weeks; 3.0 mg/kg daily maintenance dose; n=47) compared to phenytoin (2.5 mg/kg daily increasing to 5.0 mg/kg daily; n=47) showed no difference in efficacy over a 12 month period, and no excess of behavioral side-effects for phenobarbital based on Conners parent rating scales. (Pal DK, Das T, Chaudhury G, Johnson AL, Neville BGR. Randomised controlled trial to assess acceptability of phenobarbital for childhood epilepsy in rural India. Lancet Jan 3, 1998;351:19-23). (Respond: Dr Deb K Pal, Neurosciences Unit, Institute of Child Health, University College London, Wolfson Centre, London WC1N 2AP, UK).

COMMENT. Phenobarbital appears to be an acceptable first-line treatment for childhood epilepsy in a rural community in India. Compared to phenytoin, phenobarbital is equal in efficacy and has no excess of behavioral side effects. The WHO recommendation of phenobarbital for the treatment of partial and generalized tonic-clonic epilepsies in developing countries is justified. Phenytoin is slightly more expensive whereas carbamazepine and sodium valproate cost many times more than phenobarbital and generally require much closer monitoring. The reported behavioral side effects of phenobarbital may be over-estimated in comparison with alternative antiepileptic drugs.

ATTENTION DEFICIT DISORDERS

ACUTE HEPATIC FAILURE WITH PEMOLINE (CYLERT)

A 7-year-old boy with Duchenne muscular dystrophy and ADHD who developed acute hepatic failure with autoimmune hepatitis during treatment with pemoline (56 mg/day) is reported from the Children's Hospital, Cincinnati, OH. He presented with fever, vomiting, and jaundice, with thrombocytopenia and petechiae, after a period of decreased appetite. Ten-fold elevations of serum alanine aminotransferase (399 U/L) and aspartate aminotransferase (367 U/L) had been noted prior to pemoline treatment and 8 months prior to admission with hepatic failure. These may be ascribed to the muscle disease but could be consistent with pre-existing liver disease. On admission, these enzymes were markedly elevated, 1816 and 1313 U/L, respectively, and the total and direct bilirubin levels were 104 and 56 mcmol/L, respectively. Tests for hepatitis A and B were negative, but an autoimmune antibody panel, liver biosy findings, and coexistent diabetes were consistent with autoimmune hepatitis. After treatment with methylprednisolone, signs of encephalopathy gradually resolved, and at 2 year follow-up enzyme levels were moderately elevated (197 and 133, respectively) but one half the initial levels before pemoline administration. (Hochman JA, Woodard SA, Cohen MB. Exacerbation of autoimmune hepatitis: another hepatotoxic effect of pemoline therapy. Pediatrics Jan 1998;101:106-108). (Respond: Mitchell B Cohen MD, Children's Hospital Medical Center, OSB Building, 4th Floor, 3333 Burnet Ave, Cincinnati, OH 45229).

COMMENT. Since reports of acute liver failure associated with pemoline, the drug is no longer recommended as first-line therapy for ADHD. Pemoline has been linked to hepatic failure in 4 reported and a number of unreported cases. The liver toxicity is dose-dependent or due to an immune mediated hypersensitivity. The above case suggests an autoimmune process due to the drug, since pemoline withdrawal and treatment with prednisone resulted in a prompt response that was sustained after steroids were tapered. An analysis of pemoline associated hepatic failure (Shevell M, Schreiber R, Jan 1997) suggested that only one of the 4 reported cases appeared justified. Abbott Laboratories subsequently issued a warning and notice of further cases. (Ped Neur Briefs March 1997).