British Journal of Psychiatry (1992), **161**, 293–297

Annotation

Phenomenology
Its Place in Schizophrenia Research

ANN M. MORTIMER

In some quarters schizophrenia has gained the reputation of a graveyard of research. Few findings stand the test of time, most of the pieces of this particular jigsaw seem to be missing, and it is not easy to make sense of those that are available. Even 'hard' scientific findings fail to be replicated, an example being the status of D_2 receptors in drug-naive schizophrenics (Wong *et al*, 1986; Farde *et al*, 1987).

The functions of the brain are still difficult to investigate successfully with any of the scientific techniques applied elsewhere in the body: biochemistry, neurophysiology, molecular genetics, and functional imaging can thus far give limited insight owing to the immense complexity of brain structure and function, to which the unsatisfactory access afforded by living brain is an added hurdle. To some, esoteric 'high-technology' research may seem inappropriate in a disorder like schizophrenia, where one can often tell that something is radically wrong with the patient simply by wishing him a good morning. Small wonder that a recent monograph (Carpenter, 1989) stresses the importance of "distinguishing schizophrenia through psychopathology" with a scheme based on categorical 'domains' comprising various groups of accessible, familiar phenomena such as positive symptoms and disorders of affect.

Phenomenology – the precise description of psychopathology – is an obvious tool for schizophrenia research. Psychiatric training emphasises its importance in investigation, diagnosis, and management, the phenomena of schizophrenia becoming more familiar and less difficult to elicit as experience accumulates. The raw material, the talk and actions of patients, is readily accessible from ordinary psychiatric practice without any need for sophisticated technology.

There are several additional advantages to the use of clinical phenomena in schizophrenia research. They have been recognised for many years and it is hard to doubt their validity as indicators of pathology of some kind. Many are utterly alien to normal experience, as opposed to physical symptoms, such as headache, which are familiar by experience and which do not indicate specific pathology in most instances. The development of the Present State Examination (Wing *et al*, 1974*a*,*b*) has considerably sharpened the assessment of phenomena in general. A number of rating scales are now available for the appraisal of a wide range of schizophrenic phenomena in a quantitative, albeit non-parametric, manner; this ought to represent an advance on purely descriptive methods. However, within schizophrenia itself the situation is less clear, and there remains a multiplicity of criteria for diagnosis and subdivision of the disorder. In the absence of biological markers, no operationalised diagnostic scheme can claim either to be unique in identifying the condition and its variants, or to rationalise the focus of research (Cutting, 1985).

One approach to the advancement of phenomenological research is to categorise most schizophrenic phenomena into groups which are statistically valid and which might be the manifestation of a superordinate dysfunction. The dysfunction may be characterised in terms of a higher-order phenomenon, or be related to 'harder' abnormalities such as neuropsychological, neurochemical, developmental, or morphometric disorder. Such aspects of dysfunction, if meaningful, should also have aetiological, treatment, or prognostic implications: the existence or otherwise of such associations can then be scrutinised. Thus, the groups of phenomena represent an intermediate stage in the conceptualisation of schizophrenia. It must be borne in mind that clustering of phenomena does not equal clustering of individual patients (Morrison *et al*, 1990), nor are such groups sets of criteria defining diagnostic entities. Despite the latter, diagnostic criteria for both schizophrenia and its subclasses invariably include a group of phenomena: some, such as Taylor's and Langfeldt's (Brockington *et al*, 1978), are very simple.

There is a gap at present between the pattern of phenomena occurring in the individual patient, and his disease's aetiology, treatment, and prognosis, a gap which even detailed diagnosis does not fill; perhaps the validation of phenomenological groups will eventually provide a bridge. What, then, are some of the groups elucidated so far?

A working knowledge of schizophrenic phenomena reveals a piecemeal picture. For instance, most if not all Schneiderian first-rank symptoms may be construed as a dysfunction of ego boundary (Sims,

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1988). Similarly, thought-disorder phenomena may be subsumed under dysfunction of logical association. Unfortunately, the integration of basic clinical phenomena into further phenomena which are themselves utterly abstract and perhaps more difficult to quantify than the original phenomena does not help us very much, except perhaps to design a diagnostic criterion.

The positive : negative division of phenomena (Wing & Brown, 1970) has now been replicated eight times (e.g. Mortimer *et al*, 1990), although its limits and scope remain somewhat ill-defined. The dichotomy has been linked with aetiological, treatment, and prognostic issues such as longitudinal course (Pogue-Geile & Harrow, 1985; Pogue-Geile, 1989), brain structure and neurochemistry (Crow, 1989), functional imaging (Andreasen, 1989; Owens *et al*, 1985), pharmacological treatment (Kane & Mayerhoff, 1989), and neuropsychological dysfunction (Bilder *et al*, 1985; Cornblatt *et al*, 1985; Tamlyn *et al*, 1992). This list is hardly an exhaustive one, and demonstrates that the dichotomy has fuelled a large, diverse research effort. Whether it continues to stand the test of time remains to be seen, although its usefulness appears to be well established at the moment.

Three further attempts to integrate phenomena have recently been described. The first (Liddle, 1987*a*,*b*; Liddle & Barnes, 1990) is based on studies of the segregation of schizophrenic symptoms and the relationship between symptoms and neuropsychological performance in groups of chronic patients. Three syndromes are proposed: reality distortion (delusions and hallucinations), psychomotor poverty (poverty of speech and flatness of affect), and disorganisation (thought disorder and inappropriate affect). The first two appear quite similar to positive and negative syndromes, while the third may represent an artefact of factor analysis, or may reflect the primacy of the negative syndrome over several looser groupings of non-negative symptoms (see Mortimer *et al*, 1990).

The second approach (Carpenter, 1989) uses the term 'domain' for five broad categories which, according to the author, "summarise schizophrenia pathology": these are positive symptoms, cognitive and attentional disturbance, incongruity of affect, deficit (negative) symptoms, and "neurologic manifestations". Treating affective incongruity as a separate domain overcomes the problem of its being included as a negative symptom in the US (Andreasen, 1982) but as a positive or disorganisation symptom in the UK (Owens & Johnstone, 1980; Liddle & Barnes, 1990) without considering whether it should rightfully belong in either domain. The inclusion of a domain of "neurologic manifestations" is an interesting departure from the traditional psychopathological scheme, in which abnormal neurological findings are either couched in terms of psychological rather than neurological malfunction (this is particularly applied to catatonic phenomena), ignored altogether as irrelevant, or actually used as an exclusion criterion in diagnosis or selection of patients for research (Rogers, 1987). Neurological manifestations of various kinds have been described in a number of diagnostic groups in addition to schizophrenia (Hertzig & Birch, 1966; Quitkin *et al*, 1976; Bellak, 1979; Elliott, 1982). Perhaps there is as much potential for specification of schizophrenic neurological abnormalities as there has been for the specification of schizophrenic psychopathological phenomena.

Carpenter has used his domain of deficit symptoms to examine the difference between patients with and without them. Patients with deficit symptoms had worse premorbid adustment; worse neurological dysfunction; and less frontal, parietal, and thalamic glucose metabolism. Scores on some positive symptoms, response to treatment, age of onset, and duration of psychosis did not distinguish the two groups.

It could be argued that these last investigations do not represent any addition to the conceptual framework of the positive : negative dichotomy: the results of using the other four domains to examine differences between patients remains to be seen. Like the positive : negative dichotomy, the domains approach is not explanatory – presence or absence of a domain is not linked to a specific genetic, biochemical, physiological, or developmental defect, although such biological markers may become apparent as further studies progress. Clearly, the choice of domain definition is crucial if positive findings are not to be lost.

The third grouping approach does assume that its groups relate to biological markers (van Praag *et al*, 1990). 'Dimensions' of psychopathology – inertia, aggression/anxiety, and anhedonia – are supposed to relate directly to altered metabolism of the monoamines dopamine, 5-HT, and noradrenalin, respectively. This hypothesis cuts across nosological boundaries, so that an anhedonic patient would putatively suffer deranged noradrenergic function whatever the diagnosis, be it manic–depression, schizophrenia, or anything else. An impressive array of supportive biochemical and pharmacological evidence is invoked, but it is apparent that these three psychopathological dimensions cover very few of the phenomena possible in schizophrenia. For instance, dopamine agonists may induce delusions and hallucinations; therapeutically effective neuroleptic drugs

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are invariably dopamine antagonists (McKenna, 1987). It would be difficult to argue that the effects of such agonists and antagonists result from the modification of behavioural inertia according to the monoamine hypothesis. Although the point is made that symptoms are the behavioural expression of a psychological dysfunction, a simple explanation of how the rich spectrum of schizophrenia phenomena can result from broad behavioural abnormalities is probably not possible. Similarly, with regard to 5-HT, recent work (Ceulemans *et al*, 1985; Reyntjens *et al*, 1986) suggests that 5-HT antagonism may ameliorate schizophrenic disorder of affect and negative symptoms, a suggestion which does not sit easily with proposed 5-HT mediation of aggression and anxiety. A further difficulty is that if the same dysfunctions of biochemistry occur in both of the major psychoses – manic–depression and schizophrenia – then, logically, the same phenomena would be expected to emerge regardless of diagnostic category – clearly, this is not entirely the case.

The monoamine perspective represents a valiant attempt to shift the emphasis of research from tying inadequate nosology to capricious phenomena, to tying metabolism to psychological variables. Its main drawback seems to be that reality is infinitely more complex: for example, additional neurotransmitters, such as GABA (Simpson *et al*, 1989) and glutamate (Deakin *et al*, 1981), have been implicated in psychosis.

In an attempt to examine how phenomenology and other approaches are currently utilised in schizophrenia research, the author looked at the 50 research papers on schizophrenia published by the *British Journal of Psychiatry* between March 1989 and February 1991. Only two papers described trials of (psychotherapeutic) treatment. The remaining 48 papers could be divided into three groups according to the major investigative method utilised: phenomenology (24 papers), basic/clinical science (19 papers), and neuropsychology (five papers). Basic/clinical science techniques applied to schizophrenic patients included positron emission tomography (PET) scanning for glucose metabolism, estimation of cerebral spinal fluid serotonin levels, study of somatosensory evoked potentials, epidemiology/criminology, genetic studies, assessment of neurological soft signs, medication side-effects and handedness, small-intestinal permeability, and magnetic resonance imaging.

Most (37 of the 48) papers used their approach in a pure form: 19 of 24 phenomenological papers, all the neuropsychology papers, and 13 of 19 basic/clinical science papers. Twenty-seven papers studied demographic and/or illness-status variables, such as severity and chronicity, as an adjunct to the main method. A combined approach that used an additional subsidiary method distinguished the remaining 11 papers: neuropsychology was added to phenomenology in two, basic/clinical science to phenomenology in three, and phenomenology to basic/clinical science in six.

An obvious conclusion of these findings is that phenomenology is, indeed, alive and well in the minds of those who choose to research into schizophrenia. A second conclusion is that there is a very wide variety of basic/clinical science techniques which have been felt sufficiently relevant to be of use. A third, pessimistic conclusion, suggested by the dearth of studies using combined methods, is that the body of research is itself schizophrenic: it is fragmented into a vast diversity of disparate, narrow approaches which bear no relationship whatsoever to each other. Possible explanations for this lack of organisation are that individuals will investigate areas of their own interest and experience, that available facilities (especially 'high technology') will be made use of, that there is difficulty and complexity in putting together a piece of research linking a number of approaches, and that the elusive nature of aetiological factors suggests a large number of avenues worth trying.

Rather than taking a narrow approach to the investigation of schizophrenia, using purely phenomenology or anything else, it may be more profitable to set up strategies in which phenomenology, neuropsychology, and basic/clinical sciences are combined in a manner designed to facilitate understanding of the connections between the various approaches. Figure 1 illustrates a three-level model of schizophrenia: the levels are levels of understanding, and the interlinking of the three levels is what requires to be elucidated. Explanation of abnormality within each level, while requiring consistency, need not be unitary. In addition, this model implies that each level can be used only to understand a level

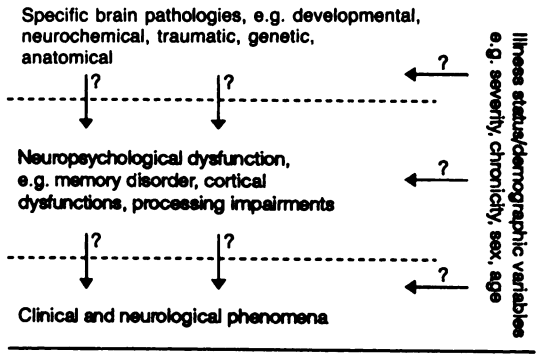


Fig. 1 Three levels of understanding in schizophrenia.

https://doi.org/10.1192/bjp.161.3.293 Published online by Cambridge University Press

directly above or below; that understanding cannot occur across an intervening level. The interposition of neuropsychological dysfunction between phenomena and brain pathology stems from increasing evidence that schizophrenia is associated with particular patterns of results on 'frontal' (Morice, 1990; Shallice *et al*, 1991), memory (McKenna *et al*, 1990; Tamlyn *et al*, 1992), and perhaps other neuropsychological tests. This suggests that a neuropsychological approach to the understanding of phenomenology might eventually be achieved (cf. Frith, 1987). That neuropsychology can also be related to brain function is, of course, amply documented in much research on the same memory and frontal functions. In short, neuropsychology may turn out to be the only way of bridging the gulf between the clinical phenomena of schizophrenia and the presumed underlying brain dysfunction.

This model of schizophrenia has parallels with Tsuang *et al*'s approach (1990) to the investigation of the heterogeneity of schizophrenia. Their 'level II indicators' (Fig. 2) are similar to the brain-pathology level, and their 'level III indicators' to the phenomenology level. In the model illustrated here, as opposed to Tsuang *et al*'s, neuropsychological abnormality is conceived of as being explanatory of phenomena and resulting from brain pathology, and as therefore deserving a level of its own. It is unclear whether this model requires an aetiological level analogous to Tsuang *et al*'s 'level I'; it may be simpler to deal with aetiological factors purely in terms of their implications for specific brain pathology.

This model includes illness status factors, such as severity and chronicity, as relevant to all three levels, since it is conceivable that interactions may take place at each level. Neurological abnormalities have been designated as phenomena, since it can be logically argued that they must result from brain pathology: this requires validation, as does their diagnostic specificity.

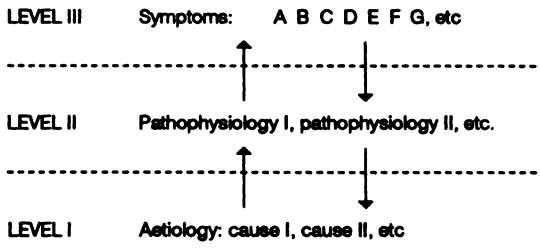


Fig. 2 Models for investigating the heterogeneity of schizophrenia (adapted from Tsuang *et al*, 1990).

Conclusions

There appear to be three views upon the nature of schizophrenia research. The first view, the 'graveyard' theory, a sort of philosophical nihilism, is fostered by the inability so far of concentrated efforts to solve the puzzle of schizophrenia. The second view implies a reductionist approach: the aetiology and pathology of schizophrenia, using analogies with 'medical' conditions such as tuberculosis, will be understood in fairly simple terms. Thus, the search for the gene or genes, the neurodevelopmental disorder, the aberrant neurotransmitter, or the anatomical site of dysfunction continues; in parts admirable, this approach is, as a whole, chaotic.

The third view (the author's) is that research may more expediently proceed on a very broad canvas, making use of all three components of the 'levels of understanding' model. This is necessitated by schizophrenia's heterogeneity, and the crevasse between phenomenology and brain disorder. I suggest that schizophrenia researchers should, therefore neither be seduced by the possibilities of 'high technology', nor limit themselves to an exclusive phenomenological approach, nor ignore the potential of neuropsychology; an integration of all three may, in time, contribute to the denouement of this fascinating condition.

Acknowledgements

The author would like to thank Dr P. J. McKenna, whose comments on this paper were most helpful.

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Ann M. Mortimer, BSc, MB, BCh, MRCPsych, MMedSci, *Senior Lecturer in Psychiatry, Charing Cross & Westminster Medical School, c/o St Bernard's Wing, Ealing Hospital, Uxbridge Road, Southall, Middlesex UB1 3EU*

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