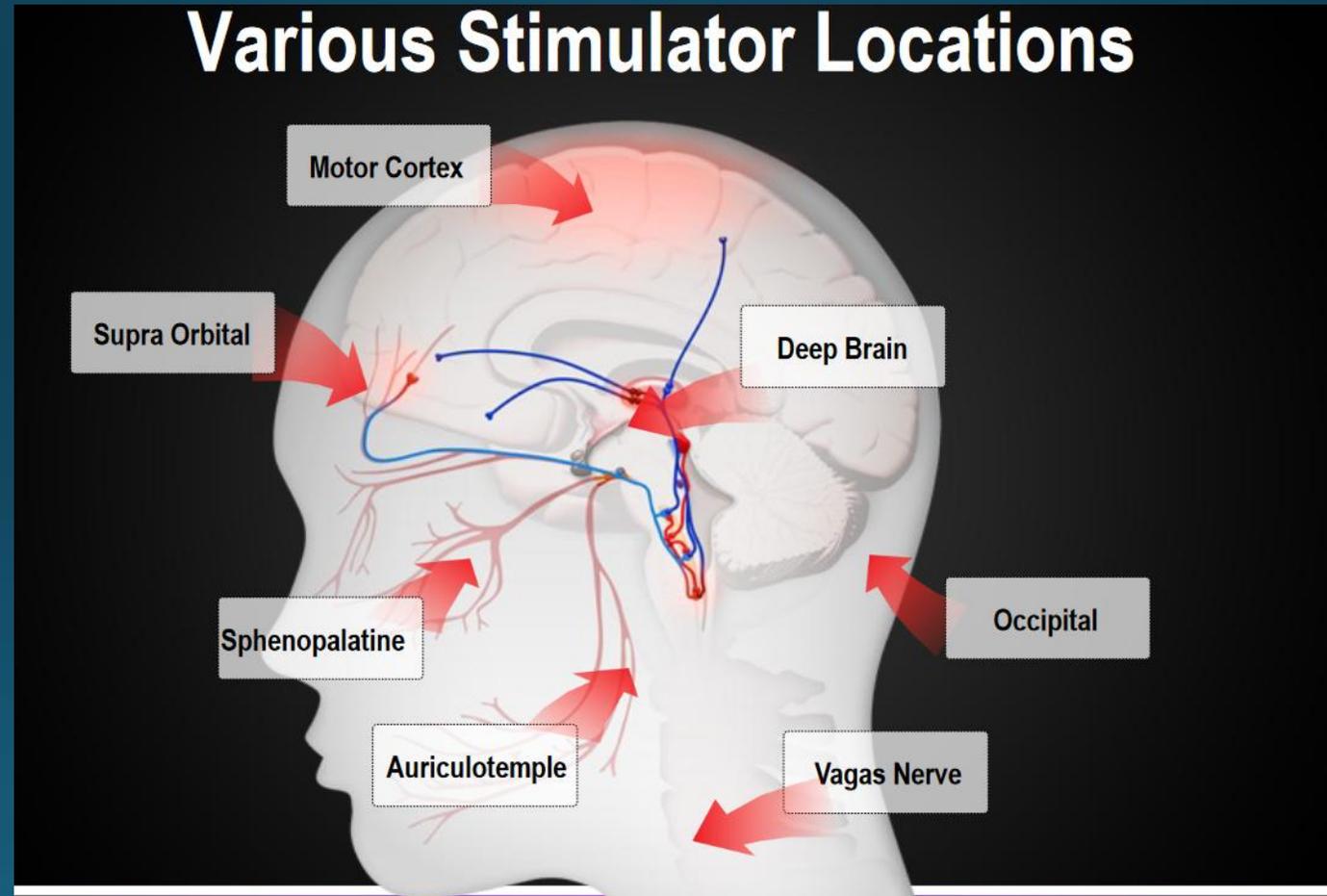
American Headache Society. *Headache*. 2019;59(1):1-18.

Diener H-C et al. *Curr Pain Headache Rep*. 2011;15(1):64-69.

Nutraceuticals

	Daily Dose ^a	American Academy of Neurology Evidence Level for Efficacy ^{29-31,b,c}	Canadian Headache Society Recommendation ^{27,d}	Canadian Headache Society Evidence Level for Efficacy ^{27,d}
Nutraceuticals				
Coenzyme Q10	300 mg	C	Strong	Low
Magnesium citrate	400-600 mg	B	Strong	Low
Riboflavin	400 mg	B	Strong	Low
Feverfew	50-300 mg	B	Strong against	Moderate

Neuromodulation



Non-Invasive Vagus Nerve Stimulation

- Hand-held device
 - – (not implanted)
- Applies pulsed stimulation to the Vagus Nerve in the neck
 - **Well tolerated**
 - **Minimal discomfort**
 - **Platysmus muscle activation implies**
- Correct placement
- FDA cleared for acute migraine, acute cluster ha, adjunctive prevention of migraine



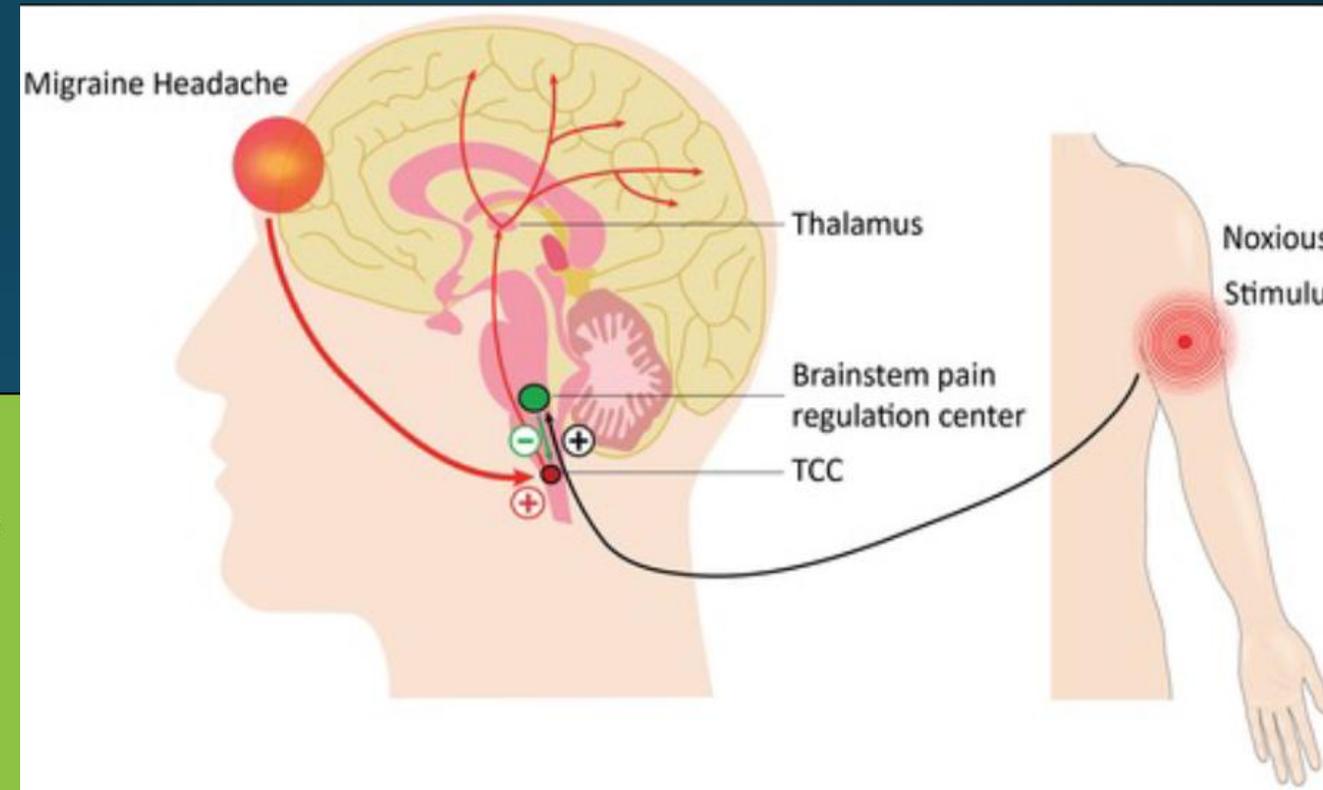
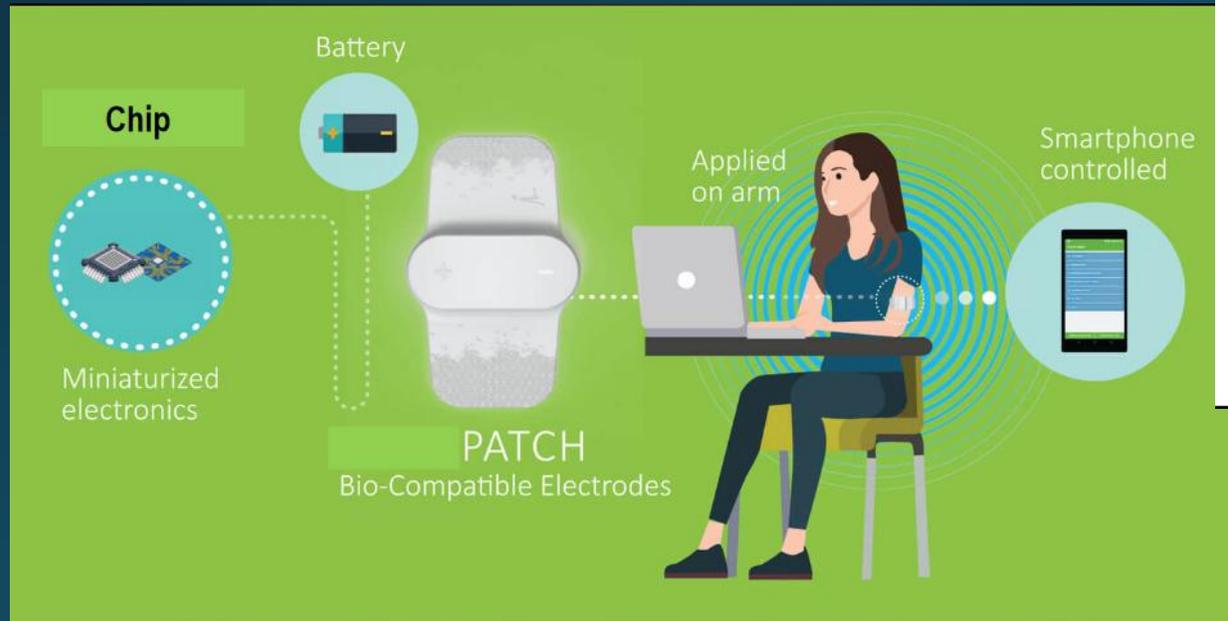
sTMS Transcranial Magnetic Stimulation

- Prevention - Migraine with Aura
 - Mean reduction of 2.8-4.0 headache days
- Acute Migraine with Aura
 - N=201
 - 164 subjects TMS
 - 37 subjects sham device
 - Pain Free
 - 2 hours (38% vs 17%)
 - 24 hours (34% vs 10%)
- sTMS mini™ is indicated for the acute and prophylactic treatment of migraine headache in adolescents (age 12 and older) and adults



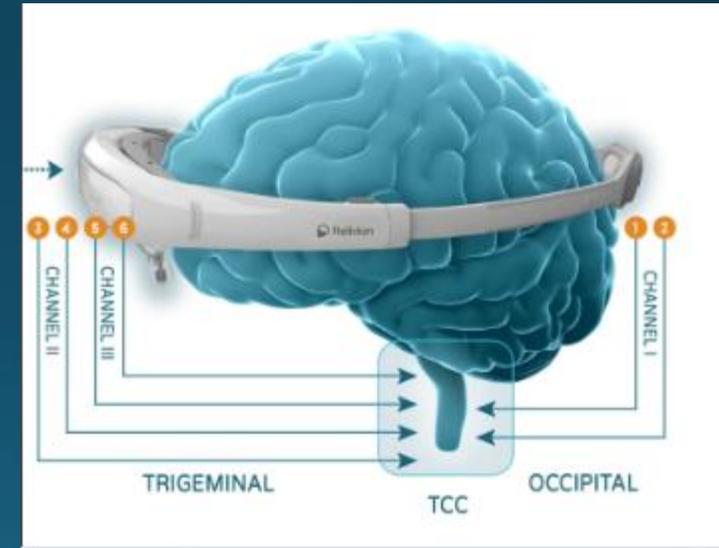
Remote Electrical Neuromodulation

- Approved for acute treatment with or without aura in adults.
- 296 participants
- 66.7% of patients achieved pain relief versus 38.8% in sham, sustained benefits out to 48 hours.

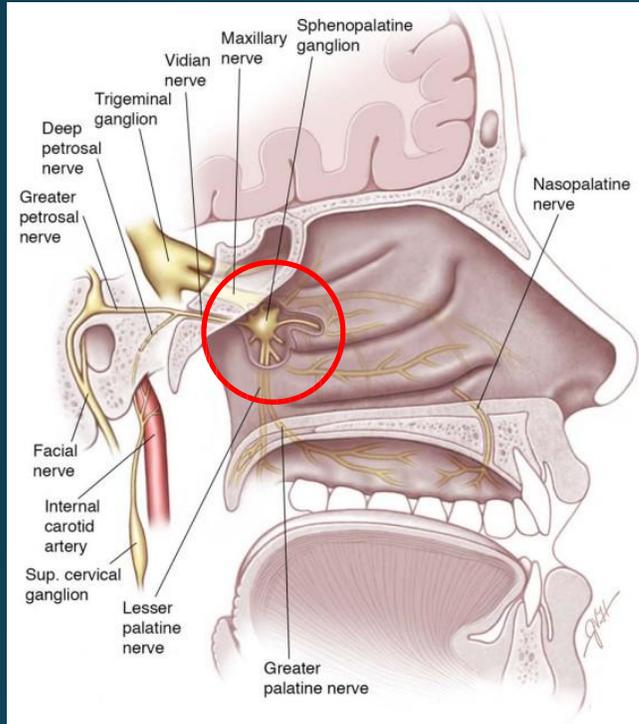


Peripheral stimulation of both trigeminal nerves and occipital nerves

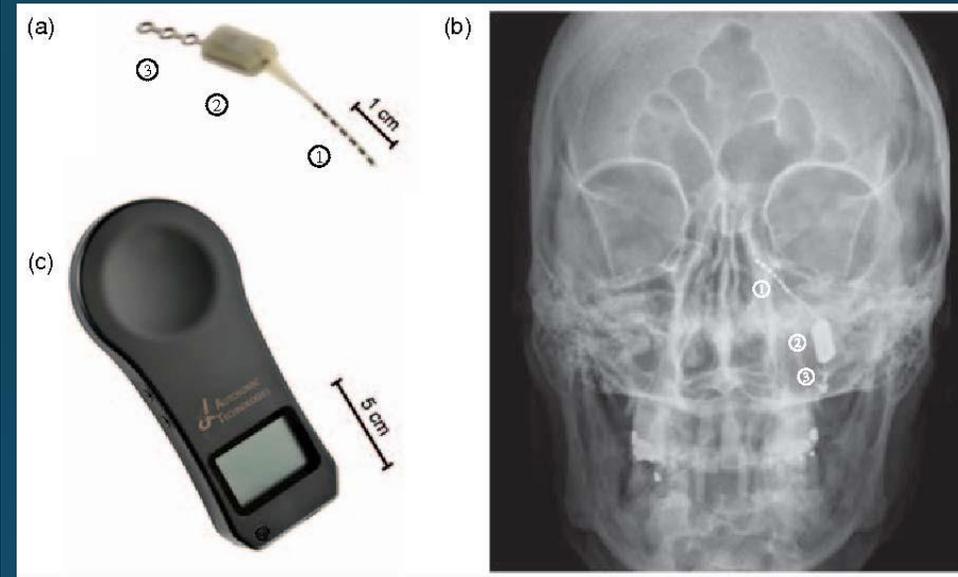
- Acute treatment of migraine
- Pain-free :
 - 1 h 29.2% vs. 16%
 - 2 h 41.7% vs. 20%.
- Headache relief:
 - 1 h 67% vs. 26% 2 h
 - 66.7% vs. 32%
- Adverse effects:
 - headache ($n = 2$), numbness/paresthesia ($n = 2$), skin irritation ($n = 1$), nausea ($n = 1$), and itching scalp ($n = 1$)



Sphenopalatine Ganglion Stimulation



Robbins MS et al. Headache. 2016

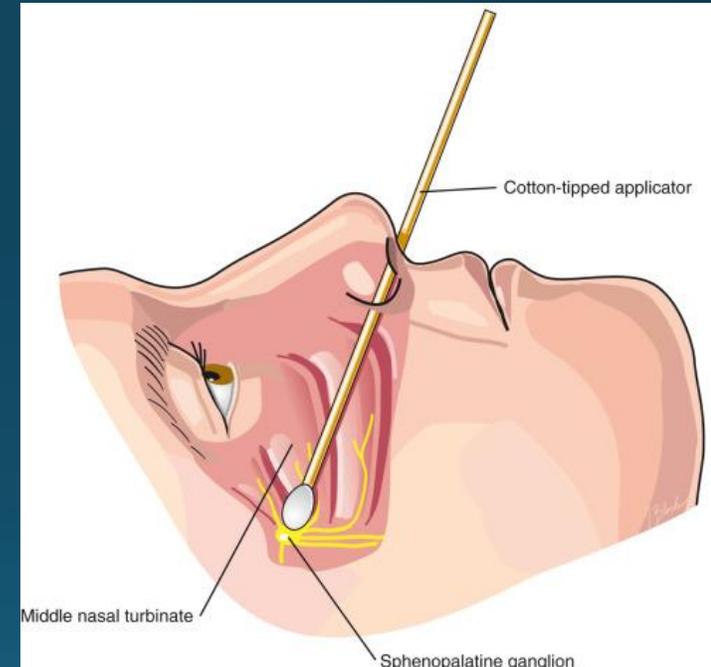


Schoenen J et al. Cephalalgia. 2013

- SPG connects with autonomic nerves
- During a migraine, PNS outflow causes vasodilation, release of inflammatory mediators
- **SPG blockade/modulation may inhibit propagation of aberrant autonomic signals**

Sphenopalatine ganglion block

- SPG located in the pterygopalatine fossa (parasympathetic, sensory and sympathetic contributions)
- Useful for cluster headache and post-dural puncture headache management, migraines, trigeminal autonomic cephalgias
- Results in anesthesia of area supplied by the maxillary branch of the 3rd cranial nerve (trigeminal nerve)
- 2 approaches: 1) injection around ganglion through intraoral pass way (inferozygomatic approach)
- 2) topical placement of local anesthetic on the nasal mucosa overlying the SPG in the postero-lateral nasal wall
- Can be ablated as well**



<https://www.sciencedirect.com/topics/medicine-and-dentistry/sphenopalatine-ganglion-block>

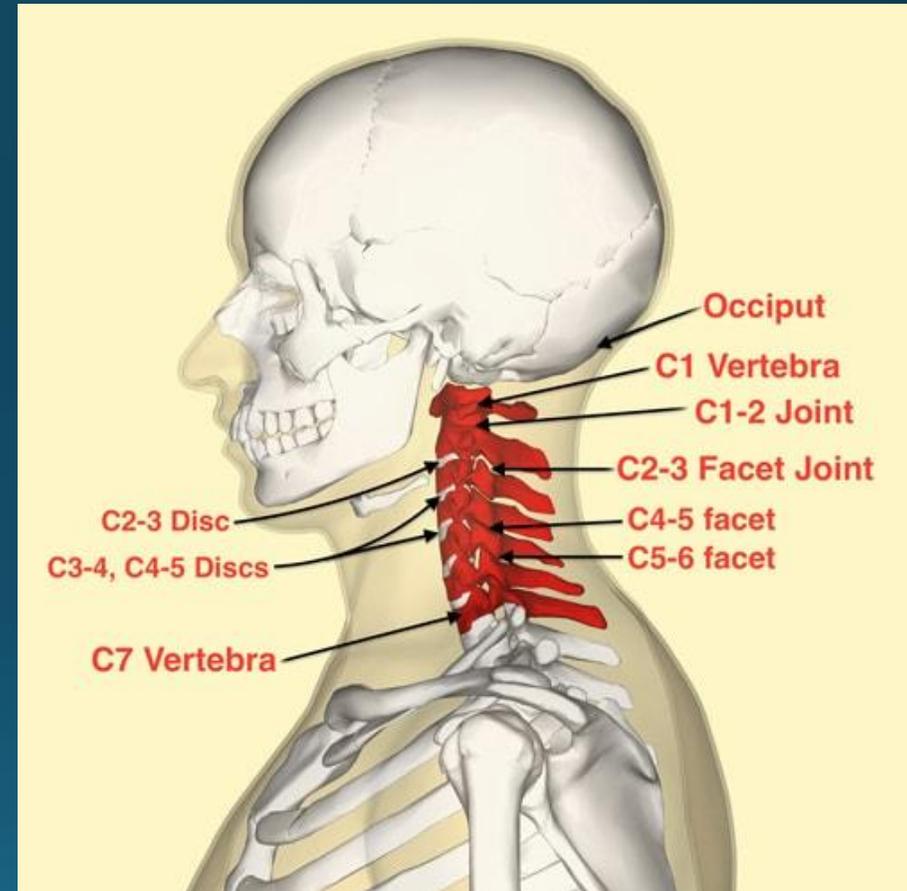


When to consider an
interventional therapy?

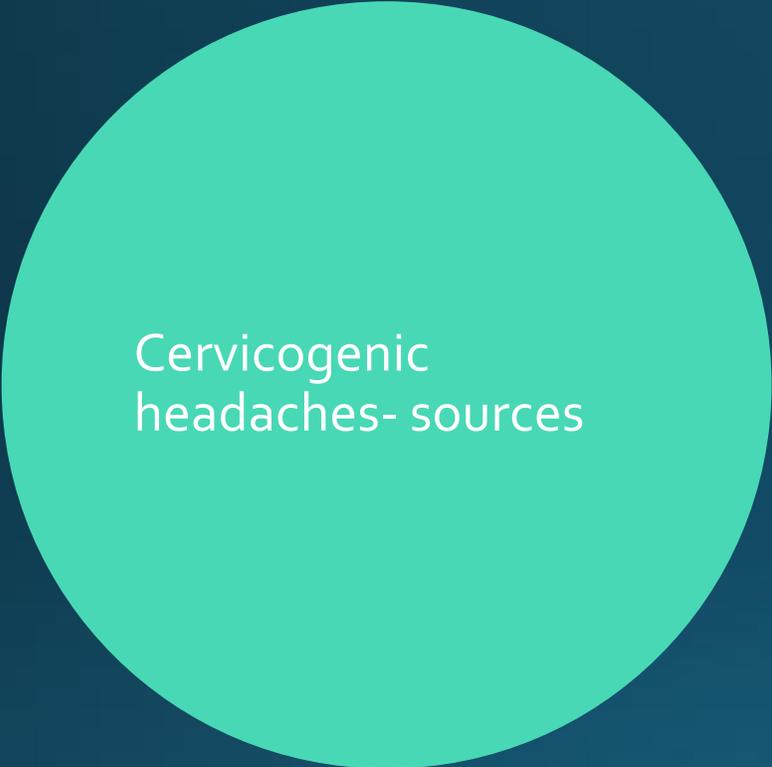
- Pharmacological therapies are the mainstay for headache management.
- Sometimes, agents cannot be used due to co-morbid medical conditions, medication interaction
- If patients fail medications, PT, CBT, stress management, acupuncture
- For such patients, interventional therapy can be effective.

Cervicogenic headaches

- SECONDARY HEADACHE DISORDER, unilateral or bilateral
- Originates in areas innervated by C1-3 spinal nerves
- 1. Must have pain with neck movement or sustained "awkward" positioning
- 2. Pain with applied pressure over the upper cervical or occipital region on the affected side.
- 3. restricted neck range of motion, provocative measures cause pain (facet loading)
- 4. ** Positive response to anesthetic blockade of the cervical spine nerves.



<https://headacheheal.com/treatments-conditions/cervicogenic-headaches/>



Cervicogenic
headaches- sources

- Pain is referred from first 3 cervical spinal nerves
- Possible sources of cervicogenic headache:
 - Atlanto-occipital joint
 - Atlantoaxial joints
 - C2-3 facet joint
 - C2-3 intervertebral disc
 - Upper cervical spinal nerves and roots
 - ** other more serious causes of occipital headaches should be ruled out – posterior cranial fossa lesions and vertebral artery dissection or aneurysm.

Trigemino-cervical complex

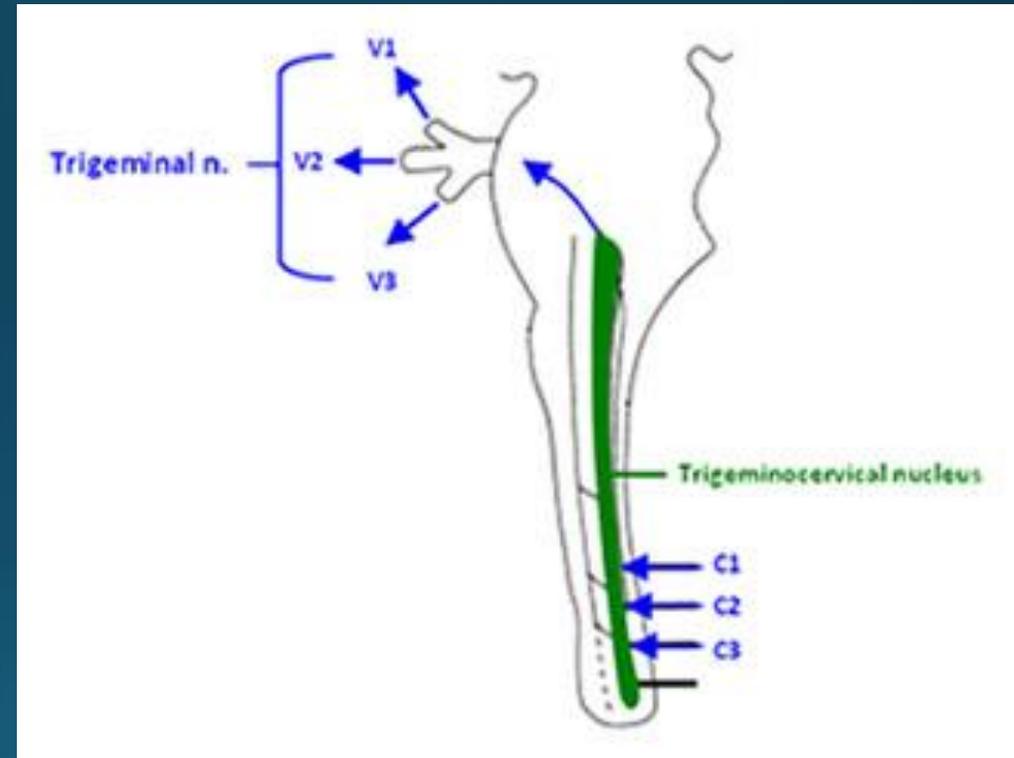


Table 2

Diagnostic criteria for cervicogenic headache according to IHS^[18].

- (A) Pain, referred from a source in the neck and perceived in 1 or more regions of the head and/or face, fulfilling criteria C and D
- (B) Clinical, laboratory, and/or imaging evidence of a disorder or lesion within the cervical spine or soft tissues of the neck known to be, or generally accepted as, a valid cause of headache
- (C) Evidence that the pain can be attributed to the neck disorder or lesion based on at least one of the following:
 - (1) Demonstration of clinical signs that implicate a source of pain in the neck
 - (2) Abolition of headache following diagnostic blockade of a cervical structure or its nerve supply using placebo or other adequate controls
- (D) Pain resolves within 3 months after successful treatment of the causative disorder or lesion

IHS = International Headache Society.

Adapted from^[18].

Cervicogenic headaches:C2 NEURALGIA



Caused by lesions affecting the C2 nerve root or dorsal ganglion



le: neuroma, meningioma, anomalous vessels.



C2 nerve root lies posterior to the lateral AA joint – irritation and entrapment of the nerve are possible.



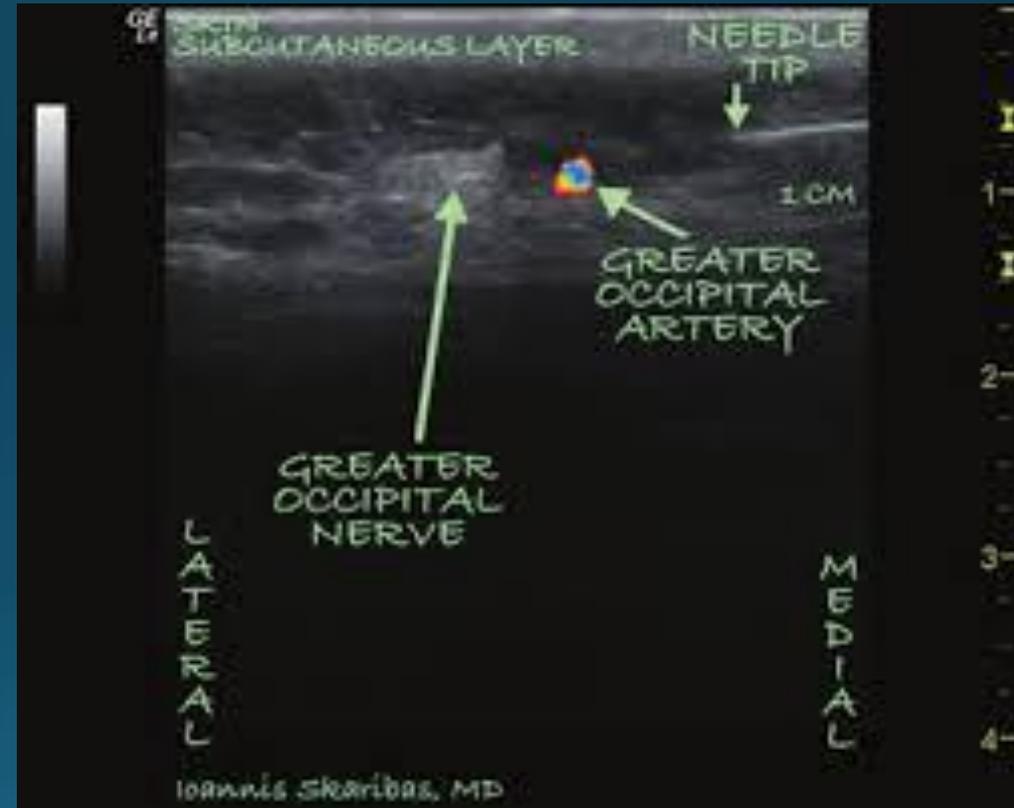
C2 neuralgia - intermittent lancinating occipital pain associated with lacrimation, ciliary injection and rhinorrhea.



Can selectively block the C2 nerve root, thermocoagulation, decompression also options

Occipital nerve blocks

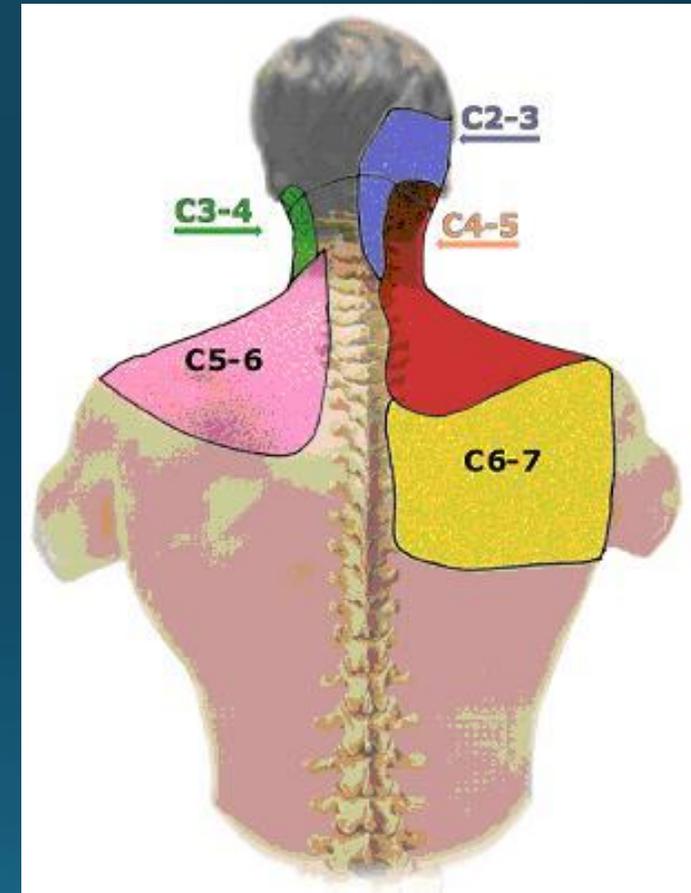
- **Occipital neuralgia**: sharp pain in the occipital region which arises from the greater and lesser occipital nerves
- Percutaneous ONB are low risk, minimally invasive therapies to manage occipital neuralgia
- Greater occipital nerve block- for pain in the parietal and occipital areas
- Lesser occipital nerve block- for pain in the frontal and temporal areas
- Ultrasound or blind techniques can be used, local anesthetic injected
- Occipital nerve stimulation



<https://migrainecenters.com/assets/Ultrasound.pdf>

Cervical facet blocks/Medial branch blocks and Radiofrequency

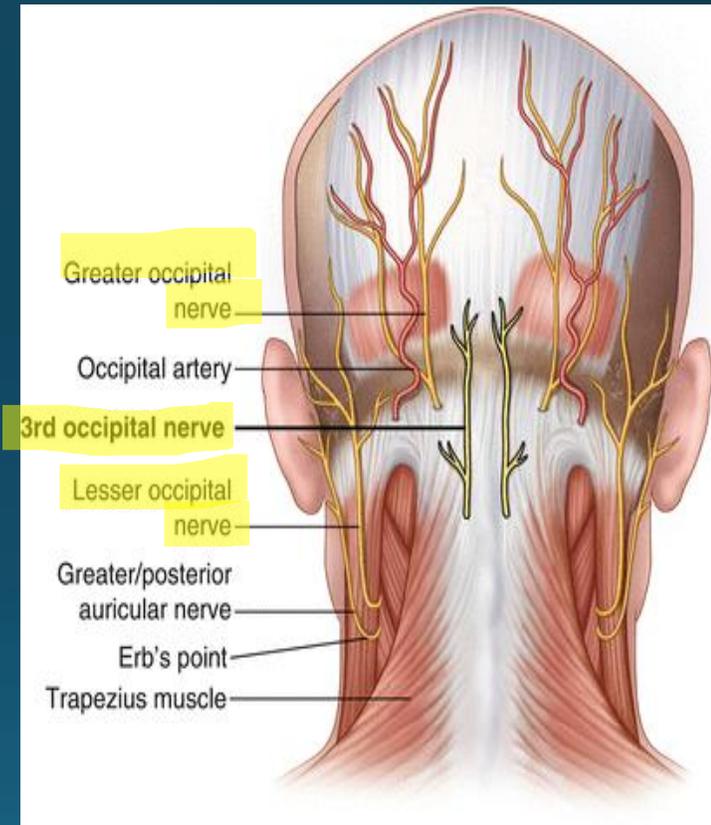
- **TON (third occipital nerve)** superficial branch from the dorsal ramus of C₃
- Courses deep into the semispinalis capitus muscle
- Lies adjacent to the greater occipital nerve
- ** Primary afferent channel along which the C_{2/3} cervical zygapophysial joints send their sensory input
- Pain from C_{2/3} and TON referred to occipital region as well as frontotemporal and periorbital regions. (vulnerable to injury from whiplash)
- Remaining cervical facet joints are innervated by 2 medial branches that arise from the dorsal rami of 2 successive spinal nerve roots



<https://centenoschultz.com/cervical-facet-syndrome-a-pain-in-the-neck/>

Cervical facet blocks/Medial branch blocks and Radiofrequency

- Typically, a local anesthetic is injected over the location of the TON or medial branches
- Ie: 0.5cc of bupivacaine or lidocaine
- Patient is asked to keep a pain log for several hours after the procedure
- 2 sets of blocks may be performed to improve patient selection for radiofrequency ablation
- Must demonstrate significant pain relief >50% for the duration of local anesthetic effect to qualify for radiofrequency ablation



Cervical radiofrequency ablation

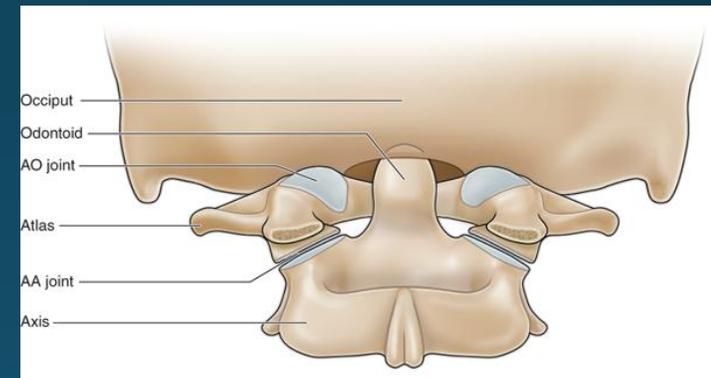
- RFA is done to generate a thermal current within the desired nerve essentially destroying the afferent pain pathway (80 degrees celsius)
- Sensory and motor testing is done to ensure that only the medial branch is ablated
- Minimal sedation for this reason
- Results can vary but >90% of patients appropriately selected for RFA have been reported to experience improvement in their headaches. ²



<https://www.bsmedicine.com/lumbar-cervical-radiofrequency-ablation-lesioning/>

Atlantoaxial joint pain – cervicogenic headache

- Lateral AA joint accounts for 16% of patients with CGH due to osteoarthritis or trauma
- Present with occipital or suboccipital pain, focal tenderness over the suboccipital area, restricted painful rotation of C1 on C2 and pain with passive rotation of C1.
- Lateral AA joint injection with local anesthetic and steroids can be effective for diagnosis and short term pain relief. Fluoroscopy, live injection with contrast vs. ultrasound.
- ** avoid injury to the vertebral artery, C2 dorsal root ganglion and nerve root to avoid serious complications



<https://accessanesthesiology.mhmedical.com/content.aspx?bookid=1158§ionid=64176391>

Cervical myofascial pain

- Pain from myofascial trigger points: small highly sensitive areas in muscle characterized by hypersensitive, palpable, taut bands of muscles that are tender on palpation, reproducing the patient's symptoms and cause referred pain.
- 4 most common muscles responsible for cervical myofascial headaches that include the : trapezius, sternocleidomastoid, splenius capitus, and temporalis.
- ? Excess release of acetylcholine which leads to sustained contraction of muscle to form a trigger point
- Tx: trigger point injections, dry needling vs. local anesthetic, botox



<https://www.njpaindoc.com/myofascial-pain-syndrome.html>

Patient scenarios to consider

Considerations for choice of treatment

- Frequency of Headache- are you thinking about abortive or preventative or both?
- Age
- Child bearing status
- Past treatments- were they effective? Well tolerated? Easy to adhere to?
- Headache/Medical/Other Co-morbidities
- Consider likelihood of adherence, drug interactions, risk of side effects
- Access

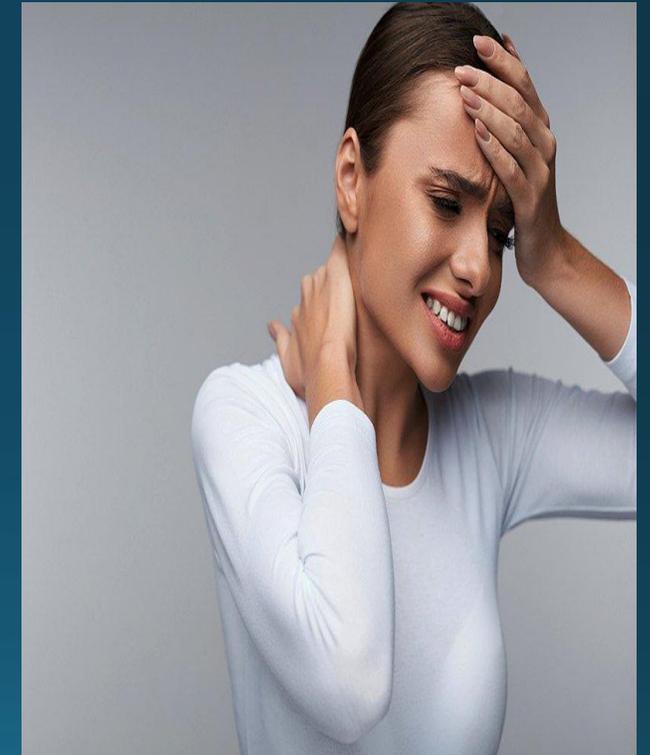
Patient with migraine diagnosis for multiple years- new pattern

HPI: 46 yr RHF originally presented in 2016 for routine f/u for episodic migraine. She typically experiences pain as well as photophobia, phonophobia, mild nausea- intensity of symptoms can increase around menses.

Now returns with near daily headaches. This is a clear change for her over the prior 6 months.

Pain radiates up from the base of the skull/right occiput and shoots forward. Also has tenderness in b/l temples "dull", not throbbing

Additionally, has neck pain, "woke up one day with it and it just won't go away". No known trauma



PMH

- Seizure disorder
(sz free since 2012)
- Migraine
- Anxiety

Allergies

- NKDA

Medications

- Propranolol LA 60 mg QD
- Frovatriptan 2.5 mg PRN
- Lamotrigine 200 mg BID
- Sertraline 50 mg daily
- Vitamin D

Physical Examination

- VS: BP 106/72 HR 54 RR 14 HT 5'4" WT 133 lb Pain intensity 0/10
- Neurologic exam: non-focal
- General examination: **Tenderness** to palpation along the right occiput with radiating pain; **Tenderness** along the b/l trapezius R>L with increased muscle tone, pain with movement of the neck and **reduced range of motion** of her neck due to pain with movement.

What are your thoughts?

Diagnosis?

Further workup?

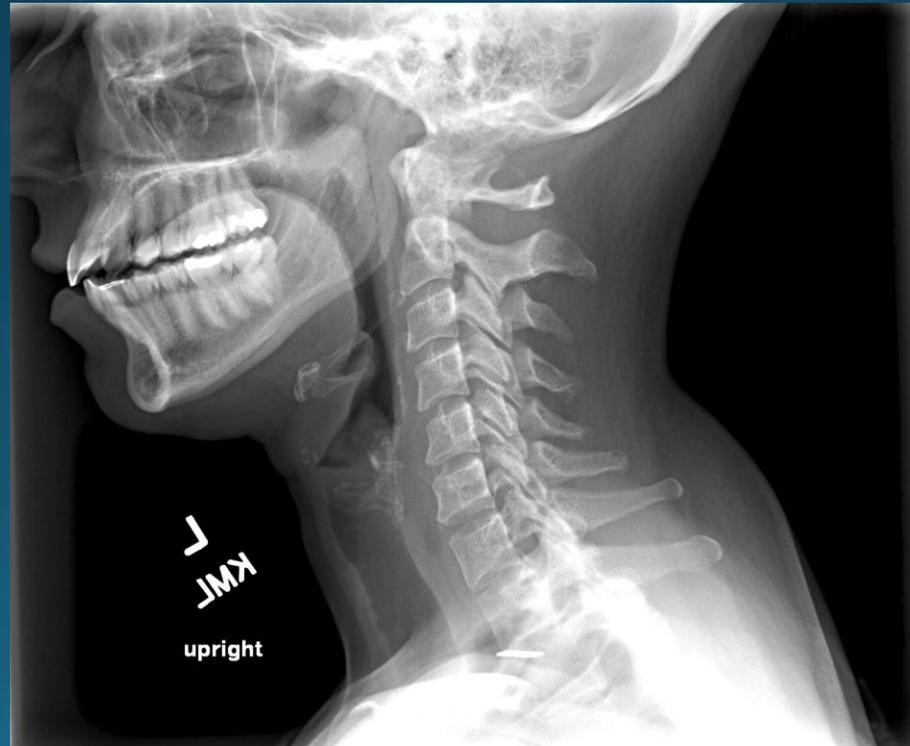


<https://cdn.mamamia.com.au/wp/wp-content/uploads/2016/03/29215315/puppy-head-tilt-feature-.jpg>

Imaging

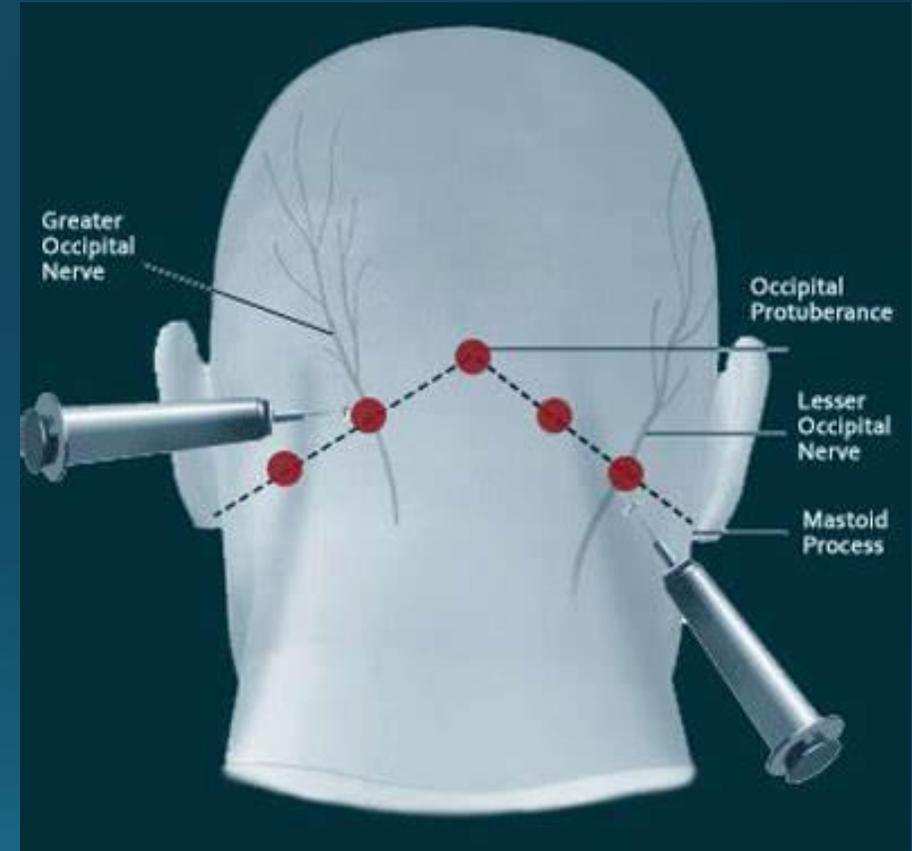
MRI brain: Normal

Cervical spine XR:
Normal



Treatment:
Right Occipital nerve block

Continued her migraine prevention (propranolol), and abortive (frovatriptan)



<https://www.bing.com/images/search?view=detailV2&id=8303052E0B9D134AA6CC84EB876CDED5CA0A00&thid=OIP.CgouEt1ZpsGV0PQ8k53yGgAAAA&mediaurl=https%3A%2F%2Fmkocompletepainnrlau.kinstacdn.com%2Fwp-content%2Fuploads%2F2016%2F05%2FOccipital-Nerve-Block-Location.gif&exp=380&expw=396&q=Occipital+Nerve+Block+Injection&selectedIndex=0&ajaxhist=0&vt=0&eim=1,2,6>

Additional treatment

- Physical Therapy and Massage therapy
- Tizanidine 4mg po qhs
- Topical lidocaine
- Scheduled follow up
- Consideration of interventional pain management

Cervicogenic headache occurring in a person with known migraine

“A referred pain perceived in the head, with a source in the neck.”

- Seen in 0.4-2.5% of the general population
- 15-20% of chronic headache population
- Average age of onset, 4th decade

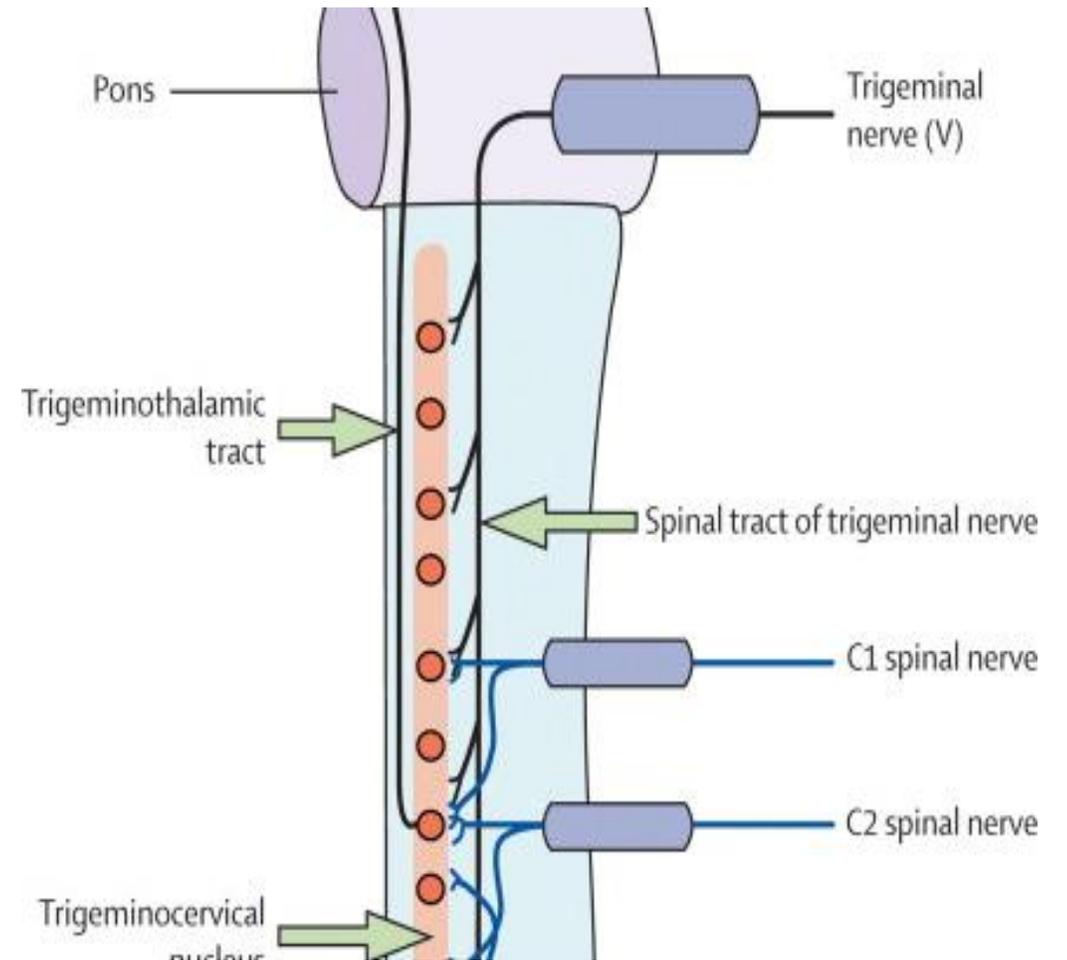
Cervicogenic headache

- Trigeminothalamic nucleus
 - Not just a headache associated with neck pain

Diagnostic Criteria

ICDH 3 11.2.1 Cervicogenic headache

<https://ichd-3.org/11-headache-or-facial-pain-attributed-to-disorder-of-the-cranium-neck-eyes-ears-nose-sinuses-teeth-mouth-or-other-facial-or-cervical-structure/11-2-headache-attributed-to-disorder-of-the-neck/11-2-1-cervicogenic-headache/>



Cervicogenic Headache

Key features

- Unilateral
- Nonthrobbing, nonlancinating, non-radicular
- Restricted range of motion
- Ipsilateral neck, shoulder, arm pain
- Fluctuating intensity, radiate from occipital to frontal
- Cervical spine imaging not sensitive enough for diagnosis



<https://www.uppercervicalhealthcentersboise.com/occipital-neuralgia--occipital-headache.html>

Cervicogenic Headache

Treatment

- Nerve blocks
- Manual therapy
- Steroid injections
- Liberation/decompression of the nerve
- Radiofrequency neurotomy
- Botox
- Myofascial trigger point injections
- Dry needling
- Behavioral
 - Biofeedback
 - Relaxation
 - CBT
- Pharmacologic treatment
 - Duloxetine
 - Gabapentin

Outcome

- She had improvement in an expected time frame
- Continues management of **episodic** migraine, as well as measures employed to treat the cervicogenic headaches

Take home

Appropriate diagnosis and multidisciplinary approach are key

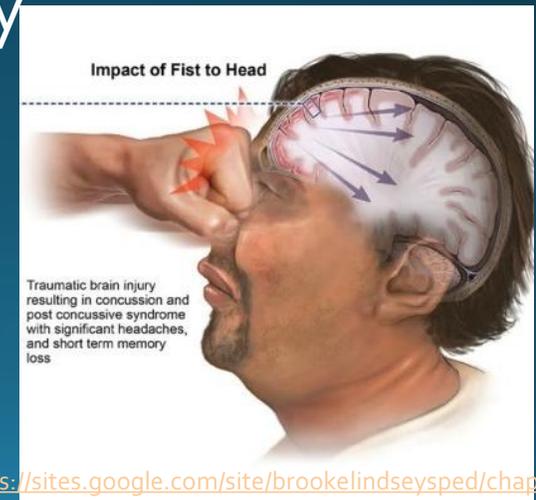
- Primary
- Neuro
- Manual therapy
- Pharmacy
- Interventional



Post concussion/traumatic headache

HPI: BR is a 55 year old RHM presented 3 months after sustaining a concussion at work, wherein he sustained multiple blows (punches/kicks) to the face, head, and neck.

He had a constellation of symptoms consistent with post concussive syndrome including daily headaches, trouble with concentration, memory, attention, irritability, depression, insomnia, imbalance, neck pain.



Symptoms

The headaches:

"whole head pounding"

"sharp shooting sensations sometimes"

+ photophobia/phonophobia/N

Increased pain with exertion, reading, watching TV

Background

- PMH

- Atrial fibrillation
- Stroke (R cerebellar)
- Gastric Ulcer
- Hypothyroidism
- Anxiety

- Social Hx

+TOB

- Allergies:

- Lisinopril: cough
- Sumatriptan: psychosis

- Medications

- Aspirin 81 mg
- Clonazepam 6 mg daily
- Flecainide 100 mg BID
- Losartan 50 mg daily
- Protonix 40 mg daily
- Levothyroxine 88 mcg daily
- Zofran PRN
- Tylenol 500-1000 mg multiple times daily

Physical examination

General Physical Exam

Vital Signs: BP: 140/100. PR: 98. RR: 16. Ht: 5 feet 10 inches
Wt: 236 pounds pain 4/10 headache.

HEENT: Normocephalic. well healed scar above the left eyebrow. Mild left ptosis , subjective blurred vision

Neck: increase muscle tone, and tenderness to palpation along the bilateral paracervical spinal muscles, and trapezius, with mildly reduced range of motion in all planes.

Coordination: positive Romberg

Case 2: BR

What are your thoughts?

Diagnosis?

Further workup?



Case 2: BR

CT/CTA head and neck

Vessel imaging was normal

Old stroke R cerebellum,
otherwise normal scan



Case 2: BR

- Treatment options
 - A short course of steroids reduce his daily Tylenol use
 - He returned 4 months later, still complaining of daily headache, but with reduced severity, and photophobia/phonophobia more sporadic.
- Escitalopram - no impact
- Botox, no difference
- Zonisamide - nausea
- Lamotrigine – rash
- Gabapentin – cognitive complaints
- Magnesium, Vit B2 – no change
- Sumatriptan – psychosis
- Neuropsychology, cognitive therapy
- Neuro-ophthalmology
- Manual therapy

Case 2: BR

He relents!!



<http://jinkchak.files.wordpress.com/2013/02/accomplished.jpg>

Findings:

“During the diagnostic portion of the study: total sleep time 124 minutes. Poor sleep efficiency of 68%. Prolonged sleep latency of 38 minutes. No REM sleep. The apnea hypopnea index is severely elevated at 54/hour. There is evidence of mild intermittent desaturation.”

“Severe obstructive sleep apnea. ”

OSA related headaches

- Can experience upon waking
- Tend to be experienced bilaterally
- Can dissipate when patient wakes, and breathing normalizes
- Regularly recur
- Can induce cluster, hypnic, chronic migraine, tension type
- The occurrence and severity tends to correlate with the severity of OSA

ICDH 3 10.1.4 Sleep apnea headache

<https://ichd-3.org/10-headache-attributed-to-disorder-of-homoeostasis/10-1-headache-attributed-to-hypoxia-andor-hypercapnia/10-1-4-sleep-apnoea-headache/>

Case 2 Outcome

He is doing well,
successfully treated
with CPAP.



Take home

- This patient experienced multiple different types of headache.
- Be mindful of other, common underlying medical contributions to headaches.
- Other things that can fall into this category include:
 - Anemia
 - Thyroid
 - Sinus disease
 - Hypertension
 - Hypoglycemia

Conclusions

- Migraine disease is common
- Multiple recent advances in migraine pathophysiology have led to numerous new migraine mechanism based medical options
- Although these newer treatments have yet to be formally integrated into new treatment guidelines past published data suggest that prior guidelines recommended treatments that although effective for some, were not effective for too many others
- For those experiencing migraine as well as those treating migraine, there have never been more opportunities to collaborate to achieve personalized successful treatment outcomes

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