

■ Critical Review of the Evidence for Residual Long-Term Speech Deficits Following Transient Cerebellar Mutism in Childhood

■ Revue critique des données probantes sur les troubles résiduels à long terme de la parole à la suite d'un mutisme ischémique transitoire durant l'enfance

Rebecca Hisson

Scott G. Adams

Abstract

This brief report critically examines the available evidence for residual long-term speech deficits following transient cerebellar mutism in childhood. Study designs include: parental surveys and retrospective chart reviews, between group comparison designs, and case studies (3). Overall, the research supports the presence of residual speech deficits (articulation, fluency, phonology, rate of speech, and dysarthria) in many individuals who underwent surgery and recovered from mutism. The results of the present report should be interpreted with consideration of the inherent limitations of the methodology used in the reviewed studies.

Abrégé

Le présent rapport sommaire examine de façon critique les données probantes disponibles sur les troubles résiduels à long terme de la parole suivant un mutisme ischémique transitoire durant l'enfance. Cette étude se fonde sur des enquêtes auprès des parents, l'examen rétrospectif de dossiers, des comparaisons entre groupes et des études de cas (3). Dans l'ensemble, la recherche corrobore la présence de troubles résiduels de la parole (articulation, fluidité, phonologie, débit de la parole et dysarthrie) chez bon nombre de personnes ayant subi une chirurgie et surmonté leur mutisme. Il faut interpréter les résultats contenus dans le présent rapport en tenant compte des limites inhérentes de la méthode employée par les études examinées.

Key words: cerebellar mutism, speech, cerebellar tumors

The primary function of the cerebellum is to coordinate the timing and force of muscular contractions so that skilled, voluntary movements are appropriate for an intended task. The cerebellum processes sensory information from all over the body and integrates that information into the execution of a movement. There are three major neural pathways that are involved in cerebellar function and connect the cerebellum to other regions of the nervous system. These include the inferior, middle and superior peduncles. The inferior peduncle transmits sensory information (i.e. joint position, muscle contraction, tendon stretch, vestibular information, etc.) from the entire body to the cerebellum, helps to monitor the timing and force of movements, and determines if muscle contractions are achieving the intended results. The middle peduncle transmits preliminary information regarding the plan of the intended movement from the cerebral cortex to the cerebellum where it can be integrated with incoming sensory information to modify and refine the intended plan of movement. The superior peduncle transmits the processed and refined information about the intended motor plan from the cerebellum to the motor areas of the cortex. This information from the superior cerebellar peduncle is then used to modify the

Rebecca Hisson, M.Cl.Sc.
(S-LP)
School of Communication
Sciences and Disorders
University of Western
Ontario
London, Ontario Canada

Scott G. Adams, PhD
School of Communication
Sciences and Disorders
University of Western
Ontario
London, Ontario Canada

final motor outflow from the motor cortex.

Cerebellar damage may be caused by stroke, tumour resection, alcohol abuse, head injury, or exposure to chemicals such as toluene and phenytoin. Cerebellar lesions may result in a dysarthria affecting the ability to coordinate the movements of the tongue, lips, and palate, as well as the synchronization of respiration and phonation (Gordon, 1996). The most typical speech pattern resulting from cerebellar lesions is that of ataxic dysarthria which is characterized by imprecise consonants, excess and equal stress (scanning speech), irregular articulation breakdown, vowel distortions, harsh voice, phoneme and interval prolongation, monopitch and monoloudness, and slow speech rate (Darley, Aronson & Brown, 1969). Clinicoanatomic studies of ataxic dysarthria have produced quite variable results. A recent review of focal cerebellar lesions found that ataxic dysarthria was associated with lesions of the vermal, paravermal, and lateral aspects of the cerebellum (Kent, Duffy, Slama, Kent & Clift, 2001). In a subsequent review, it was concluded that clinical data do not yet provide a coherent picture on the topographic correlates of cerebellar dysarthria (Ackermann, Mathiak, & Riecker, 2007).

In addition to the characteristics of ataxic dysarthria stated above, some individuals develop transient cerebellar mutism (TCM), also known as posterior fossa syndrome or cerebellar syndrome, following surgery to remove cerebellar tumours. This transient mutism, which is a state of speechlessness in a conscious patient, occurs in 7.5% to 29% of patients following tumour resection and is more common in children than adults (Huber, Bradley, Speigler & Dennis, 2006; Pollack, Polinko, Albright, Towbin, Fitz, Hoffman, & Schut, 1995; Dailey, McKhann, & Berger, 1995; Van Mourik, Catsman-Berrevoets, Yousef-Bak, Paquier & Van Dongen, 1998; Catsman-Berrevoets, Van Dongen, Mulder, Paz y Geuze, Paquier & Lequin, 1999). The posterior fossa is the most common site for brain tumours in children, accounting for about a half of all childhood central nervous system tumours (Breen, Kehagioglou, Usher, & Plowman, 2004). Cerebellar astrocytoma and medulloblastoma tumours together account for approximately one third of all childhood brain tumours (Huber et al., 2006) with medulloblastoma being the most common malignant childhood brain tumour (Ray et al. (2004). Five and 10-year survival rates for children diagnosed with medulloblastoma are reported to be 59% and 49% respectively (Ray et al., 2004).

The mechanism and structures involved in the development of cerebellar mutism are poorly understood (Duffy, 2005) but it has been suggested that mutism may result from an interruption of the pathway that connects the cerebellum to the supplementary motor cortex (Germano et al., 1998). A modified version of this hypothesis has been recently presented by Ackermann and colleagues (Ackermann et al., 2007). In their 'cerebello-cerebral diaschisis' hypothesis, Ackermann et al., (2007) suggest that posterior fossa cerebellar lesions, due to tumor surgery, cause a disruption in the normal cerebellar-

to-cortical interactions and give rise to a 'functional suppression' of mesofrontal cortical structures that are believed to be required for the initiation of speech. This hypothesis suggests that mutism is caused by remote effects (diaschisis) on the frontal cortex rather than local effects on the cerebellum. In this hypothesis, local effects on the cerebellum would be linked to ataxic dysarthria but not mutism (Ackermann, et al., 2007). Thus, this hypothesis suggests that there is an anatomical and a functional dissociation between cerebellar mutism and ataxic dysarthria.

In children who develop transient cerebellar mutism following tumour resection, well-preserved speech is observed for 24 hours up to 6 days, after which time mutism begins and lasts anywhere from several days to several months, or even years (Huber et al., 2006; van Dongen, Catsman-Berrevoets, & van Mourik, 1994; Steinbok, Cochrane, Perrin, & Price, 2003; Doxey, Bruce, Sklar, Swift, & Shapiro, 1999). As speech begins to re-emerge following the period of mutism, children often present with the characteristics of ataxic dysarthria discussed above. Therefore, the term 'mutism with subsequent dysarthria' has been used in the literature to refer to the process of the recovering cerebellar mechanism (van Dongen et al., 1994). The nature of cerebellar recovery following tumour resection is not fully understood. Some researchers describe the mutism as resolving completely into normal speech (van Dongen et al., 1994; ReKate, Grubb, Aram, Hahn, & Ratcheson, 1985; Riva & Giorgi, 2000; Pollack, 1997). Other researchers suggest that speech never fully recovers (Hudson, Murdoch & Ozanne, 1989; Huber et al., 2006; Huber-Okraïnec, Dennis, Bradley, & Spiegler, 2001; Steinbok et al., 2003). This discrepancy in the literature has been noted by researchers and some have gone so far as to suggest that "One might reasonably conclude from a review of the literature that 'cerebellar mutism' is a distressing, but transient and ultimately benign problem" (Steinbok et al., 2003, p. 180).

Objectives

The primary objective of this paper is to critically evaluate the existing literature regarding the evidence for residual long-term speech deficits following transient cerebellar mutism in childhood. The second objective is to provide evidence-based recommendations for future research.

Method

Search Strategy

Computerized databases, including Commdis Dome, CINAHL, Pubmed and Medline, were searched using the following search strategy:

((transient cerebellar mutism) OR (posterior fossa syndrome) OR (cerebellar syndrome) OR (mutism)) AND (dysarthria) AND ((tumour resection) OR (tumor resection)).

The reference lists of the articles found were also searched for relevant papers and the search was limited to articles written in English.

Search Criteria

Studies selected for inclusion in this review were required to investigate speech characteristics, involve individuals who had cerebellar tumours resected in childhood, and who developed a transient period of mutism as a result of the cerebellar lesion. In order to fulfill the requirement of 'long-term' deficits, studies were also required to have participants who had received surgery at least 2 years prior.

Data Collection

Results of the literature search yielded the following study types: parental surveys and retrospective chart reviews, between group comparison designs, and case studies (3).

Results

In their 2006 study, Huber, Bradley, Speigler and Dennis investigated the presence of residual motor speech deficits in six survivors (mean survival years was 10.78) of childhood cerebellar tumour resection who develop transient cerebellar mutism (TCM) as a result. These individuals were then compared to six individuals with cerebellar tumours who did not develop postoperative TCM and six healthy individuals in order to determine whether deficits were greater in those individuals who developed TCM than in the other two groups. Subjects were videotaped while providing a narrative in response to a picture book. Two speech-language pathologists (S-LPs) independently analyzed the videotaped narratives to determine percent dysfluencies (types of dysfluencies included in the count were blocks, prolongations, part-word repetitions, word and phrase repetitions, interjections, and phrase revisions), rate of speech and the presence of ataxic dysarthria using the Dysarthria Rating Scale. Results showed that individuals who developed TCM postoperatively were significantly more dysarthric and had slower rates of speech than either the healthy controls or the individuals who did not develop TCM postoperatively. The results also showed that patients who developed TCM following tumour resection were significantly more dysfluent than healthy controls, but were not significantly different from patients who did not develop TCM following tumour resection.

The 2003 study by Steinbok, Cochrane, Perrin, and Price used parental reports and retrospective chart reviews to determine the long-term neurological and speech outcomes of seven patients who developed TCM following cerebellar tumour resection in childhood. Subjects were identified through a search of a hospital's database and medical records were reviewed and parents were contacted to determine their child's most recent speech and neurological status. Reports from parents and chart reviews indicated that at time of follow-up (between 2.5

and 13.1 years post-surgery) one child remained mute, one child's speech returned to normal, three children had speech that was reported to be slower than normal, and two children were reported to slur their speech.

In 1989, Hudson, Murdoch, and Ozanne looked at the presence of articulation, phonological, phonetic, and motor speech deficits in two individuals who had undergone surgery in childhood for the removal of a cerebellar tumour and developed TCM postoperatively. Subjects completed the Fisher-Logemann Test of Articulation Competence, and the Khan and Lewis procedure was used to analyze the results phonetically. Participants also completed the Frenchay Dysarthria Assessment and provided an audio recorded connected speech sample which two independent S-LPs used to determine the presence or absence of the ten most prominent features of ataxic dysarthria as defined by Darley, Aronson, and Brown (1969). The first individual, who was 6 years post-operative, was described as presenting with a number of inconsistent phoneme productions, the retention of phonological processes, a mixed ataxic-flaccid dysarthria, left facial palsy, lack of volume control and 5 of the 10 ataxic dysarthria characteristics, including imprecise consonants, excess and equal stress, irregular articulatory breakdowns, prolonged phonemes and slow rate. This individual was reported to be largely unintelligible. At 4 years and 9 months post-surgery, the second individual was described as presenting with some phoneme prolongations, pitch breaks, variable pitch, lack of volume control, explosive onsets and 6 of the 10 ataxic dysarthria features including imprecise consonants, excess and equal stress, harsh voice, prolonged phonemes, prolonged intervals and slow rate.

Di Cataldo et al. (2001) presented a case study of an individual who at 25 months after surgery for the removal of a cerebellar tumour was reported to have normal speech following TCM. No formal assessment was completed.

In 1996, Jones, Kirolos, and Van Hille described a case study of an individual who underwent cerebellar tumour resection and developed TCM postoperatively. At a 2-year follow-up the individual was reported to demonstrate slurring and stuttering of speech, although no formal assessment was completed.

Discussion

When reviewing the results of these studies, it is important to consider issues related to subject selection, sample size, statistics, and methodology, as these factors may impact the strength of the evidence.

Subject Selection and Sample Size

Most of the studies involved very small sample sizes, ranging from one to seven subjects. This is likely due to the limited number of cases of TCM and the limited number of individuals who survive medulloblastoma brain tumours. As a result, the power and generalizability of the findings are compromised.

Another issue is the lack of random sampling in

the selection of the participants. The participants in the study by Huber et al. (2006) were the same individuals that participated in a previous study and the methods of participant selection were not described in either paper. Therefore it is unknown whether the methods of selection were valid and thus to what extent the results can be generalized. Steinbok et al. (2003) and Hudson et al. (1989) used hospital databases in order to identify potential subjects for their studies. This process may have had inherent biases as individuals who seek services at a particular hospital may be of a certain ethnic background or have a certain socioeconomic status. However, this background information was not provided by the authors.

Information regarding participant inclusion and exclusion criteria was not included in a number of studies (Huber et al., 2006; Steinbok et al., 2003; Di Cataldo et al., 2001). Therefore, it is unclear if factors such as medical history or the presence of premorbid speech impairments could have played a role in the outcome of these studies. Many papers also failed to report whether the subjects had received speech therapy and therefore it is unclear what role speech therapy may play in the prevention of long-term residual speech deficits in TCM. Reporting and controlling for these variables would assist in ensuring that the results obtained were due to the effects of cerebellar tumour resection and TCM and not to premorbid medical issues, premorbid speech impairments, or speech treatment.

A number of researchers failed to control for, or provide information regarding, tumour type, location or size, surgical resection technique and amount of tumour resected, or whether post-surgery treatment (e.g. radiotherapy, chemotherapy) was provided. Huber et al. (2006) matched the TCM group with the tumour resection group who did not develop TCM for tumour type and location. They also stated that there were equal numbers of radiated and nonradiated patients in each group. Steinbok et al., (2003) controlled for tumour type and location as well as the location of the surgical incision, however some individuals were reported to have received radiation while others did not. Hudson et al. (1989) provided information regarding tumour type, location and size, as well as whether radiation therapy was received, and, if so, the dosage and location of delivery. Di Cataldo et al. (2001) and Jones et al. (1996) reported tumour type and location as well as a description of the surgical procedure. In addition, Di Cataldo et al. (2001) included information regarding post-operative treatment including radiation and chemotherapy. These variables could impact the potential outcome of TCM and, therefore, controlling for them would increase the generalizability of the results obtained.

Methodological Issues

When interpreting the results of studies, it is important to consider limitations of the methodology, in particular prominent information that was not included in the research descriptions, statistics provided, and the

type of study conducted (e.g. case study, chart review, experimental).

Huber et al. (2006) and Hudson et al. (1989) failed to provide important information related to test administration and listening procedures (i.e. how many times the S-LPs were able to listen to speech samples; who administered the standardized tests, etc.). Several studies failed to provide information related to the methods and criteria that were used to determine the subjects' neurological deficits and diagnosis. This lack of information reduces the reproducibility and validity of the findings.

A number of researchers relied on subjective observations made by themselves in their case studies (Di Cataldo et al., 2001; Jones et al., 1996), or by parents through parental reports (Steinbok et al., 2003), to determine the presence of speech deficits. These studies do not provide empirical findings and, therefore, the results are less valid and reliable than experimental approaches. There are also concerns that the researchers reporting the observations were not qualified S-LPs and may have missed subtle speech deficits. Definitions or descriptions of the observations were not included and, therefore, what constitutes 'slurring of speech', for example, is unknown. Likewise, Huber et al. (2006) reported that individuals who developed TCM post-operatively were significantly more dysfluent than healthy controls, however, the type and frequency of dysfluencies observed were not provided. Therefore, whether these individuals presented with typical stuttering, consisting of more blocks and prolongations than word and phrase repetitions, remains unknown. Steinbok et al. (2003) obtained information from parents by asking whether their child's speech had returned to normal and used this information along with information from medical records to make a determination as to whether speech had returned to normal. How this determination was made and by whom was not reported. The method of how the information was obtained, and by whom, will influence the validity as well as the scope of the results. According to Steinbok et al. (2003) "...it is reasonable to assume that if a speech abnormality was noted by the parents, it is highly probable that a speech abnormality would have been identified in a formal speech assessment" (p.182). This may be true, however, the converse is not. Parents will most likely not be aware of all the aspects of speech that a trained professional would be able to detect (e.g. excess and equal stress, irregular articulatory breakdowns, prolonged phonemes and intervals, voice quality, fluency of speech) and a seemingly minor speech abnormality might not be detected or reported by a parent.

Another important aspect to discuss when appraising these articles is the manner in which speech samples were obtained. Huber et al. (2006) obtained connected speech samples using picture-prompted narrative speech task in which individuals were asked to tell a story using a children's picture storybook. The age of subjects in this study ranged from 8.75 to 31.5 years and, therefore, the use of a children's picture book may not have been appropriate for the older

children and adults in the study. Similarly, Hudson et al. (1989) used a picture stimulus and asked each child (age 8 and 16) 'what will happen next?'. Obtaining a speech sample through these means may have resulted in shorter samples that are not as comprehensive or representative of daily speech as would have been a more open discussion with the subjects. Assessing speech in a more natural environment (e.g. conversations with family members in the individual's home) and through more natural means would aid in increasing the validity of the speech samples obtained. This would result in samples that were more representative of daily speech and, therefore, more representative of the speech difficulties that the subjects face in their daily lives.

Finally, it is important to consider how speech outcomes were measured. It may not be appropriate to apply diagnostic and assessment criteria derived from the acquired dysarthrias of adulthood to those of children. The difficulties are similar to those seen when classifying the acquired aphasias in children, in that the clinical picture in childhood is different from that in adults (Catsman-Berrevoets, van Dongen & Zwetsloot., 1992, p.1108). However, Huber et al. (2006) used the Dysarthria Rating Scale in order to determine the presence of ataxic dysarthria and Hudson et al. (1989) used the Frenchay Dysarthria Assessment and the method of Darley et al. (1969) to describe the speech characteristics of their subjects. Therefore, the results obtained from these studies may have reduced validity and reliability.

Statistics

Inter-rater reliability was not reported by Huber et al. (2006) and therefore there is no way of knowing how many inconsistencies occurred and how often a consensus had to be reached. Inter-rater reliability scores would allow for the determination of the reliability of the results obtained. The researchers used an appropriate between-group ANOVA for each speech characteristic. However, with only six subjects in each group it is unlikely that the study had sufficient power to obtain a statistical difference between groups. Therefore, it is almost impossible to reject the null hypothesis and a descriptive approach might have been more appropriate for this study. The sample sizes in the remaining empirical research papers were too small to allow for statistical analyses.

Recommendations

Based on the critical review of the available literature there is evidence to suggest that some individuals continue to have speech deficits as measured by articulation, phonology, fluency of speech, rate of speech, or the presence of dysarthria characteristics years after surgery and TCM. However, several concerns regarding the research exist including; concerns regarding recruitment of participants, small sample sizes, lack of inclusion and exclusion criteria, lack of experimental designs and control groups and concerns regarding the use of adult criteria to classify the acquired dysarthrias of childhood. It is

therefore recommended that clinicians be cautious when generalizing the findings of these studies to clients in their practice, such as when providing parents with information regarding the expected speech outcomes for their child following surgery.

It is also recommended that further research be conducted to confirm the research that has been completed and to clarify this research question. Researchers working in this area are encouraged to:

1. Use experimental study designs and include control groups.
2. Develop longitudinal studies.
3. Use objective measurements for articulation, fluency, dysarthria, and rate of speech instead of relying on subjective and descriptive approaches.
4. Include relevant and important information such as inclusion and exclusion criteria, participant histories and recruitment procedures.
5. Include more participants in their studies and use random sampling.
6. Control for the use of radiation in the treatment paradigm, including whether it was delivered focally or to the whole brain, and the dose.
7. Control for the involvement of speech therapy post-surgery.
8. Obtain speech samples in more natural communication settings where interactions are more spontaneous and representative of daily conversational speech.
9. Use diagnostic tools which were developed for assessing the acquired dysarthrias of childhood.
10. Examine the relationship between the extent and location of cerebellar damage due to tumour resection and the extent of residual speech deficits.
11. Examine the relationship between the length of the period of mutism and characteristics of the subsequent recovery and the long term speech deficits.
12. Examine whether the residual deficits are so minimal as not to be perceived by the average person, and only by trained professionals through the use of diagnostic procedures.

Conclusion

The present literature review suggests that some individuals who have cerebellar tumours resected in childhood experience residual speech deficits following transient cerebellar mutism. These deficits may impact articulation, phonology, fluency, or rate of speech, or they may manifest as a complex dysarthria. They may persist beyond 2 years following surgery. This information is important for clinicians to consider when providing parents with the expected long-term speech outcomes of their child following surgery and mutism. Although the research is largely descriptive in nature and contains few subjects, there were long term speech deficits in all but

two participants in the previous studies. Until further experimental research can be completed, the findings from these studies can be used cautiously to show that there is the potential for residual long-term speech deficits following transient cerebellar mutism in childhood.

References

- Ackermann, H., Mathiak, K., & Riecker, A. (2007). The contribution of the cerebellum to speech production and speech perception: Clinical and functional imaging data. *The Cerebellum*, 6, 202-213.
- Breen SL, Kehagioglou P, Usher C, Plowman PN (2004). A comparison of conventional, conformal and intensity-modulated coplanar radiotherapy plans for posterior fossa treatment. *British Journal of Radiology*, 77(921), 768-774.
- Catsman-Berrevoets, C.E., Van Dongen, H.R., & Zwetsloot, C.P. (1992). Transient loss of speech followed by dysarthria after removal of posterior fossa tumour. *Developmental Medicine and Child Neurology*, 34, 1102-1117.
- Dailey AT, McKhann GM 2nd, Berger MS (1995). The pathophysiology of oral pharyngeal apraxia and mutism following posterior fossa tumor resection in children. *Journal of Neurosurgery*, 83(3), 467-475.
- Darley, F.L., Aronson, A.E., & Brown, J.R. (1969). Differential diagnostic patterns of dysarthria. *Journal of Speech and Hearing Disorders*, 12, 246-269.
- Di Cataldo, A., Dollo, C., Astuto, M., La Spina, M., Ippolito, S., & Papotto, M. Guiffreda, S. (2001). Mutism after surgical removal of a cerebellar tumor: two case reports. *Pediatric Hematology and Oncology*, 18, 177-121.
- Doxey, D., Bruce, D., Sklar, F., Swift, D., & Shapiro, K. (1999). Posterior fossa syndrome: Identifiable risk factors and irreversible complications. *Pediatric Neurosurgery*, 31, 131-136.
- Duffy, J.R. (2005). *Motor Speech Disorders: Substrates, Differential Diagnosis and Management*. St. Louis: Mosby Pub.
- Germano, A., Baldari, S., Caruso, G., Caffo, M., Montemagno, G., Cardia, E., & Tomasello, F. (1998). Reversible cerebral perfusion alterations in children with transient mutism after posterior fossa surgery. *Childs Nervous System*, 14, 114-119.
- Gordon, N. (1996). Speech, language, and the cerebellum. *European Journal of Disorders of Communication*, 31, 359-367.
- Huber, J., Bradley, K., Spiegler, B., & Dennis M. (2006). Long-term effects of transient cerebellar mutism after cerebellar astrocytoma or medulloblastoma tumor resection in childhood. *Childs Nervous System*, 22, 132-138.
- Huber-Okrainec, J., Dennis, M., Bradley, K., & Spiegler, B. (2001). Motor speech deficits in long-term survivors of childhood cerebellar tumours: Effects of tumor type, radiation, age at diagnosis, and survival years. *Neuro-Oncology*, 3, 371.
- Hudson, L.J., Murdoch, B.E., & Ozanne, A.E. (1989). Posterior fossa tumours in childhood: associated speech and language disorders post-surgery. *Aphasiology*, 3, 1-18.
- Jones, S., Kirolos, R.W., & Van Hille, P.T. (1996). Cerebellar mutism following posterior fossa tumour surgery. *British Journal of Neurosurgery*, 10, 221-224.
- Kent, R.D., Duffy, J.R., Slama, A., Kent, J.F., & Clift, A. (2001). Clinicoanatomic studies in dysarthria: review, critique and directions for research. *Journal of Speech, Language, and Hearing Research*, 44, 535-551.
- Pollack, I.F. (1997). Posterior fossa syndrome. *International Review of Neurobiology*, 41, 411-432.
- Pollack IF, Polinko P, Albright AL, Towbin R, Fitz C (1995). Mutism and pseudobulbar symptoms after resection of posterior fossa tumors in children: incidence and pathophysiology. *Neurosurgery*, 37(5), 885-893.
- Ray A, Ho M, Ma J, Parkes RK, Mainprize TG, Ueda S, McLaughlin J, Bouffet E, Rutka JT, Hawkins CE (2004). A clinicobiological model predicting survival in medulloblastoma. *Clinical Cancer Research*, 10(22), 7613-7620.
- Rekate, H.L., Grubb, R.L., Aram, D.M., Hahn, J.F., Ratcheson, R.A. (1985) Muteness of cerebellar origin. *Archives of Neurology*, 42, 697-698.
- 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, 1051-1061.
- Steinbok, P., Cochrane, D.D., Perrin, R., & Price, A. (2003). Mutism after posterior fossa tumour resection in children: Incomplete recovery on long-term follow-up. *Pediatric Neurosurgery*, 39, 179-183.
- van Dongen, H.R., Catsman-Berrevoets, C.E., & van Mourik M. (1994). The syndrome of cerebellar' mutism and subsequent dysarthria. *Neurology*, 44, 2040-2046.
- Vvan Mourik M, Catsman-Berrevoets CE, Yousef-Bak E, Paquier PF, van Dongen HR (1998). Dysarthria in children with cerebellar or brainstem tumors. *Pediatric Neurology*, 18(5), 411-414.

Author Note

Correspondence concerning this article should be addressed to Scott G. Adams, School of Communication Sciences and Disorders, University of Western Ontario, London, Ontario N6G 1H1. E-mail: sadams@uwo.ca

Received: May 10, 2007

Accepted: February 20, 2008

