Memory in functional psychosis

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SUMMARY Acute schizophrenic, chronic schizophrenic, and depressive patients (20 of each) were compared with normal subjects and six groups of patients with organic brain disease. They were given tests of verbal learning (left hemisphere type function) and pattern recognition memory (right hemisphere type function). All functional psychotics showed impaired memory. Acute schizophrenics were, however, only impaired on the verbal task, suggesting left hemisphere dysfunction, while chronic schizophrenics and depressives were impaired on both tasks, suggesting bilateral dysfunction.

Global impairment of memory is regarded traditionally as a characteristic of those psychiatric disorders where organic brain damage is present. There is, however, increasing evidence that memory is not intact in the functional psychoses. Johnstone *et al.* (1978) reported severe memory impairment in chronic schizophrenics in long-stay wards. Flor-Henry (1976) and Koh (1978) identified poor performances on some memory tasks in acute schizophrenics. A depressive pseudodementia is recognised clinically, and Larner (1977), Miller and Lewis (1977) and Strömberg (1977) have confirmed that memory impairment is a regular feature of depressive illness.

It has generally been assumed that memory impairment in the course of a functional psychosis is not central to the disease process itself but is a secondary phenomenon attributable to some other factor. Such factors include pre-existing brain damage as in the schizophrenia-like psychoses of epilepsy (Slater *et al.*, 1963); the side effects of treatment—leucotomy, medication, electroconvulsive therapy (ECT); and psychological factors—apathy induced by institutionalism, inattention through preoccupation with psychotic experiences. In the case of the chronic schizophrenics studied by Johnstone *et al.*, equivalent hospitalisation for a physical illness did not lead to impairment of memory, some patients with a

memory deficit had never received phenothiazines, and the presence of delusions or hallucinations on mental state examination was not critical. In depression, the administration of ECT, even bilateral or left sided applications, which are known to produce memory deficits themselves, can lead to improvement of memory (Small *et al.*, 1972; Strömberg, 1977). Such evidence suggests that impaired memory may, in part, be a central feature of the psychosis rather than a secondary phenomenon.

In view of these findings I decided to study the prevalence of one aspect of cognitive dysfunction in psychiatric conditions. I chose memory disorder as this, more than disturbances of attention or perception, has been regarded traditionally as characteristic of organic states. I used more than one task (paired-associate learning to represent verbal memory, pattern recognition for non-verbal memory) and more than one measure of the latter (total correct score to represent quantitative aspects, distribution of false negatives and false positives for qualitative aspects). My subjects were grouped into three functional (acute schizophrenia, chronic schizophrenia, depression) and six organic categories (alcoholism, confusional states, left hemisphere lesions, right hemisphere lesions, Korsakoff's syndrome, presenile dementia). The contribution of extrinsic factors mentioned earlier was examined. The effect of any previous organic brain damage contaminating the scores of the functional groups was minimised by excluding any

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patient who had a history of such—for example, a patient with the clinical picture of acute schizophrenia but a history of epilepsy was not included. The influence of treatment, length of stay in hospital, and preoccupation with psychotic themes was assessed by identifying functional subgroups who differed on these factors.

Subjects

There were nine pathological groups with 20 subjects in each, with the exception of presenile dementia where only 10 subjects were found in the period of the study. A “normal” group of 40 subjects was also studied, the larger number reflecting the desire to obtain more comprehensive normative data for the unstandardised tests used.

No patient, except in the group with confusional states, was aged over 65 years. The sex distribution, mean age, and intelligence (IQ, Mill Hill Vocabulary) are shown in Table 1. They show substantial differences, reflecting genuine characteristics of the groups, which made matching for these variables impracticable. Sex and age mismatching were, therefore, corrected in the statistical treatment of results. Only subjects whose native language was English were included and, as only three groups (chronic schizophrenia, left hemisphere lesions, dementia) differed significantly from normal on IQ (*t* test) no correction for IQ was made as it was felt that the impaired performance on the Mill Hill Vocabulary might be an effect of the pathological process itself.

“Normals” were chosen from among neurological inpatients with disease restricted to peripheral nerves or spinal cord (21) and from psychiatric outpatients attending with personality disorder, phobias, or anxiety neurosis (19). *Acute schizophrenics* were consecutive admissions to two psychiatric wards whose mental state in a standardised interview (Present State Examination, PSE, of Wing *et al.*, 1974) fitted these authors’ criteria for schizophrenia. Thirty per cent did not

co-operate with testing. The remainder were all acutely psychotic, although all were on phenothiazines. *Chronic schizophrenics* were inpatients in two long-stay wards of a large mental hospital. They had been regarded as schizophrenic throughout their stay (mean length, 16 years) but not all were floridly psychotic on the day of testing. Any who had undergone leucotomy were excluded. All had received phenothiazines and half of them ECT at some time. Twenty-six fulfilled the inclusion criteria. Four failed to co-operate with the tests, and the two most disorientated patients were also excluded. *Depressives* were consecutive admissions to two psychiatric wards with a diagnosis of depression (“psychotic” or “retarded” by the PSE and criteria of Wing *et al.*). None had received ECT in the previous year but all had been prescribed tricyclic antidepressants in the previous two weeks. *Confusional states* were diagnosed in general medical inpatients with disorientation in their mental state in association with a physical disease (for example, systemic lupus erythematosus). *Alcoholics* were alcohol-dependent subjects, abstinent for at least four weeks, with heavy daily alcohol consumption (150 ml absolute alcohol) for at least 15 years. The groups with *right or left hemisphere lesions* contained subjects with temporal lobectomy (10 in each), unilateral temporal lobe epilepsy (five in each), and miscellaneous unilateral lesions. *Korsakoff* subjects were inpatients with severe anterograde amnesia and disorientation confidently diagnosed as Korsakoff’s syndrome related to past alcohol consumption. *Dements* were neurological inpatients, under 65 years of age, in whom a pneumoencephalogram had shown cerebral atrophy.

Methods

Subjects were told that I was interested in memory and concentration in people with their condition and asked to co-operate.

Table 1 General characteristics of diagnostic groups

Group	Number	Sex		Age (yr)	IQ (Vocabulary)
		Male	Female		
Normal	40	20	20	45	99
Acute schizophrenia	20	8	12	35	98
Chronic schizophrenia	20	14	6	44	86
Depression	20	6	14	49	98
Confusional state	20	12	8	58	101
Alcoholism	20	14	6	48	94
Right hemisphere lesion	20	16	4	43	104
Left hemisphere lesion	20	11	9	40	87
Dorsakoff’s syndrome	20	15	5	53	99
Kementia	10	6	4	55	90

VERBAL MEMORY

The paired-associate learning subtest of the Wechsler Memory Scale was modified to contain 10 moderately easy pairs (for example, river-blue), and the list of these was presented twice. Responses were asked for after each list presentation by reading out the first word of the pair. The score was the percentage of correct responses out of a maximum of 20.

PATTERN RECOGNITION MEMORY

Fifty coloured reproductions of histological specimens, stimuli which are difficult to verbalise, were pasted on 3 in. $\times$ 3 in. cards. Half (items) were shown for three seconds each with instruction to memorise. The 25 items were then shuffled with the other 25 cards (fillers) and all 50 shown, with the instruction that subjects should say "yes" or "no" according to whether they thought they had seen them before.

Two measures were calculated: *total correct*—percentage of all pictures correctly identified as familiar or unfamiliar; and *percentage of false positives in total errors*, an estimate of the pattern of errors independent of total correct. The total correct score will be referred to when discussing quantitative impairment of pattern memory, the other measure reflecting qualitative aspects.

STATISTICAL ANALYSIS

The three measures for each patient were subjected to an inverse sign transformation and by analysis of covariance these were then corrected for age and sex. Analysis of variance was then carried out on the corrected scores and, following an overall significant effect of groups, a posteriori tests were then done using the corrected pooled standard error. Duncan's test (Winer, 1971) was chosen as the a posteriori test for the effect of diagnosis. This is not a conservative test but allows moderate conviction on the significance of differences. The effect of extrinsic factors was evaluated by the *t* test on nominated pairs of groups.

Results

EFFECT OF DIAGNOSIS—QUANTITATIVE MEASURES (VERBAL AND PATTERN TOTAL CORRECT MEMORY)

The raw group means in rank order are shown in Table 2. The significant differences between functional and between functional and organic groups, are shown in matrix form in Table 3.

Depressives and chronic schizophrenics were impaired on both tasks, in comparison with normal subjects, while acute schizophrenics were only impaired on the verbal task.

Chronic schizophrenics were worse than either acute schizophrenics or depressives on both tasks. Acute schizophrenics were not significantly different from depressives on either task.

Acute schizophrenics most resembled those with left hemisphere lesions or confusional states and differed from other groups on at least one measure. Chronic schizophrenics achieved a score on both tasks comparable to patients with confusional states, dementia, or Korsakoff's syndrome. Depressives could not be distinguished on the present measures from those with left hemisphere lesions or confusional states and, like schizophrenics, had a comparable verbal memory to that of dements.

EFFECT OF DIAGNOSIS—QUALITATIVE MEASURE

The raw means for the proportion of false positives in errors are presented in rank order in Table 2 with significant differences for the functional groups in Table 3.

Chronic schizophrenics made more false positives in their errors than did normal subjects, and acute and chronic schizophrenics gave relatively more false positives than depressives.

Acute schizophrenics differed from Korsakoff and demented patients; chronic schizophrenics differed from all but the right hemisphere group; and depressives differed from no organic group.

EFFECT OF EXTRINSIC FACTORS

Subgroups representing contrasting effects of treat-

Table 2 Mean scores of diagnostic groups in rank order

Verbal memory (% correct)		Pattern memory-total correct (% correct above chance)		Pattern memory-distribution of errors (% false positives in errors)	
Korsakoff	21.5	Dementia	17.2	Chronic schizophrenia	73.3
Chronic schizophrenia	32.0	Korsakoff	22.4	Acute schizophrenia	56.3
Confusion	33.0	Alcoholic	33.2	Right hemisphere lesion	49.3
Dementia	33.0	Chronic schizophrenia	34.0	Confusion	47.0
Left hemisphere lesion	44.0	Right hemisphere lesion	37.6	Alcoholic	43.7
Depression	48.5	Confusion	40.4	Depression	42.6
Acute schizophrenia	57.0	Depression	49.2	Normal	40.4
Right hemisphere lesion	64.8	Left hemisphere lesion	55.2	Left hemisphere lesion	34.8
Alcoholic	70.0	Acute schizophrenia	58.8	Dementia	28.0
Normal	78.1	Normal	62.2	Korsakoff	20.6

Table 3 Matrix of significant differences comparing functional psychoses with each other and with organic conditions

	Acute schizophrenia	Chronic schizophrenia	Depression
Acute schizophrenia	*	*	*
Chronic schizophrenia	Verbal; pattern	*	*
Depression	Error distribution	Verbal; pattern; error distribution	*
Normal	Verbal	Verbal; pattern; error distribution	Verbal; pattern
Alcoholic	Verbal; pattern	Verbal; error distribution	Verbal; pattern
Right hemisphere lesion	Pattern	Verbal	Verbal
Left hemisphere lesion		Pattern; error distribution	
Dementia	Pattern; error distribution	Error distribution	Pattern
Korsakoff	Verbal; pattern; error distribution	Error distribution	Verbal; pattern
Confusion		Error distribution	

All differences are significant at the 0.01 level using Duncan's test on corrected transformed scores.

Verbal=verbal memory, pattern=pattern memory (total correct), error distribution=distribution of false positives and false negatives in pattern memory.

General factors significantly affecting scores: verbal memory—males worse, old worse; pattern memory—old worse; error distribution—females greater % of false positives.

ment, hospitalisation, and clinical state were prepared. Differences between the contrasting subgroups are reported for verbal memory and total correct pattern memory only, based on *t* tests using corrected measures. The results are shown in Table 4.

Treatment

No patient had undergone leucotomy. An attempt to evaluate the effect of medication was abandoned as all schizophrenics were on phenothiazines, all depressives on antidepressants, and missing drug sheets or inadequate records made the identification of heavily and moderately treated subgroups impossible. Information on the administration of ECT, however, appeared relatively complete. Half the chronic schizophrenics had received ECT at some stage, and they actually performed better than those who had not. Seven depressives had received previous ECT and they were not significantly worse than those who had not been given

the treatment, although there was a trend in this direction.

Hospitalisation

The 10 chronic schizophrenics with the longest stay were worse, but not significantly so, than the 10 with the shortest stay. There was also a consistent tendency for acute schizophrenics and depressives with previous admissions to be worse on both tests than those beginning their first admission, but this only reached significance in the case of depressives on verbal memory.

Clinical state

The influence of clinical state was only assessed for chronic schizophrenics and depressives. Two measures were made in chronic schizophrenics. The ward nurses were asked to fill in Wing's (1961) ward behaviour scale, designed to measure social withdrawal and socially embarrassing behaviour. Ten patients with the highest ratings on

Table 4 Effect of extrinsic factors on memory (raw scores of subgroups expressed as percentage)

	Verbal memory		Pattern memory (total correct)	
ECT	ECT	No ECT	ECT	No ECT
Chronic schizophrenia	42.0	19.0	37.4	32.8
Depression	40.0	53.0	44.0	52.0
Hospitalisation	Long/multiple	Short/single	Long/multiple	Short/single
Chronic schizophrenia	18.5	42.0	28.0	42.4
Acute schizophrenia	50.5	63.5	51.6	66.8
Depression	37.9*	64.4	45.0	55.6
Clinical state				
Social withdrawal	Withdrawn	Not withdrawn	Withdrawn	Not withdrawn
Chronic schizophrenia	14.0†	47.0	27.6	42.8
Psychotic	Psychotic	Not psychotic	Psychotic	Not psychotic
Chronic schizophrenia	39.4	24.6	43.0	30.0
Depression	45.7	50.0	49.2	49.2

* = $P > 0.05$,

† = $P > 0.02$ between pairs of adjacent means (*t* test on corrected transformed scores)

social withdrawal were worse than the 10 with the lowest ratings, significantly so for verbal memory. Secondly, eight patients with current psychotic phenomena elicited by the PSE performed better than 12 without such phenomena, though this difference was not significant. Seven depressive patients with psychotic phenomena (for example, delusions of reference) were comparable to non-psychotic depressives on both tests.

The effect of medication was not evaluated. Previous ECT was not apparently responsible for impairment on the tests. Prolonged hospitalisation in chronic schizophrenics and repeated admissions in both acute groups were associated consistently with poor test performance, but this only reached significance in one of six comparisons (verbal memory in depressives). The presence of psychotic phenomena at the time of testing did not appear critical in determining impairment in chronic schizophrenics or depressives. The most significant finding was that socially withdrawn chronic schizophrenics were particularly impaired on verbal memory.

Discussion

The chief finding to emerge was that all three functional groups were impaired on at least one of the memory tasks. Further, chronic schizophrenics were not significantly better than demented on either task, and acute schizophrenics and depressives were not significantly better than demented on verbal memory. The conclusion which can be drawn, therefore, is that memory impairment, traditionally regarded as a characteristic feature of an organic mental state, implying the existence of brain damage of some sort, is in fact a common accompaniment of these functional disorders. There are two main alternative explanations. Either the capacity to carry out the tasks was being affected by some related factor not itself part of the psychotic process, or the observed impairment was a fundamental characteristic of the psychotic process.

The factors to be considered include pre-existing brain damage, the effect of impairment of other psychological functions (for example, motivation) on memory, and the effect of treatment. A recognised cause of brain damage, such as epilepsy, meant exclusion from one of the functional groups. The presence of psychotic phenomena (abnormal experiences and beliefs) at the time of testing might have been expected to impair attention and hence affect memory independently of diagnosis, but depressives and chronic schizophrenics free of these were no less

impaired.

Motivational factors could, however, have contributed to the different performance of acute schizophrenics on verbal learning and pattern recognition tasks, since the two tasks require different cognitive strategies and differences attributed to hemispheric involvement may have reflected other cognitive factors. This is a major defect in the study, but it was difficult to surmount because if a verbal recognition test had been selected in place of the recall task, right hemisphere involvement could have been introduced. Of treatment effects the easiest to exclude was a previous course of ECT, which did not result in poorer performance. The other two major treatment factors, institutionalism and medication, are confounded in the present study. Although there was a tendency for patients in all three groups to perform better, in the case of acute groups if they were beginning their first admission, and in the case of chronic schizophrenics if they had remained less time in hospital, this can be variously interpreted. First, it may represent a general effect of institutionalism, described in convincing fashion by Wing and Brown (1970). This might be true for the chronic schizophrenics but is less likely to apply to the acute groups, where previous admissions were in the main few and brief. Secondly, it may indicate a deleterious and cumulative effect of medication. There is only meagre evidence on this in the literature. Against such an effect is the fact that Johnstone and her colleagues found memory impairment in some chronic schizophrenics who had never been prescribed phenothiazines (Crow and Johnstone, 1977), the demonstration by Small *et al.* (1972) that memory improved in acute schizophrenics after phenothiazines and in depressives after antidepressants, and reports that other cognitive functions return to normal after medication—for example, attention in acute schizophrenics after phenothiazines (Spohn *et al.*, 1977). In favour of an adverse effect of medication on memory are the suggestion by Marsden (1976) that phenothiazines, by blocking dopamine receptors throughout the brain, affect cortical function in schizophrenics; the finding of Kendrick (1979), in a new version of his neuropsychological battery, that phenothiazines induce a dementia-like pattern of deficit in elderly depressives; and reports that lithium (only given to two depressives in the present study) impairs short-term memory (Kusumo and Vaughan, 1977). The issue is for the moment unresolved. Thirdly, the association between impaired memory and hospitalisation may reflect the influence of severity of illness: more severely

affected patients would stay longer in hospital and be more liable to readmission. This possibility is supported by the significant association between severity of chronic schizophrenia, as measured by social withdrawal, and verbal memory impairment, an association which was more pronounced than that between hospitalisation and memory impairment. It would appear, therefore, that one of the extrinsic factors—medication—cannot be discounted as a cause of the general findings, but the alternative explanation, that memory impairment is intrinsic to the functional conditions, is also consistent with these findings.

The alternative explanation derives particular support from the finding that in acute schizophrenics pattern memory is preserved while verbal memory is deficient. Subject to the criticism discussed earlier, that motivation may have been affected unequally in the two tasks, this suggests that in acute schizophrenia left hemisphere type functions are impaired while right hemisphere type functions remain intact. This idea was originally proposed by Flor-Henry, based on his findings of an association between left temporal lobe epilepsy and a schizophrenic psychosis (Flor-Henry, 1969). It has since drawn support from psychophysiological experiments where asymmetries in skin conductance pointed to a left hemisphere dysfunction (Gruzeliier, 1973), and from tachistoscopic (Gur, 1978) and dichotic listening studies (Colbourn and Lishman, 1979) where an abnormal right hemisphere bias in the perception of verbal material has been identified. Chronic schizophrenics in the present study did not show this dissociation, although those with the greatest social withdrawal were particularly poor on verbal memory. The depressives did not show the dissociation: performance on both tasks was impaired relative to normal subjects. This contradicts the opinion of Flor-Henry, who believes that right hemisphere function is impaired selectively in affective psychosis (Flor-Henry, 1976), although he does admit that specific impairment of right hemisphere type functions in depression is less striking—that is, there is some bilateral impairment—than is the specific impairment of left hemisphere type functions in schizophrenia. Other studies on the incidence of pathological mood change after unilateral brain lesions are contradictory. Gainotti (1972), Cutting (1978) and Gasparrini *et al.* (1978) found depressive mood more common after left sided lesions, while Folstein *et al.* (1977) found an association with right sided damage. Strömberg (1977) indicated that the matter may be more

complex. She found that depressive appearance and agitation were associated with poor performance on all the tests she gave (attention, verbal, and non-verbal memory) while depressive content of ideas was associated only with non-verbal memory impairment. In summary, despite some contradictions, there is some support from other sources that the impairment in memory identified in the present study may be the result of an inherent dysfunction of one or both hemispheres in the particular psychosis.

The qualitative aspect identified concerned the distribution of errors in pattern memory. The chief finding was of a difference in the relative proportion of false negatives and false positives between depressives, with a relatively high rate of false negatives, and both groups of schizophrenics, with a relatively high rate of false positives. This finding is notable on two counts. First, it was an aspect of cognitive function where acute and chronic schizophrenics most resembled one another, and which best discriminated them from other groups. (They were the only groups where the majority of their errors were false positives.) Secondly, it is rare to find an aspect of cognitive function which discriminates between acute schizophrenics and depressives. Many claims that there exists a specific cognitive deficit in schizophrenia, based on an experiment using normal controls, have been refuted when depressive control subjects are shown to have the same deficit. In this way, for example, Levy and Maxwell (1968) demonstrated that the inability to benefit from contextual constraint in a verbal memory task, previously believed to be specific to schizophrenics, also applied to depressives. It could be argued that the finding of impaired verbal memory in conjunction with preserved pattern memory in acute schizophrenics, demonstrated in the present study, is open to the same criticism, as acute schizophrenics and depressives were not significantly different from each other when the total correct score, a quantitative measure, is considered. The emergence of a significant qualitative difference is, therefore, especially notable. It has been reported that depressives tend to respond “conservatively” in memory tasks, preferring to judge an item as familiar only when they are very sure (Whitehead, 1974; Larner, 1977; Miller and Lewis, 1977), a strategy which leads them to make relatively more false negatives than other psychiatric groups, though not, in the present study, significantly more so than normal subjects. Less interest has been shown in this aspect of behaviour in

schizophrenics, despite theories which claim that they are more impaired when faced with a complex response than when called upon to appreciate an equivalently complex stimulus (Marshall, 1973). There is a suggestion, however, from Lewine's (1978) review of responses in perceptual tasks and from Koh's (1978) experiments on memory searching, that some schizophrenics do behave less conservatively than other psychiatric patients or normals, a strategy which leads them to make, as in the present study, relatively more false positives.

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References

- Colbourn, C. J., and Lishman, W. A. (1978). Lateralisation of function and psychotic illness: a left hemisphere deficit. In *Proceedings of a Conference on Hemispheric Asymmetries and Psychopathology*. In press.
- Crow, T. J., and Johnstone, E. C. (1977). Cerebral atrophy and cognitive impairment in chronic schizophrenia. *Lancet*, **1**, 357.
- Cutting, J. (1978). Study of anosognosia. *Journal of Neurology, Neurosurgery, and Psychiatry*, **41**, 548–555.
- Flor-Henry, P. (1969). Psychosis and temporal lobe epilepsy: a controlled investigation. *Epilepsia*, **10**, 363–395.
- Flor-Henry, P. (1976). Lateralized temporal-limbic dysfunction and psychopathology. *Annals of the New York Academy of Sciences*, **280**, 777–797.
- Folstein, M. F., Maiberger, R., and McHugh, P. R. (1977). Mood disorder as a specific complication of stroke. *Journal of Neurology, Neurosurgery, and Psychiatry*, **40**, 1018–1020.
- Gainotti, G. (1972). Emotional behavior and hemisphere side of lesion. *Cortex*, **8**, 41–55.
- Gasparrini, W. G., Satz, P., Heilman, K. M., and Coolidge, F. L. (1978). Hemispheric asymmetries of affective processing as determined by the Minnesota Multiphasic Personality Inventory. *Journal of Neurology, Neurosurgery, and Psychiatry*, **41**, 470–473.
- Gruzeliier, J. H. (1973). Bilateral asymmetry of skin conductance orienting activity and levels in schizophrenics. *Biological Psychology*, **1**, 21–41.
- Gur, R. E. (1978). Left hemisphere dysfunction and left hemisphere overactivation in schizophrenia. *Journal of Abnormal Psychology*, **87**, 226–238.
- Johnstone, E. C., Crow, T. J., Frith, C. D., Stevens, M., Kree, L., and Husband, J. (1978). The dementia of dementia praecox. *Acta Psychiatrica Scandinavica*, **57**, 305–324.
- Kendrick, D. (1979). A new version of the Kendrick neuropsychology battery. *British Journal of Social and Clinical Psychology*. In press.
- Koh, S. D. (1978). Remembering in schizophrenia. In *Language and Cognition in Schizophrenia*, pp. 55–59. Edited by S. Schwartz. Lawrence Erlbaum: Hillsdale, New Jersey.
- Kusumo, K. S., and Vaughan, M. (1977). Effects of lithium salts on memory. *British Journal of Psychiatry*, **131**, 453–457.
- Larner, S. (1977). Encoding in senile dementia and elderly depressives. *British Journal of Social and Clinical Psychology*, **16**, 379–390.
- Levy, R., and Maxwell, A. E. (1968). The effect of verbal context on the recall of schizophrenics and other psychiatric patients. *British Journal of Psychiatry*, **114**, 311–316.
- Lewine, R. R. J. (1978). Response complexity and social interaction in the psychophysiological testing of chronic and paranoid schizophrenics. *Psychological Bulletin*, **85**, 284–294.
- Marsden, C. D. (1976). Cerebral atrophy and cognitive impairment in chronic schizophrenia. *Lancet*, **2**, 1079.
- Marshall, W. L. (1973). Cognitive functioning in schizophrenia. *British Journal of Psychiatry*, **123**, 413–433.
- Miller, E., and Lewis, P. (1977). Recognition memory in elderly patients with depression and dementia: a signal detection analysis. *Journal of Abnormal Psychology*, **86**, 84–86.
- Slater, E., Beard, A. W., and Glithero, E. (1963). The schizophrenia-like illnesses of epilepsy. *British Journal of Psychiatry*, **109**, 95–150.
- Small, I. F., Small, J. G., Milstein, V., and Moore, J. E. (1972). Neuropsychological observations with psychosis and somatic treatment. *Journal of Nervous and Mental Disease*, **155**, 6–13.
- Spohn, H. E., Lacoursiere, R. B., Thompson, K., and Coyne, L. (1977). Phenothiazine effects on psychological and psychophysiological dysfunction in chronic schizophrenics. *Archives of General Psychiatry*, **34**, 633–644.
- Strömberg, L. S. (1977). The influence of depression on memory. *Acta Psychiatrica Scandinavica*, **56**, 109–128.
- Whitehead, A. (1974). Factors in the learning deficit of elderly depressives. *British Journal of Social and Clinical Psychology*, **13**, 201–208.
- Winer, B. J. (1971). *Statistical Principles in Experimental Design*, second edition, p. 196. McGraw-Hill: New York.
- Wing, J. K. (1961). A simple and reliable sub-classification of chronic schizophrenia. *Journal of Mental Science*, **107**, 862–875.
- Wing, J. K., and Brown, G. W. (1970). *Institutionalism and Schizophrenia*. Cambridge University Press: Cambridge.
- Wing, J. K., Cooper, J. E., and Sartorius, N. (1974). *Measurement and Classification of Psychiatric Symptoms*. Cambridge University Press: Cambridge.