

Does the cerebellar sequential theory explain spoken language impairments? A literature review

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► **To cite this version:**

M. Lupo, G. Olivito, L. Angelini, G. Funghi, F. Pignatelli, et al.. Does the cerebellar sequential theory explain spoken language impairments? A literature review. *Clinical Linguistics & Phonetics*, Taylor & Francis, 2020, 35 (4), pp.296 - 309. 10.1080/02699206.2020.1745285 . hal-03172052

HAL Id: hal-03172052

<https://hal.univ-lorraine.fr/hal-03172052>

Submitted on 18 Mar 2021

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4 **literature review.**
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38 The authors report no conflict of interest
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Abstract

During the past decades, converging evidence from clinical, neuroimaging and neuroanatomical studies has demonstrated the key role of the cerebellum in the processing of non-motor aspects of language. Although more is known about the way in which the cerebellum participates in the mechanisms involved in written language, there is ambiguous information on its role in other aspects of language, such as in non-motor aspects of spoken language.

Thus, to contribute additional insight into this important issue, in the present work, we review several original scientific papers focusing on the most frequent non-motor spoken language impairments evidenced in patients affected by cerebellar pathology, namely, verbal working memory, grammar processing and verbal fluency impairments. Starting from the collected data, we provide a common interpretation of the spoken language disorders in cerebellar patients, suggesting that sequential processing could be the main mechanism by which the cerebellum participates in these abilities. Indeed, according to the cerebellar sequential theory, spoken language impairments could be due to altered cerebellar function to supervise, synchronize and coordinate the activity of different functional modules, affecting the correct optimization of linguistic processing.

Keywords

Cerebellum, spoken language, sequential processing, verbal working memory, verbal fluency, grammar processing

Introduction

For over two centuries, the cerebellum was considered to be mainly involved in motor control and coordination (Manto et al., 2012; Mariën & Beaton, 2014; Murdoch, 2010). However, clinical and neuroanatomical studies on patients with cerebellar damage and neuroimaging data on healthy subjects have shown that the cerebellum participates in a large spectrum of affective and cognitive domains, including linguistic functions (Clausi et al., 2017, 2019; Leggio, Chiricozzi, Clausi, Tedesco, & Molinari, 2011; Lupo et al., 2015, 2019; Mariën & Beaton, 2014; Stoodley & Schmahmann, 2009; 2010; Tedesco et al., 2011).

Specifically, difficulties in the language domain are one of the main signs of “*cerebellar cognitive affective syndrome*” (CCAS), described in 1998 by Schmahmann and Sherman. This syndrome results from cerebellar damage and is characterized by impairments in executive function, spatial cognition, and language, together with personality changes (Schmahmann & Sherman, 1998). Although the role of the cerebellum in cognition has been widely acknowledged, how this brain region is involved in specific dysfunctions of complex domains, such as language, is still a matter of debate (Mariën & Beaton, 2014; Mariën & Borgatti, 2018).

Several studies on patients with cerebellar pathology have evidenced alterations in spoken language abilities, such as verbal working memory, verbal fluency, and grammar processing, that are not related to mere motor abilities (Chiricozzi, Clausi, Molinari, & Leggio, 2008; Gasparini et al., 1999; Leggio, Silveri, Petrosini, & Molinari, 2000; Mariën et al., 2014, 1996; Murdoch, 2010; Ravizza et al., 2006; Schweizer, Alexander, Gillingham, Cusimano, & Stuss, 2010; Silveri, Leggio, & Molinari, 1994).

Cerebellar involvement in non-motor language processing is supported by reciprocal crossed neural pathways between the cerebellum and frontal areas involved in language functions (Hubrich-Ungureanu, Kaemmerer, Henn, & Braus, 2002; Jansen et al., 2005; Mariën & Beaton, 2014; Murdoch, 2010; Stoodley & Schmahmann, 2009). Furthermore, data from clinical studies on cerebellar patients show that linguistic disorders follow injuries to the right or left posterolateral cerebellar hemisphere (lobules VI and Crus I/II) (Fabbro, Moretti, & Bava, 2000; Richter et al., 2007; Schmahmann & Sherman, 1998; Tedesco et al., 2011).

Despite considerable effort to characterize the anatomo-clinical configuration of non-motor linguistic dysfunctions in cerebellar disorders (Mariën & Beaton, 2014), there is no agreement about the cerebellar operational mode in these aspects.

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3 Three theories have been advanced to explain how the cerebellum modulates language processing:
4 the *crossed cerebello-cortical diaschisis* theory (Broich, Hartmann, Biersack, & Horn, 1987;
5 Mariën, Engelborghs, Fabbro, & De Deyn, 2001), the *timing theory* (Gasparini et al., 1999; Keele &
6 Ivry, 1990; Silveri et al., 1994), and the *sequencing theory* (Ackermann, Mathiak, & Ivry, 2004;
7 Fabbro et al., 2000; Leggio et al., 2011).
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11 The *crossed cerebello-cortical diaschisis* theory postulates that, in the presence of cerebellar
12 damage, linguistic deficits might result from functional depression of supratentorial language areas
13 due to a reduction in excitatory impulses through the cerebello-thalamo-cortical pathways (Broich
14 et al., 1987; Mariën et al., 2001). According to this hypothesis, language problems might arise from
15 hypoperfusion in the frontal areas of the left cerebral hemisphere to which the cerebellum is
16 connected (Ackermann, Mathiak, & Riecker, 2007; Murdoch, 2010; Silveri et al., 1994).
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19 The *timing theory* asserts that the cerebellum plays an important role in the timing of linguistic
20 functions, operating as a modular clock-like system preparing the cortical regions in a timely
21 manner to optimize speech perception (Keele & Ivry, 1990; Schwartz & Kotz, 2016; Silveri et al.,
22 1994).
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25 The *sequential theory*, on which we focus in the present mini-review, suggests that the cerebellum
26 works by simulating repetitive patterns of temporally or spatially structured events. This simulation-
27 skill allows one to create internal models of different information types in order to predict future
28 events (Ito, 2008; Leggio & Molinari, 2015). According to this hypothesis, when an incoming
29 external stimulus is received by the cerebellum, it makes a match between the internal model
30 previously generated and the new stimulus. If discrepancies are detected, the cerebellum sends
31 feedback to cortical areas to modify the response based on the new event (Leggio & Molinari,
32 2015). It has been proposed that this cerebellar operational mode could be applied to both motor
33 and cognitive domains (Leggio et al., 2011; Restuccia, Della Marca, Valeriani, Leggio, & Molinari,
34 2007).
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37 In particular, regarding the language domain, it has been hypothesized that the cerebellum acts as a
38 predictor of future states, additionally optimizing language processes (Mariën et al., 2014; Mariën
39 & Borgatti, 2018). This hypothesis is in line with the results obtained in most of the studies that
40 tested the link between sequential processing and language learning, which found that knowledge of
41 the word-order sequence in specific sentences permits a listener to better identify, or even implicitly
42 predict, the words that will follow (Conway, Bauernschmidt, Huang, & Pisoni, 2010; Elliott, 1995;
43 McClelland, Mirman, & Holt, 2006; Miller, Heise, & Lichten, 1951; Onnis, Farmer, Baroni,
44 Christiansen, & Spivey, 2008; Pisoni, 1996; Rubenstein, 1973).
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3 To contribute additional insight into this important issue, in the present work, we review the clinical
4 data describing spoken language alterations in cerebellar patients, focusing on studies that have
5 investigated verbal working memory, grammar processing and verbal fluency.
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8 Taking into account the key role of sequential processing in language abilities, we interpret the
9 results in light of cerebellar sequencing theory.
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13 **Verbal working memory**

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15 Verbal working memory (VWM) can be described as a short-term memory mechanism that allows
16 one to temporarily store a limited amount of speech-related information (such as letters, words or
17 numbers) (Marvel & Desmond, 2015). According to Baddeley's model (1992), VWM is supported
18 by a phonological loop that contains two subcomponents. The first is a passive storage process, also
19 called the phonological short-term store (ph-STs), which contains acoustic or speech-based
20 information that lasts 1–2 seconds. The second is an active articulatory rehearsal process that
21 "refreshes the phonological storage" (Marvel & Desmond, 2015). The articulatory control process is
22 further divided into two stages: In the first stage, a visual-verbal stimulus (e.g., printed words) is
23 translated into a phonological representation; in the second stage, subvocal repetition refreshes this
24 information so that it can be maintained in phonological storage (Baddeley, 1992; Mariën et al.,
25 2014).
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29 During the past decades, several clinical studies have evidenced VWM impairment in patients with
30 focal cerebellar lesions (Ben-Yehudah & Fiez, 2008; Chiricozzi et al., 2008; de Ribaupierre, Ryser,
31 Villemure, & Clarke, 2008; Justus, Ravizza, Fiez, & Ivry, 2005; Ravizza et al., 2006; Silveri, Di
32 Betta, Filippini, Leggio, & Molinari, 1998) or degenerative cerebellar damage (Cooper et al., 2012;
33 Justus et al., 2005; Tamura et al., 2018) (see Table 1). According to these findings, some studies
34 have attributed to the cerebellum the role of controller and organizer of several language inputs,
35 allowing for a correct output process (Justus et al., 2005; Silveri et al., 1998).
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39 More specifically, in a patient affected by a right cerebellar lesion, Silveri and colleagues (1998)
40 found a reduced verbal digit span and a rapid forgetting of verbal material. The authors identified
41 the patient's deficit in the phonological output buffer. This component is part of the rehearsal
42 system, where phonological information is temporally stored and then sent to various output
43 processes (i.e., planning or editing of the processes that are needed for speech). In light of this, the
44 authors hypothesized that a cerebellar lesion may affect the articulatory programme of speech
45 output within the phonological output buffer. Thus, it is likely that the cerebellum organizes and
46 controls different input information in a sequential way to guarantee the implementation of the
47 correct articulatory rehearsal processes and motor execution for regular speech planning.
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3 A similar interpretation of the role of the cerebellum as the controller of VWM function was
4 suggested by Justus and collaborators (2005), who analysed VWM deficits in patients with focal or
5 degenerative cerebellar damage by using auditory and visual word recall tasks. In these patients, the
6 authors described a reduced phonological similarity effect. The phonological similarity effect is the
7 phenomenon whereby the memory for phonologically dissimilar items is stronger than the memory
8 for phonologically similar stimuli. Thus, the absence of this effect for both aural and visual stimuli
9 reflects a phonological store impairment (Justus et al., 2005). However, the authors did not limit
10 cerebellar involvement in VMW merely to a phonological store dysfunction. Rather, they suggested
11 that its role may consist of the ongoing correction of altered sensory information, integrating
12 acoustic and phonetic representations with articulatory representations.

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14 In this context, a valid contribution to the explanation of the mechanism by which the cerebellum is
15 involved in VWM deficits was provided by Leggio and colleagues (2011). They hypothesized that
16 the cerebellum compares different activity patterns to detect discrepancies that need to be corrected
17 (i.e., between the articulatory rehearsal processes and the motor execution, between the output of
18 sub-vocal articulation and the content of phonological storage, and between acoustic/phonetic
19 representations and articulatory representations) (Desmond, Gabrieli, Wagner, Ginier, & Glover,
20 1997). To do so, the sequential characteristics of incoming data must be scanned and recognized. It
21 has been proposed that sequence-in-sequence-out processing can be considered to reflect cerebellar
22 involvement in VWM (Leggio et al., 2011).

37 **Verbal fluency**

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39 Verbal fluency is the ability to generate lists of words according to a given semantic or phonetic
40 rule, and it is usually assessed by word-generation tasks (Abwender, Swan, Bowerman, &
41 Connolly, 2001; Ruff, Light, Parker, & Levin, 1997).

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43 Several studies have described phonological and/or semantic verbal fluency impairments in patients
44 affected by focal cerebellar lesion (Alexander, Gillingham, Schweizer, & Stuss, 2012; Gottwald,
45 Wilde, Mihajlovic, & Mehdorn, 2004; Neau, Arroyo-Anllo, Bonnaud, Ingrand, & Gil, 2000;
46 Peterburs, Bellebaum, Koch, Schwarz, & Daum, 2010; Richter et al., 2007; Schweizer et al., 2010;
47 Leggio et al., 2000; Schmahamnn, 1998) or degenerative cerebellar damage (Apollonio, Grafman,
48 Schwartz, Massaquoi, & Hallett, 1993; Brandt, Leroi, O'Hearn, Rosenblatt, & Margolis, 2004; de
49 Nóbrega, Nieto, Barroso, & Montón, 2007; Fancellu et al., 2013; Nieto, Correia, de Nóbrega,
50 Montón, & Barroso, 2013; Nieto et al., 2012; Stoodley & Schmahmann, 2009; Suenaga et al., 2008;
51 Leggio et al., 2000; Schmahamnn, 1998) (see Table 1). Word retrieval requires sequencing abilities
52 that allow one to compare previous stimuli with current stimuli. Therefore, to obtain a correct
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3 response, the last word must be coupled sequentially with the new word to keep the prototypical
4 sound (phonological fluency) or category (semantic fluency) active within the working memory
5 system and recognize the correct correspondence between two consecutive words. However, while
6 semantic verbal fluency is based on well-known, already employed procedures, phonological verbal
7 fluency requires an unusual, novel and less automatized search strategy (Martin, Wiggs, Lalonde, &
8 Mack, 1994; Rosser & Hodges, 1994). Indeed, it has to be noted that phonological verbal fluency
9 results are more frequently affected by cerebellar damage than are semantic results (see Table 1).
10 This is conceivable considering that, when a strategy is well-learned, as is the case in semantic
11 retrieval, sequential processing is achieved with less cerebellar effort; conversely, when a strategy is
12 new, active cerebellar involvement is necessary to monitor and accelerate sequential information
13 processing (Leggio et al., 2011, 2000).
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24 **Grammar processing**

25 Grammar processing refers to a well-organized linguistic framework of features and structural rules
26 that regulate the composition of a sentence structure (Adamaszek & Kirkby, 2015). Indeed, the
27 composition of grammatically correct sentences depends on a correct retrieval and application of
28 grammar rules (such as the correct positioning of the noun first and the verb after or the correct
29 inflexion, as with noun-verb agreement) and requires the integration of various linguistic functions
30 controlled by different neural circuits (Ackermann et al., 2007; Adamaszek & Kirkby, 2015).
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36 Over the past 30 years, several studies have demonstrated that, in speech production, the cerebellum
37 also contributes to morphological, syntactic and lexical aspects of grammar processing
38 (Ackermann, Gräber, Hertrich, & Daum, 1999; Adamaszek, Strecker, & Kessler, 2012; Fabbro et
39 al., 2000; Gasparini et al., 1999; Justus, 2004; Mariën, Engelborghs, Pickut, & De Deyn, 2000;
40 Mariën et al., 1996; Silveri et al., 1994; Zettin et al., 1997).
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45 The first case of agrammatic speech following an infarction in the right cerebellar hemisphere was
46 described by Silveri and collaborators (1994). The patient presented with specific impairments in
47 spontaneous speech, characterized by the omission of free-standing grammatical morphemes and
48 the use of infinitives in place of inflected verb forms. The authors hypothesized that the cerebellar
49 lesion could have altered the correct sequential timing required for functional interplay among the
50 neural structures involved in the production of sentences. In accordance with this interpretation,
51 Gasparini and collaborators (1999) speculated that the expressive agrammatism observed in a
52 patient with right-hemisphere cerebellar lesion was the result of a delay in the application of
53 syntactic rules and linguistic processes caused by damage to the cerebellum. Due to this delay,
54 information temporarily stored in verbal working memory decays before all linguistic operations are
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3 successfully completed, resulting in errors (Gasparini et al., 1999; Mariën & Borgatti, 2018; Silveri
4 et al., 1994). More recent studies have confirmed that cerebellar damage may cause agrammatic
5 speech, characterized by simplification of syntactic structures, reduced sentence length and/or
6 omissions and grammatical substitutions (Ackermann et al., 1999; Adamaszek et al., 2012; Fabbro
7 et al., 2000; Gasparini et al., 1999; Justus, 2004; Mariën et al., 1996; Silveri et al., 1994; Zettin et
8 al., 1997) (see Table 1).

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10 Leggio and colleagues (2011) interpreted grammatical impairment due to cerebellar lesions to result
11 from damage to the sequential and automatic computational components of grammatical operation,
12 referring to the declarative-procedural model of language proposed by Ullman (2004). According to
13 this model, language is based on a “mental lexicon” of memorized word-specific knowledge, which
14 depends on declarative memory, and a “mental grammar”, which renders the combination of lexical
15 items into complex representations and depends on the neural system that underlies the procedural
16 memory system (Ullman, 2004). It has been hypothesized that this system plays a relevant role in
17 rule-governed structure building, such as the sequential and hierarchical combination of stored
18 forms and abstract representations into complex structures (Leggio et al., 2011; Molinari & Leggio,
19 2013).

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21 In this framework, the cerebellum has been hypothesized to be involved in the search for lexical
22 items from declarative memory and in the detection, through error-based learning of rules, that
23 underlies the regularities of complex structures (Leggio et al., 2011; Molinari & Leggio, 2013;
24 Ullman, 2004). Indeed, since complex morpho-syntactic operations, which allow for the
25 construction of syntactically correct sentences, demand the use of sequential procedures within a
26 defined period of time, cerebellar dysfunction can impair the sequential and automatic computation
27 of grammatical operations. The outcomes of this dysfunctional sequential processing are the
28 decoupling of morpho-syntactic processes that are the basis for sentence representation, a slowdown
29 in mental operations, and the decay of morphemes from working memory, resulting in sentence
30 disintegration (Leggio & Molinari, 2015; Silveri et al., 1994).

31 32 33 34 35 36 37 38 39 40 41 42 43 44 45 46 47 48 49 **Discussion**

50
51 In the present review, we summarized clinical studies that described specific non-motor spoken
52 language deficits in terms of verbal working memory, verbal fluency and grammar processing
53 impairments among cerebellar patients. These deficits were interpreted in light of sequential
54 processing theory, which describes the basic mechanism of cerebellar functioning (Leggio et al.,
55 2011; Molinari et al., 2008; Molinari & Leggio, 2013).

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3 Indeed, in non-motor spoken language function, sequential processing is required when a set of
4 linguistic elements (phonemes, syllables, words) must be organized in a non-random way according
5 to specific rules (Daltrozzo, Conway, & Smith, 2013; Gomez & Gerkin, 1999; Graf Estes, Evans,
6 Alibali, & Saffran, 2007; Mirman, Magnuson, Estes, & Dixon, 2008; Saffran, Aslin, & Newport,
7 1996; Ullman, 2004). Therefore, in this function, the cerebellum acts as a supervisor of the
8 sequence of linguistic processes by synchronizing and coordinating the activity of different
9 functional modules that are necessary to perform linguistic processes (Mariën et al., 2014; Mariën
10 & Borgatti, 2018).

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12 Strong evidence supporting the cerebellar role in non-motor spoken language is provided by
13 reciprocal neuroanatomical connections that closely connect the cerebellum to the projection areas
14 of the language-dominant hemisphere (Mariën & Beaton, 2014). Analogous to the motor system,
15 through these connections, the cerebellum might control the speed, capacity, consistency and
16 appropriateness of cognitive and linguistic processing, just like it adjusts the accuracy, rate, force
17 and rhythm of movement (Murdoch, 2010; Schmahmann, 1998).

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19 According to this view, the VWM impairments described in cerebellar patients (Ben-Yehudah &
20 Fiez, 2008; Chiricozzi et al., 2008; Cooper et al., 2012; de Ribaupierre et al., 2008; Justus et al.,
21 2005; Ravizza et al., 2006; Silveri et al., 1998; Tamura et al., 2018) can be explained in light of an
22 inability to compare the phonological information and the phonetic representations of letters and
23 words in input with the subvocal articulation/representation in output, evidencing difficulties to
24 correct existing discrepancies between these frames using sequential processing (Leggio et al.,
25 2011). Evidence confirming the use of sequential theory to describe VWM deficits has also been
26 derived from studies with healthy subjects. Indeed, Peterburs and collaborators (2019), using an
27 fMRI protocol associated with a modified version of the Sternberg Verbal working memory Task
28 (Sternberg, 1966), found significant activation of the cerebellar posterior lobule VIIIa. In this task,
29 the list length of an initial memory set of items (encoding phase) that subjects must retain over a
30 short time interval to respond appropriately to a final probe item (retrieval phase) is manipulated.
31 Thus, the authors suggested that the cerebellum may contribute to VWM by participants' capacity
32 to predict letter sequences during the retrieval process. Along a similar line, Sheu and colleagues
33 (2019) used a transcranial magnetic stimulation (TMS) protocol on the right cerebellum of healthy
34 subjects and described an alteration in VWM during the rehearsal of a sequence of encoded letters,
35 which showed cerebellar involvement when it was necessary to identify whether a probe letter was
36 in the correct position within a word, predicting upcoming verbal items in the phonological loop
37 (Sheu, Liang, & Desmond, 2019). Thus, the predicted output of the rehearsal process must be
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3 constantly compared to the content of the phonological storage, which holds the correct phoneme
4 sequence (Sheu et al., 2019).

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6 Regarding verbal fluency, existing data suggest that cerebellar patients are impaired in verbal
7 fluency abilities, with a major involvement of phonological verbal fluency (see Table 1). We
8 advance the hypothesis that verbal fluency depends on the different cognitive loads imposed by
9 semantic and phonological word searching of the mental lexicon in terms of higher or lower
10 automatized sequential control during recall, selection and comparison processes.

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12 Finally, the hypothesis of a sequential alteration in grammatical disorders was demonstrated
13 considering the morphological, syntactic and lexical impairments of speech production (Ackermann
14 et al., 1999; Adamaszek et al., 2012; Fabbro et al., 2000; Gasparini et al., 1999; Justus, 2004;
15 Mariën et al., 2000, 1996; Silveri et al., 1994; Zettin et al., 1997). In these disorders, the sequential
16 processing alteration was explained in terms of cerebellar impairment to synchronize and coordinate
17 the different modules required for a correct linguistic output by integrating the information received
18 from the ‘frontal lobe system’ (Ackermann et al., 2004; Fabbro et al., 2000). Although, in the
19 literature, there are no univocal interpretations of a sequential processing impairment in agrammatic
20 speech, it is possible to hypothesize that, similar to writing disorders, the cerebellum is strictly
21 involved in the mechanism that allows for the correct activation and timing of letters within a string
22 and placement of the letters in a specific sequential writing order (Lupo et al., 2019; Mariën &
23 Borgatti, 2018).

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25 In summary, the clinical data reported in the present review suggest that a sequencing deficit due to
26 cerebellar damage could represent a common functional impairment in a variety of non-motor
27 language functions, including verbal working memory, verbal fluency and grammar/syntax. (Leggio
28 et al., 2011, 2000; Molinari & Leggio, 2013).

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30 Unfortunately, there are still very few reports that investigate the link between non-motor spoken
31 language disorders and cerebellar sequential processing impairments. Therefore, we believe that the
32 development of experimental protocols focused on these aspects is needed to further clarify this
33 issue.

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35 Finally, we suggest that studies on this topic could have important clinical implications for the
36 assessment of cognitive rehabilitation of language impairments through sequential processing
37 training, considering the cerebellum as a potential target of non-invasive neuro-modulation
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3 techniques (Bauernschmidt et al., 2009; Daltrozzo et al., 2013; Smith, Conway, Bauernschmidt, &
4 Pisoni, 2015).
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8 **Statement of Interest**

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10 The authors report no conflict of interest.
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3 **Table title**
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5 **Table 1.** Spoken language impairments in patients affected by cerebellar damage
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Table 1. Spoken language impairments in patients affected by cerebellar damage

REFERENCE	ETIOLOGY	TASK	DEFICIT
<i>Verbal Working Memory</i>			
Silveri et al., 1998	Focal lesion (<i>RC</i>)	- Immediate serial recall of lists of verbal stimuli	Phonological output buffer
Justus et al., 2005	Cerebellar degeneration Focal lesion (<i>RC, LC</i>)	- Auditory word recall - Visual word recall	Reduction of the phonological similarity effect in both conditions
Ravizza et al., 2006	Focal lesion (<i>RC, LC</i>)	- Digit span - Spatial span - Immediate span	Verbal working memory tasks
Ben-Yehudah & Fiez, 2008	Focal lesion (<i>RC, LC</i>)	- Digit span - Visual memory span	Recalling of a no-words list
Chiricozzi et al., 2008	Focal lesion (<i>RC, LC</i>)	- Digit span - Articulatory suppression - Phonological similarity effect - Short-term forgetting - Unattended speech	Impairment of the phonological Shot-Term Store
de Ribaupierre et al., 2008	Focal lesion (<i>RC, LC</i>)	- Digit Span - 15 word list (Clapared-Rey)	Anterograde memory
Tamura et al., 2018	Cerebellar degeneration (<i>SCA type 3</i>)	- The Miyake Paired-Associate Word Learning Test - The Alzheimer's Disease Assessment Scale	Retrieval of information in episodic memory storage
<i>Verbal Fluency</i>			
Appollonio et al., 1993	Cerebellar degeneration	- Phonemic (F-A-S) - Semantic (animals, country, supermarket)	Phonemic and semantic
Schmahmann & Sherman, 1998	Cerebellar degeneration (<i>ICA; cerebellitis</i>) Focal lesion (<i>RC, LC, Bil, medial</i>)	- The Animal Naming	Semantic
Leggio et al., 2000	Cerebellar degeneration (<i>ICA</i>) Focal lesion (<i>RC, LC</i>)	-Phonemic (F-A-S) -Semantic (birds and furniture)	Phonemic
Neau et al., 2000	Focal lesion (<i>RC, LC, Bil</i>)	- Phonemic - Semantic (animals, colours, fruits and towns)	Semantic

Brandt et al., 2004	Cerebellar degeneration (<i>Genetic etiology; multisystem atrophy</i>)	- Phonemic (F-A-S) - Semantic (animals and supermarket)	Phonemic
Gottwald et al., 2004	Focal lesion (<i>RC, LC, Bil, medial</i>)	- Phonemic (L-B-S) - Semantic (animals, supermarket, hobbies)	Phonemic and semantic
de Nóbrega et al., 2007	Cerebellar degeneration (<i>Friederich Ataxia</i>)	- Phonemic (F-A-S) - The Animal Naming Test	Phonemic
Richter et al., 2007	Focal lesion (<i>RC, LC, Bil, medial</i>)	- Phonemic Verbal Fluency	Phonemic
Suenaga et al., 2008	Cerebellar degeneration (<i>SCA type 6</i>)	- Phonemic (nouns starting with the Japanese Kana character “Ka”) - Semantic (animals)	Phonemic and semantic
Stoodley & Schmahmann, 2009	Cerebellar degeneration (<i>SCA type 6, SCA type 8, ILOCA, ILOCA dominant pure cerebellar ataxia, cerebellitis, Behçet-related cerebellar ataxia</i>)	- Phonemic (F-A-S) - Semantic (animals, fruits)	Phonemic and semantic (with a trend to be more impaired on phonemic than semantic fluency)
Peterburs et al., 2010	Focal lesion (<i>RC, LC, Bil</i>)	- Phonemic (nouns starting with letter “B”) - Semantic (countries)	Phonemic
Schweizer et al., 2010	Focal lesion (<i>RC, LC</i>)	- Phonemic (F-A-S) - Semantic (animals)	Phonemic and semantic
Alexander et al., 2010	Focal lesion (<i>RC, LC</i>)	- Phonemic (F-A-S) - Semantic (animals)	Phonemic and semantic
Nieto et al., 2012	Cerebellar degeneration (<i>Friedreich ataxia</i>)	- Phonemic (F-A-S) - Semantic (animals)	Phonemic and semantic
Nieto et al., 2013	Cerebellar degeneration (<i>Friedreich ataxia</i>)	- Phonemic (F-A-S) - Semantic (animals)	Phonemic and semantic
Fancellu et al., 2013	Cerebellar degeneration (<i>SCA type1; SCA type2</i>)	- Phonemic (F-L-P) - Semantic (animals, fruits and car brand)	Phonemic and semantic
<i>Grammar Processing</i>			
Silveri et al., 1994	Focal lesion (<i>RC</i>)	- Qualitative and quantitative analysis of spontaneous speech (considering omissions and substitutions of grammatical morphemes)	Agrammatic speech and reduced verbal fluency

Zettin et al., 1997	Focal lesion (<i>RC</i>)	- Qualitative and quantitative analysis of spontaneous speech (considering omissions and substitutions of grammatical morphemes)	Expressive agrammatic disorder
Ackermann et al., 1999	Cerebellar degeneration	- Time discrimination and identification tasks	Receptive agrammatism
Gasparini et al., 1999	Focal lesion (<i>RC</i>)	Language Examination	Expressive agrammatism
Marien et al., 1996	Cerebellar degeneration (<i>SCA</i>)	- Boston Diagnostic Aphasia Examination - Aachner Aphasic Test - The Frenchay Dysathria Assessment - The Token Test	Luria's dynamic aphasia, expressive and receptive agrammatism
Fabbro, 2000	Focal lesion (<i>RC, LC, vermis</i>)	- Italian adaptation of the Bilingual Aphasia Test - Word generation	Morphosyntactic errors
Justus, 2004	Cerebellar degeneration Focal lesion (<i>RC, LC, midline</i>)	- Picture description - Language comprehension - Grammatically judgment	Grammatical morphology alterations
Adamaszek et al., 2012	Focal lesion (<i>RC, LC</i>)	Syntactic Positive Shift	Syntactic processing alterations

Abbreviations: RC= right cerebellar lesion; LC= left cerebellar lesion; Bil= Bilateral cerebellar lesion; SCA = Spinocerebellar ataxia; ICA = Idiopathic cerebellar ataxia; ILOCA; Idiopathic late onset cerebellar ataxia