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July 14, 1998

Mr. John B. Hatfield, Jr.
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219 W. Washington St.
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Re: Ronnie Lee Kimble

Dear Mr. Hatfield:

On July 12, 1998 I had the opportunity to discuss Ronnie Kimble's history of sleep complaints with you and then to interview Mr. Kimble as requested. I have subsequently reviewed the medical records you have provided with particular attention to sleep studies that he had in June 1995 and January 1997 with the related evaluations by specialists in Neurology, Otolaryngology and Sleep Disorders Medicine(Dr. DeBeck).

Mr. Kimble has given a consistent description across several years and through different interviewers, of a perception of excessive daytime sleepiness not obviously associated with impaired nocturnal sleep.

He has described episodes of being asleep on his feet, for instance while marching, and similar near automatic behavior.

Nocturnal polysomnograms(sleep studies) on the 2 dates showed fairly similar results. He does snore but does not demonstrate significant objective evidence of obstructive apnea, narcolepsy or severe pathologic daytime sleepiness. The Multiple Sleep Latency Test for measuring objective daytime sleepiness done with the 1997 study showed mild daytime sleepiness by our local laboratory criteria with a mean latency of 10.4 ~~latency of 10.4 minutes~~ and no sleep-onset REM. His earlier study in 1995 showed a mean latency of 12.4 minutes with 1 REM episode. These results indicate somewhat more daytime sleepiness than I would consider routine in a healthy young man who had been getting adequate nocturnal sleep.

It is possible that he either needs more sleep than the average person, or Alternatively, he might have a very mild incomplete form of narcolepsy which is a disorder of excessive daytime sleepiness.

TO; MR. HATFIELD July 14, 1998

RE; R. KIMBLE

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I think I would put him in the category of Idiopathic Hypersomnia. I don't believe he needs further studies now unless he develops other evidence of neurologic problems.

I am sending you some pages from a coding book which describe this disorder.

This disorder, and associated sense that he may be somewhat different from other people, may cause some degree of depression but I get no impression from Mr. Kimble that it would significantly affect his ability to make decisions or to control significant actions. I do not get the impression that he has difficulty distinguishing reality from dreams.

He does describe occasionally vivid dreams and is able to relate them the next day. To the extent that dreaming may represent some mental processing of daytime issues, we are not surprised if dreams recalled the next day seem to have threads of relevance to issues of daytime life but I can offer no formal opinion that the content of dreams actually means anything very specific.

I hope these thoughts are of help to you.

Respectfully,

Clinton D. Young, M. D.

CDY; cm

Moderate: More than two episodes of prolonged sleep periods per year. Tl symptoms produce a moderate impairment of social or occupational functio
Severe: More than two episodes of prolonged sleep episodes per year. Tl symptoms produce a severe impairment of social or occupational functio

Duration Criteria:

Acute: Duration 1 month or less.
Subacute: Duration more than 1 month but less than 6 months.
Chronic: Duration 6 months or longer.

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Idiopathic Hypersomnia (780.54-7)

Synonyms and Key Words: Dependent, idiopathic, or NREM narcolepsy; idi pathic; idiopathic central nervous system (CNS); hypersomnia; functional, mixe or harmonious hypersomnia. Idiopathic hypersomnia is the preferred term. Do not include posttraumatic hypersomnia, which is described elsewhere.

Essential Features:

Idiopathic hypersomnia *is a disorder of presumed central nervous system cause that is associated with a normal or prolonged major sleep episode and excessive sleepiness consisting of prolonged (1–2 hours) sleep episodes of non-REM sleep.*

Idiopathic hypersomnia is characterized by a complaint of constant or recurre excessive daytime sleepiness, typically with sleep episodes lasting 1 or seve hours in duration. It is enhanced in situations that allow sleepiness to beco manifest, such as reading or watching television in the evening. The major sl episode may be prolonged, lasting greater than 8 hours. The capacity to aro the subject may be normal, but some patients report great difficulty waking and experience disorientation after awakening.

Associated Features: Some patients may complain of paroxysmal episodes sleepiness culminating in sleep attacks, as in narcoleptic patients. Most often th attacks are preceded by long periods of drowsiness. Naps are usually longer th

in narcolepsy or sleep apnea, and short naps are generally reported as being nonrefreshing. Often as disabling as narcolepsy, idiopathic hypersomnia has an unpredictable response to stimulants such as the amphetamines and methylphenidate. These patients often report more side effects, such as tachycardia or irritability, and such medications tend to exacerbate the associated symptoms of headache.

Associated symptoms suggesting dysfunction of the autonomic nervous system are not uncommon. They include headaches, which may be migrainous in quality, fainting episodes (syncope), orthostatic hypotension, and, most commonly, peripheral vascular complaints (Raynaud-type phenomena with cold hands and feet).

Course: The disorder is initially progressive, but often is stable by the time of diagnosis. It appears to be lifelong.

Prevalence: This syndrome is estimated to account for 5–10% of patients who bring a complaint of sleepiness to a sleep clinic. This estimate may vary considerably depending on the criteria used to diagnose excessive sleepiness (see polysomnographic features below).

Age of Onset: At the time of presentation, most patients have had the disorder for many years. Idiopathic hypersomnia usually becomes apparent during adolescence or the early twenties. Many changes, which are frequently associated with stress or increased tension, take place in the patient's life at that time. Consequently, the disorder is often difficult to diagnose at an early stage and may be confounded with other disorders of excessive sleepiness.

Sex Ratio: There are no gender differences.

Familial Pattern: A familial manifestation of this disorder can be observed. However, studies using standard diagnostic criteria and procedures are needed to estimate the ratio of familial to isolated cases, as well as the mode of transmission.

Polysomnographic Features: Polysomnographic monitoring of nocturnal sleep usually demonstrates normal quantity and quality of sleep. Sleep at night is not disrupted as in narcolepsy. The sleep latency may be reduced in duration and the sleep period tends to be of either normal or slightly greater than normal duration. Slow wave sleep can be normal or slightly increased in amount and percentage.

Polysomnographic monitoring should rule out sleep-onset REM periods, pathological apnea indexes, and periodic movements during sleep.

Sleep latencies are typically short in the daytime in idiopathic hypersomnia. The multiple sleep latency test (MSLT) usually demonstrates a sleep latency of less than 10 minutes. The clinical severity of idiopathic hypersomnia may not closely correlate with the MSLT results, as latencies above 5 minutes are not uncommon in patients with clinically severe hypersomnia.

Other Laboratory Test Features: Human leukocyte antigen (HLA) determination may be helpful in the diagnosis. Most narcoleptic patients carry the HLA-

DR2, whereas only HLA-Cw2 incidence is elevated in idiopathic hypersomnia and the incidence of HLA-DR2 in this population is found to be either normal or even decreased.

Differential Diagnosis: Idiopathic hypersomnia must be differentiated from several other disorders of sleepiness, such as narcolepsy, sleep apnea syndromes, posttraumatic hypersomnia, periodic limb movement disorder, and sleepiness associated with affective disorders. The polygraphic features usually help to distinguish idiopathic hypersomnia from the sleep apnea syndromes, narcolepsy, and periodic limb movement disorder. It should be distinguished from long sleepers who do not have objective evidence of excessive sleepiness after a full major sleep episode.

The differential diagnosis of sleepiness associated with low-grade, chronic depression may be more difficult. Although no systematic studies have been performed on the personality profile of patients with idiopathic hypersomnia, clinical experience reveals the presence of polymorphic psychological disturbance in a large number of these patients. It is mainly polysomnographic features with short sleep latencies and normal sleep organization that can single out idiopathic hypersomnia. The diagnosis of sleepiness associated with dysthymia and related mood disorders relies primarily on the identification of depressive symptoms during the clinical evaluation, but psychometric tests may help in the diagnostic process. Patients with idiopathic hypersomnia often tend to deny subjective dysphoria, and depression should be inferred from restriction of interests, anhedonia, and observational signs of depression in facial expression or posture. A family history of mood disorder can also be helpful.

Two other syndromes of excessive sleepiness must be ruled out before diagnosing the primary form of idiopathic hypersomnia. First, sleepiness may be an early symptom of progressive hydrocephalus in children and adults. Other clinical features of hydrocephalus may be completely absent at that point. Computed tomography, skull X-ray, and electroencephalography may be necessary to eliminate this diagnosis. Secondly, 6–18 months after head trauma, patients may gradually develop posttraumatic hypersomnia showing all features of the primary form of idiopathic hypersomnia.

Diagnostic Criteria: Idiopathic Hypersomnia (780.54-7)

A. A complaint of prolonged sleep episodes, excessive sleepiness, or excessively deep sleep.
B. Presence of a prolonged nocturnal sleep period or frequent daily sleep episodes.
C. The onset is insidious, and typically before age 25 years.
D. The complaint is present for at least 6 months.
E. The onset does not occur within 18 months of head trauma.
F. Polysomnography demonstrates one or more of the following:
   1. A sleep period that is normal or prolonged in duration;
   2. Sleep latency less than 10 minutes;
   3. Normal REM sleep latency; and

4. An MSLT that demonstrates a sleep latency less than 10 minutes;
5. Less than two sleep-onset REM periods.

G. Absence of any medical or psychiatric disorder that could account for the symptom.

H. Does not meet the diagnostic criteria of any other sleep disorder causing excessive sleepiness, e.g., narcolepsy, obstructive sleep apnea syndrome, or posttraumatic hypersomnia.

Minimal Criteria: A plus B plus C plus D.

Severity Criteria:

Mild: Mild sleepiness as defined above.
Moderate: Moderate sleepiness as defined above.
Severe: Severe sleepiness as defined above.

Duration Criteria:

Acute: Not applicable.
Subacute: Duration more than 6 months but less than 1 year.
Chronic: Duration 1 year or longer.

Bibliography:

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Roth B. Narcolepsy and hypersomnia. Basel: Karger, 1980; 310.

Posttraumatic Hypersomnia (780.54-8)

Synonyms and Key Words: Posttraumatic hypersomnia, secondary hypersomnolence.

Essential Features:

Posttraumatic hypersomnia *is excessive sleepiness that occurs as a result of a traumatic event involving the central nervous system.*

This disorder clearly represents an alteration of the patient's pretrauma sleep patterns. The hypersomnia is characterized by frequent daytime sleepiness that may or may not be able to be resisted, with consequent sleep episodes. The duration of the major sleep episode may be prolonged compared with the prior sleep length.

Associated Features: The sleepiness is usually seen in the context of other posttraumatic encephalopathic symptoms, such as headaches, fatigue, difficulty