Psychological Medicine, 1999, **29**, 1175–1181. Printed in the United Kingdom

Different psychopathological models and quantified EEG in schizophrenia

A. W. F. HARRIS,[1] L. WILLIAMS, E. GORDON, H. BAHRAMALI AND S. SLEWA-YOUNAN

From the Department of Psychological Medicine, University of Sydney; School of Psychology, University of New England, Armidale; and Cognitive Neuroscience Unit, Westmead Hospital, Westmead, NSW, Australia

ABSTRACT

Background. This study compared the ability of two different models of psychopathology in schizophrenia to account for findings in the quantified electroencephalogram (qEEG) recorded from midline sites in a group of 40 subjects with schizophrenia. The first model was based on the positive and negative syndrome dichotomy, the second was a tripartite model that resembled Liddle's syndromes of psychomotor poverty, disorganization and reality distortion (Liddle, 1987*a*).

Methods. A group of 40 subjects with predominantly chronic schizophrenia was assessed with the Positive and Negative Syndrome Scale (PANSS) prior to the acquisition of their quantified electroencephalogram. The relationship between EEG data and symptomatology was explored, initially with the PANSS positive and negative subscales and then with a tripartite model derived from a principal component analysis of the 14 positive and negative subscale items.

Results. The tripartite syndrome model showed a greater concordance with the qEEG of the subjects than the dichotomous model. 'Psychomotor poverty' was significantly positively correlated with both delta and beta power and 'reality distortion' was significantly positively correlated with alpha-2 power. No significant correlations between the positive and negative syndrome dichotomy and the qEEG were observed.

Conclusions. This study lends support to the factor analysis of psychopathology, and specifically the tripartite syndrome model of schizophrenia, as a step in explicating the biological dimensions of the disorder.

INTRODUCTION

A possible approach to the complex biological and clinical heterogeneity of schizophrenia, is to regard the disorder as a number of distinct symptom based syndromes. Crow (1980) suggested that a differentiation could be made between productive (type 1) schizophrenia and deficit (type 2) schizophrenia. Andreasen, drawing upon the work of Hughlings-Jackson and Bleuler, developed a syndrome subtyping based upon a dichotomy of positive and negative symptoms (Andreasen, 1982; Andreasen & Olsen, 1982). More recently, it has been suggested that an alternate approach to schizophrenia may have greater explanatory power, conceptualizing the disorder as a number of pathophysiological processes manifested by symptom based domains (Buchanan & Carpenter, 1994).

Clinicians and researchers have found the dichotomy of positive and negative symptoms inadequate when trying to account for the range of psychopathology found in schizophrenia (Kay & Sevy, 1990; Minas *et al.* 1992). Factor analytical studies, based on a number of different symptom rating tools, have suggested that additional symptom dimensions better explain this heterogeneity (for review see: Buchanan & Carpenter, 1994; Andreasen *et al.* 1995). Though a range of such dimensions has been put forward, a model describing three domains of psychopathology has been well supported (Liddle, 1987*a*).

[1] Address for correspondence: Dr A. W. F. Harris, Department of Psychiatry, Westmead Hospital, Westmead, NSW 2145, Australia.

The three factor model breaks the positive–negative symptom dichotomy into one dimension characterized by hallucinations and delusions, referred to by Liddle (1987*a*) as the 'reality distortion' syndrome of schizophrenia. A second dimension of positive schizophrenic symptoms is characterized by the presence of thought disorder, attentional problems (Liddle, 1987*a*; Liddle & Barnes, 1990; Peralta *et al.* 1992; Thompson & Meltzer, 1993; Kawasaki *et al.* 1994) and sometimes bizarre behaviour (Gur *et al.* 1991; Thompson & Meltzer, 1993; Andreasen *et al.* 1995). In Liddle's terminology, these symptoms represent a 'disorganization' syndrome. A third factor has been defined by 'deficit' negative symptoms, such as affective flattening, emotional and social withdrawal, and avolition. Liddle refers to this dimension as one of 'psychomotor poverty'.

Quantified EEG studies that have considered the psychopathological heterogeneity of schizophrenia have either examined the positive–negative dichotomy or severity of symptomatology. Negative symptoms have been associated with increased delta activity (Guenther *et al.* 1988; Gattaz *et al.* 1992) and inversely with alpha (Merrin & Floyd, 1992) and beta (Williamson *et al.* 1989; Gattaz *et al.* 1992). It has also been associated with treatment non-responders (Harris *et al.* 1997). Overall severity of symptoms has been observed to correlate inversely with alpha-1 (Omori *et al.* 1995), to correlate with alpha mean frequency (Schellenberg *et al.* 1992) and overall improvement with beta (Czobor & Volavka, 1993).

While the tripartite symptom structure has not so far been examined in relation to qEEG, cognitive performance, neurological signs and cerebral blood flow have been found to distinguish the three syndromes (Liddle, 1987*b*, 1996).

In this study it was proposed that the tripartite symptom structure would elucidate more precise relationships with qEEG activity than does the positive–negative dichotomy alone, as it is expected that it more accurately reflects the underlying psychopathological processes. More specifically, it is hypothesized that the psychomotor poverty syndrome would be associated with increased delta power and alpha-1 power, that the disorganization syndrome would be associated with a slowing of the qEEG, and that reality distortion would correlate with increased alpha, especially alpha-2 and beta power. The negative symptom pole of the positive–negative dichotomy would share with the psychomotor poverty syndrome increases in delta and alpha-1 power. However, it is hypothesized that few, if any, correlations will be found for positive symptoms (considered as a single category) reflecting the blending of different psychopathological processes.

METHOD

Subjects

Forty subjects with schizophrenia, aged between 16 and 65 years of age, were recruited from hospital and community health centres. The subjects were predominantly male (male $N = 29$; female $N = 11$), with a mean age of 35·4 years (S.D. = 8·0 years), a mean duration of illness of 12·6 years (S.D. = 8·4 years) and an average dose of medication of 661 chlorpromazine equivalents (S.D. = 637 CPZ equi) (Hollister, 1987; van Kammen & Marder, 1995). Diagnosis was confirmed using Section G (schizophrenia and psychotic disorders) of the Composite International Diagnostic Interview (World Health Organization, 1992*a*) or by two psychiatrists according to DSM-III-R (American Psychiatric Association, 1987) or ICD-10 criteria (World Health Organization, 1992*b*). Subjects were questioned about their previous medical history and excluded on the basis of a recent history of substance abuse, or past history of substance dependence, epilepsy, other neurological disorders, mental retardation or head injury and predominant left handedness. The Positive and Negative Syndrome Scale (Kay *et al.* 1986) was used to rate individual symptoms. All subjects were asked to refrain from smoking or drinking caffeine for 3 h before the recording session.

EEG acquisition

EEGs were acquired as part of an electrophysiological battery. Only the eyes closed condition was used in this analysis. The EEGs were recorded using an electrocap (Blom & Anneveldt, 1982) from 19 sites according to the international 10–20 system (Fp1, Fp2, Fz, F3, F4, F7, F8, Cz, C3, C4, P3, P4, Pz, T3, T4, T5, T6, O1, O2). Linked earlobes served as a

reference. Both horizontal and vertical EOG activity was recorded with two bipolar electrodes placed 1 cm lateral to the outer canthus of each eye and two bipolar electrodes placed above and below the centre of the right eye. All electrode impedances were maintained at 5 kΩ or less throughout the recording. During the procedure subjects were seated in a comfortable chair and asked to limit any eye movement. All potentials were acquired on a Syn Amps (NEURO SCAN Inc) 32 channel DC system with a gain of 10000, a digitalization rate of 250 Hz and an upper bandpass filter set at 50 Hz. A continuous EEG recording was acquired until 2 min of EEG was collected. All data was stored on tape for further analysis. All EOG correction was carried out post-acquisition according to a technique described by Gratton *et al*. (1983).

Data analysis

A Fast Fourier Transform analysis of 60 artefact free 2 s epochs was used to determine absolute EEG power in the delta (1·0–3·0 Hz), theta (4·0–7·0 Hz), alpha (8·0–13·0 Hz), alpha-1 (8·0–9·0 Hz), alpha-2 (10·0–13·0 Hz) and beta (14·0–30·0 Hz) bands. Logarithmic transformations were undertaken for EEG data, for each frequency band and midline site under 'eyes closed' conditions, to control for skewness. Subsequent correlation analyses with symptom variables were therefore conducted with transformed EEG variables.

PANSS Positive and Negative subscale scores represented positive and negative symptom categories. The tripartite division was indexed by factor scores, derived from a principal component analysis of the 14 positive and negative subscale items. The General Psychopathology subscale was omitted because the focus was on the relationship between performance and dimensions of positive and negative symptoms specifically and as a means of limiting the number of items submitted to the analysis. A varimax rotation was used because the factors were essentially uncorrelated, and it produces the simplest factor structure.

The relationship between EEG data and symptomatology was then explored using two-tailed Pearson correlation analysis, initially with the PANSS positive and negative subscales and then with factor scores. Given that *a priori* hypotheses were specified, an alpha level of 0·05 was used for correlation analyses. *Post hoc* partial correlation analyses were conducted to ensure that significant associations between symptom profiles (dichotomous or tripartite) and qEEG power were not due to the confounding effects of medication or duration of illness.

RESULTS

Symptom ratings from the 14 items of the PANSS positive and negative syndrome subscales were first subjected to principal components analysis (PCA). The PCA converged with five iterations with three factors with eigenvalues > 1, accounting for 68·80% of the variance. This was comparable with previous analyses of schizophrenic psychopathology (Buchanan & Carpenter, 1994; Andreasen *et al*. 1995). Component loadings are presented in Table 1. Factor 1 was defined primarily by deficit negative symptoms, and thus represented, using Liddle's terminology, a 'psychomotor poverty' dimension. Factor 2 was defined by high loadings from both positive and negative aspects of thought disorder, and grandiosity. Given the predominance of thought disorder items, this factor reflected a 'disorganization' dimension. The third factor, defined primarily by delusions, hallucinations and paranoia items

Table 1. *PCA of 14 items of the PANSS Positive and Negative subscales* (*factor loadings* $< 0{\cdot}30$ *excluded*)

	Factors		
PANSS symptoms	1	2	3
Blunted affect	0·84	—	—
Emotional withdrawal	0·84	—	—
Social withdrawal	0·82	—	—
Poor rapport	0·78	—	—
Lack of spontaneity	0·77	−0·33	—
Conceptual disorganization	—	0·81	—
Grandiosity	—	0·81	—
Excitement	—	0·76	—
Lack of abstract thinking	—	0·64	—
Stereotyped thinking	—	0·64	0·40
Suspiciousness	—	—	0·84
Delusions	—	0·39	0·73
Hostility	—	—	0·72
Hallucinatory behaviour	—	—	0·64
Variance explained (total = 68·8%)	38·82%	19·69%	10·30%

https://doi.org/10.1017/S0033291799008855 Published online by Cambridge University Press

Table 2. *Significant results* (P *values*) *for correlation of qEEG band with symptomatology, with* post hoc *partial correlations for effect of medication and duration of illness*

			Pearson correlation *P*	Covary for medication *P*	Covary for duration of illness *P*
PANSS subscale: eyes closed					
Negative symptoms	Delta	Pz	0·019	0·018	0·001
	Delta	Pz	0·035	0·047	0·024
Factor scores: eyes closed					
Psychomotor poverty	Delta	Pz	0·007	0·002	0·009
	Beta	Pz	0·005	0·009	0·005
Disorganization	Delta	Fz	0·020	0·069	0·024
Reality distortion	Alpha-1	Fz	0·034	0·040	0·014
	Alpha-2	Fz	0·015	0·010	0·003
	Alpha-total	Fz	0·019	0·010	0·005
	Alpha-total	Cz	0·036	0·018	0·009

(suspiciousness, hostility) represented a 'reality distortion' dimension.

The only significant correlations for the PANSS subscales was between the PANSS negative subscale scores and both beta and delta at Pz. Partial correlation analyses were conducted to examine the possible confounding of medication and length of illness with the significant findings for negative subscale scores. When CPZ equivalent medication level was taken into account, the positive associations between negative subscale scores and both delta and beta at Pz remained significant (delta, $r_{55} = 0{\cdot}39$, $P = 0{\cdot}018$; beta, $r_{55} = 0{\cdot}33$, $P = 0{\cdot}047$ respectively). Both relationships also remained significant when duration of illness was partialled out (delta, $r_{35} = 0{\cdot}51$, $P = 0{\cdot}001$; beta, $r_{55} = 0{\cdot}37$, $P = 0{\cdot}024$, respectively) – the latter clearly was strengthened (see Table 2).

When the relationship between qEEG data and factor scores was examined (see Table 2), significant positive correlations were found for psychomotor poverty and both delta and beta at Pz; for disorganization and delta at Fz; and for reality distortion and alpha-1 at Fz, alpha-2 at Fz, and total alpha at Fz and Cz. Partial correlation analyses were conducted to examine the possible confounding of medication and length of illness with the significant associations between factor scores and the EEG power. When CPZ equivalent medication level was taken into account, the relationships between reality distortion and alpha-1, alpha-2 and alpha-total at Fz remained significant ($r_{52} = 0{\cdot}39$, $P = 0{\cdot}040$; $r_{55} = 0{\cdot}42$, $P = 0{\cdot}010$; $r_{55} = 0{\cdot}40$, $P = 0{\cdot}01$). The positive relationship between reality distortion and alpha-total at Cz also remained significant ($r_{52} = 0{\cdot}39$, $P = 0{\cdot}018$). The positive relationships between psychomotor poverty and both delta and beta at Pz remained significant at the corrected alpha levels ($r_{55} = 0{\cdot}49$, $P = 0{\cdot}002$; $r_{55} = 0{\cdot}43$, $P = 0{\cdot}009$ respectively). However, the positive association between disorganization and delta at Cz was no longer significant when medication was controlled for ($r_{35} = 0{\cdot}30$, $P = 0{\cdot}069$).

When controlling for duration of illness, the positive relationships between reality distortion and alpha-1, alpha-2 and alpha-total at Fz remained significant ($r_{55} = 0{\cdot}40$, $P = 0{\cdot}014$; $r_{55} = 0{\cdot}47$, $P = 0{\cdot}003$; $r_{55} = 0{\cdot}45$, $P = 0{\cdot}005$). The association between reality distortion and alpha total at Cz also remained significant ($r_{55} = 0{\cdot}42$, $P = 0{\cdot}009$). The associations between psychomotor poverty and both delta and beta at Pz remained significant at $P < 0{\cdot}005$ ($r_{55} = 0{\cdot}49$, $P = 0{\cdot}009$; $r_{55} = 0{\cdot}46$, $P = 0{\cdot}005$). The association between disorganization and delta at Cz remained significant ($r_{55} = -0{\cdot}37$, $P = 0{\cdot}024$).

DISCUSSION

This study has confirmed the greater utility of the tripartite syndrome description of schizophrenic psychopathology in identifying underlying brain dysfunction, as reflected in the qEEG, compared with the positive–negative dichotomy. The results support the stability of the negative symptom cluster of both systems of classification, but indicate the importance of dividing

https://doi.org/10.1017/S0033291799008855 Published online by Cambridge University Press

the positive symptom cluster into smaller symptom groupings since without division not a single qEEG measure correlated with the overall symptom cluster.

The separation of reality distortion from disorganization, appears to have unlocked significant new associations, particularly for reality distortion. It is possible that the combination of the two positive syndromes 'cancelled out' interactions of interest in previous studies, and that the greater specificity of the syndromal approach has tapped, as hoped, more specific underlying psychopathological networks. The strong association of the EEG alpha band, which has been associated with ongoing information flow via thalamo-cortical networks (Steriade *et al*. 1990), and reality distortion may reflect aberrant information processing leading to the hallucination and delusions. The alpha band has been seen to alter with changes in attention (Ray & Cole, 1985) and in subjects with schizophrenia under different cognitive loads (Harris *et al*. 1998).

The predicted increase in beta power in association with reality distortion was not seen. This may have been due to the effects of medication dose and chronicity of illness (which have been shown to decrease overall EEG beta and other bands) – but this was not found to be the case in this study. Although decreased beta power has been observed in response to maximal readiness, paralleled by a reduction in alpha power (Pfurtscheller, 1992), changes in beta have also been associated with cognitive and emotional processes in the absence of any changes in alpha (Ray & Cole, 1985). The lack of any cognitive or emotional valence in this eyes closed paradigm, may be the cause for the absence of any increase in beta power. Further examination of this in a more cognitively challenging paradigms would help resolve this lack of relationship between beta and reality distortion.

Disorganization was positively correlated with delta power at the frontal site, however this was no longer significant when covaried for medication, pointing to the need to examine an unmedicated group of subjects with schizophrenia. This increase in frontal delta power among other findings links disorganization with psychomotor poverty. Both factors have been correlated with cognitive dysfunction and cortical neurological signs suggestive of frontal lobe dysfunction (Liddle, 1987*b*) and both have been correlated to the level of premorbid deficits (Lenzenweger & Dworkin, 1996). Disorganization and psychomotor poverty may both be characterized, at least in part, by a slowing of the quantified EEG which may be an index of the cognitive impairment associated with these two factors and schizophrenia.

The association of beta band with psychomotor poverty or negative symptoms was unexpected, as a deficit in beta power is often seen in subjects with chronic schizophrenia (Williamson *et al*. 1989; Gattaz *et al*. 1992; John *et al*. 1994). Certainly other reports (Scarone *et al*. 1987; Guenther *et al*. 1988; Karson *et al*. 1988) suggest changes in the topography of beta power in schizophrenia, or a change restricted to faster beta frequencies (Mukundan, 1986). This will be addressed in future reports examining qEEG topography and changes in higher frequency bands.

The effect of covarying for duration of illness was either negligible or served to considerably strengthen the significance of the relationship between the symptom syndrome and qEEG measure. The latter result may indicate that the change in psychopathology over time commonly seen in people with schizophrenia (Arndt *et al*. 1995; Quinlan *et al*. 1995) may not reflect changes in the psychopathological networks responsible for the symptoms, merely their expression.

In addition to examining for the effect of duration of illness, the effect of medication on the qEEG in the schizophrenic subjects, measured in chlorpromazine equivalents, was investigated. Medication is recognized as a potential source of slowing of the EEG with an increase particularly of theta often being observed (Small *et al*. 1987; John *et al*. 1994), thus it was interesting to observe that only the strength of the relationship between disorganization and delta at Fz was diminished when the result was covaried for medication. Another result, the relationship between total alpha and the reality distortion factor at Fz, was strengthened. Beyond this little effect appeared due to medication. In our view this supports the robustness of the findings and underlines the independence of the increased delta activity, often described in schizophrenia, from medication effects.

The effect of limiting the symptom profile to the positive and negative subscales of the PANSS is difficult to determine with the size of the study group. Certainly the aim of the study was to limit the range of psychopathology examined so as to facilitate the comparison between the dichotomy of positive and negative syndromes and the tripartite separation of symptoms. However, factor analyses of the full PANSS, have identified additional factors reflecting cognitive disability, depression, hostility and anxiety (Kay & Sevy, 1990; Bell *et al*. 1994; Kawasaki *et al*. 1994; Lindenmeyer *et al*. 1994; Peralta & Cuesta, 1994) and the inclusion of factors reflecting these symptoms may provide a better fit for the qEEG. This awaits testing with a larger study group.

This study lends support to the factor analysis of the psychopathology of schizophrenia as a step in explicating the biological dimensions of the disorder. It lends weight to the tripartite separation of symptoms, suggesting that this division of symptoms better reflects underlying psychopathology than a simple dichotomy of positive and negative syndromes. In reappraising the literature of qEEG in schizophrenia, this strategy may assist in elucidating some of the contradictory findings and in refining hypotheses in future studies.

REFERENCES

American Psychiatric Association (1987). *Diagnostic and Statistical Manual of Mental Disorders*, *3rd edn. revised*. APA: Washington, DC.

Andreasen, N. C. (1982). Negative symptoms in schizophrenia: definition and reliability. *Archives of General Psychiatry* **39**, 784–788.

Andreasen, N. C. & Olsen, S. (1982). Negative v. positive schizophrenia. *Archives of General Psychiatry* **39**, 789–794.

Andreasen, N., Arndt, S., Alliger, R., Miller, D. & Flaum, M. (1995). Symptoms of schizophrenia. *Archives of General Psychiatry* **52**, 341–351.

Arndt, S., Andreasen, N. C., Flaum, M., Miller, D. & Nopoulos, P. (1995). A longitudinal study of symptom dimensions in schizophrenia: prediction and patterns of change. *Archives of General Psychiatry* **52**, 353–360.

Bell, M. D., Lysaker, P. H., Beam-Goulet, J. L., Milstein, R. M. & Lindenmeyer, J.-P. (1994). Five-component model of schizophrenia: assessing the factorial invariance of the Positive and Negative Syndrome Scale. *Psychiatry Research* **52**, 295–303.

Blom, J. L. & Anneveldt, M. (1982). An electrode cap tested. *Electroencephalography and Clinical Neurophysiology* **54**, 591–594.

Buchanan, R. W. & Carpenter, W. T. (1994). Domains of psychopathology: an approach to the reduction of heterogeneity in schizophrenia. *Journal of Nervous and Mental Disease* **182**, 193–204.

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

Czobor, P. & Volavka, J. (1993). Quantitative electroencephalogram examination of effects of respiridone in schizophrenic patients. *Journal of Clinical Psychopharmacology* **13**, 332–342.

Gattaz, W. F., Mayer, S., Ziegler, P., Platz, M. & Gasser, T. (1992). Hypofrontality on topographic EEG in schizophrenia. Correlations with neuropsychological and psychopathological parameters. *European Archives of Psychiatry and Clinical Neuroscience* **241**, 328–332.

Gratton, G., Coles, M. G. & Donchin, E. (1983). A new method for off-line removal of ocular artifact. *Electroencephalography and Clinical Neurophysiology* **55**, 468–484.

Guenther, W., Davous, P., Godet, J.-L., Guillibert, E., Breitling, D. & Rondot, P. (1988). Bilateral brain dysfunction during motor activation in type II schizophrenia measured by EEG mapping. *Biological Psychiatry* **23**, 295–311.

Gur, R. E., Mozley, P. D., Resnik, S. M., Levick, S., Erwin, R., Saykin, A. J. & Gur, R. C. (1991). Relations among clinical scales in schizophrenia. *American Journal of Psychiatry* **148**, 472–478.

Harris, A. W. F., Gordon, E., Anderson, J., Ritchie, G., McLachlan, C. & Meares, R. (1997). Change in quantified electroencephalography (QEEG) with medication and altered clinical state in the same subjects with schizophrenia. *Schizophrenia Research* **23**, 87–89.

Harris, A. W. F., Bahramali, H., Lagopoulos, J., Li, W. M., Gordon, E. & Wright, J. J. (1998). The effect of paradigm on alpha frequency in schizophrenia. *International Journal of Neuroscience* **96**, 63–72.

Hollister, L. E. (1987). Psychiatric Disorders. In *Avery's Drug Treatment*, *3rd edn*. (ed. T. S. Speight), pp. 1057–1121. ADIS Press: Auckland, NZ.

John, E. R., Prichep, L. S., Alper, K. R., Mas, F. G., Cancro, R., Easton, P. & Sverlov, L. (1994). Quantitative electrophysiological characteristics and subtyping of schizophrenia. *Biological Psychiatry* **36**, 801–826.

Karson, C. N., Coppola, R., Daniel, D. G. & Weinberger, D. R. (1988). Computerised EEG in schizophrenia. *Schizophrenia Bulletin* **14**, 193–197.

Kawasaki, Y., Maeda, Y., Sakai, N., Hiashima, M., Urata, K., Yamaguchi, N. & Kurachi, M. (1994). Evaluation and interpretation of symptom structures in patients with schizophrenia. *Acta Psychiatrica Scandinavica* **89**, 399–404.

Kay, S. R. & Sevy, S. (1990). Pyramidical model of schizophrenia. *Schizophrenia Bulletin* **16**, 537–545.

Kay, S. R., Opler, L. A. & Fiszbein, A. (1986). *Positive and Negative Syndrome Scale* (*PANSS*). Multi-Health Systems Inc.: North Tonawanda, NY.

Lenzenweger, M. F. & Dworkin, R. H. (1996). The dimensions of schizophrenia phenomenology. Not one or two, at least three, perhaps four. *British Journal of Psychiatry* **168**, 432–440.

Liddle, P. F. (1987*a*). The symptoms of chronic schizophrenia: a re-examination of the positive–negative dichotomy. *British Journal of Psychiatry* **144**, 145–151.

Liddle, P. F. (1987*b*). Schizophrenic syndromes, cognitive performance and neurological dysfunction. *Psychological Medicine* **17**, 49–57.

Liddle, P. F. (1996). Syndromes in schizophrenia and their neuropsychological and neuroanatomical correlates. In *Schizophrenia: A Neuropsychological Perspective* (ed. C. Pantelis, H. E. Nelson and T. R. E. Barnes), pp. 299–313. John Wiley & Sons: Chichester.

Liddle, P. F. & Barnes, T. R. E. (1990). Syndromes of chronic schizophrenia. *British Journal of Psychiatry* **157**, 558–561.

Lindenmeyer, J. P., Bernstein-Hyman, R. & Grochowski, S. (1994). Five factor model of schizophrenia: initial validation. *Journal of Nervous and Mental Disease* **182**, 631–638.

Merrin, E. L. & Floyd, T. C. (1992). Negative symptoms and EEG alpha activity in schizophrenic patients. *Schizophrenia Research* **8**, 11–20.

Minas, I. H., Stuart, G. W., Klimidis, S., Jackson, H. J., Singh, B. S. & Copolov, D. L. (1992). Positive and negative symptoms in the

psychoses: multidimensional scaling of SAPS and SANS items. *Schizophrenia Research* **8**, 143–156.
Mukundan, C. R. (1986). Computed EEG in schizophrenics. *Biological Psychiatry* **21**, 1221–1225.
Omori, M., Koshino, Y., Murata, T., Murata, I., Nishio, M., Sakamoto, K., Horie, T. & Isaki, K. (1995). Quantitative EEG in never-treated schizophrenic patients. *Biological Psychiatry* **38**, 303–309.
Peralta, V. & Cuesta, M. J. (1994). Psychometric properties of the Positive and Negative Syndrome Scale (PANSS) in schizophrenia. *Psychiatry Research* **53**, 31–40.
Peralta, V., de Leon, J. & Cuesta, M. J. (1992). Are there more than two syndromes in schizophrenia? *British Journal of Psychiatry* **161**, 335–343.
Pfurtscheller, G. (1992). Event-related synchronisation (ERS): an electrophysiological correlate of cortical areas at rest. *Electroencephalography and Clinical Neurophysiology* **83**, 62–69.
Quinlan, D. M., Schuldberg, D., Morgenstern, H. & Glazer, W. (1995). Positive and negative symptom course in chronic community-based patients. A two-year prospective study. *British Journal of Psychiatry* **166**, 634–641.
Ray, W. J. & Cole, H. W. (1985). EEG alpha activity reflects attentional demands, and beta activity reflects emotional and cognitive processes. *Science* **228**, 750–752.
Scarone, S., Gambini, O., Colombo, C., Cattaneo, R., Bellodi, L. & Pugnetti, L. (1987). Electrophysiological characteristics of hemispheric functioning in schizophrenic disorders. In *Cerebral Dynamics, Laterality and Psychopathology* (ed. R. Takahashi, P. Flor-Henry, J. H. Gruzelier, S. Niwa), pp. 115–124. Elsevier: Amsterdam.
Schellenberg, R., Schwarz, A., Knorr, W. & Haufe, C. (1992). EEG-Brain mapping. A method to optimize therapy in schizophrenics using absolute power and center frequency values. *Schizophrenia Research* **8**, 21–29.
Small, J. G., Milstein, V., Small, I. F., Miller, M. J., Kellams, J. J. & Corsaro, C. J. (1987). Computerised EEG profiles of haloperidol, chlorpromazine, clozapine and placebo in treatment resistant schizophrenia. *Clinical Electroencephalography* **18**, 124–135.
Steriade, M., Gloor, P., Llinas, R. R., Lopes da Silva, F. H. & Mesulam, M. M. (1990). Basic mechanisms of cerebral rhythmic activities. Report of IFCN Committee on Basic Mechanisms. *Electroencephalography and Clinical Neurophysiology* **76**, 481–508.
Thompson, P. A. & Meltzer, H. Y. (1993). Positive, negative, and disorganisation factors from the Schedule for Affective Disorders and Schizophrenia and the Present State Examination. *British Journal of Psychiatry* **163**, 344–351.
van Kammen, D. P. & Marder, S. R. (1995). Dopamine receptor antagonists. In *Comprehensive Textbook of Psychiatry*, *6th edn*. (ed. H. I. Kaplan and B. J. Sadock), pp. 1987–2022. Williams & Wilkins: Baltimore.
Williamson, P. C., Kutcher, S. P., Cooper, P. W., Snow, W. G., Szalai, J. P., Kaye, H., Morrison, S. L., Willinsky, R. A. & Mamelak, M. (1989). Psychological, topographical EEG, and CT scan correlates of frontal lobe function in schizophrenia. *Psychiatry Research* **29**, 137–149.
World Health Organization (1992*a*). *Composite International Diagnostic Interview*, *1st edn*. WHO: Geneva.
World Health Organization (1992*b*). *The ICD-10 Classification of Mental and Behavioural Disorders* (*ICD-10*). WHO: Geneva.