

## Frontal Lobology – Psychiatry’s New Pseudoscience

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The frontal lobes are the most evolutionarily advanced organ of the body. Here lies the seat of the highest human functions of thought, intellect, creativity, self-control and social interaction (Milner & Petrides, 1984; Lishman, 1987; Russell & Roxanas, 1990; Kolb & Wishaw, 1990). As one recent reviewer notes (Reading, 1991), it is hard to avoid ‘sounding metaphysical’ when going through such a list. It seems that, in accordance with psychiatry’s own late phylogenesis, the frontal lobes have only just been discovered.

The disorders outlined below have been ascribed to frontal lobe dysfunction.

*Personality disorders* (Gorenstein, 1982): the lack of concern for others (psychopathy), the indifference, and the apathy are all reminiscent of frontal lesions.

*Obsessions* (Behar *et al*, 1983): the rigidity, lack of spontaneity, and perseveration are the hallmarks of frontal lobe dysfunction.

*Delusions* (Benson & Stuss, 1990): the inability to evaluate evidence and the jumping to conclusions are suggestive of frontal pathology.

*Depression* (Robinson *et al*, 1984): the psychomotor slowing, the dysfluency of speech, and the failure to respond to the environment are surely reminiscent of frontal lobe damage.

*Mania* (Taylor & Abrams, 1986): the disinhibition, overfamiliarity, and stimulus-bound behaviour point unmistakably to the frontal lobes.

*Hyperkinesia/Conduct disorder* (Mattes, 1980): the poor impulse control and inability to delay gratification are very ‘frontal’.

*Schizophrenia* (Parfitt, 1956; Goldberg, 1985; Weinberger *et al* 1986; Reading, 1991; McGrath, 1991): the avolition, poor judgement, lack of self-care, inappropriate behaviour, poverty of thought, poor insight (David, 1990), and lack of reason are positively redolent of frontal dysfunction. (It should be noted that a substantial part of the influential work of Stuss & Benson on the function of the frontal lobes (Stuss & Benson, 1986) was based on studies of leucotomies performed on schizophrenic patients.)

To this list could be added catatonia (Taylor, 1990), thought disorder (McGrath, 1991), anorexia nervosa, and hysteria, in all of which frontal lobe pathology has been postulated (Russell & Roxanas, 1990). All psychiatry, not to mention human life, is there. Psychiatric disorders are, by definition, problems at

the highest levels of thought, so how does it help us to state that they are, by analogy and implication, the surface manifestations of frontal lobe neuropathology? Such a statement is a tautology. Rogers (1985) has argued in a different context, namely movement disorders, that the terminology with which phenomena are described brings with it prejudicial assumptions about the nature of the phenomena. Here is another conflict of paradigms where the glamour of neuroscience has displaced a humbler descriptive science, psychopathology. Does history help us understand how this stale impasse came about?

Much of the following discussion will concentrate on schizophrenia since this is where the frontal lobe hypothesis has its strongest allegiance.

### Anatomy and physiology

The frontal lobes constitute approximately one-third of the brain. Therefore, localising a disturbance to this region is rather like a person directing a visitor to an address marked ‘Europe’. Hence, genuine experts in the field refer to specific areas according to Brodmann’s maps or other anatomically specified regions. Three main ‘subcontinents’ have been identified with a degree of functional specificity – at least in experimental animals (see Goldman-Rakic (1984) and Fuster (1989) for reviews). These are the orbital, mesial and dorsolateral areas. Damage to orbitofrontal regions is said to give rise to the more ‘pseudopsychopathic’ clinical syndrome while dorsolateral prefrontal damage is said to produce a ‘pseudodepressive’ state (Blumer & Benson, 1975).

### Neuropsychology: the frontal lobes do nothing/ everything

Modern contributions to this debate began with Hebb (1945) who showed that patients with frontal lesions maintained their IQ performance much better than patients with posterior lesions (although this is not a reliable finding, see Warrington *et al* (1986) and Heinrichs (1990)). Meanwhile Goldstein (1944) struggled to encapsulate the nature of the disturbance in terms of the loss of abstract attitude, which he observed in similar patients. Later, Luria (1966), by devising his own tests to tap planning and regulation,

rather than employing large IQ batteries, was able to show striking deficits. Modern commentators now agree that the frontal lobes carry out an 'executive function' (e.g. Shallice, 1988). Therefore, while functions such as 'language' and 'memory' can be localised elsewhere in the brain, the deployment and appropriate use of these functions is the province of the frontal lobes. In Fodor's terminology (Fodor, 1983), the central systems are not 'modular' but rather, they are likely to be distributed. This presents a problem to the would-be localisationist. More worryingly, it suggests that tests of language, memory (Kopelman, 1991) and the rest, may be impaired by frontal pathology depending on the precise demands of the test, the disposition of the subject, and the degree to which the test is demanding (i.e. requires strategic planning). In summary, the paradox contained in this section's heading is true. The frontal lobes, in a sense, do everything in general and nothing in particular (but see later for an experimental approach to dealing with this).

#### Do frontal lobe tests test the frontal lobes?

Here I will discuss two procedures which have acquired the status of traditional tests of frontal lobe function. These are card sorting tasks, of which the most famous is the Wisconsin Card Sorting Test (WCST), and the Stroop test. The WCST requires the subject to arrange cards according to one of three criteria: colour, number, and shape (Milner, 1963). Patients with frontal lobe resections were found to be impaired on completing a set within one category, and, more significantly, they tended to continue sorting according to one dimension after it had been changed – perseverative errors (but see Drewe, 1974). It is the latter which are diagnostic of frontal dysfunction (Nelson, 1976).

Turning to psychiatric patients, schizophrenics have been found to perform poorly on this test (Malmo, 1974). However, other psychiatric patients (Malmo, 1974; Heinrichs, 1990), and those with diffuse brain damage, are also impaired (Robinson *et al.*, 1980). Furthermore, non-specific poor performance has been found in some studies rather than an increase in perseverative errors (Kolb & Wishaw, 1983; Mattes *et al.*, 1991). One study failed to show any difference between schizophrenics and manics and, even more worrying, found that 53% of normals performed within the brain-damaged range and 47% within the range indicative of 'focal frontal involvement' (i.e. 16 perseverative errors) (Morice, 1990). There are also examples in the literature of gross frontal lesions and 'frontal' cognitive and behavioural abnormalities where the WCST has been

normal or near normal (Eslinger & Damasio, 1985; Shallice & Burgess, 1991).

The linking of this test with *in vivo* measures of cerebral activity such as blood flow by researchers at the National Institute of Mental Health, Washington, DC, represents a major advance in neuropsychological research. It appears that schizophrenic patients fail to activate their dorsolateral prefrontal cortices (DLPFCs) while attempting to carry out this task, unlike normal controls (Weinberger *et al.*, 1986). Is the performance poor because of a failure to activate the relevant frontal regions or is the failure to activate due to poor performance (Weinberger & Berman, 1988)? In other words, does this sophisticated research tell us any more than the fact that patients are not doing the test properly? It appears that poor performance cannot be the whole explanation since patients with Huntington's disease (HD) are similarly impaired on the test (the connections between the basal ganglia and the frontal cortex and similarity between diseases of both are well known (Robbins, 1990; Reading, 1991)) yet do show the expected increase in frontal perfusion (Weinberger *et al.*, 1988). Likewise, HD and normal subjects' performance does not correlate with frontal activation. While this body of work seems to offer robust evidence of reduced frontal blood flow in some schizophrenics it raises the question of what this represents in functional, psychological terms.

The fact that such a relationship can be demonstrated in chronic schizophrenics and that perseverative errors are negatively correlated with activation of the prefrontal cortex ( $r = -0.52$ , i.e. explains 25% of the variance, in Weinberger *et al.*'s study (1986)) and left inferior frontal region, some distance from DLPFC, in another report (Sagawa *et al.*, 1990), is useful but difficult to interpret. Perhaps the results can be explained as showing that the problem with schizophrenic patients is that they rely unduly on the frontal lobes to perform the WCST. Why should this be? Berman & Weinberger (1990) suggest that 'inner speech' is a crucial element in carrying out sorting tasks, hence the left hemisphere asymmetry in blood flow in both normals and schizophrenics on this task. Perhaps the primary disturbance lies in this realm.

One final twist comes from recent work showing that schizophrenic patients, generally recalcitrant to advancement on this test (Goldberg *et al.*, 1987) (they are typically 'frontal' in this respect), do improve when offered monetary rewards (Summerfeldt *et al.*, 1991). It seems that the core deficit is in accepting the experimenter's praise as sufficient positive reinforcement. There is a distinct possibility that

offering people money increases their frontal cerebral blood flow (Warren *et al.*, 1984).

John Ridley Stroop's eponymous phenomenon describes the interference which an incongruent colour word has upon colour naming (see MacLeod (1991) for review). There are various analogues of this classical Stroop test, such as the interference varying pitch causes in a task identifying the words high and low. The Stroop test is said to reflect frontal lobe function since successful performance requires the inhibition of one, automatic response, in favour of a second, less automatic but desired, response. Empirical evidence for the frontal basis for the Stroop test comes from just one clinical study (Perret, 1974) while others have shown a left hemisphere emphasis (Golden, 1976). Again, frontal lobe patients frequently perform well on this supposedly frontal test (Shallice, 1988). A positron emission tomography study showed activity in the right cingulate gyrus during the test (Pardo *et al.*, 1990) but it is not clear to what extent this result was influenced by the colour-word stimuli or whether it pertains to the principle underlying Stroop-like effects.

While there is some doubt as to the cerebral-localising potential of Stroop tests, there is even less reason to believe that schizophrenic patients are specifically impaired. Bearing in mind that such patients may do less well on almost any task (Chapman & Chapman, 1973) due to anything from medication and distressing symptoms to boredom and wilful uncooperativeness, a basic requirement is that they should be more impaired than suitable controls. When matched psychiatric patients are used, no distinct impairment is observed (Everett *et al.*, 1989). Work by Liddle & Morris (1991), while showing correlations between Stroop performance and negative symptoms, included only chronic schizophrenic patients.

#### Frontal lobes in schizophrenia – structural measures

This section deals with structure as opposed to function, and again concentrates on schizophrenia. While temporal lobe abnormalities are being reported in neuropathological and magnetic resonance imaging (MRI) studies (Roberts, 1991), the frontal lobes look all right when compared with those of appropriate controls (e.g. Andreasen *et al.*, 1990). Most researchers seem dissatisfied with this state of affairs, such is the hold that frontal lobology has on psychiatric thinking. The frontal cortex is, as we have seen, an executive. As such it must receive information from those departments under its jurisdiction.

Therefore, the connections to and from this part of the brain are enormous (see Robbins, 1990). It is hard to find a region which is not, by some route, connected. As a result, carefully revealed abnormalities in the basal ganglia or temporal lobes are linked, however tenuously, to the frontal lobes (e.g. Goldberg, 1985; Robbins, 1990; Reading, 1991). The frontal lobe hypothesis of schizophrenia is rapidly becoming irrefutable.

#### A way forward

The attempt to construct a neuropsychology of psychiatric disorders is an exciting venture. If it is to be successful, it must go beyond crude localisationism and instead define psychiatric phenomena in terms of a breakdown or malfunction of psychological processes (see Duncan, 1986). In schizophrenia this process is beginning to take shape (Weinberger *et al.*, 1986; Weinberger & Berman, 1988; Cutting, 1990; Frith & Done, 1990). This does not mean the pseudo-scientific application of labels which convey the impression of understanding yet explain little – as is characteristic of frontal lobology – but instead it requires rather more mundane skills of observation and measurement (L'hermitte, 1986). For example, what is 'lack of self-care' when expressed in psychological terms? Is it an inability to carry out a series of over-learned grooming behaviours? Is it a domain-specific failure to detect the disapproval of others? Is it the loss of a shared value system pertaining to hygiene? Similarly, how can 'executive functions' be studied in isolation from the subordinate processes being controlled? Shallice & Burgess (1991) provided a lead in their study which took real-life tasks and broke them down into their component parts. It is possible to show that each component can be performed in isolation but not as an ordered sequence appropriate to the setting. Demonstrating a dissociation between the correct performance of complex tasks, with but not without a structure provided by the experimenter, allows strong inferences to be made about the adequacy of the executive system. In sum, the answers to these questions cannot be found by the new technologies alone but from careful experimentation informed by a thorough knowledge of normal and abnormal psychology (David, 1992).

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