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Theories of frontal lobe executive function: clinical applications

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Abstract and Keywords

Abstract

Many of the symptoms that are particularly difficult to treat are associated with damage to the frontal lobes. There are a very large number of symptoms which are collectively referred to as ‘dysexecutive symptoms’. These are not only problematic in themselves, but can also affect a patient's ability to benefit from therapy aimed at ameliorating other forms of deficit (e.g. physical therapy), and are often associated with a generally poor response to treatment (Alderman 1991). Considerable treatment advances have been made in this area in the last few years. However, in order to develop new methods we need to understand the causes of the particular symptoms. This chapter has four aims: (1) To describe some of the latest findings about the functional anatomy of the frontal lobes; (2) to describe the main clusters of frontal lobe symptoms, how they relate together, and their relative importance; (3) to explain the main theories of how the frontal lobe executive system works and how they relate to the symptoms you can expect to see day to day, and (4) to give some 'blue-sky' predictions about which therapeutic methods might be worth pursuing based on these theories.

Aim of this chapter

The frontal lobes of the human brain are involved in a myriad of functions, including language, motor and high-level perceptual skills. Critically however, they also play an important role in what are known as 'executive functions'. These are the abilities that allow a person to adapt to new situations and develop and follow their life goals. In this regard, 'executive functions' is an umbrella term for a host of functions such as those that allow people to plan and organise themselves over long periods of time; make complex high-level and abstract judgements; and organise and control their memory processes. The way these functions interact with the environment and are supported by the brain are not straightforward. As such this is a theoretically complex area, and going from pure theory to practical clinical application is therefore not always easy. However this does not mean clinical practice cannot on occasion be informed by basic science in the area. Accordingly, this chapter presents an outline of the practical rehabilitation implications of current theories and models of executive function, outlining some 'provisional principles' for the rehabilitation of the dysexecutive patient. These are not as yet empirically supported findings, but are hypotheses developed from theorising which we hope may one day be testable. As the reader will see, these principles are not especially surprising, and (p.212) in fact generally correspond to the sorts of approaches that are good practice in everyday rehabilitation settings. But this is how it should be. After all, the gifted intuitions of many rehabilitation professionals have honed the approach to the treatment of the dysexecutive patient. And if current theorising has any validity, then its implications should correspond to what has been found to work in practice.

The aim of this chapter is to try and describe why it is that these procedures have been found to work. For further information about the rehabilitation methods mentioned in this chapter the reader is directed to Burgess and Alderman (2004); Alderman and Burgess (2002); Mateer 1999; Robertson 1999; Sohlberg *et al.* 1993; and chapters in this book by Jon Evans and Andrew Worthington: Chapters 20 and 21 respectively.

Introduction

There is little doubt that deficits in the executive functions of the frontal lobe are of major concern for the rehabilitation professional. Many of the symptoms that are particularly difficult to treat are associated with frontal lobe abnormalities (e.g. apathy; Okada *et al.* 1997). Moreover, it has been argued on many occasions that frontal executive dysfunction can affect a patient's ability to benefit from therapy aimed at ameliorating other forms of deficit (e.g. physical therapy), and is often associated with a generally poor response to treatment (e.g. Alderman 1996; Tamamoto *et al.* 2000). However, the theory of how rehabilitation of executive functions might work is not as developed as for other areas of therapy (e.g. speech and language therapy; physiotherapy) or indeed other areas of cognitive rehabilitation (e.g. amelioration of memory deficits). This is largely a consequence of two interlinked factors: the myriad of symptoms of executive dysfunction, and the theoretical complexities involved in investigating (and therefore understanding) them.

For the range of symptoms of executive dysfunction, consider Table 18.1. This lists the twenty most commonly reported symptoms of frontal lobe dysfunction that were described by Stuss and Benson (1984, 1986). There are also many other symptoms that may be less common but which could also have been included (e.g. utilisation behaviour, Shallice *et al.* 1989; bizarre behaviour, Burgess and Shallice 1996a; multitasking problems, Shallice and Burgess 1991a, Burgess *et al.* 2000), attentional difficulties (Stuss *et al.* 1999; Robertson *et al.* 1997). In addition, one might also include the difficulties with spoken language, visual perception and motor control that can occur following frontal lobe damage (see Fuster 1997; Passingham 1993 for reviews) but which are traditionally considered under other topic headings in cognitive neuroscience, and so are not outlined here. A pragmatic solution is to include all of these symptoms under one topic heading (e.g. 'executive (dys)function'), but there is a danger that this implies the possibility of finding a single unifying explanation for them, and therefore a single rehabilitative method. The present evidence suggests that this will not be possible, and that the practicing clinician will need a range of techniques which can be applied to different symptoms.

What can we learn from theories of frontal lobe function?

For the reasons just described, there is often a sizeable gap between empirical evidence and theorising in this area, and its implications for rehabilitation. This chapter will try to bridge this gap as far as is currently possible. After all, every good therapy needs a theory of what is being treated, and how the intervention will work. For this reason, our theories about frontal lobe function should crucially influence the manner of our intervention. What, therefore, do current theories tell us about how we should treat our dysexecutive patients?

(p.213)

Table 18.1 Frequencies of reporting dysexecutive symptoms[3]

Symptom	Patients reporting problem (per cent)	Carers reporting problem (per cent)	Rank of disagreement[1]	Scaled disagreement in ranks[2]
Poor abstract thinking	17	21	16.5	−9
Impulsivity	22	22	19.5	−10

Symptom	Patients reporting problem (per cent)	Carers reporting problem (per cent)	Rank of disagreement[1]	Scaled disagreement in ranks[2]
Confabulation	5	5	19.5	+3
Planning	16	48	1	+8
Euphoria	14	28	5	+7
Poor temporal sequencing	18	25	15	−8
Lack of insight	17	39	3	+5
Apathy	20	27	13	−5
Disinhibition (Social)	15	23	13	−3
Variable motivation	13	15	18	−7
Shallow affect	14	23	10.5	+1
Aggression	12	25	6	+6
Lack of concern	9	26	4	+9
Perseveration	17	26	10.5	−1
Restlessness	25	28	16.5	−6
Can't inhibit responses	11	21	9	+4
Know-Do dissociation	13	21	13	−2
Distractibility	32	42	8	+1
Poor decision-making	26	38	7	−3
Unconcern for social rules	13	38	2	+10

(1) This number represents the rank size of the disagreements (in proportions reporting the symptom) between patients and controls, where 1 = largest disagreement. In other words, 1 means that carers reported this symptom much more often than patients.

(2) This number reflects the relative disagreement in rank frequency of reporting between patients and controls, scaled from -10 to +10, with 0 being absolute agreement in rank position of that symptom. On this scale, -10 means that this was a commonly reported symptom by patients, but not by carers; and +10 means that carers reported this symptom frequently, but it was relatively uncommon for patients to report it.

(3) Only ratings of 3 or 4 (out of a maximum of 4) for each item on the DEX questionnaire (Burgess *et al.* 1996a) were considered as indicating a problem. These correspond to classification of the symptom as 'often' or 'very often' observed. These results are based on data gathered as part of the study by Wilson *et al.* (1996).

Most current 'theories' of frontal lobe function may be (in the terms of Morton and Bekerian 1986) more accurately characterised as frameworks than falsifiable theories. For this reason, they are often of rather distant help when faced with a specific symptom. However, some general principles emerge. The greatest level of distinction as regards implications for rehabilitation method concerns the way in which the theory was developed. The main differences are between single account theories, 'construct-led' theories, multiple-account theories and single symptom theories. We cannot cover all of the theories in this chapter, so we have chosen a few illustrative ideas.

Single system theories

Cohen's contextual information theory

Single system theories are those that hold that damage to a single process or system is responsible for a number of different dysexecutive symptoms. A good example is the theory of Cohen and his colleagues (Cohen *et al.* 1990; Cohen and Servan-Schreiber 1992; Cohen, Braver and O'Reilly 1998), (p.214) which is derived from connectionist modelling of simple tasks such as the Stroop paradigm. Cohen *et al.*'s theory is that prefrontal cortex (PFC) serves an adaptive, task-dependent function, representing 'context information', which they define as the 'information necessary to mediate an appropriate behavioural response' (Cohen *et al.* 1998, p. 196). This information may be a 'set of task instructions, a specific prior stimulus or the result of processing a sequence of prior stimuli (i.e. the interpretation resulting from processing a sequence of words in a sentence)' (ibid.). Different functions of PFC, such as behavioural inhibition and active memory, may therefore reflect the operation of the context layer under different task conditions. Under conditions of response competition, when a strong response tendency must be overcome for appropriate behaviour, the context module plays an inhibitory role by supporting the processing of task-relevant information. On the other hand, when there is a delay between information relevant to a response and the execution of that response, then the context module plays a role in memory by maintaining that information over time.

Cohen and colleagues acknowledge that their theory is incomplete (Cohen *et al.* 1998). They maintain that for a more general account of cognitive control (as opposed to one that is constrained chiefly to explaining patterns of Stroop performance), mechanisms are required that deal with the management of interference, the identification of task-relevant information and the representation of many different information types. A strength of this theory, however, is that various aspects of it can be tested empirically. For instance, Cohen *et al.* make the prediction from the model that memory deficits should emerge earlier than inhibitory deficits in schizophrenia.

Grafman's structured event complex theory

Jordan Grafman's theory (e.g. Grafman 2002) is different from many others which seek to understand how the executive system works because it focuses not on adaptive processes supported by the frontal lobes but on the nature of representations stored within this region of the brain. Grafman terms these representations 'structured event complexes'. A structured event complex (SEC) is 'a set of events, structured in a particular sequence, that as a complex composes a particular kind of activity that is usually goal-oriented' (Grafman 2002, p. 298). A SEC is a knowledge representation of all the typical actions and sequences of events that go to make up a common event. For instance the structured event complex for going to a restaurant with a friend might include the sequence of events such as getting in your car and driving to the restaurant, ordering the meal, food being served and so forth. Grafman believes that there are many SECs within the frontal lobes, categorised according to the posterior cortical or subcortical regions to which particular frontal lobe areas are connected. This theory predicts that damage to the frontal lobes of the brain can cause deficits in a wide range of situations, depending on which set of SECs has been affected. Thus a rehabilitation suggestion from this theory is that one needs to study carefully the situations in which the patient experiences problems and then work with them one by one, without expecting much generalisation of gains to other situations.

Implications for rehabilitation

These accounts both have the advantage of parsimony. They make different predictions, however, about patterns of impairment, and therefore approaches to rehabilitation. Cohen's theory holds the promise that apparently quite different symptoms might share a common cause, and thus treatment might lead to improvement in a range of situations. In contrast, Grafman's theory suggests that behavioural impairments in particular situations may have different underlying causes (depending on the locus of the damage), and thus predicts a more restricted outcome for rehabilitation, with each problematic situation needing to be tackled separately. However they both encourage careful consideration (p.215) of the precise demands of the situations in which patients demonstrate their impairment, and both theories suggest that it might be difficult to predict the exact form of improvement in any one case. In practical terms, Grafman makes the useful point that his theory has the advantage of being easily understood by family members. He suggests that therapists might want to target for rehabilitation an (impaired) activity that has a 'mid-range frequency of experience by the patient. This gives the patient some familiarity with the activity, but the activity is not so simple…'. He suggests training using behaviour modification methods, working on situations that are targeted at specific activities relevant to the patient's daily life (for examples of these methods see Alderman and Burgess 2002).

By contrast, Cohen's theory suggests that dysexecutive patients might be helped by a system for reminding patients what they are supposed to be doing, and how far they have so far progressed in achieving their goal. A moment by moment feedback system, in other words. A system of this kind has been used by Nick Alderman and his colleagues at St Andrew's Hospital in Northampton, in the UK. Alderman *et al.* (1995) described a programme of Self-monitoring Training (SMT) which had two specific aims: first, to improve the ability of the individual to attend to multiple events; and second, once this has been established, to reduce the behaviour of concern using an appropriate operant strategy. They argued that the latter will only be effective when the ability to attend to multiple events, and in particular, to monitor one's own behaviour and modify it in response to change in the environment, is possible. The training involved the following five stages:

• ***Stage One*** – *Baseline:* The therapist first obtains a baseline of the target behaviour.

• ***Stage Two*** – *Spontaneous self-monitoring:* In the second stage, the participant was instructed to monitor the target behaviour whilst conducting some background task over a discrete time period. The participant was given an external counting device to enable them to achieve this (a mechanical ‘clicker’, whereby each time a button is pressed, a number display is advanced by one digit). At the same time, the therapist discretely monitored the behaviour using a similar device. At the end of the trial, the therapist compared their recording with that of the participant.

• ***Stage Three*** – *Prompted self-monitoring:* Stage two was repeated with one modification: each time the participant engaged in the target behaviour but did not record it, the therapist gave a verbal prompt that they should do so. The purpose of this stage of the training was to encourage the participant to monitor their own behaviour more accurately and get into the habit of routinely making a recording whenever it occurred.

• ***Stage Four*** – *Independent self-monitoring and accuracy reward:* The purpose of this stage was to withdraw external structure and facilitate self-monitoring by reinforcing accuracy within gross limits. This involved explaining to the participant that they would receive a reward at the end of the trial providing the recording they made was accurate to within 50 per cent of that made by the therapist. During the trial itself, prompts to record would not be given to the participant.

• ***Stage Five*** – *Independent self-monitoring and reduction of the target behaviour:* The aim of the final stage of training was to encourage inhibition of the target behaviour using an appropriate operant strategy. To this aim, the patient receives reward at the end of a trial providing they had met a specified criterion (e.g. a certain number of occasions of the target behaviour). During the training period, they continue to use the external counter to monitor behaviour in an effort to keep within the limit that has

> been set. With success, this target is gradually reduced until the target behaviour is eliminated, or occurs infrequently. Of course, the point is that successful participation in the operant stage of the training is only possible because it has been preceded by improvement in the accuracy of multiple-monitoring skills.

In the original case described by Alderman *et al.* (1995), considerable reduction in a very frequent, disruptive target behaviour was achieved using SMT. It had not been possible to develop inhibitory control over this behaviour previously using other operant approaches due to a gross impairment in **(p.216)** monitoring skills. Furthermore, this improvement was still evident when reassessed some months after the training had been completed. (See also an example of SMT to treat confabulation: Dayus and Van Den Broek 2000.)

Construct-led theories

Construct-led theories are those that propose a cognitive construct[1] such as ‘working memory’ or ‘fluid intelligence’ as a key function of the frontal lobes. Usually they are predicated on various patterns of performance on experimental tasks, or a characterisation of the demands of those tasks. Investigators typically go in search of the critical brain structures that they think support this construct (e.g. Duncan *et al.* 2000).

The most prevalent ideas of this type are the working memory theories of frontal lobe function. Two of the most commonly encountered are those by Petrides and colleagues (e.g. Petrides 1994) and by Goldman-Rakic and colleagues (e.g. Goldman-Rakic 1995) (see also Baddeley and Della Sala 1998).

Working memory theories

Petrides's position concerns the roles of the mid-dorsolateral and mid-ventrolateral aspects of the frontal lobes. He argues that the mid-dorsolateral region (areas 9 and 46) supports a brain system ‘in which information can be held on-line for monitoring and manipulation of stimuli’ (Petrides 1998, p. 106). By ‘monitoring’ he refers to the process of considering a number of possible alternative choices. This system enables the evaluation and monitoring of self-generated choices and the occurrence of events. The mid-ventrolateral region, on the other hand, ‘subserves the expression within memory of various first-order executive processes, such as (the) active selection, comparison and judgement of stimuli’ (p. 107). It plays a role in the maintenance of information in working memory, as well as the explicit encoding and retrieval of information from long-term memory. The distinction between frontal lobe areas involved in monitoring and manipulation on the one hand, and maintenance on the other, is supported by evidence from patients with frontal lobe damage (Petrides and Milner 1982; Owen *et al.* 1990).

In contrast, Goldman-Rakic argues that the various different frontal lobe regions all perform a similar role in working memory, but that each processes a different type of information (Goldman-Rakic 1995). Working memory is defined as the ability to ‘hold an item of information “in mind” for a short period of time and to update information from moment to moment’ (Goldman-Rakic 1998, p. 90). It is argued that dysfunction of this system can cause a variety of deficits. Problems on the verbal fluency and Stroop tasks are explained as a failure to suppress a prepotent response due to an inability to use working memory to initiate the correct response. Perseveration and disinhibition may result from the 'loss of the neural substrate necessary to generate the correct response' (p. 93). This theory is based largely on electrophysiological recording in animals (e.g., Wilson *et al.* 1993) and functional neuroimaging studies in humans (Courtney *et al.* 1996).

An interesting aspect of these theories is that they are the ones that most strongly make a connection between function and neurochemistry. Links are consistently made between working memory, dorsolateral PFC and dopaminergic systems (See Diamond 1998, for a review). Indeed, there is a link **(p.217)** here with Cohen's theory, where dopaminergic neuro-modulation is also a critical aspect of the account (e.g. Cohen *et al.* 1998, pp. 207–8).

Implications for rehabilitation

Two possible avenues for rehabilitation emerge from the working memory accounts of dysexecutive symptoms. The first is drug therapy, as suggested by the link between working memory deficits and dopaminergic system dysfunction. The uses of drugs that alter the action of dopamine in the brain are of course well developed for schizophrenia. They are less well developed for the treatment of brain injury, although evidence is now beginning to emerge (e.g. Karli *et al.* 1999; Powell *et al.* 1996; Kolb 2002). However, these theories suggest that a role might be found for patients whose pattern of deficits is consistent with working memory problems. The second possibility arising from these theories is the use of simple and varied instructions, and quite basic methods of reinforcement. The argument goes as follows:

Let us assume that Petrides is correct, and the root of many dysexecutive patients' problems is that they cannot either (i) hold in mind a number of things at one time, and/or (ii) select, compare and make judgements upon incoming stimuli. Let us also assume that Goldman-Rakic might be correct, and that working memory systems might be information- or modality-specific (e.g. verbal, visual, tactile etc.). These characterisations have quite straightforward implications for the way that rehabilitation should be conducted: keep instructions simple and unambiguous. Reasoning with someone about their behaviour requires the person to track the various arguments as they are being said, and to compare the various aspects of the argument. This may well be beyond the capabilities of the patient with working memory problems, and suggests that one should consider using simple reinforcement and reward techniques if possible.

Petrides maintains that the ventrolateral WM system is implicated in encoding and recall of complex material. If this system is damaged, the patients may have difficulty encoding for themselves the salient aspects of the learning situation. So it may be better if verbal reinforcement alone is not relied upon; actual acts of reward may be more effective. Moreover, it may be unwise to rely for treatment effect on the patient's ability to encode and actively later remember the content of previous sessions. Gradual behavioural shaping may be a better solution. Above all, one might expect just saying to a patient 'it would be better to do X because....' or general talking therapies to be relatively ineffective for dysexecutive symptoms that may be secondary to WM impairment. According to Goldman-Rakic, this might include disinhibition, which as Table 18.1 shows, is a quite common dysexecutive symptom.

Duncan's Theory of 'g'

Another construct-led theory of frontal lobe function is Duncan's frontal lobe theory of 'general intelligence' or '*g*'. (e.g. Duncan 1995; Duncan *et al.* 2000). This is quite different from the working memory theories. It suggests instead that the principal purpose of the frontal executive system is to support a single function that is used in many situations, called 'fluid intelligence' or Spearman's *g*.[2] As its implications for rehabilitation are considerable, it is worth outlining in a little detail.

Duncan's concept takes as its starting point the almost universal finding that if a very large number of (healthy) people are given a very large range of psychometric tests, the resulting correlation matrix will tend to show more positive correlations than one would expect by chance. This effect is known as **(p.218)** 'positive manifold'. Duncan follows Spearman (1927) in interpreting this positive manifold as evidence for a single cognitive process or function that is used in many (if not all) apparently different situations. This process, ability or function was called 'g' by Spearman (short for 'general intelligence'; see Duncan and Miller 2002 for further details).

It has been known for many years, however, that a single underlying function is not the only possible explanation of positive manifold. It is perfectly possible to get the same effect if there are many (but a limited number of) different cognitive processes involved in different tasks, and any one task samples from a different subgroup of them. In short, positive manifold is not proof of a single core cognitive process, yet the notion of 'g' (or fluid intelligence) persists to this day as one possible explanation of a very prevalent finding.

So what is the evidence that Duncan uses to demonstrate that the frontal lobes play a critical part in 'g'? The first is his demonstration using three frontal lobe patients. These people showed planning and organisational problems in everyday life despite normal performance on executive (e.g. verbal fluency, WCST), memory, language or perception tests, and also performed normally on the WAIS (Duncan, Burgess and Emslie 1995). However they performed poorly on Cattell's Culture Fair intelligence test, which was designed as a measure of 'fluid intelligence'. They also performed poorly on multitasking tests such as the Six Element Test (Burgess *et al.* 1996b) and the Multiple Errands Test (Shallice and Burgess 1991a).

In a second study, Duncan *et al.* (1996) found that a small group of frontal patients showed increased 'goal neglect' on a difficult speeded task involving searching arrays whilst also maintaining attendance to switching signals. Goal neglect was defined as 'disregard of a task requirement even though it has been appreciated verbally' (Duncan *et al.* 1997, p. 716). The patients also showed decrements in Cattell's intelligence test. Duncan *et al.* argued that this association of deficits, together with the finding that in the normal population performance on their goal neglect task is closely related to g, suggests a link between fluid intelligence or g, goal neglect, and the frontal lobes. Or perhaps more correctly, between fluid intelligence, the frontal lobes, and whatever *prevents* goal neglect. This process (i.e. which when damaged leads to goal neglect) is described as a 'frontal process of...constructing an effective plan by activation of appropriate goals' (Duncan *et al.* 1997, p 716).

In a study a year later, Duncan *et al.* (1997) showed generally stronger correlations in a group of 24 head-injured patients between Cattell's Culture Fair test performance and three executive tasks (Six Element Test, Verbal Fluency, and Self-Ordered Pointing) than between the executive tests themselves.[3] Finally, and most recently, Duncan *et al.* (2000) report rCBF changes in lateral frontal cortex when people are performing so-called 'high-*g*' tasks relative to 'low-*g*' ones. This result is used to argue that 'general intelligence results derives from a specific frontal system important in the control of diverse forms of behavior' (p. 399).

Implications for rehabilitation

A prediction from Duncan's view is that if one could somehow improve, circumvent, or provide compensatory aid for the damaged function (i.e. g), then one would expect to see benefit across a whole range of situations. Another way of putting this is that you would expect to see good generalisation of gains. If a task could be found that tapped the crucial processing aspect eeffectively, regardless of whether it bore any resemblance to real-world situations, then any improvements on the training task **(p.219)** would be reflected in changes in many activities of daily living. Indeed a logical conclusion would be that if one could improve a subject's performance on the measure of fluid intelligence he uses (Cattell's Culture Fair Test), then they should also show improvement in everyday life.[4]

Is there any evidence then that such generalisation occurs? Stablum *et al.* (2000) trained 10 closed head injury (CHI) and 9 anterior communicating artery aneurysm (ACoAA) patients on a dual task paradigm. Training consisted only of performing the task, and was given once a week for five weeks. Stablum *et al.* report gains in dual-task performance for both patient and control groups. This is not especially surprising since one would expect some practice effect. More surprising however was the improvement of both groups on neuropsychological tasks (e.g. Paced Auditory Serial Addition Task (PASAT), Continuous Performance Test (CPT)) that were not trained. However, there are caveats that could be applied to the results of this study (e.g. there was not a no-treatment patient group, or multiple baseline-type design; improvement on real-world tasks was not measured). Nevertheless it does provide an interesting suggestion worthy of larger-scale investigation.

Alderman (1996) gives a complimentary finding. He administered a series of dual-task paradigms to a group of head-injured patients on admission to a rehabilitation unit that specialises in the treatment of severe behavioural problems. He found that the patients who subsequently failed to benefit from rehabilitation showed significantly greater dual-task decrement (i.e. the amount that performance on one task is affected by having to do another at the same time) at admission than those who did benefit. There was no significant difference between the groups in performance in the single task conditions (which were digits forwards and backwards, the temporal judgement subtest of the BADS, and conversation). There were also small differences between the groups on other executive measures (cognitive estimates, verbal fluency, WCST and Trail-Making). The link between dual-task decrements, dysexecutive behavioural symptoms and frontal lobe lesions is supported by Baddeley and Della Sala (1998), who report that a group of frontal lobe patients with behavioural disturbance showed significant dual-task decrements relative to a frontal lobe group without dysexecutive behavioural disturbance. Performance on verbal fluency or WCST was not significantly different between the groups, although there was a non-significant trend for the patients with dysexecutive behavioural disturbance to be poorer. So perhaps one marker of a resource that is important for many situations in everyday life with executive requirements is dual-task decrement.

If these kinds of accounts are true, there is a potential consequence that clinicians might wish to consider. If no more than merely performing an executive task (as in the Stablum *et al.* 2000 study) can cause training effects that generalise to other executive tasks, then this has implications for assessment as well as treatment. Theoretically, if in one's standard assessment, five executive tasks (for instance) were administered (and in our laboratory we routinely give more than twice this number), one would expect to see order effects in performance on the tasks. Moreover assessment of change over time and the determination of its cause would be problematic. The upside is that one could bask in the knowledge that by giving a patient a standard assessment, one was also rehabilitating them at the same time!

Clearly it is important for rehabilitation that we should know whether this account of the frontal lobe executive system is a good one, since if it is true it should inform our whole treatment approach. However, Burgess and Robertson (2002) outline a series of problems for any single account theory:

1. In group studies of either neurological patients or healthy subjects, correlations between performance on different executive tasks are typically very low (see e.g. Burgess and Shallice, 1994; Robbins 1998; Miyake *et al.* 2000).
2. Group studies also show that different behavioural symptoms of the dysexecutive syndrome tend to cluster together rather than all loading on one factor (Burgess *et al.* 1998).
3. (p.220) At the single case level, symptoms such as confabulation or multitasking deficits may be seen independently of virtually any other signs (e.g. Shallice and Burgess 1991a; Burgess and McNeil 1999; Burgess *et al.* 2000).
4. Also at the single case level, deficits such as response suppression and initiation problems can doubly dissociate (Shallice 1988) on executive tests (e.g. Burgess and Shallice 1996c).
5. Different behavioural symptoms are associated with performance decrements on different clinical executive tasks (Burgess *et al.* 1998).

6. As a group, frontal lobe patients can show a range of different forms of error on the same executive test (e.g. Burgess and Shallice 1996a, c; Stuss *et al.* 2000).
7. Brain lesions in different parts of the frontal lobes can be associated with decrements on different executive tasks, and with different types of failure (e.g. Burgess *et al.* 2000; Stuss, Eskes and Foster 1994; Stuss *et al.* 1999; Stuss and Alexander 2000; Troyer *et al.* 1998).
8. Functional imaging and electrophysiological studies of the frontal lobes suggest potential fractionation of the executive system (see Picton, McIntosh and Alain 2002; D'Esposito and Postle 2002).

Together, these results suggest that although there may well be cognitive control/executive processes that are used in many different situations as the single process and construct-led accounts claim, it is doubtful that they can be complete accounts of the entire frontal cognitive system (as the authors themselves generally admit). As a consequence, some theorists have presented more complex models that attempt to take these potential fractionations into account: the multiple process theories.

Multiple process theories

These are theories that propose that the frontal lobe executive system consists of a number of components that typically work together in everyday actions, but can be examined relatively independently in experimental studies. There are large aspects of common ground amongst the various models. They take (often implicitly) as a starting point an automatic/routine vs. controlled/novel distinction in the organisation of thought and behaviour. Frontal lobe cognitive processes are aligned with the operation of controlled processing, and the purpose of these processes is to deal with novelty.

Fuster's temporal integration framework

Fuster (1997; 2002) provides one of the most concisely articulated examples of this type of theory. This states that

> The prefrontal cortex is essential for the formulation and execution of novel plans or structures of behaviour. These gestalts of action, with their goals, are represented in neuronal networks of this cortex in the form of abstract *schemas*. The simpler components of those structures of action are represented in frontal or subcortical networks at lower levels of the motor hierarchies.
>
> Fuster (1997, p. 251)

The frontal cortex exerts its influence through connective and reciprocal links with posterior cortical regions, and the overall frontal system performs three functions:

1. Working memory: the provisional retention of information for prospective action. This function is mainly supported by dorsolateral prefrontal cortical areas (DLPFC).

2. Set: The selection and preparation of particular (established) motor acts. This is also supported by DLPFC, and also the anterior medial cortex.

(p.221) 3. Inhibitory control: This function serves to suppress interference, either from external distractors, or from internal inappropriate sensory and motor memories, and is supported primarily by the orbitomedial PFC.

Stuss's anterior attentional functions

The idea in Fuster's model that the frontal lobes serve control functions over more basic schemas[5] is one of the enduring ideas in modern frontal lobe theorising (e.g. Luria and Homskaya 1964; Norman and Shallice 1986). Recently, Stuss *et al.* (1995) expanded upon how they see the relationship between the schema and the executive system might operate (see also Stuss *et al.* 2002).

Stuss *et al.* (1995) describe a schema as a network of connected neurons that can be activated by sensory input, by other schemata,[6] or by the executive control (i.e. frontal lobe) system. In turn, it can recruit other schemata to cognitive control processing so as to produce its required response(s). In addition they suggest that schemata provide feedback to the executive system concerning its level of activity. Different schemata compete for the control of thought and behaviour by means of a process called 'contention scheduling' (a concept described originally by Norman and Shallice 1986) and is mediated by lateral inhibition. They suggest that each schema contains multiple internal connections, some of which provide internal feedback. Once activated, a schema remains active for a period of time depending upon its goals and processing characteristics. This might be only a few seconds in situations such as reaction-time tasks. But over longer periods that require activity without triggering input, activation has to be maintained by repeated input for the executive control system.

The focus of Stuss *et al.*'s theorising is attention. They propose seven different attentional functions, each of which have their own neuronal correlates:

1. sustaining (right frontal),
2. concentrating (cingulate),
3. sharing (cingulate and orbitofrontal),
4. suppressing (DLPFC),
5. switching (DLPFC and medial frontal),
6. preparing (DLPFC), and
7. setting (left DLPFC).

Shallice's supervisory attentional system

The notion that the frontal lobes are crucially involved in attention is also reflected in one of the most influential modern theories of frontal lobe function. This theory was developed by Shallice and his colleagues over the last 20 years (e.g. Norman and Shallice 1986 [initially published as a technical report in 1980]; Shallice 1988; Shallice *et al.* 1989; Shallice and Burgess 1991a, b, 1993, 1996; Burgess and Shallice 1997; Burgess *et al.* 2000). The use of the term attention in this theory is broad, and refers in a general sense to the allocation of processing resources (Shallice 1988).

The first version of this theory (Norman and Shallice 1986) was principally concerned with outlining in broad terms the organisation of the executive control system over well-rehearsed behavioural (and thought) routines. There were four levels of increasing organisation. The first level consisted **(p.222)** merely of 'cognitive or action units', which were the basic abilities one has (e.g. reaching for an object, reading a word). Schemata existed at the second level. These were nests of these units that had come to be closely associated through repetition, as described above. The third level was a process called 'contention scheduling'. This was the basic triggering interface between incoming stimuli (including thoughts) and the schemata. Its purpose was to effect the quick selection of routine behaviours in well-known situations. However, of course, many situations (or aspects of them) that we encounter are not well-rehearsed. In this situation one has to consciously decide what one has to do. The cognitive system that effects this conscious deliberation was called the 'supervisory attentional system' (SAS).

In the early versions of the theory, the SAS was merely represented as a single entity. This was not because it was thought that the system comprised only one process or construct, but merely that there was little empirical evidence at that time concerning potential fractionation (see points 1–7 above). Most recently, the putative organisation of the SAS has been articulated in more detail (Shallice and Burgess 1996). In this model, the SAS plays a part in at least eight different processes: working memory; monitoring; rejection of schema; spontaneous schema generation; adoption of processing mode; goal setting; delayed intention marker realisation; and episodic memory retrieval (see Figure 18.1).

Implications for rehabilitation

The strength of the multiple process theories is that they encapsulate the results from many different types of studies, and they attempt to explain behaviour in many different kinds of situations. As such they stand a better chance of explaining a variety of dysexecutive symptoms (see Table 18.1). Their disadvantage is that they are difficult to disprove. If a new dissociation between tasks or symptoms is found, it is easy to either bolt on another process to the theory, or explain it as a refinement of one of the existing concepts. It is difficult to see at what stage such a theory would ever be completely rejected.

(p.223) For rehabilitation the suggestions of these models is more complex than for the single-process or construct-led theories. These theories suggest that one first needs to isolate the locus of the impairment(s). In

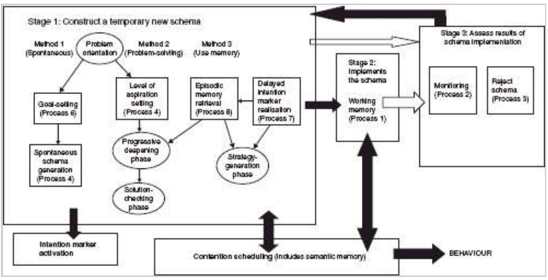

Figure 18.1 The Shallice and Burgess (1996) update of the Norman and Shallice (1980)

practical terms, this would require administration of the sets of procedures upon which the model was based (or as close as is practically possible). This is likely to be a much more time-consuming procedure than for the other types of theory: however it is less likely that an important impairment would be missed. Moreover, a number of the hypothesised processes are only tapped in certain situations, and one needs to assess a patient in a wide range of them.

"Supervisory Attentional System" model. Temporally distinct phases of supervisory system processing are depicted by circles. An operation (i.e. a change of state of one or more control variables) is depicted on a solid rectangle/square. Solid lines depict flow of control, unfilled lines represent information transfer.

Rehabilitation efforts can then be targeted to the specific situations in which the patient has problems. In this way, the implications for rehabilitation of the multiple process models are quite different from the previous theories, the suggestion of which is that rehabilitation could be performed out of the everyday situations in which the patient has problems. So are there any grounds for establishing a basic minimum set of investigations that should be performed as a first stage assessment of the dysexecutive patient?

The results of a study by Burgess *et al.* (1998) may give some hints. In this study, the carers or relatives of 92 mixed aetiology neurological patients were given a questionnaire (the DEX) (Burgess *et al.* 1996a) which asked them to rate the frequency of occurrence of the 20 most common dysexecutive symptoms in the patients they knew well. These symptoms are listed in Table 18.1. When the results were subjected to factor analysis (orthogonal rotation), five factors were selected: Inhibition (principally problems with response suppression and disinhibition); intentionality (everyday deficits in planning and decision-making); executive memory (e.g. confabulation, perseveration); and two purely affective factors – positive (e.g. euphoria) and negative (e.g. apathy) affective changes. A range of neuropsychological tests was also administered to the patients, which allowed examination of the relationships between the scores for these behavioural symptom factors and individual psychometric test performances. Burgess *et al.* found a distinct pattern of relationships between test performances and the behavioural symptoms. Performance on none of the psychometric tests was associated with either of the affective symptom factor scores. However performance on many executive tests (Cognitive Estimates, Verbal Fluency, Trail-Making and the Six Element Test of the BADS) was significantly associated with Inhibition factor scores, as were WAIS Full-Scale IQ scores. Modified Wisconsin Card Sorting Test (MWCST) performance and verbal fluency was associated with the Executive Memory factor scores. And only one test (out of a total of 22) was significantly associated with the Intentionality factor scores: the Six Element Test (Shallice and Burgess 1991a; Burgess *et al.* 1996b). From these results, Burgess *et al.* (1998) recommended that at the very least an assessment of a dysexecutive patient should include tests that measure each of these symptom clusters (the affective aspects will be dealt with below). Broadly, these would include:

1. A general measure of inhibitory abilities (impairments of which are detected by a wide range of tasks including those of intellectual function).
2. Measures of executive memory abilities both short-term (i.e. working memory) and long-term (i.e. accuracy of episodic recollection).
3. A measure of multitasking ability (the subcomponents of which will be described in detail below, but include planning, and prospective memory – including task switching).

The degree of concordance of these empirical results with the multiple-process theories outlined above is striking. Two of Fuster's three temporal integration functions are closely replicated, and if one takes a broad view of the preparatory set, then there is further agreement. Stuss's inhibition, switching, preparation and attention maintenance aspects are also all reflected in these empirical findings. Shallice's model copes particularly well. It explicitly mentions, as separate processes, inhibition (in the form of both schema rejection and adoption of processing mode); both working memory and (p.224) episodic retrieval; and both components of multitasking (prospective memory as 'delayed intention marker realisation', and planning as the set of processes in method 2 of new schema formation). Thus the multiple-process theories cope generally well with explaining the multitude of symptoms that can follow frontal lobe damage.

They also suggest a general approach to investigating the root causes of everyday dysexecutive impairments. Following formal assessment of function as outlined above, one would examine how the particular impairments contribute to disability in everyday life. This requires componential analysis of the situations that present the greatest problem (as identified, for instance by ABC analysis). It is at this point that it is most unfortunate that experimental paradigms are so often unlike real-world situations (as addressed above), since if this were not the case, much of the work would already have been done for the rehabilitation specialist and this stage would be largely redundant. The final stage would of course be intervention, the exact method of which would depend on the nature of the impairment, the situation in which it manifests itself, the *intact abilities* the patient shows, and other relevant clinical variables. In the next section we will consider examples of the treatment of each of the main variables isolated here (inhibition problems, executive memory problems, and multitasking problems).

However, the foregoing argument raises two important and interconnected matters for the rehabilitation of executive function. The first concerns the relative importance of the various symptoms, and the second concerns the patients' awareness of them. If one considers Table 18.1, it is apparent that some symptoms are reported more frequently than others by carers of dysexecutive patients. Indeed, some (e.g. planning problems) are reported as a problem by relatives in almost half of the cases that formed the sample in the study from which this data was taken (Burgess *et al.* 1998). This is even more remarkable when one considers that this group of patients was not specifically selected because they showed dysexecutive symptoms, but merely because they represented a cross-section of the sort of neurological patients that might typically be encountered in clinical practice (consisting of 59 per cent head injury; 13 per cent dementia; 8.5 per cent CVA; 6.5 per cent encephalitis; 13 per cent other conditions). Clearly one would ideally first develop treatment methods for, and target efforts towards, those symptoms that are most often observed, since these are *ipso facto* most likely to be problems in everyday life.

A frequently encountered problem, as Table 18.1 shows, is that patients are often unaware of the extent of their problems. This is not a problem that is confined to executive dysfunction: many amnesics or people with neglect etc. may be unaware of their problems, at least in the early stages of their disability. However, it is a difficulty that is both prevalent and persistent in this area, as Table 18.1 shows (see also Prigatano 1991). This should at least in part guide the agenda for dysexecutive patients' rehabilitation. All other matters being equal, treatment of a problem that the patient does not notice or acknowledge will always be more problematic than for one about which the patient complains. Table 18.1 shows that two of the symptoms where there is typically greatest disagreement between patients and carers both involve a lack of concern. This presents an interesting conundrum: how does one make a patient concerned that they are unconcerned? Perhaps considering the item of greatest absolute disagreement between patients and carers provides an answer: planning problems. One might in a simple-minded fashion suggest that a consequence of not considering the future, or the future consequences of one's actions, is likely to be a lack of concern. So if one can first facilitate this planning function, the problem with concern is likely to show concomitant improvement. So in this example, it would make most sense to target one's rehabilitation efforts first towards the deficits in planning. This argument suggests a further dysexecutive rehabilitation principle should be that when choosing which dysexecutive behaviour to treat first, do not just consider which is the most troublesome. Also consider the order in which the symptoms should be treated (see the example of Alderman and colleagues' SMT training above).

(p.225) Single-symptom theories

It is probably significant for this area of science to note that there are a greater number of general theories that seek to explain many dysexecutive symptoms at once, than there are theories more modest in their ambitions. Nevertheless there are a few areas where theorising about specific symptoms has developed to the point where it could be used to suggest rehabilitation methods. A full review of the rehabilitation implications of all possible theories of all possible dysexecutive symptoms would be beyond the scope of this article. However, as an example we will take two symptoms that are both quite common, and also for which theorising is reasonably well developed: confabulation and multitasking deficits.

Confabulation

One of the more detailed theories of confabulation is given by Burgess and Shallice (1996b). They reasoned that if confabulation is a consequence of damage to the memory control processes involved in autobiographical recollection, one should be able to demonstrate their influence in people who do not confabulate. So a group of subjects were asked to reflect aloud upon their thought processes as they tried to answer a series of simple autobiographical questions such as ‘What were you doing for the two hours before lunchtime last Sunday?’ The participants' reflections were then transcribed and each statement was classified as belonging to one of 25 different utterance types. By examining the probability of the frequency of any two statements following in sequence, it was possible to establish a prototypical recollection structure. It was found that there were three broad classes of control processing statements that were relevant to the recollection process. They could be defined in terms of their decreasing proximity to memories in the recollection structure, respectively: ‘descriptor elements’ (i.e. specifications of what it is that is being asked of the memory store); ‘editing elements’ (i.e. verification, checking and comparison operations) and ‘mediator’ elements (i.e. problem-solving routines). On the basis of these findings, Burgess and Shallice (1996b) proposed the model shown in Figure 18.2, and argued that the consequence of failure of these memory control processes would be confabulation. Indeed, it was argued that some of the errors that normals quite commonly make are similar to some of those that can be seen, with greater frequency, in confabulation. In this way, the Burgess and Shallice model is a synthesis of aspects of many other leading theories of confabulation (e.g. Papagno and Baddeley 1997; Dalla Barba *et al.* 1997; Moscovitch and Melo 1997). It predicts that a variety of forms of confabulation may be seen, either singly or in combination: those who show problems with self-initiated retrieval; those whose retrieval processing is insufficiently constrained; and those who cannot distinguish between real memories and fantasies and other thoughts. The Burgess/Shallice theory has since been used to explain the pattern of confabulation in a number of cases (e.g. Burgess and McNeil 1999; Dab *et al.* 1999; Worthington 1999).

Implications for rehabilitation

Most confabulators tend to stop confabulating of their own accord (Schnider *et al.* 2000). However some do not, and clinical experience suggests that the speed of recovery in those who will in any case improve may well be increased by cognitive intervention. Different treatment approaches are suggested by the Burgess/Shallice theory. Those whose problems are principally at the descriptor level are characterised by the ability to remember information if given sufficient cues. They are probably best helped therefore by methods to ameliorate the need for self-initiated retrieval, e.g. training in the strict use of a diary, and with reminders such as the Neuropage system (Evans, Emslie and Wilson 1998). Fortunately these methods are also likely to be of benefit for those with ‘editor’ impairments, **(p.226)**

whose memory system tends to run unfettered. But experience suggests that the key element for these patients is feedback: knowing one has a tendency to produce erroneous memories soon leads to a more cautious approach to recall, which is sometimes all that is needed. The most difficult group of confabulators is likely to be those with mediator-type impairments. These patients suffer from more generalised dysexecutive impairment, one aspect of which is manifested in their memory recall. It is likely that these patients would be helped by a more general approach rather than one which just aimed to deal with their disability in this one situation.

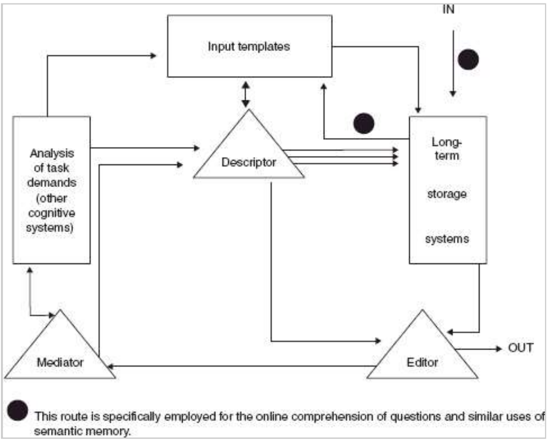

Figure 18.2 The Burgess and Shallice (1996b) model of the control processes involved in autobiographical recollection.

These predictions are largely speculative at present, and are presented in the spirit of trying to suggest links between theory and practice. However there is some early evidence for the effectiveness of these simple methods. For instance, Burgess and McNeil (1999) report the successful treatment of a patient (B.E.) who confabulated following an operation to clip an anterior communicating artery aneurysm. His particular form of confabulation was unusual in that it was stable and restricted to just one part of his life, as the following description shows:

> On one of the first days after returning home, B.E.'s wife found him getting out of bed in the morning and dressing in formal clothes. This was rare for him since he was a shopkeeper. However in addition to his shopkeeping, he used to earn extra money by performing stock-takes for other shops with his business partner. On these occasions he used to dress more formally. B.E.'s wife was alarmed, and asked him what he was doing. He replied that he had a stock-take to perform for a local shop. His wife pointed out that he had only just returned from hospital after recovering from major surgery, and told him that no arrangements had been made for his (p.227) involvement in stock-taking for the foreseeable future. However B.E. said that he remembered having a conversation with his business partner the day before when he had arranged to do some stock-taking. His wife knew that this was false, but B.E. was only stopped when he finally was at the door ready to leave (despite his wife's protestations), but couldn't remember exactly where it was that he thought he should be going.

The remarkable thing about this case was that these events occurred every day (and sometimes twice a day) for three weeks until intervention was instituted. This was causing great difficulty between him and his wife.

For treatment, B.E. was trained to keep a diary in which he wrote a detailed account of his daily activities including meals he had eaten, television programmes he had watched, and particularly any telephone calls he had made or received, however trivial they seemed at the time. This helped in two ways. First it provided a record he could check to see if had actually had the telephone conversations he was erroneously remembering. But it also helped because the additional details in his diary served as a cue for him to be able to remember things about the day before that he could not remember without prompting (i.e. a 'descriptor impairment', above). This enabled him to piece together the events of the day and usually remember a real memory that conflicted with the erroneous one. He could then reject the confabulatory memory with more confidence. This simple technique had dramatic effects in a relatively short period of time (five weeks). Although at the end he reported that he sometimes still 'felt' like he might have performed a stock-take recently, he did manage to stop himself acting on the confabulations, and the conflicts with his wife no longer occurred.

This theory-motivated intervention was just a first step. Moreover, it is clear that some paramnesic phenomena are less amenable to current treatment methods than others (see Burgess *et al.* 1996c). However it does at least suggest that our theoretical understanding of this fascinating disorder is beginning to reach the level of sophistication where rehabilitative methods might soon be developed that will be effective for at least some confabulators.

Multitasking deficits

Mesulam (1986) articulated an observation that had been discussed clinically for many years, but never formally investigated. This was that some frontal lobe patients may show marked problems with higher-level cognitive functions such as planning and organisation in everyday life, but appear normal or near-normal on formal laboratory testing. The years since Mesulam's observation have seen considerable interest in these cases, partly because they have presented a problem for rehabilitation and medico-legal assessment of disability, and partly because they are good evidence of the potential fractionation of the executive system.

As a consequence, we now know a fair amount about the neuropsychological profile of these cases (see Burgess 2000a, for review). The problems that are noticed in everyday life by carers and relatives are typically reported as: tardiness and general disorganisation, unreliability, absent-mindedness (by which is usually meant prospective memory failures) and failures to complete even quite basic tasks such as shopping or preparing a meal. Typically, tasks are started but never completed, with patients often breaking off in the middle of one task to start another, but never returning to the first. Despite these obvious difficulties in everyday life, they may fail none of the traditional tests of executive function (e.g. WCST, fluency, cognitive estimates, Tower of London; Stroop; Trail-Making), nor show any problems on formal testing with WAIS Full-Scale IQ, language, memory, motor or visuospatial perception.

Shallice and Burgess (1991a) explained this pattern as being due to the fact that traditional neuropsychological tasks are typically very well-structured (see Goel *et al.* 1997; Burgess *et al.* 2005, for discussion on this point). More specifically, they do not typically require the following: the creation, **(p.228)** maintenance and execution of delayed intentions; the ability to recognise the need for, selfinitiate and carry out complex meta-strategies; dovetailing of tasks to be time-effective; prioritisation of tasks; and deciding for oneself in the absence of feedback whether a result is satisfactory. In other words, traditional executive tests do not measure many of the abilities that make a person effective in the real world.

The cardinal situation that does tap these characteristics, however, can be called 'multitasking'.[7] Burgess (2000b) describes eight features of the situation, the first five of which are axiomatic, plus a further three (6–8) that are usually true of everyday life multitasking:

1. Many tasks: A number of discrete and different tasks have to be completed.
2. Interleaving required: Performance on these tasks needs to be dovetailed in order to be time-effective.
3. One task at a time: Due to either cognitive or physical constraints, only one task can be performed at any one time.
4. Delayed intentions: The times for returns to task are not signalled directly by the situation.
5. No immediate feedback: there is no moment by moment performance feedback of the sort that participants in many laboratory experiments will receive. Typically, failures are not signalled at the time they occur.
6. Interruptions and unexpected outcomes: Unforeseen interruptions, sometimes of high priority will occasionally occur, and things will not always go as planned.
7. Differing task characteristics: tasks usually differ in terms of priority, difficulty and the length of time they will occupy.
8. Self-determined targets: People decide for themselves what constitutes adequate performance.

The most typical multitasking test used in the clinic is the Six Element Test (see above). In this test, participants are given three simple sets of tasks (e.g. dictation, writing down the names of some common objects), each of which has two sub-components, A and B. (For instance A and B of dictation task are describing a holiday and describing any memorable event). Subjects are given 10 minutes for the task overall, and are told three things: First, it is not possible to do everything in the allotted 10 mins; second, but you must try to do at least some of each subtask. Third, you are not allowed to do an A section of a task followed immediately by the B section of the same task.
Clearly, dealing with situations like these may require a number of different behavioural stages. However, since a given process may be used at many different stages, it may be that the processing demands may be characterised more simply than might at first be imagined. In fact there is some evidence that this is the case. Burgess *et al.* (2000) administered a procedure similar to the Six Element Test (called the Greenwich procedure) to 60 people with circumscribed cerebral lesions and 60 age- and IQ-matched controls. In addition to the basic test measures, the task was given in a way that allowed consideration of the relative contributions of task learning and remembering, planning, plan-following and remembering one's actions to multitasking performance. A basic finding was that this sort of procedure is sensitive to a range of cognitive

problems – despite no differences between the controls and patients on measures of premorbid (NART) or current fluid intelligence (Raven's Advanced Progressive Matrices), the patients showed significant impairment on most of the variables (a similar finding is reported by Levine *et al.* 2000). At a more specific level, lesions in different brain regions were associated with impairment at different stages in the multitasking procedure. Lesions to a large region of superior posterior medial cortex including the left posterior cingulate and forceps major **(p.229)** gave deficits on all measures except planning. Remembering task contingencies after a delay was also affected by lesions in the region of the anterior cingulate, and rule-breaking and failures of task switching were additionally found in people with lesions affecting medial and more polar aspects of Brodmann's areas 8, 9 and especially 10. A theory of the relationships between the cognitive constructs underpinning multitasking was tested using structural equation modelling. The results suggest that there are three primary constructs that support multitasking: retrospective memory, prospective memory and planning. Burgess *et al.* (2000) tentatively suggested that left anterior and posterior cingulates together play some part in the retrospective memory demands, while the prospective memory and planning components make demands upon processes supported by rostral PFC and RDLPFC respectively.

It seems likely that the principal contribution of the PFC to multitasking lies with supporting the planning and especially the prospective memory that is required. Impairment of this prospective memory component typically manifests itself in two ways on tasks such as the Six Element Test (SET). First, subjects may fail to switch tasks when they intended.[8] Second, they may break the task rules (in the case of the SET, this means doing two subsections of the same type consecutively). Interestingly, a recent study by Alderman *et al.* (2003) suggests that these two types of failure might be unrelated. Fifty mixed aetiology neurological patients were given a simplified version of Shallice and Burgess's (1991a) Multiple Errands Test (MET). The MET is a real-world shopping test, carried out in a shopping precinct. Participants have to buy several items and carry out errands whilst following a set of quite arbitrary rules. Alderman *et al.* found that the patient group could be divided into those who tended to forget to carry out tasks, but did not break rules, or those who broke the rules but completed tasks. This suggests that different rehabilitative methods should be used for the two groups.

Mechanisms of recovery

We have discussed some of the leading theories of how the frontal lobes exert their control over behaviour. And we have outlined in broad terms some of the implications for the rehabilitation of patients with executive control deficits. In some cases the form of rehabilitation might be termed ‘cognitive prosthetics’: in other words the aim is to improve function rather than ameliorate the cognitive impairment by the use of external aids (e.g. the Neuropage system (Wilson *et al.* 1997)). In other cases, rehabilitation may reduce the need for the impaired ability by changing the environment. A third type aims to establish behaviour patterns that rely on intact systems: in other words to alter the construct

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demands of the old function (for illustration, a rather simple example might be where a patient with severe speech production problems is encouraged to write messages to communicate). A fourth type, however, aims to improve the actual cognitive impairment itself. But how might such an improvement occur in the damaged brain?

The current primary evidence comes from studies of motor skill deficits. In a recent study by Liepert *et al.* (2000), the cortical motor output area of a hand muscle was mapped in thirteen stroke patients using transcranial magnetic stimulation before and after treatment. The rehabilitation consisted of twelve days of constraint-induced movement therapy. They found that before treatment the cortical representation area of the affected hand muscle was significantly smaller than the contralateral side. However, following treatment the muscle output area in the affected area was significantly enlarged, and the motor performance of the paretic limb was improved. Liepert *et al.* (2000) (p.230) claim that the shift in the centre of the output in the affected hemisphere was suggestive of the recruitment of adjacent brain areas (see also Traversa *et al.* 1997).

However, the positive effect afforded by functional reorganisation has an enemy. Nudo *et al.* (1996) discuss how a subtotal lesion confined to a small portion of the representation of one hand can result in a further loss of hand territory in the adjacent, undamaged cortex (in squirrel monkeys). Fortunately however, retraining of skilled hand use resulted in prevention of the loss of hand territory adjacent to the infarct, and in some instances the hand representations expanded into regions formerly occupied by representations of the elbow and shoulder. These results give a strong impetus for early rehabilitation intervention: it is possible that treatment is not only about recovery of function, but also about prevention of further loss of function (see Kolb 2002, for further discussion).

Interestingly, this may provide one explanation for a common sequence of events in patients who have sustained frontal lobe damage, but are otherwise

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goodwill amongst friends and relatives, who have often had enough of the tiresome behaviour, and the patient's failure to move on in their lives. This may settle into a longer-term mild to moderate social isolation, with the patient seeming more impaired many years on than they were initially, thus contradicting the truism of rehabilitation that some spontaneous recovery can always be expected. The most obvious explanation for this sequence of events is the interaction between the patient's behaviour and its social and financial consequences. However the recent experimental findings raise the possibility that following the initial insult, there is further detrimental neural change.

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In any case from a theoretical standpoint there is good reason to be concerned about the onset of social isolation. Many theorists believe that a principle function of the frontal lobes is to deal with novel situations (see Burgess 1997 for a review). For this reason, finding tasks that can serve as foci for rehabilitation efforts is difficult: once someone has performed a given problem-solving task, it becomes less novel, and therefore stresses less the processes one intends to address. However, social interaction fulfils most of the criteria one would want as a 'rehabilitation exercise' since no two interactions are exactly the same (novelty). Moreover, successful interaction requires representing the states of mind, moods and emotions of others, self-generation of thoughts and words, fine judgement, and often some basic planning, inhibition and so forth. In other words very many of the functions of the frontal lobes. For this reason, there is good theoretical reason to recommend as important for rehabilitation that the dysexecutive patient receives much social interaction.

Discussion

The predictions from pure theory for rehabilitation that have been covered in this chapter are summarised in Table 18.2. These are extractions from pure theoretical work and as such should be treated with caution: they certainly do **not** have the status of being clinically tested guidelines. Our aim is, however, to try to bridge the gap between the basic scientific research in the area and clinical practice. This gap often appears very large, perhaps insurmountable. However it is our contention that this is a mirage created partly by the differences in terminology between the two areas and partly by the differing demands, concerns, and priorities of researchers and clinicians.

It is trite to state that it is difficult to understand how the executive system of the brain works, but true nevertheless. The executive system is the high-level interface between the person and the environment. It is the way that people decide what they want to achieve, decide how they will go on to achieve it, and then assess the level of their own success. In this way the behavioural outcome at any one stage **(p.231)**

Table 18.2 Suggestions for rehabilitation emerging from pure

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(i.e. loss of goal).
2. Some dysexecutive patients fail to carry out intended tasks despite being able to recall (when prompted) what it is that they have to do. Consider the use of simple interrupts in their treatment.
3. Keep instructions and advice simple and unambiguous.
4. Use simple reinforcement and reward techniques if possible.
5. Pre- and post-treatment evaluation of dysexecutive problems requires assessment of competence in a wide range of situations.
6. Generalisation in improvement from one situation to another might be limited.
7. When deciding which dysexecutive problem to treat first, do not necessarily start with the most troublesome.

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8. Consider also the best order, since in the long term treatment of certain problems may benefit greatly by initial groundwork elsewhere.

is influenced by the person's past experience and their current perception of their situation, as well as their pure executive function abilities, and abilities in other areas. It is little wonder therefore that there is such variety in the symptoms that patients will show, and variability in the effects of brain damage to any one region. However this should not mean that there are no underlying guiding principles, and the clinician is in a good position to discover them. As for the importance of developing behavioural and cognitive therapies for rehabilitation of executive dysfunction, it is clear that this should be a priority. Some theorists have argued that the greatest advance in rehabilitation for this area still awaits us: perhaps some practical application of stem cell research, for instance, that will enable the affected area of the brain to 'repair' itself physically. What is often missed in this speculation is that even if structural repair were perfectly achievable, this alone will never lead to *functional* repair. The processes and representations in the brain have evolved over a person's development since birth, and it is implausible that structural repair alone would replicate the effects of this complex process. Instead it seems likely that the analogy would be with physiotherapy, for instance, after successful surgery to repair a serious limb injury. Even though the limb may now have the potential to be functional, it still needs in most circumstances to be trained, and that training has to progress in a safe and progressive way, guided by the principles extracted from our understanding of how the limb, and other relevant structures, operate. This means that even if we do put faith in pure science research to achieve what we cannot at present (i.e. to produce perfect structural repair to the damaged brain), we will still need to understand the principles behind enabling the brain to once again be functional.

It is almost certain that there is no one rehabilitative method alone that is suitable for all impairments, and although for the critical (damaged) processes to be targeted, the training situations don't have to be real-world ones, we need first to know that they tap the processes that are actually used in the real world. In this regard it is vital that practical rehabilitative research continues, and that clinicians and theorists meet and share ideas with each other.

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Trust number 061171. It is based in part on ideas first presented in Burgess and Robertson (2002). We are grateful to Barbara A. Wilson, Nick Alderman, Jon J. Evans and Hazel Emslie for letting us describe data collected as part of their study in Table 18.1. (p.232)

Notes:

(1) 'Construct' here just refers to an idea. A theoretical characterisation of, or explanation for a set of empirical results. It is unusual for such ideas in this area of science to be expressed in a form where there could ever be a single disproof. Often they are post-hoc explanations of patterns of data, and only rarely does one construct compete directly with another. On occasion, many different constructs may be evoked as explanations of the same data set.

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(2) Current working memory theories of frontal lobe function might also be included under this category, given that it has been used to explain such a wide range of dysexecutive symptoms (especially in non-human primates), and performance in such a wide range of situations.

(3) However this result was largely due to the very low correlation between Verbal Fluency and the Six Element Test (see also Burgess 2000). In fact the correlation between the Six Element Test and the Self-Ordered Pointing Task was actually larger than for two of the three correlations that involved Cattell's test.

(4) Of course the suggestion is not that 'training' someone in the performance of a specific version of this test would be helpful. The improvement would need to be such that it could be demonstrated on versions of the test that had not previously been encountered.

(5) The term 'schema' as used in these theories is generally equivalent to Piaget's (1952) notion of 'schema'. Other practically equivalent terms used in this area are 'scripts' or 'memory organisation packets' (see Schank 1982; Grafman *et al.* 1995).

(6) 'Schemata' is the plural of 'schema'.

(7) Multitasking can be distinguished from 'multiple-task' performance principally by the 'ill-structuredness' of the situation, and by the requirement of the activation of delayed intentions (see Goel and Grafman 1995 and Burgess 2000b for discussion).

(8) Switching an abnormally large number of times also occurs in patients. However the relation of this rare pattern to switch failures is not known.

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