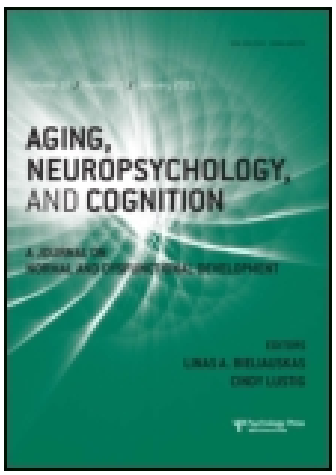
On: 21 May 2015, At: 09:07
Publisher: Routledge
Informa Ltd Registered in England and Wales Registered Number: 1072954 Registered office: Mortimer House, 37-41 Mortimer Street, London W1T 3JH, UK



Aging, Neuropsychology, and Cognition: A Journal on Normal and Dysfunctional Development

Publication details, including instructions for authors and subscription information:
http://www.tandfonline.com/loi/nanc20

Frontal-Striatal Circuits in Cognitive Aging: Evidence for Caudate Involvement

David C. Rubin

Published online: 09 Aug 2010.

To cite this article: David C. Rubin (1999) Frontal-Striatal Circuits in Cognitive Aging: Evidence for Caudate Involvement, Aging, Neuropsychology, and Cognition: A Journal on Normal and Dysfunctional Development, 6:4, 241-259

To link to this article: http://dx.doi.org/10.1076/1382-5585(199912)06:04;1-B;FT241

Aging, Neuropsychology, and Cognition
1999, Vol. 6, No. 4, pp. 241-259

1382-5585/99/0604-241$15.00

Frontal-Striatal Circuits in Cognitive Aging: Evidence for Caudate Involvement*

David C. Rubin
Duke University

ABSTRACT

Changes in cognition with aging have been claimed to be due in large part to a decline in frontal lobe function. However, at our present state of knowledge, the emphasis on the frontal lobes to the exclusion of the rest of the frontal-striatal circuits of which they are a part is unwarranted. To argue this point, I consider another anatomical candidate within these circuits, the caudate. Evidence is presented that the caudate decreases in size with age as much as the frontal lobes and that damage to either the frontal lobes or the caudate is accompanied by declines in inhibitory processes, executive control, and cognitive speed similar to those seen in normal aging. Separating the unique contributions of the frontal lobes and the caudate to these circuits is difficult but should be the focus of future studies of the biological basis of cognitive aging.

Changes in the frontal lobes have been claimed to be the neurological basis of many of the cognitive declines that occur in aging (Albert & Kaplan, 1980; Daigneault & Braun, 1993; Demspter, 1992; Mittenberg, Seidenberg, O'Leary, & DiGiulio, 1989; Moscovitch & Winocur, 1992; Shimamura, 1994; Shimamura & Jurica, 1994; West, 1996; Whelihan & Lesher, 1985), even in papers with no other neural claims (Craik & Jennings, 1992). I argue that the standard documented declines in cognitive aging cannot be attributed to declines in frontal lobe function to the exclusion of declines in the rest of the frontal-striatal circuit. To substantiate this argument, I focus on one structure in that circuit, the caudate (technically the head of the caudate), which also declines with age. I choose the caudate because the strongest support can be made for this structure; the most data are available for it. For the caudate I will show that when its functioning declines, cognitive changes occur that are similar to those attributed to frontal lobe damage. Thus, attributing changes in normal aging to frontal lobe decline as opposed to caudate decline, though widely held, is at best premature. Oddly enough, I could find only two earlier arguments for the role of the caudate in cognitive aging. In a book on subcortical dementias (i.e., dementias often involving caudate damage), Van Gorp and Mahler (1990) presented a chapter relating subcortical dementia to normal aging. More directly, Hicks and Birren (1970) proposed that the caudate has a central role in cognitive aging because damage to it causes cognitive slowing.

Three major empirical observations consistently appear in the literature on cognitive aging: (a) decreases in the functioning of inhibitory mechanisms, (b) decreases in executive control or planning, and (c) cognitive slowing as measured by increases in response time. Other common categories, such as decreases in sensory function (Park, 1998), are not central to the arguments made here because they are usually not

* We wish to thank Herb Crovitz, John Dalrymple-Alford, Debra Due, Daniel Greenberg, Cindy Lustig, Naftali Raz, and J. A. Saint-Cyr for their comments and the McDonnell-Pew Cognitive Neuroscience Program and an Erskine Fellowship from the University of Canterbury for support.
Address correspondence to: David C. Rubin, Department of Psychology: Experimental, Box 90086, Duke University, Durham, NC 27708-0086, USA. Tel.: (919) 660-5732. Fax: (919) 660-5726. E-mail: rubin@psych.duke.edu.
Accepted for publication: October 19, 1999.

attributed to frontal-lobe or caudate decline (Moscovitch & Winocur, 1992; Shimamura, 1994; West, 1996). Slowing is relatively easy to distinguish, but it is harder to classify tasks between inhibition and executive control. For instance, the Wisconsin Card Sort Test involves both inhibition of past correct categories and executive control in the form of self-initiated planning. For the purposes of this paper, the exact classification of a particular task is not important as long as similar results are obtained in aging, frontal lobe damage, and caudate damage. Although some theorists would argue for one of these three categories as a mechanism at the expense of the others (e.g., Dempster, 1992; Hasher & Zacks, 1988; Salthouse, 1996), I keep them as summary terms for independent sets of observations; a presentation in terms of independent mechanism rather than classes of observations appears too ambitious at this time (Light, 1991). In particular, the age-related declines in tasks taken to be evidence for a decline in frontal lobe function will be shown to also decline with damage to the caudate. These parallel declines in observable performance hold no matter what theoretical framework is used to summarize them.

The claim is not that the decreases in inhibitory mechanisms, executive function, and cognitive speed that occur in aging occur in the absence of other changes; they do not. Nonetheless, the claim of a more steep decline in inhibition, executive control, and speed than in other classes of behavior is not a distortion of the consensus view of the literature. For instance, in reviewing the literature on cognitive aging, with no claims about physiological processes, Park (1998) notes, '' There are four important mechanisms that have been hypothesized to account for age differences in cognitive functioning: a) the speed at which information is processed; b) working memory function, c) inhibitory function, and d) sensory function'' (pp. 49-50). From her review, it is clear that much of Park's second mechanism is close to what I am calling executive control and that her fourth mechanism is not one which has its main neural basis in the frontal lobes or caudate.

For a more complete review of the neuropsychological differences that accompany both aging and frontal lobe damage than that presented here the reader should see Moscovitch and Winocur (1992), Shimamura (1994), and West (1996). They note that not all changes in aging can easily be attributed to decline in frontal lobe function, and that declines in mnemonic function also occur that can be most likely attributed to declines in function of the hippocampus and related circuitry. But in terms of the relation of frontal lobe damage and aging Moscovitch and Winocur (1992) note, ''There is a remarkable consistency between the memory deficits seen in human patients and experimental animals with frontal lesions and those seen in normal aging. Indeed, there is not one instance where a significant discrepancy is found'' (p. 353). Thus, the claim that cognitive aging is similar to frontal lobe damage is on good empirical grounds that will not be challenged here. My claim is simply that the parallels are at least as strong between aging and caudate damage and until work is done to separate the effects of caudate and frontal lobe contributions, the caudate and especially circuits involving both structures must be considered.

CAUDATE CHANGES WITH AGE AND DISEASE

There is evidence that the caudate, like the frontal lobe, is differentially affected by aging. Such evidence is prerequisite to the arguments to be made here. Using autopsy data reported in the literature from a total of 8,000 brains, Eggers, Haug, and Fischer (1984) found an estimated decrease from ages 25 to 75 of 12% for the frontal cortex and 15% for the caudate but no decrease for striate cortex or the parieto-occipital lobe. Using magnetic resonance imaging (MRI), Jernigan, Archibald, Berhow, Sowell, Foster, and Hesselink (1991) found a correlation of –.49 between age and caudate volume, the largest of the twelve areas they measured, and correlations of –.40 and –.37 for areas containing the frontal lobes. Using MRI, Krishnan et al. (1990) also found a large decline in caudate volume with

age, $r = -.69$. This decline in caudate volume with age was independent of and additive to reduced caudate volume from depression, ruling out depression as a factor (Krishnan et al., 1992). In an exhaustive review of the neuroimaging literature, Raz (in press) found roughly equal loss with age for the frontal lobes and caudate ($r = -.47$ for both), although if the gray matter caudate were compared only to the gray matter of the frontal lobes, the frontal lobe loss would be greater ($r = -.57$).

Evidence for changes in caudate function from outside aging come from studies of patients with lesions to that area and from patients with Parkinson's and Huntington's disease. Parkinson's disease results from damage to the substantia nigra. The substantia nigra synthesizes dopamine and projects to the caudate. In the course of the disease, when dopamine levels drop 70% or more from normal levels, symptoms appear (Lezak, 1995). Replacement of the dopamine with L-dopa causes a reversal of symptoms for a period of time (e.g., Malapani, Pillon, Dubois, & Agid, 1994). Huntington's disease involves the atrophy of the caudate nucleus itself as well as other basal ganglia structures and a decrease in dopamine function (Bäckman, Robins-Wahlin, Lundin, Ginovart, & Farde, 1997), which may be seen as the interaction of the disease process with normal age changes (Finch, 1979). Using MRI, Jernigan, Salmon, Butters, and Hesselink (1991) found the largest decrease of the twelve areas they measured in patients with Huntington's disease was the caudate, both when compared to controls and to patients with Alzheimer's disease.

There are considerable data to support the similarities of caudate damage to aging and to frontal lobe damage. For instance, Gabrieli, Singh, Stebbins, and Goetz (1996) note that when factors such as severity of disease and depression are controlled, patients with Parkinson's disease ''often exhibit a pattern of intact and impaired cognition and memory that is strikingly similar to that shown by patients with frontal-lobe lesions'' (p. 323). Similarly, for the same groups and reservations about specifying the exact nature of the conditions and the patients, Brown and Marsden (1990) note that ''the behavioral parallels are, nevertheless, impressive'' (p. 22). In listing similarities among subcortical dementias, of which Parkinson's and Huntington's disease are examples, for a book that serves as a text for many in neuropsychology, Lezak (1995) provides a list remarkably similar to the one Park (1998) gave for aging and to the one used here: ''slowed mental processing, disturbances of attention and concentration, executive disabilities including impaired ability to manipulate concepts or generate strategies, visuospatial abnormalities, and a memory disorder that primarily affects retrieval rather than learning'' (p. 231). Talking about the individual diseases, she notes, ''By and large, the cognitive deficits associated with Parkinson's disease are similar – and often identical – to cognitive disorders that occur with frontal lobe damage, particularly with involvement of the prefrontal cortex'' (p. 224), and ''most of the cognitive deficits of patients with Huntington's disease are akin to frontal lobe disorders'' (p. 232). Thus, it is claimed by leaders in their respective fields that aging is like damage to the frontal lobes and that damage to the caudate is like damage to the frontal lobes. All this is missing is the logical completion that aging is like damage to the caudate.

In addition to the similarity of Parkinson's and Huntington's disease to frontal-lobe damage and normal aging, several observations suggest a relationship between the functioning of the caudate (and other structures in the frontal-striatal circuit) to normal aging. First, these two diseases strike most often in middle age or later life. Second, the dopaminergic system declines, though less dramatically than in Parkinson's disease, over the course of aging (Antonini et al., 1993; Bäckman, Ginovart, Dixon, Robins-Wahlin, Whalin, Halldin, & Farde, in press; Gabrieli, 1995; Kish, Zhong, Hornykiewicz, & Haycock, 1995; Volkow et al., 1998; Wang, Chan, Holden, Dobko, Mak, Schulzer, Huser, Snow, Ruth, Calne, & Stoessl, 1998). Third, even with the duration of the disease controlled, using positron-emission tomography (PET) it has recently been shown that the uptake of fluorodopa in the caudate of patients with Parkinson's disease is more impaired in

older patients (de la Fuente-Fernandez et al., 1998).

Parkinson's and Huntington's disease are different from each other and from frontal lobe damage in their etiology and cognitive effects, and all three are different from normal aging. Because the argument to be made here is that subcortical areas of the frontal-striatal circuits are also important in cognitive changes with aging, I focus on their similarities. Once this basic argument is accepted, the differences of Parkinson's and Huntington's disease from each other and from frontal-lobe damage, rather than their similarities, would become the focus of understanding which structures are most related to specific cognitive changes. However, for now, one major difference between Parkinson's and Huntington's disease must be clear to evaluate the arguments made here. Parkinson's disease is caused by a drop in dopamine in the caudate and other areas of the striatum, *and* also in the frontal lobes. Thus, there may be some affects of Parkinson's disease that are caused by a decline in frontal lobe functioning that is independent of the subcortical damage (see Gabrieli et al., 1996 and Taylor, Saint-Cyr, & Lang, 1986 for discussions of this view). Because of this, the cognitive measures from Parkinson's disease cannot be taken as pure evidence for caudate damage, as opposed to frontal-striatal circuit damage. In contrast, Huntington's disease is caused by damage to the caudate, and damage to the frontal lobes is secondary to this damage. Brandt and Bylsma (1993) review the neuroimaging data from patients with Huntington's disease that show that structural measures of caudate, but not frontal, atrophy correlate strongly with many cognitive measures, including many of those reviewed here. They also review studies using PET and single photon emission tomography that show decreased metabolism in the caudate, but not the frontal lobes, that correlates with cognitive measure.

FRONTAL-STRIATAL CIRCUITS

Why do frontal lobe and caudate damage produce similar behaviors? Figure 1 is adapted from a now classic article by Alexander, DeLong, and Strick (1986) and maintains the nomenclature of that article. It lays out the idea of parallel, discrete, nonoverlapping circuits that send information from specific cortical areas to specific basal ganglia to specific thalamic nuclei and then back to the same areas of cortex. Figure 1 shows two of the five parallel topographic circuits from the original article. Recent work argues for an even finer grained topographic parallel structure that in humans blurs the clear distinction among these separate pathways into a more continuous and interrelated system (Alexander & Crutcher, 1990; Cummings, 1993; Goldman-Rakic & Selemon, 1990; Graybiel, 1990; Houk, Davis, & Beiser, 1995; Parent & Hazrati, 1995; Wise, Murray, & Gerfen, 1996). The two pathways shown pass through both the head of the caudate and prefrontal cortex. The remaining three circuits pass through the motor cortex, oculomotor cortex, and the anterior cingulate at the cortical level and the putamen, body of the caudate, and ventral striatum at the striatal level, respectively. These five frontal-striatal circuits are involved in many aspects of complex behavior and cognition. The two circuits shown, which pass through the head of the caudate, are involved in cognitive tasks of interest here, while the other three parallel frontal-striatal circuits are involved in motor and other activity. In particular, the circuit through the putamen is more related to motor slowing and decreases in sensorimotor coordination than the more ''frontal'' tasks considered here. Research with primates has shown that these pathways have the behavioral effects that would be expected from human neuropsychology. For instance, in 1967 Divac, Rosvold, and Szwarcbart performed lesions in four distinct areas of the caudate in monkeys. All lesions produced impairments that would have been produced in the monkey with selective lesions to the cortical area projecting to the damaged area of caudate. Moreover, these pathways are involved in timing (Meck, 1996), consistent with their role in cognitive slowing.

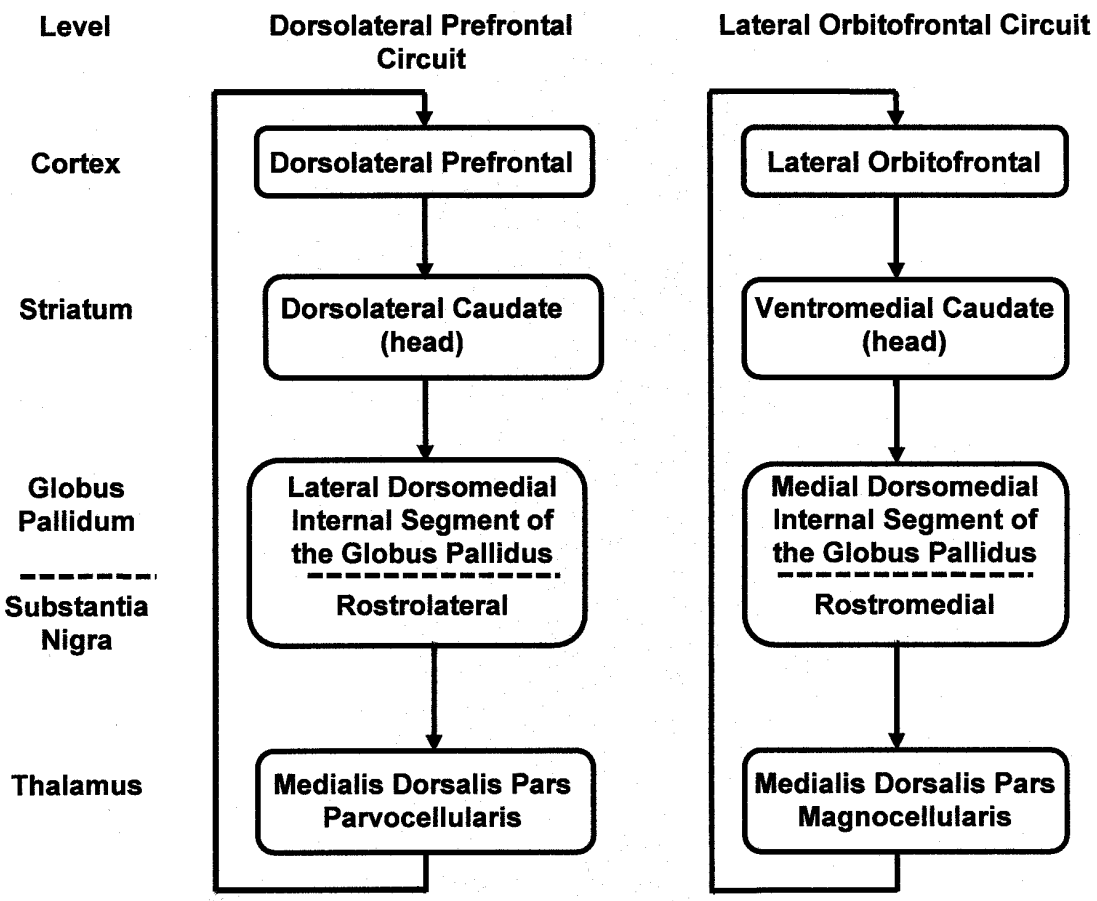


Fig. 1. A schematic drawing of the frontal-caudate loops after Alexander, DeLong, and Strick (1986).

The idealized pathways shown in Figure 1 account for many of the connections made to the structures shown. These connections within the circuits ensure that damage to any one structure will cause the other structures to have abnormal inputs and over time may lead to secondary damage in other structures. Because there is a circuit, damage to the frontal lobe can lead to decline in function and eventually structure in the caudate, and damage to the caudate can lead to decline in function and eventually structure in the frontal lobe, even when the initial damage is restricted to one structure or the other (Cummings, 1993; Wise et al., 1996).

THE NATURE OF THE ARGUMENT

The form of the claim that many aspects of cognitive aging are due to decreases in frontal lobe function can be stated as follows. 1a) Central to the effects of aging on cognition is a decrease in function X as indexed by tests x_1, x_2, x_3, . . . 1fl) Central to the effects of frontal lobe damage on cognition is a decrease in function X as indexed by tests x_1, x_2, x_3, . . . 2fl) With increasing age there is a decrease in frontal lobe functioning as measured by anatomical volume, neurotransmitter level, or functional imaging. 3fl) Therefore the frontal lobe changes with normal aging are central to changes in cognitive aging.

I wish to challenge this argument by adding propositions 1c, 2c, and 3c, which parallel 1fl, 2fl, and 3fl except that the word *caudate* is substituted for the words *frontal lobes.* 1c) Central to the effects of caudate damage on cognition is a decrease in function X as indexed by tests x_1, x_2, x_3, . . . 2c) With increasing age there is a decrease in caudate functioning as measured by anatomical volume, neurotransmitter level, or functional imaging. 3c) Therefore the caudate changes with normal aging are central to changes in cognitive aging. Evidence for 2c has been presented already; evidence for 1c comprises most of the rest of the paper. At the end of the paper, I will return to the claim that frontal-striatal circuits that include the caudate and areas of the frontal lobes that it projects to through the thalamus are in fact the neural basis for the changes noted and that more work has to be done if localization more specific than this is to be stated. But for ease of reviewing the literature, which usually considers either cortical or subcortical structures and not circuits, the case first will be made for the caudate. The aim is not to substitute one neural structure for another, but to shift focus from structures to circuits. Given the existing evidence, however, the only way to do this is by concentrating on structures.

Please note the logic of the case being made. I wish to show that the generally held view that changes in cognition with aging are due in large part to changes in the frontal lobe can be challenged by showing that another anatomical structure is as strongly implicated by existing data. Therefore, I use less primary data and spend time making the case for the frontal lobes, except to specify the particular functions and tests (i.e., the X, x_1, x_2, and x_3, in the formal argument) so that the same tests can be used for the caudate. All I need show for the frontal lobes propositions is that they are not straw men, that their role in cognitive aging is received wisdom, that respected scholars have attributed functions to them that perhaps should be attributed more diffusely. However, when it comes to claims that the caudate is involved, primary data are needed and are provided.

Also note that counter arguments that terms like inhibition and executive control are too vague to fill the roles of ''function X,'' or that the tests used to measure a function do not represent a psychometrically coherent and exclusive group (Duncan, Johnson, Swales, & Freer, 1997; Salthouse, Fristoe, & Rhee, 1996), or that all diseases tend to cause decreases in inhibition, executive control, and speed of processing are not criticisms of the basic argument being made here. To the extent that these criticisms are valid, the basic claim that changes in cognition with aging can be attributed to decreases in frontal lobe functioning are called into question, which is what I wish to do. That is, once these terms and tests are accepted for use in cognitive aging and frontal lobe studies as shown by citations to published studies, they cannot be seen as too vague or commonly occurring when damage to other neural structures is considered. However, once we move from arguing this initial step that the search for the neural substrate of aspects of cognitive aging must be extended beyond the frontal lobes to the harder task, not undertaken here, of actually investigating which neural structures and circuits account for which aspects of behavioral change, it rapidly becomes apparent that more precise terminology and theory would be helpful.

At several points quotes from or reference to review articles or authorities are used in place of a full discussion in order to supplement the evidence presented. The validity of the argument rests with the evidence, not the authority, but this tack is used both for the sake of brevity and to remind the reader that most of what is presented here is received wisdom in its own field. I try to make my argument as forcefully and clearly as possible so that any flaws will be easily apparent and can be corrected by others. For each of the three classes of empirical observations, decreases in (a) inhibition, (b) executive control, and (c) cognitive speed, I begin by briefly reviewing the behavioral evidence from normal aging and frontal lobe damage. This sets up the context for demonstrating that decreases in inhibition, executive control, and speed also occur with declining caudate function. Where possible, I choose tasks that have been used with all three participant populations.

DECREASED INHIBITION

Aging

One of the most widespread findings in the cognitive psychology of aging is a decrease in inhibitory processes (Dempster, 1992; Hasher & Zacks, 1988). Unlike the relatively theory-neutral, direct relationship between cognitive slowing and response times on cognitive tasks, empirical evidence for inhibition comes from what might on the surface seem to be an unrelated complex of tasks. Despite some theoretical differences among researchers about the nature of inhibition, however, prototypical tasks that measure inhibition have widespread theoretical agreement. In reviewing the aging literature, Dempster (1992) surveys studies using the Wisconsin Card Sorting Test, the Stroop Test, selective attention tasks, and interference in the Brown-Peterson short-term memory task as measures of inhibition. The results are robust. For instance, the same year that Dempster's review was published, Daigneault, Braun, and Whitaker (1992) demonstrated losses with aging on both the Wisconsin Card Sorting Test and on the Stroop Test. The Wisconsin Card Sorting Test requires participants to discover the category that the experimenter has chosen and to switch to an unannounced new category when the experimenter switches category. For example, sorting by color will be correct for a time, but then, without warning, the correct sort will be on number or shape. When a switch in category occurs, the previously correct category must be inhibited. Thus, the Wisconsin Card Sorting Test involves many processes. It is included in this section on decreased inhibition only because it is most often considered as a measure of inhibitory processes in the cognitive aging literature. The Stroop Test is a test of inhibition in that the color of the ink in which words are typed needs to be reported and the difficulty comes in inhibiting responding with the actual word typed when it is a color name (e.g., inhibiting reading the word *red* when you are supposed to report the color of the ink, which is blue).

Frontal Lobe Damage

Dempster (1992), in arguing for inhibition as the basis for a ''unified theory of cognitive development and aging'' (p. 45), notes that the frontal lobes are the first cortical lobes to show signs of change with aging and that the tasks used to measure inhibition are often associated with frontal lobe damage. In reviewing memory and frontal lobe function Shimamura (1995) made a list of tasks similar to Dempster's as well as proposing the unifying concept of ''a disruption in inhibitory control of extraneous activity'' (p. 803) in order to explain what the tasks share at a theoretical level. Poor performance on the Wisconsin Card Sorting Test, especially when it is caused by an increase in the number of perseverations, is an indication of a decrease in inhibition. This test has long been used as a test of frontal lobe damage (Milner, 1963). There is enough overlap in performance with age-matched controls on this task that with small samples there are not always significant differences in overall performance measures. When there is a lack of a statistically significant difference in overall performance, there may still be differences in perseverations (Nelson, 1976).

Caudate Decline

In reading the literature on aging, frontal lobe damage, and Parkinson's and Huntington's disease together, the similarity in theory and data on inhibitory processes is striking. The only major difference is that researchers studying frontal lobe damage (and often aging) attribute the cause of decreases in inhibitory processes to frontal lobe damage whereas most researchers studying Parkinson's and Huntington's disease attribute the cause of decreases in inhibitory processes to the caudate.

Dubois, Boller, Pillon, and Agid (1991), reviewing the literature on Parkinson's disease, cite eight studies showing deficits in the Wisconsin Card Sort Test with Parkinson's disease, including the standard frontal lobe findings of greater number of trials to complete the first category, dissociation between knowing and doing, and more perseverative errors; later work (Cooper, Sagar, Jordan, Harvey, & Sullivan, 1991; Paulsen et al., 1995) and reviews (Mahurin, Feher, Nance, Levy, & Pirozzolo, 1993; Taylor & Saint-Cyr, 1995) confirm these find-

ings. Perseverative errors are the most clear inhibitory problem because they occur when patients fail to inhibit responses that were once correct, but now are wrong. These typical frontal-lobe deficits need not be part of general cognitive loss and can occur in the absence of other intellectual declines (Pillon, Dubois, Ploska, & Agid, 1991; Taylor et al., 1986). Patients with Parkinson's disease show a deficit on the Stroop task (Brown & Marsden, 1988; Hietanen & Trevainen, 1986).

Another set of inhibition related tasks reviewed by Dubois et al. (1991) center on the inability of patients with Parkinson's disease to shift attentional set or alternate between different conceptual categories. For instance, when deficits in recall versus recognition are next discussed, it will be noted that patients with Parkinson's disease are slower in listing all the members of a semantic category such as the names of animals or fruits. However, the deficit is even greater when they have to alternate between the two categories listing an animal, then a fruit, then an animal, and so forth, and the deficit is greater when the patients are off their L-dopa medication (Gotham, Brown, & Marsden, 1988). Inhibiting the prior category and then returning to it appears to be the problem. Similarly, in the Trail Making Test, Part B, the patients must connect consecutively numbered and lettered dots (e.g., 1-a-2-b-3-c-4-d . . .), which is an analog of the category shifting problem. In several studies, patients with Parkinson's disease have been shown to be poorer at this task relative to controls and relative to their own performance on Part A, which does not have a category alternation. Detailed studies that isolate various components of the task demonstrate that in patients with Parkinson's disease L-dopa medication has major effects on shifting ability (Hayes, Davidson, Keele, & Rafal, 1998; Owen et al., 1993). Moreover, although there are similarities among patients with frontal lobe damage and Parkinson's disease, differences can be noted in component processes that show promise of separating the roles of the various structures in the frontal-striatal circuit (Owen et al., 1993).

Especially relevant for the claims made here is a study of 7 patients with unilateral caudate lesions (Mendez, Adams, & Lewandowski, 1989). Because stroke and not Huntington's or Parkinson's disease was the cause of the computerized tomography (CT) or MRI confirmed caudate damage, the study provides converging evidence from humans that the caudate damage and not other aspects of the diseases is the causal factor for the cognitive deficit. Mendez et al. used a battery of 12 standard neuropsychological tests for a total of 17 reported subtests. Even with only 7 patients and 7 control participants, there were differences in tests that are held to measure decreased inhibition. On the Brown-Peterson Test the scores were 18.57 versus 28.00 ($t = 2.43$) for patients and control participants, respectively, and on the Wisconsin Card Sort Test they were 1.71 versus 5.14 ($t = 3.50$).

Patients with Huntington's disease show problems in both inhibiting responses to stimuli similar to ones that require responses and in alternating responses to letters and numbers (Sprengelmeyer, Lange, & Hömberg, 1995). Patients with Huntington's disease are also worse in reversal learning where a formerly correct response is now wrong and has to be inhibited, showing marked perseveration of the previously correct response (Lange, Sahakian, Quinn, Marsden, & Robbins, 1995).

EXECUTIVE CONTROL

Aging

In the cognitive aging literature, executive control or strategic planning has been shown to decline more rapidly with age than other functions (Baddeley, 1986; Craik & Byrd, 1982; Park, 1998). Differences in executive control or planning are often measured by differences between self-initiated and stimulus controlled tasks and by differences between recall (which requires a search strategy) and recognition (in which only a judgment of familiarity is required).

It is difficult to attribute tasks such as the Wisconsin Card Sorting Test to just the decreased inhibition or the executive control category and its results could be considered again here; all that really matters is that the same test

shows declines in the populations being compared here. The Self-Ordered Pointing Task (Petrides & Milner, 1982), which was developed to test frontal lobe damage, is another task that could go in either this section or the previous one. It consists of a person pointing to one of a set of cards on each trial. The cards appear in different locations on each trial and the person's task is to point to a different card each time. The task has shown a marked decline in aging that can be attributed to both a failure to inhibit past responses and to a lack of effective strategies to help remember what cards have been pointed to before (Daigneault & Braun, 1993; Daigneault, Braun & Whitaker, 1992; Shimamura & Jurica, 1994).

A case with clear face validity for executive control is the Tower of Hanoi problem because of its need for planning. In this test, discs must be moved among pegs with the restriction that a larger disc can never be placed on top of a smaller disc. The goal is to get all the discs to an indicated peg. Studies have shown age declines in this task (Brennan, Welsh, & Fisher, 1997; Vakil & Agmon-Ashinazi, 1997).

Verbal fluency, the number of words that come from a category that a person can list in a short time, is a measure of search strategy, especially when the category is not an existing semantic one, like *animals*, but is a relatively novel one such as *all the words beginning with the letter S.* This is because the category *all words beginning with the letter S* is an ad hoc category requiring a more complex and novel search strategy than a more structured category like *animals* (Rubin, 1990). Such fluency declines with age from young to older adulthood (Ardila & Rosselli, 1989; Isingrini & Vazou, 1997; Rosen, 1980), though the decline after age 50 appears to be in semantic category fluency tasks (Kozora & Cullem, 1995; Tomer & Levin, 1993).

In most studies, free recall declines more rapidly with increasing age than cued recall or recognition, presumably because more of a search strategy is needed for recall. In general, the aging literature supports this claim, though there are studies in which recall, cued recall, and recognition all decline at the same rate with age or fail to decline with age (Kausler, 1982). The free recall versus cued recall difference is especially apparent in categorized lists where category-title cues provide an effective retrieval strategy (Hultsch, 1975; Laurence, 1967). A skeptical reader of the literature could find the age difference in recall versus recognition not clearly supported once all methodological differences are controlled, especially differences in the ease of the tasks (Salthouse, 1991). However, for an equal degree of learning in the same task, recall usually shows a larger deficit with age.

Frontal Lobe Damage

Patients with frontal lobe damage show deficits on tests that require planning such as the Tower of Hanoi or in tasks that resemble the Self-Ordered Pointing Task (Owen, Downes, Sahakian, Polkey, & Robbins, 1990). Verbal fluency is another test that has long been used as a measure of frontal lobe damage, especially left or bilateral damage (Benton, 1968; Janowsky, Shimamura, Kritchevsky, & Squire, 1989; Pendleton, Heaton, Lehman, & Hulihan, 1982; Troyer, Moscovitch, Winocur, Alexander, & Stuss, 1998). The poorer performance of patients with frontal lobe damage appears to be caused by a decrease in the ability to switch from one group of related words to another (Troyer et al., 1998). Similarly, patients with frontal lobe damage show a larger deficit on recall than recognition compared to controls (Janowsky et al., 1989). Although there is reason to question the proposition that all tests used to measure inhibition and executive control in patients with frontal lobe damage form a psychometrically coherent diagnostic group (Duncan et al., 1997; Salthouse et al., 1996), for current purposes the characterization of frontal lobes as being involved in tasks said to measure executive control is well supported.

Caudate Decline

As with older adults and patients with frontal lobe damage, in Huntington's and Parkinson's disease there is a decrease in performance on the Self-Ordered Pointing Test (Gabrieli et al., 1996; Rich, Bylsma, & Brandt, 1996) and in Parkinson's disease in a task that is similar to it (Robbins et al., 1994). Patients with Parkinson's

and Huntington's disease have a deficit in the Tower of Hanoi task that varies with the disease and its severity (Lange et al., 1995; Lawrence et al., 1996; Robbins et al., 1994; Saint-Cyr, Taylor, & Lang, 1988).

A decrease in verbal fluency with caudate damage is also apparent. There is considerable support for difficulty with this task in Parkinson's (Cooper et al., 1991; Dalrymple-Alford, Kalders, Jones, & Watson, 1994; Dubois et al., 1991; Litvan, Mohr, Williams, Gomez, & Chase, 1991; Pillon, Dubois, Lhermitte, & Agid, 1986; Suhr & Jones, 1998) and Huntington's disease (Butters, Salmon, Heindel, & Granholm, 1988; Butters, Wolfe, Granholm, & Martone, 1986; Hodges, Salmon, & Butters, 1990; Rosser & Hodges, 1994; Rothlind & Brandt, 1993; Suhr & Jones, 1998). Butters et al. (1988) compared patients with Alzheimer's, Korsakoff's, and Huntington's disease with the same Dementia Scale rating scores to young and old control participants. All groups listed words beginning with particular letters and words from the category *animals*. Patients with Huntington's disease ''were the most consistently and severely impaired in their attempts to retrieve information'' (p. 69), with the most dramatic differences from the other clinical populations on the letter task. Rosser and Hodges (1994) found similar results. In contrast, Suhr and Jones (1998) found similar levels of deficits in patients with Huntington's, Parkinson's, and Alzheimer's disease with similar levels of performance on general tests of cognitive performance, though all clinical groups did more poorly than their age-matched controls, and patients with Huntington's disease had more repetitions of words already given.

The Mendez et al. (1989) study of 7 patients with unilateral caudate lesions found clear differences in retrieval difficulty. Using their reported t tests as descriptive indexes monotonically related to the size of their effects, the biggest difference between the 7 lesion and 7 control patients was in the long-term free recall of the California Verbal Learning Test (4.00 vs. 11.14, $t = 7.07$), and there was no significant difference on the long-term recognition subscale of the same test (15.00 vs. 15.29, $t = 0.76$). This is strong evidence for the recall versus recognition difference. On the Benton Word Fluency Test, there was a difference of 42 versus 76 words listed ($t = 2.63$).

The recall versus recognition difference is used in the clinical literature to provide a differential diagnosis between cortical dementias (e.g., Alzheimer's and amnesias caused by hippocampal or temporal lobe damage) and subcortical dementias (e.g., dementias caused by Parkinson's and Huntington's disease). Cortical dementias result in an anterograde loss that affects both recall and recognition, whereas subcortical dementias affect free recall much more than recognition or cued recall (Dubois et al., 1991; Gabrieli, 1995; Gabrieli et al., 1996; Maruyama, 1997; Moss, Albert, Butters, & Payne, 1986). In fact, on repeated recall of the same list, patients with Huntington's disease tend to recall different items on each trial, which is much more unusual for controls, suggesting a fluctuating retrieval strategy for stored information (Cain, Ebert, & Weingartner, 1977).

In addition to the tasks just reviewed, which can easily be compared across the aging, frontal lobe, and caudate groups, recent studies of patients with Parkinson's disease have tested Baddeley's (1986) model of executive control more directly by using dual task performance. Although these dual task tests are difficult to compare across groups because there is no standard pair of dual tasks to use, they do show disproportionate decline in the patients with Parkinson's disease (Brown & Marsden, 1991), even when the level of performance on the tasks performed individually is equated (Dalrymple-Alford et al., 1994).

COGNITIVE SLOWING

Aging

There is abundant evidence of a slowing of cognitive processes in aging (Cerella, 1985, 1991; Hicks & Birren, 1970; Noble, Baker, & Jones, 1964; Salthouse, 1994a, 1994b, 1996). Over a whole host of published studies using different tasks, the time taken by older adults is a linear function of the time taken by young adults; older participants have almost twice the response time

of the younger participants. That is, independent of the task or experimental condition within the task and independent of the theory used to motivate the study, the reaction time of 65-year-old participants is almost twice that of 20-year-old participants. Cerella (1991) concluded ''that the effects in some 288 experimental conditions are primarily determined by a single aspect of the information processing requirement, namely, task duration. The evidence is near-to-overwhelming that age is experienced, at least to a first approximation, as some sort of generalized slowing . . . success over such a diversity of data suggests that aging effects stem from some elementary aspect of the biology of the nervous system'' (pp. 220-221). Although Cerella's meta-analysis is compelling, it causes problems for many theories in the literature (also see Rubin, 1985 and Salthouse, 1992 for similar arguments). The 288 experimental conditions reanalyzed were not performed by their original experimenters to test for general slowing, but a variety of other more complex and more local theories of aging.

One way that the finding does fit into current theories is through the work of Salthouse and his colleagues. In a typical study, approximately 200 participants varying in age perform memory tasks such as paired-associate learning of a list (Salthouse, 1994a) or a measure of working memory span (Salthouse, 1994b). They also perform tasks that measure cognitive speed. Large, consistent age differences are found in both kinds of tasks and Salthouse then asks, in several different ways, how speed of processing is related to memory. In path analyses, age enters into the predictions most heavily by its effect on speed. Speed affects the degree of learning (i.e., immediate or zero lag recall), which affects percentage forgetting. Speed also has a direct effect on the percentage correct. Thus age has its main effects through cognitive response speed. More directly, the age-related variance in the measures of learning is greatly reduced if speed is controlled. Thus, many effects in cognitive aging can be described by a general slowing in cognitive processes.

Frontal Lobe Damage

Lezak (1995) mentions slowing as a symptom of frontal lobe damage and there is some direct evidence for this (Alivastos & Milner, 1989). However, cognitive slowing is not mentioned as a symptom of frontal lobe damage in the other sources reviewed to describe frontal lobe damage. Shimamura (1994), who has published extensively on the frontal lobes and on aging, attributes some aspects of cognitive decline with aging to the frontal lobes and others to the medial temporal lobes. However, he attributes cognitive slowing to neither of these specific structures but to a loss in general neuronal efficacy. Evidence that the main cause of slowing is location specific damage to the caudate rather than a general loss of neuronal efficacy is presented later, but for now Shimamura's view emphasizes the lack of mention of cognitive slowing in cases of frontal lobe damage. One can rarely claim that a particular symptom never accompanies a condition as diffuse as frontal lobe damage, and as noted earlier cognitive slowing does at times accompany frontal lobe damage, but the lack of emphasis on slowing in the literature compared with the emphasis on decreased inhibition and executive control implies that, when present, it is much less noticeable to most researchers. In contrast, cognitive slowing is central to many kinds of caudate damage. Thus, a first candidate for separating the roles of caudate and frontal lobe functions in cognitive aging should be the effects of slowing. The relative decline in specific tasks involving inhibition, executive control, and speed would be especially informative if differential effects were found in the different populations.

Caudate Decline

The strongest nonlaboratory evidence that the caudate is involved in a general cognitive slowing comes from work with Parkinson's disease. Rather than causing a general dementia, the cognitive effects of Parkinson's disease, at least at the early stages, are isolated and specific (Dubois et al., 1991). A major defining symptom of the disease is *bradyphrenia*, which as a symptom is a subjective general cognitive slowing or intellectual inertia, but which can be seen

in the laboratory ''as a symptom defined as a measurable lengthening of normal information-processing time'' (Dubois et al., 1991, p. 215). Although there is some disagreement about the definition and use of the term bradyphrenia as a description of all patients with Parkinson's disease, all that is needed here to parallel the aging literature is ''a measurable lengthening of normal information-processing time'' as measured by increased reaction times in the laboratory and that is available. In a meta-analysis of 70 studies that tested simple and choice reaction times in over 1,500 patients with Parkinson's disease and 1,500 controls, Wang, Thomas, and Stelmach (1998) found that patients were about one standard deviation slower than controls. Cognitive slowing occurs in both Parkinson's and Huntington's disease (Hanes, Andrewes, & Pantelis, 1995; Sprengelmeyer, Canavan, Lange, & Hömberg, 1995; Willingham, Koroshetz, Treadwell, & Bennett, 1995). Though it is not as pronounced a general symptom in Huntington's disease (Lezak, 1995), Huntington's disease often results in larger reaction time deficits than Parkinson's disease (Sprengelmeyer, Canavan, Lange, & Hömberg, 1995; Willingham, Koroshetz, Treadwell, & Bennett, 1995), again a difference that could be used in probing the localization of function.

Many of the studies report results on only a handful of patients and negative findings occur, and some of the positive findings could be attributed to executive control instead of slowing (Dubois et al., 1991). There is, however, strong evidence of slowing in fairly simple tasks that cannot easily be considered as heavily involving executive control or inhibition. For instance, Sprengelmeyer et al. (1995) found slowing for patients with Parkinson's and Huntington's disease with both a simple and a choice reaction time task in which much of the motor component is removed by timing only until a finger is removed from a rest location. Similarly, Willingham et al. (1995), for different simple and choice reaction time tasks, found slowing in patients with Parkinson's and Huntington's disease.

Hicks and Birren (1970) came to the conclusion that slowing is central rather than peripheral because with age there is a cognitive slowing but no, or at most little, decrease in nerve conduction velocity in both humans and laboratory animals. For instance, they noted that reflex latencies on long nerves do not change with age. Having established that the slowing is central and not peripheral and noting that many kinds of brain damage of a diffuse or unknown localization cause slowing, Hicks and Birren (1970) reviewed the evidence that damage to the caudate and related circuits reliably produces slowing, and in a later paper Birren and Fisher (1995) considered the role of subcortical white matter. In either case, slowing cannot easily be attributed to a general decrease in neuronal efficacy but must be central and probably, in part, subcortical.

As is found in the aging literature, slowing often increases relative to control populations as the task becomes more difficult (Malapani et al., 1994). Thus, with the sample sizes usually tested, slowing may not be present at a level that is statistically reliable when simple reaction time tasks are used but can become quite marked when the tasks become more complex cognitively (Hicks & Birren, 1970). Similarly, two simple reaction tasks which produce no noticeable slowing in patients with Parkinson's disease compared to controls can, when performed simultaneously, produce a marked differential (Malapani et al., 1994), and a simple reaction time might show no difference whereas one involving a decision will (Tachibana, Aragane, Miyata, & Sugita, 1997). The pattern is similar to that reviewed by Cerella and others in the cognitive aging literature (where the reaction time difference in young and old participants is proportional to the difficulty of the task as measured by the time taken by the young participants) and argues strongly against a motor interpretation of slowing with caudate damage.

DISCUSSION

Omissions

My goal was to argue as forcibly as possible that claims that the frontal lobes were centrally involved in cognitive aging were really claims that

the frontal-striatal circuits were centrally involved in cognitive aging. In doing so I made three major omissions. First, the presentation has been only in terms of the grossest of gross anatomical structures. As the frontal-caudate circuits are primarily dopaminergic circuits, whereas the hippocampal circuit is a primarily cholinergic circuit, our increasing knowledge of neurotransmitters (e.g., Cooper, Bloom, & Roth, 1996; Graybiel, 1990) should in the future be a great aid to our understanding of the behavioral changes that accompany aging. Similarly, our knowledge of more detailed anatomical structure can provide insight into behavior (Wise et al., 1996).

The second omission was that of the relation of the frontal-caudate circuits to other systems held to be important in cognitive aging. The behavioral and neuropsychological literature suggests that human cognition is supported by multiple memory systems. Consider two, which were among the first described (Mishkin, Malamut, & Bachevalier, 1984). One includes connections among the hippocampus, amygdala, mammillary bodies, medial thalamus, and rhinal and ventromedial prefrontal cortex. This medial temporal lobe system also declines with age, as noted earlier. The second memory system includes connections among the caudate, putamen, globus pallidus, ventral portions of the thalamus, substantia nigra, and frontal cortex. Others would add most of cortex, especially frontal cortex, to this second system (e.g., see the Houk et al., 1995 edited volume). In terms of function, Mishkin et al. (1984) have emphasized the hippocampal system for conscious, declarative, explicit, episodic memory and the caudate system for unconscious, skilled, implicit memory, although later work has shown that the caudate system is also involved in the explicit, conscious search that uses working memory (Gabrieli, 1995). From neuropsychological studies of various forms of amnesia it appears that the hippocampal system is needed to store new memories and its bilateral destruction produces post traumatic amnesias, but that the caudate system is needed for the explicit as well as implicit recall, as opposed to recognition, of stored material (Butters, 1984; Gabrieli, 1995; Moss et al. 1986).

The third omission was the hard task of sorting out what the various components of the frontal-striatal circuits actually do. Although the claims made here were restricted to the caudate, they might be extended to other parts of the frontal-striatal circuit if data were available (Cummings, 1993; Wise et al., 1996) or to other frontal-striatal circuits. Thus, instead of asking ''Could it be the caudate?'' we could say, ''Perhaps the putamen?'' The putamen, which appears at the level of the caudate in one of the three pathways not shown in Figure 1, is more involved in motor coordination than the cognitive tasks considered here. Its frontal-striatal circuits, which are better understood than those through the caudate (Alexander et al., 1986), pass through the supplementary motor areas at the cortical level. Consistent with regulation of the more motoric aspects of cognition, the putamen also receives a greater proportion of inputs from sensory, as opposed to association, cortices than the caudate and has somatotopic organization. According to a review of autopsy data on 8,000 brains (Eggers et al., 1984) and a review of imaging studies (Raz, in press), it decreases in size about as much as the caudate (15% each from 25 to 75 for the autopsy data; $r = -.44$ for the putamen versus –.47 for the caudate for the imaging studies). It would be a natural structure to be involved in age-related slowing and declines in motor coordination, which is why the studies of cognitive slowing (i.e., slowing that increases with cognitive vs. motoric difficulty) in patients with subcortical lesions cited earlier were careful to separate out motor effects (e.g., Sprengelmeyer et al., 1995; Willingham et al., 1995). In a recent study (Volkow et al., 1998), when age effects were partialed out, dopamine D_2 receptor availability in the putamen correlated only with finger tapping, whereas availability in the caudate correlated with finger tapping, the Wisconsin Card Sort Test, and the Stroop Test. Combining the frontal-striatal circuits through both the caudate and the putamen could account for many of the cognitive and sensorimotor, common cause, declines found in aging (Baltes & Lindenberger, 1997) rather than just declines commonly attributed to the frontal lobes.

In general, we need to begin asking which changes in behavior with aging are due to which neural structures in the frontal-striatal circuit and perhaps even which are best viewed as properties of the circuit as a whole. This is becoming common in studies of Parkinson's and Huntington's disease (Lange et al., 1995; Lawrence et al., 1996; Massman, Delis, Butters, Levin, & Salmon, 1990; Partiot et al., 1996; Robbins et al., 1994; Sprengelmeyer et al., 1995) and even in studies of aging that concentrate on the striatum (Volkow et al., 1998), but this cannot be done in aging research if all effects are attributed to frontal lobe decline. From this initial search, however, it appears that global concepts like inhibition, executive function, and slowing are too general for the task. The challenge is to develop concepts that can organize the existing data and provide new tests of localization of function (see Owen et al., 1993; Partiot et al., 1996; and Wise et al., 1996, for examples not involving aging). This call for theory and others like it (Brown & Marsden, 1990; Taylor & Saint-Cyr, 1995) are easy. Producing an adequate theory is harder (Light, 1991). Similarly, assembling and reviewing evidence of similarities in different populations as was done here is easy compared to finding methods and participants described in enough detail to attribute differences in behavior to damage in different areas of the brain.

Thus, tasks for the future are: (a) the inclusion of information about anatomical structure and neurotransmitters into our thinking about behavior; (b) considering how the frontal-striatal circuit integrates with the rest of the brain to produce the differences noted in cognitive aging; and (c) the subdivision of the behavioral deficits, which were summarized here using the categories of decreases in inhibition, executive control, and cognitive speed, into more fine-grained descriptions, preferably along theoretically motivated dimensions that can be more easily attributed to individual components of the frontal-striatal circuits. For now, this paper is both a cautionary tale about the difficulty of localizing function in particular brain structures and a claim that we have, in fact, made an error. Much of what has been attributed to cortex is probably due to subcortical structures.

Could It Be Just the Frontal Lobes?

Aging is a complex process involving all aspects of human experience and physiology. It would be odd to find that most of the changes reside in one particular brain structure. On the other hand, localization of specific functions exists. Moreover, localization of function is an attractive, almost seductive, idea. If you know where something is, then it must be real and you certainly know more about it than someone who cannot even find it. Here I have taken a widely held attribution of changes in behavior to one neural location and argued that it should really be attributed to a circuit involving that location by focusing on another location in that circuit. If one had to choose either the frontal lobes or the caudate as the cause of cognitive aging, it would be a hard choice; but if anything, the caudate accounts for more of the data because a clearer case can be made for slowing. The only reason that I can imagine for the literature's current choice of the frontal lobes to the exclusion of the caudate is that the choice is based on the additional, unstated assumption that a loss in a higher function must be due to loss in a higher structure; only cortical as opposed to subcortical gray matter could be involved in executive control. However, such a binary choice between structures as the neural basis of all the effects of cognitive aging reviewed here is not needed and is not the optimal route to follow.

Nevertheless, given all the literature reviewed, the skeptic might ask if it is possible that all processing of the cognitive tasks considered is taking place in the frontal lobes (or caudate) and that all the caudate (or frontal lobes) does is passively recirculate the information without changing it, serving only as a distribution center to other parts of the brain. It is unlikely. It would be a very odd design for gray matter structures. But it is possible. Studies that measure changes in size or changes in activity in either the frontal lobes or the caudate, but not both, are useless on this issue, even if they measure relevant behaviors and relate them to neural measures. The structure not measured could

show more change. Studies that measure neural change in both structures are a bit more relevant, but not definitive. For example, the observation that the caudate shrinks at least as much as the frontal lobes with age may mean only that a structure that is passive with respect to cognition deteriorates more and causes a lack of input to the important structure.

The studies that are the most useful are those that measure neural and behavioral change in two or more samples with damage to two or more structures, but even here the conclusions will not be straightforward without detailed measures. One reason is that double dissociations of gross measures will not be as likely in highly interconnected structures and therefore detailed functions for each structure will have to be postulated (see Owen et al., 1993, or Partiot et al., 1996, for an example). Another reason is that the damaged area in the frontal lobe sample would have to project to the area of the caudate that is damaged in the caudate sample or differences would be observed even if one structure were totally passive; that is, the same circuit would have to be damaged in both samples for a comparison to be made.

These reminders of the complexity of the neuropsychology aside, the evidence presented here suggests that when careful studies are conducted with the intent of finding the roles of structures in the frontal-striatal circuit in cognitive aging, both the frontal lobes and the caudate will be important in related but distinct ways. Until then, when faced with the claim that the frontal lobe decline causes much of cognitive aging, one should ask of the evidence, could it be the caudate? If the answer is yes, then circuits instead of structures need to be considered.

REFERENCES

Albert, M. S., & Kaplan, E. (1980). Organic implications of neuropsychological deficits in the elderly. In L. W. Poon, J. L. Fozard, L. S. Cermak, D. Arenberg, & L. W. Thompson (Eds.), *New directions in memory and aging: Proceedings of the George A. Talland Memorial Conference* (pp. 403-432). Hillsdale, NJ: Lawrence Erlbaum.

Alexander, G. E., & Crutcher, M. D. (1990). Functional architecture of basal ganglia circuits: Neural substrates of parallel processing. *Trends in Neuroscience*, *13*, 266-271.

Alexander, G. E., Delong, M. R., & Strick, P. L. (1986). Parallel organization of functionally segregated circuits linking basal ganglia and cortex. *Annual Review of Neuroscience*, *9*, 357-381.

Alivisatos, B., & Milner, B. (1989). Effects of frontal or temporal lobectomy on the use of advance information in a choice reaction time task. *Neuropsychologia*, *27*, 495-503.

Antonini, A., Leenders, K. L., Reist, H., Thomann, R., Beer, H-F., & Locher, J. (1993). Effect of age on D_2 dopamine receptors in normal human brain measured by positron emission tomography and ^{11}C-raclopride. *Archives of Neurology*, *50*, 474-480.

Ardila, A., & Rosselli, M. (1989). Neuropsychological characteristics of normal aging. *Developmental Neuropsychology*, *5*, 307-320.

Bäckman, L., Ginovart, N., Dixon, R. A., Robins-Wahlin, T.-B., Whalin, Å., Halldin, C., & Farde, L. (in press). Age related cognitive deficits are mediated by changes in the striatal dopaminergic system. *American Journal of Psychiatry*.

Bäckman, L., Robins-Wahlin, T.-B., Lundin, A., Ginovart, N., & Farde, L. (1997). Cognitive deficits in Huntington's disease are predicted by dopaminergic PET markers and brain volumes. *Brain*, *120*, 2207-2217.

Baddeley, A. (1986). *Working memory.* Oxford: Clarendon Press.

Baltes, P. B., & Lindenberger, U. (1997). Emergence of a powerful connection between sensory and cognitive functions across the adult life span: A new window to the study of cognitive aging. *Psychology and Aging*, *12*, 12-21.

Benton, A. (1968). Differential behavioral effects in frontal lobe disease. *Neuropsychologia*, *6*, 53-60.

Birren, J. E., & Fisher, L. M. (1995). Aging and speed of behavior: Possible consequences for psychological functioning. *Annual Review of Psychology*, *46*, 329-353.

Brandt, J. A., & Bylsma, F. W. (1993). The dementia of Huntington's disease. In R. W. Parks, R. F. Zec, & R. S. Wilson (Eds.), *Neuropsychology of Alzheimer's disease and other dementias* (pp. 265-282). New York: Oxford University Press.

Brennan, M., Welsh, M. C., & Fisher, C. B. (1997). Aging and executive function skills: An examination of a community-dwelling older adult population. *Perceptual and Motor Skills*, *84*, 1187-1197.

Brown, R. G., & Marsden, C. D. (1988). An investigation of the phenomenon of ''Set'' in Parkinson's disease. *Movement Disorders*, *3*, 152-161.

Brown, R. G., & Marsden, C. D. (1990). Cognitive function in Parkinson's disease: From description to theory. *Trends in Neuroscience*, *13*, 21-29.

Brown, R. G., & Marsden, C. D. (1991). Dual task performance and processing resources in normal subjects and patients with Parkinson's disease. *Brain*, *114*, 215-231.

Butters, N. (1984). The clinical aspects of memory disorders: Contributions from experimental studies of amnesia and dementia. *Journal of Clinical Neuropsychology 6*, 17-36.

Butters, N., Salmon, D. P., Heindel, W., & Granholm, E. (1988). Episodic, semantic, and procedural memory: Some comparisons of Alzheimer and Huntington disease patients. In R. D. Terry (Ed.), *Aging and the Brain* (pp. 63-87). New York: Raven Press.

Butters, N., Wolfe, J., Granholm, E., & Martone, M. (1986). An assessment of verbal recall, recognition and procedural memory. *Cortex*, *22*, 11-32.

Cain, E., Ebert, M., & Weingartner, H. (1977). An outline for the analysis of dementia: The memory disorder of Huntington's disease. *Neurology*, *27*, 1087-1092.

Cerella, J. (1985). Information processing rates in the elderly. *Psychological Bulletin*, *98*, 67-83.

Cerella, J. (1991). Age effects may be global, not local: Comment on Fisk and Rogers (1991). *Journal of Experimental Psychology: General*, *120*, 215-223.

Cooper, J. A., Sagar, H. J., Jordan, N., Harvey, N. S., & Sullivan, E. V. (1991). Cognitive impairment in early, untreated Parkinson's disease and its relationship to motor disability. *Brain*, *114*, 2095-2122.

Cooper, J. R., Bloom, F. E., & Roth, R. H. (1996). *The biochemical basis of neuropharmacology.* New York: Oxford University Press.

Craik, F. I. M., & Byrd, M. (1982). Aging and cognitive deficits: The role of attentional resources. In F. I. M. Craik & S. Trehub (Eds.), *Aging and cognitive processes* (pp. 191-211). New York: Plenum Press.

Craik, F. I. M., & Jennings, J. M. (1992). Human memory. In F. I. M. Craik & T. A. Salthouse (Eds.), *The handbook of aging and cognition* (pp. 51-110). Hillsdale, NJ: Lawrence Erlbaum.

Cummings, J. L. (1993). Frontal-subcortical circuits and human behavior. *Archives of Neurology*, *50*, 873-880.

Daigneault, S., & Braun, C. M. (1993). Working memory and the Self-Ordered Pointing Task: Further evidence of early prefrontal decline in normal aging. *Journal of Clinical and Experimental Neuropsychology*, *16*, 881-895.

Daigneault, S., Braun, C. M. J., & Whitaker, H. A. (1992). Early effects of normal aging on perseverative and non-perseverative prefrontal measures. *Developmental Neuropsychology*, *8*, 99-114.

Dalrymple-Alford, J. C., Kalders, A. S., Jones, R. D., & Watson, R. W. (1994). A central executive deficit in patients with Parkinson's disease. *Journal of Neurology, Neurosurgery, & Psychiatry*, *57*, 360-367.

de la Fuente-Fernandez, R., Kishore, A., Snow, B. J., Schulzer, M., Lee, C. S., Ruth, A. J., & Stoessl, A. J. (1998). Effects of age on caudate dopaminergic function in Idiopathic Parkinsonism. *Parkinsonism and Related Disorders*, *4*, 1-5.

Dempster, F. N. (1992). The rise and fall of the inhibitory mechanism: Toward a unified theory of cognitive development and aging. *Developmental Review*, *12*, 45-75.

Divac, I., Rosvold, H. E., & Szwarcbart, M. K. (1967). Behavioral effects of selective ablation of the caudate nucleus. *Journal of Comparative and Physiological Psychology*, *63*, 184-190.

Dubois, B., Boller, F., Pillon, B., & Agid, Y. (1991). Cognitive deficits in Parkinson's disease. In S. Corkin, J. Grafman, & F. Boller (Eds.) *Handbook of Neuropsychology*, Vol. 6. (pp. 195-240). New York: Elsevier.

Duncan, J., Johnson, R., Swales, M., & Freer, C. (1997). Frontal lobe deficits after head injury: Unity and diversity of function. *Cognitive Neuroscience*, *14*, 713-741.

Eggers, R., Haug, H., & Fischer, D. (1984). Preliminary report on macroscopic age changes in the human prosencephalon: A stereologic investigation. *Journal fuer Hirnforschung*, *25*, 129-139.

Finch, C. E. (1979). The relationship of aging changes in the basal ganglia to manifestations of Huntington's Chorea. *Annals of Neurology*, *7*, 406-411.

Gabrieli, J. (1995). Contribution of the basal ganglia to skill learning and working memory in humans. In J. C. Houk, J. L. Davis, & D. G. Beiser (Eds.), *Models of information processing in the basal ganglia* (pp. 287-294). Cambridge, MA: MIT Press.

Gabrieli, J. D. E., Singh, J., Stebbins, G. T., & Goetz, C. G. (1996). Reduced working memory span in Parkinson's disease: Evidence for the role of a frontalstriatal system in working and strategic memory. *Neuropsychology*, *10*, 322-332.

Goldman-Rakic, P. S., & Selemon, L. D. (1990). New frontiers in basal ganglia research. *Trends in Neuroscience*, *13*, 240-243.

Gotham, A. M., Brown, R. G., & Marsden, C. D. (1988). 'Frontal' cognitive function in patients with Parkinson's disease 'on' and 'off' levodopa. *Brain*, *111*, 299-321.

Graybiel, A. M. (1990). Neurotransmitters and neuromodulators in the basal ganglia. *Trends in Neuroscience*, *13*, 244-253.

Hanes, K. R., Andrewes, D. G., & Pantelis, C. (1995). Cognitive flexibility and complex integration in Parkinson's disease, Huntington's disease, and schizophrenia. *Journal of the International Neuropsychological Society*, *1*, 545-553.

Hasher, L., & Zacks, R. T. (1988). Working memory, comprehension, and aging: A review and a new

view. In G. H. Bower (Ed.), *The psychology of learning and motivation*, (Vol. 22, pp. 193-225). San Diego, CA: Academic Press.

Hayes, A. E., Davidson, M. C., Keele, S. W., & Rafal, R. D. (1998). Toward a functional analysis of the basal ganglia. *Journal of Cognitive Neuroscience*, *10*, 178-198.

Hicks, L. H., & Birren, J. E. (1970). Aging, brain damage, and psychomotor slowing. *Psychological Bulletin*, *74*, 377-396.

Hietanen, M., & Teräväinen, H. (1986). Cognitive performance in early Parkinson's disease. *Acta Neurologica Scandinavia*, *73*, 151-159.

Hodges, J. R., Salmon, D. P., & Butters, N. (1990). Differential impairment of semantic and episodic memory in Alzheimer's and Huntington's disease: A controlled prospective study. *Journal of Neurology, Neurosurgery & Psychiatry*, *53*, 1089-1095.

Houk, J. C., Davis, J. L., & Beiser, D. G. (Eds.). (1995). *Models of information processing in the basal ganglia.* Cambridge, MA: MIT Press.

Hultsch, D. F. (1975). Adult age differences in retrieval: Trace-dependent and cue-dependent forgetting. *Developmental Psychology*, *11*, 197-201.

Isingrini, M., & Vazou, F. (1997). Relation between fluid intelligence and frontal lobe functioning in older adults. *International Journal of Aging & Human Development*, *45*, 99-109.

Janowsky, J. S., Shimamura, A. P., Kritchevsky, M., & Squire, L. R. (1989). Cognitive impairment following frontal lobe damage and its relevance to human amnesia. *Behavioral Neuroscience*, *103*, 548-560.

Jernigan, T. L., Archibald, S. L., Berhow, M. T., Sowell, E. R., Foster, D. S., & Hesselink, J. R. (1991). Cerebral structure on MRI, Part I: Localization of age-related changes. *Biological Psychology*, *29*, 55-67.

Jernigan, T. L., Salmon, D. P., Butters, N., & Hesselink, J. R. (1991). Cerebral structure on MRI, Part II: Specific changes in Alzheimer's and Huntington's diseases. *Biological Psychology*, *29*, 68-81.

Kausler, D. H. (1982). *Experimental psychology and human aging.* New York: Wiley.

Kish, S. J., Zhong, X. H., Hornykiewicz, O., & Haycock, J. W. (1995) Striatal 3,4-dihydroxyphenylalanine decarboxylase in aging: Disparity between postmortem and positron emission tomography studies? *Annals of Neurology*, *38*, 260-264.

Kozora, E., & Cullum, C. M. (1995). Generative naming in normal aging – total output and qualitative changes using phonemic and semantic constraints. *Clinical Neuropsychologist*, *9*, 313-320.

Krishnan, K. R. R., Husain, M. M., McDonald, W. M., Doraiswamy, P.M., Figiel, G. S., Boyko, O. B., Ellinwood, E. H., Jr., & Nemeroff, C. B.. (1990). In vivo stereological assessment of caudate volume in man: Effect of normal aging. *Life Science*, *47*, 1325-1329.

Krishnan, K. R. R., McDonald, W. M., Escalona, P. R., Doraiswamy, P. M., Chul, N., Husain, M. M., Figiel, G. S., Boyko, O. B., Ellinwood, E. H., Jr., & Nemeroff, C. B. (1992). Magnetic Resonance Imaging of the caudate nuclei in depression. *Archives of General Psychiatry*, *49*, 553-557.

Lange, K. W., Sahakian, B. J., Quinn, N. P., Marsden, C. D., & Robbins, T. W. (1995). Comparison of executive and visuospatial memory function in Huntington's disease and dementia of Alzheimer type matched for degree of dementia. *Journal of Neurology, Neurosurgery, and Psychiatry*, *58*, 598-608.

Laurence, M. W. (1967). Memory loss with age: A test of two strategies for its retardation. *Psychonomic Science*, *9*, 209-210.

Lawrence, A. D., Sahakian, B. J., Hodges, J. R., Rosser, A. E., Lange, K. W., & Robbins, T. W. (1996). Executive and mnemonic functions in early Huntington's disease. *Brain*, *119*, 1633-1645.

Lezak, M. D. (1995). *Neuropsychological assessment* (3rd ed.). New York: Oxford University Press.

Light, L. L. (1991). Memory and aging: Four hypotheses in search of data. *Annual Review of Psychology*, *42*, 333-376.

Litvan, I., Mohr, E., Williams, J., Gomez, C., & Chase, T. N. (1991). Differential memory and executive functions in demented patients with Parkinson's and Alzheimer's disease. *Journal of Neurology, Neurosurgery & Psychiatry*, *54*, 25-29.

Mahurin, R. K., Feher, E. P., Nance, M. L., Levy, J. K., & Pirozzolo, F. J. (1993). Cognition in Parkinson's disease and related disorders. In R. W. Parks, R. F. Zec, & R. S. Wilson (Eds.), *Neuropsychology of Alzheimer's disease and other dementias* (pp. 309-349). New York: Oxford University Press.

Malapani, C., Pillon, B., Dubois, B., & Agid, Y. (1994). Impaired simultaneous cognitive task performance in Parkinson's disease: A dopamine-related dysfunction. *Neurology*, *44*, 319-326.

Maruyama, T. (1997). Deficit of explicit memory in Parkinson's disease demonstrated by auditory-verbal and visual-design learning tasks. *Nippon Rinsho-Japanese Journal of Clinical Medicine*, *55*, 195-201.

Massman, P. J., Delis, D. C., Butters, N., Levin, B. E., & Salmon, D. P. (1990). Are all subcortical dementias alike? Verbal learning and memory in Parkinson's and Huntington's disease patients. *Journal of Clinical and Experimental Neuropsychology*, *12*, 729-744.

Meck, W. H. (1996). Neuropharmacology of timing and time perception. *Cognitive Brain Research*, *3*, 227-242.

Mendez, M. F., Adams, N. L., & Lewandoski, K. S. (1989). Neurobehavioral changes associated with caudate lesions. *Neurology*, *39*, 349-354.

Milner, B. (1963). Effects of different brain lesions on card sorting. *Archives of Neurology*, *9*, 90-100.

Mishkin, M., Malamut, B., & Bachevalier, J. (1984). Memories and habits: Two neural systems. In G. Lynch, J. L. McGaugh, & N. M. Weinberger (Eds.), *Neurobiology of Memory* (pp. 65-77). New York: Guilford Press.

Mittenberg, W., Seidenberg, M., O'Leary, D. S., & DiGiulio, D. V. (1989). Changes in cerebral functioning associated with normal aging. *Journal of Clinical and Experimental Neuropsychology*, *11*, 918-922.

Moscovitch, M., & Winocur, G. (1992). The neuropsychology of memory and aging. In F. I. M. Craik & T. A. Salthouse (Eds.), *The handbook of aging and cognition* (pp. 315-372). Hillsdale, NJ: Lawrence Erlbaum.

Moss, M. B., Albert, M. S., Butters, N., & Payne, M. (1986). Differential patterns of memory loss among patients with Alzheimer's disease, Huntington's disease and alcoholic Korsakoff's syndrome. *Archives of Neurology*, *43*, 239-246.

Nelson, H. E. (1976). A modified card sorting test sensitive to frontal lobe deficits. *Cortex*, *12*, 313-324.

Noble, C. E., Baker, B. L., & Jones, T. A. (1964). Age and sex parameters in psychomotor learning. *Perceptual and Motor Skills*, *19*, 935-945.

Owen, A. M., Downes, J. J., Sahakian, B. J., Polkey, C. E., & Robbins, T. W. (1990). Planning and spatial working memory following frontal lobe lesions in man. *Neuropsychologia*, *28*, 1021-1034.

Owen, A. M., Roberts, A. C., Hodges, J. R., Summers, B. A., Polkey, C. E., & Robbins, T. W. (1993). Contrasting mechanisms of impaired attentional set-shifting in patients with frontal lobe damage or Parkinson's disease. *Brain*, *116*, 1159-1175.

Parent, A., & Hazrati, L-N. (1995). Functional anatomy of the basal ganglia. I. The cortico-basal ganglia-thalamo-cortical loop. *Brain Research Review*, *20*, 91-127.

Park, D. C. (1998). Cognitive aging, processing resources, and self-report. In N. Schwarz, D. C. Park, B. Knauper, & S. Sudman (Eds.), *Cognition, aging, and self-reports* (pp. 45-70). Philadelphia: Psychology Press.

Partiot, A., Vérin, M., Pillon, B., Teixeira-Ferreira, C., Agid, Y., & Dubois, B. (1996). Delayed response tasks in basal ganglia lesions in man: Further evidence for a striato-frontal cooperation in behavioural adaptation. *Neuropsychologia*, *34*, 709-721.

Paulsen, J. S., Salmon, D. P., Monsch, A. U., Butters, N., Swenson, M. R., & Bondi, M. W. (1995). Discrimination of cortical from subcortical dementias on the basis of memory and problem-solving tests. *Journal of Clinical Psychology*, *51*, 48-58.

Pendleton, M. G., Heaton, R. K., Lehman, R. A. W., & Hulihan, D. (1982). Diagnostic utility of the Thurstone Word Fluency Test in neuropsychological evaluations. *Journal of Clinical Neuropsychology*, *4*, 307-317.

Petrides, M., & Milner, B. (1982). Deficits on subject-ordered tasks after frontal- and temporal-lobe lesions in man. *Neuropsychologia*, *20*, 249-262.

Pillon, B., Dubois, B., Lhermitte, F., & Agid, Y. (1986). Heterogeneity of cognitive impairment in progressive supranuclear palsy, Parkinson's disease, and Alzheimer's disease. *Neurology*, *36*, 1179-1185.

Pillon, B., Dubois, B., Ploska, A., & Agid. Y. (1991). Severity and specificity of cognitive impairment in Alzheimer's, Huntington's, and Parkinson's diseases and progressive supranuclear palsy. *Neurology*, *41*, 634-643.

Raz, N. (in press). Aging of the brain and its impact on cognitive performance: Integration of structural and functional findings. In F. I. M. Craik and T. A. Salthouse (Eds.), *Handbook of aging and cognition – II.* Mahwah, NJ: Erlbaum.

Rich, J. B., Bylsma, F. W., & Brandt, J. (1996). Self-ordered pointing performance in Huntington's disease patients. *Neuropsychiatry, Neuropsychology and Behavioral Neurology*, *9*, 99-106.

Robbins, T. W., James, M., Owen, A. M., Lange, K. W., Lees, A. J., Leigh, P. N., Marsden, C. D., Quinn, N. P., & Summers, B. A. (1994). Cognitive deficits in progressive supranuclear palsy, Parkinson's disease, and multiple system atrophy in tests sensitive to frontal lobe dysfunction. *Journal of Neurology, Neurosurgery, and Psychiatry*, *57*, 79-88.

Rosen, W. G. (1980). Verbal fluency in aging and dementia. *Journal of Clinical Neuropsychology*, *2*, 135-146.

Rosser, A., & Hodges, J. R. (1994). Initial letter and semantic category fluency in Alzheimer's disease, Huntington's disease, and progressive supranuclear palsy. *Journal of Neurology, Neurosurgery & Psychiatry*, *57*, 1389-1394.

Rothlind, J. C., & Brandt, J. (1993). A brief assessment of frontal and subcortical functions in dementia. *Journal of Neuropsychiatry and Clinical Neurosciences*, *5*, 73-77.

Rubin, D. C. (1985). Memorability as a measure of processing: A unit analysis of prose and list learning. *Journal of Experimental Psychology: General*, *114*, 213-238.

Rubin, D. C. (1990). Directed graphs as memory representations: The case of rhyme. In R. W. Schvaneveldt (Ed.), *Pathfinder associative networks: Studies in knowledge organization* (pp. 121-133). Norwood, NJ: Ablex.

Saint-Cyr, J. A., Taylor, A. E., & Lang, A. E. (1988). Procedural learning and neostriatal dysfunction in man. *Brain*, *111*, 941-959.

Salthouse, T. A. (1991). Mediation of adult age differences in cognition by reductions in working mem-

ory and speed of processing. *Psychological Science*, *2*, 179-183.
Salthouse, T. A. (1992). Influence of processing speed on adult age differences in working memory. *Acta Psychologica*, *79*, 155-170.
Salthouse, T. A., (1994a). Aging associations: Influence of speed on adult age differences in associative learning. *Journal of Experimental Psychology, Learning, Memory, and Cognition*, *20*, 1486-1503.
Salthouse, T. A. (1994b). The nature of the influence of speed on adult age differences in cognition. *Developmental Psychology*, *30*, 240-259.
Salthouse, T. A. (1996). The processing-speed theory of adult age differences in cognition. *Psychological Review*, *103*, 403-428.
Salthouse, T. A., Fristoe, B. N., & Rhee, S. H. (1996). How localized are age-related effects on neuropsychological measures? *Neuropsychology*, *10*, 272-285.
Shimamura, A. P. (1994). Neuropsychological perspectives on memory and cognitive decline in normal human aging. *Seminars in the Neurosciences*, *6*, 387-394.
Shimamura, A. P. (1995). Memory and frontal lobe function. In M. S. Gazzaniga (Ed.), *The cognitive neurosciences* (pp. 803-813). Cambridge, MA: MIT Press.
Shimamura, A. P., & Jurica, P. J. (1994). Memory interference effects and aging: Findings from a test of frontal lobe function. *Neuropsychology*, *8*, 408-412.
Sprengelmeyer, R., Lange, H., & Hömberg, V. (1995). The pattern of attentional deficits in Huntington's disease. *Brain*, *118*, 145-152.
Sprengelmeyer, R., Canavan, A. G. M., Lange, H. W., & Hömberg, V. (1995). Associative learning in degenerative neostriatal disorders: Contrasts in explicit and implicit remembering between Parkinson's and Huntington's diseases. *Movement Disorders*, *10*, 51-65.
Suhr, J. A., & Jones, R. D. (1998). Letter and semantic fluency in Alzheimer's, Huntington's, and Parkinson's dementias. *Archives of Clinical Neuropsychology*, *13*, 447-454.
Tachibana, H., Aragane, K., Miyata, Y, & Sugita, M. (1997). Electrophysiological analysis of cognitive slowing in Parkinson's disease. *Journal of Neurological Sciences*, *149*, 47-56.
Taylor, A. E., & Saint-Cyr, J. A. (1995). The neuropsychology of Parkinson's disease. *Brain and Cognition*, *28*, 281-296.
Taylor, A. E., Saint-Cyr, J. A., & Lang, A. E. (1986). Frontal lobe dysfunction in Parkinson's disease. *Brain*, *109*, 845-883.
Tomer, R., & Levin, B. E. (1993). Differential-effects of aging on 2 verbal fluency tasks. *Perceptual and Motor Skills*, *76*, 465-466.
Troyer, A. K., Moscovitch, M., Winocur, G., Alexander, M. P., & Stuss, D. (1998). Clustering and switching on verbal fluency: The effects of focal frontal- and temporal-lobe lesions. *Neuropsychologia*, *36*, 499-504.
Vakil, E., & Agmon-Ashkenazi, D. (1997). Baseline performance and learning rate of procedural and declarative memory tasks: Younger versus older adults. *Journals of Gerontology Series B-Psychological Sciences and Social Sciences*, *52*, 229-234.
Van Gorp, W. G., & Mahler, M. (1990). Subcortical features of normal aging . In J. L. Cummings (Ed.), *Subcortical dementia* (pp. 231-250). New York: Oxford University Press.
Volkow, N. D., Gur, R. C., Wang, G-J., Fowler, J. S., Moberg, P. J., Ding, Y-S., Hitzemann, R., Smith, G., & Logan, J. (1998). Association between decline in brain dopamine activity with age and cognitive and motor impairment in healthy individuals. *American Journal of Psychiatry*, *155*, 344-349.
Wang, J., Thomas, J. R., & Stelmach, G. E. (1998). A meta-analysis on cognitive slowing in Parkinson's disease: Are simple and choice reaction times differentially impaired? *Parkinsonism and Related Disorders*, *4*, 17-29.
Wang, Y., Chan, G. L., Holden, J. E., Dobko, T., Mak, E., Schulzer, M., Huser, J. M., Snow, B. J., Ruth, T. J., Calne, D. B., & Stoessl, A.J. (1998). Age-dependent decline of dopamine D1 receptors in human brain: A PET study. *Synapse*, *30*, 56-61.
West, R. L. (1996). An application of prefrontal cortex function theory to cognitive aging. *Psychological Bulletin*, *120*, 272-292.
Whelihan, W. M., & Lesher, E. L. (1985). Neuropsychological changes in frontal functions with aging. *Developmental Neuropsychology*, *1*, 371-380.
Willingham, D. B., Koroshetz, W. J., Treadwell, J. R., & Bennett, J. P. (1995). Comparison of Huntington's and Parkinson's disease patients' use of advance information. *Neuropsychology*, *9*, 39-46.
Wise, S. P., Murray, E. A., & Gerfen, C. R. (1996). The frontal cortex – basal ganglia system in primates. *Critical Reviews in Neurobiology*, *10*, 317-356.