

dendritic spines persist after birth suggesting a dysgenetic process, and neuronal membranes in Down syndrome are abnormally hyperexcitable. The mechanism of increased susceptibility to seizures in children with Down syndrome may involve an interplay between hyperexcitable membranes, altered dendritic spines, and reduction of inhibitory interneurons. The present study suggests that seizures in patients with Down syndrome are frequently caused by additional factors including hypoxia and infection.

The prevalence, onset, and type of seizure disorders and seizure control were studied in 405 individuals with Down syndrome aged six months to 45 years at the Departments of Pediatrics and Neurology, Rhode Island Hospital, Providence, RI. The prevalence of seizures in this group of patients was 8.1%. The onset of seizures occurred before one year of age in 40% and in the third decade of life in another 40%. Infantile spasms and tonic-clonic seizures with myoclonus were most common in the younger age group and partial simple or partial complex seizures and tonic-clonic seizures occurred in the older patients. A bimodal distribution of seizures in patients with Down syndrome was noted. (Pueschel SM et al. Seizure disorders in Down syndrome. Arch Neurol March 1991; 48:318-320).

HEADACHE DISORDERS

MANAGEMENT OF HEADACHE: A DEVELOPMENTAL PERSPECTIVE

Developmental issues related to the assessment and treatment of childhood headache are discussed from the Department of Psychology, University of South Alabama, Mobile, Alabama. The three major areas of development are cognitive, self-regulation, and psychosocial development. Young children use "what", "where", and "who" before they can use "how", "why", and "when". "What is a headache?" is more likely to produce an intelligible response than "How do people get headaches?" Knowing the child's explanation of headache will help provide better explanations of medical and psychological procedures for children. Children's reports of the quality of headache pain are variable and may be exaggerated in the 9 to 11 age group and minimized in the 6 to 8 year olds. The headache record is the predominant outcome measure for determining treatment success, and the diary format should divide the day according to relevant and easily remembered events of meals and bedtime. An open ended format for measuring intensity such as a vertical thermometer scale is preferred to a fixed response scale of 0-5 that assumes equal increments in intensity. Headache duration might be compared to the length of a favorite television show, recess, or other activity rather than actual time estimates which may be problematic for children. "Before" and "after" are confusing for preschoolers although they understand the concepts of "first" and "last".

A shift from an external health locus of control to the acknowledgment of one's own role in headache control occurs with increasing

age. Treatments that incorporate self-regulation such as autogenic thermal feedback training require an understanding of an internal control mechanism. From a medical perspective the foundation for self-management and self-regulation of behavior develops as the child applies autonomy to managing the external world. Awareness of autonomy, initiative and industry issues may facilitate successful treatment of childhood headache. A shift in family attention to headache free days could be a strong treatment reinforcer for children, focusing less attention on the headaches. (Marcon RA, Labbe EE. Assessment and treatment of children's headaches from a developmental perspective. Headache Sept 1990; 30:586-592).

COMMENT. Adding a developmental perspective to psychological interventions in the management of childhood headache is likely to increase treatment effectiveness. The management of headache in children is different from that of adults and requires an assessment of cognitive and affective development and a knowledge of children's concepts of illness and pain. Factors in precipitating headaches such as diet and exercise may be more important in children than in adults and will require special attention. The use of long-term investigational medications in children is undesirable and relaxation techniques and dietary modification may be more effective and appropriate.

PATHOPHYSIOLOGY OF VASCULAR HEAD PAIN

The mechanism of head pain, provocative stimuli, and receptors involved in drug treatment are reviewed from the Stroke Research Laboratory, Neurosurgery and Neurology, Massachusetts General Hospital, Boston, MA. A primitive defensive fiber network surrounds the brain and its blood vessels which is derived principally from trigeminal ganglia neurons. Headaches develop from activity within the trigeminal vascular system and are controlled by serotonin agonists that couple with 5-HT receptors on unmyelinated C fibers to block activity in the trigeminovascular system. Neuropeptide release and neurogenic inflammation within the coverings of the brain are inhibited. Altered physiologic states (stress, fatigue), bright lights and sleep deprivation may precipitate migraine and act by modulating neuronal activity and synaptic events within cortical gray matter. Local increases in cortical blood flow modulated by trigeminovascular fibers facilitate removal of potentially noxious chemicals. (Moskowitz MA. The visceral organ brain: Implications for the pathophysiology of vascular head pain. Neurology Feb 1991; 41:182-186).

COMMENT. That vasospasm contributes to monosodium glutamate-induced headache has been demonstrated by Merritt JE and Williams PB in the Department of Pharmacology, Eastern Virginia Medical School, Norfolk, VA. (Headache Sept 1990; 30:575-580). The direct effects of glutamate and its metabolite glutamine on arterial contractility were examined using rings of rabbit aorta. High concentrations of glutamate caused significant concentration dependent contractions. At high concentrations