The Neuropsychology of Schizophrenia

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From being an area primarily on the periphery of mainstream behavioural and cognitive science, neuropsychology has developed in recent years into an area of central concern for a range of disciplines. We are witnessing not only a revolution in the way in which brain–behaviour–cognition relationships are viewed, but a widening of interest concerning developments in neuropsychology on the part of a range of workers in a variety of fields, Major advances in brain-imaging techniques and the cognitive modelling of the impairments following brain damage promise a wider understanding of the nature of the representation of cognition and behaviour in the damaged and undamaged brain.

Neuropsychology is now centrally important for those working with brain-damaged people, but the very rate of expansion in the area makes it difficult to keep up with findings from current research. The aim of the Brain Damage, Behaviour and Cognition series is to publish a wide range of books which present comprehensive and up-to-date overviews of current developments in specific areas of interest.

These books will be of particular interest to those working with the brain-damaged. It is the editors' intention that undergraduates, postgraduates, clinicians and researchers in psychology, speech pathology and medicine will find this series a useful source of information on important current developments. The authors and editors of the books in this series are experts in their respective fields, working at the forefront of contemporary research. They have produced texts which are accessible and scholarly. We thank them for their contribution and their hard work in fulfilling the aims of the series.

CC and DJM
Sydney, Australia and Ipswich, UK
Series Editors
Conference speakers and discussants  


Front Row, left to right: R. Bentall, C. Frith, P. Liddle, R. Hoffman, J. Cutting, A. David, D. Rogers, S. Lewis.
Foreword

Early in 1991, we had the idea for a book on the neuropsychology of schizophrenia, for which there seemed to be an obvious need. The trouble with such ideas is knowing whether they are shared by colleagues and peers. We decided that the best way to test this was to organise an international symposium on the topic at the Institute of Psychiatry in London. This took place on the 10th and 11th October of that year. The response from invited speakers was extremely encouraging, as was the meeting’s attendance. The book was the natural next step. However, books of conference proceedings litter the shelves of libraries and offices, standing in testament to wasted human endeavour, not to mention wasted natural resources. We were determined that this should not be just “the book of the conference”; the publishers agreed. To ensure this, contributors were chosen because of their eminence in the field as well as for their skill as communicators. Their brief was wide, in that we asked for detailed reviews, empirical data, and theoretical speculation, all in the same chapter! The symposium served to focus the minds of the contributors into producing early drafts, which were precirculated and later mulled over in the light of discussions during and after the symposium. Eventually, completed chapters emerged, which we believe cover the breadth and depth of this exciting hybrid discipline. We trust their efforts were worthwhile.

The meeting and hence this book would not have been possible without the generous support of The Wellcome Trust. Lundbeck Pharmaceuticals and Sandoz (UK) also provided sponsorship, for which we are grateful.
Finally we thank the staff of the Institute of Psychiatry, in particular Mrs Lee Wilding for her administrative help.

ASD and JC
October 1992
The idea that there is a “neuropsychology of schizophrenia” is of relatively recent origin. Indeed, the term “neuropsychology” itself only dates back three or four decades. The concept to which it refers, however, is far from new.

The thrust of Kraepelin’s and most of his fin de siècle German contemporaries’ research into schizophrenia was towards elucidating the biological basis of the condition, which included questions such as whether particular parts of the brain were damaged more than others. The spate of higher-order neurological deficits uncovered in the last half of the nineteenth century—aphasia, agnosia, apraxia, alexia, etc.—led many psychiatrists to suppose that schizophrenia, like these other conditions, would turn out to have a specific link with some damaged brain site, e.g. Broca’s aphasia with a lesion in the left second and third frontal gyri. A minority of more neurologically-orientated physicians (e.g. Wernicke and Kleist) did, in fact, develop just such a detailed system (without any real neurobiological evidence, however). Kraepelin, although committed to this approach, was rigorous enough to realise that he could not as yet justify such schemes.

Bleuler’s incorporation of Freud’s and Jung’s psychodynamic explanations of psychiatric phenomena paved the way for a multitude of non-neuropsychological approaches—psychodynamic, social, existential, behavioural, and cognitive models—which persisted as the mainstream of thinking for over 70 years. All these merely adopted some current
psychological model of mind (or non-mind in the case of behavioural) and schizophrenia was slotted into these in the most plausible way possible.

As late as the early 1980s, when one of us was contemplating a book on the matter, the title *Neuropsychology of Schizophrenia* was suggested to a publisher, who was enthusiastic about the title as anything to do with the brain was becoming a selling-point in the United States. By the time the literature had been reviewed, however, it was clear that, apart from Kraepelin's, Wernicke's, and Kleist's modest beginnings, there was no corpus of work on the neuropsychology of schizophrenia, and the name was changed to *The Psychology of Schizophrenia* (Cutting, 1985). Ten years on, the situation is radically different. All the chapters in this book reflect this to a greater or lesser degree. Even those contributors who are still promoting what are essentially "preneuropsychological era" models, feel obliged to give their views some neuropsychological relevance.

Is there a common definition of the neuropsychology of schizophrenia? As already mentioned, neuropsychology *per se* is a new term, although its origin may have had psychiatric connections. Perhaps the first use of the word was in 1913, when Sir William Osler spoke at the opening of the Henry Phipps Psychiatric Clinic of the Johns Hopkins Hospital, Baltimore (Bruce, 1985). This was just 2 years after Bleuler's description of *The Schizophrenias* (Bleuler, 1911-1950). The second landmark was the appointment of Karl Lashley as Research Professor of Neuropsychology at Harvard in 1937. However, Donald Hebb's book, *The Organisation of Behaviour: a Neuropsychological Theory*, published in 1949, finally established neuropsychology on the intellectual map. Coming more up to date, the first journal devoted to the topic was launched in 1963—*Neuropsychologia*, edited by Henry Hecaen.

One way to avoid the arbitrariness of definitions is to look at current usage. Unfortunately this takes us even further from consensus. A recent target article in the journal *Behavioural and Brain Sciences* by Gray et al. (1991) carried the same title of this book, and Hemsley (Chapter 6) summarises many of the underlying theoretical points made in that article. For Gray and colleagues, neuropsychology is any study of behaviour in relation to the hardware of the brain (see also Robbins, 1990). In this case the brain refers to any brain, including that of the rat. The "neuro" prefix comes in because of experimental manipulation of that organ, either through lesioning or pharmacological agents. The limitations and advantages of inferences about human behaviour derived from animal work have been much debated over the years. Clearly they have an important place in providing the foundation to human neuropsychological theories.

Another usage is the application of psychometric tests derived from studies on populations with known brain lesions, to psychiatric patients. This is perhaps the most traditional form of psychiatric neuropsychology.
It can be subdivided into the use of broad test batteries, which show patterns of deficits in, for example, visual–spatial or linguistic skills, which in turn may be interpreted in terms of right and left hemisphere problems (e.g. Flor-Henry & Gruzelier, 1983; see also Liddle, Chapter 3), and the use of more specific, custom-built tests intended to examine in detail a single psychological function or brain area, e.g. the use of the Tower of London and Wisconsin Card Sort test (WCST) (Shallice et al., 1991), or chimeric faces, a right posterior hemisphere test, (David & Cutting, 1990; see also Ellis and de Pauw, Chapter 18; Pantelis and Nelson, Chapter 13; Cutting, Chapter 14). This approach is, in essence, argument by analogy; the analogy may hold at a number of levels. For instance, at the behavioural level, the apathy and amotivation of the frontal-lobe-patient may resemble that of the chronic schizophrenic, as well as at the neuropsychological level, when there may be frequent perseverative errors on the WCST in both patient groups. Dunkley and Rogers (Chapter 11) go as far as to say that dementia praecox is as much a dementia as that of the Alzheimer type.

There is no reason why the neuropsychologist should be wedded to psychiatric nosology and indeed this may lead research into blind alleys. O'Carroll (1992) in a recent review showed how neuropsychology could act at different clinical levels from the diagnostic, e.g. schizophrenia (Dunkley and Rogers, Chapter 11; Pantelis and Nelson, Chapter 13), through the syndromic or subsyndromic, e.g. positive syndrome (Hemsley, Chapter 6), negative syndrome (Frith, Chapter 9), the “reality distortion” of Liddle (Chapter 3) to the symptom level, e.g. delusions (Bentall, Chapter 19), delusional misidentification (Ellis and de Pauw, Chapter 19), and hallucinations (Slade, Chapter 15; David, Chapter 17).

Fortunately, advances in neuroscience and neuroimaging have allowed these analogies, particularly with respect to brain localisation, to be tested. The structure of the brain region of interest may be examined with the detailed morphometry of the psychoneuropathologist or through CT and magnetic resonance brain imaging. Similarly, functional imaging utilising metabolism or cerebral blood flow (Early et al., Chapter 2; Liddle, Chapter 3) may serve not only to test the pattern of activation at rest or in response to a cognitive challenge (Weinberger et al., 1986; see also Nuechterlein et al., Chapter 4; Fleming et al., Chapter 12), but also the assumptions about the regional specificity of the test in question, based on patient data and crude clinicopathological correlations, which often turn out to be spurious. Also, the power of pharmacological agents to modify and produce abnormal psychological phenomena requires localisation in terms of neurotransmitter sites as well as functionally specific anatomical regions (Early et al., Chapter 2).

Early and colleagues report two separate findings in non-medicated schizophrenics: (1) an increase in blood flow (relative to the rest of the brain)
in a subcortical region on the left; and (2) a tendency to ignore stimuli presented in the right visual field. Although this is in support of some lateralized deficit—neurobiological and psychological—in schizophrenia, it is somewhat idiosyncratic. The neurobiological finding is in line with several studies showing left hemisphere overactivity and right hemisphere underactivity in the condition. The psychological finding is curious, however, because visual neglect in subjects with focal lateralised brain damage is virtually always restricted to the left visual field, unless there is anomalous lateralisation of the search control mechanism. Moreover, right hemisphere damage also impairs search in the right visual field more than does left hemisphere damage in that (right) field.

Liddle (Chapter 3) reports different patterns of blood flow in different subtypes of schizophrenia. Patients with predominantly poverty of speech, flat affect, and decreased spontaneous movement exhibited reduced left-sided dorsolateral frontal perfusion. Patients with predominantly thought disorder and inappropriate affect exhibited decreased right-sided ventral frontal perfusion. One patient with catatonia resembled the former group.

It is not surprising that different symptom clusters in schizophrenia are associated with different blood flow patterns. Different mental activities provoke different blood flow patterns in normals, of course. Liddle's results, however, are intriguing because they go some way towards mapping the cerebral dysfunction in different subtypes, though whether they reflect an end-stage cerebral dysfunction or some primary problem is not clear. The patients were all medicated.

As mentioned earlier, neuropsychology owes its distinctiveness from the rest of psychology by virtue of its reference to the brain. However, this has led to an obsession with brain localisation. While the location of schizophrenic disturbances to a cortical area would be a valuable achievement, the story would not end there. In fact this would provide but one link in a complete neuropsychology of schizophrenia chain, which would stretch from neural ultrastructure and chemical transmission to the most abstract of phenomenal experiences. Neuropsychology can justifiably regard as its legitimate domain the intervening steps between the abstract levels of representation—hallucinations, delusions, etc. and “pure” psychological phenomena, e.g. memory and attention. No direct reference may be made to the brain provided two underlying assumptions are held, namely, that these psychological processes may become distorted by dysfunction at a lower level—in the brain—and that schizophrenia is the manifestation of just such a distortion. In other words, cognitive psychology may be applied usefully to the study of schizophrenia but some reference to the biological underpinnings to cognition must be made eventually (Fleminger, Chapter 20; Bentall, Chapter 19; Slade, Chapter 15). This is the chief difference between the work done in the 1980s and earlier work.
Much of the earlier endeavour may be described as "preneuropsychological" only insofar as neural bases for such phenomena as described by Green and Nuechterlein (Chapter 5) have yet to be elucidated fully. Green and Nuechterlein report abnormalities in processing visual information in schizophrenia, measured by a technique known as "backward masking", in which two stimuli are presented for recognition, at different interstimulus intervals, with the purpose of evaluating the potency of the "sensory register" or "icon"; manics were equally impaired. This study illustrates the typical "preneuropsychology of schizophrenia methodology" amongst psychological studies on schizophrenia, and is included as an excellent example of its kind. Green and Nuechterlein attempt a bold integration of their findings (personal communication). They suggest that the interruption of information from a target involves the interaction of transient and sustained visual channels, which do not appear to interact before the level of the primary visual cortical area. The deficit observed may be an example of a general sensory gating deficit, which involves the failure to attenuate the second of two paired stimuli (in this case the mask). Gating is believed to involve mesolimbic dopamine pathways in the brain.

McKenna and colleagues (Chapter 10) adopt the traditional approach to the psychology of schizophrenia: examining one mental function—memory—and comparing its potency in schizophrenics and normals. What gives this chapter a neuropsychological slant is the choice of memory, as this is conventionally regarded as relatively intact in schizophrenics, but impaired if brain damage is present. McKenna et al. find gross memory deficits in their schizophrenics, especially in the realm of semantic memory. This has less in common with the everyday concept of memory but refers to a store of knowledge about the world. McKenna et al. speculate that access to this store may be unreliable, giving rise not only to deficit but also to "productive" symptoms such as illogicality and delusions. This has some resemblance to Cutting's theory regarding impaired real-world knowledge and schizophrenia. Cutting (Chapter 14) puts forward a strong case for this to be the result of right hemisphere dysfunction, having first outlined the ground rules for such an inference (see also Cutting, 1990).

Fleming et al. (Chapter 12) propose that the memory deficit in schizophrenia is a result of some higher-order control-mechanism disorder, which is itself a consequence of frontal lobe dysfunction. This chapter conforms to the newer neuropsychological approach of trying to relate some psychological deficit in schizophrenia to a comparable psychological deficit in subjects with known brain disease. It is theoretical, but draws on data from such diverse sources as twin studies and cerebral blood flow conducted at the United States' National Institute of Mental Health.

Pantelis and Nelson (Chapter 13) set out to examine theoretical formulations, such as those of Fleming et al., by administering recognised
frontal lobe tests to schizophrenics. The authors find evidence for frontal lobe dysfunction in schizophrenia but extend this anatomical localisation to subcortical connections. The “frontal lobe hypothesis” of schizophrenia is fashionable at the moment and, although not all neurobiological procedures confirm it, certainly has to be taken seriously on the evidence of results such as these.

In the case of Frith’s work on the theory of mind (Chapter 9), there has been very little time to examine brain–cognition relationships in detail. Frith approaches the problem of the psychological deficit in schizophrenia from two relatively novel angles. The first relies on new findings on the nature of cognitive development in infants. The second incorporates the tendency in experimental psychological research to talk in terms of metapsychological entities, i.e. to go beyond the base mental functions (attention, perception, memory, etc.) and examine how the person’s whole mind tackles complex problems, including how it monitors and evaluates its own activity. The chapter is a theoretical discussion of how different levels of metapsychological development/maldevelopment might give rise to specific schizophrenic symptoms. The discussion does have a strong neuropsychological element, in that putative abnormalities in schizophrenia are related to functions of various parts of the brain (see also Frith & Done, 1988). In any event, psychological “facts”, be they metapsychological or whatever, will remain so in spite of advances in the neurosciences. In this sense they are not reducible (Churchland, 1988). Nevertheless, the two-way flow of information between neurological and psychological sciences, whose interface is neuropsychology, can only serve to illuminate the study of schizophrenia and other enigmas. Exemplifying this is the work contained in Nuechterlein et al.’s chapter (Chapter 4). Nuechterlein and co-workers studied PET scans and EEG coherence, during an attention task—the continuous performance test—known to be sensitive to right hemisphere activity, and found impairment in schizophrenia. These results are consistent with other neurobiological and psychological studies implicating right hemisphere dysfunction in schizophrenia, although prefrontal areas may also be implicated.

One study which is in some way an exception is that by Bentall (Chapter 19), who presents a coherent account of the nature and cognitive underpinnings of paranoid delusions. What is distinctive about this chapter is that it underplays the role of brain dysfunction or even abnormal perceptual experiences. Instead, Bentall portrays delusions as extensions of normal beliefs and this is backed up by experimental evidence from patients. Like Fleminger’s chapter (Chapter 20), Bentall’s is complementary to the entirely brain-based accounts contained elsewhere in this book.

This discussion leads on to another hybrid label: cognitive neuropsychology. This is a new branch of psychology as well as a very old one (see Ellis &
1. INTRODUCTION

Young, 1988; McCarthy & Warrington, 1990). Here the aim of study is to elucidate the cognitive subcomponents of psychological processes. The existence of these subcomponents or abstract "modules" (Fodor, 1983), is inferred from their absence in cases of focal brain damage or disease. The study of patients with cerebral lesions is therefore only a means to an end. Can this approach be tailored to psychiatric disorders? Ellis and de Pauw (Chapter 18) provide a rationale concerning the origin of Capgras syndrome, one form of delusional misidentification, and David (Chapter 17) outlines the sort of models of normal cognition that might become distorted to produce key psychological symptoms such as auditory hallucinations. Ellis and de Pauw provide us with a comprehensive and critical review, finally espousing what the authors refer to as cognitive neuropsychiatry. It is predicted that such an approach may be seen more often in the future (David, 1993).

The levels of explanation and investigation, as well as the parallels between the traditional subject matter of neuropsychology and that of psychopathology, are shown in Table 1.1. The example of a cerebrovascular accident affecting the left hemisphere is compared with schizophrenia.

Neuropsychology is not only defined by its subject matter, or indeed by its aims. It has as its hallmark a methodology, part statistical technique, part theory, and part experimental strategy. This is the dissociation of function or, more distinct still, the double dissociation of function (Teuber, 1955). This may be applied to a single case or to a series. It describes a pattern of results in which one task is impaired, usually markedly so, in relation to another or, in the case of a double dissociation, there are at least two subjects, one of whom has a dissociation of function in one direction and the other has it in the other direction. This notion has been analysed in detail and refined (Shallice, 1988) but remains a potent force behind neuropsychological research. Dissociations, be they single or double, are rarely described overtly in schizophrenia research yet as leading commentators

### TABLE 1.1

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>Pathology</th>
<th>Brain localisation</th>
<th>Clinical syndrome</th>
<th>Phenomenology</th>
<th>Cognition</th>
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<tbody>
<tr>
<td>Neurology</td>
<td>Genetic Smoking HBP, etc.</td>
<td>Ischaemia/infarction</td>
<td>e.g. left hemisphere</td>
<td>&quot;Stroke&quot; Aphasias Alexia</td>
<td>e.g. optic aphasia Phonological dyslexia</td>
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The example of a cerebrovascular accident affecting the left hemisphere is compared with schizophrenia.
argued some two decades ago, much research founders on this issue. Chapman and Chapman (1973) described what has become known as the “differential deficit problem” in such research. They argued that what may be taken for a dissociation may in fact be an artifact of a failure of the two tasks in question to be matched accurately on such factors as reliability and, especially, difficulty. As schizophrenics are liable to do badly on most tasks for a variety of theoretically trivial reasons, the experimenter must convince the sceptic that the bad performance is specific to a particular task, justified and predicted \textit{a priori}. The sceptic will be persuaded only by a clear demonstration of adequate or good performance on a well-matched control task, or better still, if performance is superior on the control task, in relation to a comparison group, a version of the double dissociation (see Hemsley, Chapter 6).

Hemsley reports a study using a classic behavioural technique derived from animal research. The technique is known as “latent inhibition” and tests a subject’s ability to inhibit a conditioned response to two stimuli. Schizophrenics were less likely than normals to be influenced by the earlier conditioning, and achieved an association between one of the stimuli and a new stimulus \textit{more readily}. Hemsley argues that this behaviour, i.e. being less influenced by previous associations between events in a subject’s current behaviour, is a core problem in schizophrenia and explains most of the complex features of the condition. Although this is not primarily a neuropsychological approach, the search for dissociations is. Hemsley also invokes some speculation concerning the involvement of the left hemisphere in the task he employs.

Encompassed in this book are some novel approaches to the study of schizophrenia; these include research from a neurodevelopmental framework. The observation that disturbances in cognitive and motor function may precede the onset of schizophrenia has given much impetus to new theories concerning both aetiology and pathogenesis (Jones et al., Chapter 8; Walker, Chapter 7). Walker relies on one of the harder pieces of evidence for pinpointing the neuropsychological substrate of schizophrenia—lateralised motor pathology in children who subsequently become schizophrenic. She also uses one of the most innovative and direct techniques of all the contributors here. She obtained “home movies” of the children of families in whom one child subsequently became schizophrenic. Comparing developmental abnormalities in the schizophrenic-to-be child with its normal-to-be sibling the most striking difference was an increased incidence of left-body side abnormalities, i.e. emanating from right hemisphere dysfunction. This study is outstanding, for its ingenuity and for its combining neuropsychological and developmental research principles.

Jones and colleagues examined the intelligence quotients of subjects who eventually become schizophrenic, comparing these with those eventually
developing affective disorder. The preschizophrenic subjects had lower intelligence quotients than the preaffectives. They concluded that this represented an earlier onset of the condition in schizoprenia as opposed to affective psychosis. Jones and colleagues take overall intellectual performance as evidence of brain ability, and note deficits in schizophrenia vis-à-vis controls, especially males, the gender prone to "developmental" disorders.

These apparent continuities and discontinuities between childhood and adult psychopathology force a third question on to the neuropsychological research agenda. Not only must we find out what happened to the brain and where, but also when. The temporal dimension does not end with the onset of the disorder. Whether or not the lesion or lesions responsible for schizophrenia are static, the clinical state appears to change with time (Dunkley and Rogers, Chapter 11; Pantelis and Nelson, Chapter 13). Dunkley and Rogers examine the Kraepelinian notion that schizophrenics do indeed have a dementia, not explained by the social understimulation endemic in a psychiatric hospital, nor by the cognitive side-effects of physical treatment. They show, conclusively, that this is the case. These authors argue that there is some pervasive neuropsychological deficit in schizophrenia, providing a nice link between what the originator of the concept of schizophrenia (Kraepelin) believed and the current tendency to look back to Kraepelin and ignore the social psychiatric movement (1930s–70s) in between. Nuechterlein and colleagues (Chapter 4) are interested in neuropsychological accounts of schizophrenia, both as a state and trait. Their approach is complementary to Dunkley and Rogers' in that it capitalises on variations in course, relapses and remissions, in the quest for vulnerability markers. Such vulnerability spans cognition, functional relatedness of brain regions, an index of which is E.E.G. coherence, and focal defects in metabolic activity revealed on P.E.T. images.

The beginnings of other approaches derived from cybernetics are to be found in the computational models referred to by Ellis and de Pauw (Chapter 19) and David (Chapter 17), and Fleminger's proposal for the action of top down processes in delusion formation (Chapter 20). Fleminger presents a schematic model of delusions, which says as much about the factors that maintain delusions as generate them. Though primarily non-neuropsychological, it deserves a place here because of a willingness to view both neurobiological abnormalities and extreme cognitive bias as important contributing variables.

Hoffman and Rapaport (Chapter 16) explore two general explanations of the nature of schizophrenic auditory hallucinations. Are they an effect of incompetent perception of ambient speech? or are they the by product of a subject's own internal subvocalisations? They conclude in favour of the latter explanation, basing their argument on some intriguing experiments.

Slade (Chapter 15) also puts forward an explanation of the nature of hallucinations. The essence of this is that they are the result of an interaction
between the four factors: (1) stress; (2) predisposition; (3) current environmental stimulation; and (4) mood. The argument is theoretical and represents one of the most typical preneuropsychological models of schizophrenia, namely a multifactorial explanation. Although not in any direct sense neuropsychological, Slade’s proposal is pertinent to neuropsychological formulations themselves, as it highlights non-brain factors, which any pure neuropsychologist should consider. Slade’s pioneering use of the single case methodology in his early formulations has a distinctly contemporary ring. The neuropsychological element in these two chapters is small but is complemented by David’s comprehensive review. After reviewing critically the neurological literature on auditory hallucinations of all types, David narrows in on the neuropsychology of inner speech and attempts to illustrate how disturbances of this mechanism may give rise to typical psychotic experiences. Despite their different theoretical orientations, all of these authors writing about hallucinations, emphasise inner speech as the critical element, which any study of schizophrenic patients’ “voices” must address.

Hoffman and Rapaport (Chapter 16) also allude to computer simulations and network models of cognition, which we predict will become a major focus over the next decade. As for our earlier justification for using the “neuro” prefix, which permitted reference (implicit and explicit) to the brains of both humans and animals, we may have to bend the rules even further to include silicon chip-supported artificial brains.

The chapters that follow all examine the general issue of the psychology of schizophrenia. The neuropsychological component is examined from the standpoint of brain imagery, neuropsychological test results, phenomenology, neurodevelopment, and electrophysiological studies. The theoretical stance of different authors varies considerably, but we hope that the “neuropsychology of schizophrenia theme” can be traced in all the chapters, if not in a direct reference then in the formulation of some complementary psychological proposal as yet partially or yet-to-be assimilated.
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