Insomnia Classification Brings New Terminology

BY BRUCE JANCIN

Denver Bureau

Denver — Recent changes in the International Classification of Sleep Disorders definition of insomnia reflect a significant evolution in expert opinion regarding the fundamental nature of these disorders, according to speakers at a satellite symposium held in conjunction with the annual meeting of the Associated Professional Sleep Societies.

"One of the major differences that you'll find in the ICSD-2 versus -1 is that we now have insomnia defined as a disorder, and that all of the specific categories of insomnia subtypes have to meet the same basic general definition for insomnia disorder," explained Daniel J. Buysse, M.D., professor of psychiatry at the University of Pittsburgh.

"The important thing, as we're educating our colleagues in the medical field, is that for many, many years we have been taught—and we've taught others—that insomnia is a symptom, not a disorder. I think that this is really undergoing an evolution. We're starting to think of insomnia as a disorder that's commonly comorbid with other medical and psychiatric condi-

tions and may often warrant treatment in and of itself, not as part of the treatment for the other disorders," he added.

The ICSD-2 categorization of insomnia released last year by the American Academy of Sleep Medicine was formed on a template provided by an earlier academy working group that published the first formal research diagnostic criteria for insomnia (Sleep 2004;27:1567-96).

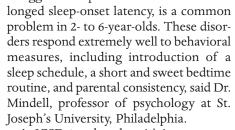
The core ICSD-2 criteria for diagnosis of any insomnia disorder are that the patient must experience one or more sleep-related complaints that occur despite adequate opportunity and circumstances for sleep and result in some form of daytime functional impairment. Some of these manifestations of sleep-related daytime impairment are obvious, such as sleepiness, irritability, and difficulty in concentrating. Others are not necessarily so, including GI symptoms, tension headaches, and error-proneness at work.

Jodi A. Mindell, Ph.D., noted that physicians who work with children will find they're encountering some new terminology as a result of the ICSD-2 changes. For example, two of the most common childhood sleep disorders listed in ICSD-1—sleep-onset association disorder and limit-

setting sleep disorder—have been combined in ICSD-2 into a single rubric: behavioral insomnia of childhood. Within this new category are three subtypes: sleeponset association type, limit-setting type, and the combined type that involves both.

The sleep-onset association subtype typically involves children aged 6 months

to 3 years who experience repeated nighttime awakenings that require parental intervention because of an inability to self-soothe. The limit-setting subtype, involving bedtime struggles and pro-



As ICSD-2 makes clear, it's important to check for physiologically based sleep problems in every child with a sleep problem.

Among the most common are restless legs syndrome and periodic limb movement disorder, which has a strong family history component. In addition, obstructive sleep apnea has a 1%-3% prevalence in childhood, with a peak during ages 2-7 years; the most common cause is large tonsils, she said at the symposium sponsored

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Parasomnias, in-

'All of the specific cludin ing, so insomnia subtypes have to meet the same basic general definition.' children reason

DR. BUYSSE

cluding sleepwalking, sleep terrors, and confusional awakening, are common in young children. "For some reason, people think they don't start until the preschool

years, but we see them often even in 1-yearolds," Dr. Mindell said.

Dr. Buysse noted that paradoxical insomnia is a new term introduced in ICSD-2. It replaces sleep-state misperception, the former term for a patient's subjective feelings that a sleep problem is present in the absence of objective findings.

"It's basically a mismatch between what you might record with polysomnography and what the patient says," he said. ■

Quantitative EEG Diagnostic Of Vascular Dementia Severity

BY KERRI WACHTER

Senior Writer

PORTO, PORTUGAL — Quantitative EEG shows promise as a clinical diagnostic tool that is sensitive enough to distinguish mild from moderate subcortical dementia, according to one study presented at the Fourth International Congress on Vascular Dementia.

"Quantitative EEG results correlated strongly with mental impairment in subcortical vascular dementia," said Dr. Malgorzata Gawel, of the Medical University of Warsaw.

The white matter lesions and lacunar ischemic stroke that are associated with subclinical vascular dementia lead to the destruction of neurons and neuronal synapses and also cause cholinergic deficits. In turn, cholinergic deficits lead to changes in synaptic bioelectric potential, which may be picked up in EEG signals. In particular, these changes result in a slowing of background brain activity and the presence of slow brain waves (delta and theta), Dr. Gawel said.

The researchers used visual and quantitative EEG to evaluate 31 patients with subcortical vascular dementia (mean age 72 years, 19 women) and 14 healthy controls (mean age 70 years, 8 women). Subcortical vascular dementia was diagnosed using the National Institute of Neurological Disorders and Stroke—Association Internationale pour la Recherche et l'Enseignement en Neurosciences (NINDS-AIREN) criteria and criteria developed by Timo Erkinjuntti of the University of Helsinki.

The group with dementia was further divided into two subgroups based on severity of cognitive impairment. The first group (16 patients) had mild cognitive impairment, defined as a score of 19-23 on the Mini-Mental State Examination (MMSE). The second group (15 patients) had moderate cognitive impairment, defined as a score of 11-18 on the MMSE.

EEG was performed for 20 minutes with eyes closed. Visual inspection of EEGs involved an 8-point scale, with 1 being normal and 8 representing severe abnormalities. There was no significant difference between visual EEG results for the two vascular dementia subgroups. There was a significant difference on the visual EEG results between the control group and both subcortical vascular dementia subgroups. There was no correlation between visual EEG results and cognitive impairment measured by the MMSE.

Quantitative EEG was performed for 5 minutes, following the visual EEG. The researchers calculated the power ratio of alpha waves to theta waves (A/T); the ratio of alpha waves to delta waves (A/D); and the ratio of alpha waves to the sum of theta and delta waves (A/T+D). They also calculated the mean waves frequency for all derivations (amplifier/electrode configurations), with particular interest in the occiptal and temporal derivations.

There was significant difference between the quantitative EEG results for the patients with mild and moderate dementia for all parameters. There was a significant correlation between all parameters and cognitive impairment.

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