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EDITORIAL

'Narrow localizationalism' in psychiatric neuropsychology[1]

INTRODUCTION: STRUCTURE AND FUNCTION

Luria (1973) used the term 'narrow localizationism' to refer to a tradition in late nineteenth- and early twentieth-century clinical neurology of using observations of deficits following areal brain injury of various types to map discrete psychological processes and functions onto circumscribed areas of the brain. Luria (1973, 1980) propounded an alternative view of higher brain function: namely, the concept that behavioural functions involve the operation of flexible cerebral *functional systems* for their manifestation. According to this approach, a given act or behaviour is not the result of an invariant connectionistic activation by a particular cerebral locus or centre, but is 'performed by variable (variative) mechanisms bringing the process to a constant (invariant) result' (Luria, 1973, p. 28).

According to this conception, behaviour may be performed, and the solution to problems carried out, by a variety of adaptive cognitive strategies which are, in turn, related to varying and modifiable patterns of dynamic cerebral activity, and which can allow the problem to be approached in different ways, depending on the circumstances and on the constraints on the neurocognitive system. Thus,

> The most significant feature of a functional system is that, as a rule, it is based on a complex dynamic 'constellation' of connections, situated at different levels of the nervous system, that, in the performance of the adaptive task, may be changed with the task itself remaining unchanged. (Luria, 1980, p. 22.)

The use of psychological tests to assess cerebral functioning has had a long tradition in clinical neuropsychology, so that at present there exists a substantial armamentarium of 'neuropsychological tests' which are utilized for both the purpose of research and for clinical assessment (Golden, 1978; Goldstein, 1981; Lezak, 1983; McFie, 1975). However, the problems associated with the use of such psychometric instruments to 'diagnose' the locus and laterality of brain dysfunction in various patient populations is only beginning to be addressed systematically (Goldstein, 1981; Kiernan, 1981).

Some notes of dissatisfaction with the use of such measures have been expressed in the past, although thoughtful attention to these matters has frequently not characterized the approach of many studies in psychiatric neuropsychology, nor has it guided the use of neuropsychological assessment techniques in the routine clinical evaluation setting. Thus, over two decades ago, Smith (1962) expressed his dissatisfaction with the idea of regarding cognitive processes as unitary 'functions', which could then be localized by the use of psychological tests.

> Despite the variety of definitions of the nature of impairment of 'higher' mental functioning, all tests assumed to measure either discrete components and/or the aggregate of such functions – all involve various modalities. (Smith, 1962, p. 317.)

This represents a position very close to that of Luria (1973, 1980), but with specific reference to formal psychological testing, a method that Luria largely eschewed because of its association with narrow localizationist concepts of structure and function (Goldstein, 1981; Luria, 1980; Luria & Majovski, 1977). As problematical as the use of neuropsychological tests to assess locus of pathology may be in the case of brain-injured patients, it is all the more acute in the growing field of psychiatric neuropsychology, where the use of neuropsychological tests and related evaluation techniques with psychiatric patients has led to the development of a neuropsychological typology of psychiatric syndromes, such that different classes of psychopathology have come to be associated with dysfunction in particular brain regions or hemispheres (Flor-Henry, 1976, 1979; Flor-Henry

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et al. 1981; Insel *et al.* 1983; Marin & Tucker, 1981; Taylor *et al.* 1981; Wexler, 1980; Yeudall, 1977). The consequences of such a typology are twofold: first, it biases and guides theoretical model-building in psychiatric neuropsychology, and constrains the empirical questions which direct research in this area; and, secondly, in routine clinical assessment situations it reinforces the practice of thinking of 'impaired' performance on neuropsychological tasks in terms of 'deficits' in cognitive functioning which are then referrable to 'lesions' of various structures or locations in the brain. The problems are, for the most part, similar in both the research and clinical settings.

METHODOLOGICAL ISSUES

It could be argued that much of the inferential hypothesis-building in psychiatric neuropsychology violates the usual rules for the establishment of structure-function relationships, which tend to rely on two basic methodological constructs, as described by Jones (1983). In the method of *double dissociation*: first, membership in subject groups is allocated on the basis of known lesion type and/or lesion location; secondly, group membership is found to predict reciprocal impairments in functional performance; thirdly, it is concluded that particular functions are characteristically sustained at known neural locations. In the method of *reciprocal disability*: first, membership in subject groups is allocated on the basis of reciprocal impairments in functional performance; secondly, the impairments are shown retrospectively to be the consequences of neural damage of certain types and/or in certain locations; thirdly, it is concluded that particular functions are sustained at particular neural locations.

It can be seen that the development of structure-function constructs in psychiatric neuropsychological research tends to follow the first and third steps of the reciprocal disability model: that is, subjects are classed according to differences in performance on neuropsychological tasks, and conclusions are drawn as to the locus of function and dysfunction based on task performance. What is characteristically missing, however, is the crucial second step: impaired performance is, in these cases, *regarded inferentially* as being the result of localized or lateralized brain dysfunction, based on phenotypic similarities in task performance between the psychiatric subjects and individuals with known brain damage. The middle validating step – demonstration of the right lesion in the right location – is typically absent.

Inferences about the diagnostic and localizing value of neuropsychological tests frequently follow the line of reasoning that, if damage to cerebral structure X is known to produce a decline in performance on task T, then any new subject or class or subjects having relatively poor performance on T must have a lesion at X (Miller, 1983). However, the Lurian principle of performance of an invariant task by involvement of variable mechanisms suggests that poor performance on T may relate to factors other than having a lesion at X. This is especially the case in psychiatric applications, where the deficits on testing are rarely of the exact type and degree as that found in structurally impaired neurological patients. More commonly, the patterns of performance deficit produced by various psychiatric patients only approximate that of brain damaged individuals in terms of type or quality. Thus, it is common to hear neuropsychologists speak of 'frontal-looking', or 'right hemisphere-ish' findings in psychiatric patients, with the understanding that 'lesions', in the traditional neurological sense are not the real diagnostic issue. However, it is an entirely different matter to regard a psychiatric syndrome as a 'frontal' or 'right hemisphere' disease, based on findings from neuropsychological tests.

DEFICIT INTERPRETATION IN NEUROLOGICAL AND PSYCHIATRIC SUBJECTS

It would seem plausible to argue that the concept of narrow localizationism is embodied in assertions such as the following:

> [neuropsychological] test procedures developed with brain damaged patients can be exactly duplicated in the investigation of functional psychiatric disorders. As a result, the comparability of response deficits in functional and organic syndromes may be objectively assessed. (Gorenstein, 1982, p. 369.)

Such a statement is all the more disturbing when one realizes that clinicians and research workers

have questioned the localizational constructs underlying cerebral organization in even the most 'organic' of organic brain syndromes (Kiernan, 1981; Mehler *et al.* 1984; Smith, 1962; Tucker, 1981).

Even if we could safely assume that impairment on test *T* implies some sort of genuine dysfunction in a cerebral system which involves structure *X*, and that qualitatively similar relationships can be found with most major psychiatric syndromes of neuropsychological interest, problems remain with regard to the meaning of 'brain damage' as educed from psychometric measures.

For example, diffuse or non-focal cerebral impairment can produce lateralized- or focal-looking test findings, even in subjects with known brain damage. Among tasks found by Spreen & Benton (1965) to be sensitive to impaired performance in cases of diffuse, non-focal cerebral damage were the Wechsler–Bellvue Similarities subtest, the Block Design subtest and a test of visual retention, all measures commonly regarded as reflecting lateralized dysfunction (Lezak, 1983; McFie, 1975).

Russell (1979) found that different types of brain damage produced different patterns of performance on the Wechsler Adult Intelligence Scale (WAIS): (1) a normal pattern; (2) a 'right hemisphere' and diffuse degenerative pattern; and (3) a 'left hemisphere' pattern. The results from the diffuse degenerative group were not significantly different from those of the right hemisphere group. Russell's (1979) patients with left hemisphere damage did not show significantly lower Verbal *v.* Performance IQs, whereas the left hemisphere-damaged patients studied by Bornstein (1983) did obtain significantly lower Verbal IQs. In agreement with Russell (1979), however, Bornstein (1983) found that both right hemisphere and bilateral cerebral injury groups obtained significantly lower Performance IQs. Bornstein (1983) warned against the all-too-common practice of automatically regarding VIQ–PIQ discrepancies as reliable indices of lateralized cerebral dysfunction.

Using the Wisconsin Card Sorting Test (WCST), a well-studied measure believed to be a sensitive index of frontal lobe impairment (Drewe, 1974; Malmo, 1974; Milner, 1963), Robinson *et al.* (1980) found that there were no significant differences in performance between subjects with focal frontal lesions and those with diffuse cerebral impairment, both groups showing deficits on the task. The authors concluded that the WCST may not be helpful in distinguishing focal lobe lesion cases from those involving diffuse generalized cerebral dysfunction.

This finding assumes particular importance in the present context, since the WCST is one of the 'frontal lobe tests' which have been used in attempts to document impaired frontal lobe functioning in psychopaths, as in the study conducted by Gorenstein (1982). In this study, response perseveration (persistence of an ineffectual response strategy in the face of negative feedback) was cited as one of the indices of impaired frontal lobe functioning, based on an analogy with past research on perseverative responding in frontal-lesioned subjects, and despite evidence that perseveration, as a clinical finding and as a feature of structured task performance, is frequently seen as an accompaniment of damage to brain regions other than the frontal lobe and is also found in diffuse cerebral dysfunction of many types and from many sources (Damasio, 1979; Lishman, 1978; Goldberg & Tucker, 1979).

Hare (1984), using similar 'frontal-lobe' measures (including the WCST), was unable to confirm frontal dysfunction in his group of incarcerated psychopaths, suggesting that there may not in fact be anything particularly 'frontal' about this group. He raised the possibility that differences between his and Gorenstein's (1982) findings may relate to the fact that Gorenstein (1982) used psychopaths from a psychiatric population, whereas Hare's (1984) subjects came from a prison population. Nevertheless, given that individuals with antisocial or psychopathic behaviour tendencies are known to display a range of deficits on neuropsychological testing (Brickman *et al.* 1984; Robbins *et al.* 1983; Yeudall, 1977), it is not surprising that many of them may be found to show certain behavioural patterns and response characteristics (e.g. perseveration) which are qualitatively similar to those seen in individuals with organic brain damage (e.g. frontal lobe). However, to assert that, because test findings in psychopaths are similar to test findings in frontal lobe patients, the former have brain dysfunction focally equivalent to the latter, is an example of the type of narrow localizationist syllogism that serves little constructive purpose in psychiatric neuropsychology.

A similar issue arises in the efforts to make attributions to hemispheric laterality of cerebral dysfunction in schizophrenia, based on neuropsychological test findings. Much of the literature on

neuropsychological impairment in this syndrome has implicated left hemisphere dysfunction as underlying the cognitive deficits educed from test results (Flor-Henry, 1976, 1979; Goldstein, 1978; Marin & Tucker, 1981; Seidman, 1983). This is frequently because performance on verbal-linguistic measures ('left hemisphere tests') is found to be worse than that on visuospatial tasks ('right hemisphere tests'). In individual assessment situations, however, clinicians do find patterns of neuropsychological performance in schizophrenics which appear to be 'atypical' from the perspective of the consensus of laterality research. For example, West (1984) reported that a sample of schizophrenics with formal thought disorder displayed particularly poor performance on a puzzle task sensitive to visual organization deficits that the author believed to reflect impaired right hemisphere functioning. In fact, the schizophrenics' performance on this task was as impaired as that of a sample of neurological patients with confirmed right hemisphere lesions. Miller (1984*b*) described clinical observations of severely impaired performance by many individual schizophrenic patients on such visuospatial (and thus supposedly 'right hemisphere') tasks as the WAIS-R Block Design and Object Assembly subtests (Wechsler, 1981) and the Benton Visual Retention Test (Benton, 1974), as compared with comparatively better performance on verbal-linguistic ('left hemisphere') measures. Aylward *et al.* (1984), reviewing the literature on intelligence and intelligence testing in schizophrenia, concluded that 'the consistent tendency for schizophrenic patients to manifest higher VIQs than PIQs would seem to refute the analogy drawn between schizophrenic patients and patients with left hemisphere lesions' (p. 455). Thus, the data at this point are hardly conclusive in establishing schizophrenia as a 'left hemisphere' disease.

TASK ANALYSIS AND PROCESS MODELS

Miller (1984*b*) has suggested that, instead of using lateralized test findings to argue for this or that hemisphere as being the primary 'locus' of dysfunction in schizophrenia, clinicians and research workers should take a closer look at the component task demands of the tests being used to measure particular 'functions'. For example, visuospatial problem-solving deficits in schizophrenic patients need not automatically imply a 'lesion' in the right-hemisphere substrate for perceptual functioning in general; rather, the novelty and complexity of such tasks may so tax a disordered neurocognitive system as to preclude the use of overlearned and familiar verbal cuing strategies which are normally utilized in the solution of most multimodal tasks, even tasks with a more ostensibly 'visuospatial' component. Subjects will frequently be observed to autoarticulate solution strategies to supposedly 'non-verbal' tasks and problems; the uniquely human facility with language enables most normal subjects to apply a variety of solution strategies to particular tasks by means of a delicate and varied intercognitive network of associations (Lenneberg, 1967; Luria, 1973, 1980; Vygotsky, 1962).

In fact, those enamoured of a narrow localizationist (or lateralizationist) point of view could just as easily argue that, based on the above analysis, schizophrenia is indeed a 'left hemisphere' disease, since the problems in solving complex visuospatial tasks can now be interpreted as involving an autoverbalization deficit, which is by definition a verbal disorder, which implies left hemisphere dysfunction, which puts schizophrenia 'back' in the left hemisphere, and so on.

The point is that schizophrenia, or psychopathy, or any other psychiatric syndrome or symptom, is not likely to 'live' in any particular hemisphere or lobe. Simply reasoning by analogy from the test results of brain-injured subjects is not likely to aid in the specification of the cerebral bases for these disorders (Miller, 1984*a*, 1985) as long as we ignore the necessity of pursuing the type of process analyses which will help us to analyse the various task demand components of our measures, as well as the types of cognitive processes and solution strategies which are involved in the performance of these tasks (Kiernan, 1981; Luria, 1973, 1980).

This issue is not unique to psychiatric neuropsychology, nor is it new. Teuber & Weinstein (1956) observed that aphasic patients showed significantly greater impairment than non-aphasic patients on a visuospatial task involving perception of a hidden figure. At variance with traditional localizationist models, this finding was interpreted as a non-specific or general mental defect which occurs in some obligatory association with more localized or specific deficits. From our present

perspective, the issue is remarkably close to that of verbal mediation of visuospatial problem-solving in schizophrenics. Both Teuber & Weinstein's (1956) aphasics and West's (1984) thought-disordered schizophrenics may have performed particularly poorly on their respective visuoperceptual tasks because both lacked some crucial cognitive-linguistic faculty which facilitates problem-solving of various types. However, because language disorders usually occur with left hemisphere damage, this does not automatically mean that schizophrenia is a left hemisphere disease. Yes, the left hemisphere, or portions of it, may be involved in the cognitive impairments which characterize schizophrenic thought disorder, just as psychopathic behaviour may involve frontal lobe interactions with other cortical and subcortical systems that mediate appropriateness of response and sensitivity to modifying feedback (Ackerly, 1964; Damasio, 1979; Stuss & Benson, 1984). But here we are beginning to talk about the operation of functional systems, not the 'loci' of narrow localizationism.

Over two decades ago, Smith (1962) concluded:

> The use of...psychological tests in clinical and experimental neurological studies of brain damage has emphasized a growing appreciation for the need to develop new contexts for studying and defining mental functions and brain mechanisms. (p. 321.)

We still await the realization of these 'new contexts', but a beginning is being made in efforts to pursue the development of *process models* (Kiernan, 1981). Such an approach involves an analysis of the task demands inherent in different tests of cognitive functioning, as well as examinations of the various and complex problem-solving modalities which individuals use to negotiate the demands of what may superficially appear to be simple or unidimensional tasks (Ben-Yishay *et al.* 1970; Bond & Bechtel, 1984; Grant & Berg, 1948; Hadano, 1984; Schorr *et al.* 1982). The use of ancillary techniques – such as CAT and PET scans, or evoked potentials – to examine brain functioning in psychopathological groups will undoubtedly tell us much about structure and function in a *physiological sense*, but caution must always be exercised in making conceptual leaps to conclusions about *psychological* functioning.

> It is a mistake to assume that increased neuroanatomic precision alone will permit us to localize more specifically such complex functions. We will always be limited by the conceptual system that is least understood. It is fundamental to research in neuropsychology that we cannot localize functions that we do not understand. (Kiernan, 1981, p. 346.)

By looking closely at the task demands of the neuropsychological measures we employ, we may begin to appreciate the rich neurocognitive dynamics that human problem-solving invariably involves, both in its normal and pathological modes of operation. In a related manner, moving towards a conceptualization of the brain as a three-dimensional functionally interactive complex of systems will foster the development of cerebral models possessed of commensurate richness, flexibility and complexity. Psychiatric neuropsychology – indeed, neuropsychology as a whole – is now in a position to shed non-productive notions of narrow localizationism and to pursue a course of clinical and experimental investigation and model-building that does appropriate justice to both dimensions of the mind–body continuum.

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REFERENCES

Ackerly, S. S. (1978). A case of paranatal frontal lobe deficit observed for thirty years. In *The Frontal Granular Cortex and Behavior* (ed. J. M. Warren and K. Akert), pp. 192–218. McGraw-Hill: New York.

Aylward, E., Walker, E. & Bettes, B. (1984). Intelligence in schizophrenia: meta-analysis of the research. *Schizophrenia Bulletin* **10**, 430–459.

Benton, A. L. (1974). *Revised Visual Retention Test: Clinical and Experimental Applications* (4th edn). Psychological Corporation: New York.

Ben-Yishay, Y., Diller, L., Gerstman, L. & Gordon, W. (1970). Relationship between initial competence and ability to profit from cues in brain damage individuals. *Journal of Abnormal Psychology* **75**, 248–259.

Bond, J. & Bechtel, H. A. (1984). Comparison of the Wisconsin Card Sorting Test and the Halstead Category Test. *Journal of Clinical Psychology* **40**, 1251–1255.

Bornstein, R. A. (1983). Verbal IQ–Performance IQ discrepancies on the Wechsler Adult Intelligence Test-Revised in patients with unilateral or bilateral cerebral dysfunction. *Journal of Consulting and Clinical Psychology* **51**, 779–780.

Brickman, A. S., McManus, M., Grapentine, W. L. & Alessi, N.

(1984). Neuropsychological assessment of seriously delinquent adolescents. *Journal of the American Academy of Child Psychiatry* **23**, 453–457.

Damasio, A. (1979). The frontal lobes. In *Clinical Neuropsychology* (ed. K. M. Heilman and E. Valenstein), pp. 360–412. Oxford University Press: New York.

Drewe, E. A. (1974). The effect of type and area of brain lesion on Wisconsin Card Sorting Test performance. *Cortex* **10**, 159–170.

Flor-Henry, P. (1976). Lateral temporal-limbic dysfunction and psychopathology. *Annals of the New York Academy of Sciences* **280**, 777–795.

Flor-Henry, P. (1979). On certain aspects of the localization of the cerebral systems determining emotion. *Biological Psychiatry* **14**, 677–698.

Flor-Henry, P., Fromm-Auch, D., Tapper, M. & Shopflocher, D. (1981). A neuropsychological study of the stable syndrome of hysteria. *Biological Psychiatry* **16**, 601–625.

Goldberg, E. & Tucker, D. (1979). Motor perseveration and long-term memory for visual forms. *Journal of Clinical Neuropsychology* **1**, 273–288.

Golden, C. J. (1978). *Diagnosis and Rehabilitation in Clinical Neuropsychology*. Charles C. Thomas: Springfield, Ill.

Goldstein, G. (1978). Cognitive and perceptual differences between schizophrenics and organics. *Schizophrenia Bulletin* **4**, 160–185.

Goldstein, G. (1981). Some recent developments in clinical neuropsychology. *Clinical Psychology Review* **1**, 245–268.

Gorenstein, E. E. (1982). Frontal lobe functioning in psychopaths. *Journal of Abnormal Psychology* **91**, 368–379.

Grant, D. A. & Berg, E. A. (1948). A behavioral analysis of degree of reinforcement and ease of shifting to new responses in a Weigl-type card sorting problem. *Journal of Experimental Psychology* **38**, 404–411.

Hadano, K. (1984). On Block Design constructional disability in right and left hemisphere brain-damaged patients. *Cortex* **20**, 391–401.

Hare, R. D. (1984). Performance of psychopaths on cognitive tasks related to frontal lobe funtion. *Journal of Abnormal Psychology* **93**, 133–140.

Insel, T. R., Donnelly, E. F., Lalekea, M. L., Alterman, I. S. & Murphy, D. L. (1983). Neurological and neuropsychological studies of patients with obsessive-compulsive disorder. *Biological Psychiatry* **7**, 741–751.

Jones, G. V. (1983). On double dissociation of function. *Neuropsychologia* **21**, 397–400.

Kiernan, R. J. (1981). Localization of function: the mind–body problem revisited. *Journal of Clinical Neuropsychology* **3**, 345–352.

Lenneberg, E. H. (1967). *Biological Foundations of Language* Wiley: New York.

Lezak, M. (1983). *Neuropsychological Assessment* (2nd edn). Oxford University Press: New York.

Lishman, W. A. (1978). *Organic Psychiatry· The Psychological Consequences of Cerebral Disorder*. Blackwell: Oxford.

Luria, A. R. (1973). *The Working Brain*. Basic Books: New York.

Luria, A. R. (1980). *Higher Cortical Functions in Man* (2nd edn). Basic Books: New York.

Luria, A. R. & Majovski, L. V. (1977). Basic approaches used in American and Soviet clinical neuropsychology. *American Psychologist* **32**, 959–968.

Malmo, H. P. (1974). On frontal lobe functions: psychiatric patient controls. *Cortex* **10**, 231–237.

Marin, R. S. & Tucker, G. J. (1981). Psychopathology and hemisphere dysfunction: a review. *Journal of Nervous and Mental Disease* **169**, 546–554.

McFie, J. (1975). *Assessment of Organic Intellectual Impairment*. Academic Press: New York.

Mehler, J., Morton, J. & Jusczyk, P. W. (1984). On reducing language to biology. *Cognitive Neuropsychology* **1**, 83–116.

Miller, E. (1983). A note on the interpretation of data derived from neuropsychological tests. *Cortex* **19**, 131–132.

Miller, L. (1984*a*). Neuropsychological concepts of somatoform disorders. *International Journal of Psychiatry in Medicine* **14**, 31–46.

Miller, L. (1984*b*). Hemispheric asymmetry of cognitive processing in schizophrenics. *Psychological Reports* **55**, 932–934.

Miller, L. (1985). Neuropsychological assessment of substance abusers: review and recommendations. *Journal of Substance Abuse Treatment* **2**, 5–17.

Milner, B. (1963). Effects of different brain lesions on card sorting: the role of the frontal lobes. *Archives of Neurology* **9**, 100–110.

Robbins, D. M., Beck, J. C., Pries, R., Jacobs, D. & Smith, C. (1983). Learning disability and neuropsychological impairment in adjudicated, unincarcerated male delinquents. *Journal of the American Academy of Child Psychiatry* **22**, 40–46.

Robinson, A. L., Heaton, R. K., Lehman, R. A. W. & Stilson, D. W. (1980). The utility of the Wisconsin Card Sorting Test in detecting and localizing frontal lobe lesions. *Journal of Consulting and Clinical Psychology* **48**, 605–614.

Russell, E. W. (1979). Three patterns of brain damage on the WAIS. *Journal of Clinical Psychology* **35**, 611–620.

Schorr, D., Bower, G H. & Kiernan, R. (1982). Stimulus variables in the Block Design task. *Journal of Consulting and Clinical Psychology* **50**, 479–487.

Seidman, L. J. (1983). Schizophrenia and brain dysfunction: an integration of recent neurodiagnostic findings. *Psychological Bulletin* **94**, 195–238.

Smith, A. (1962). Ambiguities in concepts and studies of 'brain damage' and 'organicity'. *Journal of Nervous and Mental Disease* **135**, 311–326.

Spreen, O. & Benton, A. L. (1965). Comparative studies of some psychological tests for cerebral damage. *Journal of Nervous and Mental Disease* **140**, 323–333.

Stuss, D. T. & Benson, D. F. (1984). Neuropsychological studies of the frontal lobes. *Psychological Bulletin* **95**, 3–28.

Taylor, M. A., Redfield, J. & Abrams, R. (1981). Neuropsychological dysfunction in schizophrenia and affective disease. *Biological Psychiatry* **16**, 467–478.

Teuber, H. L. & Weinstein, S. (1956). Ability to discover hidden figures after cerebral lesions. *Archives of Neurology and Psychiatry* **76**, 369–379.

Tucker, D. M. (1981). Lateral brain function, emotion and conceptualization. *Psychological Bulletin* **89**, 19–46.

Vygotsky, L. S. (1962). *Thought and Language*. MIT Press: Cambridge, Mass.

Wechsler, D. (1981). *Wechsler Adult Intelligence Scale-Revised: Manual*. Psychological Corporation: New York.

West, E. D. (1984). Right hemisphere dysfunction in schizophrenia. *Lancet* i, 344.

Wexler, B. E. (1980). Cerebral laterality and psychiatry: a review of the literature. *American Journal of Psychiatry* **137**, 279–291.

Yeudall, L. T. (1977). Neuropsychological assessment of forensic disorders. *Canadian Mental Health* **25**, 7–15.