Biobehavioral Considerations in the Diagnosis and Treatment of Primary Headache Disorders

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Epidemiology
Scope of the problem

Epidemiology

• Incidence- Rate of onset of new cases over a defined time period in a defined population. Helpful for identifying risk factors and disease associations

• Prevalence- The proportion of a defined population that has migraine during a particular time period. 1 year period or lifetime prevalence. Helpful in estimating scope and distribution of problem

Migraine Severity

• AMS II
  – Patient report pain as extremely severe or severe during attacks
  – 62% experienced 1 or more severe attacks per month
  – 25.2% experienced more than 4 attacks per month

Prevalence of Migraine in Men and Women in AMS I & II

CHRONIC DAILY HEADACHE

4.1% of 13,000 General Public
30%-80% Headache Clinic Population

Childhood Precursors to Migraine

- Motion sickness
- Episodic syndromes of childhood
  - Vertigo
  - Abdominal pain
- Low threshold for headache
  - e.g. “ice cream headache”

Incidence: Age of First Migraine

Peak Incidence (population-based)

<table>
<thead>
<tr>
<th>M</th>
<th>F</th>
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</thead>
<tbody>
<tr>
<td>Migraine with aura</td>
<td>5-6</td>
</tr>
<tr>
<td>Migraine without aura</td>
<td>10-11</td>
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</tbody>
</table>

Women and Migraine

- Menstrual cycle (cyclic ↓ estrogen levels)
  - 60% to 70% report related attacks
  - ≈ 60% experience ↑ frequency of attack
- Pregnancy (noncyclic ↑ levels of estrogen)
  - 55% to 90% report ↓ frequency or absence
- Perimenopause (↓ estrogen production)
  - many report exacerbation of migraine
- After menopause (noncyclic ↓ levels of estrogen)
  - two thirds report marked improvement

Headache History

Grade the headache by its intensity/severity

<table>
<thead>
<tr>
<th>Incapacitating (operational definition)</th>
<th>Moderate - severe</th>
<th>Dull</th>
</tr>
</thead>
</table>

note characteristics of pain for each intensity headache

- frequency
- location/laterality
- character of pain
- medication usage and relief
- time of onset/duration/ pain patterns

How often are you clearheaded?

“If I don’t take the pills, all my headaches will be incapacitating.”

Onset

- Age headache onset
- Developmental issues (menarche, postpartum, etc)
- Life circumstances
- Periods of increased headache versus headache remission
- Length of time at current frequency/severity

FREQUENCY AND INTENSITY

Migraine is episodic

Chronic daily headache often daily and constant

Cluster typically 1-3 attacks / day

Character of Pain

- Migraine - throbbing, deep
- Cluster - boring, sharp
- Tension-type headache - squeezing, steady

Time of onset and duration

<table>
<thead>
<tr>
<th>MIGRAINE</th>
<th>CLUSTER</th>
<th>TTHA</th>
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</thead>
<tbody>
<tr>
<td>Anytime, often perimenstrual, 4-72 hours.</td>
<td>Attacks often occur with circadian periodicity, can be chronic</td>
<td>Episodes vs. chronic, 30-180 minutes</td>
</tr>
</tbody>
</table>
Associated Symptoms

migraine - GI disturbance, photo, sono
cluster - unilateral autonomic

Behavior During Attacks

migraine - hibernates
cluster - movement, pacing

Look at cognitions (“I can’t handle this”; “I know what to do to manage this attack”)

Worrisome headache red flags 
SNOOP

• Systemic symptoms (fever, weight loss) or Secondary risk factors (HIV, systemic cancer)
• Neurologic symptoms or abnormal signs (confusion, impaired alertness or consciousness)
• Onset: sudden, abrupt or split second
• Older: new onset and progressive headache, especially in middle age ≥50 (giant cell arteritis)
• Previous headache history: first headache or different (change in attack frequency, severity or clinical features)

Headache History 
Behavioral Assessment

• Habit history
• Sleep
• Mood/ anxiety
• Coping skills
• Locus of control/self-efficacy
• Functional capacity/ disability
• Family history and dynamics
• Vocational history (absences/loss of productivity)

Behavioral Assessment 
Treatment Adherence

• Does patient understand therapy rationale?
• Did patient receive adequate drug or behavioral Rx?
• Has patient adhered to therapy regimens?
• Did “rebound” problems affect outcome?
• At what point does patient medicate?
• What skills need to be developed for better management?

Psychological/ Biofeedback 
Interventions

• Did learning occur?
• Type of feedback- sites trained
• Appropriate therapy modalities
• Concurrent medication overuse/rebound problems
Locus of control

- Internal = perception that life events and circumstances (headache) are the results of one’s own actions, a sense of control
- External = perception that life events and circumstances are beyond one’s own control. Reliance on fate, chance, other people.

Locus of Control

Internal
- Patient is task-specific
- “good historian”
- Action-oriented
- “I have a plan”
- sets realistic goals

External
- Helplessness (“fix me”)
- Fatalistic / global
- “suffering” or “hope” without action orientation
- Looking for “magic pill”
- “Yes...but”

Why Study Comorbidity?

- Complicates differential diagnosis
- Creates therapeutic opportunities
- Imposes therapeutic limitations
- Allows treatment of the “whole person”

Association Between Migraine and Depression: IHS-Based Community Studies

<table>
<thead>
<tr>
<th>Reference</th>
<th>Association</th>
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<tr>
<td>Breslau (1998)</td>
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<tr>
<td>Swartz et al (2000)</td>
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<td>2.3</td>
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</table>

Low & Merikangas, 2003

Association Between Migraine and Bipolar Disorder: IHS-Based Community Studies

<table>
<thead>
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<th>Reference</th>
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<tr>
<td>Migraine without aura</td>
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<td>Bipolar II Migraine with aura</td>
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<td>Merikangas et al (1990)</td>
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<td>Bipolar spectrum</td>
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Low & Merikangas, 2003
Association Between Migraine and Anxiety: Community Studies

<table>
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<tr>
<th>Reference</th>
<th>Panic</th>
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<td>Breslau et al (2001)</td>
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<td>Merikangas et al</td>
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Psychiatric Comorbidity

- Onset of anxiety generally precedes the onset of migraine, whereas the onset of major depression follows the onset of migraine. Anxiety may appear in childhood, followed by migraine then depression.
- 88% of patients with migraine and major depression have at least 1 anxiety disorder.
- Over 50% of patients with mood or anxiety disorders present exclusively with physical symptoms to primary care physicians.

Phases of a Migraine Attack

**Prodrome**

- The first symptoms of migraine
  - Mood changes
  - Changes in vision, hearing, or smell
  - Fatigue and feeling tired
  - Forgetfulness or slowness in thinking
  - Food craving especially things like chocolate
  - Pain in the muscles of the head and neck
  - Nasal stuffiness or nasal drainage
  - Yawning

**Aura: The Electrical Phase of Migraine**

- Approximately 15% of migraines are preceded by an aura.
- Common auras are scintillating lights in the vision, black spots or voids in the vision, and numbness and tingling in the hands or face.
- Auras should resolve entirely within an hour after they start.

MIGRAINE WITH AURA (FORMERLY “CLASSIC” MIGRAINE)

- Complex array of symptoms reflecting focal cortical or brainstem dysfunction
- Gradual evolution: 5–20 minutes (<60 minutes)
- May or may not be associated with headache
- Visual > sensory > motor, language, brainstem

**Headache phase**

- 4-72 hours
- Throbbing (83% F, 84% M)
- Worsened by activity
- Fronto-temporal
- Unilateral (59F, 51M)

  - Nausea (72% F, 60% M)
  - Scalp and pericranial tenderness
  - Heightened sensory perception (72F, 66M)
  - Hibernation

**Postdrome**

- Limited food tolerance
- Impaired concentration
- Fatigue and weakness “washed out”
- Irritability
- Muscle aches
- Excessive yawning

**Clinical Features of Migraine**

- Frequency
  - Intermittent
  - 1-4 per month on average
- Location
  - Unilateral
  - Pulsating
- Character
  - Moderate to severe intensity
  - Exacerbated by routine activity
- Intensity
  - Inhibits or prohibits daily activities
  - Duration
  - 4-72 hours
- Associated symptoms
  - Nausea or vomiting
  - Photophobia

**IHS Criteria for Migraine**

- At least 5 Attacks
- Headache lasting 4 to 72 hours
  - (2 to 48 hours in children)
- At least 2 of the following:
  - Unilateral location
  - Pulsating quality
  - Moderate to severe intensity (inhibits or prohibits daily activities)
  - Exacerbated by routine activity
- At least 1 of the following:
  - Nausea and/or vomiting
  - Photophobia and phonophobia
- Not attributable to other causes

**Chronic Daily Headache**

- Chronic migraine
  - with medication overuse
  - without medication overuse

- Chronic tension-type headache
  - with medication overuse
  - without medication overuse

- New daily persistent headache
  - with medication overuse
  - without medication overuse
**Chronic Migraine, IHS/AHS 2006**

- At least 15 days of HA/month
- At least 4 hours/day untreated
- At least 8 days/month meet criteria for migraine
- No need for previous history of episodic migraine or transformation
- Does not meet criteria for NDPH, CTTH


**Most CDH/CM evolves from episodic migraine**

- Thus, most CDH is transformed migraine, transformed from episodic migraine to daily headache, often by medication overuse
- Many, but not all patients remember their period of transformation

**Transformed Migraine Sufferer**

- Episodic Migraine
- Chronic Headache
- Severe Impairment
- Moderate Impairment
- Mild Impairment
- Normal Neurological Function
- Sleep Disorder
- Anxiety

**Rebound Headache**

The worsening of head pain in chronic headache sufferers, caused by the frequent and excessive use of immediate relief medications.”

**Medication Overuse Headache**

**Rebound Headache**

- Most patients with MOH have a history of episodic migraine
- Chronic migraine/CDH is often caused or maintained by medication and caffeine overuse
- A self-sustaining rhythm of predictable and escalating medication use of q3-6 hours
- Headaches increase in frequency and intensity and become refractory to acute care and preventive treatments
- Medication withdrawal results in acute escalation of headache

Kudrow, 1982; Rapoport, Weeks, Sheftell, Bockin et al. 1985

**Medication Overuse Headache**

**New Criteria 2005**

A. Headache present on at least 15 days/month fulfilling criteria C and D.
B. Regular overuse [≥10-15 days/month] for ≥3months of one or more drugs that can be taken for acute and/or symptomatic treatment of headache.
C. Headache has developed or markedly worsened during medication overuse.
D. Headache resolves or reverts to its previous pattern within 2 months after discontinuation of overused medication.

Silberstein SD, et al. The International Classification of Headache Disorders, 2nd
### Medication Overuse Headache Systematic Review (17 studies)

**Improvement**

- Drug withdrawal 30-50%
- Drug withdrawal + prophylaxis 72-85% (< 12m)
  50-66% (3 - 5 y)


### Incidence and Predictors for Chronicity of Headache in Patients with Episodic Migraine

- Clinic based study
- They followed 532 consecutive patients with episodic migraine for one year
- Sixty-four (14%) developed chronic daily headache
- Risk factors were high frequency at baseline and medication overuse
- The odds of developing CDH were 19.4 times higher in those overusing acute medication

Katsarava, Schneeweiss, et al. Neurology, 2004

### General Treatment Considerations for chronic migraine with medication overuse

- Keep a headache diary
- Explicit plan for tapering or discontinuing overused agents
- Discourage abortive Rx for mild/moderate HA’s in short term
- Acute treatment limits
- Initiate bridge therapy for withdrawal headaches
- Start prophylaxis/relaxation/biofeedback
- Use nonpharmacological strategies
- Frequent revisits during “washout” period

### Key Features of Cluster Headache

- Predominance in men- 5:8:1
- Unilaterality (often periorbital)
- Excruciating, boring pain “like a hot poker in the eye”
- 30-90 minutes in duration, on average
- Associated symptoms:
  - ipsilateral reddening and tearing of the eye
  - partial Horner’s syndrome
  - miosis
  - nasal congestion and/or rhinorrhea
  - paces- can’t sit or lie still
  - clockwise regularity
  - often nocturnal attacks

### Tension-Type Headache

- Can be associated or not associated with pericranial tenderness
- Diagnosed by manual palpation
- In new criteria, no use of surface EMG or pressure algometry

### Chronic Tension-Type Headache

A disorder evolving from episodic tension-type headache, with daily or very frequent episodes (>15days/mo) of headache lasting minutes to days. The pain is typically bilateral, pressing or tightening in quality and of mild to moderate intensity, and it does not worsen with routine physical activity. There may be mild nausea, photophobia or phonophobia.
Mechanisms of Migraine

Overview of Migraine Pathophysiology

- Migraine is a result of episodic changes in CNS physiologic function in a hyperexcitable brain
- Aura is probably generated by cortical spreading depression (CSD)
- Migraine pain has multiple mechanisms and the syndrome is mediated by the trigeminovascular system
- There may be a brainstem generator for the migraine headache which may modulate TGVS nociceptive input

TGVS Theory

- Dura is an important source of head pain
- Dura and its blood vessels are densely innervated by neuropeptide containing trigeminal and upper cervical sensory nerve fibers
- TGVS stimulation (animal, human studies) causes NI of dural vessels by releasing CGRP and SP
- Effective anti-migraine agents block NI

Central Sensitization

- The longer a migraine goes, the more neurons are activated, and the more brain becomes involved
- Neuronal pathways become sensitized in stages; peripheral neurons are activated early in the attack (mild-moderate) and central neurons are activated later in the attack (full blown migraine)
- Central sensitization is a time dependent physiologic event and leads to allodynia where nonpainful stimuli are perceived as painful

Areas of red indicate cerebral blood flow increases (P < 0.001).
(Weller et al. 1995)

(Adapted from Hargreaves, Shephard 1999)

Burstein and Jakubowski Ann Neurology, 2004
Central Sensitization

- Acute treatment works best before central neurons are recruited and central sensitization and allodynia develop
- Recurrent episodes of migraine may produce chronic sensitization of higher order neurons producing chronic neuronal discharge and chronification of migraine

Burstein et al., *Ann Neurology* 2000; Burstein et al., *Headache* 2002

Key features for migraine understanding

- Brain stem hyperexcitability
- Spreading cortical depression
- Pain has neurovascular and other mechanisms mediated by TGVS disinhibition
- Central processing
- Central sensitization

Migraine psychophysiology

- TPA and peripheral blood flow data is equivocal (? nature of the stressor and active versus passive coping) although cold hands the norm
- General autonomic instability
- Electrophysiology data shows evidence of hyperexcitable brain. Abnormal CNV (slow event-related potential) and auditory evoked potentials suggests lack of habituation or potentiation


General Treatment Principles I

- The history is the heart of diagnosis and Rx
- Review patient goals
- Diary to see patterns/ assess outcome
- Educate
- UNDO
  - headache inducing medications
  - ineffective or unnecessary medications (including vitamins, herbs)
  - overuse of immediate relief medications

General Treatment Principles II

- Avoid/ limit triggers
- Lifestyle management/ self-regulation
- Evaluate relevant comorbidities
- Optimize treatment of acute attacks
- Assess need for preventive therapy
- Flexible plan with periodic reassessment

Treatment of Acute Migraine

- Tailor treatment to the attack and to the individual
- Know all medications (prescriptive, nonprescriptive) the individual is taking
- Are any therapies contraindicated due to medical history or risk factors?
- Treatment is based on attack severity, time to peak intensity, patient’s preference, N/V
- Assess adherence to regimen
- Medication overuse may lead to treatment failure
Triptan Formulations

- **Sumatriptan**
  - Oral - 25, 50, 100 mg
  - Nasal - 5, 20 mg
  - Autoinjector - 6 mg, 4 mg.

- **Zolmitriptan**
  - Oral - 2.5, 5 mg
  - ODT - 2.5, 5 mg
  - Nasal - 5 mg

- **Naratriptan**
  - Oral - 1, 2.5 mg

- **Rizatriptan**
  - Oral - 5, 10 mg
  - ODT - 5, 10 mg

- **Almotriptan**
  - Oral - 2.5 mg

- **Frovatriptan**
  - Oral - 2.5 mg

- **Eletriptan**
  - Oral - 20, 40 mg

(ODT = Orally disintegrating tablet)

Preventive Treatment for Migraine

- **Episodic**- pretreat prior to known trigger such as exercise or sexual activity
- **Intermittent (subacute)**- time limited exposure to provoking agent such as menstruation
- **Chronic**

Migraine Preventive Drugs

**Facts**

- Most drugs are used based on:
  - Open-label studies
  - Anecdotes
  - Poorly controlled trials
  - Variable types of outcome parameters
  - Poorly defined migraine diagnosis

**Migraine Preventives for Adults**

**Antidepressants**

- **Tricyclics**
  - Amitriptyline,
    Nortriptyline,
    Desipramine
  - 10 - 100 mg QHS

- **SSRIs**
  - Standard antidepressant doses

**Cardiovascular Agents**

- **Beta Blockers**
  - Propranolol* LA
  - Nadolol, atenolol, timolol*

- **Verapamil**
  - 120 - 480 mg QD

**Neuronal Stabilizers**

- **Divalproex sodium***
  - Delayed Release
    250 - 1000 mg
  - Extended Release
    500 - 1000 mg

- **Topiramate**
  - 50 - 200 mg

- **Gabapentin**
  - 900 - 2400 mg

**Other Approaches**

- **Magnesium**
  - 500 mg

- **Vitamin B2**
  - 200 - 400 mg

- **Coenzyme Q10**
  - 150 - 300 mg

- **Petasites Hybridus**
  - 75 mg, BID

* = FDA approved for migraine prevention

(Adapted from Hargreaves, Shephard 1999)
Behavioral and Psychophysiologic Approaches to Headache Management

Methodological Issues
- Medication confounds
- Inadequate diagnostic criteria
- Poor description of sample
- Many treatments are packages
- Higher rates of improvement in EMG biofeedback for episodic rather than chronic tension type headache but studies are confusing
- No relationship to pretreatment pericranial surface EMG levels, pressure-pain thresholds or ES2 duration in TTHA studies. Some relationship to finger temperature increase in M studies.

Psychobiological Model
- Abandon organic/psychogenic distinction, for primary HA, in most cases, but evaluate behavioral issues and psychiatric comorbidity
- Conditions that control chronic headache are multidimensional involving cognitive/emotional/behavioral factors as well as biological processes

Psychobiological Model
- As a headache disorder becomes more severe and chronic, faulty learning and behavior become important maintenance factors and may be part of the chronification process.

Multiaxial Assessment
I. Headache diagnosis; frequency, intensity, and level of disability
II. Medication use, overuse, misuse
III. Stress-related risk factors
IV. Comorbid Axis I and II psychiatric disorders

Behavioral Analysis
- Antecedents: Events or triggers that precede migraine or periods of increased headache.
- Behavior: Actions taken during prodrome, headache or escalation of pain. May experience cephalalgia phobia (pre-emptive meds), may increase or decrease activities, pain behaviors
- Consequents: The impact and effect on the environment. Reinforcement, family responses, changes in pain levels effects previous behaviors

Modification of Lake, 2001

Adapted from Lake AE. Medical Clinics of North America. 2001;85:1055-1074
Pain and Learning
- Operant conditioning - pain behavior is affected by its consequences
  - reinforcement (secondary gain)
  - avoidance learning
- Classical conditioning - biological reactions can be conditioned to associated stimuli
  - fear reactions
  - avoidance learning

Psychiatric Comorbidity
migraine and CDH
- Depression/Dysthymia (bidirectional)
- Anxiety/Panic
- Bipolar disorder
- Sleep disorder

Axis II
- Persistent, inflexible patterns of behavior that lead to distress and impaired functioning.
- Low pain tolerance
- Affective instability

Axis II Signs
- Patient is overly-preoccupied with his or her relationship with the physician to the detriment of treatment.
- Patient over idealizes the physician, then devalues him or her when high initial expectations are not met.
- Patient ignores customary boundaries that characterize professional relationships.
- Patient shows an attitude of excessive entitlement, together with disregard for the physicians and staff feelings or needs.

Adapted from Griffith, 2005

Headache and psychiatric comorbidity (multi-axial examples)

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<thead>
<tr>
<th>Axis I</th>
<th>Axis I</th>
<th>Axis I</th>
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<tr>
<td>Medication overuse</td>
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Goals of Nonpharmacological Treatment
- Reduced frequency/severity of headache
- Reduced headache-related disability
- Reduced reliance on poorly tolerated or unwanted pharmacotherapies
- Enhanced personal control of pain
- Reduced headache-related distress and psychological symptoms
Behavioral Medicine Program
- Time-limited and goal oriented
- Active participation and personal responsibility
- Education following a coping skills model
- Self-monitoring with headache diary
- Dietary and behavior changes
- Relaxation / biofeedback to foster self-regulation
- Cognitive strategies to enhance coping
- Maximize adherence to drug regimens
- Diagnose and treat comorbid psychiatric problems

General Hints for Headache Control
- Reduce or eliminate caffeine
- Maintain consistent biological rhythms
- Sleep/wake patterns consistent incl. weekends
  - Avoid oversleeping
  - Same bedtime/ time of awakening
- Eat nutritious meals at regular intervals
- Increase aerobic exercise

Relaxation Training
- A core component of behavioral rx for primary headache disorders.
- A self-regulation strategy that teaches patients to consciously reduce muscle tension and autonomic arousal.
- Includes progressive relaxation training, autogenic training, abdominal breathing, imagery. Training progresses to briefer time periods to evoke relaxation response.
- It is often delivered alone or part of biofeedback (BFT) or cognitive behavioral therapy (CBT)

What is biofeedback?
- The use of instrumentation to monitor and display physiological responses that are out of the patients awareness so that they can be “modified” in a more adaptive direction.
- Feedback gives immediate, “objective” information and is usually combined with a relaxation-based therapy.
- Modalities include surface EMG, thermal, HR, PWA, EDR, EEG.

Biofeedback as Self-Regulation
- Enhances internal locus of control
- Learn a non-specific “low arousal” physiologic response and use as a coping skill
- Encourage generalization to the natural environment
- Integrate into “action plans” to better manage exacerbations of pain or fear
- Non-threatening environment to begin to explore psychological issues

Biofeedback Program
Step 1. Clinical interview and assessment
  - Patient begins headache diary

Step 2. Teach body awareness of tension and overarousal
  - Introduce diaphragmatic breathing
  - Make progressive relaxation tape while EMG is monitored

Step 3. Use EMG biofeedback to discriminate between relaxed and tense muscles

Step 4. Introduce passive relaxation using imagery and breathing as relaxation cues
Biofeedback Program

Step 5. Continue EMG training until patient can reliably decrease muscle tension by approximately 50%
- Emphasize scalp, facial, neck, and shoulder relaxation

Step 6. For migraine sufferers, use passive and autogenic training with thermal biofeedback
- Goal is to increase finger temperature to 95°F, or 1°F per minute

Step 7. Conduct frequent short generalization exercises
- Identify prodromal signs and use techniques early

Cognitive-Behavior Therapy

- Attempt to foster an internal locus of control and modify distress-related thoughts
- Rehearse adaptive cognitive and behavioral responses to the development of a migraine
- Accurately interpret body signals
- Develop “action plans”
- Reduce anxiety and depression
- Recognize triggers

Stress Management Training

Acute Migraine

- Preparing for a migraine
- The beginning of the headache
- As intensity builds
- Coping with thoughts and feelings at critical moments
- Self-reflection and evaluation

US Headache Consortium Guidelines

reasons for nonpharmacological therapy

- Patient preference
- Poor responder to preventative meds
- Medical contraindications
- Poor tolerance for pharmacologic interventions
- Pregnancy, planned pregnancy, or nursing
- History of excessive use of acute agents
- High stress level or deficient coping skills

Four Additional Reasons

- Effective
- Augment pharmacologic therapy
- Children and adolescents
- Maximize long term success

Behavioral Treatment for Migraine

Evidence

- Relaxation training
- Thermal biofeedback with relaxation training
- EMG biofeedback
- Cognitive-behavioral therapy

(all considered modestly effective - Grade A)
- 32-49% reduction in headache index

- Behavioral + pharmacologic additive
  (Grade B)

Campbell, Penzien & Wall (2000) Evidence-based guidelines for migraine headaches: Behavioral and physical treatments. AAN Website
Poor Responders to Behavioral Treatment

- Cluster headache
- Continuous or daily chronic daily headache (much better results if 1 or 2 headache-free days per week)
- Medication overuse interferes with the effectiveness of biofeedback although biofeedback can assist in withdrawal and may reduce dropouts
- Menstrual migraine- equivocal data, may depend on the interaction of stressful factors with hormonal changes

Behavioral Treatment
Children and Adolescents

- Cochrane review reported on 15 RCT’s of chronic or recurrent headache.
- Authors concluded that there is very good evidence that psychological treatments, principally relaxation and cognitive behavioral therapy, are effective in reducing the severity and frequency of chronic headache in children and adolescents.

Behavioral Treatment of Migraine
Children and Adolescents

- Most studies were small and only 7 RCT’s used IHS diagnostic criteria.
- Concluded that moderate evidence for an effect of RT, RT + BFT, RT + BFT +/- CBT compared with waiting list controls.
- Limited evidence for an effect of RT+CBT compared with attention placebo.

Combined Treatment for Transformed Migraine Complicated by Analgesic Overuse

- 61 consecutive pts. with transformed migraine and analgesic overuse were briefly hospitalized and treated pharmacologically alone or combined with biofeedback-assisted relaxation.
- Similar levels of improvement for both groups until one year post hospitalization.
- At year 3, combined treatment group had fewer days of HA, reduced amount of analgesics and significantly less relapse.

Stress Management vs. TCA’s in Chronic Tension-type Headache

- 203 adults with CTTHA (mean, 26 HA d/mo) randomly assigned to TCA, placebo, stress management therapy, stress management+TCA
- Outcome of > 50% reduction in HA index over 8 month period
- TCA 38% (faster onset), SM 35%, SM+TCA 64%

Neurofeedback

- Patients learned to control their high negative slow cortical potentials and to habituate and normalize the cortical preactivation level decreasing hyperexcitability and reducing migraine frequency