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Cerebellar mutism

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Abstract
Introduction Cerebellar mutism (CM) is defined as a peculiar form of mutism that may complicate the surgical excision of posterior cranial fossa tumor. The incidence is variable in the literature, occurring in up to one third of cases in some series. Commonly occurring peculiar features of CM are delayed onset following surgery, limited duration, and spontaneous recovery usually associated with dysarthria.
Methods A review has been performed concerning anatomical substrates and circuits actually considered to be involved in the development of cerebellar mutism, as well as risk factors for its development that have been documented in the literature. Attention has also been given to the long-term prognosis and the possibilities of rehabilitation that can be considered in these children, which has been compared with the authors' institutional experience.
Results and conclusions Tumor infiltration of the brainstem seems to represent the most relevant feature related to the development of CM, along with the histological diagnosis of medulloblastoma. On the other hand, hydrocephalus does not represent an independent risk factor. The higher rate of CM in children seems to be related to the higher incidence in children of tumors with malignant histology and brain stem involvement. Surgical technique does not seem to have a definite role; in particular, the use of a telovelar approach as compared to vermian split to reach the fourth ventricle extension of the tumor has not been demonstrated to prevent the development of cerebellar mutism. Concerning long-term prognosis, around one third of the children who develop cerebellar mutism after surgery have a persistent dysarthria, the remaining ones showing a residual phonological impairment. Long-term dysarthric features tend to be more severe and less prone to recovery in children presenting at diagnosis with associated combined procedural memory and defective neurocognitive functions.

Keywords Posterior fossa tumors · Mutism · Posterior cranial fossa syndrome · Mutism and subsequent dysarthria · Medulloblastoma · Ependymoma · Astrocytoma

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Definition

Phonation is a complex activity, requiring the synergistic activity of 40 muscles and 18 nerves controlled at a higher level by several intricate neural networks, involving cortical and subcortical structures, as well as the brainstem and cerebellum [1]. Beside the pure motor aspect, the uttered phonemes are enriched by a cognitive value to constitute the basis of *language*.

Mutism is defined as the total absence of speech in an awake and conscious patient. This condition includes a wide range of disorders, variously mixing the motor and cognitive impairments. Indeed, *aphasia*, typically resulting from cerebral cortical lesions, is a disorder of the cognitive component with or without a motor component whereas *aphonia*, typically resulting from peripheral nerve disruption, and *anarthria* (also known as *oroapraxia*), in which performance of learned motor skills of face, lip, and tongue are affected without an apparent neuroanatomic lesion, would be purely motor

impairment. On the other hand, *selective or elective mutism* represents a refusal of speech usually following psychological trauma.

Cerebellar mutism (CM) is defined as a peculiar form of mutism that may complicate the surgical excision of a posterior cranial fossa tumor. The incidence is variable through the literature, occurring in up to one third of cases in some series [2, 3]. Peculiar features of CM are delayed onset following surgery, limited duration, and spontaneous recovery usually associated with dysarthria [2, 3]. Due to these characteristics, CM has been also defined in the literature as *akinetic mutism*, *transient cerebellar mutism*, and *mutism with subsequent dysarthria* and these definitions have been used interchangeably so far. Though simply considered a form of severe dysarthria merging into anarthria by some authors [4], as supported by cases of mutism associated to difficulty in performing simple orofacial movements [5, 6], the movements of lip, tongue, and palate are usually normal in patients affected by CM, thus complicating the interpretation of this condition. Furthermore, behavioral abnormalities, such as apathy, eating dysfunction, poor oral intake, eye closure, urinary retention, and personality changes, such as depressed affect, whining, irritability and agitation, and emotion instability, may often accompany CM, thus configuring more complex syndromes. In general, *cerebellar mutism syndrome* (CMS) includes ataxia, hypotonia, and irritability. *Posterior fossa syndrome* (PFS) additionally includes cranial nerve deficits, neurobehavioral changes, and urinary retention or incontinence [7]. Interestingly, a similar syndrome, variably associating linguistic, cognitive, and behavioral-affective disturbances, has been described in adults with cerebellar lesions and defined *cerebellar cognitive affective syndrome* (CCAS) [8, 9].

However, this terminology is largely inconsistent through the literature and many of the abovementioned terms have been used interchangeably. Some authors propose to distinguish CMS with neurological deficits related to cerebellar functions from PFS that includes symptoms related to cranial nerves or brainstem deficits [10]. Finally, the severity of the syndrome may largely vary, although duration is the only factor considered through the literature to date for grading it [11].

Anatomical circuitry

The anatomical substrate of CM is not fully understood, although a recent pathogenetic hypothesis is based upon three main steps. The impairment of dentato-thalamo-cortical tract would be the first event. The subsequent disturbance of the cerebello-cerebral circuitry would cause a decrease of the cerebellar facilitating activity. This phenomenon, also known as diaschisis, would result in the hypofunction of cerebral cortical areas [12]. In this context, the dentate nucleus has been identified as the signaling output channel of cerebellum, whereas the role of the cerebellar structures acting upstream in the linguistic input generation is unclear. Indeed, the data on cerebellar representation of language control are still debated, as confirmed by a recent consensus paper that thoroughly addresses the involvement of the cerebellum in several aspects of language, such as speech and language perception, motor speech planning, verbal working memory, phonological and semantic verbal fluency, syntax processing, the dynamics of language production, reading, and writing [13].

The following paragraphs would briefly focus on the role of different cerebellar structures involved in this circuitry, according to the integrated functional and morphologic radiological data available in the literature to date.

Vermis

The vermis and the paravermal areas, through neural connections with deep cerebellar nuclei, are involved in coordination of laryngeal and respiratory functions, which are the basis of fluent speech, as demonstrated experimentally in animals [14–16]. The inferior vermis, receiving tactile information from the limbs and also from the head and face, has been firstly considered responsible for speech initiation [17]. Hence, some authors related splitting of the vermis to CM [6], but this hypothesis was not confirmed. The splitting limited to the inferior third of the vermis is a safe procedure to approach fourth ventricle tumors, that is not associated to a higher risk of CM [18, 19]. On the other hand, evidence from patients suffering cerebellar stroke in the territory of the superior cerebellar artery advocated the role of the superior portion of vermis and the contiguous paravermal cortex [20]. This is further supported by the clinical observation of a case of preoperative mutism secondary to an intralesional focal hemorrhage of the upper vermis [21].

Additionally, the role of the vermis would concern not only motor aspects of language but also complex and social behavior processing. Indeed, according to the functional topography of cerebellum, the vermis would represent the cerebellar limbic system, thus being involved in the modulation of emotions, whereas the lateral hemispheres would take part in modulating thought, language, and ability to plan [22, 23]. Interestingly, imaging studies performed in children affected by autism strongly linked speech delays with hypoplasia of the whole cerebellum or of the vermis [24, 25], in particular of its anterior I–V portion [26], and the similarities between CM and autistic spectrum disorder would not be limited to this anatomical feature [27]. Finally, vermis hypoplasia has been correlated to an impairment of the phonological short-term memory, whose integrity would be necessary for the language acquisition [28].

Lateral hemispheres cortex

Lesion studies significantly related speech disorders to the superior surface of the hemisphere, namely the paravermal regions at the level of lobules VI and VII [29]. This was initially confirmed by a case of isolated dysarthria due to a small infarct placed in this region on the left side [30]. Thereafter, studies on cerebellar ischemic lesions confirmed the hypothesis of a role of the superior part of the lateral hemisphere, showing that an infarct within superior cerebellar artery region causes speech deficits but suggested that the right-sided was more important [20, 31].

More recently, functional imaging studies documented the presence of a link between left frontal language regions and the contralateral cerebellum, showing an activation of the lateral posterior hemisphere (lobule VI, VIIb, Cr I, Cr II) contralateral to the language-dominant cerebral hemisphere [32] and some fMRI-based studies even place the tongue representation in the cerebellar lobules VI–VII [33]. Interestingly, language-impaired patients showed a smaller right side posterolateral lobule VIIIa associated with a smaller left inferior frontal cortex language area if compared to the contralateral side, whereas unimpaired patient showed an opposite asymmetry [26]. Furthermore, the crossed lateralization of language in the cerebellum has been recently confirmed to be irrespective of handedness or typical or atypical language representation [34]. Therefore, the so-called lateralized linguistic cerebellum [35] could be involved in the pathogenesis of CM.

The role of the midline structures would not rule out the influence of the lateral hemispheres, which could take part in different aspects of complex language production. In fact, functional imaging studies, based on PET and fMRI, reported bilateral foci of activation in the ventrolateral thalamus and in the cerebellum during singing and speaking. In the cerebellum, two foci of activation were reported: one in the vermis and one in the lobule VIa of the lateral hemisphere [36–40].

Thus, the generation of the linguistic cerebellar input would need the cooperation of different cortical cerebellar structures, analogous to cerebral structure organization. The synergistic activation of these structures would eventually reflect the dichotomy between the motor and cognitive aspects of language. Indeed, overt speech or articulation would be controlled by vermal and paravermal regions of lobules V–VI and VII–VIII, consistently with the hypothesized location of cerebellar sensorimotor homunculi, whereas the cognitive aspects of language would depend on the function of the lateral right cerebellum. This explanation is ontogenetically and phylogenetically founded, since the vermis, also known as *paleocerebellum*, would intervene in the less evolved aspects of language, whereas the lateral hemispheres, or *neocerebellum*, would take part in the higher aspects of language. On these grounds, the functional topography of cerebellum may eventually account for the cohort of symptoms eventually associated with CM [23, 41].

Finally, the cognitive modulation exerted by the cerebellar hemispheres has been hypothesized to reflect as a mirror the cerebral counterpart, with the right cerebellar hemisphere being associated with logical reasoning and language processing and the left cerebellum mediating right-hemispheric functions including attentional and visuo-spatial skills [42]. This hypothesis could be confirmed by the evidence of a location-related pattern in speech disorders due to cerebellar injuries [43].

Dentate nucleus and efferent fibers

Although literature data about the role of cerebellar nuclei are scarce, the first evidence of the involvement of the dentate nucleus in the pathogenesis of mutism comes from the clinical report of two cases of transient mutism following stereotactic lesioning in both dental nuclei for the treatment of dyskinetic syndrome [44]. The dentate nucleus receives projections from large areas of the ipsilateral cerebellar cortex, including the vermis [15], and projects its fibers, passing through the superior cerebellar peduncles, to the contralateral red nucleus and ventrolateral thalamus [45, 46]. This pathway, reaching the cerebral cortex through the thalamo-cortical projection, would connect the regulating cerebellar structures to the executing cerebral areas [47]. Thus, the dentate nucleus could be the output station of signal directed to the motor domain, targeted primary motor, and premotor areas of the cerebral cortex, as well as to the non-motor domain. In the monkey, the motor channels were localized in the dorsal portion of the dentate and the non-motor in the ventral portion of the dentate nucleus, and a unique molecular marker differentially recognize these two domains [48, 49]. This dichotomy would reflect respectively the motor speech aspects and the cognitive related aspects of language.

Finally, the role of the fibers leaving the cerebellum and conveying the signals generated by the cerebellar cortical structures and integrated by the dentate nucleus is crucial. These fibers running through the superior cerebellar peduncle into the brainstem may be injured by the presence of a cerebellar tumor or by the surgical procedure to resect the tumor [50]. Indeed, tractography has confirmed the qualitative and quantitative impairment of these fibers in patients with CM [51, 52]. The incomplete maturation of myelin in children may account for the susceptibility of these fibers to injury [53]. Interestingly, diffusion abnormalities of proximal efferent cerebellar pathways have been recently documented by means of intraoperative MRI in children developing CM [54].

In conclusion, the complexity of neural structures involved in the linguistic functions does not allow definitive conclusions on the pathogenesis of CM. Probably the same clinical epiphenomenon, that is CM, may be caused by an injury

affecting the median or paramedian structures of the cerebellum [21, 29, 55, 56], the dentate nucleus [44, 57, 58] or the proximal dentato-rubral or dentato-thalamo-cortical way [47, 59]. Although bilateral damage of neural structures has been advocated [60, 61] for the pathogenesis, an injury to the dominant-side pathway is probably necessary and sufficient by itself to cause the onset of CM.

Factors considered as possibly influencing the development of cerebellar mutism

Though several factors have been claimed to contribute to the development of CM after posterior fossa tumor surgery, it is not easy to create a unifying hypothesis and pathogenetic mechanism. In the past, the adopted surgical technique has been considered as one of the contributing factors; specifically, the most implicated step of the surgery was splitting of the vermis to access fourth ventricular tumors. In 1995, Dailey et al. reviewed the pertinent literature concerning cerebellar mutism and collected 33 published cases, which was added to their personal experience in nine cases. Looking at the data, 34 of the overall 42 tumors involved the vermis, consequently requiring the partial or total removal of the affected part of this structure by itself, but vermian split was clearly reported only in less than half of the cases (17/42 = 40.5 %). In spite of this, the authors concluded that to avoid mutism, the inferior vermis must be preserved [6]. Based on this assumption, numerous efforts have been made to avoid the splitting of the vermis or to minimize the extent of the incision [62]. Kellogg and Piatt firstly proposed that approaching the fourth ventricle through the cerebello-medullary fissure might yield exposure comparable to what can be achieved by splitting the vermis and could minimize the risk of developing CM; however, this paper was substantially an anatomical report based on the authors' experience in 11 cases and the results of several cadaveric dissections, no mention being given on the rate of postoperative CM in their experience [63]. El-Bahy used this approach to the fourth ventricle in 16 patients, and CM was not observed in any patient. Again, among these patients, only three harbored medulloblastomas while the remaining patients had laterally located tumors and nontumoral lesions, which are not usually associated with the development of CM after surgery. Thus, it is difficult to conclude whether this approach was beneficial [64]. The most beneficial results of the telovelar approach in preventing CM were reported by Von Hoff et al., who observed no case of CM after surgery for ependymoma in 23 children [65]. Zaheer et al. reported their personal experience with the telovelar approach in 20 children, six of whom (6/30 = 30 %) developed CM after surgery; this relatively high rate in spite of the avoidance of split of the vermis was explained by the authors with their prospectively planned strategy for an aggressive tumor removal, with the suggestion that avoiding vermian split might be relatively less relevant in the pathogenesis of postoperative mutism when compared with edematous reaction as a response to surgical manipulation of the deep medial cerebellar structures [66]. Viewing the issue from the opposite side, authors reporting vermian split as their surgical route for midline tumors actually report this complication only in around 25 % of the operated cases. Among the eight children with CM who were operated on at our unit, a split of the inferior vermis was performed only in two case (2/8 = 25 %), whereas the remaining six children (6/8 = 75 %) underwent surgery through a telovelar approach.

According to some authors, one of the further factors that might influence the development of CM in spite of sparing the vermis is the choice of a bilateral telovelar fissure opening. In fact, this kind of difference has been found inconstantly. The development of CM has been reported after a unilateral approach [66] as well as after a bilateral dissection [67, 68], and some authors also reported no case of CM after accessing the fourth ventricle through a bilateral telovelar route [69]. Among the other alternative technical suggestions that have been proposed to preserve the inferior vermis and deep medial cerebellar structures, Hermann et al. proposed a combined transventricular and supracerebellar approach to access fourth ventricular tumors. No instances of CM occurred postoperatively in their experience [70]. Apart from the limited number of children treated by the authors (the series consisted of four children), it should be mentioned that this surgical route should be considered only for tumors of the higher portion of the fourth ventricle; moreover, the semisitting position is required with potential hazards especially in young children and infants [70]. It should also be remembered that CM has been reported also after damage of the superior vermis, which might occur through this surgical route. A common thread in the abovementioned series is the suggestion to reduce as much as possible the retraction on the whole complex of midline and deep medial cerebellar structures, surrounding the fourth ventricle tumors. In this direction, El Beltagy et al. recently reported on the help that might be obtained by the use of high definition intraoperative ultrasonography. These authors prospectively operated on 60 children affected by the fourth ventricular tumors, 30 of them with the aid of intraoperative ultrasonography and 30 with a conventional microsurgical technique. Only 1 of the 30 children operated on with the help of high definition ultrasound (1/30 = 3.3 %) developed CM postoperatively, whereas 6 cases (6/30 = 30 %) developed this complication in the conventionally operated on group. This complication was also more severe and longer lasting in this last group. The authors concluded that the lower incidence and severity of CM among the ultrasound-aided surgery cases might be attributed to applying less retraction force on the cerebellar nuclei, vermis, and superior cerebellar peduncles during the exploration and surgical manipulation, thus

diminishing edema in these structures and preventing acute and permanent vascular insults [71]. In fact, postoperative edema is currently considered, the factor explaining the delayed onset of CM after surgery.

Metabolic (FDG PET/CT) [72] and DTI studies have recently confirmed that the regions other than the inferior vermis could be involved by surgical damage leading to the development of CM. Ojemann et al. acquired DTI images in 12 children operated on for a posterior fossa tumor. In all the patients who developed posterior fossa syndrome, the superior cerebellar peduncle could not be discerned on either side (5/12 = 41.6 %). In agreement with this finding, the superior cerebellar peduncle could be recognized in all the patients who did not develop this complication and in controls [52]. Previous studies seem to confirm these authors' findings [73]. Morris and colleagues describe a cohort of 26 patients, of whom 13 developed a posterior fossa syndrome. On a group level, those patients with bilateral dentate-thalamo-cortical pathway injury were predisposed to the development of CM [59]. This finding supports a model of disruption of the white matter bundles containing efferent axons within the superior cerebellar peduncle as a critical underlying pathophysiological component. It is worth noting that the location of the cerebellar tracts connecting the dentate to the thalamus is consistent with the known anatomical location of the superior cerebellar peduncle, sitting immediately adjacent to the lateral wall of the fourth ventricle. As a detail to be pointed out, the need for bilateral damage to the dentate nuclei areas and white matter tracts connecting to the thalamus as suggested by Ojemann et al. is actually not uniformly accepted.

In our experience, the involvement of the right dentate nucleus, either exclusively or more rarely in the context of bilateral invasion, was not constantly noticed in patients showing preoperative language impairment (PLI) and developing CM.

On the other hand, the location of the presumed damage corresponds to the prevalent location of the most frequent histological diagnoses related to the development of CM, namely medulloblastomas and midline cerebellar/brainstem astrocytomas which is documented in most series [50, 52, 74–77]. The significance of location is confirmed when considering different subtypes of midline astrocytomas. According to El Beltagy et al., children with pylomixoid variants have a higher tendency to develop CM if compared with pylocitic forms, and this could be explained by their higher rates of brain stem adhesion or invasion [71].

In our experience, seven out of eight children who developed CM presented a specific pattern of brainstem infiltration, with the involvement of the upper right quarter of the IV ventricle floor, eventually suggesting a disruption of the proximal efferent pathway of the cerebellum.

A more aggressive surgical strategy could also have contributed to the increase of the rate of CM in pediatric series reported in the last two decades [52]. Though areas of brainstem invasion are actually considered for removal only in the presence of preserved intraoperative neurophysiological parameters, no such physiological parameters exist for monitoring the deep medial cerebellar structures connected to the brainstem, areas actually dealt aggressively, especially when a pathology such as medulloblastoma or astrocytoma exists where the extent of resection is a predictor of long-term survival and/or cure.

In partial disagreement with direct surgical damage of deep medial cerebellar structures, Szathmari et al. [75] were not able to find any correlation between dentate nucleus involvement and CM. The only significant factor on postoperative MR images was represented by the evidence of cerebellar atrophy. They hence suggested as possible pathogenetic mechanism a surgical ischemic lesion and/or a preoperative compression of anatomic structures surrounding the tumor [78]. A vascular insult as a possible cause of CM was also suggested by Wells et al. These authors compared preoperative and postoperative MR images of 11 children who developed CM after surgical removal of a posterior fossa tumor with those of 18 cases that did not present this complication. Cerebellar edema was observed in 92 % of all the patients. There were trends for more middle and superior cerebellar peduncle edema ($p = 0.051$ and 0.074, respectively) in patients with CM compared with those without. A review of MR imaging performed 1 year after resection revealed significantly more atrophy of total cerebellum and vermis ($p < 0.01$) as well as more brainstem atrophy ($p < 0.05$) in affected patients. Finally, a review of MR imaging performed 1 year after resection revealed significantly more atrophy of total cerebellum and vermis ($p < 0.01$) as well as more brainstem atrophy ($p < 0.05$) in affected patients [79]. In transient forms, a vascular insult with vasogenic edema could explain why CM more frequently starts a few days after surgery and tends to spontaneously regress after a few weeks.

Hydrocephalus has also been hypothesized to be related to CM. Most of the data are however based on personal experiences and relatively small series [76, 80]. Larger series and multicenter studies have not confirmed this finding [11, 52, 78]. In our experience, the rate and severity of preoperative hydrocephalus was not significantly different in patients who were complicated by CM. Instead, the incidence of persistent hydrocephalus was significantly higher in children with CM (75 versus 25 % in patients without CM, $p < 0.05$). However, it is difficult to hypothesize a specific role of postoperative hydrocephalus, since it was managed by perioperative EVD in all cases. As previously hypothesized, persistent hydrocephalus could be considered a concomitant favoring factor or a consequence of the higher incidence of malignancies in the group of patients developing CM that is not directly related to the pathogenesis of CM. From a deeper consideration of the data present in the papers suggesting a role for ventricular

dilation, it appears that most probably, hydrocephalus could have exacerbated the severity of the mutism but acted as a concurrent pathogenetic factor together with a deep medial cerebellar surface and midline location of the tumors.

A further interesting factor contributing to the concept that location of the tumor has a primary role is raised by the analysis of preoperative neurocognitive evaluation data. In a relatively recent paper by our group, we were able to identify a selected group of patients who showed language impairment in the preoperative period (reduction of spontaneous language and or phonological disorders) predicting the development of CM after tumor removal; in fact 7 of the 11 children with preoperative language impairment (PLI) developed CM in the postoperative period, whereas none of those who did not show defective language function before surgery became mute after tumor removal. When we reviewed preoperative MR images, tumor location was deep and extended to the dentate nuclei in 90.9 % of the children with language impairment before surgery, a significantly higher percentage compared with children with normal language performances at admission (65.2 %). The right side and posterosuperior part of the dentate nuclei were more frequently involved by the tumor in children with abnormal results at the neurocognitive evaluation [74]. Though limited in number, the available preoperative DTI studies are in line with this finding, with invasion of the dentate-thalamic tract on tractography having been described as a predictive for the development of CM [52].

In conclusion, the midline location, the histotype of medulloblastoma, and the invasion of brainstem are the only factors consistently related with the onset of CM according to a recent systematic review of the literature [81].

Long-term prognosis and rehabilitation of children developing cerebellar mutism after posterior cranial fossa tumor removal

Over two thirds of the children who developed CM following tumor resection show long-term motor speech deficits [82]. However, although functional MR studies have located selective verbal functions in different regions of the cerebellum [31, 83, 84], reported findings are unspecific not allowing to foresee a different outcome for different selective language functions in children operated on for tumors located in different areas of the cerebellum. What is accepted is that long-term language impairment is substantially a motor impairment and depicted in the context of the so-called cerebellar ataxic dysarthria. This kind of disorder present two types of distinct features: (1) in the first type, the disorder is represented by an extreme form of pure dysarthria in the context of a preserved language perception and remaining neurocognitive functions in the normal range; these children have normal involuntary palatal, lip, and tongue movements, but none of them can imitate tongue or lip movement on command. (2) The second type is characterized by an apraxic language disorder, which is accompanied by a transient dysmetria of thought [85], namely these patients present procedural memory and praxic disorders, overall sustained by a global defective function of the cerebellum in recognition of sensory stimuli, and organization of operative schemes. The language of these children is not dysarthric but slow and monotonous, lacking normal prosody. Despite a normal tendency to communicate, they make use of a telegraphic language, often structured in the form of simple noun/verb sentences that frequently omit grammatical elements and often include uninflected verbs.

A number of factors have been proposed to influence the rate of recovery from these long-term consequences of CM. According to some authors, an earlier age (e.g., <5 years) would result in a worse outcome due to the disruption early in life of neural networks involved in speech and language organization. However, the role of early age and long-term outcome has not been confirmed by other authors. Huber et al. compared adults and young survivors of childhood (<16 years) onset of posterior fossa tumor and showed that dysarthria outcomes were strikingly similar, despite the age of onset and number of years after surgery at the time of the evaluation [86]. Recovery appeared to continue up to 1 year being followed by a substantial steady state. Our experience confirms these data; in particular, the number of years after surgery at last neurocognitive evaluation had no obvious impact, in our experience, on dysarthria outcome. Overall, neurocognitive evaluations in our series showed the absence of a clear association between Full Scale IQ (FSIQ) scores and language; additionally, no significant differences were found between Verbal IQ (VIQ) and Performance IQ (PIQ) scores, suggesting that the outcome in terms of speech function was not dependent on a prevalence of deficits on either of the two studied functions. On the contrary, both literature and personal findings demonstrate that the severity of associated neuropsychological deficits in fields as attention, memory, processing, speed imagery, and behavior in the immediate postoperative period significantly influence the severity of long-term dysarthric speech deficits [11], confirming that complete recovery is much more complex and usually incomplete in children affected by the second type of ataxic dysarthria.

Rehabilitation programs should be organized differently for the two previously described different types of long-term ataxic dysarthria. For the first type (the purely dysarthric disorder), rehabilitation should be based on a program of training aimed at the reorganization of words output schemes, combined with exercises of coordination and sensorimotor integration. For the second type (the one featured by a combined procedural memory and defective neurocognitive functions), the objective of the rehabilitation program should aim to strengthening the child phonological awareness, sounds representation, and their sequences planning; the rehabilitation

Table 1 Synopsis of patients with preoperative language impairment

Gender	Age	Pathology	Tumor site	Brainstem infiltration	Dentate nucleus involvement	Approach	Vermis split	Preoperative hydrocephalus	Postoperative hydrocephalus	Immediate postoperative outcome of PLI	Long-term outcome
M	3	MB	IV v	Y	Yes (right)	Suboccipital	N	Y	Y	CM	Dysarthria
M	6	PA	R hemisphere	N	Yes (right)	Suboccipital	N	Y	Y	Improved	Normal
M	10	PA	IV v	Y	Yes (right)	Suboccipital	N	Y	Y	CM	Phonological impairment
M	8	MB	IV v-L CPA	Y	Yes (bilateral)	Suboccipital	N	Y	Y	CM	Dysarthria
F	2	PA	Vermis	N	Yes (bilateral)	Suboccipital	N	Y	N	Improved	Normal
F	11	PA	L cerebellar peduncle	N	Yes (left)	Suboccipital	N	N	N	Stable	Normal
F	9	PA	Vermis	Y	Yes (bilateral)	Suboccipital	Y	Y	Y	CM	Phonological impairment
F	10	MB	IV v-R CPA	Y	Yes (right)	Suboccipital	N	Y	Y	CM	Phonological impairment
F	7	PA	Vermis-R hemisphere	N	Yes (right)	Suboccipital	N	Y	N	Improved	Normal
M	6	MB	IV v	N	Yes (right)	Suboccipital	N	Y	Y	CM	Dysarthria
F	1	E III	Vermis	Y	N	Suboccipital	N	Y	N	CM	Phonological impairment
M	5	MB	IV v	N	N	Suboccipital	N	Y	N	Improved	
M	8	MB	IV v	N	Yes (right)	Suboccipital	Y	Y	N	CM	Dysarthria
M	7	PA	R hemisphere	N	N	Suboccipital	N	N	N	Improved	Normal
F	4	PA	Vermis-L hemisphere	N	Yes (left)	Suboccipital	N	Y	Y	Improved	Normal

PA pilocytic astrocytoma, *MB* medulloblastoma, *E III* anaplastic ependymoma, *R* right, *L* left, *CM* cerebellar mutism, *PLI* preoperative language impairment

should hence include an auditory and visual attention stimuli program [87].

Institutional experience

Children with posterior cranial fossa tumors admitted to the Paediatric Neurosurgical Department, A. Gemelli Hospital, Rome, Italy, in the period 2006–2013 were studied in order to identify risk factors involved in the development of CM. Each patient received neurological examination, brain magnetic resonance imaging (MRI) studies, and a neuropsychological, neuromotor, and behavioral assessment at the admission and during the postoperative period, prior to chemotherapy or radiotherapy in order to rule out the effects of adjuvant treatments, as described in a previous paper [74]. Children less than 24 months or subjects showing severe neurological impairment or abnormal emotional status in the preoperative or postoperative period, which prevented a reliable language assessment, were excluded from the study. Overall, we studied 48 children (26 M/22 F). The mean age at diagnosis was 7.92 years (median 7.5 years) with a range of 2–17 years. Location of the tumor was midline in 30 cases (62.4 %), midline with lateral extension, and exclusively lateral in 9 cases each (18.8 %). MRI showed brainstem compression in 37 children (77.1 %) and dentate nucleus involvement in 35 cases (72.9 %) (Table 1).

All children underwent the surgical resection of the tumor through a median suboccipital craniotomy except 3 cases operated on through a paramedian approach. Splitting of the inferior cerebellar vermis was performed in 15 cases (31.3 %). Intraoperative evidence of brainstem infiltration was noticed in 21 children (43.7 %). Gross total tumor resection was achieved in 33 children (68.8 %), while a minimal residual tumor hardly adherent to the brainstem was left in place in the remaining 15 patients.

Pilocytic astrocytoma was the most common histological diagnosis (25 cases, 52.1 %), followed by medulloblastoma (20 cases, 41.7 %) and ependymoma (3 cases, 6.2 %). Preoperative hydrocephalus was present in 38 cases (79.2 %), with moderate to severe ventricular dilation in most of the cases (78.9 %). A concomitant external ventricular drain (EVD) was inserted during tumor resection in 33 children. Hydrocephalus persisted postoperatively in 16 children (42.1 % of patients with preoperative hydrocephalus), who underwent endoscopic third ventriculostomy (ETV). Two patients finally required further treatment of hydrocephalus. CSF infection occurred in 4 patients (8.3 %), who were treated with intravenous antibiotic therapy.

The preoperative neuropsychological evaluation identified a preoperative language impairment (PLI) in 15 cases (31.3 %). This specific neurolinguistic disorder consists of variable association of impaired non-motor cognitive and neurolinguistic processes that were largely described in a previous paper [74]. Indeed, PLI was characterized by persistently reduced spontaneous language and by a decreased mean length of utterance, as impairment of words and morphemes production. The word finding abilities and the capacity to generate words according to a given a phonological rule were also impaired. Additionally, impairment of imagery was constantly noticed, thus configuring a sort of *palsy of thought*.

After surgery, 8 out of 48 children developed CM (16.7 %), all of them showing PLI. Thus, CM did not occur in any patient without PLI and occurred in 53.3 % of patients with PLI. Among patients with PLI who were not complicated by CM, this disorder improved in 6 cases and remained stable in the remaining case after surgery. No statistically significant difference were noticed in the main demographic, radiological, and surgical features of patients suffering from CM except for infiltration of the brainstem ($p < 0.05$) and persistent hydrocephalus.

In all cases of CM, a recovery of speech production was observed, with a latency period largely ranging from 4 days to 6 months. Four children suffered from persistent dysarthria, while the remaining 4 children presented phonological disorders. In conclusion, speech rarely normalizes completely after CM [88].

Concerning the long-term outcome (mean follow-up 5.3 years), 4/15 patients presenting with preoperative language impairment presented a persistent ataxic dysarthria (in all cases of type 1, namely a pure dysarthric disorder). All of these patients harbored medulloblastomas and had developed CM in the immediate postoperative period. Brainstem infiltration (present in 2/4 cases) and involvement of the right dentate nucleus (present in 3/4 cases) were the only two related risk factors.

The remaining 11 children all showed a good recovery of their language impairment, that was complete in children not showing postoperative CM. Recovery occurred at a mean time distance of 8.6 months from surgery (min. 3.2 months, max. 14.5 months), no further improvement being observed beyond 15 months. Pathological diagnosis of pylocitic astrocytoma (8/11 cases) and the absence of brainstem infiltration (no brainstem infiltration in 7/11 cases) were the two most favoring factors for the long-term disappearance of the language defects; needless to say the benign histology was the premise for a more intense rehabilitation program which might have had a relevant role for the recovery of these children (Table 1).

Conclusions

Cerebellar mutism which manifests after surgery for a posterior fossa tumor is a more complex phenomenon than originally considered. In fact, beyond being a phenomenon which manifests in the early postoperative period, cerebellar mutism

might be predicted based not only on MR features of the tumor at diagnosis but also by a preoperative neurocognitive evaluation. Tumor infiltration of the brainstem and preoperative language impairment represent in this context the most relevant predictive features. Children developing cerebellar mutism also tend to present, at least partially, long-term persistence of phonological disorders, which manifests in the form of an ataxic dysarthria in most cases. This tends to be more severe and less prone to recovery in children presenting at diagnosis with associated combined procedural memory and defective neurocognitive functions.

Conflict of interest The authors declare that this paper has not been submitted elsewhere. The authors have not received financial assistance or inducement and declare that they have no known conflicts of interest to disclose.

References

1 Jurgens U (2002) Neural pathways underlying vocal control. Neurosci Biobehav Rev 26:235–258
2 Gudrunardottir T, Sehested A, Juhler M, Schmiegelow K (2011) Cerebellar mutism: review of the literature. Childs Nerv Syst 27: 355–363
3 Pitsika M, Tsitouras V (2013) Cerebellar mutism. J Neurosurg Pediatr 12:604–614
4 Nagatani K, Waga S, Nakagawa Y (1991) Mutism after removal of a vermian medulloblastoma: cerebellar mutism. Surg Neurol 36:307–309
5 van Mourik M, Catsman-Berrevoets CE, van Dongen HR, Neville BG (1997) Complex orofacial movements and the disappearance of cerebellar mutism: report of five cases. Dev Med Child Neurol 39: 686–690
6 Dailey AT, McKhann 2nd GM, Berger MS (1995) The pathophysiology of oral pharyngeal apraxia and mutism following posterior fossa tumor resection in children. J Neurosurg 83:467–475
7 Gudrunardottir T, Sehested A, Juhler M, Grill J, Schmiegelow K (2011) Cerebellar mutism: definitions, classification and grading of symptoms. Childs Nerv Syst 27:1361–1363
8 Schmahmann JD, Sherman JC (1998) The cerebellar cognitive affective syndrome. Brain 121(Pt 4):561–579
9 De Smet HJ, Paquier P, Verhoeven J, Marien P (2013) The cerebellum: its role in language and related cognitive and affective functions. Brain Lang 127:334–342
10 Thomale UW, Driever PH (2013) Inconsistent terminology for cerebellar mutism. Childs Nerv Syst 29:717–718
11 Robertson PL, Muraszko KM, Holmes EJ, Sposto R, Packer RJ, Gajjar A, Dias MS, Allen JC (2006) Incidence and severity of postoperative cerebellar mutism syndrome in children with medulloblastoma: a prospective study by the Children's Oncology Group. J Neurosurg 105:444–451
12 van Baarsen KM, Grotenhuis JA (2014) The anatomical substrate of cerebellar mutism. Med Hypotheses 82:774–780
13 Marien P, Ackermann H, Adamaszek M, Barwood CH, Beaton A, Desmond J, De Witte E, Fawcett AJ, Hertrich I, Kuper M, Leggio M, Marvel C, Molinari M, Murdoch BE, Nicolson RI, Schmahmann JD, Stoodley CJ, Thurling M, Timmann D, Wouters E, Ziegler W (2014) Consensus paper: language and the cerebellum: an ongoing enigma. Cerebellum 13:386–410
14 Larson CR, Sutton D, Lindeman RC (1978) Cerebellar regulation of phonation in rhesus monkey (Macaca mulatta). Exp Brain Res 33:1–18
15 Tolbert DL, Bantli H, Bloedel JR (1978) Organizational features of the cat and monkey cerebellar nucleocortical projection. J Comp Neurol 182:39–56
16 Al-Afif S, Staden M, Krauss JK, Schwabe K, Hermann EJ (2013) Splitting of the cerebellar vermis in juvenile rats—effects on social behavior, vocalization and motor activity. Behav Brain Res 250: 293–298
17 Allen G, Buxton RB, Wong EC, Courchesne E (1997) Attentional activation of the cerebellum independent of motor involvement. Science 275:1940–1943
18 Di Rocco C (1999) Cerebellar astrocytomas. In: Choux M, Di Rocco C, Hokley AD, Walker ML (eds) Pediatric neurosurgery. Churchill Livingstone, London, pp. 427–460
19 Ersahin Y (1998) Is splitting of the vermis responsible for cerebellar mutism? Pediatr Neurosurg 28:328
20 Ackermann H, Vogel M, Petersen D, Poremba M (1992) Speech deficits in ischaemic cerebellar lesions. J Neurol 239:223–227
21 Frassanito P, Massimi L, Caldarelli M, Di Rocco C (2009) Cerebellar mutism after spontaneous intratumoral bleeding involving the upper cerebellar vermis: a contribution to the physiopathogenic interpretation. Childs Nerv Syst 25:7–11
22 Schmahmann JD (1991) An emerging concept. The cerebellar contribution to higher function. Arch Neurol 48:1178–1187
23 Stoodley CJ, Schmahmann JD (2010) Evidence for topographic organization in the cerebellum of motor control versus cognitive and affective processing. Cortex 46:831–844
24 Courchesne E (1991) Neuroanatomic imaging in autism. Pediatrics 87:781–790
25 Tavano A, Grasso R, Gagliardi C, Triulzi F, Bresolin N, Fabbro F, Borgatti R (2007) Disorders of cognitive and affective development in cerebellar malformations. Brain 130:2646–2660
26 Hodge SM, Makris N, Kennedy DN, Caviness Jr VS, Howard J, McGrath L, Steele S, Frazier JA, Tager-Flusberg H, Harris GJ (2010) Cerebellum, language, and cognition in autism and specific language impairment. J Autism Dev Disord 40:300–316
27 Tasdemiroglu E, Kaya M, Yildirim CH, Firat L (2011) Postoperative cerebellar mutism and autistic spectrum disorder. Childs Nerv Syst 27:869–878
28 Misciagna S, Iuvone L, Mariotti P, Silveri MC (2010) Verbal short-term memory and cerebellum: evidence from a patient with congenital cerebellar vermis hypoplasia. Neurocase 16:119–124
29 Lechtenberg R, Gilman S (1978) Speech disorders in cerebellar disease. Ann Neurol 3:285–290
30 Amarenco P, Chevrie-Muller C, Roullet E, Bousser MG (1991) Paravermal infarct and isolated cerebellar dysarthria. Ann Neurol 30:211–213
31 Urban PP, Marx J, Hunsche S, Gawehn J, Vucurevic G, Wicht S, Massinger C, Stoeter P, Hopf HC (2003) Cerebellar speech representation: lesion topography in dysarthria as derived from cerebellar ischemia and functional magnetic resonance imaging. Arch Neurol 60:965–972
32 Jansen A, Floel A, Van Randenborgh J, Konrad C, Rotte M, Forster AF, Deppe M, Knecht S (2005) Crossed cerebro-cerebellar language dominance. Hum Brain Mapp 24:165–172
33 Wildgruber D, Ackermann H, Grodd W (2001) Differential contributions of motor cortex, basal ganglia, and cerebellum to speech motor control: effects of syllable repetition rate evaluated by fMRI. NeuroImage 13:101–109
34 Mendez Orellana C, Visch-Brink E, Vernooij M, Kalloe S, Satoer D, Vincent A, van der Lugt A, Smits M (2014) Crossed cerebrocerebellar language lateralization: an additional diagnostic feature for assessing atypical language representation in presurgical functional MR imaging. AJNR Am J Neuroradiol

35 Marien P, Engelborghs S, Fabbro F, De Deyn PP (2001) The lateralized linguistic cerebellum: a review and a new hypothesis. Brain Lang 79:580–600

36 Herholz K, Pietrzyk U, Karbe H, Wurker M, Wienhard K, Heiss WD (1994) Individual metabolic anatomy of repeating words demonstrated by MRI-guided positron emission tomography. Neurosci Lett 182:47–50

37 Hirano S, Kojima H, Naito Y, Honjo I, Kamoto Y, Okazawa H, Ishizu K, Yonekura Y, Nagahama Y, Fukuyama H, Konishi J (1996) Cortical speech processing mechanisms while vocalizing visually presented languages. Neuroreport 8:363–367

38 Paus T, Perry DW, Zatorre RJ, Worsley KJ, Evans AC (1996) Modulation of cerebral blood flow in the human auditory cortex during speech: role of motor-to-sensory discharges. Eur J Neurosci 8:2236–2246

39 Perry DW, Zatorre RJ, Petrides M, Alivisatos B, Meyer E, Evans AC (1999) Localization of cerebral activity during simple singing. Neuroreport 10:3979–3984

40 Price CJ, Wise RJ, Warburton EA, Moore CJ, Howard D, Patterson K, Frackowiak RS, Friston KJ (1996) Hearing and saying. The functional neuro-anatomy of auditory word processing. Brain 119(Pt 3):919–931

41 Stoodley CJ, Schmahmann JD (2009) Functional topography in the human cerebellum: a meta-analysis of neuroimaging studies. NeuroImage 44:489–501

42 Baillieux H, De Smet HJ, Dobbeleir A, Paquier PF, De Deyn PP, Marien P (2010) Cognitive and affective disturbances following focal cerebellar damage in adults: a neuropsychological and SPECT study. Cortex 46:869–879

43 Riva D, Giorgi C (2000) The cerebellum contributes to higher functions during development: evidence from a series of children surgically treated for posterior fossa tumours. Brain 123(Pt 5):1051–1061

44 Fraioli B, Guidetti (1975) Effects of stereotactic lesions of the dentate nucleus of the cerebellum in man. Appl Neurophysiol 38:81–90

45 Carpenter MB (1976) Human neuroanatomy. Williams & Wilkins, Baltimore

46 Akakin A, Peris-Celda M, Kilic T, Seker A, Gutierrez-Martin A, Rhoton Jr A (2014) The dentate nucleus and its projection system in the human cerebellum: the dentate nucleus microsurgical anatomical study. Neurosurgery 74:401–424 discussion 424-405

47 Frim DM, Ogilvy CS (1995) Mutism and cerebellar dysarthria after brain stem surgery: case report. Neurosurgery 36:854–857

48 Dum RP, Li C, Strick PL (2002) Motor and nonmotor domains in the monkey dentate. Ann N Y Acad Sci 978:289–301

49 Dum RP, Strick PL (2003) An unfolded map of the cerebellar dentate nucleus and its projections to the cerebral cortex. J Neurophysiol 89:634–639

50 McMillan HJ, Keene DL, Matzinger MA, Vassilyadi M, Nzau M, Ventureyra EC (2009) Brainstem compression: a predictor of postoperative cerebellar mutism. Childs Nerv Syst 25:677–681

51 Soelva V, Hernaiz Driever P, Abbushi A, Rueckriegel S, Bruhn H, Eisner W, Thomale UW (2013) Fronto-cerebellar fiber tractography in pediatric patients following posterior fossa tumor surgery. Childs Nerv Syst 29:597–607

52 Ojemann JG, Partridge SC, Poliakov AV, Niazi TN, Shaw DW, Ishak GE, Lee A, Browd SR, Geyer JR, Ellenbogen RG (2013) Diffusion tensor imaging of the superior cerebellar peduncle identifies patients with posterior fossa syndrome. Childs Nerv Syst 29:2071–2077

53 Ildan F, Tuna M, Erman T, Gocer AI, Zeren M, Cetinalp E (2002) The evaluation and comparison of cerebellar mutism in children and adults after posterior fossa surgery: report of two adult cases and review of the literature. Acta Neurochir 144:463–473

54 Avula S, Kumar R, Pizer B, Pettorini B, Abernethy L, Garlick D, Mallucci C (2015) Diffusion abnormalities on intraoperative magnetic resonance imaging as an early predictor for the risk of posterior fossa syndrome. Neuro-Oncology 17:614–622

55 Dietze Jr DD, Mickle JP (1990) Cerebellar mutism after posterior fossa surgery. Pediatr Neurosurg 16:25–31 discussion 31

56 Ferrante L, Mastronardi L, Acqui M, Fortuna A (1990) Mutism after posterior fossa surgery in children. Report of three cases. J Neurosurg 72:959–963

57 Cakir Y, Karakisi D, Kocanaogullari O (1994) Cerebellar mutism in an adult: case report. Surg Neurol 41:342–344

58 Ammirati M, Mirzai S, Samii M (1989) Transient mutism following removal of a cerebellar tumor. A case report and review of the literature. Childs Nerv Syst 5:12–14

59 Morris EB, Phillips NS, Laningham FH, Patay Z, Gajjar A, Wallace D, Boop F, Sanford R, Ness KK, Ogg RJ (2009) Proximal dentatothalamocortical tract involvement in posterior fossa syndrome. Brain 132:3087–3095

60 Crutchfield JS, Sawaya R, Meyers CA, Moore 3rd BD (1994) Postoperative mutism in neurosurgery. Report of two cases. J Neurosurg 81:115–121

61 Ersahin Y, Mutluer S, Saydam S, Barcin E (1997) Cerebellar mutism: report of two unusual cases and review of the literature. Clin Neurol Neurosurg 99:130–134

62 Mussi AC, Rhoton Jr AL (2000) Telovelar approach to the fourth ventricle: microsurgical anatomy. J Neurosurg 92:812–823

63 Kellogg JX, Piatt Jr JH (1997) Resection of fourth ventricle tumors without splitting the vermis: the cerebellomedullary fissure approach. Pediatr Neurosurg 27:28–33

64 El-Bahy K (2005) Telovelar approach to the fourth ventricle: operative findings and results in 16 cases. Acta Neurochir 147:137–142 discussion 142

65 von Hoff K, Kieffer V, Habrand JL, Kalifa C, Dellatolas G, Grill J (2008) Impairment of intellectual functions after surgery and posterior fossa irradiation in children with ependymoma is related to age and neurologic complications. BMC Cancer 8:15

66 Zaheer SN, Wood M (2010) Experiences with the telovelar approach to fourth ventricular tumors in children. Pediatr Neurosurg 46:340–343

67 Matsushima T, Abe H, Kawashima M, Inoue T (2012) Exposure of the wide interior of the fourth ventricle without splitting the vermis: importance of cutting procedures for the tela choroidea. Neurosurg Rev 35:563–571 discussion 571-562

68 Han S, Wang Z, Wang Y, Wu A (2013) Transcerebellomedullary fissure approach to lesions of the fourth ventricle: less is more? Acta Neurochir 155:1011–1016

69 Tomasello F, Conti A, Cardali S, La Torre D, Angileri FF (2015) Telovelar approach to fourth ventricle tumors: highlights and limitations. World Neurosurg 83:1141–1147

70 Hermann EJ, Rittierodt M, Krauss JK (2008) Combined transventricular and supracerebellar infratentorial approach preserving the vermis in giant pediatric posterior fossa midline tumors. Neurosurgery 63:ONS30–ONS35 discussion ONS35-37

71 El Beltagy MA, Atteya MM (2013) The benefits of navigated intraoperative ultrasonography during resection of fourth ventricular tumors in children. Childs Nerv Syst

72 Gedik GK, Sari O, Koktekir E, Akdemir G (2014) Fluorodeoxyglucose positron emission tomography/computed tomography findings in a patient with cerebellar mutism after operation in posterior fossa. Asian J Surg. doi:10.1016/j.asjsur.2014.01.004

73 Kusano Y, Tanaka Y, Takasuna H, Wada N, Tada T, Kakizawa Y, Hongo K (2006) Transient cerebellar mutism caused by bilateral damage to the dentate nuclei after the second posterior fossa surgery. Case report. J Neurosurg 104:329–331

74 Di Rocco C, Chieffo D, Frassanito P, Caldarelli M, Massimi L, Tamburrini G (2011) Heralding cerebellar mutism: evidence for

pre-surgical language impairment as primary risk factor in posterior fossa surgery. Cerebellum 10:551–562

75 Korah MP, Esiashvili N, Mazewski CM, Hudgins RJ, Tighiouart M, Janss AJ, Schwaibold FP, Crocker IR, Curran Jr WJ, Marcus Jr RB (2010) Incidence, risks, and sequelae of posterior fossa syndrome in pediatric medulloblastoma. Int J Radiat Oncol Biol Phys 77:106–112

76 Catsman-Berrevoets CE, Van Dongen HR, Mulder PG, Paz y Geuze D, Paquier PF, Lequin MH (1999) Tumour type and size are high risk factors for the syndrome of "cerebellar" mutism and subsequent dysarthria. J Neurol Neurosurg Psychiatry 67:755–757

77 Dubey A, Sung WS, Shaya M, Patwardhan R, Willis B, Smith D, Nanda A (2009) Complications of posterior cranial fossa surgery—an institutional experience of 500 patients. Surg Neurol 72:369–375

78 Szathmari A, Thiesse P, Galand-desme S, Mottolese C, Bret P, Jouanneau E, Guyotat J, Lion-Francois L, Frappaz D (2010) Correlation between pre- or postoperative MRI findings and cerebellar sequelae in patients with medulloblastomas. Pediatr Blood Cancer 55:1310–1316

79 Wells EM, Walsh KS, Khademian ZP, Keating RF, Packer RJ (2008) The cerebellar mutism syndrome and its relation to cerebellar cognitive function and the cerebellar cognitive affective disorder. Dev Disabil Res Rev 14:221–228

80 Turgut M (1998) Transient "cerebellar" mutism. Childs Nerv Syst 14:161–166

81 Reed-Berendt R, Phillips B, Picton S, Chumas P, Warren D, Livingston JH, Hughes E, Morrall MC (2014) Cause and outcome of cerebellar mutism: evidence from a systematic review. Childs Nerv Syst 30:375–385

82 Morgan AT, Liegeois F, Liederkerke C, Vogel AP, Hayward R, Harkness W, Chong K, Vargha-Khadem F (2011) Role of cerebellum in fine speech control in childhood: persistent dysarthria after surgical treatment for posterior fossa tumour. Brain Lang 117:69–76

83 Schmahmann JD, Pandya DN (2008) Disconnection syndromes of basal ganglia, thalamus, and cerebrocerebellar systems. Cortex 44:1037–1066

84 Ackermann H, Mathiak K, Riecker A (2007) The contribution of the cerebellum to speech production and speech perception: clinical and functional imaging data. Cerebellum 6:202–213

85 Schmahmann JD (1998) Dysmetria of thought: clinical consequences of cerebellar dysfunction on cognition and affect. Trends Cogn Sci 2:362–371

86 Huber JF, Bradley K, Spiegler B, Dennis M (2007) Long-term neuromotor speech deficits in survivors of childhood posterior fossa tumors: effects of tumor type, radiation, age at diagnosis, and survival years. J Child Neurol 22:848–854

87 Walker D, Thomas SA, Talbot EJ, Bennett EJ, Starza-Smith A, Da Silva SL (2014) Cerebellar mutism: the rehabilitation challenge in pediatric neuro-oncology: case studies. J Pediatr Rehabil Med 7:333–340

88 Steinbok P, Cochrane DD, Perrin R, Price A (2003) Mutism after posterior fossa tumour resection in children: incomplete recovery on long-term follow-up. Pediatr Neurosurg 39:179–183