(0.2% to 15.1%) and cardiologists (54.8% to 68.6%) after the advisory. The change in the use of a modified screen was 7.4% to 34.5% for noncardiologists and no increase for cardiologists (7.8% to 5.9%). The proportion of noncardiologists willing to prescribe stimulant medications in children with potential or actual cardiac issues showed a considerable decrease. These changes in practice following the advisory have occurred despite the lack of studies to address the actual cardiac risks of stimulant medications. Consensus recommendations are needed to determine whether screening before starting treatment is necessary and which children may be treated cautiously. (Conway J, Wong KK, O'Connell C, Warren AE. Cardiovascular risk screening before starting stimulant medications and prescribing practices of Canadian physicians: Impact of the Health Canada Advisory. **Pediatrics** October 2008;122:e828-e834). (Respond: Jennifer Conway MD. E-mail: jennifer.conway@iwk.nshealth.ca).

COMMENT. A full cardiac screen consists of all of the following: ask about a history of congenital heart disease, family history of sudden death, and family history of early coronary infarct, record the pulse and blood pressure, check for murmur, and obtain ECG. A modified screen differs only in that the ECG is performed selectively for children with abnormal exam.

In the US, the American Academy of Pediatrics, contrary to an American Heart Association statement advising pre-treatment ECG, considers routine ECG before starting stimulant therapy for ADHD to be unnecessary. Cardiac history and examination are recommended, and ECG and cardiac consultation, only if clinically indicated. (Perrin JM et al. **Pediatrics** 2008;122:451-453; **Ped Neur Briefs** Sept 2008;9:66).

SLEEP DISORDERS

OLFACTORY DYSFUNCTION AND HYPOCRETIN IN NARCOLEPSY

CSF orexin A (hypocretin-1) is decreased or absent in narcoleptic patients with cataplexy. Researchers at Christian-Albrechts University Kiel, Germany, analyzed olfactory sensation of 10 adult patients and 10 controls. Orexin-A was applied intranasally in 7 of the patients, and odor detection thresholds for 2-phenyl-ethyl alcohol were measured. Patients showed significantly lower scores for olfactory threshold, discrimination, and identification, separately, and for the total scores. In all patients, the odor detection olfactory threshold score increased after intranasal orexin A compared to placebo. Lack of CNS orexin is involved in the pathophysiological mechanism underlying olfactory dysfunction in narcolepsy. (Baier PC, Weinhold SL. Huth V, Gottwald B, Ferstl R, Hinze-Selch D. Olfactory dysfunction in patients with narcolepsy with cataplexy is restored by intranasal orexin-A (hypocretin-1). **Brain** Oct 2008;131:2734-2741). (Respond: Dr Paul Christian Baier, Department of Psychiatry and Psychotherapy, Christian-Albrechts University Kiel, Niemannsweg 147, 24105 Kiel, Germany).

COMMENT. Orexin A and B are neuropeptides synthesizd by neurons in and around the lateral hypothalamus and olfactory tract. Orexin is involved in sleep wake regulation. Olfactory dysfunction, an early predictor of Parkinsonism, is also a sign of narcolepsy with cataplexy. Correction of the associated orexin A defciency in the CSF by intranasal

administration will restore the olfactory sensation of patients with narcolepsy. The authors comment that orexin A intranasally is, theoretically, a promising treatment for narcolepsy and may be considered for future trial.

DIAGNOSIS AND MANAGEMENT OF NARCOLEPSY REVIEWED

Researchers at Duke University Medical Center, and Veterans Affairs Medical Center, Durham, NC, review the epidemiology, pathophysiology, diagnosis, and treatment of pediatric narcolepsy. Narcolepsy is a disorder of rapid eye movement (REM) sleep characterized by excessive daytime somnolence, associated with sleep paralysis, hypnagogic (when falling asleep) and hypnopompic (when awakening) hallucinations, and cataplexy. Prevalence is 0.05% in the US and Europe; 0.18% in Japan; and 0.002% in Israel; greater in males than females. Onset highest in second decade, with peaks at 14 yrs and 35 yrs. Etiology is unknown, possible neurodegenerative with autoimmune component. Patients with narcolepsy and cataplexy share the same HLA genotype. Predominantly sporadic, sometimes familial, only 25-31% concordance in twin studies. CSF levels of hypocretins less than 110 pg/ml are diagnostic of narcolepsy with cataplexy. Obesity with narcolepsy is associated with low 24 hr leptin levels, a hormone secreted by adipose tissue. Narcolepsy is idiopathic or secondary (symptomatic) and caused by hypothalamic tumors, head trauma, multiple sclerosis, vascular, and encephalitic disorders. In addition to the classic tetrad of narcolepsy symptoms, semi-purposeful, automatic behavior is common during the day, sometimes misdiagnosed as epilepsy, and frequent noctural awakenings. Obesity and obstructive sleep apnea frequently coexist. The interval between symptom-onset to diagnosis is about 10 years. Misdiagnosis is common in children with narcolepsy, leading to delay in treatment. The history combined with polysomnography and mean sleep latency (MSLT) <8 minutes are used in diagnosis in adults and in children older than 8 years. Two or more sleep onset REM sleep periods (SOREMP) in a MSLT within 15 min of sleep onset are consistent with narcolepsy. MRI is normal in idiopathic narcolepsy. Treatment includes lifestyle changes, brief naps, caffeine, reduced carbohydrates or Atkins diet, and pharmacotherapy (methylphenidate, amphetamines, and non-approved pediatric use of modafinil (Provigil), and sodium oxybate (Xyrem). Sodium oxybate is particularly effective in cataplexy in adults. Antidepressants have also been used to treat cataplexy and hypnogogic hallucinations in adults. (Peterson PC, Husain AM. Pediatric narcolepsy. **Brain Dev** Nov 2008;30:609-623). (Respond: A.M. Husain, E-mail: Aatif.husain@duke.edu)

COMMENT. A high index of suspicion is required in the diagnosis of narcolepsy. This review provides an excellent account of the diagnosis and treatment of narcolepsy. A sleep specialist reports the highest success rate for correct diagnosis among neurologists (55%), psychiatrists (11%), and pediatricians (0%). (Kryger MH et al. **Sleep** 2002;25:36-41).

From the Archives: Idiopathic narcolepsy: a disease sui generis. By Adie WI. **Brain** 1926;49:257-306 and The narcolepsies. By Kinnear Wilson SA. **Brain** 1928;51:63-109. Adie describes cases seen at Queen Square and misdiagnosed as epilepsy, and summarizes earlier reports by Gelineau (1880). Examples of patients are two soldiers courts-martialed for falling asleep on listening-post duty. Adie coins the term cataplexy associated with narcolepsy. He considers the etiology an endocrine-nervous disorder, a disease sui generis, a proposal