1. Differential Diagnosis of Craniofacial Pain

**Differential Diagnosis of Craniofacial Pain**

*Steven J. Scrivani*

Associate Professor
The Craniofacial Pain and Headache Center
Tufts University-New England Medical Center
and
Pain and Analgesia Imaging and Neuroscience (P.A.I.N.) Group
Brain Imaging Center
McLean Hospital
Harvard Medical School
2007

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2. International Headache Society

**International Headache Society**

*International Classification of Headache Disorders II (ICHD II) Cephalalgia, 2004*

**14 CATEGORIES**

- The Primary Headaches: 1-4
- The Secondary Headaches: 5-12
- Cranial Neuralgias, central and primary facial pain and other headache disorders: 13
- Others: 14

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3. The Primary Headaches

**The Primary Headaches (1-4)**

1. Migraine
   *without aura
   *with aura
2. Tension-type headache
3. Cluster headache and other trigeminal autonomic cephalalgias
4. Other primary headaches

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4. The Secondary Headaches

**The Secondary Headaches (5-12)**

5. Attributed to head and/or neck trauma
6. Attributed to cranial or cervical vascular disorder
7. Attributed to non-vascular intracranial disorder
8. Attributed to a substance or its withdrawal
9. Attributed to infection
10. Attributed to disorder of homeostasis
11. HA or facial pain attributed to disorder of cranium, neck, eyes, ears, nose, sinuses, teeth, mouth, or other facial or cranial structures
12. Attributed to psychiatric disorder

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5. Headache attributed to head and/or neck trauma

Headache attributed to head and/or neck trauma (5.1-7)

5.1 – Acute post-traumatic HA
   * moderate or severe head injury
   * mild head injury
5.2 – Chronic post-traumatic HA
5.3 – Acute HA attributed to whiplash injury
5.4 – Chronic HA attributed to whiplash injury
5.5 – HA attributed to traumatic intracranial hematoma
5.6 – HA attributed to other head and/or neck trauma
5.7 – Post-craniotomy HA

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6. Headache attributed to cranial or cervical vascular disorder...

Headache attributed to cranial or cervical vascular disorders (6.1-7)

6.1 – Ischemic stroke or TIA
6.2 – Non traumatic intracranial hemorrhage
6.3 – Unruptured vascular malformation
6.4 – Arteritis
6.5 – Carotid or vertebral artery disorder
6.6 – Cerebral venous thrombosis
6.7 – Other intracranial vascular disorders
   (CADASIL, MELAS, pituitary apoplexy)

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7. Headache attributed to a substance or its withdrawal (8.1-4)...

**Headache attributed to a substance or its withdrawal (8.1-4)**

8.1 – HA induced by acute substance use or exposure
   - NO donor-induced
   - PDE inhibitor-induced
   - Alcohol induced
   - HA induced by food components and additives
   - Cocaine induced
   - Cannabis induced

8.2 – Medication overuse HA

8.3 – HA as an adverse event attributed to chronic medication

8.4 – HA attributed to substance withdrawal
   - Caffeine-withdrawal
   - Opioid-withdrawal
   - Estrogen-withdrawal

8. Headache or facial pain attributed to

**Headache or facial pain attributed to disorders of cranium, neck, eyes, ears, nose, sinuses, teeth, mouth or other facial or cranial structures (11.1-8)**

11.1 – Cranial bones
11.2 – Neck
   - Cervicogenic headache
11.3 – Eyes
11.4 – Ears
11.5 – Sinus disorders (“Sinus headache”)
11.6 – Teeth, jaws or related structures
11.7 – TMJ disorders (TMD)
9. Cranial neuralgias, central and primary facial pain and oth...

### Cranial neuralgias, central and primary facial pain and other headaches (13.1-19)

13.1 – Trigeminal neuralgia
13.2 – Glossopharyngeal neuralgia
13.8 – Occipital neuralgia
13.9 – Neck-tongue syndrome
13.12 – Constant pain caused by compression, irritation or distortion of cranial nerves or upper cervical roots by **structural lesions**

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10. Cranial neuralgias, central and primary facial pain and oth...

### Cranial neuralgias, central and primary facial pain and other headaches, cont.

13.13 – Optic Neuritis
13.15 – Head or facial pain attributed to herpes zoster  
*post-herpetic neuralgia*
13.16 – Tolosa-Hunt syndrome
13.18 – Central causes of facial pain  
anesthesia dolorosa  
central post-stroke pain  
*facial pain attributed to multiple sclerosis*  
persistent idiopathic facial pain  
burning mouth syndrome

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11. Unusual Causes of Headache

Unusual Causes of Headache

“Headache is a symptom of many disorders and diseases-systemic and cerebral. It is the manifestation of any pathologic process that leads to:

1. activation of nociceptors on the cranial and dural vessels;
2. dysmodulation of central pain pathways;
3. stimulation of meningeal nociceptors; or
4. stretching/distortion of cranio cervical muscles, joints, or fascia.”


12. “Red Flags” in the Headache History

“Red Flags” in the Headache History

- HA accompanied by unconsciousness
- First-worst HA (appearing suddenly)
- HA accompanied with neurological abnormalities during and/or after the HA
- HA associated with fever or stiff neck
- HA developing after 50 years of age
- A change in characteristic response to previous treatments of HA
- HA associated with alterations in behavior and personality
- HA initiated by Valsalva maneuver
13. **"SSNOOPP" for Secondary HA**

- Systemic symptoms
- Secondary risk factors – underlying disease
- Neurological symptoms or abnormal signs
- Onset: sudden, abrupt, or split-second ("First, Worst")
- Older: new onset and progressive HA, especially in older-age group
- Pattern change
- Previous HA history: attack frequency, severity, or clinical features

David Dodick, MD – Mayo Clinic, Scottsdale, AZ

14. **The Primary Headaches (1-4)**

1. Migraine
   *without aura
   *with aura
2. Tension-type headache
3. Cluster headache and other trigeminal autonomic cephalalgias
4. Other primary headaches

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What is Migraine?

- Repeated attacks of headache
  - Moderately or severely painful
  - Frequent or infrequent
  - Last a few hours to a couple of days
- Often only one side of the head hurts
- Often experience loss of appetite, nausea, and vomiting

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How Migraine Stacks Up Against Other Common Diseases

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17. Most Patients’ Headaches Are Severe or Extremely Severe

![Pie chart showing the percentage of severe headaches]

National Headache Foundation, American Migraine Study II: Migraine in the United States. Burden of Illness and Patterns of Treatment

18. Migraine Takes Quality Time Out From Your Life

![Pie chart showing the percentage of work ability]

National Headache Foundation, American Migraine Study II: Migraine in the United States. Burden of Illness and Patterns of Treatment
19. **Migraine Takes Time Out From Your Life**

![Migraine Takes Time Out From Your Life](image)

In the past 3 months...

<table>
<thead>
<tr>
<th>Amount</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>9 million</td>
<td>Missed Work or School</td>
</tr>
<tr>
<td>14 million</td>
<td>Functioned less than half as well at work/school</td>
</tr>
<tr>
<td>21 million</td>
<td>Were unable to do chores/household work</td>
</tr>
<tr>
<td>18 million</td>
<td>Functioned less than half as well at household chores</td>
</tr>
<tr>
<td>16 million</td>
<td>Missed family or leisure activity</td>
</tr>
</tbody>
</table>

National Headache Foundation

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20. **Unnecessary Suffering**

![Unnecessary Suffering](image)

Unnecessary Suffering

- More than half of people with migraine suffer for at least a year before they are diagnosed with migraine
- 38% suffer for 3 or more years

National Headache Foundation. American Migraine Study II: Migraine in the United States: Burden of Illness and Patterns of Treatment

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21. Migraine Pathophysiology

Migraine Pathophysiology

- Genetic “predisposition”
- The “sensitive” brain
- The “triggering” events
- The “neurological” aura
- The “pain” phase
- Associated “features”

22. How Migraine Works

How Migraine Works

1. Migraine originates deep within the brain
2. Electrical impulses spread to other regions of the brain.
3. Changes in nerve cell activity and blood flow may result in visual disturbance, numbness or tingling, and dizziness
4. Chemicals in the brain cause blood vessel dilation and inflammation of the surrounding tissue
5. The inflammation irritates the trigeminal nerve, resulting in severe or throbbing pain
Migraine Pathophysiology: Phases

- Initiation
- Activation of peripheral nociceptors
- Transmission in the primary afferent neurons
- Activation and sensitization of the trigeminal brainstem complex
- Integration of cortical and subcortical areas


Protective Factors

- Regular sleep
- Regular meals
- Regular exercise
- Biofeedback
- Healthy lifestyle

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25. Triggers and Risk Factors

Triggers and Risk Factors

Migraine headaches are often triggered by specific things.

26. Triggers: Changes in Daily Cycles

Triggers: Changes in Daily Cycles

- Menses
- Late hours
- No breakfast
- Overslept
27. Triggers: Environment or Diet

**Triggers: Environment or Diet**

- Weather
- Diet
- Altitude
- Smoking
- Some medications
- Bright light
- Alcoholic beverages

28. Triggers: Mental

**Triggers: Mental**

- Anger
- Fear
- Anxiety
- Depression
29. What You Might Feel Before or During an Attack

<table>
<thead>
<tr>
<th>Feeling of well-being</th>
<th>Drowsiness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Talkativeness</td>
<td>Depression</td>
</tr>
<tr>
<td>Surge of energy</td>
<td>Irritability</td>
</tr>
<tr>
<td>Hunger</td>
<td>Tension</td>
</tr>
<tr>
<td>Decreased appetite</td>
<td>Restlessness</td>
</tr>
</tbody>
</table>

30. What You Might Experience During an Attack

<table>
<thead>
<tr>
<th>Nausea</th>
<th>Sensitivity to sound</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vomiting</td>
<td>Scalp tenderness</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>Pale color</td>
</tr>
<tr>
<td>Sweating</td>
<td>Pulsing temple</td>
</tr>
<tr>
<td>Cold hands</td>
<td>Pressure pain</td>
</tr>
<tr>
<td>Sensitivity to light</td>
<td></td>
</tr>
</tbody>
</table>

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31. Migraine without Aura

**Migraine without Aura**

**Diagnostic criteria:**

A. At least 5 attacks fulfilling criteria B–D
B. Headache attacks lasting 4–72 hours (untreated or unsuccessfully treated)
C. Headache has at least two of the following characteristics:
   1. unilateral location
   2. pulsating quality
   3. moderate or severe pain intensity
   4. aggravation by or causing avoidance of routine physical activity (e.g., walking or climbing stairs)
D. During headache at least one of the following:
   1. nausea and/or vomiting
   2. photophobia and phonophobia
E. Not attributed to another disorder

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32. Migraine with Aura

**Migraine with Aura**

**Diagnostic criteria:**

- A. At least 2 attacks fulfilling criterion B
- B. Migraine aura fulfilling criteria B and C for one of the subforms 1.2.1–1.2.6
- C. Not attributed to another disorder

**The aura is the complex of neurological symptoms that occurs just before or at the onset of migraine headache.**

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33. Migraine Aura

Migraine Aura


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34. Tension-Type Headache

Tension-Type Headache

A. Frequency – days to weeks to daily (< or > 15 d/month x 3 months or more)
B. Headache lasts hours or may be continuous
C. Headache has at least two of the following characteristics:
   1. bilateral location
   2. pressing/tightening (non-pulsating) quality
   3. mild or moderate intensity
   4. not aggravated by routine physical activity such as walking or climbing stairs
   ** Pericranial tenderness

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35. Tension-Type Headache

**Tension-Type Headache**

- Typically pressing, tight pain
- Mild-to-moderate pain intensity
- Bilateral
- Doesn’t worsen with physical activity
- Nausea absent but photophobia or phonophobia may be present
- Attacks last hours to days
- Termed chronic if more than 15 d/mo for 6 mo

36. Cluster HA and Other TACs

**Cluster HA and Other TACs**

Cluster headache
- Episodic cluster headache
- Chronic cluster headache

Paroxysmal hemicrania
- Episodic paroxysmal hemicrania
- Chronic paroxysmal hemicrania (CPH)

Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT)
37. Cluster Headache

Cluster Headache

- Occurrence: 6 times more common in men than women
- Onset usually third or fourth decade
- Attacks
  - Often awaken the patient
  - Come in clusters and recur at regular, often annual, intervals
  - Common triggers: alcohol and nitroglycerin
  - Characteristic: unilateral and periorbital; excruciating, burning, and knife-like pain; often associated with lacrimation, conjunctival injection, rhinorrhea, and miosis
  - Last 15 min to 3 h

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38. Paroxysmal Hemicrania

Paroxysmal Hemicrania

- Chronic paroxysmal hemicrania is a headache disorder with pain intensity/location similar to cluster, BUT attacks are shorter and more frequent (up to 30 times/d)

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39. Chronic Daily Headache

Chronic Daily Headache

A Primary Headache Syndrome
(Organic causes excludes)

- Occurs >> 15 days a month
- Last >> 4 hours a day
- Often for multiple days at a time

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40. Chronic Daily Headache

Chronic Daily Headache

- Up to 30% of headache-center patients complain of daily headache.
- Controversy: Is this a separate category or the result of a "transformation" of a previously known episodic disorder into a daily one?
- Persons with either migraine or tension-type headache may develop this syndrome.
- Postulated contributing factors
  - Medication overuse
  - Stress
  - Hypertension
  - Psychologic disturbances

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41. Chronic Daily Headache

Chronic Daily Headache

- Transformed migraine ("chronic migraine"), with or without medication overuse
- Chronic tension-type headache, with or without medication overuse
- New daily, persistent headache, with or without medication overuse
- Hemicrania continua, with or without medication overuse

Silberstein SD, et al. 1996

42. Headache Attributed to As Substance or Its Withdrawal

Headache Attributed to As Substance or Its Withdrawal

- Headache attributed to acute substance use or exposure – nitrates, carbon monoxide, alcohol, cocaine
- Medication overuse headache – overuse of abortive headache medications
- Headache as an adverse event attributed to chronic medication – for other conditions
- Headache attributed to substance withdrawal – caffeine, ergots, triptans, opioids, estrogens

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43. Analgesic Rebound Headache / Medication-Induced Headache

**Analgesic Rebound Headache / Medication-Induced Headache**

- Worsening of headache 3-4 hours after analgesic wears off
- Withdrawal phenomena, where patients experience an escalation in symptoms after the discontinuance of medication
- Other pharmacologic and nonpharmacologic therapies are rendered essentially ineffective in the face of overuse syndromes


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44. Common Medications Associated with MOH

**Common Medications Associated with MOH**

- Ergots
- Triptans
- NSAIDs
- Combination analgesics
- Opioids
- Others

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45. Pharmacotherapy for Migraine

Pharmacotherapy for Migraine

46. Headache Treatments

Headache Treatments

- Educate patients about their condition and its treatment
- Encourage them to participate in their own care
- Identify and remove triggers
- Start a “wellness program” – exercise, diet, balanced meals, adequate and regular sleep, smoking cessation, etc.
- Pharmacotherapy
- Physical therapies
- Behavioral psychological therapies
- CAM therapies

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47. Self Treatment Efforts: What You Can Do For Your Migraines...

Self Treatment Efforts:
What You Can Do For Your Migraines

- Rest
- Biofeedback
- Ice/heat
- Massage
- Exercise
- Avoid triggers
- Seek treatment early
- Keep a headache diary
- Take medications as directed by your doctor

Many options are available for migraine relief – ask your doctor what’s right for you

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48. Pharmacologic Management of Headache: Symptomatic Therapies...

Pharmacologic Management of Headache: Symptomatic Therapies

- Stratified, NOT step-care, approach!
- NSAIDs, combination therapies including acetaminophen/aspirin/caffeine, or butalbital combinations should be used for mild-to-moderate pain
- Oral/parenteral triptans and dihydroergotamine should be used for severe migraine or for those individuals with less severe pain but who have not responded to other agents
- REMEMBER: overuse of symptomatic therapies can lead to an analgesic rebound headache syndrome

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Pharmacologic Management of Migraine: Prophylactic Therapies

- Indications
  - ≥3 headaches/mo with disability or
  - Lack of efficacy with symptomatic therapies or
  - Presence of headache types with any risk for neurologic injury
- Therapy should be individualized

Pharmacologic Management of Headache: Prophylactic Therapies

- Proven types of prophylactic agents include: beta-blockers, antidepressants, calcium channel blockers, NSAIDs, anticonvulsants, methysergide, and alpha-adrenergic agents
- Other types of prophylactic agents that have been used but whose role has not been as clearly established include topiramate and botulinum toxin
51. Medicines to Stop a Migraine Attack

Medicines to Stop a Migraine Attack

- Non-prescription medications – use with care and tell your doctor
  - NSAIDs (e.g., ibuprofen, naproxen)
  - Aspirin, acetaminophen, caffeine combination (avoid using more often than twice a week, especially if using several agents or if you drink a lot of coffee, tea, or caffeinated soda)
- Prescription medications
  - Triptans
  - Dihydroergotamine (DHE)
  - Others

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52. Other Factors Involved in the Choice of Medication

Other Factors Involved in the Choice of Medication

- How fast it works
  - Nasal spray allows for fast onset of migraine relief
- How long it keeps working
- Presence of migraine symptoms you may have
- Other medications you may be taking

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53. Fast Relief vs. Long-lasting Relief

Fast Relief vs. Long-lasting Relief

- Injections yield the fastest relief
- A scientific study compared DHE injection with an injectable triptan
- The triptan worked slightly faster
- DHE worked longer, so fewer patients had a return of their headaches


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54. Options for Preventive Treatment

Options for Preventive Treatment

- Divalproex sodium/sodium valproate & Topiramate (anticonvulsant)
- Propranolol (beta-blocker)
- Timolol (beta-blocker)
- Methysergide (serotonin antagonist)
- Other anticonvulsants
- Other beta-blockers
- Antidepressants
- NSAIDs (eg, aspirin)
- Other serotonin antagonists

These are medicines you take every day to prevent headaches. Choice based on comorbid conditions.

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### Pharmacological Therapy

#### Acute
- Acetaminophen
- ASA
- NSAIDs
- Opioids
- Barbiturates
- Benzodiazepines
- Neuroleptics/Antiemetic
- Corticosteroids
- Triptans

#### Preventive
- Antiepileptics
- Antidepressants
- Beta blockers
- Calcium channel blockers
- Serotonin antagonists
- NSAIDs
- Opioids?
- "Muscle relaxants?"
- Others

---

### Acute ( Abortive) Medication Therapy

#### Specific
- Ergots
- Triptans

#### Nonspecific
- Aspirin
- NSAIDs
- Opioids
- Barbiturates
- Combinations
- Others

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57. **DHE**

- Dihydroergotamine
  - Nasal Spray
  - Injectable

58. **Acute Therapy for Migraine**

**Acute Therapy for Migraine**

*Group 1: Proven pronounced statistical and clinical benefit*

- Acetaminophen + ASA + caffeine
- ASA
- DHE SC, IV, IN
- Ibuprofen
- Naproxen
- Naratriptan
- Rizatriptan
- Sumatriptan
- Zolmitriptan

*The US Headache Consortium*
59. Acute Therapy for Migraine

**Acute Therapy for Migraine**

Group 2: Moderate statistical and clinical benefit

- Acetaminophen + codeine
- Butalbital + ASA + caffeine, codeine
- Chlorpromazine IV
- Diclofenac
- Ergotamine + caffeine + phenobarbital
- Flurbiprofen
- Ketorolac IM
- Lidocaine IN
- Meperidine IM, IV
- Naproxen

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60. Acute Therapy for Migraine

**Acute Therapy for Migraine**

Group 3: Conflict of inconsistent evidence

- Butalbital + ASA + caffeine
- Ergotamine + caffeine
- Metoclopramide IM, PR

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61. Acute Therapy of Migraine

**Acute Therapy of Migraine**

Group 4: Statistically or clinically ineffective
- Acetaminophen
- Chlorpromazine IM
- Lidocaine IV

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62. Acute Therapy of Migraine

**Acute Therapy of Migraine**

Group 5: Statistical and clinical benefits unknown
- Dexamethasone IV
- Hydrocortisone IV

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63. **Triptans**

- Almotriptan
  - Tablets
- Eletriptan
  - Tablets
- Rizatriptan
  - Tablets
  - Orally disintegrating tablets (MLT)
- Naratriptan
  - Tablets
- Frovatriptan
  - Tablets
- Sumatriptan
  - Subcutaneous
  - Nasal spray
  - Tablets
- Zolmitriptan
  - Tablets
  - Orally disintegrating tablets (ZMT)
  - Nasal Spray

64. **Triptans: Efficacy Headache Relief at 2 hours**

- **Sumatriptan**
  - SC 6 mg 87%
  - Nasal 20 mg 64%
  - Tablet 50 mg 60%
- **Rizatriptan**
  - Tablets 10 mg 77%
  - MLT 10 mg 66%
- **Zolmitriptan**
  - Tablets 2.5 mg 65%
- **Naratriptan**
  - Tablets 2.5 mg 65% (4 hrs)
- **Amlotriptan**
  - Tablets 12.5 mg 65%
65. **Triptans**

<table>
<thead>
<tr>
<th>Triptans</th>
<th>QOE</th>
<th>Scientific Effect</th>
<th>Clinical Effect</th>
<th>AEs</th>
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<tbody>
<tr>
<td>Sumatriptan</td>
<td>A</td>
<td>+++</td>
<td>+++</td>
<td>1+(3+)</td>
</tr>
<tr>
<td>Rizatriptan</td>
<td>A</td>
<td>+++</td>
<td>+++</td>
<td>1+</td>
</tr>
<tr>
<td>Zolmitriptan</td>
<td>A</td>
<td>+++</td>
<td>+++</td>
<td>1+</td>
</tr>
<tr>
<td>Naratriptan</td>
<td>A</td>
<td>+/+</td>
<td>+/+</td>
<td>+/-</td>
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</tbody>
</table>

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66. **Preventive Therapies for Migraine**

<table>
<thead>
<tr>
<th>Preventive Therapies for Migraine</th>
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<tr>
<td>Alpha 2 agonists</td>
<td>B</td>
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</tr>
<tr>
<td>Antiepileptics</td>
<td>A/B</td>
<td>++++/++</td>
<td>++++/++</td>
</tr>
<tr>
<td>Antidepressants</td>
<td>A/B</td>
<td>++++/++</td>
<td>+++</td>
</tr>
<tr>
<td>TCAs</td>
<td>A/B</td>
<td>++++/++</td>
<td>+++</td>
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<td>SSRIIs</td>
<td>B/C</td>
<td>+/?</td>
<td>+</td>
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<td>A/B</td>
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<tr>
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<td>NSAIDs</td>
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<td>+/+</td>
<td>+/+</td>
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</table>

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67. Daily scheduled opioids for intractable head pain

**Daily scheduled opioids for intractable head pain: Long-term observations of a treatment program**

Saper, J.R. MD, FACP, FAA; Lake, A.E. III PhD; Hamel, R.L. PA-C; Lutz, T.E. BA; Branca, B. PhD; Sims, D.B. RN; Kroll, M.M. RN, BSN

- 160 sequential patients participating in the program, 70 who remained on DSO for at least 3 years qualified for inclusion in an efficacy analysis.
- Patients completed structured questionnaires at each medical visit as part of routine clinical care.
- The authors assessed medical records during treatment, and during the 2 years before starting DSO.
- The primary clinical efficacy variable was percentage improvement in the severe headache index (frequency x severity of severe headaches/week).

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68. Daily scheduled opioids for intractable head pain

**Daily scheduled opioids for intractable head pain: Long-term observations of a treatment program, cont.**

Saper, J.R. MD, FACP, FAA; Lake, A.E. III PhD; Hamel, R.L. PA-C; Lutz, T.E. BA; Branca, B. PhD; Sims, D.B. RN; Kroll, M.M. RN, BSN

- Analysis of the medical records found 41 (26%) of the original 160 patients with >50% improvement.
- Patients reported larger improvements on a visual analog scale (mean improvement = 70%) than shown by the medical record (mean improvement = 46%), p < 0.00001.
- Problem drug behavior (dose violations, lost prescriptions, multi-sourcing) occurred in 50% of patients, usually involving dose violations.
- 74% of those treated either failed to show significant improvement or were discontinued from the program for clinical reasons.
- The relatively low percentage of patients with demonstrated efficacy and unexpectedly high prevalence of misuse have clinical relevance.

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69. Nonpharmacologic Management of Headache

Nonpharmacologic Management of Headache

- Diet
- Exercise
- Biofeedback/relaxation training
- Acupuncture
- Consistent sleep/wake cycles

70. Other CAM Therapies

Other CAM Therapies

<table>
<thead>
<tr>
<th>Decrease??</th>
<th>Increase??</th>
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</thead>
<tbody>
<tr>
<td>Food additives</td>
<td>Caffeine</td>
</tr>
<tr>
<td>Food products</td>
<td>Magnesium</td>
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<td>Alcohol</td>
<td>Vitamins</td>
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<tr>
<td>Chocolate</td>
<td>Antioxidants</td>
</tr>
<tr>
<td>Caffeine</td>
<td>Botulinum toxin</td>
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<td>Estrogen</td>
<td>Estrogen</td>
</tr>
</tbody>
</table>

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71. Topical Agents

Topical Agents

- NSAIDs
- Local anesthetics
- Corticosteroids
- Anticonvulsants (AEDs)
- Capsaicin
- Clonidine
- Ketamine
- Ice

Cannabinoids

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72. Magnesium

Magnesium

- Circulates largely unbound to protein
- Excretion: renal (T½ = 4hr)
- Side effects: well tolerated
- Vasoconstriction
- Altered affinity for serotonin receptors
- Noncompetitive NMDA RA
- Stress has been shown to result in Mg depletion
- Does not cross BBB when given IV

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Magnesium for Headache

Magnesium for Headache

- RCT support efficacy
- Quality of evidence – Level B
- Efficacy rating – Group 2
  (US Headache Consortium Guidelines)


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MigraHealth and MigraLief

MigraHealth and MigraLief

* Magnesium 300 mg, Riboflavin 400 mg and Feverfew 100 mg

- DB-PC, RCT
- MigraHealth vs. riboflavin 25 mg (active placebo)
- 49 patients completed a 3 month trial
  Primary outcome: 50% or greater reduction in migraines
  Secondary outcome: change in mean number of migraines, migraine days, migraine index, or triptan dose

- No significant difference between active and “placebo” group for both outcome measures
- Compared to baseline, both groups showed a significant reduction in secondary outcome measures


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75. Vitamin B2 (Riboflavin)

**Vitamin B2 (Riboflavin)**

- Increases mitochondrial energy efficiency?
- Animal data – antioxidant
- Riboflavin deficiency linked to RA?

* Migraine
* Rheumatoid arthritis
* Muscle disorders

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76. Vitamin B2 (Riboflavin)

**Vitamin B2 (Riboflavin)**

- Placebo-controlled, double-blind open trial of riboflavin 400 mg/day
- Migraine preventive study
- Statistically significant in reducing the attack frequency, headache days, and migraine index
- The proportion of patients improved by at least 50% in “headache days” were 15% for placebo and 59% for riboflavin

Schoenen, et al. 1998

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77. Coenzyme Q 10 (ubiquinone)

Coenzyme Q 10 (ubiquinone)

- Vitamin-like substance made by the liver and found in plants and meats (in mitochondria)
- Antioxidant properties and produces ATP to power cellular reactions
- Minimal research and little data
  * Strengthen heart muscle – cardiac disease
  * Stimulate immune function – breast cancer
  * Promote weight loss in obesity
  * Alleviate diabetes
  * Periodontal disease

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78. CoQ 10

CoQ 10


- RCT
- 42 subjects
- 100 mg TID vs. placebo
- Outcome – significant reduction in HA frequency by 50%
- 47.6% - CoQ 10 group
- 14.4% - placebo group

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Feverfew

**Feverfew (Tanacetum parthenium)**

“febrifuge” – chases away fever

- Effective against inflammation, fever, swelling and menstrual pains
- Parthenolide – interferes with the aggregation of platelets and secretion of serotonin, may decrease the production of prostaglandin, and neutralizes histamine. Acts as an antagonist to vasoconstriction.

* Migraine preventive
* Arthritis pain
* Menstrual pain
* Stimulates the appetite
* Relieves depression

---

80.

Feverfew

**(Feverfew)**

Overall, these studies suggest that feverfew may be beneficial for the prevention of migraine attacks. However, the effectiveness has not been established beyond reasonable doubt. More data is needed to determine which dose and formulation should be prescribed, and how effective it is. Future trials should measure clinically relevant outcomes in accordance with International Headache Society (IHS) criteria.


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81. St. John’s Wort (Hypericum perforatum)

St. John’s Wort (\textit{Hypericum perforatum})

- Antidepressant activity – hypericin has MAOI-like properties. Also thought that xanthones and flavonoids have additional antidepressant activity.
- Antiviral activity against numerous viral types
  * Anxiety and depression disorders
  * SAD
  * Insomnia
  * Some interest in viral illnesses
- NIMH does not currently recommend the use of St. John’s Wort. NIH has ongoing clinical trials (NICAM)

82. Butterbur (Petasites hybridus)

Butterbur (\textit{Petasites hybridus})

- Highly toxic plant used in Germany for > 20 years
- Petadolex – highly purified commercial product available in the US
- Double-blind, placebo-controlled randomized trial confirmed efficacy

Women’s Issues and Headache

Pure Menstrual Migraine & Menstrually Associated Migraine

1. Falls in endogenous or exogenous estrogen concentrations are the provocative factor in menstrual attacks
2. Falls in progesterone do not trigger migraine
3. Eliminating or minimizing the premenstrual decline in estrogen decreases the likelihood that menstrual migraine will occur
4. Increasing the magnitude of the decline in estrogen will aggravate migraine

Loder E, Martin Y. Women's Issues in Headache. Headache. ACP Series, 2004
85. Oral Contraceptives and Migraine

**Oral Contraceptives and Migraine**

- Lack of consensus
- Effect variable and unpredictable
- Increased risk of stroke in migraineurs and OC users (migraine with aura)
- OCs should not be used solely to treat migraine
- Risks and benefits should be thoroughly discussed and understood
- Lowest possible estrogen dose should be used
- Trials of 3-6 months?

86. Pregnancy and Headache

**Pregnancy and Headache**

- 55%-90% of women – migraines improve with pregnancy, especially in the second and third trimesters
- However, a woman may experience her first migraine with pregnancy, and other disorders associated with headache occur more frequently or exclusively with pregnancy
- These include preclampsia, stroke, cerebral venous thrombosis, subarachnoid hemorrhage, pituitary tumor

Pregnancy and Headache

• Medications for headache should be used judiciously in pregnant and lactating women

FDA Use-in-Pregnancy Risk Category
(A, B, C, D, X)
Medications and Their Compatibility with Breastfeeding
(AAP)

Menopause and Migraine

• Physiological menopause can benefit migraine
• HRT has a variable effect on migraine, but can help “migraine triggered by hormonal fluctuations”
• HRT women have lower pain threshold and tolerance
• Synthetic estrogens preferred
• Phytoestrogens and herbal treatments??
Botulinum Toxin – A (BTX-A)

- Purified neurotoxin complex
- Fermentation of “Hall strain” of *Clostridium botulinum* type A
- Inhibits release of acetylcholine from presynaptic boutons of cholinergic neurons at the NMJ and at autonomic nervous system effector organs (e.g., glands)
- Cleaves synaptic vesicle-associated membrane protein complex (SNAP-25), a component of the protein complex responsible for docking and fusion of the synaptic vesicle to the cellular membrane
BTX-A

- Partial chemical denervation of muscle
- Localized reduction in muscle activity
- May have a direct antinociceptive effect??

  Pain relief before there is any muscle-relaxing action
  Pain relief without muscle weakness

- Latency for clinical effect within the first 2 weeks after injection
- Maximum clinical benefit at approximately 6-8 weeks
- Duration of effect up to 3-4 months

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BTX-A

- Single vial of 100 U of sterile vacuum-dried BTX-A
- Stored frozen prior to use
- Reconstituted with sterile, nonpreserved saline
- Stored in the refrigerator before injection
- Must be used within 4 hours of being reconstituted

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93. Side Effects

**Side Effects**

- Site-of-injection – pain, inflammation, infection, hematoma
- Medication-related – inadvertent muscle weakness, alteration in salivary consistency, antibody formation

Systemic complications are uncommon – “flu-like” syndrome

94. Antibody Formation & Resistance

**Antibody Formation & Resistance**

- Nonhuman protein that may evoke antibody formation
- Prevalence of treatment resistance is unknown (estimated 5-10% in cervical dystonia)
- Increase risk – high doses & short intervals between doses
- When resistance to one serotype develops, switching to a different serotype may restore the therapeutic response
95. Contraindications and Cautions

Contraindications and Cautions

- Known allergy
- Infection or inflammation at the proposed injection site
- Pregnancy or lactation
- Inability of the patient to cooperate?
- High levels of anxiety or fearfulness?
- Anatomic abnormalities
- Difficult or impossible injection
- Comorbid disease
- Coagulopathy, including therapeutic anticoagulation

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96. How Does BTX Decrease Pain??

How Does BTX Decrease Pain??

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Mechanisms of Analgesic Activity??

1. Reduced muscle spindle afferent discharge from Ia gamma-motoneurons to the spinal cord. Abolishes the hyperactivity of the muscle and the pain that is due to tonic contraction. Because the spinal afferents have supraspinal projections, the change in their firing pattern caused by BTX may also cause changes in sensory processing at higher levels of the CNS.

2. Decreasing prolonged muscle contraction may decrease various substances that induce local muscle ischemia and sensitizes muscle nociceptors.
Mechanisms of Analgesic Activity??

3. Suppression of neurogenic inflammation

Mechanisms of Analgesic Activity??

4. Alter the release of neurotransmitters other than acetylcholine.
   (Substance P, CGRP, NK1, glutamate)
101. Mechanisms of Analgesic Activity??

**Mechanisms of Analgesic Activity??**

5. Central nervous system effects
   * Animal studies suggest retrograde transport of BTX into the CNS
   * CNS neuroplasticity induced by alterations in afferent input

102. FDA Approval (1989)

**FDA Approval (1989)**

- Strabismus
- Essential blephrospasm
- Cervical dystonia
- Cosmetic facial procedures (wrinkle reduction)
103. Other Common Uses

Other Common Uses

- Dystonia
- Spasticity
- Achalasia
- Hyperhidrosis
- Hyperkinetic disorders (tremor)
- Spasmotic dysphonia
- Headache
- Myofascial pain disorders

104. Chronic Headaches

Chronic Headaches

- Daily patterns of HA pain and temporalis muscle EMG activity
- 3-day period in 36 patients and 36 controls
- No correlation between HA pain and temporalis muscle EMG activity
- “Stress” and pain correlated

105. Chronic Headache (Migraine, Tension-type, CDH)

Chronic Headache
(Migraine, Tension-type, CDH)

- Both open-label and DB, P, RCT studying “fixed-site,” “follow the pain” or a combination approach have demonstrated significant reduction in migraine frequency, severity, and duration, as well as decreased use of acute medications.
- The most prominent reductions are in those with “severe” migraine

106. Botulinum Toxin

Botulinum Toxin

Porta, et al: 1999

- Randomized, single-blind trial
- Botulinum toxin vs. methylprednisolone
- ETTH or CTTH
- Injections at cranial muscle tender points
- At 30 and 60 days postinjection – both had a significant reduction in pain scores.
- At 60 days – statistically significantly greater reduction in the botulinum toxin group
107. Botulinum Toxin

Botulinum Toxin


- Double-blinded, placebo-controlled, randomized trial of 40 patients (3 months)
- CTTH
- Neck and temporalis muscle injections
- Significant increase in headache free days in the botulinum toxin group
- Lower overall headache scores in botulinum toxin group

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108. Botulinum Toxin and EMG

Botulinum Toxin and EMG


- 8 patient with CTTH randomly assigned to botulinum toxin vs. placebo
- Pericranial muscles, post. neck and SCM muscles
- Clinical Outcome - no significant differences
- EMG – no reduction in resting muscle activity in botulinum toxin group (>> at 12 weeks)

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Botulinum Toxin Type A

- Multicenter, open-label trial to evaluate the efficacy of BTX-A for migraine management.
- Efficacy was categorized as either complete response with total symptom elimination, partial response with >50% reduction in headache severity and frequency, or no beneficial response.
- 51% of patients treated with BTX-A as migraine prophylaxis reported a complete response to localized head and neck BTX-A injections, with a mean duration of 4.1 months.
- An additional 38% reported partial improvement, with a mean response period of 2.7 months.


Botulinum Toxin

Botulinum Toxin


- Chronic migraine as a preventive therapy
- 123 patients in 12 center trial
- Randomized to 0, 25, 75 U
- Glabellar, frontalis and temporalis
- 0-30 days: 25 U significantly better than 0 and 75
- 31-90 days: 25 U and 75 U were significantly better than placebo on patient global assessment only
- 75 U: more treatment-related adverse events

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Botulinum Toxin, cont.

- First double-blind, placebo-controlled, randomized clinical trial
- 123 patients who had experienced between two and eight moderate-to-severe migraine episodes over a 3-month period
- A single injection of either placebo, low-dose (25 U), or high-dose (75 U) BTX-A.
- Injections were performed anteriorly, in the frontalis, glabellar region, and temporalis.


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Botulinum Toxin, cont.

- Low-dose BTX-A group experienced a mean decrease of 1.88 moderate-to-severe migraines compared with the placebo group \((P = .042)\). Furthermore, patients in the low-dose group reported a significant reduction in the incidence of migraine-associated vomiting compared with those in the placebo group \((P = .012)\).
- The high-dose BTX-A therapy did not have a significant effect on migraine pain and associated symptoms, however. In fact, at the higher dose, there was an increase in adverse effects.
- Silberstein et al suggested that the lack of BTX-A activity at this higher concentration may actually be due to a lower number of migraine headaches at baseline in this group compared with the low-dose group.
- In this trial, BTX-A was well tolerated, with no adverse effects observed in the low-dose group compared with the placebo group.


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Botox CDH

- This was a subgroup analysis of an 11-month, randomized double-blind, placebo-controlled study of BoNT-A for the treatment of adult patients with 16 or more headache days per 30-day periods conducted at 13 North American study centers.
- Patients were injected with BoNT-A or placebo and assessed every 30 days for 9 months.
- The following efficacy measures were analyzed per 30-day periods: change from baseline in number of headache-free days; change from baseline in headache frequency; proportion of patients with at least 30% or at least 50% decrease from baseline in headache frequency; and change from baseline in mean headache severity.


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Botox CDH, cont.

- 228 not taking prophylactic medication and were included in this analysis.
- 117 received BoNT-A, 111 received placebo injections.
- Mean frequency of headaches per 30 days at baseline was 14.1 for the BoNT-A group and 12.9 for the placebo group (P=.205).
- After two injection sessions, the maximum change in the mean frequency of headaches per 30 days was −7.8 in the BoNT-A group compared with only −4.5 in the placebo group (P=.032).


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115. **Botox CDH, cont.**

**Botox CDH, cont.**

- The between-group difference favoring BoNT-A treatment continued to improve to 4.2 headaches after a third injection session ($p = .023$).
- BoNT-A treatment at least halved the frequency of baseline headaches in over 50% of patients after three injection sessions compared to baseline.
- Statistically significant differences between BoNT-A and placebo were evident for the change from baseline in headache frequency and headache severity for most time points from day 180 through day 270.


116. **Differential Diagnosis of Craniofacial Pain**

**Differential Diagnosis of Craniofacial Pain**

*Neuralgiform (Neurogenic)*

- Trigeminal nerve involvement
  
  *Trigeminal Neuralgia (TN)*
  
  - Idiopathic
  
  - Symptomatic

*Neuropathic Pain Disorder*  

- Other cranial nerve involvement
117. Trigeminal Neuralgia

**Trigeminal Neuralgia**
“**Trigeminal Neuralgia Equivalents**”

1. Paroxysmal pain
2. Trigger areas
3. Unilateral
4. No sensory deficit
5. Restricted to the distribution of the trigeminal nerve
6. No obvious source of pathology

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118. Characteristics of Patients

**Characteristics of Patients**


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<thead>
<tr>
<th>AVERAGE AGE</th>
<th>PRESENT</th>
<th><strong>COMBINED</strong></th>
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<td>61.5</td>
<td>65</td>
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<table>
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<td>16%</td>
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<td>3%</td>
<td>40%</td>
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<tr>
<td>V-1, V-2, V-3</td>
<td>4%</td>
<td>13%</td>
</tr>
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</table>

**Tew JM, van Loveren H, 1995**

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Classification of TN

- “Classical” or Primary TN – Idiopathic
- “Symptomatic” or Secondary TN – Associated with another disease process

- Pre-trigeminal neuralgia
- Atypical TN

Etiology??

*Peripheral vs. Central ?*
*Ectopic afferent activity ??
*Chronic irritation / inflammation ??
*Demyelination ??
*Vascular compression ??
*Seizure activity / epilepsy ??
121. Trigeminal Neuralgia Therapeutic Options

Trigeminal Neuralgia Therapeutic Options

The disorders you can “F…”

Ferment,
Fascinate,
Fry,
Freeze,
Fondle,
Fibrillate,
Free-radical,

However, not “figure out”?

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122. Headache and Craniofacial Pain Disorders I: Slide 122

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123. Anticonvulsants (AEDs)

Anticonvulsants (AEDs)

- Phenytoin
- Carbamazepine
- Baclofen
- Clonazepam
- Gabapentin
- Lamotrigine
- Topiramate
- Oxcarbazepine
- Tiagabine
- Levetiracetam
- Zonisamide
- Pregabalin

Lidocaine
Mexiletine

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124. Pharmacological Effect and Mechanism of Pain Relief

Pharmacological Effect and Mechanism of Pain Relief

- CBZ Na chan
  OXC
- CLO GABA
- BAC GABA-B
- GBP Na, Ca chan
- LTG Na chan, inh GLU
- TOP Na chan, GABA, AMPA
- TGB GABA RI

“Suppress spontaneous ectopic neuronal activity”
“Reduce neuronal hypersensitivity”
“Segmental inhibition of signaling”

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125. AEDs for TN – RCTs

**AEDs for TN – RCTs**


126. Pharmacological Therapy for Trigeminal Neuralgia

**Pharmacological Therapy for Trigeminal Neuralgia**

61% of patients with TN
54 patients prospectively followed:

- Excellent pain control (no pain) 27 (50%)
- Good pain control (satisfactory) 19 (35%)
- Fair/Poor pain control (unsatisfactory) 8 (15%)


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Pharmacological Therapy

152 patients – retrospective survey

- Gabapentin (Neurontin) = 39.3%
- Carbamazepine (Tegretol) = 60.0%
- Gabapentin + other = 78%
- Carbamazepine + other = 70%
- Other AEDs – baclofen, clonazepam, topiramate
- Side effects – N = 12%**  
  Teg = 31%

Negative outcome co-factors – duration & tactile triggers

Scrivani S.J., Mathews E.S., Rabinovitch A. American Academy of Orofacial Pain  
(abstract presentation)

Surgical Interventions

- Peripheral Procedures
- Intracranial Procedure
  - Radiofrequency Thermal Rhizotomy
  - Microvascular Decompression
  - Gamma Knife Radiosurgery
  - Cyberknife Robotic Radiosurgery

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129. Acute Therapy

**Acute Therapy**

- Trigeminal blocks:
  - Local anesthetic +/- steroid
  - Botulinum toxin
- IV drug:
  - Lidocaine
  - Depacon (valproic acid)
  - Cerebryx (fosphenytoin)

130. Differential Diagnosis of Craniofacial Pain

**Differential Diagnosis of Craniofacial Pain**

*Neuralgiform (Neurogenic)*

- Trigeminal nerve involvement
  - Trigeminal Neuralgia (TN)
    - Idiopathic
    - Symptomatic
  - Neuropathic Pain Disorder
- Other cranial nerve involvement
131. Neuropathic Facial Pain “Old & New Names”

Neuropathic Facial Pain
“Old & New Names”

- Atypical Facial Pain (AFP)
- Atypical TN
- Atypical Odontalgia (“Persistent toothache”)
- Deafferentation Pain Syndrome
- Phantom Tooth Syndrome
- Sympathetically Maintained Pain

Persistent Idiopathic Facial Pain
Chronic Regional Pain Syndrome I & II

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132. Persistent Idiopathic Facial Pain

Persistent Idiopathic Facial Pain

- Pain (constant, diffuse, burning, deep pain possible paroxysms of sharp)
- Unrelated to associated tissue damage
- Disproportionate to the stimulation of nociceptors
- Often not associated with neuroanatomical distributions
- Second and third division
- Cutaneous hypersensitivity
- Associated psychopathology
- Multiple associated signs and symptoms or dysfunction

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133. Chronic Neuropathic Facial Pain

**Chronic Neuropathic Facial Pain**

*“Trigeminal Neuropathic Pain Disorder”*

*IHS – “Idiopathic Persistent Facial Pain”*

- Complex, multi-factorial process
- Regional
- Pain is the primary symptom
- Additional symptoms which occur together; the sum of signs of any morbid state - a Syndrome

“CRPS type I and II”

---

134. CRPS of the Head and Neck

**CRPS of the Head and Neck**

Does it exist ???

YES !

What is the pathophysiologic mechanism ?

Not known !

What do we call it ?

Neuropathic facial pain !

How should we treat it ?

In any way possible ! .....BUT
135. Burning Mouth/Tongue (Oral Burning)

Burning Mouth/Tongue (Oral Burning)

Neuropathic Pain Syndrome

+ Taste Abnormality??

136. Treatment of Neuropathic Craniofacial Pain

Treatment of Neuropathic Craniofacial Pain

“Multidisciplinary Approach”
137. Pain Management

**Pain Management**

- Pharmacological Therapy
- Injection Therapy (Local anesthesia)
- Intravenous Therapy
- Exercise Therapy
- Physical Medicine
- Behavioral Medicine
- Psychiatric Medicine
- Surgical Therapy
- Complementary & Alternative Medicine

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138. Voltaire’s Cynicism

**Voltaire’s Cynicism**

...Doctors pour drugs of which they know little, for diseases of which they know less, into patients of whom they know nothing...
139. Prudent Polypharmacy!!!

140. Pharmacological Therapy

- NSAIDs
- Opioids
- Tricyclic and Tetracyclic Antidepressants
- SSRIs/SNRIs/NDRIs
  - Venlafaxine
  - Duloxetine
- Anticonvulsants (AEDs)
  - Pregabalin
Pharmacological Therapy

- Other Neuromodulator Agents
  - Neuroleptics
  - Alpha adrenergic agent
  - NMDA-R antagonists
- Serotonin (5-HT) receptor agents
- Botulinum toxin??

NMDA-Receptor Antagonists

- MK-801
- Ketamine
- Memantine (Nemanda) & Amantadine
- Dextromethorphan
  - NMDA-receptor blocker, Ca, Na channels?
  - Attenuates glutamate neurotoxicity
  - Attenuates and reverses development of tolerance to opioids
- Methadone??
Topical Agents

- NSAIDs
- Opioids (Transmucosal)
- Local anesthetics (Lidocaine)
- Corticosteroids
- Anticonvulsants (AEDs)
- Capsaisin
- Clonidine
- Ketamine *Cannabinoids
- Cold/Ice

Advances in Neuropathic Pain

Advances in Neuropathic Pain: Diagnosis, Mechanisms, and Treatment Recommendations

First Line Treatment Recommendations:
- Gabapentin
- Lidocaine Patch 5%
- Opioids
- Tramadol
- Tricyclic Antidepressants

145. CAM Use for Headache & Facial Pain

CAM Use for Headache & Facial Pain

- Survey of headache patients at CPMC
- 98% familiar with at least 1 intervention
- 44% familiar with between 1-13
- Most familiar - massage, acupuncture, exercise, meditation, nutritional therapy, relaxation therapy, chiropractic therapy
- 85% used CAM therapies
- 60% perceived CAM to be effective


146. Complementary and Alternative Medicine (CAM)

Complementary and Alternative Medicine (CAM)

- Meditation
- Hypnosis
- Guided Imagery
- Biofeedback
- Relaxation Therapy
- CBT
- Prayer and Spirituality
- Homeopathy
- TCM
- Bodywork and Movement Therapy

- Acupuncture
- Ayurvedic Medicine
- Physical Medicine
- Chiropractic Therapy
- Energy Medicine
- Dietary Medicine
- Herbal Medicine
- Massage Therapy
- Naturopathy
- Neural Therapy
- Magnet Therapy

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147. Local Anesthetic Nerve Block Injections

Local Anesthetic Nerve Block Injections

Somatic block
Sympathetic block

Botulinum Toxin??

148. Electrical Stimulation

Electrical Stimulation

• TENS
• Peripheral Nerve Stimulation (Implant)
  Trigeminal nerve branches and ganglion
  Greater occipital nerve
• Trigeminal Ganglion Stimulation
• Deep Brain Stimulation
• Brainstem Stimulation (Trigeminal tract)
• Motor Cortex Stimulation
149. The Spectrum of Pain in Herpes Zoster

The Spectrum of Pain in Herpes Zoster

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150. PHN: Clinical Presentation

PHN: Clinical Presentation

- Constant baseline pain (burning, aching)
- Spontaneous, intermittent pain (lancinating, jabbing)
- Allodynia (mechanical, cold/warm)
- Sensory abnormalities (itching, numbness, tingling)
- Sensory deficits (absent or diminished thermal, tactile)
- Skin pigmentation changes and/or scarring

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151. Vasculitides with Headache & Facial Pain

Vasculitides with Headache & Facial Pain

- Polyarteritis nodosa group
- Hypersensitivity angitis group
  - Drug-induced angitis
- Cryoglobulinemic vasculitis
- Infection-related vasculitis
- Paraneoplastic vasculitis
- Granulomatoses
  - Wegeners granulomatosis
  - Lymphomatoid granulomatosis
  - Lethal midline granuloma
  - Sarcoidosis
- Giant cell arteritis
  - Temporal arteritis
  - Takayasu arteritis
- Connective tissue disorders
  - SLE
  - RA
  - Scleroderma
  - SS
  - Bechet’s syndrome
  - Cogan syndrome
  - Mixed CT disease

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152. Temporal Arteritis

Temporal Arteritis

- Age > 50 years
- Headache, neck pain, jaw claudication, scalp tenderness, facial pain, visual loss
- Thickened, nodular, pulseless superficial temporal artery
- Headache is mild to severe, and of acute or gradual onset; the patient is typically without a history of HA or deviation from his or her chronic HA pattern
- Associated with polymyalgia rheumatica, especially in elderly patients

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153. Superficial Temporal Artery

154. Temporal Ateritis

- Untreated or inadequately treated may result in unilateral or bilateral blindness (up to 50%)
- Oculomotor disturbances
- Vertigo and hearing impairment
- Cervical myelopathy
- Unilateral or bilateral limb bruits and claudication
- Brainstem strokes and TIs
Temporal Arteritis

- Blood studies – elevated ESR, CRP
- Others
- Temporal artery biopsy – false negatives due to skipped lesions (bilateral biopsies)
- Treatment with corticosteroids