Chronic Daily Headache: Classification, Diagnosis, and Management

Case Study and Commentary, Morris Levin, MD

Abstract

- **Objective:** To review the classification, diagnosis, and management of chronic daily headache (CDH).
- **Methods:** Qualitative assessment of the literature.
- **Results:** Many patients present to their physicians with frequent or even daily head pain. Careful evaluation of these patients is essential to rule out a number of possible secondary causes of persistent headache, such as trauma, vascular causes, altered intracranial pressure, and neoplastic and infectious processes. Proposed subdivisions of primary CDH include chronic migraine, chronic tension-type headache, new daily persistent headache, and hemicrania continua. All of these may be exacerbated by frequent use of analgesics (analgesic rebound), and all imply a duration of more than 4 hours and a frequency of greater than 15 days per month. A number of medications are available for use in the treatment of CDH, but no controlled double-blind studies have examined pharmacotherapy for CDH.
- **Conclusion:** CDH is a common and frustrating condition. Successful treatment hinges on correct diagnosis. Fortunately, for most causes of CDH treatment is usually at least partially effective.

Many patients present to their physicians with frequent or even daily head pain [1]. In these patients, headache pain is often not severe every day, and generally migraine aura symptoms or accompaniments such as nausea are lacking or infrequent. Nonetheless, these patients often find the recurrent head pain maddening. What to call this headache type is the question. In recent years the term “chronic daily headache” (CDH) has been used by most authors to describe headaches of more than several hours’ duration not due to an underlying “secondary” cause occurring on the majority of days. Estimates of CDH prevalence vary, but several population-based studies suggest that 4% to 5% of all people have CDH [2–4].

In 1988, when the International Headache Society (IHS) produced its groundbreaking “classification and diagnostic criteria for headache disorders, cranial neuralgias, and facial pain,” CDH was not well described [5]. As a result, this diagnostic category was not even listed. The closest the IHS classification came to describing CDH was the term “chronic tension-type headache,” which was simply intended to refer to a very frequent form of tension-type headache. Unfortunately, it did not describe very well the patients who had migrainous features, who were in analgesic rebound, who had new onset of frequent headache (with no features of tension-type headache), or other groups of patients, such as those with postconcussive daily headaches. The revised classification soon to be published by the IHS Classification Committee will include a new category, “chronic migraine,” but it is unlikely that there will be a broad attempt to sort out CDH completely.

It has become clear that the category of CDH includes some important subcategories. Stephen Silberstein and colleagues have proposed a classification scheme for primary
CDH that includes chronic migraine, chronic tension-type headache, new daily persistent headache, and hemicrania continua (Table 1). All of these may be exacerbated by frequent use of analgesics (analgesic rebound), and all imply a duration of more than 4 hours and a frequency of greater than 15 days per month.

Chronic migraine is thought by many to originate from intermittent migraine, with the increase in frequency perhaps resulting from a natural progression of the disease. Many have termed this “transformed migraine” or “evolved migraine” [6] to imply that the problem began with intermittent migraine. This certainly seems to be true in some cases but not all [7]. In some patients, frequent analgesic use is such a key contributing factor that discontinuing analgesics reduces headache frequency back to the level of intermittent migraine.

Chronic tension-type headache tends to be diagnosed in patients with a history of episodic tension-type headache and who have no migraine features such as nausea or auras. Location is generally diffuse. Here, too, analgesic rebound can play a major role, converting intermittent tension-type headaches to the chronic form. Both episodic and chronic forms do not seem to be linked to a muscular etiology (as was thought in the past) and, like migraine, may have a genetic basis [8].

New daily persistent headache (NDPH) was first described by Vanast in 1986 and refers to the relatively acute onset of headache that persists on a frequent basis [9]. Some patients can clearly date the onset of these headaches to a viral or other infectious disease. Chronic tension-type headaches are very similar morphologically to NDPH (absence of migrainous features and diffuse location). Previous tension-type headache theoretically excludes NDPH, but this is problematic because of the high prevalence of intermittent tension-type headache, which in some patients might be coincidental. Chronic posttraumatic headaches are also similar to NDPH.

Hemicrania continua is a relatively rare condition that consists of persistent unilateral head pain varying in intensity that is unresponsive to virtually all known migraine, cluster, and tension-type headache treatment except for indomethacin. In the “remitting form,” head pain can disappear for prolonged periods of time, reminiscent of the timing pattern of cluster headache [10].

Cluster headache, paroxysmal hemicrania, SUNCT (short-lasting, unilateral neuralgiform pain with conjunctival injection and tearing), cranial neuralgias, and stabbing headaches can also occur daily or nearly daily. These are all very distinctive and almost never lead to pain beyond 4 hours’ duration. The exception is the patient with cluster headaches who describes exacerbations of peri orbital pain with autonomic features (ptosis, lacrimation, congestion) but with underlying persistent aching pain between exacerbations of severe pain. This article presents 2 cases that illustrate important issues in evaluation, classification, and treatment of patients with CDH, with emphasis on the evidence (where available) supporting diagnostic and therapeutic decision making.

**CASE ONE**

**Initial Presentation**

A 36-year-old woman presents with complaints of “increasing headaches” that are becoming harder to treat successfully.

**History**

Hemicranial throbbing headaches began in her teens, often occurring around the onset of menses. They were severe at times and sometimes led to missed days at school and, after college, missed work. Nausea was a prominent accompaniment initially but recently has been less prominent. She carried 2 pregnancies to term during which headaches were mild or nonexistent.

She describes prodromal symptoms of blurred vision and at times some visual distortions, including “wavy lines around things.” These symptoms last for only a few minutes. She has never had any cognitive changes at the time of her headaches but does admit to being irritable over the last few months. She has also had some crying spells and feels “trapped” by her duties at home. Sleep is poor. She does no regular exercise.

Over the past year, the headache frequency has increased to approximately 4 days per week. Pain is now often diffuse or “like a band around my head.” Over-the-counter analgesics like ibuprofen, acetaminophen, and combination medications are sometimes helpful but not consistently. She finds that she uses some analgesic medication on a daily basis. She usually does not miss work due to headache but once a month or so she is disabled by a severe headache, more like the headaches she had in her teens and twenties, that is unresponsive to medication.

**Physical Examination**

Vital signs, general examination, and neurological examination

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**Table 1. Proposed Classification for Chronic Daily Headache**

| 1. Chronic migraine (aka transformed migraine) |
| 2. Chronic tension-type headache |
| 3. New daily persistent headache |
| 4. Hemicrania continua |

This patient has CDH, beginning approximately 1 year ago. Possible etiologies include the range of primary CDH (chronic migraine, chronic tension-type headache, hemicrania continua) as well as the many possible secondary causes of headaches (Table 2). Searching for posttraumatic, vascular and infectious causes as well as intracranial neoplasm or altered pressure is essential in a patient reporting a change in headache pattern.

Previous migrainous symptoms (nausea, worsening with menses, improvement with pregnancy) suggest chronic migraine, transformed type. The visual aura symptoms are also suggestive but are vague enough to implicate central nervous system involvement by another mechanism, such as occipital or ocular pathology. Tension-type headache is also possible but less likely. Few features of cluster headache or hemicrania continua are present, so these are unlikely.

There are no prominent symptoms suggestive of intracranial pathology, and neurologic exam is normal. There are several markers for depression, and this may be a contributing factor. She uses over-the-counter medications, so there may be an element of analgesic rebound.

- When is neuroimaging warranted in CDH?

The temptation to order neuroimaging such as CT or magnetic resonance imaging (MRI) is high when patients report chronic head pain and are unresponsive to medication. Guidelines published by the American Academy of Neurology [11] suggest that patients with headache and normal examinations do not benefit from neuroimaging (Table 3). This is almost always a sensible initial approach unless there are clues suggesting more ominous problems, such as change in personality or cognition or a subacute increasing pattern of pain, or in the setting of chronic systemic illness such as AIDS or cancer.

This patient fits so well into the transformed migraine scenario that initial diagnostic testing is quite unlikely to help. On the other hand, if symptoms worsen or pain is intractable to several appropriate treatment approaches, neuroimaging as well as testing for systemic illness such as thyroid dysfunction, inflammatory disease, or other metabolic problems is reasonable.

- What factors in lifestyle and medication use are important areas for initial management?

There is ample evidence for depression in this patient, either situational or perhaps even major depression, which must be

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### Table 2. Differential Diagnosis of Chronic Daily Headache

<table>
<thead>
<tr>
<th>Primary causes</th>
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<tbody>
<tr>
<td>Chronic migraine</td>
</tr>
<tr>
<td>Transformed (from intermittent migraine headache)</td>
</tr>
<tr>
<td>De novo</td>
</tr>
<tr>
<td>Associated with analgesic rebound</td>
</tr>
<tr>
<td>Chronic tension-type headache</td>
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<tr>
<td>Transformed (from intermittent tension-type headache)</td>
</tr>
<tr>
<td>De novo</td>
</tr>
<tr>
<td>Associated with analgesic rebound</td>
</tr>
<tr>
<td>New daily persistent headache</td>
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<tr>
<td>Hemicrania continua</td>
</tr>
<tr>
<td>Cluster headache with persistent underlying headache</td>
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<tr>
<td>Typical cluster headache</td>
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<tr>
<td>Chronic cluster headache</td>
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</tbody>
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<table>
<thead>
<tr>
<th>Secondary causes</th>
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<tbody>
<tr>
<td>Medication and toxins</td>
</tr>
<tr>
<td>Medication overuse headache (analgesic rebound)</td>
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<tr>
<td>Medication effects</td>
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<tr>
<td>Persistent toxin exposure</td>
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<tr>
<td>Posttraumatic</td>
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<tr>
<td>Chronic subdural hematoma</td>
</tr>
<tr>
<td>Postconcussive headache</td>
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<tr>
<td>Exacerbation of existing primary headache</td>
</tr>
<tr>
<td>Vascular causes</td>
</tr>
<tr>
<td>Cerebral arteritis (primary CNS or systemic)</td>
</tr>
<tr>
<td>Cerebral venous thrombosis (due to increased intracranial hypertension)</td>
</tr>
<tr>
<td>Unruptured AVM</td>
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<tr>
<td>Altered intracranial pressure</td>
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<tr>
<td>Idiopathic intracranial hypertension (“pseudotumor cerebri”)</td>
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<tr>
<td>Intracranial hypotension (post LP; spontaneous CSF leak)</td>
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<tr>
<td>Neoplasm</td>
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<tr>
<td>Solid brain parenchymal tumor: primary, metastatic, lymphoma</td>
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<tr>
<td>Skull-based tumor: primary, metastatic</td>
</tr>
<tr>
<td>Meningeal neoplastic disease</td>
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<tr>
<td>Infection</td>
</tr>
<tr>
<td>Chronic viral or other meningoencephalitis (HIV, malaria, EBV)</td>
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<tr>
<td>Abscess(es)—bacterial, fungal, cysticercosis</td>
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</tbody>
</table>

AVM = arteriovenous malformation; CNS = central nervous system; CSF = cerebrospinal fluid; EBV = Epstein-Barr virus; LP = lumbar puncture.

are unremarkable. A computed tomography (CT) scan of the head done 8 years ago was normal.

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addressed. Finding resources for help with household chores, establishing a support group, making some fallback plans for possible time off work when necessary, and finding resources for counseling are all essential. Sleep regulation is also necessary. Many patients with CDH will report worsening with jet lag or other changes in their sleep pattern. Basic sleep hygiene guidelines include maintaining a consistent wake-up time, avoiding naps, and avoiding exercise or central nervous system–stimulating activities close to bedtime. Some clinical evidence suggests that regular exercise is also very beneficial for patients with chronic headaches, although some will report intensification of headache with exercise.

Use of analgesic medication can lead to worsening of CDH via the mechanism of analgesic rebound. Frequent use of analgesic medication seems to lead to increasing frequency and intensity of headaches [12]. Patients in analgesic rebound also seem to respond poorly to prophylactic medication [13]. The pathophysiology of analgesic rebound is still not fully understood, although synaptic changes are postulated to play a role. It seems to be unique to headache disorders, as other pain conditions seem not to be prone to the development of analgesic rebound. This patient was taking enough over-the-counter medications to lead to rebound headaches, but her case raises the controversial question of which comes first: the CDH pattern (which leads to heavy analgesic use) or analgesic overuse (which leads to frequent headaches).

A crucial component in the management of CDH is assessment of progress. A record of headache frequency (kept by the patient) is mandatory because without a record it may be impossible to judge the effectiveness of treatment. In addition, a log showing headache frequency, related symptoms, medication use, menstrual cycle, and sleep patterns can help patients with CDH to see clearly how various factors affect their headaches.

• What psychological conditions are associated with CDH?

The incidence of psychiatric disease in CDH is estimated to be as high as 90% [14]. Illnesses encountered frequently include depression, anxiety, panic disorder, dysthymia, and bipolar disorder. A list of possible comorbid psychiatric conditions associated with CDH is found in Table 4.

Patients with chronic migraine seem to have a higher rate of psychiatric comorbidity than those with chronic tension-type headache [15]. The causal relationships between psychiatric conditions and CDH are not clear. Mood and anxiety disorders may contribute to CDH pain, but the reverse may clearly occur as well. For example, one study found that comorbid depression often improves when headaches improve [16]. From a practical standpoint, these conditions must be addressed in order for patients to be satisfied with their headache treatment.

• What medications are available for CDH prophylaxis?

Unfortunately, there are no controlled, double-blind studies that have examined pharmacotherapy for CDH. Some studies have looked at patients with recurring migraines, with data supporting the use of a number of medications, including β blockers, cyclic antidepressants, calcium channel blockers, divalproex sodium, methysergide, and more recently, topiramate. As a result these medications have been employed by many for prophylaxis of the various types of primary CDH, and open-label studies are promising for all.

There are several studies supporting the use of amitriptyline in patients with chronic tension-type headache [17–19]. There is some evidence that divalproex sodium is effective in CDH [20]. Other anticonvulsants, such as zonisamide and levetiracetam, and the antispasmodic agent tizanidine may also prove useful in CDH prophylaxis. Choosing from among these possibilities is generally a matter of personal choice, but most would probably place amitriptyline, and other cyclic antidepressants, at the top of the list (Table 5).

One of the major problems in drawing firm conclusions
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regarding efficacy of treatment in CDH is the variability in diagnostic criteria used by different investigators. As the distinctions between types of primary CDH become clearer, this issue will be much less problematic.

• What nonpharmacologic or other treatments are potentially beneficial to patients with CDH?

Biofeedback, relaxation training, and cognitive behavioral therapy have been useful in patients with chronic headaches [21,22], but no blinded, controlled studies are available for CDH. Acupuncture seems not to be useful [23]. The U.S. Headache Consortium published their evidence-based guidelines on the management of migraine in 2000 and concluded that “relaxation training, thermal biofeedback combined with relaxation training, EMG biofeedback, and cognitive behavioral therapy are all somewhat effective in preventing migraine when compared with controls” [24]. Many feel these recommendations pertain to CDH, but further study is necessary.

Botulinum toxin therapy has been used for patients with intractable CDH with some striking anecdotal successes, but evidence at this time is primarily suggestive.

• What acute medications are reasonable choices for “breakthrough” headaches?

Most authors have extrapolated the very positive results of controlled studies of triptans and other acute migraine medications to include exacerbations of CDH. This seems very reasonable in patients with chronic migraine. It is also logical in patients with the tension-type headache form of CDH on the basis of the so-called “Spectrum Study” [25], which showed that sumatriptan will successfully treat tension headaches, particularly in patients who also have migraine headaches. Isometheptene can be helpful as well, although it is less potent than triptan medication.

Much of the head pain experienced by patients with chronic tension-type headache is mild enough to respond to nonsteroidal antiinflammatory drugs (NSAIDs) such as ibuprofen and naproxen. There are no controlled studies providing clear evidence of this approach, but intermittent tension-type headache has been shown to respond well to NSAID medication [26].

Follow-up of Case One

The patient was educated about the importance of analgesic rebound and was successfully weaned off all previous analgesics. She found that naproxen sodium was effective for the more moderate headaches and responded very well to an oral triptan medication when she experienced severe migrainous headaches. Sodium valproate was effective in reducing the headache frequency by 50% at a dose of 1000 mg daily but led to some weight gain and mild tremor. At the patient’s request, it was tapered and discontinued. When this happened, there was an increase in moderately severe headaches. Her sleep problem persisted and she was placed on nortriptyline 25 mg in the evening, which was later increased to 50 mg at bedtime. This seemed to control not only the headaches (bringing frequency down to about 1 to 2 mild headaches per week), but also seemed to help sleep significantly. Nonpharmacological sleep hygiene measures were also helpful. Mood was improved. Counseling was still recommended, but the patient preferred not to start this. She did become involved in regular exercise, which she felt was helpful.

CASE TWO

Initial Presentation

A 41-year-old man presents complaining of frequent diffuse aching head pain which began following a work site injury 14 months ago.

History

The patient, a lumber yard worker, was well until hitting his right frontal region on a protruding 2 × 4 at work. He does not recall losing consciousness but remembers lying on the ground looking up and not knowing where he was before being taken to the emergency room by ambulance.

Table 5. Pharmacologic Agents Used in the Treatment of Chronic Daily Headache

<table>
<thead>
<tr>
<th>Antidepressants</th>
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<tr>
<td>Amitriptyline</td>
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<td>Nortriptyline</td>
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<tr>
<td>Doxepin</td>
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<td>β Blockers</td>
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<td>Propranolol</td>
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<td>Nadolol</td>
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<tr>
<td>Atenolol</td>
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<tr>
<td>Calcium channel blockers</td>
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<tr>
<td>Verapamil</td>
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<tr>
<td>Amlodipine</td>
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<tr>
<td>Flunarizine</td>
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<tr>
<td>Anticonvulsants</td>
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<tr>
<td>Valproate</td>
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<td>Gabapentin</td>
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<td>Topiramate</td>
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<td>Antispasmodics</td>
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<tr>
<td>Tizanidine</td>
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</table>

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The head pain is nearly constant, and since the accident he has also had bouts of spinning sensations with nausea, neck pain, difficulty concentrating, memory lapses, poor sleep, and irritability. He lost his job because his employer “is a jerk.” There is a lawsuit pending regarding the accident.

He admits to occasional “regular headaches” that would respond to acetaminophen or combination over-the-counter medications prior to his injury. He drinks 3 beers daily on average and smokes 1 pack of cigarettes per day. He drinks 4 cups of coffee daily “to stay awake.” He is now taking approximately 6 to 8 acetaminophen-aspirin-caffeine tablets per day along with 3 to 4 butalbital-acetaminophen-caffeine tablets for head and neck pain.

Physical Examination

The patient appears healthy looking. Vital signs, general examination, and neurological examination are unremarkable. CT scan of the head done in the emergency room on the day of his accident was normal.

• What are possible ways that head trauma can lead to CDH?

Trauma can induce subdural or epidural hematoma. Superficial nerve injury can lead to neuralgia, particularly in the supraorbital nerve and greater occipital nerves. Unidentified cervical spine injury can lead to cervicogenic headache. Posttraumatic cerebrospinal fluid leaks can lead to low pressure headaches, which can persist if dural tears do not heal. Head injury can also lead to intracranial or subarachnoid hemorrhage that can lead to cerebrospinal fluid absorption problems and intracranial hypertension or hydrocephalus. But the majority of patients who experience persistent headaches following nonpenetrating head injury do not have evidence for any of these. These are termed “posttraumatic headaches” and are generally bilateral, non-throbbing, and mild to moderate in intensity. Headache can be quite frequent as it is in this patient, and when it occurs on more than 15 days per month it often closely resembles chronic tension-type headache.

• What are the diagnoses in this case? Should any further testing be done?

In addition to posttraumatic CDH, this patient seems to have other posttraumatic symptoms (ie, vertigo, nausea, cognitive dysfunction, poor sleep and irritability), consistent with postconcussive syndrome (PCS). In addition, analgesic rebound seems certain, and nicotine, ethanol, and caffeine overuse complicate this patient’s headache disorder as well. Previous tension-type headache is likely, so chronic tension-type headache is a possible diagnosis as well.

Subdural hematoma is unlikely given the normal examination, but repeat CT or MRI of the head would be appropriate to rule this out as well as the possibility of posttraumatic hydrocephalus (also unlikely without the usual exam finding of truncal ataxia). In addition, evidence of parenchymal injury should be sought; while this finding is not likely to change treatment, it would provide important information regarding prognosis and disability. In addition, it is important to know whether there has been any hepatotoxicity due to ethanol or acetaminophen use, so liver enzymes should be checked.

• What is postconcussive syndrome? What is its pathogenesis?

PCS refers to a collection of symptoms that occur in a number of victims of closed head injury. The most common symptom is headache, which interestingly can take the form of any of the intermittent primary headache types or CDH. In addition, cognitive and mood disturbances, vertigo, sleep dysfunction, and other symptoms can occur (Table 6). Some patients with PCS improve, including the headaches, but others seem to follow a more or less disabling chronic course.

“Diffuse axonal injury” due to inertial forces acting on deeper cerebral structures is often proposed as the mechanism for the production of this syndrome. There is a great deal of controversy regarding diagnosis and treatment of PCS, which can be complicated by considerations of secondary gain. Attempts to devise diagnostic tests for PCS, including late evoked potentials and neuropsychological testing, have been disappointing.
**CHRONIC DAILY HEADACHE**

- **What treatments should be initiated in this patient?**

Due to the intense frustration most of these patients feel, counseling is essential. Neuropsychological assessment is important in determining cognitive deficits and strengths and establishing levels of disability. Individual symptoms such as headache, depression, and sleep dysfunction can be treated in many patients successfully by appropriate medications, such as antidepressants and soporifics. Cognitive difficulties are usually resistant to treatment. Medications useful in other types of CDH are reasonable choices, including β blockers, cyclic antidepressants, and antiepileptics. Insomnia may respond to soporifics. Mood disturbances might respond to selective serotonin reuptake inhibitor antidepressants. Fatigability and attentional problems can sometimes respond to stimulant medications.

- **What is the most effective approach in reversing analgesic rebound?**

This patient will almost certainly not improve unless analgesic rebound is reversed. Here, the offending agents include the over-the-counter combination preparation acetaminophen-aspirin-caffeine and the butalbital-acetaminophen-caffeine prescription combination. These 2 preparations are widely prescribed and notorious for their propensity to cause analgesic rebound. In this case, they must all be discontinued. In addition, the 4 cups of coffee daily is another likely contributor to rebound headache and must be reduced. Nicotine and ethanol should also be discontinued as quickly as possible.

The difficulty most patients experience discontinuing even one of the many substances this patient is habituated to suggests that he will not be willing or able to comply with these plans. In addition, discontinuing butalbital, even in moderate doses, can produce unpleasant or even dangerous side effects. Substituting phenobarbital or benzodiazepine medications, which can slowly be tapered, is one option. Corticosteroids in short courses can also make discontinuation of rebound-producing drugs easier by reducing withdrawal symptoms.

However, if outpatient treatment fails, inpatient treatment can be successful. In the hospital, rebound-producing agents are discontinued while medications useful in reducing withdrawal symptoms are titrated. Prophylactic medications can be initiated at the same time. Intravenous dihydroergotamine has been shown to be particularly helpful in gaining control over the intractable head pain [27,28]. Psychological comorbidity can be assessed while in hospital and treatment plans can be initiated.

**Follow-up of Case Two**

The patient is very reluctant to discontinue the butalbital-containing medication but was finally convinced to taper and stop this medication and the other over-the-counter medications when liver enzymes were found to be mildly elevated. These levels normalized after several weeks. Amitriptyline 75 mg at bedtime in addition to clonazepam 0.5 to 1 mg on occasion for insomnia was helpful in improving sleep and reducing head pain. Isometheptene was a reasonably effective analgesic/abortive agent. He was not able to quit smoking cigarettes but was able to reduce. He discontinued alcohol completely. He reduced his caffeine intake to 3 cups daily. Neuropsychological testing revealed significant difficulties with attention, and a stimulant medication seemed to improve performance on a second battery of tests. Vocational counseling was extremely helpful.

**Conclusion**

CDH can be due to a number of primary and secondary causes. Analgesic rebound is a common contributory factor. Lack of consistent classification of CDH has made systematic study of treatment difficult, but there are a number of promising prophylactic and analgesic pharmacologic options. In addition, nonpharmacologic therapies and lifestyle adjustment is often extremely helpful.

**References**

CASE-BASED REVIEW


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EVALUATION FORM: Chronic Daily Headache: Classification, Diagnosis, and Management

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Part 1. Please respond to each statement. Strongly Agree Strongly Disagree

5 4 3 2 1

I was provided with new information pertinent to my practice. ❑ ❑ ❑ ❑ ❑
I reaffirmed a specific skill or knowledge. ❑ ❑ ❑ ❑ ❑
This article will help with clinical decision making. ❑ ❑ ❑ ❑ ❑
Relevant clinical outcomes are addressed. ❑ ❑ ❑ ❑ ❑
The case is communicated in a manner that kept my interest. ❑ ❑ ❑ ❑ ❑
The case presentation is realistic and effective. ❑ ❑ ❑ ❑ ❑
I could easily interpret the tables and figures. ❑ ❑ ❑ ❑ ❑
My attitude about this topic changed in some way. ❑ ❑ ❑ ❑ ❑

Additional comments: ______________________________________________________________________________________
________________________________________________________________________________________________________

Part 2. Please complete the following sentence.

As a result of reading this case study, I . . .
❑ see no need to change my practice.
❑ will seek more information before modifying my practice.
❑ intend to change the following aspect(s) of my practice: (Briefly describe)
________________________________________________________________________________________________________
________________________________________________________________________________________________________


Signature: ___________________________ Date: ___________________________

Part 4. Identifying information: Please PRINT legibly or type the following:

Name: ___________________________ Fax number ___________________________
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Medical specialty: ___________________________

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