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The Future of Diagnosis

ANTHONY DAVID

'Cognitive neuropsychiatry' is a new hybrid label (David, 1993; Ellis & de Pauw, 1994). A journal soon to be launched with that as its title defines the term as "the study of cognitive processes underlying psychological and behavioural abnormalities, including psychotic symptoms, with and without organic brain disease". Cognitive neuropsychiatry is the cousin of cognitive neuropsychology (Ellis & Young, 1988), a discipline which seeks to understand basic cognitive mechanisms supporting language, perception, movement, etc. using inferences prompted by the study of pure cases of (usually) deficits, in patients with (usually) circumscribed, acquired brain lesions (Caramazza, 1986; Shallice, 1988; McCarthy & Warrington, 1990). For example, explanations are offered for prosopagnosia, the inability to recognise familiar faces while the ability to match unfamiliar faces, recognise equally complex objects and people from their descriptions, is preserved.

Charlton holds up this fledgling academic cross-discipline as an alternative to psychiatric diagnosis since the object of enquiry in cognitive neuropsychiatry is at the phenomenological level rather than the syndromic or diagnostic. In attacking psychiatric classification, Charlton catches a tide against the practice of psychiatry fanned by other critics (e.g. Bentall, 1990). In my view, despite its concern for phenomenal experience across diagnostic categories, single case study methodology and relative disinterest in cerebral localisation of dysfunction, cognitive neuropsychiatry can happily coexist with conventional diagnoses. In our present state of ignorance, treatment is best determined by syndrome (psychosis, deficit syndrome, mania, depression, and so on; see Williams, 1979) not cognitive microstructure, while diagnostic group (or possibly syndrome) is a reasonable basis from which to search for aetiology. True, success in uncovering the aetiology of schizophrenia and manic–depression is lacking, but there is no theoretical reason why this need be so. A theory of auditory hallucinations in the third person has to be psychological – in explaining a psychological phenomenon – and could be challenged only on psychological grounds. In the meantime, the discovery of a gene for schizophrenia would neither add nor detract from such a theory. Any coexisting psychological theory of the *aetiology* of schizophrenia would, however, have to be revised if not abandoned.

An analogy can be drawn with the syndrome of stroke. Many of the most remarkable pure cases of specific deficits in neuropsychology, such as prosopagnosia, arise following stroke. Theories which account for the specificity of the cognitive deficit have nothing to say about the aetiology of stroke, which after all, could have caused aphasia, hemiplegia, or ataxia depending on the 'luck' of the individual and where the vascular occlusion occurred. This should not deflect researchers from looking for aetiological and risk factors underlying stroke since their discovery could have major implications for health. It is only because of our primitive state of knowledge about aetiology in psychiatry that we at times conflate these two aims – explanation of mechanism and discovery of cause.

Charlton offers a simple model of the mind based on the personal computer. This I think is flawed. First, because it requires an operator to interpret output and is thus incorrigibly dualist (Dennett, 1991), and second it supposes that psychosis is a matter of disintegration of basically sound subsystems. While this view is reasonable and accounts for much of the non-specific findings in the literature on neuropsychological deficits in schizophrenia (David, 1994*a*), it is far from confirmed. Indeed, the view that delusions are faulty interpretations of good sensory data (see Garety & Hemsley, 1994) has been challenged (Maher, 1974) and does not seem to

https://doi.org/10.1192/S0007125000064266 Published online by Cambridge University Press

explain delusional memory (David & Howard, 1994). Similarly, the cognitive neuropsychological literature on cases of anosognosia (lack of awareness of disease or bodily change) belies the neat separation between lower level dysfunction (neurological) and higher level dysfunction (psychiatric). A fact later conceded by Charlton. The patient who believes they have three upper limbs, knows that this is impossible yet cannot override this input through correct inference (Halligan *et al*, 1993).

A further illustration of the rationale for cognitive neuropsychiatry can be gained through a thought experiment: consider a theory concerning the circulation of the blood. Since the internal structure of the mind is not directly observable (although we are not entirely ignorant about it) we have to infer its workings from function and dysfunction. Imagine the same was true of the cardiovascular system. An intuitive model of blood circulation and oxygenation, gleaned from the external appearances of the body, would be that the heart is a pump which sends blood to the lungs for oxygenation and then onwards around the body returning to the heart. Then someone describes a case of a man who has a good pulse but is cyanosed and develops oedema. The physician suspects that the heart may be malfunctioning such that blood is pooling in the peripheral organs and not reaching the lungs. However, this is incompatible with the simple theoretical model advanced above since there should also be evidence of poor cardiac output (say, a thready pulse). The theory begins to creak. Next, another perceptive clinician describes a second case in whom the pulse is thready, blood pressure is low, and there is severe breathlessness. There is no peripheral oedema. Again all the signs point to malfunction of the heart but the pattern does not fit with the currently accepted modular architecture of the circulation (i.e. separate, relatively independent components, each with a specific job). A new theory is required and states that there must be two pumps: one which collects blood from the bodily organs and pushes it through the lungs, and another which conveys the oxygenated blood to the rest of the body. Note that this new theoretical model is silent on the matter of where these two pumps might be located, only that there are (at least) two. The fact that we now know that they are joined in a single organ is immaterial.

Cognitive neuropsychiatry conforms to this same logic but tries to explain such things as third person auditory hallucinations rather than oedema. The models involve language input and output systems, monitors and loops, but exist as hypotheses attempting to explain phenomena. The cognitive neuropsychiatrist must take into account the meaningful content of the hallucination (basic language output must be intact); the similarity with normal inner speech (a normal mechanism out of control rather than an entirely aberrant mechanism) and so on, when proposing his or her theory.

This methodology carries several assumptions. In the heart analogy we assume that everyone has lungs, two hearts (!) and oxygen-carrying blood. If not, the models can have no general application. We are not saying everyone's heart is the same but that everyone has circulation. If we were to 'average' the two 'pure' cases we would end up with a picture of heart failure which concealed the modularity of the heart and succeeded in describing neither patient's condition satisfactorily. In the same way most people who suffer from hallucinations seem to be unable to differentiate self- from other-generated voices. However, they have no difficulties distinguishing between different external voices nor in understanding the content of voices, internal or external. Other patients do have problems understanding and producing meaningful speech. To 'average' these abilities and disabilities would be to obscure such differences. Charlton assumes, rightly in my opinion, that mental structure is as invariant as physical structure and supports this claim by reference to evolution. He writes, "the basic structure of the human mind is identical in all races and societies . . .". By placing psychiatry in this evolutionary context, he makes an important contribution which I hope will be developed. In the case of stroke, a lesion to the left hemisphere will cause aphasia regardless of the level of literacy of the individual. However, dyslexia is a different matter. Much effort has gone in to explaining acquired deficits of reading (Coltheart *et al*, 1987) but this really is a deficit confined to literate people. Presumably, in evolutionary terms, a more fundamental defect of symbol recognition upon which reading is grafted must be involved, but it reminds us that differences between individuals may exist at the 'deep' cognitive level.

A further assumption already alluded to, is that it need not matter what the cause of the dysfunction is. Infarction or cardiomyopathy in the heart case, genetics, aberrant development, chemical imbalance in the case of psychiatric illness. Finally, the fact that it is a lesion of the left hemisphere which causes aphasia is not important since a right-sided lesion may also occasionally do this (in a person with crossed dominance for language). Hence single case methods are unreliable for localisation. The cognitive neuropsychological approach is only interested in the fact of aphasia in the midst of otherwise normal functioning (i.e. modularity (Fodor, 1983)), not where the lesion is.

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I have taken the time to argue for a cognitive neuropsychiatric methodology so that it may survive critics of Charlton's more general radicalism. Where I join with him on the barricades is the plea for a theoretically motivated psychological approach to psychiatric classification and in his belief that this will lead to a sensible and non-arbitrary classificatory system. Cognitive neuropsychiatry has first to demonstrate it can do this. It is early days and the results are mixed (Shallice *et al*, 1991; David & Lucas, 1993; David, 1994*b*). Cognitive probes of the sort envisaged by Charlton will need to be developed and tested before older methods are abandoned. Cognitive neuropsychiatry could turn out to be the saviour of psychiatric diagnosis, rather than its nemesis, leading it out of the current dark ages into a new era of rational enlightenment (Charlton, 1993).

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Method in Madness

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To both practitioners and outsiders, psychiatry is "a medical discipline long on disorders and short on explanations" (McHugh, 1995). Worse, the taxonomy of those disorders does not seem to be motivated in any principled fashion (Henderson, 1994); successive revisions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) are not always guided by the types of empirical evidence that would provoke classificatory change in other scientific domains. And worse still, there seems to be little consensus about what would constitute an explanation for any psychiatric phenomenon; it is difficult to seek the 'true' causes of disturbed cognition, affect, or praxis without having a moderately clear picture of what would count as an explanation (right or wrong!). In the absence of coherent modes of explanation (that is, theory), the collection and interpretation of data (whether clinical, epidemiological, or experimental) can only be piecemeal and capricious. That is, there are more constraints on how to support or disconfirm a theory than there are on how to revise a nosology.

As a pre-theoretical discipline, psychiatry is especially open to misdirection and the disastrous consequences that can follow from fad and fashion (McHugh, 1995). We can thus only applaud when

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