

organic disease before the hysterical episodes. Psychological features included a model (54%), stressful event (46%), separation or loss of relative (46%), previous hysterical symptoms (33%), la belle indifference (19%). Treatment consisted of stopping unwarranted investigation, PT and OT, and psychologic counseling. At discharge, 61% were completely recovered or had appreciably improved. A core group of 13 (21%) did not respond. (Grattan-Smith P et al. Clinical features of conversion disorder. Arch Dis Child April 1988;63:408-414).

COMMENT: In all but 3 of the 36 children presenting with an abnormality of gait, pain and, less frequently, anesthesia were prominent features. These associated symptoms are helpful in the differentiation from a dystonic gait, frequently misdiagnosed as hysterical in nature. The infrequent occurrence of organic disease as a prelude to conversion symptoms in this study is unusual. Gait disturbances of a hysterical nature may be preceded by minor trauma and pseudoseizures are frequently accompanied by true seizures requiring treatment with anticonvulsant drugs.

SEIZURE DISORDERS

EPILEPSY WITH OCCIPITAL CALCIFICATIONS

Four patients, aged 13-22 yrs, with focal epilepsy, and bilateral occipital corticosubcortical calcifications without facial cutaneous angioma were followed at the Neurological Institute, University of Bologna Medical School, Via Ugo Foscolo, Bologna, Italy, and were found to develop a severe encephalopathy with progressive mental impairment. The age at onset of seizures was 3-8 years and psychomotor function was normal while seizures remained controlled from 1-2 years. Unexpectedly, the seizures recurred and were refractory to medication. Concomitantly, all patients had progressively severe mental impairment, and the EEG's showed progressive slowing of the background activity. During non-REM sleep, fast polyspike bursts, diffuse and with greater prominence in both occipital regions, were observed. CT's showed occipital calcifications and skull X-ray in one patient showed double-contoured curvilinear calcifications. The authors regarded a diagnosis of atypical Sturge-Weber syndrome as questionable. (Gobbi G et al. Epilepsy with bilateral occipital calcifications: A benign onset with progressive severity. Neurology June 1988;38:913-920).

COMMENT: A case of Sturge-Weber-Dimitri disease without facial nevus (Taly AB et al. Neurology 1987;37:1063), published after submission of this paper and noted by the authors as an addendum, was found to have bilateral leptomeningeal angioma. Bilateral calcification and bilateral ectodermal angioma in Sturge-Weber syndrome may not have been reported often but they occur in my experience. A progressive epileptic encephalopathy also may occur, particularly as a sequel to status epilepticus with Sturge-Weber disease. In the present cases the cause for the deterioration was unclear. This experience should prompt consideration of early neurosurgical excision in similar cases with unilateral calcified lesions, despite initial responsiveness to anticonvulsant medication.