

Reye syndrome with aspirin use in the US is coincidental. (Orlowski JP et al. A catch in the Reye. Pediatrics 1987;80:638-642).

**COMMENT.** Rupert Murdoch, the Australian journalist and publisher, would approve of this apt title, a refreshing innovation for our generally plain medical style of writing. Despite the conclusions drawn here and supported by a study from Japan, which also failed to show an association between aspirin and Reye syndrome, it is unlikely that the pediatric usage of aspirin will be resumed in the US. The massive public education campaign launched by the Government in 1982 to caution parents against aspirin use for colds, influenza or chicken pox has been very successful, notwithstanding the persisting mystery regarding the true cause or causes of Reye syndrome.

### NUTRITION, DIET AND THE NERVOUS SYSTEM

#### ADVERSE REACTIONS TO FOOD ADDITIVES

As part of a multicentre study of food additive intolerance commissioned by the UK Ministry of Agriculture, Fisheries and Food, the prevalence of reactions to food additives was studied in a survey population by the Depts of Dermatology and Community medicine, Wycombe General Hospital, High Wycombe, Bucks, and St. Thomas' Campus, London University. Of 18,582 respondents to questionnaires, 7.4% had reactions to food additives, 15.6% had problems with foods, and 10% had symptoms related to aspirin. The incidence of a personal history of atopy reported in 28% of all respondents was significantly higher in those reacting to additives, food, and aspirin (50%, 47.5%, and 36% respectively). A preponderance of reactions occurred in children, boys more than girls. Older patients were affected less often and with a female preponderance.

Abnormal behavior and mood changes were mainly related to additives whereas headache was associated with foods more frequently than additives. Of 44 individuals (7% of 649 interviewed) who reported monosodium glutamate sensitivity, 13 (30%) suffered headache, and 8 (18%) had behavioral or mood changes. Headache was related to food intolerance in 14% of those interviewed but had not previously been regarded as migrainous in nature. Of 81 reactive subjects who completed an additive challenge with annatto or azo dye, only 3 showed consistent reactions. The authors estimated the prevalence of food additive intolerance in the study population at 0.01-0.23%. (Young E et al. J Roy Coll Physicians London 1987;21:241-247).

**COMMENT.** The debate in the UK on food additives and behavior waxes while in the USA interest wanes, with more attention being given to sugar and the effectiveness of stimulants in therapy (see Ped Neur Briefs 1987;1:5,22,38). In the same issue of the JRCP London, Pollock I and Warner JO at the Brompton Hospital report a follow-up of children with food additive intolerance showing that symptoms were mainly transient, 76% showing no reaction on rechallenge studies, and Lessof MH at Guy's Hospital reviews the literature and concludes that more reliable diagnostic tests and toxicological screening methods are needed. A food intolerance databank has been compiled at the Leatherhead Food Research Association, UK, that will provide constantly updated information on food product composition and brands free from

ingredients most commonly associated with food intolerance (milk, egg, wheat, soya bean, cocoa, BHA and BHT, sulfur dioxide, benzoate, glutamate and azo colours).

#### SUGAR, ASPARTAME AND BEHAVIOR

The effects of glucose, sucrose, saccharin, and aspartame on aggression and motor activity in 30 boys, ages 2-6 years, were studied at the Child Psychiatry Branch and Laboratory of Developmental Psychology, NIMH, Bethesda, MD. Eighteen boys were recruited or selected as "sugar responders" and 12 male playmates were "non responders". Single doses of sucrose, 1.75g/kg; glucose, 1.75g/kg; aspartame, 30mg/kg; or saccharine administered in a randomized, double-blind design produced no significant effect on aggression or on teacher ratings of behavior. Actometer counts for 2 hours after ingestion of aspartame were lower than those following other sweeteners. Parent ratings of activity and aggression after home challenges with sweeteners failed to show differences between substances for either the alleged "responders" or "non responders". Consistent with baseline measures, parents rated responders more hyperactive than playmates who were not believed to be sugar reactive. No parent differentiated between sugar and nonsugar trials. Mean daily sucrose intake and total sugar consumption correlated with duration of aggression against property for the alleged sugar responsive group but acute sugar loading did not increase aggression or activity in preschool children. (Krnesi MJP et al. Effects of sugar and aspartame on aggression and activity in children. Am J Psychiatry 1987;144:1487-1490).

COMMENT. Connors CK at the Children's Hospital, Washington, D.C. reports that deleterious effects of sugar on children with attention deficit may be demonstrated if the challenge follows a high carbohydrate breakfast but the effects are blocked or reversed by a protein load. The beneficial and protective effects of a protein diet are correlated with neuroendocrine changes and the prevention of the serotonergic effects of sugar on behavior and attention (personal communication and in Diet and Behavior, Lubbock, Texas Tech Univ Press). Diets low in protein and high in carbohydrates might be expected to cause increases in spontaneous activity, as demonstrated in animal studies, but these effects are not necessarily related to swings in blood sugar concentrations. For recent reviews of the effects of dietary nutrients and deficiencies on brain biochemistry and behavior see Yehuda S. Intern J Neuroscience 1987;35:21-36; and Nutrition Reviews/Supplement May 1986;44:1-250.

#### PAROXYSMAL DISORDERS

##### HEMISPHERECTOMY FOR CHILDHOOD EPILEPSY

Seventeen patients treated for hemiplegic epilepsy by hemispherectomy between 1950 and the present day have been followed up at the National Centre for Children with Epilepsy, The Park Hospital for Children, Oxford. The causes of the seizures were associated with perinatal complications in 8 and early febrile status epilepticus, prolonged and unilateral, in 9. Two of the 17 had congenital abnormalities: Sturge-Weber disease in one and a heterotopia found at operation in the other. Habitual seizures began at age 1 to 10 yrs after an interval of relative freedom varying