

CHLORAL HYDRATE: A POTENTIAL CARCINOGEN

The long-term health effects of chloral hydrate are discussed and its carcinogenicity in mice are reported from the University of California, Davis, and the California School of Public Health, Berkeley. In a group of 8 mice receiving 10 mg/kg, 6 developed hepatic adenomas or carcinomas, an incidence significantly greater than that of 2/19 controls with carcinomas. Of 24 mice receiving 166 mg/kg/daily intake of chloral hydrate in drinking water for 104 weeks, 17 developed hepatic adenomas or carcinomas compared to 3 of 20 controls. Other recent studies have shown chloral hydrate to be genotoxic, causing chromosome changes in vivo and in vitro. (Salmon AG, Kizer KW et al. Potential carcinogenicity of chloral hydrate - a review. Clin Toxicol 1995;33:115-121). (Respond: Dr Kenneth Kizer, Department of Community and International Health, School of Medicine, TB-168, University of California, Davis, CA 95616).

COMMENT. Many of us who have stocked chloral hydrate in large quantities for use in sedating children for EEGs and other short procedures may want to reconsider its safety and substitute an alternative. Certainly, its chronic long-term use as a sedative in mentally retarded children should be discouraged until further studies are completed. Chloral hydrate is a metabolite of trichloroethylene, a known carcinogen.

HEREDO-METABOLIC DISORDERS

PRESENTING SIGNS OF MITOCHONDRIAL DISEASE

Clinical features of 51 patients confirmed with mitochondrial respiratory chain disease and clinical investigations most helpful in diagnosis of different phenotypes are reported from the Divisions of Clinical Neuroscience and Neurobiology, University of Newcastle upon Tyne, UK. Ages ranged from birth to 55 years, and 21 patients were <16 years. Presenting symptoms in order of frequency included ptosis and ophthalmoplegia (20), lactic acidosis (10), seizures (6), myopathy (6), failure to thrive (6), and ataxia (5). Features other than ptosis and ophthalmoplegia identified as clues to respiratory chain dysfunction were as follows: 1) lactic acidosis with deafness, short stature/failure to thrive, or basal ganglia calcifications on CT; 2) family history of neurological disease with maternal transmission; and 3) proximal myopathy and CNS disease. In addition to well-recognized syndromes (MERRF and MELAS) many had non-specific encephalopathies. The most useful confirmatory diagnostic test was histochemical analysis of muscle. (Jackson MJ, Bindoff LA et al. Presentation and clinical investigation of mitochondrial respiratory chain disease. A study of 51 patients. Brain April 1995;118:339-357). (Respond: Dr LA Bindoff, Division of Clinical Neuroscience, The Medical School, University of Newcastle upon Tyne, Framlington Place, Newcastle upon Tyne NE2 4HH, UK).

COMMENT. Mitochondrial respiratory chain disease is manifested by a large variety of syndromes, but histological and chemical analysis of skeletal muscle is frequently diagnostic. Succinate dehydrogenase, cytochrome c oxidase activity, and DNA studies in muscle may be performed on a needle biopsy specimen. Elevated CSF lactate is a good