

1.06

**Epilepsy and Pregnancy: Maternal-Newborn Pathology.** R. Meischenguiser (Fleni, Buenos Aires, Argentina).

We report our experience and the clinical history of women with epilepsy and their ongoing or planned pregnancy. Despite reiterated counseling on prevention of conception, ~50% of such women consult us once their pregnancy has already been confirmed; ~30% are aged  $\leq 18$  years. The different complications posed by pregnancy may include: (a) variation in the number of seizures; (b) bleeding, miscarriages, and preterm babies in greater proportion than that in the general population; and (c) possible prolonged delivery.

The newborn may be affected by (a) anomalies or malformations, not always detected by ultrasound or by determination of alpha-feta protein (AFP) level, which may be high, with no connection to the abnormalities, which necessitates further complete study of the mother; (b) excitement, depression, or rejection of food, owing to deprivation of or treatment with antiepileptic drugs; and (c) hemorrhage, which cannot be related solely to low levels of vitamin K. These difficulties make it necessary to avoid problems through the joint efforts of neuropsychiatrists, neonatal supervisors, and obstetricians.

In 26 pregnancies from 1990 to 1996, we noted bleeding in 3 of 26; in 1 of 26 mothers, treated with valproate (VPA), the infant, born prematurely, died at 48 h. One of 26 mothers, treated with VPA and ethosuximide, miscarried (in the 10th week); the fetus had anomalies. One infant born to a mother treated with phenobarbital was of low birthweight and died at 48 h. Another infant died at age 25 days. The mother had received phenobarbital. AFP levels were normal in 12 of 26 pregnancies and high, without malformations in 2 of 14. Ultrasound showed 15 of 26 fetus to be normal.

1.07

**Anticonvulsants: Conception Anomalies.** R. Meischenguiser, N. Tineti, E. Guerschberg (Hospital Pirovano, Buenos Aires, Argentina).

Evaluation of newborns of epileptic mothers and updated international statistics indicate a greater percentage of anomalies (5–9%) in such infants than in the general population (1.5%). We wished to determine whether the high incidence of anomalies is due to high doses of AEDs, a direct effect of AEDs or their metabolites, a significant genetic role, or to a single cause or a combination of factors. Is any AED reliable? No. Anomalies can result with use of any AED and be major or minor as reflected in the need for medical or surgery care of newborns to ensure their good future quality of life.

The main causes of malformations in newborns are metabolic interference, alterations in intracellular pH, and folate deficiency. Most of these undesirable effects can be produced by use of any of the major AEDs as monotherapy and especially as polytherapy, which in certain combinations can produce anomalies with an incidence of anomalies as high as 60%.

AED treatment must be correctly managed, both quantitatively and qualitatively; such protective measures can help resolve the challenge that these patients represent.

In 26 pregnancies studied from 1990 to 1996, 1 of 26 ended in miscarriage (week 10) with malformed fetuses [AEDs were valproate (VPA) and ethosuximide]. For 1 in 26 with Term Birth Living Child (TBLC) (died at 48 hours), and in 1 of 26 low birthweight infants, (died at 25 days) phenobarbital was the AED received by the mother. In 1 of 26 preterm infants (32 weeks) who died at 48 hours, VPA was AED received by the mother. AED levels were low in 12 of 15 of the pregnancies assessed. Twenty of the mothers had received monotherapy and 6 had received polytherapy. Anomalies were minor in 2 of 26 pregnancies, resulting from carbamazepine (CBZ) in one case and from VPA in the other.

## Clinical Epileptology

1.08

**Primary Generalized Epilepsy and the Crucial Role of Arousal.** Ernst Niedermeyer (Department of Neurology, The Johns Hopkins University, Baltimore, MD, U.S.A.).

Evidence that shows arousing stimuli are capable of triggering seizures (myoclonus, also absences and generalized tonic-clonic seizures) in primary generalized epilepsy (PGE). Paroxysmal arousal effects are readily demonstrable in the EEG during non-REM sleep (spiky K complexes) and also during hyperventilation. Clinical data are even more convincing: Most seizures occur soon after the patient awakens, mostly after a night of insufficient sleep. The ensuing post-sleep drowsiness is punctuated by short arousals associated with bisynchronous spike bursts, myoclonus (also absences, mainly at age <12 years) and a tonic-clonic convulsion may ensue. In a small subgroup of patients with PGE, photosensitivity is the preponderant epileptogenic mechanism (either with or without associated paroxysmal arousal).

The importance of sufficient sleep cannot be overemphasized in patients with PGE. The problem is compounded by the fact that most such patients are adolescents and young adults who feel healthy and wish to enjoy life to the utmost. Even optimal antiepileptic drug treatment (with valproate as primary medication) may not control the seizures. The patients must be made aware of their special vulnerability to avoid relapses throughout adulthood.

1.09

**Refractory Medical Epilepsy: Treatment and Follow-up with Schmidt's Scale.** Marcelo B. Devilat and Jaime M. Carrizosa (Pediatric Service of Neurology and Psychiatry, Hospital Luis Calvo Mackenna, Chilean Group of Epilepsy, Santiago, Chile).

Epilepsy treated adequately but with persistent seizures is termed refractory epilepsy. Schmidt's scale differentiates refractory epilepsy from inadequately treated epilepsy. We report the clinical characteristics of patients with refractory epilepsy, as well as the seizure evolution according to Schmidt's pharmacologic scale. Between February and November 1996, we made a prospective follow-up of 15 patients with four or more seizures a month who were receiving antiepileptic drug (AED) treatment. The patients had the following clinical characteristics associated with refractory epilepsy: early age at onset and long duration of the epileptic illness, absence of idiopathic etiology, epileptic syndromes difficult to treat medically, mental handicap, and polytherapy. At first evaluation, Schmidt's scale identified 60% (9 patients) as undertreated; at the last evaluation, 20% (3 patients) remained in that category. In a mean of 6.6 months, with changes effected in AED regimen, seizure frequency was reduced in 77.9%. In epileptic referral centers, a pharmacologic evaluation scale is recommended to optimize AED treatment in patients considered refractory to therapy. Schmidt's scale differentiates inadequately treated patients and selects patient with refractory epilepsy as candidates for new AED trials or epileptic surgery.

1.10

**Neurocysticercosis as a Main Etiology in Late-Onset Epilepsy in a Selected Population in Mexico.** A. Rangel-Guerra Ricardo, Claudio Muñiz Landeros, and Hector R. Martinez (Neurology Service, University Hospital U.A.N.L. "Dr. Jose Eleuterio Gonzalez," Monterrey, Mexico).

Late-onset epilepsy is usually secondary to a structural lesion in the CNS. Since the advent of magnetic resonance imaging (MRI), this issue has not been analyzed in underdeveloped countries.

We performed a prospective study involving 216 patients, divided into groups of patients aged <20 years (102 patients) and >20 years (late onset, 114 patients). All patients with late-onset epilepsy were assessed by MRI. Complete physical, neurological, biochemical, and EEG studies were performed in all cases.

In the present series, seizures were divided into cryptogenic and symptomatic. Those of late-onset cases were divided into those in patients aged <50 years in which the most frequent etiologies were cryptogenic and/or secondary to neurocysticercosis, and those in patients aged >50 years, in which the etiologies were predominantly due to neurocysticercosis and ischemic vascular disease.