seizures. Polysyndactyly is the single most variable main sign, both in degree and in presence, even within the same family. A review of 18 published cases and the present family shows that the main manifestations are cranial facial anomalies and the abnormality of the corpus callosum. Most families have been reported from northeastern Switzerland and the Mediterranean basin. The present family supports a chromosomal recessive inheritance (Gelman-Kohan Z et al. Further delineation of the acrocallosal syndrome. Eur J Pediatr Sept 1991; 150:797-799).

COMMENT. The acrocallosal syndrome (Schinzel syndrome) and other syndromes which include callosal agenesis are reviewed in **Progress in Pediatric Neurology**, Ed. Millichap JG, Chicago and London, PNB Publishers 1991, 310-312. At least four distinct syndromes include callosal agenesis as a major component: Aicardi (with infantile spasms, hypsarrythmia, chorioretinal lacunae and coloboma), Schinzel syndrome (with macrocephaly and polysyndactyly), Anderman syndrome (with anterial horn cell disease); and Shapiro syndrome (with recurrent hypothermia).

SEIZURE DISORDERS

NEURONAL MIGRATION DISORDERS

Thirty patients with intractable partial epilepsy and a pathological or radiological diagnosis of neuronal migration disorders (NMD) were studied at the Montreal Neurological Institute and Hospital, Montreal, Quebec, Canada. Age at onset of epilepsy ranged from 4 months to 21 years (mean 5.7). Prenatal etiological factors in 8 patients included maternal x-ray exposure or trauma, diabetes, advanced maternal age, uterine abnormality. All patients had partial epilepsy: complex partial (67%), partial motor (73%), and secondary generalized seizures (73%). 8 patients (27%) had tonic or atonic drop attacks, and 6 of these had lesions involving the central region. Status epilepticus had occurred in 30%. A below average IQ was found in 1/2 of the patients. MRI was superior to CT, but did not distinguish between pachygyria, cortical dysplasia or tuberous sclerosis. White matter subcortical abnormalities were demonstrated more frequently than cortical abnormalities. In 22 of 26 surgically treated patients, the histological diagnosis was focal cortical dysplasia (12), and forme fruste of tuberous sclerosis (10). Those with a histological picture of tuberous sclerosis had none of the classical clinical diagnostic signs, but 80% had delayed psychomotor development and all 10 patients had multilobar unilateral or bilateral EEG spiking. These findings differed from patients with focal cortical dysplasia whose developmental milestones were normal and epileptogenic discharges were confined to one lobe or area of the brain (Palmini A, Andermann F et al. Focal neuronal migration disorders and intractable partial epilepsy: a study of 30 patients. Ann Neurol Dec 1991; 30:741-749).

COMMENT. The results of surgical treatment of the above 26 patients are reported in the same journal (Palmini A et al. Ann Neurol Dec 1991; 30:750-757). Good or excellent seizure control was achieved in 42% and moderate control in 25%. The extent of lesion removed was most strongly correlated with the surgical outcome; 77% receiving complete or 50% excision of the lesion had a good prognosis. When excision of epileptogenic tissue was based on scalp EEG and electrocorticography studies there was no correlation with surgical outcome. The lack of specificity of the MRI in the diagnosis of tuberous sclerosis in this study is also alluded to by Fryer AE, Geneticist at the Royal Liverpool Hospital, England in an editorial concerning tuberous sclerosis (J Roy Soc Med Dec 1991; 84:699-701). Although MRI is more sensitive than CT, CT is more specific and potentially less confusing in interpretation. A thorough clinical examination including ophthalmoscopy and ultraviolet light examination of the skin is essential to exclude the diagnosis in anyone suspect or at risk. Prenatal diagnosis is only possible by fetal echocardiography but this examination is unreliable before 26 weeks and has a high rate of false negatives.

MMR IMMUNIZATION AND FEBRILE SEIZURES

The risk of seizures following MMR or MR immunization is evaluated in a retrospective cohort study conducted among 18,364 Tennessee children from The Department of Preventive Medicine, Vanderbilt University School of Medicine, Nashville, TN. Of 100 children (0.5%) with a confirmed seizure between the first MMR or MR and age 36 months, 77 had febrile seizures, 15 afebrile seizures and 8 acute symptomatic seizures. The incidence of febrile seizures was 4.3, 5.1, and 2.8 per 1,000 person-years in the 3 age groups, 361-540, 541-720, and 721-1,080 days, respectively. 4 children had febrile seizures in the 7-14 days following MMR or MR immunization, compared with 72 in the interval 30 or more days following MMR or MR with a relative risk of 2.1. The authors concluded that the increase in febrile seizures in the 7-14 day interval following MMR was coincident with the occurrence of fever post-immunization (Griffin MR et al. Risk of seizures after measles-mumps-rubella immunization. Pediatrics Nov 1991; 88:881-885).

COMMENT. This review of seizure occurrence was confined to those patients seen at a hospital and would not include seizures treated at home or at outpatient facilities. The authors admit that the ascertainment of seizures was incomplete but would include more serious seizures or neurologic events.

Measles was the cause of fever in 1.2% of 7,000 cases of febrile seizures reported in the world literature between 1929 and 1964 (Millichap JG, Febrile convulsions 1968 Macmillan, New York).