International Journal of Language & Communication Disorders

INT J LANG COMMUN DISORD, MAY–JUNE 2018,
VOL. 53, NO. 3, 515–525

Research Report

Cognate effects and cognitive control in patients with parallel and differential bilingual aphasia

Lize Van der Linden†, Nele Verreyt‡, Miet De Letter§, Dimitri Hemelsoet¶, Peter Mariën‖#, Patrick Santens¶, Michaël Stevens‡, Arnaud Szmalec†‡ and Wouter Duyck‡

†Psychological Sciences Research Institute, Université catholique de Louvain, Louvain-la-Neuve, Belgium
‡Department of Experimental Psychology, Ghent University, Ghent, Belgium
§Department of ORL & Logopaedic and Audiologic Sciences, Ghent University Hospital, Ghent, Belgium
¶Department of Neurology, University Hospital Ghent, Ghent, Belgium
‖Department of Neurology, ZNA Middelheim Hospital, Antwerp, Belgium
#Clinical and Experimental Neurolinguistics, Vrije Universiteit Brussel, Brussels, Belgium

(*Received April 2017; accepted November 2017*)

Abstract

Background: Until today, there is no satisfying explanation for why one language may recover worse than another in differential bilingual aphasia. One potential explanation that has been largely unexplored is that differential aphasia is the consequence of a loss of language control rather than a loss of linguistic representations. Language control is part of a general control mechanism that also manages non-linguistic cognitive control. If this system is impaired, patients with differential aphasia could still show bilingual language activation, but they may be unable to manage activation in non-target languages, so that performance in another language is hindered.
Aims: To investigate whether a loss of cognitive control, rather than the loss of word representations in a particular language, might underlie differential aphasia symptoms.
Methods & Procedures: We compared the performance of seven bilinguals with differential and eight bilinguals with parallel aphasia with 19 control bilinguals in a lexical decision and a flanker task to assess bilingual language co-activation and non-linguistic control respectively.
Outcomes & Results: We found similar cognate effects in the three groups, indicating similar lexical processing across groups. Additionally, we found a larger non-linguistic control congruency effect only for the patients with differential aphasia.
Conclusions & Implications: The present data indicate preserved language co-activation for patients with parallel as well as differential aphasia. Furthermore, the results suggest a general cognitive control dysfunction, specifically for differential aphasia. Taken together, the results of the current study provide further support for the hypothesis of impaired cognitive control abilities in patients with differential aphasia, which has both theoretical and practical implications.

Keywords: bilingualism, aphasia, parallel aphasia, differential aphasia, cognate effect, executive functions.

What this paper adds

What is already known on the subject

Patients with differential aphasia might have impaired cognitive-control abilities, so that they are unable to inhibit fully (words in) their most dominant language, resulting in loss of functionality in the non-dominant language. At present, the only direct empirical evidence for this hypothesis is a single case study that reported impaired control by showing preserved language interactivity, but impaired language and non-linguistic control abilities in a patient with differential aphasia.

Address correspondence to: Lize Van der Linden, Psychological Sciences Research Institute, Université catholique de Louvain, Place Cardinal Mercier 10, B-1348 Louvain-la-Neuve, Belgium; e-mail: lize.vanderlinden@uclouvain.be

ISSN 1368-2822 print/ISSN 1460-6984 online
DOI: 10.1111/1460-6984.12365

What this paper adds to existing knowledge
The available case study only tested control participants and one patient with differential aphasia, so that it remains unclear whether the observed problems are specific to differential aphasia or to aphasia in general. Here, we compared unusually large groups of patients with differential and parallel aphasia. We found preserved language interactivity and impaired non-linguistic control only in the group of patients with differential aphasia. Additionally, this study showed that patients with parallel aphasia do not suffer from non-linguistic control difficulties, providing further support for the hypothesis that cognitive control difficulties underlie differential language loss, and not all manifestations of aphasia.

What are the potential or actual clinical implications of this work?
This study highlights the importance of considering the assessment and training of cognitive control abilities in patients with differential aphasia.

Introduction

Bilinguals are typically considered as individuals who regularly use two languages, and at present about half the world's population is bilingual (Grosjean 2010). Therefore, it is not surprising that many patients with aphasia are bilingual. Aphasia refers to a general impairment in understanding, formulating or using verbal messages, in spoken and/or written modality, caused by an acquired brain dysfunction in language-related areas (Damasio 1992).

Interestingly, bilinguals who suffer from aphasia do not always have the same amount of impairment in both languages, and different recovery or impairment patterns can be identified (Paradis 2004, Giussani *et al.* 2007). Paradis (2004) described six different patterns of bilingual aphasia. A first type, where both languages recover similarly, is called parallel recovery. Differential recovery occurs when one language recovers more than the other. Selective recovery is an extreme case of differential recovery in which one language does not recover at all. For some patients, one language only starts recovering when the other has fully recovered, which is called successive recovery. There can also be an alternation in recovery: one language starts recovering but weakens again when the other recovers. A last recovery pattern is blended recovery, where patients uncontrollably switch and mix their languages.

The occurrence of differential language loss in bilingual aphasia is interesting for functional and neuroanatomical theories of bilingualism. It shows that languages can be selectively impaired and are therefore likely to be represented in distinct brain structures. However, bilinguals do not have two distinct representational systems, one for each language, but rather one integrated lexicon that contains word representations of both languages (Van Heuven *et al.* 1998). Evidence for this integrated lexicon has often been found with so-called cognate effects. Cognates are words that have the same meaning and a similar orthography and/or phonology in both languages (e.g., the Dutch–English cognates *winter* or *appel–apple*). Typically, cognates are processed faster and more accurately than non-cognates (Duyck *et al.* 2007). This cognate effect is explained by convergent activation spreading from the cognate's similar semantic and orthographic/phonological representations across languages, and hence suggests co-activation of multiple languages. These cross-lingual interactions are not only found during word recognition, but also during production and comprehension, and even when processing language in a monolingual context (e.g., Duyck *et al.* 2007). Furthermore, it has been demonstrated that, at least for balanced bilinguals,[1] languages mostly rely on common brain areas (Abutalebi *et al.* 2001).

From the findings reported above it is not clear how brain damage to a language area may lead to (greater) loss of one specific language, or why bilinguals with aphasia sometimes recover one language more than the other. Already more than a century ago, Pitres (1895) argued that a deficit in language control may underlie differential aphasia rather than an actual loss of language representations. Accordingly, differential aphasia is not the result of a lesion in the language's neural substrate, causing the loss of word representations in one particular language, but rather the result of a problem with ignoring the irrelevant and selecting the relevant, target language. Therefore, according to this view, patients with differential aphasia (PWDA) do not lose a language in itself, but rather the capability to control the co-activation of the most preserved language while attempting to use the most impaired language.

Typically, cross-language intrusions are rare for bilinguals (Poulisse 2000), despite the interactions between languages reported above. This indicates that bilinguals can select only those words belonging to the target language. It has been proposed that this control is done by inhibiting co-activated words in the irrelevant language (Green 1998). The PWDA might be unable to inhibit fully (words in) their most dominant language (L1), because this language is more active and requires more inhibition, so that especially performance in the other,

non-dominant language (L2) is hampered. This inhibition mechanism is not only assumed to manage dual-language activation of bilinguals but also serves as a more general control mechanism, which is involved in non-linguistic control. Consequently, a language-control impairment not only might lead to language control difficulties (in this case, differential aphasia) but also might be associated with a non-linguistic control impairment. The aim of the current study is to relate differential aphasia to such general cognitive control impairment and to explore indications of preserved cross-language activation in PWDA despite (language) control problems.

Previous studies have already found evidence for cross-lingual interactions in bilingual aphasia by studying the preservation of cognate effects. Detry *et al.* (2005), for instance, ran a word–picture verification task, a picture-naming task and a translation task and found cognate effects on all tasks in a French–Italian bilingual patient with aphasia. This finding demonstrates that lexical interactions may at least emerge between languages in patients with aphasia, supporting (partly) preserved lexico-semantic functionality. However, no study thus far has focused on the relative degree of language loss or differentiated between parallel and differential aphasia and, therefore, may not answer the question at hand.

As far as we know, only two case studies have investigated cognate effects specifically in a PWDA. Lalor and Kirsner (2001) described an English–Italian PWDA showing larger deficits in Italian (L2) than in English (L1). They found a partial cognate effect in naming: low-frequent Italian cognates with high-frequent translations were read faster than low-frequent Italian cognates with low-frequent translations. In addition, they also ran a generalized lexical decision (LD) task in which the patient had to decide whether a visually presented word was an existing word, irrespective of the language. They did not find a cognate effect in reaction times (RTs) (probably because of high variability), but they did in accuracy. According to the authors, this indicates co-activation of the cognate in both languages, facilitating performance. The non-linguistic control performance of this patient, however, was not assessed, as this paper appeared long before the current discussion of non-linguistic and language control in the bilingualism literature.

Like Lalor and Kirsner (2001), Verreyt *et al.* (2013) examined cognate effects in a French–Dutch PWDA, but they manipulated language control demands across three different LD tasks. The LD tasks all contained cognates, L1 non-cognates, L2 non-cognates and non-words. There was one generalized and two selective LD tasks (one for each language). While the patient had to indicate whether the presented word was an existing word in a particular language in the selective tasks, he had to indicate whether the word on the screen existed, irrespective of the language, in the generalized task. Language control was not required in the latter task, because all known words, in any language, led to a positive response. In the selective variants, on the other hand, the irrelevant language had to be inhibited because the recognition of the presented L1 non-cognate would lead to an incorrect response in the L2-selective LD task, and vice versa. Verreyt *et al.* only observed cognate facilitation in the generalized LD task. Moreover, cognate interference was found in the L2-selective LD task, indicating that the patient was unable to inhibit the activation of his L1 and was, therefore, falsely rejecting cognates. Finally, neither facilitation nor interference of cognates were found in the L1-selective LD task, indicating that L2 words were inhibited. These findings were taken as evidence that words in the most impaired language in PWDA can still be activated, and lead to facilitation under conditions where no language control is needed, as shown by the cognate effect in the generalized LD task. The inhibition of the L1, on the other hand, is difficult, which hinders the recognition of L2 words. Verreyt *et al.* also examined non-linguistic control abilities using a flanker task (Eriksen and Eriksen 1974). The patient showed more difficulties in ignoring the irrelevant arrows than a control group, revealing problems in dealing with competing responses. Altogether, this case study offers the only relatively direct empirical support for Pitres' (1895) hypothesis of general cognitive control difficulties in PWDA, showing impaired language and non-linguistic control, but preserved cross-lingual interactivity.

Overview of the study aims

In the present study, we aimed to gather further empirical evidence for Pitres' hypothesis of a control deficit in differential aphasia beyond the case study of Verreyt *et al.* (2013). To this end, we compared cross-lingual interactions and non-linguistic control performance of a group of PWDA and a control group. Examining control difficulties on a small group level, rather than a single-case difference, is necessary to establish the generalizability of Verreyt *et al.*'s case study findings. Also note that the control participants for the flanker task in Verreyt *et al.* did not suffer from aphasia. In order to guarantee that potential control problems are only characteristic for differential language loss, rather than for aphasia in general, here we also compared PWDA with a group of bilingual patients with parallel aphasia (PWPA). The Pitres hypothesis only predicts control deficiencies for the former group, whereas cross-lingual co-activation should be observed for both. To the best of our knowledge, no previous study has contrasted (non-linguistic) cognitive control abilities of different (quite large) groups of bilingual patients

with aphasia (seven PWDA and eight PWPA), which is plausible given that differential aphasia is quite rare.

Methods

Participants

Fifteen bilingual patients with aphasia were recruited in the University Hospital of Ghent and the ZNA Middelheim Hospital Antwerp, Belgium. The patients were referred to us by the neurologist (D.H.), the speech and language therapist (M.D.L.) or the neurolinguist (P.M.). Inclusion criteria were the following: (1) very good knowledge of Dutch and French or English before the acute onset of vascular aphasia (as assessed by a language questionnaire); (2) formally diagnosed aphasia in Dutch based on the Aachen Aphasia Test (AAT; Graetz *et al.* 1992); and (3) relatively spared comprehension, also based on aphasia test scores and on the assessment by the speech and language therapist. Patients suffering from a developmental disorder, from a serious cognitive or depressive illness, or from a motor impairment were excluded from the study. All patients were tested between 2 and 4 weeks post-stroke and had been receiving speech therapy in Dutch at the moment of their participation. During hospitalization in the first 2 weeks post-stroke, 1 h of therapy was given daily. After hospitalization and at the moment of testing, the speech–therapy rehabilitation was continued with a common frequency of three times for 30 min per week. To examine pre-onset language proficiency and language use, we administered a language proficiency test, which was completed by the patient accompanied by a close family member and contained self-rated proficiency questions. To assess language functions in Dutch, we used the Dutch version of the AAT. We developed a French experimental version of the AAT to assess French language functions.[2] For English, the Comprehensive Aphasia Test (CAT; Swinburn *et al.* 2004) was assessed. Average percentile scores for each patient and each language were calculated. These scores summarized the percentile scores on the different subtests of the AAT (spontaneous speech, token test, repetition, writing, naming, and comprehension) and CAT (cognitive screen, language comprehension, and expressive language) respectively.

Demographic data are shown in table 1. Pre-onset, all patients were highly proficient bilinguals with the same bilingual profile. The patients were assigned to the group of PWDA if they showed significant differences in their language scores (average percentile AAT/CAT scores) across both languages. To this end, 95% confidence intervals around the language scores were calculated for each patient. A difference was considered as significant if the confidence intervals around the language scores did not overlap. Seven patients were classified as PWDA, eight as PWPA. Most of the patients were Dutch–French bilinguals (four

Table 1. Detailed demographic data of the patients with differential and parallel aphasia

			Bilingualism		AoA (years)		Pre-onset proficiency		%ile AAT/CAT	
Subject	Age (years)	Gender	L1	L2	L1	L2	L1	L2	L1	L2
Patients with differential aphasia (PWDA)										
D01	41	F	Dutch	French	0	8	5.00	4.00	78.00	42.00
D02	24	F	French	Dutch	0	2	5.00	4.00	47.00	71.80
D03	53	F	Dutch	French	0	11	5.00	4.00	78.60	62.40
D04	41	M	Dutch	English	0	13	5.00	4.00	66.60	42.20
D05	77	M	French	Dutch	0	2	5.00	4.00	86.00	73.60
D06	41	F	Dutch	French	0	16	5.00	3.67	78.80	55.60
D07	62	M	French	Dutch	0	3	5.00	4.00	66.80	46.80
Total	48.43				0.00	7.86	5.00	3.95	71.69	56.34
	(17.24)				(0.00)	(5.70)	(0.00)	(0.12)	(12.92)	(13.36)
Patients with parallel aphasia (PWPA)										
P08	63	M	Dutch	English	0	13	5.00	4.00	77.40	73.20
P09	80	F	Dutch	French	0	11	5.00	2.67	86.20	85.40
P10	45	F	Dutch	French	0	11	5.00	4.00	92.60	87.80
P11	56	M	Dutch	English	0	12	5.00	3.66	93.80	88.60
P12	41	F	Dutch	French	0	7	5.00	3.00	92.00	91.20
P13	61	F	Dutch	French	0	1	5.00	5.00	78.40	88.20
P14	63	M	Dutch	French	0	13	5.00	3.33	57.80	70.60
P15	59	F	Dutch	French	0	20	5.00	4.00	10	21.2
Total	58.50				0.00	11.00	5.00	3.71	73.53	75.76
	(11.98)				(0.00)	(5.42)	(0.00)	(0.72)	(28.28)	(23.32)

Note: Shown are bilingualism (L1/L2 based on the age of acquisition—AoA), average pre-onset proficiency (scale), average percentiles (%ile) on the Aachen Aphasia Test (AAT) for Dutch and French, and Comprehensive Aphasia Test (CAT) for English. Standard deviations are shown in parentheses.

Table 2. Demographic data of the control subjects, patients with parallel differential aphasia (PWDA) and patients with parallel aphasia (PWPA)

	Control subjects, $N = 19$	PWDA, $N = 7$	PWPA, $N = 8$	Difference
Male/female	4/15	3/4	3/5	$\chi^2(2) = 1.50$, $p = .47$
Age (years)	55.68 (12.37)	48.43 (17.60)	58.50 (11.98)	$F(2,33) = 1.13$, $p = .34$
Education (years)	15.16 (2.52)	15.00 (2.45)	14.75 (2.96)	$F < 1$
L1 proficiency (scale)	5.00 (0.00)	5.00 (0.00)	5.00 (0.00)	$F < 1$
L2 proficiency (scale)	3.89 (0.80)	3.95 (0.12)	3.71 (0.72)	$F < 1$

Note: The male/female ratio was compared by means of a chi-squared test. One-way ANOVAs were conducted to compare the three groups in terms of Age, Education, L1 and L2 proficiency. Standard deviations are presented within parentheses.

PWDA and six PWPA). However, some of the patients were French–Dutch bilinguals (three PWDA) or Dutch–English bilinguals (two PWPA). Demographic data of both patient groups were compared by means of independent samples *t*-tests. Pre-onset, L1 and L2 proficiency as well as the proficiency difference between L1 and L2 were similar for both patients groups, $t < 1$ for all tests. Post-aphasia onset, there was no difference in L1 and L2 language functions, $t < 1$ and $t(13) = 1.94$, $p = .08$ respectively. The difference on (post-aphasia onset) AAT/CAT scores between the L1 and L2, on the other hand, differed significantly between the two groups, $t(13) = 5.13$, $p < .001$, indicating smaller differences in L1 and L2 proficiency for PWPA than for PWDA, as intended. Taken together, this shows better recovery of the L2 for PWPA than for PWDA, while there was similar recovery of the L1.

We also tested 19 control subjects that were recruited among family and friends of the patients and the authors. The control subjects were matched with the patients for age, sex, education and self-rated proficiency in L1 and L2 (based on the language background questionnaire) (table 2). More precisely, one-way analyses of variance (ANOVAs) showed that the three groups (control group, PWDA and PWPA) did not differ in age, years of education, (pre-onset) L1 proficiency and (pre-onset) L2 proficiency. Furthermore, a chi-squared test indicated that there was no difference in the male/female ratio across the three groups.

Stimuli and materials

Lexical decision (LD) task

Cognate effects, as a marker of preserved cross-lingual co-activation, are more likely to emerge in PWDA when language control demands are low (Verreyt *et al.* 2013). Therefore, we administered a generalized LD task in which all words (cognates and non-cognates) required a 'Yes' response, whereas the non-words required a 'No' response. Because both Dutch–French and Dutch–English bilinguals were included in the study, we developed a Dutch–French and a matched Dutch–English version of the task. The stimuli used in the LD tasks were 30 Dutch–French/English cognates, 30 Dutch non-cognates, 30 French/English non-cognates and 90 non-words. Cognates and non-cognates were matched for word length, frequency and neighbourhood size using WordGen (Duyck *et al.* 2004). In both versions, 14 cognates were identical cognates (e.g., the Dutch–English *baby*) and 16 cognates were non-identical cognates (e.g., the Dutch–English *schip–ship*).

Flanker task

Each stimulus of the flanker task consisted of five arrows horizontally presented on the screen. All the arrows pointed either in the same (congruent) direction or the flanking arrows pointed in the opposite (incongruent) direction of the central arrow. We included 40 congruent and 40 incongruent trials.

Procedure

The participants completed an LD task and a flanker task. The order of the tasks was counterbalanced across participants. Both tasks were programmed in E-prime 1.0 (Schneider *et al.* 2002).

Lexical decision (LD) task

Each trial started with the visual presentation of a black fixation cross ('+') on a white screen for 500 ms, followed by a letter string. The letter string was presented in 18-point Courier New font and remained on the screen until response. There was an inter-trial interval of 700 ms. The participants were asked to indicate as quickly and accurately as possible whether or not the letter string was an existing word, irrespective of the language. They were instructed to press the right green key (return) for words and the left red key (capslock) for non-words. The task started with a practise block of 10 trials, which contained four non-words, three L1 non-cognates and three L2 non-cognates that were presented in a random order. The experimental block was divided into two parts of 90 trials (15 cognates, 15 L1 non-cognates, 15 L2 non-cognates and 45 non-words) with a break in between them.

Flanker task

Each trial started with the presentation of a black fixation cross ('+') on a white screen for 500 ms, followed by a horizontal array of five equally sized and spaced arrows that remained on the screen until response. There was an inter-trial interval of 700 ms. All participants were asked to focus on the central arrow and to press the right button (return) when it pointed to the right and the left button (capslock) when it pointed to the left. They were instructed to ignore the flanking arrows and to answer as quickly and accurately as possible. The task started with a practice phase of 12 (six congruent and six incongruent) trials presented in a random order. The experimental block contained two blocks of 40 (20 congruent and 20 incongruent) trials with a break in between them.

Results

For both tasks, preliminary data treatment was as follows. First, RTs for incorrect responses and outliers were excluded from analyses on RT data. Outlier analyses were carried out using adjusted box plot methods on each condition for each group (Hubert and Vandervieren 2008), because a box plot distribution of the RTs for the three groups showed a much larger variability in the group of PWDA compared with the other two groups. Second, we fitted models with maximum likelihood estimation using the lmer function for RT and the glmer function for accuracy (ACC) from the lme4 package in R (Bates *et al.* 2015). We used Satterthwaite approximation to estimate the degrees of freedom for planned comparisons (Kuznetsova *et al.* 2015).

As in other studies examining bilingual aphasia, only the results based on ACC will be interpreted because of the very high variability typically observed on RTs for PWDA (Lalor and Kirsner 2001, Verreyt *et al.* 2013). The results on RTs on both tasks are nevertheless reported for completeness. Mean ACC and RTs for both the LD and the flanker task can be found in table 3.

Lexical decision (LD) task

We entered Group and Cognate status (and their interaction) as fixed effects and Subject as random effect into the model for both the ACC and RT data.

Accuracy

For ACC, we did not include a random intercept for Cognate status, because the conducted maximum likelihood comparison showed that the data did not justify its inclusion, $\chi^2(2) = 1.00$, $p = .61$. The logistic modelling did not reveal a main effect of Group, $\chi^2(2) = 4.11$, $p = .13$. The main effect of Cognate status did reach significance, $\chi^2(1) = 18.80$, $p < .001$, showing higher ACC for cognates than for non-cognates. Importantly, the size of this cognate effect did not differ across groups, $\chi^2 < 1$, indicating that the groups did not differ with respect to cross-lingual interactions.

Table 3. Mean accuracy rates and reaction times (RTs) for control subjects, patients with differential aphasia (PWDA), and patients with parallel aphasia (PWPA) on the different status levels for the lexical decision (LD) task (cognates and non-cognates) and the different congruency levels for the flanker task (congruent and incongruent)

	LD task		Flanker task	
	Cognates	Non-cognate	Congruent	Incongruent
Accuracy (%)				
Control subjects	97.40	95.20	99.21	99.47
	(15.91)	(21.39)	(8.86)	(7.24)
PWDA	89.52	84.54	99.29	96.07
	(30.70)	(36.19)	(8.44)	(19.46)
PWPA	95.00	88.61	99.06	99.38
	(21.84)	(31.81)	(9.65)	(7.89)
RTs (ms)				
Control subjects	730.55	783.23	519.01	532.77
	(258.99)	(258.55)	(124.09)	(121.87)
PWDA	1021.12	1171.19	766.86	792.23
	(642.61)	(928.54)	(429.67)	(410.01)
PWPA	1218.13	1303.92	685.03	697.98
	(930.34)	(1170.78)	(286.02)	(292.93)

Note: Standard deviations are shown in the parentheses.

Reaction times (RTs)

Outlier analyses on RTs resulted in the exclusion of 3.77% of the data for the control group, 10.97% for PWDA and 6.17% for PWPA. The higher outlier rate for PWDA compared with the other groups is consistent with the finding that RTs are highly variable in PWDA (Verreyt *et al.* 2013, Lalor and Kirsner 2001). We included a random slope for Cognate status into the model on RT data, because maximum likelihood modelling supported the conclusion, $\chi^2(2) = 14.82$, $p < .001$. The linear modelling revealed a main effect of Group, $\chi^2(2) = 10.84$, $p < .01$. More precisely, the control group was overall faster than PWDA, $t(28.30) = -2.83$, $p < .01$. There were no RT differences between the control group and PWPA or between PWDA and PWPA, $t < 1$ for both tests. There was a main effect of Cognate status, $\chi^2(1) = 10.10$, $p < .01$, showing faster RTs on cognates compared with non-cognates. The interaction of Group and Cognate status was also significant, $\chi^2(2) = 7.08$, $p = .03$. Planned comparisons on this interaction revealed a cognate effect for PWDA, $t(24.77) = 3.91$, $p < .001$, but not for the control group nor for PWPA, $t < 1$ for both tests. Moreover, the cognate effect was larger for PWDA than for both the control group,

$t(23.95) = 2.40$, $p = .02$, and PWPA, $t(23.92) = 2.38$, $p = .03$. The cognate effect was similar for the control group and PWPA, $t < 1$.

Flanker task

We entered Group and Congruency (and their interaction) as fixed effects and Subject as random effect into the model for both ACC and RT data.

Accuracy

For ACC, the random slope for Congruency was not significant, $\chi^2 < 1$ and was therefore not included in the model. The logistic modelling showed that the main effects of Group and Congruency were not significant, $\chi^2(2) = 1.72$, $p = .42$ and $\chi^2(1) = 1.36$, $p = .24$ respectively. The crucial interaction between Group and Congruency, on the other hand, was significant, $\chi^2(2) = 6.57$, $p = .04$. Planned comparisons on the interaction revealed that both the control group and PWPA showed no congruency effect (congruent versus incongruent trials), $z < 1$ for both groups. Crucially, however, PWDA did have a significant congruency effect, $z = -2.31$, $p = .02$, showing higher ACC on congruent compared with incongruent trials. These results suggest weaker non-linguistic control skills for PWDA (despite the necessarily small number of participants).

Reaction times (RTs)

Based on outlier analyses, 4.50% of the RT data were excluded for the control group, 7.13% for PWDA and 3.78% for PWPA. Thus, similar to the generalized LD task, PWDA had a higher outlier rate. The random slope for Congruency was not significant, $\chi^2 < 1$, and was therefore not included in the final model. Linear modelling revealed a main effect of Group, $\chi^2(2) = 11.02$, $p < .01$. More specifically, PWPA as well as PWDA showed overall slower RTs compared with the control group, $t(31.80) = 2.30$, $p = .03$ and $t(31.80) = 2.88$, $p < .01$ respectively. The patient groups did not differ from each other, $t < 1$. The main effect of Congruency was also significant, $\chi^2(1) = 10.51$, $p < .01$, indicating slower RTs on incongruent trials than on congruent trials. There was no interaction between Group and Congruency, $\chi^2(2) = 2.10$, $p = .35$. Taken together, these results show no difference in congruency effect in terms of RTs for PWDA, although both patient groups were slower than healthy control subjects.

Discussion

In the current study we examined the Pitres (1895) control impairment hypothesis for bilingual PWDA. More precisely, we investigated the preservation of bilingual cross-lingual interactions and impaired non-linguistic control in a group of PWDA and contrasted their performance with that of a healthy control group and a group of PWPA.

To investigate the occurrence of retained cross-lingual interactions, we used a generalized LD task with cognates. We observed cognate effects that did not differ across the three groups. This indicates that the groups, also the group of PWDA that experiences larger functionality loss in one language, showed an equal amount of cross-language lexical interactivity. This finding therefore argues against a strict localized account for differential aphasia, which proposes that the differential impairment of one language is due to selective damage to the language-specific area. This would predict smaller lexical activation in one language in particular and a smaller (or absent) cognate effect for PWDA. The observation of cognate effects is in line with other studies reporting effects of bilingual co-activation in patients with aphasia in general (e.g., Detry *et al.* 2005) and with studies that assessed cognate effects in PWDA in particular (Lalor and Kirsner 2001, Verreyt *et al.* 2013).

The results of the flanker task, which was used to examine non-linguistic control abilities, support the hypothesis of impaired control specifically in PWDA. Both the control group and the PWPA performed almost at ceiling level, so that they did not show a congruency effect for the present task. However, PWDA did significantly make more errors on incongruent trials than on congruent trials. These findings confirm that worse performance in the flanker task by the single PWDA in Verreyt *et al.* (2013), relative to non-aphasia controls, was indeed typical for differential aphasia, rather than for aphasia altogether. Together, the pattern of results in Verreyt *et al.* and the current group study are consistent with the notion that PWDA show worse non-linguistic control relative to PWPA (and control participants).

Our joint findings of a typical cognate effect and an increased congruency effect for PWDA support Pitres' hypothesis (1895) that a control deficit underlies non-parallel language loss in bilingual patients with aphasia. What is still not clear from the current study, however, is whether and how a deficit in non-linguistic control might lead to different non-parallel types of aphasia. As noted in the introduction, Paradis (2004) described five other types besides parallel aphasia, which are essentially all non-parallel types of aphasia. With the results of the current study in mind, we might assume that Paradis' different types of non-parallel aphasia vary in terms of language control abilities. Patients with selective aphasia for instance, who are completely unable to recover one of their known languages, might suffer

extreme problems in inhibiting the L1, even more than PWDA, who can recover both languages at least to some extent. We could not further investigate this, as no such patients appeared during the (four) years that the current study was running. Also, note that Paradis did not differentiate between variants of differential aphasia with greater loss in the L2 and those with greater loss in the L1. It is clear that the current control hypothesis would predict that differential aphasia always appears with greater loss in the L2, as was indeed the case for all PWDA that we identified over the years in the university hospital. Actually, cognitive control problems should always manifest themselves to the greatest extent if the L1 needs to be inhibited in order to use the L2, because the stronger language needs more inhibition (Green 1998, Meuter and Allport 1999). Future research should shed more light on the dissociation between more diverse types of non-parallel bilingual aphasia in terms of language control abilities.

The results of the current study also have practical implications. At present, very little is known about the best method for language recovery in bilingual aphasia. There is a lot of debate whether linguistic training should be done in one or in both languages of a bilingual patient. Some researchers have found that the effects of language therapy in one language generalize to the other untrained language (e.g., Marangolo *et al.* 2009), while others were not able to observe such generalization (e.g., Abutalebi *et al.* 2009). Our results suggest a cognitive control deficit underlying differential aphasia, which is not limited to the linguistic domain. A direct re-education of non-linguistic control abilities in addition to the typical linguistic treatment of aphasia might have an additional beneficial effect on recovery. Some studies already showed positive effects of non-linguistic control training for the recovery of bilingual aphasia (e.g., Brownsett *et al.* 2014). Thus, additional training of non-linguistic skills may have more positive consequences than only training word representations.

Neuro-anatomically, the brain regions responsible for control might involve frontal attentional and subcortical mechanisms (Price *et al.* 1999). An important aim for future research might be to link the pattern of aphasia with the localization of brain damage, to disentangle neuro-anatomic representations. Given that none of our patients exclusively showed very apparent frontal damage (e.g., haemorrhage), brain damage specifically leading to differential aphasia may be hard to detect. In fact, the brain damage in the patients of the current study was very diverse for both the PWDA and PWPA (table 4), and no conclusive pattern emerged. A difference between the two groups of patients that should be mentioned, however, is that the insular lobe was more frequently impaired in the group of PWDA ($n = 4/6$) than in the group of PWPA ($n = 1/8$).[3] The insular lobe serves as a crucial region that mediates language function and has been found to be implicated in receptive language, expressive language and language production (Oh *et al.* 2014). Interestingly for the present study, the insula is also involved in extreme language control, as it occurs for instance in simultaneous interpreting (e.g., Hervais-Adelman *et al.* 2015). This is consistent with the present hypothesis that explains differential aphasia in terms of impaired language control. Nevertheless, the focus of future work should perhaps be on disconnectivity rather than localized damage, and sophisticated connectivity analyses between brain networks responsible for language control and networks responsible for language processing may be necessary. The origin for the importance of such disconnectivity is relatively clear: language control involves the same neural network as non-linguistic control (Abutalebi and Green 2007). This language control network consists of the anterior cingulate cortex (ACC), the prefrontal cortex (PFC; Brodmann area (BA)47) and the head of caudate (HC). The ACC has been shown to contribute in response monitoring, but also in language switching, language selection and in cross-linguistic conflict resolution. The PFC is important for response control in general, such as response selection and suppression (Green and Abutalebi 2013). Finally, the HC is important for translation, language selection and switching in production and comprehension. However, the HC also plays a key role in non-linguistic cognitive functioning, such as in goal-directed behaviour (Grahn *et al.* 2009). It might be that there is a decreased connectivity between (one of) these control structures and other brain networks that are responsible for language processing and are typically interacting (Abutalebi *et al.* 2009). For instance, the insular lobe has direct anatomical connections to the PFC (Jakab *et al.* 2012). A decreased connectivity between the insula and the PFC might thus explain how different brain damage can lead to a common pattern of differential language loss. This is however speculative, and because aphasia is often associated with noisy patterns of neural damage, requires further empirical testing. A detailed behavioural and brain connectivity analysis of a very clear selective or differential aphasia case may test this in the future.

Despite the theoretical and practical contributions of our study, we would also like to address some of its limitations. First, we only examined non-linguistic control abilities with the flanker task. We chose this task because it is the most frequently used task in the literature to investigate non-linguistic control, and because it was not evident to submit vulnerable patients to very extensive testing batteries, in addition to the intensive speech therapy and neurological follow-up. It would be interesting to examine non-linguistic control

Table 4. Overview of the anatomical damage of patients with differential aphasia (PWDA) and patients with parallel aphasia (PWPA)

Subject	Lesion side	Complete MCA	Anterior MCA	Posterior MCA	Cortical/ subcortical	Basal ganglia	Haemorrhagic component	Thalamus	Insular lobe	Frontal lobe	Temporal lobe	Parietal lobe	Occipital lobe
PWDA													
D01	L&R[a, b]	No	Yes	Yes	Cortical	No	No	No	Yes	Yes	Yes	Yes	Yes (L&R)[a]
D03	L&R[a]	No	No	Yes	Cortical	No	No	No	Yes	No	Yes	Yes (L&R)	No
D04	L	No	Yes	No	Subcortical	Yes	No	No	No	Yes	No	Yes	No
D05	L	No	No	No	No	No	Yes	Yes	No	No	No	No	No
D06	L	No	No	Yes	Both	No	Yes	No	Yes	No	Yes	Yes	No
D07	L[b]	No	No	Yes	Both	Yes	No	No	Yes	No	Yes	Yes	No
Total (*n*)	n.a.	0	2	4	n.a.	2	2	1	4	2	4	5	1
PWPA													
P08	L	No	No	Yes	Cortical	No	No	No	No	No	No	Yes	No
P09	L[b]	No	No	Yes	Cortical	No	No	No	Yes	No	Yes	No	No
P10	L&R[a, b]	No	No	No	Both	No	Yes	No	No	Yes	No	yes (L&R)[a]	No
P11	L	No	Yes	No	Subcortical	Yes	No	No	No	No	No	No	No
P12	L&R[b]	No	No	No	Subcortical	No	No	Yes (L&R)	No	No	No	No	No
P13	L[b]	No	Yes	Yes	Cortical	No	No	No	No	Yes	Yes	No	No
P14	L	No	No	Yes	Cortical	No	No	No	No	No	Yes	Yes	No
P15	L	No	No	Yes	Both	No	Yes	No	No	No	Yes	Yes	No
Total (*n*)	n.a.	0	2	5	n.a.	1	2	1	1	2	4	4	0

Notes: The data of one patient with differential aphasia, patient D02, could not be obtained from the treating neurologist.
[a]Left lesion more severe than the right lesion.
[b]Multifocal brain damage.
MCA, middle cerebral artery; n.a. = not applicable.

with other tasks to detect control difficulties in PWDA. Additionally, we only addressed cross-lingual activation with a LD task and we did not examine actual language control. This only provided evidence for bilingual language processing of words under conditions of low language control demands. Further examination towards bilingual activation during language production is warranted to generalize our observations to language abilities in general. Third, French language abilities were tested with an experimental version (French translation of the Dutch AAT) and scores on this test were interpreted according to the norms for the Dutch AAT. It can therefore not be excluded that observed differences between Dutch and French may to some extent reflect differences in test difficulty rather than in language impairment. However, if only task difficulty would drive the effects, patients in the current study should all have obtained an overall higher (or lower) score in French compared with Dutch. This is however not what we observed. While the PWPA obtained a similar score for Dutch and French, two PWDA obtained a higher score for French than Dutch and three PWDA obtained the reverse pattern (table 1), which makes an explanation based on task difficulty only less plausible. A final important remark is that all patients were tested 2–4 weeks post-stroke and, hence, they were all within the acute phase of aphasia. This has the advantage that patients did not yet receive much linguistic treatment, excluding possible training effects. However, during the acute phase, recovery patterns might still evolve due to neuroplasticity (Robertson and Fitzpatrick 2008) and spontaneous recovery of some language functions is common (Lazar *et al.* 2008). This plasticity might have influenced the results of the current study, because difficulties might still vary from day to day. However, the observation of non-linguistic control difficulties when both languages are recovered more differentially supports the control impairment hypothesis of differential aphasia, regardless of whether or not patients are still spontaneously recovering.

Taken together, these findings provide new insights in the underlying mechanism of differential aphasia by further supporting the hypothesis that differential aphasia is not due to selective loss of the most affected language. Our results provide a two-sided argument for a control deficit underlying differential aphasia. First, the similar cross-lingual activation observed for PWDA shows that these patients do not primarily differ from PWPA in terms of bilingual lexical interactions, at least when language control demands are low (such as in the generalized LD task). Second, the increased number of errors in the non-linguistic control task suggests that the pattern of language loss may instead be attributed to a cognitive control dysfunction that also generalizes to the non-verbal domain.

Acknowledgements

This paper is dedicated to the memory of our dear colleague and friend, Professor Peter Mariën, who passed away on 1 November 2017, a few days before it was accepted for publication. ***Declaration of interest:*** The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

Notes

1. In unbalanced bilinguals there is evidence for both overlapping and distinct brain regions representing both languages (Briellmann *et al.* 2004). The patients included in the current study, however, can all be regarded as balanced bilinguals.
2. Because no standardized test in French was available, we developed a direct, literal translation of the Dutch AAT. We applied the Dutch norms to calculate the percentile scores, though we are aware that an identical score or change in scores on the same test in two different languages is not necessarily interpretable in the same way (Ivanova and Hallowell 2013). However, test results here are not used as a fine-grained assessment of preserved function. Instead, a difference in score with the Dutch version is only used for categorization in two groups of aphasia (parallel versus differential).
3. Information about the brain damage of one PWDA could not be obtained from the treating neurologist.

References

Abutalebi, J., Cappa, S. F. and Perani, D., 2001, The bilingual brain as revealed by functional neuroimaging. *Bilingualism: Language and Cognition*, **4**, 179–190.

Abutalebi, J. and Green, D., 2007, Bilingual language production: the neurocognition of language representation and control. *Journal of Neurolinguistics*, **20**, 242–275.

Abutalebi, J., Rosa, P. A. D., Tettamanti, M., Green, D. W. and Cappa, S. F., 2009, Bilingual aphasia and language control: a follow-up fMRI and intrinsic connectivity study. *Brain and Language*, **109**, 141–156.

Bates, D., Mächler, M., Bolker, B. M. and Walker, S. C., 2015, Fitting linear mixed-effects models using lme4. *Journal of Statistical Software*, **67**, 1–48.

Briellmann, R. S., Saling, M. M., Connell, A. B., Waites, A. B., Abbott, D. F. and Jackson, G. D., 2004, A high-field functional MRI study of quadri-lingual subjects. *Brain and Language*, **89**, 531–542.

Brownsett, S. L. E., Warren, J. E., Geranmayeh, F., Woodhead, Z., Leech, R. and Wise, R. J. S., 2014, Cognitive control and its impact on recovery from aphasic stroke. *Brain*, **137**, 242–254.

Damasio, A. R., 1992, Aphasia. *New England Journal of Medicine*, **326**, 531–539.

Detry, C., Pillon, A. and De Partz, M. P., 2005, A direct processing route to translate words from the first to the second language: evidence from a case of a bilingual aphasic. *Brain and Language*, **95**, 40–41.

Duyck, W., Desmet, T., Verbeke, L. P. C. and Brysbaert, M., 2004, WordGen: a tool for word selection and nonword generation in Dutch, English, German, and French. *Behavior Research Methods, Instruments, and Computers*, **36**, 488–499.

Duyck, W., Van Assche, E., Drieghe, D. and Hartsuiker, R. J., 2007, Visual word recognition by bilinguals in a sentence context: evidence for nonselective lexical access. *Journal of Experimental Psychology: Learning Memory and Cognition*, **33**, 663–679.

ERIKSEN, B. A. and ERIKSEN, C. W., 1974, Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception and Psychophysics*, **16**, 143–149.

GIUSSANI, C., ROUX, F. E., LUBRANO, V., GAINI, S. M. and BELLO, L., 2007, Review of language organisation in bilingual patients: what can we learn from direct brain mapping? *Acta Neurochirurgica*, **149**, 1109–1116.

GRAETZ, P., DE BLESER, R. and WILLMES, K., 1992, *Akense Afasie Test. Nederlandstalige versie* (Lisse: Swetz & Zeitlinger).

GRAHN, J. A., PARKINSON, J. A. and OWEN, A. M., 2009, The role of the basal ganglia in learning and memory: neuropsychological studies. *Behavioural Brain Research*, **199**, 53–60.

GREEN, D. W., 1998, Mental control of the bilingual lexico-semantic system. *Bilingualism: Language and Cognition*, **1**, 67–81.

GREEN, D. W. and ABUTALEBI, J., 2013, Language control in bilinguals: the adaptive control hypothesis. *Journal of Cognitive Psychology*, **25**, 515–530.

GROSJEAN, F., 2010, Bilingualism, biculturalism, and deafness. *International Journal of Bilingual Education and Bilingualism*, **13**, 133–145.

HERVAIS-ADELMAN, A., MOSER-MERCER, B. and GOLESTANI, N., 2015, Brain functional plasticity associated with the emergence of expertise in extreme language control. *NeuroImage*, **114**, 264–274.

HUBERT, M. and VANDERVIEREN, E., 2008, An adjusted boxplot for skewed distributions. *Computational Statistics and Data Analysis*, **52**, 5186–5201.

IVANOVA, M. V. and HALLOWELL, B., 2013, A tutorial on aphasia test development in any language: key substantive and psychometric considerations. *Aphasiology*, **27**, 891–920.

JAKAB, A., MOLNÁR, P. P., BOGNER, P., BÉRES, M. and BERÉNYI, E. L., 2012, Connectivity-based parcellation reveals interhemispheric differences in the insula. *Brain Topography*, **25**, 264–271.

KUZNETSOVA, A., BROCKHOFF, P. B. and CHRISTENSEN, R. H. B., 2015, *lmerTest: Tests in Linear Mixed Effects Models*. Retrieved from https://cran.r-project.org/package=lmerTest.

LALOR, E. and KIRSNER, K., 2001, The role of cognates in bilingual aphasia: implications for assessment and treatment. *Aphasiology*, **15**, 1047–1056.

LAZAR, R. M., SPEIZER, A. E., FESTA, J. R., KRAKAUER, J. W. and MARSHALL, R. S., 2008, Variability in language recovery after first-time stroke. *Journal of Neurology, Neurosurgery and Psychiatry*, **79**, 530–534.

MARANGOLO, P., RIZZI, C., PERAN, P., PIRAS, F. and SABATINI, U., 2009, Parallel recovery in a bilingual aphasic: a neurolinguistic and fMRI study. *Neuropsychology*, **23**, 405–409.

MEUTER, R. F. I. and ALLPORT, A., 1999, Bilingual language switching in naming: asymmetrical costs of language selection. *Journal of Memory and Language*, **40**, 25–40.

OH, A., DUERDEN, E. G. and PANG, E. W., 2014, The role of the insula in speech and language processing. *Brain and Language*, **135**, 96–103.

PARADIS, M., 2004, *A Neurolinguistic Theory of Bilingualism* (Amsterdam: John Benjamins).

PITRES, A., 1895, Etude sur l'aphasie chez les polyglottes. *Revue de Médicine*, **15**, 873–899.

POULISSE, N., 2000, Slips of the tongue in first and second language production. *Studia Linguistica*, **54**, 136–149.

PRICE, C. J., GREEN, D. W. and VON STUDNITZ, R., 1999, A functional imaging study of translation and language switching. *Brain*, **122**, 2221–2235.

ROBERTSON, I. H. and FITZPATRICK, S. M., 2008, The future of cognitive neurorehabilitation. In Stuss, D. T., G., W. and H., R. I. (eds), *Cognitive Neurorehabilitation* (New York: NY: Cambridge University Press), 566–574.

SCHNEIDER, W., ESCHMAN, A. and ZUCCOLOTTO, A., 2002, *E-Prime User's Guide* (Pittsburgh, PA: Psychology Software Tools).

SWINBURN, K., PORTER, G. and HOWARD, D., 2004, *The Comprehensive Aphasia Test* (Hove: Psychology Press).

VAN HEUVEN, W. J. B., DIJKSTRA, T. and GRAINGER, J., 1998, Orthographic neighborhood effects in bilingual word recognition. *Journal of Memory and Language*, **39**, 458–483.

VERREYT, N., DE LETTER, M., HEMELSOET, D., SANTENS, P. and DUYCK, W., 2013, Cognate effects and executive control in a patient with differential bilingual aphasia. *Applied Neuropsychology*, **20**, 221–230.