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The Lateralized Linguistic Cerebellum: A Review and a New Hypothesis

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During the past 2 decades the collaboration across disciplines and the methodologic and conceptual advances of contemporary neuroscience have brought about a substantial modification of the traditional view of the cerebellum as a mere coordinator of autonomic and somatic motor functions. Growing insights in the neuroanatomy of the cerebellum and its interconnections, evidence from functional neuroimaging and neurophysiological research, and advancements in clinical and experimental neuropsychology have established the view that the cerebellum participates in a much wider range of functions than conventionally accepted. This increase of insight has brought to the fore that the cerebellum modulates cognitive functioning of at least those parts of the brain to which it is reciprocally connected. This article reviews the recently acknowledged role of the cerebellum in cognition and addresses in more detail experimental and clinical data disclosing the modulatory role of the cerebellum in various non-motor language processes such as lexical retrieval, syntax, and language dynamics. In agreement with the findings indicating a topographical organization of the cerebellar structures involved in language pathology we advance the concept of a ''lateralized linguistic cerebellum.'' In our view crossed cerebral diaschisis processes, reflecting a functional depression of supratentorial language areas due to reduced input via cerebellocortical pathways, might represent the relevant pathomechanism for linguistic deficits associated with cerebellar pathology.

EARLY EVIDENCE AND DEVELOPMENT OF CONCEPTS

In the 19th century the long-lasting view was established that the cerebellum subserves motor functions. Bloedel and Bracha (1997) distinguished during the 20th century five periods in the conceptual growth and development of insights in cerebellar functioning: (1) coordination of goal-oriented voluntary movement and orientation of the body and the head in space; (2) regulation and integration of sensory information for cutanomuscular and proprioceptive reflex organization; (3) regulation of vestibulo-ocular movements and posture of the head; (4) learning of classically conditioned withdrawal responses; and (5) modification of linguistic, cognitive, and affective behavior. These conceptual expansions have been overshadowed by the prominent role of the cerebellum in motor functioning. In the early reports, for instance, a possible causal relationship between cerebellar pathology and accompanying cogni-

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tive deficits remained unexplored (e.g., Combettes, 1831; Vulpian, 1866; Whyte, 1898; Vogt & Astwazaturow, 1912; Curschmann,1922; Akelaitis, 1938; Knoepfel & Macken, 1947; Schut, 1950; Joubert, Eisenring, Robb, & Andermann, 1969; Landis, Rosenberg, Landis, Schut, & Nyhan, 1974). In the late 1970s and early 1980s new neuroimaging techniques created increased interest in the nonmotor functions of the cerebellum and case reports began to appear alluding to the possible pathogenic role of the cerebellum in various cognitive dysfunctions (Bolthauser & Isler, 1977; Hamilton, Frick, Takahashi, & Hopping, 1983; Fehrenbach, Wallesch, & Claus, 1984). Soon thereafter, functional neuroimaging studies brought to the fore that even in the complete absence of any motor activity the cerebellum is activated during the performance of various cognitive and linguistic tasks (Petersen, Fox, Posner, Mintun, & Raichle, 1988, 1989; Ryding et al., 1993). Following the notion that the cerebellum may be crucially involved in nonmotor functions, experimental and clinical studies started to explore the new concept of ''cerebellar cognition.''

CEREBELLAR COGNITION

In the late 1980s, studies grounded the view that the cerebellum contributes to executive functions, such as mental planning, sequential reasoning, and mental operations closely associated with the functional role of the (pre)frontal cortex (Leiner, Leiner, & Dow, 1986, 1989; Grafman, Litvan, Massaquoi, Stewart, Sirigu, & Hallett, 1992; Hallett & Grafman, 1997). Keele and Ivry (1989, 1991) demonstrated that the cerebellum seems to act as an ''internal clock'' during any process requiring temporal computations.

Probably due to dysfunction of cerebello-thalamo-cortical pathways, cerebellar damage can disrupt selective attentional processes (orienting, distributing, and shifting attention). Attentional processes largely depend on coordinated interactions between the reticular activating system, and the frontal and parietal lobes (e.g., Mesulam, 1981; Posner & Petersen, 1990; Corbetta, Miezin, Dobmeyer, Shulman, & Petersen, 1993). Within this integrated system the cerebellum has been considered to enhance neural responsiveness in advance to stimulation (e.g., Yeo, Hardiman, & Glickstein, 1985; Thompson, 1986). The view that the cerebellum coordinates the direction of selective attention and as a consequence subserves the execution of cortically generated commands for the enhancement and inhibition of different sources of sensory information has been confirmed in large series of behavioral, neurophysiological, and neuroimaging studies (Courchesne, 1985; Thompson, 1986; Akshoomoff & Courchesne, 1994; Le & Hu, 1996; Akshoomoff, Courchesne, & Townsend, 1997).

Botez, Gravel, Attig, and Vézina (1985) first underscored the role of cerebellofrontal and cerebelloparietal associative loops as neural substrates of mild frontal- and parietal-like symptoms encountered in a patient with reversible cerebellar ataxia after chronic phenytoin intoxication. Many of their subsequent studies further explored the role of the cerebellum in visuospatial and visuoconstructive procedures (Botez, Léveillé, Lambert, & Botez, 1991; Botez-Marquard & Botez, 1993; Botez-Marquard, Léveillé, & Botez, 1994; Botez-Marquard & Routhier, 1995; Botez-Marquard, Pedraza, & Botez, 1996). Closely related to the view that the cerebellum participates in visuospatial functions, Silveri, Misciagna, Leggio, and Molinari (1997, 1999) reported a patient with cerebellar atrophy who showed typical features of spatial or afferent dysgraphia. They explained this writing disorder as an uncoupling of motor planning and proprioceptive feedback due to cerebellar damage and postulated that the functional substrate of afferent dysgraphia includes a defective interplay between the left cerebellum and the contralateral supratentorial structures.

TABLE 1
Cerebellar Involvement in Neurocognitive Functions

Cognitive domain	Function	Reference
Executive planning	Frontal problem solving	e.g., Grafman et al., 1992
	Cognitive planning	e.g., Grafman et al., 1992
	Sequencing of plans	e.g., Hallett & Grafman, 1997
Temporal sequencing	Judgment of time duration	e.g., Ivry & Keele, 1989
	Timing of plans and actions	e.g., Hallett & Grafman, 1997
	Judgment of velocity of movement	e.g., Ivry & Diener, 1991
	Discrimination of vowel duration	e.g., Ackermann et al., 1996
	Discrimination of VOT	e.g., Ackermann et al., 1996
Attention	Enhancement of neural responsiveness	e.g., Yeo et al., 1985
	Direction of selective attention	e.g., Akshoomoff et al., 1997
Visuoperception	Visuospatial processing	e.g., Silveri et al., 1997
	Visuoconstruction	e.g., Botez-Marquard et al., 1994
Learning	Motor skill learning	e.g., Marr, 1969; Tach, 1997; Poldrack & Gabrieli, 2001
	Procedural & associative learning	e.g., Bracke-Tolkmitt et al., 1989
Memory	Long-term memory	e.g., Appollonio et al., 1993
	Phonological short-term memory	e.g., Paulesu et al., 1993
Imagery	Visuomotor imagery	e.g., Decety et al., 1990

Note. VOT = voice onset time.

Experimental and clinical studies have demonstrated that the cerebellum is involved in many different components of memory such as procedural learning (eyeblink classical conditioning, motor adaptation learning, and motor skill learning), paired-associative learning, working memory, phonological short-term memory, and long-term memory (Marr, 1969; Brindley, 1969; McCormick, Lavond, Clark, Kettner, Rising, & Thompson, 1981; Bracke-Tolkmitt, Linden, Canavan, Rockstroh, Scholz, Wessel, & Diener, 1989; Appollonio, Grafman, Schwartz, Massaquoi, & Hallett, 1993; Pascual-Leone, Grafman, Clark, Stewart, Massaquoi, Lou, & Hallett, 1993; Paulesu, Frith, & Frackowiak, 1993; Jenkins, Brooks, Nixon, Frackowiak, & Passingham, 1994; Grafton, Hazeltine, & Ivry, 1995; Vallar & Papagno, 1995; Molinari, Leggio, Solida, Ciorra, Misciagna, Silveri, & Petrosini, 1997; Tach, 1997; Thompson, Bao, Cipriano, Grethe, Kim, Thompson, Tracy, Weninger, & Krupa, 1997; Vallar, Di Betta, & Silveri, 1997; Poldrack & Gabrieli, 2001) (Table 1).

Most of the available data seem to corroborate the hypothesis that the cerebellum subserves cognitive operations at a modulatory level in between the cognitive processes per se and their relative executive phases (Silveri & Misciagna, 2000).

CEREBELLAR SPEECH AND LANGUAGE DISTURBANCES

Articulation and Phonation

Ataxic dysarthria. In 1917 Holmes extensively described disturbed muscular control of speech after cerebellar lesions and contributed to the view that the cerebellum plays a crucial role in motor speech production. He defined the typical cerebellar speech characteristics as slow, monotonous, staccato, scanned, indistinct, remarkable irregular, jerky, explosive, slurred, and labored. Darley, Aronson, and Brown (1975) included these alterations in phonation and articulation in the category of ‘‘ataxic

dysarthria'' and identified imprecise production of consonants and vowels, irregular articulatory breakdown, excess and equal stress, and harsh voice quality as the cardinal symptoms.

Holmes (1917, 1922), and many investigators after him, maintained that the responsible lesion for ataxic dysarthria could be situated in either one or both cerebellar hemispheres. However, more recent studies addressing topographic aspects of cerebellar induced motor speech abnormalities have shown that dysarthria most frequently follows damage to the superior anterior vermal and paravermal regions (Lechtenberg & Gilman, 1978; Amarenco, Chevrie-Muller, Roullet, & Bousser, 1991; Amarenco, Roullet, Goujon, Cheron, Hauw, & Bousser, 1991; Ackermann, Vogel, Petersen, & Poremba, 1992; Barth, Bogousslavsky, & Regli, 1993). In addition, Lechtenberg and Gilman (1978) and Amarenco et al. (1991) showed that dysarthria resulted more frequently from left than right cerebellar lesions. This laterality effect is supported by the majority of functional neuroimaging studies with PET and fMRI on articulated speech (see Fiez & Raichle, 1997, for a review).

Apraxia of speech. Apraxia of speech (anarthria, verbal apraxia, and speech apraxia) consists of a selective impairment of speech movements following the inability to properly and smoothly convert phonological knowledge into verbal–motor commands (Lebrun, 1990; Rosenbek, 1999). The condition is typically characterized by inconsistent misarticulations, phonetic alterations of vowel and consonant production, articulatory groping inducing sequential errors, flattened voice volume, prosodic abnormalities, slow articulation, scanning speech, and ''islands of fluent oral-verbal output.'' Dronkers (1996) recently localized the crucial anatomical seat of this articulatory planning and coordination disorder in the ''precentral gyrus of the language dominant insula, directly anterior to the central insular sulcus.''

Apraxia of speech shares striking semiological similarities with ataxic dysarthria. These similarities, already implicated by prior terms as ''ataxic aphasia'' and ''cortical dysarthria'' to denote speech apraxia, seem to indicate a close funcitonal cooperation between the left anterior insular and opercular speech area of the language dominant hemisphere and the right hemisphere of the cerebellum. A study of Mariën, Pickut, Engelborghs, Martin, and De Deyn (2001) supports this hypothesis. In this report an 83-year-old right-handed patient is described with a unique infarction restricted to the left anterior insula (the sulcus circularis insulae and the gyri breves insulae) and the adjacent part of the intrasylvian frontal opercular cortex (the gyrus frontalis inferior, especially the pars opercularis) (Fig. 1). At onset the patient developed severe apraxia of speech that evolved into mere mutism within a few hours. After rapid recovery from mutism, apraxic speech symptoms persisted. In-depth language investigations during the lesion phase additionally disclosed an isolated phonological agraphia which receded within a few weeks. A Tc-99m-ECD SPECT study of the brain demonstrated hypoperfusions involving the gyrus frontalis inferior and gyrus precentralis of the left cerebral hemisphere and the contralateral right hemisphere of the cerebellum (Fig. 2). This pattern of perfusional defects, representing crossed cerebellar diaschisis, reflects the distant metabolic impact of the cerebral lesion on the contralateral cerebellar hemisphere and adds evidence to the view that these sites are anatomically and functionally closely interconnected (Baron, Bousser, Comar, & Castaigne, 1980; Pantano, Baron, Samson, Bousser, Derouesne, & Comar, 1986; Abe, Ukita, Yorifuji, & Yanagihara, 1997; Engelborghs, Pickut, Mariën, Opsomer, & De Deyn, 2000).

Additional evidence for the presumed underestimated role of the right cerebellum in apraxic speech manifestations was recently obtained in two personal observations of pure and persistent apraxia of speech. In both these patients with focal frontoopercular lesions of the language dominant hemisphere, a SPECT scan of the brain

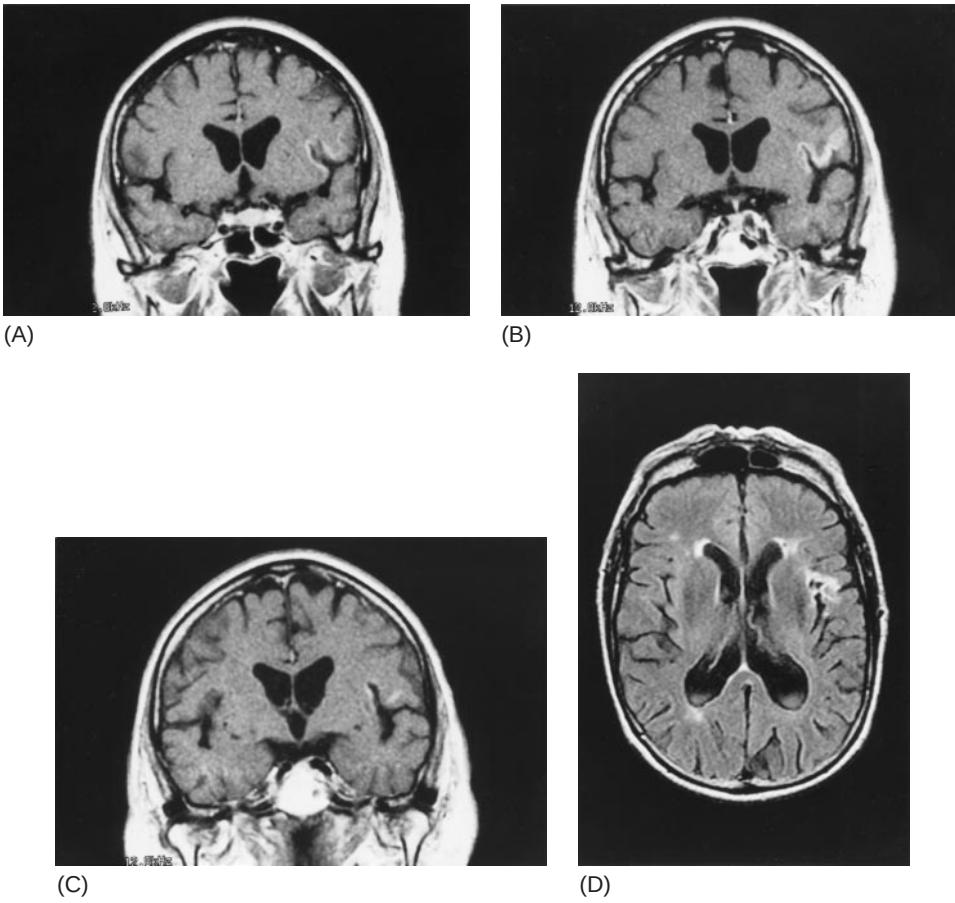


FIG. 1. Coronal T1-weighted brain MRI slices after gadolinium **(A–C)** and axial FLAIR slice **(D)** disclose in the left cerebral hemisphere a focal anterior insulo-opercular lesion occupying the sulcus circularis insulae, the gyri breves insulae, the gyrus frontalis inferior, and the gyrus precentralis. Several small bilateral white matter lesions of presumed vascular origin are shown as well.

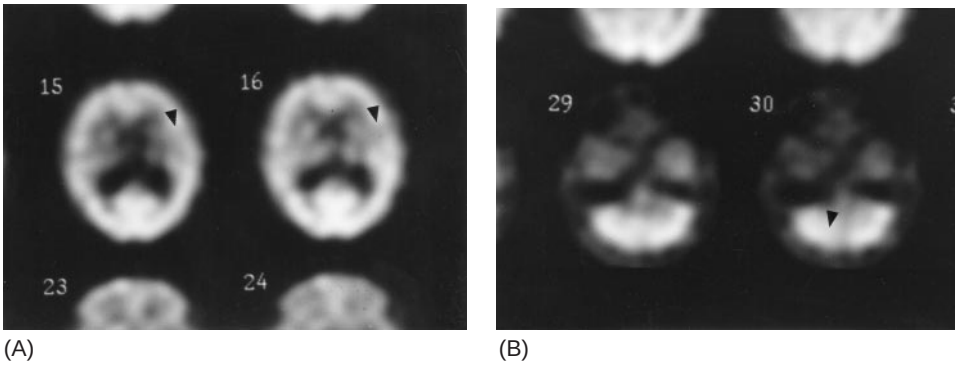


FIG. 2. Tc-99m-ECD SPECT scan of the brain performed 9 days after stroke demonstrating **(A)** a relative hypoperfusion in the left gyrus frontalis inferior and left gyrus precentralis and **(B)** crossed cerebellar diaschisis.

also disclosed crossed cerebellar diaschisis. These findings seem to indicate that diaschisis-related phenomena affecting the right hemisphere of the cerebellum might constitute an important factor in apraxia of speech. To unravel the presumed role of the right hemisphere of the cerebellum in apraxic speech manifestations future studies are needed that concentrate on a close semiological comparison between apraxia of speech and ataxic dysarthria following right cerebellar lesions.

Language Processing

Introduction. Leiner et al. (1986, 1989) discussed the functional expansion of the cerebellum as a consequence of structural changes that evolved during hominid evolution. Having traced newly evolved connections in the human brain, they postulated that the enlarged size of the dentate nucleus (the new ventrolateral and older dorsomedial part) gave rise to new neural connections. These new connections, which evolved concomitantly with the human dentate nucleus, end in some expanded prefrontal areas which send back new connections. The composition of these loops consists of the phylogenetically new parts of the lateral cerebellum sending projections to the contralateral Brodmann's areas 6, 44, and 45 via the nucleus ventralis intermedius and nucleus ventralis anterior of the thalamus (Engelborghs, Mariën, Martin, & De Deyn, 1998) and backward projections from the prefrontal areas to the lateral cerebellum via the pons and the parvocellular part of the red nucleus (Leiner et al., 1986, 1989). The discovery of major reciprocal neural pathways between the cerebellum and the frontal areas of the language dominant hemisphere [Broca's area and the supplementary motor area (SMA)] constitutes a hallmark in the development of the concept of cerebellar contribution in nonmotor lingustic processes. In the past 10 years, a close cooperation across disciplines has established the view that the right cerebellum plays a crucial role within the language network.

Verbal fluency and word retrieval. Petersen et al. (1988, 1989) reported the results of innovative PET activation procedures that provided preliminary evidence in support of the hypothesis of cerebellar involvement in nonmotor language (Leiner et al., 1986). Their paradigm required subjects (1) to repeat a visually presented noun (motor task) and (2) to generate a semantically associated verb for a visually presented noun and to say this verb aloud (cognitive and motor task). Subtracting motor activation of the first task from motor activation of the second task (cognitive and motor) allowed identification of activated areas during cognitive word association. These tasks, which reflect the capacity to generate words according to a given rule, are generally considered to depend on a close cooperation between verbal and executive abilities and are clinically widely applied to explore frontal lobe functioning. During mere verbal–motor performance, an activated area in the superior anterior lobe of the cerebellum was found just lateral to the loci involved in finger and eye movement. The verbal association task strikingly activated a totally different area: the inferior lateral part of the right cerebellum, which projects to the left prefrontal language areas. Despite variations on the original task design, several studies have consistently reproduced activation of the right lateral cerebellum during word generation tasks (Raichle, Fiez, Videen, MacLeod, Pardo, Fox, & Petersen, 1994; Martin, Haxby, Lalonde, Wiggs, & Ungerleider, 1995; Grabowski, Frank, Brown, Damasio, Boles-Ponto, Watkins, & Hichwa, 1996). Leiner et al. (1989) interpreted the simultaneous activation of the right cerebellum and Broca's language area during word generation as the reflection of accelerated transmission of signals between these two centers during word finding.

Clinical studies on patients with cerebellar disease have confirmed the role of the right cerebellum in word production. Fiez, Peterson, Cheney, and Raichle (1992)

conducted the first specially designed study on word generation in a patient who presented with sematic retrieval deficits after a vascular lesion of the right cerebellar hemisphere. Despite high-level conversational skills and normal performance on standard language tests, the patient failed in various semantic word generation tasks. Leggio, Solida, Silveri, Gainotti, and Molinari (1995) conducted both phonological and semantic verbal fluency studies in three etiologically distinct patient groups with cerebellar pathology. The phonological tasks required the subjects to produce during 1 min as many words as possible with initial phonemes F, A, and S. The semantic verbal fluency tasks consisted of the generation of as many words as possible belonging to the semantic categories ''birds'' and ''furniture.'' One group had atrophic lesions (mainly of the vermal and paravermal regions) and both other groups had restricted focal lesions (lateral part of the left or right cerebellar hemisphere). Their results showed that (1) the cerebellar lesioned group performed at a lower level than the matched controls irrespective of the task involved (phonological or semantic); (2) atrophic patients obtained better results than patients with focal lesions, although they had more severe ataxic impairments; (3) in comparison with the control group, the atrophic patients performed only significantly worse on the phonological task; and (4) patients with focal damage of the left cerebellum performed slightly better than patients with right cerebellar damage. These findings, which extend the functional role of the cerebellum in linguistic processes, reveal a close association between (1) medial cerebellar lesions and the prevalence of motor deficits and (2) lateral, especially right cerebellar, damage and verbal fluency deficits. In a subsequent study, Leggio, Silveri, Petronsini, and Molinari (2000) basically confirmed these findings and explicitly demonstrated that verbal fluency deficits in their study population could not be attributed to motor speech impairment. Treating the dysarthria score as a covariate with the total verbal output for the phonological and semantic verbal fluency tasks, all statistical differences were confirmed. In addition, the authors interpreted the difference in cerebellar effects between phonological and semantic verbal fluency along the view of the role of the cerebellum in planning, strategy formation, and learning of procedures. They conceived that cerebellar damage affects phonological processes to a greater extent than semantic processes because phonological tasks depend on unusual novel and less automized searching strategies than semantic tasks.

Disorders of grammatical production. The role of the right cerebellum in nonmotor language functions has recently been expanded by evidence derived from patients with agrammatism. Silveri, Leggio, and Molinari (1994) and Zettin, Cappa, D'Amico, Rago, Perino, Perani, and Fazio (1997) reported two patients in whom a right cerebellar lesion caused expressive agrammatism. In two other agrammatic patients with vascular right cerebellar damage, more extensive linguistic defects were described (Mariën, Saerens, Nanhoe, Moens, Nagels, Pickut, Dierckx, & De Deyn, 1996; Mariën, Engelborghs, Pickut, & De Deyn, 2000; Gasparini, Di Piero, Ciccarelli, Ciccarelli, Cacioppo, Pantano, & Lenzi, 1999). Similar observations were made in a child with cerebellitis (Riva, 1998), a group of patients with infiltrative cerebellar damage (Fabbro, Moretti, & Bava, 2000), and in two children after posterior fossa surgery (Riva & Giorgi 2000).

Agrammatism has long been considered a pure syntactic disorder affecting both language production and comprehension. In-depth psycholinguistic and neurolinguistic investigations have broadened the concept. The observation of various combinations of differential syntax impairments (Tossot, Mounin, & Lhermitte, 1973; Caramazza & Zurif, 1976; Miceli, Mazzucchi, Menn, & Goodglass, 1983; Kolk, vanGrunsven, & Keyser, 1985; Jerema, Kadzielawa, & Waite, 1987; Caramazza & Hillis, 1989), sometimes even only affecting one specific modality (e.g., Bub &

Kertesz, 1982; Howard, 1985), led to the hypothesis that a distinct neuroanatomic organization subserves different types of syntactic disorders: a primarily syntactic component being localized in the dominant frontal lobe and a primarily morphological component being located in the dominant postcentral perisylvian cortex (Nadeau, 1988; Nadeau & Gonzalez Rothi, 1992).

Silveri et al. (1994) for the first time reported a consistent correlation between focal damage of the right cerebellum and agrammatic symptoms. They described a 67-year-old right-handed patient who, after a right cerebellar stroke, presented with a right-sided cerebellar syndrome, ataxic dysarthria, and transient expressive agrammatism. Linguistic analysis of the agrammatic manifestations showed the deficit to be of the morphological type (relevant omissions of auxiliaries and clitic pronouns and many substitutions of bound grammatical morphemes). Repeated structural neuroimaging studies did not reveal any supratentorial abnormality to account for the observed language deficits. SPECT, however, evidenced a relative hypoperfusion in the entire left cerebral hemisphere and more stable and consistent in the left posterior, temporal region. During follow-up, the perfusion defects paralleled the clinical course of motor and linguistic symptoms. Four months after onset of neurological symptoms, amelioration of the motor deficits and agrammatic symptoms was reflected by a marked improvement of left cerebral hemispheral perfusion. Silveri et al. (1994) interpreted this selective speech production impairment as a ''peripheral disorder'' reflecting a linguistic behavioral adaptation to a deficit outside the mental linguistic system. The deficit was not considered to affect syntactic competence but the online application of syntactic rules that put the grammatical morphemes in accordance. They claimed that if the temporal computation of morphosyntactic operations underlying sentence construction is disrupted by cerebellar damage, the application of syntactic rules is decoupled from phonological working memory operations. Zettin et al. (1997) also accounted for the sentence production impairment of their patient with a hemorrhagic stroke of the right cerebellum as a deficit lying outside the linguistic system. They viewed the disturbance as a compensatory mechanism to circumvent a disorder that goes beyond the strictly articulatory level. In their view, the temporal decoupling between the computation of syntactic rules and the application of grammatical morphemes temporarily stored in working memory is caused by a deregulation of articulatory planning. They considered a defect in working memory, itself unlikely given the normal performance of their patient in span tasks.

In the patients reported by Mariën et al. (1996, 2000) and Gasparini et al. (1999), a vascular lesion of the right cerebellar hemisphere induced a structural impairment at the syntactic selection level, producing agrammatic manifestations in different language modalities of both receptive and expressive language. Given the evidence of structurally impaired syntactic knowledge, the agrammatic symptoms in these patients could not be explained as the compensatory result of a timing disorder lying outside the lingustic system. Following the observation that additional aphasic deficits may accompany expressive and receptive agrammatism we maintained that right cerebellar lesions may provoke aphasia (Mariën et al., 1996). The observations of Riva (1998), Fabbro et al. (2000), and Riva and Giorgi (2000), in an etiologically different population, corroborate this view.

Cerebellar induced aphasia. Hassid (1995) described a 17-year-old left-handed man with a right cerebellar hemisphere infarction. In addition to classic cerebellar motor symptoms, including dysarthria, formal testing revealed moderate anomia on the Boston Naming Test (Kaplan, Goodglass, & Weintraub, 1983), mild difficulties in auditory reception and reading, and severe difficulties in writing and mathematics. Computerized tomograpy (CT) and magnetic resonance imaging (MRI) of the brain only disclosed a right-sided wedge-shaped cerebellar infarction. SPECT scan images

of the brain showed a relative hypoperfusion in the right cerebellar hemisphere and in the frontal, temporal, and parietal regions of the left cerebral hemisphere, consistent with an infarction in the right hemisphere of the cerebellum associated with crossed cerebral diaschisis. In support of the notion of the role of the cerebellum in cognition, Hassid (1995) concluded from these observations that cognitive abnormalities after cerebellar infarction can be easily overlooked and that standardized cognitive assessments in patients with focal cerebellar lesions may be more reliable in accurately delineating subtle, but significant, cognitive abnormalities. The finding of aphasic symptoms accompanying right cerebellar damage in this left-hander was not discussed.

Mariën et al. (1996, 2000) reported a 73-year-old right-handed patient who developed dynamic aphasia (Luria & Tsvetkova, 1967; Luria, 1977), receptive and expressive agrammatism, and dysarthria after a vascular lesion in the right cerebellar hemisphere. Formal language investigations by means of an extensive test battery consisting of the Frenchay Dysarthria Assessment (Enderby, 1983), the Aachener Aphasie Test (Graetz, De Bleser, & Willmes, 1992), the Boston Diagnostic Aphasia Examination (BDAE) (Goodglass & Kaplan, 1983), the Token Test (De Renzi & Vignolo, 1962), and the Boston Naming Test (Kaplan, Goodglass, & Weintraub, 1983; Mariën, Mampaey, Vervaet, Saerens, & De Deyn, 1998) and in-depth analysis of spontaneous speech samples revealed, in addition to mild dysarthria, that the core element of the aphasic syndrome consisted of a striking dissociation between profoundly affected propositional speech and rather well-preserved, externally guided language in nominative, repetition, and comprehension tasks. Despite normal confrontational naming and phonological skills, self-generated speech was severely reduced, adynamic, fragmented, and characterized by severe word-finding difficulties. In addition, linguistic analysis revealed a structural impairment at the syntactic selection level, indicative of a frontal disturbance. Although the neuroanatomical correlates of the aphasia type, which closely resembled transcortical motor aphasia, cluster in the frontal lobe of the language dominant cerebral hemisphere, repeated structural imaging studies with CT and MRI did not disclose a lesion in the expected supratentorial areas. Repeated 99mTc-HMPAO SPECT studies, however, yielded positive findings to account for the language symptoms. In addition to a marked hypoperfusion in the right hemicerebellum, SPECT revealed a left frontoparietal hypoperfusion which involved the gyrus frontalis medius and inferior as well as the gyrus precentralis and postcentralis. Along the lines of linguistic improvement, a less pronounced hypoperfusion was found in the frontal areas 6 months after onset. In association with near remission of the aphasic symptoms, near normalization of the perfusion pattern in the left frontoparietal area was found 5 years postonset. Since it was first recognized by Broich, Hartmann, Biersack, and Horn (1987), this phenomenon of so-called ''crossed cerebello-cerebral diaschisis'' has been amply documented (e.g., Kimura, Nakamura, Matsumura, Morohashi, Ueoka, Hasegawa, & Yonekura, 1989; Yokoji, Ide, Matsubaru, & Takamori, 1989; Botez et al., 1991; Rousseaux & Steinling, 1992; Deguchi, Takeuchi, Yamada, Touge, & Nishioka, 1994; Sönmezoglu, Sperling, Henriksen, Tfelt-Hansen, & Lassen, 1993). In contrast, however, to an already well-documented range of cognitive correlates of ''cerebral diaschisis'' (e.g., Meyer, Hata, & Imai, 1987; Meyer, 1991), the literature makes only scant mention of cognitive dysfunctions associated with ''crossed cerebello-cerebral diaschisis'' (Attig, Botez, Hublet, Verdonck, Jacqui, & Capon, 1991; Boni, Valle, Gioffi, Bonetti, Perrone, Tofani, & Mani, 1992; Botez-Marquard et al., 1994; Silveri et al., 1994). In the light of their findings, Mariën et al. (1996, 2000) proposed that a possible explanation for aphasia following right cerebellar damage might be found in a loss

of excitatory impulses through cerebello-ponto-thalamo-cortical pathways (Sönmezoglu et al., 1993). Consequently, aphasia in cerebellar pathology does not imply representation of language functions at the level of the cerebellum but reflects as a diaschisis phenomenon (Von Monakow, 1914) diminished or abolished function of the remote supratentorial ''language zones'' due to reduced input via cerebellocortical pathways.

Gasparini and co-workers (1999) contended the view that neurolinguistic impairments after right cerebellar damage constitute diaschisis-related phenomena. They described a 51-year-old right-handed patient in whom formal language testing disclosed expressive and receptive agrammatism, stereotypies, and word-finding difficulties consequent to a vascular lesion in the superior lateral area of the right hemisphere of the cerebellum. Functional neuroimaging did not reveal any supratentorial abnormalities of perfusion distribution. Following this observation they suggested, along the lines of the view that the cerebellum primarily acts as a timing mechanism in the modulation of cognitive functions, that right cerebellar lesions induce a slower timing in sentence representation.

Fabbro et al. (2000) thoroughly investigated language functions of four right-handed patients with tumoral cerebellar lesions before and after surgery. Irrespective of lesion type and lesion localization (vermis and left and right cerebellum), all four patients displayed linguistic dysfunctions, mainly affecting morphosyntactic knowledge and lexical retrieval. After surgery, only two patients partially recovered. Fabbro et al. (2000) related the linguistic deficits to an alteration of language control processes rather than to a structural impairment of specific components of the language system. In their view, the vermis and portions of the cerebellar hemispheres operate within a large functional language network as an organizational control mechanism via the frontal lobe system. Rapid recovery of linguistic disturbances following acute cerebellar damage was attributed to partial functional reactivation of linguistic centers after regression of diaschisis phenomena.

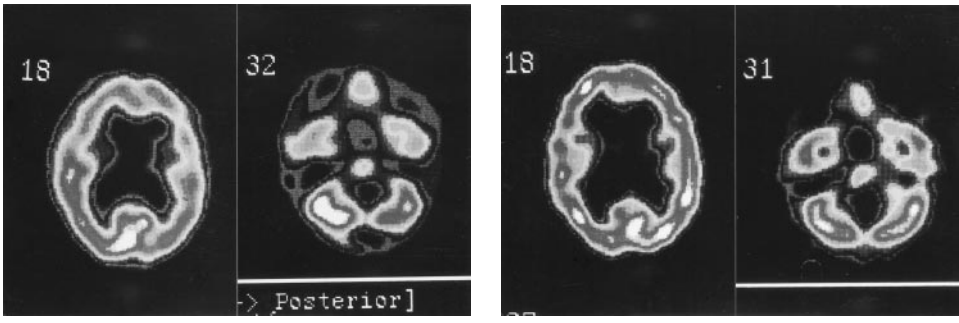
Transient cerebellar mutism syndrome. So-called ''posterior fossa syndrome'' or ''transient cerebellar mitism syndrome with subsequent dysarthria'' (Van Dongen, Catsman-Berrevoets, & van Mourik, 1994) following resection of posterior fossa tumors in children constitutes a well-recognized behavioral disorder. Though it has sporadically been described in adults (e.g., Salvati, Missori, Lunardi, & Orlando, 1991; D'Avanzo, Scuotto, Natale, Scotto, & Ciofi, 1993; Çakir, Karakisi, & Koçanaogullari, 1994; Dunwoody, Alsagoff, & Yuan, 1997) and in association with brain stem tumor surgery (e.g., D'Avanzo et al., 1993; Frim & Ogilvy, 1995; Ersahin, Mutluer, Saydam, & Barçin, 1997), the syndrome most frequently occurs in children who underwent vermian tumor surgery (estimated incidence up to 15%) (Pollack, 1997). Other etiologies such as traumatic cerebellar injury (e.g., Yokota, Nakazawa, Kobayashi, Taniguchi, & Yukihide, 1990), brain stem infarction following traumatic injury of the vertebral artery (Miyakita, Taguchi, Sakakibara, Matsuzawa, & Kitagawa, 1999), and viral infections of the cerebellum (Riva, 1998) have exceptionally been reported. The condition of speechlessness, frequently associated with a spectrum of abundant behavioral changes typically develops with a latency of 1–4 days after surgery and recedes after a period of weeks to 4 months. Aside from a residual dysarthria recovery is generally complete. Levisohn, Cronin-Golomb, and Schmahmann (2000) and Riva and Giorgi (2000) recently identified, however, long-term clinically relevant cognitive and affective changes in children with resection of posterior fossa tumors.

Several risk factors for the development of the posterior fossa syndrome have been proposed: preoperative hydrocephalus, tumor location (Pollack, Polinko, Albright, Towbin, & Fitz, 1995), tumor type and size (Catsman-Berrevoets, van Dongen,

Mulder, Paz y Geuze, Paquier, & Lequin, 1999), rostrocaudal length of the vermian incision (Dailey, 1995; Pollack et al., 1995), acute bilateral cerebellar injury (Rekate, Grubb, Aram, Hahn, & Ratcheson, 1985), dentate nucleus injury (Ammirati, Mirzai, & Samii, 1989; Çakir et al., 1994), postoperative edema within the brachium pontis and/or brachium conjunctivum, postoperative hydrocephalus and meningeal reactions (Humphreys, 1989; Ferrante, Mastronardi, Acqui, & Fortuna, 1990), transient dysfunction of the A9 and A10 mesencephalic dopaminergic cell-groups and ascending activating reticular system (Catsman-Berrevoets, van Dongen, & Zwetsloot, 1992), postoperative arterial spasms causing ischemia and disturbed cerebellar perfusion (Nagatani, Waga, & Nakagawa, 1991).

Riva and Giorgi (2000) not only reported for the first time in a pediatric population long-lasting cognitive deficits but also linguistic dysfunctions after vermal medulloblastoma resection. In the early phase of recovery from mutism, two of six surgically treated children presented with a predominantly expressive language syndrome. This syndrome essentially consisted of prosodic abnormalities (bradylalia and flattened intonation) and expressive syntax disturbances ''reminescent of the agrammatical language frequently encountered in aphasic patients (including children) with acquired left frontal lesions.'' Formal language assessments later revealed excellent auditory–verbal comprehension, normal repetition, ''severe lack of spontaneity in terms of active language, and [a tendency] to speak very little even after being encouraged to do so.'' Three years after the operation the syntax disturbances had resolved but language quality was considered poor. In contrast to the four children with a classic presentation of the syndrome, the linguistically impaired children had (aside from a partial excision of the vermis) an additional lesion of the right cerebellar hemisphere. The authors consequently related the linguistic manifestations to focal damage of the right cerebellum and concluded in the absence of functional imaging data that ''it is impossible to determine whether the deficits . . . are directly due to the cerebellar lesion or to diaschisis arising from the sudden interruption of the reciprocal connections between the different cerebral regions and the cerebellum.''

Our findings seem to corroborate the observations of Riva and Giorgi (2000) and might further contribute to the understanding of the pathophysiological substrate of the intriguing spectrum of behavioral disturbances that may follow ischemia or tumor resection in the posterior fossa. First, our adult case with cerebellar-induced aphasia following a lesion of the right cerebellar hemisphere sheds some light on the above-raised issue (Mariën et al., 1996, 2000). In agreement with Riva and Giorgi's observations (2000) a genuine aphasic syndrome was found that typologically resembled Luria's frontal dynamic aphasia with agrammatism. As indicated by SPECT, these aphasic symptoms likely resulted from diaschisis affecting the contralateral prefrontal cortical areas probably through cerebello-ponto-thalamo-cortical pathways. Second, we also encountered in several children with resection of posterior fossa tumors almost identical aphasic disturbances (unpublished observations). Given the overt clinical resemblances that the frontal-like nature of the behavioral alterations observed in these children we started to investigate these patients with SPECT and an extensive neuropsychological test battery. The preliminary results of this study reveal a correlation between type and extent of the behavioral dysfunctions and the area and degree of crossed cerebral diaschisis and support the pathophysiological view on cerebellar-induced language disturbances as a diaschisis phenomenon. For instance, in a 10-year-old right-handed boy surgically treated for a pilocytic astrocytoma, 99mTc-HMPAO SPECT disclosed severe bifrontal perfusion defects during a 3-week period of akinetic mutism (Fig. 3A). When the boy gradually recovered, he displayed dynamic aphasia, agrammatism, and slight behavioral alterations. Repeated SPECT

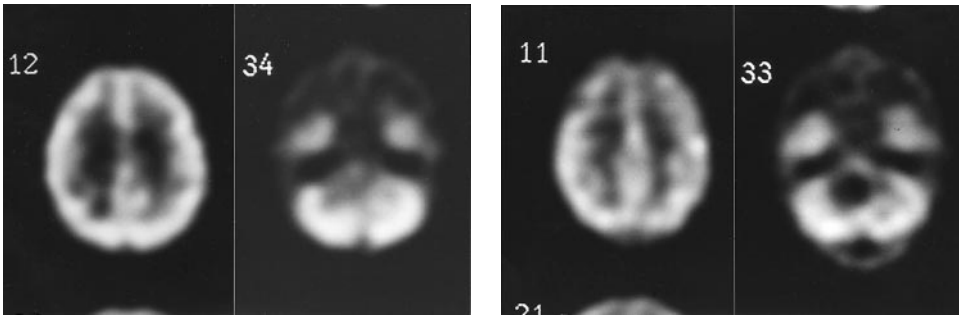


(A) (B)

FIG. 3. **(A)** 99mTc-HMPAO SPECT of the brain performed during a 3-week period of akinetic mutism following posterior fossa surgery. In addition to a pronounced relative (anteroposterior) bifrontal hypoperfusion, a marked relative hypoperfusion in the left hemisphere of the cerebellum is shown. **(B)** Repeated SPECT performed during the early phase of recovery from akinetic mutism reveals clearly improved perfusion of the frontal lobes with minimal perfusion decrease in the parietal lobe of the left cerebral hemisphere. A slight asymmetry of the cerebellar hemisphere persists (relative hypoperfusion of the left cerebellar hemisphere).

showed a reduction of the frontal perfusion deficits which were, however, still significantly more pronounced in the left frontal region (Fig. 3B). In several other children with posterior fossa tumors a similar parallelism was found between the regional distribution and extent of perfusion deficits and the type and course of cognitive symptoms. In some of the patients under consideration this correlation was even found in the preoperative phase. Preoperative cognitive assessments in a child with a cerebellar medulloblastoma demonstrated an isolated linguistic deficit consisting of significant diminution of self-generated speech. This finding was reflected by a left frontal hypoperfusion on SPECT (Fig. 4A). After tumor resection spontaneous speech worsened and correlated with an aggravation of hypoperfusion in the left frontal areas (Fig. 4B).

Though carefully controlled studies with large groups of patients are needed to draw sound conclusions, these findings support the view that aphasic dysfunctions



(A) (B)

FIG. 4. **(A)** Preoperative 99mTc-HMPAO SPECT of the brain performed in a 5-year-old right-handed child with a cerebellar medulloblastoma and linguistic deficits. A relative hypoperfusion of the right cerebellar hemisphere is shown. Although there is no breach of the cerebral cortex there is less physiological activation of the left frontal lobe. **(B)** Postoperative SPECT demonstrates a large scintigraphic defect in the cerebellum slightly off-center to the right. A strikingly increased hypoperfusion deficit in the left frontal lobe is shown.

observed after lesions of the right cerebellar hemisphere might be interpreted as a direct effect of diaschisis via the cerebello-ponto-thalamo-cortical pathways.

TOWARD THE CONCEPT OF CROSSED CEREBELLAR APHASIA

The ''Lateralized Linguistic Cerebellum''

Contemporary investigations increasingly show that the cerebellum is topographically organized in subserving a wide range of cognitive, linguistic, and affective functions. Schmahmann et al. (Schmahmann, Loeber, Marjani, & Hurwitz, 1998; Schmahmann, Doyon, McDonald, Holmes, Lavoie, Hurwitz, Kabani, Toga, Evans, & Petrides, 1999; Schmahmann, 2000) provided preliminary evidence for at least three functionally distinct cerebellar areas: (1) the so-called sensorimotor cerebellum, located rostral to the primary fissure in the anterior lobe with a secondary representation in lobules VIII/IX of the cerebellar hemispheres; (2) the ''cognitive cerebellum,'' located in lobule VI and VII at the vermis and extending into lobule VI and crus I and II of lobule VIIA and lobule VIIb of the cerebellar hemispheres; and (3) the ''limbic cerebellum'' that encompasses the phylogenetically older cerebellar fastigial nucleus, the vermis, and the flocculonodular lobes. Within this frame of topographic functional representations, a robust amount of clinical and experimental evidence seems to indicate that the modulatory function of the cerebellum in nonmotor linguistic processes are represented in a highly restricted way in what might be called the fourth cerebellar area or the ''lateralized linguistic cerebellum.'' As reviewed above, it has been demonstrated that the right hemisphere of the cerebellum is at least crucially involved in (1) the integrated subsystem of working memory that subserves several language processes, (2) articulatory planning, (3) a variety of linguistic operations implicated in semantic and phonological word retrieval, (4) syntactic processing, and (5) the dynamics of language processing (Table 2).

Crossed Cerebellar Aphasia: A New Avenue in Crossed Aphasia and Cerebellar Cognition?

As an exception to so-called Broca's doctrine, assigning left-hemisphere dominance for language to dextrals and right hemisphere dominance for language to sinistrals, Byrom Bramwell (1899) introduced, more than a century ago, the term ''crossed phasia.'' This concept denotes the exceptional condition in which an aphasic syndrome results from a cerebral lesion ipsilateral to the dominant hand (i.e., aphasia in left-handers following a left cerebral hemisphere lesion and aphasia in right-handers following a right cerebral hemisphere lesion). Since then, more than 200 cases of crossed aphasia in dextrals (CAD) have been documented in the literature, which evidence that exceptions may occur in the neurobiological organization of lateralized brain functions (Mariën, Engelborghs, Vignolo, & De Deyn, in press). Following close anatomical and functional interconnections between the ''lateralized linguistic cerebellum'' and the contralateral language dominant hemisphere it seems plausible to hypothesize that similar anomalous configurations may hold for the functional organization of linguistic functions at the cerebellar level. Though this new avenue of ''crossed cerebellar aphasia'' (cerebellar induced aphasia resulting from a lesion in the left cerebellar hemisphere in right-handed individuals) is yet to be explored, the literature contains one single case study that seems to corroborate this concept. Fabbro et al. (2000) reported a 59-year-old right-handed bilingual woman with an astrocytoma of the left cerebellar hemisphere. Formal language investigations performed 2 and 3 weeks after surgery revealed fluent spontaneous speech with some

TABLE 2
Cerebellar Involvement in Speech and Language Functions

Linguistic level	Function	Disturbance	Lateralization	References
Articulation	Muscular speech control	Ataxic dysarthria	Left cerebellar hemisphere more frequent than the right	Holmes, 1917; Lechtenberg & Gilman, 1978
	Covert articulation		Right cerebellar hemisphere	Fiez & Raichle, 1997
	Articulatory planning	Speech apraxia	Right cerebellar hemisphere	Mariën et al., 2001
Speech perception	Discrimination of VOT and vowel duration		Right cerebellar hemisphere	Ackermann et al., 1996
Spelling	Visuospatial organization	Afferent dysgraphia	Left cerebellar hemisphere	Silveri et al., 1997, 1999
Linguistic processing	Verbal associations	Lexical retrieval deficit	Right cerebellar hemisphere	Petersen et al., 1988, 1989
	Generation of synonyms, translations, rhyming words	Lexical retrieval deficit	Right cerebellar hemisphere	Klein et al., 1995
	Word completion	Lexical retrieval deficit	Right cerebellar hemisphere	Buckner et al., 1995
	Semantic associations	Lexical retrieval deficit	Right cerebellar hemisphere	Fiez et al., 1992
	Phonological generation	Disturbed verbal fluency	Right cerebellar hemisphere	Leggio et al., 1995
	Expressive grammar	Expressive agrammatism	Right cerebellar hemisphere	Silveri et al., 1994; Zettin et al., 1997
	Syntactic knowledge	Agrammatism	Right cerebellar hemisphere	Mariën et al., 1996; 2000; Riva, 1998; Gasparini et al., 1999; Fabbro et al., 2000; Riva & Giorgi, 2000
	Language dynamics	Dynamic aphasia	Right cerebellar hemisphere	Mariën et al., 1996, 2000; Riva, 1998; Gasparini et al., 1999; Fabbro et al., 2000; Riva & Giorgi, 2000

Note. VOT = voice onset time.

morphosyntactic errors, poor grammatical comprehension, and poor mental arithmetic skills. Propositionizing and reading were the most impaired linguistic levels, whereas syntax was the most impaired linguistic skill. The authors remarked that the linguistic deficits of this patient only differed in lesion site. Given the growing amount of evidence that similar lesions of the right hemisphere of the cerebellum induce similar language deficits it seems likely that the clinical anatomical profile of this patient represents the phenomenon of ''crossed cerebellar aphasia'' following anomalous lateralization of the ''linguistic cerebellum'' in the left cerebellar hemisphere.

CONCLUSION

Neuroanatomic, functional, and clinical investigations have provided converging evidence in support of the view that the cerebellum is crucially implicated in a variety of nonmotor cognitive and neurolinguistic processes. That our understanding of the contribution of the cerebellum to these processes is currently still in a preliminary stage is essentially due to the historic neglect of the nonmotor role of the cerebellum, but also follows the fact that the cerebellum primarily acts as a modulator of cognition. If this modulating function is impaired, behavioral deficits arise that are quantitatively and qualitatively different from the deficits produced by lesions of the supratentorial structures. Therefore standard test batteries which focus on the detection of cognitive and neurolinguistic impairments are often not sensitive enough to reveal and objectify the ''subtle'' deficits that may follow cerebellar damage. In addition, the possibility of inadequate assessment even seems to increase since the impairments induced by cerebellar damage often evolve rapidly. As a consequence, refinement of cognitive test methodologies and the development of specifically adapted clinical investigation tools are required to further explore and delineate the exact role of the cerebellum in cognitive and neurolinguistic dysfunctions. As suggested in this review one such new and intriguing avenue in cerebellar cognitive research seems to be the further development of the concept of a functionally lateralized linguistic cerebellum and its modulatory role in nonmotor linguistic disorders such as apraxia of speech, classic aphasia syndromes, and aphasia in atypical populations. In this contribution the advanced view that linguistic deficits following cerebellar pathology do not imply representation of linguistic functions at the cerebellar level, but reflect functional deactivation of the supratentorial language areas due to reduced input via cerebellocortical pathways puts emphasis on diaschisis processes as the relevant pathomechanism for cerebellar induced language disorders.

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