Child Development, January/February 2000, Volume 71, Number 1, Pages 44–56

Close Interrelation of Motor Development and Cognitive Development and of the Cerebellum and Prefrontal Cortex

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Motor development and cognitive development may be fundamentally interrelated. Contrary to popular notions that motor development begins and ends early, whereas cognitive development begins and ends later, both motor and cognitive development display equally protracted developmental timetables. When cognitive development is perturbed, as in a neurodevelopmental disorder, motor development is often adversely affected. While it has long been known that the striatum functions as part of a circuit with dorsolateral prefrontal cortex, it is suggested here that the same is true for the cerebellum and that the cerebellum may be important for cognitive as well as motor functions. Like prefrontal cortex, the cerebellum reaches maturity late. Many cognitive tasks that require prefrontal cortex also require the cerebellum. To make these points, evidence is summarized of the close co-activation of the neocerebellum and dorsolateral prefrontal cortex in functional neuroimaging, of similarities in the cognitive sequelae of damage to dorsolateral prefrontal cortex and the neocerebellum, of motor deficits in "cognitive" developmental disorders, and of abnormalities in the cerebellum and in prefrontal cortex in the same developmental disorders.

INTRODUCTION

In general, motor development and cognitive development have been studied separately. They have generally been viewed as independent phenomena, although occurring in the same organism over the same time period. Indeed, cognitive development, as befits its exalted status, has generally been viewed as the last aspect of development to fully mature. Developmental psychologists have tended to forget that motor development is equally protracted. Fine motor control, bimanual coordination, and visuomotor skills are not fully developed until adolescence, just as the most complex cognitive operations such as accurately representing transformations, flexibly manipulating information held in mind, and simultaneously taking into account multiple facets of a problem show developmental improvements into adolescence.

Motor development and cognitive development may be much more interrelated than has been previously appreciated. Indeed, they may be fundamentally intertwined.

Similarly, until very recently, prefrontal cortex and the neocerebellum were not thought to participate in similar functions.[1] Dorsolateral prefrontal cortex is thought to be critical for the most complex cognitive abilities, whereas the cerebellum has been considered critical primarily for motor skills. In keeping with the lofty status of dorsolateral prefrontal cortex, its protracted developmental timetable and dramatic expansion during primate evolution have been emphasized. The fact that the neocerebellum, which also has undergone dramatic expansion during primate evolution, is late-maturing as well has received less attention, although there has been evidence for over 55 years (see Dow, 1942) that phylogenetic development of the neocerebellum and of prefrontal cortex have proceeded in parallel.

It is suggested here that the cerebellum is important not only for motor functions but also for cognitive functions. Indeed, the cerebellum is important for the very same cognitive functions for which dorsolateral prefrontal cortex is critical. Most cognitive tasks that require dorsolateral prefrontal cortex also require the neocerebellum.

[1] The region of prefrontal cortex on which this paper focuses is the "dorsolateral prefrontal cortex," i.e., Areas 46 and 9. In the human brain, dorsolateral prefrontal cortex extends above and below the superior frontal sulcus, past the medial frontal sulcus to the inferior frontal sulcus. It is bordered posteriorally by Area 8 and anteriorally by Area 10. The portion of the cerebellum on which this paper focuses is the "neocerebellum," i.e., the phylogenetically newer regions of the cerebellum. These regions mature later during ontogeny than other cerebellar regions and are interconnected with the cerebral cortex. The neocerebellum includes the posterior lobe of the lateral hemispheres of cerebellar cortex (lobules HVI through HVIII, Larsell & Jansen, 1972), lobules VI and VII of the vermis in the medial portion of cerebellar cortex, and one of the deep cerebellar nuclei (the dentate nucleus). Like the rest of the neocerebellum, the dentate nucleus has increased in size in parallel with prefrontal cortex.

EVIDENCE FROM NEUROIMAGING OF THE CLOSE INTERRELATION OF DORSOLATERAL PREFRONTAL CORTEX AND THE NEOCEREBELLUM

Functional neuroimaging studies consistently find that when a cognitive task increases activation in dorsolateral prefrontal cortex it also increases activation in the contralateral cerebellum. When one sees decreased dorsolateral prefrontal cortex activation (e.g., when a task has been practiced and requires less concentration) one also sees a concomitant decrease in cerebellar activation. Activation in these two regions is strikingly correlated and closely coupled.

This co-activation of dorsolateral prefrontal cortex and the contralateral neocerebellum has been found with the "verb generation" task (Raichle et al., 1994), where on each trial participants are given a noun and asked to quickly generate a related verb. Co-activation has been found on the "verbal fluency" task (Schlosser et al., 1998), where participants are given one minute to say as many different words as they can think of that begin with a specified letter (F, then A, and finally S). It has also been found with the "Wisconsin card sorting" test (Berman et al., 1995; Nagahama et al., 1996). The Wisconsin card sorting test is a classic test of the functions of prefrontal cortex. Participants are given cards that can be sorted by color, shape, or name and must deduce the correct sorting criterion. After several consecutively correct sorts, the experimenter, without warning, changes the correct sorting criterion. Indeed, co-activation of dorsolateral prefrontal cortex and the cerebellum has been repeatedly found with several nonmotor, working memory tasks (Awh et al., 1996; Desmond et al., 1995; Desmond, Gabrieli, Wagner, Ginier, & Glover, 1997; deZubicaray et al., 1998; Grasby et al., 1994; Jonides et al., 1997; Paulesu et al., 1995; Paulesu, Frith, & Frackowiak, 1993).

Moreover, similar aspects of tasks activate the neocerebellum and dorsolateral prefrontal cortex. For example, increasing the memory load increases activation in both regions (e.g., Braver et al., 1997; Desmond et al., 1997; Diamond et al., 1999; Rypma, Prabhakaran, Desmond, Glover, & Gabrieli, 1999). The cerebellum and prefrontal cortex participate as critical parts of a neural circuit that is important when (1) a cognitive task is difficult as opposed to easy, (2) a cognitive task is new as opposed to familiar and practiced, (3) conditions of the cognitive task change, as opposed to when they remain stable and predictable, (4) a quick response is required, as opposed to longer response latencies being acceptable, and (5) one must concentrate instead of being able to operate on "automatic pilot."

EVIDENCE FROM BRAIN-DAMAGED PATIENTS OF THE CLOSE ASSOCIATION BETWEEN DORSOLATERAL PREFRONTAL CORTEX AND THE NEOCEREBELLUM

Lesions of prefrontal cortex can cause hypometabolism in the contralateral cerebellum (Fulham, Brooks, Hallett, & DiChiro, 1992; Muira et al., 1994; Tanaka et al., 1992). Similarly, cerebellar damage can cause frontal hypometabolism (Boni et al., 1992).

Cerebellar patients (especially if their damage is in the posterior lobe in the lateral hemisphere of the cerebellum and/or lobules VI and VII of the cerebellar vermis) often fail cognitive tasks linked to prefrontal cortex, such as "verbal fluency" (Appollonio, Grafman, Schwartz, Massaquoi, & Hallett, 1993; Schmahman & Sherman, 1998), verb generation (Fiez et al., 1996), planning tasks (Botez, Botez, Elie, & Attig, 1989; Grafman et al., 1992; Leiner, Leiner, & Dow, 1986; Schmahman & Sherman, 1998), tasks requiring the learning and memory of arbitrary associations (Bracke-Tolkmitt et al., 1989), set-shifting tasks (Schmahman & Sherman, 1998), working memory tasks (Fiez et al., 1996; Schmahman & Sherman, 1998), and source memory tasks (Ciranni, Dodson, & Shimamura, 1998). Indeed, there is even a case of a frontal-looking aphasia caused by a focal hypometabolism in the right cerebellum (Marien et al., 1996). The match between tasks affected by cerebellar damage and those affected by prefrontal damage is by no means exact, of course; for example, cerebellar patients are not impaired on the Wisconsin card sorting test (Bracke-Tolkmitt et al., 1989; Daum et al., 1993; Fiez, Petersen, Cheney, & Raichle, 1992).

Projections from dorsolateral prefrontal cortex reach the neocerebellum (Leiner, Leiner, & Dow, 1989; Middleton & Strick, 1997; Schmahmann & Pandya, 1995) and projections from the neocerebellum reach dorsolateral prefrontal cortex (Middleton & Strick, 1994; Sasaki, Jinnai, Gemba, Hashimoto, & Mizumo, 1979; Yamamoto, Yoshida, Yoshikawa, Kishimoto, & Oka, 1992). The cross-talk between the neocerebellum and dorsolateral prefrontal cortex is greatly increased in higher primates, especially humans.

HOW MIGHT THE CEREBELLUM AID IN COGNITIVE PERFORMANCE?

The cerebellum is quite remarkable anatomically. It has an exquisite lattice structure that is strikingly precise and regular, and the human cerebellum has more neurons than all the rest of the nervous system combined (Andersen, Korbo, & Pakkenberg, 1992). Two types of cells found in the cerebellar cortex are gran-

ule cells and Purkinje cells. Granule cells are tiny and numerous (up to 7 million per cubic centimeter in the layer below the Purkinje cells). Purkinje cells are large and have elaborate dendritic trees.

One source of input to the cerebellum is called the "climbing fibers." It consists of axons from the inferior olivary nucleus (inferior olive). Each Purkinje cell receives input from only one climbing fiber, but each climbing fiber may synapse on 10 Purkinje cells. Climbing fibers also terminate on granule cells. The granule cells in turn send out branching axons that give rise to the "parallel fibers." These rows of parallel fibers travel in a plane perpendicular to the parallel rows of Purkinje cells (hence the lattice structure). Each parallel fiber synapses on the dendrites of multiple Purkinje cells, and each Purkinje cell receives input from as many as 200,000 parallel fibers, the highest convergence ratio in the nervous system. A message conveyed by a single parallel fiber is relayed to successive Purkinje cells at precise, minutely different temporal intervals.

Cerebellar cortex can be thought of as being constructed of modules perpendicular to the cortical surface and parallel to each other (Ito, 1984; Leiner, Leiner, & Dow, 1991). The basic circuitry of each module is similar to that of every other cerebellar module (Bloedel, 1992), but each has its own unique set of inputs and outputs. Rather than evolve entirely new structures to subserve cognitive functions, it would make sense if nature built upon what was already there. Thus, it would make sense if evolution built upon the exquisitely precise and intricate cerebellar modules, recruiting existing ones, or adding additional ones, to help serve cognitive functions.

There is no question that the neocerebellum is involved in motor learning (Glickstein & Yeo, 1990; Houk, Buckingham, & Barto, 1996; Ito, 1984). Since all forms of learning share many requirements, it is not unreasonable that the neocerebellum might participate in other forms of learning as well. In motor learning, cerebellar neurons are most active during the early stages of learning a task (Flament, Ellermann, Ugurbil, & Ebner, 1994; Friston, Frith, Passingham, Liddle, & Frankowiak, 1992; van Mier et al., 1994) or when conditions change. For example, Purkinje cell firing is significantly greater when a participant moves a handle against a novel load than against a known load (Gilbert & Thach, 1977). Similarly, prism adaptation (i.e., reaching with the novel visual input provided by prisms) is impaired in patients with cerebellar damage (Weiner, Hallett, & Funkenstein, 1983).

Once a motor task is no longer novel, cerebellar firing decreases. Neuroimaging studies (cited in the first section above) are finding that on cognitive tasks, too, the cerebellum is most active when the task is novel or when conditions change, and cerebellar participation decreases as the task becomes familiar or practiced. Evidently, it is when you must pay close attention and concentrate, when you must learn something new for cognitive or motor performance, that the cerebellum is recruited most heavily.

There are several hypotheses for why this is so. Based on the architecture and circuitry of the cerebellum, Marr (1969) and Albus (1971) proposed that the cerebellum functions as a modifiable pattern detector, which could be used in the learning of motor skills. It is easy to see how such a system might be useful in the acquisition of cognitive skills as well. Fiez and colleagues (1992) suggest that the cerebellum may play a role in error detection or in the ability to learn from errors. The cerebellar patient on whom they report seemed oblivious to his errors and showed an abnormal lack of improvement with practice. In a similar vein, Ghez (1991) has talked about the cerebellum's involvement in comparing intention and performance. Thach (1998) speculates that the cerebellum may provide the mechanism for combining response elements into larger groupings, so that a coordinated response is elicited in a contextually-appropriate way. Leiner and colleagues (1991) and Schmahmann (1996) propose that the cerebellum may serve to enable other parts of the brain to perform their functions more efficiently or optimally.

Certainly there are many reports that performance is slower and more variable, inaccurate, and effortful in the absence of cerebellar input. This is true whether one is talking about cognitive functions, such as shifting attention (Courchesne et al., 1994), or motor functions (Holmes, 1939). Courchesne et al. (1994) propose that the neocerebellum aids cognitive performance by learning to precisely predict the occurrence of anticipated stimuli and by improving sensitivity so that those stimuli can be perceived even in the presence of noise. Thus, for example, in the superior colliculus neuronal firing to a visual stimulus is enhanced when the stimulus is preceded by stimulation of the neocerebellum and, in the presence of background luminance sufficient to reduce collicular responding to noise, stimulation of the cerebellum enables collicular responding to demonstrate consistent visual target detection (Crispino & Bullock, 1984).

Finally, Ivry and Keele (Ivry, 1993; Ivry & Keele, 1989; Keele & Ivry, 1990) have a well-developed theory that the lateral hemispheres of the cerebellum perform critical timing functions important for motor, sensory, and cognitive tasks. For example, certain modules of the cerebellum are critical for learning and/or retention of the classically conditioned eyeblink response (Clark, McCormick, Lavond, & Thompson, 1984; Woodruff-Pak, Logan, & Thompson, 1990).

Ivry and Keele hypothesize that the reason for this is that the lateral cerebellum is critical for measuring the time interval between the warning stimulus (a tone) and the noxious stimulus (an air puff), and in using that temporal calculation so that the eye blinks at exactly the right moment. Precise timing is critical here for if the eye blinks too late it fails to protect the eye, and if it occurs too early, the blink ends before the airpuff is presented and again fails to protect the eye. Acquisition of the conditioned eyeblink response depends crucially upon the cerebellar cortex and the interpositus nucleus (Chen, Bao, Lockard, & Kim, 1996; Chen, Bao, & Thompson, 1999; Kim & Thompson, 1997; Thompson, 1990; Thompson et al., 1997; Yeo & Hardiman, 1992). This has been demonstrated in humans by PET (Logan & Grafton, 1995) and by work with patients with cerebellar damage or atrophy (Daum et al., 1993; Solomon, Stowe, & Pendlebury, 1989; Topka, Valls-Sole, Massaquoi, & Hallett, 1993).

That infants show robust conditioning even in the first couple months of life (e.g., Fagen & Rovee-Collier, 1982; Rovee-Collier, 1990, 1997), might lead one to wonder if the role of the cerebellum in the classically conditioned eyeblink response is inconsistent with evidence that the neocerebellum does not reach full maturity until at least puberty. It is not inconsistent for several reasons. First, infants are not very good at acquiring the classically conditioned eyeblink response (Ivkovich, Collins, Eckerman, Krasnegor, & Stanton, 1999; Lipsitt, 1990). Second, even if the neocerebellum does subserve functions that aid conditioning during infancy, that would still be consistent with the neocerebellum not reaching full maturity for another 10 years or so. A brain system that is not fully developed can subserve certain functions early in development and more sophisticated functions later in development when it is more mature (e.g., Diamond, Prevor, Callender, & Druin, 1997). Third, there is no evidence that the forms of conditioning that are robust in early infancy (e.g., the conjugately reinforced footkick response to cause an appealing mobile to move) depend upon the cerebellum, much less the neocerebellum. It has been demonstrated that the cerebellum is critical for aversive conditioning of reflexive responses to noxious stimuli (the eyeblink response to an airpuff to the eye and the leg flexion response to footshock), but the cerebellum is not involved in all forms of conditioning. A case in point is autonomic conditioning (electrodermal responses to an impending airpuff to the eye or heartrate conditioning to impending shock), which does not require the cerebellum (e.g., Daum & Schugens, 1996; Daum et al., 1993; Lavond, Lincoln, McCormick, & Thompson, 1984).

EVIDENCE FROM DEVELOPMENTAL DISORDERS, A: MOTOR PROBLEMS FOUND IN CHILDREN WITH "COGNITIVE" DISORDERS

Motor coordination problems are common in children with Attention Deficit Hyperactivity Disorder (ADHD), although the cognitive deficits associated with this disorder have received far more attention. At least half of all ADHD children have poor motor coordination and fit the diagnosis for developmental coordination disorder (Barkley, DuPaul, & McMurray, 1990; Denckla & Rudel, 1978; Gillberg, 1995; Hartsough & Lambert, 1985; Hellgren, Gillberg, Gillberg, & Enerskog, 1993; Kadesjo & Gillberg, 1998; Piek, Pitcher, & Hay, 1999; Stewart, Pitts, Craig, & Dieruf, 1966; Szatmari, Offord, & Boyle, 1989). AHDH children tend to have motor problems associated with cerebellar dysfunction (e.g., problems with balance, with rapid alternating movements, and with consistently producing movements of the correct distance or correct timing) as well as motor problems with less specific bases (e.g., poor handwriting). Four studies report finding the cerebellum to be smaller in ADHD boys than in normal controls (Berquin et al., 1998; Castellanos 1997; Castellanos et al., 1996; Mostofsky et al., 1998). Rapoport (personal communication) reports that, in their MRI study of over 200 children, the largest difference in the brains of ADHD and non-ADHD children is the smaller cerebellum in ADHD children. For example, Kadesjo and Gillberg found that roughly 50% of the ADHD children in their Swedish sample also met the diagnosis for developmental coordination disorder, and 50% of the children with developmental coordination disorder met the diagnosis for ADHD.

Movement deficits are also evident in children with dyslexia or specific language disorder, although of course the deficits that have been focused upon in these children are in the linguistic domain. Children who are dyslexic, like children who are clumsy, have difficulties with continuous tapping tasks compared to same-aged peers (Geuze & Kalverboer, 1994; Wolff, Michel, Ovrut, & Drake, 1990). Wolff and colleagues note in particular a problem with timing precision on bimanual tasks that require the integration of asynchronous responses. Timing precision is a function that has been attributed to the cerebellum (e.g., Ivry, Keele, & Diener, 1988; Keele & Ivry, 1990). Hill and colleagues report that children with specific language disorder show a deficit in the production of familiar hand postures similar to that seen in children with developmental coordination disorder (Hill, Bishop, & Nimmo-Smith, 1998).

Many investigators have also reported movement disturbances in children who are autistic (for a review see Leary & Hill, 1996). For example, even among high functioning autistic individuals and those with a more benign variant of autism (Asperger's syndrome), Manjiviona and Prior (1995) found that 67% and 50% respectively showed a clinically significant level of motor impairment. Hughes (1996) found young people with autism to have problems in executing goal-directed motor acts even in very simple situations. Slavoff and Bonvillian (1997) report that all of the autistic children they tested scored below age norms on the Peabody Developmental Motor Scales. Page and Boucher (1998) studied 25 autistic children and found that 55% had manual impairments and 18% had additional gross motor impairments.

To be sure, motor impairments are not the most prominent characteristic of ADHD, dyslexia, specific language disorder, or autism. The point is simply that along with the prominent cognitive deficits apparent in these disorders many children appear to have concomitant motor problems, and the suggestion is that perhaps the cognitive and motor systems are not as totally separate as has traditionally been thought.

To be sure, as well, cognitive and motor strengths or weaknesses do not always co-vary. For example, not all ADHD children have motor impairments and not all children with a disorder of motor coordination have cognitive impairments. One of many possible reasons for the diversity in outcomes is the marked segregation and specificity of inputs and outputs within the cerebellum. Even within a single cerebellar nucleus, different subregions of dorsolateral prefrontal cortex (Areas 46 and 9) project to, and receive projections from, different subregions of the nucleus. For example, the most ventral portions of the dentate nucleus of the cerebellum project to Area 46 whereas projections to Area 9 arise rostrocaudally in the middle third of the dentate nucleus (Middleton & Strick, 1997). Other cerebellar modules do not project to dorsolateral prefrontal cortex at all. Because of the different neural systems in which different modules of the cerebellum participate, it is easy to see how dysfunctions that have slightly different regional extents within the cerebellum might produce very different outcomes.

EVIDENCE FROM DEVELOPMENTAL DISORDERS, B: ABNORMALITIES FOUND IN THE CEREBELLUM AND IN PREFRONTAL CORTEX IN THE SAME DISORDER

Ciesielski, Harris, Hart, and Pabst (1997) and Lesnik, Ciesielski, Hart, and Sanders (1998) report that the cerebellum (especially the posterior lobe) and prefrontal cortex are more susceptible than other brain regions to damage from intrathecal chemotherapy for leukemia during early childhood. These investigators attribute this vulnerability to the protracted period of postnatal maturation of the neocerebellum and prefrontal cortex.

Several studies report finding the cerebellum, especially posteriorally, to be smaller in boys with ADHD than in typically developing boys of the same age (Berquin et al., 1998; Castellanos et al., 1996; Mostofsky, Reiss, Lockhart, & Denckla, 1998). Similarly, several studies report finding significant reductions in the size of frontal cortex: Casey et al. (1997), Castellanos et al. (1996), and Filipek et al. (1997) all report volumetric analyses of structural magnetic resonance (MR) scans showing a significant size reduction in frontal cortex in ADHD children compared to healthy controls. Hynd, Semrud-Clikeman, Lorys, Novey, and Eliopulos (1990) found an absence in ADHD children of the right-greater-than-left frontal asymmetry found in normally developing children.

Functional neuroimaging studies show unusual prefrontal cortex activity in persons with ADHD. Reduced prefrontal activity has been reported in both children and adults with ADHD (in children: Amen, Paldi, & Thisted, 1993, using SPECT [blood flow]; in adults: Zametkin et al., 1990, using PET [cerebral glucose utilization]). Elevated prefrontal activity in ADHD children has also been reported (Vaidya et al., 1998, using functional magnetic resonance imaging (fMRI) [blood oxygen uptake]). Thus far, I have seen no studies of cerebellar activation in ADHD individuals.

In vivo evidence of a reduced cerebellum in autistic individuals has been reported by two independent groups of researchers (Courchesne, Hesselink, Jernigan, & Yeung-Courchesne, 1987; Courchesne, Yeung-Courchesne, Press, Hesselink, & Jernigan, 1988; Gaffney, Kuperman, Tsai, & Minchin, 1987; Gaffney, Kuperman, Tsai, Minchin, & Hassanein, 1988). Others, too, have reported cerebellar hypoplasia in autistic individuals (Bauman & Kemper, 1985; Murakami, Courchesne, Press, Yeung-Courchesne, & Hesselink, 1989). Indeed, in a review of numerous studies, Courchesne (1991) found consistent evidence of pathology in the neocerebellum, especially the posterior lobe and lobules VI and VII of the vermis, in autistic individuals. It should be noted, however, that not all studies have found cerebellar abnormalities or dysfunction in autistic children (Ekman et al., 1991; Minshew, Luna, & Sweeney, 1999).

Zilbovicius et al. (1995) report delayed maturation of frontal cortex in autistic children. They investigated the metabolic maturation of frontal cortex us-

ing regional cerebral blood flow. They found hypoperfusion in frontal cortex in autistic children 3 to 4 years of age, similar to the pattern of perfusion seen in much younger normal children. Chugani et al. (1997) report asymmetries of serotonin synthesis in frontal cortex, the thalamus, and the dentate nucleus of the cerebellum in autistic boys. They found decreased serotonin synthesis in frontal cortex and the thalamus and increased serotonin synthesis in the contralateral cerebellum.

This last finding reminds us that prefrontal cortex and the neocerebellum are interconnected parts of a neural system and that a dysfunction in one component of the system can affect the other components. Hua and Houk (1997) have proposed a model whereby the cerebellum aids in the development of premotor cortex. One could imagine a similar model of how dysfunction in the subregions of the cerebellum connected with prefrontal cortex might impair the proper development of prefrontal cortex. A final possible reason for why abnormalities in both the neocerebellum and prefrontal cortex might occur in the same disorders is that, because both prefrontal cortex and the neocerebellum have an extended period of maturation, insults too late in development to affect the maturation of other neural structures can have profound consequences for both prefrontal and cerebellar development.

THE IMPORTANCE OF THE CAUDATE NUCLEUS FOR COGNITIVE, AS WELL AS MOTOR, FUNCTIONS

Although this review has focused on the cerebellum as a neuroanatomical structure important for movement that appears (1) to function in a circuit with prefrontal cortex, (2) to play a role in cognitive functions, and (3) to be affected in children with cognitive neurodevelopmental disorders, a similar argument could be made with reference to the caudate nucleus. The caudate nucleus is a C-shaped structure that roughly parallels the lateral ventricle. It is considered part of the collection of structures known as the striatum, which in turn is part of the neural system known as the basal ganglia. The basal ganglia, and the caudate specifically, is important for movement control, such as selecting the proper movement, the appropriate muscles to perform a movement, or the appropriate force with which to execute the movement (e.g., DeLong & Georgopoulos, 1981; Groves, 1983; Stelmach & Worringham, 1988). For example, reduced dopamine in the caudate, and in the basal ganglia more generally, as occurs in Parkinson's disease, is often associated with akinesia (difficulty initiating movements and a poverty of spontaneous movement) and hypertonia (muscle rigidity), whereas loss of cell bodies in the caudate, characteristic of Huntington's chorea, is associated with hyperkinesia (excessive movement, often in jerky bursts and performed involuntarily) and hypotonia (decreased muscle tone) (e.g., Albin, Young, & Penney, 1989; Bowen, 1976; Halliday et al., 1998).

The caudate is the major output structure of dorsolateral prefrontal cortex (e.g., Selemon & Goldman-Rakic, 1985, 1988). Prefrontal cortex and the caudate nucleus are critical elements in a neural circuit (see, e.g., Alexander, DeLong, & Strick, 1986). Damage to the caudate, as in Parkinson's disease, often produces cognitive deficits similar to those found after prefrontal damage (Lees & Smith, 1983; Owen et al., 1992; Pantelis et al., 1997; Taylor, Saint-Cyr, & Lang, 1987; Taylor, Saint-Cyr, Lang, & Kenny, 1986; for reviews, see Lang, Paul, Robbins, & Marsden, 1993; Taylor, Saint-Cyr, & Lang, 1990). It should be noted, however, that some of the cognitive deficits associated with Parkinson's disease and with prefrontal pathology appear to be dissociable (Owen et al., 1993; Robbins et al., 1994). In addition to the neuroanatomical abnormalities found in frontal cortex and the cerebellum in ADHD children, studies report size reductions and reduced left–right asymmetry in the caudate nucleus in ADHD children (Castellanos et al., 1996; Filipek et al., 1997; Hynd et al., 1993). In addition, four functional neuroimaging studies report reduced caudate activity in ADHD children performing a cognitive task compared to age-matched controls (Lou, Henriksen, & Bruhn, 1990; Lou, Henriksen, Bruhn, Borner, & Nielsen, 1989; Teicher, Ito, Glod, & Barber, 1996; Vaidya et al., 1998).

Hence, the caudate nucleus, like the cerebellum, appears to participate in neural systems important for motor and for cognitive functions. The caudate's roles in both functions, and the critical role of dopamine in both functions and in the functions of both prefrontal cortex and the caudate nucleus (on *dopamine and prefrontal cortex*, see, e.g., Diamond, 1996; Goldman-Rakic, Lidow, Smiley, & Williams, 1992; Lewis, Foote, Goldstein, & Morrison, 1988; Williams & Goldman-Rakic, 1995; on *dopamine and the caudate*, see, e.g., Aosaki, Graybiel, & Kimura, 1994; Graybiel, 1990; Kostowski, 1972), provide additional reasons why cognitive development and motor development may be more intertwined than has been previously appreciated.

MOTOR FUNCTIONS OF FRONTAL CORTEX

Dorsolateral prefrontal cortex subserves cognitive functions such as enabling us to hold information in mind so that we can remember what it is we are sup-

posed to do, to work with the information held in mind to organize and reorganize it, to resist distraction and stay on task, to resist the temptation to respond too early, and to inhibit one action that might perhaps be our first inclination when another behavior is more appropriate. All of these cognitive functions are clearly important for skilled motor performance. Thus, it makes sense that not only may the cerebellum and striatum play a role in cognition, but dorsolateral prefrontal cortex may contribute to motor performance. Dorsolateral prefrontal cortex has extensive interconnections with regions of frontal cortex more directly involved in motor functions such as premotor cortex and the supplementary motor area (SMA) (on *premotor cortex*, see Barbas & Pandya, 1987; Dum & Strick, 1991; Kunzle, 1978; on *SMA*, see Tanji, 1994; Wiesendanger, 1981). Premotor cortex is important for functions such as the planning of, preparation for, and sensory guidance of movement (e.g., Goldberg, 1987; Humphrey, 1979; Passingham, 1985, 1988; Wise, 1985). SMA is important for functions such as bimanual coordination and the generation and execution of motor sequences (e.g., Brinkman, 1984; Dick, Benecke, Rothwell, Day, & Marsden, 1986; Gaymard, Pierrot-Deseilligny, & Rivaud, 1990; Goldberg, 1985; Orgogozo & Larsen, 1979; Roland, Larsen, Larsen, & Skinhoj, 1980; Romo & Schultz, 1992). Premotor cortex and the SMA in turn have strong interconnections with motor cortex, which is also a region within frontal cortex. Hence, dorsolateral prefrontal cortex is positioned to be in close communication not only with subcortical regions important for motor function but with cortical centers important for movement as well.

CONCLUSIONS

It has been suggested here that motor development and cognitive development may be more interrelated than has been previously appreciated. When there are perturbations, genetic or environmental, that affect the motor system (as in developmental coordination disorder) or cognition (as in ADHD) it is often the case that both motor and cognitive functions are affected, not just one or the other. The caudate nucleus and the neurotransmitter, dopamine, play roles in neural systems subserving cognitive and motor functions. Developmental psychologists tend to focus on the protracted developmental progression in cognitive achievements, often forgetting that motor development is equally protracted. Similarly, the protracted period of prefrontal maturation is often emphasized, whereas the protracted period of cerebellar maturation has received less attention. Study after study using functional neuroimaging has found close co-activation of the neocerebellum and dorsolateral prefrontal cortex during performance of cognitive tasks. When prefrontal activation is increased on a task, cerebellar activation is increased in the contralateral hemisphere, and when prefrontal activation is decreased, cerebellar activation is decreased in the contralateral hemisphere, although motor demands remain constant. It appears that the cerebellum may not only subserve motor function, but may play a role in cognition as well. Conversely, prefrontal cortex, through its connections with cortical and subcortical centers important for movement control, may play a role in motor function, not simply in cognition.

ACKOWLEDGMENT

The author would like to acknowledge the support provided by National Institutes of Health grant R01 #HD35453.

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