Movement and Allied Disorders in Childhood

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16 Developmental Dysexecutive Syndrome: Does it Exist? A Neuropsychological Perspective

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ACTION CONTROL: NORMAN AND SHALLICE'S MODEL

In 1980, Norman and Shallice[1] proposed a general account of the control of action. The model was intended to account both for everyday slips of action (such as reaching for your keys at your front door, even when the door is not locked) to neurological disorders of action control (such as the perseverative behaviours of patients with frontal lobe damage). A schematic summary of their model is shown in Fig. 16.1.

Essentially, the model assumed that actions can be controlled in two

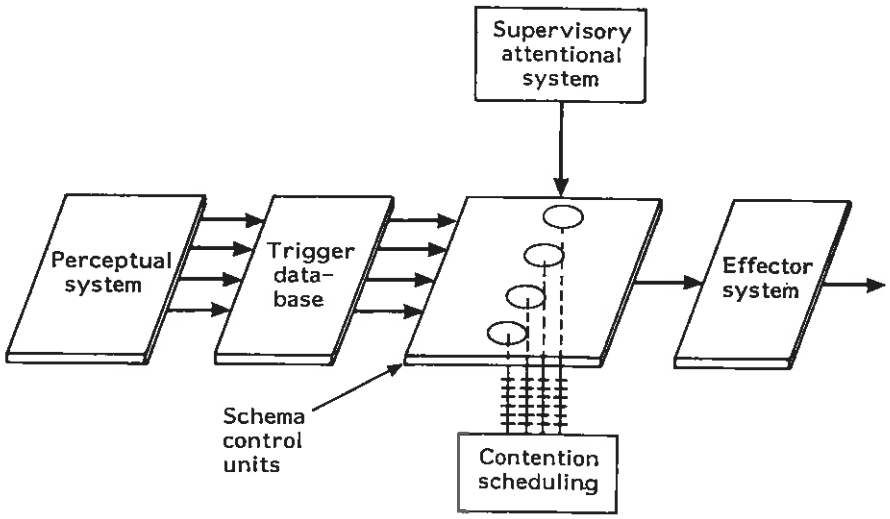

Figure 16.1. A schematic summary of Norman and Shallice's model of control of action

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different ways. The first way is most clearly seen during well-learned, routine skills, in which strong learning effects allow actions to be performed relatively 'automatically'. An example that is often cited is driving a car. Experienced drivers can usually drive familiar routes whilst doing other things (having a conversation, thinking about a problem at work, etc.), even to the extent that when they stop to think about it, they may not be able to remember anything about the driving itself over the preceding 10 minutes or so. In such situations, action control is occurring in a complex and fluent fashion—the driver was breaking at the appropriate points during the journey, possibly overtaking other cars, changing gear appropriately, monitoring speed, and avoiding parked, obtruding vehicles, etc. Since the context for such driving is highly familiar, the 'automatic' action control system functions very successfully.

In Norman and Shallice's model, this automatic level of action control is explained as follows: well-learned actions are each represented by action-schemas, and built into each schema is information about their relative importance in given familiar contexts. It is the action-schemas which 'drive' the actions. As the contexts change, a process which the authors call 'contention scheduling' operates, whereby different possible schemas 'contend' with one another to determine which will be activated and executed.

However, the model also assumes that when the context becomes less familiar—for example, when novel events occur, then 'willed' action control can operate. This second action control system effectively allows the organism to act in a more 'deliberate' fashion, overriding the automatic activation of routine action schemas. This second system is called the 'supervisory attentional system' (or SAS). Thus, in the driving example above, on perceiving a less familiar event—e.g. a child who has fallen over in the street, the driver might stop the conversation s/he is engaged in, and 'decide' to stop the car and get out to help the child. It is the SAS that allows for such conscious decisions.

In Norman's subsequent work[2], this model was applied to the everyday *slips of action*, such as driving towards work on a weekend, when one really intended to drive to the supermarket. In this example, the routine action-schema for driving to work is activated by perceptual input but without any supervisory control, leading to the error. In contrast, Shallice[3] extended the theory in neuropsychological directions. He argued persuasively that patients with frontal lobe damage have a deficit in the SAS, leading to abnormalities in the planning, organizing and control of willed action. We turn to explore this neuropsychological direction in more detail next.

ACTION CONTROL AND FRONTAL LOBE DAMAGE

Three tasks on which patients with frontal lobe damage typically show difficulties are the Wisconsin card sorting test (WCST), the test of

verbal fluency, and the Tower of London. These will now be briefly reviewed.

In the WCST, the subject is presented with a pack of cards with different numbers of shapes on each. Some shapes are circles, some triangles, some crosses or stars. The shapes vary in colour, number and form. The patient is asked to sort the cards into piles by following a rule. In the standard version the patient is not told the rule, but is given feedback as to whether s/he is correct or incorrect. In this way, the patient infers the rule the experimenter has in mind. (In modifications of this task, the patient is actually told the rule to sort the cards by.) The rule might be 'sorting by colour'. When it is clear that the patient is following this first rule, then the rule is changed without prior warning (e.g. sorting by number). The patient must now inhibit the 'learned' rule and discover the new rule. Again, once this has been followed clearly, the rule is again switched to a new one. Normal subjects find such rule-switching fairly easy and make few errors. In contrast, patients with frontal lobe damage typically persevere with the first rule they use, even when asked to try to switch to a new rule[4,5]. The brain region implicated in this rule switching is the left dorsolateral prefrontal cortex.

Similarly, on the verbal fluency task, patients are asked to come up with as many words as possible beginning with a specific letter (e.g. 'F'), in a one-minute period. Typically, patients with frontal lobe damage will produce relatively few new words (three or four), instead repeating the same few words that initially come to mind[6]. The normal person typically can produce at least a dozen, and usually as many as 30 or 40. The frontal lobe damaged patient may also break the rule and/or seek cues in the external environment. The brain region implicated in this verbal fluency task is the orbitofrontal cortex.

On the Tower of London problem (which is an adaptation of an older game, the Tower of Hanoi), the patient is shown a model of three pegs, with three differently coloured balls arranged on the pegs. This arrangement is called the 'initial position', and the patient is told to move the balls in order to reproduce a new pattern (the 'goal position'), in the fewest number of moves, and by only moving one ball at a time. Patients with left anterior frontal damage perform worse on this task[3].

Shallice's hypothesis is that such patients begin an activity, but then cannot interrupt it to change their behaviour flexibly, because they have an impairment in the SAS. The SAS in their case cannot interrupt ongoing actions to 'deliberately' switch to new actions. Thus, regarding the WCST, the patient with damage to the SAS continues to sort according to the previous rule, rather than changing to the new rule. Regarding the verbal fluency task, the idea is that because this is an unfamiliar task, there are no overlearned strategies for retrieving the information from memory, so the subject must produce novel retrieval strategies, normally a function of the SAS. Regarding the Tower of London task, the suggestion is that the task

requires conscious planning, and inhibition of action prior to planning. Deficits in the SAS neatly explain four kinds of error: perseveration, poor generativity, impulsivity, and poor planning.

Shallice's hypothesis was also intended to account for a fifth symptom that such patients may show: distractibility. This is explained as follows: in the absence of an SAS, action-schemas can be triggered by any passing stimulus in the environment. Paradoxically, then, an impaired SAS can lead to two 'opposite' symptoms: perseveration and distractibility. The most extreme form of distractibility is seen in what L'Hermitte[7] described as 'utilization behaviour', where the patient grasps and uses any object that is presented to him or her, regardless of whether it is appropriate to do so. For example, seeing the clinician's spectacles lying on the table, the patient will pick them up and put them on his or her own face. Utilization behaviour has been reported in patients with frontal lobe damage (although many patients with frontal damage do not show utilization behaviour). Note that utilization behaviour has also been reported in patients with thalamic damage[8].

ACTION CONTROL AND WORKING MEMORY

Baddeley[9] makes an interesting link between the SAS from Norman and Shallice's model and the 'central executive' component in his own model of working memory. Here a small digression to summarize Baddeley's model of working memory will be made. Essentially, Baddeley's model proposes that short-term memory (STM) acts primarily as a working memory system, with three components (shown in Fig. 16.2). The first component is the central executive, which controls the allocation of attention and which coordinates the other two components, characterized as 'slave systems'. The two slave systems are summarized as follows:

1. The 'phonological loop'—this contains a phonological store, capable of holding phonological information-trace for about 1–2 s; and contains an articulatory control process, which reads such phonological traces and can rehearse them. It can also convert written information into a phonological code, and place it in the phonological store.
2. The 'visuo-spatial sketch pad'—this is held to be responsible for setting up and manipulating visuo-spatial images. Evidence from dual task paradigms suggest this is independent of the phonological loop.

In Baddeley's model, the central executive is held to allocate attention 'at will' to one or other of these two slave systems, enabling the subject to concentrate on phonological or visuo-spatial information processing. As mentioned above, Baddeley equates the central executive with the SAS. If this proposal is correct, then patients with frontal damage, who are held to

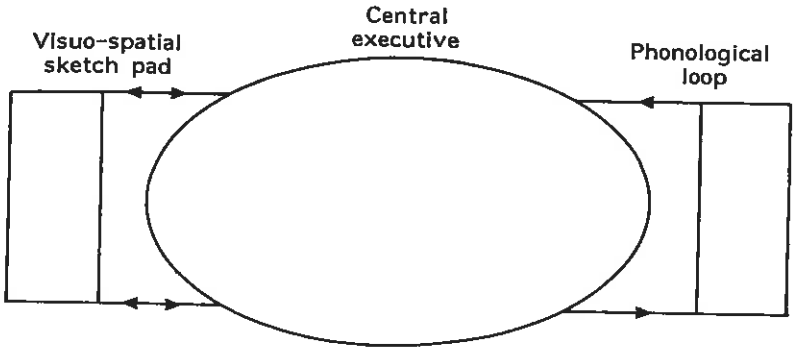

Figure 16.2. The three components of Baddeley's working memory system

have an impaired SAS, should also have problems in working memory. Shallice[5] and Baddeley and Wilson[10] summarize the evidence for 'frontal amnesia', and the evidence tying it to impairments in the SAS/central executive. On the basis of the evidence for SAS/central executive involvement in both attention-shifting, planning and working memory, Baddeley[11] coined the term *dysexecutive syndrome* to describe those patients with an impaired SAS/central executive.

DEVELOPMENTAL DYSEXECUTIVE SYNDROME

One obvious criticism of the SAS model is that it raises the 'homunculus' problem. That is, it implies that there is a system in the brain which controls all other systems, and which does so by exercising 'choice'. Whenever neural systems resemble homunculi, they pose problems, for the obvious reason that it is not clear what is controlling the homunculus. Another little homunculus inside the first one? An infinite regress looms large here. However, this argument can be levelled at many modular neurocognitive systems, and is not a fatal criticism. It simply means that the model needs greater specification in order to explain how the homunculus problem is to be avoided.

For the moment, let us accept both Shallice's and Baddeley's proposals about acquired frontal lobe damage in adulthood, and dysexecutive syndrome. Here we asked the question 'Is there a disorder which is a *developmental* instance of dysexecutive syndrome?' We aim to answer this question by briefly reviewing studies in which tests of the SAS have been used with clinical populations in childhood. In particular, we focus on autism, Gilles de la Tourette syndrome (GTS), attention deficit and hyperactivity disorder (ADHD), obsessive–compulsive disorder (OCD) and phenylketonuria (PKU). We approach this review highly selectively, by

simply considering the use of the three tests above, as they have been applied to these five childhood neuropsychiatric samples.

AUTISM

Autism is a neurodevelopmental disorder characterized by abnormal social and communicative development, an absence of pretend play in childhood, and the presence of repetitive, perseverative, quasi-obsessional behaviour[12–14]. It is the latter symptoms that make it plausible that frontal lobe damage is involved in the disorder, given the resemblance to perseverative behaviour in adults with frontal damage. Adults with autism are significantly impaired on the WCST, relative to normal controls[15,16], and on the verbal fluency test, relative to dyslexic and normal controls[16]. In the latter study, the mean number of categories completed by the subjects with autism on the WCST was $x = 1.7$ (SD = 1.77), whereas for the dyslexic control subjects, the mean number of categories completed was $x = 4.13$ (SD = 1.19). Similarly, on the verbal fluency test, the mean number of words generated by the subjects with autism in 1 min was $x = 30.8$ (SD = 10.21), whereas for the subjects with dyslexia the mean number of words generated was $x = 37.73$ (SD = 9.8). Two studies[17,18] also reported a deficit on the WCST by subjects with autism, and the latter study[18] also found deficits on the other SAS test, the Tower of Hanoi.

Using a related measure (the Stockings of Cambridge, in which the subject has to move three balls from one 'billiard stocking' presented on a computer screen into another one, to match a new pattern of balls), Hughes and colleagues[19] also found deficits. Ozonoff and colleagues[20] also found that such executive function measures differentiated siblings of children with autism from siblings of children with mental handicap, suggesting that since autism has a genetic component[21], there may be a genetic abnormality in the development of executive function/the SAS in children with autism and their close relatives.

ATTENTION DEFICIT AND HYPERACTIVITY DISORDER (ADHD)

ADHD is a childhood disorder diagnosed when there are developmentally inappropriate degrees of inattention, impulsiveness and hyperactivity. The symptoms are usually present in a range of settings but can be confined to just one (e.g. at home). It is a common disorder, possibly occurring in as many as 3% of children, and is 5–10 times as common in males as in females in clinic populations (and three times as common in community samples). The disorder has its onset before the age of 7 years. The diagnosis is excluded in the presence of pervasive developmental disorder including autism. The diagnosis is only made if the behaviours are excessive for that child's mental age, and not only his or her chronological age. Thus, care

must be taken in diagnosing ADHD in children with mental handicap. (For a full review of ADHD, see chapter 5.)

Chelune and colleagues[22] found that children with ADHD performed worse on the WCST than matched controls, and this was also reported by Gorenstein *et al.*[23]. This has not been consistently found, however (see Fischer *et al.*[24]; Loge *et al.*[25]). It may be that this impairment is more strongly linked to the conduct disorder that frequently co-occurs with ADHD, since the impairment has been found in children with conduct disorder[26]. Grodzinsky and Diamond[27] found boys with ADHD were impaired on the verbal fluency test, and this has also been reported by Felton *et al.*[28]. (Some failed replications of this have also been reported: see Grodzinsky and Diamond[27] for a discussion.)

PHENYLKETONURIA (PKU)

PKU is a classic inborn error of metabolism in which there is failure to metabolize phenylalanine (Phe) to tyrosine resulting in an accumulation of Phe in the blood, which becomes toxic[29]. (For a full review of PKU see chapter 7.) Early dietary treatment prevents the development of the severe mental handicap which is seen in this condition. However, even with dietary treatment, some mild deficits have been reported in executive function[30]. In this study, a variant of the verbal fluency test was used, namely a semantic fluency test, in which the subject has to name as many members of a specific semantic category as possible in 40 seconds. They also used the Tower of Hanoi. On the semantic fluency test, children with PKU produced a mean of $x = 14.09$ (SD = 4.57) words, whilst the control group produced a mean of $x = 20.82$ (SD = 3.71) words. On the Tower of Hanoi, the group with early-treated PKU were also significantly impaired. Pennington *et al.*[31] also reported that a group of older, early-treated children with PKU showed deficits on the WCST. Recently Diamond[32] has reported similar findings in very young children with early-treated PKU, using infancy measures of executive function.

GILLES DE LA TOURETTE SYNDROME (GTS)

GTS is a tic disorder characterized by the presence of multiple motor and one or more vocal tics. (For a full review see chapters 1, 2 and 3.)

Bornstein[33] found deficits on the WCST in children with GTS, and found that these correlated with obsessive–compulsive rather than the tic characteristics. Baron-Cohen *et al.*[34] found performance on a verbal fluency test task to be unimpaired in patients with GTS but they found impairments on the Tower of London task. In another study[35] they found a more specific deficit in what they term 'intention editing', as tested by the 'yes and no game', in which the subject has to answer a long series of closed questions

without using the words 'yes' or 'no'. Subjects with GTS report more subjective difficulty in stopping themselves answering 'yes' or 'no', and made more errors on this task than normal children who were much younger.

OBSESSIVE–COMPULSIVE DISORDER (OCD)

OCD is a disorder with its onset typically in adolescence or early adulthood, although increasingly it is recognized that it may begin much earlier. The essential clinical feature is the presence of time-consuming obsessions and/or compulsions. Although the possible neurological basis of these symptoms was recognized in the last century, it has been regarded as a neurotic disorder of presumed psychological origin. (For a full review see chapter 4.) More recently, the presence of 'soft' neurological signs and impairments on traditional neuropsychological tests have been recognized.

Zielinski[36] reported that on the controlled oral word association (COWA) test (a word fluency test), and on the WCST, there were no group differences between patients with OCD and matched normal controls. Boone *et al.*[37] also report no group differences. However, Laplane *et al.*[38] reported that the majority of their patients with OCD were significantly impaired on a modified test of verbal fluency. This was also reported by Head *et al.*[39], who also found deficits in OCD patients on the WCST (short form). This was partially replicated by Christensen *et al.*[40].

NORMAL CHILDREN AND NEUROBIOLOGICAL FACTORS

It is worth pointing out that these tests are very sensitive to developmental factors. For example, Levin *et al.*[41] found that between the age groups of 7–8 years versus 9–12 years, major changes are seen on the WCST and on the verbal fluency test in normal children. On the latter test, the 7–8-year-old normal children generated a mean of $x = 15.9$ (SD = 6.3) words in 1 min, whilst the 9–12-year-olds generated a mean of $x = 26.3$ words (SD = 7.9). This implies that the 'growth' of executive function may be continuing until at least the end of childhood, possibly reflecting the maturation of the neural systems involved.

Regarding the neural basis to SAS, it has been reported that the WCST activates the dorsolateral prefrontal cortex (DLPFC) in normal subjects, as shown with single photon emission tomography (SPET)[42,43]. A position emission tomography (PET) study of word finding in four normal subjects[44] showed an increase in left dorsolateral prefrontal blood flow, consistent with an earlier study showing frontal activation[45]. Subjects with schizophrenia (who typically also have difficulty with the WCST) show reduced blood flow in the DLPFC during performance of this task[46]. Morris *et al.*[47] report DLPFC activation using SPET during a Tower of London task, with

normal subjects. It is important to note that whilst frontal areas are implicated, it is likely that extrafrontal areas are also involved.

CONCLUSIONS

This review suggests that none of the five developmental disorders we have considered uniquely constitutes a developmental dysexecutive syndrome, in that, as measured by the three tests of WCST, Verbal Fluency, and/or the Tower of London/Hanoi, all five disorders have been reported to show deficits. Either this must mean that they *all* suffer from developmental dysexecutive syndrome—i.e. that it is a disorder that is so common that it is present in a wide range of neuropsychiatric syndromes—or it means that these tests are very sensitive to almost any kind of pathology. Either way, it throws doubt on the specificity of developmental dysexecutive syndrome.

We suggest that for theories of action control to have any explanatory power in relation to the range of disorders we have reviewed here, far more specific mechanisms will need to be postulated—possibly subcomponents of the SAS—each of which may be uniquely impaired in any specific neuropsychiatric disorder, thus accounting for the uniqueness of the symptoms observed. Our earlier proposal of an impaired intention editor in GTS is one example of this, which we suspect is indeed a subcomponent of the SAS[48]. In autism, the deficit in attributing mental states to themselves and others[49–51] appears to be unique to the syndrome (given an appropriate developmental level), and is therefore unlikely to be secondary to any SAS deficits. Future research needs to focus on demonstrating the specific links between such cognitive architecture, brain and behaviour.

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