growing brain and used only when tumor recurrence has been demonstrated. After radical excision, the rate of recurrence was lowest, with a 10 year recurrence-free survival rate of 88%. After subtotal removal, the recurrence-free survival rate, 10 yrs post-op, was 37%; this rate was significantly higher (72%) when subtotal removal was followed by irradiation, but deafness and severe neuropsychological and intellectual sequelae were frequent complications of irradiation. Post-operative mortality was low in pre-chiasmatic cases and high in retro-chiasmatic tumors. Surgical statistics may improve with newer techniques. (Pierre-Kahn A et al. Traitement des craniopharyngiomes de l'enfant. Analyse retrospective de 50 observations. Arch Fr Pediatr Mars 1988;45:163-167).

COMMENT: The neuropsychological deficits ascribed to irradiation in this report may be explained in part by the location of the tumor. Cognitive defects have been correlated with frontal lobe abnormalities seen on MRI in 4 patients with craniopharyngioma (Stelling MW et al. Am J Dis Child 1986;140:710).

INFECTIOUS DISEASE

TREATMENT OF AIDS ENCEPHALOPATHY

A 3-year old boy who had acquired HIV infection transplacentally and developed AIDS encephalopathy is reported from the Depts of Paediatrics and Immunology, Newcastle General Hospital, Newcastle upon Tyne, England. During hemophilus influenza pneumonia at 26 months his speech regressed to expressive aphasia and he developed spastic diplegia with inability to walk. CT scan showed cerebral atrophy. CSF showed no cells and normal glucose and protein; IgG antibodies to HIV were increased. Treatment with intravenous gammaglobulin 300 mg/kg and oral zidovudine (Retrovir-Wellcome) 100 mg/m^2 4x daily every 4 weeks for 8 months led to considerable clinical improvement and an almost normal CT. Spasticity regressed allowing him to run unaided and his speech in single words became articulate. (Matthews J et al. AIDS encephalopathy with response to treatment. Arch Dis Child May 1988;63:545-547).

COMMENT: AIDS encephalopathy may be acute and rapidly progressive (15%), subacute but progressive (18%), and static with cognitive deficits (28%). A plateau course is apparent in many. The reported case was subacute in onset and without treatment further progression might have been expected. (See Ped Neur Briefs 1988;2:1).

PSYCHOGENIC DISORDERS

HYSTERICAL GAIT

In a study of the clinical features of conversion disorder in 52 children admitted to the Royal Alexandra Hospital for Children, Camperdown, New South Wales, Australia, hysterical gait disturbance was the main complaint in 71%, and pain, paresthesia or anesthesia in 77%. So called classical conversion symptoms such as blindness and globus were relatively rare. The disorder was rare below 8 years of age and girls outnumbered boys three to one. Spring and summer (Sept-Nov and Jan-Mar in Australia) accounted for 75% of admissions, coinciding with the end of year exams and the beginning of the new school year. Only 6 children had

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 an 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. Concommitantly, 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.