(Respond: Timothy E Wilens MD, Pediatric Psychopharmacology Program, Masachusetts General Hospital, Harvard Medical School, 55 Parkman St, Yawkey 6A, Boston, MA 02114. E-mail: twilens@partners.org).

COMMENT. If we start stmulant treatment, will it increase the risk of drug abuse in my child with ADHD at a later age? This is a common concern and question of parents faced with the prospect of long-term therapy with stimulants for ADHD in a child of school age. Studies have demonstrated that ADHD is a risk for SUDs (Wilens TE et al. **J Nerv Ment Dis** 1997;185:475-482). Since stimulants are potential drugs of abuse, the assumption follows that they may increase the risk of cigarette smoking and SUDs when usd to treat ADHD. Opinions vary but most studies, including a meta-analysis, show no increase or even a protective effect against subsequent cigarette smoking or SUDs in adolescents and adults following early stimulant therapy for ADHD. (Wilens TE et al. **Pediatrics** 2003;111;179-185). The above group of investigators previously demonstrated a reduced risk for SUDs in adolescent boys who previously received stimulant therapy for ADHD (Biederman J, Wilens TE, et al. **Pediatrics** 1999;104:e20). The present study finds similar results in adolescent girls with ADHD who had received early stimulant treatment, even in those with comorbid conduct disorder. In addition to a reduction in risk of SUDs, cigarette smoking was also reduced, and risk of alcohol abuse or dependence was not increased. The results of this and similar studies should allay parental concerns about risk of later development of substance abuse in a child treated with stimulants for ADHD.

"Around the clock" or "intermittent" stimulant therapy: Pros and Cons. In the above study, "a life-time history of stimulant medication" was a criterion for selection of drug-exposed subjects. A continuous or intermittent dose regimen (with drug holidays) was not itemized. Opinions vary on the pros and cons of each method of treatment. Baron, David A, Temple University School of Medicine, at a recent Annual Chairs in Psychiatry Summit, favors "need to treat around the clock" "because the symptoms of the 'disease' are continuous." (**NeuroPsychiatry Review** Oct 2008;9(10): courtesy of Millichap, Martin G, Dept Health and Human Services, Waukesha, WI). Many would characterize ADHD as a 'syndrome' or 'symptom complex,' not a disease, and justification is tenuous for "around the clock" drug treatment, with its attendant adverse effects. Further studies are needed, comparing long-term effectiveness and toxicity of 'continuous' and 'intermittent' therapy of ADHD.

CARDIOVASCULAR RISK SCREENING BEFORE STARTING STIMULANTS FOR ADHD IN CANADIAN PRACTICE

Health Canada released a statement advising against stimulants in ADHD patients with cardiac disease in May 2006, after isolated reports of sudden death The impact of this advisory on 1) physicians' cardiovascular assessment of all children with ADHD before starting stimulant medications, and 2) on the treatment of children with potential or real cardiac disease was assessed by questionnaires mailed to noncardiologists and pediatric cardiologists in Canada from the Department of Pediatrics, IWK Health Centre, Dalhousie University, Halifax, Nova Scotia. Of a total of 2326 questionnaires distributed, 717 (31%) were returned. The proportion performing a full screen increased for both noncardiologists

(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