five patients. The EEG showed excess slow wave activity, of varied degree, in every case (table).

An abnormal EEG occurs frequently in SC. Diffuse slowing of spontaneous rhythms is the commonest reported change, with an incidence of between 55 and 87 per cent.[3] Its occurrence in all five of the patients in this series may reflect the fact that each exhibited disturbed behaviour at the time of study. Behavioural disorders occur frequently in SC[2] and furnish an important diagnostic clue. They may be subtle and overlooked and distractibility or dysphoria attributed to chorea per se.

The absence of pathological change on CT brain scan has been a constant finding to date and is consistent with the poverty of lesions seen at autopsy.

We are not aware of a published account documenting a series of cases of SC, SEP recordings or analysis of CSF for oligoclonal immunoglobulin (Ig) G bands. It is generally accepted that value for CSF total protein and white cell count in SC are normal,[4] although the evidence for this is not well documented. Our findings substantiate this traditional doctrine. The absence of oligoclonal bands is somewhat at variance with the result of an apparent selective increase in CSF IgG reported previously.[5] In this case, however, the evidence for local CSF IgG synthesis was based on an elevated IgG/total protein ratio where spuriously high values may be found when, as happened in that patient, there exists an abnormally high serum IgG. The detection of oligoclonal bands is an accurate method for demonstrating intrathecal synthesis of IgG. The negative result in all of our patients tested argues against a primarily antibody mediated pathologic immune reaction within the central nervous system being responsible for SC.[6 7]

What role cortical SEP results may have in the differential diagnosis of chorea has yet to be determined. Our findings differ from those in a recent report of a single case of SC in which central sensory conduction time was prolonged.[8] However, this patient exhibited psychomotor retardation, tonic gaze deviation, facial weakness, hyperactive deep tendon reflexes and the plantar response was extensor, all of which are atypical signs for SC.[2] Normal results in SC stand in contrast to the situation in Huntington's disease, where the early cortical components are either reduced in size or absent.[9] This disparity may be explained by differences in the pathologic anatomy of the two diseases. Cortical SEPs also have been reported to reveal abnormalities in patients with Wilson's disease,[10] whereas results in benign hereditary chorea and chorea gravidarum (personal observations) have been normal.

The results of this study emphasise that EEG abnormalities and associated behavioural disturbances are frequent concomitants to chorea in patients with SC. While clinical features remain the cornerstone in diagnosing SC, our experience suggests that the stereotyped pattern of results may allow standard neurodiagnostic tests to be of some discriminatory value.

We thank Professor P Bartel for scientific advice, Mr Y Chetty, Dr RA Dupont and Mrs J Bester provided technical help.

RF GLEDHILL
Division of Neurology, Department of Internal Medicine, Univeristy of Pretoria and Kalafong Hospital, South Africa
PD THOMPSON
University Department of Clinical Neurology, Institute of Neurology, Queen Square, London

Correspondence to: Dr RF Gledhill, Neurosciences Department, Rashid Hospital, PO Box 4545, Dubai, United Arab Emirates.

1 Veasy LG, Wiedmeier SE, Orsmond GS, *et al.* Resurgence of acute rheumatic fever in the intermountain area of the United States. *N Engl J Med* 1987;**316**:421–7.
2 Thiebaut F. Sydenham's chorea. In: Vinken PJ, Bruyn GW, eds. *Handbook of Clinical Neurology*. Vol. 6. Amsterdam: North Holland. 1968:409–34.
3 Ch'ien LT, Economides AN, Lemmi H. Sydenham's chorea and seizures. Clinical and electroencephalographic studies. *Arch Neurol* 1978;**35**:382–5.
4 Fishman RA. *Cerebrospinal fluid in diseases of the nervous system*. Philadelphia: WB Saunders. 1980:321.
5 Gledhill RF. Selective increase in cerebospinal fluid immunoglobulin G in a patient with Sydenham's chorea. *J Neurol Neurosurg Psychiatry* 1986;**49**:602–3.
6 Waksman BH. Immunity and the nervous system: Basic tenets. *Ann Neurol* 1983; **13**:587–91.
7 Weiner HL, Hauser SL. Neuroimmunology II: Antigenic specificity of the nervous system. *Ann Neurol* 1982;**12**:499–509.
8 Ganji S, Duncan C, Frazier E. Sydenham's Chorea: clinical EEG, CT scan and evoked potential studies. *Clin Electroenceph* 1988; **19**:114–22.
9 Noth J, Engel L, Friedmann H-H, Lange HW. Evoked potentials in patients with Huntington's disease and their offspring. 1. Somatosensory evoked potentials. *Electroenceph Clin Neurophysiol* 1984;**59**:134–41.
10 Chu N-S. Sensory evoked potentials in Wilson's disease. *Brain* 1986;**109**:491–507.

Table Results of neurodiagnostic tests

Case	Age (y)	Duration of chorea (mo)	EEG	CSF: White cells (× 10⁶/L)	CSF: Total # protein (g/L)	CSF: Oligoclonal μ IgG bands
1	7	¼	Diffuse excess theta	1*	0·10	Neg
2	12	¼	Excess theta with posterior dominance	0	0·19	Neg
3	11	3	Excess theta with right dominance	3*	0·22	Neg
4	16	9	Diffuse excess theta	0	0·23	Neg
5	13	12	Diffuse delta and excess theta	0	0·13	Neg

95 per cent reference range (black adults) 0·10 to 0·50 g/L.
μ Two or more bands, not present in serum, identification by agar gel electrophoresis with silver stain.
* Lymphocytes.
Neg = Negative.

MATTERS ARISING

Outpatient referrals

I was astonished to read that among 7836 successive new outpatient referrals analysed by G D Perkin,[1] that "conversion hysteria" with 297 examples in ten years was number six in the top twenty of his diagnoses and constituted 3·8% of the total. Moreover, conversion hysteria was twice as common as either Parkinson's or post traumatic syndrome and almost three times as common as depression. In my experience, depression is a very common symptom and presents in many guises often with somatic symptoms. Conversion hysteria is, by contrast, a very rare disorder and I do not think that I make this diagnosis more than once or twice a year in the whole of my clinical practice. Does Dr Perkin have special criteria for the diagnosis of conversion hysteria or is his outpatient practice biased heavily by referrals from psychiatric colleagues?

I was also surprised that no diagnosis was made in 26·5% of patients—surely an unusually high percentage. I should have thought that 5% would be nearer the mark. I recognise that in an outpatient clinic one may make an inaccurate diagnosis but I do not think that one should make no diagnosis at all in more than a quarter of the patients.

E A NIEMAN,
St Mary's Hospital, London, United Kingdom

1 Perkin GD. An analysis of 7836 successive new outpatient referrals. *J Neurol Neurosurg Psychiatry* 1989;**52**:447–8.

Dr Perkin replies:

The criteria that I use for the diagnosis of conversion hysteria are the presence of physical signs which by their nature can only be explained on the basis of an elaborated disability on the part of the patient. I would disagree that the condition is rare. The cases that I discussed were seldom, if ever, referred by the psychiatrists. They appear to share Dr Niemann's view about the scarcity of the problem. I have analysed some of this data in greater detail elsewhere,[1] where, I found that approximately half the patients with features of conversion hysteria had the additional characteristics of Briquet's syndrome. That particular syndrome with or without evidence of conversion hysteria has been found remarkably frequently in those patients who are frequent attenders of medical outpatient clinics.

I find Dr Niemann's use of the word "should" in the last but one line of his comment, a curious one. It almost suggests a compulsive need on the part of the neurologist to make a diagnosis in his patients. Indeed that compulsive need, I suspect, often produces spurious diagnoses which may possibly help the doctor but do little to assist the patient. It maybe that all patients who develop giddiness on turning their head have vertebro-basilar insufficiency. I rather doubt it and because of this doubt, this would be one category of patients that I would leave undiagnosed in many circumstances. If, on