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The Lateralized Linguistic Cerebellum: Fact or Fallacy?

Peter Mariën[1], Sebastiaan Engelborghs[1], Franco Fabbro[2] & Peter de Deyn[1]

[1]Department of Neurology, General Hospital Middelheim, Antwerp, Belgium; and Laboratory of Neurochemistry and Behavior, Born-Bunge Foundation, University of Antwerp, Belgium
[2]Unit of Neurolinguistics, IRCCS "E.Medea", San Vito al T. (PN), Italy

During the past two 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. In this contribution the recently acknowledged role of the cerebellum in cognition is reviewed and 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 are addressed. In agreement with the findings of studies 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[1] processes, reflecting a functional depression of supratentorial language areas due to reduced input via cerebello-cortical pathways might represent the relevant pathomechanism for linguistic deficits associated with cerebellar pathology.

1. Cerebellar induced language disturbances

1.1. Introduction

Leiner et al. (1986, 1989) discussed the functional expansion of the cerebellum as a consequence of structural changes that evolved during homonid 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 et al., 1998), and backward projections from the prefrontal areas to the lateral cerebellum via the pons and the

Contact author: Peter Mariën, Department of Neurology, General Hospital Middelheim, Lindendreef 1, B-2020 Antwerp, Belgium. E-mail: petermarien@skynet.be

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 non-motor linguistic processes. In the past ten years, a close cooperation across disciplines has established the view that the cerebellum plays a crucial role within the language network.

1.2. 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 non-motor language (Leiner et al., 1986). 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. A 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. 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 et al. (1992) conducted the first specially designed study on word generation in a patient who presented with semantic 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 a various semantic word generation tasks. Leggio et al. (1995) conducted both phonological and semantic verbal fluency studies in three etiologically distinct patient groups with cerebellar pathology. 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 study showed that: 1) the cerebellar lesioned group performed at a lower level than the matched controls irrespectively 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 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 et al. (2000) basically confirmed these findings and demonstrated that verbal fluency deficits in their study population could not be attributed to motor speech impairment. 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 cere-

bellar damage affects phonological processes to greater extent than semantic processes because phonological tasks depend on unusual novel and less automized searching strategies than semantic tasks.

1.3. Disorders of grammatical production

The role of the right cerebellum in non-motor language functions has recently been expanded by evidence derived from patients with agrammatism. Silveri et al. (1994) and Zettin et al. (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 et al., 1996, 2000; Gasparini et al., 1999). Similar observations were made in a child with cerebellitis (Riva, 1998), a group of patients with infiltrative cerebellar damage (Fabbro et al., 2000), and in two children after posterior fossa surgery (Riva & Giorgi, 2000).

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. 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, 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 behavioural adaptation to a deficit outside the mental linguistic system. The deficit was not considered to affect syntactic competence but the on-line 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 de-coupled 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 de-coupling 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 both recep-

tive 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 linguistic system. Following the observation that additional aphasic deficits may accompany expressive and receptive agrammatism Mariën et al. (1996, 2000) maintained that right cerebellar lesions may provoke aphasia. The observations of Riva (1998), Fabbro et al. (2000) and Riva and Giorgi (2000) in an etiologically different population corroborate this view.

1.4. Cerebellar induced aphasia

Hassid (1995) described a 17-year-old left-handed man with a right cerebellar hemisphere infarction. In addition to classical cerebellar motor symptoms including dysarthria, formal testing revealed moderate anomia on the Boston Naming Test (Kaplan et al., 1983), mild difficulties in auditory reception and reading, and severe difficulties in writing and mathematics. CT and 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 region of the left cerebral hemisphere, consistent with an infarction in the right hemisphere of the cerebellum associated with crossed cerebral diaschisis. 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 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 ^{99m}Tc-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 six months after onset. In association with near remission of the aphasic symptoms, near normalisation of the perfusion pattern in the left frontoparietal area was found five years post onset. Since it was first recognized by Broich et al. (1987), this phenomenon of so-called 'crossed cerebello-cerebral diaschisis' has been amply documented. In contrast however to an already well-documented range of cognitive correlates of 'cerebral diaschisis', the literature makes only scant mention of cognitive dysfunctions associated with 'crossed cerebello-cerebral diaschisis'. 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 cerebello-cortical pathways.

Gasparini et al. (1999) contended the view that neurolinguistic impairments after right cerebellar damage constitute diaschisis related phenomena. 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 four right-handed patients with tumoural cerebellar lesions before and after surgery. Irrespective of lesion type and lesion localization (vermis, 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 these 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.

1.5. Transient cerebellar mutism syndrome

So-called 'fossa posterior syndrome' or 'transient cerebellar mutism syndrome with subsequent dysarthria' (Van Dongen et al., 1994) following resection of posterior fossa tumours in children constitutes a well-recognized behavioural disorder. Though it has sporadically been described in adults (e.g. Salvati et al., 1991) and in association with brain stem tumour surgery (e.g. D'Avanzo et al., 1993), the syndrome most frequently occurs in children who underwent vermian tumour surgery (estimated incidence up to 15%) (Pollack, 1997). Other etiologies such as traumatic cerebellar

injury (e.g. Yokota et al., 1990), brain stem infarction following traumatic injury of the vertebral artery (e.g. Miyakita et al., 1999) and viral infections of the cerebellum (e.g. Riva, 1998) have exceptionally been reported. The condition of speechlessness, frequently associated with a spectrum of abundant behavioural changes typically develops with a latency of one to four days after surgery and recedes after a period of weeks to four months. Aside from a residual dysarthria recovery is generally conceived complete. Levisohn et al., (2000) and Riva and Giorgi (2000), recently identified, however, long-term clinically relevant cognitive and affective changes in children with resection of fossa posterior tumours.

Several risk factors for the development of the fossa posterior syndrome have been proposed: preoperative hydrocephalus, tumour location, tumour type and size, rostrocaudal length of the vermian incision, acute bilateral cerebellar injury, dentate nucleus injury, postoperative oedema within the brachium pontis and/or brachium conjunctivum, post-operative hydrocephalus and meningeal reactions, transient dysfunction of the A9 and A10 mesencephalic dopaminergic cell-groups and ascending activating reticular system, postoperative arterial spasms causing ischemia and disturbed cerebellar perfusion.

Riva and Giorgi (2000) not only reported for the first time in a paediatric 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 and expressive syntax disturbances 'reminescent of the agrammatical language frequently encountered in aphasic patients (including children) with acquired left frontal lesions'. Formal language assessments 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 classical 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 (Mariën et al., 2001) corroborate the observations of Riva and Giorgi (2000) and further contribute to the understanding of the pathophysiological substrate of the intriguing spectrum of behavioural disturbances that may follow ischemia or tumour resection in the posterior fossa. Firstly, 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. Secondly, we also encountered in several children with resection of fossa posterior tumours almost identical aphasic disturbances (unpublished observations). Given the overt clinical resemblances and the frontal-like nature of the behavioural 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 behavioural dysfunctions and the area and degree of crossed cerebral diaschisis and support the pathophysiological view on cerebellar induced language disturbances as a diaschisis phenomenon.

2. 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. (1998, 1999) and Schmahmann (2000) provided preliminary evidence for at least three functionally distinct cerebellar areas: the so-called 1) sensorimotor cerebellum, 2) the 'cognitive cerebellum', and 3) the 'limbic cerebellum'. 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 non-motor linguistic processes are represented in a highly restricted way in what might be called the fourth cerebellar area or the 'lateralized linguistic cerebellum'. Mariën et al. (2001) demonstrated that the right hemisphere of the cerebellum is at least crucially involved in: 1) the integrated sub-system 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. The fact that only a very limited number of patients have been reported (e.g. Fabbro et al., 2000) who display structural language defects after a *left* cerebellar lesion might be explained by the fact that in a subgroup of the right-handed population language functions are anomalously lateralised. In line with this view, patients with language disturbances following a left cerebellar leison might represent 'crossed cerebellar aphasia'.

3. 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 non-motor 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 non-motor 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, behavioural 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 anvenue in cerebellar cognitive research seems to be the further development of the concept of a functionally lateralized linguistic cerebellum and its modulatory role in non-motor linguistic disorders such as apraxia of speech, classic aphasia syndromes and aphasia in atypical populations. In this contribution we advance the view that linguistic deficits following cerebellar pathology do not imply representation of linguistic functions at the cerebellar level, but reflect functional de-activation of the supratentorial language areas due to reduced input via cerebello-cortical pathways puts emphasis on diaschisis processes as the relevant pathomechanism for cerebellar induced language disorders.

Samenvatting

De klassieke opvatting dat het cerebellum louter fungeert als een coördinator van autonoom motorische en somatische functies is tijdens de afgelopen twee decades in belangrijke mate gewijzigd. In deze bijdrage wordt de recent onderkende rol van het cerebellum in cognitieve processen geschetst. In het bijzonder wordt aandacht besteed aan experimentele en klinische onderzoeken die de modulerende rol van het cerebellum aantonen binnen zuiver linguïstische processen zoals de syntaxis, de taaldynamiek en de woordvinding. In het licht van de bevindingen die een topografische organisatie van het cerebellum aantonen binnen taalprocessen, wordt het concept van een 'gelateraliseerd linguïstisch cerebellum' uitgewerkt en worden taalstoornissen na cerebellaire aandoeiningen teruggevoerd op de pathofysiologische hypothese van 'crossed cerebral diaschisis' die de reflectie vormt van een verminderde functionaliteit van de supratentoriële taalcentra als gevolg van een gereduceerde input van excitatoire stimuli.

Note

1 Constantin Von Monakow elaborated in 1914 the concept of diaschisis. He defined the phenomenon as the result of a focal lesion that causes diminished function of areas of the central nervous system situated either much below the actual lesion, symmetrically in the opposite hemisphere or within a functional system of zones working collectively. When depression of the excitability of brain regions belonging to a single functional system occurs, the whole system may be brought into a state of temporary inactivity.

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