In contrast to earlier studies comparing patients with GE and TLE in whom the psychoses associated with GE were said to be briefer and have a better prognosis than those of TLE (Dongier, 1960), the patients with GE and psychosis reported by Perez *et al*, have psychoses that were even more chronic and severe than those of patients with the nuclear schizophrenic syndrome. Indeed, the authors note that most of their patients with nuclear schizophrenia do not become institutionalised and lived "reasonably satisfactory lives in the community". As the authors are aware, the Schneider first rank symptoms are by no means specific for schizophrenia and do not correlate highly with other diagnostic profiles for schizophrenia or with prognosis (Kendell *et al*, 1979). However, whether these relatively less handicapped persons would be generally considered more schizophrenic than those with the severe psychosis and cognitive defects is less the point than the fact that a subgroup of psychosis has been discerned that is characterised by a constellation of Schneiderian symptoms and with an anatomic reference in the temporal lobe predominantly lateralised to the left. Patients with more severe chronic interictal psychosis have evidence of more widespread neurophysiologic and neuropathologic (e.g., CT scan) changes underlying a syndrome characterised by more "negative" symptoms, earlier onset of seizures and greater cognitive impairment.

It is not difficult to accept that Schneiderian symptoms, more than half of which pertain directly to pathologic auditory perception or verbal material, are associated with pathology in the left temporal lobe (Stevens, 1973). Notably lacking in first rank symptoms, however, are the formal thought disorder, disturbances of affect and autism characteristic of Kraepelin, ICD9 or DSM III criteria for schizophrenia. Although not as clearly spelled out in their report, it appears that chronic interictal psychosis, albeit not of the Schneiderian (NS) variety, is according to their study equally common percentage-wise in adults with GE as in TLE. This study is thus a definite contribution toward delineating the underlying anatomy of Schneider's first rank symptoms rather than schizophrenia. It is of interest that clonazepam, a benzodiazepine with potent anticonvulsant activity is reportedly useful in certain patients with Schneiderian symptoms, but who were classified as schizoaffective or manic (Chouinard *et al*, 1983; Greenspan & Levin, 1985).

JANICE R. STEVENS

NIMH,
St. Elizabeths Hospital,
Washington DC 20032, USA

References

BEAR, D. M. & FEDIO, P. (1977) Quantitative analysis of interictal behavior in temporal lobe epilepsy. *Archives of Neurology*, **34**, 454–467.

CHOUINARD, G., YOUNG, S. N. & ANNABLE, L. (1983) Antimanic effect of clonazepam. *Biological Psychiatry*, **18**, 451–466.

CROW, T.(1980) Molecular pathology of schizophrenia: more than one disease process. *British Medical Journal*, **280**, 66–68.

DONGIER, S. (1960) Statistical study of clinical and electroencephalographic manifestations of 536 psychotic episodes occurring in 516 epilepsies between clinical seizures. *Epilepsia*, **1**, 117–142.

FLOR-HENRY, P. (1969) Psychosis and temporal lobe epilepsy. *Epilepsia*, **10**, 363–395.

GREENSPAN, D. & LEVIN, D. (1985) Use of clonazepam in a patient with schizoaffective disorder. *American Journal of Psychiatry*, **142**, 774–775.

HERMANN, B. P. & WHITMAN, S. (1981) Behavioral and personality correlates of epilepsy: A review, methodological critique, and conceptual model. *Psychological Bulletin*, **95(3)**, 451–497.

KENDELL, R. E., BROCKINGTON, I. F. & LEFF, J. P. (1979) Prognostic implications of six alternative definitions of schizophrenia. *Archives of General Psychiatry*, **36**, 25–31.

LINDSAY, J., OUNSTED, C. & RICHARDS, P. (1979) Long term outcome in children with temporal lobe epilepsy: III. Psychiatric manifestations in adult life. *Developmental Medicine and Child Neurology*, **21**, 630–636.

MARQUIS-ASSIS, L. (1976) Discussion in *Epileptic Seizures—Behaviour—Pain* (ed. W. Birkmayer). Baltimore: University Park Press.

MIGNONE, R. J., DONNELLY, E. F. & SADOWSKY, D. (1970) Psychological and neurological comparisons of psychomotor and non-psychomotor epileptic patients. *Epilepsia*, **11**, 345–359.

PARNAS, J. & KORSGAARD, S. (1982) Epilepsy and psychosis. *Acta Psychiatrica Scandinavica*, **66**, 89–99.

PEREZ, M. M., TRIMBLE, M. R., MURRAY, N. M. F. & REIDER, I. (1985) Epileptic psychosis: an evaluation of PSE profiles. *British Journal of Psychiatry*, **146**, 155–163.

RODIN, E. A., KATZ, M. & LENNOX, K. (1976) Differences between patients with temporal lobe seizures and those with other forms of epileptic attacks. *Epilepsia*, **17**, 313–320.

SLATER, E., BEARD, A. W. & GLITHERO, E. (1963) The schizophrenia-like psychoses of epilepsy, *British Journal of Psychiatry*, **109**, 95–150.

—— & MORAN, P. A. P. (1969) The schizophrenia like psychoses of epilepsy: relation between ages of onset. *British Journal of Psychiatry*, **115**, 599–600.

SMALL, J., HAYDEN, M. & SMALL, I. (1966) Further psychiatric investigations of patients with temporal and non-temporal lobe epilepsy. *American Journal of Psychiatry*, **123**, 303.

STANDAGE, K. F. & FENTON, G. W. (1975) Psychiatric symptom profiles of patients with epilepsy: a controlled investigation. *Psychological Medicine*, **5**, 152–160.

STEVENS, J. R. (1966) Psychiatric implications of psychomotor epilepsy. *Archives of General Psychiatry*, **14**, 461.

—— (1973) Psychomotor epilepsy and schizophrenia: a common anatomy? In *Epilepsy: Its Phenomena in Man* (ed. M. A. B. Brazier). New York: Academic Press.

Paroxysmal Kinesigenic Choreoathetosis (PKC) and Hypomania

DEAR SIR,

PKC is a rare, involuntary movement disorder. The movements are tonic, dystonic, or choreoathetotic,

https://doi.org/10.1192/bjp.147.3.322 Published online by Cambridge University Press

and attacks occur spontaneously or may be induced by movement, startle, or anxiety. Sporadic and familial cases have been reported. Onset is usually during childhood, and the condition does not progress. Response to anticonvulsant therapy, including carbamazepine, and, in one case, L-DOPA (Loong & Ong, 1973) is usually excellent. EEGs and CAT scans have been recorded as normal. The pathophysiology is unknown but it has been postulated that the condition is an unusual form of epilepsy originating in the subcortical grey matter (Kinast *et al*, 1980). Although an initial diagnosis of hysteria has often been made (Kinast *et al*, 1980; Waller, 1977) associated psychiatric illness has not been clearly described except by Kertesz (1967) who described several patients with PKC as anxious or depressed and one who committed suicide.

I observed a case in which the patient was a 32-year-old man who presented in a clearly hypomanic state with cheerful mood, a variety of grandiose delusions, and pressure of speech. He denied any abnormal experiences and was reasonably orientated. A urinary drug screen was negative. Over the next 24 hours he remained much the same but on several occasions was noted to be behaving in a very odd manner. He had received phenothiazines during this time. Eventually he collapsed to the floor in what appeared to be a painful dystonic episode affecting his left side. Despite his distress he managed to convey that he suffered from PKC. This episode was relieved immediately by i.v. diazepam and a subsequent neurological referral confirmed that these episodes were consistent with the diagnosis of PKC (although one episode was terminated by a sharp command to relax). He was able to tell us that his condition was usually well-controlled on phenytoin, but that whilst trying to concentrate on his 'creative writings' he decided to omit the drug. His mental state did not entirely settle, but he discharged himself and follow-up has not been possible.

Despite rather inadequate information I am documenting this case for two reasons. Firstly, this rare condition has been confused with a hysterical conversion symptom, and might so have been in this case had the patient not provided the correct diagnosis. Secondly, this may be a case in which there is a co-existence of hypomania and a hypothesised right subcortical epileptic focus. It would be interesting to hear of anyone else's experience of this rare condition.

NEIL JOUGHIN

St. George's Hospital, Blackshaw Road, Tooting, London SW17

References

KERTESZ, A. (1967) Paroxysmal kinesigenic choreoathetosis: an entity within the paroxysmal choreoathetosis syndrome. *Neurology*, **17**, 680–690.

KINAST, M. *et al* (1980) Paroxysmal choreoathetosis: report of five cases and review of the literature. *Pediatrics*, **65(1)**, 74–77.

LOONG, S. C. & ONG, Y. Y. (1973) PKC: Report of Chinese cases. *Neurology*, **23**, 749–755.

WALLER, D. A. (1977) Paroxysmal kinesigenic choreoathetosis or hysteria? *American Journal of Psychiatry*, **134(12)**, 1439–1440.

Water Intoxication in Psychiatric Patients

DEAR SIR,

We have read with interest the article by Singh *et al* on "Water intoxication in psychiatric patients" (*Journal*, February 1985, **146**, 127–131). In these four reported cases, inappropriate antidiuretic hormone (ADH) secretion might represent a contributory cause of the psychosis. We wish to report three cases, supporting the hypothesis of two forms of the syndrome of inappropriate secretion of ADH (SIADH) and of hyperdopaminergic activity.

Case 1. A 43-year-old woman, resident in a mental hospital since the age of 30 for chronic psychosis, was treated with perphenazine (16 mg daily) and levomepromazine (120 mg daily). She was admitted to a general ward for generalised seizures and coma, without any other abnormality. Serum sodium was 117 mmol/l, urea 1.6 mmol/l, serum osmolality 219 mOsm/kg, and urine osmolality 141 mOsm/kg, but ADH serum level was 2.8 pg/ml. With water restriction, total clinical and biochemical recovery occurred within a week. Ten days later, plasma and urine osmolality, serum sodium, free water clearance, and ADH values were studied in a water loading test: urinary dilution was maximal with a positive free water clearance, at sufficiently low plasma osmolality (250 mOsm/kg) and ADH release was suppressed (plasma level from 1.8 pg/ml to 1.7 pg/ml). But the urine became hypertonic to plasma prematurely when plasma osmolality increased; ADH also increased to 2.1 pg/ml.

Case 2. A 26-year-old woman was admitted for generalised seizures and coma, after having drunk eight litres of tea. She was diagnosed as having a narcissistic personality disorder and as being polydipsic for two years. She was not treated with any medication. On admission, serum sodium was 110 mmol/l, urea 1.5 mmol/l, plasma osmolality 128 mOsm/kg, but the plasma ADH level was 2 pg/ml. Her clinical and biological status improved with water restriction, and a water loading test, ten days later, revealed no abnormalities: the ADH level was correctly suppressed.

Case 3. A 55-year-old woman, resident in a psychiatric hospital for 11 years because of chronic psychosis, was treated with pipamperone (160 mg daily) and levomepromazine (75 mg daily), and had tardive dyskinesia. She was admitted for generalised seizures and coma, after having drunk large quantities of water: serum sodium was

https://doi.org/10.1192/bjp.147.3.322 Published online by Cambridge University Press