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Acta Psychologica 115 (2004) 271–289

acta psychologica

www.elsevier.com/locate/actpsy

Cortico-subcortical contributions to executive control

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Abstract

The term "executive functions" refers to a range of cognitive processes, their common feature being the coordination of information processing and action control. Cortico-subcortical circuits which connect the prefrontal cortex (PFC), the basal ganglia and the cerebellum via the thalamus are believed to serve as neuroanatomical substrates of executive processing. This paper focuses on information processing related to executive functions by the PFC and related subcortical regions. Findings are mainly derived from neuropsychological investigations of brain-damaged patients but also from imaging studies in healthy subjects. There is evidence for subtle differences between these regions with respect to the cognitive mechanisms contributing to inhibition of habitual responses, task management/multitasking and set shifting, although the data base is sparse so far.

PsycINFO classification: 2225; 2340; 2520; 3297
Keywords: Neuropsychology; Executive control; Prefrontal cortex; Striatum; Cerebellum

1. Introduction

The term "executive functions" refers to the coordination and control of cognitive operations to attain specific goals (Logan, 1985; Norman & Shallice, 1986). Neuropsychological research aims to determine the complex architecture of the cognitive processes underlying executive control and their neuroanatomical correlates (Baddeley, 1996). Research is based on the investigation of brain damaged patients, the study of the effects of normal ageing, animal models as well as neuroimaging

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doi:10.1016/j.actpsy.2003.12.010

techniques in healthy humans. In neuropsychology, the term executive functions has long been used as a synonym for frontal lobe function, implying a parallel of the functional and the anatomical which is—however—not necessarily justified (see Baddeley, Della Sala, Papagno, & Spinnler, 1997; Mayes & Daum, 1997), and the terms executive function or executive control are now predominantly used.

The need for an executive control mechanism has been postulated for non-routine situations requiring a supervisory system (Norman & Shallice, 1986) or strategic information processing (Logan, 1985), e.g. in relation to the control of attentional or mnemonic resources (Baddeley, 1996; Baddeley & Hitch, 1974). In contrast to these traditional unitary executive function views, recent approaches have emphasised its multifaceted nature and a division into several potential subcomponents. In a frequently cited classification, Smith and Jonides (1999) distinguished between mechanisms relating to (a) attention and inhibition, (b) task management, (c) planning, (d) monitoring and (e) coding. There is, however, no consensus on the number and the precise nature of subcomponents, and recent research has concentrated on those subprocesses which are relatively well defined in both theoretical and empirical terms and can therefore be submitted to experimental investigation. Following this rationale, this review is based on *inhibition* and *task management* which are thought to represent the most elementary features of executive control (Smith & Jonides, 1999). With respect to *task management*, *multitasking* will also be considered. The former relates to the ability to perform and to coordinate more than one task at a time (e.g. in divided attention settings), while the latter involves the self-generated organisation of several actions to attain a certain goal. In addition, evidence for the neuropsychological basis of *set shifting* is included in this review, because of the strong interest of cognitive science in set shifting procedures and the associated accumulating data base (see Hübner, Kluwe, Luna-Rodriguez, & Peters, 2004). *Set shifting* refers to the control processes involved in the flexible shift of attention and response preparation from one set of stimulus–response (S–R) rules to another. Evidence for the neuropsychological basis of these three subcomponents will be presented in the following sections. Results from standard neuropsychological tests such as the Wisconsin Card Sorting Test (WCST) will not be considered in detail, since such tests address a wide range of different cognitive demands (in addition to executive control) and suffer from poor specificity and selectivity (see Anderson, Damasio, Jones, & Tranel, 1991). They also involve the danger of defining executive dysfunction by reference to tests used to putatively measure it (see Mahurin, 1999).

In parallel to the development of a multicomponent view of executive functions, the idea of the prefrontal cortex (PFC) as the sole substrate of executive processing has been abandoned in favour of a systems view. According to Gazzaniga, Ivry, and Mangun (1998), "Executive functions do not reside in a single structure but result from the interplay of diverse cortical and subcortical neural systems". The systems in question entail reciprocal projections between the PFC and the anterior cingulate cortex (ACC) as well as subcortical structures (e.g. the cerebellum, thalamus and the basal ganglia). The systems perspective was adopted for several reasons. Animal studies using tracing methods have revealed the nature of several closed and open segregated loops connecting prefrontal and subcortical structures; and their potential

functional implications have also been described (Alexander, Crutcher, & DeLong, 1990; Joel & Weiner, 1994; Middleton & Strick, 1997). Patients with lesions outside the PFC frequently show executive impairments (Elias & Treland, 1999; Godefroy et al., 1992; Kramer, Reed, Mungas, Weiner, & Chui, 2002), and functional neuroimaging in healthy subjects performing executive function tasks consistently yields activations in the ACC, the posterior neocortex, the striatum and the cerebellum in addition to the lateral PFC (e.g. D'Esposito et al., 1995; Dove, Pollmann, Schubert, Wiggins, & von Cramon, 2000; Konishi et al., 1998; Taylor, Kornblum, Lauber, Minoshima, & Koeppe, 1997).

Taken together, this paper aims to review the evidence for cortical and subcortical processing, focussing on two related issues. First, are the cognitive processes underlying executive control "diffusely" organised in cortico-subcortical networks, leading to comparable impairments after damage to different components of the networks? Second, if not, what are the specific contributions of the different components? Neuronal circuits can be viewed as hierarchically organised modules with specific contributions of each component to processing and output organisation; disturbances at different levels within cortico-subcortical pathways would thus be expected to lead to distinct neuropsychological changes (see Zoppelt & Daum, 2003). It has generally been argued that fronto-subcortical networks serve to create and maintain representations of novel S–R associations; disruptions may lead to the use of inappropriate prepotent S–R associations (see Godefroy, Cabaret, Petit-Chenal, Pruvo, & Rousseaux, 1999). While the PFC is thought to play a critical role in the dynamic on-line organisation of the cognitive system which allows performance of different tasks in response to changing demands and goals, the nature of the contribution of the basal ganglia is less well specified (see Rogers et al., 1998). As to the fronto-cerebellar circuit, it has been suggested that the PFC may be primarily concerned with the generation of specific operations, while the cerebellum serves to optimise and to automatise the processes in question (see Daum, Snitz, & Ackermann, 2001), and a dissociation of this kind may also apply to executive control.

The empirical findings which are considered in this review are mainly derived from studies of brain-lesioned patients and complemented with neuroimaging studies. Despite rapid recent advances in neuroimaging methodology, multiple co-activations are usually observed and it is difficult to determine on the basis of neuroimaging studies alone which brain regions are critically involved in what aspect of cognitive function. Neuropsychological studies of patients with selective lesions to different regions play an important role in the evaluation of the distinct nature of information processing by each brain region.

The following sections are organised according to the brain regions which form part of the fronto-subcortical networks. Findings on the role of the PFC (the lateral PFC and the ACC) are presented first, since the lateral PFC in particular has traditionally been discussed in neuropsychology as the core region for supervisory control. This is followed by a more detailed description of the fronto-subcortical networks and the potential contribution of the subcortical components, i.e. the basal ganglia and the cerebellum. As outlined above, the presentation in each section will centre on response *inhibition*, *task management/multitasking* and *set shifting* as

representative executive control mechanisms. The final part aims at an integration and evaluation of the available evidence and issues relevant for future neuropsychological research.

2. The prefrontal cortex

The PFC has long been associated with the control, supervision or management of cognitive operations carried out elsewhere in the brain (Alexander, DeLong, & Strick, 1986; Fuster, 1993; Goldman-Rakic, 1995; Luriia, 1973). Although the association of specific aspects of executive functions to specific PFC subregions remains to be determined (see Burgess, 2000), the lateral PFC and the ACC are known to play a critical role. Both regions are anatomically connected and are frequently seen to be co-activated in functional neuroimaging studies.

2.1. The lateral prefrontal cortex

Lesions to the lateral parts of the PFC in humans generally lead to deficits in planning and problem solving, reasoning and generation of sets of appropriate responses on standard neuropsychological tests; the cognitive changes may be accompanied by a range of behavioural and affective changes (see Godefroy, 2003; Channon, 2004). Different areas within the lateral PFC are concerned with different aspects of working memory depending upon the exact task demands (Smith & Jonides, 1998, 1999). In recent years, cognitive impairments of PFC lesion patients were also described with respect to more sophisticated executive function tasks.

2.1.1. Inhibition

The ability to inhibit prepotent response tendencies has mainly been studied by procedures such as the Stroop test or stop signal reaction time (SSRT) tasks (Logan, 1994) as well as saccade tasks (see Reuter & Kathmann, 2004). Patients with right PFC lesions showed increased error rates in the interference condition of the Stroop test relative to naming coloured patches (Vendrell et al., 1995) and also longer SSRTs compared to healthy controls (Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003). In the latter study, impairments were particularly severe after lesions to the right inferior frontal gyrus. The finding of deficits of PFC lesion patients in inhibiting strong habitual response tendencies was confirmed in a study by our group. Patients with selective PFC lesions showed disproportionately longer reaction times (RTs) in the Stroop interference condition; comparable impairments were, however, not observed when weaker prepotent response tendencies induced by experimental manipulation had to be inhibited (Daum, Heyder, Koch, & Schwarz, in preparation). In the latter condition, a continuous performance task (CPT), subjects had to press one key to an A–X letter sequence which occurred 70% of the time, and a different key to any other letter pair sequence (AY, BX or BY, see Braver, Barch, & Cohen, 1999). The presentation of the letter A induces response preparation to the AX-key which needs to be suppressed when Y appears after A. Subjects with

PFC lesions did not show increased RTs on the AY trials, despite significant problems in the Stroop interference condition. A similar dissociation between Stroop interference and inhibition of experimentally induced prepotent responses was reported by Brass, Derrfuss, Matthes-von Cramon, and von Cramon (2003). These results suggest differences in inhibition mechanisms between strong habitual overlearned responses and experimentally induced (weaker) response tendencies.

Consistent with lesion studies, functional imaging studies yielded activation of the left inferior frontal gyrus in the interference condition of the Stroop test (Taylor et al., 1997), and activation in the right inferior frontal cortex in the incongruent condition of a flanker task (Hazeltine, Poldrack, & Gabrieli, 2000). In this task, a coloured target was flanked by matching (congruent) or different (incongruent) stimuli and responding to the incongruent distractors had to be inhibited. Successful motor response inhibition in a stop task was associated with activation of the right inferior PFC (Rubia, Smith, Brammer, & Taylor, 2003). The exact site of activation within the PFC has, however, been found to be related to subjective strategy in inhibiting cognitive set (Konishi, Jimura, Asari, & Miyashita, 2003).

2.1.2. Task management/multitasking

Patients with focal PFC lesions (and behavioural problems) were impaired at the simultaneous performance of a digit span task and a visual task (Baddeley et al., 1997). In a comparable procedure an initial basic two-choice task on an individual stimulus is followed by a decision whether a second stimulus is identical to the first one. PFC patients were impaired on this task as well (Godefroy et al., 1999). Patients with PFC lesions showed disproportionate problems on a multitasking procedure, a variant of the Six Elements Task (Levine et al., 1998). The lesion study findings are supported by fMRI activation of the PFC during dual-task relative to single task performance in healthy subjects (D'Esposito et al., 1995).

The problems of PFC patients with the coordination of several tasks have been discussed in terms of inadequate strategy use (Levine et al., 1998). A fine grained analysis of individual patients suggested that different cognitive subprocesses contributing to multitasking performance are impaired after lesions to different PFC regions (Burgess, Veitch, de Lacy, & Shallice, 2000). The ability to learn and remember rules was mainly impaired after ACC lesions, the ability to form appropriate plans after right dorsolateral PFC lesions and the ability to follow plans and rules after damage in the region of the left superior frontal gyrus (Burgess et al., 2000).

2.1.3. Set shifting

Set shifting problems of PFC lesion patients as assessed by a variant of the WCST have been interpreted in terms of deficits in disengaging attention from a previously relevant stimulus dimension (Owen et al., 1993). In a procedure involving switching between letter- and digit-naming, PFC lesion patients showed increased switch costs, particularly when working memory load was high, i.e. when arbitrary S–R rules had to be remembered (Rogers et al., 1998). The two tasks were presented in an alternating AABBAABB design and switch costs were determined by RT differences between trials requiring a switch (AB or BA respectively) and no-switch trials (AA, BB). In a

recent study, increased shift costs were observed for alternate letter and shape processing, independent of working memory load (Ravizza & Ciranni, 2002). This pattern was interpreted in terms of problems of PFC patients in reconfiguring stimulus and response sets (Meiran, 2000).

Consistent with the lesion results, PFC activations were observed in association with set shifting during performance of the WCST (Konishi et al., 1998, 1999), when switching between processing of different visual dimensions was required (Pollmann, Weidner, Muller, & von Cramon, 2000) or when a task switching condition was compared to a task repetition condition (Dove et al., 2000). There was, however, some degree of variation between the loci of activation in the fMRI studies, and regions outside the PFC were also frequently involved.

2.1.4. Conclusion

In summary, the lateral PFC seems to be involved in the *inhibition* of habitual responses, in particular when strong overlearned response tendencies need to be inhibited, and the allocation of processing resources in *task management* and *multitasking* paradigms. There is also evidence of a PFC contribution to the reconfiguration of stimulus and response sets during *set shifting*, which may be related to working memory load.

2.2. Anterior cingulate cortex (ACC)

The ACC and the PFC are functionally connected, integrating the ACC in the system of executive control (see Cohen, Botvinick, & Carter, 2000). Single case studies of ACC lesions were found to show similarities to lateral PFC lesions, with deficits in sustained attention and mild dysfunction on standard executive tests (Cohen et al., 1999; Ochsner et al., 2001). Its exact role with respect to the executive function subcomponents as introduced by Smith and Jonides (1999) is still under discussion. The preliminary evidence available so far comes from neuroimaging studies. There is as yet no conclusive evidence with respect to the effect of ACC lesions on experimental tasks of *inhibition*, *task management/multitasking* and *set shifting*.

The involvement of the ACC in *inhibition* of habitual responses was investigated in a neuroimaging study of the Stroop test (MacDonald, Cohen, Stenger, & Carter, 2000). The results yielded evidence of a dissociation between the left dorsolateral PFC contributing to task preparation and the ACC contributing to performance monitoring. In further studies using interference conditions in variants of the Stroop test or CPT procedures, the ACC was consistently found to be activated, in addition to the (right) lateral PFC (e.g. Braver, Barch, Gray, Molfese, & Snyder, 2001; Garavan, Ross, & Stein, 1999). Taken together, these data provide evidence for a distributed PFC–ACC network, in which the components interact in accordance with specific task demands (Haxby, Petit, Ungerleider, & Courtney, 2000).

Related views have suggested that the ACC plays an important evaluative role during the on-line detection of errors and response conflict (Carter, Botvinick, & Cohen, 1999; Carter et al., 2000; Suchan, Zoppelt, & Daum, 2003), but the exact nature of processing in the ACC during executive tasks remains to be determined. Addi-

tionally, the functional relevance of frequent co-activations in subcortical regions is as yet unclear.

3. Cortico-subcortical circuits

In recent views, information processing and integration with respect to executive control is mediated by fronto-striatal and fronto-cerebellar circuits (Robbins, 2000; Zoppelt & Daum, 2003). These circuits can be viewed in terms of hierarchically organised modules with specific contributions of each component to input processing and output organisation (Zoppelt & Daum, 2003).

3.1. Fronto-striatal circuits

Five major circuits connect the frontal cortex and the basal ganglia (Fig. 1); three may possibly contribute to the processes underlying executive control (Alexander et al., 1986; Mesulam, 1990). The first loop, the "motor" loop, involves projections from the supplementary motor, premotor, motor and somatosensory cortices to the putamen which in turn projects via the globus pallidus and the thalamus back to the supplementary motor cortex. The second loop, the "oculomotor loop", entails projections from the frontal eyefield via the caudate nucleus to the globus pallidus, which projects back to the frontal eyefield via the thalamus. The third circuit, the

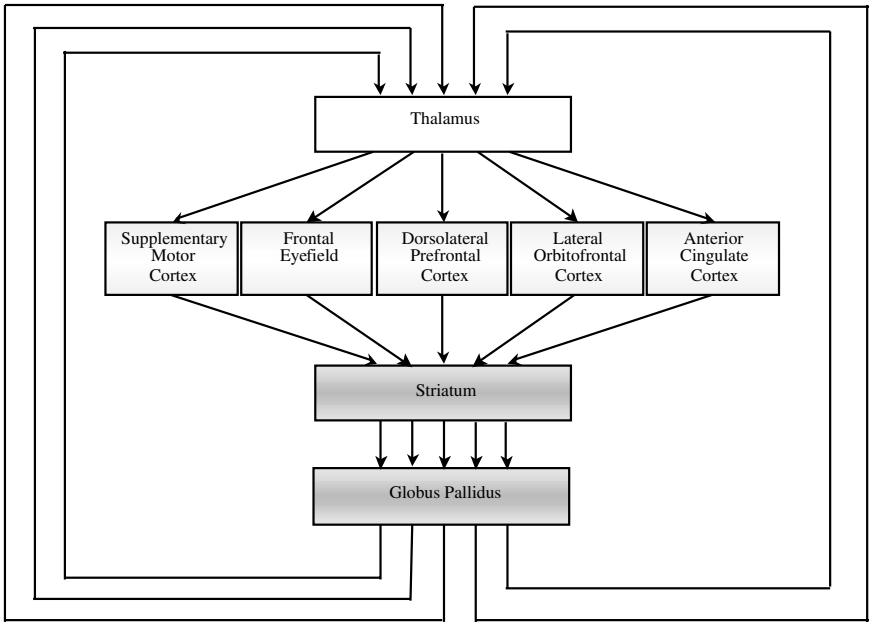

Fig. 1. Frontal-subcortical circuits between the PFC and basal ganglia (after Alexander et al., 1986).

''dorsolateral prefrontal loop'', originates from the dorsolateral PFC which projects via the dorsolateral caudate nucleus to the lateral globus pallidus which in turn projects back to the dorsolateral PFC via the dorsomedial nucleus (parvocellular region) of the thalamus. The fourth circuit, the ''lateral orbitofrontal loop'', entails projections from the orbitofrontal cortex via the ventromedial caudate nucleus to the medial globus pallidus which projects back to the orbitofrontal cortex via the dorsomedial nucleus of the thalamus (magnocellular region). The fifth circuit involves projections between the ACC, the ventral striatum, the rostrolateral globus pallidus and the mediodorsal nucleus of the thalamus (Alexander et al., 1986).

3.1.1. Degenerative disorders

Dysfunction of the subcortical components of the three non-motor circuits illustrated in Fig. 1 (i.e. the ''dorsolateral prefrontal, lateral orbitofrontal and anterior cingulate'' loops) generally leads to cognitive and behavioural impairments that are comparable to the deficits seen after PFC lesions. Patients with degenerative basal ganglia disorders such as Parkinson's disease (PD) or Huntington's disease (HD) show deficits in problem solving, reasoning, concept formation and complex memory tasks which require the self-initiated strategic organisation of encoding and retrieval. These deficits are generally attributed to dysfunction of the dorsolateral PFC-basal ganglia circuit (Stocchi & Brusa, 2000; Zoppelt & Daum, 2003). The other two non-motor circuits are mainly associated with dysfunction of affect and motivation (Cummings, 1993), although interactions of executive and affective problems may occur in PD (Breitenstein, Daum, & Ackermann, 1998).

3.1.1.1. Inhibition. PD patients showed deficits in the standard Stroop interference task (Dujardin, Degreef, Rogelet, Defebvre, & Destee, 1999) or variants of this procedure (Pollux & Robertson, 2002). These findings have been interpreted in terms of PD patients' difficulty in inhibiting irrelevant S–R mappings in order to overcome the irrelevant prepotent response (Pollux & Robertson, 2002). Whether the cognitive changes are directly related to degenerative changes of the caudate nucleus in conditions such as PD or HD is, however, unclear. While PET-studies of PD patients yielded a negative relationship between dopaminergic function of the caudate nucleus and Stroop-interference (Bruck et al., 2001; Rinne et al., 2000), quantitative MRI-analysis yielded an association of impairment on the Stroop test with the degree of diffuse cerebral atrophy rather than with caudate nucleus degeneration (Alegret et al., 2001).

3.1.1.2. Task management/multitasking. PD patients who showed inhibition deficits on the Stroop interference tests were unimpaired in a procedure involving the rehearsal of three consonants and simultaneous counting (Dujardin et al., 1999); coordination of articulatory suppression and span tasks was also intact in PD (Fournet, Moreaud, Roulin, Naegele, & Pellat, 1996). Early studies did, however, yield deficits of PD patients on dual-task procedures involving the coordination of motor and non-motor tasks (Brown & Marsden, 1991; Dalrymple-Alford, Kalders, Jones, & Watson, 1994) as well as deficient coordination of two non-motor tasks

(Sharpe, 1992, 1996). Similarly, HD patients were found to be impaired on a divided attention test which required the simultaneous processing of auditory and visual stimuli (Sprengelmeyer, Lange, & Homberg, 1995).

3.1.1.3. Set shifting. Set shifting as examined by a variant of the WCST yielded deficits of (medicated) PD patients, which were interpreted in terms of problems in re-engaging attention to a previously irrelevant dimension ("learned irrelevance"); unmedicated PD patients showed a combined learned irrelevance and perseveration deficit (Owen et al., 1993). The poor performance of PFC lesion patients on the same task was mainly due to perseveration.

In a complex task involving switches between letter- and digit-naming, PD patients showed increased error rates in the presence of normal switch costs, this finding being attributed to fatigue effects (Rogers et al., 1998). PFC lesion patients on the other hand, generally showed disorganised behaviour early in practise and increased time costs with switches. Switch costs in PD were increased when shifting between two perceptual dimensions or sensory channels was required (Hayes, Davidson, Keele, & Rafal, 1998; Ravizza & Ivry, 2001). PD patients performed normally on an alternate letter- and shape processing task that PFC patients were impaired on, but did show difficulties with ambiguous stimuli when the irrelevant features were harder to ignore (Ravizza & Ciranni, 2002). A similar pattern was reported by Cools, Barker, Sahakian, and Robbins (2001).

The apparent similarity of some of the effects of lateral PFC lesions and PD on executive processing has been conceptualised in terms of inefficient recruitment of the PFC in basal ganglia disorders (Elias & Treland, 1999; Stocchi & Brusa, 2000). It is, however, as yet unclear whether the cortical and subcortical components of the dorsolateral fronto-striatal circuit contribute in a comparable fashion to executive control. There is preliminary evidence of subtle differences in the deficit pattern. Whereas patients with lateral PFC lesions need more steps to arrive at the correct solution in planning tasks, PD patients need longer planning times, but not more steps (Robbins et al., 1994). HD patients show both, increased planning times as well as inefficient strategies, indicating both striatal and frontal dysfunction (Lawrence et al., 1996).

3.1.2. Selective basal ganglia lesions

Focal vascular lesions of the striatum were generally found to lead to deficits on standard neuropsychological tests of executive functions. Striate infarcts were e.g. associated with impaired verbal fluency (Eslinger & Grattan, 1993), poor WCST performance and increased susceptibility to interference (Godefroy et al., 1992, 1994; Mendez, Adams, & Lewandowski, 1989; Strub, 1989). There is some debate, however, whether the presence of executive impairments is dependent upon additional cortical damage (Godefroy et al., 1992).

3.1.2.1. Inhibition. In a direct comparison of the effects of selective PFC and basal ganglia lesions on inhibition, Rieger, Gauggel, and Burmeister (2003) observed deficits of both groups relative to controls with respect to the time needed to inhibit

an ongoing activity in a stop signal task. Patients with vascular basal ganglia lesions showed deficits with respect to both, the inhibition of a strong habitual response in the interference part of the Stroop test and a weaker experimentally induced response tendency in a CPT (see above, Daum et al., in preparation). As outlined above, patients with selective PFC damage of comparable etiology were only impaired when inhibition of very strong, overlearned response tendencies was required.

3.1.2.2. Task management/multitasking. The effects of vascular striatal damage on task management was assessed by a divided attention task which required the continuous parallel processing of auditory and visual stimuli (Zimmermann & Fimm, 2002) and the Six Elements Test from the Behavioural Assessment of the Dysexecutive Syndrome (BADS) battery (Wilson, Alderman, Burgess, Emslie, & Evans, 1996), which entails the self-generated coordination of six different tasks within 10 min. Striatal damage led to impairments on the former, but not on the latter task, indicating a task management deficit when demands on the on-line coordination of attentional resources between different sensory channels were high (Daum et al., in preparation).

3.1.2.3. Set shifting. So far, data on set shifting abilities in patients with basal ganglia damage are sparse. Patients with unilateral vascular lesions of the caudate nucleus, putamen and the anterior limb of the internal capsule showed increased perseveration on the WCST similar to PFC lesion patients (Eslinger & Grattan, 1993). A comparable pattern was reported in a single case study of a patient suffering cerebral angiitis which predominantly affected the right neostriatum (Keri et al., 2002).

3.1.2.4. Conclusions. In summary, the investigation of the subcortical contribution to executive control has often focused on patients with degenerative disorders of the basal ganglia, such as PD or HD, vascular lesions have been investigated in much less detail. With respect to response *inhibition*, it seems that the basal ganglia play a critical role in the overcoming of habitual responses as shown in several samples of PD patients and also in a sample of patients with vascular lesions. The lack of a correlation between caudate dysfunction and *inhibition* (Alegret et al., 2001) might be related to striato-frontal dopaminergic deafferentation which is not necessarily seen on MRI-scans but may still be of functional relevance. It is unclear inhowfar the deficits of PD patients concerning *task management* are related to demands on working memory and/or motor demands of a task (Brown & Marsden, 1991; Bublak, Muller, Gron, Reuter, & von Cramon, 2002). Movement control may disproportionately draw upon attentional resources and affect behavioural performance in PD, in spite of intact information processing.

Similarly, *set shifting* difficulties have been attributed to depleted attentional resources rather than to internal control deficits in PD (Tamura, Kikuchi, Otsuki, Kitagawa, & Tashiro, 2003; Woodward, Bub, & Hunter, 2002). In a more detailed analysis based on imaging studies of WCST performance, Monchi, Petrides, Petre, Worsley, and Dagher (2001) attributed activity in the orbitofrontal loop to processing of negative feedback which signals the need for a mental shift to a new response

set, whereas the motor loop via the putamen was associated with implementation of the new response set immediately following the shift. It has been argued that disrupted interactions between the striatum and the PFC may underlie set shifting deficits (Robbins, 2000; Robbins & Rogers, 2000). The contribution of the basal ganglia has been discussed in terms of selection and inhibition of competing cognitive and motor programmes and in terms of overcoming set inertia associated with the current set in order to engage a new set (Ravizza & Ivry, 2001).

3.2. Fronto-cerebellar circuitry

The second cortico-subcortical circuit which is likely to be involved in the mediation of executive functions entails reciprocal pathways between the dorsolateral PFC and the cerebellum (Daum & Ackermann, 1997; Daum et al., 2001; Schmahmann, 1997). Projections originating from the lateral cerebellum project via the dentate nucleus and the thalamus to the PFC which projects back to the cerebellum via pontine nuclei (Fig. 2).

It has been hypothesized that the PFC areas of this circuit are identical to the prefrontal regions involved in working memory (Middleton & Strick, 1994). This view is supported by similarities in the pattern of neuropsychological executive deficits in patients with dorsolateral PFC lesions and patients with cerebellar lesions. They include deficits in planning, problem solving and reasoning (Hallett & Grafman, 1997), verbal fluency and other word generation abilities (Appollonio, Grafman, Schwartz, Massaquoi, & Hallett, 1993) as well as concept formation and the strategic organisation of encoding and retrieval (Burk et al., 1999, 2003). Neuroimaging studies have also consistently yielded cerebellar activations for a range of tasks leading to prefrontal activations (Hallett & Grafman, 1997). Dysfunction of fronto-cerebellar pathways may form the substrate of executive impairments in patients with degenerative cerebellar diseases which involve combined brainstem-cerebellar damage (Daum & Ackermann, 1997; Daum et al., 2001; Suchan & Daum, 2000).

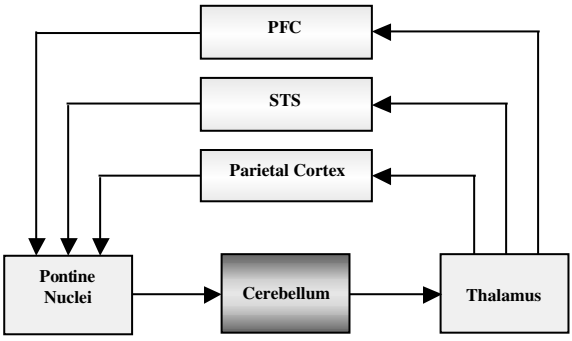

Fig. 2. Connections between the cerebellum and the neocortex (after Schmahmann, 1997). PFC = prefrontal cortex, STS = superior temporal sulcus.

3.2.1. Inhibition

Ischemic cerebellar lesions have been associated with poor performance on the interference condition of the Stroop test, at least in the acute stage after the ischemic event; at one-year follow-up these effects were significantly reduced (Neau, Arroyo-Anllo, Bonnaud, Ingrand, & Gil, 2000). Patients with selective vascular cerebellar damage showed intact inhibition effects during Stroop interference and the CPT task (see above, Daum et al., in preparation); the patients of this study were assessed when the acute effects of the ischemic event had subsided. Consistent with the latter finding, the cerebellum was not activated in association with withholding a response to a NoGo signal (Watanabe et al., 2002).

3.2.2. Task management/multitasking

Deficits of cerebellar patients were observed on a dual task procedure which required the simultaneous performance of a specified movement and the identification of auditory targets (Lang & Bastian, 2002). The impairment pattern was attributed to problems in automaticity of a recently practiced movement, and not to reduced attentional mechanisms. By contrast, cerebellar dysfunction due to tumour resections or hematomas was associated with reduced performance on a divided attention task involving the simultaneous processing of visual and auditory input (Gottwald, Mihajlovic, Wilde, & Mehdorn, 2003). Focal vascular cerebellar damage, on the other hand, did not have a detrimental effect either on the same divided attention task that the tumour removal patients were impaired on or the multitasking abilities tapped by the Six Elements Test of the BADS (Daum et al., in preparation). Etiology of the lesion and associated differences in extent and site of cerebellar dysfunction may thus contribute significantly to the pattern of impairment.

3.2.3. Set shifting

Earlier studies have raised the issue of a cerebellar involvement in the rapid shifting of attention between different sensory channels or between different dimensions within a single channel (Akshoomoff & Courchesne, 1992; Courchesne et al., 1994). When alternate processing of visual and auditory stimuli was compared to focused attention in single tasks (Ravizza & Ivry, 2001), cerebellar patients showed disproportionate deficits in the alternation condition, particularly with short inter trial intervals, when rapid successive responses were required. The deficit did, however, become smaller when motor demands were reduced. Increased effort in producing and monitoring motor responses may thus contribute to an important degree to the attentional set shifting deficit reported for cerebellar patients (Ravizza & Ivry, 2001).

3.2.4. Conclusions

In summary, there is no evidence for a cerebellar involvement in response *inhibition* within the context of executive control. The findings for *task management/multitasking* are less consistent, and the potential cerebellar contribution appears to be related to factors such as motor demands and degree of automaticity involved

in adequate performance. For *set shifting*, the pattern also seems to be related to demands for rapid or fluent processing.

4. Conclusion and further directions

The present review aimed to evaluate evidence for the neuropsychological basis of executive control, focusing on *inhibition*, *task management/multitasking* and *set shifting* as relevant subprocesses in a multicomponent view of executive function (see Smith & Jonides, 1999). More specifically, the issue of interest concerns the question whether the cognitive processes underlying executive control are "diffusely" organised in fronto-subcortical networks, leading to comparable impairments after damage to different components of the networks. An alternative view suggests specific contributions of the different components, with distinct neuropsychological profiles after lesions to the cortical and subcortical part of the networks.

The present evidence from neuropsychological studies seems to support the idea of subtle processing differences between frontal and subcortical components. With respect to *inhibition*, the results suggest differences in inhibitory mechanisms between habitual overlearned responses and experimentally induced (weaker) response tendencies. Whereas the PFC seems to be associated with the inhibition of habitual overlearned responses, the basal ganglia are involved in both the inhibition of habitual and newly implemented response tendencies. There is no clear evidence for a significant cerebellar role in inhibition. Taken together, the data indicate an interesting dissociation between the processes involved in the inhibition of strong, habitual response tendencies and weaker response tendencies created temporarily in a specific situation. The relevance and nature of this distinction remains to be further elucidated in future research.

For *task management/multitasking*, an important role of the PFC has been established, while the involvement of the basal ganglia may be related to motor demands and the rapid processing and coordination of sensory input. The cerebellum does not seem to be consistently involved in the coordination of task management/multitasking. The PFC is thus involved in the coordination of attentional resources between different sensory or response channels. The features of a striatal contribution and the differences to PFC processing remain to be determined in more detail.

For *set shifting*, the PFC has been related to the reconfiguration of stimulus and response sets, and its contribution may be particularly critical in conditions of high working memory load. Whether or not set shifting was intact in degenerative basal ganglia dysfunction was dependent upon task difficulty. Similarly, a potential cerebellar involvement in the mediation of set shifting performance appeared to be related to demands on rapid fluent processing.

In summary, despite an increasing number of comparative neuropsychological studies in recent years, no firm conclusions can be drawn as yet with respect to the specific contribution of cortical and subcortical components of frontostriatal and frontocerebellar circuits to executive functions. The subcortical participation in particular has frequently been investigated in patients with neurodegenerative

disorders which do not only affect the basal ganglia or the cerebellum, respectively, but also a number of other brain systems and a range of neurotransmitters. It is thus as yet unclear, e.g. whether executive impairments in PD reflect mechanisms at the level of the PFC or the striatum (Robbins, 2000). Factors such as severity of disease and degree of motor dysfunction, medication or co-existing affective changes may significantly contribute to the pattern of cognitive dysfunction in patients with PD or HD, even in early stages (e.g. Uekermann et al., 2003). Patients with degenerative disorders are thus no good model of focal pathology of the basal ganglia or the cerebellum.

The study of executive functions in patients with focal lesions has so far mainly been based on standard neuropsychological tests such as the WCST or verbal fluency tests which are relatively "impure" tasks addressing a range of different cognitive demands (see Burgess, 2000; Godefroy, 2003) although recently tests with high ecological validity have also been used (see Channon, 2004). As concerns the issue of cortico-subcortical networks, a suitable strategy would be to assess groups of patients with dysfunction of comparable etiology (i.e. vascular damage). Restricting pathology to focal lesions would greatly increase the direct comparability of the cognitive profiles after cortical and subcortical damage. Although variation of performance is still to be expected within each clinical group, analysis of individual cases with respect to identification and specification of critical within-region lesions will be a useful strategy. Ideally, high resolution MRI should be used to identify the exact site of a lesion within each region, in order to determine whether the area in question forms part of the loop under investigation. This is particularly important for the fronto-striatal loops, because of their close anatomical proximity. The determination of double dissociations between different aspects of executive functions and different brain areas would also help to further clarify the issue of the potential fractionation of executive control (see Burgess, 2000). Such clinical neuropsychological approaches should be combined with neuroimaging studies using identical experimental procedures in healthy subjects, the common aim being the identification of critical between-region processing differences.

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