restaurant MSG headache). Red wine contains more phenolic flavonoids than white wine, which might explain the reported red wine sensitivity of some migraineurs; sensitivity may be related to a low level of phenosulphotransferase P, an enzyme that detoxicates the flavonoid phenols. Alcohol-induced headache has been related to an increase in body water. Ice cream headache is an accepted phenomenon, but the mechanism is controversial. Fasting is a generally accepted migraine trigger, but hypoglycemia may not be the mechanism. Elimination diets have had mixed results in controlling migraine. The author concludes that apart from a sensitivity to red wine and other alcoholic drinks, proposed dietary triggers require additional factors to induce migraine attacks. (Rose FC. Food and Headache. Headache Q 1997;8:319-329). (Reprints: F Clifford Rose MD, London Neurological Centre, 110 Harley Street, London W1N 1AF, UK).

COMMENT. Diet and headache is a controversial topic that needs further scientific study. Anecdotal evidence and limited controlled experiments are very persuasive for items such as chocolate, milk, cheese, coffee, aspartame, nitrites, MSG, and red wine. However, results of elimination diets and challenge experiments are sometimes conflicting in groups of patients, and individual differences in sensitivity or combinations of factors may explain the controversies. After working with Dr John Wilson in his clinic at Great Ormond Street Hospital, London, I am convinced of a relation between dietary factors and headache in children. (For reviews of articles on Diet and Migraine, see Millichap JG, ed. Progress in Pediatric Neurology Vol I, 1991;pp146-150; Vol II, 1994;pp166-168; Vol III, 1997;pp170-171). My experience in the United States, however, is hampered by a reluctance of parents to embark on a time-consuming, hypoallergenic dietary therapy, often requiring the added expense of nutritional counselling and supervision. At best, parents and patients can sometimes be persuaded to try the avoidance of caffeine and aspartame-containing diet sodas, often consumed in extraordinary quantities by young children, and the restriction of chocolate may also be attempted. A suggestion that milk and other dairy products may be the offending headache triggers is frequently received with a look of disbelief. Patients have been educated in reliance on the "pill."

ATENTION DEFICIT AND COMORBID DISORDERS

METHYLPHENIDATE-INDUCED OBSESSIVE-COMPULSIVENESS

An 8-year-old boy with a transient but severely debilitating obsessive-compulsive disorder (OCD), induced by a brief course of methylphenidate (MPH) (10 mg/daily) for treatment of mild, uncomplicated attention deficit hyperactivity disorder, is reported from the University of Illinois College of Medicine, Rockford, Illinois. After 2 weeks of MPH treatment, school work improved, but OC behaviors emerged: avoidance of touching objects, repeated hand washing. He also developed tics involving head and neck. The past history was negative for psychiatric comorbidity, recent streptococcal infection, and familial anxiety disorders. Withdrawal of MPH was followed by gradual recovery from the OCD over a 3 month period. (Kouris S. Methylphenidate-induced obsessive-compulsiveness. J Am Acad Child Adolesc Psychiatry Feb 1998;37:135). (Respond: Dr Steven Kouris, University of Illinois College of Medicine, Rockford, IL).

COMMENT. Obsessive compulsive disorder (OCD) is a rarely reported complication of psychostimulant therapy for ADHD. The author cites only two previous references. Dextroamphetamine has been implicated more often than methylphenidate (MPH). Tic disorder is a comorbid complication and a more

frequent side effect of MPH than OCD. The above report should prompt a greater awareness of the potential risk of OCD in addition to tics in ADHD children treated with MPH. As with MPH-induced seizures, the incidence of this side effect may be higher than the literature documents.

Psychiatric, neuropsychological, and psychosocial features of DSM-IV subtypes of ADHD were assessed in 413 children and adolescents referred to the Pediatric Psychopharmacology Unit, Massachusetts General Hospital, Boston, MA. Combined-type subjects showed the greatest psychiatric impairments compared to other subtypes, but no differences in cognitive or psychosocial functioning. Inattentive subjects were more likely to require extra help in school. Hyperactive-impulsive patients were not different from controls on measures of depression, social functioning, IQ, and academic achievement. (Faraone SV, Biederman J, Weber W, Russell RL. I Am Acad Child Adolesc Psychiatry Feb 1998;37:185-193).

MUSCLE DISORDERS

CONGENITAL MYASTHENIC SYNDROMES: END-PLATE AChR LACK

Two families with 5 affected members suffering from congenital myasthenic syndrome are reported from University Hospital, Bonn, Germany. The age at onset of ptosis, extraocular weakness, and exercise intolerance was in childhood or adolescence. The course was slowly progressive. The distribution of muscle weakness was more proximal than distal. All patients had abnormal decremental response on low-frequency 3-Hz nerve stimulation, and no repetitive responses to single nerve shock. All improved with anti-acetylcholinesterase drugs. Intercostal muscle biopsies from one patient in each family showed type 1 fiber predominance, and marked reduction in number of secondary synaptic clefts per neuromuscular junction and in the expansion of the postsynaptic area. Acetylcholine-receptor (AChR) density was reduced, and AChR-associated protein utrophin was deficient in the end-plate, on immunohistochemical analysis. Both patients showed a defect in the development or maintenance of the postsynaptic clefts. (Sieb JP, Dorfler P, Tzartos S et al. Congenital myasthenic syndromes in two kinships with end-plate acetylcholine receptor and utrophin deficiency. Neurology Jan 1998;50:54-61). (Reprints: Dr JP Sieb, Department of Neurology, University Hospital, Sigmund-Freud-St 25, D-53105 Bonn, Germany).

COMMENT. Since the clinical description of a *congenital* myasthenic syndrome (Millichap, JG, Dodge PR. Neurology 1960;10:1007), as distinguished from the *neonatal transient* form, several congenital myasthenic syndromes have been identified. Engel AG et al at the Mayo Clinic have reported patients and families with endplate acetylcholine and AChR deficencies, a slow-channel syndrome, defects in resynthesis of ACh and kinetics of AChR, and abnormal interaction of acethylcholine with its receptor. (see Progress in Pediatric Neurology III, 1997;pp346-347, for reviews of Mayo Clinic reports).

The Bonn families with congenital myasthenic syndrome had defects in the postsynaptic clefts with deficiencies in end-plate acetylcholine receptor and utrophin (dystrophin-related protein). The authors note that their patients resemble reports of a so-called congenital paucity of secondary synaptic clefts syndrome (CPSC) (Smit et al. 1988; Wokke et al. 1989). These familial syndromes have different clinical features and absent anti-AChR antibodies that distinguish them from autoimmune myasthenia gravis.