measures are considered: a) alert healthcare workers to the risks of bilirubin encephalopathy; b) instruct mothers more fully before discharge; c) more liberal use of infant formula supplements; d) lower plasma bilirubin limits for phototherapy and exchange transfusion; e) screen all term and near-term infants; f) use skin jaundice detection device that corrects for melanin content. (Ebbesen F. Recurrence of kernicterus in term and near-term infants in Denmark. Acta Paediatr Oct 2000;89:1213-1217). (Respond: Dr Finn Ebbesen, Department of Paediatrics, University Hospital of Aalborg, DK-9000 Aalborg, Denmark).

COMMENT. A recent increase in the cases of kernicterus in term or near-term infants in Denmark has raised concerns regarding the primary and secondary healthcare recognition and management of the problem. A larger prospective study of infants with elevated bilirubin levels should be performed prior to considering a population-based screening program.

Prediction and prevention of hyperbilirubinemia is currently a concern in the USA. (Newman TB et al. Arch Pediatr Adolesc Med Nov 2000;154:1140-1147). The predictors of extreme neonatal hyperbilirubinemia (> or = to 428 mcmol/L) determined in 11 Northern California Kaiser Permanente hospitals and a cohort of >51,000 term newborns included the following: 1) family history of jaundice in a newborn; 2) exclusive breastfeeding; 3) cephalhematoma. No case of kernicterus was diagnosed.

Diagnosis of kernicterus. The characteristic neurological findings of kernicterus (athetosis, impaired vertical gaze, and auditory loss or imperception) may not evolve until 4 years of age. In the neonatal period, the diagnosis is suspected when an infant with hyperbilirubinemia becomes drowsy, hypertonic and opisthotonic, the Moro reflex is absent, and the cry abnormal. Clonic convulsions occur in about 10% of cases. Early classic references to kernicterus as a form of cerebral palsy are by Byers RK, Paine RS, and Crothers B (Pediatrics 1955;15:248) and Perlstein MA (Charles C Thomas, 1961).

SEIZURE DISORDERS

BRAIN VOLUME REDUCTION WITH INTRACTABLE EPILEPSY

Cerebral, cerebellar, and hippocampal volumes were measured by quantitative magnetic resonance imaging on 112 children, ages 4 - 18 years, with epilepsy syndromes, determined by video-EEG telemetry, at Sydney Children's Hospital, Randwick; St Vincent's Hospital, Victoria; and New Children's Hospital, Westmead, New South Wales, Australia. A significant reduction in cerebral (13%) and cerebellar (8%) volume was present in the epilepsy group compared with 44 controls. This included partial epilepsies such as frontal lobe epilepsy. Hippocampal asymmetry was more sensitive than volume reduction as a marker for mesial temporal lobe epilepsy. Volume reduction was independent of age of onset and duration of epilepsy, suggesting that brain volume reduction is present at the onset of epilepsy and is not the result of intractable seizures. IQ was significantly correlated with cerebral and cerebellar volume, but not with duration or age of onset of epilepsy. (Lawson JA, Vogrin S, Bleasel AF, Cook MJ, Bye AME. Cerebral and cerebellar volume reduction in children with intractable epilepsy. Epilepsia November 2000;41:1456-1462). (Reprints: Dr Ann ME Bye, Department of Paediatric Neurology, Sydney Children's Hospital, Randwick, 2031 New South Wales, Australia).

COMMENT. While MRI data may not be reliable in localizing an epileptogenic area, measurements of brain volume reduction in partial epilepsies may prove of value in prognosis, especially in potential surgical cases. The correlation of IQ with brain volume in the above MRI study of children with epilepsy corroborates similar findings in normal children and adolescents at Johns Hopkins University (Reiss AL et al. Brain 1996;119:1763; and Progress in Pediatric Neurology III, 1997;p294).

Mitochondrial complex I deficiency is reported in the hippocampal epileptic focus of patients with temporal lobe epilepsy. (Kunz WS et al. Ann Neurol November 2000;48:766-773). Mitochondrial dysfunction is a possible mechanism involved in the neuronal excitability in temporal lobe epilepsy.

USE AND ABUSE OF THE ELECTROENCEPHALOGRAM

The utility of the electroencephalogram (EEG) to evaluate clinical situations in epilepsy is reviewed from the Department of Clinical Neurophysiology, King's College Hospital, London, UK. Based on an analysis of articles available in Medline, Cochrane, and the Internet, and personal experience, the utility of the EEG is not satisfactorily addressed. A referral for an EEG should contain a clear statement of the clinical problem and the reasons for obtaining an EEG. Lack of communication between the clinician and the neurophysiologist may result in abuse of the EEG and minimize its usefulness. The diagnosis of epilepsy is made essentially on clinical evidence, and a referral for a routine EEG for this purpose is usually an abuse. In patients with epilepsy, the first EEG will confirm the diagnosis in 80%. Epileptiform activity in the EEG considerably enhances the likelihood of epilepsy. It may also answer a frequent question "Is it epilepsy?" Video EEG telemetry or ambulatory EEG may be required to distinguish epilepsy vs nonepileptic attack disorder, or absence vs daydreaming. The EEG may be used to distinguish a seizure type or syndrome and its associated clinical prognosis; it can rarely determine etiology. It may provide the first localizing evidence for a lesion, and its utility in presurgical evaluation of epilepsy has not been entirely displaced by imaging techniques. In children, the EEG can help to determine when it is safe or appropriate to discontinue antiepileptic drugs. Interpretation of the EEG requires detailed information regarding the clinical situation, and close liaison between the referring clinician and the EEG department. (Fowle AJ, Binnie CD. Uses and abuses of the EEG in epilepsy. Epilepsia 2000;41(Suppl 3):S10-S18). (Reprints: Dr Adrian J Fowle, Department of Clinical Neurophysiology, King's College Hospital, Denmark Hill, London SE5 9RS, UK).

COMMENT. The authors conclude that the EEG has many uses in the evaluation of epilepsy, but attention to detail is essential in the referral request. Our own Epilepsy Center at Children's Memorial Hospital, Chicago, directed by Drs Nordli, Stack, and Kelly, requires a detailed referral form to be completed, when requesting an EEG. If this practice was more generally adopted, the utility of the EEG in the management of epilepsy would be increased.

EEG in evaluation of the first nonfebrile seizure is addressed in a recent report of the quality standards subcommittee of the American Academy of Neurology, Child Neurology Society, and American Epilepsy Society (Hirtz D, Ashwal S, Berg A et al. Neurology Sept (1 of 2) 2000;55:616-623). (Reprints: QSS, American Academy of Neurology, 1080 Montreal Ave, St Paul, MN 55116). Based on a review of available evidence in the literature, routine EEG was recommended as part of the diagnostic evaluation of the first nonfebrile seizure in children. The