

# A specific pattern of executive dysfunctions in transcortical motor aphasia

Lilla Zakariás<sup>1,2</sup>, Attila Keresztes<sup>3</sup>, Gyula Demeter<sup>3</sup>, and Ágnes Lukács<sup>3</sup>

<sup>1</sup>Institute of Education, Eötvös Loránd University, Budapest, Hungary

<sup>2</sup>Neurology and Rehabilitation Centre, Flór Ferenc Hospital, Kistarcsa, Hungary

<sup>3</sup>Department of Cognitive Science, Budapest University of Technology and Economics, Budapest, Hungary

*This is an Accepted Manuscript of an article published by Taylor & Francis in Aphasiology on 13/09/2013, available online:*  
<http://dx.doi.org/10.1080/02687038.2013.835783>

## A specific pattern of executive dysfunctions in transcortical motor aphasia

Lilla Zakariás<sup>1,2</sup>, Attila Keresztes<sup>3</sup>, Gyula Demeter<sup>3</sup>,  
and Ágnes Lukács<sup>3</sup>

<sup>1</sup>Institute of Education, Eötvös Loránd University, Budapest, Hungary

<sup>2</sup>Neurology and Rehabilitation Centre, Flór Ferenc Hospital, Kistarcsa, Hungary

<sup>3</sup>Department of Cognitive Science, Budapest University of Technology and Economics, Budapest, Hungary

5 AQ1

*Background:* Recent studies imply that executive functions (EF) are closely related to our ability to comprehend and produce language. A number of findings suggest that functional communication and language recovery in aphasia depend not only on intact language abilities but on EF as well. Some patients with transcortical motor aphasia (TMA) show language deficits only in tasks in which conflicting representations must be resolved by executive processes. In line with these results, others have proposed that TMA should be referred to as “dysexecutive aphasia”. EF in aphasia have mostly been studied using neuropsychological tests, therefore there is a need for systematic experimental investigations of these skills.

10

*Aims:* 1. To investigate EF in TMA, and to test whether executive dysfunctions are specific to TMA. 2. To experimentally measure different components of EF: updating working memory representations and inhibition of prepotent responses.

20

*Methods & Procedures:* Five individuals with TMA, five patients with conduction aphasia and ten healthy controls participated. We designed four nonverbal tasks: to measure updating of working memory representations, we used a visual and an auditory *n*-back task. To assess inhibition of prepotent responses, we designed a Stop-signal and a nonverbal Stroop task. All tasks involved within-subject baseline conditions.

25

*Outcomes & Results:* We found certain EF deficits in both groups of individuals with aphasia as compared to healthy controls. Individuals with TMA showed impaired inhibition as indexed by the Stop-signal and the nonverbal Stroop tasks, as well as a deficit of updating of working memory representations as indexed by the auditory *n*-back task. Participants with conduction aphasia had difficulties in only one of the tasks measuring inhibition, but no clear evidence for impairment of updating of working memory representations was found.

30

*Conclusions:* Although the results show different patterns of EF deficits in the groups with aphasia, the findings clearly demonstrate that EF deficits are not specific to participants with TMA. Based on these results, and on earlier data highlighting the role of executive processes in functional communication and language recovery, we suggest that tests of EF should be an inherent part of clinical aphasia assessment.

35

**Keywords:** Executive functions; Cognitive control; Working memory; Conduction aphasia; *n*-back; Aphasia assessment.

---

Address correspondence to: Lilla Zakariás, Institute of Education, Eötvös Loránd University, Budapest, Hungary. E-mail: [lilla.zakarias@gmail.com](mailto:lilla.zakarias@gmail.com)

We thank Klára Marton for discussions on the study and her comments on a previous version of the manuscript, and László Jenei for his help in analysing neurological data.

Recent studies imply that executive functions (EF) are closely related to our ability to comprehend and produce language (Novick, Trueswell, & Thompson-Schill, 2010). EF are generally defined as a range of cognitive processes that enable us to control and regulate various cognitive processes and thereby behaviour (e.g., Miyake et al., 2000). These functions do not add up to a unitary construct, and are always considered as a set of functions or components, such as shifting between tasks or mental sets, updating and monitoring of working memory representations and inhibition of prepotent responses (Miyake et al., 2000).

More and more studies investigating aphasia emphasise the role of EF in successful communication, particularly in conversation (Alexander, 2006; Frankel, Penn, & Ormond-Brown, 2007; Green et al., 2010; Penn, Frankel, Watermeyer, & Russell, 2010; Purdy, 2002; Ramsberger, 2005). These findings suggest that conversational success depends not only on language ability but on EF as well. Based on the model of Barkley (1997), Penn et al. (2010) suggested separate roles for inhibition and working memory in discourse features: these EF components seem to be important in maintaining focus, initiating new topics, planning and monitoring our communicative performance, including shifting between communication strategies to successfully convey information (Ramsberger, 2005) or effectively generating self-repair to error correction (Penn et al., 2010).

Others have suggested that EF also play a role in recovery from aphasia (Green et al., 2010; Penn et al., 2010; Ramsberger, 2005). For instance, differences in executive abilities may account for different language recovery patterns in bilingual aphasia (Green et al., 2010). Moreover, it seems that executive deficits also have an influence on therapy outcome, because the ability to generate, select and apply strategies is important in utilising trained methods (Keil & Kaszniak, 2002).

In line with these results, some studies have investigated the influence of cognitive therapy, particularly EF training, on language improvement (Hardin & Ramsberger, 2004, cited by Helm-Estabrooks & Albert, 1991; Helm-Estabrooks, Connor, & Albert, 2000; Ramsberger, 2005). After delivering attention-training programme, Helm-Estabrooks et al. (2000) revealed improved performance on tasks measuring auditory comprehension and visual analytic reasoning. Similarly, Hardin and Ramsberger (2004) noted that attention/executive training can lead to improvement of transactional success in conversation.

In brief, understanding executive processes is relevant to understanding aphasia (e.g., Code, Tree, & Dawe, 2009; Green et al., 2010), and although EF and their relationship to certain language symptoms have already been investigated in aphasia (e.g., Alexander, 2006; Penn et al., 2010), research specifically addressing the relationship between executive deficits and different types of aphasia is scarce (Keil & Kaszniak, 2002).

## TMA: A special case of executive dysfunctions?

According to theories linking EF with language symptoms, transcortical motor aphasia (TMA) seems to be of outstanding relevance (Alexander, 2006; Ardila, 2010; Luria, 1973; Novick, Kan, Trueswell, & Thompson-Schill, 2009; Robinson, Blair, & Cipolotti, 1998). In classical terminology, TMA, one of the eight aphasia syndromes, is characterised by nonfluent output, anomia, good auditory comprehension, and relative to spontaneous speech, almost preserved repetition. However, the characteristics

of TMA, and associated lesion sites can vary greatly. Based on such variations, some authors suggested that there are different forms of TMA (e.g., the distinction between TMA and dynamic aphasia, a type of aphasia first described by Luria, 1973). The nature of symptoms and the overlap between brain regions affected in TMA and those associated with EF have led some researchers to propose that language symptoms in TMA arise due to executive dysfunctions (Alexander, 2006; Luria, 1973). 90

TMA can involve many different brain regions overlapping to a large extent with regions associated with EF. Reviewing clinical–anatomical studies, Alexander (2003) pointed out that patients diagnosed with TMA had diverse lesions in many different areas of the left frontal lobe and in structures deep into them. The most common lesion sites were in the dorsolateral frontal cortex (BA 45, 46, 9), typically extending into the deep white matter, the ventrolateral (BA 44, 45, 47) and medial frontal lobe (BA 24, 32), including the supplementary motor area (BA 6, 32). Given the large overlap of these areas with neural networks involved in EF (for localisation of EF, see Botvinick, Cohen, & Carter, 2004; Milham et al., 2001; Miyake et al., 2000; Smith, Jonides, Marshuetz, & Koeppe, 1998), Alexander (2006) posited two different executive processes that subservise complex language use. According to his account, the left medial frontal lobe is critical for the activation of language responses, whereas the left lateral frontal areas are necessary to exert control (e.g., inhibition, suppressing, sustaining and monitoring) over procedures implementing syntax and narrative discourse. Disruption of these control processes will, depending on the site and the extension of the lesion, manifest in language use in aphasia to different degrees. More specifically, the above-mentioned EF disturbances might lead to the impairment of complex syntax implementation, lexical selection and difficulties in narrative discourse in TMA. In the framework proposed by Alexander (2006), the level of control procedure impairment, partly associated with lesions in different frontal loci, determines the level of language impairment, leading to hierarchically organised types of aphasia related to TMA. 95 100 105 110

In a similar vein, Ardila (2010) proposed that TMA is not a primary aphasic syndrome in terms of the underlying impairment. Rather, TMA is “an executive function defect specifically affecting language use” (Ardila, 2010, p. 374–375). He argued that TMA patients’ primary language skills are intact, but demonstrate the characteristics of dysexecutive syndrome specifically with regard to verbal processes. Hence, he also has proposed that TMA should be referred to as “dysexecutive aphasia”. 115

Convergent evidence for TMA language symptoms as manifestations of an executive deficit is provided by a few case studies. Some authors, investigating the purest form of TMA, dynamic aphasia, reported patients whose language deficits arose only under certain conditions (Luria, 1973; Novick et al., 2009; Robinson et al., 1998). Following injury to the left inferior frontal gyrus, these patients presented a conspicuous lack of verbal fluency only on tasks in which conflicting representations had to be resolved by executive processes. They have been characterised by an almost complete lack of spontaneous speech in contrast to well-preserved naming, comprehension, repetition and reading skills. 120 125

Robinson et al. (1998) described the case of A. N. G., who presented extremely reduced speech during conversation but had no difficulty in a confrontation-naming task or when she had to generate sentences from a pictorial scene. Moreover, the authors revealed that A. N. G. had difficulties on the structurally analogous tasks depending on how many verbal responses could be activated. For example, in a sentence-completion task, she appropriately completed almost all the sentences where 130

only few continuations were possible, whereas her performance was significantly impaired when trying to complete sentences with more response options. The authors concluded that difficulties of patients with dynamic aphasia can be explained by the inability to select from competing response options. According to Robinson and colleagues, this might also explain the nonfluency of A. N. G.'s spontaneous speech. 135

Novick et al. (2009) also investigated a single patient's, I.G.'s, performance on several conflict-resolution tasks. These included a proactive-interference task using letter stimuli, a picture-naming task using stimuli of varying name agreement (low-agreement stimuli depicting objects with multiple names, e.g., couch, sofa, loveseat vs. high-agreement stimuli depicting objects with a unique name, e.g., apple), a verbal-fluency task and a comprehension task involving syntactic ambiguity. Similar to Robinson et al. (1998), Novick and colleagues concluded that "I. G. had a general conflict resolution impairment which affects his ability to produce and comprehend language under specific conditions" (Novick et al., 2009, p. 528). 140 145

### Measuring EF in TMA

Despite the growing interest in the relationship between EF and aphasia, and in particular TMA, "pure" nonverbal executive skills have not yet been systematically investigated in this type of aphasia. Following a review of studies focusing on EF in aphasia Keil and Kaszniak (2002) concluded that performance on most of the widely used EF tests require language-related processes, which poses serious limitations on their use in populations with aphasia. In addition, they suggested that tests meant to measure EF in individuals with aphasia should mitigate psychomotor slowing and avoid motor processing speed confound (Keil & Kaszniak, 2002). 150 155

As a further step in the understanding of the exact nature of executive processes in TMA, our study aimed to assess two different components of EF which are crucial for language abilities like lexical selection, successful conversation and narrative discourse, in a group of individuals with TMA. Based on the framework of Penn et al. (2010) we focused on updating and monitoring of working memory representations and inhibition of dominant responses. Working memory processes have been proposed to support shifting, maintaining topics during conversation, integrating new information with current communicative content and organising communicative behaviour across time (Frankel et al., 2007; Penn et al., 2010). Inhibition of dominant responses, according to this framework involves two different types of inhibition processes that are involved in different aspects of language processing and production. The ability to stop a prepotent response is proposed to be necessary to recognise and to stop ineffective communicative strategies and to shift to an effective one. Inhibition-based interference control, on the other hand, would make it possible to sustain the topic of a conversation, and the communicative goal in the face of distractors, and inhibiting irrelevant information. This type of inhibition is also important for selecting appropriate lexical and syntactic representations in cases of competition (e.g., Novick, Trueswell, & Thompson-Schill, 2005; Schnur, Lee, Coslett, Schwartz, & Thompson-Schill, 2005). 160 165 170 175

Similar distinctions have been made by Novick et al. (2005) who suggested different inhibitory processes for the resolution of response-based and representational conflict (i.e., response inhibition versus inhibition-based interference control in the Penn et al., 2010 framework). Recently, it has been suggested that two classical inhibitory paradigms, the Stop-signal task and the Stroop task not only differ in their complexity 180

but also in the type of conflict that has to be resolved during performing them. Whereas the Stop-signal task is supposed to tap the resolution of response-based conflict, the Stroop task is more likely to tap resolution of representational conflict. In line with these suggestions, successful performance on the Stop-signal task have been correlated with activations in medial frontal areas, whereas success in the Stroop task have been shown to correlate with ventrolateral frontal activity (Milham et al., 2001; Novick et al., 2005). Therefore, in assessing inhibitory functions in aphasia, we used both the Stroop and the Stop-signal paradigm. 185

To see whether any pattern of impairment found is specific to TMA, we also included a group of patients with conduction aphasia as controls. Two major reasons motivated our choice of a group with conduction aphasia. First, we intended to include a control group with different neural networks underlying symptoms and possible cognitive dysfunctions, but with similar level of auditory comprehension necessary to perform the experimental tasks. Second, although working memory, and in particular verbal working memory deficits have been reported in both types of aphasia, the way these deficits manifest in language seems to be different. In addition, in conduction aphasia, these deficits have been primarily related to an impaired storage capacity of the phonological loop (e.g., Friedmann & Gvion, 2003; Gvion & Friedmann, 2012), disturbances in TMA have been associated also to impaired manipulation of representations stored in working memory. Taken together, we expected to find different patterns of performance on tasks measuring EF. 190 195 200

We designed four nonverbal tasks. In order to avoid confounds summarised by Keil and Kaszniak (2002), all tasks involved within-subject baseline conditions. The four tasks were variations of widely used EF tasks: a nonverbal Stroop and a Stop-signal task measuring inhibition of prepotent responses (Logan, 1994; Milham et al., 2001; Novick et al., 2005; Stroop, 1935) and two variations (one in the auditory and one in the visual modality) of the *n*-back task to measure updating of working memory representations (Miyake et al., 2000). Based on earlier findings we expected to detect a specific pattern of executive deficits in TMA that would be clearly different from that observed among healthy controls and in conduction aphasia. 205 210

## METHODS

### Participants

A total of five individuals with TMA (age:  $M = 58$  years,  $SD = 13.60$ ; 1 female, 4 males; education:  $M = 12.6$ ,  $SD = 2.6$ ;  $RAVEN_{age\ corrected}$ :  $M = 48.92$ ,  $SD = 13.81$ ) participated. As controls, a group with conduction aphasia ( $n = 5$ ; age:  $M = 53$  years,  $SD = 4.84$ ; 1 female, 4 males; education:  $M = 11.2$ ,  $SD = 2.05$ ;  $RAVEN_{age\ corrected}$ :  $M = 33.60$ ,  $SD = 7.56$ ) and a group of healthy participants ( $n = 10$ ; age:  $M = 59.5$  years,  $SD = 12.26$ ; 6 female, 4 males; education:  $M = 12.9$ ,  $SD = 2.96$ ;  $RAVEN_{age\ corrected}$ :  $M = 53.77$ ,  $SD = 11.69$ ) were recruited. Healthy controls were matched in age and education. All participants with aphasia have had a single left hemisphere infarct, confirmed by CT or structural MRI, except one, who has had a traumatic injury, also to the left hemisphere. The mean time post-onset was 8.4 months for the group with TMA and 8.6 months for the group with conduction aphasia. All of them spoke Hungarian as their primary language and were right-handed. They had been recruited and tested at two rehabilitation centres in Budapest, Hungary: the Flór Ferenc Hospital and the National Institute for Medical Rehabilitation. 215 220 225



Their language impairment was classified by the Western Aphasia Battery (Kertész, 1982; Hungarian adaptation: Osmáné Sági, 1991) complemented with the Boston Naming Test (Kaplan, Goodglass, Weintraub, & Segal, 1983). Neurological assessment showed no visual problems for any of the patients, and all of them reported hearing within normal limits. Kruskal–Wallis tests showed that there were no significant differences between the subject groups in terms of age ( $H(2) = 3.71$ , ns.), education ( $H(2) = 0.93$ , ns.) and intelligence ( $H(2) = 4.79$ , ns.).

Table 1 summarises the characteristics of all participants with aphasia. In the current study, we used the diagnostic label “transcortical motor aphasia” to refer to patients with aphasia whose language output was nonfluent, extremely reduced, fragmentary echoic and perseverative after 1 month post-onset. Their performance was also impaired on picture-naming, but to a remarkably lower extent than in spontaneous speech. Naming reflected word-finding difficulties, most frequently hesitations, pauses and perseverative errors. Comprehension at the word level as well as at the level of one-part commands was intact, but showed problems at the level of two-part commands. Repetition was good, nearly normal for all participants. During therapy, output became more fluent but still anomic, especially in conversation. In naming, they demonstrated only a milder anomia with long latencies and hesitations. Comprehension and repetition developed to a normal level.

Participants with conduction aphasia showed good auditory comprehension, fluent spontaneous speech interrupted by phonemic paraphasias and self-correction of errors. Compared to spontaneous speech, repetition was severely impaired. Word-finding problems were prominent in naming, coupled with phonemic paraphasias and pauses.

## Materials, designs and procedures of the EF tasks

To assess EF, we designed four nonverbal tasks that reduced the influence of impaired linguistic ability on task performance. We focused on two major processes related to EF, updating of working memory representations and inhibition of prepotent responses. All experiments were run by E-Prime (Psychology Software Tools, Pittsburgh, PA, Version 1.2) except for the auditory  $n$ -back task that was programmed and run by Presentation<sup>®</sup> software (Version 14.1) on an IBM T40p thinkpad. Participants used the buttons on the keyboard to respond. All participants completed the experiments in two sessions, each lasting 1–1.5 hours, depending on the length of self-paced pauses between the experimental tasks.

### *Tasks measuring updating of working memory representations*

We designed two modified  $n$ -back tasks, one relying on auditory processing and the other relying on visual processing. The  $n$ -back task is generally used to index updating of information maintained in working memory (e.g., Miyake et al., 2000).

#### *Auditory $n$ -back task*

Participants were exposed to a stream of tones. One tone was presented on each trial and participants had to respond when the stimulus presented was identical to the one appearing in  $n$  trials before. We varied  $n$  within subjects, and all participants performed the  $n$ -back task with  $n = 1$ , then with  $n = 2$ . In both conditions, the task

TABLE 1  
Background description of the participants with aphasia

Participant	Age	Gender	Education	Aetiology	Lesion	Time post-onset (months or days)	Aphasia type (WAB profile)	Aphasia quotient (WAB AQ)	WAB subtests' scores					
									Spontaneous speech		Comprehension	Repetition	Naming	Boston scores
									IC	Flu				
H. J.	63	M	17	CVA	Infarct of the left MCA	7.5 m	TMA	76.1	6	5	9.85	10	7.2	29
T. I.	66	M	11	CVA	Left frontal infarct	12 m	TMA	85.4	9	8	7.9	10	7.8	41
K. J.	69	M	11	CVA	Left fronto-parietal infarct	9.5 m	TMA	90.1	9	9	9.05	10	8	43
N. F.	57	M	11	CVA	Left frontal infarct	12 m	TMA	94	9	9	10	10	9	57
P. T.	35	F	13	CVA	Left frontal and insular infarct	1 m	TMA	68.4	7	5	9.6	8	4.6	29
V. V.	55	M	11	CVA	Infarct of the left MCA	2 m	Conduction	63.6	7	6	8.2	5.2	5.4	22
O. Gy.	48	M	13	TBI	Bilateral parietal contusion	6 m	Conduction	44.8	5	6	7.6	1.6	2.2	4
Sz. I.	59	M	13	CVA	Left parietal infarct	14 m	Conduction	62	7	6	8.3	4.7	5	18
K. J.	48	M	11	CVA	Left fronto-temporal infarct	21 m	Conduction	43.1	5	5	7.35	1	3.2	5
Cz. J.	55	F	8	CVA	Left fronto-temporal infarct	5 d	Conduction	77.4	8	8	9.3	6.4	7	28

Note: CVA: cerebrovascular accident, TBI: traumatic brain injury, MCA: middle cerebral artery, TMA: transcortical motor aphasia, IC: information content, Flu: fluency.



consisted of 5 blocks of 30 trials. Blocks were separated by self-paced resting periods. The first blocks in both conditions were used as practice blocks. The results show data from Blocks 2–5 in both conditions. On each trial, a sound was sampled from a pool of eight pure frequency sounds (ca. half sounds starting from the standard musical note A5: 440 Hz, 490 Hz, 540 Hz, 590 Hz, 640 Hz, 690 Hz, 740 Hz and 790 Hz). Sampling was fully randomised so that on each trial the chance of sampling a sound that was presented  $n$  trials before was one to four. In each trial, the sound was presented for 300 ms, followed by a silent period of 1000 ms, during which participants had time to respond. Trials were separated by a 500 ms intertrial interval. In the practice blocks, all trials were followed by a 1000 ms feedback trial if the participant pressed the response button. No feedback was provided in Blocks 2–5.

### *Visual n-back task*

Participants were exposed to a stream of pictures from 14 different semantic categories (e.g., dogs, windows). One picture appeared on each trial and participants had to respond by pressing the ENTER on the keyboard when the stimulus presented was from the same semantic category as the one presented  $n$  trials before. We varied  $n$  within subjects, and all participants performed the  $n$ -back task with  $n = 1$ , then with  $n = 2$ . In both conditions, the task consisted of 60 trials. On each trial, a picture was sampled from a pool of pictures of a given semantic category. Sampling was pseudorandomised so that in both conditions for all participants, 10 trials required a hit response. In each trial, the picture was presented in the middle of the screen for 2500 ms. Trials were separated by a 500 ms intertrial interval.

### *Tasks measuring inhibition*

We used two modified inhibition tasks to measure different types of conflict resolution (Lukács, Kemény, Fazekas, Ladányi, & Németh, in prep.). The Stop-signal task is generally used to index the ability to resolve response-based conflict through inhibition (Logan, 1994; Milham et al., 2001), while the Stroop task is generally used to assess the ability to resolve representational conflict through inhibition (Novick et al., 2005; Stroop, 1935).

### *Stop-signal task*

On each trial, a stimulus (either a circle or a square) appeared in the middle of the screen for 2000 ms, and participants had to respond as fast as possible by pressing the corresponding button on the keyboard (“c” for circle, “b” for square). Trials were separated by a 250 ms fixation trial (a fixation cross was presented in the middle of the screen and the participant had to fixate on it until the next trial was presented). On some trials, a loud tone was presented after the onset of the stimulus that signalled to the participants that they should refrain from responding (stop trials). Delay of tone onset (Stimulus onset Asynchrony—SOA) was varied within subjects, so that it was increased from 50 ms to 350 ms by steps of 50 ms, through seven blocks. Each block consisted of 60 trials, with 15 stop trials and 45 go trials (trials where the loud tone was absent) randomly intermixed. The seven blocks were separated by self-paced resting periods. Following previous work by Logan (1994), we used proportion of correct rejections (not pressing any button on a stop trial) as a measure of inhibition.

### *Nonverbal Stroop task*

On each trial an arrow was presented on the screen for 3000 ms, with four possible directions (left, right, up, down) and four possible positions relative to the centre of the screen (left, right, over, below). We varied congruency of position and direction within subjects. In the congruent condition, direction matched position (e.g., an arrow pointing to the left, presented on the left side of the screen). In the incongruent condition, direction did not match position (e.g., an arrow pointing to the left, presented on the right side of the screen). Participants had to press the arrow button on the keyboard corresponding to the direction of the arrow on the screen as fast as possible.

This experiment started with 60 control trials that also helped participants to familiarise with matching directions to buttons. In each control trial, an arrow appeared in the middle of the screen (pointing to either of the four possible directions) and participants had to press the corresponding arrow button on the keyboard. (Note that there were no congruent or incongruent trials during these control trials.) Congruent and incongruent trials were blocked so that each participant performed a block of 60 congruent trials followed by a block of 60 incongruent trials. The two blocks were separated by a self-paced resting period. As a measure of inhibition, we used the difference between proportions of errors in the incongruent vs. congruent conditions.

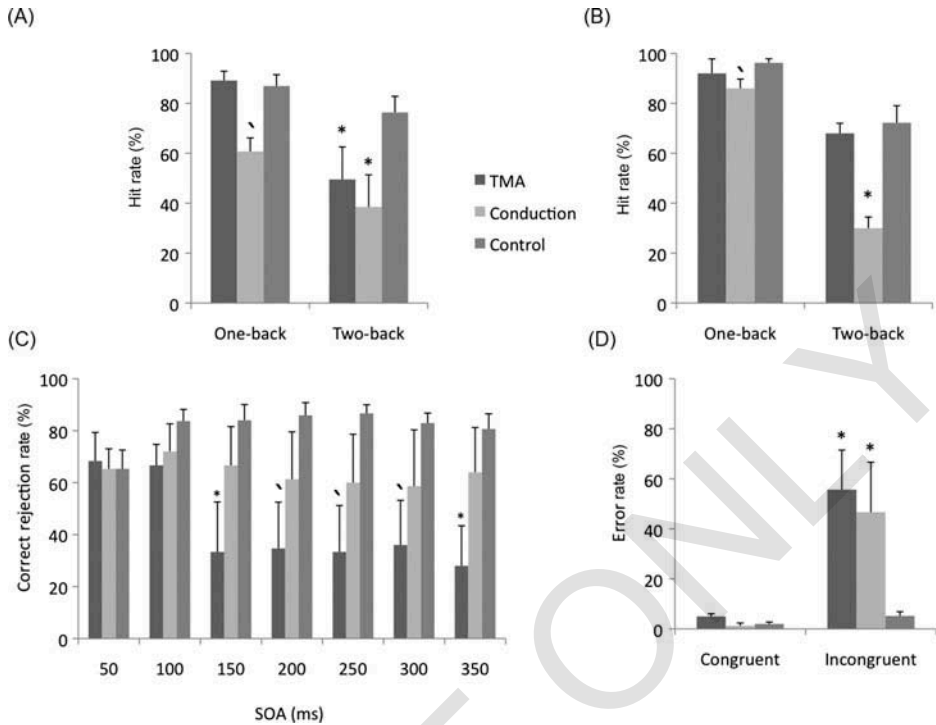
## RESULTS

Data were screened for outliers. We excluded all data points that were more than two standard deviations from the group mean (we performed this analysis separately for healthy controls and participants with aphasia). Altogether, less than 3% of the data were removed. For each experimental task, we compared the performance of participants with TMA and conduction aphasia to that of healthy controls. For all comparisons reported we used the nonparametric equivalent of the independent *t*-test, the Mann–Whitney test.

### *Auditory *n*-back task*

Average hit rates for the one-back condition and the two-back condition in the three groups are presented in Figure 1(A). Mann–Whitney tests showed that TMA participants' hit rate in the two-back condition was significantly lower than that of healthy controls,  $U = 5.0, p = .05$ . This occurred despite the fact that their performance did not differ in the one-back condition,  $U = 15, p = .64$ . That is, impaired updating performance was accompanied by an intact ability to discriminate between sounds. Hit rates of patients with conduction aphasia in the two-back condition also differed from that of healthy controls,  $U = 5.5, p = .05$ . However, their hit rates were already lower (at the level of tendency) than that of healthy controls in the one-back condition,  $U = 7.5, p = .1$ .

Follow-up Mann–Whitney tests showed that false alarm rates did not differ significantly from those of healthy controls, in either the one-back ( $U = 9, p = .20$  for both TMA vs. controls, and conduction vs. controls contrasts) or the two-back condition ( $U = 11, p = .33$  for the TMA vs. controls contrast, and  $U = 16, p = .83$  for the conduction vs. controls contrast).



**Figure 1.** Indicators of executive functions measured in four tasks for the three experimental groups. Asterisks indicate significant differences between the patient and the control groups ( $*p < .05$ ), primes indicate tendencies for differences between the patient and the control groups ( $'p < .1$ ). (A) Auditory  $n$ -back task, with  $n = 1$  and  $n = 2$ . Updating of working memory is assessed by hit rates in the two-back condition. (B) Visual  $n$ -back task, with  $n = 1$  and  $n = 2$ . Updating of working memory is assessed by hit rates in the two-back condition. (C) Stop-signal reaction time task, with seven levels of stimulus onset asynchrony (SOA) defined as the onset time of the Stop-signal minus the time onset of the target stimulus. Resolution of response-based conflict through inhibition is assessed by the rate of correct rejections on trials where a Stop-signal occurred (stop trials). (D) Nonverbal Stroop task, with a congruent and an incongruent condition. Resolution of representational conflict through inhibition is assessed by the difference in error rates between congruent and incongruent conditions. Asterisks in this panel mean that this difference in both patient groups was significantly larger than in the control group. Error bars represent standard error of the mean.

### Visual $n$ -back task

Average hit rates for the one-back condition and the two-back condition in the three groups are presented in Figure 1(B). Mann–Whitney tests showed that performance of patients with TMA did not differ from that of healthy controls in either conditions,  $U = 18$ ,  $p = .83$  for the one-back, and  $U = 20.5$ ,  $p = .79$  for the two-back conditions. In contrast, patients with conduction aphasia performed worse than healthy controls in the one-back condition at the level of tendency,  $U = 7$ ,  $p = .065$ , and their performance was significantly worse than that of healthy controls in the two-back condition,  $U = 1$ ,  $p = .002$ .

Again, follow-up Mann–Whitney tests showed that false alarm rates did not differ significantly from those of healthy controls, in either the one-back ( $U = 14$ ,  $p = .80$  for

the TMA vs. controls contrast, and  $U = 9.5$ ,  $p = .13$  for the conduction vs. controls contrasts) or the two-back condition ( $U = 15$ ,  $p = .52$  for the TMA vs. controls contrast, and  $U = 11.5$ ,  $p = .46$  for the conduction vs. controls contrast).

370

### Stop-signal task

We plotted the percentage of correct rejections at all seven SOAs for the three groups in Figure 1(C). This measure shows how often participants could successfully stop responding on stop trials, i.e., refrain from pressing any response button, when a Stop-signal required them to do so.

375

As can be seen in Figure 1(C), the pattern of performance changed, as a function of SOAs, in different ways in the three groups. Mann–Whitney tests confirmed this pattern. At SOAs 150 through 350 TMA patients refrained from stopping their answer on stop trials less often than healthy controls, although this difference did not reach the level of significance at all SOAs ( $U = 8.5$ ,  $p = .039$  at SOA = 150 ms,  $U = 8.0$ ,  $p = .051$  at SOA = 200 ms,  $U = 8.5$ ,  $p = .059$  at SOA = 250 ms,  $U = 8.5$ ,  $p = .061$  at SOA = 300 ms, and  $U = 6$ ,  $p = .019$  at SOA = 350 ms.) The same comparisons between the conduction aphasia and the control groups did not yield any significant differences (all  $Us > 13$ , ns.).

380

### Nonverbal Stroop task

385

We plotted the error rates in both the congruent and the incongruent conditions for the three groups in Figure 1(D). A larger difference in error rates between the congruent and the incongruent condition indicates a lower degree of representational conflict resolution through inhibition.

Mann–Whitney tests showed that difference in error rates between the congruent and the incongruent condition among TMA participants was significantly higher than among healthy controls,  $U = 0.0$ ,  $p = .003$ . Similarly, patients with conduction aphasia also produced significantly more errors in the incongruent than in the congruent condition compared to healthy controls,  $U = 3.5$ ,  $p = .024$ .

390

## DISCUSSION

395

In the current study, we used four nonverbal tasks to investigate different components of EF in TMA and in conduction aphasia in an attempt to test whether executive dysfunctions are specific to TMA. Our results demonstrate executive deficits among individuals with transcortical motor *and* with conduction aphasia. Importantly, our data revealed different patterns of performance in the two aphasia types.

400

Deficits of EF among individuals with TMA was evident on several measures: compared to healthy controls, these participants were impaired in resolving response-based conflict (as shown by their performance on the Stop-signal task), in resolving representational conflict through inhibition (as shown by results of the nonverbal Stroop task) and in updating working memory representations (as shown by results of the auditory *n*-back task).

405

Importantly, these results are in line with theories (Alexander, 2006; Ardila, 2010; Luria, 1973) predicting that TMA patients will present extensive EF deficits. Earlier, it has been shown that impaired ability to resolve representational conflict results in difficulties in lexical selection and in impairment of syntax (e.g., Alexander, 2006;

410

Novick et al., 2009). These in turn might explain word-finding difficulties, hesitations and reduced grammar in a nonfluent spontaneous speech in TMA, and in dynamic aphasia. Poor ability to resolve representational conflicts can also lead to unwanted interruptions, sudden topic changes and a general difficulty to stay on topic in narrative discourse (Penn et al., 2010). On the other hand, the inability to resolve response-based conflict can lead to perseverations of communicative strategies. In addition, the disrupted ability to update working memory representations can disturb management of the temporal integration of conversations (Penn et al., 2010). Although this updating deficit was not observed in our visual *n*-back task, it is possible that the two-back condition of the task was not demanding enough to tap the differences between healthy controls and participants with TMA, and that administering a three-back condition might reveal significant differences. Taken together, we suggest that TMA patients have deficits in both inhibition and updating which might explain a range of narrative discourse problems often observed in TMA (e.g., Alexander, 2006).

Our findings also clearly demonstrate that TMA is not the only type of aphasia exhibiting executive dysfunctions: individuals with conduction aphasia also performed poorer than healthy controls on several EF measures.

First, as evidenced by results of the Stroop task, compared to healthy controls, these participants were impaired in resolving representational conflict. Second, they performed generally worse than healthy controls on the auditory *n*-back task. Their performance was already below that of healthy controls in the one-back condition which might be attributed to deficits of both auditory discrimination and working memory. For the interpretation of these results, it is important to note that (see Figure 1(A)) the pattern of performance in the two groups was similar, i.e., increasing working memory load (from one-back to two-back) did not decrease performance in conduction aphasia more than in healthy controls, as one would expect in case of a marked deficit of updating functions (Vasic, Walter, Sambataro, & Wolf, 2009; Waltz et al., 2004). Unfortunately, the fact that the possible deficits in working memory and auditory discrimination are confounded in this task puts limitations on the use of auditory *n*-back task in investigating EF in conduction aphasia. Third, in the visual modality, we observed a clear deficit of updating working memory representations in conduction aphasia. Because the pictures used in this task depicted everyday objects for which both object names and category names are easy to verbalise, participants could lean in part on subvocal rehearsal (Baddeley, Eysenck, & Anderson, 2009). Subvocal rehearsal is known to be affected in conduction aphasia (Buchsbaum et al., 2011), and this might have resulted in their lower performance in the two-back condition of this task.

In brief, the poor level of performance on the *n*-back tasks might be caused by different impairments in the two aphasia types. Whereas in TMA, the deficit of updating seems to be the main factor explaining the results of the *n*-back task, in conduction aphasia, deficits in subvocal rehearsal (in the case of the visual *n*-back task) and auditory discrimination (in the case of the auditory *n*-back) may also contribute to poor performance.

The different patterns of performance displayed in the two types of aphasia on the two inhibitory tasks (with both groups showing deficits on the nonverbal Stroop task, but only the TMA group showing a deficit on the Stop-signal task) support the view (Milham et al., 2001; Novick et al., 2005) that these tasks indeed measure distinct components of inhibitory executive processes. Novick et al. (2009, 2010) suggested that the resolution of representational conflict, conventionally indexed by the Stroop

task, is associated with language abilities. Accordingly, the same authors as well as others (Robinson et al., 1998) have suggested that this type of conflict resolution is associated with TMA. Our results provide support for this suggestion but also show that the impairment of representational conflict resolution is present in conduction aphasia as well. The results of the Stop-signal task used in our study provide evidence for the involvement of response-based conflict resolution in TMA, but not in conduction aphasia. Based on Penn et al. (2010) we suggest that these inhibitory deficits might have separable contributions to narrative discourse impairment in TMA, and lead together to the overall pattern of language symptoms in TMA.

Our study is the first systematic assessment of EF in aphasia to demonstrate clear and extensive executive deficits among TMA patients. We hope that it might serve as a starting point for future research addressing the exact relationship between different EF components and language abilities. The most intriguing question for this line of research is the causal relationship between EF deficits and language disorders. Earlier results suggest that EF deficits might manifest in language symptoms in TMA. In conduction aphasia, however, it is possible that the observed EF deficits are only associated with language symptoms, but are not causal in their development. Whatever the answer to these questions, examination of EF components and training of EF should be an essential part of clinical aphasia assessment and rehabilitation.

Manuscript received 30 March 2013  
 Manuscript accepted 14 August 2013  
 First published online Day Month Year

480  
 AQ6

## REFERENCES

- Alexander, M. P. (2003). Transcortical motor aphasia: A disorder of language production. In M. D'Esposito (Ed.), *Neurological foundations of cognitive neuroscience* (pp. 165–174). London: MIT.
- Alexander, M. P. (2006). Impairments of procedures for implementing complex language are due to disruption of frontal attention processes. *Journal of the International Neuropsychological Society*, *12*(2), 236–247.
- Ardila, A. (2010). A proposed reinterpretation and reclassification of aphasic syndromes. *Aphasiology*, *24*(3), 363–394.
- Baddeley, A., Eysenck, M. W., & Anderson, M. C. (2009). *Memory*. Hove: Psychology Press.
- Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin*, *121*(1), 65.
- Botvinick, M. M., Cohen, J. D., & Carter, C. S. (2004). Conflict monitoring and anterior cingulate cortex: An update. *Trends in Cognitive Sciences*, *8*(12), 539–546.
- Code, C., Tree, J. J., & Dawe, K. (2009). Opportunities to say “yes”: Rare speech automatisms in a case of progressive nonfluent aphasia and apraxia. *Neurocase*, *15*(6), 445–458.
- Frankel, T., Penn, C., & Ormond-Brown, D. (2007). Executive dysfunction as an explanatory basis for conversation symptoms of aphasia: A pilot study. *Aphasiology*, *21*(6–8), 814–828.
- Friedmann, N., & Gvion, A. (2003). Sentence comprehension and working memory limitation in aphasia: A dissociation between semantic-syntactic and phonological reactivation. *Brain and Language*, *86*(1), 23–39.
- Green, D. W., Grogan, A., Crinion, J., Ali, N., Sutton, C., & Price, C. J. (2010). Language control and parallel recovery of language in individuals with aphasia. *Aphasiology*, *24*(2), 188–209.
- Gvion, A., & Friedmann, N. (2012). Does phonological working memory impairment affect sentence comprehension? A study of conduction aphasia. *Aphasiology*, *26*(3–4), 494–535.
- Hardin, K. H., & Ramsberger, G. (2004). *Treatment of attention in aphasia: A case study*. Poster presented at Clinical Aphasiology Conference, Park City, UT.
- Helm-Estabrooks, N., & Albert, M. L. (1991). *Manual of aphasia therapy*. Austin, TX: Pro-Ed.
- Helm-Estabrooks, N., Connor, L. T., & Albert, M. L. (2000). Treating attention to improve auditory comprehension in aphasia. *Brain and Language*, *74*, 469–472.



- Kaplan, E., Goodglass, H., Weintraub, S., & Segal, O. (1983). *Boston naming test*. Philadelphia, PA: Lea & Febiger.
- Keil, K., & Kaszniak, A. W. (2002). Examining executive function in individuals with brain injury: A review. *Aphasiology, 16*(3), 305–335.
- Kertész, A. (1982). *The Western aphasia battery*. New York, NY: Grune-Stratton. 515
- Logan, G. D. (1994). On the ability to inhibit thought and action: A users' guide to the stop signal paradigm. In D. Dagenbach & T. H. Carr (Eds.), *Inhibitory processes in attention, memory, and language* (pp. 189–239). San Diego, CA: Academic Press.
- Lukács, Á., Kemény, F., Fazekas, K., Ladányi, E., & Németh, K. (in prep.) Verbal and nonverbal executive functions in Specific Language Impairment. 520
- Luria, A. R. (1973). *The working brain: An introduction to neuropsychology*. Basic Books. AQ4 AQ5
- Milham, M. P., Banich, M. T., Webb, A., Barad, V., Cohen, N. J., Wszalek, T., & Kramer, A. F. (2001). The relative involvement of anterior cingulate and prefrontal cortex in attentional control depends on nature of conflict. *Cognitive Brain Research, 12*(3), 467–473.
- Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex “frontal lobe” tasks: A latent variable analysis. *Cognitive Psychology, 41*(1), 49–100. 525
- Novick, J. M., Kan, I. P., Trueswell, J. C., & Thompson-Schill, S. L. (2009). A case for conflict across multiple domains: Memory and language impairments following damage to ventrolateral prefrontal cortex. *Cognitive Neuropsychology, 26*(6), 527–567. 530
- Novick, J. M., Trueswell, J. C., & Thompson-Schill, S. L. (2005). Cognitive control and parsing: Reexamining the role of Broca's area in sentence comprehension. *Cognitive, Affective, & Behavioral Neuroscience, 5*(3), 263–281.
- Novick, J. M., Trueswell, J. C., & Thompson-Schill, S. L. (2010). Broca's area and language processing: Evidence for the cognitive control connection. *Language and Linguistics Compass, 4*(10), 906–924. 535
- Osmán Sági, J. (1991). Az afázia klasszifikációja és diagnosztikája. *Ideggyógyászati Szemle, 44*, 339–362.
- Penn, C., Frankel, T., Watermeyer, J., & Russell, N. (2010). Executive function and conversational strategies in bilingual aphasia. *Aphasiology, 24*(2), 288–308.
- Purdy, M. (2002). Executive function ability in persons with aphasia. *Aphasiology, 16*(4–6), 549–557. 540
- Ramsberger, G. (2005). Achieving conversational success in aphasia by focusing on non-linguistic cognitive skills: A potentially promising new approach. *Aphasiology, 19*(10–11), 1066–1073. 540
- Robinson, G., Blair, J., & Cipolotti, L. (1998). Dynamic aphasia: An inability to select between competing verbal responses? *Brain, 121*(1), 77–89.
- Schnur, T. T., Lee, E., Coslett, H. B., Schwartz, M. F., & Thompson-Schill, S. L. (2005). When lexical selection gets tough, the LIFG gets going: A lesion analysis study of interference during word production. *Brain and Language, 95*(1), 12–13. 545
- Smith, E. E., Jonides, J., Marshuetz, C., & Koeppel, R. A. (1998). Components of verbal working memory: Evidence from neuroimaging. *Proceedings of the National Academy of Sciences, 95*(3), 876–882.
- Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18*, 643–662. 550
- Vasic, N., Walter, H., Sambataro, F., & Wolf, R. C. (2009). Aberrant functional connectivity of dorsolateral prefrontal and cingulate networks in patients with major depression during working memory processing. *Psychological Medicine, 39*(6), 977.
- Waltz, J. A., Knowlton, B. J., Holyoak, K. J., Boone, K. B., Back-Madruga, C., McPherson, S., . . . Miller, B. L. (2004). Relational integration and executive function in Alzheimer's disease. *Neuropsychology, 18*(2), 296–305. 555