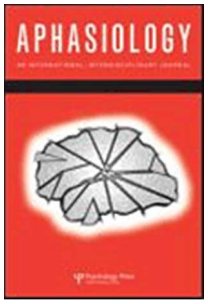

Syntactic priming in bilingual patients with parallel and differential aphasia.

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Syntactic priming in bilingual patients with parallel and differential aphasia.

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Abstract

Background: Syntactic priming is the phenomenon by which the production or processing of a sentence is facilitated when that sentence is preceded by a sentence with a similar syntactic structure. Previous research has shown that this phenomenon also occurs across languages, i.e., hearing a sentence in one language can facilitate the production of a sentence with the same structure in another language. This suggests that syntactic representations are shared across languages.

Aims. The aim of the current study is to investigate this cross-lingual syntactic priming in patients with bilingual aphasia. To address this aim, we asked the following three research questions: (1) Do patients with bilingual aphasia show priming effects within and across languages? (2) Do these priming effects differ from the priming effects observed in control participants? and (3) Does the pattern of priming effects interact with the type of aphasia?

Methods and procedures. We tested two groups of patients: one group had similar impairments in both languages (parallel aphasia); in the other group, the impairments were larger in one of the languages (differential aphasia). We investigated syntactic priming within and across languages by means of a dialogue experiment.

Outcomes and results. We found significant cross-lingual priming effects in both patient groups as well as in a control group. In addition, the effect size of both patient groups was similar to that of the control group.

Conclusion. These findings support models that incorporate shared syntactic representations across languages, and are in favour of a non-localised account of differential aphasia in bilingual aphasia.

Key words: bilingualism, aphasia, syntactic priming, differential aphasia, parallel aphasia

Introduction

In psycholinguistics, bilinguals are individuals who master and use two or more languages, but are not necessarily equally proficient in both (Grosjean, 1989). As the world's population is becoming more bilingual, it is not surprising that the number of bilingual patients with aphasia increases as well (Lorenzen & Murray, 2008). Aphasia is defined as a general impairment in understanding, formulating or using verbal messages, in spoken and/or written modality, caused by brain dysfunction to language-related areas. The main cause of aphasia is a stroke, but a tumour, an infection or degenerative brain diseases can also lead to aphasia.

Aphasia in bilingual patients does not always affect both languages to the same extent, nor do both languages always recover to the same degree. More specifically, Paradis (2004) described six different recovery patterns in bilingual aphasia (Table1).

Analogously to these *recovery* patterns, some impairment patterns might be described using similar terms (i.e., parallel, differential, and selective impairment). For example, a patient with one language that is more affected than the other, or with qualitatively different impairments in one language compared to the other, is diagnosed with differential aphasia (Agliotti & Fabbro, 1993; Goral, Levy, Obler, & Cohen, 2006; Meinzer, Obleser, Flaisch, Eulitz, & Rockstroh, 2007).

Initially, the phenomena of selective and differential aphasia were explained by the idea that languages of a multilingual are represented separately in distinct areas in the brain. It was hypothesised that selective impairment of one language was due to selective damage to the specific brain area representing that particular language. However, much evidence has now been gathered in healthy bilinguals falsifying the hypothesis of language-specific brain areas. First, at a functional level, the two languages of a bilingual always seem to be strongly interacting. A strong illustration of

this constant interlingual interaction is the cognate effect. Cognates are words with the same meaning and a similar form in different languages (e.g.,English–Dutch [film]–[film]). It was found that cognates are processed faster than non-cognates (i.e. the cognate facilitation effect, Dijkstra, Grainger, & Van Heuven, 1999), even if the non-target language should not be activated for the task at hand. For example, Dutch-English cognates are processed faster than non-cognates when reading Dutch sentences in an exclusively Dutch context (Duyck, Van Assche, Drieghe, & Hartsuiker, 2007). The effect also generalizes to word production: pictures with cognate names are named faster than pictures with non-cognate names (Costa, Caramazza, & Sebastian-Galles, 2000). This is commonly explained by convergent activation spreading from the cognate's similar representations across languages. According to cascade models of language production and comprehension, the production or comprehension of a word activates the semantic, orthographic and phonological representation of that word (Dell, 1986). This activation spreads to words with a related meaning, orthography or phonology. In the case of cognates, orthographic and phonologic nodes in both languages become activated, which leads to a higher activation, and therefore faster comprehension or production.

In addition to the behavioural evidence for cross-lingual interactions, neuroscience studies have found that the languages of a multilingual person are represented in overlapping areas in the brain (Hernandez, Martinez, & Kohnert, 2000; Illes et al., 1999). For example, Vingerhoets et al. (2003) investigated brain activation for word generation, word fluency and picture naming in Dutch, English and French within the same subjects, and found largely overlapping brain activation in the three languages (See also Klein, Zatorre, Milner, Meyer, & Evans, 1994).

At first sight, the behavioural and imaging evidence for a single integrated language system may seem contradictory with the existence of differential and selective aphasia: if both languages rely on the same (or highly overlapping) neural structures,

how may brain damage have larger effects in one language than in the other? This question may be traced back to a 1895 claim of Pitres, which may account for this discrepancy. He claimed that every language could be independently inhibited, temporarily or permanently. Thus bilingual aphasia should not be the result of a lesion in the neural substrate of a language, but rather the result of a functional inhibition of the language. (Pitres, 1895). In other words, selective or differential aphasia is here explained by a problem of language control, i.e., in the selection of words in the intended language, and the inhibition of words in unattended languages, rather than to a selective lesion in language-specific neural representations themselves.

In the present paper, Pitres' hypothesis will be applied to the syntax level. In addition to cross-lingual interaction effects at the word level, the syntactic priming literature has also revealed cross-lingual interactions at the syntactic level. Research has shown that the processing and production of a sentence is facilitated when the sentence is preceded by a sentence with a similar syntactic structure. For example, after hearing a passive sentence, a person will be inclined to produce a passive sentence rather than an active one. This is called *syntactic priming* (Bock, 1986), and is quite a robust effect. It has been found in a range of paradigms, such as written sentence completion (Pickering & Branigan, 1998), sentence recall (Potter & Lombardi, 1998), and spoken dialogue experiments, in which participants describe pictures to each other (Branigan, Pickering, & Cleland, 2000).

Important for the current study is that syntactic priming has also been found across languages. Hartsuiker, Pickering and Veltkamp (2004) found syntactic priming for transitive sentences in a dialogue experiment with Spanish-English bilinguals. They found that the bilinguals produced an English (L2) passive sentence more often after hearing a passive Spanish (L1) sentence than after an active Spanish sentence (See also Hartsuiker & Pickering, 2008; Meijer & Tree, 2003; Shin & Christianson, 2009 for

cross-lingual priming with datives). Cross-lingual syntactic priming has also been found in the opposite direction (L2-L1). Schoonbaert and colleagues studied syntactic priming with dative sentences in a group of Dutch–English bilinguals, and found significant priming effects within and across languages (Schoonbaert, Hartsuiker, & Pickering, 2007).

One of the mechanisms proposed to account for this cross-lingual priming is that residual activation of the structure of the previously heard or produced sentence might influence the choice for a current structure. Pickering and Branigan (1998) incorporated this assumption in the model of language production by Levelt (Levelt, Roelofs, & Meyer, 1999), to support syntactic priming effects. This model stated that in monolingual language production, the encoding of syntactic information (e.g., the arguments of a verb) is situated on the lemma level. Pickering and Branigan claim that the lemma nodes are connected to category nodes, indicating the word type, and combinatorial nodes, representing in which grammatical constructions the word can occur. When a verb can occur in an active and a passive construction, the lemma node of the verb will be connected with two different combinatorial nodes (i.e., an active node and a passive node). Thus, when the verb is used in a passive sentence, both the lemma node of the verb and the passive combinatorial node will become activated. The model further assumes that these combinatorial nodes are shared between lemma nodes, implying that for instance every verb that can be used in the passive voice will be connected to the passive combinatorial node. Syntactic priming effects are explained as follows: hearing (or producing) a passive sentence will activate the verb and the passive combinatorial node. When the next sentence is produced, the previously activated passive combinatorial node will still be residually active, and will facilitate the subsequent production of a passive sentence.

Previous research has also provided evidence for shared syntactic representations across languages (see above). Therefore, Hartsuiker et al. (2004) extended the model of Pickering and Branigan (1998) to bilinguals. They claim that syntactic information in proficient bilinguals is shared between languages, i.e., lemma nodes of verbs in both languages are connected to the same combinatory nodes (see Figure1).

In this study, we aimed to investigate whether patients with bilingual aphasia still show cross-lingual syntactic priming effects, and whether such effects depend on the type of aphasia (here: parallel vs. differential loss across languages). Until now, only two studies investigated syntactic priming in aphasic patients. This is regrettable, given the fact that aphasia is often not only characterized by dysfunctions at the lexical level, but also at the grammatical level. Applying this paradigm from monolingual psycholinguistic literature may add to our understanding of these dysfunctions. Because syntactic priming increases the availability of a grammatical structure, one would assume that patients who show impaired sentence production might benefit from this phenomenon. This is indeed what was found by Saffran and Martin (1997) on syntactic priming in aphasic patients. They investigated syntactic priming in a small group of patients with impaired sentence production. Firstly, participants completed a baseline exercise, in which they were asked to describe pictures that were not preceded by a prime. This was followed by a dialogue experiment. Here, patients heard a prime sentence, which they were asked to repeat. Subsequently they were asked to describe a target picture. This was done both with dative and transitive sentences. After the dialogue experiment the items used in the baseline test were administered again. During the dialogue experiment significant priming effects were observed with transitive, but not with dative sentences. Analysis of the baseline performance showed that patients produced more passives after the dialogue experiment than before. This suggests that there was also a long-term

priming effect, illustrating the potential value of this kind of research for therapeutic settings (i.e. extensively practising certain grammatical structures in therapy might enhance daily communication in patients with aphasia).

In the second study, Hartsuiker and Kolk (1998) extended these findings by comparing a group of Broca-aphasic patients with matched control participants. They also used transitive and dative sentences in a dialogue experiment, framed somewhat differently. Firstly, participants were told that they participated in a memory experiment, in which they were asked to indicate whether they had already seen the sentence or picture during the session or not. The participants were asked to read the sentences out loud and to describe the pictures "to facilitate the recognition". No specific instructions were given in the second condition; participants were only asked to describe pictures and read sentences out loud. In the third condition, participants were explicitly asked to use the grammatical structure of the previous sentence. In general, participants had to carry out the same crucial task in each of the three conditions, namely describing pictures that were preceded by prime sentences. The patient group showed significant priming effects for passives, double object datives and prepositional datives, in the three conditions. The size of the priming effects did not differ across conditions. However, in the control group significant priming effects were only observed in the third condition, in which they were specifically asked to re-use the structure of the previously heard sentence. The authors explained the null priming effects in control subjects as a consequence of task difficulty, or of the use of a relatively small control group.

The finding that aphasic patients show priming effects even though spontaneous production is impaired, suggests that grammatical representations in themselves are not 'lost', but instead harder to functionally access. Both studies showed that aphasic patients used hardly any passive sentences spontaneously, whereas they did produce

passives during and after the experiment. This suggests a positive influence of syntactic priming on the quality of language (sentence) production.

The present study

With the current study we aimed to go a step further than the existing syntactic priming studies in monolingual aphasia, by investigating *cross*-lingual syntactic priming in *bilingual* aphasic patients. To our knowledge, cross-lingual syntactic priming has never been investigated in relation to aphasia. We also aimed to assess whether such syntactic priming patterns are dependent on relative language loss by comparing a group of patients with parallel aphasia (i.e., having expressive and receptive impairments to the same extent in both languages) and a group of patients with differential aphasia (i.e., having larger impairments in one language compared to the other, or showing different impairments in one language compared to the other). More specifically, we studied the following three research questions: (1)Do patients with bilingual aphasia show priming effects within and across languages? (2)Do these priming effects differ from the priming effects observed in control participants? and (3)Does the pattern of priming effects interact with the type of aphasia[1]? For the latter, we were specifically interested in the difference between patients with parallel aphasia and patient with differential aphasia. Testing patients with differential aphasia allowed us to investigate whether we could still find syntactic priming from the most affected language as the prime language: are syntactic representations that are most dysfunctional still able to influence production in another language? Within the view that the underlying mechanism of selective and differential aphasia is a problem in cognitive

[1] Caveat. With "type of aphasia" we do not refer here to the classical distinction between Broca and Wernicke aphasia., but rather to the distinction between parallel and differential aphasia.

control and not in a brain area representing a single language (Pitres, 1895), we expected that patients with differential aphasia could still show syntactic priming from their most affected language, because the representations themselves are intact, as loss of functionality in spontaneous productions could reflect a language control problem instead. If this hypothesis is correct, production in the most affected language fails because there is a problem in activation or inhibition of the target or non-target language, respectively, however not because the target language representations themselves are dysfunctional. In the syntactic priming paradigm, however, the primes in the most affected language need to be comprehended, not produced. This requires less language control, because language selection is not strictly necessary for comprehension (one may just rely on bottom-up activation from the input), unlike production (Costa & Santesteban, 2004). As such, activation of syntactic representations after comprehension of the prime in the most affected language might still transfer to production of the same grammatical structure in the best-preserved language.

Method

We investigated these research questions by using a dialogue experiment in which a confederate and a participant were asked to describe pictures to each other (Bernolet, Hartsuiker, & Pickering, 2007; Branigan et al., 2000). We used four language-conditions: two conditions in which we tested within-language priming (L1-L1, L2-L2) and two conditions in which we tested between-language priming (L1-L2, L2-L1). Firstly, our paradigm and stimuli were piloted in a group of age-matched non-aphasic control subjects, to make sure they elicited priming effects. Consequently, we used the same paradigm and materials to assess syntactic priming in our patients with parallel vs. differential aphasia.

Participants

The *control group* consisted of 19 Dutch-French bilinguals who were matched with our patient group on age, sex, education, and self-rated proficiency in Dutch and French (Table2).

The patient group consisted of six bilingual aphasic patients. All the patients were referred to us by the neurology department of Ghent University Hospital. We used following inclusion criteria: (1)having a very good knowledge of French and Dutch before the onset of aphasia (as assessed by a language questionnaire[2]); (2)being diagnosed with aphasia based on the Aachen Aphasia Test (AAT); and (3)having relatively good remaining comprehension (also based on AAT scores and on the assessment by the speech and language therapist). Patients suffering from a developmental or neurodegenerative disease, from an infection or tumour, or from a serious cognitive or depressive illness were excluded from the study. None of the patients had had a stroke previous to the one causing the current aphasic symptoms. The vision of all the patients was normal or corrected to normal (see also Table3 and Table4). All patients were early, balanced bilinguals. L1 and L2 were determined based on Age of Acquisition. In the group of patients with differential aphasia, L1 was consistently the best-preserved language.

Materials

We assessed the proficiency of the patient in speaking, writing and reading in Dutch and French before the onset of aphasia on a 5-point Likert scale, the context in which they used both languages, and the frequency of use (days/week), in both the patient and the

[2] This questionnaire was filled out based on both the answers of the patients and their closest family member(s), present in the hospital on the day of testing.

close family member(s). To this end, we used a comprehensive, self-developed questionnaire.

To test the language strengths and deficits we used the Aachen Aphasia Test in Dutch (Graetz, De Bleser, & Willmes, 1992) and a self-developed French parallel version. The patients were assigned to the parallel or differential aphasia group based on the comparison of the scores on the subtest 'Naming' in Dutch and French. If these scores did not differ significantly on a paired t-test (determined by AAT software), the patient was assigned to the parallel aphasia group; if they did differ, the patient was assigned to the differential aphasia group. We opted to base the group assignment on the Naming subtest, because naming proficiency is highly relevant for the experimental task (i.e., describing pictures with a sentence). In addition we administered Part C of the Bilingual Aphasia Test (BAT) for Dutch and French (Paradis & Libben, 1987). This test assesses recognition of translation equivalents.

Before running the experiment, we administered a baseline task to measure how frequently the participants produced active and passive sentences without priming. The baseline task included twenty pictures that the participants were asked to describe without hearing a prime sentence. Ten pictures were supposed to be described in Dutch, ten pictures in French. For every language, eight pictures showed a transitive action, the other two pictures were filler sentences showing an intransitive action.

The syntactic priming task was a dialogue experiment. The *target stimuli* were 296 different pictures. We used pictures from the stimulus set of Bernolet et al. (2009) and from the different language versions of the BAT (of course we excluded the stimuli of the Dutch and the French version of the BAT). We included 132 critical trials, separated by either one or two filler trials. The target pictures depicted an action with a transitive verb (eliciting an active or a passive sentence), and the words needed to describe the picture (agent, patient and infinitive of the verb). We added these words to

prevent that word finding difficulties interfered with the focus of this study, accessing syntactic representations (see also Hartsuiker & Kolk, 1998b). The words were presented at the right side of the picture so that participants would be more likely to look at the picture first[3]. The order of the words varied across pictures. In addition, all pictures were mirrored relative to the vertical axis, because it was found that the spatial position of the agent might influence the preference for an active or a passive structure (Hartsuiker & Kolk, 1998). Both the original and the mirrored pictures were randomly used. When the patient showed inability to read the words, the experimenter read them aloud. The prime and the target sentence never contained the same verb, to avoid a lexical boost (Figure2a).

Since it was shown that animacy of the agent (i.e. the instigator of the action) and patient (i.e. the person (or animal) undergoing the action) might influence syntactic priming (Arai, Van Gompel, & Scheepers, 2007), our stimuli were controlled for this variable. We included equal amounts of three types of pictures: animate-animate (AA – animate agent and patient) pictures, inanimate-animate (IA – inanimate agent and animate patient) pictures, and inanimate-inanimate (II-inanimate agent and patient) pictures. We included 44 trials of each type (11 in each Language condition). In addition to the target pictures, we included 41 *filler pictures* in each language condition, showing an action with an intransitive verb (Figure2b).

Procedure

[3] This was not formally assessed, however the participants quickly understood the importance of firstly looking to the pictures, and then to the words. By only looking at the words, probability for erroneous sentences was high, because both nouns could act as an agent or a patient.

The study was approved by the Ghent University Hospital Ethics Committee. Before administering the tests, the patients and their closest family member were asked for their permission to be included in the study. Written informed consent was obtained in both the patient and the control group. In the patient group, we first administered the AAT, the BAT and the language questionnaire. The control group filled in the language questionnaire as well. Subsequently we administered the baseline task and the syntactic priming experiment.

The experiment was set up as a dialogue experiment. It was programmed in Eprime (version 2.1). During the experiment, one experimenter acted as the confederate (i.e. pretending to take part in the experiment as a participant), the other sat next to the participant to guide him through the experiment. The confederate and the participant each sat in front of a computer screen, and the participant was told that they would be describing pictures to each other. To avoid unnecessary complexity, we blocked language trials (L1-L1,L1-L2,L2-L1,L2-L2). Before each language block, the participant was told in which language he/she was supposed to produce a sentence. The sequence of a trial was as follows (Figure3): (a)the confederate read the prime sentence; (b)the participant saw two pictures (one with a blue background, one with a red background), and had to indicate which picture fitted the sentence he just heard by pressing the corresponding button (i.e., *the verification task*); (c)the participant saw a picture he had to describe using the words next to the picture; (d)the confederate coded the target sentence. The confederate coded[4] the target sentences produced by the participant as *active* (sentences with an active surface structure, including when the verb form was morphologically incorrect), *passive* (sentences with a passive surface structure, i.e.,

[4] All experiment runs were taped and listened to for a second time in case of uncertainty.

having an auxiliary and a past participle, in which the patient takes the function of sentence subject and the agent is expressed as an oblique object), or *other.* In step (c), participants were instructed to firstly look at the picture, and then to the words. We included the verification task (steps a and b) for two reasons. First, we wanted to make sure the participants listened carefully to and comprehended the prime; secondly, the accuracy in the verification task could function as a measure of language comprehension.

Design

The independent variables were *Syntactic structure* of the prime sentence (active vs. passive), *Prime Language* (L1/L2), and *Target Language* (L1/L2). This resulted in four language-conditions (L1-L1,L1-L2,L2-L1,L2-L2). The language-conditions were administered in blocks, and the order of these language blocks was counterbalanced. The dependent variable was the structure used to describe the target sentence[5].

Results

Control group

*Pre-experimental baseline***.** Baseline results show a low frequency of passive target descriptions. In control subjects, on average 5.3%(SD=0.08) of the pictures in the Dutch subset were described with a passive sentence, whereas on average 3.3%(SD=0.09) of the pictures were described with a passive sentence in French. A Chi-

[5] Analyses with agent and patient agency as an additional factor yielded similar results with respect to the crucial findings described below. Because of the design complexity, the factor is therefore not included in the analyses in the main text.

square test shows that these average proportions do not differ significantly ($\chi^2_{(3)}$=4.57,p>0.20).

Verification task. The accuracy on the verification task is very high. The control subjects responded inaccurately in only 1.9% of the trials.

Priming experiment. We used the Lme4 package in R (Version 2.12.2; CRAN project; The R foundation for Statistical Computing, 2009). Table5 and Figure4 show the priming effects in the control subjects. These were calculated as the difference between (amount of passive targets following a passive prime) minus (amount of passive targets following an active prime) (Pickering & Branigan, 1998). Trials in which participants did not use a correct active or passive sentence were excluded from the analysis (3.1% of the trials). We ran a binary logistic regression with one random factor[6] (mixed logit model, see Jaeger (2008)). The fixed-effect variables were *Prime* (active/passive), *Prime Language* (L1/L2*),* and *Target Language* (L1/L2). We included the two-way and three-way interactions between these variables. Finally, we included a random intercept for *Subject* (other random effects did not significantly improve the log-likelihood of the models). The dependent variable was *Target structure*. A significant main effect of *Prime* was found, which is the priming effect (i.e., more passive target sentences after a passive prime compared to an active prime) (χ^2(1)=24.94,p<.000). We also found a significant effect of *Prime Language* (χ^2(1)=4.52,p<.034), and a marginally significant interaction between *Prime* and *Prime Language* (χ^2(1)=3.72,p<.054). Further analyses show that the estimated β is 0.34 with an L1 prime (p<.042), whereas β is 0.80 with an L2 prime (p<.00). This might suggest that priming effects are somewhat larger with L2-primes, however this effect seems mainly caused by the L2-L2 vs. L1-L2 comparison.

[6] We first selected a structure for the random effects to then add the fixed effects. Finally the model was reduced by removing non-significant fixed effects and the model diagnostics were assessed.

Patient groups

Pre-experimental baseline. In the patient groups, on average 10.4%(SD=0.12) of the target pictures were described with a passive structure in Dutch, in French, this was only the case for on average 4.2%(SD=0.10) of the target pictures. A Chi-square test shows that the proportion of passive target sentences does not differ significantly between Dutch and French ($\chi^2_{(2,6)}$=1.83,p>0.40), nor between patients with parallel and differential aphasia ($\chi^2_{(2,6)}$=1.33,p<0.51). The baseline proportion of passive target descriptions does not differ either between patients or control subjects ($\chi^2_{(36,25)}$=6.90,p> 0.08).

Verification task. The group of patients with parallel aphasia has a mean error percentage of 1.9% (0.5% after L1-primes, 1.4% after L2-primes), patients with differential aphasia 3.3% (1.1% after L1-primes, 2.2% after L2-primes). To analyse the error data we ran a binary logistic regression with one random factor (mixed logit model). The fixed-effects variables were *Aphasia type* (parallel/differential aphasia), *Prime* (active/passive), and *Prime Language* (L1/L2). All two-way and three-way interactions were included, as was a random intercept for Subject (other random effects did not significantly improve the log-likelihood of the models). The dependent variable was Accuracy.

The effect of *Aphasia Type* is not significant (p>0.1), suggesting that patients with parallel and differential aphasia do not differ in the amount of errors on the verification task. We find a significant effect of *Prime Language* ($\chi^2_{(1,6)}$=5.49,ß=-1.51,p<0.02), showing that more errors were made with L2-primes (3.6%) compared to L1-primes (1.6%). No other effects reach significance (p>0.08).

Priming experiment. We ran a fully specified binary logistic regression with one random factor (mixed logit model). The fixed-effect variables were *Prime* (active/passive), *Prime Language* (L1/L2), and *Target Language* (L1/L2). We also included a random intercept for *Subject*. The dependent variable was *Target structure*.

We find a significant main effect of *Prime*, implying a syntactic priming effect (more passives after a passive prime than after an active prime; $\chi^2_{(1,3)}$=6.49,*p*<0.01). We also find a significant main effect of *Target Language* ($\chi^2_{(1,3)}$=8.40,*p*<0.004), meaning that more passives were produced with L2-targets. The interaction between *Prime* and *Target Language* is not significant ($\chi^2_{(1,3)}$=0.1439,p>0.7) (Figure6).

Because both our patient groups are very small, we were not able to statistically compare the magnitude of the priming effects in the different language-conditions between the two patient groups. As an alternative we discuss the numerical differences in the magnitude of the priming effects, as if it were a multiple case study.

Table6 and Figure5 show that both patient groups show considerable priming effects in each language condition, which are comparable with the priming effects observed in the control group. To be able to compare the priming effects in the patient groups with the control group statistically, we calculated the 95% confidence interval of the parameter of the factor *Prime* in the control group. The estimated parameter of *Prime* is 0.58(SE=0.12), and the confidence interval is [0.34,0.82]. For the patients with parallel aphasia, the estimated parameter for *Prime* is 0.35(SE=0.27) with a confidence interval of [-0.11,0.81]. For the patients with differential aphasia, the parameter for Prime is 0.69(SE=0.3), with a confidence interval of [0.18,1.20]. This suggests that both patient groups show approximately equally large priming effects across language-conditions compared to the control group.

The patients with parallel aphasia show more priming with L2-primes (10.6%) compared to L1-primes (4.7%). This pattern[7] was also observed in the control subjects (9.2% with L2-primes vs. 7.3% with L1-primes). Interestingly, the patients with differential aphasia show the opposite effect, with larger priming effects from the best-preserved language (8.4% with L2-primes vs. 15% with L1-primes). When comparing between-language priming (the average effect in the L1-L2 and L2-L1 condition) and within-language priming (the average effect in the L1-L1 and L2-L2 condition), patients with parallel aphasia show more priming when the prime language and the target language are the same (within-language priming, 11%) compared to between-language priming (4.3%). This pattern is again similar in the control group (10.2% within-language priming vs. 6.3% between-language priming). Interestingly, the patients with differential aphasia show almost equal priming effects within languages (11%) and between languages (12.4%).

Discussion

The aim of the current study was to investigate syntactic priming in bilingual aphasia. To address this aim, we asked the following research questions: first, do patients with bilingual aphasia show syntactic priming within and between languages? Secondly, do these priming effects differ across aphasia patterns? We included two types of aphasia patients: to investigate whether relative language loss influences such cross-lingual syntactic interactions, we contrasted patients with parallel aphasia (i.e., having similar impairments in both languages) and patients with differential aphasia (i.e., the impairments in one language are more severe than in the other language). Our third

[7] We opted not to speculate about differences in the size of the effects, because of the differences in group size, which make it difficult to compare the size and the strength of the priming effects between the patient groups and the control group.

research question was whether these priming effects differ from the priming effects observed in control participants. Therefore we compared the priming effects of the patients with the effects of a group of matched control participants.

We administered a dialogue experiment in four language-conditions: two within-language-conditions (L1-L1 and L2-L2) and two between-language-conditions (L1-L2, L2-L1). Our results show that patients with bilingual aphasia did show a robust, statistically significant syntactic priming effect: they produced more passive sentences after hearing a passive prime than after hearing an active prime, both within and across languages. This is the first demonstration of cross-lingual syntactic priming in bilingual aphasic patients. Both control subjects and aphasic patients show considerable priming effects in all four language-conditions, but some interesting differences across groups also emerged. Within-language priming was stronger than between-language priming for both control participants and patients with parallel aphasia, whereas patients with differential aphasia showed equally strong cross-lingual as intralingual priming. Control participants and patients with parallel aphasia showed stronger priming effects from L2 primes, whereas patients with differential aphasia showed stronger priming effects from the first-acquired (and also best-preserved) language (L1). So, patients with parallel aphasia behaved much more similarly to controls than patients with differential aphasia.

Finding syntactic priming effects in patients with bilingual aphasia replicates two previous findings of syntactic priming in aphasic patients (Hartsuiker & Kolk, 1998; Saffran & Martin, 1997). However, Hartsuiker and Kolk (1998b) did find larger priming effects in the patient group than in control subjects, which we did not observe. A possible explanation might be that Hartsuiker and Kolk only tested patients with Broca's aphasia, who showed severe syntactic deficits, whereas our patients were diagnosed with

different types of aphasia and had relatively smaller syntactic deficits[8]. It is plausible that patients with strong grammatical/syntactic impairment benefit more from syntactic activation triggered by prime sentences when producing sentences. The lack of severe syntactic deficits in our patients might explain why they did not show a larger tendency than the control subjects to rely on the previous structure. This is consistent with our observation that the cross-lingual priming effect for patients with differential aphasia was larger when producing sentences in the most affected language (15.2%) than the cross-lingual effect for sentence production in the most preserved (but still affected) language (9.5%). Another important difference with Hartsuiker and Kolk, is that they did not find priming effects in the control group. We did observe priming effects in our control subjects, which is in line with previous studies showing cross-lingual priming effects for transitive sentences in control subjects .

Although all groups showed syntactic priming in all language-conditions, one of the most interesting findings in this study is that differential aphasia patients also showed strong syntactic priming effects with L2-primes, even though this is the most affected language. The overall 8.4% priming effect with L2-primes is comparable with the L2-priming effect of patients with parallel aphasia (10.6%) and control subjects (9.2%). Only looking at L2-L1 cross-lingual priming, differential patients even showed stronger priming effects (9.5%) than parallel (5.9%) patients and control (7.1%) participants. This suggests that the most impaired language is not “lost”; syntactic representations in themselves are intact and still able to influence syntactic processing in the other language, if language control demands are low. In the syntactic priming paradigm, the prime in the most affected language (L2) only has to be comprehended, not produced

[8] It may also be the case that the current study did not have adequate power to detect significant differences. It would definitely be interesting to replicate these findings in larger groups of participants.

(so that the dominant language does not necessarily need to be inhibited, as is the case in production), so that activation in the syntactic representations triggered by comprehension is strong enough to influence subsequent production in the best preserved language. However, it remains unclear why the L2-L1 priming effect is larger for the group of patients with differential aphasia compared to the group of patients with parallel aphasia.

In patients with parallel aphasia and control participants, we found that L2-primes (9.9%)[9] elicited larger priming effects compared to L1-primes (6%)[9]. This might be explained by a complexity effect, in which syntactically more complex sentences generalize to syntactically less complex sentences, but the reverse does not occur (Thompson, Shapiro, Kiran, & Sobecks, 2003). One might argue that this complexity effect could also be reflected in more generalization from a less frequent language (L2) to a more frequent language (L1), than vice versa. However, it remains unclear why this effect could not be found in patients with differential aphasia. Replication with larger groups of patients is needed to confirm this pattern of results, and to further elaborate this effect in patients with differential aphasia.

Because the priming effects of parallel aphasia patients were more similar to the effects found in control subjects than the effects of differential aphasia patients, and because the latter still showed strong L2-L1 priming, this provides evidence for a non-localized account of differential language loss, e.g. in terms of language control (see above). A possible network underlying language control was recently described by Abutalebi and Green (2007). The network consists of the prefrontal cortex, the anterior cingulate cortex, the inferior parietal cortex and the basal ganglia. Damage to these components might lead to the control deficits underlying bilingual aphasia. This view is

[9] These percentages reflect average priming effects of patients with parallel aphasia and control subjects across language-conditions.

consistent with Pitres' account, which already suggested that differential or selective aphasia may not be due to loss of the language representations themselves, but rather to a problem in controlling languages. To further disentangle the role of each component of this control network in differential bilingual aphasia, additional (imaging) research is needed.

The results are in line with other studies demonstrating cross-lingual syntactic priming (Bernolet et al., 2007; Hartsuiker et al., 2004; Kantola & Van Gompel, 2011; Loebell & Bock, 2003; Schoonbaert et al., 2007; Shin & Christianson, 2009) showing that people tend to re-use syntactic structures, even across languages (see Introduction). In addition, they confirm the predictions based on the bilingual syntactic priming model of Hartsuiker et al. (Hartsuiker et al., 2004), discussed in the introduction, and provide further evidence for shared syntactic representations across languages. In this model, both within- and across-languages priming effects are explained in terms of residual activation in syntactic representations after comprehension of the prime. Because the model assumes shared syntactic representations across languages, it predicts cross-lingual priming effects as long as these syntactic representations are intact. As such, this model is compatible with accounts that explain differential aphasia in terms of language control. Important to notice however, is that the model of Hartsuiker et al. did predict similar priming effect sizes in between- and within-language priming, which is not completely in line with what we found here. Yet, Cai and colleagues recently contested this prediction by assuming that not only the combinatorial node remains activated, but the language node as well, inhibiting other language nodes (Cai, Pickering, Yan, & Branigan, 2011). This would suggest larger within-language than between-language priming effects (as was found in our patients with parallel aphasia and in our control subjects, but not in our group of patients with differential recovery). Further research will

be needed to apply these models to syntactic priming effects in patients with bilingual aphasia.

The finding of cross-lingual priming effects is also interesting from a therapeutic perspective, because it implies that training in one language might also be beneficial for the other language. This is in line with previous studies showing that language therapy in one language might generalise to another (untrained) language (Edmonds & Kiran, 2006; Filiputti, Tavano, Vorano, De Luca, & Fabbro, 2002; Kiran & Edmonds, 2004; Marangolo et al., 2009; Miertsch, Meisel, & Isel, 2009; for a conflicting view, see Abutalebi, Rosa, Tettamanti, Green, & Cappa, 2009; Galvez & Hinckley, 2003; Meinzer et al., 2007). Given the current findings, agrammatic symptoms in bilingual patients may benefit from training in, and transfer from, other languages than the dominant language, both for parallel and differential aphasia patients. An interesting finding concerning therapy effects is that aphasia patients seem to show longer lasting priming effects in certain conditions: patients with parallel aphasia produce a passive sentence after an active prime in 33% of the trials of the L1-L2 condition, and patients with differential aphasia produce a passive sentence after an active priming in 33% of the trials of the L2-L2 condition. This never occurred in the control group. This suggests that priming lasts longer in the aphasic group, that is, the passive construction is not inhibited in an active condition, but it is still triggered. In addition, this only seems to occur when the target language is L2, and most often in aphasia patients. Further research is needed to identify the conditions under which training effects last longer and generalize across languages. In addition, the requirements of language therapy should be further investigated. An interesting path for future research would be to sort out why aphasia patients do not benefit from hearing sentences in daily life, and why they do benefit from language therapy. It might be the case that they do benefit from hearing sentences in daily life, but this is less visible than after therapy. Also, linguistic input for patients with

aphasia might be less grammatically diverse, because people take into account possible comprehension problems.

To summarise, this is the first demonstration of cross-lingual syntactic priming in a group of patients with bilingual aphasia. The pattern of the effects was comparable to the pattern observed in a group of matched control subjects. Moreover, patients with differential aphasia also showed cross-lingual priming from the most affected language to the best-preserved language. This shows that the least recovered language can still influence syntactic processing in the other language. Our results are largely in line with the model proposed by Hartsuiker et al. (2004), and support a control-based account of different bilingual aphasia patterns. Nevertheless, these results were obtained in a small group of patients, so further research with more patients is needed to confirm our findings. In addition, the current findings should be replicated with other syntactic structures and language combinations.

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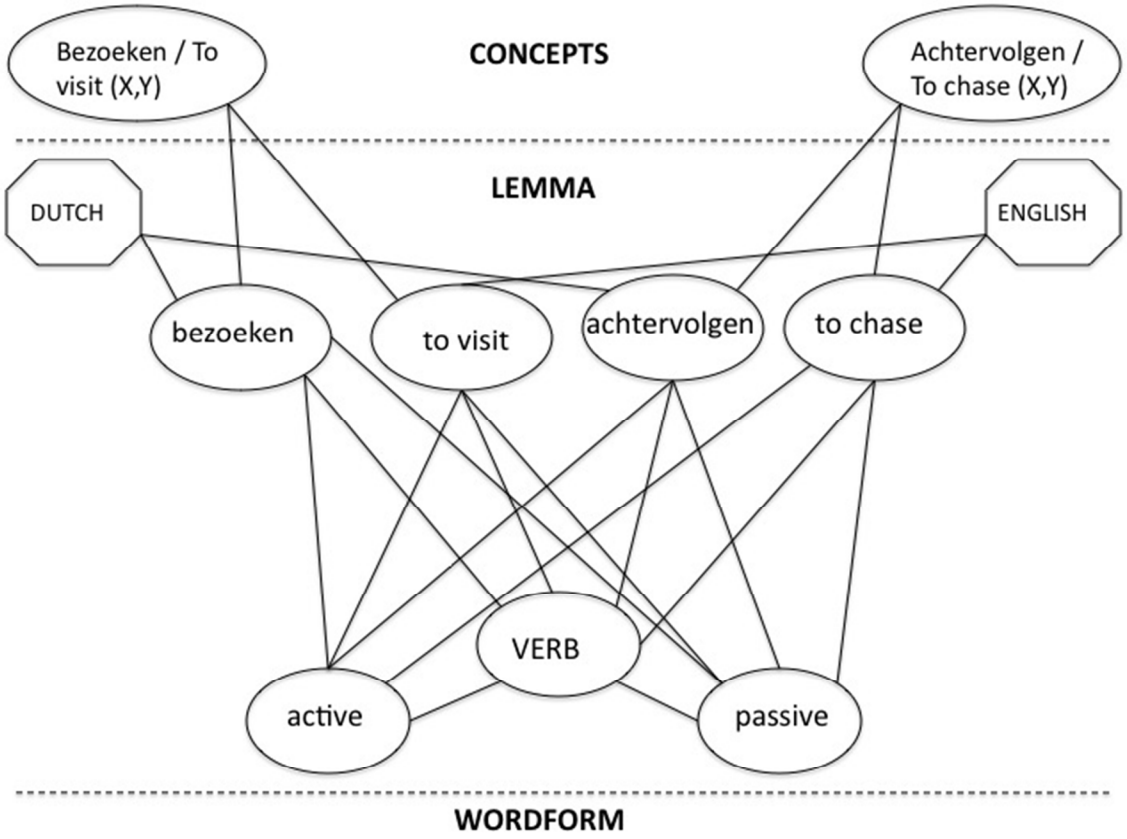
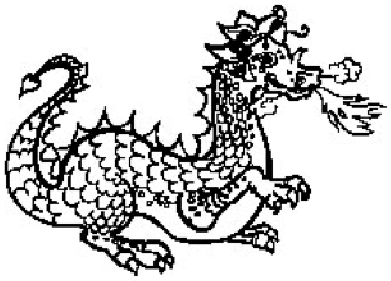


Figure 1. The model for cross-lingual syntactic priming proposed by Hartsuiker et al. (2004) applied to a Dutch-English example. In this model, the lemma nodes of the verbs [bezoeken] / [to visit] and [achtervolgen] / [to chase] are connected to a shared active combinatory node, and a shared passive combinatory node. Each lemma node is also connected to a category node for verb, and a language node (Dutch or English).

a

VASTHOUDEN
VROUW
BABY

b



HURLER
DRAGON

Figure 2. Example of (a) a target picture (with the words "to hold", "woman" and "baby") and (b) a filler picture (with the words "to roar" and "dragon").

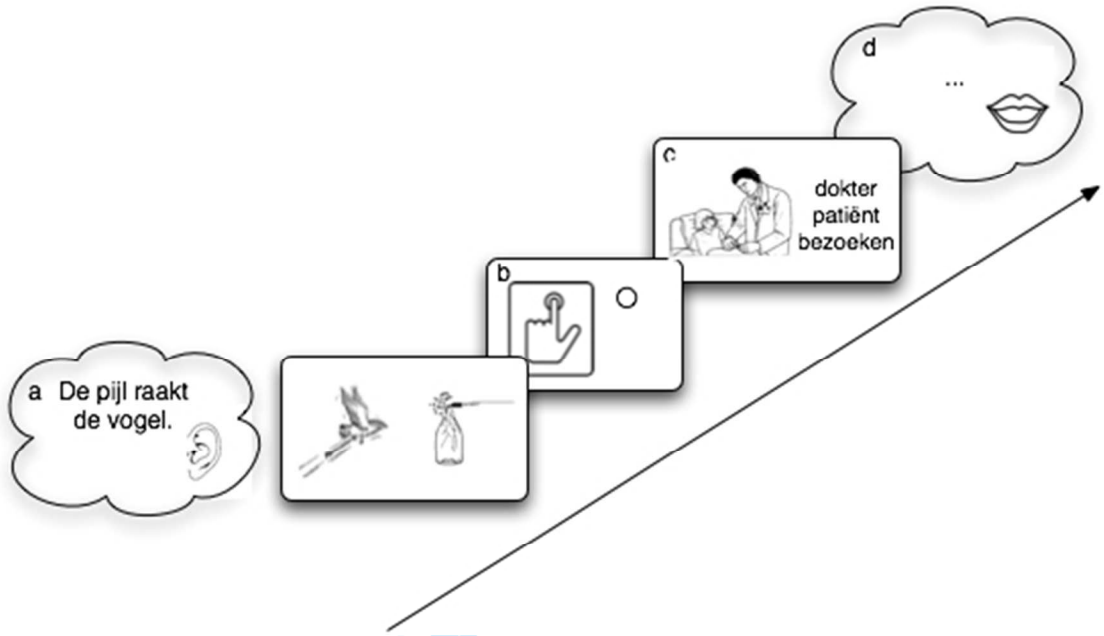


*Figure 3. Sequence of a trial as seen by the participant in the dialogue experiment; (a) participants hear a sentence and see two pictures; (b) participants indicate which picture matches the sentence they just heard (= **verification task**); (c) participants see a picture to describe; (d) participants describe the pictures.*

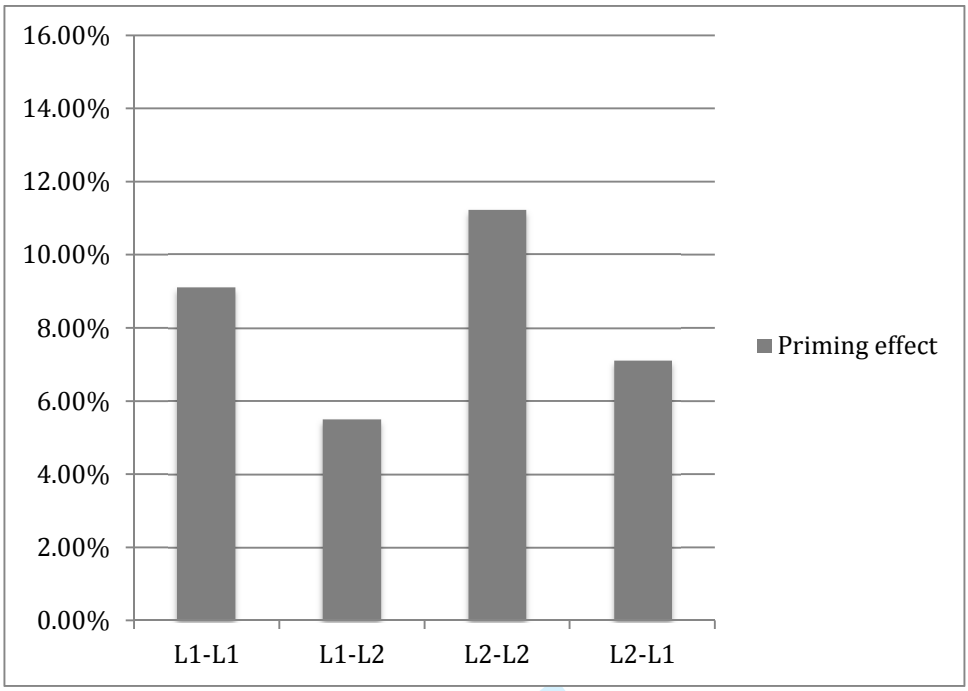


Figure 4. The size of the priming effect in the four language conditions for the control subjects. The priming effects are calculated as the difference between (amount of passive targets following a passive prime) minus (amount of passive targets following an active prime) (Pickering & Branigan, 1998).

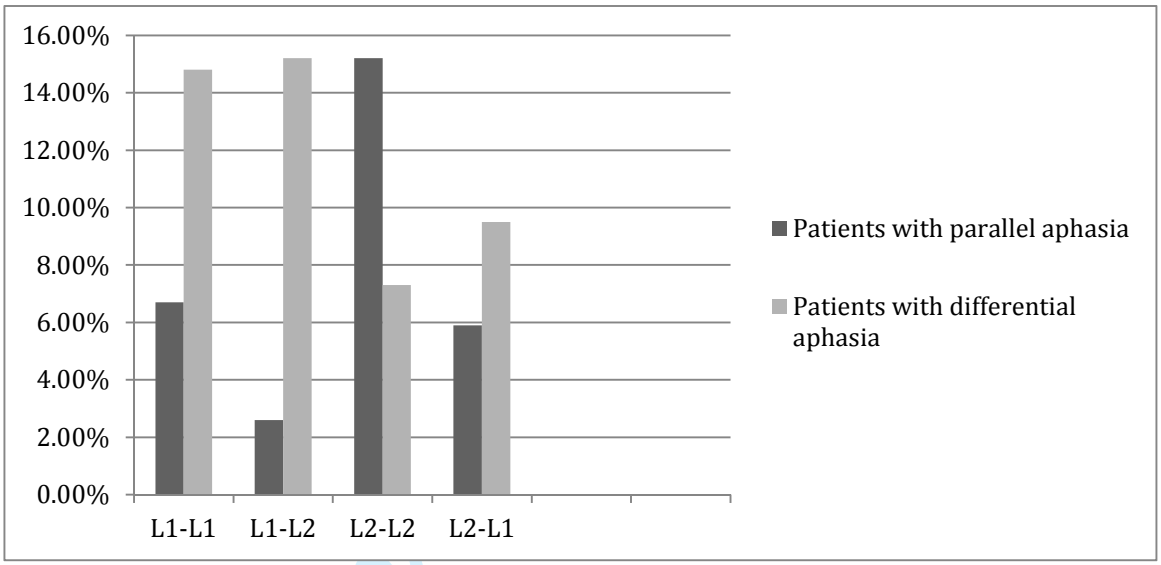


Figure 5. Size of the syntactic priming effect in the group of patients with parallel and differential aphasia. The priming effects are calculated as the difference between (amount of passive targets following a passive prime) minus (amount of passive targets following an active prime) (Pickering & Branigan, 1998).

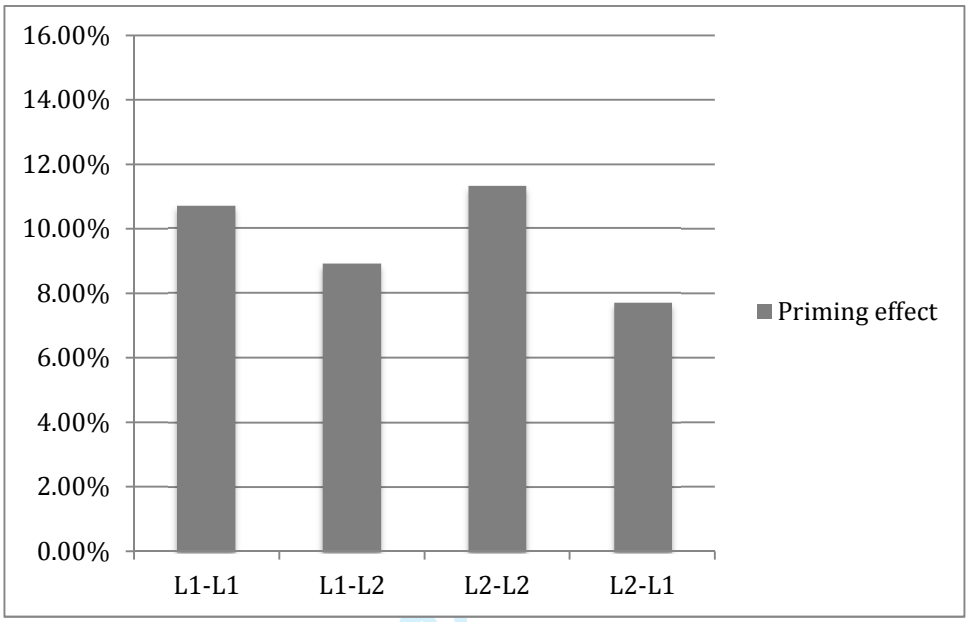


Figure 6. Size of the syntactic priming effect in the group of patients. The indicated values reflect the averages across type of aphasia (patients with parallel and patients with differential aphasia). The priming effects are calculated as the difference between (amount of passive targets following a passive prime) minus (amount of passive targets following an active prime) (Pickering & Branigan, 1998).

Recovery pattern	Description	References
Parallel recovery	Both languages recover with the same speed and/or to the same extent.	(Marangolo, Rizzi, Peran, Piras, & Sabatini, 2009)
Differential recovery	Recovery is more pronounced in one language compared to the other, the recovery in both languages differs qualitatively.	(Aglioti & Fabbro, 1993; Goral, Levy, Obler, & Cohen, 2006; Meinzer, Obleser, Flaisch, Eulitz, & Rockstroh, 2007)
Selective recovery	One language does not recover at all.	
Successive recovery	One language only starts to recover when the other one has fully recovered.	
Antagonistic recovery	An alternation in the recovery of both languages.	
Blended recovery	Patients uncontrollably switch and mix their languages during recovery.	(Adrover-Roig et al., 2011; Fabbro, Skrap, & Aglioti, 2000; Leemann, Laganaro, Schwitter, & Schnider, 2007; Marien, Abutalebi, Engelborghs, & De Deyn, 2005; Riccardi, Fabbro, & Obler, 2004)

Table 1. Recovery patterns described by Paradis (2004).

	Control group (n=19)	Patient group (n=6)	Group difference
Age (years)	55.58 (12.38)	59.17 (16.70)	$t_{(23)}$ = .570, p> .574
Sex (m/f)	4/15	1/5	$\chi^2_{(1)}$= .055, p> .815
Education (years)	15.11 (2.49)	14.17 (3.13)	$t_{(23)}$ = -.758, p> .456
L1 proficiency (pre-onset)	5.00 (0.0)	5.00 (0.0)	Ns.
L2 proficiency (pre-onset)	3.81 (0.76)	3.72 (0.53)	$t_{(23)}$ = -.243, p> .81

Table 2. Demographic data of the participant groups; L1 and L2 proficiency was rated on a 5-point Likert scale, for speaking, reading and writing.

Subject	Age	Sex	Bilingualism (L1-L2)	(previous) profession	Aetiology
Patients with differential aphasia					
N.D.	41	F	Dutch-French	Geriatric nurse	Ischemia in left MCA area
H.D.M.	77	M	French-Dutch	Technical Engineer	Hemorrhage in left thalamus
D.J.	53	F	Dutch-French	Lawyer	Ischemia in left posterior MCA area
Patients with parallel aphasia					
J.C.	80	F	Dutch-French	Housewife	Ischemia in left posterior MCA area
K.H.	45	F	Dutch-French	Secretary	Hemorrhage left frontal area
I.T.	59	F	Dutch-French	Secretary	Hemorrhage left parieto-temporal

Table 3. Demographic data of the patients; all patients are early, balanced bilinguals; L1 and L2 are based on Age of Acquisition; MCA=Middle Cerebral Artery.

Subject	AAT			Naming	Aphasia Pattern	BAT – part C	
	Subtest (max. score)	L1 (%ile)	L2 (%ile)			L1-L2 (/5)	L2-L1 (/5)
N.D.	SS (30)	27	15	L1 > L2	Wernicke aphasia *Differential aphasia* (Dutch better preserved than French)	4	2
	TT (50)	7 (90)	31 (53)*				
	RE (150)	108 (52)	101 (44)				
	WL (90)	80 (83)	25 (27)*				
	NA (120)	**113 (88)**	**58 (36)***				
	LC (120)	98 (77)	84 (50)				
H.D.M.	SS (30)	26	19	L1 > L2	Amnestic aphasia *Differential aphasia* (French better preserved than Dutch)	5	4
	TT (50)	6 (91)	19 (71)*				
	RE (150)	146 (96)	143 (92)				
	WL (90)	66 (65)	69 (68)				
	NA (120)	**113 (88)**	**90 (67)***				
	LC (120)	100 (80)	94 (70)				
D.J.	SS (30)	22	14	L1 > L2	Broca aphasia *Differential aphasia* (Dutch better preserved than French)	5	5
	TT (50)	29 (57)	34 (48)				
	RE (150)	138 (84)	124 (69)				
	WL (90)	57 (56)	49 (49)				
	NA (120)	**111 (97)**	**82 (55)***				
	LC (120)	118 (99)	107 (91)				
J.C.	SS (30)	25	24	Ns.	Amnestic aphasia *Parallel aphasia*	5	5
	TT (50)	7 (90)	6 (91)				
	RE (150)	126 (72)	139 (85)				
	WL (90)	90 (100)	82 (87)				
	NA (120)	**110 (96)**	**102 (84)**				
	LC (120)	96 (73)	100 (80)				

K.H.	SS (30)	21	22	Ns.	Amnestic	5	5
	TT (50)	11 (84)	15 (77)		aphasia		
	RE (150)	148 (98)	146 (96)		*Parallel*		
	WL (90)	90 (100)	88 (97)		*aphasia*		
	NA (120)	**113 (98)**	**113 (98)**				
	LC (120)	102 (83)	95 (71)				
I.T.	SS (30)	18	13	Ns.	Wernicke	5	5
	TT (50)	50 (6)	50 (6)		aphasia		
	RE (150)	0 (1)	84 (31)		*Parallel*		
	WL (90)	6 (12)	47 (47)		*aphasia*		
	NA (120)	**30 (23)**	**24 (21)**				
	LC (120)	49 (8)	13 (1)				

Table 4. Patient scores and percentiles on Aachen Aphasia Test (AAT) in Dutch and French, difference in Naming scores, aphasia pattern and scores on the Bilingual Aphasia Test (BAT) Part C in both directions. SS = Spontaneous Speech, TT = Token Test, RE = Repetition, WL = Written Language, NA = Naming, LC = Language Comprehension (auditory and reading), (*) significant difference between the scores. Caveat: For the subtest "Token Test", the score reflects the amount of errors (so a higher score implies a lower performance); BAT-part C assesses recognition of translation equivalents.

		Control Group *(N=19)*		
Condition	**Prime**	**active targets (%)**	**passive targets (%)**	**priming effect (%)**
L1 – L1	A	90.0	10.0	9.1
	P	80.9	19.1	
L2 – L2	A	78.0	22.0	11.2
	P	66.8	33.2	
L1 – L2	A	81.8	18.2	5.5
	P	76.3	23.7	
L2 – L1	A	82.4	17.7	7.1
	P	75.2	24.8	

Table 5: Priming effects in the control subjects; "A" = Active prime, "P" = Passive prime; Priming effects are calculated as the difference between (amount of passive targets following a passive prime) minus (amount of passive targets following an active prime) (Pickering & Branigan, 1998).

		Patients with parallel aphasia N=3			Patients with differential aphasia N=3		
Condition	**Prime**	**active targets (%)**	**passive targets (%)**	**priming effect (%)**	**active targets (%)**	**passive targets (%)**	**priming effect (%)**
L1 – L1	A	85.7	14.4	6.7	84.2	15.8	14.8
	P	79.0	21.1		69.4	30.6	
L2 – L2	A	79.5	20.5	15.2	66.7	33.3	7.3
	P	64.3	35.7		59.4	40.6	
L1 – L2	A	66.7	33.3	2.6	82.9	17.1	15.2
	P	64.1	35.9		67.7	32.3	
L2 – L1	A	85.4	14.6	5.9	82.5	17.5	9.5
	P	79.5	20.5		73.0	27.0	

Table 6. Results of the priming experiment in the patient groups; "A" = Active prime, "P" = Passive prime. Priming effects are calculated as the difference between (amount of passive targets following a passive prime) minus (amount of passive targets following an active prime) (Pickering & Branigan, 1998).