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EDITORIAL

Cognitive neuropsychiatry?[1]

Given that the dominant and most productive approaches to psychiatric research hitherto have come from epidemiology and neuroscience, the suggestion that it would be valuable to return to qualitative assessments, paper and pencil tests and single-case studies is liable to be treated with derision. Nevertheless, such a strategy, which goes under the heading of cognitive neuropsychology, has already begun to assert itself in the field of psychology and, predictably after the usual ten-year delay, is now encroaching on psychiatry. Like most trumpeted 'new advances' it turns out that this is an old one, going back at least a century.

The founding fathers of neuropsychology, Wernicke, Lichtheim, Dejerine, Lissauer *et al.* were 'diagram makers'; that is, they attempted to construct what would now be termed information processing models of normal cognition by inference from the pattern of defects seen in rather unusual cases in the neurological clinic. Unusual in that they suffered from specific deficits which forced the conclusion that psychological functions, once lumped together, could in fact be dissociated. Hence language input processes were separate and could be put out of action, while output processes were left more or less intact (Wernicke, 1874; Lichtheim, 1885). Or, more dramatically, a person might be unable to identify an object by name yet retain knowledge about the object (Lissauer, 1880; see Jackson, 1988). More dramatically still, is the patient who cannot recognize a lone class of object such as the human face yet recognizes items of similar appearance and complexity (Bodamer, 1947). A single 'ideal' case with one of these 'pure' deficits can thus become the lynch-pin of theoretical development. Better still is the identification of a pair of cases where one has lost function X but has a normal function Y and the other has the opposite pattern of abilities/disabilities, the magical double dissociation (Teuber, 1955). Even in the last few decades dramatic discoveries have been made based on this method. The dissociation between short- and long-term memory and between short-term memory (Warrington & Shallice, 1969) and language comprehension are two examples of current interest (Vallar & Shallice, 1990). The limitations and possibilities for scientific inference of this method have been discussed in detail by Shallice (1979, 1988). It has taken hold for at least two reasons apart from these contemporary successes: the rigour of analyses by Shallice (1988), Caramazza (1986) and others, and the philosophical underpinnings articulated by Fodor (1983) in the principle of modularity.

Within neuropsychology this renaissance has not received a universal welcome (Caplan, 1988). Others propose that the 'syndromic approach' has served medicine well, particularly with regard to localizing disturbance to a brain region. Some cognitive neuropsychologists (with notable exceptions; McCarthy & Warrington, 1990) have been open to criticism from cognitive neuroscientists for taking an excessively cognitive approach (see e.g. Morton, 1984) which is disinterested in localization and indeed can be caricatured as regarding the constraints of plausible neurophysiology as a trifling matter, a nuisance, to be ignored in favour of increasingly rarefied, abstract models. Others take issue from a more pragmatic base, reminding researchers of the assumptions of normality and universality that cognitive neuropsychologists bring to their work. That is, assumptions of what the patient 'would have been expected to do' were it not for brain injury; that the patient's cognitive apparatus is entirely representative of all other human cognitive systems; and that the deficits observed reveal a stable underlying process and not a unique and

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haphazard reorganization (Newcombe & Marshall, 1988). Also, they state that pure cases could occur because of exceptions to functional organization rather than being exemplars of it.

Such misgivings were voiced towards the end of the nineteenth century by neurologists such as Freud (1891/1953) and Marie (1906) who attacked the diagram makers for the inconsistencies of their models, their inability to account for exceptions and neurological implausibility. These sceptical views began to hold sway in the early half of the century and were epitomized by the English neurologist, Head (1926), in a chapter in his textbook describing the proliferation of ill-conceived diagrams, ominously entitled 'Chaos'[1]. What with this and the combined assaults of mass-action (Lashley, 1929) and Behaviourism, the cognitive neuropsychological approach seemed to die until resurrected by authors such as Geschwind (1965). It now flourishes with several monographs (e.g. Ellis & Young, 1988; McCarthy & Warrington, 1990), symposia, at least one university degree course and a journal devoted to it. The purpose of this article is not to re-run these arguments but to examine their applicability to psychological research in psychiatric disorders.

SINGLE-CASE STUDIES

These have always had a place in psychiatric research. Ironically, in view of his previous stance, Freud wrote some of the most famous case studies in the medical literature such as Anna O. and the Rat Man. However, modern commentators question the validity of Freud's 'hypothesis testing' as described by him. Psychologists often use a single patient and an A–B–A–B intervention design to examine the specific effects of treatment (Aldridge, 1991). Indeed '$N = 1$' studies of this kind have a respectable pedigree within epidemiology (Guyatt *et al.* 1986). This avoids the potentially misleading conclusion from group studies that treatment A is somewhat effective for condition X compared with placebo – based on a statistically significant improvement. This averaging procedure may disguise a genuine and dramatic response in some individuals and a complete lack of efficacy in others. Similarly, the deceptively simple case-study methodology can be allied with state-of-the-art neuroimaging as in the work by Hawton *et al.* (1990) who looked at hypofrontal cerebral blood flow with serial single-photon emission tomography scans in a psychotic patient before, during and after treatment.

The black swan approach, or finding the exception which disproves the rule, is a powerful example of single-case methodology. Again, this cannot be regarded as a safe test of aetiology or even treatment efficacy; it is a test of a theory. The discovery of normal speech in a patient following left hemispherectomy disproves the theory that the right hemisphere is incapable of supporting articulate language. Similarly, one pair of identical twins discordant for a disorder refutes the notion of absolute genetic risk, and so on. Unfortunately, in most cases we would not recognize a black swan even if we saw it since rarely do we have such a clear understanding of mechanism, as in the above examples. The single-case approach can only be applied to unambiguously specified theoretical models and mechanisms, not disease causation, for which the epidemiological approach is usually necessary.

PROBLEMS FACING COGNITIVE NEUROPSYCHIATRY

Problems arise out of the distinctly different subject-matter of traditional neuropsychology and of psychiatry. The former is used to dealing with deficits, often gross and fixed – aphasia, apraxia, amnesia etc. There are few neuropsychological studies of hyper-functions (e.g. hyperlexia), and here the emphasis is on the underlying deficit or the discrepancy between the unusual ability and other deficiencies (Pennington *et al.* 1987). There are a few exceptions such as a recent description of hyperlalia (Yamdori *et al.* 1990) and the careful dissection of exceptional artistic ability in autistic children where explanations have been in terms of highly developed or super-efficient access to modules such as visual memory (O'Connor & Hermelin, 1987). Psychiatric disturbances, on the

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other hand, if framed in cognitive terms, are usually aberrant behaviours, or perhaps excessive activities, even when viewed in Jacksonian terms as the loss of inhibition. The Capgras' delusion is not the failure to recognize a familiar person but mistaken beliefs about their identity. Hallucinations are not failures to perceive sensory stimuli but, by definition, they represent the perception or internal generation of images in the absence of stimuli. There are few psychological models which incorporate abnormal increases in function. The nearest is the anatomically based 'hyper-connection' model of behavioural disturbances in association with temporal lobe epilepsy which proposes that certain beliefs may attract unusually strong affects because of increased linkage between cortical and limbic brain areas (Bear, 1979).

Of interest in this context is the study by Shallice *et al.* (1991) which is a case series of five schizophrenic patients. Curiously, given the senior authors' theoretical position, the study takes a conservative neuropsychological rather than cognitive neuropsychological approach. It addresses the 'localization' in the brain of the psychological impairment. The authors administered a battery of tests aimed at detecting dysfunction in different regions. They found that 'tests sensitive to frontal lobe lesions' were performed particularly badly. The notion that negative symptoms of schizophrenia are analogous to frontal lobe deficits is popular and plausible. However, these deficits are presumed to be somewhat stable and to give rise to loss of functions, in this case planning and motivation. If 'dementia' was substituted in the text for schizophrenia, there would be no change in the sense of the article. However, a truly cognitive neuropsychological approach would have no interest in aetiology or diagnosis but in the manifest disturbance. Such studies do not examine 'stroke', 'epilepsy' or 'Alzheimer's dementia' which are not psychological constructs but, for example, 'phonological dyslexia' and 'specific impairments of semantic memory'. Hence the problem of clinical heterogeneity which troubles Shallice *et al.* (1991) as it does most conventional psychiatric researchers, is of no concern to the cognitive neuropsychologist who is interested in solitary disturbances of function that may converge from a variety of pathological routes on to a presumed final common path. While old-style neuropsychology may confirm the dementia of dementia praecox, it cannot of itself explain the phenomenology of psychosis.

Psychotic disturbances are not stable deficits but unstable, transient phenomena, over and above the deficits with which they may be associated. Associations do not allow strong inferences. For example, one learns little about expressive language by knowing that an extensor plantar response on the right is often associated with aphasia. The co-occurrence of these phenomena reflects merely a shared anatomical location in the left anterior part of the cortex; there is no functional relationship between the two. In schizophrenia, an equivalent association exists between the so-called positive and negative symptoms. A truly *cognitive neuropsychiatric* approach would be to seek out a patient with positive symptoms but without negative symptoms (and vice versa), surely not a difficult task, although one wonders whether an important journal would consider publishing such a case. This is a pity because it counters indisputably any psychological theory, though not a neurological one, which links the two symptom-types. Similarly, the study of global 'cognitive impairment' in schizophrenia is theoretically (though not practically) trivial as it is in patients with epilepsy since both presumably have damaged brains. More interesting is the fact that both conditions can occur in people of the highest intelligence with no manifest cognitive impairment – note, another dissociation. The real challenge to cognitive neuropsychiatry is to provide a cognitive account of the essence of psychosis: abnormal beliefs, perceptions and inexplicable behaviour – evanescent, mysterious and fleeting though these sometimes are. The neurological equivalent would be to study the cognitions that occur during an epileptic seizure.

Fixed delusions, continuous hallucinations and intractable thought disorder may be more amenable to cognitive neuropsychiatric investigation. Nevertheless, such symptoms are often immensely distracting, making prolonged testing under such circumstances difficult. The subjective nature of these also poses challenges to this methodology. How can one be sure that the patient is experiencing hallucinations at that moment? Cooperation in psychiatric subjects is variable and unreliable. This too places limits on testing. Finally, it is worth mentioning that the variability of psychiatric disturbances is not unique. For instance, patients with acquired dyslexia or agnosia may

show marked fluctuations in performance from day to day or even hour to hour. Methods for dealing with this which will have to be devised in cognitive neuropsychiatry may feed back to its neuropsychological counterpart in a mutually beneficial way.

APPLICATION OF COGNITIVE NEUROPSYCHOLOGICAL MODELS TO PSYCHIATRIC DISORDERS

Perhaps the first example of a cognitive neuropsychological case study applied to a psychiatric disorder was by Brockington in his unpublished M.Phil. thesis (1972). This was a detailed analysis of a patient with a chromosomal defect whose main symptom was an inability to cook for herself. Brockington devised a number of naturalistic cooking tests, for which he obtained normative data, in order to distil the essence of the problem, which turned out to be one of sequencing and planning a complex action. Slade published a number of papers around this time (Slade, 1972, 1973) on auditory hallucinations. These were essentially treatment studies but were also used to validate and develop the then current theories of the mechanism and genesis of hallucinations. A recent study by David & Lucas (1993) has also addressed auditory-verbal hallucinations but from an explicitly cognitive neuropsychological perspective, drawing on current models of short-term memory and language processes. This approach was only feasible because an intelligent, cooperative patient with continuous hallucinations was found.

An explicit use of cognitive neuropsychological models as a framework for understanding a psychiatric symptom was provided by Ellis & Young (1990) in their analysis of delusional misidentification syndromes (DMS). (Indeed Hadyn Ellis must by credited or blamed for the first public use of the term cognitive neuropsychiatry at a symposium, held at the Institute of Psychiatry in October, 1991.) This was promising from the start for a number of reasons. First, they had a fairly simple yet well substantiated model of face recognition based on studies of normal and clinical subjects including cases of prosopagnosia (Bruce & Young, 1986). Second, evidence has been accumulating (e.g. Lewis, 1987) to suggest that most cases of DMS have coarse organic brain disease so that it was likely that these disorders might 'behave' like neurological disorders. Finally, a number of paradigms and test materials, with norms for comparison, were available. The authors offered testable hypotheses as to the pattern of cognitive deficits which would be expected in different DMS according to the point in their information processing model at which the abnormality arose. Experimental work to date has been less decisive than hoped (Young *et al.* 1990) and it is too early to say whether this will lead to substantial advances in our understanding of DMS or delusions in general. Psychiatric patients, it seems, are rarely 'pure' and never 'ideal'.

THE FUTURE

In a review by Jones & Murray (1991), the authors stated that 'Genes code for proteins, not for auditory hallucinations in the third person'. While this is undeniable, they did not offer an alternative explanation for these particular symptoms. The subjective nature of these phenomena is no longer considered a barrier to scientific enterprise (Marcel & Bisiach, 1988). The only way to understand psychological phenomena is in terms of psychology. Hopefully, such work will progress in tandem with that which addresses aetiology. The only barrier is our own failure to exploit the progress made in other fields and a curious aversion to theory. Cognitive neuropsychiatry, if it can overcome formidable practical problems, could become an immensely powerful means of achieving conceptual growth.

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